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Muscle contributions to vertical and fore-aft accelerations are altered in subjects with crouch gait

Katherine M. Steele [a], Ajay Seth [b], Jennifer L. Hicks [b], Michael H. Schwartz [c,d], Scott L. Delp [a,b,*]

[a] Department of Mechanical Engineering, Stanford University, Stanford, CA, USA
[b] Department of Bioengineering, Stanford University, Stanford, CA, USA
[c] The James R. Gage Center for Gait and Motion Analysis, Gillette Children's Specialty Healthcare, St. Paul, MN, USA
[d] Departments of Orthopaedic Surgery and Biomedical Engineering, University of Minnesota, Minneapolis, MN, USA

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ABSTRACT

The goals of this study were to determine if the muscle contributions to vertical and fore-aft acceleration of the mass center differ between crouch gait and unimpaired gait and if these muscle contributions change with crouch severity. Examining muscle contributions to mass center acceleration provides insight into the roles of individual muscles during gait and can provide guidance for treatment planning. We calculated vertical and fore-aft accelerations using musculoskeletal simulations of typically developing children and children with cerebral palsy and crouch gait. Analysis of these simulations revealed that during unimpaired gait the quadriceps produce large upward and backward accelerations during early stance, whereas the ankle plantarflexors produce large upward and forward accelerations later in stance. In contrast, during crouch gait, the quadriceps and ankle plantarflexors produce large, opposing fore-aft accelerations throughout stance. The quadriceps force required to accelerate the mass center upward was significantly larger in crouch gait than in unimpaired gait and increased with crouch severity. The gluteus medius accelerated the mass center upward during midstance in unimpaired gait; however, during crouch gait the upward acceleration produced by the gluteus medius was significantly reduced. During unimpaired gait the quadriceps and ankle plantarflexors accelerate the mass center at different times, efficiently modulating fore-aft accelerations. However, during crouch gait, the quadriceps and ankle plantarflexors produce fore-aft accelerations at the same time and the opposing fore-aft accelerations generated by these muscles contribute to the inefficiency of crouch gait.

1. Introduction

Many individuals with cerebral palsy walk in a crouch gait pattern characterized by excess hip and knee flexion [1]. Walking in a crouched posture can lead to joint pain [2] and bone deformities [3] and is inefficient [4,5]. Understanding how muscles accelerate the mass center during crouch gait may provide insight about the underlying mechanics and inefficiencies associated with this gait pattern.

Previous studies have documented how muscles accelerate the mass center during unimpaired gait [6–8] and have shown that the same muscles that accelerate the mass center upward also modulate fore-aft acceleration [9]. Examining muscle contributions to mass center acceleration can provide insight into the role of individual muscles and the control strategy used during gait. During unimpaired gait, the vasti and gluteus maximus accelerate the mass center upward and backward in early stance, the gluteus medius accelerates the mass center upward in mid stance, and the ankle plantarflexors accelerate the mass center upward and forward in late stance [6–8]. This coordinated muscle activity uses different muscle groups at different periods of stance to efficiently modulate vertical and fore-aft mass center accelerations.

How muscles modulate vertical and fore-aft accelerations of the mass center during crouch gait and how these accelerations change with crouch severity is not well understood. Previous work has shown that similar muscles accelerate the mass center upward during the single-limb stance phase of both mild crouch gait and unimpaired gait [10]. However, the role of muscles during the double support phase, when fore-aft accelerations are largest, is not known for crouch gait. The ability of muscles such as the gluteus medius and soleus to extend the hip and knee [11] and accelerate the mass center upward [12] has been shown to decrease in a crouched posture; however, the effect of crouch severity on muscle contributions to mass center acceleration has not been investigated. Alterations in the ability of muscles to

* Corresponding author at: Clark Center, Room S-321, Stanford University, Mail Code 5450, 318 Campus Drive, Stanford, CA 94305-5450, USA.
Tel.: +1 650 723 1230; fax: +1 650 723 8544.
E-mail address: delp@stanford.edu (S.L. Delp).

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generate vertical and fore-aft accelerations of the mass center may change the strategy used to accelerate the mass center during crouch gait and contribute to the in efficiency of this gait pattern.

The goals of this study were to determine whether: (1) the magnitude and timing of muscle contributions to vertical and fore-aft accelerations differ between crouch gait and unimpaired gait, and (2) the muscle contributions to vertical and fore-aft accelerations change with crouch severity. To calculate muscle contributions to mass center accelerations we created three-dimensional musculoskeletal simulations of gait for typically developing children and children with cerebral palsy and varying degrees of crouch gait (Fig. 1). Examining how individual muscles contribute to vertical and fore-aft accelerations can elucidate the mechanics of crouch gait and inform treatment strategies.

2. Methods

Nine children with cerebral palsy were selected from a database of subjects who had undergone motion analysis at Gillette Children's Specialty Healthcare. The selection criteria for the subjects included: (1) a diagnosis of spastic diplegic cerebral palsy, (2) a minimum knee flexion angle during stance greater than 15°, and (3) a tibial and femoral torsion deformity less than 30° [13]. We also required that the subjects did not use an assistive device during the motion analysis and had at least two consecutive force plate strikes. Nine subjects with crouch gait were divided into three groups based on minimum knee flexion angle during stance: 15–30° knee flexion was defined as mild crouch gait, 30–50° was defined as moderate crouch gait, and 50° or larger was defined as severe crouch gait (Table 1).

