Original Clinical Article

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Influence of impaired selective motor control on gait in children with cerebral palsy

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Abstract

Purpose Spastic cerebral palsy (CP) is characterized by four neuromuscular deficits: weakness, short muscle-tendon unit, muscle spasticity and impaired selective motor control (SMC). We examined the influence of impaired SMC on gait in children with bilateral spastic CP. Delineating the influence of neuromuscular deficits on gait abnormalities can guide surgical and therapeutic interventions to reduce long-term debilitating effects of CP.

Methods The relationship between impaired SMC and gait was assessed using multivariate linear regression analysis of Selective Control Assessment of the Lower Extremity (SCALE) in relation to stance phase knee flexion and temporal-spatial gait parameters calculated using 3D kinematics for 57 children with bilateral spastic CP, ages seven to 11 years.

Results Mean SCALE values were 5.8 (0 to 10, SD 3.0) and 5.7 (0 to 10, SD 2.9) for right and left legs, respectively. Multivariate linear regression models, including right and left SCALE and height, significantly predicted right and left knee flexion at initial contact ($R = 0.479$, $p = 0.003$; $R = 0.452$, $p = 0.007$, respectively) and right and left knee flexion in midstance ($R = 0.428$, $p = 0.013$; $R = 0.407$, $p = 0.022$, respectively). The model significantly predicted right and left step length ($R = 0.645$, $p = 0.000$; $R = 0.523$, $p = 0.001$, respectively) and predicted gait velocity ($R = 0.444$, $p = 0.008$). The model including SCALE did not predict step width.

Conclusion Results indicate impaired SMC predicts increased knee flexion at initial contact, and reduces step length and velocity. Understanding the influence of impaired SMC on gait can inform decisions regarding therapy and surgery, such as hamstring lengthening.

Level of Evidence Level II Retrospective Study

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Keywords: gait; cerebral palsy; selective motor control; temporal-spatial parameters; flexed-knee gait

Introduction

Cerebral palsy (CP) is the most common movement disorder in children, with a worldwide prevalence of approximately three per 1000 live births, and a higher prevalence of 60 to 150 per 1000 among preterm infants weighing < 1500 grams at birth.[1] A diagnosis of CP is often made based on MRI, delay of motor milestones and the presence of gait abnormalities in young children, which range in severity from mild, e.g. toe-walking, to severe, e.g. crouched, internally rotated gait.[2] Although the initial brain injury is non-progressive, the musculoskeletal impairments and functional limitations associated with CP are progressive.[3] Three main classes of CP include spastic, dyskinetic and ataxic. Spastic CP, which involves injury to the corticospinal tract (CST) as well as other brain regions, affects approximately 87% of children with CP[4] and results in four interrelated neuromuscular deficits: muscle weakness, short muscle-tendon unit, spasticity and impaired selective motor control (SMC). These deficits arise from the brain injury that interrupts descending excitatory and inhibitory signals. While the influence of weakness,[5] short muscle-tendon unit,[6] and spasticity[7] on gait has been studied, the impact of impaired SMC on gait is not well delineated. Understanding how neuromuscular deficits contribute to gait abnormalities can help guide more strategic treatment for children with CP.[8-10]

Impaired SMC is defined as 'impaired ability to isolate the activation of muscles in a selected pattern in response to demands of a voluntary posture or movement'.[11] A

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synergistic mass movement pattern is defined as a simultaneous, obligatory flexor or extensor pattern at two or more joints.[12,13] These flexor or extensor synergies interfere with isolated joint movements resulting in obligatory muscle co-activation of flexor or extensor muscles during functional activities such as gait.[14] The Selective Control Assessment of the Lower Extremity (SCALE) is an observation-based measure for children with spastic CP that has been found to reliably quantify the SMC of lower limb joints in children with CP.[15] The SCALE scores for children with spastic CP have also been found to correlate with gross motor function, specifically the Gross Motor Functional Classification Scale (GMFCS) levels.[15]

The benchmark for delineating gait abnormalities in children with CP in terms of joint kinematics and kinetics is 3D gait analysis, as well as temporal-spatial (TS) gait parameters such as velocity, step length, percentage single limb support in the gait cycle (%SLS), percentage stance in the gait cycle (%stance), and step width which reflect functional severity of gait abnormalities (Fig. 1).[16] TS parameters are more easily recorded in the clinical setting and often used as a metric for measuring gait improvement. Assessment of SCALE in relation to lower limb gait kinematics and TS parameters may clarify how SMC impacts gait and can help researchers and clinicians better understand the functional implications of impaired SMC in children with spastic CP.

