

The effect of walking speed on hamstrings length and lengthening velocity in children with spastic cerebral palsy

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ABSTRACT

Children with cerebral palsy often walk with reduced knee extension in terminal swing, which can be associated with short length or slow lengthening velocity of hamstrings muscles during gait. This study investigated the role of two factors that may contribute to such short and slow hamstrings: walking speed and spasticity. 17 children with spastic cerebral palsy and 11 matched typically developing children walked at comfortable, slow, and fast walking speed. Semitendinosus muscle-tendon length and velocity during gait were calculated using musculoskeletal modeling. Spasticity of the hamstrings was tested in physical examination. Peak hamstrings length increased only slightly with walking speed, while peak hamstrings lengthening velocity increased strongly. After controlling for these effects of walking speed, spastic hamstrings acted at considerably shorter length and slower velocity during gait than normal, while non-spastic hamstrings did not (all $P < 0.001$). These data are important as a reference for valid interpretation of hamstrings length and velocity data in gait analyses at different walking speeds. The results indicate that the presence of spasticity is associated with reduced hamstrings length and lengthening velocity during gait, even at constant walking speed.

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1. Introduction

Children with cerebral palsy (CP) often walk with reduced knee extension in terminal swing. This reduction in knee extension leads to a decrease in step length and reduced walking speed, which may limit functional performance. Reduced knee extension in terminal swing is often associated with reduced hamstrings length and lengthening velocity during gait [1–4]. Arnold et al. [3] showed that about 35% of subjects walking with reduced knee extension had short and in most cases also slow hamstrings during gait; 30% had slow but not short hamstrings and another 35% had neither short nor slow hamstrings. They also showed that surgically lengthening of muscles that were short or slow, resulted in improved length and velocity during gait and in increased knee extension in terminal swing [4]. This suggests that hamstrings length and velocity during gait are important measures to consider in treatment planning. Moreover, since many of the underlying impairments as well as treatment options lay at muscle rather than joint level, considering muscle-tendon behavior during gait seems

to have added value over conventional gait analysis techniques alone that examine joint kinematics and kinetics.

However, the underlying causes of short or slow hamstrings during gait are not well understood. Several factors may cause hamstrings to be short or slow during gait, two of which are walking speed and spasticity. First, children with CP generally walk slower than typically developing children. Walking speed has been shown to influence hamstrings lengths and velocity in healthy subjects walking in crouch [5]. However, no studies have investigated the effect of walking speed on hamstrings length and velocity in CP patients. Therefore, it is not known to what extent differences in hamstrings length or velocity between patients and control subjects can be attributed to differences in walking speed.

Second, hamstrings spasticity may contribute to short and slow hamstrings during gait. Cheung et al. [6] compared spastic threshold velocity as measured during physical examination with muscle-tendon lengthening velocity during gait in spastic hamstrings and quadriceps muscle, and found a significant correlation. Jonkers et al. [7] found that peak rectus femoris length and lengthening velocity were reduced in spastic muscles in stiff knee gait, with peak lengths during gait decreasing with increasing rectus femoris spasticity scores. These studies suggest that muscle-tendon length and velocity during gait are related to spasticity, but evidence is limited.

Moreover, walking speed and spasticity effects may interact, first because the presence of spasticity may limit walking speed,

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and second because walking speed may influence the effects of spasticity, due to its velocity-dependency [8]. Therefore, studying speed effects in spastic children is particularly relevant.

The aims of this study were:

- (1) to investigate the effect of walking speed on hamstrings length and lengthening velocity during gait in children with CP;
- (2) to investigate to what extent spasticity as measured in physical examination is related to hamstrings length and lengthening velocity during gait; and
- (3) to study the interacting effects of walking speed and spasticity.

2. Methods

2.1. Subjects

17 children with spastic CP and 11 typically developing (TD) children, matched in age, height and weight, participated in this study. Characteristics of the children with CP (mean \pm standard deviation) were: age 8.9 ± 2.1 years (range 6–12); height 136 ± 13 cm; weight 33 ± 10 kg; and of the TD children: age 8.2 ± 1.8 years (range 6–12); height 134 ± 12 cm and weight 32 ± 13 kg. All children with CP were clinically diagnosed with spastic CP (13 bilateral, 4 unilateral), were able to walk independently without walking aids, were classified on the gross motor function classification scale (GMFCS) as level I–II [9], had no prior orthopedic surgery, rhizotomy or baclofen treatment, and had no prior botulinum toxin treatment within the previous 16 weeks. All children and their parents provided informed consent. The study protocol was approved by the Medical Ethics Committee of the VU University Medical Center.

