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Muscular contributions to hip and knee extension during the single limb stance phase of normal gait: a framework for investigating the causes of crouch gait

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Abstract

Crouch gait, a troublesome movement abnormality among persons with cerebral palsy, is characterized by excessive flexion of the hips and knees during stance. Treatment of crouch gait is challenging, at present, because the factors that contribute to hip and knee extension during normal gait are not well understood, and because the potential of individual muscles to produce flexion or extension of the joints during stance is unknown. This study analyzed a three-dimensional, muscle-actuated dynamic simulation of walking to quantify the angular accelerations of the hip and knee induced by muscles during normal gait, and to rank the potential of the muscles to alter motions of these joints. Examination of the muscle actions during single limb stance showed that the gluteus maximus, vasti, and soleus make substantial contributions to hip and knee extension during normal gait. Per unit force, the gluteus maximus had greater potential than the vasti to accelerate the knee toward extension. These data suggest that weak hip extensors, knee extensors, or ankle plantar flexors may contribute to crouch gait, and strengthening these muscles—particularly gluteus maximus—may improve hip and knee extension. Abnormal forces generated by the iliopsoas or adductors may also contribute to crouch gait, as our analysis showed that these muscles have the potential to accelerate the hip and knee toward flexion. This work emphasizes the need to consider how muscular forces contribute to multijoint movements when attempting to identify the causes of abnormal gait.

Keywords: Dynamic simulation; Muscle function; Gait; Cerebral palsy

1. Introduction

Children with cerebral palsy frequently walk with excessive flexion of their hips and knees during the stance phase. This movement abnormality, called crouch gait, is problematic because it increases patello-femoral force (Perry et al., 1975), impedes toe clearance, and dramatically increases the energy requirements of walking (Campbell and Ball, 1978; Rose et al., 1990; Stout and Koop, 2004). Persistent hip and knee flexion typically worsen if not corrected (Sutherland and Cooper, 1978; Gage and Schwartz, 2004) and can lead to altered patellofemoral joint mechanics and chronic knee pain (Rosenthal and Levine, 1977; Lloyd-Roberts et al., 1985; Bleck, 1987; Sutherland and Davids, 1993).

The biomechanical causes of the excessive hip flexion and knee flexion in persons with cerebral palsy are often unclear, making it challenging to determine the most appropriate treatment. In some cases, abnormally "short" or "spastic" hamstrings are presumed to limit knee extension, and surgical lengthening of the hamstrings is performed (Bleck, 1987; Perry and Newsam, 1992;

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DeLuca et al., 1998; Abel et al., 1999). In other cases, diminished plantar flexion strength is thought to be a factor, and ankle–foot orthoses are prescribed (Rodda and Graham, 2001; Gage, 2004b). Other hypothesized causes of crouch gait include malrotation of the femur, tibia, and foot (Schwartz and Lakin, 2003; Gage, 2004b), tight hip flexors (Roosth, 1971; Reimers, 1973; Bleck, 1987; Novacheck et al., 2002), weak hip extensors (Wiley and Damiano, 1998), weak knee extensors (Damiano et al., 1995; Beals, 2001; Gage, 2004b), and poor balance (Gage and Schwartz, 2004). The outcomes of treatments aimed at correcting crouch gait, at present, are variable; some individuals walk with dramatically improved extension of their hips and knees following treatment (e.g., DeLuca et al., 1998; Novacheck et al., 2002), while others show little improvement or get worse.

Successful treatment of crouch gait is difficult, in part, because the factors that contribute to hip and knee extension during normal gait are not completely understood, and because the potential of individual muscles to produce flexion or extension of the joints during stance has not been rigorously evaluated. Indeed, a theoretical framework for elucidating which muscles are likely to contribute to a patient's abnormal gait does not exist. Establishing such a framework is complicated because the muscles that influence the motions of the joints are not necessarily intuitive—a muscle that spans one joint has the potential to accelerate other joints, and biarticular muscles can produce accelerations of the joints that oppose their applied moments (Zajac and Gordon, 1989; Zajac et al., 2002). For instance, several studies have shown that knee motions during the swing phase are influenced not only by muscles that cross the knee, but also by muscular moments that are generated at other joints (e.g., Piazza and Delp, 1996; Kerrigan et al., 1998; Anderson et al., 2004).

