Original Research Article: Effect of Plantar Flexor Muscle Fatigue on Postural Control

Tyler Grey, Daren Redguard, Rebecca Wengle, Peter Wegscheider

School of Kinesiology, The University of Western Ontario, London, Ontario, Canada

Abstract

Objective. Previous studies have demonstrated that various factors alter postural stability. Our aim was to examine the effect of plantar flexor fatigue on postural stability in quiet standing.

Methods. Fifteen healthy male university students (age, 21.3 ± 1.7 y; height, 1.83 ± 0.06 m; weight, 81.6 ± 9.4 kg) were instructed to stand on a force plate before and after calf fatiguing exercise. The sensory systems were controlled by blindfolding subjects and having them stand on a flat firm surface, without moving their head. Fatigue was achieved through repetitive weighted plantar-flexor exercise. Standing balance was assessed by using a force plate to calculate Center of Pressure (CoP) displacement.

Results. Plantar flexor fatigue led to significant ($p < 0.05$) postural control impairments in the frontal and sagittal planes compared to non-fatigue (control). Fatigue led to significant changes in M/L (1.66 ± 0.85 mm and 3.30 ± 1.24 mm for control and fatigue, respectively) and A/P (4.48 ± 1.70 mm and 8.89 ± 3.74 for control and fatigue, respectively) CoP variance.

Interpretation. Lower limb fatigue led to significant postural control impairments. Interestingly fatigue in the plantar flexors, primary responsible for control in A/P directions, led to significant postural sway in the M/L directions. Therefore, it is possible that other muscle groups (i.e. hip and knee flexors and extensors) are used to correct posture, as the plantar flexors are not at full functioning capacity. It can be concluded that under a sensory controlled environment, postural control is significantly impaired by lower limb fatigue, and can possibly be supported by other muscle groups.

Introduction

Postural sway can be described as the displacement of the Center of Mass (CoM) in relation to the Base of Support (BoS), and is directly related to postural stability control and balance (1). Postural sway is increased (indicating diminished stability) by many factors, including both muscular fatigue and impaired vision (2–4).

Muscular fatigue is a complex phenomenon and has been defined as a reduction in the force-generating capacity, regardless of the performed task (1). Lower limb muscle fatigue is speculated to be a leading factor in ankle joint athletic injuries since many of these injuries occur at the end of an activity when the athlete is fatigued (2). There are two main fatigue-related mechanisms that can contribute to the diminished force-generating capacity: somatosensory activity and muscular strength. During fatigue, somatosensory activity (efferent and afferent signals) is less successful in transmitting neural signals and as a result the muscle is stimulated to a lesser extent (1). This will lead to reduced motor control and therefore decreased stability. Strength is also diminished during fatigue as the proprioceptive and kinesthetic properties of the joints are altered through increased threshold of muscle spindle discharge, disrupting afferent feedback, and consequently limiting the muscle's ability to control the joint (3). Therefore, fatigue of postural musculature, such as the gastrocnemius and soleus, has led to diminished postural control and increased difficulty to stabilize balance (5).

Fatigue may also lead to a loss of postural muscular control due to diminished use of sensory information (1,3,4,6,7). It is understood that visual

stabilization of postural sway is based on how effectively body oscillations can be detected relative to stationary environmental surfaces (8). If subjects are blindfolded, less sensory information is available to control stability. The subject must undergo a reconfiguration of their postural control system in order to adapt to new conditions. The destabilizing effects due to the absence of vision has shown their effect when a subject is absent of vision for more than six seconds (9). Therefore, the removal of vision input emphasizes a reliance on the vestibular and somatosensory systems to control posture.

These sensory changes have a dramatic effect on the ankle with respect to posture. In regards to postural sway, the ankle is primarily responsible for control of anterior-posterior (A/P) movement of the Center of Pressure (CoP) and less responsible for medial-lateral (M/L) movement (10). The magnitude of CoP variability in each plane that the ankle controls has not yet been established. The ankle plays a central role with regard to postural corrections in single limb stance and although still vital, the ankle plays less of a role in double limb stance (10).