2.2. Design

The children walked along a 10 m walkway, first at self-selected comfortable walking speed (CWS), and subsequently at $70 \pm 5\%$ (SLOW) and $130 \pm 5\%$ (FAST) of CWS, in random order. Walking speed was recorded online and controlled by giving instant feedback to the children. After sufficient practice attempts, three successful trials were collected for each speed condition. The children were measured twice in two separate sessions, in order to obtain a large and reliable data sample per child, and as part of a larger study. This resulted in a total of six trials per condition. The sessions took place at the same time of day, 17.6 ± 11.6 days apart, without any interventions in between the two sessions. For logistic reasons, two children could be measured only once.

3D kinematic data were collected for the trunk, pelvis, thigh, shank and foot, using a motion capture system (Optotrak, Northern Digital, Waterloo, ON). Data on the right leg were collected in the TD group and on both legs in the CP group. A technical cluster of three markers was attached to each segment. While standing in anatomical position, bony landmarks were indicated in order to anatomically calibrate the technical cluster frames [10].

All children with CP underwent a standard physical examination in one of the two sessions, all performed by the same person. Spasticity was measured in the hamstrings with a standardized clinical spasticity test (SPAT) [11], which is based on the Modified Tardieu Scale [12]. In this test muscles were stretched at slow and very fast speed. Based on these measurements, the muscles were grouped according to the level of spasticity: SPAS2 = severe spasticity (presence of a clear catch at fast stretch, SPAT score 2 or 3); SPAS1 = mild spasticity (increase in muscle resistance somewhere in the range of motion at fast stretch, without a catch, SPAT score 1); SPAS0 = no spasticity.

2.3. Analysis

3D kinematic data were analyzed with open-source software (www.Body-Mech.nl; MatLab[®]; The Mathworks). Initial contact (IC) was calculated from the forward foot velocity, and defined as the instants at which this velocity became lower than 20% of its maximal value [5]. One successful stride (IC to IC), for each leg separately, was selected for each trial, and left and right legs were included independently. It is acknowledged that muscle behavior may be influenced by other muscles in the ipsilateral leg, or even by muscles in the contralateral leg. However, since it was not possible to account for any such influence, muscles of all legs were evaluated independently. For one patient, data on only one leg were available for technical reasons, resulting in a total of 33 legs in the CP group and 11 legs (all right legs) in the TD group.

Actual walking speed during the successful stride was calculated as the average forward velocity of the pelvis markers over the full stride, and nondimensionalized by $\sqrt{g \cdot L_{leg}}$ [13], with L_{leg} the leg length, calculated as the summed length from trochanter major to lateral epicondyle to lateral malleolus. Stride length was calculated as the forward progression of the pelvis markers over the stride and nondimensionalized by L_{leg} .

Semitendinosus (ST) was considered representative for the hamstrings, and ST length was calculated with SIMM musculoskeletal modeling software [14,15]. This model has been validated [16,17] and previously used for calculation of

hamstrings length in CP [1–4]. The standard generic model was used and scaled to the individual subject sizes, using 3D kinematic data from the anatomical landmarks. Muscle-tendon length was filtered using an 8 Hz low-pass symmetric filter and differentiated in order to obtain muscle-tendon velocity. Both muscle-tendon lengths and velocities were time-normalized to 100% gait cycle, and nondimensionalized by L_{ref} and $\sqrt{g \cdot L_{leg}}$, respectively, with L_{ref} the anatomical reference length with all joint angles set at zero, calculated with SIMM. Peak muscle-tendon length and lengthening velocity, as reached in (terminal) swing, were calculated for all trials.

2.4. Statistics

A repeated measures analysis of variance (ANOVA), with Bonferroni adjustment for multiple comparisons, was used to investigate whether walking speed and stride lengths differed between the three speed conditions and between subgroups.

