The effect of excessive tibial torsion on the capacity of muscles to extend the hip and knee during single-limb stance

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Abstract

Excessive tibial torsion, a rotational deformity about the long axis of the tibia, is common in patients with cerebral palsy who walk with a crouch gait. Previous research suggests that this deformity may contribute to crouch gait by reducing the capacity of soleus to extend the knee; however, the effects of excess external torsion on the capacity of other muscles to extend the stance limb during walking are unknown. A computer model of the musculoskeletal system was developed to simulate a range of tibial torsion deformities. A dynamic analysis was then performed to determine the effect of these deformities on the capacity of lower limb muscles to extend the hip and knee at body positions corresponding to the single-limb stance phase of a normal gait cycle. Analysis of the model confirmed that excessive external torsion reduces the extension capacity of soleus. In addition, our analysis revealed that several important muscles crossing the hip and knee are also adversely affected by excessive tibial torsion. With a 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%. Since a tibial torsion deformity reduces the capacity of muscles to extend the hip and knee, it may be a significant contributor to crouch gait, especially when greater than $30°$ from normal, and thus should be considered by clinicians when making treatment decisions.

1. Introduction

Many children with cerebral palsy walk with excessive flexion of their hips and knees, a movement pattern known as crouch gait. Tibial torsion deformities, characterized by excess twisting about the bone's long axis, are commonly observed in patients with cerebral palsy who walk with a crouch gait. Previous researchers have postulated that excess tibial torsion reduces the ability of muscles to extend the joints (e.g. [1–4]), which may contribute to the crouched posture observed in some patients. This bony misalignment can often be corrected with a derotation osteotomy (e.g. [5]); however, the indications for surgery are unclear and the outcomes are variable. Understanding if this deformity is a significant contributor to diminished hip and knee extension is an important step in improving the treatment of patients with excess tibial torsion and crouch gait.

There are two possible biomechanical links between crouch gait and tibial deformities. First, excess tibial torsion may reduce the plantar flexion moment arms of muscles crossing the ankle joint. Previous work has established that the soleus and gastrocnemius play an important role in supporting the body during the mid- and late-stance phases of gait [6,7]. If the plantar flexion moment arms of the soleus or gastrocnemius are diminished as a result of the altered musculoskeletal geometry, their capacity to extend the joints and support the body may be reduced, possibly contributing to crouch gait.

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A second possibility is that excess tibial torsion alters the dynamic interactions between muscles and the underlying skeletal system during gait. Analyzing the actions of muscles during movement is complex given the body's multiarticular nature [8]. When a muscle applies a force to a body segment, that segment is accelerated. However, the acceleration of that segment is resisted by the inertia of adjoining segments, which generates intersegmental forces that accelerate the other joints in the body. Therefore, a muscle that only crosses the ankle joint, such as soleus, has the potential to accelerate not just the ankle, but also the hip and knee. This "plantar flexion–knee extension couple" is well established for the soleus [9–11]. Many other muscles have also been demonstrated to accelerate joints they do not cross during gait [12–16]. Moreover, during single-limb stance, the acceleration of the part of the foot that is in contact with the ground must be zero. Since any muscle activation will generate forces on the foot, an equal and opposite force must be applied to the foot by the ground for the foot to remain static. The intersegmental forces generated by a muscle activation and the resulting foot–ground interaction depend on both the orientation of the body segments and bony geometry. Thus, the joint accelerations from a particular muscle activation will change as the body progresses through the gait cycle and also in the presence of a bony deformity like tibial torsion. This means that excess tibial torsion, a transverse plane misalignment of the lower leg, can alter the capacity of muscles to accelerate joints in the *sagittal* plane at the *knee* and *hip*.

Previous research suggests that excess tibial torsion may affect the dynamic capacity of muscles to extend the joints during gait. Schwartz and Lakin [4] demonstrated with a computer model that an external tibial torsion deformity reduces the capacity of soleus to extend the knee during single-limb stance. This study is an important first step in understanding the biomechanical links between tibial torsion and crouch gait; however, several unresolved issues remain. The multiarticular nature of the body means that tibial torsion can affect the capacity of the soleus and other lower limb muscles to extend not only the knee, but also the hip.

