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Understanding compensatory strategies for muscle weakness during gait by simulating activation deficits seen post-stroke

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ABSTRACT

Musculoskeletal simulations have been used to explore compensatory strategies, but have focused on responses to simulated atrophy in a single muscle or muscle group. In a population such as stroke, however, impairments are seen in muscle activation across multiple muscle groups. The objective of this study was to identify available compensatory strategies for muscle weakness during gait by simulating activation deficits in multiple muscle groups. Three dimensional dynamics simulations were created from 10 healthy subjects (48.8 ± 13.3 years, self-selected speed 1.28 ± 0.17 m/s) and constraints were set on the activation capacity of the plantar flexor, dorsiflexor, and hamstrings muscle groups to simulate activation impairments seen post-stroke. When the muscle groups are impaired individually, the model requires that the plantar flexor, dorsiflexor, and hamstrings muscle groups are activated to at least 55%, 64%, and 18%, respectively, to recreate the subjects' normal gait pattern. The models were unable to recreate the normal gait pattern with simultaneous impairment of all three muscle groups. Other muscle groups are unable to assist the dorsiflexor muscles during early swing, which suggests that rehabilitation or assistive devices may be required to correct foot drop. By identifying how muscles can interact, clinicians may be able to develop specific strategies for using gait retraining and orthotic assistance to best address an individual's needs.

1. Introduction

Stroke is a leading cause of disability in the US. A common goal of stroke survivors is to regain community ambulation [1] and therefore, gait retraining is an important part of rehabilitation after stroke. Gait retraining often attempts to address abnormal gait kinematics [2] that are related to functional deficits such as decreased walking speed after stroke. Such abnormal gait patterns occur in part due to compensation strategies used by the patient in an attempt to make up for inadequate muscle function [2]. By identifying how muscles may adopt different activation patterns to achieve ambulation in response to muscle impairment, the specific gait deficits observed in individual stroke survivors may be more readily understood.

Musculoskeletal simulations have been used to explore possible compensatory strategies in response to reduced force capability of a single muscle [3]. Goldberg and Neptune (2007) used a forward dynamic simulation of normal walking to observe muscle compensations in response to plantar flexor, quadriceps, and hamstrings weakness over a gait cycle. This study highlighted the ability of the plantar flexor muscle group to compensate for weak hip and knee flexors and extensors. However, the model was unable to reproduce a normal walking pattern when the plantar flexor strength was reduced as a group. A study by Jonkers et al. examined the contributions of individual muscles during stance, concluding that a combination of multiple muscles is likely required to compensate for weakness in a single muscle [4]. A study by Steele et al. determined the minimum isometric force requirements for varying muscle groups in children with cerebral palsy during crouch gait by systematically reducing maximum isometric force of muscle groups [5]. Similarly, a recent study by van der Krogt et al. studied the ability of models to reproduce healthy gait in response to muscle weakness induced by a reduction in maximum isometric force in various muscle groups [6]. This study concluded that weakness of individual muscles results in increased activation of the weak muscle and an overall increase in total muscle activation and cost of walking.

Because these studies manipulate the model parameters corresponding to maximum isometric force, they indirectly explore the effects of atrophy in a single muscle or muscle group on gait, and on the body's ability to compensate with other muscles

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to maintain normal gait. In a stroke population, however, there is often significant impairment of muscle activation. This is an important distinction, as atrophy and activation failure have different roles in the force production capacity of muscle. With activation impairment, the passive forces of the muscle are not weakened, and force production can exceed that of a fully activated atrophied muscle. This is an important distinction when considering the level of activation required for a muscle to produce a given force, as passive forces require no volitional effort. No modeling studies have looked at the effects of reduced muscle force due to simulated muscle activation impairment.

For many musculoskeletal simulations, the problem of muscle redundancy is solved using a cost function that minimizes a function of muscle activation [7]. Thus, predicted muscle patterns may differ when weakness is emulated by reduced maximum isometric force compared to activation levels. Consider the case when muscle is weakened by reducing maximum isometric force by 50%, the optimizer would require 100% activation of the muscle to reach its maximum force potential. In contrast, a muscle which has a reduction in maximum activation, but preservation of its maximum isometric force, the optimizer would require only 50% activation force (even less, if the preservation of passive forces is also considered) to achieve the same maximum force. In this scenario, an optimizer that minimizes muscle activation would calculate a greater cost at the same force production for a muscle with reduced maximum isometric force compared to reduced maximum activation. Similarly, a cost function which minimizes muscle stress [6] (the ratio of force produced to maximum force) would also calculate a greater cost at the same force for a muscle with reduced maximum isometric force compared to reduced maximum activation.

