Muscles that influence knee flexion velocity in double support: implications for stiff-knee gait

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Abstract

Adequate knee flexion velocity at toe-off is important for achieving normal swing-phase knee flexion during gait. Consequently, insufficient knee flexion velocity at toe-off can contribute to stiff-knee gait, a movement abnormality in which swing-phase knee flexion is diminished. This work aims to identify the muscles that contribute to knee flexion velocity during double support in normal gait and the muscles that have the most potential to alter this velocity. This objective was achieved by perturbing the forces generated by individual muscles during double support in a forward dynamic simulation of normal gait and observing the effects of the perturbations on peak knee flexion velocity. Iliopsoas and gastrocnemius were identified as the muscles that contribute most to increasing knee flexion velocity during double support. Increased forces in vasti, rectus femoris, and soleus were found to decrease knee flexion velocity. Vasti, rectus femoris, gastrocnemius, and iliopsoas were all found to have large potentials to influence peak knee flexion velocity during double support. The results of this work indicate which muscles likely contribute to the diminished knee flexion velocity at toe-off observed in stiff-knee gait, and identify the treatment strategies that have the most potential to increase this velocity in persons with stiff-knee gait.
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1. Introduction

Knee flexion velocity at toe-off is an important factor in generating swing-phase knee flexion during normal gait (Mochon and McMahon, 1980; Mena et al., 1981; Piazza and Delp, 1996). Low knee flexion velocity at toe-off is a potential contributor to stiff-knee gait, a movement abnormality associated with stroke and cerebral palsy in which swing-phase knee flexion is diminished. Stiff-knee gait is commonly attributed to excessive activity of the rectus femoris muscle, which is thought to limit knee flexion by producing an excessive knee extension moment during swing (Perry, 1987; Sutherland et al., 1990). However, Goldberg et al. (2003) observed that many stiff-knee patients with

cerebral palsy do not exhibit excessive knee extension moments during swing phase, but instead walk with a low knee flexion velocity at toe-off. Goldberg et al. (2003) showed that a simulated increase in knee flexion velocity at toe-off resulted in an increased range of knee flexion in swing, suggesting that interventions that increase this velocity have the potential to improve peak knee flexion during swing for stiff-knee patients. To determine which interventions have the most potential to alter knee flexion velocity at toe-off, the muscles that contribute to this velocity must be identified.

Knee flexion velocity increases dramatically during double support. Comparison of observed muscle activity and gait kinematics has led to the belief that gastrocnemius makes a large contribution to knee flexion during late stance, with additional contributions from poplitius and occasionally gracilis (Perry, 1992). Rectus femoris (Perry, 1992) and possibly vasti (Winter,

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1991) are in some cases active during this period and are thought to limit knee flexion. However, because muscles can accelerate joints that they do not span (Zajac and Gordon, 1989), a muscle's role in producing movement cannot be deduced from these observations alone. Dynamic simulations that estimate muscle forces and accurately represent the geometry of the lower limbs during the gait cycle can be used to assess how muscles accelerate the joints. Yamaguchi and Zajac (1990) identified gastrocnemius and iliopsoas as muscles that accelerate the knee into flexion during double support. In a more recent study, gastrocnemius, as well as soleus and vasti, were found to accelerate the knee into extension during double support (Neptune et al., 2001, Fig. 8).

The objectives of this work were: (1) to identify the muscles that contribute to knee flexion velocity during double support in normal gait, and (2) to rank the potential of individual muscles to alter this velocity. We addressed these objectives by perturbing muscle forces during double support in a forward dynamic simulation of normal gait and observing the resulting changes in peak knee flexion velocity. The results of this study not only contribute to our understanding of muscle function in normal walking, but also suggest the potential causes of low knee flexion velocity at toe-off in persons with stiff-knee gait and possible treatment strategies to increase knee flexion velocity.

