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Muscle contributions to support and progression during single-limb stance in crouch gait

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ABSTRACT

Pathological movement patterns like crouch gait are characterized by abnormal kinematics and muscle activations that alter how muscles support the body weight during walking. Individual muscles are often the target of interventions to improve crouch gait, yet the roles of individual muscles during crouch gait remain unknown. The goal of this study was to examine how muscles contribute to mass center accelerations and joint angular accelerations during single-limb stance in crouch gait, and compare these contributions to unimpaired gait. Subject-specific dynamic simulations were created for ten children who walked in a mild crouch gait and had no previous surgeries. The simulations were analyzed to determine the acceleration of the mass center and angular accelerations of the hip, knee, and ankle generated by individual muscles. The results of this analysis indicate that children walking in crouch gait have less passive skeletal support of body weight and utilize substantially higher muscle forces to walk than unimpaired individuals. Crouch gait relies on the same muscles as unimpaired gait to accelerate the mass center upward, including the soleus, vasti, gastrocnemius, gluteus medius, rectus femoris, and gluteus maximus. However, during crouch gait, these muscles are active throughout single-limb stance, in contrast to the modulation of muscle forces seen during single-limb stance in an unimpaired gait. Subjects walking in crouch gait rely more on proximal muscles, including the gluteus medius and hamstrings, to accelerate the mass center forward during single-limb stance than subjects with an unimpaired gait.

1. Introduction

Crouch gait, a common movement pattern among individuals with cerebral palsy, is characterized by excessive flexion of the hip, knee, and ankle during the stance phase of gait. This walking pattern is inefficient (Rose et al., 1989; Waters and Mulroy, 1999) and if left untreated can lead to joint pain (Jahnsen et al., 2004), formation of boney deformities (Graham and Selber, 2003), and loss of independent gait (Johnson et al., 1997; Opheim et al., 2009). Clinicians try to identify muscles that can be strengthened, surgically lengthened, or otherwise treated to enable a more erect and efficient walking pattern. Little is known, however, about how individual muscles contribute to joint and mass center motions during crouch gait; thus, it is difficult to design treatment plans that target muscles most likely to improve gait dynamics.

Humans have developed an efficient walking pattern to achieve forward progression while supporting body weight. Several studies (e.g. Anderson and Pandy, 2003; Neptune et al., 2004; Arnold et al., 2005; Kimmel and Schwartz, 2006; Liu et al., 2006) have examined how muscles accelerate the joints and mass center during unimpaired gait. These studies have shown that during early stance, the vasti and gluteus maximus support the body and slow forward progression, while in late stance the gastrocnemius and soleus support body weight and propel the body forward (Neptune et al., 2001; Anderson and Pandy, 2003; Liu et al., 2006). Liu et al. (2008) demonstrated that the roles of these muscles are maintained over a range of walking speeds.

Changes in joint kinematics and muscle activation patterns during crouch gait alter how muscles contribute to joint and mass center accelerations. Hicks et al. (2008) analyzed the potential of individual muscles to accelerate the hip and knee, per unit force, in crouched walking postures. This analysis revealed that a crouched posture markedly reduces the potential of several major lower extremity muscles to generate extension accelerations of the hip and knee and increases the joint flexion accelerations due to gravity. While this prior study determined the direction (i.e. flexion or extension) of the accelerations generated by important muscles, the magnitudes of the accelerations generated by muscles depend on muscle forces, which have not been estimated for subjects with crouch gait.

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Determining how muscles contribute to joint angular accelerations and mass center accelerations during crouch gait can clarify the role of muscles during this abnormal walking pattern and elucidate biomechanical consequences of treatments, such as surgically lengthening muscles. Thus, the goal of the present study was to quantify angular accelerations of the hip, knee, and ankle generated by stance-limb muscles during the single-limb stance phase of crouch gait by creating and analyzing the first subject-specific dynamic simulations of crouch gait. Additionally, we characterized how muscles accelerate the mass center, which provides a holistic view of how muscles contribute to motion of the body during gait. The simulations are freely available at www.simtk.org, enabling other researchers to reproduce the results of this study and to perform additional analyses.

2. Methods

2.1. Subjects

Ten subjects with spastic diplegic cerebral palsy – age: 8.1 ± 1.7 yrs, height: 1.25 ± 0.09 m, weight: 27.1 ± 9.1 kg, leg length: 0.65 ± 0.06 m (mean ± SD) – were selected from a database of subjects examined at the Gillette Children's Specialty Healthcare, St. Paul, MN. Each subject included in the study: (1) walked with a mild crouch gait (minimum knee flexion 15–40° during stance), (2) did not walk in equinus and achieved at least 0° of dorsiflexion during his or her physical exam, (3) had no previous surgeries, and (4) had no significant torsional skeletal deformities (less than 30° of tibial torsion and femoral anteversion). All subjects and/or guardians provided informed written consent for the data collection; analyses of the data were performed in accordance with the regulations of all participating institutions.

