MU-MCW Biomedical Engineering Department

Announcement of Public Dissertation Defense

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10:00 am
Engineering Hall, Room 323
Marquette University

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ABSTRACT

MECHANISMS OF IMPAIRED MOTOR UNIT FIRING BEHAVIOR IN THE VASTUS LATERALIS MUSCLE AFTER STROKE

The purpose of this dissertation research project was to examine the role of impaired motor unit firing behavior on force generation after a stroke. We studied the relationship between intrinsic motoneuron properties and inhibitory sensory pathways to deficient motoneuron activity in the vastus lateralis muscle after a stroke. Individuals with stroke often have deficits with force generation and volitional relaxation. Current models of impaired force output after a stroke focus primarily on the pathology within the motor cortex because of decreased descending drive. Though this is an important aspect for deficient motoneuron output, it is incomplete because motoneurons receive other inputs that can shape motor output. Because the motoneuron is the last site of signal integration for muscle contractions, using methods that study motor unit activity can provide a window to the activity in the spinal circuitry.

This research study utilized a novel algorithm that decomposed electromyography (EMG) signals into the contributions of the individual motor units. This provided the individual firing instances for a large number of concurrently active motor units during isometric contractions of the knee extensor muscles. In the first aim, the association between the hyperemic response and motor unit firing rate modulation to intermittent, fatiguing contractions was investigated. It was found that the magnitude of blood flow was lower for individuals with stroke compared to healthy controls, but both groups increased blood flow similarly in response to fatiguing contractions. This did not relate to changes in muscle fiber contractibility for the participants with stroke; rather, participants better able to increase blood flow showed greater modulation in motor unit firing rates. To further investigate how ischemic conditions impact motor unit output, the second aim used a blood pressure cuff to completely occlude blood flow through femoral artery with the intent of activating inhibitory afferent pathways. We found that ischemic conditions had a greater inhibitory effect on motor unit output for individuals with stroke compared to healthy controls, likely because of hyper-excitable group III/IV afferent pathways. The final aim investigated how stroke related changes in the intrinsic excitability of the motoneurons affected prolonged motor unit firing during voluntary relaxation. A serotonin reuptake inhibitor was administered to quantify motoneuron sensitivity to neuromodulatory inputs. This study found that the serotonin reuptake inhibitor reduced delays in muscle relaxation and may have reduced persistent inward current contributions to prolonged motor unit firing. In conclusion, while damage to the motor cortex is a major component to poor functionality, the intrinsic properties of the motoneuron and sensory pathways to the motoneuron pool are essential for understanding deficient motor control after a stroke.

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