The aim of the present study was to determine the mechanisms by which excess external tibial torsion contributes to diminished knee and hip extension. We created a computer model of the musculoskeletal system to simulate a range of tibial torsion deformities. We determined the changes in moment arms of soleus and gastrocnemius as a function of tibial torsion angle to assess the possibility that excess tibial torsion contributes to crouch gait by altering plantar flexion moment arms. We also determined the effect of excess external torsion on the capacity of muscles to extend the hip and knee during single-limb stance to assess the possibility that the deformity alters the dynamic interactions between muscles, the skeletal system and the ground during gait. The muscles most affected by excess external tibial torsion and the degree of deformity resulting in a substantial decrease in extension capacity were examined to help establish indications for a derotational osteotomy.

2. Methods

2.1. Musculoskeletal model

A computer model of the musculoskeletal system with a deformable tibia (Fig. 1) was developed to determine the effect of external tibial torsion on: (1) muscle moment arms and (2) dynamic muscle extension capacities, which we define as the potential of muscles to accelerate the hip and knee into extension during gait. The model used in this study had 14 segments, 11 joints, 23 degrees of freedom, and 92 muscles [17]. The upper body (head, trunk, and arms) consisted of a single segment connected to the pelvis by a 3-degree-of-freedom ball and socket joint. Each leg was composed of a femur, patella, and combined tibia-fibula segment and each foot was represented by talus, calcaneus, and metatarsophalangeal segments. The hip was modeled as a 3-degree-of-freedom ball and socket joint and the knee as a planar joint with constraints to represent the tibiofemoral and patellofemoral joint kinematics [18]. The ankle, subtalar, and metatarsophalangeal joints were modeled as 1-degree-of-freedom revolutes. Muscle paths, bone geometry, and segment inertial parameters were based on previous studies [17,19,20]. The equations describing the model were generated using the SIMM Dynamics Pipeline [21] and SD/Fast (Parametric Technologies, Needham, MA).

2.2. Tibial torsion deformity

A torsional deformity was modeled as a gradual rotation about the long axis of the tibia (Fig. 2). In the undeformed model, the angle between the knee flexion axis and the ankle plantar flexion axis, measured about the long axis of the tibia, was 12° [22]. We created models with additional external torsion up to 60° larger than this baseline in 10° increments. The deformity was modeled in two parts: (1) a rotation of the foot, ankle joint, and distal third of the tibia (Fig. 2, inner box) by the entire torsion angle, and (2) a linearly varying twist of the tibia beginning just above the distal third of the tibia and continuing to a location just distal to the origin of the soleus and the patellar tendon attachment site (Fig. 2, outer box). All proximal muscle attachments on the tibia were unaltered.