The purpose of this study was to quantify the angular accelerations of the hip and knee induced by individual muscles during the single limb stance phase of normal gait and to rank the potential of the muscles to alter the accelerations of the joints. The actions of the gluteus maximus, hamstrings, vasti, soleus, gastrocnemius, and other muscles were determined using a three-dimensional, muscle-actuated dynamic simulation of walking (Anderson and Pandy, 2001). The results of this study offer insight into the dynamical actions of muscles during walking and establish a framework for the identification of factors that may cause excessive hip flexion and knee flexion in persons with cerebral palsy.

2. Methods

The flexion/extension accelerations of the hip and knee induced by muscles during the single limb stance phase were calculated based on the dynamic optimization solution for a half cycle of normal gait solved by Anderson and Pandy (2001). The musculoskeletal model used to generate this solution (Fig. 1A) was a 10-segment, 23-degree-of-freedom linkage actuated by 54 muscles (Anderson and Pandy, 1999, 2001). The pelvis was modeled as a rigid segment that was allowed to rotate and translate in three dimensions with respect to the ground. Each hip was modeled as a ball-and-socket joint, each knee as a hinge joint, each ankle–subtalar joint as a universal joint, and each metatarsal joint as a hinge joint. The head, arms, and torso were represented as a single segment that articulated with the pelvis via a ball-and-socket joint located at approximately the third lumbar vertebra. The inertial properties of the segments were based on the regression equations of McConville et al. (1980) and anthropometric measures obtained from five healthy adult males (Anderson and Pandy, 2001). Contact between each foot and the ground was characterized by five stiff spring–damper units distributed under the sole of the foot. Ligaments were represented as angle-dependent joint torques (Davy and Audu, 1987) that prevented non-physiological hyperextension of the joints.

Each of the 54 muscle–tendon actuators in the model was represented as a three-element, Hill-type muscle in series with an elastic tendon (Zajac, 1989). The force-generating properties, attachment sites, and path geometries of the muscles were based on data reported by Delp et al. (1990). The muscle excitation–contraction dynamics were characterized by a first-order differential equation (Zajac, 1989). The rise and decay time constants for muscle activation were assumed to be 22 and 200 ms, respectively (Zajac, 1989; Pandy et al., 1990). The excitation patterns of the muscles were determined by solving a dynamic optimization problem, in which the performance criterion was to minimize the metabolic energy consumed per unit distance traveled on level ground. The solution to this optimization problem produced a forward simulation of a half cycle of normal gait (Fig. 1B). The excitation patterns of the muscles and the resulting joint angular displacements and ground reaction forces generated during the simulation compare favorably to experimental data (Anderson and Pandy, 2001).

The contributions of individual muscles to the angular accelerations of the joints were determined using the equations of motion for the model, as described by Zajac et al. (2002), and the decomposition of the ground reaction force described by Anderson and Pandy (2003):

$$\ddot{\vec{q}} = \overleftrightarrow{I}(\vec{q})^{-1} \cdot \{\overleftrightarrow{R}(\vec{q}) \cdot \vec{f}_M + \Gamma(\vec{q}, \dot{\vec{q}}) + \vec{G}(\vec{q}) + \vec{C}(\vec{q}, \dot{\vec{q}}^2) + \overleftrightarrow{S}(\vec{q}) \cdot \vec{f}_s\}, \quad (1)$$

$$\vec{f}_S = \vec{f}_S^M + \vec{f}_S^\Gamma + \vec{f}_S^G + \vec{f}_S^C. \quad (2)$$

