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Do the hamstrings operate at increased muscle–tendon lengths and velocities after surgical lengthening?

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Abstract

Children with crouch gait frequently walk with improved knee extension during the terminal swing and stance phases following hamstrings lengthening surgery; however, the mechanisms responsible for these improvements are unclear. This study tested the hypothesis that surgical lengthening enables the hamstrings of persons with cerebral palsy to operate at longer muscle–tendon lengths or lengthen at faster muscle–tendon velocities during walking. Sixty-nine subjects who had improved knee extension after surgery were retrospectively examined. The muscle–tendon lengths and velocities of the subjects' semimembranosus muscles were estimated by combining kinematic data from gait analysis with a three-dimensional computer model of the lower extremity. Log-linear analyses confirmed that the subjects who walked with abnormally short muscle–tendon lengths and/or slow muscle–tendon velocities preoperatively tended to walk with longer lengths (21 of 29 subjects, $p < 0.01$) or faster velocities (30 of 40 subjects, $p < 0.01$) postoperatively. In these cases, surgical lengthening may have slackened the subjects' tight hamstrings and/or diminished the hamstrings' spastic response to stretch. Other subjects walked with muscle–tendon lengths and velocities that were neither shorter nor slower than normal preoperatively (22 of 69 subjects), and the semimembranosus muscles of most of these subjects did not operate at increased lengths or velocities after surgery; in these cases, the subjects' postsurgical improvements in knee extension may have been unrelated to the hamstrings surgery. Analyses of muscle–tendon lengths and velocities may help to distinguish individuals who have “short” or “spastic” hamstrings from those who do not, and thus may augment conventional methods used to describe patients' neuromusculoskeletal impairments and gait abnormalities.

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1. Introduction

Children with cerebral palsy frequently walk with excessive knee flexion during the terminal swing and stance phases of the gait cycle. Abnormally “tight” hamstrings, due to spasticity (e.g., Baumann et al., 1980;

Crenna, 1998; Tuzson et al., 2003) or static contracture (Baumann et al., 1980; Sutherland and Davids, 1993), are thought to cause the excessive knee flexion in many cases. Thus, individuals who exhibit a crouched gait often undergo surgical lengthening of the hamstrings, usually in combination with other orthopaedic procedures, in an effort to diminish the excessive flexion of their knees and improve their gait mechanics.

Although many individuals walk with improved knee extension following hamstrings surgery (e.g., DeLuca

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et al., 1998; Abel et al., 1999), the source of the excessive knee flexion and the mechanisms responsible for patients' postoperative improvements are not well understood. It is often inferred, based on gait analysis and physical examination, that the limited range of knee extension exhibited by patients during walking is caused by an exaggerated, velocity-dependent resistance of the hamstrings to stretch (i.e., spasticity) or by excessive passive forces, generated by the stretched sarcomeres of hamstrings comprised of abnormally short muscle fiber lengths (i.e., contracture). When spasticity of the hamstrings contributes to an individual's crouched gait, surgical lengthening of the hamstrings may improve knee extension by attenuating the muscles' exaggerated response to stretch, thereby enabling "spastic" hamstrings to lengthen at greater muscle–tendon velocities during walking. When contracture of the hamstrings contributes to an individual's crouched gait, surgical lengthening of the hamstrings may improve knee extension by slackening the stretched sarcomeres and decreasing the passive tension in the muscles (Delp and Zajac, 1992), thereby enabling "short" hamstrings to operate at longer muscle–tendon lengths during walking. However, it is unknown whether surgical lengthening indeed allows patients' hamstrings to operate at greater muscle–tendon velocities or longer muscle–tendon lengths. Our study examines this issue.

Previous analyses of the muscle–tendon lengths have shown that some patients with a crouched gait walk with hamstrings that are shorter than normal (Hoffinger et al., 1993; Delp et al., 1996; Schutte et al., 1997; Thompson et al., 2002). We hypothesized that individuals who walk with abnormally "short" muscle–tendon lengths preoperatively are likely to walk with longer lengths following hamstrings surgery (perhaps indicating that the surgery slackened the tight hamstrings). Other studies have reported that some patients with a crouched gait walk with hamstrings that are activated at lengthening velocities slower than normal, particularly during terminal swing when the hamstrings are rapidly stretched (Crenna, 1998). We hypothesized that individuals who walk with abnormally "slow" muscle–tendon velocities preoperatively are likely to walk with faster velocities postoperatively (perhaps indicating that the surgery attenuated the hamstrings' spastic response to stretch).

