3.6 MODELING AND SIMULATION OF NORMAL AND PATHOLOGICAL GAIT

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The diagnosis and treatment of gait abnormalities in children with cerebral palsy is challenging. A combination of several factors, including muscle spasticity, muscle weakness, bony malalignment, and neurological impairment may contribute to a patient's movement abnormality. In theory, correcting these factors with the appropriate treatment will improve the patient's gait pattern. Identifying the set of factors to target with treatment is difficult, however, since the abnormal gait pattern and set of contributing factors differ between patients. Further, the human body is a complex 3-dimensional linkage, and consequently muscles often have non-intuitive roles during locomotion that are difficult to discern from examining electromyographs (EMG) and joint motions (Fig. 3.6.1). Modeling and simulation of the musculoskeletal system is a powerful tool for quantifying muscle function during pathological gait, which can in turn help identify why a specific patient walks with an abnormal gait, and enable us to design an appropriate treatment plan.

'Modeling', in the context of gait, is a term that often conjures images of 3-dimensional musculoskeletal models and complex dynamic simulations. It is important to recognize that the term 'model' simply refers to a set approximations used to represent a system of interest – in this case, the human body. For example, a model of the musculoskeletal system is needed to perform a conventional gait analysis. The process of inverse dynamics, which calculates joint angles and moments during gait (Davis et al. 1991), requires a model of the body that represents the rotational axes of the joints and the inertial properties of the body segments (i.e. masses and moments of inertia). Recent advances in the field of biomechanics allow us to extend this model in several ways to rigorously define the roles of skeletal alignment, muscle activations, muscle–tendon dynamics, and other factors in generating a normal gait pattern.

We can first extend the model used for inverse dynamics with a representation of 3-dimensional musculoskeletal geometry (Fig. 3.6.1, orange box). We represent each muscle as a path or set of paths between the muscle's origin and insertion, possibly with wrapping surfaces or via points to approximate the more complex geometries of tendon sheaths or overlapping muscles (Delp et al. 1990, Van der Helm et al. 1992). With this addition to the musculoskeletal model, we can calculate muscle moment arms, lengths and velocities as a function of a subject's joint kinematics (Hoffinger et al. 1993, Delp et al. 1996, Schutte et al. 1997, Thompson et al. 1998). This type of model enables us to quantify, for example, the lengths and velocities of a patient's hamstrings muscles to determine if the muscles are

slow or short during gait (Arnold et al. 2006a, b). We can also represent common bony deformities, like femoral anteversion, which alter muscle origins and insertions, and quantify the resulting changes in the moment arms or lengths of muscles (Schutte et al. 1997, Arnold et al. 2001, Arnold and Delp 2001).

As a next step, we can apply forces – along muscle paths, at the foot–ground interface, or from gravity – to the musculoskeletal model and observe the motions that result. In order to link forces to motions, we must formulate the model's equations of motion, which relate the accelerations of all the joints in the body to the forces applied to the body and the inertial parameters, position, and velocity of the body segments (Fig. 3.6.1, yellow box). By representing the action of forces on the body, we can gain valuable insight into the often non-intuitive role of muscles in supporting the body against gravity and propelling the body forward, as illustrated for several important lower extremity muscles in Videos 3.6.1–6. For example, as a consequence of dynamic coupling between joints in the body, muscles can accelerate joints they do not cross (Zajac and Gordon 1989, Riley and Kerrigan 1999, Arnold et al. 2005, Kimmel and Schwartz 2006). This phenomenon is well established for the soleus, often called the plantarflexion/knee-extension couple, as illustrated in Figure 3.6.2 and Video 3.6.6. The action of biarticular muscles also depends on dynamic coupling. For example, the hamstrings muscles may produce a knee-flexion acceleration through the muscles' knee-flexion moment and may generate a knee-extension acceleration through the muscles' hip-extension moment, via dynamic coupling, as shown in Video 3.6.3 (Arnold et al. 2005). The net acceleration the hamstrings generate at the knee depends on the orientation of the body segments and the muscle's hip-to-knee moment-arm ratio, an important consideration when examining the function of this muscle group often implicated in crouch gait. The musculoskeletal model's equations of motion capture the complexity of dynamic coupling and allow us to quantify muscle function in the presence of bony deformities, like excess tibial torsion or femoral anteversion, or in pathological locomotion patterns.

The next level of complexity in biomechanical models is forward-dynamic simulation of a subject's gait, driven by muscle tendon actuators (see Fig. 3.6.1, red box and Video 3.6.7). Dynamic simulations represent muscle activation dynamics, linking muscle activations to forces as a function of a muscle's cross-sectional area, length, and velocity (Zajac 1989). We then use mathematical optimization to find a solution for the set of muscle activations that drives the model to follow a specified gait pattern and is consistent with experimentally measured EMG (Anderson and Pandy 2001, Thelen et al. 2003, Thelen and Anderson 2006). With a muscle-driven simulation of gait, we can assess how changes in a muscle's activation level or timing affect gait kinematics. For example, we can 'turn off' the rectus femoris muscle in the simulation of a child with stiff-knee gait to determine if this muscle inhibits knee flexion in swing phase (Reinbolt et al. 2008). Alternatively, we can increase the force applied by a muscle like the psoas or gastrocnemius in the simulation to determine if strengthening one of these muscles might similarly improve the patient's swing-phase knee flexion (Goldberg et al. 2004).

The musculoskeletal modeling tools described above have the potential to enhance our understanding of muscle function in normal and pathological gait and, in turn, improve the

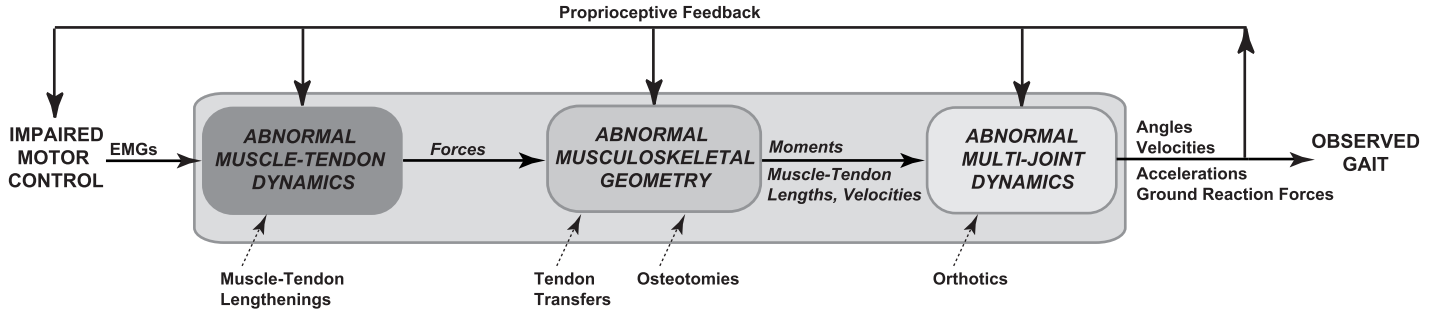

Fig. 3.6.1 Many factors contribute to movement abnormalities in persons with cerebral palsy. Gait analysis is used routinely to record EMG patterns, joint angles, and ground reaction forces during walking, but the transformation between EMG patterns and coordinated multi-joint movement is complex (gray-shaded region). Furthermore, to make treatment decisions clinicians must try to predict how the motions induced by muscles might change after treatment. Typically, treatments alter muscle-tendon dynamics or musculoskeletal geometry, which are difficult changes to measure. Computational models that characterize patients' muscle-tendon dynamics (red box), musculoskeletal geometry (orange box), and multi-joint dynamics of the body (yellow box) during walking may enhance the interpretation of motion analysis studies and improve the planning of treatments.

