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The effect of plantar flexor muscle fatigue on postural control

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ABSTRACT

Objective: Previous studies have demonstrated that ankle muscle fatigue alters postural sway. Our aim was to better understand postural control mechanisms during upright stance following plantar flexor fatigue.
Method: Ten healthy young volunteers, 25.7 ± 2.2 years old, were recruited. Foot center-of-pressure (CoP) displacement data were collected during narrow base upright stance and eyes closed (i.e. blindfolded) conditions. Subjects were instructed to stand upright and as still as possible on a force platform under five test conditions: (1) non-fatigue standing on firm surface; (2) non-fatigue standing on foam; (3) ankle plantar flexor fatigue, standing on firm surface; (4) ankle plantar flexor fatigue, standing on foam; and (5) upper limb fatigue, standing on firm surface. An average of the ten 30-s trials in each of five test conditions was calculated to assess the mean differences between the trials. Traditional measures of postural stability and stabilogram-diffusion analysis (SDA) parameters were analyzed.
Results: Traditional center of pressure parameters were affected by plantar flexor fatigue, especially in the AP direction. For the SDA parameters, plantar flexor fatigue caused significantly higher short-term diffusion coefficients, and critical displacement in both mediolateral (ML) and anteroposterior (AP) directions. Long-term postural sway was different only in the AP direction.
Conclusions: Localized plantar flexor fatigue caused impairment to postural control mainly in the Sagittal plane. The findings indicate that postural corrections, on average, occurred at a higher threshold of sway during plantar flexor fatigue compared to non-fatigue conditions.

1. Introduction

Maintenance of postural control requires coordinated interaction of various sensory components such as visual, vestibular, cutaneous, musculotendinous receptors, muscle spindles and Golgi tendon organs (Massion, 1994; Maurer et al., 2000). Among the sensory inputs relevant to postural control, muscle proprioception has been demonstrated to play a dominant role (Kavounoudias et al., 1999; Gurfinkel et al., 1995; Di Fabio et al., 1990). Lower limb muscle fatigue is proposed to be responsible for most ankle joint athletic injuries since many of these injuries occur at the end of an activity when the athlete is fatigued (Simoneau et al., 2005). It is theorized that lower limb muscle fatigue may impair the proprioceptive and kinesthetic properties of joints by increasing the threshold of muscle spindle discharge, disrupting afferent feedback, and subsequently alter conscious joint awareness (Gribble and Hertel, 2004a,b); this could be due to the inability of the stabilizing musculature to produce or sustain the required muscle tone under fatigued conditions, leading to decreased functionality of the proprioceptive muscle receptor system (Forestier et al., 2002; Johnston et al., 1998). Therefore, altered somatosensory input due to muscle fatigue could result in deficits in neuromuscular and postural control (Gribble and Hertel, 2004a,b).

One way of quantifying an aspect of neuromuscular control is through measures of postural sway. An increase in postural sway might indicate impairment of postural control resulting in functional postural instability (Tropp et al., 1984). Lundin et al. (1993) found that plantar- and dorsi-flexor fatigue induced through isokinetic exercises caused significant increase in mediolateral sway (ML-sway) in young adults. Yaggie and McGregor (2002) found increased ML- and AP-sway during the forward lean test after plantar, dorsi flexor, and invertor-evertor muscle fatigue. Muscle fatigue induced through isokinetic exercise of the ankle, knee, and hip increased postural sway (Johnston et al., 1998; Gribble and Hertel, 2004a,b). However, Adlerton and Moritz (1996) found no increase in body sway in one leg stance after fatiguing exercises of the calf muscles, and Gribble and Hertel (2004a,b) found that ankle muscle fatigue did not led to postural control impairment.