To test these hypotheses, we developed a computer model of the lower extremity that accurately characterizes the muscle–tendon lengths and moment arms of the medial hamstrings muscles (Arnold et al., 2001). We used this model to retrospectively estimate the muscle–tendon lengths and velocities for 69 subjects treated for a crouched gait, at the body positions corresponding to each subject's measured gait kinematics, before and after hamstrings surgery. We purposely selected subjects who walked with improved knee extension postopera-

tively, with the goal of identifying the mechanisms most likely responsible for these improvements.

2. Methods

The 69 subjects who were analyzed were selected from the motion analysis laboratory databases at two children's medical centers. All had spastic cerebral palsy, were able to walk without orthoses or other assistance, were 6 years of age or older (mean 10 yrs, range 6–22 yrs), and walked with at least 20° of knee flexion in one or both limbs at initial contact (averaged over 0–4% of the gait cycle) or terminal swing (averaged over 96–100% of the gait cycle) preoperatively. We included subjects with varying degrees of stance-phase knee extension (i.e., with a jump-knee gait or a crouch gait (Sutherland and Davids, 1993)) and varying levels of involvement. All of the subjects had undergone orthopaedic surgery, including surgical lengthening of the hamstrings, and physical therapy between their pre- and postoperative exams, and all walked with improved knee extension after surgery (Table 1). To be considered "improved," a subject's knee flexion at initial contact or terminal swing had to be diminished by at least 10°, or be within two standard deviations of the knee angles

Table 1
Selected characteristics of the subjects before and after hamstrings lengthening surgery^a

	Normal ^b	Pre ^c	Post ^c
Mean walking speed (m/s)	1.2±0.1	1.0±0.2 ^f	1.0±0.2
Knee flexion at initial contact ^d (deg)	7±6	32±10 ^f	17±9 ^g
Maximum knee extension in stance ^d (deg)	3±5	18±15 ^f	7±11 ^g
Popliteal angle ^e (deg)	—	50±12	37±13 ^g

^aA subject was classified as having undergone a hamstrings lengthening if the surgery included one or more of the following procedures: intramuscular tenotomy or z-lengthening of the semitendinosus (56 subjects and 5 subjects, respectively), transfer of the semitendinosus to the adductor magnus or the lateral femoral condyle (3 subjects and 5 subjects, respectively), lengthening of the semimembranosus (66 subjects), or lengthening of the biceps femoris long head (11 subjects).

^bMean±1 SD for the 45 unimpaired subjects during normal gait.

^cMean±1 SD for the 69 subjects with cerebral palsy, before and after hamstrings lengthening surgery.

^dPositive values indicate knee flexion, negative values indicate knee hyperextension.

^ePopliteal angle quantifies the degree to which the knee can be passively extended with the pelvis stabilized, the ipsilateral hip flexed 90°, and the contralateral limb flexed; a value of 0° indicates full knee extension.

^fDiffers significantly from the normal values for $p < 0.05$, based on t -tests for differences in the mean values.

^gDiffers significantly from preoperative values for $p < 0.05$, based on paired t -tests for differences in the mean values.

averaged for 45 unimpaired subjects during normal gait. We used a 10° threshold because this value corresponds to approximately two standard deviations of the knee angles averaged for the unimpaired subjects, it represents a clinically significant change, and is unlikely to have been caused by skin motion artifacts or other measurement inaccuracies.

One limb per subject, the limb with the greater degree of preoperative stance-phase knee flexion that met all other inclusion criteria, was selected for analysis. A limb was excluded from the study if its musculo-skeletal geometry about the hip or knee had been surgically altered prior to the preoperative exam, or if the hamstrings, psoas, or adductors had been injected with botulinum toxin within 6 months prior to either exam. Subjects who had previously undergone a tendo-Achilles lengthening (16 of the 69 subjects) were included in the study if the surgery had been performed at least 24 months prior to the preoperative exam. None of the subjects had undergone a rhizotomy or a neurectomy, and none were taking baclofen (oral or pump).

Each subject underwent three-dimensional gait analysis (Davis et al., 1991) and clinical assessment. The subjects' joint angles during walking were computed as described by Kadaba et al. (1990), and one representative barefoot walking trial from each subject's pre- and postoperative exam was selected for further study. All subjects and/or their parents provided informed written consent for the collection of these data. Retrospective analyses of the data were performed in accordance with the regulations of all participating institutions.