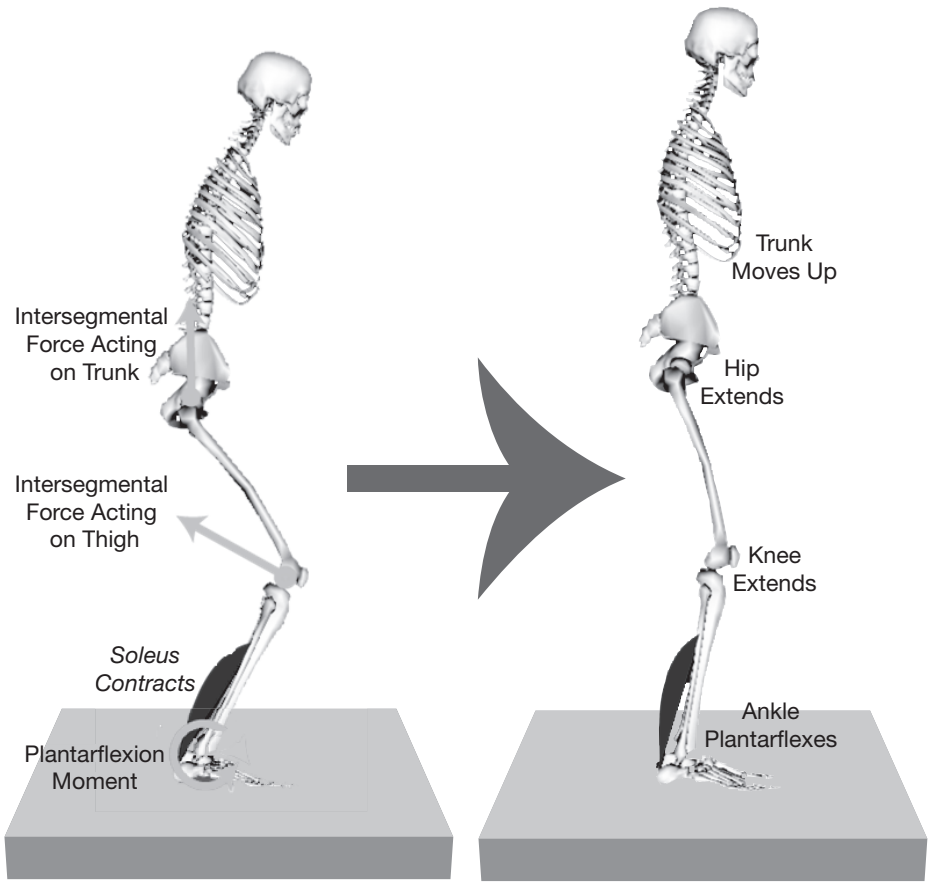

Fig. 3.6.2 Action of soleus as a result of dynamic coupling. *Left*: The force applied by soleus, a uniarticular muscle spanning the ankle, not only generates an ankle plantarflexion moment (bottom orange arrow), but also induces intersegmental forces throughout the body. The magnitudes and directions of these intersegmental forces depend on the force applied by the muscle, the moment arms of the muscle, the inertial properties of the segments, and the configuration of the body. In this example, the force applied by soleus produces a counter-clockwise angular acceleration of the shank. This acceleration requires the location of the knee joint to accelerate to the left and upward. The inertia of the thigh resists this acceleration, resulting in an intersegmental force at the knee (middle orange arrow). The intersegmental force at the knee accelerates the thigh, which in turn induces an intersegmental force at the hip (top orange arrow), and so on. *Right*: As a consequence of the intersegmental forces induced by soleus, the muscle accelerates not only the ankle, but all the joints of the body. At the body position shown, soleus accelerates the ankle toward plantarflexion, the knee toward extension, the hip toward extension, and the trunk upward. Over time, these accelerations give rise to changes in position. Thus, due to dynamic coupling, soleus does not function solely as an 'ankle plantarflexor' – in many situations, it does much more. In a similar fashion, other muscles induce intersegmental forces and accelerate joints that they do not span. (Figure adapted from Anderson et al. 2006.)

diagnosis and treatment of patients with gait disorders. Several challenges must be overcome to ensure these tools are useful in a clinical setting. First, although new freely available modeling tools (Delp et al. 2007) improve access to simulation, and increased computer processor speed reduces the time and cost required to generate muscle-driven dynamic simulations, creating a subject-specific simulation of each child who visits a gait analysis lab may not be practical. Second, expertise is required to interpret the results of any biomechanical analysis, since simulations cannot yet predict how a patient will walk after treatment. We can use several strategies to mitigate these concerns. First, we must use models of appropriate complexity to answer carefully posed questions about the cause of a particular gait abnormality or the consequence of a treatment. For example, we can determine if a muscle is slow or short during a patient's gait using a purely kinematic model – in this case we do not require a formulation of the model's equations of motion or a representation of muscle activation dynamics. Second, we must continue to validate our models and translate the insights gained from simulations into screening techniques that can be applied in a clinical setting. In the sections that follow, we discuss three clinical applications of modeling and simulation that utilize these principles. We conclude with a discussion of the remaining challenges for the modeling and simulation of pathological gait that will drive continued research.

Kinematic models to estimate hamstrings length and velocity in crouch gait

Many children with cerebral palsy walk with excessive knee flexion in terminal swing and stance, a movement pattern known as crouch gait. Spasticity or contracture of the hamstring muscles is thought to restrict knee extension, thus contributing to the crouch gait of some patients (Baumann et al. 1980, Sutherland and Davids 1993, Crenna 1998, Tuzson et al. 2003). Hamstrings lengthening surgery is commonly prescribed to address a patient's diminished knee extension during gait. While some patients demonstrate improved knee kinematics, stride length, and locomotor efficiency after a hamstrings lengthening procedure (DeLuca et al. 1998, Abel et al. 1999), other patients fail to adopt a more erect gait posture after surgery or may experience negative complications, including the development of excessive anterior pelvic tilt or a stiff-knee gait pattern (Thometz et al. 1989, Hsu and Li 1990, DeLuca et al. 1998). The variable success of this procedure provides an impetus to better identify good candidates for hamstrings lengthening and understand the mechanisms of improvement after surgery. Common clinical indications for a hamstrings lengthening include excessive knee flexion during gait and/or a large popliteal angle; however, these measures do not directly assess whether a patient's hamstrings are short or slow during gait and are thus inhibiting the patient's knee extension. In a series of investigations, we examined whether analyses of the muscle-tendon lengths and lengthening velocities of patients' hamstrings during walking can help determine if a patient is likely to benefit from hamstrings surgery (Arnold et al. 2006a, b).

