

Joint Biomedical Engineering Seminar Series

MU ▪ MCW ▪ UWM

Friday, March 31, 2017
12:00 noon
Olin Engineering, Room 202

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ABSTRACT

MUSCLE COORDINATION CONTRIBUTES TO FUNCTION AFTER STROKE; PROPRIOCEPTION CONTRIBUTES TO CONTROL OF POSTURE, MOVEMENT

Each year, approximately 750,000 Americans experience new or recurrent stroke. Half of stroke survivors experience persistent motor impairment, including deficits in reaching. Reaching is an important behavior because it facilitates interaction with objects in the environment, thus contributing to quality of life and independence. Effective reaching involves coordination of agonist/antagonist muscle pairs, as well as coordination of multiple control actions for stabilizing and moving the arm.

In this dissertation, I present three studies in which I recorded isometric torque production, single joint flexion and stabilization, and clinical measures of function and impairments after stroke to evaluate the extent to which changes in coordination of muscles and control actions contribute to deficits after stroke.

In Aim 1, stroke participants (SP) and neurologically intact participants (NI) isometrically tracked step-change torque targets to investigate coordination between elbow agonist/antagonist muscle pairs. SP had marked hypertonia of the primary flexor muscle, which led to increased compensatory activity in the primary extensor muscle. These stroke-related deficits of muscle coordination degraded ability to generate, maintain, and relax cued torque production.

In Aim 2, SP and NI performed sequential combinations of elbow reach and stabilization tasks to investigate coordination between control actions contributing to stabilization and movement of the arm. Impaired proprioception in SP was associated with impairments in stabilizing the arm against a perturbation. Surprisingly, SP with intact proprioception had greater impairments in reaching than did SP with impaired proprioception. These results support the supposition that deficits of somatosensation can differentially impact neural control of limb stabilization and movement.

Aim 3 used correlation and forward regression to quantify the extent to which deficits of muscle coordination (Aim 1) and control (Aim 2) contribute to deficits of motor function after stroke. The amount of phasic muscle activation required to hold a moderate, steady torque was found to be inversely related to motor function after stroke.

Taken together, the three studies revealed that stroke-related deficits in coordination timing and magnitude of muscle activation impact clinically-measured function, and that somatosensory deficits can differentially impair neuromotor stabilization and movement control.

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