Effect of Plantar Flexor and Dorsiflexor Fatigue on Unilateral Postural Control

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The purpose of this study was to determine the effect of plantar flexor and dorsiflexor fatigue on postural sway amplitude during unilateral, or one-legged, stance. It was hypothesized that plantar flexor and dorsiflexor fatigue would increase unilateral postural sway amplitude. Eight uninjured male subjects participated in pre- and postfatigue unilateral stability tests. Selected parameters describing medial–lateral (ML) and anterior–posterior (AP) postural sway were measured on a Chattecx Balance System before and after an isokinetic fatigue protocol. The fatigue protocol resulted in a significant increase in ML postural sway amplitude (p < 0.05) and an increase in AP sway amplitude (p = 0.065). Previously, links have been established between increased postural sway amplitude and ankle joint injury. Thus, fatigue of the plantar flexors and dorsiflexors, which increased postural sway amplitude, may render the ankle joint susceptible to injury. Induced ankle muscle fatigue may represent a valid paradigm to study the causes of traumatic ankle joint injury.

Some 30% to 50% of all injuries reported by athletic teams occur at the ankle joint, with the lateral ligamentous complex most frequently affected through inversion injury (Weiker, 1984). Because of the acute and chronic debilitating effects of these common injuries, the underlying mechanisms of ankle injuries have been examined by many investigators (Cornwall & Murrell, 1991; Gauffin, Tropp, & Odenrick, 1988; Konradsen & Bohsen Ravn, 1991; Tropp, 1986; Tropp, Ekstrand, & Gillquist, 1984). Cornwall and Murrell (1991) reported that unilateral postural sway amplitude was significantly greater in a group of subjects with a history of ankle injury than in a group of uninjured subjects, and that the increased sway amplitude was evident as long as 2 years following the injury. Tropp et al. (1984) examined unilateral postural sway in a group of soccer players and found a significantly greater predisposition for ankle injury in subjects with pathological sway amplitudes than in subjects with normal sway amplitudes (pathological defined as exceeding the mean value of a control group by two

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standard deviations). This predictive relationship between sway amplitude and ankle joint injury was established in previously uninjured subjects as well as in chronically injured subjects.

Paulos, Noyes, Grood, and Butler (1991) stated that reinjuries of the knee ligaments often occur during the latter stages of an athletic activity and intimated that as the knee joint muscles fatigue they lose some of their capacity to control and protect the knee joint. This raises the possibility that acute muscular fatigue at the ankle joint would similarly place that joint at greater risk of injury. Therefore, induced fatigue might represent a valuable experimental paradigm to study ankle joint control relative to ankle joint ligament injury. The purpose of this study was to determine the effect of plantar flexor and dorsiflexor fatigue on postural sway amplitude during unilateral, or one-legged, stance. It was hypothesized that plantar flexor and dorsiflexor fatigue would increase medial–lateral (ML) and anterior–posterior (AP) unilateral postural sway amplitudes.

Methods

Subjects

Eight males (mean age = 30.0 ± 6.2 years, mean stature = 182.6 ± 2.5 cm, mean weight = 807.4 ± 83.4 N) volunteered to participate in the study and provided informed consent. All of the subjects were right-leg dominant, as determined by the criterion of the preferred foot for kicking a ball. None of the subjects reported recent lower extremity injury or a history of chronic ankle injuries.

Data Collection

Conditions. Each ankle was tested with at least 3 days between tests. The ankle that was tested first for each subject was randomized. Low-top athletic shoes were worn during the entire protocol to provide comfort during the fatigue protocol. The shoes were not removed prior to the stability testing to minimize the time period between the fatigue protocol and the postfatigue unilateral stability test.

