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Postural steadiness and ankle force variability in peripheral neuropathy

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Abstract

Introduction—The purpose was to determine the effect of peripheral neuropathy (PN) on motor output variability for ankle muscles of older adults, and the relation between ankle motor variability and postural stability in PN patients.

Methods—Older adults with (O-PN) and without PN (O), and young adults (Y) underwent assessment of standing postural stability and ankle muscle force steadiness.

Results—O-PN displayed impaired ankle muscle force control and postural stability compared with O and Y groups. For O-PN, the amplitude of plantarflexor force fluctuations was moderately correlated with postural stability under no-vision conditions ($r = 0.54$, $P = 0.01$).

Discussion—The correlation of variations in ankle force with postural stability in PN suggests a contribution of ankle muscle dyscontrol to the postural instability that impacts physical function for older adults with PN.

Keywords

aging; older adults; physical function; postural control; balance

Introduction

Peripheral neuropathy (PN) degrades sensory function (Brown et al., 2004) and reduces the quality of critical afferent feedback necessary for supraspinal centers to optimize motor output and stabilize posture. Motor neuropathy may follow sensory neuropathy, negatively impacting efferent signals to skeletal muscles (Yamamoto et al., 2001). This decline in neural function results in postural deficits that can reduce functional ability and quality of life, and increase fall risk, morbidity, and mortality. Given the projected increase in the older adult population and the dominant diabetic and chemotherapeutic etiology of many PN cases, the societal health burden of PN will increase in the coming decades (Martyn & Hughes, 1997).

As with the postural instability that can accompany normal aging, functional disability can arise in PN due to balance impairment (Nardone & Schieppati, 2007) secondary to degraded sensation (Richardson & Ashton-Miller, 1996). Peripheral neuropathy exacerbates sensory dysfunction and postural instability to an even greater degree than normal aging (Yamamoto et al., 2001). Plantar sensation and muscle mechanoreceptor-based proprioceptive signaling are important to postural stability and are negatively impacted by PN (van Deursen & Simoneau, 1999). These initial symptoms which continues to progress after onset of PNt, contribute substantially to postural instability as impaired plantar sensation decreases and/or degrades the information provided to supraspinal centers about shifting plantar pressures and postural sway (Wang & Lin, 2008). The negative impact of PN on postural stability has been demonstrated by clinical tests including center of gravity sway velocity (Lim, Kim, Noh, Yoo, & Moon, 2014), the Berg Balance Scale (Ghanavati, Shaterzadeh Yazdi, Goharpey, & Arastoo, 2012), and average sway velocity (Zhang, Manor, & Li, 2015). This reduced postural stability is likely to result in falls (DeMott, Richardson, Thies, & Ashton-Miller, 2007; Tofthagen, Overcash, & Kip, 2012).

The degradation of submaximal muscle force production in aging has been studied in a variety of muscle groups, including the plantarflexor (PF) muscles (Tracy, 2007a). A small amount of information also suggests that ankle force production is correlated with postural stability (Kouzaki & Shinohara, 2010). The impact of motor output variability at the ankle on postural stability is supported by the observations that change in the soleus electromyogram (sEMG) precedes alteration of center of pressure and that torque output at the ankle is reflected by center of mass displacement during quiet standing (Masani, Popovic, Nakazawa, Kouzaki, & Nozaki, 2003). For example, the coefficient of variation (CV) of isometric PF force output at 2.5% of maximal voluntary contraction has been shown to be correlated with the coefficient of variation of the center of pressure during quiet standing (Kouzaki & Shinohara, 2010).

In the context of older adults or special populations, the interpretation of studies that have correlated ankle force steadiness with postural stability is unclear. These investigations have either focused solely on young adults (Oshita & Yano, 2012) or have pooled the values from young and old adults together (Kouzaki & Shinohara, 2010) to compute correlations. Furthermore, no previous investigations have characterized motor output variability at the ankle for PN patients. Thus, no information exists on the correlation of impaired ankle force steadiness with the excessive postural instability observed in PN patients. This type of information could inform interventions to improve ankle function, postural stability, and reduce the risk of falls in PN patients.

The goal of this study was to characterize postural control and ankle force steadiness in older adults with peripheral neuropathy (O-PN), older healthy adults (O), and young healthy adults (Y). It was hypothesized that postural and ankle function would be impaired for older adults, and that even greater impairments would be observed in older adults with PN. It was also hypothesized that PF and dorsiflexor (DF) force fluctuations would be correlated with postural fluctuations to a greater degree in the subject groups with more impaired postural stability (O and O-PN). This outcome would suggest that the degraded ankle muscle output of aging and further degradation found with neuropathy impacts real-world function such as

postural stability. It was expected that the strongest correlation between ankle force output and postural stability would be demonstrated by O-PN due to the greater changes in that group. Furthermore, it was expected that the correlations between force output and postural stability would be greatest without visual feedback due to the greater reliance on proprioceptive input and the large changes in postural stability that occur for older adults and PN patients when visual input is not available as a sensory input (Teasdale, Stelmach, Breunig, & Meeuwssen, 1991). A second aim of this study was to determine the role of visuomotor processing in both ankle force output and postural control in O-PN, O, and Y. It was hypothesized that the results of this study would align with previous investigations wherein visuomotor processing is affected by age (Tracy, 2007b; Tracy, Dinunno, Jorgensen, & Welsh, 2007). Therefore, we expected the removal of visuomotor processing to reduce force fluctuations for O-PN and O, but not for Y. As has been observed previously, removal of visual feedback was hypothesized to have the greatest detrimental effect on postural control in O-PN, followed by O, with lesser impact in Y. The results have been presented previously in abstract form (Tracy, Massie, Long, & Hitchcock, 2010).