To address the utility of a musculoskeletal model in identifying good candidates for hamstrings lengthening surgery, we retrospectively analyzed the muscle-tendon lengths and velocities of 152 subjects treated at two different clinical centers for crouch gait (Arnold et al. 2006a). We estimated the subjects' preoperaitve and postoperative muscle-tendon

lengths and velocities by combining kinematic data from a standard gait analysis with a 3-dimensional model of the lower extremity (Fig. 3.6.3). The lower-extremity model represented the geometry of the pelvis, femur, tibia, and the hip and knee joints, as well as the 3-dimensional paths of the hamstrings muscles (Arnold et al. 2001). We calculated the distance along the modeled path of the semimembranosus from origin to insertion at every 2% increment of the gait cycle for each subject to estimate the hamstrings' lengths and calculated the rate of change of this distance to estimate the hamstrings' lengthening velocity. We normalized the muscle-tendon lengths and velocities based on the average peak values measured in normal gait, which eliminated variations due to subject size (Arnold et al. 2001). To examine the relationship between hamstrings length and velocities and the outcome of surgery, we cross-classified subjects in a series of multi-way contingency tables based on the subjects' preoperative and postoperative gait kinematics, preoperative and

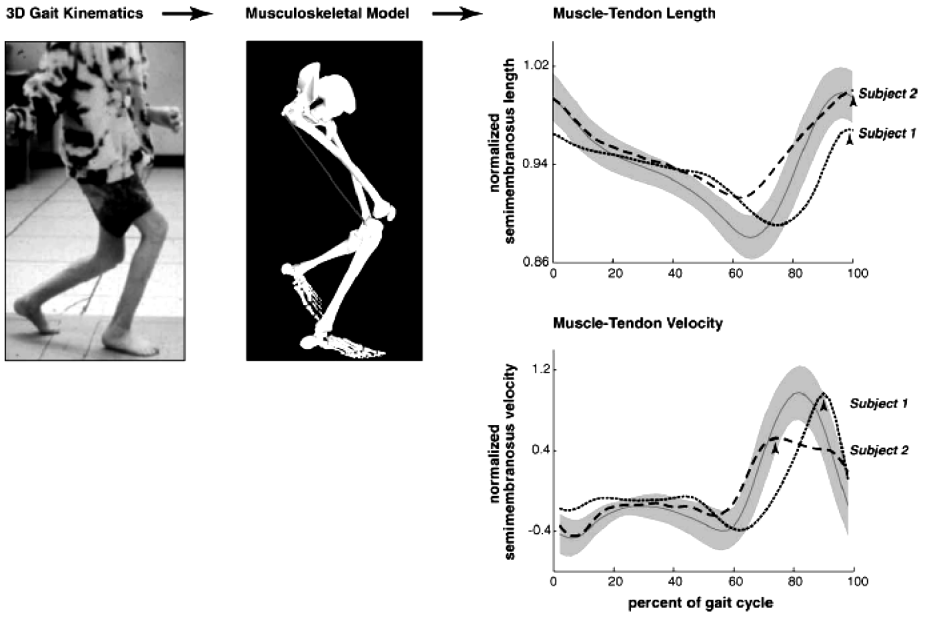

Fig. 3.6.3 Estimations of subjects' semimembranosus muscle-tendon lengths and velocities during walking. A computer model of the lower extremities (*center*) was used in combination with the subjects' joint angles and stride durations measured during gait analysis (*left*) to plot the muscle-tendon lengths and lengthening velocities of the semimembranosus vs gait cycle (*right*). The muscle-tendon lengths and velocities corresponding to each subjects' gait (e.g. Subjects 1 and 2, dashed lines) were normalized and were compared to the lengths and velocities averaged for unimpaired individuals (mean ± 2 S.D., shaded region) to determine if the subjects' hamstrings were operating at peak muscle-tendon lengths shorter than normal, or peak muscle-tendon velocities slower than normal (peak lengths and velocities indicated by arrows). Some of the subjects, such as Subject 2, walked with semimembranosus velocities that were substantially slower than normal. Such analyses may help to distinguish patients who have 'short' or 'spastic' hamstrings from those who do not, and thus may augment conventional methods used to describe patients' neuromusculoskeletal impairments and gait abnormalities. (Figure from Arnold et al. 2006.)

postoperative hamstrings lengths and velocities, and whether they received a hamstrings lengthening as part of their treatment. Using a log-linear analysis, we assessed whether subjects' outcomes were related to their length/velocity classifications (i.e. were the hamstrings short or slow before treatment and longer or faster after treatment?) and/or surgery classifications (i.e. did the subject receive a hamstrings lengthening?)

Our analysis of a diverse group of subjects treated for crouch gait at two leading clinical centers revealed that subjects with hamstrings that were neither short nor slow were less likely to exhibit improved knee kinematics after a hamstrings lengthening procedure (Arnold et al. 2006a). Further, the subjects who received an 'unnecessary' hamstrings surgery tended to have unimproved or worsened pelvic tilt. Within the subset of subjects who had improved knee extension after surgery, patients with short or slow hamstrings preoperatively tended to have longer or faster hamstrings after surgery (Arnold et al. 2006b). Evidence from prior experimental studies suggests that, in some instances, excessive passive forces generated by the hamstrings contribute to excessive knee flexion (Abel et al. 1999, Buczek 2002). Our examination of hamstrings length during gait, before and after surgery, corroborate these prior studies and suggest that surgically lengthening short hamstrings may improve knee extension by allowing these muscles to operate at increased lengths. Similarly, previous studies suggest that in some instances abnormal hamstrings excitation, triggered by spasticity, may limit the lengthening velocity of the hamstrings during gait (Perry and Newsam 1992, Crenna 1998, Granata et al. 2000, Tuzson et al. 2003). Our investigation of hamstrings velocities support these findings and suggest that surgical lengthening of 'spastic' hamstrings may allow them to elongate with greater muscle-tendon velocities.

One argument against using an additional modeling step to calculate muscle-tendon lengths and velocities is that perhaps the same conclusions could be drawn from a subject's conventional gait analysis or clinical exam data. However, the presence of short or slow hamstrings was not evident from standard measures like hip or knee kinematics and popliteal angle (Arnold et al. 2006a). Since the estimates of muscle-tendon length used in our studies are based on a detailed model of musculoskeletal geometry and joint kinematics, they simultaneously account for muscle moment arms at the hip and knee, as well as the patient's 3-dimensional gait kinematics during walking.

In these studies, we demonstrated that a musculoskeletal model can help to identify subjects with abnormally short or slow hamstrings, and thus augment conventional gait analysis in identifying appropriate candidates for surgery. Estimating muscle-tendon lengths and velocities with a relatively simple modeling framework helped explain the functional consequences of surgery – we observed that in successful outcomes, the hamstrings operate at longer lengths or faster speeds, enabling more normal knee kinematics. A limitation of this type of analysis is that it does not allow us to estimate active or passive muscle-tendon forces. Further work, using muscle-driven dynamic simulations, is needed to quantify hamstrings activation and the joint accelerations this muscle group produces in both normal and pathological gait. We also recognize that multiple factors may contribute to the crouch gait of each subject. For example, muscle weakness or poor skeletal alignment may prevent improvement after a hamstrings surgery even if the length and velocity of the hamstrings are corrected to normal values. Thus the focus of the investigation in the next section is the

contribution of bony deformities and crouched walking postures to the diminished knee extension observed in patients with crouch gait.

