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Weakening iliopsoas muscle in healthy adults may induce stiff knee pattern

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ABSTRACT

Objective: The goal of the present study was to investigate the relationship between iliopsoas muscle group weakness and related hip joint velocity reduction and stiff-knee gait (SKG) during walking in healthy individuals.

Methods: A load of 5% of each individual's body weight was placed on non-dominant thigh of 15 neurologically intact, able-bodied participants (average age: 22.4 ± 0.81 years). For 33 min (135 s $\times$ 13 repetitions $\times$ 5 s rest), a passive stretch (PS) was applied with the load in place until hip flexor muscle strength dropped from 5/5 to 3+/5 according to manual muscle test. All participants underwent gait analysis before and after PS to compare sagittal plane hip, knee, and ankle kinematics and kinetics and temporo—spatial parameters. Paired t-test was used to compare pre- and post-stretch findings and Pearson correlation coefficient (r) was calculated to determine strength of correlation between SKG parameters and gait parameters of interest ($p < 0.05$).

Results: Reduced hip flexion velocity (mean: 21.5%; $p = 0.005$) was a contributor to SKG, decreasing peak knee flexion (PKF) (-20%; $p = 0.0008$), total knee range (-18.9%; $p = 0.003$), and range of knee flexion between toe-off and PKF (-26.7%; $p = 0.001$), and shortening duration between toe-off to PKF (-16.3%; $p = 0.0005$).

Conclusion: These findings verify that any treatment protocol that slows hip flexion during gait by weakening iliopsoas muscle may have great potential to produce SKG pattern combined with reduced gait velocity.

Spastic paretic stiff-knee gait (SKG) is among the most common gait abnormalities diagnosed in clinics (80% of ambulatory children with cerebral palsy [CP]), and it is defined by diminished and delayed peak knee flexion (PKF) angle in swing phase.[1] It can cause tripping in swing phase and increases energy expenditure during walking.[1–3] Excessive activity of rectus femoris muscle during swing or pre-swing phase of gait is major cause of SKG.[1–8] In such cases, treatment is directed at rectus femoris. It may be 1) surgically treated by transferring distal insertion[1,7] or by performing intramuscular lengthening,[6] 2) treated with chemo-denervation by injecting neuromuscular blocking agents,[8] or 3) treated using physiotherapy (i.e., stretching rectus femoris or strengthening antagonist muscles).[1] Although these procedures are widely applied in clinics, outcomes remain varied. They are mentioned as inconsistent,[1–3] variable,[9] not always beneficial,[3] and not always persistent.[10] Therefore, understanding of influences on normal knee flexion (KF) in swing phase is greatly needed.[3]

Due to occurrence of hip flexor muscle contracture in CP, operations that lengthen psoas through myofascial lengthening of common iliopsoas tendon or iliopsoas lengthening (in more severe cases) are commonly performed prior to or concomitant with rectus femoris transfer.[6,11,12] Children with CP are generally weaker, especially in multi-joint muscles (such as iliopsoas), and walk more

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slowly than their typically developing peers.[11–13] Iliopsoas is primary hip flexor muscle group and it directly affects hip flexion velocity during swing phase. These treatment procedures for correction of hip flexor muscle contracture may reduce hip flexion velocity. No previous reports have investigated the relationship between iliopsoas weakness related to reduced hip flexion velocity and SKG pattern in able-bodied individuals.

It has been reported that electromyographic (EMG) activity of rectus femoris muscle has a significant relationship to first half of pre-swing phase of gait.[16] In pre-swing phase, iliopsoas is primarily active during normal walking;[7] however, rectus femoris muscle may compensate for weak iliopsoas muscle, which promotes knee extension and may theoretically reduce KF velocity for patients with SKG pattern.[14–16]

Muscle-driven, simulation-based studies have demonstrated that abnormal muscle activation prior to swing phase influences KF velocity and alters PKF time and angle.[3,5,16–18] According to these studies, reducing iliopsoas force theoretically influences PKF, causing SKG. Moreover, these studies using simulation models demonstrated that in some cases, increased hip flexion improves KF in swing phase,[17–19] though this has not yet been validated in able-bodied participants.

Therefore, this study aimed to investigate relationship between weakness of iliopsoas muscles and related hip joint velocity reduction and SKG during walking in healthy individuals. The hypothesis of this study was that there would be reduction in peak knee angle and range of KF during gait.

