# 11
# THE ROLE OF MUSCULOSKELETAL MODELS IN PATIENT ASSESSMENT AND TREATMENT

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The management of gait abnormalities in persons with cerebral palsy is a challenging issue. Theoretically, gait abnormalities can be diminished by first identifying the biomechanical factors that contribute to abnormal movement and then either decreasing the muscle forces that disrupt normal movement (e.g. via muscle-tendon lengthening or botulinum toxin injections), 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 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 requiring a different treatment. Treatment planning is further complicated because there is currently no scientific basis for determining how patients' neuro-musculoskeletal impairments contribute to abnormal movement. The static muscle tests performed during a patient's physical exam (Chapter 5) and the kinematic, kinetic, and electromyographic (EMG) data obtained from gait analysis (Chapters 6–9) are not always sufficient to identify the biomechanical source of a patient's abnormal gait or to predict the consequences of treatments. This limitation exists, in part, because the transformation from EMG patterns to motion is extremely complex (Fig. 11.1) and because the effects of common surgical procedures on muscle-tendon mechanics and musculoskeletal geometry are not easily measured. This chapter describes how computer simulations of the musculoskeletal system can be used, in combination with gait analysis, to enhance our understanding of movement abnormalities and to provide a theoretical basis for planning treatments.

Imagine the following hypothetical scenario. A child with a troublesome gait abnormality visits a cerebral palsy clinic. The child undergoes a routine physical exam, a gait analysis, and perhaps a medical imaging study. A computer model of the child's musculoskeletal system is created that characterizes the force-generating capacity of the muscles, the geometric relationships between the muscles, tendons and bones, the kinematics of the joints, and the inertial properties of the body (see the shaded region of Fig. 11.1). The model is driven by a set of muscle excitation signals, and the resulting motion of the model is governed by mathematical equations that describe the activation dynamics of muscle, the contraction dynamics of muscle, and the multi-joint dynamics of the body during walking. The muscle excitation signals are specified such that the computer model "walks"

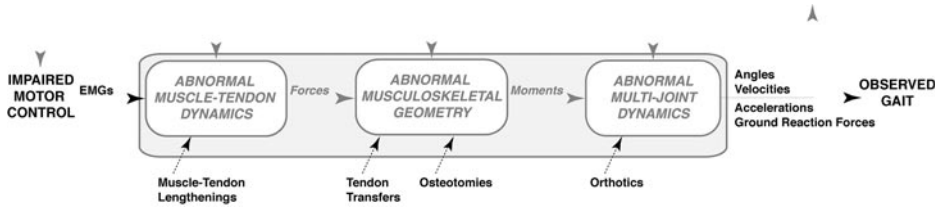


**Fig. 11.1.** Many factors contribute to movement abnormalities in persons with cerebral palsy. Gait analysis is used routinely to record EMG patterns, joint angles, and ground reaction forces during walking, but the transformation between EMG patterns and coordinated multi-joint movement (shaded region) is complicated. Furthermore, to make treatment decisions clinicians must try to predict how the motions induced by muscles might change after treatment. Typically, treatments alter the muscle-tendon dynamics or the musculoskeletal geometry, and these changes are not easily measured. Computational models that characterize patients' muscle-tendon dynamics, musculoskeletal geometry, multi-joint dynamics of the body during walking may enhance interpretation of motion analysis studies and improve the planning of treatments.

in a way that resembles the child's abnormal gait. Analysis of the motions produced by muscles identifies the specific causes of the child's abnormal movements. The model is used to evaluate several possible treatments and a comprehensive surgical plan and/or physical therapy regimen is designed, based on the child's clinical exam, the gait analysis and the modeling study. The child returns for a postoperative gait analysis one year after treatment. As predicted by the model, the child's walking ability has improved dramatically.