Here, we examine the influence of impaired SMC on gait kinematics and TS parameters in children with bilateral spastic CP, including knee flexion at initial contact (IC) and midstance, step length, velocity, %SLS, %stance and step width. Achieving a normal step length requires the ability to fully extend the knee in terminal swing, when the hip is flexed, a movement that requires intact SMC. In contrast, achieving full knee extension in midstance, when the hip is extended, does not necessarily require normal SMC. Therefore, we hypothesized that the severity of impaired SMC as measured by SCALE would correlate to magnitude of knee flexion at IC and to step length.

Materials and methods

Patients and parameters

Data was obtained from all children with spastic CP GMFCS I to III who were seen in the Motion & Gait Analysis Lab at Lucile Packard Children's Hospital at Stanford, California from August 2010 (when SCALE measurements were first initiated) through to October 2018, and who met inclusion criteria. Only data from the first visit that SCALE was collected was analyzed; thus patients with multiple gait analysis studies were included only once. The inclusion criteria were as follows: 1) age seven to 11 years, 2) diagnosis of spastic CP and 3) the ability to walk independently without assistive devices for at least 6 m. Only data from children GMFCS I and II were included in the analysis. Four children with GMFCS III (who used assistive devices that did not limit stride) were reported separately for visual inspection in Figure 2, one participant used forearm crutches, two participants used reverse walkers and one child walked with hand-held assist. Children with musculoskeletal injury, who had > 15° of passive range of knee extension or > 15° of knee extension in midstance, who had a history of dorsal rhizotomy surgery, or who had orthopaedic surgery within one year prior to evaluation, were excluded. Children with a diagnosis of hemiplegia with a SCALE score of 10 in one limb with no hip, knee or ankle contracture (> 5° flexion at extension on passive range of movement (ROM)) were also excluded. TS parameters were calculated using 3D motion capture; impaired SMC was evaluated using SCALE. Right and left lower limbs were reported separately for each patient to capture the range of severity of impaired SMC and its correlation to a range of gait parameters, bilaterally. Both the involved and less involved side SCALE values were included for all participants given the bilateral nature of SCALE and TS gait parameters. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

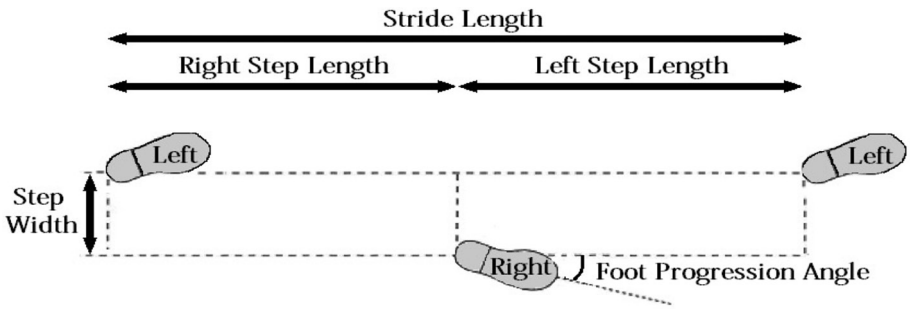

Fig. 1 Temporal-spatial parameters of gait.