A model of the lower extremity (Arnold et al., 2001) was used in combination with subjects' joint angles and stride durations obtained from gait analysis to estimate the muscle–tendon lengths and velocities of the hamstrings at the body positions corresponding to each subject's pre- and postoperative gait (Fig. 1). The model characterizes the three-dimensional geometry of the pelvis, femur and proximal tibia, the kinematics of the hip and tibiofemoral joints, and the paths of the semimembranosus and semitendinosus muscles. The line of action of each muscle was defined by a series of line segments and ellipsoidal wrapping surfaces

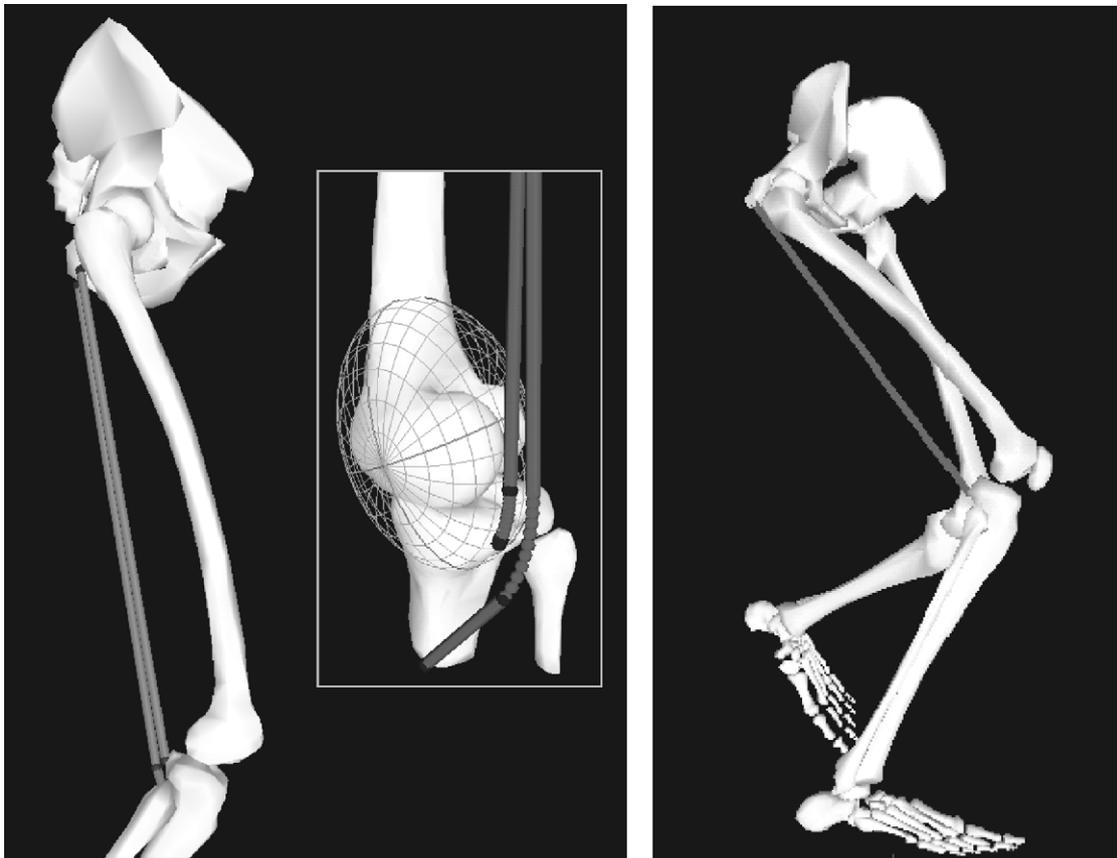


Fig. 1. Computer model of the lower extremity used to estimate the muscle–tendon lengths and velocities of the medial hamstrings at the body positions corresponding to subjects' pre- and postoperative gait kinematics. Ellipsoidal wrapping surfaces (see inset) were used to constrain the paths of the semimembranosus and semitendinosus to slide over the gastrocnemius and posterior femoral condyles with knee extension. The length changes of the muscles computed with this model compare favorably to the length changes determined experimentally on cadaveric specimens over a range of hip and knee angles (Arnold et al., 2001).

(van der Helm et al., 1992) and via points were introduced to simulate the underlying femoral condyles and other anatomical constraints. The model has been tested extensively in previous studies (Arnold et al., 2000, 2001).

The muscle–tendon lengths and velocities of the semimembranosus were determined for each subject at every 2% of the gait cycle (e.g., Figs. 2 and 3). Because the length changes of the semimembranosus and semitendinosus are similar during walking (Schutte et al., 1997; Arnold et al., 2001), we considered the semimembranosus to be representative of the medial hamstrings for this analysis. Muscle–tendon length was calculated as the distance along the modeled path of the semimembranosus between the muscle’s origin and insertion. Muscle–tendon velocity was estimated by computing the numerical derivative of the muscle–tendon length data with respect to time and applying a zero-phase digital filter with a cutoff frequency of 8 Hz (2nd-order Butterworth filter, MATLAB, The MathWorks, Natick, MA). We identified the maximum, or “peak” lengths and velocities of the subjects’ semimem-

branosus during walking, since these measures correspond to times in the gait cycle when short or spastic hamstrings are most likely to restrict knee extension.