This scenario offers one vision of how musculoskeletal simulations might someday be used to improve the treatment of gait abnormalities in persons with neuromuscular disorders. However, creating biomechanical models that enable accurate prediction of treatment outcomes remains a formidable, multifaceted challenge. Most biomechanical studies of muscle function to date have relied on "generic" models, based on measurements of muscle architecture, musculoskeletal geometry, neuromuscular excitation patterns, and multi-joint movement kinematics from a relatively small number of unimpaired subjects. Only a few investigators have attempted to characterize the bone geometry or the muscle force-generating properties of children with deformities, spasticity or contracture (Tardieu and Tardieu 1987, Rose et al. 1994, Lundy et al. 1998, Arnold and Delp 2001). Dynamic simulations that resemble normal gait, driven by as many as 54 muscle-tendon actuators, have been created (Yamaguchi and Zajac 1990, Taga 1995, Gerritsen et al. 1998, Anderson and Pandy 2001, Neptune et al. 2001). However, no simulation has been developed that can explain how a particular child's impairments contribute to abnormal gait, or can predict how an individual will ambulate following orthopaedic surgery. Furthermore, constructing a patient-specific model for every child with a gait abnormality would be costly and labor-intensive. Given these difficulties, is the hypothetical scenario outlined above realistic, or overly optimistic? What is the role of musculoskeletal modeling in patient assessment and treatment?

We believe that musculoskeletal simulations have tremendous potential to enhance the management of movement abnormalities in persons with cerebral palsy. However, models that are used to guide treatment decisions must be formulated, tested, and interpreted with

care, in the knowledge of the underlying limitations of the models and the conditions that determine when, and for which patients, the results of a simulation are applicable. Models do not need to include patient-specific representations of all the neuromuscular and musculoskeletal elements involved in the production of movement to be valuable. Rather, we envisage that many of the insights needed to improve treatment outcomes will come from analyses of models with varying complexity.

Models of the musculoskeletal system can facilitate the assessment and treatment of gait abnormalities in several ways. First, models can provide information about a range of biomechanical parameters that are not easily measured, such as the lengths and moment arms of muscles, the force- and moment-generating capacities of muscles, and the multi-joint accelerations produced by muscles during movement. A relatively simple model that characterizes musculoskeletal geometry, for example, can be used in conjunction with joint angles obtained from gait analysis to estimate the lengths of muscles during normal and pathologic gait (Hoffinger et al. 1993, Delp et al. 1996, Schutte et al. 1997, Thompson et al. 1998). Knowledge of the muscle-tendon lengths may be useful because a "short" muscle that restricts movement can often be surgically lengthened or injected with botulinum toxin. Analyses of the muscle-tendon lengths during movement may help to distinguish patients who walk with abnormally short muscles from those who do not walk with short muscles, and thus may provide a basis for identifying patients who would benefit from treatment. Studies to test this hypothesis are under way.

Second, musculoskeletal models enable users to pose "what if?" questions, introduce changes to a model, and quantify the biomechanical consequences. For instance, a model that describes muscle-tendon mechanics and musculoskeletal geometry can be used to determine how the moment-generating capacities of muscles are altered by tendon lengthenings (Delp and Zajac 1992, Delp et al. 1995), tendon transfers (Dul et al. 1985, Delp et al. 1994, Lieber and Friden 1997), and other musculoskeletal procedures. These surgeries are often performed on persons with cerebral palsy in an effort to produce a more normal balance of the moments about the joints during movement, but are not always successful. A model that allows surgical effects to be quantified may help investigators to design more effective orthopaedic procedures. Without a model, evaluating the outcome of a treatment is often difficult, if not impossible, because of the many uncontrolled parameters of clinical trials.

