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Effects of muscle strength and activation profile on foot drag in a simulated SCI rat

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Introduction

Spinal cord injury (SCI) can lead to decreases in overall motoneuron output, changes in muscle activation patterns, and atrophy of the affected muscles. Depending upon the severity of SCI, foot drag can occur during the stance-to-swing transition during locomotion. Our preliminary experimental data suggests that muscles co-activate following SCI, which may cause foot drag. To assess the relative roles of neural and muscular changes, a 3D neuro-musculoskeletal model of the rat hindlimb is being developed. Initially, a 2D, one degree-of-freedom ankle model with gastrocnemius (GAS) and tibialis anterior (TA) muscles was developed. Given that GAS can produce more ankle torque than TA, this simulation study tested the hypothesis that GAS motoneuronal deactivation profile dominates the ability of the foot to transition from stance to swing. Our results suggest that even small changes in the ability to precisely de-activate muscles could lead to kinematic deficits commonly observed following SCI.

Methods

Anatomical data collected from female Long-Evans rats, supplemented with data from literature, was used to implement the musculoskeletal model in SimMechanics/Simulink using Virtual Muscle [1]. Ankle and muscle dynamics were simulated using an imposed ankle trajectory from kinematic analysis of a normal rat walking on a treadmill. Relative GAS deactivation and TA activation onset times were varied within physiologically relevant ranges based on simplified locomotor electromyogram profiles. Three muscle strength (mass) parameter sets were analyzed: normal (100% each), moderate SCI [2] (100%TA, 80% GAS), and severe (50% each). Positive moments coinciding with the transition from stance-to-swing phase were defined as foot swing and negative moments as foot drag.

Results

As predicted, GAS deactivation dominated movement at the stance-to-swing transition. With normal muscle strength, GAS must be de-activated at least 0.2 sec before the TA onset to allow forward ankle rotation, preventing foot drag. Since GAS activity inhibits foot swing, the required transition delay was reduced in the moderate SCI condition. Under the severe condition only the magnitude of the moments was changed (Figure 1).

Discussion

Confirming our hypothesis, the musculoskeletal model revealed that movement dynamics are not equally sensitive to different aspects of neural output. Failure to precisely deactivate GAS could result in foot drag following SCI. Although muscle activation is an important contributor to movement dynamics, intrinsic muscle properties also substantially affected motor output. Future models will examine the role of the stretch reflex, as it is known to change after SCI, possibly affecting foot drag.

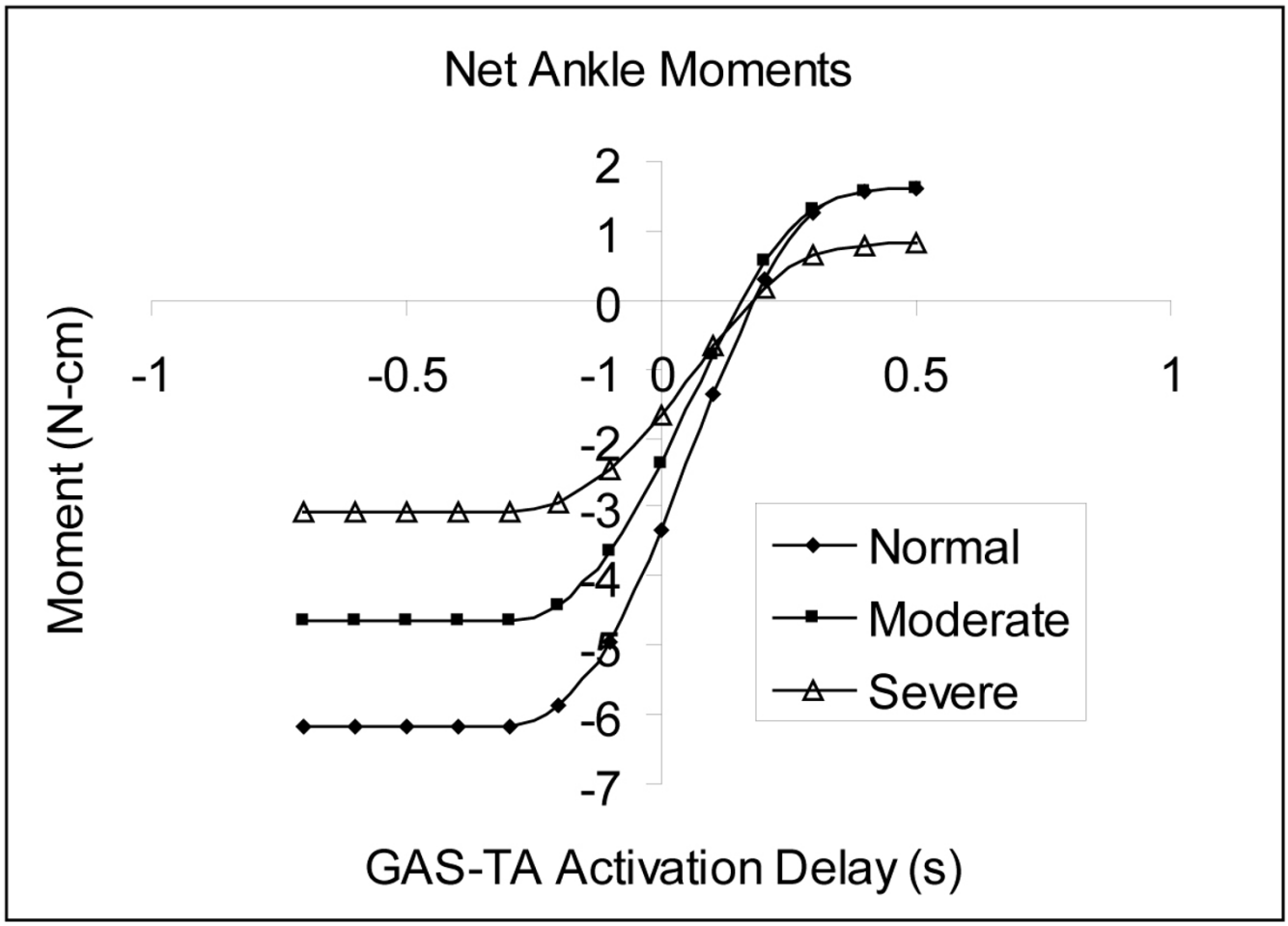

Figure 1
Maximum ankle moments at the stance-to-swing transition at different activation delays and muscle strengths.

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