

## **Computer modeling of gait abnormalities in cerebral palsy: application to treatment planning**

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The treatment of gait abnormalities in persons with cerebral palsy is challenging. Theoretically, gait abnormalities can be diminished by decreasing the muscle forces that disrupt normal movement (e.g. via muscle–tendon lengthenings or tone-altering medications) and/or increasing the muscle and ground reaction forces that have the potential to improve movement (e.g. via strengthening exercises, orthoses, or derotational osteotomies). However, different patients exhibit varying degrees of neurologic impairment, spasticity, weakness, and bone deformity, suggesting that gait deviations arise from a variety of sources, each of which requires a different treatment. Treatment planning is further complicated because there is currently no scientific basis for determining how patients' neuromusculoskeletal impairments contribute to abnormal movement. This paper describes how biomechanical models can be used, in combination with experimental data, to enhance our understanding of gait abnormalities and to provide a theoretical basis for planning treatments. Two examples are presented, and suggestions for future work are discussed.

*Keywords:* Modeling; Treatment planning; Weakness; Bone deformity

### **1. Introduction**

The management of gait abnormalities in children with cerebral palsy is challenging. Musculoskeletal surgeries and other treatments, such as tone-altering medications, orthoses and physical therapy are often prescribed in an effort to improve the alignment of patients' limbs, prevent the progression of bone deformities, and increase walking ability. However, different patients with cerebral palsy exhibit varying degrees of neurologic impairment, spasticity, weakness, muscle contracture, and bone deformity, suggesting that gait deviations arise from a variety of sources, each of which requires a different treatment. Treatment planning is further complicated because there is currently no scientific basis for determining how patients' neuromusculoskeletal impairments contribute to abnormal movement (figure 1). This paper describes how biomechanical models can be used, in combination with experimental data, to enhance our understanding of gait abnormalities and to

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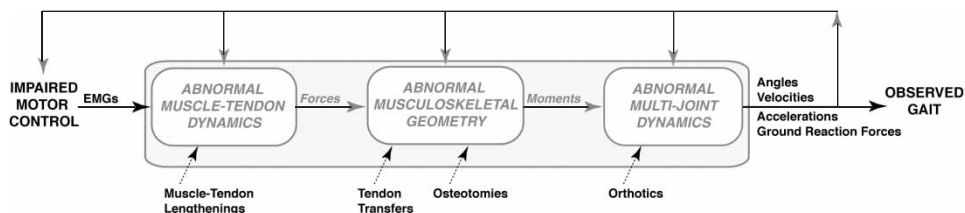


Figure 1. Many factors contribute to movement abnormalities in persons with cerebral palsy. Gait analysis is used routinely to record electromyographic (EMG) patterns, joint angles, and ground reaction forces during walking, but the transformation between EMG patterns and coordinated multijoint movement (shaded region) is complicated. Furthermore, to make treatment decisions clinicians must try to predict how the motions induced by muscles might change after treatment. Computational models that characterize patients' muscle-tendon dynamics, musculoskeletal geometry, and multijoint dynamics during walking may enhance interpretation of motion analysis studies and improve the planning of treatments. Reproduced with permission from Arnold and Delp (2004).

provide a theoretical basis for planning treatments. Two examples are presented, and suggestions for future work are discussed.

## 2. Analysis of hip muscle moment arms during internally rotated gait

Children with cerebral palsy frequently walk with excessive internal rotation of the hip. Spastic medial hamstrings or adductors are thought to contribute to the excessive internal rotation in some patients based on electromyographic (EMG) evidence that the muscles are active during walking, and on the presumption that these muscles generate an internal hip rotation moment (e.g. Sutherland *et al.* 1969, Chong *et al.* 1978). Surgical lengthening of these muscles is often expected to decrease excessive internal rotation (e.g. Tachdjian 1990). However, the extent to which the hamstrings and adductors contribute to hip internal rotation is unclear, and the changes in hip rotation following surgery are inconsistent. We have performed a series of studies to determine which muscles have the greatest potential to rotate the hip in persons with femoral deformities who walk with a crouched, internally rotated gait (Arnold *et al.* 1997, Delp *et al.* 1999, Arnold *et al.* 2000, Arnold and Delp 2001). These studies have provided new guidelines for the treatment of excessive hip internal rotation.