Induced acceleration analysis to quantify muscle function in subjects with crouch gait and tibial torsion

The excessive knee flexion exhibited by children with crouch gait tends to worsen over time if patients do not receive appropriate treatment (Sutherland and Cooper 1978, Bell et al. 2002). This chronic knee flexion increases the energy costs of walking (Campbell and Ball 1978, Waters and Lunsford 1985, Rose et al. 1990) and can lead to knee joint degeneration (Rosenthal and Levine 1977, Lloyd-Roberts et al. 1985, Bleck 1987). As discussed in the previous section, spasticity or contracture of the hamstrings may contribute to the excess knee flexion observed in some patients. Bony deformities like tibial torsion, which alter the dynamic coupling between joints (Fig. 3.6.2), may also contribute to crouch gait by reducing the capacity of muscles to accelerate the joints into extension, as suggested by several previous investigators (Stefko et al. 1998, Schwartz and Lakin 2003, Selber et al. 2004, Ryan et al. 2005). Correcting the deformity with a tibial derotation osteotomy is thought to improve bony alignment and help to restore normal muscle function (see Chapter 5.2 for a clinical discussion of tibial torsion and derotation surgery), but this hypothesis has not been fully validated, nor is this surgery always successful. Simply walking in a crouched posture may also alter dynamic coupling and the joint angular accelerations generated by muscles during gait – changes that must be quantified to understand the implications of muscle weakness or abnormal activation. In the next set of investigations, we quantified the effect of crouched gait postures and the presence of a tibial deformity on the capacity of the lower-extremity muscles to extend the hip and knee joints (Hicks et al. 2007, 2008).

As in the previous section, we performed our analysis with a 3-dimensional model of the musculoskeletal system (Fig. 3.6.4) that represented the geometry of the joints and the paths of muscles in the lower extremity (Delp et al. 1990). The model in this study had 10 segments to represent the upper body and right and left legs, and 13 degrees of freedom to represent the articulations at the back, hip, knee and ankle joints. We included 92 muscles paths in the model, but focused our analysis on the major muscles capable of extending the hip or knee, including gluteus maximus, gluteus medius, vasti, hamstrings, and soleus (Arnold et al. 2005). Since we also sought to quantify the effect of tibial deformities on the joint accelerations induced by muscles, we developed a technique to simulate a torsional deformity of the tibia (Fig. 3.6.4, highlighted region), which modified muscle attachments sites and the relative orientation of the knee and ankle joints (Hicks et al. 2007). To calculate the joint accelerations that result from applying muscle forces or gravity to the body, we represented the inertial parameters of the body segments in the model (Delp et al. 1990) and the interaction between the foot and the ground (Anderson and Pandy 2003). By specifying these additional model properties, we were able to formulate the dynamic equations of motion which relate muscle forces to joint accelerations as a function of the body's position (Delp and Loan 2000).

To quantify the effect of tibial torsion and crouched postures on the capacity of muscles to extend the hip and knee, we used a technique commonly referred to as an induced

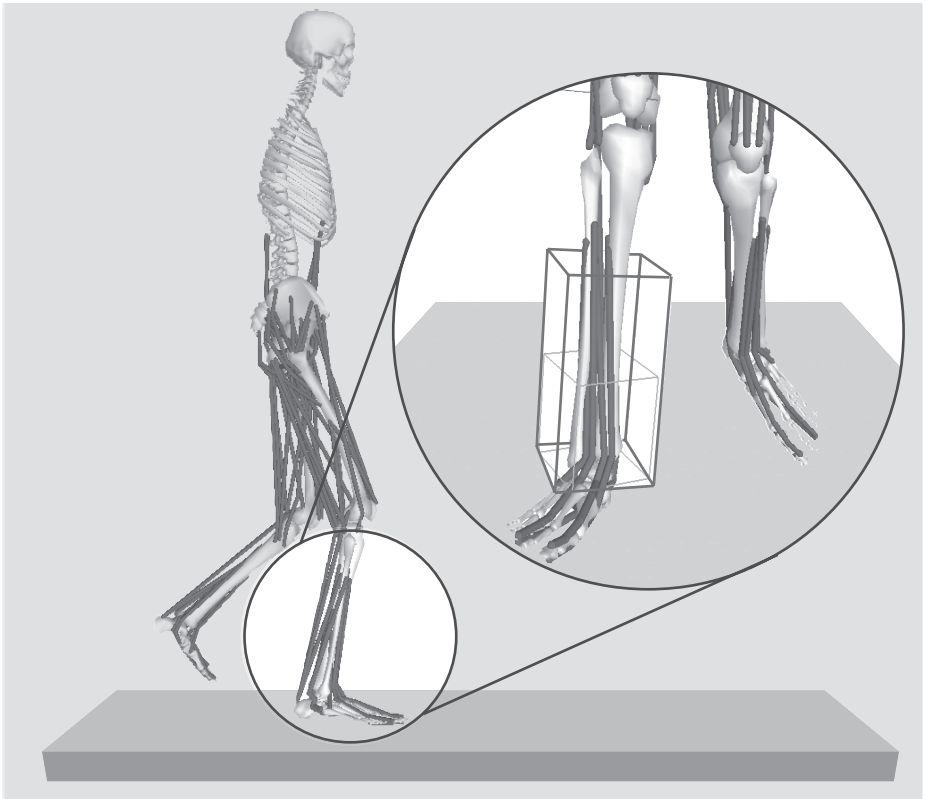

Fig. 3.6.4 Three-dimensional model of the musculoskeletal system used to determine the effect of tibial torsion deformities and crouched postures on muscle extension capacities. The model had 10 segments, 13 degrees of freedom, and 92 muscle paths. The model is shown with joint angles corresponding to the beginning of single limb support for a crouch gait. We added a deformable tibia to the model to simulate a range of torsional deformities (highlighted region). This deformity was implemented using two boxes (pink and blue): the inner box (pink), the ankle axis, and the foot were rotated by the torsion angle specified. There was a linear decrease in tibial torsion angle between the top of the inner box and the top of the outer box (blue). All bone deformation was distal to the proximal muscle attachments on the tibia.

acceleration analysis (Zajac and Gordon 1989). We positioned the musculoskeletal model with the joint angles from a normal or crouch gait pattern (Videos 3.6.8–10) and applied, in turn, a 1 Newton muscle force along the path of each of the lower-extremity muscles. Using the model's equations of motion, we then calculated the resulting accelerations of the knee and hip joint. These induced accelerations represent the *capacity* of each individual muscle to accelerate the joint towards extension, independent of the muscle's cross-sectional area, activation level, muscle-tendon length, or lengthening velocity. We similarly applied the force of gravity to each of the body segments and calculated the joint accelerations induced by gravity. To quantify the effect of tibial torsion on muscle extension capacities, we repeated this analysis for models with a range of excess, external torsion.

