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Muscular coordination of knee motion during the terminal-swing phase of normal gait

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Abstract

Children with cerebral palsy often walk with diminished knee extension during the terminal-swing phase, resulting in a troublesome "crouched" posture at initial contact and a shortened stride. Treatment of this gait abnormality is challenging because the factors that extend the knee during normal walking are not well understood, and because the potential of individual muscles to limit terminal-swing knee extension is unknown. This study analyzed a series of three-dimensional, muscle-driven dynamic simulations to quantify the angular accelerations of the knee induced by muscles and other factors during swing. Simulations were generated that reproduced the measured gait dynamics and muscle excitation patterns of six typically developing children walking at self-selected speeds. The knee was accelerated toward extension in the simulations by velocity-related forces (i.e., Coriolis and centrifugal forces) and by a number of muscles, notably the vasti in mid-swing (passive), the hip extensors in terminal swing, and the stance-limb hip abductors, which accelerated the pelvis upward. Knee extension was slowed in terminal swing by the stance-limb hip flexors, which accelerated the pelvis backward. The hamstrings decelerated the forward motion of the swing-limb shank, but did not contribute substantially to angular motions of the knee. Based on these data, we hypothesize that the diminished knee extension in terminal swing exhibited by children with cerebral palsy may, in part, be caused by weak hip extensors or by impaired hip muscles on the stance limb that result in abnormal accelerations of the pelvis.

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

1. Introduction

Many children with cerebral palsy walk with diminished knee extension during the terminal swing and stance phases. Failure to extend the knee in swing is problematic, often resulting in a "crouched" posture at initial contact and an abnormally short stride. Tight hamstrings, resulting from an exaggerated reflex response or from excessive passive forces, are thought to cause the diminished knee extension in most cases (e.g., Baumann et al., 1980; Crenna, 1998; Sutherland and Davids, 1993; Tuzson et al., 2003). Thus, children with a crouched gait often undergo hamstrings lengthening surgery.

Unfortunately, it is difficult to predict which patients will benefit from hamstrings surgery; some walk with dramatically improved knee extension following treatment (DeLuca et al., 1998; Novacheck et al., 2002), while others show negligible increases in knee extension. Restoring terminal-swing knee extension in these children is challenging, in part, because the factors that extend the knee during normal walking are not well understood. Knee motions during swing are often described as "ballistic" (e.g., McGeer, 1990; Mochon and McMahon, 1980), analogous to the passive motion of a multi-link pendulum. However, metabolic energy is consumed by swing-limb

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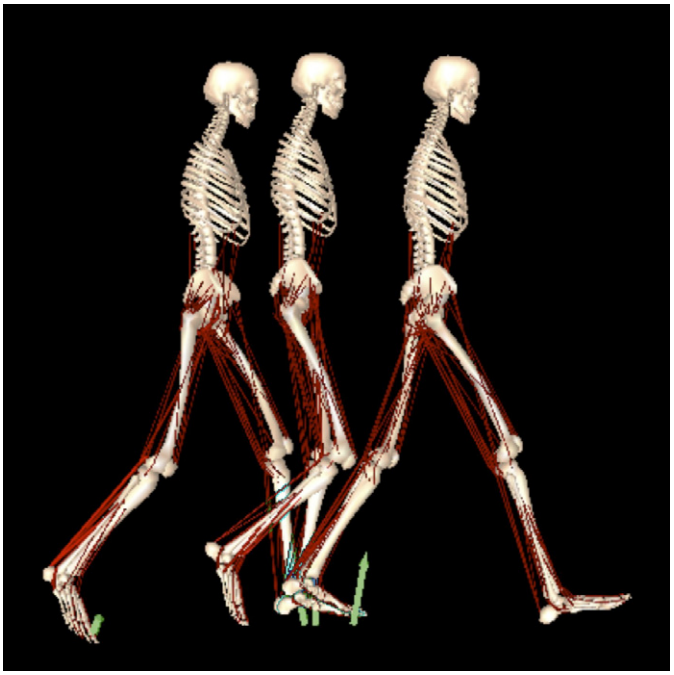

Fig. 1. Muscle-driven simulation of swing phase that reproduces the gait dynamics of a representative subject, Subject 4. To create this simulation, a musculoskeletal model with 21 degrees of freedom and 92 muscle-tendon actuators was scaled to the mass (31.7 kg) and height (1.4 m) of an 11-year-old subject who walked at a self-selected speed of 1.3 m/s. The simulation is shown at the instants just prior to toe-off (*left*), just prior to initial contact (*right*), and at peak swing-phase knee flexion (*center*).

muscles (Doke et al., 2005; Marsh et al., 2004), and some muscles—including the hamstrings—are active (Perry, 1992; Winter, 1990). The extent to which muscles influence knee motions late in swing has not been rigorously investigated. Determining how individual muscles contribute to knee motions is difficult because a muscle can accelerate joints it does not span and segments other than those to which it attaches (Zajac and Gordon, 1989; Zajac et al., 2002). For instance, several studies have shown that knee motions in early swing are influenced not only by muscles that cross the knee, but also by moments generated at other joints (Anderson et al., 2004; Kerrigan et al., 1998; Piazza and Delp, 1996).

The purpose of this study was to evaluate the angular accelerations of the knee induced by muscles, gravity, and the passive dynamics of the body during the mid- and terminal-swing phases of normal gait. We achieved this by analyzing a series of muscle-driven simulations. Our results offer insight into the actions of muscles during walking and establish a framework for the identification of factors that may limit terminal-swing knee extension in persons with cerebral palsy.

2. Methods

Muscle-driven simulations of the swing phase were generated that reproduced the measured gait dynamics of six typically developing children walking at self-selected speeds. The subjects' ages ranged from 10 to 14 years (mean 12.3 years), and their walking speeds from 1.1 to 1.4 m/s (mean 1.3 m/s). Each subject underwent gait analysis at the Gillette Children's Specialty Healthcare, St. Paul, MN. A 12-camera system (Vicon Motion Systems, Lake Forest, CA) was used to record the three-dimensional locations of markers secured to the torso, pelvis, and lower extremities during static and walking trials. Markers were placed over skeletal landmarks according to a standard clinical protocol (Davis et al., 1991), supplemented with torso markers at the seventh cervical vertebra and distal to the clavicles. The subjects' hip and knee centers were estimated using functional techniques (Schwartz and Rozumalski, 2005), and their joint angles were computed (e.g., Kadaba et al., 1990). Surface EMG was recorded bilaterally from the medial and lateral hamstrings, rectus femoris, gastrocnemius, and anterior tibialis (Motion Lab Systems, Baton Rouge, LA). Four force plates (AMTI, Watertown, MA) were used to record the ground reaction forces and moments. One barefoot trial with consecutive force plate strikes, per subject, was selected for analysis. All subjects and/or their parents provided informed consent for the collection of these data. Retrospective analyses of the data were performed in accordance with the regulations of all participating institutions.