2.3. Muscle moment arms

We determined the effect of tibial torsion on muscle moment arms as follows. For the undeformed model and each model with excess tibial torsion, the plantar flexion moment arms of the soleus, medial gastrocnemius, and lateral gastrocnemius were determined throughout the ankle range of motion, with the knee fully extended. Moment arms were calculated using the partial velocity method [21]. The moment arm for each muscle was plotted as a function of additional tibial torsion angle, and the maximum percentage change from normal was determined. The potential effects of a hindfoot varus or valgus deformity, which may accompany a tibial torsion deformity, were not included in this analysis.
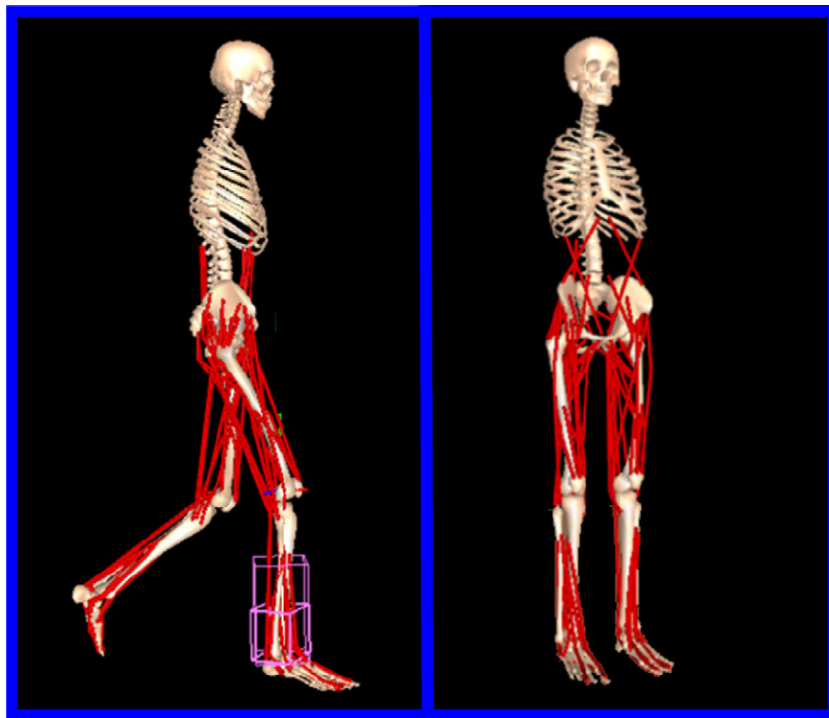
Fig. 1. Three-dimensional model of the musculoskeletal system. The model has 14 segments, 11 joints, 23 degrees of freedom, and 92 muscles. This model was used to evaluate the effect of torsional deformities of the tibia (see box on right tibia) on muscle moment arms and dynamic muscle extension capacities.

2.4. Dynamic muscle extension capacities

We used an induced acceleration analysis (e.g. [11]) to determine the effect of excess external torsion on the dynamic extension capacity of soleus and other muscles during gait. The undeformed model and each model with excess tibial torsion were positioned at the joint angles corresponding to the single-limb stance phase of normal gait. The single-limb stance joint angles were based on gait data from 10 able-bodied children (mean age 12 ± 1.5 years and mean walking speed 1.3 ± 0.1 m/s), collected at Gillette Children's Specialty Health Care, St. Paul, MN. All motion data were acquired using a Vicon 512 motion capture system operating at 60 Hz

(Oxford Metrics, Oxford, UK). The gait data for each of the 10 subjects were normalized to a percent of the gait cycle and then all cycles were averaged. Muscle capacities to accelerate the joints were calculated at each 2% of single-limb stance (15–40% of the gait cycle) for the right leg.

At each position of single-limb stance we assessed the dynamic extension capacity of each muscle in the model. To compute the joint accelerations induced by a muscle, a unit muscle force and its corresponding contribution to the ground reaction force were applied to the model. The resulting angular accelerations of the hip and knee per unit force were then calculated using the model's equations of motion. These accelerations, determined for each
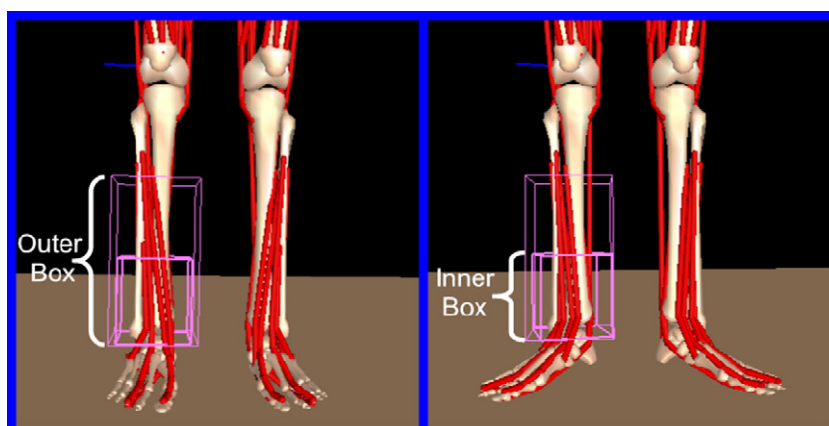