Analyzing the dynamics of the musculoskeletal model revealed that a crouched gait posture reduces the capacity of several major muscles to extend the knee (Hicks et al. 2008), including gluteus maximus, posterior gluteus medius, vasti and soleus (Fig. 3.6.5, white bars). The exception was the hamstrings muscle group, whose extension capacity was

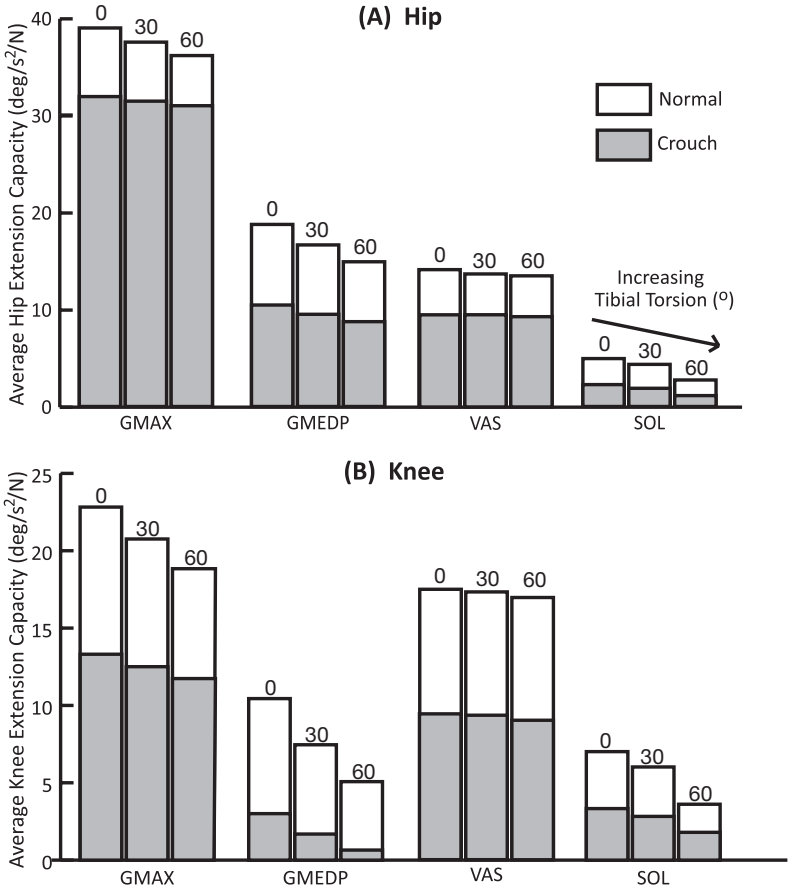

Fig. 3.6.5 The effect of external tibial torsion and crouched gait postures on the capacity of muscles to extend the hip (A) and knee (B) during single limb stance. The muscles shown, including gluteus maximus (GMAX), the posterior compartment of gluteus medius (GMEDP), vasti (VAS), and soleus (SOL), are the major muscles responsible for generating hip and knee extension accelerations in the single support phase of gait. The white bars show the average extension acceleration during normal single limb stance for each muscle in a model with 0°, 30° and 60° of excess, external tibial torsion. The gray bars show the average extension capacity during a representative moderate crouch gait for a model with 0°, 30° and 60° of excess tibial torsion. The capacity of all of these muscles to extend the hip and knee joint was substantially reduced when the model was positioned in a crouched gait posture. A torsional deformity of the tibia also reduced the capacity of several of these muscles, in particular the posterior gluteus medius and soleus, to extend the hip and knee joints. (Figure from Hicks et al. 2008.)

maintained in a crouched posture. Crouched gait postures also increased the flexion accelerations induced by gravity at the hip and knee. Several important muscles crossing the hip and knee were adversely affected by excessive external tibial torsion (Fig. 3.6.5, gray bars) when walking in either a normal or crouched gait posture (Hicks et al. 2007, 2008). For example, with an external tibial torsion deformity of 30°, the capacities of soleus, posterior gluteus medius, and gluteus maximus to extend both the hip and knee were all reduced by over 10% from the values calculated for an undeformed model.

The negative impact of both crouched postures and tibial deformities increased with the severity of the impairment, suggesting that crouch gait is a downward cycle. In a crouched posture, the joint-flexion accelerations induced by gravity increase and the capacity of muscles to generate joint-extension accelerations decrease, so an individual in a crouch gait must generate more muscle force to maintain a crouched posture. This increase in required muscle force is consistent with experimental studies that show greater muscle activity in flexed postures (Hsu et al. 1993) and reports of increased energy expenditure for crouch gait (Campbell and Ball 1978, Waters and Lunsford 1985, Rose et al. 1990). Larger muscle forces also increase joint loading, a likely contributor to knee abnormalities, like patella alta, observed in patients with crouch gait (Rosenthal and Levine 1977, Lloyd-Roberts et al. 1985). A tibial deformity tended to have the greatest impact on the soleus and gluteal muscles, which suggests that the deformity may be particularly deleterious in patients with pre-existing weakness of the gluteal or plantarflexor muscles. More optimistically, the findings of this study suggest that small improvements in gait posture, as a result of physical therapy or surgery, may help to reverse the natural progression of crouch gait. For example, our findings suggest that correcting a patient's tibial alignment may lead to small improvements in the capacity of the subject's muscles to extend the joints, which may lead to a more erect posture, and thus further improvement in muscle extension capacities.

The dynamic analysis used in these studies can be adapted to analyze the gait kinematics and bone geometry of individual patients to help estimate, for example, the contribution of a patient's tibial deformity to his or her crouch gait. This analysis requires a minor amount of additional computation beyond conventional inverse dynamics and may help to identify patients whose crouch gait would improve with targeted strength training or from surgery to correct bony malalignment. A notable restriction of this type of analysis is that it only allows us to calculate the capacity or potential of a muscle to accelerate the joints. The actual accelerations produced by a muscle also depend on its activation level, cross-sectional area, length, and velocity. The forces generated by muscles via activation during gait will be addressed in the next section on muscle-driven simulation.

Muscle-driven forward-dynamic simulations to understand stiff-knee gait

Many individuals with cerebral palsy walk with diminished knee flexion in the swing phase of gait, a movement abnormality known as stiff-knee gait. The insufficient knee flexion observed in these patients is often attributed to excessive swing-phase activation of the rectus femoris (Waters et al. 1979, Perry 1987, Sutherland et al. 1990), a biarticular muscle that generates a flexion moment at the hip and an extension moment at the knee. Stiff-knee gait is commonly treated with a rectus femoris transfer, a surgery in which the distal tendon

of the muscle is detached from the patella and reattached to a site posterior to the knee (Gage et al. 1987, Perry 1987). The surgical transfer is thought to convert the rectus femoris to a knee-flexor (see Chapter 5.2 for a clinical discussion of the rectus femoris transfer), thereby eliminating the excessive swing-phase knee-extension moment produced by the muscle. The outcome of this procedure is sometimes unsuccessful and research by Asakawa and colleagues (2004) suggested that scarring after the transfer surgery may prevent the rectus femoris from acting as a knee-flexor. Analysis of the multi-joint motions produced by a muscle like rectus femoris is complex, but there is a strong clinical motivation to understand the possible contribution of abnormal rectus femoris activity to stiff-knee gait. We have developed forward dynamic simulations of able-bodied subjects and persons with stiff-knee gait to determine how the forces generated by rectus femoris and other muscles influence knee flexion during swing (Goldberg et al. 2003, 2004, Reinbolt et al. 2008). Quantifying the factors that influence knee flexion in swing can help us understand the biomechanical consequences of the rectus transfer procedure and identify appropriate candidates for surgery.