2. Methods

Our analysis was based on the muscle forces and kinematics predicted by a dynamic optimization solution for a half-cycle of normal gait (Anderson and Pandy, 2001a). The model used to generate this solution was a 10 segment, 23 degree-of-freedom linkage controlled by 54 musculotendon actuators. 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. Interactions of the feet with the ground were modeled using five independent visco-elastic elements distributed under the sole of each foot. Each actuator was modeled as a three-element, Hill-type muscle in series with tendon (Zajac, 1989) with musculotendon parameters based on Delp et al. (1990). See Anderson and Pandy (1999) for details concerning the model. The joint angular displacements, ground reaction forces, muscle excitation patterns, and time/distance parameters (forward velocity of $1.36\,\mathrm{m\,s^{-1}}$, step length of $0.76\,\mathrm{m}$) of the gait produced by the solution were similar to those obtained from healthy subjects (Anderson and Pandy, 2001a). See Anderson and Pandy (2001b) for the time histories of muscle forces predicted by the optimal solution.

Forward dynamic simulations of gait were performed in which the unaltered muscle excitations from the optimal solution served as the controls and the forces of individual muscles were perturbed from their original values during the period of double support. For each perturbed simulation, the peak knee flexion velocity during double support was recorded to determine the effect of the perturbation. Perturbations were made by either scaling the force in the muscle during double support by a percentage of the unperturbed force or by adding (or subtracting) a fixed force in Newtons to the unperturbed force throughout double support (Fig. 1). The change in peak knee flexion velocity due to a scaled perturbation is dependent on the force output of a muscle during the simulation; it characterizes how much
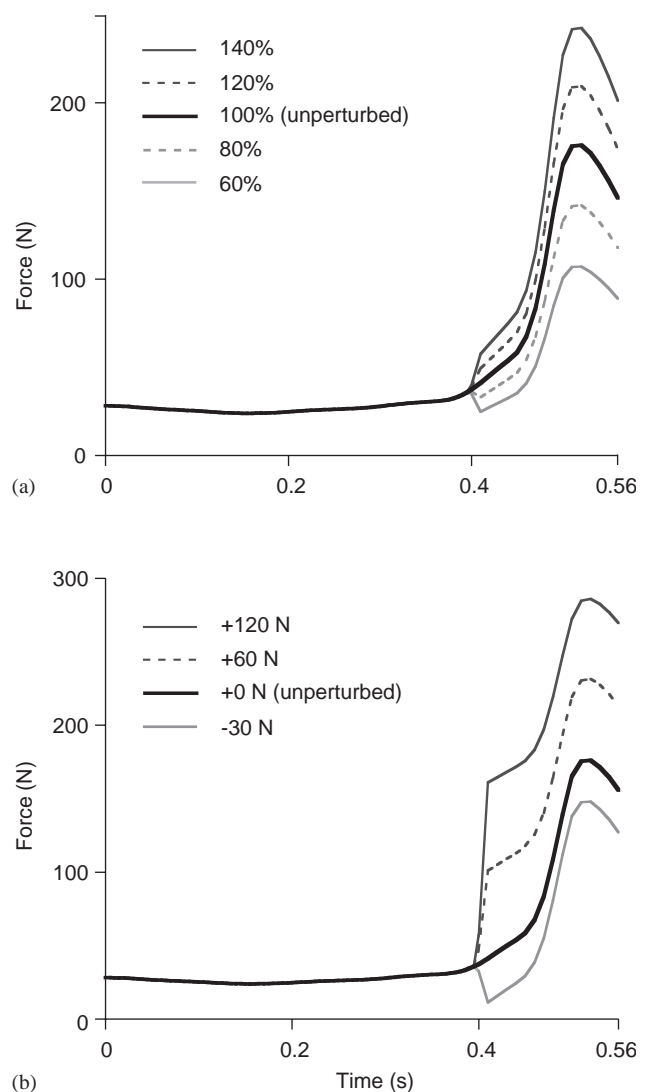

Fig. 1. Examples of perturbed muscle force trajectories. Muscle forces were perturbed only during the period of double support (from 0.4 to 0.56 s). Forces were perturbed in one of two ways: (a) the force was scaled by a percentage of the unperturbed force, or (b) a fixed perturbation in Newtons was added to or subtracted from the unperturbed force trajectory.

the force in that muscle influenced peak knee flexion velocity during the simulation. The change in peak knee flexion velocity due to a fixed perturbation in Newtons is independent of the unperturbed force in a muscle and characterizes the potential of a muscle to influence the velocity based on its geometry (i.e. muscle moment arms and muscle path). Therefore, the potential of a muscle to contribute to peak knee flexion velocity could be determined whether or not the muscle was active during the perturbation period.