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The research studies introduced above utilized CoP-based summary statistics as measures of balance function. Unfortunately, these measures do not provide a specific understanding of underlying postural control mechanisms related to ankle muscle fatigue. In the present study, stabilogram-diffusion analysis (SDA) was used (Collins and De Luca, 1995; Wolff et al., 1998) to explore the underlying mechanism of reduced postural control under fatigued conditions. Stabilogram-diffusion analysis provides a number of parameters that have a physiologically meaningful interpretation. Among these are diffusion coefficients obtained by plotting the mean squared CoP displacement as a function of the time interval. This plot demonstrates two distinct regions: a short-term region and a long-term region. The short-term (*Drs*) and long term (*Drl*) diffusion coefficients, which characterize the effective stochastic activity of open-loop and closed loop postural control mechanisms, respectively, are derived from the slopes of the short-term and long-term regions of this plot. It has been suggested that in the short-term region, the postural control system operates in an open-loop mode and does not directly rely on sensory information over short time intervals, whereas the long-term region reflects closed-loop control mechanisms the human postural control system operate with sensory feedback (Collins and De Luca, 1993, 1995). The transition between open-loop and closed-loop control has been termed the critical point, the coordinates of which reflect the average time interval (critical time, *Ctx*) and sway displacement (critical displacement, *Cdx*) where closed-loop control begins to dominate sway behavior. The SDA method has been adopted by a number of research groups who have shown that SDA parameters are sensitive to the effects of age (Rougier and Farenc, 2000), vision (Wolff et al., 1998; Mitchell et al., 1995), Parkinson disease (Laughton et al., 2003), balance impaired older adults (Melzer et al., 2010), and reduced cutaneous sensation (Winter et al., 1996; Meyer et al., 2004). Interpretation of the SDA may offer more insight into the nature of the process controlling the CoP trajectories and may be more sensitive than the traditional CoP measures. We believe that under calf muscle fatigue, which is known to interfere with stability, SDA will reveal deficiencies in balance control. We hypothesize that under calf muscle fatigue, subjects (1) will show an increase in short term postural sway, before closed-loop feedback mechanisms are called into play, and (2) stabilogram-diffusion analysis (SDA) parameters would be different for anteroposterior (AP) directions but not mediolateral (ML).

2. Methods

2.1. Participants

A convenience sample of 10 healthy young volunteers (7 female and 3 male subjects), 25.7 ± 2.2 years old, were recruited. The exclusion criteria were: history of lower-extremity injury or neurologic deficits. Participants provided informed consent, in accordance with approved procedures by the Helsinki ethics committee in Soroka University Medical Center, Beer-Sheva, Israel.

2.2. Experimental protocols

2.2.1. Postural stability protocol

During the postural stability test, subjects stood upright and barefoot in a narrow base stance (heels and toes touching), with their eyes closed and covered (i.e. wearing blindfolds), and their arms placed at the sides of their body, no arm movements were allowed during the trial. The subjects were instructed to stand as still and stable as possible on a force platform. A total of ten 30-s trials were conducted for each of the five test conditions (50 postural stability trials for each subject): (1) non-fatigue, upright standing on force plate, this condition served as a control session; (2) non-fatigue, upright standing on foam; (3) plantar flexor fatigue, upright standing on force plate following fatigue exercises; (4) plantar flexor fatigue, standing on foam following fatigue exercises; and (5) upper limb fatigue, this condition served as a control session to the plantar flexor fatigue conditions to find whether local muscle fatigue or general fatigue resulted any postural sway changes.

The center of pressure (CoP) and ground reaction force data during quiet-standing were collected with a Kistler 9287 force platform (Kistler Instrument Corp., Winterthur, Switzerland). The force platform data were sampled at a frequency of 100 Hz and stored on hard disk for later processing. Force platform data were analyzed using code written in Matlab (Math Works Inc., Cambridge, MA, USA) to extract traditional postural sway parameters: (1) Mediolateral CoP range (mm) (ML-sway range); (2) Anteroposterior CoP range (mm) (AP-sway range); (3) mean velocity of CoP sway (mm/s); and (4) sway area (mm^2) – the elliptical area of the CoP points. These parameters were computed for each 30 s trial, and then averaged for each set of 10 trials to obtain an average value for each parameter and for each subject, in each experimental condition. In addition, the stabilogram-diffusion analysis (SDA) as described by Collins and De Luca (1993, 1995), was performed on the CoP trajectories using MatLab code (Math Works Inc., Cambridge, MA, USA). The current analysis was focused on the short-term (*Dxs*, *Dys*) and long-term (*Dxl*, *Dyl*) diffusion coefficients as well as critical time (*Ctx*, *Cty*) and critical displacement (*Cdx*, *Cdy*), which reflect effective stochastic activity of open-loop and closed-loop postural control mechanisms in the ML and AP directions. These parameters are derived from the slopes of the short-term and long-term regions of the linear stabilogram-diffusion plot (Collins and De Luca, 1993, 1995).

2.2.2. Fatigue protocols

During the plantar flexors fatigue protocol the subjects stood upright and barefoot and were instructed to conduct as many concentric-eccentric ankle plantar flexor contractions as possible until exhaustion (e.g. the subjects continued until they insisted on stopping, ranged from 18 to 26 contractions/30 s). Verbal encouragement was given to ensure maximal effort.