A dynamic model of the musculoskeletal system was used, in combination with the experimental data, to create a three-dimensional simulation of each subject's swing phase (Fig. 1). We represented the musculoskeletal system as a 21-degree-of-freedom linkage actuated by 92 muscle-tendon units. The pelvis was allowed to rotate and translate in three dimensions with respect to the ground. The head, arms, and torso were represented as a single rigid segment that articulated with the pelvis via a ball-and-socket joint (Anderson and Pandy, 1999). Each hip was modeled as a ball-and-socket joint, each knee as a planar joint with constraints that specify the tibiofemoral and patellofemoral translations as a function of knee flexion angle (Yamaguchi and Zajac, 1989), and each ankle-subtalar complex as two revolute joints (Inman, 1976). Inertial properties of the segments were based on the regression equations of McConville et al. (1980). We represented each muscle-tendon unit as a Hill-type muscle in series with an elastic tendon (Schutte et al., 1993; Zajac, 1989). The attachment sites, path geometry, and force-generating properties of the muscles were based on data reported by Delp et al. (1990), and were refined for the hamstrings and adductors based on work by Arnold and Delp (2001), Arnold et al. (2000). The model was created using SIMM and the Dynamics Pipeline (Delp and Loan, 2000). The equations of motion were derived using SD/Fast (Parametric Technologies Corporation, Needham, MA). Variations of this model have been used in other studies (e.g., Thelen and Anderson, 2006), and additional details are provided elsewhere (Anderson and Pandy, 2001; Delp et al., 1990).

We generated a forward simulation of each subject's swing phase using a five-step procedure:

Step 1 was to scale the model to match the anthropometry of each subject. We scaled the dimensions of the torso, pelvis, thigh, shank, and foot based on the relative distances between pairs of markers measured experimentally and the corresponding markers in the model. The muscle attachments were scaled with the segments, and the optimal muscle fiber lengths and tendon slack lengths were scaled proportionally so that the force-generating properties of the muscles were preserved. The mass properties of the segments were scaled proportionally so that the measured mass of each subject was reproduced.

Step 2 was to use a least-squares formulation (Lu and O'Connor, 1999) to compute a set of desired joint angles for tracking, consistent with each scaled model, based on the marker trajectories, joint constraints, and joint angles from gait analysis.

Step 3 was to eliminate dynamic inconsistencies between the joint kinematics to be tracked, as computed in Step 2, and the subjects' measured ground reaction forces and moments. We did this by making small adjustments to the desired pelvis translations and torso orientations (Thelen and Anderson, 2006).

Step 4 was to solve for a set of muscle excitations which, when applied to the model along with the measured ground reaction forces and moments, reproduced the desired kinematics (Thelen and Anderson, 2006). We resolved muscle redundancy at each time step by minimizing the

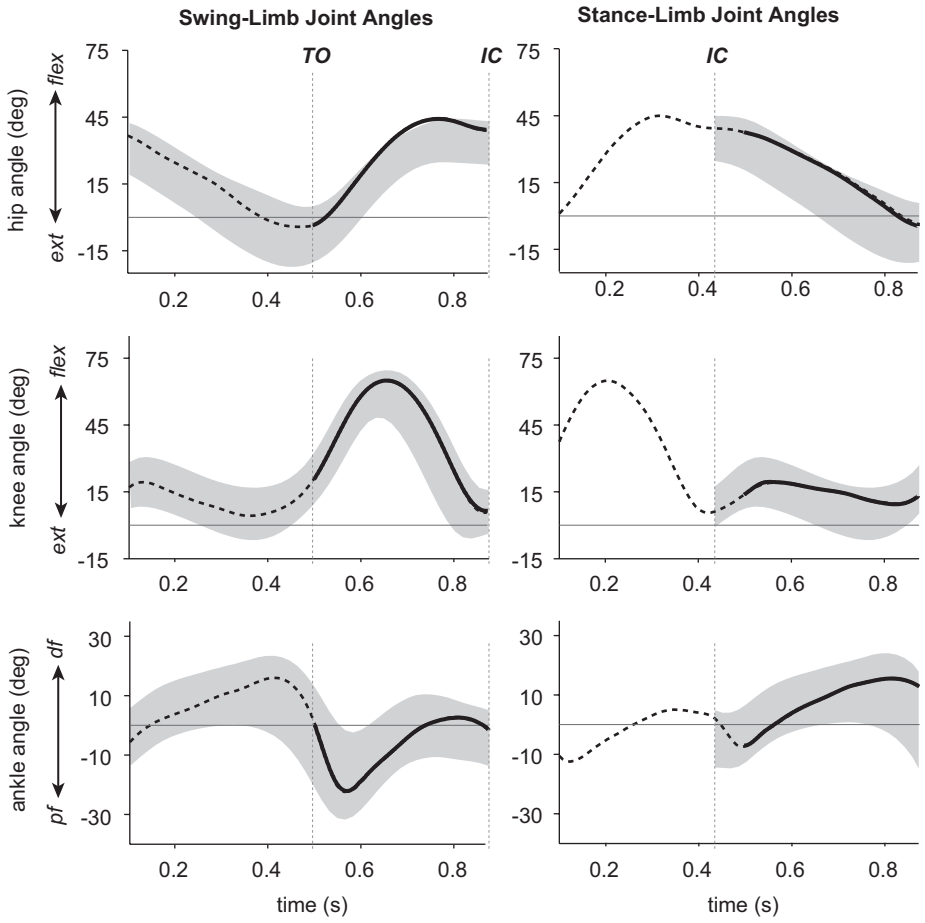

Fig. 2. Sagittal plane hip, knee, and ankle angles vs. time as determined experimentally (*dotted lines*) and as generated by the muscle-driven simulation (*solid lines*) for Subject 4. Averaged data ± 2 SD for a group of 29 unimpaired subjects (*shaded region*) are shown for comparison. For each of the six subjects in this study, data from gait analysis were collected for two or more successive strides. Simulations were generated that accurately tracked each subject's measured kinematics from toe-off (TO) through the end of the swing phase (IC) of the first stride.

sum of the muscle activations squared, weighted by the muscle volumes. Steps 2–4 are described in more detail elsewhere (Delp et al., in press; Thelen and Anderson, 2006).