A linear generalized estimating equation (GEE) analysis was applied to investigate the relationship of spasticity and walking speed with the outcome measures peak muscle-tendon length and peak muscle-tendon lengthening velocity (SPSS v15.0.0; exchangeable working correlation structure and robust estimation of the covariance matrix). This analysis accurately controls for differences in walking speed, and estimates the individual contributions of the independent variables to the outcome measures. Three independent variables were included in the model: (1) spasticity group as categorical variable (TD, SPAS0, SPAS1 and SPAS2); (2) walking speed as continuous variable; and (3) the interaction of group and walking speed. Walking speed was centered around the mean nondimensional walking speed of 0.40, by subtracting this value from the measured walking speed. Centering allowed for a meaningful interpretation of main effects when interaction was present in the model [18], in which case the main effect could be interpreted as the effect of spasticity group at nondimensional walking speed of 0.40. This resulted in the following model:

$$\text{Outcome} = B_0 + B_1(\text{group}) + B_2 * (\text{walking speed} - 0.40) + B_3(\text{group}) * (\text{walking speed} - 0.40)$$

with B_0 the value of the outcome measure in TD (reference group), at a walking speed of 0.40; B_1 the difference between the CP groups and TD at a speed of 0.40 (main effect of group); B_2 the slope of the outcome measure versus speed curve for TD (main effect of speed); and B_3 the difference in slope between groups (interaction). Post hoc analyses were performed with SPAS0 and SPAS1 as reference groups to determine the significance of all pair-wise comparisons between groups. P -values of less than 0.05 were considered to be statistically significant.

3. Results

Based on the physical examination, 6 of the 33 semitendinosus muscles were assigned to SPAS0, 15 to SPAS1 and 12 to SPAS2. Of the ‘sound’ limbs of the unilaterally involved children, 3 muscles were assigned to SPAS0 and 1 to SPAS1.

Nondimensional walking speed differed significantly between the three conditions ($P < 0.001$, Table 1). The CP group walked slower than the TD group ($P < 0.001$); CWS in the CP group was close to SLOW in the TD group ($P = 0.25$), and FAST in the CP group was close to CWS in the TD group ($P = 0.85$). Walking speed in SPAS2 was significantly slower than in SPAS0 and SPAS1 ($P < 0.05$). Stride length was lower in the CP group than in the TD group, and decreased with increasing levels of spasticity ($P < 0.001$, Table 1).

Peak ST length and lengthening velocity both increased with walking speed (Figs. 1 and 2, Table 2: B_2 , P_2). The effect of walking speed on ST length was small (Fig. 2A). B_2 , which indicates the slope of the peak length versus walking speed curve, was 0.029 ± 0.012 in the reference group TD. This means that peak ST length increased with 2.9% of reference length for each unit of nondimensional walking speed. Or in more meaningful terms: as walking speed almost doubled from slow (0.36) to fast speed (0.66), peak length increased with approximately 1% of reference length, which came down to about 3.5 mm. The effect of walking speed on peak lengthening velocity was more pronounced (Fig. 2B). B_2 was 0.174 ± 0.022 for the TD group. Thus, as walking speed almost doubled from slow to fast speed, peak ST velocity increased with approximately 0.050, or 40%.

Peak ST length and lengthening velocity were lower in more spastic muscles (Figs. 1 and 2, Table 2: B_1 , P_1). Both SPAS1 and SPAS2 had shorter peak length and slower peak velocity than TD.

Table 1
Nondimensional walking speed and stride length (means ± S.D.).

	Condition	TD	CP-ALL	SPAS0	SPAS1	SPAS2
Absolute speed	SLOW	0.36 ± 0.03 ^{T012}	0.26 ± 0.06 ^{T1}	0.30 ± 0.02 ^{T2}	0.28 ± 0.05 ^{T2}	0.23 ± 0.06 ^{T01}
	CWS	0.51 ± 0.04 ⁰¹²	0.39 ± 0.07 ^{T1}	0.42 ± 0.03 ^{T2}	0.41 ± 0.06 ^{T2}	0.35 ± 0.08 ^{T01}
	FAST	0.66 ± 0.05	0.51 ± 0.07 ^{T1}	0.56 ± 0.03 ^{T2}	0.53 ± 0.05 ^{T2}	0.47 ± 0.08 ^{T01}
Relative speed (speed/CWS)	SLOW	0.71 ± 0.03	0.68 ± 0.06	0.73 ± 0.02	0.67 ± 0.07	0.67 ± 0.05
	CWS	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00	1.00 ± 0.00
	FAST	1.29 ± 0.03	1.34 ± 0.13	1.33 ± 0.10	1.30 ± 0.12	1.37 ± 0.14
Stride length	SLOW	1.60 ± 0.13 ^{T12}	1.27 ± 0.28 ^{T1}	1.46 ± 0.12 ^{T2}	1.32 ± 0.28 ^{T2}	1.14 ± 0.31 ^{T01}
	CWS	1.87 ± 0.12 ^{T12}	1.46 ± 0.25 ^{T1}	1.61 ± 0.04 ^{T2}	1.58 ± 0.18 ^{T2}	1.28 ± 0.31 ^{T01}
	FAST	2.11 ± 0.13 ^{T12}	1.68 ± 0.23 ^{T1}	1.85 ± 0.08 ^{T2}	1.76 ± 0.12 ^{T2}	1.39 ± 0.30 ^{T01}