Postural Sway Measurement. Pre- and postfatigue measurements of unilateral postural sway were obtained with a Chattecx Balance System (Chattecx Corporation, Chattanooga, TN). This instrument measured vertical reaction forces beneath the support foot with four force transducers located under the medial and lateral aspects of the heel and forefoot. The vertical reaction force signals from each transducer (sampled at 100 Hz) were transformed to represent the relative loading of each force transducer as a percentage of the total load (body weight). The time-related changes of the relative loading of the force transducers were evaluated separately in the AP and ML directions. These time-related changes of relative loading, or weight transfer, have recently been shown to be significantly related to excursions of the center of pressure measured with strain gauge force platforms (Grabiner, Lundin, & Feuerbach, 1993).

Subjects were positioned on the Balance System with the support foot parallel with the AP direction of the load cells, the arms crossed over the chest, and the knee of the nonsupport leg flexed approximately 45°. The subjects were instructed to minimize postural sway amplitude and were provided real-time visual feedback of the relative loading of the force transducers through a computer.
monitor placed at eye level approximately 1 m in front of the subjects. A 10-s warm-up and familiarization trial was performed prior to a 25-s trial during which the postural sway data were collected.

**Force Measurements.** Pre- and postfatigue measurements of maximum isometric plantar flexion and dorsiflexion force and rate of force generation were obtained on a Kin-Com dynamometer (Chattecx Corporation, Chattanooga, TN). The subjects were positioned on the Kin-Com in a supine position with arms crossed over the chest. The hip and knee joints were both flexed approximately 10° with the approximate plantar flexion–dorsiflexion axis of rotation aligned with the axis of rotation of the Kin-Com. Velcro straps around the shank, toe, and ankle and a canvas buckled strap around the waist secured the subjects to the Kin-Com.

Following a brief warm-up and familiarization period, the subjects, with an ankle position of 25° plantar flexion (with reference to the foot perpendicular to the tibia), performed two isometric maximum voluntary effort (MVE) plantar flexion contractions followed by two MVE dorsiflexion contractions. Two contractions were performed to reduce the possibility of recording anomalous data. The instructions to the subjects for the MVE trials were to generate their maximum effort as rapidly as possible, and then to immediately relax. There was an approximate 10-s delay between trials to allow for data storage. The force signals were digitized at 1 kHz and were stored for processing on a microcomputer.

**Fatigue Protocol**

After the prefatigue postural sway and force measurements were completed, the fatigue protocol was performed. While subjects were in the supine position previously described, their ankles were passively and isokinetically (30°/s) moved through a range of motion of 50° (5° dorsiflexion to 45° plantar flexion). Each subject performed a total of 5 sets of 15 repetitions with rest intervals of 10 s between sets. The first two sets consisted of concentric–eccentric dorsiflexion contractions through the 50° range of motion, during which the subjects were encouraged to maximally assist (concentric) or resist (eccentric) the isokinetic motion of the Kin-Com. The remaining three sets were performed in the same manner with concentric–eccentric plantar flexion contractions replacing dorsiflexion contractions.

Immediately following the fatigue protocol, four postfatigue isometric plantar flexion–dorsiflexion MVE trials were performed, identical to the prefatigue trials. The subjects then performed the postfatigue unilateral stability test, identical to the prefatigue test.

**Data Reduction**

**Postural Sway Variables.** Pre- and postfatigue variables extracted from the Balance System data for both ML and AP directions were mean center of balance (COB) and postural sway amplitude. Mean COB was expressed as the relative load (body weight) positioning over the four load cells averaged over the 25-s trial. For example, an AP value of +5.0% indicated that the anterior load cells were loaded 5.0% more than they would be if all the load cells were loaded equally. Similarly, a positive ML value indicated a shift toward the lateral border of the foot. Both ML and AP sway amplitude values were characterized
by the standard deviations of the respective COB data (Feuerbach & Grabiner, 1993).

**Force Variables.** Maximum isometric plantar flexion and dorsiflexion force and rate of force generation were extracted from each pre- and postfatigue MVE condition. Maximum rate of force generation was determined by finding the maximum value of the first derivative with respect to time of the force curve. The force data were smoothed with a recursive second-order Butterworth filter with a cutoff frequency of 6 Hz.