Step 5 was to compare the simulations with each subject's measured gait data (Figs. 2–4), and to refine the muscle excitations as needed. In most cases, our algorithm produced excitations that were similar to the subjects' measured EMG patterns and to EMG on/off times published in the literature. In some cases, however, one or more of the muscles were excited at inappropriate times unless we constrained the solution of the algorithm (Step 4), forcing those muscles to be inactive at those times. We implemented the necessary constraints for each simulation, solved for a refined set of muscle excitations, and verified that the resulting coordination patterns were plausible (e.g., Fig. 4). We also verified that the joint angles of each simulation matched the subjects' measured joint angles to within a few degrees (e.g., Fig. 2), and that the joint moments were consistent with the moments computed from the experimental data (e.g., Fig. 3).

We analyzed the contributions of individual muscles to the angular accelerations of the swing-limb knee using a perturbation technique (Liu et al., 2006). At each 10 ms time step in each simulation, for each muscle in the model, we introduced a 1 N perturbation in the muscle's force. All other muscles were constrained to apply the same force trajectories that they applied in the unperturbed simulation. We integrated the equations of motion over a 20 ms interval to determine the changes in the accelerations of the swing-limb segments and joints per unit force. We then scaled these accelerations by the muscle's average force over the perturbation interval to determine the net accelerations attributable to that muscle, independent of other factors such as gravity. Interactions between the stance-limb foot and the ground were characterized by a set of rotational and translational spring-damper units located at the average center of pressure, as computed over the perturbation interval (Thelen and Anderson, 2006). Hence, the ground reaction forces and moments were allowed to change in response to the perturbations in force. The translational stiffness and damping coefficients (5000 kN/m and 500 N/m/s, respectively) were scaled depending on the percentage of body weight supported by the foot. The rotational stiffness and damping coefficients (20,000 N m/rad and 100 N m/rad/s^{1}, respectively) were scaled depending on the orientation of the foot, approximating a fixed-foot constraint in the interval between foot flat and heel off (Anderson and Pandy, 2003). Analogous methods were used to determine the knee motions induced independently by gravity and velocity-related forces (i.e., Coriolis and centrifugal forces).

3. Results

During normal walking at self-selected speeds, the knee is rapidly accelerated toward flexion during preswing, reaching its peak flexion velocity near toe-off (Fig. 5A). During the remainder of the swing phase, the knee is accelerated toward extension (Fig. 5B, *extension phase*), then toward flexion (Fig. 5B, *braking phase*) as the knee's

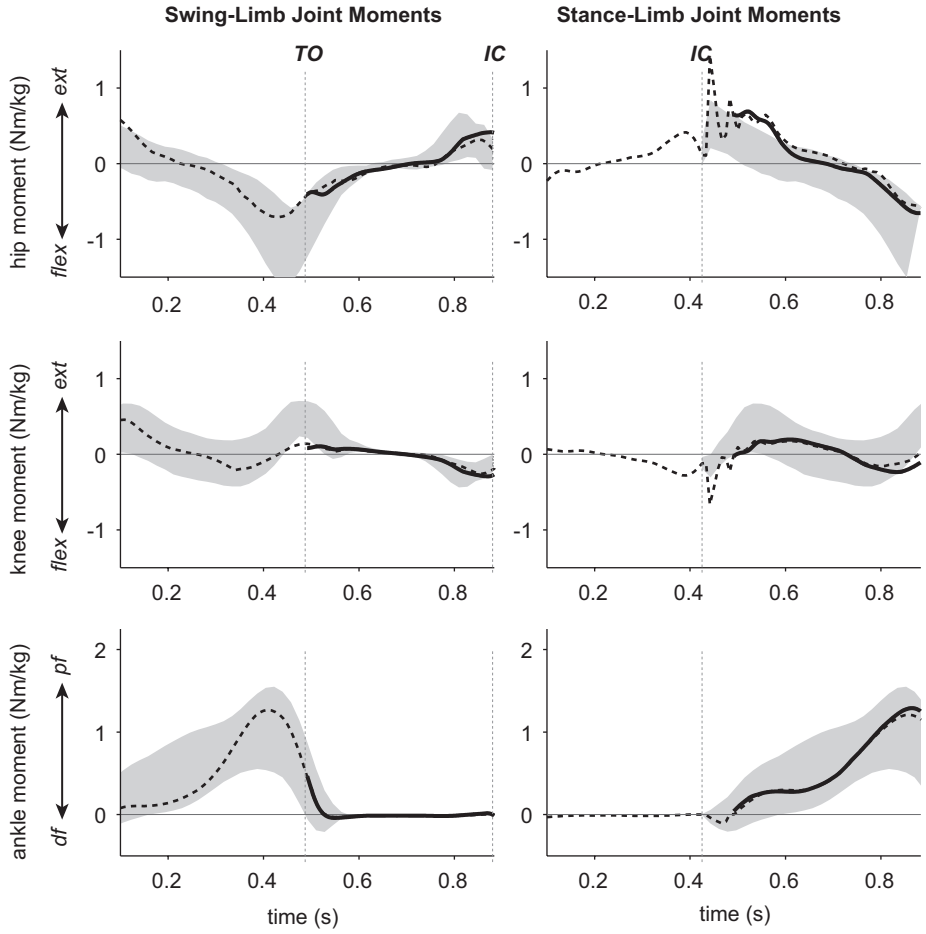

Fig. 3. Sagittal plane hip, knee, and ankle moments vs. time as determined experimentally (*dotted lines*) and as generated by the muscle-driven simulation (*solid lines*) for Subject 4. Averaged data ± 2 SD for a group of 29 unimpaired subjects (*shaded region*) are shown for comparison.

extension motion is slowed prior to contacting the ground. The knee reaches its peak flexion between 25% and 40% of the swing phase, and thereafter it extends.

In our simulations, muscles generated about half (57 ± 4%) of the knee extension acceleration during the extension phase (Figs. 5C and 6A, *dark gray bars*); the other half was provided by velocity-related forces that arose from the rotational motions of the limb segments (Figs. 5C and 6A, *light gray bars*). Muscles generated nearly all of the knee flexion acceleration during the braking phase (Figs. 5C and 6B, *dark gray bars*). These findings were consistent for all six subjects.

Muscles on the stance limb—not the swing limb—made the largest net contribution to extension of the swing-limb knee during the extension phase (Fig. 7A, *dark gray bars*). The net action of muscles on the swing limb, by contrast, was to accelerate the knee toward flexion (Fig. 7A, *light gray bars*). Examination of the motions induced by individual muscles revealed that the knee was powerfully accelerated toward extension in our simulations by the stance-limb hip abductors (Fig. 8B). These muscles provide vertical support (Anderson and Pandy, 2003; Kimmel and Schwartz, 2006; Liu et al., 2006) and typically generate large forces in early and mid-stance. The swing-limb hip flexors, biceps femoris short head, and ankle dorsiflexors, all of which are activated during early swing, contributed to knee flexion (Fig. 8A). Passive forces produced by the swing-limb vasti and residual forces produced by the uniarticular ankle plantarflexors, remaining from their activity during stance, opposed knee flexion in our simulations (Fig. 8A).

