How is voluntary movement disrupted in the presence of muscle afferentation?

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Multiple computational models of neuromuscular control include cortico-spinal drive as the primary (or only) command signal to a muscle. However, actual α-motoneuron activation results from summation of excitatory and inhibitory descending, propriospinal, sensory and proprioceptive synaptic inputs. Muscle spindles provide homologous and heteronomous proprioceptive inputs encoding muscle fiber length and velocity, while γ-motoneurons can modulate these spindle outputs, the extent to which the spindle feedback inputs alters limb kinematics is unknown. Here we model the functional effects of excitatory spindle afferent signals on limb kinematics to quantify whether and how the open loop descending cortico-spinal drive to α-motoneurons needs to be adjusted to counterbalance spindle afferent signals. Similar to (Hagen and Valero-Cuevas 2017), we used a 31-muscle Macaque arm model in MuJoCo and generated 100 open loop α-motoneuron commands that produced random free arm movements lasting 2 seconds starting from rest. We then systematically added excitatory monosynaptic spindle afferent to each muscle. We compared the baseline motion to the resulting disrupted trajectories and endpoint location after using five incremental feedback gains, proportional to the lengthening and eccentric velocity of each muscle. As expected, movements inducing greater fiber lengthening and eccentric velocities tended to be more disrupted as gain increased. But these trajectory and endpoint disruption were neither linear nor necessarily kinematically significant. Our findings highlight that each arm movement must have a distinct, nonlinear compensatory interaction between α and γ motoneuron drives, which can range from subtle to strong. Moreover, our conceptual approach to computational neuromuscular control and learning should be broadened to encompass dynamic muscle afferentation.

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