In this series of investigations, we generated muscle-actuated simulations of gait for both able-bodied individuals and children who walked with diminished swing-phase knee flexion. An example of a muscle-driven simulation of able-bodied gait is shown in Video 3.6.7. As in the previous section, we began with a computer model of the musculoskeletal system that represented the three-dimensional geometry of the bones and joints and paths of the muscle-tendon units (Delp et al. 1990, Anderson and Pandy 1999). We then scaled the model to represent the experimentally measured size of the subject and generated the model's equations of motion, which relate applied forces to joint accelerations (Delp and Loan 2000). To create muscle-driven simulations, we represented the muscle-tendon units as actuators that generate forces as a function of the muscle's activation level, length, and velocity (Zajac 1989, Delp et al. 1990). We then solved for the muscle activations that enabled the model to follow the experimentally observed joint trajectories of the subject (Fig. 3.6.6). The muscle activations were determined using a mathematical optimization that minimized the difference between the measured gait kinematics and the motion of the model, while remaining consistent with experimentally-measured EMG (Anderson and Pandy 2001, Thelen et al. 2003, Thelen and Anderson 2006). Once this subject-specific simulation was generated, we made systematic changes to the simulation to address specific questions relevant to stiff-knee gait. For example, in one investigation, we altered the flexion velocity of the knee joint immediately prior to swing to quantify the effect of this parameter on peak knee flexion (Goldberg et al. 2003). We additionally examined the effect of perturbing the activation level or timing of the rectus femoris and other muscles to quantify the joint accelerations generated by the lower-extremity muscles during the double support and swing phases of gait (Goldberg et al. 2004, Reinbolt et al. 2008).

In our first simulation-based study of stiff-knee gait, we investigated the validity of one of the most common clinical indications for rectus transfer surgery: overactivation of the rectus femoris during early swing, which is thought to create excessive knee extension moments that restrict swing-phase knee flexion. Analysis of a large group of patients with stiff-knee gait demonstrated that these subjects do not exhibit larger than normal knee

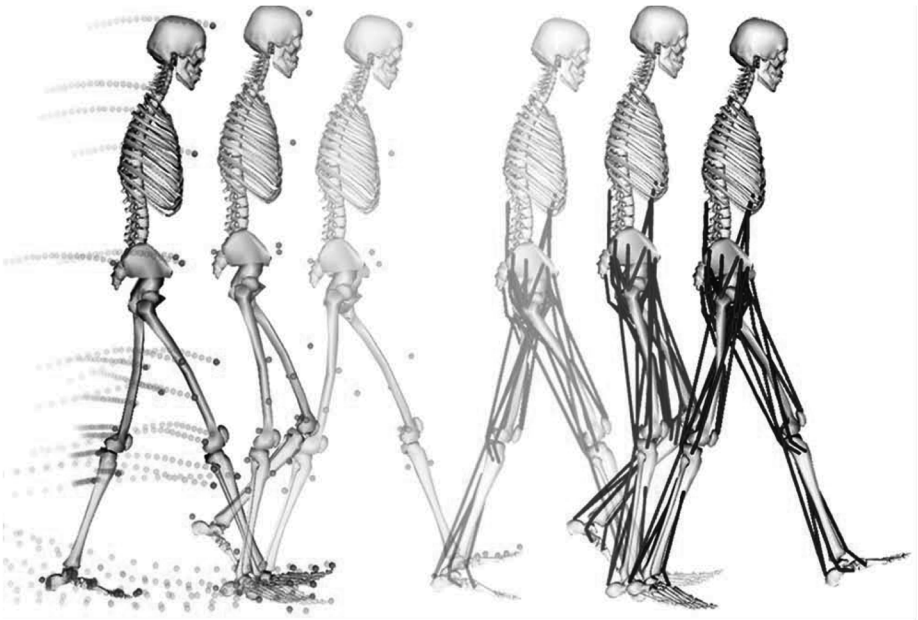

Fig. 3.6.6 Process for computing muscle activations in a dynamic simulation. A 3-dimensional, full-body musculoskeletal model with 21 degrees of freedom and 92 muscle-tendon actuators was used in conjunction with the subject's gait analysis data to create each subject-specific simulation. We scaled the body segment lengths and inertial properties for a generic model according to distances on the model and corresponding measures on the subject from experimental markers. We then used numerical optimization to solve for the set of muscle activations that drove the model to follow the subject's experimentally measured gait kinematics. The muscle excitations were constrained to be consistent with measured EMG patterns. The resulting simulations produced joint motions that were within a few degrees of measured joint motions.

extension moments in swing, but rather show reduced knee-flexion velocities at toe-off (Goldberg et al. 2003). This suggests that abnormal muscle activity prior to swing phase reduces the velocity of the knee going into swing, which results in the diminished and delayed knee flexion observed in these subjects. Our simulations of stiff-knee gait support this hypothesis. In particular, increasing subjects' knee flexion velocity at toe-off to normal values in the simulations increased the knee range of motion for all subjects by at least 7°. This first set of simulations indicated that the mechanism for improvement after rectus transfer surgery may be an alteration of rectus femoris function in pre-swing, which could improve knee flexion velocity at toe-off. Thus the focus of our next investigation was the influence of the rectus femoris and other lower-extremity muscles on knee flexion velocity at toe-off.

To establish a baseline for muscle function in pre-swing, we quantified the influence of each lower-extremity muscle on peak knee-flexion velocity in the double support phase of normal gait (Goldberg et al. 2004). Starting from a normal simulation of the double support phase, we systematically perturbed the force level in each muscle from its normal value.

We then stepped forward in time via numerical integration and observed the resulting peak knee-flexion velocity. We compared the perturbed and unperturbed simulations to determine the effect of increasing or decreasing a given muscle force on the peak knee-flexion velocity achieved. As expected, we found that increasing the force in the rectus femoris during double support decreased peak knee-flexion velocity (Fig. 3.6.7). Several other muscles had a significant impact on knee flexion as well. Increased force by the vasti or soleus had the effect of reducing peak knee-flexion velocity. In contrast, increased force by iliopsoas, gastrocnemius or hamstrings increased the peak knee-flexion velocity achieved.

This investigation of muscle function in normal gait has several clinical implications. First, the results of this analysis provide further evidence that diminished knee-flexion velocity at toe-off and abnormal pre-swing activation of rectus femoris may be an appropriate indication for surgery. These findings also shed light on why many patients treated for crouch or equinus gait subsequently develop a stiff-knee gait pattern (Thometz et al. 1989, Damron et al. 1993). Musculotendinous lengthening of the hamstrings or gastrocnemius muscles is commonly performed on these patients and may diminish the muscle's force-generating capacity, and thus its ability to create a knee-flexion moment during double support. Alternately, targeted strengthening of the iliopsoas, gastrocnemius, or hamstrings muscles may benefit many patients with stiff-knee gait.