During the braking phase, both swing- and stance-limb muscles contributed to deceleration of the knee (Fig. 7B). The swing-limb ankle dorsiflexors (Fig. 9A) and the stance-limb hip flexors (Fig. 9B), in particular, accelerated the swing-limb knee toward flexion. Other muscles generated forces that accelerated the knee toward extension, notably the swing-limb hip extensors (Fig. 9A) and the uniarticular ankle plantarflexors (passive). The stance-limb hip abductors also induced terminal-swing knee extension in our simulations (Fig. 9B).

4. Discussion

Classic texts on walking frequently assert that the knee extends passively under the influence of gravity and/or

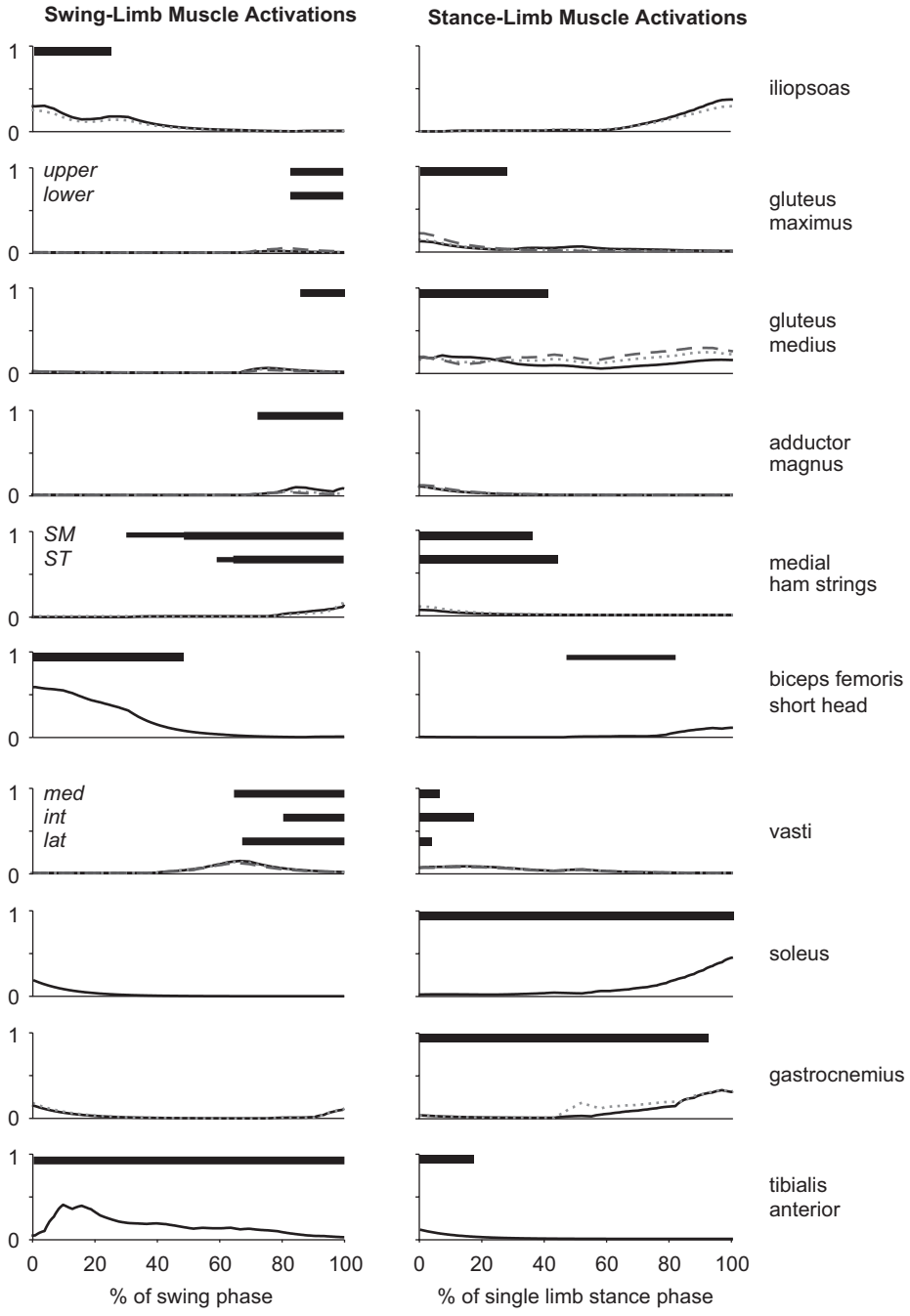

Fig. 4. Activation patterns for 42 of the 92 muscle-tendon actuators on the swing limb (*left*) and stance limb (*right*) used to drive the simulation of Subject 4. The activation patterns of muscles with similar actions (e.g., iliacus and psoas) and muscles represented by multiple compartments (e.g., gluteus medius) are displayed on the same plot (*represented by different line types*). Corresponding EMG on/off times published by Perry (1992), scaled to the subject's measured stance and swing phases, are overlaid for comparison (*solid bars; the thinner bars indicate inconsistencies in EMG timing as documented by Perry*). Note that our musculoskeletal model has the isometric force-generating capacity of an adult, while Subject 4 has the anthropometry of an 11-year-old child. The magnitudes of the muscle activations, therefore, reflect the relatively small activations (and forces) needed to track the subject's gait dynamics.

velocity-related forces during the mid- and terminal-swing phases, analogous to the passive dynamics of a multi-link pendulum (e.g., Boakes and Rab, 2006; Gage, 2004; Perry, 1992; Whittle, 1996). In this study, we quantified the angular accelerations of the swing-limb knee induced by muscles, gravity, and the passive dynamics of the body in six children with normal gait. In contrast to the classic texts, our analysis suggests that both muscular and velocity-related forces, but not gravity, make important contributions to terminal-swing knee motions during walking, at self-selected speeds.