The typically developing children were selected from a group of subjects who also visited Gillette Children's Specialty Healthcare for motion analysis and whose gait has been previously simulated [7]. Previous studies demonstrated consistent contributions to vertical and fore-aft accelerations during gait in typically developing children; thus, we chose three subjects with an age range similar to the subjects with crouch gait (Table 1).

Motion analysis data was collected using a 12-camera system (Vicon Motion Systems, Lake Forest, CA) and a standard marker measurement protocol [14]. Ground reaction forces were measured with four force plates (AMTI, Watertown, MA). All subjects walked barefoot at their self-selected speed. Surface electromyography (EMG) was recorded from the rectus femoris, hamstrings, gastrocnemius, and tibialis anterior (Motion Laboratory Systems, Baton Rouge, LA) for nine of the subjects. The nine subjects who had EMG data available included three typically developing subjects, three mild crouch gait subjects, one moderate crouch gait subject, and two severe crouch gait subjects. The EMG signals were sampled at 1080 Hz, band-pass filtered between 20 and 400 Hz, rectified, and low-pass filtered at 10 Hz. The magnitude of the EMG signal was normalized for each muscle group from zero to one based on the minimum and maximum values observed during the motion analysis. Since EMG was not available for all subjects, we averaged the processed EMG signals for each group of subjects for comparison to simulated muscle activations.

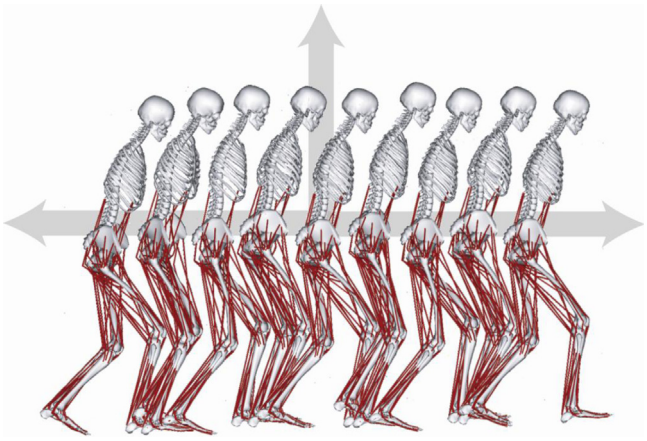

Fig. 1. Musculoskeletal model of an individual with cerebral palsy and crouch gait. Vertical and fore-aft accelerations of the mass center were calculated by analyzing muscle-driven simulations.

Table 1
Subject characteristics (average ± standard deviation).

	N	*N* (with EMG)	Age (years)	Height (cm)	Mass (kg)	KFA[a] (°)
Unimpaired	3	3	10 ± 3	144 ± 16	36 ± 9	−2 ± 4
Mild crouch	3	3	9 ± 1	124 ± 10	24 ± 4	18 ± 2
Moderate crouch	3	1	11 ± 2	136 ± 6	43 ± 31	34 ± 2
Severe crouch	3	2	14 ± 2	157 ± 12	41 ± 8	64 ± 20

[a] KFA: minimum knee flexion angle during stance.

To create dynamic musculoskeletal simulations of gait, we used a musculoskeletal model with 19 degrees of freedom and 92 musculotendon actuators (lower extremities from Ref. [15] and torso from Ref. [16]). The degrees of freedom in the model included three translations and three rotations of the pelvis, a ball-and-socket joint between the pelvis and the torso located at the third lumbar vertebrae, ball-and-socket joints at each hip, a custom joint with coupled translations and rotations at each knee, and a revolute joint at each ankle. This model has previously been used to model typically developing children [7,9] and children with cerebral palsy [10,11,17]. The model was scaled according to anthropometric measurements for each subject using OpenSim [18]. We scaled the maximum isometric force of all muscles by height-squared [19].

Inverse kinematics, which minimizes the difference between experimental marker trajectories and markers placed on the model, was used to calculate joint angles (Fig. 2A). Joint moments were calculated for each subject using inverse dynamics (Fig. 2B).

A dynamic simulation of one gait cycle was generated for each subject. The residual reduction algorithm (RRA) was used to reduce residuals at the pelvis [18]. Residuals are non-physiological forces and moments applied at the pelvis that balance dynamic inconsistencies resulting from experimental errors and modeling assumptions. RRA reduced these residuals by making small changes to measurements that may have experimental error including the position of the torso mass center (changes were less than 2 cm in magnitude) and the kinematics (changes were less than 2° for all joint angles). Using the adjusted model and kinematics determined from RRA, the computed muscle control algorithm (CMC) was used to estimate the muscle forces required to track each subject's kinematics [20,21]. At each time step, CMC determines the distribution of muscle activations, such that model accelerations match the experimental accelerations for all degrees-of-freedom. The algorithm accounts for muscle activation and contraction dynamics, which includes the muscle force–length–velocity relationship. The distribution of muscle activations was determined by minimizing the sum of squared activations at each time step. The estimated muscle activations from CMC were qualitatively compared to the average EMG signal for each gait pattern; constraints on muscle excitations were used when the simulated muscle activity was inconsistent with the EMG signals.

An induced acceleration analysis was used to compute the contributions of individual muscles to vertical and fore-aft mass center accelerations [6,22–24] at each time point of a subject's gait simulation. This analysis solves the model's equations of motion,