In one study, we evaluated the hip rotation moment arms of the medial hamstrings and adductors using highly accurate musculoskeletal models of three individuals with cerebral palsy that we constructed from magnetic resonance images (figure 2). Analysis of these models, at the limb positions corresponding to each subject's internally rotated gait, revealed that the semimembranosus, semitendinosus, adductor brevis, adductor longus, and gracilis had *external* rotation moment arms or very small internal rotation moment arms throughout the gait cycle, in all three subjects (Arnold *et al.* 2000). These findings indicate that neither the medial hamstrings nor the adductors are likely to be important contributors to excessive internal rotation of the hip and that other causes of internal rotation should be considered with planning treatments for these patients.

We used our musculoskeletal models to examine other potential causes of excessive hip internal rotation (Arnold *et al.* 1997, Delp *et al.* 1999). Our experimental

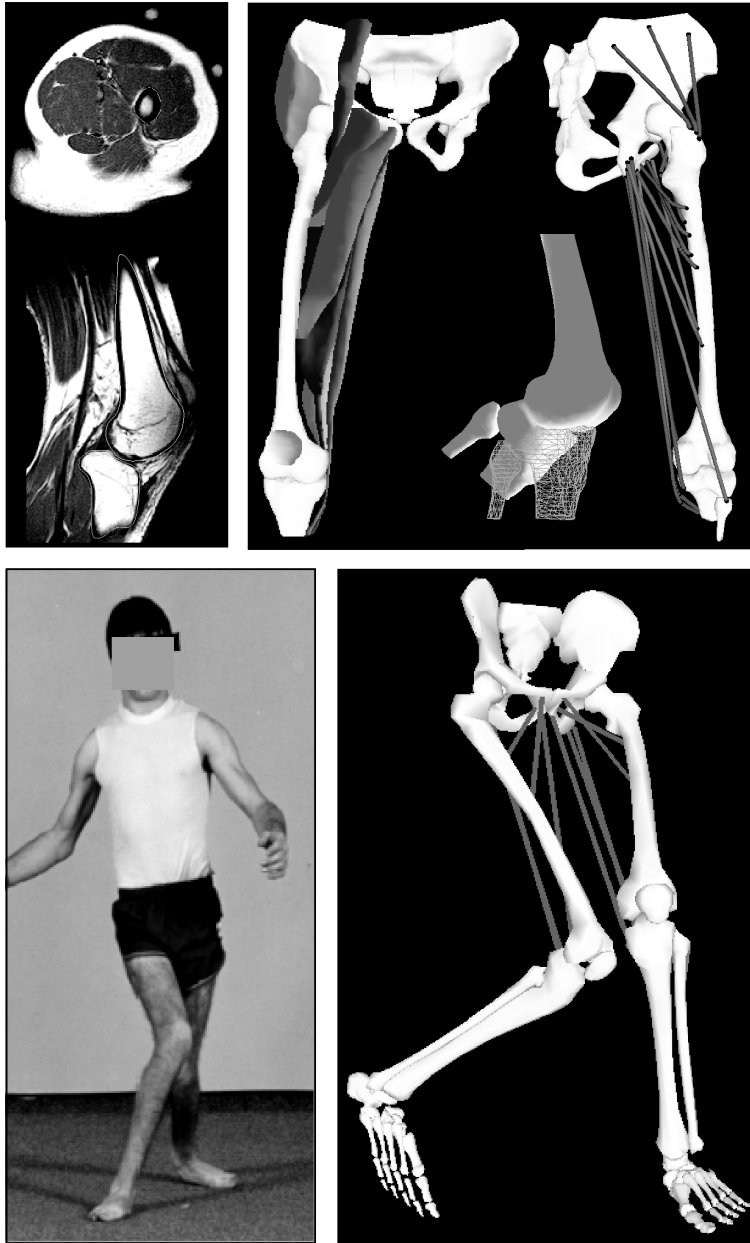


Figure 2. Determination of hip rotation moment arms during crouched, internally rotated gait. Musculoskeletal models of subjects with cerebral palsy were created from magnetic resonance images. For each subject, three-dimensional surface representations of the muscles and bones were generated from two-dimensional contours segmented manually from each of approximately 200 images (top left). Surfaces from overlapping series of images were registered to obtain an accurate representation of each subject's anatomy at the 'scanned' limb position. Kinematic models of the hip and the knee were implemented, and the muscle lines of action were defined (top right). The rotational moment arms of the medial hamstrings, adductors, and other muscles were evaluated at the body positions corresponding to each subject's internally rotated gait (bottom). Reproduced with permission from Arnold and Delp (2004).

studies of moment arms in cadavers and model-based analyses have shown that the rotational moment arms of the gluteus medius and minimus increase dramatically with hip flexion (Delp *et al.* 1999). Since excessive flexion of the hip frequently accompanies internally rotated gait (Bleck 1987), and since the gluteal muscles are typically active and play an important role in walking (Perry 1992), we suggest that the excessive hip flexion of patients, which increases the internal rotation moment arms of the gluteus medius and minimus, is more likely than the hamstrings or adductors to cause internal rotation. Furthermore, we have observed that the gluteus maximus has a large capacity for external rotation when the hip is extended (Delp *et al.* 1999); thus, strengthening or enhancing activation of the gluteus maximus in persons with crouched, internally rotated gait may help to correct both the excessive hip flexion and internal rotation. These studies of hip muscle moment arms highlight the need for musculoskeletal models that can account for altered bone geometry and abnormal joint kinematics when hypothesizing the causes of gait abnormalities and planning treatments.

