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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 × 13 repetitions × 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 chemodenervation 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

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