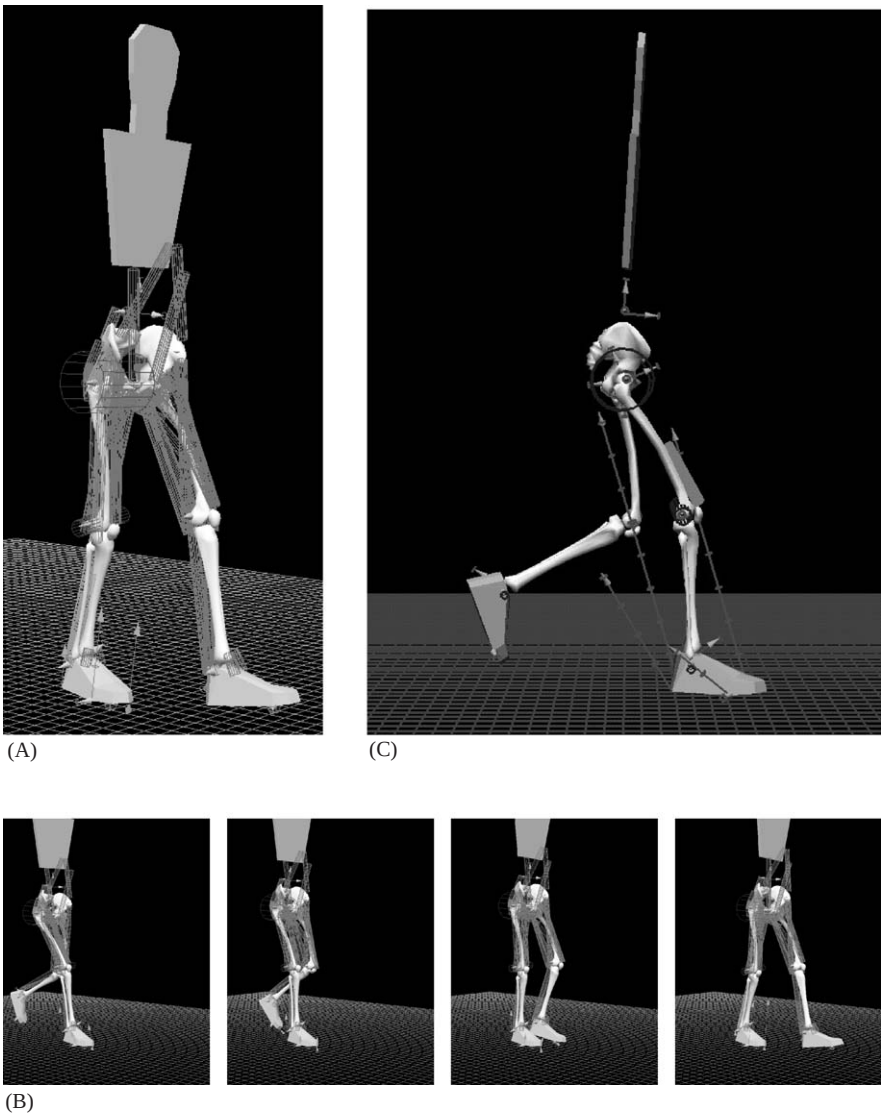

Fig. 1. Muscle-actuated dynamic model with 10 segments, 23 degrees of freedom, and 54 muscle–tendon actuators (A) used to quantify the contributions of individual muscles to hip and knee extension during the single limb stance phase of normal gait (B). At each time step in the simulation, a muscle's contributions to the angular accelerations of the joints were calculated by applying that muscle's force and the corresponding portion of the ground reaction force caused by that muscle to the model (C).

In these equations, $\vec{q}$ is the vector of generalized coordinates, $\overleftrightarrow{I}(\vec{q})^{-1}$ is the inverse of the system mass matrix, $\vec{f}_M$ is the vector of muscle forces, $\overleftrightarrow{R}(\vec{q})$ is the matrix of muscle moment arms, $\Gamma(\vec{q},\dot{\vec{q}})$ is the vector of ligament torques, $\vec{G}(\vec{q})$ is the vector of gravitational forces, $\vec{C}(\vec{q},\dot{\vec{q}}^2)$ is the vector of centripetal and Coriolis forces, $\vec{f}_s$ is the vector of spring and damper forces that characterize the interaction of the feet with the ground, $\overleftrightarrow{S}(\vec{q})$ is a matrix that converts the foot–ground spring forces into generalized forces (Kane and Levinson, 1985), and $\vec{f}_S^M$, $\vec{f}_S^\Gamma$, $\vec{f}_S^G$ and $\vec{f}_S^C$ are the contributions made to the foot–ground spring forces by the muscle forces, ligament torques, gravitational forces, and centripetal and Coriolis forces, respectively. At each time step in the simulation, a muscle's contributions to the instantaneous accelerations of the generalized coordinates were calculated by applying that muscle's force, as generated during the simulation, and the corresponding portion of the ground reaction force caused by that muscle (Fig. 1C):

$$\ddot{\vec{q}}_i = \overleftrightarrow{I}(\vec{q})^{-1} \cdot \{\overleftrightarrow{R}(\vec{q}) \cdot \vec{f}_{M_i} + \overleftrightarrow{S}(\vec{q}) \cdot \vec{f}_S^{M_i}\}. \quad (3)$$