Peak knee flexion velocity (Fig. 2a), rather than the knee flexion velocity at toe-off, was chosen as the variable of interest because it could be consistently identified for each simulation and could be measured even in cases when toe-off was not reached before the end of a perturbed simulation. (Toe-off was not reached when a perturbation in muscle force resulted in a delay in toe-off. In these cases, it was not possible to simulate to the time of the new toe-off because the controls for the simulation (i.e., the muscle excitations) ended at the toe-off time of the unperturbed simulation.) Peak knee flexion velocity was found to correlate closely with knee flexion velocity at toe-off in normal subjects (Fig. 3).

We analyzed the change in peak knee flexion velocity of the pre-swing limb due to perturbations in each muscle in both limbs of the lower extremity, including iliopsoas, adductor magnus, anterior gluteus medius and minimus, posterior gluteus medius and minimus, gluteus maximus, sartorius, gracilis, hamstrings (combined semimembranosus, semitendonosus, and biceps femoris long head), rectus femoris, vasti (combined vastus medialis, lateralis, and intermedius), biceps femoris short head, gastrocnemius, and soleus. A complete list of all muscles analyzed is given in Appendix A. Each muscle was perturbed individually. Perturbations were made in increments of 5.0 N or 5% for the fixed and scaled perturbations, respectively, until any one of three conditions occurred: (1) the change in velocity with perturbation size became non-linear, (2) the perturbed sagittal plane joint angles of the hip, knee, or ankle deviated by more than one standard deviation from the unperturbed kinematics, or (3) a muscle force became negative. Linearity was defined as the data being fit by a line with $R^2 > 0.99$. One standard deviation was defined as that of the experimental subject data used to validate the original optimization solution (Anderson and Pandy, 2001a). This criterion was enforced to prevent perturbations from resulting in unrealistic joint configurations.

The change in peak knee flexion velocity due to a perturbation (Fig. 2b, ΔV) was plotted against perturbation size. The range of allowable perturbations, as determined by the conditions given above, differed between muscles (Fig. 4 and in Appendix A the table given). The change in peak knee flexion velocity with perturbation size exhibited a large linear range for most muscles. This enabled the slope of the data to be used to
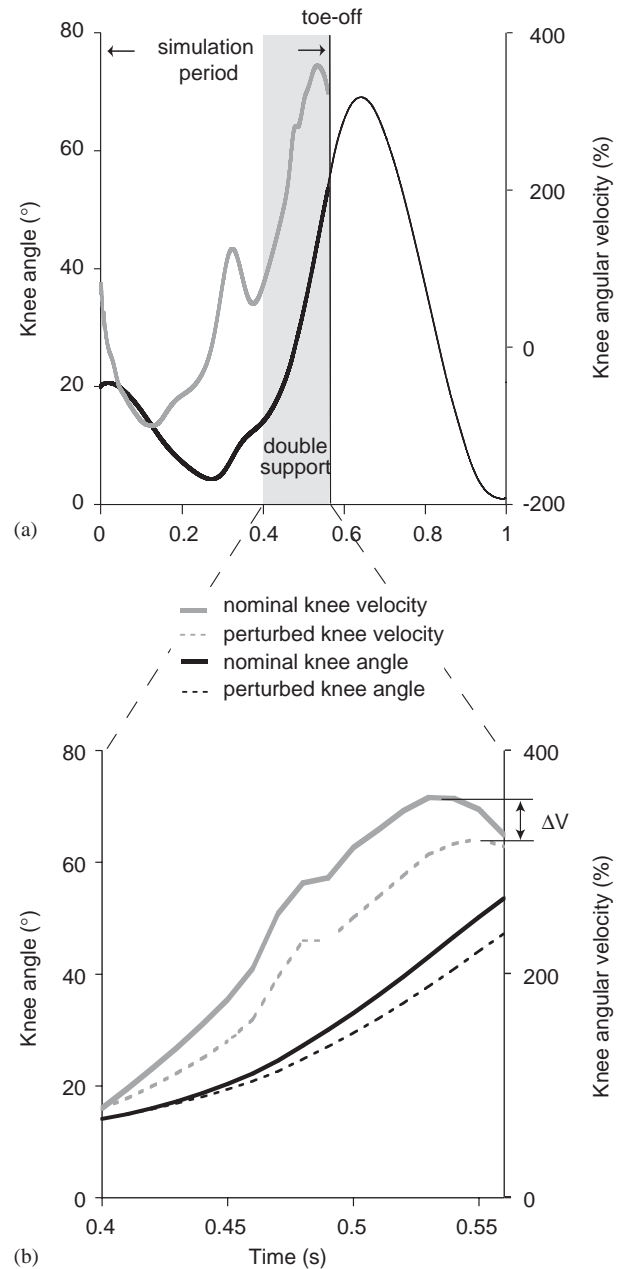