Third, musculoskeletal simulations provide a powerful theoretical framework for examining cause-and-effect relationships between the excitation patterns of muscles and the multi-joint accelerations of the body during movement. Clinical assessments of muscle function during walking are often based upon a muscle's EMG activity and the joint moments to which the muscle contributes. However, these are not the only factors that determine the actions of a muscle on the body. A muscle that crosses one joint has the potential to accelerate other joints, and biarticular muscles can produce angular accelerations of the joints that oppose their applied moments (Hollerbach and Flash 1982, Zajac and Gordon 1989). For example, the soleus exerts only an ankle plantarflexion moment, yet Zajac and Gordon (1989) demonstrated that the soleus can accelerate the knee into extension more than it accelerates the ankle into plantarflexion. To identify which muscles may produce abnormal movement, therefore, we need a model that characterizes the coupled dynamics

of the limb segments, the muscle-tendon mechanics, and the musculoskeletal geometry.

The remainder of this chapter reviews several studies in which biomechanical models of varying complexity have been used to enhance the analysis of a particular gait abnormality or improve the design of a treatment plan. Each example first introduces the clinical question that motivated the development of a model and then describes selected simulation results. The chapter concludes with a brief discussion of some of the limitations of current musculoskeletal simulations. Consideration of these limitations suggests areas for future research.

**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, among other factors, are thought to contribute to the excessive internal rotation in many patients based on EMG evidence that the muscles are active during walking, and on the presumption that these muscles generate an internal hip-rotation moment (Sutherland et al. 1969, Chong et al. 1978). Surgical lengthening of these muscles is often expected to decrease excessive internal rotation (Hoffer 1986, Root 1987, 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. The rotational moment arm of a muscle about the hip determines whether the muscle has the potential to produce an internal or an external hip rotation moment. Therefore knowledge of the muscle moment arms is needed to establish a scientific rationale for muscle-tendon surgeries intended to reduce internal rotation moments.

Determination of hip-rotation moment arms in patients with cerebral palsy is difficult for two main reasons. First, rotational abnormalities of the hip are often accompanied by excessive anteversion of the femur (Bleck 1987), a torsional bone deformity that may alter the lines of action and moment arms of muscles about the hip. Second, the muscle moment arms must be evaluated over the range of limb positions assumed by persons with cerebral palsy during walking; this frequently includes exaggerated flexion of the hips and knees in addition to increased internal rotation of the hip. We have performed a series of studies to determine which muscles have the greatest potential to rotate the hip in children with femoral deformities who walk with a crouched, internally rotated gait (Arnold et al. 1997, 2000, Delp et al. 1999, 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 (Fig. 11.2). 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). Hence, none of these muscles could have generated a substantial hip internal-rotation moment in these subjects, whose gait abnormalities and femoral deformities might be considered typical of patients who walk with excessive internal rotation of the hip.

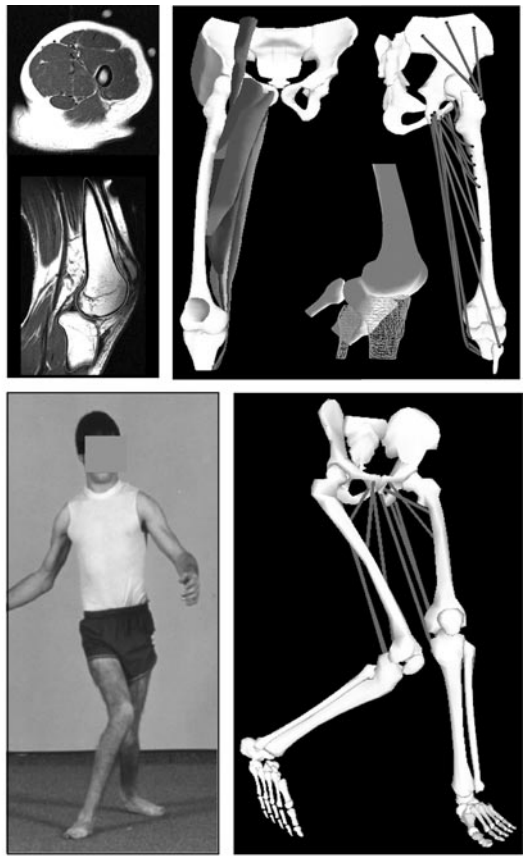

**Fig. 11.2.** Determination of hip rotation moment arms during crouched, internally rotated gait. Musculoskeletal models of subjects with cerebral palsy were created from magnetic resonance images. For each subject, three-dimensional surface representations of the muscles and bones were generated from 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 (top center). 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).