All other muscular forces, gravitational forces, and force terms arising from angular velocities were set to zero. The portion of the ground reaction force caused by a particular muscle was estimated by applying that muscle's force to the model in isolation and calculating the reaction forces necessary to constrain each spring

point in contact with the ground to have zero acceleration (Anderson and Pandy, 2003).

Two descriptions of the muscle actions during single limb stance were examined. First, the angular accelerations of the hip and knee induced by the gluteus maximus, hamstrings, vasti, soleus, gastrocnemius, and other muscles were quantified to determine which of the muscles enabled hip and knee extension in the simulation of normal gait. Second, the muscle-induced accelerations of the hip and knee per unit force were calculated to assess the "dynamic potential" of each muscle to accelerate the limb segments toward flexion or extension. This measure of a muscle's actions, obtained by setting $\vec{f}_{Mi} = 1$, computing the corresponding $\vec{f}_S^{Mi}$, and substituting these quantities into Eq. (3), does not depend on the muscle excitations or forces applied during the simulation. Hence, this analysis evaluated the relative potential of a muscle to generate (or limit) hip and knee extension if, for example, the muscle was activated inappropriately or was producing excessive passive force.

3. Results

Gravity, in combination with its contribution to the ground reaction force, accelerated both the hip and the knee toward flexion during the stance phase in our simulation. The effects of gravity were resisted by muscles crossing the hip, knee, and ankle (Fig. 2). In early single limb stance, the hip extension moment generated by the gluteus maximus and the knee extension moment generated by the vasti strongly accelerated *both* the hip and knee toward extension. Notably, the gluteus maximus accelerated the knee toward extension almost as much as the vasti in our simulation. In mid- and late stance, the posterior portion of the gluteus medius and the soleus also contributed substantially to hip and knee extension. Hence, our dynamic analysis of the muscle actions corroborates previous studies (e.g., Winter, 1980; Perry, 1992; Jonkers et al., 2003b; Neptune et al., 2004) that have suggested that the hip extensors, knee extensors, and ankle plantar flexors all help to control hip and knee extension during the stance phase of normal gait. The hamstrings, which generated a hip extension moment and a knee flexion moment in early stance, accelerated the hip toward extension in our simulation, but had very little effect on the stance-phase motion of the knee.

The muscles with the greatest potential to accelerate the knee toward extension in our model, per unit force and averaged over single limb stance, were the gluteus maximus, vasti, adductor magnus, and soleus (Fig. 3B). This result suggests that the gluteus maximus has a greater capacity to induce knee extension, per unit force, than the vasti during stance. The muscles with the most potential to accelerate the hip toward extension were the gluteus maximus, adductor magnus, hamstrings, and vasti (Fig. 3A).

The muscles with the greatest potential to accelerate the hip and knee toward flexion in our model, per unit force and averaged over single limb stance, were the sartorius, iliopsoas, and tensor fascia latae (Fig. 3). These muscles, with their large hip flexion moment arms, had a greater capacity to induce knee flexion than the biceps femoris short head, which crosses only the knee. The adductor brevis, adductor longus, and pectineus (ADDS in Fig. 3) and the gracilis (not shown) also had the potential to produce hip and knee flexion during the mid- and late stance phases in our model.

The potential of the biarticular hamstrings, rectus femoris, and gastrocnemius muscles to induce angular accelerations of the knee during single limb stance was small relative to other muscles (Fig. 3B). This was due to dynamic coupling; in particular, each of these muscles produced moments at adjacent joints that had opposing actions at the knee. For example, the knee flexion moment generated by the hamstrings accelerated the knee toward flexion, but the hip extension moment generated by the hamstrings accelerated the hip and knee toward extension. During single limb stance, both the rectus femoris and the hamstrings had the potential to weakly accelerate the knee toward *extension* in our model. The gastrocnemius had the potential to accelerate the knee toward flexion during single limb stance, opposite in direction to the uniarticular soleus.