Fig. 2. Knee flexion angle and angular velocity of the pre-swing limb (a) for the unperturbed simulation and (b) due to a perturbation in muscle force. (a) The simulation period for the pre-swing limb began at single support and continued through double support, ending at toe-off. (The knee flexion angle after toe-off is shown for clarity only.) (b) The dotted black and gray curves show an example of the knee flexion angle and velocity resulting from a perturbation in an individual muscle force during the period of double support. The peak in the knee flexion velocity was diminished and delayed due to the perturbation. This change in peak knee flexion velocity (ΔV) is a function of perturbation size and is used to calculate the influence and potential influence of each muscle on peak knee flexion velocity. Note that the final value of the knee flexion velocity in the perturbed case is not the knee flexion velocity at toe-off, as toe-off was delayed past the end of the simulation period and was never reached.

evaluate the effect of muscles on peak knee flexion velocity. In the case of the fixed perturbation, the slope ($\Delta V/\Delta F$) has the units of (°/s)/N and will be referred to as the potential influence of a muscle on peak knee flexion velocity, as this value characterizes how much a muscle could influence the peak knee flexion velocity if it were to generate force during double support. In the case of the scaled perturbation, the slope (ΔV/(force/ unperturbed force)) has the units of °/s and will be referred to as the influence of a muscle on peak knee flexion velocity, as this value characterizes how much a
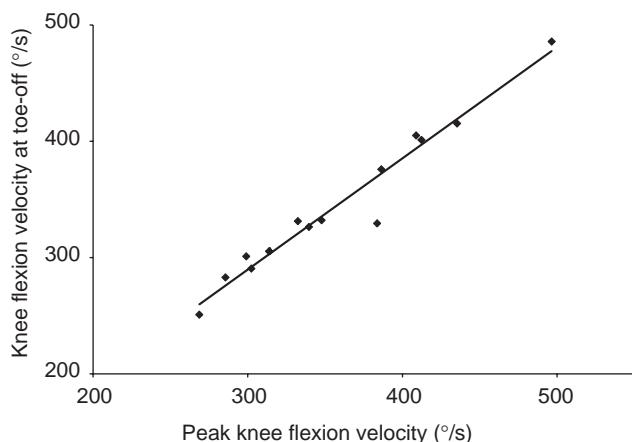

Fig. 3. Knee flexion velocity at toe-off vs. peak knee flexion velocity for 14 unimpaired subjects. The linear relationship shows that the two values of knee flexion velocity are closely correlated. The normal subject data were collected as described in Goldberg et al. (2003). Toe-off was taken to be the time at which the vertical ground reaction force dropped below 5% body weight.

muscle contributed to generating peak knee flexion velocity during the simulation. Only results for the most influential and clinically relevant muscles will be discussed. Potential influence data for all muscles are summarized in the table given in Appendix A.