We normalized the muscle–tendon lengths and velocities based on the averaged peak length and averaged peak velocity, respectively, of the semimembranosus during normal gait. The means and standard deviations of the peak muscle–tendon lengths and velocities during normal gait were determined from the gait kinematics of the 45 unimpaired subjects. The model was not scaled to the subjects prior to normalization; this decision was based on a previous study (Arnold et al., 2001), which showed that scaling the model along its anatomical axes did not improve the accuracy of the normalized lengths estimated with the model. Hence, differences in the subjects’ muscle–tendon lengths and velocities before and after surgery reflect changes in their measured gait kinematics, not changes in size.

We evaluated our hypotheses—that changes in the muscle–tendon lengths and velocities of the subjects’ hamstrings after surgery would be related to the lengths

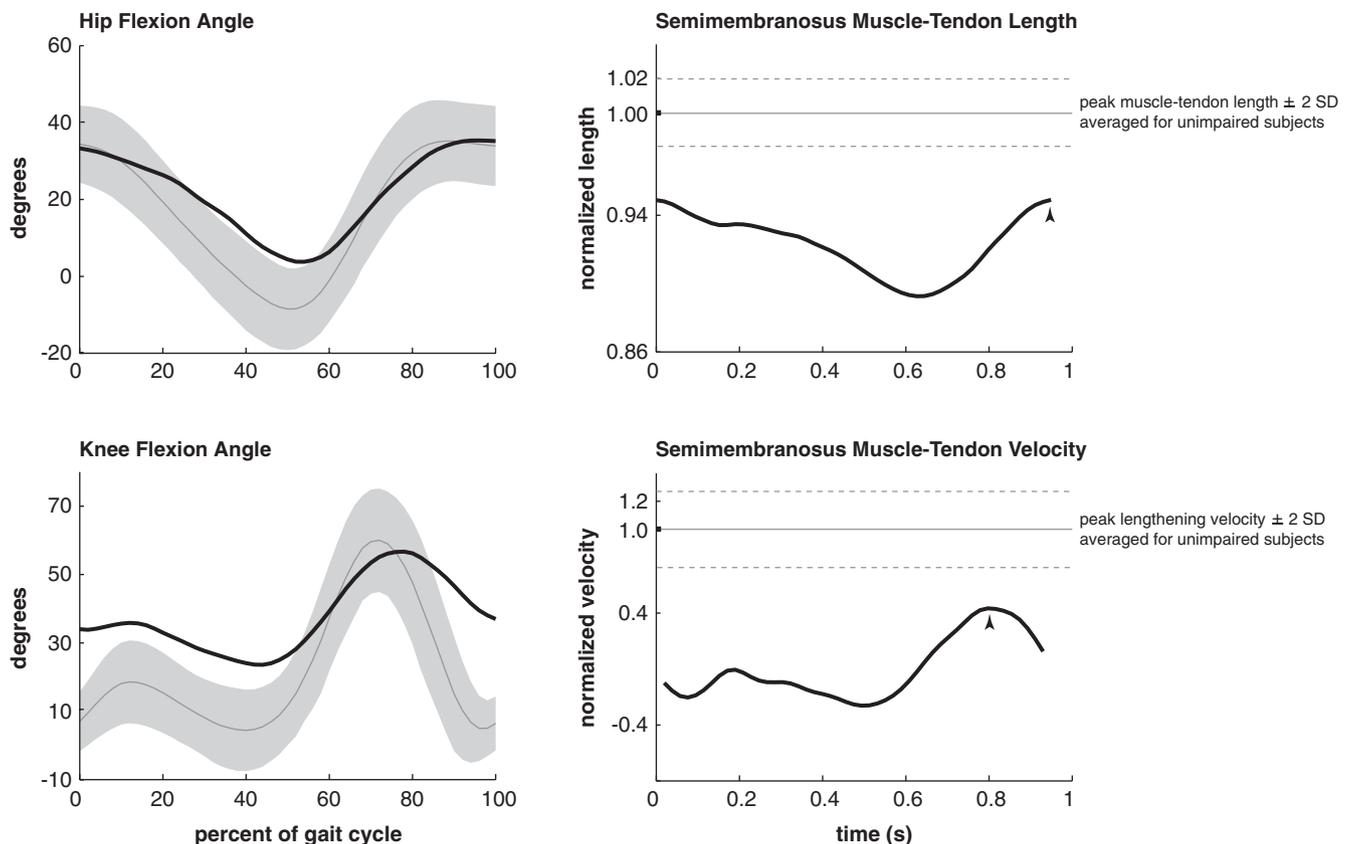


Fig. 2. Hip and knee flexion angles vs. gait cycle (solid curves, left) and semimembranosus muscle–tendon lengths and velocities vs. time (solid curves, right) for a representative subject with cerebral palsy whose hamstrings operated at peak lengths and velocities (see arrows) that were substantially “shorter” and “slower” than normal. The hip and knee flexion angles averaged for unimpaired subjects during normal gait ($mean \pm 2$ SD, shaded regions, left) and the peak muscle–tendon length and velocity averaged for unimpaired subjects during normal gait ($mean \pm 2$ SD, thin and dotted lines, right) are shown for comparison. A normalized muscle–tendon length (velocity) of 1 corresponds to the averaged peak length (averaged peak lengthening velocity) of the semimembranosus during normal gait.

