Long-term motor dysfunction in the lower limb is common after stroke. One potential contributor is motor compensation, a behavior in which functions originally performed by the paretic limb are performed by the non-paretic limb. Compensation in chronic stroke may contribute to long-term motor dysfunction by limiting functional ability, impairing future recovery, and eliciting maladaptive neuroplasticity. The purpose of this dissertation was to describe how compensation is related to motor function and brain activation during lower limb pedaling and identify elements that produce this behavior.

To achieve this purpose, we evaluated muscle activation and motor performance when compensation was prevented. During unilateral pedaling, paretic muscle activation increased but motor performance deteriorated. During bilateral uncoupled pedaling, paretic muscle activation further increased. However, subjects were unable to coordinate movements of the legs, and motor performance further deteriorated. These results suggest that compensation improves motor performance but limits paretic motor output. Because motor performance was worse during bilateral uncoupled than unilateral pedaling, impaired interlimb coordination may be a primary factor leading to compensation. As a follow-up, we determined whether altered interlimb spinal reflex pathways contribute to impaired interlimb coordination after stroke. Interlimb cutaneous reflexes were elicited during pedaling, and we assessed whether the amplitude or phasing were altered. Interlimb reflexes were altered and correlated with impairments in interlimb coordination. These data suggest that stroke-related changes in interlimb reflex pathways undermine interlimb coordination. Finally, we assessed whether altered motor commands and performance, such as seen with compensation, are related to decreased pedaling-related brain activation after stroke. Brain activation was measured during volitional pedaling and during passive pedaling, when between-group differences were minimized. Between-group differences in brain activation persisted during passive pedaling, suggesting that altered motor commands and pedaling performance do not account for reduced brain activation after stroke.

Overall, these studies provide insight into rehabilitative interventions that may decrease long-term motor dysfunction in the lower limb after stroke. One potential strategy is to enhance paretic muscle activity by preventing compensation while simultaneously employing efforts to improve interlimb coordination, possibly by manipulating interlimb reflex pathways.