Based on these observations, we hypothesized that the rotational moment arms of the medial hamstrings and adductors are shifted toward external rotation by excessive femoral anteversion and/or by exaggerated hip flexion, knee flexion or hip internal rotation. We tested this hypothesis using a model of the lower extremity with a "deformable" femur that estimates the moment arms at the body positions of patients who walk with crouched, internally rotated gait (Arnold et al. 2001). We determined that the semimembranosus, semitendinosus and gracilis muscles in our model had negligible or external rotation-moment arms when the hip was internally rotated or when the knee was flexed—the body

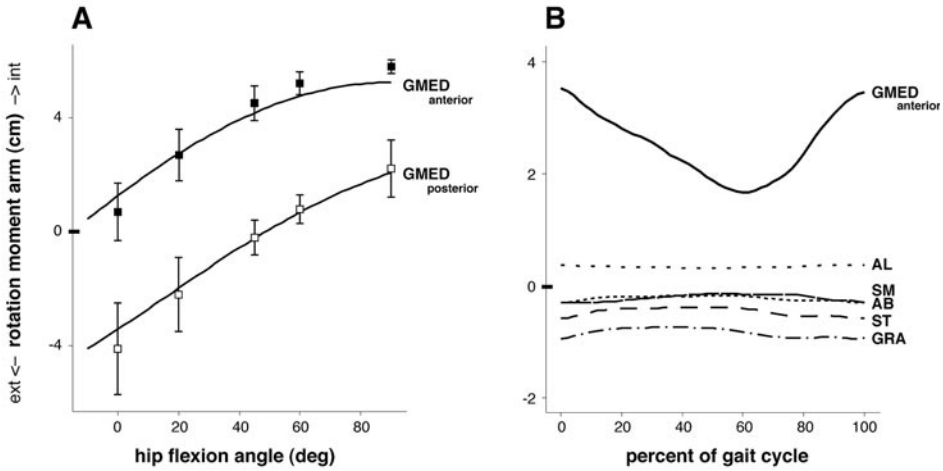


**Fig. 11.3.** Hip rotation moment arms of the gluteus medius versus hip-flexion angle (A) and gait cycle (B). Rotational moment arms of the anterior (filled squares) and posterior (open squares) compartments of the gluteus medius determined experimentally (mean ± 1 S.D. for four specimens) and calculated with a musculoskeletal model (solid lines) increase dramatically with hip flexion (A). This suggests that internally rotated gait may be a result of excessive hip flexion, which shifts the rotational moment arms of the gluteal muscles toward internal rotation. Internal rotation moment arms of the anterior compartment of the gluteus medius during walking, computed with an magnetic resonance-based model of a subject with cerebral palsy, are approximately four times larger than the rotational moment arms of the medial hamstrings or adductors (B).

positions that children with cerebral palsy commonly assume during walking. When the femur was excessively anteverted, the rotational moment arms of the adductor brevis, adductor longus, pectineus and proximal compartments of the adductor magnus in our model shifted toward external rotation. These results indicate that neither the medial hamstrings nor the adductors are likely to contribute substantially 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 studies of hip-rotation moment arms in cadavers have shown that the rotational moment arms of the gluteus medius and gluteus minimus increase dramatically with hip flexion (Delp et al. 1999). The moment arms computed with our musculoskeletal models are consistent with this observation (Fig. 11.3). Since excessive flexion of the hip frequently accompanies internally rotated gait (Bleck 1987, Gage 1991), 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. We have further 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.