4. Discussion

This study may have important implications for the evaluation and treatment of crouch gait. Our analysis of the multijoint accelerations induced by muscles during single limb stance confirms that the gluteus maximus, vasti, and soleus make substantial contributions to hip and knee extension during normal gait. This suggests that diminished force in the hip extensors (Wiley and Damiano, 1998), knee extensors (Damiano et al., 1995; Beals, 2001; Gage, 2004b), or ankle plantar flexors (Rodda and Graham, 2001; Gage, 2004b) may contribute to crouch gait, and strengthening these muscles—particularly gluteus maximus—may help to improve both hip and knee extension. Other impairments that limit the capacity of the gluteus maximus, vasti, or soleus to accelerate the joints toward extension, such as excessive external tibial torsion (Schwartz and Lakin, 2003; Gage, 2004b), may also be contributing factors. Our study further suggests that abnormal forces generated by contracture of the iliopsoas (Roosth, 1971; Reimers, 1973; Bleck, 1987; Novacheck et al., 2002) or spasticity of the adductors may cause crouch gait in some cases, since these muscles have a large potential to

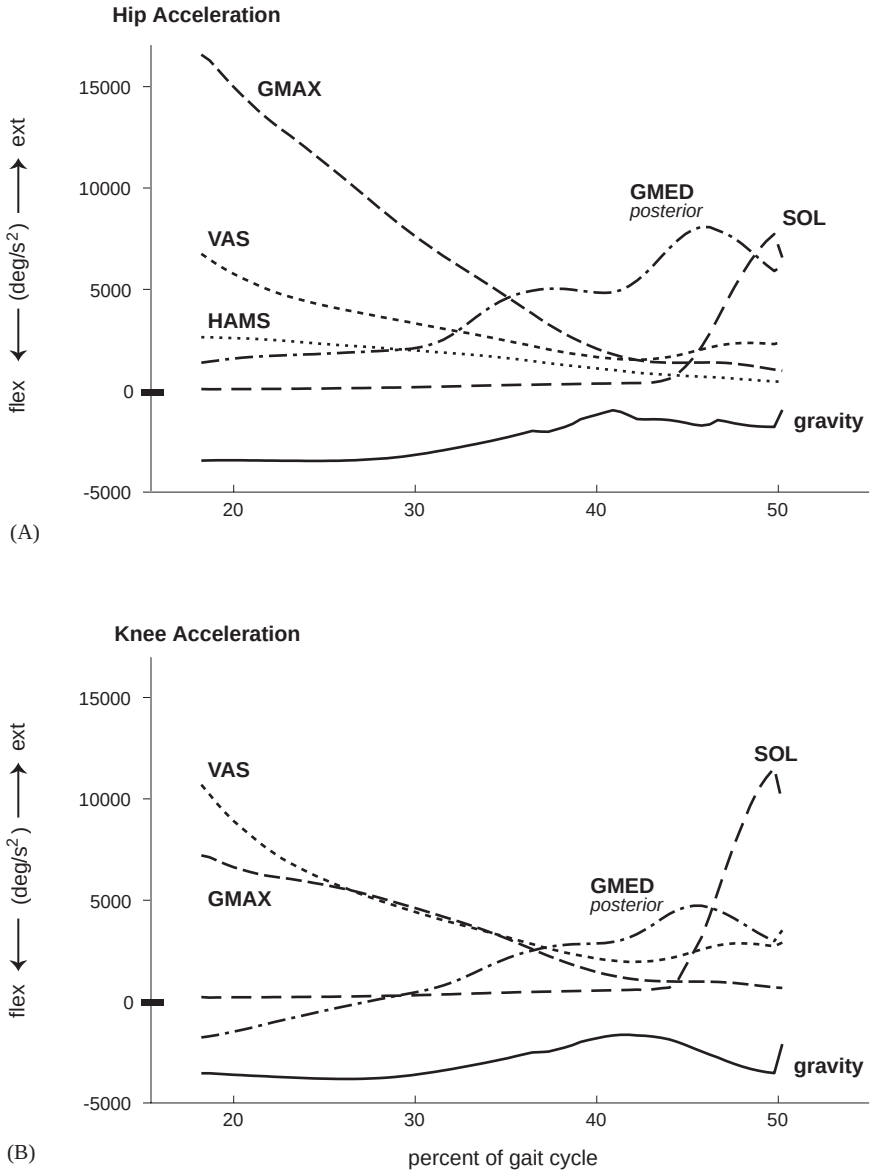

Fig. 2. Contributions of the gluteus maximus (GMAX), vasti (VAS), hamstrings (HAMS), soleus (SOL), posterior compartment of the gluteus medius (GMED), and gravity to the angular accelerations of the hip (A) and knee (B) during the single limb stance phase of normal gait. The gluteus maximus and vasti induce hip and knee extension throughout single limb stance, even after their excitations have ceased in the simulation, because the forces produced by the muscles decay at a rate determined by the time constant for muscle deactivation.

accelerate the hip and knee toward flexion. The hamstrings had little effect on stance-phase knee motion in our model; this unexpected result suggests that abnormally short or spastic hamstrings, a reputed cause of crouch gait, may not be the direct source of excessive stance-phase knee flexion in some patients. This work emphasizes the need to consider how muscular forces contribute to multijoint movements when attempting to identify the causes of abnormal gait.

Clinicians who treat gait abnormalities have long recognized that the gluteus maximus and hamstrings make important contributions to hip extension (e.g., Waters et al., 1974; Perry, 1992; Rab, 1994; Whittle, 1996) and that the vasti and soleus make important contributions to knee extension (e.g., Sutherland, 1966; Perry and Newsam, 1992; Rab, 1994; Gage et al., 1995) during normal walking. It has generally *not* been recognized, however, that the vasti and soleus also induce hip extension, and that the gluteus maximus induces knee extension, when these muscles generate force during the stance phase. For example, Whittle (1996) speculated that hip extension is achieved by inertia and gravity in mid-stance, when contraction of the gluteus maximus and hamstrings ceases. By contrast, our study shows that the soleus and posterior portion of the gluteus medius strongly accelerate the hip toward