### 3. Analysis of muscle actions during stiff-knee gait

Many individuals with cerebral palsy walk with insufficient knee flexion during the swing phase, or stiff-knee gait. This movement abnormality is often attributed to excessive activation of the rectus femoris (Gage *et al.* 1987, Perry 1987), a biarticular muscle that generates both hip flexion and knee extension moments. Stiff-knee gait is commonly treated by rectus femoris transfer, a procedure in which the distal tendon of the muscle is detached from the patella and reattached posterior to the knee. However, the surgical outcomes are inconsistent and sometimes unsuccessful. We have developed forward dynamic simulations of the swing limb to identify factors that influence knee flexion during normal gait and to determine how forces generated by the rectus femoris during swing affect knee flexion.

A model of the lower extremity with five segments (pelvis, thigh, patella, shank, and foot), three degrees of freedom (flexion-extension of the hip, knee, and ankle), and 12 muscle-tendon actuators was created (Piazza and Delp 1996). Each muscle-tendon actuator generated force as a function of its activation, length, and velocity (Zajac 1989). The excitation patterns of the muscles, the motions of the pelvis, and the angles and angular velocities of the joints at toe-off were specified as inputs to the simulation. The resulting kinematics of the limb were calculated by integrating the equations of motion of the model forward in time. A simulation of the swing phase of normal gait was developed using muscle excitations that were derived from published intramuscular EMG recordings (Perry 1992). Other simulations were conducted using an exaggerated excitation input to the rectus femoris to clarify the dynamical actions of the muscle at the knee.

Our simulations confirmed that overactivity of the rectus femoris inhibits knee flexion during swing and, thus, may cause stiff-knee gait. Additionally, our analyses revealed that several other factors, such as weakened hip flexors or stance phase factors that diminish the angular velocity of the knee at toe-off, may also be responsible for decreased knee flexion during the swing phase (Piazza and Delp 1996).

To gain more insight into the factors that contribute to stiff-knee gait, we have begun to analyze simulations that reproduce the swing limb trajectories of patients. In one case study, for example, we created a model of a subject with stiff-knee gait

who walked with excessive activity of the rectus femoris (Goldberg *et al.* 2001). We used inverse dynamics, in combination with kinematic data from gait analysis, to calculate the subject's muscular joint moments during the swing phase. We then used these calculated moments to drive a forward dynamic simulation. When we prescribed the initial kinematic conditions for the simulation based on the subject's measured joint angles and angular velocities at toe-off, the knee motions of the model reproduced the subject's stiff-knee gait. However, when normal kinematic conditions at toe-off were input into the simulation, without changing the muscular joint moments, the peak flexion of the knee during swing was greater than during normal gait (figure 3). These data suggest that abnormal muscular moments in swing phase were not the primary cause of diminished knee flexion in this subject, and may explain why this subject's knee flexion did not improve following rectus femoris transfer surgery. In summary, we believe that kinematic conditions at toe-off should be considered along with rectus femoris activity before surgery is performed in an attempt to correct stiff-knee gait. This study emphasizes the need for rigorous,