JOURNAL OF CHILDREN'S ORTHOPAEDICS

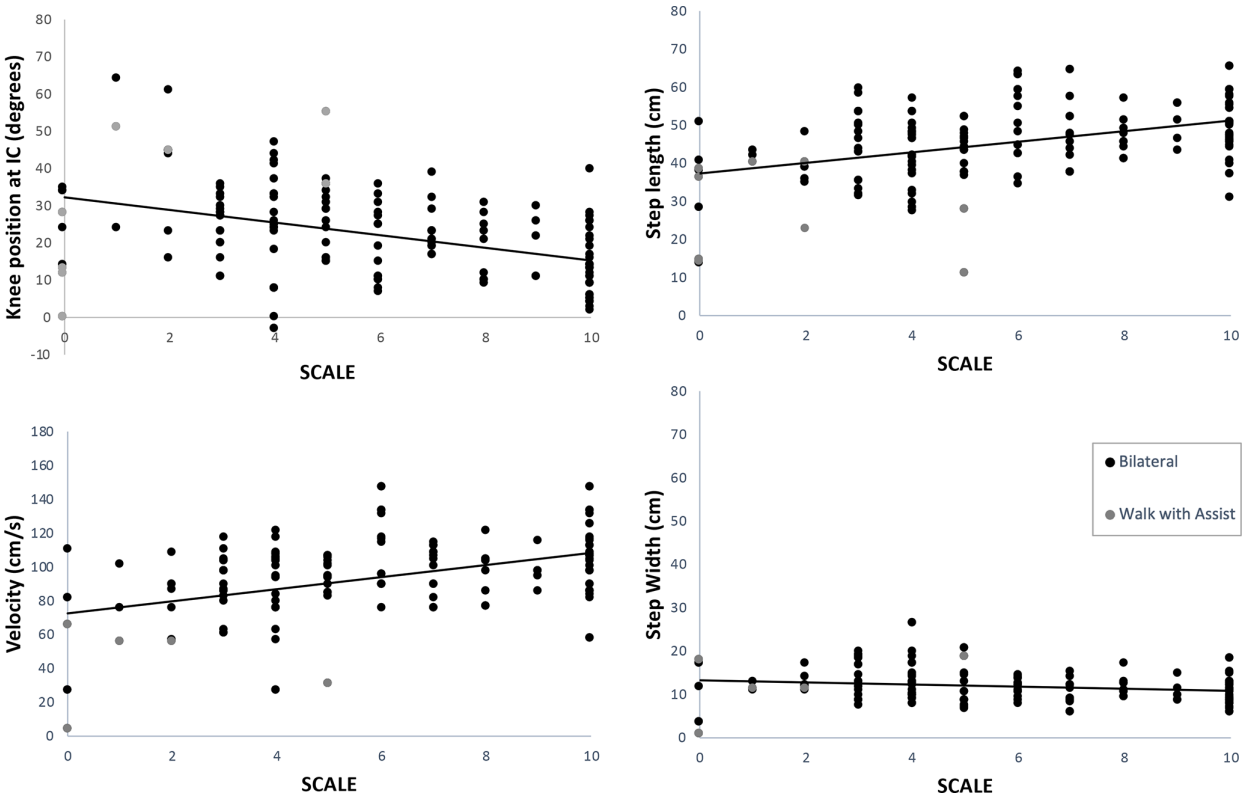

Fig. 2 Scatterplot demonstrating the relationship of the Selective Control Assessment of the Lower Extremity (SCALE) and temporal-spatial and kinematic parameters: (**a**) SCALE *versus* knee position at initial contact (IC); (**b**) SCALE *versus* step length; (**c**) SCALE *versus* velocity; (**d**) SCALE *versus* step width.

Gait parameters recorded from 3D analysis of gait kinematics

Walking was tested barefoot at a self-selected speed along a 6 m path, and marker trajectories were recorded with an eight-camera optoelectronic system for 3D motion analysis (Motion Analysis Corporation, Santa Rosa, California) at a sampling rate of 100 Hz. A modified Helen Hayes marker set for lower limbs was used: 18 reflective markers were placed on the sacrum, the left and right anterior superior iliac spines, the mid-shaft femurs, the lateral knee joint axes, the mid-shaft tibias (shanks), the lateral malleoli, the calcanei, the dorsum of foot between the second and third metatarsal heads, the right and left acromia and the left scapula. For a static calibration trial, four additional markers were placed on the right and left medial knee joint axes and medial malleoli. OrthoTrak software version 6.6.4 (Motion Analysis Corporation) was used to calculate joint kinematics and TS parameters from an average of three representative trials. TS velocity was calculated from the sacral marker, cadence was calculated from number of steps in a trial divided by trial time, step length was calculated from distance along the direction of progression from ipsilateral foot strike to subsequent contralateral foot strike, step width was calculated from the distance between ankle centres along the axis of the room perpendicular to the direction of walking, and stance and SLS as a percent of the gait cycle as determined from timing of ipsilateral and contralateral foot strike. Knee position at IC and maximum knee extension in midstance was measured in degrees of flexion.