**Analysis of muscle moment-generating capacity after tendon surgery**

Persistent plantarflexion of the ankle, termed equinus gait, is one of the most common movement abnormalities among cerebral palsy patients. Equinus gait is frequently caused by contracture (i.e. shortening of the fibers) of the triceps surae. Either isolated contracture of the gastrocnemius or combined contracture of the gastrocnemius and soleus may be present. When only the gastrocnemius is contracted, surgical lengthening of the gastrocnemius aponeurosis is usually successful in restoring the normal range of ankle motion while maintaining plantarflexion strength (Rose et al. 1993). However, tendo-Achilles lengthening, which is the procedure commonly performed to treat combined contracture of the gastrocnemius and soleus, is less effective. If the Achilles tendon is not lengthened enough, passive plantarflexion moment continues to cause ankle equinus after surgery (Sharrard and Bernstein 1972, Lee and Bleck 1980). By contrast, if the Achilles tendon is lengthened too much, the active force-generating capacity of the muscles can be compromised, resulting in disabling muscle weakness (Sutherland and Cooper 1978, Segal et al. 1989).

The force-generating capacity of a muscle after tendon surgery is influenced by the architecture of the muscle-tendon complex (i.e. the lengths and arrangement of the muscle fibers). Since the gastrocnemius and soleus exhibit different architectures, one might expect these muscles to respond differently to tendon lengthening (Delp and Zajac 1992). These effects are difficult to quantify in clinical studies because individual muscle forces cannot be measured without invasive techniques. However, a musculoskeletal model that accounts for differences in the muscle architectures can be used to evaluate how tendon-lengthening affects the muscles' force- and moment-generating characteristics.

We have used a musculoskeletal model to examine the trade-off between restoring range of ankle motion and maintaining plantarflexion strength in cases of combined contracture of the gastrocnemius and soleus (Delp et al. 1995). We first developed a model that represents the normal force- and moment-generating characteristics of the major muscles crossing the ankle. We then altered the model to represent contracture of the gastrocnemius and soleus. The force-length properties of each muscle-tendon complex were derived by scaling a dimensionless model of muscle and tendon by four parameters: peak isometric muscle force, optimal muscle-fiber length, tendon slack length, and pennation angle (Zajac 1989). Values of these parameters were specified based on experimental data published in the literature (Wickiewicz et al. 1983, Friederich and Brand 1990). To represent contracture of the gastrocnemius and soleus, the optimal fiber lengths of the muscles were decreased by 45% (Ziv et al. 1984). This decrease in the fiber lengths caused a substantial increase in the passive moments generated by the muscles, which is consistent with clinical observations (Bleck 1987, Tardieu and Tardieu 1987). We simulated the effects of tendo-Achilles lengthening by elongating the tendons of both the contracted gastrocnemius and the contracted