**Data Analysis**

A $2 \times 2$ analysis of variance (ANOVA) (Test Day $\times$ Fatigue Condition) with repeated measures across each factor was performed on the postural sway and force variables. The level of significance was set at 0.05.

**Results**

Examples of AP and ML COB data from the first 10 s of one of the 25-s trials of one subject are shown in Figure 1. The positive AP COB values reflect that the subject shifted his weight over the anterior load cells. The positive ML COB values reflect a shift toward the lateral border of the foot.

The results of the ANOVAs revealed that there were no significant differences as a function of test day ($p > 0.05$). This was tantamount to revealing that there were no significant interankle (right to left) differences. Thus, the data were pooled across test day (across ankle) with the appropriate correction for degrees of freedom.

The ANOVAs revealed a significant anterior shift of the COB ($p < 0.05$) but no significant change in the lateral location of the COB ($p > 0.05$) subsequent to the fatigue protocol (Figure 2). An increase in AP sway amplitude ($p = 0.065$) and a significant increase in ML sway amplitude ($p < 0.05$) were seen following the fatigue protocol (Figure 3). The significant reductions in maximum isometric plantar flexion and dorsiflexion force and rate of force development ($p < 0.05$) (Figures 4 and 5) demonstrate that the ankle joint musculature was fatigued prior to the postfatigue unilateral postural sway measurement.

**Discussion**

Although the fatigue protocol involved sagittal plane motion, it was assumed that the muscles that produce inversion and eversion were fatigued because these muscles also produce plantar flexion and dorsiflexion. For example, tibialis anterior acts to dorsiflex and invert the foot, peroneus longus and peroneus brevis act to plantar flex and evert the foot, and peroneus tertius acts to dorsiflex and evert the foot.

It is possible that a portion of the acute decrease in isometric force was due to the muscle damage that has been associated with eccentric contractions, which ultimately produces delayed onset muscle soreness. Lieber, Woodburn, and Friden (1991) stated that of the 69% decrease in rabbit tibialis anterior isometric force 1 hr after 1,800 eccentric contractions, 30% was due to fatigue and 26% was due to muscle damage. Thus, a major portion of the reduction of force in Lieber et al.'s study appeared to be due to fatigue even after 1 hr of
recovery. Therefore, it is probable that the majority of the acute force reduction seen in the present study was produced by fatigue.

Consistent with expectations, a significant increase in ML unilateral postural sway amplitude and an increase in AP unilateral postural sway amplitude \( (p = 0.065) \) were observed following the fatigue of the plantar flexors and dorsiflexors (Figure 3). These results support the hypothesis that fatiguing the plantar flexors and dorsiflexors would increase unilateral postural sway amplitude. It is tempting to postulate that acute fatigue of the ankle joint musculature and the associated reduction in motor control act as a mechanism for some ankle injuries. Because acute muscular fatigue produced an increase in postural sway amplitude and abnormally large sway amplitude values have been linked with ankle injury

![Figure 1](Image)

Figure 1 — AP and ML center of balance (a and b, respectively) from the first 10 s of one of the 25-s trials of one subject.
The increases in unilateral postural sway amplitude found in the present study were seemingly not as substantial as the differences between subjects predisposed to ankle joint injury and those who are not (Tropp et al., 1984). In other words, postfatigue values were not greater than the prefatigue values by two standard deviations. In the present study postural sway was divided into ML and AP components and was expressed as the standard deviation of the relative weight positioning over the four load cells of the balance system. Tropp et al. (1984) calculated postural sway amplitude as the area of a two-dimensional confidence ellipse of the coordinates of the center of pressure values measured on a force plate. Thus, the relatively small increases in AP and ML sway amplitude

(Cornwall & Murrell, 1991; Tropp et al., 1984), the possibility exists that fatigue of the ankle joint musculature predisposes the ankle joint to injury.
Figure 3 — Mean AP and ML postural sway amplitude (a and b, respectively) in the pre- and postfatigue conditions. *significantly different from prefatigue condition (p < 0.05).

seen in the present study may produce relatively large increases in the area of such a confidence ellipse. The differences in data analysis between the present study and that of Tropp et al. (1984) would, most likely, account for the differences between the two studies in regard to control versus experimental sway amplitude values.

