ABSTRACT
REFLEX GAIN EFFECTS ON FORCE REGULATION AND ITS CLINICAL IMPLICATIONS POST STROKE

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Marquette University, 2015

Amplified stretch reflex gain may contribute to limited force regulation and walking ability post stroke. Additionally, neuromuscular fatigue may exacerbate reflex gain, which could further limit walking endurance. This increased reflex gain may be caused by cortical disinhibition, which allows Ia afferents to amplify excitatory synaptic inputs to motoneuron pools and corresponding muscle fibers (motor units). This cortical disinhibition is presumably caused by stroke-related motor cortex damage. Although, other excitatory synaptic sources to motor units contribute to motor control, increased reflex gain may be one contributor that affects stroke survivors more than healthy controls. However, researchers infrequently report stroke-related reflex gain effects on force regulation. Our goal was to quantify stroke related stretch reflex gain with (out) a fatiguing condition and relate the findings to clinical function.

To investigate reflex gain effect on force regulation in a non-fatiguing condition, we examined stimulus frequency in the soleus H-reflex response of stroke survivors and healthy controls. The H-reflex is an electrical analog of the stretch reflex and gives insight into the monosynaptic sensory pathway. Stroke survivors had less H-reflex depression as compared to controls. Additionally, the slowest walking stroke survivors had the least amount of H-reflex depression.

We implicated amplified stretch reflex gain further by investigating patellar tendon tap (TT) responses with a fatiguing knee extensor task in stroke survivors and healthy controls. Additionally, we explored the contributions of voluntary muscle strength (MVC), neural drive (VA) and involuntary muscle property (RT) responses. Central mechanisms affect force regulation after fatigue because stroke survivors had less RT and MVC reduction and greater VA reduction as compared to controls. More specifically, increased reflex gain may contribute because stroke survivors had higher post TT responses and less change in TTs when compared to controls after fatigue. Furthermore, stroke survivors with fewer baseline central synaptic dysfunctions had less clinical leg impairments and were faster walkers.

These dissertation results indicate increased reflex gain as one contributing mechanism for abnormal motor control post stroke. Moreover, stroke survivors’ clinical and quantitative laboratory measures may be related, which can enhance the understanding of force regulation and, thus lead to optimized treatments.