Previous studies have also demonstrated the influence of sensory systems on fatigue and postural sway by controlling different factors (4,9,11,12). In one particular study, vision was withdrawn and as a result, subjects exhibited a similar increase in CoP displacement in both fatigue and non-fatigue conditions (9). However, when vision was restored the subjects were able to immediately cope with the destabilizing effects induced by muscular fatigue, reducing postural sway (9). Furthermore, in the control condition and in absence of visual information, subjects were able to accomplish a rapid re-calibration and reorganization of postural control to take into account the new sensory conditions by switching from visual to proprioceptive control (9). Therefore, a complex relationship exists between the different sensory systems (vision and somatosensory) involved in the regulation of posture. The present study looked at postural sway when vision and somatosensory information was compromised.

The aim of our project is to better understand postural control mechanisms during quiet bipedal stance following plantar flexor fatigue. In order to determine the

effects of plantar flexor fatigue on postural sway, we will measure the CoP of blindfolded subjects, before and after fatiguing exercise. We hypothesize that there would be a significant increase in postural sway, as measured by the CoP variability in the A/P and M/L directions, following fatiguing exercise.

Methods

Subjects

Fifteen physically active young subjects voluntarily participated in this study. All subjects were healthy and not involved in a training program during their participation in the study. Participation was limited to subjects without a history of hip, knee, ankle or lower leg injuries, including medial tibial stress syndrome (shin splints) and compartment syndrome, in the past two years. Subject characteristics are presented in Table 1. All subjects gave their informed consent and were non-smokers and non-obese ($BMI < 30\text{kg/m}^2$). None of the subjects presented a history of known neurological disease, musculoskeletal problems or vestibular impairment. This study was approved by The University of Western Ontario Research Ethics Board for Health Sciences Research Involving Human Subjects.

Standing protocol

Subjects were blindfolded, asked to stand as motionless as possible on the force plate, arms at their sides, and feet shoulder-width apart while wearing socks. This primary task was executed under two states of muscular fatigue (no-fatigue and fatigue). The no-fatigue condition served as a control session (CON) executed first, with the fatigue session (FAT) executed immediately (<10 sec) after a fatiguing procedure. The standing trials were 20 seconds long to ensure that there was enough time to detect differences (6). In order to remove visual feedback, the subjects were blindfolded for both trials. Both the control and fatigue standing trials were administered once per subject.

Fatiguing protocol

Subjects performed two-legged plantar-flexion exercise while wearing a weighted-vest (10% of total body weight) until subjective fatigue. Subjects stood on the edge of a raised platform (soles of the feet resting on

a 12cm tall ledge while the heel was over the edge) and were instructed to repeatedly rise onto their tiptoes with their body upright. The subjects performed these movements to the beat of a metronome and received verbal encouragements from an experimenter. The frequency of the metronome was 80 beats/min (6) with the subject rising every other beat. The number of heel raises was counted. The ledge was located close to the force platform to reduce the time in between fatiguing exercise and FAT measurements. Volitional fatigue was achieved when the subject could no longer raise the heel properly (could not attain maximum plantar flexor range of motion, with body in a straight and upright position). Immediately following volitional fatigue, each subject was instructed to stand on the force platform where the CoP measures were obtained. After the fatiguing exercise and balance trial, a standard visual analog scale was used to determine the subjects' level of fatigue. This response was then converted into an RPE (Rate of Perceived Exertion) value on a scale of 1 to 10.

Apparatus

Ground reaction force data was used to calculate the CoP sway from the force plate (Kistler model 9287B) as the distance that the subjects swayed in the A/P and M/L directions. The sway produced by the machine reflects the degree of data scatter about a subject's centre of balance (13). The force plate signals were amplified (AMP 9865C), converted from analog to digital form through an A/D converter (sampled for 20 seconds at a sampling rate of 100 Hz, 16 bit, Model# 779407-01, National Instruments), and written to disk using a custom program written in LabVIEW (National Instruments).

Statistical Analysis

The variability of the CoP in the A/P and M/L directions was calculated from the force plate data. Data are presented as means $\pm$ SD. Statistical analysis was done using t-tests and a Bonferroni post-test correction was applied to eliminate multiple comparison biases. Statistical significance was declared when $p < 0.05$.