3. Results

Sartorius and gracilis, biarticular muscles that have flexion moment arms at the hip and knee during double support, had the largest potential to increase peak knee flexion velocity (Fig. 5). These muscles generate hip flexion moments and knee flexion moments, both of which promote knee flexion. Rectus femoris, a biarticular muscle that has a flexion moment arm at the hip and an extension moment arm at the knee, had the potential to decrease peak knee flexion velocity, indicating that the knee extension moment it generated had a larger effect on the knee than the hip flexion moment it generated. Gluteus maximus and vasti, uniarticular muscles that extend the hip and knee, respectively, had large potentials to decrease knee flexion velocity, whereas biceps femoris short head, a uniarticular knee flexor, had a large potential to increase knee flexion velocity. Gastrocnemius and soleus had opposing potential influences on peak knee flexion velocity; increased force in gastrocnemius increased the peak knee flexion velocity, while increased force in soleus decreased it.

Iliopsoas and gastrocnemius contributed the most to peak knee flexion velocity during double support
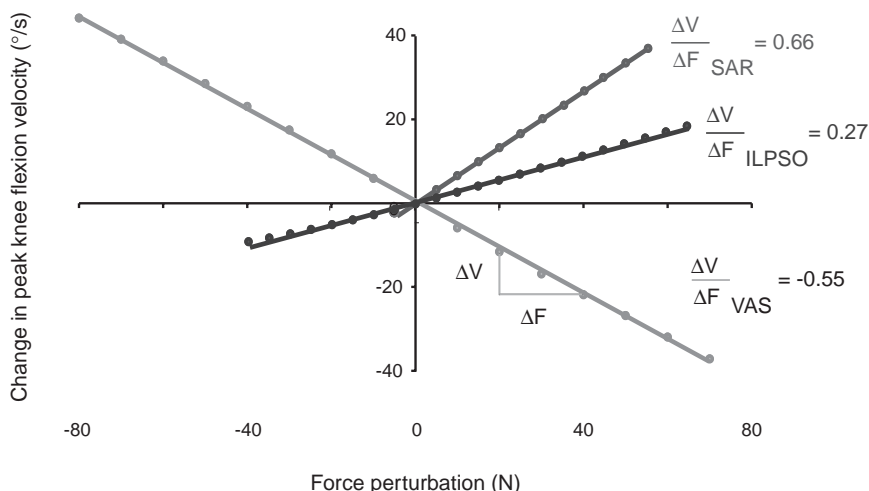

Fig. 4. Potential influence of sartorius (SAR), iliopsoas (ILPSO), and vasti (VAS) on the peak knee flexion velocity during double support. The potential influence of a muscle is the change in the peak knee flexion velocity (ΔV) per unit change in muscle force (ΔF), and is measured by the slope of a line fit to the data. A negative slope indicates that an increase in the force of the muscle would result in a decrease in peak knee flexion velocity during double support. The range of perturbation sizes made in each muscle was restricted such that each of three criteria were simultaneously met: maintenance of linearity, restriction of the perturbed kinematics to be within one standard deviation of the unperturbed kinematics, and maintenance of a positive muscle force (see text for details). The three muscles shown are examples of each criteria serving as the limiting criteria. The range of perturbations made in ilopsoas was restricted to maintain linearity, in sartorius to prevent negative muscle forces, and in vasti the limiting criteria was the limitation on the perturbed kinematics. The perturbation ranges for all muscles are given in the table in Appendix A.

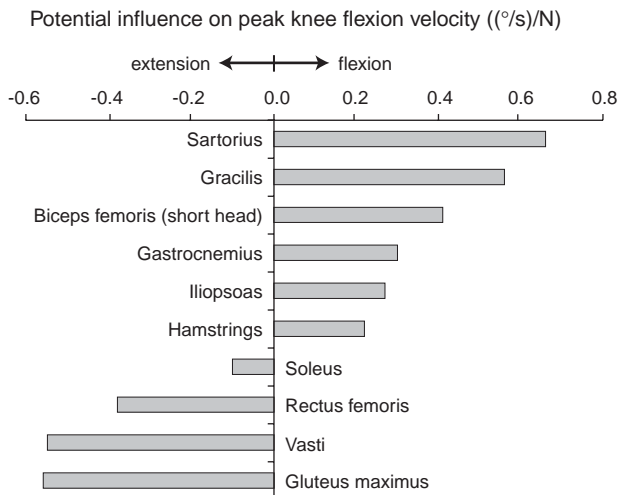Potential influence on peak knee flexion velocity ((°/s)/N)

