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

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INTRODUCTION

Spindle afferents provide 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].

The velocity-dependent stretch reflexes (i.e., Ia afferents) in lengthening muscles (i.e., 'antagonist' in the single-joint system) can, if not modulated, disrupt or stop joint rotations induced by the shortening muscles (i.e., 'agonists' in the single-joint system) compromise movement accuracy [6-7].

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

GOAL

Investigate (i) how velocity-dependent stretch reflexes perturb limb movements in the general case of numerous multi-articular muscles, and (ii) whether modulation of velocity-dependent stretch reflex gain can mitigate these disruptions.

METHODS

I. Open-loop and closed-loop simulations with unmodulated reflex gain

We used a 25-Hill-type muscles computational model of *Rhesus Macaque* arm [8] to simulate (i) 1,100 open-loop movements with feedforward alpha-MN commands only (Fig. 1A) and (ii) their closed-loop simulation by adding unmodulated positive homonymous muscle velocity feedback (i.e., velocity-dependent stretch reflex) (Fig. 1B).

II. Closed-loop simulations with modulated reflex gain

We modulated stretch reflex gain as per **idealized α-γ co-activation** (Fig. 2A) and by **scaling the gain by the homologous α-drive to γ-MNs** via an α-MN collateral [9-13] (Fig. 2B)

Analysis

We quantified disruption (Fig. 3) to the open-loop movements endpoint trajectories (i.e., cumulative residual) and final position (i.e., terminal error) of the hand, and the effect on the disruptions when stretch reflex gain was modulated.

RESULTS

I. **Unmodulated velocity-dependent stretch** reflexes cause **large, variable disruptions** of the endpoint trajectories in **task-dependent ways**