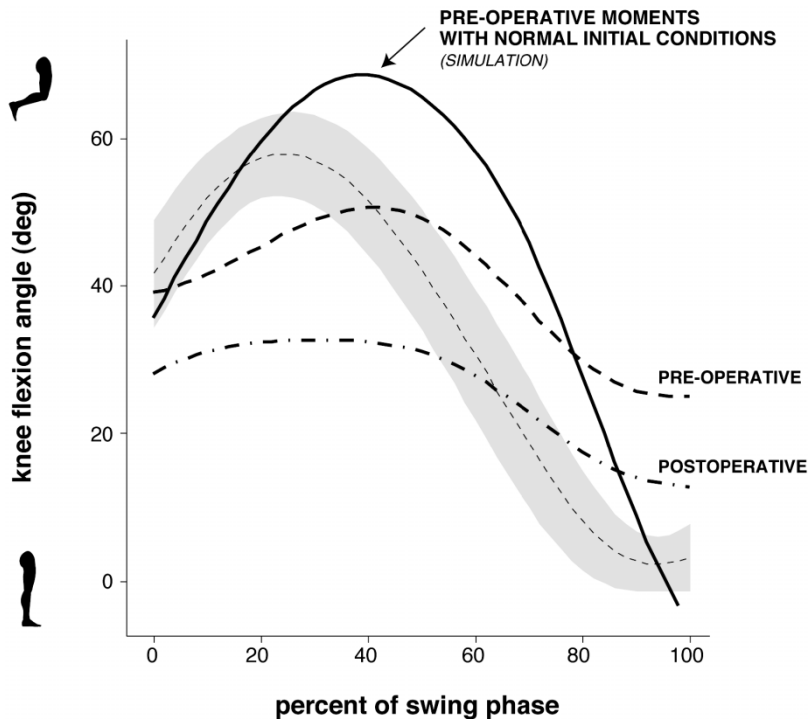


Figure 3. Knee flexion angle vs. percent of swing phase, generated from patient-specific, forward dynamic simulations. When the initial kinematic conditions for the simulation (i.e. knee angle, knee velocity, and hip velocity at toe-off) were prescribed based on the subject's measured joint angles and angular velocities, the knee motions of the model reproduced the subject's stiff-knee gait (dashed lines). When normal kinematic conditions at toe-off were input into the simulation, without changing the subject's muscular joint moments, the resulting knee motion resembled normal gait (c.f. solid line and shaded region). This result suggests that abnormal muscular moments in swing were not the primary cause of the subject's diminished knee flexion. Reproduced with permission from Arnold and Delp (2004).

dynamics-based analyses of the actions of muscles when attempting to determine the causes of a patient's gait abnormality.

#### 4. Discussion and future directions

Musculoskeletal simulations have provided important insights into the pathomechanics of gait abnormalities and the functional consequences of treatments. However, the limitations of current models must be reduced, and the accuracy with which models represent individuals with neuromusculoskeletal impairments must be tested, before simulations can be widely used to guide treatment decisions for patients. Some issues to be resolved in future studies are outlined below.

First, methods to accurately and efficiently characterize the musculoskeletal geometry of children with cerebral palsy need to be developed. This is imperative because the results of simulations are often sensitive to the accuracy with which the lengths and moment arms of muscles can be estimated with a model. To date, studies of muscle function during movement have typically relied on 'generic' models of adult subjects with normal musculoskeletal geometry. We have modified generic models to simulate bone deformities (Arnold *et al.* 2001, Arnold and Delp 2001), osteotomies (Free and Delp 1996, Schmidt *et al.* 1999), and tendon transfer surgeries (Delp *et al.* 1994). However, more work is needed to understand how variations in musculoskeletal geometry due to size, age, deformity, or surgery might influence the predictions of a model, and to determine when, and under what conditions, simulations based on generic models are applicable to individual patients.

Second, the model of muscle-tendon mechanics that we have used in simulations must be further tested. While this model captures many features of muscle force generation in unimpaired subjects, it does not account for adaptations that can occur in persons with neuromuscular disorders. Muscle-tendon models that characterize the effects of pathology, surgery, and other treatment modalities on the muscle force-generating characteristics are needed.

Perhaps the most profound limitation of existing biomechanical models is their exclusion of central nervous system control. Certainly, the incorporation of accurate representations of sensory-motor control into dynamic simulations of abnormal movements is one of the most critical challenges that must be overcome if models are to be developed that can predict the outcomes of treatments.

We believe that musculoskeletal simulations are necessary for explaining the biomechanical causes of movement abnormalities and the consequences of common interventions; this information is essential for developing improved treatment plans. Ultimately, controlled clinical studies are required to determine if the insights gained from musculoskeletal models can indeed improve treatment outcomes.

#### Acknowledgments

We would like to thank Stephen Piazza, Bill Hess, Deanna Asakawa, Silvia Blemker, Saryn Goldberg, Peter Loan, Ken Smith, Carolyn Moore, Stephen Vankoski, Claudia Kelp-Lenane, Julie Witka, and Rob Novak for help in the anatomical experiments, computer modeling, and data analysis. We are also grateful to Eugene

Bleck, Norris Carroll, Luciano Dias, James Gage, Tom Novacheck, Michael Schwartz, Sylvia Ounpuu, Jacquelin Perry, George Rab, and Felix Zajac for their helpful comments related to movement deformities and musculoskeletal modeling. This work was supported by NIH RO1HD33929 and RO1HD37639.

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