Patients and methods

Fifteen able-bodied participants (13 male, 2 female) aged 22.4 ± 0.81 years (weight: 70.5 ± 10.15 kg; height: 175.6 ± 4.2 cm) with no neurological or musculoskeletal problems were included in the study. Participants had no prior history of neurological or musculoskeletal disorders and had not participated in any physical sporting activities for 3 days prior to undergoing study tests. The institutional review board of the Istanbul University Faculty of Medicine approved study design. The 15 participants were more than minimum sample size needed to ensure power of 90% confidence level and to detect statistical significance at a 2-sided significance level of 0.05 ($\beta = 0.2$) by considering average change in 4 SKG parameters as effect (19.6%) between 2 conditions and using calculation method as basis for descriptive studies.[20]

First, according to applied stretching and loading protocol in the present study, hip flexor muscle strength of non-dominant side was measured using manual muscle test (MMT).[23] Non-dominant side was stretched and loaded to create appropriate condition to interpret the results. Non-dominant side was arbitrarily selected for stretching. In order to emphasize unilateral hip flexion velocity reduction and to increase weight on thigh segment for iliopsoas to more efficiently mimic asymmetric hip flexor weakness, a load equal to 5% of each participant's body weight (BW) was firmly strapped to front of distal part of non-dominant thigh (Fig. 1a). Passive stretch (PS) as described by Fowles et al,[22] which was reported to produce 25% loss in maximum voluntary force after 30 min of prolonged muscle stretch, was performed on participants' non-dominant iliopsoas muscle with the weight attached while he/she was lying supine on a bed (Fig. 1b). Only skin around sacral marker was marked before removal for stretching protocol; remaining markers were left in place during stretching. Placement of all markers was visually checked and sacral marker was replaced immediately after stretching protocol. Stretch was limited by participants' tolerance to pain (Fig. 1b); researcher verbally confirmed whether stretch was under participant's pain threshold every 30 s. After 33 min of stretching (135 s × 13 repetitions) with 5 s of rest

between stretches, MMT was performed with the weight on thigh to determine if strength level had decreased 5/5 to 3+/5. All participants were tested by the same researcher to avoid inter rater difference. Unlike the work of Fowles et al,[22] in this case, if desired strength reduction was not achieved, an additional stretching protocol of 5 repetitions was performed. After that, gait analysis was conducted with the weight on non-dominant thigh within 1 min (loading protocol). All participants underwent gait analysis at self-selected speed before and after stretching and loading protocols. Gait analysis was carried out at the Istanbul University Gait Analysis Laboratory using optoelectronic system with 6 cameras and 2 force plates (ELITE 2002; BTS Bioengineering, Milan, Italy) between June 2014 and October 2014 based on description of Davis et al (modified Helen Hayes).[21] Sagittal plane A-kinematic parameters: 1) pelvic tilt, 2) hip flexion, 3) KF, and 4) ankle plantar flexion (PF) velocities; B-kinetic parameters: 1) hip power (H3) and moment, 2) knee power and moment, and 3) ankle power and moment of stretched side; and C-temporal—spatial parameters: 1) mean velocity (m/s), 2) cadence (step/min), 3) stance time (m/s), 4) stride time (m/s), and 5) step length (mm) were gait parameters of interest. Loss of hip power (H3) in late stance was selected as main gait parameter.[7,16] Gait analysis parameters measured immediately after (within 1 min), 1—2 min, and 3—4 min after stretching were also compared. Average of 3 gait trials pre-stretch and gait trial and measurements immediately after stretch (full contact with force platform) were compared for statistical analysis. Joint velocity was reported as degrees per second by normalizing joint velocity individually with stride time (seconds) for each participant before averages were compared. All parameters were compared for 2 different walking conditions: walking before stretch (pre-stretch, unloaded) (Pre-S) and walking after stretch (post-stretch, loaded with 5% BW on thigh) (Post-S).

Gait characteristics of 6 healthy individuals of similar age, height, weight (25 ± 2.9 years, 177 ± 5.3 cm, 73 ± 11.3 kg, respectively) who walked, at self-selected speed, as slowly as participants in post-stretch condition in this study (average: 0.89 ± 0.05 m/s) were compared with post-stretch condition results using Student's t-test ($p < 0.05$).

Four gait parameters were selected to determine SKG pattern: P1) PKF angle, P2) range of KF between toe-off and PKF, P3) total range of KF, and P4) time of PKF in swing.[2] For each participant, if average value of criteria was more than 2 standard deviations below average of normal value, it was indicated as stiff. Limb was considered stiff if ≥ 3 criteria were met and not stiff if 1 or none of the criteria were observed.[2] If 2 SKG indicators were observed, limb was classified as borderline case. Normality test (Shapiro—Wilk test) was used to determine whether or not they were normally distributed. As all parameters were normally distributed, paired t-test was used to compare pre- and post-stretch conditions, and Pearson correlation coefficient (r) was calculated to determine strength of correlation between SKG parameters and gait parameters for both conditions using Rovai et al guidelines (95% confidence interval).[24] P value of <0.05 was considered to indicate significant result.

