Disruptions of voluntary movement by velocity-dependent stretch reflexes can vary greatly within and across movements: Implications to sensorimotor control

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The primary motor cortex does not uniquely or directly produce the alpha drive (α-drive) activations to arm muscles during voluntary movement. Rather, α-drive activations emerge from the synthesis of signals from multiple descending tracts, fusimotor gains, sensory inputs, and spinal interneurons. This includes the inhibition of velocity-dependent stretch reflexes provided by the primary (Ia) muscle spindle sensory signals in eccentrically contracting muscles, which otherwise would disrupt voluntary movement [1,2]. I.e., *"Inhibition is as important as excitation."* Spinal circuits for such homonymous (from the same muscle) and reciprocal (from other muscles) inhibition of stretch reflexes have been proposed mostly for the special (and experimentally tractable) case of single joints with clear agonist and antagonist muscles [3,4], but require careful real-time balance of fusimotor (gamma static (γ-static) and gamma dynamic (γ-dynamic) motoneurons) gains [5,6] in ways that are not entirely understood and are likely difficult to conclusively establish experimentally. Importantly, these circuits for the mono-articular case [3,4] are not guaranteed to generalize to the natural condition of numerous muscles crossing multiple joints during the production of large and fast 3-dimensional limb movements in the presence of gravity.

We address this long-standing problem of inhibition of stretch reflexes *during voluntary movement* first posed by Sherrington [1] by extending a computational model of the Rhesus Macaque (*Macaca mulatta*) [7] to include simulated monosynaptic velocity-dependent stretch reflexes for all its 25 Hill-type muscles. In each movement, five randomly-selected muscles were activated from zero to 60 % of maximum, while the remaining 20 muscles reached only 4% of maximum in a similar way (fig.1A). This distribution of high and low activations mitigated co-contraction and enabled both small and large arm movements. We first generated random sets of open-loop (i.e., baseline) s-shaped α-drive time histories lasting 2 s to produce 1,100 point-to-point movements, holding at a terminal position, each starting from the same reference posture, and exploring the workspace of the arm (fig.1B). We then evaluated the perturbation of each trajectory by velocity-dependent stretch reflexes that disrupt the baseline activation of any eccentrically contracting muscle (fig.2). The gain of the stretch reflex was chosen to be compatible with available data [8], and we conservatively excluded conduction delays to quantify best-case scenario disruptions.

We found that velocity-dependent stretch reflexes produced varied small and large disruptions in both movement trajectory and terminal position (fig.3 cases 5 & 518). Moreover, increasing the gain of the stretch reflex did not always produce monotonic disruptions, and could even provoke changes in the direction and endpoint of the trajectory (fig.3 cases 884 & 122). While there are limitations to this study such as the exclusion of γ-static drive (we assume its keeps spindles from going slack) and the choice of muscle coordination patterns (we avoid a particular muscle coordination strategy to highlight the generality of this approach), they do not challenge our main message that disruptions of voluntary movement by velocity-dependent stretch reflexes can vary greatly within and across movements.

These results are, to our knowledge, the first to quantify the effects of velocity-dependent stretch reflexes for the natural condition of numerous muscles crossing multiple joints during the production of large limb movements. The case-dependent effects of velocity-dependent stretch reflexes motivate and direct a rigorous neuromechanical approach to understand muscle afferentation during voluntary movement—which has often been taken for granted or ignored in the neuromuscular control literature [9]. In addition, these results provide a powerful conceptual foundation that may begin to explain the disruption of voluntary movement in neurological conditions, which has been historically attributed to disruption of γ drive or presynaptic inhibition of α-motoneurons of still uncertain or unknown cortical or subcortical origin [10,11]. Our preliminary extensions of this work also show that di-synaptic propriospinal mechanisms could mitigate these perturbations, and serve as a low-level mechanism to complement cortical, subcortical, cerebellar, and other propriospinal circuits to address the longstanding problem of the sensorimotor control of afferented muscles in health and disease.

References: [1] Sherrington, Q.J. Exp.Physiol,1913; [2] Sherrington, N. Foundation (Ed.), Nobel Lectures, Physiol. & Medicine, 1932; [3] Windhorst, Neurophysiol, 2022; [4] Pierrot,et al., CUP press, 2005; [5] Loeb, 1984; [6] Prochazka et al., 1985; [7] Chan and Moran, J. Neural Eng., 2006; [8] Krutky, et al., J. Neurophysiol, 2009; [9] Hagen, et al., J. Biomech, 2027; [10] Sanger, et.al, J. Neural. Eng., 2010; [11] Krakauer, MIT press, 2022.