SCALE and ROM

SCALE was used as a clinical tool to quantify voluntary SMC in patients with CP, assessing hip flexion/extension in a side-lying position and knee extension/flexion, ankle dorsiflexion/plantarflexion, subtalar inversion/eversion and toe flexion/extension in a sitting position. These five movements were graded at each joint as 'normal' (2 points), 'impaired' (1 point) or 'unable' (0 points), for a maximum of 10 points per leg. SCALE measurements were obtained by a physical therapist, and a total of two physical therapists conducted measurements for all participants. SCALE measurements have a high reliability; prior studies of mean interrater reliability have been found to be 0.88 to 0.91 ($p < 0.001$).[15]

As reported by Fowler et al (2009),[15] a grade of 'Normal' is given when the desired movement sequence

is completed within the verbal count without movement of untested ipsilateral or contralateral lower extremity joints. A grade of 'Impaired' is given when the patient isolates movement during part of the task, but demonstrates any of the following errors: movement occurs in only one direction; observed movement is < 50% of the approximate available passive ROM found during the passive demonstration; movement occurs at a non-tested joint (including mirror movements); or the time for execution exceeds the approximate three-second verbal cadence. A grade of 'Unable' is given when the requested movement sequence is not initiated or when it is performed using a synergistic mass flexor or extensor pattern.

Passive ROM measurements

Knee and ankle passive ROM values were measured by a physical therapist with the patient in the supine position and reported in degrees of knee flexion and ankle plantarflexion, respectively.[17] A total of two physical therapists conducted measurements for all participants. Knee passive ROM limitation was defined as maximum knee extension measured in degrees of flexion; ankle passive ROM limitation was defined as maximum ankle dorsiflexion measured in degrees plantarflexion.

Statistical analysis

Data was inspected for normalcy using q-q plots of distribution and parametric tests were applied. Individual correlations of SCALE and passive ROM of the knee and ankle in relation to gait parameters were assessed using the Pearson correlation, adjusted for height. Based on the sample size, four independent variables were included in a multivariate linear regression model; the relationship between SCALE values and gait parameters was assessed using a multivariate linear regression model that included height, right, and left SCALE values of each participant. All statistical analyses were performed using SPSS version 24.0 (SPSS Inc., Chicago, Illinois).

Results

In all, 57 children ages seven to 11 years with bilateral spastic CP met the inclusion criteria, were seen in the gait lab from August 2010 to October 2018 for kinematic testing, and also had SCALE measurements. The mean age of participants was 9.33 years (7 to 11, SD 1.31). Age was well distributed (Table 1): six participants were aged seven years, 11 aged eight, 11 aged nine, 16 aged ten and 13 aged 11 years. All but two participants had a SCALE score < 10 in at least one limb. Mean SCALE values for the right and left limbs of children with CP were 5.8 (0 to 10, SD 3.0) and 5.7 (0 to 10, SD 2.9), respectively (Table 1). SCALE was correlated with ipsilateral Gait Deviation Index (GDI) (Table 2).

SCALE was negatively correlated with ipsilateral knee flexion at IC (R = -0.415, p = 0.000) (Table 2); this relation

Table 1 Participant demographics

	Bilateral spastic cerebral palsy independent walkers n = 57		Bilateral spastic cerebral palsy walks with assistive devices (not included in statistical analysis) n = 4	
	Mean	SD	Mean	SD
Height (cm)	134.27	9.96	9.25	2.06
Age (yrs)	9.33	1.31	1.75	2.36
Right SCALE	5.77	2.99	1.50	2.38
Left SCALE	5.74	2.91	0.25	0.50
SCALE difference (absolute values)	2.25	2.03	60.32	12.15
Right GDI	74.84	13.30	58.43	7.20
Left GDI	75.38	11.68	-1.25	8.54
Passive ROM* (right knee)	-1.49°	9.95°	-2.50°	6.45°
Passive ROM* (left knee)	-3.95°	8.65°	-11.25°	8.54°
Passive ROM* (right ankle)	-3.16°	13.01°	-15.00°	4.08°
Passive ROM* (left ankle)	-2.02°	13.59°	9.25°	2.06°
Knee contractures (n) Knee (n right, n left)	12 (10R, 6L)		1 (1R, 0L)	
Ankle contractures (n) Ankle (n right, n left)	16 (9R, 11L)		1 (1R, 1L)	
Prior treatment (n)				
None	28		1	
Bone surgery	0		2	
Soft-tissue surgery	17		1	
Botox	6		0	
Unknown	2		0	

*passive range of movement (ROM) limitation defined as maximum knee extension or ankle dorsiflexion, measured in degrees of flexion or plantarflexion, respectively. Contractures defined as > 5° flexion at passive maximum knee extension or ankle dorsiflexion

SCALE, Selective Control Assessment of the Lower Extremity; GDI, Gait Deviation Index.

remained significant when participants with knee flexion contractures > 5° (n = 12) were eliminated (R = -0.348, p = 0.001). SCALE also negatively correlated with ipsilateral knee flexion in midstance (R = -0.320, p = 0.001) (Table 2).