Results

Sagittal plane kinematic alterations

Mean and range of pelvic tilt were not significantly different in Post-S condition compared to Pre-S condition (Table 1). In Post-S state, maximum hip flexion velocity between mid-stance and mid-swing significantly decreased from 169.53°/s (1.9 ± 0.3°/fr) to 133.19°/s (1.7 ± 0.3°/fr) (mean difference: 21.5%; p = 0.005). Similarly, maximum KF velocity decreased significantly from 299.59°/s
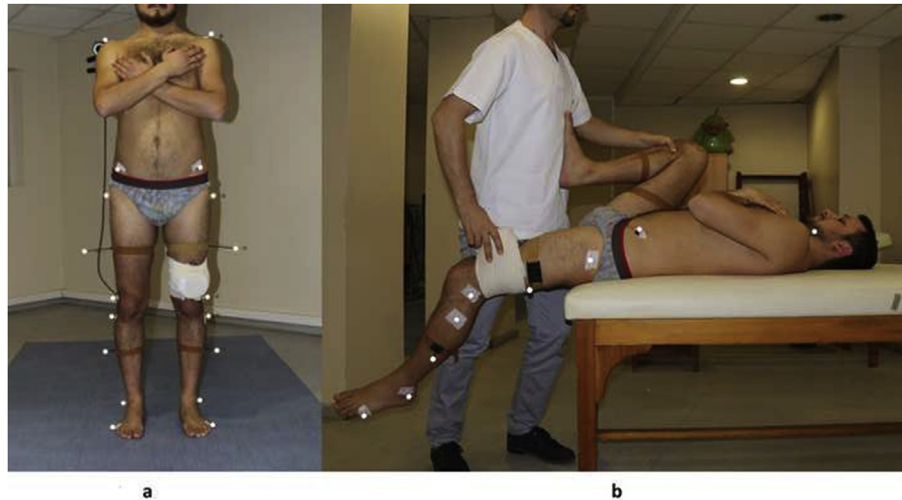
Fig. 1. Demonstration of (a) weight placement and (b) iliopsoas muscle stretching position.

Table 1
Stiff knee gait parameters and the other kinematic gait parameters of interest.

Parameters	Involved side (stretch & weight) (mean ± SD)			Uninvolved side (no stretch or weight) (mean ± SD)		
	Pre-stretch	Post-stretch + weight	P-value	Pre	Post	P-value
1-Peak knee flex (°)	55.3 ± 3.9	44.4 ± 8.3	**<0.01***	55.2 ± 4.8	53.8 ± 3.4	0.26
2-Range in early swing (°)	28.6 ± 87.6	21.0 ± 9.8	**0.01***	23.7 ± 8.2	22.9 ± 7.2	0.62
3-Total range (°)	58.8 ± 3.5	47.7 ± 11.5	**<0.01***	57.1 ± 4.3	54.6 ± 5.2	0.12
4-Duration from toe-off to PKF (% GC)	13.1 ± 2.7	10.9 ± 2.9	**<0.01***	11.7 ± 2.9	11 ± 2.7	0.20
Range of pelvic tilt	3.1 ± 0.7	3.3 ± 1.2	0.71	3.6 ± 1.3	3.5 ± 1.1	0.93
Mean pelvic tilt (°)	8.8 ± 3.4	8.8 ± 3.7	0.29	6.7 ± 3.4	7.5 ± 3.6	0.18
Hip flex vel @ toe-off (°/s)	125.8 ± 35.5	107.2 ± 25.3	0.15	124.5 ± 25.5	110.1 ± 15.1	**0.04***
Max hip flex vel 20% GC & PKF(°/s)	169.5 ± 29.7	133.2 ± 35.3	**<0.01***	170.6 ± 3	144.7 ± 17.5	**<0.01***
Time of max hip flex vel (% GC)	66,8 ± 2.7	65,7 ± 5.2	0.52	68.2 ± 0.2	68.8 ± 2.8	0.58
Knee flex vel @ toe-off (°/s)	267.3 ± 35.5	205.9 ± 46.9	**<0.01***	267.7 ± 36.5	233.4 ± 43.8	**<0.01***
Max knee flex vel between 20% GC- PKF (°/s)	299.6 ± 27.7	234.4 ± 41.6	**<0.01***	283.7 ± 31.4	251.4 ± 23.0	**<0.01***
Time of PKF velocity (% GC)	62.5 ± 1.9	62.7 ± 2.5	0.55	61.5 ± 1.9	65 ± 3.0	**<0.01***
Peak knee ext in stance (°)	−3.5 ± 3.6	−2.3 ± 6.1	0.34	−1.9 ± 3.8	−0.8 ± 5.2	0.37
Time of PKF (% GC)	72.5 ± 1.4	72.1 ± 2.8	0.52	71.9 ± 2.1	74.1 ± 2.2	**0.01***
Knee flex @ toe-off (°)	26.7 ± 8.8	23.4 ± 8.8	**<0.01***	31.5 ± 8.6	30.9 ± 6.7	0.76
Time of toe-off (% GC)	59.4 ± 2.8	61.2 ± 3.2	**0.02***	60.2 ± 3.2	63.0 ± 4.2	**<0.01***
PF at toe-off (°)	−7.9 ± 3.8	−4.5 ± 6.4	0.09	−11.0 ± 4.3	−9.9 ± 5.3	0.42
PF vel @ toe-off (°/s)	−149.5 ± 62.4	−84.5 ± 45.6	**<0.01***	−120.6 ± 79.3	−110.9 ± 53.5	0.55
PF vel between midstance & PKF (°/sec)	−175.2 ± 37.6	−113.7 ± 41.8	**<0.01***	−177.9 ± 31.8	−152 ± 9±39.0	**0.03***
% GC of max PF vel (°/sec)	57.9 ± 1.8	57.8 ± 3.4	**0.93**	57.3 ± 2.0	59.3 ± 5.6	0.25
Peak PF (°)	−13.7 ± 4.8	−9.9 ± 5.3	**0.04***	−14.7 ± 5.2	−14.0 ± 6.0	0.46
Ankle range of motion (°)	24.4 ± 4.1	20.3 ± 3.7	**<0.01***	25.2 ± 4.2	24.1 ± 5.2	0.28