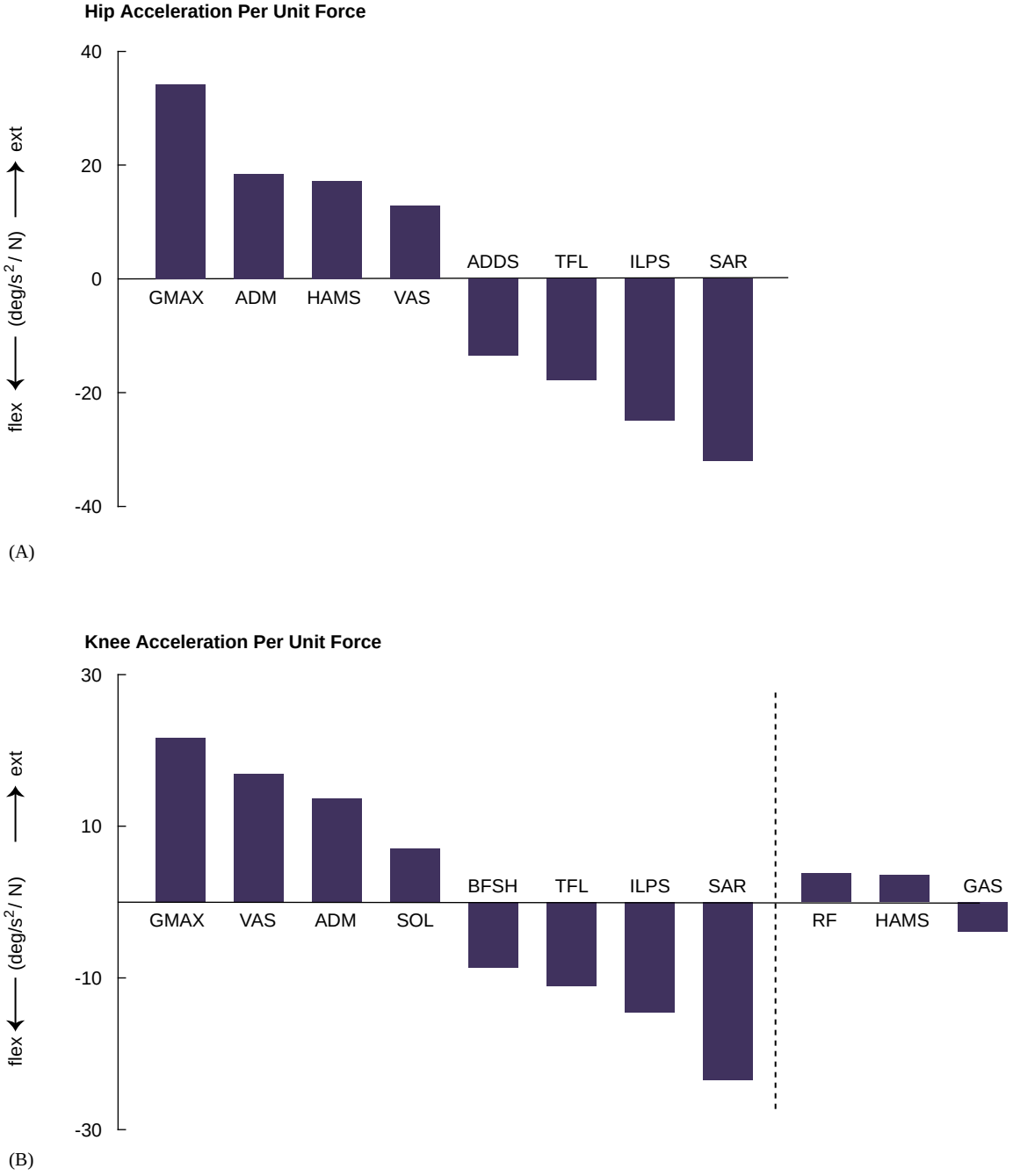

Fig. 3. Angular accelerations of the hip (A) and knee (B) per Newton, averaged over the period of single limb stance (17–50% of the gait cycle), induced by the gluteus maximus (GMAX), vasti (VAS), hamstrings (HAMS), adductor magnus (ADM), adductor brevis, longus, and pectineus (ADDS), iliopsoas (ILPS), sartorius (SAR), tensor fascia latae (TFL), rectus femoris (RF), biceps femoris short head (BFSH), soleus (SOL) and gastrocnemius (GAS).

extension in mid-stance, counteracting the hip flexion acceleration induced by gravity (Fig. 2). Gage (1991, 2004a) postulated that in early stance, the gluteus maximus contributes to knee extension via its action through the iliotibial band. Our study supports Gage's assessment of the function of gluteus maximus; however, our analysis indicates that the joint intersegmental reaction forces caused by the muscle's hip extension moment enable the gluteus maximus to contribute to knee extension, even without transmission of force through the iliotibial band. We believe that simulation-based analyses of the muscle actions, in combination with measured gait kinematics and electromyographic (EMG) recordings, have tremendous potential to advance our understanding of normal and pathological movements.

Several previous studies have used models to examine the co-functional roles of the hip extensors, knee extensors, and ankle plantar flexors in stabilizing the hip and knee during single limb stance. Winter (1980) analyzed the hip, knee, and ankle moments generated by subjects during walking, and concluded that prevention of collapse during weight bearing is accomplished by muscles at all three joints. Pandy and Berme (1989a,b) showed