The upper limb fatigue was achieved by performing push-ups until subjects insisted on stopping. The upper limb fatigue protocol involved multiple joints and many muscle groups in the upper limbs and served as a control task for the localized plantar flexor fatigue conditions. During the trials subjects were asked to report the level of fatigue at the end of the concentric-eccentric ankle plantar flexor contractions using the Borg visual analog scale (Borg, 1973). In addition, during the fatigue trials, heart rate was monitored by a Polar watch (model AXN 500), to explore differences between fatigue and non-fatigue conditions and between plantar flexor and upper limb fatigue.

To ensure that balance measurements during fatigue were obtained in a real fatigued state, despite a presumable short recovery time of the calf muscles (Johnston et al., 1998), various rules were respected, as previously described in detail elsewhere (Vuillerme et al., 2001). The fatiguing exercise took place next to the force platform to minimize the time-interval between the fatiguing exercise and the CoP measurements; postural control was assessed for 30 s, immediately after the fatigue exercises; the fatiguing exercise was repeated prior to each postural test; to ensure that fatigue was reached during the Plantar flexors fatigue protocol, surface electromyographic signals (sEMG) were recorded from the first two subjects' Lateral Gastrocnemius and Soleus muscles of the dominant leg during each of the 30 s postural sway trials. Surface EMG electrodes (Delsys, Inc., Boston, Massachusetts; DE 2.1 sin-

gle-differential, parallel-bar configuration, Contact Material 99.9% Ag, contact Spacing 10. 0 mm, with detection area of 10 mm^2) were attached to the mid-bellies of the soleus and Gastrocenemius muscles bellies, with adhesive skin interfaces as described by Basmajian and De-Luca (1985). The sEMG was recorded continuously on a portable data logger (Delsys Ltd., Boston, MA, USA). The EMG raw signals were sampled at 2048 Hz, and filtered using a second order zero phase-lag Chebyshev Type II with a low-pass cut-off frequency at 10 Hz and a high-pass cut-off at 400 Hz, using a roll-off of 12 dB/oct. The signals were stored for later processing with data acquisition and analysis software (EMGworks, Delsys, Inc.). Fast Fourier Transform was applied to assess the frequency spectrum and mean median power (MMP) frequency of the EMG signal (Stulen and De Luca, 1982), using code written in MatLab (Math Works Inc., Cambridge, MA, USA).

2.2.3. Sample size estimation

Sample size of 10 subjects was determined by using the CoP sway velocity, where the CoP sway velocity reflects unsteadiness in balance control between fatigue and non-fatigue conditions (Vuillerme et al., 2002). We found that 10 subjects would be required to detect a two-sided difference in COP sway velocity between fatigue (15 ± 5.0 mm/s) and non-fatigue conditions (10 ± 5.0 mm/s). A significance level of 0.05 and 80% power was chosen for a clinically meaningful estimate.

2.3. Statistical analysis

To compare means for each dependent variable (the traditional postural sway parameters and the SDA parameters) between five independent trial conditions, we used separate one-way ANOVA for each dependent variable, followed by Post-hoc Tukey comparisons to see exactly which pairs of trial conditions are significantly different. Additional one-way ANOVA followed by Post-hoc Tukey comparison were performed to explore difference in mean heart rate and mean Borg fatigue scores between the five independent trial conditions. Statistical significance for all tests was accepted at $p < 0.05$. All data were analyzed using SPSS 15 software (SPSS Inc., Chicago, IL, USA).

3. Results

A total of 500 postural stability trials from 10 subjects aged 25.7 ± 2.2 years old were assessed and analyzed (50 trials from each subject). The heart rate, Borg scale, and sEMG characteristics of the participants (described in Figs. 1–3) were measured to explore whether subjects achieved fatigue during the performance fatigue exercises.

Heart rate (HR) results are presented in Fig. 1. The mean HR for the non-fatigue conditions was significantly lower compared to both the plantar flexor fatigue condition and the upper limb fatigue condition (89 ± 16 beats per minute (bpm) vs. 109 ± 15 bpm, $p = 0.01$; and 89 ± 16 bpm vs. 110 ± 16 bpm, $p = 0.009$, respectively). Heart rate for the plantar flexor fatigue task was not significantly different from the upper limb fatigue (109 ± 15 vs. 110 ± 16, $p = 0.757$).