Ext: extension; flex: flexion; GC: gait cycle; max: maximum; PF: plantar flexion; PKF: peak knee flexion; s: second; vel: velocity; (−): plantar flexion direction. Values for uninvolved side are in italic type. Significant p values are bolded.
*Statistically significant between pre-stretch and post-stretch conditions (p < 0.05).

(3.4 ± 0.2°/fr) to 234.42°/s (2.9 ± 0.4°/fr) (mean difference: 21.6%; p = 0.0004) (Table 1). Compared to Pre-S condition, the 4 selected SKG parameters decreased significantly in Post-S condition: mean difference in percentage was 20% for PKF (10 participants: <45.5°), 26.7% for range of KF between toe-off and mid-swing (time of PKF) (6 participants: <15.2°), 18.9% for total range of KF (13 participants: <52°), and 16.3% for toe-off to PKF time difference (1 participant: <7.6 fr) during swing phase (Table 1, Fig. 2b). Peak PF angle and PF velocity between mid-stance and mid-swing as well as total PF range of motion showed significant decrease in Post-S condition (p = 0.009, p = 006, and p = 0.044, respectively) (Table 1). For uninvolved side, step length was less and stance time was significantly higher in Post-S condition than in Pre-S condition. Additionally, in Post-S condition, peak hip extension (p = 0.08) decreased and PF increased (p = 0.006) in uninvolved side relative to involved side (Table 1). No significant difference was found in other kinematic

parameters on uninvolved side (Fig. 2a, b, c). Peak hip and KF velocities were also found to be slower in uninvolved side (Table 1).

Sagittal plane kinetic alterations

H3 hip power generation in pre-swing decreased significantly in Post-S condition (0.38 ± 0.16 wt/kg to 0.14 ± 0.04 wt/kg; p = 0.02). In Post-S condition, knee extension moment was reduced (from 0.11 ± 0.17 Nm/kg to 0.02 ± 0.25 Nm/kg; p = 0.02) in late stance relative to Pre-S condition (Fig. 2e, Table 2). Knee power generation in terminal stance and absorption in pre-swing (K3) phases did not change significantly (from 0.49 ± 0.23 wt/kg to 0.65 ± 0.31 wt/kg; p = 0.06 and from 0.822 ± 0.57 to −0.29 ± 0.32; p = 0.09, respectively) (Fig. 2j). Ankle power generation dropped from 2.27 ± 0.48 wt/kg to 1.61 ± 0.58 wt/kg; p = 0.014 in Post-S condition (Fig. 2k, Table 2).
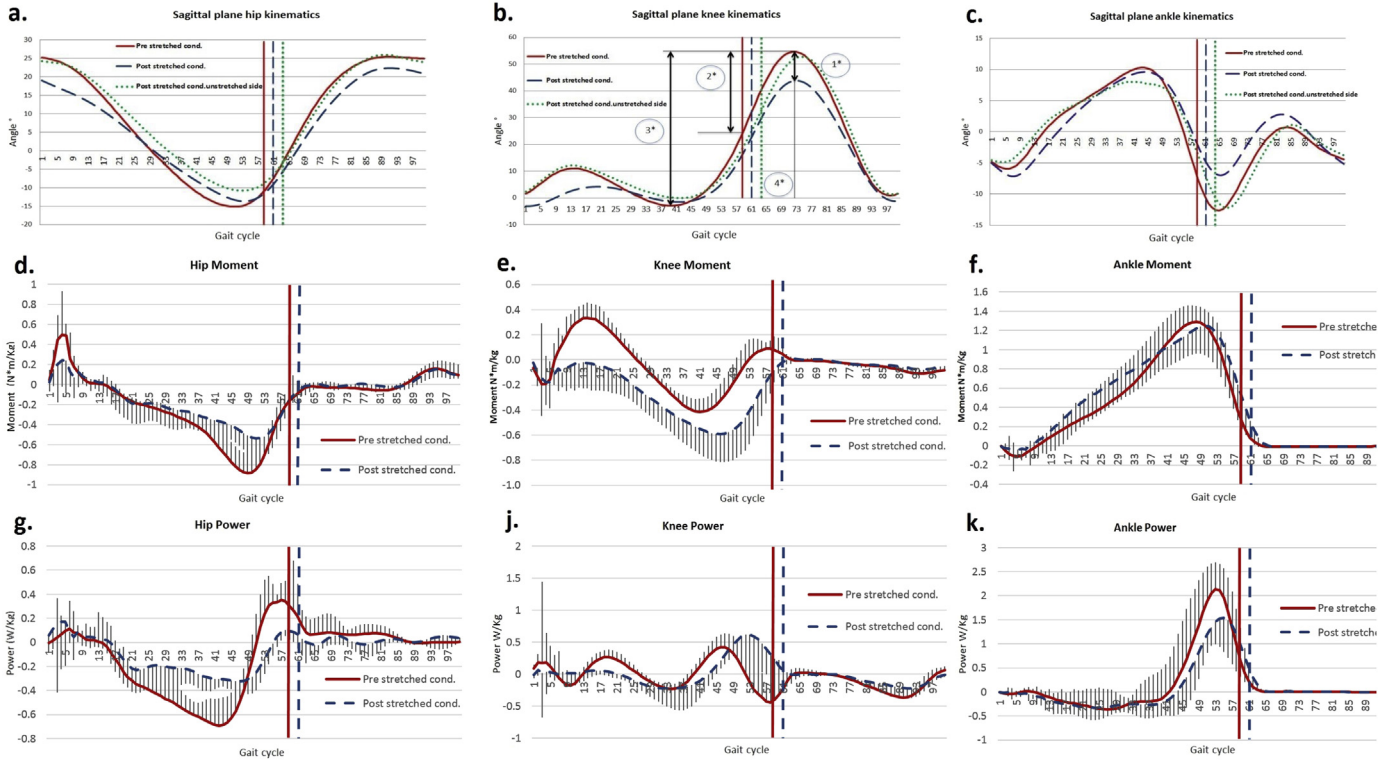
Fig. 2. Averaged values of all participants' (a) hip, (b) knee, and (c) ankle kinematics in pre-stretch (solid line), post-stretch involved side (dashed line), and post-stretch uninvolved side (dotted line). Averaged moments of (d) hip, (e) knee, and (f) ankle, and averaged power of (g) hip (j) knee, and (k) ankle. Four averaged stiff-knee gait parameters: 1) peak knee flexion angle, 2) range of knee flexion between toe-off and peak knee flexion, 3) total knee range of motion in sagittal plane, and 4) duration of time between toe-off and peak knee flexion. All 4 stiff-knee gait parameters decreased in post-stretched condition (dashed line) compared with pre-stretched condition (solid line). (*) Indicates statistically significant different parameter (b).