Knee passive ROM limitation correlated to ipsilateral knee flexion at IC, but was more strongly correlated to knee flexion in midstance (Table 2). Ankle passive ROM was correlated with knee flexion at IC, and more weakly correlated to knee flexion in midstance. SCALE correlated to ipsilateral passive knee extension limitation (R = -0.495, p = 0.000).

A multivariate linear regression model including right and left SCALE and height revealed a significant association between the model and gait velocity (R = 0.444, p = 0.008), and explained 15% (adjusted R^2 = 0.151) of gait velocity variance (Fig. 2, Table 3a). There was a significant association between the model including SCALE and step length: the model predicting to right step length (R = 0.645, p = 0.000; adjusted R^2 = 0.383) and left step length (R = 0.523; p = 0.001; adjusted R^2 = 0.232) explained 38% and 23% of right and left step length variance, respectively (Table 3a). These associations remained when the n = 12 patients who had flexion contractures > 5° of flexion at maximum knee extension were eliminated.

The model including SCALE was not significantly associated with cadence. The model demonstrated that lower SCALE values were significantly associated with shorter ipsilateral SLS and contralateral stance as percent of the gait cycle (Table 3a), however, the model was not significantly associated with step width (Table 3a).

Discussion

This work examines how gait is influenced by impaired SMC, one of four major neuromuscular deficits in spastic CP. The multivariate linear regression model revealed that impaired SMC, as measured by SCALE, was significantly associated with knee flexion at IC, with shortened step length, and with decreased velocity in children with spastic CP. The results support the hypothesis that severity of impaired SMC as measured by SCALE correlate to magnitude of knee flexion at IC and to step length. The relationship between SCALE and knee flexion at IC in children with CP may be due to their inability to fully extend the knee at the end of swing, when the hip is flexed, a movement that requires intact SMC. Similarly, the relationship between SCALE and knee position at midstance in children with CP may be due to their inability to fully extend the knee with ankle dorsiflexion. Given the correlations found between SCALE with step length and velocity, that velocity is the product of step length and cadence, and that SCALE was not correlated to cadence, the results indicate that the reduced velocity associated with impaired SMC is in large part driven by reduced step length. Furthermore, impaired SMC was not significantly associated with step width, a gait deficit often associated with ataxic, not spastic, CP (Table 3a, b). This is expected as muscle synergies indicative of impaired SMC would not likely impact step width directly. The four children with GMFCS III used assistive devices that did not limit stride and were plotted separately for visual inspection in Figure 2, however, they were not included in any statistical analyses. We report them to show that children who do rely on assistive devices have TS parameters that fall on the graph where expected for their SCALE values.

The individual partial correlations and the models including right and left SCALE were significant for SCALE in relation to ipsilateral knee flexion at IC, step length and velocity. Within the model there appeared to be a lack of additional significance derived from the contralateral SCALE value (Table 3a, b).

Neuromuscular correlates of gait

The neuromuscular deficits of spastic CP, muscle weakness, short muscle-tendon length, spasticity and impaired SMC[5,14,18] are interrelated, influence gait in different ways and contribute to equinus, flexed-knee, and a stiff-knee gait,[8] all of which may reduce step length. Short muscle-tendon unit and impaired SMC may contribute

Table 2 Partial correlations with height adjustment of Selective Control Assessment of the Lower Extremity (SCALE) and passive knee extension and ankle dorsiflexion range of movement (ROM) limitation in relation to ipsilateral gait temporal-spatial parameters and ipsilateral knee kinematics at initial contact (IC) and at midstance. Right-most column shows bilateral correlation of Gait Deviation Index (GDI) with SCALE and passive knee extension and ankle dorsiflexion ROM.