The Borg scores were monitored to compare subjects' level of exertion for the different test conditions. Results are presented in Fig. 2. The mean Borg score for the non-fatigue conditions (0.46 ± 0.63) was significantly lower than that for plantar flexor fatigue (5.36 ± 2.11, $p < 0.001$) and the upper limb fatigue (6.47 ± 1.58, $p < 0.001$). Borg score during the plantar flexor fatigue task was not significantly different than during upper limb fatigue ($p = 0.163$).

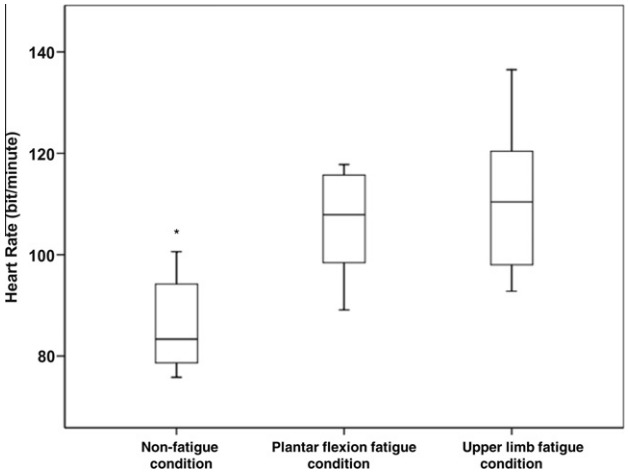

Fig. 1. Comparison of subjects' heart rates in the different tasks, presented as average HR, range of HR, and standard deviation. $^*p < 0.05$ of non-fatigue vs. plantar flexor fatigue vs. upper limb fatigue.

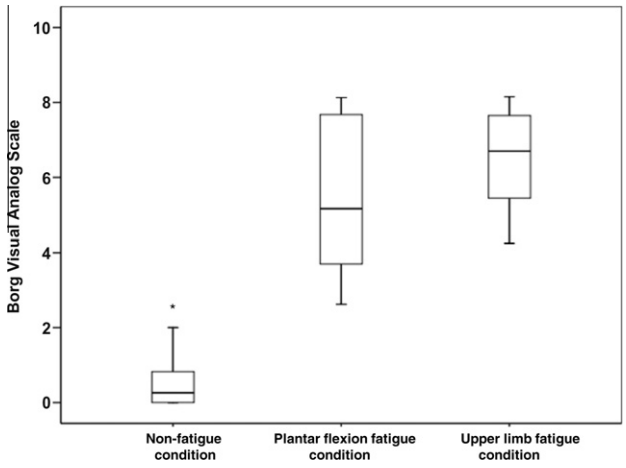

Fig. 2. Comparison of subjects' effort estimations according to a Borg scale in the different tasks presented as average Borg score and standard deviation. $^*p < 0.05$ of non-fatigue vs. plantar flexor fatigue vs. upper limb fatigue.

Fig. 3 shows the mean median frequency (MMF) of the spectral EMG signal of the two muscles for two subjects during the postural stability trials while standing on the force plate. Test number one in Fig. 3 shows the MMF value before the fatigue protocol and test numbers 2–11 shows MMF during 10 postural stability trials after fatigue. A linear regression between MMF values over time for each of the measured muscles provided R^2 values ranging from 0.48 to 0.83 ($p < 0.05$). The MMF during the non-fatigue trial in subject #1 was 100.4 Hz, 10% higher than during the average MMF during the fatigue trials (90.4 Hz); similarly, MMF in subject #2 was 130.4 Hz during the non-fatigue trial and 112.9 Hz during the fatigue trials. The Soleus muscle displayed a decrease in the average MMF during the fatigue trials compared to the non-fatigue trial (36 Hz vs. 24.8 Hz in Subject #1 and 122.8 Hz vs. 102.6 Hz in Subject #2). Decreases in MMF during sustained contractions are typically considered to be a sign of muscle fatigue.