Table 2
Kinetic gait parameters of interest.

Kinetic parameters	Involved side (stretch & weight) (mean ± SD)		
	Pre-stretch	Post-stretch + weight	P-value
H3 hip power (wt/kg)	0.38 ± 0.16	0.14 ± 0.04	**0.02***
Hip flex mom in pre-swing (Nm/kg)	−0.93 ± 0.15	−0.56 ± 0.18	**<0.01***
Knee power gen. in late stance (wt/kg)	0.49 ± 0.23	0.65 ± 0.31	0.06
Knee ext mom. in late-stance (Nm/Kg)	0.11 ± 0.17	0.02 ± 0.25	**0.02***
A2 peak ankle power gen. (wt/kg)	2.27 ± 0.48	1.61 ± 0.58	**0.01***
Peak ankle PF mom (Nm/kg)	1.33 ± 0.17	1.3 ± 0.09	0.82

Ext: extension; flex: flexion; kg: kilograms; gen: generation; mom: moment; Nm: Newton meter; PF: plantar flexion; PKF: peak knee flexion; s: seconds; wt: weight.
Significant p values are bolded.
*Statistically significant between pre-stretch and post-stretch conditions ($p < 0.05$).

Temporal-spatial alterations

In Post-S condition, mean walking velocity (m/s) and cadence decreased noticeably (24.6% and 10.9%, respectively). Stance time, stride time, and time of toe-off were 15.04%, 10.35%, and 3%, respectively, longer in Post-S condition than in Pre-S condition (Table 3). Comparison of both sides after stretching demonstrated that all joint velocities ($p < 0.001$) and P1 ($p = <0.001$), P2 ($p = <0.001$), and P3 ($p = 0.02$) parameters were lower in involved side (Table 3).

Table 3
Temporal-spatial gait parameters of interest.

Interested parameters	Involved side (stretch & weight) (mean ± SD)			Uninvolved side (no stretch or weight) (mean ± SD)		
	Pre-stretch	Post stretch + weight	P-value	Pre	Post	P-value
Mean velocity (m/s)	1.2 ± 0.1	0.9 ± 0.2	**<0.01***	–	–	–
Cadence (step/min)	105.6 ± 7.7	94.1 ± 8.4	**0.01***	–	–	–
Stance time (m/s)	679.7 ± 75.7	781.3 ± 93.6	**0.02***	*692 ± 80.1*	*818.0 ± 120.7*	***0.02****
Stride time (m/s)	1139 ± 80.3	1257 ± 33	**0.04***	–	–	–
Step length (mm)	667.1 ± 64.2	565.6 ± 99.0	**<0.01***	*679.1 ± 47.9*	*593.2 ± 73.3*	***<0.01****

Flex: flexion; GC: gait cycle; m: meters; mm: millimeters; s: seconds. Values for uninvolved side are demonstrated in italic type.
*Statistically significant between pre-stretch and post-stretch ($p < 0.05$). Significant p values are bolded.