Bilateral limbs, n=114		Velocity	Cadence	Step length	%Stance	%SLS	Step width	Knee flexion at IC	Knee flexion at midstance	GDI
SCALE	Pearson R	**0.325**	0.024	**0.347**	0.004	**0.423**	**-0.211**	**-0.415**	**-0.320**	**0.426**
	Significance	0.000	0.798	0.000	0.965	0.000	0.025	0.000	0.001	0.000
Knee extension ROM	Pearson R	**-0.191**	0.162	**-0.289**	0.046	**-0.203**	0.045	**0.401**	**0.589**	-0.100
	Significance	0.042	0.086	0.002	0.626	0.031	0.638	0.000	0.000	0.140
Ankle dorsiflexion ROM	Pearson R	-0.069	0.114	-0.062	-0.135	**-0.227**	0.144	**0.331**	**0.293**	**-0.134**
	Significance	0.466	0.229	0.514	0.155	0.016	0.129	0.000	0.002	0.046

Bold = significant, R/p < 0.05 level (two-tailed)

%Stance, percentage stance of gait cycle; %SLS, percentage single limb support of gait cycle

Table 3a Multivariate linear regression model including Selective Control Assessment of the Lower Extremity (SCALE) in relation to gait temporal-spatial parameters in children with spastic cerebral palsy, with independent variables: right SCALE, left SCALE, height

Bilateral spastic CP subjects, N= 57	Velocity	Cadence	Right step length	Left step length	Right % stance	Left % stance	Right %SLS	Left %SLS	Step width
R	**0.444**	.257	**.645**	**.523**	**.468**	**.392**	**.391**	**.468**	0.295
P	0.008	.113	.000	.001	.000	.002	.002	.000	.182
R^2	0.197	.066	.416	.273	.219	.153	.153	.219	.087
Adjusted R^2	0.151	.034	.383	.232	.192	.124	.124	.192	.035
	Beta, p	**Beta, p**	**Beta, p**	**Beta, p**	**Beta, p**	**Beta, p**	**Beta, p**	**Beta, p**	**Beta, p**
R SCALE	.094, .461	.233, .074	.357, .002	.247, .045	.220, .065	-.451, .000	.451, .000	-.220, .065	-.106, .437
L SCALE	.088, .534	.046, .726	.092, .446	.162, .230	-.573, .000	.105, .399	-.105, .399	.573, .000	-.122, .417
Height	.365, .012	-.103, .343	.414, .001	.307, .026	.111, .265	.049, .637	-.049, .635	-.111, .265	-.171, .259
Standard error	20.3	38.2	7.29	7.71	4.90	4.97	4.97	4.89	4.01

Bold = Beta significance < 0.05 level (two-tailed)

IC, initial contact; %Stance, percentage stance of gait cycle; %SLS, percentage single limb support of gait cycle

Table 3b Multivariate linear regression model including Selective Control Assessment of the Lower Extremity (SCALE) in relation to gait knee kinematics in children with spastic cerebral palsy, with independent variables: right SCALE, left SCALE, height.

Bilateral spastic CP subjects, N= 57	Right knee flexion at IC	Left knee flexion at IC	Right knee flexion midstance	Left knee flexion midstance
R	**.479**	**.452**	**.428**	**.407**
P	.003	.007	.013	.022
R^2	.229	.204	.183	.165
Adjusted R^2	.186	.159	.137	.118
	Beta, p	**Beta, p**	**Beta, p**	**Beta, p**
R SCALE	-0.085, 0.498	-0.152, 0.232	0.313, 0.017	-0.208, 0.112
L SCALE	-0.461, 0.001	-0.155, 0.272	-0.243, 0.091	-0.269, 0.064
Height	0.007, 0.961	-0.296, 0.040	-0.184, 0.200	-0.192, 0.188
Standard error	10.95	10.46	11.53	11.06

Bold = Beta significance < 0.05 level (two-tailed)

IC, initial contact; %Stance, percentage stance of gait cycle; %SLS, percentage single limb support of gait cycle

to equinus gait,[19,20] flexed-knee gait,[6,10] and stiff knee gait[8,10] (Fig. 3). The current results indicate specifically that impaired SMC predicted to knee flexion at IC, reduced step length and reduced velocity in children with flexed-knee gait.

We found that knee passive ROM correlated to knee flexion at IC, but more strongly correlated to knee flexion in midstance, and negatively correlated to ipsilateral step length (Table 2). Knee flexion contracture would be expected to contribute to knee flexion in stance phase, and 12 of 57 participants had knee flexion contracture. These findings further support the hypothesis that impaired SMC may be associated with increased knee flexion at IC and reduced step length.

We found that ankle passive dorsiflexion ROM demonstrated a correlation with ipsilateral knee flexion at IC and to knee flexion in midstance and did not correlate to ipsilateral step length or velocity. The association of ankle passive ROM limitation to knee flexion at IC may be expected because ankle plantarflexion at IC with forefoot contact, biomechanically imposes a slight flexed-knee posture.