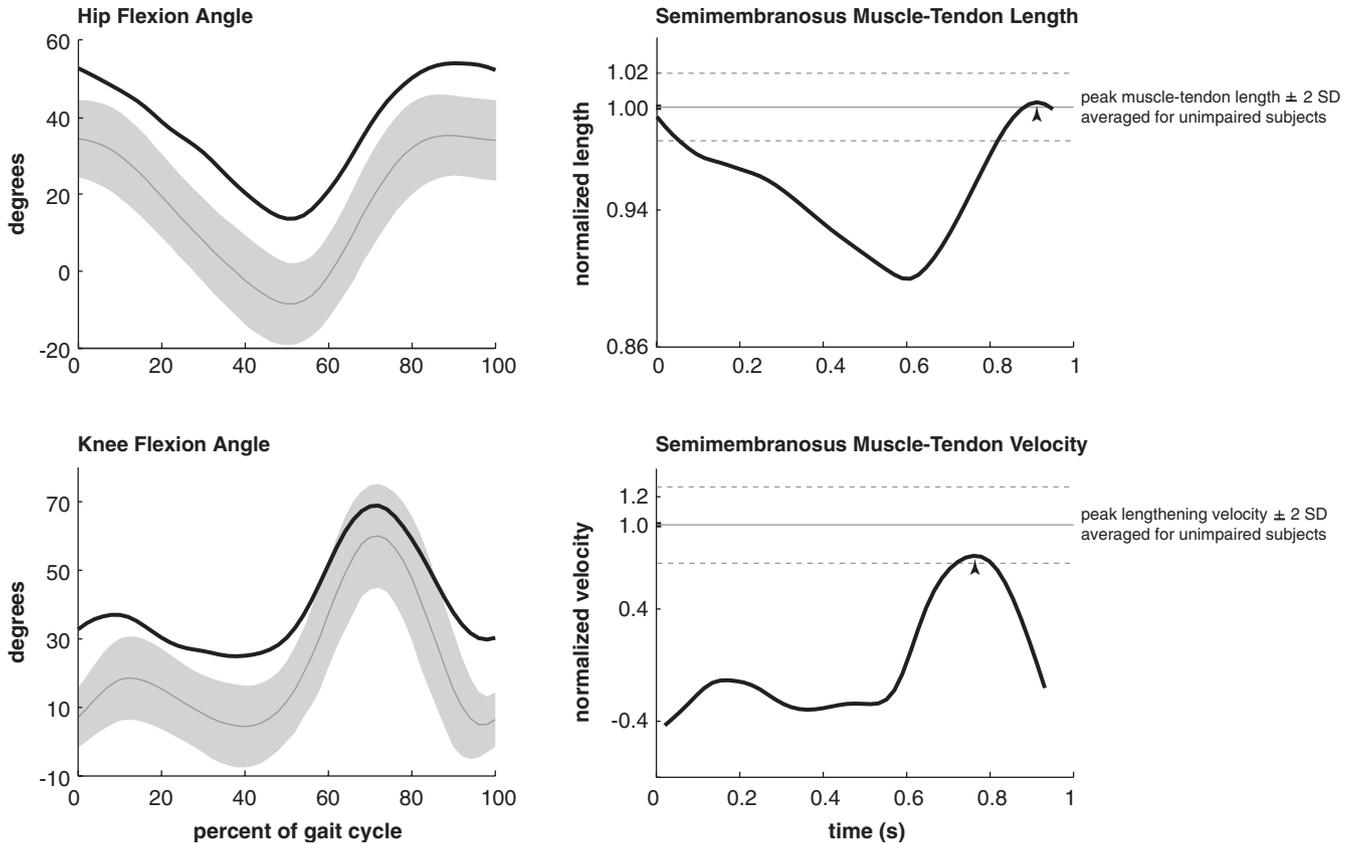


Fig. 3. Hip and knee flexion angles vs. gait cycle (solid curves, left) and semimembranosus muscle–tendon lengths and velocities vs. time (solid curves, right) for a representative subject with cerebral palsy whose hamstrings operated at peak lengths and velocities (see arrows) that were neither “shorter” nor “slower” than normal. The hip and knee flexion angles averaged for unimpaired subjects during normal gait (mean ± 2 SD, shaded regions, left) and the peak muscle–tendon length and velocity averaged for unimpaired subjects during normal gait (mean ± 2 SD, thin and dotted lines, right) are shown for comparison. A normalized muscle–tendon length (velocity) of 1 corresponds to the averaged peak length (averaged peak lengthening velocity) of the semimembranosus during normal gait.