Similar to the present study, Seliga et al. (1991) reported an increase in postural sway amplitude following submaximal, aerobic bicycling. Seliga et al. (1991) reasoned that central fatigue (diminished central nervous system stimulation to muscle) and localized muscular fatigue were factors associated with the observed increases in sway amplitude. These investigators might be suggesting that decreases in the maximum force-generating capability of the muscles in-
involved in postural control result in increases in postural sway amplitude. However, Tropp (1986) found no correlation between postural sway amplitude and ankle joint pronator muscle strength. Lentell, Katzman, and Walters (1990) also reported that maximum inversion and eversion moments did not correlate with unilateral postural sway amplitude. It is possible that a reduction in muscle force was not the mechanism that produced unilateral postural sway amplitude increases in the present study. Muscle forces required to maintain postural control are relatively small compared to the maximum force-generating capability of the involved muscles.

Konradsen and Bohsen Ravn (1991) found a high degree of correlation (Spearman’s rho = 0.92) between delays in peroneal muscle reaction time (onset
of EMG following sudden ankle inversion) and increases in postural sway amplitude. This delay in peroneal reaction time results in a delay of muscular force generation, which is similar to the electromechanical delays in force generation seen with muscular fatigue (Hakkinen & Komi, 1983; Hortobagyi, Lambert, & Kroll, 1991). Delays in muscle force generation may be a "pathological mechanism” described by Tropp et al. (1984) that leads to increases in unilateral postural sway amplitude and results in ankle joint injury during high levels of athletic activity. Thus, if the forces required for the correction of an unstable placement of the foot are delayed due to fatigue, then the ankle joint would be at risk of injury.

Some investigators (Lentell et al., 1990; Tropp, 1986) have suggested that
measurements of unilateral postural sway amplitude characterize ankle joint proprioception. Fatigue-induced proprioceptive deficits, therefore, may account for the increases in unilateral postural sway amplitude seen in the present study. Skinner, Wyatt, Hodgdon, Conard, and Barrack (1986) found a significant proprioceptive deficit at the knee joint as a result of fatigue due to endurance running. Fatigue of the ankle joint musculature may have a similar consequence on ankle joint proprioception. Impairment of the ability to control the ankle joint and foot during some types of vigorous physical activity would likely increase the probability of the foot being placed in an unstable position. This impairment of control could result in ankle joint injury, particularly if the corrective/protective responses at the ankle joint are delayed.

In addition to the increases in unilateral sway amplitude, plantar flexion–dorsiflexion fatigue produced an anterior shift of body weight compared to that of the nonfatigued state (Figure 2a). This postfatigue shift might reflect the adoption of a postural control strategy that increases the role of the intrinsic toe flexors (flexor digitorum brevis and longus) in maintaining balance. This muscle group has been shown to play a role in the maintenance of postural control, especially at the anterior limits of postural stability (Schieppati, Nardone, & Giordano, 1992) and may be more active after other plantar flexors fatigue than before the plantar flexors fatigue.

**Conclusions**

A fatigue protocol involving isokinetic plantar flexion and dorsiflexion contractions produced a significant increase in ML unilateral postural sway amplitude compared to that of a nonfatigued state. Previously, a link had been established between a pathological postural sway amplitude and ankle joint injury (Cornwall & Murrell, 1991; Tropp et al., 1984). Thus, by extension, fatigue of the plantar flexors and dorsiflexors, which produced an increase in postural sway amplitude compared to that of the nonfatigued state, may render the ankle joint susceptible to injury. Possible pathological mechanisms associated with muscular fatigue that could lead to ankle joint injury are delays in force-generating capabilities of the ankle musculature and decreases in ankle joint proprioception.

**References**


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