Gravity did not contribute substantially to angular motions of the knee in our simulations (Figs. 5C and 6, *white bars*) because it accelerated *all* segments of the swing

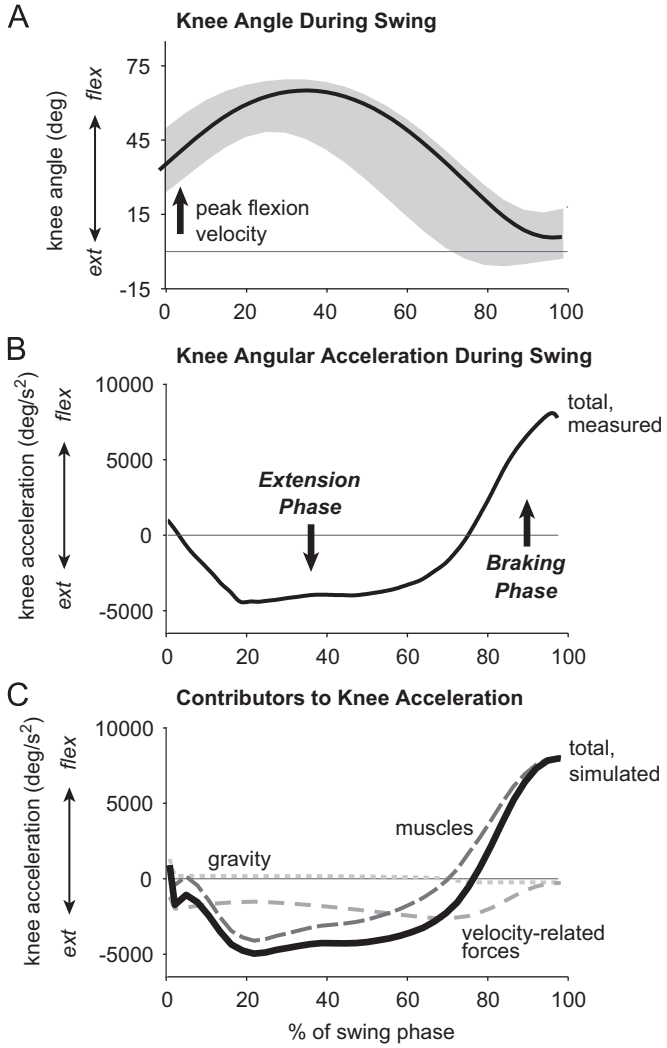

Fig. 5. Knee flexion angle (A) and angular acceleration during the swing phase as determined experimentally (B) and as generated by the muscle-driven simulation of Subject 4 (C). Prior to toe-off, the knee is rapidly accelerated toward flexion. Shortly after toe-off, the knee stops accelerating toward flexion, and starts accelerating toward extension due to the actions of muscles and velocity-related forces. In late swing, the knee stops accelerating toward extension and starts accelerating toward flexion due to the actions of muscles. The *extension phase* is defined as the interval during which the knee is accelerated toward extension; the *braking phase* is defined as the interval during which the knee is accelerated toward flexion.

limb downward, nearly uniformly. If we had analyzed a simpler model consisting of only the swing limb, in which the trajectory of the pelvis was prescribed, then gravity would have accelerated the knee toward extension during the extension phase. This explanation has been noted previously (Anderson et al., 2004).

Muscles on the stance limb, particularly the hip abductors, extensors, and flexors, had a major influence on motions of the swing-limb knee in our simulations (Figs. 8B–9B). These muscles, in combination with their induced ground reaction forces, accelerated the pelvis, simultaneously inducing reaction forces at the swing-limb hip that accelerated the thigh and knee (Fig. 10). For instance, during early and mid-stance, corresponding to the extension phase of the swing-limb knee (Fig. 5B), the hip abductors and extensors produced forces that accelerated the pelvis center of mass upward and rotated the pelvis posteriorly. As a result, the swing-limb thigh was accelerated forward relative to the pelvis, and the swing-limb hip and knee were accelerated toward extension (Fig. 10A). In late stance, corresponding to the braking phase of the swing-limb knee (Fig. 5B), the hip flexors produced forces that accelerated the pelvis center of mass backward and rotated the pelvis anteriorly. As a result, the swing-limb thigh was accelerated backward relative to the pelvis, and the swing-limb hip and knee were accelerated toward flexion (Fig. 10B). If we had analyzed a simpler model consisting of only the swing limb, in which the trajectory of the pelvis was prescribed (e.g., Piazza and Delp, 1996), then these actions of the stance-limb muscles would not have been elucidated.

Our conclusion that motions of the swing-limb knee are sensitive to the forces generated by stance-limb hip muscles is consistent with other studies. For example, Mena et al. (1981) analyzed a planar, three-segment model of the swing limb and showed that when the prescribed trajectory of the hip was exaggerated, the motions of the knee were abnormal. Anderson et al. (2004) used a simulation to identify the contributions of muscles and toe-off kinematics to peak knee flexion during early swing, and reported that the net effect of stance-limb muscles, particularly the gluteus maximus and gluteus medius/minimus, was to oppose knee flexion. Wang et al. (2005), using a torque-driven simulation, showed that reasonable stepping motions could be generated simply by controlling pelvis motion.

Muscles on the swing limb crossing the hip, knee, and ankle also generated forces that affected knee motions in our simulations. However, the net effect of swing-limb muscles was small relative to stance-limb muscles, particularly during the braking phase (Fig. 7B), because several of the swing-limb muscles induced opposing accelerations of the knee (Fig. 9A). For example, the ankle dorsiflexors generated forces that accelerated the knee and ankle toward flexion. The hip extensors, by contrast, accelerated the hip and knee toward extension (Fig. 11B).

It is commonly thought that the hamstrings are activated in terminal swing to restrain both hip flexion and knee extension in preparation for contacting the ground (e.g., Boakes and Rab, 2006; Perry, 1992; van de Crommert et al., 1996). Therefore, we were surprised to discover that the hamstrings in our simulations did *not* contribute substantially to motions of the swing-limb knee (e.g., Fig. 9), even though they were actively generating force in terminal swing. This was due to dynamic coupling: the hamstrings' knee flexion moment accelerated the knee toward flexion, but the hamstrings' hip extension moment accelerated the knee toward extension. Further analysis of the muscle actions revealed that the hamstrings decelerated the forward motion of the swing-limb shank (Fig. 12). The