3.2. The effect of plantar flexor fatigue on postural control

3.2.1. Sway parameters summary statistics

Summary statistic parameters are shown in Table 1. There was no significant increase in ML-sway range during Plantar flexor fatigue trials compared to non-fatigue trials and between plantar

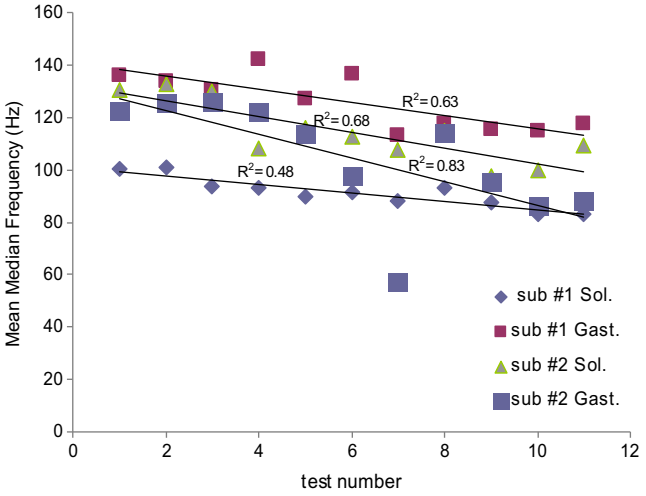

Fig. 3. Mean median frequency (MMF) of the lateral Gastrocnemius and Soleus, measured during the non-fatigue trial (test #1) and each of the ten 30 s postural sway trials (tests #2 to #11) in 2 subjects. Mean Median frequency (MMF) slope decreases during the fatigue trials (R^2 = 0.48–0.83, $p < 0.05$). This decrease in the MMF is typically considered to be a sign of muscle fatigue.

flexor fatigue trials compared with upper limb fatigue trials (35.5 ± 5.8 vs. 32.9 ± 7.6 mm, $p = 0.11$, and 35.5 ± 5.8 vs. 32.9 ± 7.0 mm, $p = 0.09$, respectively). ml-velocity increased significantly during the plantar flexor fatigue trial compared to non-fatigue trials ($p = 0.01$) but was not significantly different from upper limb fatigue ($p = 0.127$).

There was a significant increase across all AP-sway measures during plantar flexor fatigue. The AP-sway range was greater compared with non-fatigue trials and compared with upper limb fatigue trials (37.3 ± 9.8 mm vs. 28.6 ± 6.5 mm, $p = 0.001$; and 37.3 ± 9.8 mm vs. 29.2 ± 7.7 mm, $p = 0.001$, respectively). Similarly, the AP-velocity showed a significant increase during plantar flexor fatigue compared with non-fatigue trials and upper limb fatigue (18.5 ± 3.1 mm^2/s vs. 16.4 ± 2.1 mm^2/s, $p = 0.014$; and 18.5 ± 3.1 mm^2/s vs. 17.0 ± 1.8 mm^2/s, $p = 0.03$, respectively).

In addition, the sway area was significantly greater during plantar flexor fatigue trials compared with non-fatigue trials and compared with the upper limb fatigue trials (114.2 ± 33.2 mm^2/s vs. 82.9 ± 28.4 mm^2/s, $p = 0.003$; and 114.2 ± 33.2 mm^2/s vs. 87.4 ± 23.04 mm^2/s, $p = 0.001$, respectively). There were no significant differences between the non-fatigue and elbow Extensors fatigue trials.

For the standing on foam trials the results showed significant increases across all traditional sway parameters after the plantar flexor fatigue compared with non-fatigue trials, apart from ML-sway range (Table 2). For example, AP-sway range, AP-sway velocity, and sway area were significantly greater (75.9 ± 14.4 mm vs. 62.8 ± 10.6 mm, $p = 0.004$; 38.8 ± 7.96 mm^2/s vs. 34.2 ± 4.3 mm^2/s, $p = 0.05$; 416.4 ± 114.4 mm^2/s vs. 320.6 ± 69.4 mm^2/s, $p = 0.026$, respectively).

Table 2
The effect of plantar flexor muscle fatigue on postural stability – while standing on thick foam.

	Non-fatigue	Plantar-flexion fatigue	*p*-Value
ML-sway range (mm)	62.8 ± 10.8	66.3 ± 11.7	0.186
AP-sway range (mm)	62.8 ± 10.6	75.9 ± 14.4	0.004
ML-sway velocity (mm^2/s)	34.6 ± 4.8	41.3 ± 7.4	0.009
AP-sway velocity (mm^2/s)	34.2 ± 4.3	38.8 ± 7.96	0.05
Sway area (mm^2)	320.6 ± 69.4	416.4 ± 114.4	0.026

Presented a mean ± SEM.