Studies using musculoskeletal models have identified a shortened semitendinosus during gait as contributing to two-thirds of the cases of flexed-knee stance.[6] However, the influence of impaired SMC on gait metrics derived from musculoskeletal models require further study. In a prior study, Rha et al (2016),[10] analyzed data collected over two-year study period and found that while a short semitendinosus during gait correlated with increased knee flexion at IC and midstance, SCALE was a stronger correlate of knee flexion at IC than short semitendinosus during gait or short hamstring passive ROM, which were more strongly associated with increased knee flexion in midstance.

Impaired SMC in spastic CP: influence on gait

Results of the current study indicate that impaired SMC predicted a lack of full knee extension at IC, thereby reducing step length and velocity. This may be expected because a flexion synergy can limit knee extension in terminal swing, just prior to IC, when the hip is flexed, leading to a flexed-knee posture at IC which reduces step length. Additional gait events may also be influenced by impaired SMC. Observational analysis indicates that impaired SMC can slow the transition from stance phase hip and knee extension to swing phase hip and knee flexion, leading to gait asymmetry and poor foot clearance in swing, which can also reduce step length.[21] Furthermore, an extensor synergy may trigger ankle plantarflexion (ankle extension) in terminal swing when the knee is extending, leading to a forefoot IC.[18,19]

Our results support the findings of Fowler et al (2010)[21] who reported that impaired SMC, as measured by SCALE, was significantly correlated to increased coupling of hip and knee joint movement in swing phase, with loss of inter-joint coordination in mid-terminal swing phase.

Chruscikowski et al (2017)[22] studied impaired SMC in relation to global gait impairment in 194 patients with bilateral CP and found a negative correlation between

JOURNAL OF CHILDREN'S ORTHOPAEDICS

SCALE and global gait impairment, as measured by the Gait Profile Score, suggesting that impaired SMC negatively affects overall gait function. Our prior studies found that children with mild to severe spastic CP consistently demonstrate co-activation of the quadriceps and gastrocnemius on EMG, distinguishing spastic CP from idiopathic toe walking.[19,20] Our current results build on this prior work and further clarify the implications of impaired SMC on gait and support the conclusion of prior studies that individuals with greater SMC impairment as measured by SCALE demonstrate reduced complexity of neuromuscular control during gait through increased flexion/extensor synergy.[23]

Understanding the relationship between SMC, kinematics, and TS parameters can help to clarify the impact of SMC on gait.[24] While joint kinematics, kinetics, and muscle activity are important for describing specific gait pattern abnormalities associated with CP,[25] gait velocity has important functional relevance. Gait velocity is highly correlated with energy expenditure, such that humans innately choose a free-walking speed that is most energy efficient.[26] Therefore, quantifying gait velocity in relation to SMC is fundamental to characterizing the functional limitations imposed by impaired SMC on ambulatory ability.[27]

Impaired SMC in spastic CP: treatment

Treatment for impaired SMC in children with spastic CP is currently being developed and has demonstrated promising early results, including robotic assisted passive and active movement therapy. Emerging evidence points to the rubrospinal tract playing a compensatory role subsequent to CST injury in stroke patients,[14] and if this is indeed the case in CP, the rubrospinal tract may respond to intensive training treatments. Intensive treatments, such as those used to improve balance[28] and manual dexterity[29] have proven beneficial. Treatments utilizing high dosage intensive exercise, in patterns outside of synergy patterns, such as knee extension combined with ankle dorsiflexion, may prove beneficial in improving impaired SMC and gait. Wu et al (2011)[30] found that combining passive stretching and active movement training outside of extension synergy with high repetition exercises resulted in significant increases in passive and active ROM, dorsiflexor and plantarflexor muscle strength, SCALE and functional outcome measures. With knees extended, the 12 participants in the study voluntarily plantarflexed and dorsiflexed their ankle first with robotic assistance and then with robotic resistance. The ROM target of the computer games was scaled to the individual participant's ROM. All participants improved in balance ($p = 0.003$) and distance walked as part of functional outcome measures ($p = 0.02$). This study suggests that treatments combining robotic devices with motivating games involving intensive exercise patterns outside of synergy patterns, such as hip flexion combined with knee extension, and knee extension combined with ankle dorsiflexion, may prove beneficial and warrant further investigation.[31]