Abbreviations: TD, typically developing; CP-ALL, all cerebral palsy patients grouped together; SPAS0, 1 and 2: increasing levels of spasticity in CP; CWS, comfortable walking speed. ^{T012} indicate significant difference ($P < 0.05$) of subgroup to TD^(T), SPAS0⁽⁰⁾, SPAS1⁽¹⁾ and SPAS2⁽²⁾, respectively. All speed conditions were significantly different from each other ($P < 0.001$).

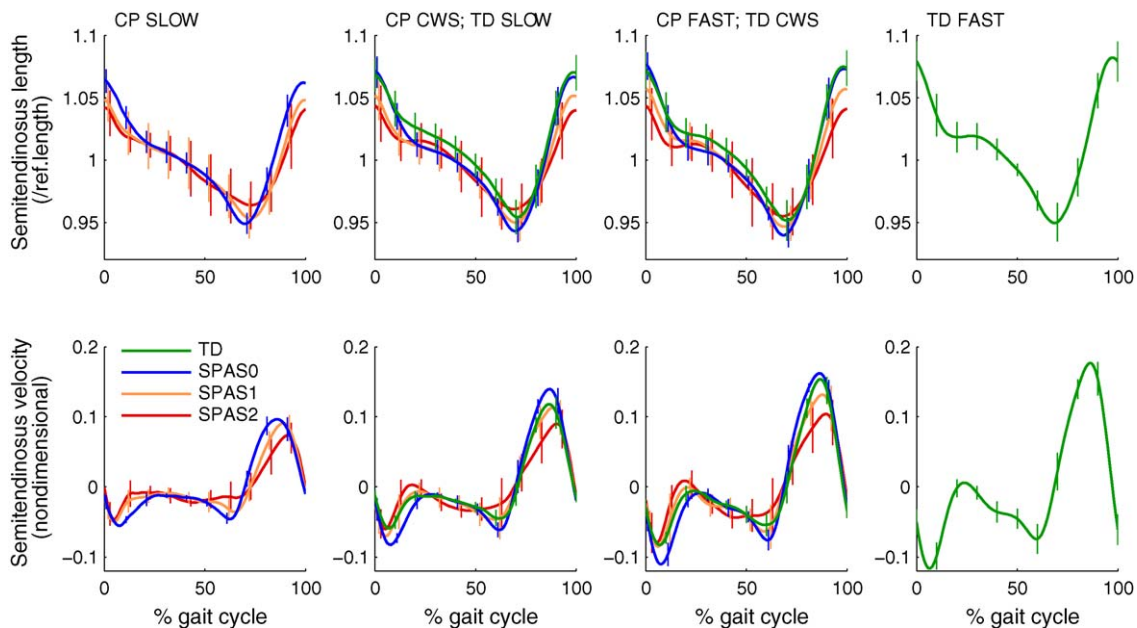


Fig. 1. Semitendinosus muscle-tendon length (top row) and velocity (bottom row) versus gait cycle, for the typically developing group (TD; $n = 11$) and the cerebral palsy (CP) groups: SPAS0 ($n = 6$), SPAS1 ($n = 15$) and SPAS2 ($n = 12$), indicating increasing levels of spasticity. CWS: comfortable walking speed. Data at comparable walking speeds (CP CWS and TD SLOW; CP FAST and TD CWS; see Table 1) are plotted together for better comparison.

Non-spastic muscles in CP were not shorter or slower than normal. At the average nondimensional walking speed of 0.40, peak length in TD was $7.6 \pm 0.4\%$ longer than reference length (B_0). Peak length in SPAS2 was $2.9 \pm 0.7\%$ of reference length shorter than in TD (B_1). Peak

lengthening velocity in SPAS2 was 0.028 ± 0.008 or about 20% lower than normal.