and velocities of their hamstrings before surgery—by performing two log-linear tests. For each test, we cross-classified the 69 subjects in a multi-way frequency table based on two design variables (preoperative length and preoperative velocity) and one response variable (either postoperative change in length or postoperative change in velocity). The first design variable indicated whether the subjects walked with hamstrings that were abnormally short preoperatively, or whether their muscle–tendon lengths were within the normal range. The second design variable indicated whether the subjects walked with hamstrings that were abnormally slow preoperatively, or whether their muscle–tendon velocities were within the normal range. A subject’s hamstrings were classified as “short” preoperatively if the peak length of the semimembranosus during walking was less than the peak length during normal gait by two standard deviations or more; otherwise, a subject’s hamstrings were classified as “not short.” A subject’s hamstrings were classified as “slow” preoperatively if the peak velocity of the semimembranosus during walking was less than the peak velocity during normal

gait by two standard deviations or more; otherwise, a subject’s hamstrings were classified as “not slow.” The response variable indicated whether the hamstrings operated at longer muscle–tendon lengths (or faster muscle–tendon velocities) following hamstrings surgery. A subject’s hamstrings were classified as “longer” if the maximum postoperative length of the semimembranosus during walking was greater than the preoperative length by an amount corresponding to a 10° change in knee angle at initial contact, as averaged for the unimpaired subjects; otherwise, the subject’s hamstrings were considered to be “not longer.” A subject’s hamstrings were classified as “faster” if the maximum postoperative velocity of the semimembranosus during walking was greater than the preoperative velocity by 2.7 cm/s; otherwise, the subject’s hamstrings were considered to be “not faster.” This velocity change corresponds to a 10° increase in knee extension computed over 250 ms, the approximate time interval of terminal swing.

Likelihood ratio χ^2 statistics were evaluated to determine whether the subjects’ pre- and postoperative

muscle–tendon lengths and/or velocities were related. A test statistic was considered to be significant for p -values less than 0.05. We evaluated our hypotheses using log-linear tests, rather than simpler χ^2 tests, because the preoperative peak lengths and velocities of the subjects' hamstrings were not independent. The log-linear approach enabled us to account for these interactions when testing for associations between the lengths and velocities of the subjects' hamstrings during walking and the changes detected after surgery.

3. Results

Consistent with our hypotheses, the subjects in our study who walked with abnormally short semimembranosus muscle–tendon lengths preoperatively tended to walk with longer peak lengths following hamstrings lengthening surgery (21 of 29 subjects, $p < 0.01$, Table 2), suggesting that surgical lengthening may have slackened these subjects' tight hamstrings. The subjects who walked with abnormally slow muscle–tendon velocities preoperatively tended to walk with faster peak semimembranosus velocities postoperatively (30 of 40 subjects, $p < 0.01$, Table 3), suggesting that, in most of these cases, surgical lengthening may have diminished the spastic response of the subjects' hamstrings to stretch. Approximately one-third of the subjects who were

Table 2

Numbers of subjects who walked with longer^a semimembranosus muscle–tendon lengths after surgical lengthening, cross-classified by the subjects' preoperative peak semimembranosus lengths^b and velocities^c during walking

Preoperative length	Preoperative velocity	Change in muscle–tendon length	
		Longer	Not longer
Short	Slow	15	7
	Not slow	6	1
Not short	Slow	7	11
	Not slow	8	14

Likelihood ratio χ^2 (3 degrees of freedom) for 2-way interactions = 16.15, $p < 0.01$. Significant 2-way interactions include [preoperative length * preoperative velocity] and [preoperative length * change in length].

^aA subject's hamstrings were classified as operating at *longer* muscle–tendon lengths after surgical lengthening if the peak postoperative length of the semimembranosus was greater than the preoperative length by an amount corresponding to a 10° change in knee angle at initial contact, as averaged for 45 unimpaired subjects during normal gait.

^b*Short* defined as peak muscle–tendon length shorter than 2 SD from the peak length averaged for 45 unimpaired subjects during normal gait.

^c*Slow* defined as peak muscle–tendon velocity slower than 2 SD from the peak velocity averaged for 45 unimpaired subjects during walking.