The compensation strategies employed by patients with activation impairment may be different than those with muscle atrophy, particularly for neurologically impaired populations such as stroke, who often demonstrate preservation or enhancement of passive muscle forces [8] concurrent with activation impairments. Recently, Barber et al. demonstrated that young adults with spastic CP may have a greater ability to produce passive force through altered muscle-tendon properties. Consideration and preservation of passive properties in musculoskeletal simulations may be of increased importance when studying pathologic gait [9].

The objective of this study was to identify available compensatory strategies for muscle weakness during gait by simulating activation deficits in multiple muscle groups. We created three dimensional dynamic simulations from healthy walking data. The effects of activation impairment were explored by unilaterally constraining the activation of the plantar flexor, dorsiflexor, and hamstrings muscle groups in the simulations to emulate activation deficits seen post-stroke. Muscle groups were impaired individually in a first group of simulations, then simultaneously in a second set of simulations. Muscle coordination and function were compared between the healthy and impaired simulations to identify plausible compensation strategies. We hypothesized that with a combination of plantar flexor, dorsiflexor, and hamstrings impairment, the model would no longer be able to reproduce healthy gait.

2. Methods

A total of 10 healthy subjects (48.8 ± 13.3 years, self-selected speed 1.28 ± 0.17 m/s) participated in this study. All subjects read and signed an informed consent form approved by the Human Subjects Review Board at the University of Delaware. Data were collected on an instrumented split belt treadmill (Bertec Corp., 2000 Hz) using an 8 camera motion capture system (Motion Analysis Corp., 200 Hz) with the subjects walking at their self-selected speed.

A 23 degree of freedom model with 54 muscle actuators was used to generate a 3D, forward dynamic simulation based on motion capture walking trials from each subject [10]. Inverse kinematics and residual reduction analysis (RRA) were run on all models with the results of RRA within acceptable limits for simulations as outlined in the OpenSim User's Guide. Using Computed Muscle Control (CMC) [11], the muscle forces and activations required to reproduce normal gait kinematics and joint torques were computed. Constraints were set on the activation capacity of the plantar flexor (medial gastrocnemius, soleus, tibialis posterior), dorsiflexor (tibialis anterior), and the primary hamstrings (biceps femoris long and short head) muscle groups to simulate activation impairment seen post-stroke [12–16]. Each muscle group was progressively impaired individually and CMC was repeated at each level of impairment until the model could no longer generate the joint torque (within 5%) required to produce the subject's normal gait pattern, as quantified by the reserve actuator torque (Fig. 1). From this, the minimum level of activation required for each muscle group was determined through trial and error refinement with a minimum step of 0.5% activation. CMC was run a final time to re-optimize muscle controls with the imposed muscle activation limitations. The final calculated set of muscle activations, forces and resulting kinematics were analyzed to determine compensation strategies.

To quantify the changes in muscle activation, we calculated percent activation as the average activation of a muscle over a period of time. This value was computed during the second double support phase of gait while the limb of interest is the trailing limb for the plantar flexors, first half of swing for the dorsiflexors, and first half of stance for the hamstrings. Paired *t*-tests were used to compare the average percent activation between the normal and impaired simulations for each muscle in the model.

A muscle force perturbation was performed to quantify individual muscle contribution to joint and center of mass accelerations [17]. This was done for the plantar flexors during the second double support, in light of recent studies that have highlighted the importance of plantar flexor function in post-stroke gait [16,18], and the dorsiflexors during the first half of the swing phase of gait, to examine the effect of impaired function on foot drop. The accelerations induced by each muscle were averaged over the gait phase of interest. Paired *t*-tests were used to assess significant ($p < 0.05$) differences for each muscle between the normal and impaired simulations.

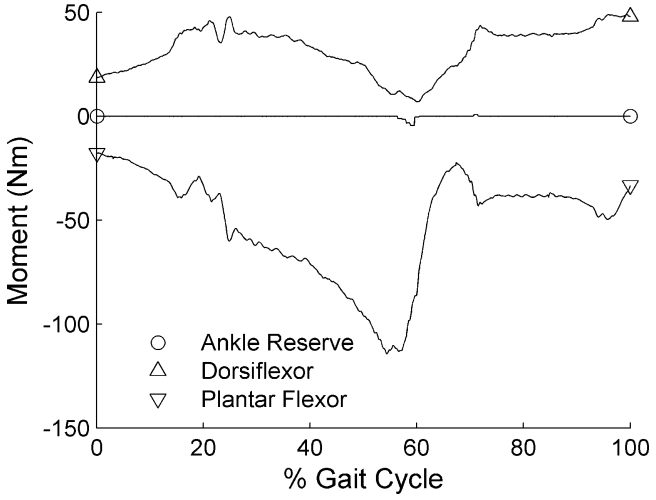

Fig. 1. Plantar flexor, dorsiflexor and ankle reserve moments for a representative subject at the greatest level of plantar flexor impairment allowed by study criteria. Ankle reserve moment reaches ~5% of plantar flexor moment just prior to 60% of the gait cycle.