that knee extension was diminished when ankle plantar flexion activity was excluded from a model of single limb stance. Yamaguchi and Zajac (1990) demonstrated, using a muscle-actuated simulation, that the vasti and soleus were critical for stabilizing the knee during mid-stance. Siegel et al. (2001) examined the strategies used by subjects with quadriceps weakness to prevent collapse of their knees; they reported that the subjects' knees were accelerated into extension by their hip extension moments, ankle plantar flexion moments, and/or their contralateral plantar flexion moments in early stance. The results of our investigation are generally consistent with the actions attributed to muscles in these previous studies.

Jonkers et al. (2003a,b) inferred the actions of muscles during normal gait using a series of simulations in which the activation of each muscle in a two-dimensional model was systematically set to zero. They reported that the primary contributors to hip extension in single limb stance were the vasti, hamstrings, and gluteus maximus; the primary contributors to knee extension were the vasti, soleus, and gastrocnemius. However, because the state variables—and therefore the forces generated by other muscles—were altered in the perturbed simulations, the relative potential of muscles to accelerate the joints could not be determined. Our study builds on the work of Jonkers et al. by quantifying the potential of individual muscles to produce angular accelerations of the hip and knee during walking. The muscle actions determined from our study are qualitatively similar to those reported by Jonkers et al., with the exception of the actions of gastrocnemius (and smaller muscles not reported).