To explore the contribution of abnormal pre-swing rectus femoris activation to diminished swing-phase knee flexion, we next examined a series of simulations of patients with stiff-knee gait (Reinbolt et al. 2008). In each subject-specific simulation, we eliminated the activation of the rectus femoris first during the pre-swing period, and then during the swing period, and observed the resulting peak knee-flexion angle in swing (see Fig. 3.6.8

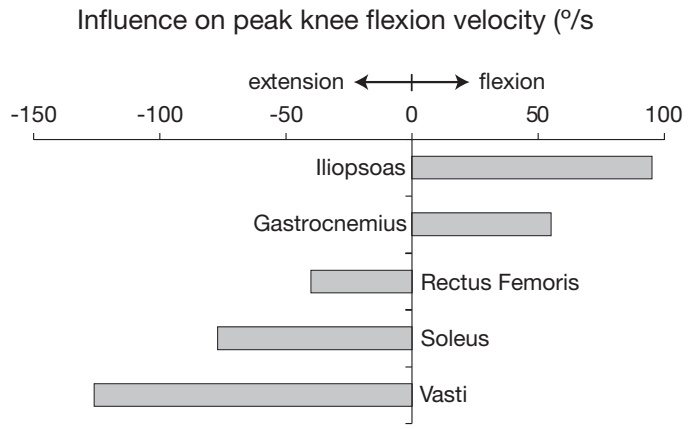

Fig. 3.6.7 The influence of selected muscles on peak knee-flexion velocity during double support. The influence of each muscle was calculated as the slope of the plot of change in peak knee-flexion velocity vs perturbation size in the simulation. The influence is a function of both the potential of each muscle to induce knee flexion velocity and the force exerted by the muscle during the simulation. Iliopsoas and gastrocnemius contributed the most to peak knee flexion during double support. Forces in vasti, rectus femoris and soleus decreased the knee-flexion velocity during double support. All other muscles had influences of less than 26°/s. (Figure from Goldberg et al. 2004.)

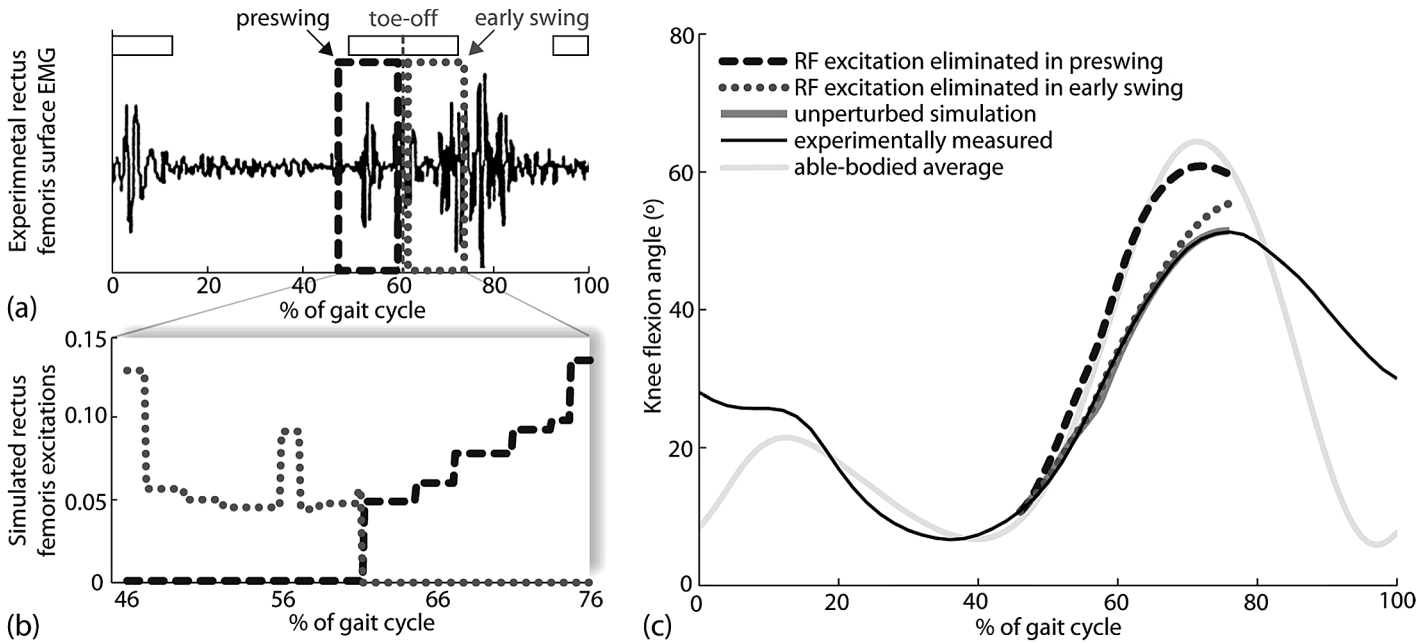

Fig. 3.6.8 Example of methods used to determine increase in peak knee-flexion angle in swing when rectus femoris activity was eliminated during pre-swing and separately during early swing. (a) Rectus femoris surface EMG of a subject with stiff-knee gait was recorded over an entire gait cycle. Normal rectus femoris EMG is indicated by horizontal white bars. Toe-off is indicated by a vertical dashed line at 61% of the gait cycle. Two time periods were selected for analysis: early swing (i.e. the period from toe-off to peak knee flexion) and pre-swing (i.e. the period before toe-off equal in duration to early swing). (b) Two simulation experiments were conducted by eliminating rectus femoris activity during pre-swing (dashed line) and separately during early swing (dotted line) to determine the muscle's effect on peak knee flexion. (c) Simulated changes in knee-flexion angles were different when rectus femoris activity was eliminated during pre-swing (dashed line) or early swing (dotted line). The unperturbed simulation (thick solid line) and experimentally measured (thin solid line) knee angles are shown for comparison. Normal knee flexion (shaded line) and 2 S.D. of the normal curve (shaded region) are shown as well. (Figure from Reinbolt et al. 2008.)

and Video 3.6.11). We found that eliminating abnormal rectus activation in either pre-swing or swing increased the peak knee flexion achieved during subjects' gait simulations. Improvements in peak knee flexion tended to be larger when subjects' pre-swing rectus activity was eliminated. These results confirm that pre-swing rectus femoris activity is at least as important as early swing activity and, for many subjects with stiff-knee gait, may limit knee flexion more than activity in early swing. Indeed, patients with a good surgical outcome tend to show an increased knee-flexion velocity at toe-off and a decrease in their knee-extension moment during double support (Goldberg et al. 2006). Our muscle-driven simulations have demonstrated that in evaluating rectus femoris activity for treatment of stiff-knee gait, both pre-swing and early swing EMG should be examined.