Results

A summary of the subject characteristics and fatiguing exercise is presented in Table 1. A comparison of the control and fatigue CoP variability in the A/P and

M/L directions is presented in Figure 1. Fatigue CoP variability was significantly greater ($p < 0.05$) than the control in the A/P direction ($8.89 \pm 3.74\text{mm}$ and $4.48 \pm 1.7\text{mm}$, respectively). Fatigue CoP variability was significantly greater ($p < 0.05$) than the control in the M/L direction ($3.30 \pm 1.24\text{mm}$ and $1.66 \pm 0.85\text{mm}$,

respectively).

Figure 1: Comparisons of control and fatigue variability in the medial/lateral and anterior/posterior direction. Significant differences from control are identified by * ($p < 0.05$). Error bars are SD

Figure 2: Spaghetti plot of the CoP displacements in the x and y direction over time, in the fatigue and control conditions, of one representative subject.

Table 1. Subject and fatigue level characteristics

	n	Age (yr)	Body mass (kg)	Height (m)	# of heel raises	RPE
Mean	15	21.3	81.6	1.83	257	8.6
SD	-	1.7	9.4	0.06	169	1.2

Discussion

The purpose of this study was to examine the effect of plantar flexor fatigue on postural stability in quiet standing. In the present investigation, fatigue of the plantar flexors led to significant increases in sway variability in the A/P and M/L directions. This suggests that the plantar flexors are necessary for stability during quiet stance and it is possible that smaller intrinsic muscles are unable to fully compensate for fatigued plantar flexors.

Our findings for A/P sway confirmed our hypothesis of greater variance of A/P CoP fluctuations as a result of plantar flexor fatigue. This is consistent with previous literature (2,5–8,11,14,15); however, other researchers (1,3,16) have found conflicting results. Contradictory findings may arise as there are many factors that contribute to the results, including; control and experimental conditions, fatiguing protocols, etc. Corbeil (1) found no increase in A/P sway. This difference from our study can plausibly be attributed to the difference in fatigue protocol; the Corbeil study performed the fatigue protocol in a bent-knee, seated position; thus, not allowing full range of motion of the gastrocnemius muscle. Other notable differences in protocols include the positioning of the feet and whether subjects wore socks, as in the current study, or were bare-footed; Vuillerme et al. (12) demonstrated that increased cutaneous feedback (wearing socks for example) could possibly attenuate postural sway under a fatigued-state. Deterioration in the A/P postural control is indicated by an increased A/P CoP variance seen in the fatigue condition, which could be due to neurological impairments of the ankle joint. More specifically, a decreased motor output as well as decreased sensitivity of the proprioceptors (6). Alternatively one can consider increased A/P variability as a function of control reorganization, thus a major contribution could be a result of deliberate increase in sway to effectively activate the

vestibular system(5). Gimmon et al. (2) proposed that the neuromuscular system is compromised in its ability to maintain sufficient muscle tension, leading to compensatory contractions during fatigue that result in overcompensations, manifested as larger displacements of CoP. However, it has been proposed that slowed conduction of afferent signals during fatigue will lead to delayed efferent signals and limit the ability to make compensatory movements (14).

Low intensity exercise to fatigue is associated with excitation contraction coupling failure within skeletal muscle leading to long recovery times (17); however, Adlerton et al. (10) have shown recovery time from fatigue to be only 5.9 and 1.5 minutes for the knee extensors and ankle dorsiflexors, respectively. It can be assumed that fatigue in the plantar flexors have a relatively quick recovery time, which our study controlled by having participants perform fatigue close to the force plate and allowing measurements to be obtained well under a minute.

Although the plantar flexors are not associated with CoP movement in the M/L direction, significant changes were seen in this plane. Fatigue of the plantar flexors led to significant changes in M/L sway variance. These results can be supported (8,11,15,16) by previous research conducted in the field, while other researchers (1–4,14) have produced confounding results. A previous study found that sway parameters were affected more in the A/P direction and less in the M/L direction with plantar flexor fatigue (2). It was reasoned that since the hip abductor and adductors are responsible for M/L movement and since they were not fatigued, less variance was seen in this plane. However, another study found substantial impairment in M/L postural control when proximal hip and ankle musculature were fatigued (14); it was concluded that gross changes occur during postural instability at the hip and knee. It is possible that during fatigue, smaller musculature is unable to correct for small