Gait velocity reduction was only moderately correlated with 2 of the 4 SKG parameters (PKF: r = 0.56, p = 0.03; knee range: r = 0.57, p = 0.02) and had very weak correlation with P3 and P4 (Table 2). However, difference in maximum hip flexion velocity was found to be highly correlated with maximum KF velocity (r = 0.86, p < 0.0001), which is highly correlated with 3 of the SKG parameters, also highly correlated with PKF (P1: r = 0.72; p = 0.03) and total knee range (P3: r = 0.73; p = 0.002) (Fig. 3).

In order to determine associations between SKG parameters, Pearson correlations were calculated (Table 4). Maximum KF velocity was found to be very highly correlated with knee range (r = 0.93; p < 0.0001), highly correlated with PKF (r = 0.73; p < 0.0001) and moderately correlated with range in early swing (r = 0.58; p = 0.02). Maximum knee velocity was highly correlated with maximum hip velocity (r = 0.86; p < 0.0001). Comparison of findings in Post-S condition with individuals walking slowly showed that all 4 averaged SKG parameters in post-S condition were different (P1: 58.34°±7.4°; P2: 10.04°±8.7°; P4: 6.5 ± 3.4 fr, p < 0.001; P3: 54.68°±4.6°, p = 0.04).

Discussion

This study not only describes the biomechanical consequences of hip flexor muscle weakness, but also tested 1 of the factors that can cause slower KF during pre-swing. Hip flexor weakness decreased hip flexion velocity as well as KF velocity, resulting in SKG pattern that met 3 of the 4 SKG parameters.[2]

Once hip flexion velocity was reduced, present study results indicated that PKF and total knee range were reduced by 20% (55°−44°) and 19% (58°−47°), respectively (Table 1). Hip velocity reduction of 27% caused 22% decrease in KF velocity. However, in Goldberg et al.'s work, mean PKF decreased by about 27% (from 66° to 48°), and total knee range was reduced by 50% (60°−30°) relative to age-matched control CP children with SKG pattern.[2] In addition, though participants in the present study had no neuromuscular problem, rectus femoris spasticity, or positive score on Ely test, when only considering gait analysis results,

Table 4
Correlations of gait parameters of interest and stiff knee gait parameters.

Parameters	Mean velocity (m/s) r (95% CI) p		Max knee flex vel in GC (°/s) r (95% CI) p		Max hip flex vel. in (°/s) r (95% CI) p	
P1-PKF (°)	0.56*	0.03	0.87**	0.00	0.72*	0.00
P2-Range in early swing (°)	0.25	0.24	**0.59***	0.02	0.40	0.14
P3-Total range (°)	**0.57***	0.02	**0.90****	0.00	**0.73****	0.00
P4-Duration in early swing (% GC)	0.18	0.52	0.23	0.42	0.24	0.38

CI: confidence interval; ext: extension; flex: flexion; GC: gait cycle; m: meters; max: maximum; PF: plantar flexion; PKF: peak knee flexion; s: seconds; vel: velocity. Significant p values are bolded.
*Significant result (p < 0.05), **Highly significant result (p < 0.01).

some participants could be considered candidates for orthopedic surgery, according to Goldberg et al (11 participants had <48° PKF, 2 had <30° total knee range, 4 had <4° KF range in early swing).[2]