Fig. 2. Geometric method for deforming the tibia. The model on the left has an undeformed tibia, while the model on the right has an additional 30° of external torsion. The tibial torsion deformity was implemented using two boxes. The inner box, ankle axis, and foot were rotated by the torsion angle specified. There was a linear decrease in tibial torsion angle between the top of the inner box and the top of the outer box. All bone deformation was distal to any proximal muscle attachments on the tibia.

muscle at each position of single-limb stance, represent the dynamic capacity or potential of the muscle to accelerate the joints.

The contribution of the muscle to the ground reaction force was determined using a decomposition approach developed by Anderson and Pandy [23]. The interaction between the stance limb and the ground was represented by five contact points that were distributed over the sole of the foot. A unit muscle force was applied to the model and the resulting ground reaction force was determined by solving for the minimum total force that would constrain the acceleration of each contact point to be zero. Optimization was performed using CFSQP (AEM Design, Tucker, GA), a sequential programming algorithm for solving nonlinear optimization problems.

We compared the extension capacities of the muscles for our model without excess tibial torsion to previously published results [12]. The absolute magnitudes of the acceleration potentials and relative potentials between the muscles were in good agreement (Fig. 3). It should be noted that the previous results were obtained from a different model of the musculoskeletal system and separate analysis software. We focused our analysis on a subset of muscles based on two criteria: (1) the muscle had at least a $5°/s^2/N$ potential to accelerate the hip or knee into extension during single-limb stance and (2) the muscle was known to be active during single-limb stance [24]. The muscles meeting these criteria included gluteus maximus, posterior gluteus medius, vasti, hamstrings, and soleus. For each of these muscles, the potential to accelerate the hip and knee as a function of additional torsion was determined and plotted as a percent of the muscle's capacity in the undeformed model.
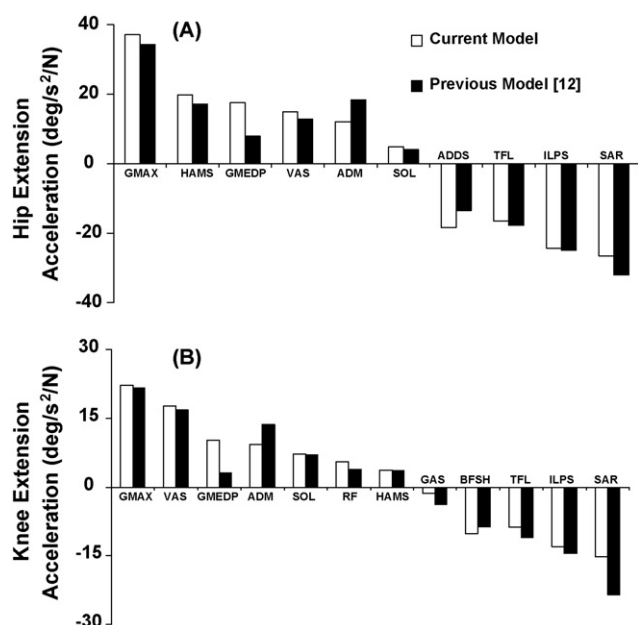

Fig. 3. The capacity of selected muscles to accelerate the hip (A) and knee (B) toward extension in a model with normal tibial geometry, averaged over single-limb stance. The results of this analysis are shown in white. The results of a previously-published analysis [12], using a different model and different simulation software, are shown in black. Muscles analyzed include gluteus maximus (GMAX), hamstrings (HAMS), posterior compartment of the gluteus medius (GMEDP), vasti (VAS), adductor magnus (ADM), soleus (SOL), adductor brevis, longus, and pectineus (ADDS), tensor fascia latae (TFL), iliopsoas (ILPS), sartorius (SAR), rectus femoris (RF), gastrocnemius (GAS), and biceps femoris short head (BFSH).