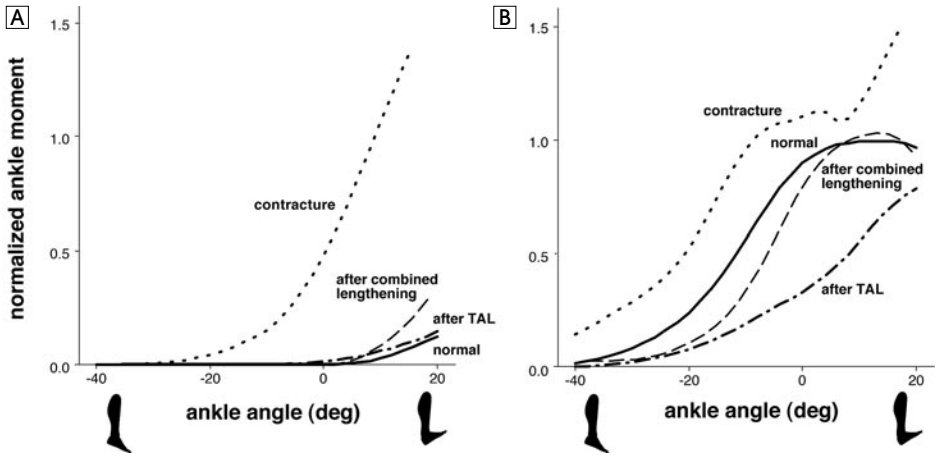


**Fig. 11.4.** Passive ankle moment (A) and total ankle moment (B) generated by a model of the triceps surae versus ankle angle. After simulated tendo-Achilles lengthening (TAL), the passive plantarflexion moment developed by the contracted triceps surae is restored approximately to normal (A), but the total moment-generating capacity of the triceps surae is substantially less than normal (B). After combined tendo-Achilles lengthening and gastrocnemius aponeurosis lengthening, the passive moment developed by the triceps surae is greater than normal (A); however, the total moment-generating capacity of the triceps surae is nearly normal (B). Muscles were assumed to be at their maximum level of activation when the total moments were calculated, and the moments were normalized by the maximum active moment of the normal triceps surae.

soleus. The effects of gastrocnemius aponeurosis lengthening were simulated by elongating the tendon of the gastrocnemius while the soleus tendon remained unaltered. The theoretical effectiveness of the simulated surgeries was evaluated based on the potential of each procedure to reproduce normal passive and active moment-generating characteristics of the triceps surae about the ankle.

In the simulations, neither tendo-Achilles lengthening nor gastrocnemius aponeurosis lengthening alone was an effective treatment for combined contracture of the gastrocnemius and soleus. Lengthening of the gastrocnemius aponeurosis did not diminish the excessive passive moment developed by the contracted soleus. Lengthening of the Achilles tendon by 2 cm restored the passive moment of the contracted muscles to near normal (Fig. 11.4A); however, this change in tendon length decreased the total moment-generating capacity of the triceps surae substantially (Fig. 11.4B).

The moment-generating characteristics of the triceps surae could be restored more effectively in the model if the contracted gastrocnemius and the contracted soleus were corrected independently. After simulated lengthening of both the Achilles tendon and the gastrocnemius aponeurosis by 1 cm, the total moment-generating capacity of the triceps surae was slightly less than normal (Fig. 11.4B), but greater than after a 2 cm lengthening of the Achilles tendon alone (Fig. 11.4B, dashed and dot-dashed curves). These results suggest that independent lengthening of the gastrocnemius and soleus can account for differences in the architectures of these muscles, and theoretically could provide a more efficacious

means for correcting equinus gait and preserving plantarflexion moment-generating capacity in cases of combined contracture. Although independently adjusting the muscles is slightly more complicated than tendo-Achilles lengthening, Saraph and colleagues (2000) reported favorable outcomes in a 2-year follow-up of 22 patients. This example illustrates how a model of musculoskeletal geometry and muscle-tendon mechanics can help explain and enhance our understanding of the biomechanical effects of treatments.

**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, Sutherland et al. 1990), 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 to one of several sites posterior to the knee. However, the surgical outcomes are inconsistent and sometimes unsuccessful, in part because the biomechanical factors that contribute to stiff-knee gait have not been adequately characterized. Analysis of the multi-joint motions produced by the rectus femoris is complex; the hip-flexion moment it generates has the potential to increase knee flexion while the knee-extension moment it generates acts to decrease knee flexion. We have developed forward dynamic simulations of the swing limb to identify factors that influence peak knee flexion during normal gait, and to determine how abnormal forces generated by the rectus femoris during swing affect knee flexion.

We calculated the accelerations of the swing limb from muscle excitation patterns. 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 swing limb were calculated by numerically integrating the equations of motion of the model forward in time. A simulation of the swing phase of normal gait was developed using muscle excitation patterns that were derived from published intramuscular EMG recordings (Perry 1992). Other simulations were conducted using an exaggerated excitation input to the rectus femoris (i.e. excitation of the rectus femoris at 30% of its maximum level throughout the swing phase) to clarify the dynamical actions of the rectus femoris at the knee.