changes, and therefore the larger muscle groups overcompensate with gross movements resulting in a larger displacement of the CoP (3). This is similar to our findings whereby one subject's CoP had substantially more variability with fatigue. Normally during quiet stance, small changes are corrected by the ankle strategy through the stretch reflex of the plantar flexor and peroneal muscles; with fatigue, the ability to correct these changes becomes more difficult (16). In support of our findings, it is possible that the proprioceptors of the ankle joint were desensitized and the lateral (peroneal) muscles (responsible for M/L stability by ankle eversion, as well as minor plantar flexion) were unable to stabilize the joint, in the M/L plane, post-fatigue. Increased latency of the peroneal muscles would cause oscillations laterally during decreased support, and then medially with lateral contraction (16). In this case, smaller intrinsic muscles are relied on for structural support when larger musculature is fatigued. The movements caused by these muscles are sufficient to correct changes but apparently not for full stabilization of the joint.

In the present study, vestibular and somatosensory systems were controlled for by having the subjects stand in quiet stance on a flat surface without any cutaneous stimulation. It is proposed that vision also contributes to compensate for muscular fatigue (9). Under conditions of fatigue, it is shown that the vestibular and somatosensory systems have an increased role in maintaining upright posture (11). In a study by Pinsault and Vuillerme (11), the effect of plantar flexor fatigue during quiet standing was determined under the effects of altered vestibular and neck somatosensory conditions. While adopting a head-tilted backward posture to alter vestibular and somatosensory information, the effects of plantar flexor muscle fatigue was accentuated (11). In a second trial, adhesive bandages were placed over and around the skin of the neck to increase cutaneous feedback, the destabilization of the fatigued plantar flexors was alleviated (11). To demonstrate the importance of vision, a study by Vuillerme et al. (9) determined the effects of vision and muscular fatigue on postural control. With vision withdrawn in the no-fatigue condition, subjects were able to recalibrate and switch from visual to proprioceptive control. This compensation was not seen in the fatigue condition. In both conditions when vision was restored, subjects were able to

compensate for the destabilization caused by fatigued calf muscles. Thus, all sensory and motor components of postural control work in conjunction to keep the body stabilized. Therefore, with all feedback systems controlled for, the sole effect of muscular fatigue on postural control (statistically significant increases in CoP variability in the A/P and M/L directions, in the fatigue condition compared to control) was determined in the current study.

Limitation

Our study was limited in determining fatigue as a result of not using electromyography and instead basing level of fatigue on participants' subjective report. By using an RPE type determination of fatigue, the results are susceptible to various factors such as level of fitness, psychological state, and environmental conditions (18).

A limitation exists in fatiguing studies as illustrated by Roerdink et al. (5), who hypothesized that postural musculature compensated a fatigued state by deliberately increasing the magnitude of sway in order to exploit the vestibular system to correct posture. Thus, it is possible that our results could be the consequence of a functional adaptation in postural musculature to deliberately increase sway, in order to activate postural control mechanisms.

The present study used force plate data to record changes in sway variability; however, goniometers and/or video analysis could be used in tandem to determine change in balance strategy from ankle (distal corrective movements) to hip strategy (larger, proximal movements) post-exercise.

Future directions

Future studies should incorporate EMG data of lower limb musculature to better understand compensatory measures made by subjects under fatigued conditions. Muscle groups that are not primarily responsible for plantar flexion may assist the fatigued gastrocnemius and soleus muscles by becoming more active in postural control. Non-invasive surface EMG's can be used to measure activity of inverters, evertors, dorsiflexors and plantar flexors of the lower leg. A protocol to ensure a minimum fatigue level should be

developed using EMG data or a dynamometer. Surface EMG has been used previously by Gimmon et al. (2) to assure that their participants reached a fatigued state following the fatiguing protocol. A threshold for decrease in muscular strength should be established wherein participants who don't meet the threshold are A) excluded from the study, or B) asked to continue exercise until they reach an adequate level of fatigue (past threshold).

Conclusion

Our hypothesis of increased postural sway following fatigue was confirmed by an increase in A/P and M/L CoP variance under the fatigued-state. Therefore, a relationship appears to exist between muscle fatigue of the plantar flexors at the ankle and impaired postural control. Slowed conduction of afferent signal from the fatigue altered state of the muscle may lead to slowed propagation of efferent signals to help maintain posture. Theoretically, this will place the fatigued individual at a greater risk for musculoskeletal injury and therefore steps should be taken during the conditioning of athletes and with injury rehabilitation to help prevent muscle fatigue.

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