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TITLE: Exaggerated Modulation of Dorsiflexor MEPs During Plantarflexion Correlates with Gait Dysfunction Post-stroke

Purpose/Hypothesis: Plantarflexion is critical to gait, notably to produce forward propulsion, momentum, and limb advancement during swing. Plantarflexor power generation is impaired after stroke. Paradoxically stroke rehabilitation targets the dorsiflexors (DFs) in an effort to assure adequate foot clearance during walking. Here our goal was to determine how well stroke survivors modulate DF excitability during paretic limb plantarflexion (PF). We used transcranial magnetic stimulation (TMS) during isolated PF to test our hypotheses: [1] stroke survivors reveal less DF inhibition during PF than Controls, and [2] impaired DF inhibition is related to common measures of gait dysfunction.

Number of subjects: 20

Materials/methods: 12 stroke survivors (age 67 ± 10 , 5.8 ± 3.5 years post stroke) and 8 healthy controls (age 60 ± 10) performed both isometric and dynamic PF contractions. Participants held PF torque at 10-20% MVC for 1s, following which TMS was delivered at 120% of resting motor threshold. Motor evoked responses (MEPs) measured in medial gastrocnemius (MG), soleus (SOL), and tibialis anterior (TA) were normalized to background EMG and the change in MEP_{area} between isometric and dynamic conditions calculated. Self-selected (SSWS) and fastest comfortable (FCWS) walking speeds were measured for each participant. Clinical measures taken in stroke survivors included: lower extremity Fugl-Meyer Motor Assessment (FMA), Short Physical Performance Battery (SPPB), and Dynamic Gait Index (DGI).

Results: Controls reveal mild, but non-significant facilitation ($61.9\pm 24\%$, mean \pm SE) of TA MEPs in dynamic, relative to isometric, PF. As a group, stroke survivors revealed a wide range of responses. During PF movements stretch-mediated EMG, observed in the TA of Controls and 6 stroke survivors (SME+), was absent in 6 stroke survivors (SME-). The SME- group produced marked increases in TA MEP_{area} ($393.5\pm 119\%$, $p=0.02$), while the SME+ group ($87.4\pm 75\%$) was similar, to Controls. The change in TA MEP_{area} revealed a significant negative linear relationship with SPPB, DGI, FCWS, and most notably, SSWS ($R^2=0.459$, $p=0.001$).

Conclusions: A sub-population of stroke survivors experience exaggerated DF excitability during dynamic PF. The significant linear relationship between increased TA MEP_{area} and gait dysfunction suggests that DF over-activity may limit the amount of PF power that can be achieved in the affected limb during pre-swing following stroke. DF over-activity may result from either increased synaptic efficacy to the DF motoneuron pools or impairment of the reciprocal inhibition circuitry.

Clinical Relevance: SME- stroke survivors revealed slower walking speeds and greater motor impairment than either Controls or SME+ stroke survivors. These individuals appear unable to modulate DF activity during important PF tasks (e.g., during pre-swing) which could be a key contributor to decreased walking speed and clinical scores, especially tests specifically tuned to gait.

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