Fig. 5. The potential influence of selected muscles on the peak knee flexion velocity during double support. The values characterize the change in peak flexion velocity (ΔV) due to a 1 N change in muscle force (ΔF) throughout the period of double support and were calculated as shown in Fig. 4. Muscles not shown had potential influences of less than 0.25 (°/s)/N.
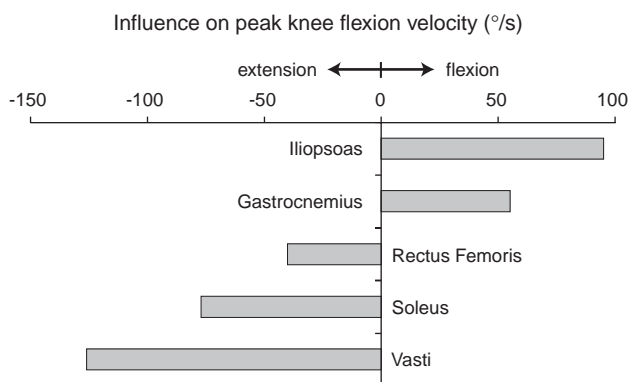
Influence on peak knee flexion velocity (°/s)

Fig. 6. The influence of selected muscles on the peak knee flexion velocity during double support. The influence was calculated as the slope of plots of change in peak knee flexion velocity vs. perturbation size as a percentage of unperturbed muscle force. All other muscles had influences of less than 26°/s.

(Fig. 6). This was because both muscles had a large potential to increase knee flexion velocity (Fig. 5) and exerted relatively large forces during double support in the simulation. Forces in vasti, rectus femoris, and soleus decreased the knee flexion velocity during double support. The ranking of the influence of these muscles on peak knee flexion velocity (Fig. 6) can be explained by examining the forces they produced during double support (Anderson and Pandy, 2001b) and their potentials to decrease knee flexion velocity (Fig. 5). The high influence of vasti was due to the combination of passive forces produced during double support and a large potential to decrease knee flexion velocity. During double support, vasti generated an average force of 225 N; this is less than 4% of the muscle's peak isometric

force. Rectus femoris demonstrated a relatively large potential to decrease the knee flexion velocity; however, this potential and the muscle force generated by rectus femoris during double support were both lower than those of vasti. Soleus exerted large forces during double support, but had a small potential to influence knee flexion velocity.

4. Discussion

Our results indicate that during normal gait, iliopsoas and gastrocnemius are the largest contributors to peak knee flexion velocity during double support, while vasti, soleus, and rectus femoris are the muscles that act to decrease this velocity (Fig. 6). Abnormal force production by any one of these muscles would alter the knee flexion velocity at the end of stance, and subsequent peak knee flexion angle in swing. Thus, therapeutic interventions that target these muscles could be effective in altering this velocity in subjects who walk with stiff-knee gait.

Of the clinically relevant muscles analyzed, increased force in the quadriceps (Fig. 5, vasti and rectus femoris) was found to have the largest potential to decrease the peak knee flexion velocity during double support. While rectus femoris over-activity during swing phase is often implicated in stiff-knee gait, our results show that over-activity in the late stance phase could also be an important factor. This result is consistent with the findings of Sung and Bang (2000), who reported that injections of lidocane to the rectus femoris muscle resulted in a significant increase in knee flexion velocity at toe-off in 16 out of 31 stiff-knee patients.