Unlike children with CP, in the present study, PKF and total knee range decreased without enhancing knee extensor moment during pre-swing[1,4,7,11] in Post-S condition (Fig. 2e), which is commonly thought to occur due to increased rectus femoris activity. Instead, KF moment increased and hip flexion moment decreased (Table 2). These alterations may be due to decreased gait velocity[25] or compensation for hip flexor muscle weakness, which reduces peak KF velocity in pre-swing.[2,5,11] Additionally, peak PF angle at time of toe-off (from −13.71° to −9.92°), maximum PF velocity in gait cycle (GC) and peak PF velocity (from −175.2°/s to −113.7°/s), total ankle range (from 24.4° to 20.3°), and power generation in pre-swing (Fig. 2k, Table 2), the parameter reported as slowing KF velocity in swing,[2,3,5] unexpectedly declined. Although PF was not the focus of the stretching or loading protocols in the present study, it was not observed to compensate for the relatively weakened iliopsoas as 1 of the 2 energy generators of gait.[5] These changes in ankle movement may be due to the 5% BW or as result of reduced walking velocity, but surprisingly, were very similar to problems seen in the
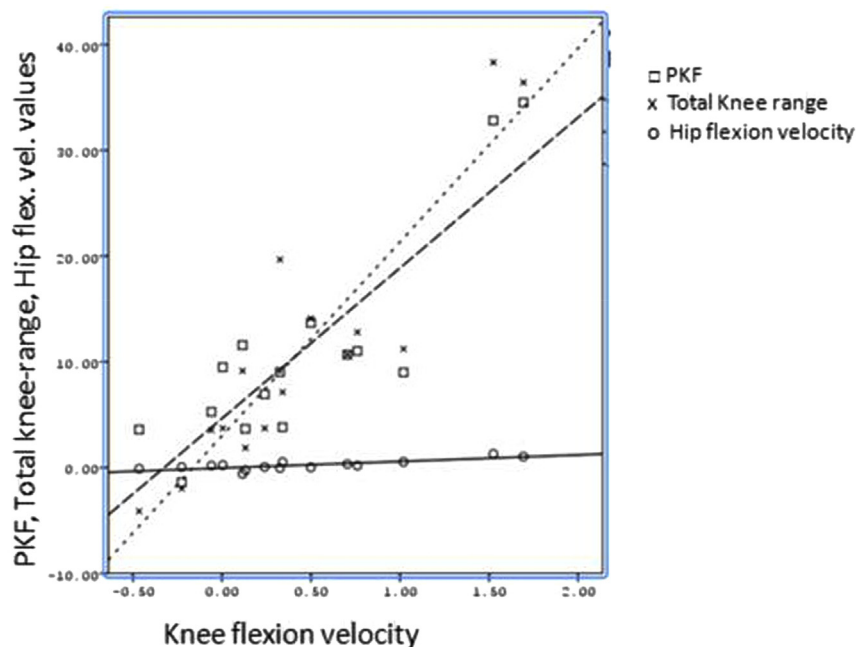

Fig. 3. Correlation of knee flexion velocity difference with difference of peak knee flexion (PKF) angle (PKF "□" dashed line), total knee flexion range ("x" dotted line) and hip velocity ("o" solid line) between pre-stretch and post-stretch conditions. Flex: flexion; vel: velocity.

clinic in patients with neurological injuries.[2,6] Compensation mechanism would be different for CP patients because of the nature of the disease, although this study demonstrated that SKG may be seen without increased knee extension moment in pre-swing for patients without neurological problem. EMG activity of hamstrings, psoas, rectus femoris, gastrocnemius, and soleus were not recorded in this study, and it is suggested for future studies.

In the present study, there was no difference in timing of PKF between the 2 conditions (72.46 vs 72.13 of GC%), which was one of the SKG parameters (P4). It has been reported as delayed in children with CP.[6] Delayed PKF may be related to the nature of CP, which includes co-spasticity around joints and motor control problems.

Average gait velocity (1.20 ± 0.15 cm/s) and KF in Pre-S condition were similar to those of control groups reported in the literature (340 ± 60°/s).[2] In Post-S condition, gait velocity dropped, which could be considered the reason for changes in some SKG parameters such as reduced PKF and delayed time of PKF.[25] However, instead of gait velocity reduction, it was found that KF velocity was the primary factor for most of the SKG parameters. Additionally, age-matched, slowly walking, healthy individuals had significantly different SKG parameters than Post-S condition, which clearly indicates that reduced walking speed did not influence SKG parameters. Therefore, it was obvious that majority of changes to SKG parameters are significantly associated with reduced KF velocity and hip velocity.

For the uninvolved side, increased PF (from −9.9° to −14.0°) might be compensation for reduced swing leg velocity (Table 1).[1−7] Reduced peak hip extension on uninvolved side might be the reason for reduced swing duration, and therefore reduced step length on the involved side (Fig. 2b and c). Moreover, although all joint velocities were lower after stretching in uninvolved side, the differences between both limbs were very high and sufficient to generate SKG pattern after stretching (Table 1).

Recovery in parameters of interest was checked by recording gait trials between 1 and 2 min and between 3 and 4 min after first Post-S trial (Fig. 4). Maximum hip flexion velocity (p = 0.08), mean gait velocity (p = 0.86), and 2 of the 4 SKG parameters (P2, P4) were not significantly different (p = 0.49, 0.92 respectively) compared to Pre-S condition, and significance level of difference dropped for the other 2 SKG parameters (PKF: p = 0.08; range: p = 0.01) after 3−4 min. These findings demonstrate that the effect of PS and weight caused temporal muscle weakness.

Fowles et al noted that maximum muscle force drops after 30 min of prolonged PF muscle stretch.[22] Loss of force generation might be partly due to changes in length−tension relationship or plastic deformation of connective tissue, which were not related to absolute muscle weakness.[22] Marek et al revealed that the same motor activation and peak torque were reduced by static stretching for vastus lateralis and rectus femoris muscles as well as for PF.[26]

Limitations of the study

Recording EMG activity of PF, rectus femoris, and iliopsoas might help identify reasons for biomechanical alterations during walking. Additionally, due to quick recovery period after stretching protocol, obtaining kinetic data of stretched side within a minute was the main target of the study. Therefore, clean force plate kinetic data of unstretched side were limited in Post-S and kinetic alterations of unstretched side could not be demonstrated.