Table 3

Numbers of subjects who walked with faster^a semimembranosus muscle–tendon velocities after surgical lengthening, cross-classified^b by the subjects' preoperative peak semimembranosus lengths and velocities during walking

Preoperative length	Preoperative velocity	Change in muscle–tendon velocity	
		Faster	Not faster
Short	Slow	16	6
	Not slow	2	5
Not short	Slow	14	4
	Not slow	8	14

Likelihood ratio χ^2 (3 degrees of freedom) for 2-way interactions = 18.61, $p < 0.01$. Significant 2-way interactions include [preoperative length * preoperative velocity] and [preoperative velocity * change in velocity].

^aA subject's hamstrings were classified as lengthening at *faster* muscle–tendon velocities after surgical lengthening if the peak postoperative velocity of the semimembranosus was greater than the preoperative velocity by an amount corresponding to a 10° change in knee extension over the terminal swing phase, assuming a 0.25 s interval between peak knee flexion and extension.

^b*Short* and *slow* classifications are defined in Table 2.

examined walked with semimembranosus muscle–tendon lengths and muscle–tendon velocities that were neither abnormally short nor slow preoperatively (22 of 69 subjects), and the hamstrings of most of these subjects did *not* operate at longer peak lengths or faster peak velocities postoperatively. In these subjects, spasticity or contracture of the hamstrings may not have been the source of the excessive knee flexion, and the improvements in knee extension exhibited by the subjects after treatment may not have been related to the hamstrings lengthening surgery.

4. Discussion

Excessive knee flexion during the terminal swing and stance phases is often attributed to “short” or “spastic” hamstrings that restrict knee extension; thus, children with a crouched gait frequently undergo hamstrings lengthening surgery. Many individuals achieve dramatic improvements in knee extension, stride length, and walking efficiency following surgery (e.g., DeLuca et al., 1998), yet others show little improvement or worsen. We believe that treatments for the excessive knee flexion could be designed more effectively if the mechanisms responsible for patients' postoperative improvements were better understood. This study tested the hypothesis that surgical lengthening enables the hamstrings of persons with cerebral palsy to operate at longer muscle–tendon lengths or lengthen at faster muscle–tendon velocities during walking.

Our analysis of the subjects' muscle–tendon lengths is consistent with studies by other investigators who have postulated that excessive passive forces, generated by abnormally short hamstrings, contribute to the excessive knee flexion in some cases. Buczek et al. (2002), for example, derived subject-specific regression equations to predict the onset of passive tension in the hamstrings as a function of the joint angles and showed, for six subjects with crouch gait, that the subjects' predicted onset of passive tension during walking occurred concurrently with the development of abnormal knee flexion moments and diminished knee extension angles in swing. Abel et al. (1999) demonstrated that patients' passive ranges of knee motion are frequently increased following hamstrings lengthening surgery. Muscle biopsy studies confirming the presence of abnormally short muscle fibers in the hamstrings of persons with crouch gait, to our knowledge, have not been reported; however, Tardieu et al. (1982) have documented the presence of abnormally short fibers in the gastrocnemius muscles of some children with cerebral palsy, and Friden and Lieber (2003) have determined that the upper extremity muscles of some patients develop excessive passive forces at abnormally short sarcomere lengths. This evidence from previous studies suggests that contracture of the hamstrings is a plausible contributor to excessive knee flexion. Approximately one-third of the subjects in our study had semimembranosus lengths during walking that were substantially shorter than normal preoperatively, and most of these subjects walked with longer muscle–tendon lengths postoperatively (Table 2). These data corroborate the results of previous studies, and suggest that surgical lengthening of the hamstrings may improve knee extension by allowing “short” hamstrings to operate at increased muscle–tendon lengths.

Our analysis of the subjects' muscle–tendon velocities is also consistent with previous studies which have suggested that spasticity of the hamstrings limits knee extension in some cases. Perry and Newsam (1992), for instance, have shown that many persons with a crouched gait exhibit premature firing of the hamstrings during the swing phase, when the hamstrings are maximally stretched. Granata et al. (2000) measured subjects' joint angular velocities during normal and crouch gait and reported that the subjects with crouch gait, on average, walked with knee extension velocities that were substantially slower than normal (40 subjects, $p < 0.05$). Tuzson et al. (2003) determined the knee angular velocity that evoked an electromyographic response in subjects' hamstrings during dynamometer-controlled stretches and showed that these “spastic threshold velocities” were highly correlated with the subjects' maximum knee extension velocities during walking for about half of the subjects with cerebral palsy who were tested (7 of 18 subjects, $p < 0.001$).