Peak ST velocity showed a significant overall interaction effect of walking speed and spasticity, and a similar trend was seen for

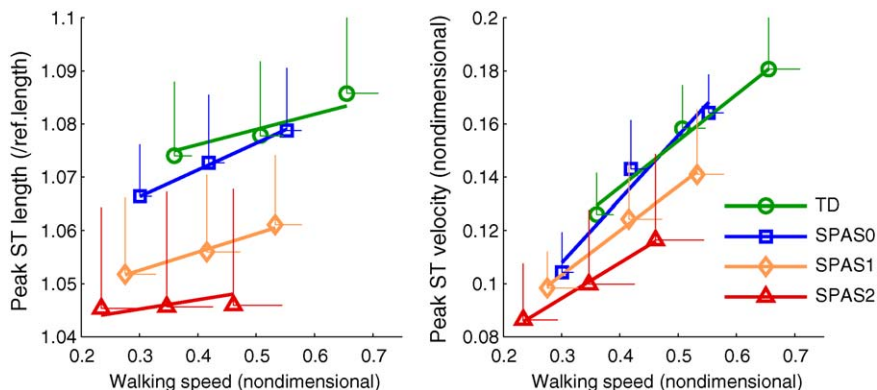


Fig. 2. (A) Peak semitendinosus (ST) muscle-tendon length and (B) lengthening velocity versus walking speed, for the typically developing (TD) group and the cerebral palsy groups: SPAS0 ($n = 6$), SPAS1 ($n = 15$) and SPAS2 ($n = 12$), indicating increasing levels of spasticity. Symbols and thin lines represent means and standard deviations of measured data, bold lines represent modeled data from the generalized estimating equation analysis.

Table 2
GEE results showing the effects of spasticity group, walking speed and their interaction.

Outcome	Group	Intercept at $v = 0.40$	Group effect		Speed effect		Group \times speed effect	
		$B_0 \pm$ S.E.	$B_1 \pm$ S.E.	P_1	$B_2 \pm$ S.E.	P_2	$B_3 \pm$ S.E.	P_3
Peak length	TD	1.076 \pm 0.004	0 ^b *12	0.000	0.029 \pm 0.012	0.000	0	0.060
	SPAS0	id. ^a	-0.005 \pm 0.006 ^{*12}		id.		0.022 \pm 0.015 ^{*2}	
	SPAS1	id.	-0.020 \pm 0.006 ^{*10}		id.		0.006 \pm 0.015	
	SPAS2	id.	-0.029 \pm 0.007 ^{*10}		id.		-0.011 \pm 0.016 ^{*0}	
Peak velocity	TD	0.136 \pm 0.004	0 ^{*12}	0.001	0.174 \pm 0.022	0.000	0 ^{*0}	0.006
	SPAS0	id.	-0.005 \pm 0.007 ^{*2}		id.		0.065 \pm 0.031 ^{*12}	
	SPAS1	id.	-0.016 \pm 0.006 ^{*T}		id.		-0.008 \pm 0.027 ^{*0}	
	SPAS2	id.	-0.028 \pm 0.008 ^{*10}		id.		-0.040 \pm 0.031 ^{*0}	

GEE: Generalized Estimating Equation: Outcome = $B_0 + B_1(\text{group}) + B_2 * (\text{speed} - 0.40) + B_3(\text{group}) * (\text{speed} - 0.40)$.

E.g. peak length in SPAS0 at a speed of 0.30 is equal to: $1.076 + -0.005 + 0.029 * (0.30 - 0.40) + 0.022 * (0.30 - 0.40) = 1.066$.

TD, typically developing; SPAS0, SPAS1 and SPAS2 indicate increasing levels of spasticity.

P -values indicate significance of main effects of spasticity group (P_1), walking speed (P_2) and group \times walking speed (P_3).

^{*1012} indicate significant difference of subgroup to TD^(T), SPAS0⁽⁰⁾, SPAS1⁽¹⁾ and SPAS2⁽²⁾, respectively.

^a B_0 and B_2 values are identical in all groups; the difference between groups is indicated by B_1 and B_3 .

^b Set to zero because TD was used as reference group.

peak length (Table 2: B_3 , P_3). Peak velocity increased more with walking speed in SPAS0 than in all other groups, but no difference was found between SPAS1 or SPAS2 and TD. Peak length increased more with walking speed in SPAS0 than in SPAS2.

4. Discussion

This study investigated the role of two factors that may contribute to hamstrings length and velocity during gait, i.e. walking speed and spasticity. When walking speed was reduced, both peak ST length and velocity decreased, with relatively small changes in peak length. Spastic muscles were shorter and slower during gait than non-spastic muscles, even after controlling for walking speed.