Our stretching method could also affect other hip flexor muscles of iliopsoas such as sartorius. Relationship between iliopsoas weakness and SKG should also be confirmed in future studies for individuals with CP and stroke. Stretching done by the same researcher might have increased reliability of the present study although, for future studies, it may lead bias if randomized control studies are designed.

In contrast to Fowles et al,[22] by placing the 5% BW on 1 leg after same side iliopsoas stretching protocol, it was possible to reduce hip power generation in pre-swing (p = 0.0261) and additionally, to mimic a relatively weak iliopsoas muscle that had to work with a heavier thigh, as observed in neurologically affected people. Cause of alterations may related to changing center mass of thigh causing pendulum effect in walk, which may require more muscle force by changing natural frequency of swinging limb.
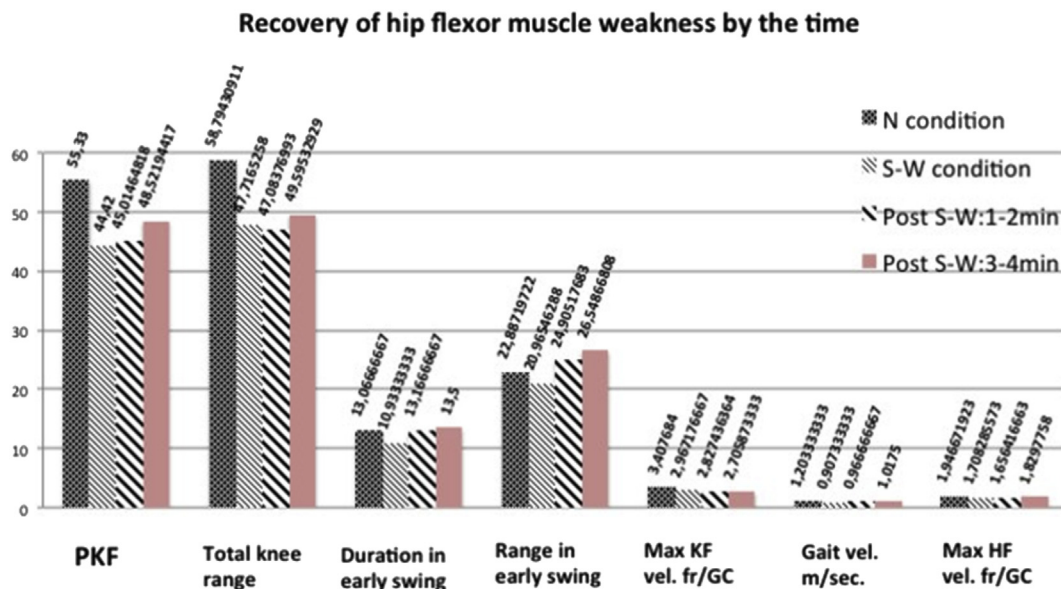

Fig. 4. Comparison of pre-stretch and post-stretch. Post-stretch: 1−2 min (walking trial between 1 and 2 min after post-stretch condition), post-stretch: 3−4 min (walking trial between 3 and 4 min after post-stretch condition). Peak knee flexion (PKF), total knee range, duration in early swing, maximum knee flexion velocity (max KF vel), maximum hip flexion velocity (max HF vel fr/GC), and gait velocity (gait vel m/sec) approached pre-stretch levels over time. Fr: frame, GC: gait cycle, m: meters, N: pre-stretch; S−W: stretch + weight; sec: seconds.

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Muscle strength refers to the amount of force that can be generated, whereas power generation, which is highly correlated with walking performance, determined not only amount of force, but also speed at which force is applied. In the literature, when measuring muscle strength of PF, such as during MMT, dynamometric measurements have been found to be only moderately correlated with power generation during walking and weakly correlated with mobility performance.[27] Khan and Williams declared that strength testing of primary muscles responsible for forward propulsion (hip flexors, PF, etc.) should be targeted for power generation during walking rather than static testing techniques like MMT or hand-held dynamometry.[27] Therefore, MMT scores were verified with H3 hip power generation in the present study. Moreover, Cuthbert and Goodheart[28] reviewed the validity and reliability of using MMT in clinics and found that there was evidence for good reliability and validity. Observational cohort studies have demonstrated good external and internal validity, and 12 randomized controlled trials revealed that MMT findings do not depend on examiner bias.[29] However, MMT may be thought of as limiting the reliability of the present study; thus, for future studies, hip flexor muscle force should be measured using a manual dynamometer or isokinetic measuring tools and correlated with gait performance.

Conclusion

The present study confirmed that any treatment protocol that weakens iliopsoas muscle force or reduces hip flexion velocity might have the potential to cause SKG in healthy individuals. The effect of iliopsoas weakness may be different in children with CP or stroke survivors. In fact, it might have more debilitating influence, which should be investigated.

Conflicts of interest

None declared.

Acknowledgements

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