Neptune et al. (2004) reported the hip and knee accelerations induced by four muscles during stance. They showed that the gluteus maximus and vasti accelerated the joints toward extension in early to mid-stance, and that the soleus accelerated the joints toward extension in mid- to late stance, consistent with our analysis. However, the relative contributions of the muscles to extension differed from those estimated in our study. For example, the peak knee accelerations induced by the gluteus maximus and soleus in single limb stance, as reported by Neptune et al., were approximately 2500 and 22,000°/s^2, respectively. The peak knee accelerations induced by the muscles in our study were approximately 7200 and 12,000°/s^2, respectively. We hypothesize that differences in the musculoskeletal models contributed to these discrepancies. The model used by Neptune et al. did not include gluteus medius, and the contribution of gluteus maximus to the ground reaction force was smaller than estimated in our study (Anderson and Pandy, 2003). If the gluteus medius had been included, or if the gluteus maximus had contributed more to the ground reaction force, then the knee extension acceleration attributed to the soleus in Neptune et al.'s analysis would likely have been smaller. Differences in the muscle excitation patterns used to generate the simulations could also be a factor. For example, the soleus developed large forces only in late stance in our simulation (Anderson and Pandy, 2001). If the soleus had been activated earlier, more consistent with EMG recordings (Perry, 1992), then it would have contributed to knee extension earlier. Despite such discrepancies in the magnitude and timing of the muscle-induced accelerations, the similarities between our data and the work of Neptune et al. (2004) and Jonkers et al. (2003a,b) suggest that a consistent description of the muscle actions during normal gait is emerging.

It is important to consider some of the limitations of this study. First, our estimates of the hip and knee accelerations induced by muscles during normal gait (Fig. 2) depend on the forces applied by the muscles during the simulation. We believe that the active and passive forces generated by most muscles in our simulation are reasonable. However, the excitation patterns of some muscles, such as soleus and adductor magnus, differ slightly from EMG recordings. The adductor magnus, for example, ceases its activity early in the loading response phase. If the adductor magnus generated force for a longer duration in our simulation, our analysis (Fig. 3) suggests that it could have contributed substantially to hip and knee extension. The role of the adductor magnus as a hip extensor has been recognized previously (e.g., Perry, 1992); however, its role as a knee extensor warrants further investigation. Fortunately, our ranking of the muscles' potential to induce hip and knee extension depends only on the muscles' moment arms and the configuration of the body, and not on the magnitude of the muscle forces predicted. Hence, we are confident in the potential actions of the muscles reported in this study.

Second, we analyzed the muscle actions at the body positions corresponding to normal gait, and we used a model with normal musculoskeletal geometry. The potential of the muscles to accelerate the joints might be different at the body positions corresponding to crouch gait, or using a model that represents bone deformities. Persons with crouch gait typically walk with excessive flexion, adduction, and internal rotation of their hips in addition to exaggerated flexion of their knees. Frequently, they exhibit bone deformities. To better understand the muscle actions during crouch gait,

dynamic simulations that reproduce the musculoskeletal geometry and the gait dynamics of individuals with cerebral palsy are needed. The data reported in this study establish a baseline for assessing how the muscle actions might change with variations in bone geometry or posture.

Third, the muscle-induced accelerations reported in this study describe the functions of individual muscles acting in isolation. To identify the biomechanical causes of crouch gait, it may be necessary to consider how excessive force in one muscle might change the forces in other muscles, either as a result of altered excitation patterns or intrinsic muscle mechanics.

Improving the treatment of crouch gait and other movement abnormalities in persons with cerebral palsy is challenging because currently there is no theoretical basis for determining the biomechanical causes of an individual's abnormal gait. Our examination of the key factors that control hip and knee extension during normal gait—using a dynamic model that enabled the multijoint accelerations induced by individual muscles to be quantified—is an essential step toward explaining the pathomechanics of crouch gait and the consequences of common interventions.

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