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PLANTARFLEXOR WEAKNESS AND CONTRACTURE ALONE CAN CAUSE SIGNIFICANT KINEMATIC ADAPTATIONS DURING WALKING

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INTRODUCTION

Deficits in the ankle plantarflexor (PF) muscles, such as weakness and contracture, are commonly observed in conditions such as cerebral palsy and stroke. While these deficits likely contribute to observed gait pathologies, elucidating a cause-effect relationship is difficult due to the often co-occurring biomechanical and neural deficits. Physics-based musculoskeletal simulations in which kinematics are generated *de novo* can help to untangle these effects as deficits can be systematically introduced into the model. In this work, we used a simulation and optimization framework to train a musculoskeletal model to walk without impairments and with either PF weakness or contracture in order to study how each condition would affect the kinematics of the model's self-selected gait (Fig 1).

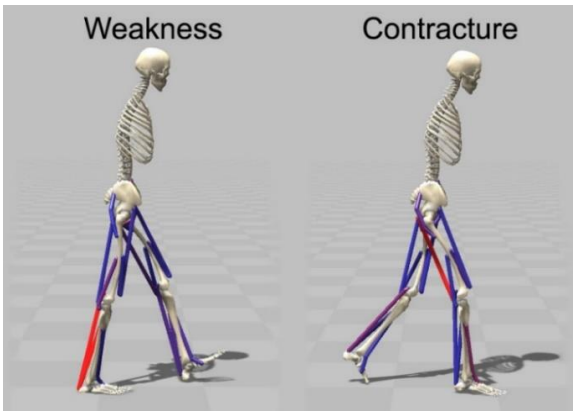

Fig 1: Our musculoskeletal models with PF weakness (left) and contracture (right) learning to walk in our simulation and optimization framework.

METHODS

We used a custom software package, SCONE (http://scone.software), to implement our gait controller and solve the single-shooting optimization problem. This package used OpenSim version 3.3 [1] to implement our musculoskeletal model and integrate the equations of motion to generate 30-second simulations.

We used a planar, 9-degree-of-freedom (dof) musculoskeletal model based on a model by Delp *et al*. [2]. The model included a 3-dof planar joint for the pelvis, and 1-dof hip, knee, and ankle joints for each leg. Each leg was actuated by 9 Hill-type musculotendon units with a compliant tendon, representing the major sagittal plane muscles. For our unimpaired model, we adjusted musculotendon parameters based on a model by Rajagopal *et al*. [3]. We modelled PF weakness by reducing the peak isometric forces of the soleus and gastrocnemius muscles to 6.25% of their unimpaired values, and we modelled PF contracture by reducing the optimal fiber lengths of the soleus and gastrocnemius muscles to 55% of their unimpaired values. Ligaments were modelled as variable stiffness springs that engaged during hyperextension and hyperflexion of the joints. Contact forces between the foot and the ground were computed using the Hunt-Crossley contact model [4], with contact spheres at the heels and toes to represent the foot, and a contact plane to represent the ground.

We modeled neural control using a series of state-based controllers based on the model developed by Geyer and Herr [5]. Briefly, this model uses a set of low-level controllers to calculate excitation for each muscle with a combination of constant signals, positive and negative feedback from the muscle length, velocity, and force, and proportional-derivative (PD) controllers to stabilize pelvis orientation. A high-level controller determines when the low-level controllers are active based on the phase of gait. In this work, the high-level controller transitioned between 5 phases of gait: early stance, mid-stance, terminal stance, swing, and landing preparation.

We assessed performance of a simulation using the following objective function, *J:*

$$J = J_{\mathrm{cot}} + w_{\mathrm{spd}} J_{\mathrm{spd}} + w_{\mathrm{inj}} J_{\mathrm{inj}} + w_{\mathrm{hd}} J_{\mathrm{hd}}.$$

This function sought to minimize the gross cost of transport (J_{cot}) while maintaining a minimum speed (J_{spd}), avoiding injury (J_{inj}), and stabilizing

the head (J_{hd}). We computed gross cost of transport, J_{cot}, by summing the basal and per-muscle metabolic rates [6, 7]. The speed penalty, J_{spd}, was applied if the speed averaged over a step was less than 0.75 m/s. This minimum speed value was set to encourage forward motion, but was low enough that the corresponding penalty term was inactive for any optimized solution. If the simulation was terminated early due to the model falling, the speed was set to 0 m/s for the remainder of the 30 seconds, yielding a large penalty. The injury penalty, J_{inj}, discouraged hyperextension or hyperflexion of the joints by penalizing ligament use. To promote head stability, J_{hd} penalized excessive accelerations of a point at the center of the head [8]. We manually adjusted weights (w_{spd}, w_{lig}, w_{hd}) to balance these competing objectives. We used the Covariance Matrix Adaptation Evolution Strategy algorithm [9] to solve for 90 design variables, which include the controller and initial pose parameters, that minimize our objective function, J.

RESULTS AND DISCUSSION

Our results support previous work that suggests PF weakness or contracture alone plays an integral role in adaptations seen in ankle kinematics [10, 11] (Fig 2). Our model with PF weakness adopted a calcaneal gait, characterized by excessive dorsiflexion throughout stance, and our model with PF contracture adopted an equinus gait, characterized by excessive plantarflexion throughout stance.

A commonly observed adaptation seen in gait pathologies is crouch gait. While it has been hypothesized that PF weakness may contribute to individuals adopting a crouch gait [12], our results did not support this notion as our model with PF weakness did not adopt a gait with increased knee flexion throughout stance. Conversely, our model with PF contracture did adopt a gait with increased knee flexion in stance, which suggests that joint contractures may contribute to crouch gait. Furthermore, researchers have previously observed that equinus and crouch gait can co-occur [13], and our simulations suggest that PF contracture alone may contribute to both of these gait adaptations.

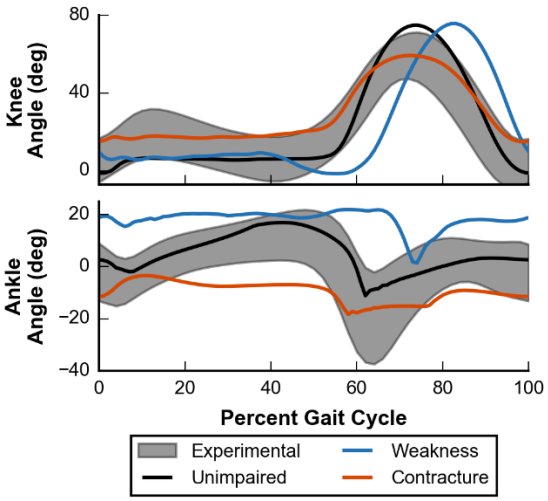

Fig 2: Knee (top) and ankle (bottom) kinematic trajectories of experimental (gray area) [14] and simulation (lines) cases. Three simulation cases are shown: unimpaired (black), PF weakness (blue), and PF contracture (orange)

CONCLUSIONS

We used a simulation and optimization framework to study the role of muscle weakness and contracture in gait adaptations. We found that with PF weakness, the model adopted a calcaneal gait without a crouch, and that with PF contracture, the model adopted an equinus gait with crouch. This work highlights how simulations can help us to better understand how specific deficits contribute to observed gait pathologies.

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