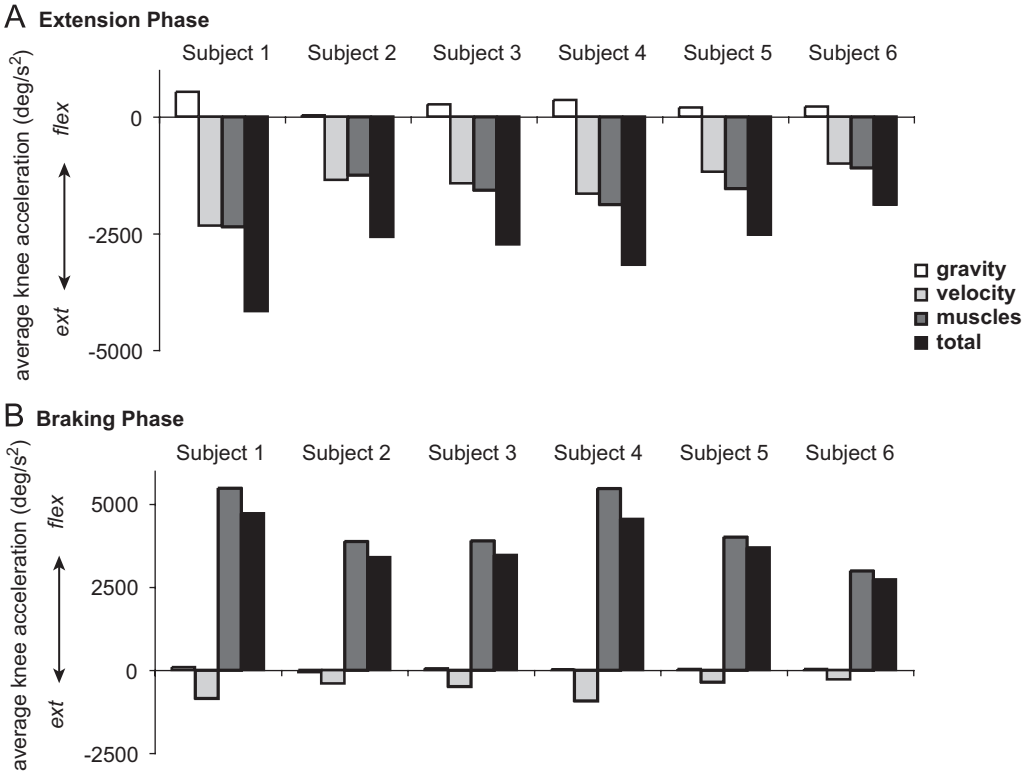

Fig. 6. Angular acceleration of the swing-limb knee induced by gravity, velocity-related forces, and muscles, averaged over the extension phase (A) and over the braking phase (B), for the six subjects in this study. Subjects are numbered in order of decreasing walking speed.

hamstrings decelerated the shank, without substantially influencing rotational motions of the knee, because they simultaneously accelerated the pelvis forward and the hip into extension.

It is pertinent to consider some of the limitations of this study. First, our estimates of the motions induced by muscles depend on the forces applied by the muscles during the simulations. We fine-tuned the timing of the muscle excitations based on detailed comparisons with measured and published EMG recordings (Fig. 4), and we verified that the simulations accurately reproduced the subjects' measured gait data (Figs. 2 and 3); thus, we believe that the forces generated by most muscles in our simulations are reasonable. Nevertheless, the forces produced by some muscles, such as the swing-limb vasti and stance-limb hip abductors, remain questionable. Our tracking algorithm chose not to excite the vasti in terminal swing, inconsistent with EMG recordings, because these muscles were shortening too rapidly to generate much force. If the vasti had generated more force, then they would have made larger contributions to terminal-swing knee extension (Fig. 9A). The gluteus medius in our simulations exhibited prolonged excitation during stance as compared to EMG data. Hence, our analysis may have exaggerated the contribution of the stance-limb hip abductors to swing-limb knee extension, particularly in the late extension phase. The forces produced by the back muscles in our simulations also remain questionable. We did not attempt to measure or track motions of the subjects' arms. As a result, the back muscles may have been activated, in part, to compensate for unmodeled forces on the torso.

We analyzed the muscle actions at the body positions corresponding to normal walking at self-selected speeds. The potential of the muscles to accelerate the knee might be different at the body positions corresponding to crouch gait, or at speeds much faster or slower than normal. To better understand the muscle actions during crouch gait, simulations that reproduce the musculoskeletal geometry and gait dynamics of individuals with crouch gait are needed. The data reported in this study establish a baseline for assessing how the muscle actions might change with variations in bone geometry, walking speed, or posture.

The muscle-induced accelerations reported in this study describe the actions of individual muscles or groups of muscles acting in isolation. To identify the source of a patient's diminished knee extension, it may be necessary to consider how excessive or insufficient force in one muscle might change the forces in other muscles. For example, a patient with crouch gait may exhibit diminished activity of the gluteus maximus in terminal swing to compensate for