Crenna (1998) used a musculoskeletal model to estimate the muscle–tendon velocities of subjects' hamstrings during crouch gait and noted that the subjects' hamstrings were activated at abnormally low lengthening velocities. Together, the data from these previous studies suggest that abnormal excitation of the hamstrings, triggered by spasticity, may limit the velocity at which the hamstrings can elongate during walking, contributing to excessive knee flexion in some cases. Evidence from our study corroborates these findings, and suggests that surgical lengthening of the hamstrings may improve knee extension by allowing “spastic” hamstrings to elongate at greater muscle–tendon velocities. Indeed, more than half of the subjects in our study had peak muscle–tendon velocities during walking that were substantially slower than normal preoperatively, and most of these subjects walked with hamstrings that lengthened at increased velocities postoperatively (Table 3).

Not all of the subjects in our study walked with longer peak semimembranosus lengths or faster peak semimembranosus velocities postoperatively, even though all of the subjects exhibited improved knee extension. In particular, about one third of the subjects (22 of 69 subjects) had muscle–tendon lengths and velocities that were neither abnormally short nor slow preoperatively, and the hamstrings of most of these subjects did *not* operate at increased lengths or velocities after surgery. In these individuals, impairments other than spasticity or contracture of the hamstrings may have contributed to the excessive knee flexion, and treatments other than surgical lengthening of the hamstrings may have contributed to the improved knee extension. Most of these subjects (18 of the 22 subjects) underwent a gastrocnemius recession or tendo-Achilles lengthening in conjunction with the hamstrings lengthening. The degree to which these procedures influenced the subjects' knee angles during walking is unclear; however, Baddar et al. (2002) have demonstrated that knee extension at initial contact can be significantly improved following gastrocnemius-soleus recession, *without* hamstrings lengthening. Other factors have been postulated to contribute to crouch gait, including tightness of the hip flexors (Bleck, 1987), weakness of the hip extensors (Beals, 2001), and malrotation of the foot (Schwartz and Lakin, 2003), and procedures related to these factors were performed on the subjects in this study, including derotational osteotomy of the femur (7 of 22 subjects), derotational osteotomy of the tibia (4 of 22 subjects), and lengthening of the psoas above the pelvic brim (4 of 22 subjects). To rigorously explain the mechanisms responsible for these subjects' improved knee extension angles during walking, more efficacious methods for identifying which factors contribute to an individual's abnormal gait are needed.

Some subjects in our study who walked with abnormally short semimembranosus lengths preoperatively did not walk with longer lengths postoperatively (8 of 29 subjects), and some subjects who walked with abnormally slow semimembranosus velocities preoperatively did not walk with faster lengthening velocities postoperatively (10 of 40 subjects), contrary to our hypotheses. One possible explanation for these findings is that we did not account for variations in walking speed when comparing the subjects' peak muscle–tendon lengths and velocities during walking to the peak lengths and velocities during normal gait. Six of these 18 subjects walked at speeds that were less than 80% of normal preoperatively. In four of these cases, the subjects may have been misclassified as having hamstrings that were abnormally “short” and/or “slow,” when in fact the hamstrings were operating at muscle–tendon lengths and velocities within the normal, speed-matched range. Four of the 18 subjects walked at speeds that were substantially slower postoperatively than preoperatively; in these cases, the subjects may have been classified as having hamstrings that were “not faster” postoperatively due to the decrease in walking speed. Another possible explanation for these findings is that our gait data were collected over a 14-year period, at two different institutions, and our analyses did not account for potential variations in treatment philosophy, operative technique, or postoperative physical therapy.

We determined the muscle–tendon lengths and velocities of subjects' semimembranosus muscles during walking; we did not estimate the subjects' passive or active muscle–tendon forces. Analysis of the muscle–tendon lengths can indicate whether a subject's hamstrings are operating at lengths shorter or longer than normal, but such analyses cannot explain why a subject's hamstrings are operating at those lengths. Analysis of the muscle–tendon velocities, similarly, can indicate whether a subjects' hamstrings are lengthening at velocities slower or faster than normal, but such analyses cannot explain why a subject's hamstrings are lengthening at those rates. Hence, our discovery that the subjects who walked with short semimembranosus muscle–tendon lengths preoperatively tended to walk with longer lengths and improved knee extension postoperatively, and our finding that the subjects who walked with slow semimembranosus muscle–tendon velocities preoperatively tended to walk with faster velocities and improved knee extension postoperatively, is suggestive—but not necessarily indicative—of a causal link. While it is plausible that surgical lengthening of the hamstrings contributed to the subjects' improved knee extension by slackening short hamstrings and/or attenuating the hamstrings' spastic response to stretch, our study does not prove that these explanations are correct.

This study revealed statistically significant relationships between subjects' pre- and postoperative semimembranosus muscle–tendon lengths and muscle–tendon velocities during walking. These findings may have important implications for treatment. Analyses of the three-dimensional musculoskeletal geometry may help to distinguish individuals who have functionally short or spastic hamstrings from those who do not, and thus may augment conventional methods used to describe patients' gait deviations and neuromusculoskeletal impairments.

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