I. Open-loop simulation of arm movement

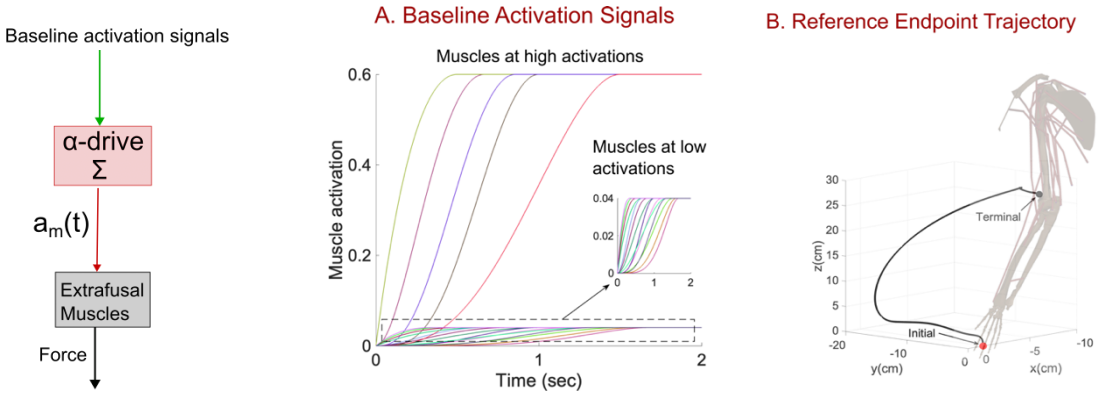

Figure 1: **Sample time history of open-loop α-motoneuron activations to muscles to produce large movements (case 5 out of 1,100).** (A) For each open-loop movement, five randomly-selected muscles were activated from zero to 60% of maximum following a random S-shaped activation profile (beta function clamped at its peak), while the remaining 20 muscles reached only 4% of maximum in a similar way (inset) to prevent excessive co-contraction and enable large movements throughout the workspace of the limb. (B) The ensuing reflex-free reference trajectory of the endpoint (distal head of the third metacarpal) for the sample activations in (A) is shown (black trace) from the starting position (red dot) to the terminal position (black dot).

II. Closed-loop simulation of arm movement

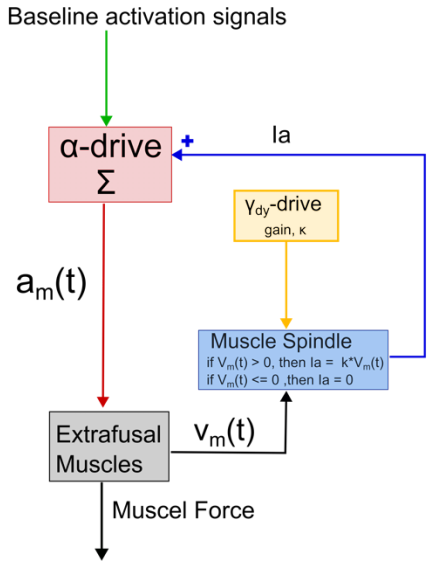

Figure 2: Schematic view of the closed-loop simulation of movements with velocity-dependent stretch reflex feedback.
The -drive to each muscle was the same as the baseline activation signal in open-loop simulation (fig.1a). Each of the 25 extrafusal muscles had a muscle spindle which received muscle velocities as inputs and generated Ia afferent (Ia) as follows. If the muscle is eccentrically lengthening (i.e., positive muscle velocities, V_m (t) > 0), the muscle velocity was multiplied by a gain k, from the gamma dynamic drive (d_y-drive). For each of the 1,100 movements we increased gain k from 1 to 10 in steps of 1.

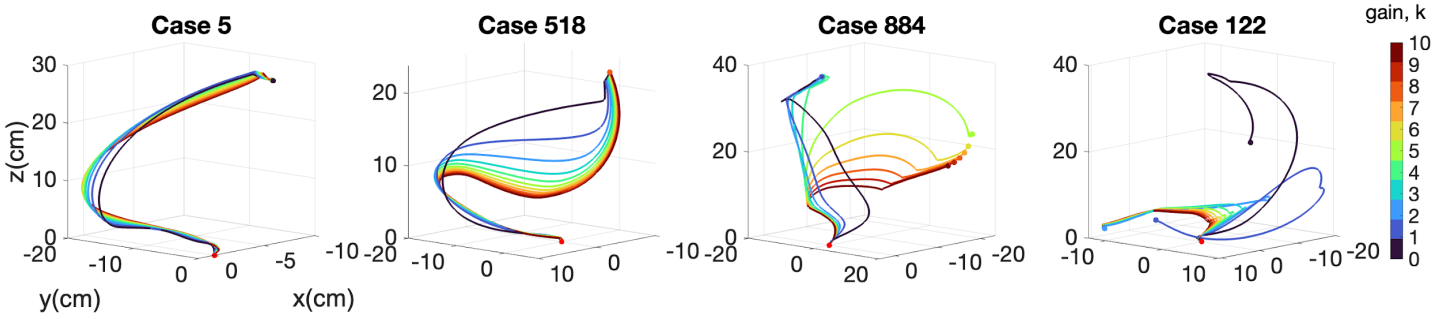

Figure 3: **Increasing the gain k of velocity-dependent stretch reflexes from 1 to 10 increases deviations in the movement trajectory and/or terminal position in a case-dependent way.** Four representative cases (out of 1,100) show that increasing the reflex gain progressively disrupts mostly the endpoint trajectory or both endpoint trajectory and terminal position. The open-loop baseline trajectories are shown in black.