Our simulations confirmed that overactivity of the rectus femoris inhibits knee flexion during swing, and thus may cause stiff-knee gait (Fig. 11.5). 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 biomechanical factors that contribute to stiff-knee gait, we have begun to develop and analyze simulations that reproduce the swing limb trajectories

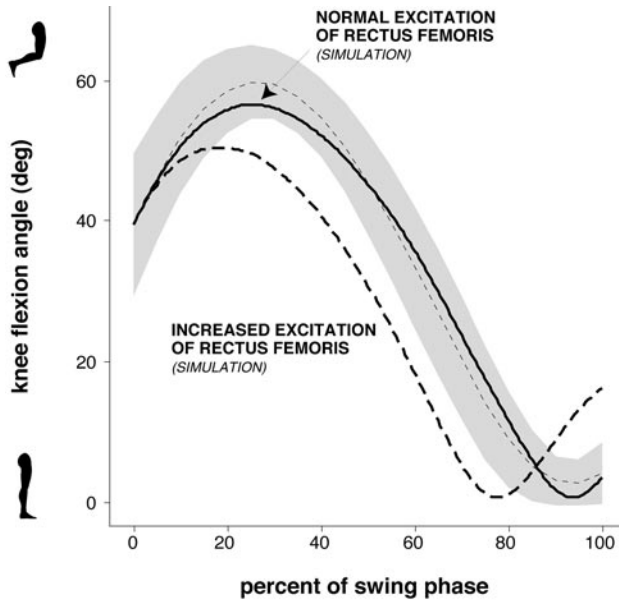


**Fig. 11.5.** Knee-flexion angle versus percentage of swing phase, generated from muscle-actuated, forward dynamic simulations of the swing limb. The knee angle for the nominal simulation, in which all of the muscle-tendon actuators were supplied with normal excitations (solid curve) compares favorably with experimental measurements of knee flexion during normal gait (mean ± 1 S.D. for 10 unimpaired subjects, shaded region). When the excitation input to the rectus femoris was exaggerated (dashed curve), knee flexion during the swing phase was limited. This result confirms that overactivity of the rectus femoris may cause stiff-knee gait.

of individual patients. In one case study, for example, we created a dynamic model of a subject with stiff-knee gait who walked with excessive and prolonged activity of the rectus femoris (Goldberg et al. 2001). We used an inverse dynamics formulation, 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 (Fig. 11.6). These data suggest that abnormal muscular moments 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 on the rectus femoris in an attempt to correct stiff-knee gait. This study emphasizes the need for rigorous, dynamics-based analyses