Motion analysis data were collected and kinematics determined using a 12-camera system (Vicon Motion Systems, Lake Forest, CA) and a standard marker measurement protocol (Davis et al., 1991). Four force plates (AMTI, Watertown, MA) were used to record ground reaction forces and moments, which were sampled at 1080 Hz and low-pass filtered at 20 Hz. Our analysis focused on single-limb support, because a lack of consecutive force plate strikes precluded the analysis of double-support. Subjects walked at a self-selected walking speed of 0.92 ± 0.18 m/s. Surface electromyography (EMG) signals were recorded for nine of the ten subjects from the medial hamstrings, biceps femoris long head, rectus femoris, gastrocnemius, and tibialis anterior (Motion Laboratory Systems, Baton Rouge, LA). EMG was sampled at 1080 Hz, band-pass filtered between 20 and 400 Hz, rectified, and low-pass filtered at 10 Hz. EMG for each muscle was normalized from zero to one based on the minimum and maximum values of that muscle over all trials for each subject.

2.2. Dynamic simulation

A generic musculoskeletal model (lower extremities from Delp et al., 1990 and torso from Anderson and Pandy, 1999) with 19 degrees of freedom and 92 musculotendon actuators was scaled to each subject according to his or her anthropometric measurements (Fig. 1). The degrees of freedom included a ball-and-socket joint located at approximately the third lumbar vertebra between the pelvis and torso, ball-and-socket joints at each hip, a planar joint with coupled translations at each knee, and revolute joints at each ankle. A subject-specific simulation of the single-limb stance phase of gait was generated using OpenSim, a freely available biomechanical simulation package (Delp et al., 2007, www.simtk.org). Double-support was not included in this study due to a lack of consecutive force plate strikes in the small set of subjects who met our inclusion criteria. Kinematic and ground reaction force data were imported into OpenSim, where the computed muscle control algorithm determined the muscle excitations that generated a forward dynamic simulation that minimized the difference between the experimental and simulated gait kinematics (Thelen et al., 2003; Thelen and Anderson, 2006). Estimated muscle activations were scaled in the same manner as the EMG signals, and compared to the experimentally measured EMG signals to evaluate the fidelity of the simulation's muscle activation timing.

To test the sensitivity of the results to errors in muscle activation timing, we constrained the simulated activations to match the shape and normalized magnitude of the EMG for the 10 muscles for which EMG was recorded for one subject (see Supplementary Figs. 1–3). When there was a disagreement between the EMG and simulated activations, the normalized magnitude of the EMG was used to define maximum and minimum bounds on the magnitude of the simulated activations. Although adding these constraints changed the magnitude of the contribution of each muscle, the direction (e.g., flexion/extension) did not change. Since these changes in magnitudes did not alter the conclusions drawn from analyzing this simulation, we chose not to constrain activations for other subjects.

A perturbation analysis was used to determine the joint angular accelerations and mass center accelerations generated by each muscle in the model (Liu et al.,

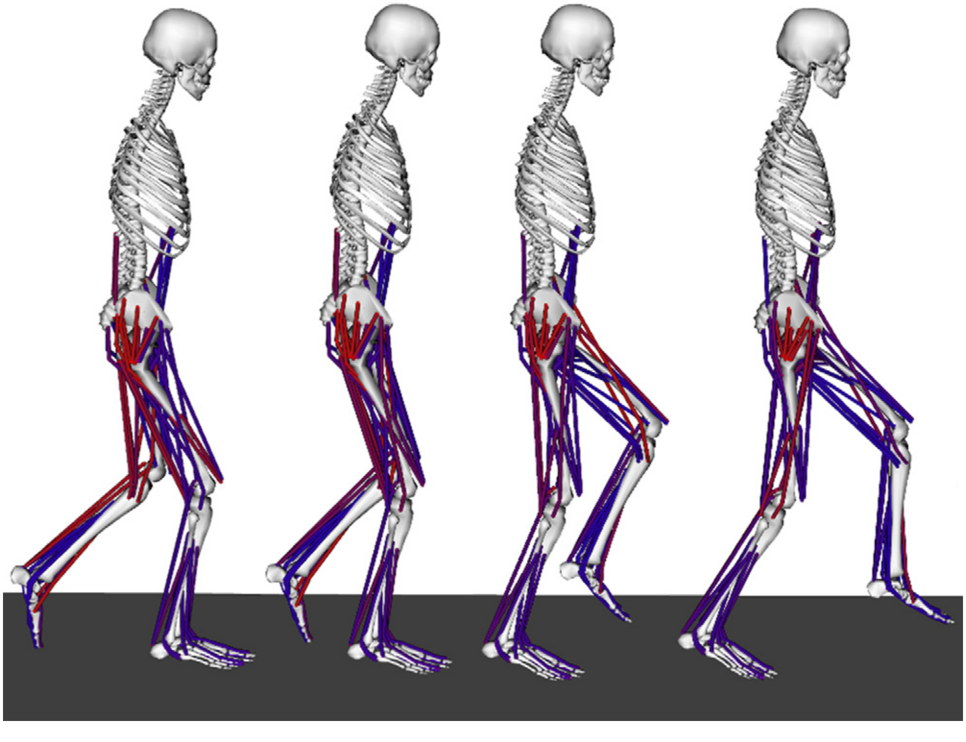

Fig. 1. OpenSim model shown at different phases of single-limb stance during crouch gait. The muscles shown in red are highly activated, while those in blue have a low activation level, as determined using the computed muscle control algorithm. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)