3.2.2. Stabilogram-diffusion parameters

Plantar flexor fatigue was associated with significantly greater short-term effective diffusion coefficients in the ML direction (*Dxs*) compared to the non-fatigue and Upper limb fatigue conditions ($p = 0.017$ and $p = 0.02$, respectively; Table 3 and Fig. 1). The critical (mean-squared) displacement in the ML direction (*Cdx*) was significantly greater during plantar flexor fatigue compared to upper limb fatigue ($p = 0.047$) and not significant when compared with the non-fatigue condition ($p = 0.1$). The critical time interval (*Ctx*), and the long-term effective diffusion coefficients in the ML direction (*Dxl*) were not statistically different between task conditions. In addition, there were no significant differences between non-fatigue and upper limb fatigue condition.

The SDA parameters in AP direction showed that during plantar flexor fatigue there was a significant increase in short-term effective diffusion coefficient (*Dys*) compared to non-fatigue ($p = 0.02$) and marginally different compared to the upper limb fatigue ($p = 0.1$; Table 3 and Fig. 1). The long-term effective diffusion coefficients in the AP direction (*Dyl*) during plantar flexor fatigue were significantly greater compared with the no-fatigue and compared with upper limb fatigue ($p = 0.017$ and $p = 0.009$, respectively). The critical (mean-squared) displacement (*Cdy*) showed a tendency to differ between the plantar flexor fatigue trials compared with upper limb fatigue and the non-fatigue trial ($p = 0.09$ and $p = 0.1$, respectively). The critical time interval (*Cty*), was significantly different between plantar flexor fatigue compare with no-fatigue ($p = 0.02$) and plantar flexor fatigue compared with upper limb fatigue ($p = 0.04$).

During standing on foam, plantar flexor fatigue was associated with significantly greater short-term effective diffusion coefficients in the ML direction (*Dxs*) when compared with the non-fatigue conditions ($p = 0.05$; Table 4 and Fig. 1), while the long-term effective diffusion coefficients in the ML direction (*Dxl*), were not statistically different between task conditions ($p = 0.9$). Also, the critical (mean-squared) displacement (*Cdx*) was marginally increased after plantar flexor fatigue when compared to non-fatigue trials ($p = 0.08$). The critical time interval (*Ctx*) was not statistically different between task conditions. The SDA parameters in AP direction showed that during the plantar flexor fatigue there was a significant increase in short-term effective diffusion coefficients in the AP direction (*Dys*) when compared with the non-fatigue trials

Table 1
The effect of plantar flexor muscle fatigue on the traditional postural sway parameters – standing on firm surface.

	Non-fatigue	Plantar flexion fatigue	Upper limb fatigue	*p*-Value non-fatigue vs. Plantar flexion fatigue	*p*-Value non-fatigue vs. upper limb fatigue	*p*-Value plantar flexor fatigue vs. upper limb fatigue
ML-sway range (mm)	32.9 ± 7.6	35.5 ± 5.8	32.9 ± 7	0.109	0.998	0.091
AP-sway range (mm)	28.6 ± 6.5	37.3 ± 9.8	29.2 ± 7.7	0.001	0.718	0.001
ML-sway velocity (mm^2/s)	15.6 ± 2.7	18.1 ± 3.6	16.5 ± 1.8	0.011	0.126	0.127
AP-sway velocity (mm^2/s)	16.4 ± 2.1	18.5 ± 3.1	16.96 ± 1.8	0.014	0.299	0.03
Sway area (mm^2)	82.9 ± 28.46	114.2 ± 33.2	87.4 ± 23.04	0.003	0.416	0.11

Results presented as mean ± SEM.

Table 3
The effect of plantar flexors muscle fatigue on stabilogram diffusion analysis parameters – standing on firm surface.

	Non-fatigue	Plantar flexion fatigue	Upper limb fatigue	*p*-Value non-fatigue vs. Plantar flexion fatigue	*p*-Value non-fatigue vs. upper limb fatigue	*p*-Value plantar flexion fatigue vs. upper limb fatigue
Stabilogram-diffusion parameters mediolateral direction						
Short-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dxs*)	39.5 ± 4.7	51.6 ± 6.1	39.2 ± 3.6	0.017	0.8	0.02
Long-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dxl*)	2.2 ± 0.7	2.1 ± 0.3	1.97 ± 0.3	0.7	0.7	0.7
Critical (mean-squared) displacement in mm^2 (*Cdx*)	59.5 ± 6.9	71.4 ± 7.5	60.7 ± 8.5	0.1	0.6	0.047
Critical time intervals in s (*Ctx*)	0.8 ± 0.03	0.78 ± 0.06	0.79 ± 0.05	0.6	0.8	0.8
Stabilogram-Diffusion Parameters anteroposterior direction						
Short-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dys*)	19.9 ± 4.9	31 ± 6.6	20.9 ± 4.9	0.1	0.9	0.02
Long-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dyl*)	2.3 ± 0.7	6.1 ± 1.9	2.7 ± 0.8	0.017	0.6	0.009
Critical (mean-squared) displacement in mm^2 (*Cdy*)	43.3 ± 5.3	51.9 ± 8.3	38.7 ± 4.2	0.1	0.2	0.09
Critical time intervals in s (*Cty*)	0.68 ± 0.01	1.04 ± 0.07	0.69 ± 0.04	0.02	0.8	0.04