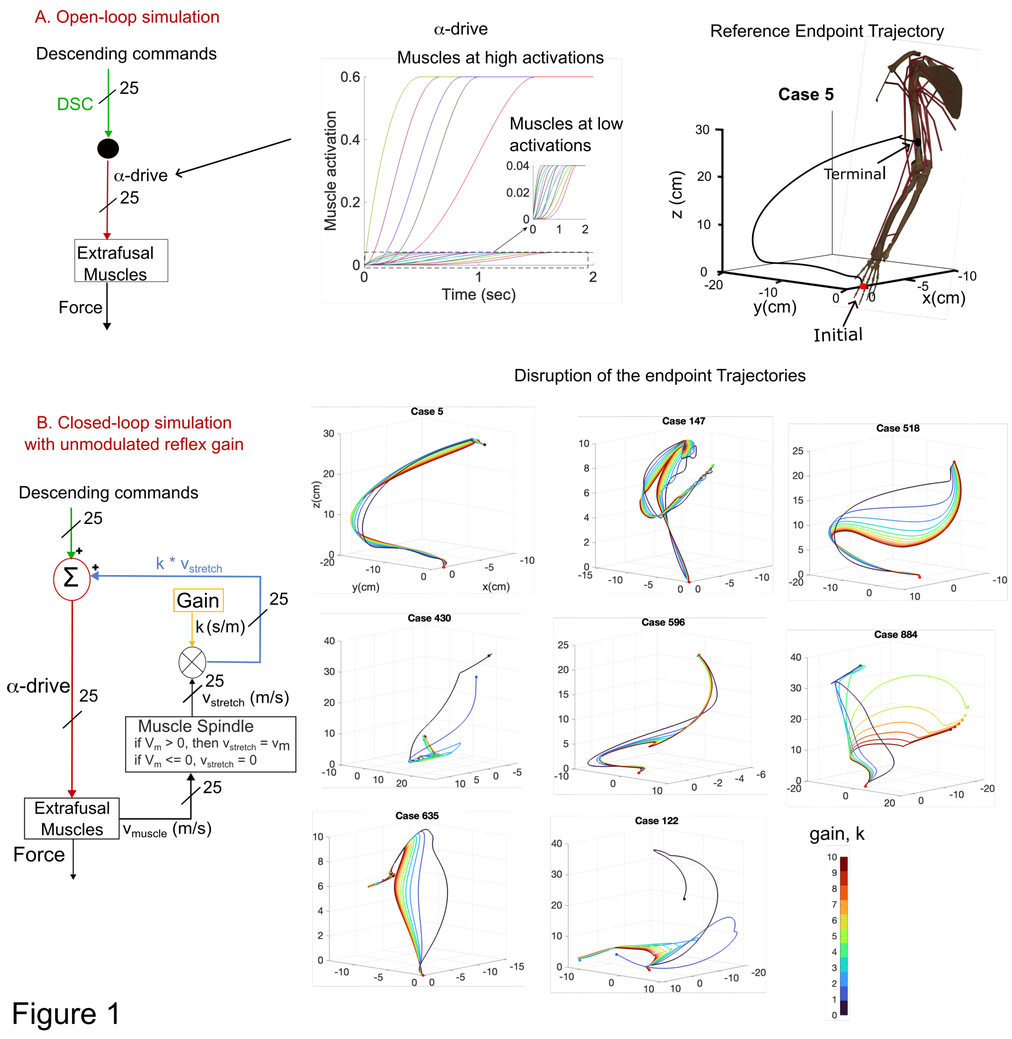

Figure 1

II. **idealized α-γ co-activation** and an **α-MN collateral to γ-MNs** can mitigate disruptions caused by the unmodulated velocity-dependent stretch reflexes

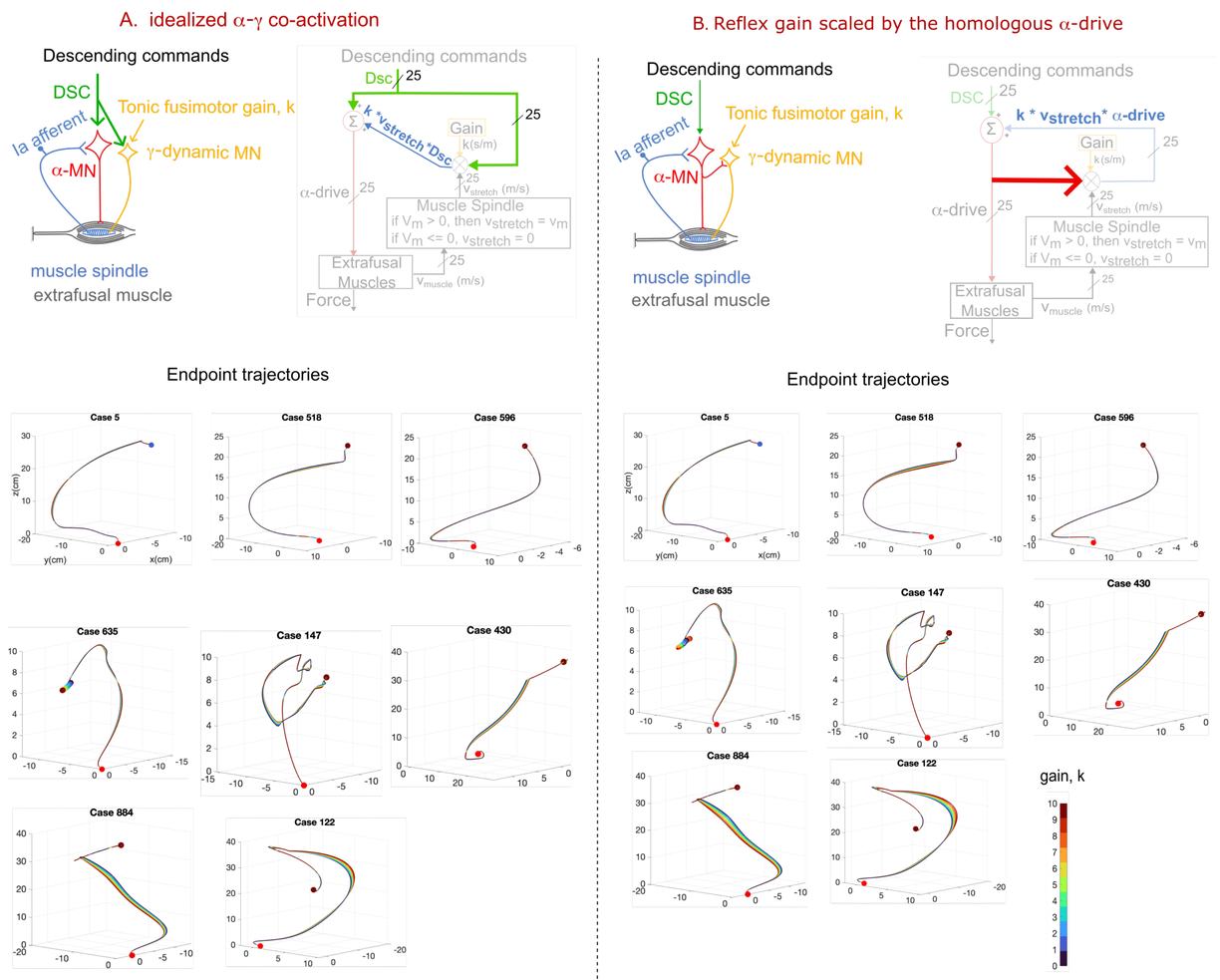

Figure 2

RESULTS (cont.).