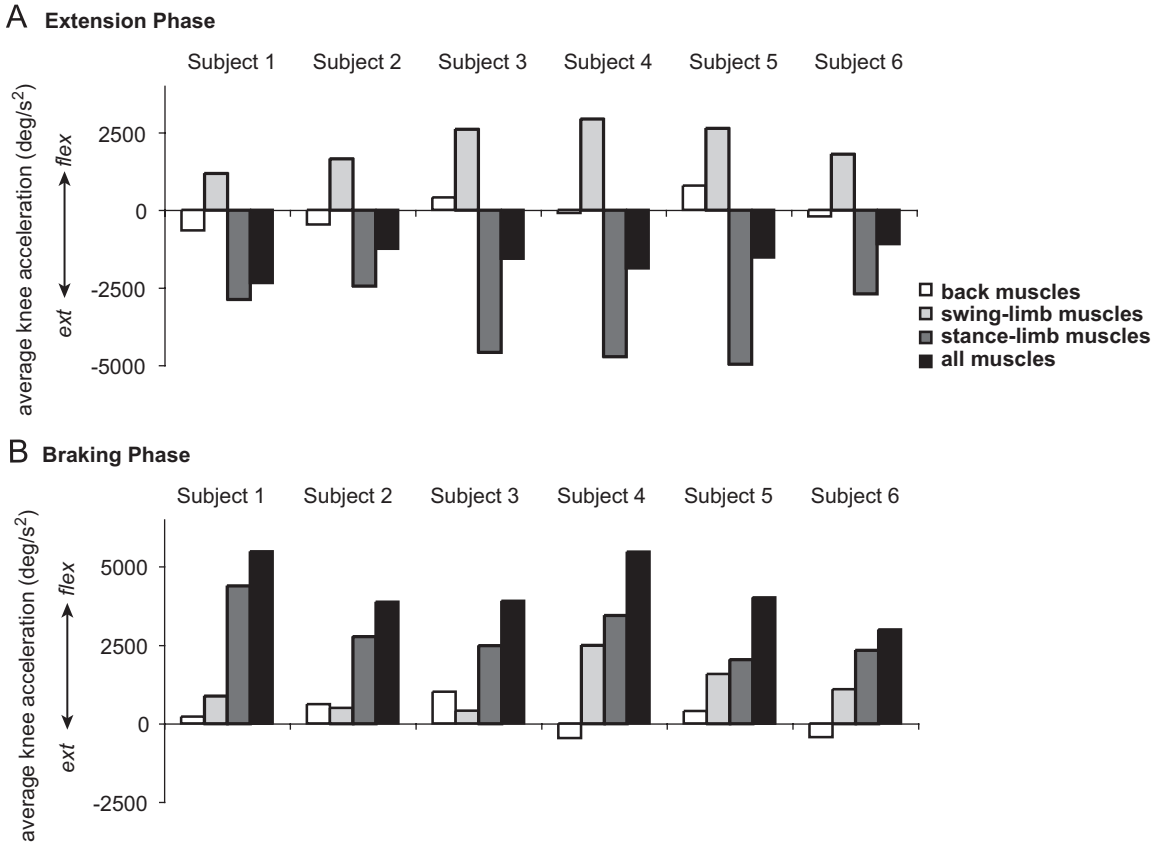

Fig. 7. Angular acceleration of the swing-limb knee induced by back muscles, swing-limb muscles, and stance-limb muscles, averaged over the extension phase (A) and over the braking phase (B), for the six subjects in this study. Subjects are numbered in order of decreasing walking speed.

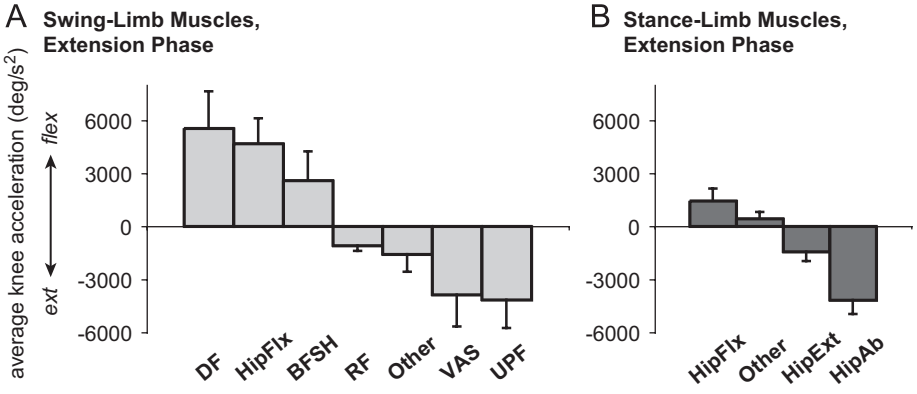

Fig. 8. Angular acceleration of the swing-limb knee induced by individual muscles or groups of muscles on the swing limb (A) and on the stance limb (B), averaged over the extension phase. *DF*, the ankle dorsiflexors, includes tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius. *HipFlx*, the hip flexors, includes iliacus, psoas, tensor fasciae latae, and sartorius. *BFSH* is the biceps femoris short head. *RF* is the rectus femoris. *VAS* includes vastus medialis, vastus intermedius, and vastus lateralis. *UPF*, the uniarticular ankle plantarflexors, includes soleus, tibialis posterior, flexor digitorum longus, flexor hallucis longus, peroneus longus, and peroneus brevis. *HipExt*, the stance-limb hip extensors, includes gluteus maximus, hamstrings, and adductor magnus. *HipAb*, the stance-limb hip abductors, includes gluteus medius and gluteus minimus. *Other* includes all other muscles of the corresponding limb in the model.

the excessive force produced by tight hamstrings. If the hamstrings' force was reduced, and the gluteus maximus' force increased, then the patient's knee extension might improve—not because the hamstrings were the direct source of the excessive knee flexion, but because the gluteus maximus powerfully accelerates the knee toward extension.

Identifying the factors that influence terminal-swing knee extension during normal gait is an important step toward explaining the causes of crouch gait and the consequences of common interventions. The results of this study suggest that diminished knee extension in swing could potentially arise from weak hip extensors on the swing limb, or by impaired hip muscles on the stance limb

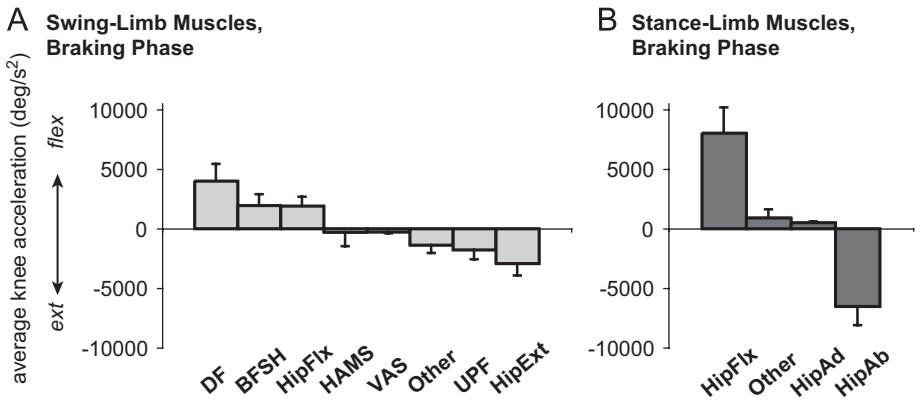

Fig. 9. Angular acceleration of the swing-limb knee induced by individual muscles or groups of muscles on the swing limb (A) and on the stance limb (B), averaged over the braking phase. *DF*, the ankle dorsiflexors, includes tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius. *BFSH* is the biceps femoris short head. *HipFlx*, the hip flexors, includes iliacus, psoas, tensor fasciae latae, and sartorius. *HAMS*, the hamstrings, includes semimembranosus, semitendinosus, and biceps femoris long head. *VAS* includes vastus medialis, vastus intermedius, and vastus lateralis. *UPF*, the uniarticular ankle plantarflexors, includes soleus, tibialis posterior, flexor digitorum longus, flexor hallucis longus, peroneus longus, and peroneus brevis. *HipExt*, the swing-limb hip extensors, includes gluteus maximus and adductor magnus. *HipAd*, the stance-limb hip adductors, includes pectineus, adductor brevis, adductor longus, and gracilis. *HipAb*, the stance-limb hip abductors, includes gluteus medius and gluteus minimus. *Other* includes all other muscles of the corresponding limb in the model.