3. Results

3.1. Muscle moment arms

Introducing a tibial torsion deformity to the musculoskeletal model changed the moment arms of the ankle plantar flexors only slightly (Fig. 4). Soleus and lateral gastrocnemius showed a small decrease in plantar flexion moment arm with additional external torsion, while medial gastrocnemius showed a slight increase in plantar flexion moment arm. The percentage decrease for soleus was about 1% for an additional torsion angle of 60°. The lateral gastrocnemius showed a 2% decrease and the medial gastrocnemius showed a 3% increase. The patterns and percentage changes were similar throughout the ankle range of motion typically observed during gait.

3.2. Dynamic muscle extension capacities

In the undeformed model, the gluteus maximus had the greatest potential to extend the hip per unit force ($37°/s^2/N$), followed by hamstrings ($20°/s^2/N$), then posterior gluteus medius ($18°/s^2/N$), vasti ($15°/s^2/N$), and soleus ($5°/s^2/N$) (Fig. 3A). The gluteus maximus also had the greatest potential to extend the knee ($22°/s^2/N$), followed by vasti ($18°/s^2/N$), then posterior gluteus medius ($10°/s^2/N$), and soleus ($7°/s^2/N$) (Fig. 3B).

Excess external torsion reduced the hip and knee extension capacity during single-limb stance for nearly all of the muscles examined. The capacity of soleus to extend the hip decreased by 50% with 60° of additional external torsion (Fig. 5A). The capacity of the posterior gluteus medius to extend the hip decreased by 20%, the gluteus maximus by 8%, and the vasti by 4% with a deformity of 60°. There was a negligible change in the potential of the hamstrings to extend the hip.

The capacity of both the soleus and posterior gluteus medius to extend the knee decreased by 50% with a 60° external deformity (Fig. 5B). The gluteus maximus showed a reduction of just under 20% and the vasti a reduction of 4%. The relationship between tibial torsion angle and extension potential was nonlinear for the soleus and approximately
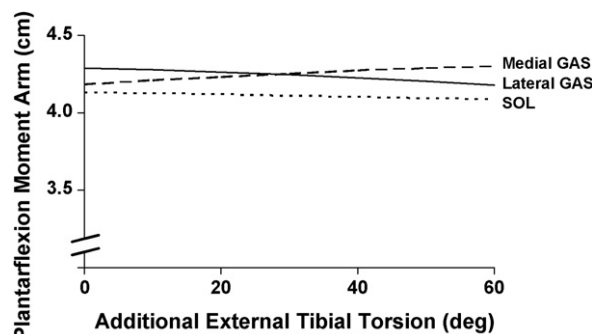

Fig. 4. The effect of excess external tibial torsion on the moment arms of the plantar flexors in an upright standing position.
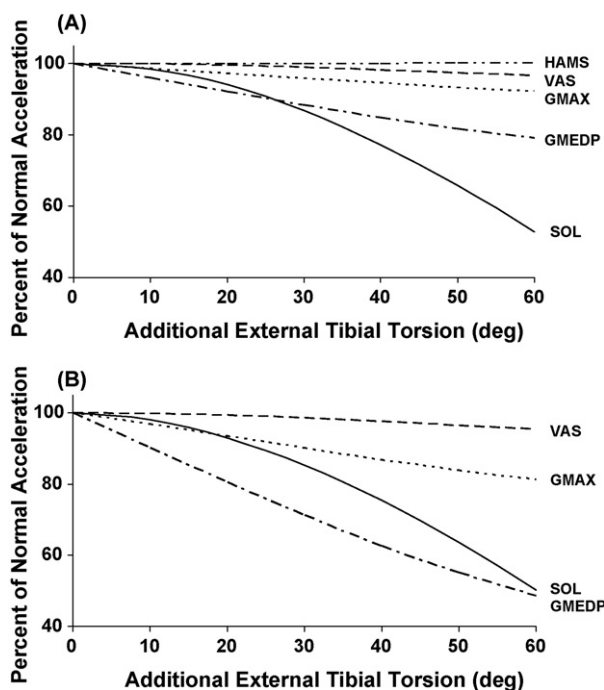
Fig. 5. The effect of excess external torsion on the average capacity of muscles to extend the hip (A) and knee (B) during single-limb stance. The accelerations per unit force are given as a percent of the values for the normal or undeformed model.

linear for the other muscles. Although the extension capacity was averaged over single-limb stance, the reduction in extension capacity as a result of a tibial torsion deformity tended to be similar throughout single-limb stance.