Results presented as mean ± SEM.

Table 4
The effect of plantar flexor muscle fatigue on Stabilogram diffusion analysis parameters – Standing on foam.

	Non-fatigue	Plantar flexion fatigue	*p*-Value non-fatigue vs. plantar flexion fatigue
Stabilogram-diffusion parameters mediolateral direction			
Short-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dxs*)	214 ± 21	274.2 ± 32.8	0.05
Long-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dxl*)	1.3 ± 0.5	1.25 ± 0.4	0.9
Critical (mean-squared) displacement in mm^2 (*Cdx*)	269 ± 31	313 ± 36.3	0.08
Critical time intervals in s (*Ctx*)	0.67 ± 0.05	0.62 ± 0.04	0.13
Stabilogram-diffusion parameters anteroposterior direction			
Short-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dys*)	164.5 ± 15.7	243.1 ± 35	0.03
Long-term effective diffusion coefficients in $mm^2 s^{-1}$ (*Dyl*)	0.6 ± 1.2	3.8 ± 1.5	0.07
Critical (mean-squared) displacement in mm^2 (*Cdy*)	254.5 ± 26.5	413.3 ± 45.3	0.004
Critical time intervals in s (*Cty*)	0.82 ± 0.04	0.96 ± 0.06	0.03

Results presented as mean ± SD.

($p = 0.03$), while differences in long-term effective diffusion coefficients in the AP direction (*Dyl*) were marginally significant ($p = 0.07$; Table 4 and Fig. 1). The critical (mean-squared) displacement (*Cdy*) and the critical time interval (*Ctx*) were significantly greater after plantar flexor fatigue compared to non-fatigue ($p = 0.004$ and $p = 0.03$, respectively).

4. Discussion

In the current study, ranges of the CoP displacements were affected by localized plantar flexor fatigue and not by upper limb fatigue, although subjects were fatigued during both fatigue protocols as indicated by the Borg scores, heart rates, and the sEMG results (Figs. 1–3). It may be concluded that postural sway was affected mainly by localized plantar flexor fatigue and that upper limb fatigue (involving upper limb a muscular group) had negligible effects. Previous authors (Corbeil et al., 2003; Gribble and Hertel, 2004a,b; Tropp et al., 1984; Yaggie and McGregor, 2002) have reported similar findings of altered postural control following localized fatigue in the ankle joint muscles.

During plantar flexor fatigue, sway parameters were mainly affected in the AP direction and less in the ML direction. Winter et al. (1996) proposed that the ankle plantar- and dorsi-flexors play a dominating role in control of AP movements, while hip abductors and adductors mainly control ML sway. Our results showed that plantar flexor fatigue caused an increase in short-term stochastic activity in the AP (*Dys*), and ML directions (*Dxs*), as well as an increased critical displacement in the AP and ML directions (*Cdx* and *Cdy*) and critical time in the AP direction (*Cty*). The greater values for *Dxs*, and *Cdx* during plantar flexor fatigue indicate an increase in stochastic activity during short-term intervals when sway tends to drift away from an equilibrium point in an open-loop control mode and a higher sway displacement before closed-loop feedback mechanisms dominate sway behavior, respectively. Since subjects performed the postural tasks with their eyes closed, it is likely that plantar flexor proprioception (muscle spindles and Golgi tendon organs) played an important role in the postural control loop. Consequently, a decrease in performance could be related to fatigue affecting the proprioceptive feedback. This would be consistent with an increased critical displacement (*Cdy*) in combination with increased long-term stochastic activity (*Dyl*, long-term diffusion coefficient) in the AP direction as it would indicate a decrease in sensitivity of the postural control loop and lower quality of control across time intervals when closed-loop control dominates behavior. The increase in long-term stochastic activity (*Dyl*, long-term diffusion coefficient) indicates greater amplitude and/or frequency of sway in the long-term region (dominated by low frequency sway), which would further suggest a less effective closed-loop control of postural sway.