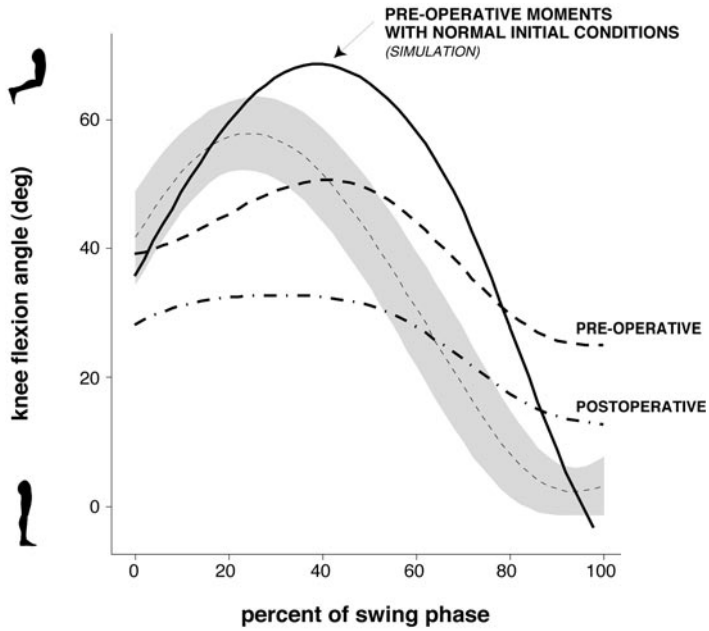


**Fig. 11.6.** Knee-flexion angle versus percentage of swing phase, generated from patient-specific, forward dynamic simulations of the swing limb. When the initial kinematic conditions for the simulation (i.e. knee angle, knee velocity, and hip velocity at toe-off) were prescribed based on the subject's 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 (solid line and shaded region). This result suggests that abnormal muscular moments were not the primary cause of the subject's diminished knee flexion.

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

**Discussion and future directions**

Musculoskeletal simulations can provide clinically useful insights into the pathomechanics of gait abnormalities and the functional consequences of treatments, as evidenced by the examples presented in this chapter. However, the limitations of current models must be reduced, and the accuracy with which models represent individuals with neuro-musculoskeletal impairments must be tested, before simulations can be widely used to guide treatment decisions for patients. Some of the important issues to be resolved in future studies are outlined below.

First, methods to accurately and efficiently characterize the musculoskeletal geometry and the joint kinematics 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. 1997, 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. One approach might be to develop patient-specific models from magnetic resonance images or ultrasonography scans that can estimate muscle lengths, moment arms, and joint kinematics *in vivo* (Sheehan et al. 1998, Wilson et al. 1999, Ito et al. 2000, Maganaris 2000). However, using image data alone to determine the lengths and moment arms of muscles at the wide range of body positions assumed during walking would require extensive imaging protocols to capture the muscle and joint geometry in many limb configurations. We suggest that a hybrid approach, which combines medical images with generic musculoskeletal models, offers a promising, tractable way to construct representative models of patients. For instance, it may be possible to transform a generic model to represent a range of individuals with cerebral palsy using multidimensional scaling techniques, algorithms for deforming bones, and a few patient-specific parameters derived from image data or experimental measurements (Chao et al. 1993, Arnold and Delp 2001). We have begun to develop and evaluate such models (Arnold et al. 2001, Arnold and Delp 2001), and we believe that additional efforts are warranted.

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. For example, the model does not account for complexities associated with activation of spastic muscle, such as potential alterations in recruitment or rate modulation (Tang and Rymer 1981). Although we have attempted to account for decreases in the muscle-fiber lengths that may occur with contracture, our simulations have not considered the effects of muscle-tendon remodeling, such as alterations in the peak force of a muscle (Williams and Goldspink 1978) or changes in the elasticity of tendon (Woo et al. 1982). Muscle-tendon models that characterize the effects of pathology, surgery, and other treatment modalities on the muscle force-generating characteristics are needed to verify the accuracy of existing simulations and to enhance the value of new ones.

Perhaps the most profound limitation of the models described in this chapter is their exclusion of central nervous system control. Our analysis of equinus gait did not consider how tendon surgery or postoperative physical therapy might affect muscle-force production through its influence on motor control. The dynamic simulations of stiff-knee gait were performed open loop; that is, the synthesized motions had no ability to modulate the muscle excitation patterns through reflexes, as occurs *in vivo*. Certainly, the incorporation of accurate representations of sensorimotor 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.

Before any model can be used to make treatment decisions, the model must be tested.

Sensitivity studies should be performed to determine whether the conclusions one draws from analysis of a model are sensitive to variations in the model parameters. If possible, simulation results should be compared with experimental data to verify that a particular model is of sufficient complexity to answer the clinical question being posed. Ultimately, controlled clinical studies are required to determine if the insights gained from a model can indeed improve treatment outcomes.

We believe that computer models of the neuro-musculoskeletal system play an important role in the assessment and treatment of gait abnormalities in persons with cerebral palsy. 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.

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