4. Discussion

The results of this analysis indicate that excess external torsion of the tibia may contribute to crouch gait by reducing the capacity of muscles to extend the hip and knee during gait. A tibial torsion deformity did not significantly affect muscle moment arms in our model—the plantar flexion moment arms of the soleus and gastrocnemius changed less than 3% at the largest tibial torsion angle tested. Rather, deforming the tibia diminished the potential of several important stance phase muscles to extend both the hip and knee by altering the skeletal platform on which the muscles act. As demonstrated by previous researchers [9–11], several muscles were found to have a considerable potential to extend joints they do not cross. The gluteal muscles had an extension potential during single-limb stance at not only the hip, but also the knee, while soleus had an extension potential at both the hip and knee. Excess tibial torsion resulted in a marked decrease in the capacity of the gluteals and soleus to extend the hip and knee and thus their capacity to support an upright posture during gait.

Two important clinical questions are: (1) when is external tibial torsion a significant contributor to a crouch gait

pattern? and (2) when should excess torsion be corrected with a derotational osteotomy? Further investigation is needed to definitively answer these questions; however, the results of this study suggest a few possible guidelines to aid surgical planning. First, major changes in the capacity of muscles to extend the joints were observed when the deformity was 30° larger than normal. For example, with an additional torsion of 30°, the potentials of soleus, posterior gluteus medius, and gluteus maximus to extend the knee were all reduced by greater than 10% and their potentials to extend the hip were all reduced by greater than 15%. This result is consistent with clinical observations of a large group of patients with diplegic cerebral palsy examined at Gillette Children's Specialty Healthcare (Fig. 6). In particular, when external tibial torsion, as determined from the bimalleolar axis, was 30° or more above normal, there was a significant increase in the likelihood that a patient walked with a crouch gait (p-value = 0.01 by the Chi-square test).

The deformity tended to have the greatest impact on the soleus and posterior gluteus medius. For example, with a tibial torsion angle of 60° greater than normal, the capacity of both soleus and posterior gluteus medius to extend the knee was reduced by 50%. This suggests that the deformity may be particularly deleterious in patients with pre-existing weakness of the gluteal or plantar flexor muscles or those receiving soft-tissue surgeries that could weaken these muscles.

Schwartz and Lakin [4] found that the potential of the soleus to extend the knee was reduced in the presence of external tibial torsion, which is consistent with our results. However, they found that the soleus generated a knee flexion acceleration when the tibial torsion deformity was 50°. Soleus always generated a knee extension acceleration in our analysis, even with 60° of excess tibial torsion. The
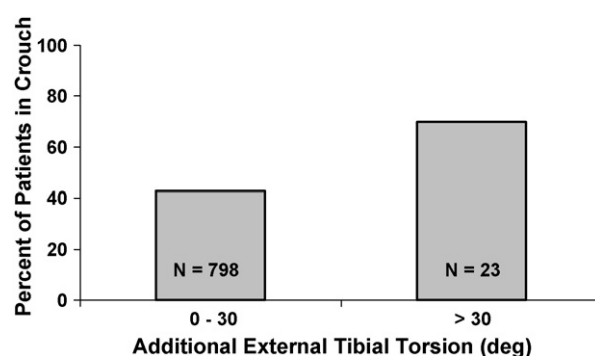