Although these studies provide valuable insight into the contribution of rectus femoris to stiff-knee gait, the mechanism by which patients improve after a rectus femoris transfer is still unclear. The transfer was originally intended to convert the rectus to a knee flexor, but several studies have shown that instead the muscle's knee extension capacity is merely reduced (Riewald and Delp 1997, Asakawa et al. 2002), possibly as a result of scarring between the rectus femoris and underlying vasti (Asakawa et al. 2004). An alternate mechanism of improvement is the reduction of the muscle's knee-extension moment, with preservation of its hip-flexion moment. We have begun a preliminary study to understand how surgical technique or scarring may influence outcome and to investigate the relative importance of the hip- and knee-moment arms of a transferred rectus (Fox et al. 2009). We created muscle-driven simulations of ten children with stiff-knee gait and altered the simulations to represent transfer of the rectus femoris to the sartorius or the iliotibial band, with and without scarring. Our preliminary results suggest that, while all surgeries improve the peak knee flexion observed in a simulation, scarring reduces the magnitude of improvement. For all procedures, the primary mechanism for improvement was the reduction of the knee-extension moment generated by rectus femoris, with a small additional benefit provided by maintenance of the muscle's hip flexion moment. While gait analysis tools alone are useful for characterizing stiff-knee gait, this series of modeling studies has demonstrated that dynamic simulation is an additional valuable tool to explore the underlying biomechanical causes of diminished knee flexion and the mechanisms leading to improvement after treatment.

Discussion and future directions

Modeling and simulation of the musculoskeletal system can provide insights into the pathomechanics of gait abnormalities and the functional consequences of treatments, as evidenced by the three examples presented in this chapter. The series of investigations we have described provide general guidelines, based on biomechanical principles, that can be used in combination with the data collected during a gait analysis to more rigorously identify the potential causes of an individual subject's movement disorder and more effectively plan treatment. For example, the methods used to estimate hamstrings lengths and velocities in the first investigation are currently used in several clinical centers to determine if a patient has short or slow hamstrings during gait, and thus may benefit from a lengthening procedure. Similarly, our analysis of models with tibial torsion deformities suggests that excess torsion

may contribute to a patient's diminished knee extension when his or her external torsion is 30° or larger than normal. Our study of stiff-knee gait has also provided clinically useful guidelines. While one of the common clinical indications for a rectus transfer is over-activation in the swing phase, our simulations suggest that pre-swing rectus activity, which can reduce the flexion velocity of the knee joint at toe-off, may be a more important parameter in selecting appropriate candidates for surgery. In addition to these general guidelines, advances in technology and computational algorithms continue to decrease the time required to generate simulations of the pathological gait of individual subjects. Still, 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 of the important issues to be resolved in future studies are outlined below.

First, we must continue to refine and validate our models of the musculoskeletal geometry of children with cerebral palsy. The results of modeling and simulation studies, like the three examples presented in this chapter, are often sensitive to parameters like bony alignment, joint geometry, or muscle origin and insertion sites, which can alter muscle moment arms and muscle lengths and velocities during movement. Our analyses typically begin with a generic model of the musculoskeletal system that is scaled based on a subject's experimentally measured body size. We have developed techniques to model common bone deformities, like tibial torsion (Hicks et al. 2007) or femoral anteversion (Arnold et al. 2001), which deform the scaled musculoskeletal model based on clinically measured bony alignment. We can similarly modify generic models to simulate osteotomies (Free and Delp 1996, Schmidt et al. 1999) and tendon transfer surgeries (Delp et al. 1994). To validate our technique for modeling femoral anteversion, we compared the muscle-tendon lengths estimated by our deformable model to lengths determined from magnetic resonance images of four subjects with cerebral palsy (Arnold et al. 2001). We found very good agreement, with most length differences less than 5 mm in magnitude. Similar imaging studies are warranted to determine if the current modeling framework accurately represents variations due to other deformities, like tibial torsion or patella alta, or differences as a consequence of age, gender, or surgical treatment. In general, creating image-based models for all subjects is not likely to be practical, so we instead suggest a continued refinement of the hybrid approach. In particular, we recommend the use of subject-specific models that incorporate multi-dimensional scaling and algorithms for deforming bones, muscles, or joints based on a few patient-specific parameters from imaging or experimental measures (Chao et al. 1993, Arnold et al. 2001, Arnold and Delp 2001, Hicks et al. 2007).

The model of muscle-tendon mechanics used in simulations must also be further tested. While the current model captures many features of force generation in unimpaired subjects, it does not account for changes that may occur in neuromuscular disorders like cerebral palsy. 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). In addition, 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) due to pathology or treatment.

Muscle-tendon models that characterize the effects of pathology, surgery, and other treatment modalities on muscle force-generating characteristics are needed to verify the accuracy of existing simulations and to enhance the value of new ones. A minimally invasive microendoscopy technique for imaging muscle (Llewellyn et al. 2008) may improve models of muscle-tendon mechanics in children with neuromuscular pathologies like cerebral palsy. This new imaging modality allows high-speed, real-time imaging of sarcomere dynamics in vivo, which will allow us to investigate how muscle architecture and dynamics change as consequence of pathology or treatment.

Perhaps the most profound limitation of the models described in this chapter is their exclusion of central nervous system control. For example, our analysis of the effect of tibial torsion on the capacity of muscles to extend the knee and hip joints did not consider how the nervous system may modulate muscle activation to compensate for this deformity. Additionally, 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. In general, the central nervous system may adapt to compensate for poor balance, muscle weakness, or in response to surgery or other treatment; however, our current modeling and simulation framework does not account for these adaptations. The incorporation of an accurate representation of sensorimotor control into dynamic simulations of abnormal movements is one of the critical challenges for developing models that can accurately simulate the outcome of treatment.

Continued work is also needed to ensure that the results generated by musculoskeletal simulations are accurate and clinically relevant. Sensitivity studies are valuable, allowing us to determine when the results of a particular analysis are strongly dependent on model parameters like joint geometry or muscle moment arms. Simulation results should be compared with experimental data to verify that a particular model is of sufficient complexity to answer the question posed. It is also important to examine whether the surgical recommendations provided by modeling and simulation studies lead to positive treatment outcomes in patients. To this end, we have begun a retrospective analysis of a large database of patients treated for crouch gait to determine if correcting the factors – like muscle spasticity, muscle weakness, or bony deformities – which contribute to a patient's excessive knee flexion, as determined by modeling and simulation, leads to good surgical outcomes. Since subject-specific modeling of each subject who visits a gait lab may not be practical, we have also begun to develop statistical models that use readily available preoperative variables from a subject's clinical exam or gait analysis to predict the likelihood that a particular treatment will be successful. The quantity of available preoperative data is prohibitively large, so we are using the knowledge gained in previous modeling and simulation studies to define a smaller subset of preoperative variables that enable robust predictions of surgical outcome and 'make sense' in terms of standard clinical and biomechanical knowledge. To ensure that computer modeling and simulation is accessible to researchers and clinicians, we have developed a freely available software package for musculoskeletal modeling and dynamic simulation, called OpenSim (Delp et al. 2007). This software is currently used by over 1000 researchers for a wide variety of applications, including the study of pathological gait.

We believe that computer models of the neuromusculoskeletal system play an important role in the assessment and treatment of gait abnormalities in persons with cerebral palsy, as illustrated by the examples presented in this chapter. Musculoskeletal simulations are necessary for explaining the biomechanical causes of movement abnormalities and the consequences of surgical procedures; this information is essential for developing improved treatment plans.

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