Hamstring lengthening is frequently performed to improve stance phase knee extension by increasing the muscle-tendon unit length. Although terminal swing knee extension may improve following lengthening, improve-

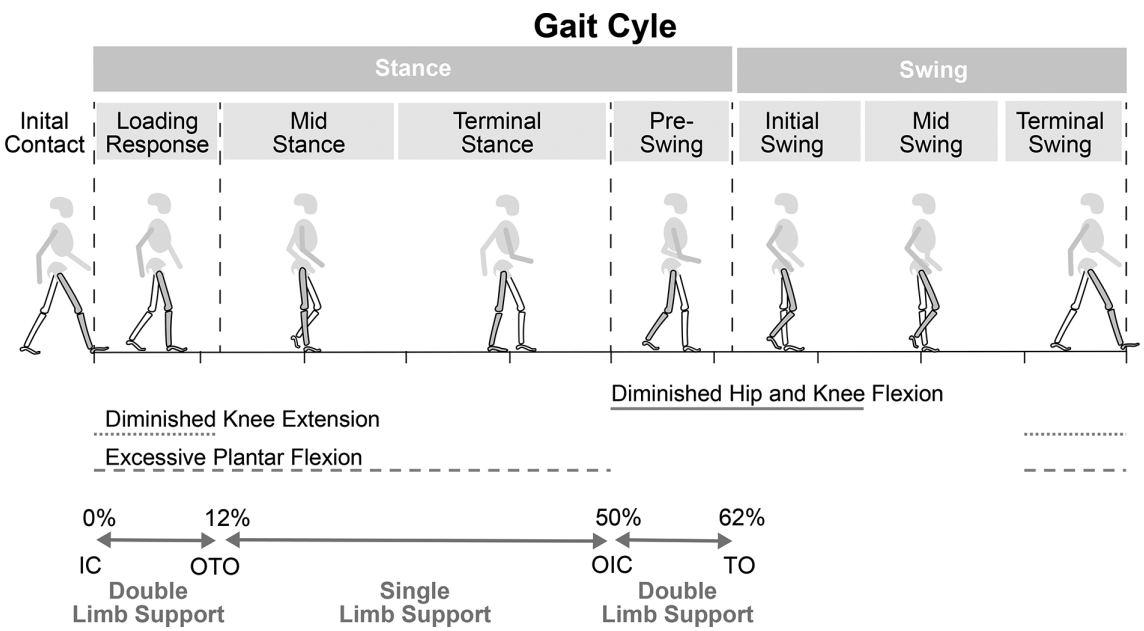

Fig. 3 Common gait abnormalities and impaired selective motor control. IC, initial contact; OTO, opposite toe-off; OIC, opposite foot initial contact; TO, toe-off.

ments are not consistent across patients, and peak knee extension does not typically approach normative values.[21] Variation in SMC impairment may explain these findings. Patients with poor SMC ability may be unable to dissociate hip and knee movement during swing regardless of hamstring length. In contrast, patients with better SMC ability, initially constrained by biomechanical factors, may be better able to utilize their increased ROM following hamstring lengthening. An understanding of the influence of SMC on stance and swing phase mechanics during gait may help direct treatment and establish more realistic goals for interventions, in particular for hamstring lengthening.

Study limitations include the retrospective nature of the analysis and a sample size that limited multivariate analysis. Further research is needed to investigate larger populations with multicenter investigations and to clarify the influence of impaired SMC on gait, particularly on limb motion in early swing in relation to gait symmetry and foot clearance. It is important to develop and evaluate treatment for impaired SMC in persons with spastic CP.

Conclusion

Results suggest that impaired SMC as measured with SCALE predicted increased knee flexion at IC, decreased step length and reduced gait velocity in children with spastic CP. Delineating the influence of motor disorders on gait can more specifically guide targeted treatment to improve neuromuscular function and gait in spastic CP.

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COMPLIANCE WITH ETHICAL STANDARDS

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ETHICAL STATEMENT

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Not required for this work.

ICMJE CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interest.

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AUTHOR CONTRIBUTIONS

J. Y. Zhou: research aims, data collection, data analysis, writing the manuscript.
E. Lowe: data collection, data analysis.
K. Cahill-Rowley: research aims, data analysis, revising the manuscript.
G. B. Mahtani: data collection, data analysis.
J. L. Young: data analysis, manuscript revision, clinical input.
J. Rose: research aims, data collection, data analysis, writing the manuscript, clinical input.

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