Fig. 6. Clinically-observed correlation between excess external tibial torsion and crouch gait. The data presented here represent 821 patients with diplegic cerebral palsy examined at Gillette Children's Specialty Healthcare since 1994. The amount of additional torsion was determined using measurements of the bimalleolar axis. A bimalleolar angle of 10° was taken as the normal or baseline value, to correspond with our undeformed musculoskeletal model. Patients were classified as being in crouch gait if their knee flexion was 20° or greater at initial contact and at least 15° throughout stance [25]. Subjects with an external tibial torsion deformity of 30° or larger ($N = 23$) had a significantly greater likelihood of walking with a crouch gait ($p = 0.01$), as determined by the Chi-square test.

different results most likely arise from the different techniques used to model the foot–ground interaction. In the current study, the ground reaction force was calculated using a decomposition technique that did not constrain the force to act through a pre-determined point. In the study by Schwartz and Lakin [4], the ground reaction force during the simulation was assumed to act through the center of pressure. The location of the center of pressure on the foot was taken from normal gait data, and when the deformity was introduced, this point was assumed to rotate with the foot. This approach may have exaggerated the effects of the deformity, since it is likely that the center of pressure moves medially on the foot when excess external tibial torsion is present.

A few limitations must be considered when interpreting our results. In this study, we analyzed the potential of a muscle to accelerate the joint per unit muscle force. The actual angular acceleration of a joint induced by a muscle depends on the muscle's activation level as well as its force-generating capacity, which depends in turn on the muscle's physiological cross-sectional area and the muscle's length and velocity during the movement. For example, in this analysis, the potential of the soleus to extend the hip and knee was small relative to the other muscles examined (Fig. 3); however, the soleus generates a large force during single-limb stance [10,12], so even small changes in its extension potential may have a significant impact on gait.

Second, several assumptions were made in modeling the tibial torsion deformity. Little information is available about the typical morphology of a tibial torsion deformity, although it is commonly believed to occur in the distal portion of the bone. The most important modeling consideration, at least for the dynamic analysis, is the relative twist between the knee flexion axis and the ankle plantar flexion axis, which should be accurately represented in our model. The actual morphology of the deformity may be more important when examining changes in muscle moment arms. Nevertheless, the effect of tibial torsion on muscle plantar flexion moment arms is still likely to be small relative to the changes in dynamic muscle extension capacity.

The capacity of muscles to extend the joints depends on the geometry of all the bones and the orientations of all the joints. As a result, the presence of other bony deformities may impact the changes in muscle capacities that result from tibial torsion. Concomitant bone deformities, including excessive femoral anteversion and varus-valgus deformities of the hindfoot, are common in the patient population with cerebral palsy and excess tibial torsion. These concomitant bony abnormalities could possibly offset or exacerbate the effects of a tibial torsion deformity on the muscle extension capacities. Also, the gait pattern used to position the model could impact the results of the analysis. In this study, the model was positioned at joint angles corresponding to a normal gait pattern. Walking with a crouch gait alone changes the potential of muscles to extend the joints [26], so

how muscles are affected by excess tibial torsion and which muscles are affected may change in the presence of crouch gait kinematics. Additionally, subjects may attempt to compensate for excess torsion, for example, by increasing the internal rotation of their hip. Since we positioned the model with normal kinematics, we were not able to assess the effectiveness of this compensation. Further investigation is warranted to determine the interacting effects of bone deformities and abnormal gait kinematics.

Since excessive external tibial torsion reduces the capacity of several stance phase muscles to extend the hip and knee, it may be a significant contributor to crouch gait, especially when the torsion angle is $30°$ greater than normal. The deformity had the greatest impact on soleus and posterior gluteus medius, suggesting that correcting excessive tibial torsion may be particularly important in patients with weak plantar flexors or gluteal muscles. The analysis technique developed in the study provides a powerful framework for examining how changes in bone geometry affect the capacity of muscles to accelerate the joints during gait or other movements. This framework could be used in future investigations to examine the effects of other bone deformities on muscle function or to assess the effect of an individual patient's bone geometry on his or her gait.

Conflict of interest

The authors declare that they have no competing interests.

Acknowledgements

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