We studied the effect of walking speed in CP patients, since it can differ considerably between gait analyses pre- and post-treatment, or between subjects, for many reasons. For good interpretation, it is important to understand the separate effects of walking speed on gait parameters. Our result that peak ST length increased only slightly with walking speed indicates that differences in peak length between patients and control subjects, or between pre- and post-treatment analyses mostly reflect deviations in pelvis or leg positioning in terminal swing, or differences in step length, that are not attributable to differences in walking speed per se. As derived from Table 1, stride length was indeed lower in the more spastic groups than in TD, even at constant walking speed (e.g. compare SLOW in TD with CWS in CP). This could partly explain the differences in ST length. Contrarily, the strong increase in peak ST velocity with walking speed indicates that differences between gait analyses in hamstrings velocity can to a large extent results from differences in walking speed. This reinforces the result of other studies (e.g. [5,19–21]) that walking speed is an important factor to consider when interpreting gait data.

With our methodology the effects of walking speed could be separated from the effects of spasticity, showing that spastic muscles are shorter and slower than non-spastic muscles, even at constant walking speed and over a range of speeds. Although no other studies controlled for walking speed in this way, our findings are in line with previous results on the relationship between spasticity and hamstrings length during gait in CP patients [6]. These results together indicate that spasticity may be an important contributor to short and slow hamstrings during gait. The close relationship between physical examination spasticity scores and muscle-tendon length and velocity during gait also strengthens the need to study gait at muscle level, rather than joint and segment

level alone, in order to better understand underlying causes of gait deviations in CP.

Of course hamstrings behavior during gait can be influenced by other factors than walking speed and spasticity alone. For example, short passive hamstrings length (contractures) or increased intrinsic muscle stiffness may restrict hamstrings length and velocity during gait [22]. Furthermore, muscle weakness, poor selective control or inadequate push-off can affect swing leg behavior, and thereby hamstrings length and velocity during gait [23]. The difference between groups may therefore be caused not only by direct effects of spasticity, but also by secondary effects or other related impairments. Based on the concept of spasticity, changes in muscle-tendon velocities directly caused by spasticity are expected to evoke involuntary muscle (reflex-)activation [24]. Future study of electromyographic data will provide more insight into these effects. Moreover, further study is recommended on a large set of patient data, which would allow for multivariate analyses in which more of the possible underlying causes of short and slow hamstrings could be included, as well as further subgroup analyses.

Since spasticity has been defined as a velocity-dependent increase in muscle tone [8], it could be expected that effects of spasticity would increase with higher walking speeds. We did find interaction effects of spasticity and walking speed. First, children with more spastic ST muscles walked slower than children with less spastic muscles (Table 1), thereby avoiding high muscle lengthening velocities. Second, length and velocity in non-spastic muscles in CP increased more with walking speed compared with spastic and TD muscles. This may reflect a possible compensation strategy at faster speeds, to compensate for more involved muscles, for example to allow for a larger or faster step at the least affected body side in asymmetric gait. Further investigation of kinematics and detailed musculoskeletal modeling is necessary to study this and other possible compensation strategies to walk with slow and short muscles when faster walking speeds are required or imposed.

Spastic muscle length and velocity tended to increase somewhat less with walking speed than in non-spastic muscles on average, but this effect was small and only significant compared to non-spastic CP muscles. A factor to consider may be that we linearized the relation between peak hamstrings length or velocity and walking speed for the area under consideration. This simplification appeared reasonable, and non-linear approximations did not considerably improve the goodness of fit. However, peak ST velocity in the TD and SPAS0 groups seemed somewhat non-linear, with lower slope at faster walking speeds. Future study

with a broader range of walking speeds could reveal a more complex relationship of muscle-tendon lengths and velocities with walking speed, as has been shown for other kinematic, kinetic and electromyographic variables [21]. This may also reveal more significant interaction effects of walking speed and spasticity.

5. Conclusions

This study investigated the separate effects of walking speed and spasticity on hamstrings length and lengthening velocity in children with CP. Peak hamstrings length increased only slightly with walking speed; therefore, differences in peak hamstrings length between patients and control subjects, or between pre- and post-treatment analyses will mostly reflect deviating pelvis or leg positioning in terminal swing, or differences in step length, and can only for a small part be attributed to differences in walking speed per se. Peak hamstrings lengthening velocity increased strongly with walking speed; therefore differences in hamstrings velocity can to a large extent result from differences in walking speed. These data are important as a reference for valid interpretation of hamstrings length and velocity data in gait analyses at different walking speeds. Even when controlled for walking speed, spastic hamstrings were considerably shorter and slower during gait than normal, while non-spastic hamstrings were not.

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Conflict of interest statement

The authors declare that they have no conflicting interests.

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