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

Grace Niyo, Lama Almofeez, Andrew Erwin, Francisco J. Valero-Cuevas
University of Southern California

The primary motor cortex does not uniquely or directly produce alpha motoneurone (α-MN) drive to muscles during voluntary movement. Rather, α-MN drive emerges from the synthesis and competition among excitatory and inhibitory inputs from multiple descending tracts, spinal interneurons, sensory inputs, and proprioceptive afferents. One such fundamental input is velocity-dependent stretch reflexes in lengthening (antagonist) muscles, which are thought to be inhibited by the shortening (agonist) muscles. It remains an open question, however, the extent to which velocity-dependent stretch reflexes disrupt voluntary movement, and whether and how they are inhibited in limbs with numerous multi-articular muscles. We used a computational model of a Rhesus Macaque arm to simulate movements with feedforward α-MN commands only, and with added velocity-dependent stretch reflex feedback. We found that velocity-dependent stretch reflex caused movement-specific, typically large and variable disruptions to arm movements. These disruptions were greatly reduced when modulating fusimotor feedback as idealized α-γ co-activation or an α-MN collateral to homologous γ-MNs (which scaled the velocity-dependent stretch reflexes to its α-MN output). We conclude that such α-MN collaterals are a tenable, but previously unrecognized, propriospinal circuit in the mammalian fusimotor system. These collaterals could collaborate with the posited (but yet to be clarified) α-γ co-activation, and the few β-MNs in mammals, to create a flexible fusimotor ecosystem to enable voluntary movement. By locally and automatically regulating the highly nonlinear neuro-musculo-skeletal mechanics of the limb, this fusimotor ecosystem could be a critical low-level enabler of learning, adaptation, and performance via brainstem, cerebellar and cortical mechanisms.