Our results suggest that the rectus femoris transfer procedure often used to treat stiff-knee gait has the potential to increase peak knee flexion velocity during double support. This finding is consistent with Ounpuu et al. (1993) (Fig. 1), who reported increased velocity at toe-off in stiff-knee subjects who have undergone a rectus femoris transfer. In this procedure, the distal insertion of the rectus femoris muscle is transferred from the patella to a site posterior to the knee (Gage et al., 1987). The transfer procedure aims to convert rectus femoris from a hip flexor/knee extensor to a hip flexor/knee flexor, like sartorius. The rationale is that any over-activity of the transferred muscle during swing phase would contribute to knee flexion, rather than oppose it (Gage et al., 1987; Perry, 1987). However, there is evidence that the transferred rectus femoris muscle does not generate a knee flexion moment (Riewald and Delp, 1997; Asakawa et al., 2002), and thus is not converted into a muscle that functions like sartorius when transferred. Instead, the procedure likely decreases or eliminates the knee extension moment generated by rectus femoris while preserving its capacity to generate a

hip flexion moment. If the knee extension moment were eliminated, the potential of rectus femoris to influence knee flexion velocity would be similar to that of iliopsoas.

Iliopsoas and hamstrings each had potential to increase the peak knee flexion velocity in double support in our simulation (Fig. 5). Many patients who walk with stiff-knee gait also walk with a crouch, defined as persistent knee flexion throughout the stance phase (Sutherland and Davids, 1993). Psoas, gracilis, and hamstrings are muscles that are often surgically length-ened to alleviate crouch gait (Gage, 1990). Reducing the force output of these muscles could compromise the knee flexion velocity generated during double support. This finding may explain why many cerebral palsy patients develop stiff-knee gait after being treated for crouch gait (Thometz et al., 1989; Damron et al., 1993), and suggests that when treating crouch gait, the choice of which muscles to lengthen and the extent to which they are lengthened should take into account the contributions these muscles may make to knee flexion velocity prior to swing.

Gastrocnemius and soleus were found to have opposing effects on peak knee flexion velocity during double support (Figs. 5 and 6); increased force in gastrocnemius increased the peak knee flexion velocity, while increased force in soleus decreased this velocity. It is common for patients with cerebral palsy to walk with tight, spastic plantarflexors (Bleck, 1987). Tight plantar-flexors are often treated with a lengthening of the two muscles' common tendon (Olney et al., 1988; Greene, 2000), reducing the force output of both muscles. The opposing effects that these muscles have on knee flexion velocity during late stance provides an additional rationale for surgical interventions that treat these muscles separately (Delp et al., 1995; Saraph et al., 2000).

Our finding that gastrocnemius increases knee flexion velocity during double support is in agreement with Perry's observation that gastrocnemius contributes to knee flexion in the late stance (Perry, 1992), and the finding of Yamaguchi and Zajac (1990) that gastro-cnemius accelerates the knee into flexion during this period. However, it is inconsistent with Fig. 8 of Neptune et al. (2001) that shows that gastrocnemius induces a knee extension acceleration during double support. The calculation of induced accelerations is sensitive to the procedure used to compute the contributions of muscles to the ground reaction force. The perturbation method described here does not require a decomposition of the ground reaction force and thus avoids this complication. There are also differences between the model used in this study and the model used by Neptune et al. (2001), including the moment arm of gastrocnemius about the knee, the model of the foot, and the model of foot–floor contact, all of which potentially affect the motions induced by gastrocnemius.

Perturbing muscle forces in a dynamic simulation is a powerful technique for understanding how muscles influence movement. Other researchers have used similar techniques to investigate the effects of altering indivi-dual muscle forces (Piazza and Delp, 1996; Jonkers et al., 2003) or joint moments (Riley and Kerrigan, 1998) on gait. Two factors should be considered when performing perturbation studies and interpreting the results. First, it is important to limit the size of a perturbation such that the change in the variable of interest depends linearly on the size of the perturbation. This ensures that the results of a perturbation analysis (e.g., Δvelocity/Δforce in the case of potential influence) will be insensitive to the perturbation size, thus enabling muscle function to be characterized consistently. Second, it must be recognized that perturbing the force in one muscle affects the force produced by all other muscles in the system. When perturbations in muscle force are made, the resulting changes in the kinematics alter the lengths and velocities of all the muscles in the system, and thus alter their forces. While these secondary effects may obscure the function of individual muscles, they reflect the complex interactions of the musculoskeletal system.