There are additional physiological mechanisms that could explain these findings. Reports have shown impaired muscle spindle sensitivity following prolonged exercise in animals (Nelson and Hutton, 1985; Pedersen et al., 1998), possibly due to the influence of metabolites or/and inflammatory substances (Pedersen et al., 1998) or through the modulation of reflex pathways originating

from small-diameter muscles afferents (Bigland-Ritchie et al., 1986). Sense of position and movement in humans was altered under muscle fatigue (Forestier et al., 2002; Pedersen et al., 1999). Consequently, the ability of the plantar flexor muscles to provide small compensatory contractions in response to input from afferent pathways may be affected by fatigue. Experiments by Walsh et al. (2004) demonstrated that subjects with arm muscle fatigue made systematic errors in forearm position-matching tasks further supporting this notion.

During muscle fatigue, ankle proprioceptive information may be discarded by the postural control system at a central level causing an increased reliance on visual information (Ivanenko et al., 1999, 2000) or haptic cues from the fingers (Lackner et al. 2000). In the current study, such visual and haptic cues were unavailable, which would limit the ability of the system to compensate for the effect of plantar flexor fatigue. This would have forced the system to rely on available sensory information, mainly somatosensory and vestibular, where proprioceptive feedback from fatigued muscles would likely have been distorted and unreliable and thereby caused a decrease in performance as measured in our postural control parameters. Furthermore, an increase in short-term postural sway during quiet standing could result from increased agonist to antagonist co-activation. Laughton et al. (2003) found that short-term postural sway was significantly correlated with ankle muscle co-activity. Greater co-activation may assist in enhancing joint proprioception by increasing the firing rate and recruitment of primary afferents, thereby enhancing the functional behavior of the associated closed-loop postural control mechanism.

An additional factor relates to the peripheral effect of muscle fatigue. The metabolic milieu of the muscle is altered during exercise-induced fatigue and the capacity of the system to provide fast and accurate contractions is diminished. Thus, the neuromuscular system becomes unable to maintain constant and accurate muscle tension. Gribble and Hertel (2004a,b) claimed that compensatory contractions occur in such a fatigued state that results in overcompensations that translate into larger displacements of the CoP. Moreover, Amann (2011) recently discussed that peripheral muscle fatigue may affect the central projection of thin fiber muscle afferents that provide inhibitory feedback to the central nervous system and thereby influence the magnitude of central motor drive during fatigue. The purpose of this feedback-loop would be to regulate and restrict the development peripheral muscle fatigue, thereby help to prevent excessive disturbance of muscle homeostasis and potential harm to the organism (Amann, 2011). This might be a factor affecting changes in postural control parameters seen in the present study.

This study has several limitations. First, sEMG was used to validate the fatigue protocol only for the first two subjects. It might be argued that not all subjects developed muscle fatigue; although we used the Borg scale as a recognized method for measuring subjective estimates of fatigue that has been used widely for that purpose. Second, the data came from a fairly small sample that was drawn from a defined healthy young population. Consequently, these results cannot be generalized to older adults, extremely weak elderly persons or to athletes. Further study should involve larger sample sizes, and different populations. Theoretically, fatigue might be a risk for fall and injury and therefore studying the effect of ankle muscle fatigue in old fallers vs. non-fallers is needed to explore underlying postural control mechanisms in these populations and its relations to falls.

In conclusion, our results showed directional specific effects of plantar flexor fatigue that were evident as changes in various postural sway parameters, mainly in the AP direction. Stabilogram-diffusion analysis indicated effects of fatigue both across short-time intervals where open-loop control dominates behavior as well as over long-term intervals where closed-loop control mechanisms dominate behavior. We discussed several non-exclusive factors related to fatigue of sensory and motor mechanisms that could explain these results. To further clarify these findings, future studies could include measurements of plantar flexor H-reflex and maximal M-wave responses to assess motor-neuron pool excitability under control and fatigued conditions to help tease out whether effects of localized muscle fatigue on postural control are mainly central or peripheral.

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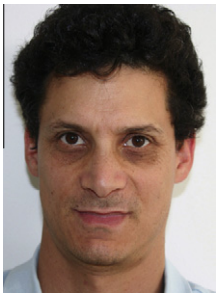

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