III. Over all 1,100 arm movement, **the cumulative residual and terminal error** were large at higher reflex gains and **became significantly reduced** when modulating reflex gain as per **idealized α-γ co-activation** (B) and via **homologous α-MN collateral drive** (C). However, in a few movements (Cases. 705, 785, 1009, 827, 51, 159, 675) the disruptions were relatively larger.

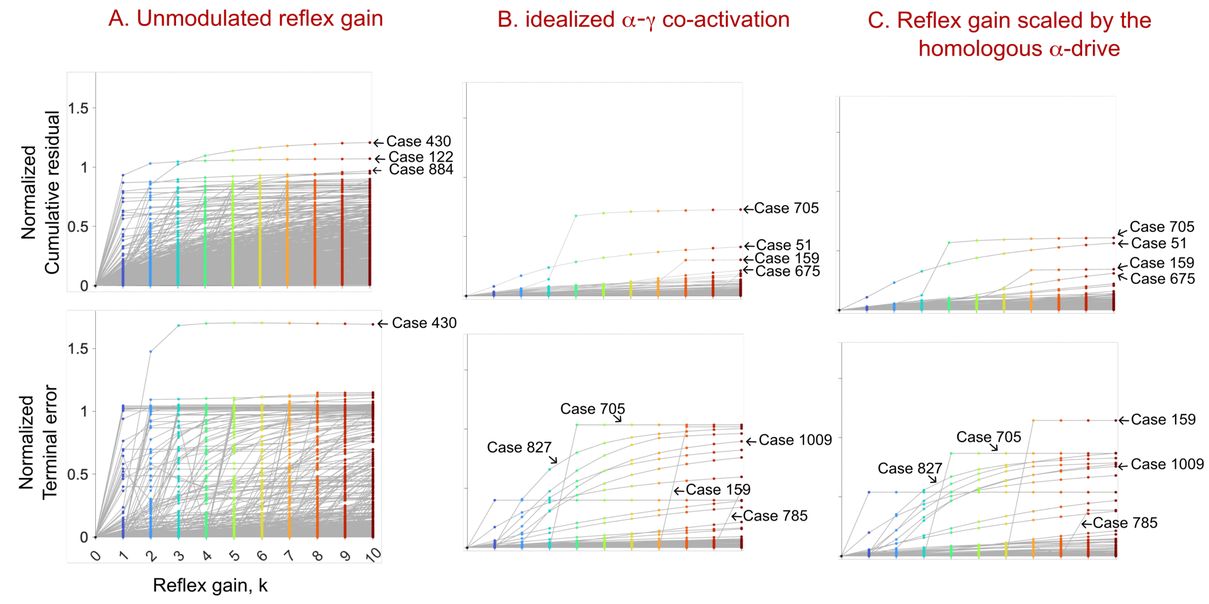

Figure 3

DISCUSSION

- Our results show that the disruptions of the movements caused by the velocity-dependent stretch reflexes are large, variable, and task-dependent enough to need inhibition, as has been proposed,but never quantified, by Sherrington [13] and others [7,14,15].

- Both idealized α-γ co-activation and an excitatory collateral from α-MN axon to γ-MNs can effectively mitigate the disruptions caused by unmodulated velocity-dependent stretch reflex.

- α-γ co-activation hinge on the assumption that the system has sufficiently accurate knowledge of the time-varying variables that determine musculotendon lengths and velocities. However, time delays and uncertainty can conspire to pollute such estimates if they rely on the supra-spinal processing of sensory signals to estimate body state or create appropriate motor actions.

- Our proposed mechanism simply scales velocity-dependent stretch reflex by an excitatory α-MN collateral. Such collateral projection among MNs have long been observed in studies of the cat and mouse spinal cord [9-13]. This mechanism is an alternative to α-γ co-activation that is evolutionary and physiologically plausible at the level of a homologous α-to-γ collateral.

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