Since the configuration of the body affects muscle function, conclusions drawn from a simulation in which muscle forces are perturbed are only directly applicable to the kinematics observed during the simulation. Our results, therefore, describe the potential of muscles to influence peak knee flexion velocity for normal gait. While these results suggest muscles that are likely contributors to diminished knee flexion velocity in stiff-knee gait, confirmation of these findings will require an analysis based on stiff-knee kinematics.

The perturbation analyses performed in this study identified muscles that contribute substantially to peak knee flexion velocity in double support during normal gait and the muscles that have the most potential to alter this velocity. These results indicate muscles that likely contribute to the diminished knee flexion at toe-off observed in the gait of stiff-knee subjects, and indicate what interventions could result in increased or decreased knee flexion velocity in late stance. In the future, we will perform this analysis using forward dynamic simula-tions that track the kinematics from stiff-knee subjects. This will enable us to draw firm conclusions about the potential of muscles to improve peak knee flexion during swing for individuals with stiff-knee gait.

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Appendix A

Perturbations were performed for the following muscles in the pre-swing limb and in the contralateral (loading-phase) limb: iliopsoas, adductor longus brevis, adductor magnus, anterior gluteus medius (combined anterior portions of the gluteus medius and gluteus minimus), posterior gluteus medius (combined posterior portions of the gluteus medius and gluteus minimus), gluteus maximus, tensor fasciae latae, sartorius, gracilis, hamstrings (semimembranosus, semitendonosus, and biceps femoris long head), rectus femoris, vasti (combined vastus medialis, lateralis and intermedius), biceps femoris short head, gastrocnemius, soleus, other plantarflexors (combined peroneus brevis, peroneus longus, tibialis posterior, flexor digitorum longus, and flexor hallucis longus), dorsiflexors (combined peroneus tertius, extensor digitorum tibialis anterior, and extensor hallucis longus), piriformis, pectinius, combined flexor digitorum longus/brevis and flexor hallicus longus/brevis, and combined extensor digitorum longus/brevis and extensor hallucis longus/brevis.

The potential influences for the muscles that had the largest potentials to influence knee flexion velocity are provided in the following table.

Muscle	Potential influence ($°/s/N$)	Range of perturbations	Limit on range lower/ upper
Sartorius	0.66	$0 < \Delta N < 55$	F/K
Gracilis	0.56	$0 < \Delta N < 60$	F/K
Gluteus maximus	−0.56	$0 < \Delta N < 20$	F/K
Vasti	−0.55	$−80 < \Delta N < 70$	K/K
Biceps femoris (short head)	0.41	$−15 < \Delta N < 150$	F/K
Rectus femoris	−0.38	$−35 < \Delta N < 120$	F/K
Gastrocnemius	0.30	$0 < \Delta N < 90$	F/K
Iliopsoas	0.27	$−40 < \Delta N < 65$	L/K
Hamstrings	0.22	$0 < \Delta N < 310$	F/K
Adductor magnus	−0.19	$−55 < \Delta N < 125$	F/L
cl. Gracilis	0.16	$−5 < \Delta N < 260$	F/K
cl. Rectus femoris	−0.13	$−10 < \Delta N < 35$	F/L
cl. Gastrocnemius	0.14	$−10 < \Delta N < 140$	F/L
cl. Vasti	−0.14	$−20 < \Delta N < 20$	F/L
Gluteus medius/ minimus, anterior	0.11	$−120 < \Delta N < 240$	F/K
Soleus	−0.10	$−80 < \Delta N < 280$	F/K

All other muscles exhibited a potential influence less than $0.10°/s/N$.
cl. indicates a muscle from the contralateral limb, which is the limb in loading phase.
F = perturbation was limited to prevent muscle from exerting negative force; L = perturbation was limited to maintain a linear fit to data of $R^2 > 0.99$;
K = perturbation was limited to keep kinematics within 1 SD of the unperturbed kinematics.

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