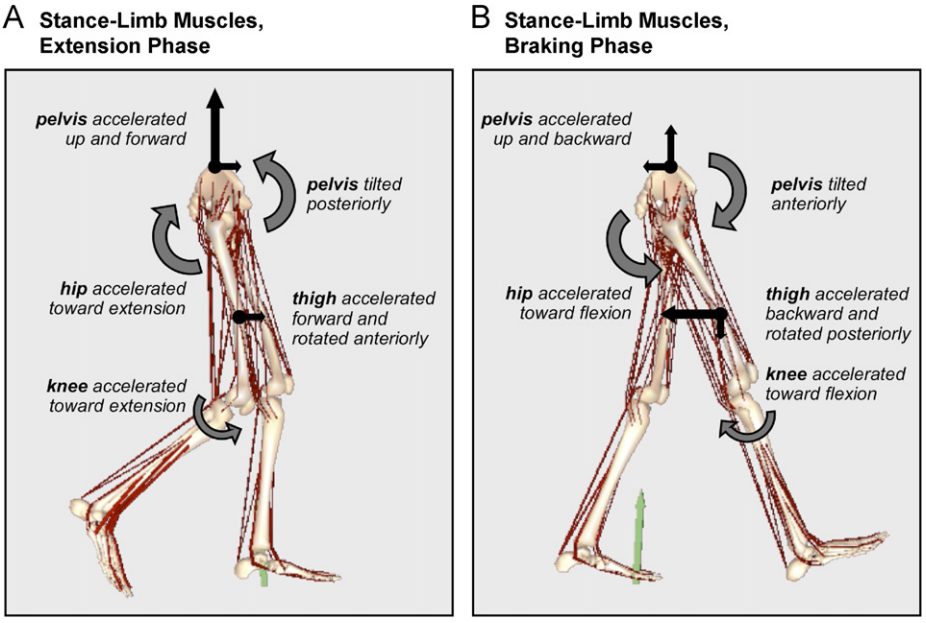

Fig. 10. Motions of the pelvis and swing limb induced by all stance-limb muscles during the extension phase (A) and braking phase (B). Straight arrows represent translational accelerations, and curved arrows represent angular accelerations. All arrows are scaled proportional to their magnitudes. Accelerations of the thigh are calculated relative to the pelvis. Stance-limb muscles accelerated the center of mass of the entire model (*not shown*) upward and backward during the extension phase, and upward and forward during the braking phase, consistent with previous studies (Liu et al., 2006).

that result in abnormal accelerations of the pelvis. Our next step, to test these hypotheses, is to analyze simulations of patients with diminished knee extension in swing.

Conflict of interest

There is no conflict of interest.

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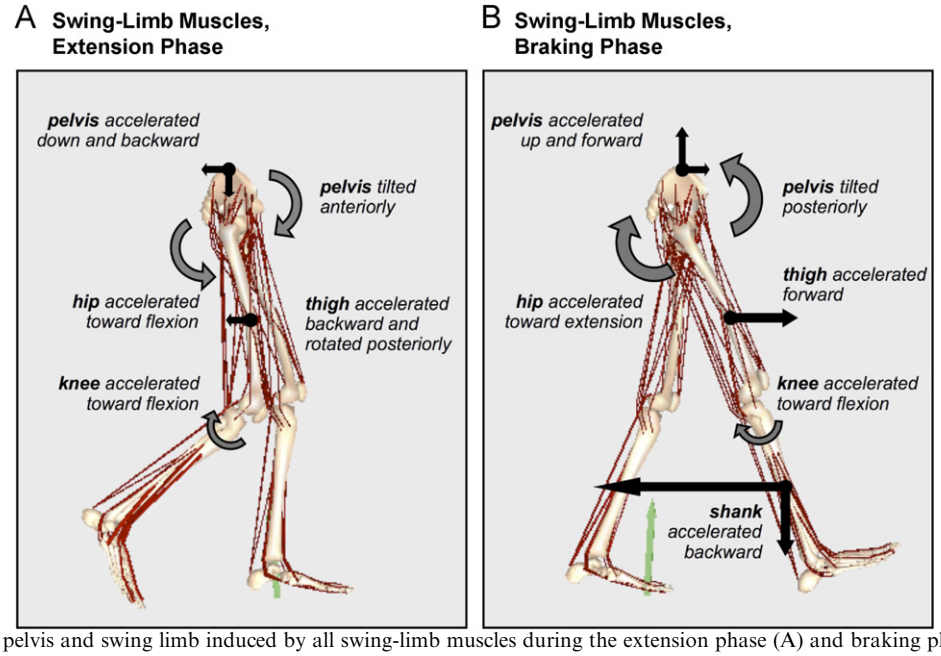

Fig. 11. Motions of the pelvis and swing limb induced by all swing-limb muscles during the extension phase (A) and braking phase (B). Straight arrows represent translational accelerations, and curved arrows represent angular accelerations. All arrows are scaled proportional to their magnitudes. Accelerations of the thigh are calculated relative to the pelvis, and accelerations of the shank are calculated relative to the thigh.

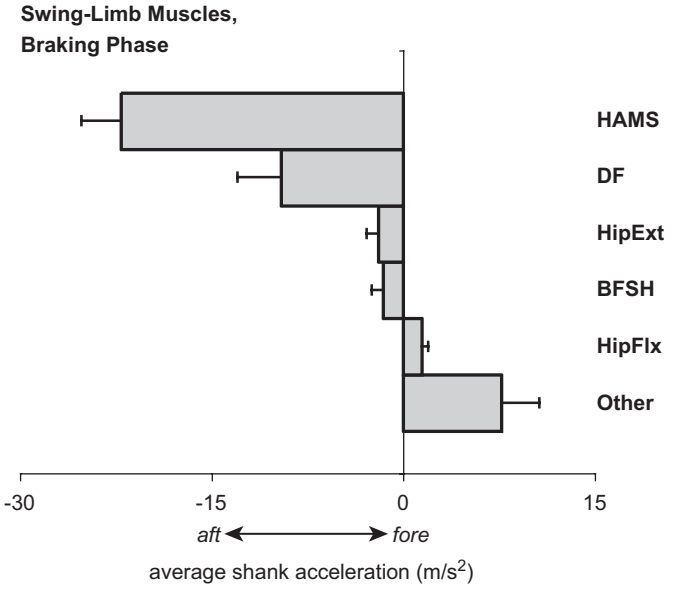

Fig. 12. Fore-aft acceleration of the swing-limb shank induced by groups of muscles on the swing limb, averaged over the last half of the braking phase. *HAMS*, the hamstrings, includes the semimembranosus, semitendinosus, and biceps femoris long head. *DF*, the ankle dorsiflexors, includes tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius. *HipExt*, the hip extensors, includes gluteus maximus and adductor magnus. *BFSH* is the biceps femoris short head. *HipFlx*, the hip flexors, includes iliacus, psoas, tensor fasciae latae, and sartorius. *Other* includes all other muscles of the swing limb in the model.

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