Materials and Methods

Subjects

Thirty-two O-PN (17 female, 15 male), 32 O (23 female, 9 male), and 12 Y (6 female, 6 male) participants were studied (Table 1). Written confirmation of PN diagnosis was obtained from each patient's physician; the etiology of the PN was not conveyed. Exclusion criteria included overt neurological and/or neuromuscular conditions (with the exception of peripheral neuropathy in the O-PN group), as well as use of psychoactive or tremor-inducing medications. The O-PN and O subjects were of similar age ($P = 0.33$). O-PN and Y were of similar height ($P = 0.99$) and were taller than the O group ($P < 0.05$). Body mass and body mass index was similar between O-PN, O, and Y ($P > 0.05$). After orientation to the protocol, subjects provided written informed consent prior to participation in the study. All study procedures were approved by the Human Research Committee at Colorado State University.

Visits

Each participant was assessed on two separate days. The first visit included orientation to the protocol and postural assessment in the Clinical Biomechanics Laboratory. At a similar time on another day subjects performed ankle force production and control tasks in the Neuromuscular Function Laboratory. The interval between visits ranged from 2–5 days.

Force production assessment

Force assessments were performed with the subject seated in a custom, instrumented experimental chair with the hips at 100 degrees, and knees and nondominant ankle restrained to 90 degrees. A seated position was used specifically to isolate DF and PF forces about the ankle, thereby focusing measures on this joint alone. For the O-PN subjects, the most symptomatic foot was used. The ankle was secured to a custom foot plate fixed to the chair. Load cells were secured under the foot plate to register DF or PF force. The load cell position was adjustable for different foot lengths. Tension (dorsiflexion) and compression

(plantarflexion) force was measured with the load cell directly beneath the first metatarsophalangeal joint so that the force applied was in line with the measurement axis of the load cell. The foot was secured to the foot plate to maintain the forefoot position on the load cell and the center of rotation of the ankle (lateral malleolus) relative to the center of rotation of the platform. The torso and pelvis were restrained with straps and the lower leg was restrained with a rigid mechanism that prevented upward movement during plantarflexion tasks. The purpose of the restraints was to isolate the muscle group of interest as best as possible.

Maximal voluntary contraction (MVC) force was assessed for the DF and PF muscles. Subjects slowly ramped up their force to maximal over the course of 3 seconds and then exerted maximal force for 2–3 seconds while exhaling. This was performed at least three times until the maximal force from two trials was within 5% of each other. This was accomplished within four trials for most subjects. Strong verbal encouragement was provided. At least 60s rest was provided between MVC trials. Visual feedback of the muscle force was provided via a line scrolling horizontally across a monitor 75 cm in front of the participant.

Isometric force steadiness tasks (2.5% MVC) with (VIS) or without (NVIS) visual feedback were randomized for DF and PF. The order of muscle group was also randomized. To maximize signal-to-noise ratio, the lowest capacity load cell (Transducer Techniques, Temecula, CA) possible was used depending on the target force for the trial. The force signal was observed online and digitized at 1000 Hz (1401 Plus, Cambridge Electronic Design, UK) for subsequent offline analysis (Spike 2, Cambridge Electronic Design, UK). Isometric force steadiness tasks in the VIS condition involved the display of a target force as a bold static horizontal line. The exerted force was represented by another bold horizontal line that moved vertically with changes in force. Subjects were instructed to match the force output line with the target line and hold the force as steadily as possible. For the NVIS force steadiness tasks, the monitor feed was turned off, and the subjects were verbally coached up to the target in approximately three seconds and were instructed to hold the force at that level as steadily as possible with no further verbal coaching. Two 10s trials in each condition (VIS, NVIS) were completed in randomized order.

For the VIS and NVIS force trials, the force signal was first detrended using the DC remove function in Spike 2 with a 1s time constant (Welsh, Dinunno, & Tracy, 2007). This procedure had the effect of removing < 0.5 Hz drift away from the target that can occur during the NVIS condition, while preserving the force fluctuations of interest (Tracy et al., 2007). The standard deviation (SD) of force was calculated from a 10s period immediately after target acquisition and attainment of steady-state. The coefficient of variation (CV) of force was calculated using the detrended SD of force ($SD/\text{mean force} \times 100$). The CV of force is a normalized measure of the amplitude of force fluctuations suitable for comparison between subjects with different strength levels (Tracy et al., 2007). In addition, a single CV of force value, representative of a subject's overall normalized force steadiness, was calculated by averaging the CV of force across the VIS and NVIS conditions.

Postural Assessment

Subjects stood on two force platforms embedded in a rigid platform (4060-10, Bertec Corp., Columbus, OH). The heels were separated by 10% of the subject's height. Each participant completed trials in random order under these conditions, 1) with visual feedback (eyes open), standing as still as possible, focused on a mark on a wall 4m ahead, 2) without visual feedback (eyes closed), standing as still as possible. A guard rail surrounded the force platforms and an investigator remained behind the subject in the event of balance loss, which did not occur. Center of pressure (COP) data was digitized at 100 Hz for subsequent offline analysis (Motus 8.5, Vicon, USA); the net COP value across the left and right force plates was used for analysis. Each postural trial consisted of 62 seconds of data collection. The middle 60 seconds of each trial was analyzed. The data were low-pass filtered at 10 Hz (4th-order recursive Butterworth). From these recordings, two center-of-pressure based measures in the anterior-posterior (A/P) direction were utilized as indicators of postural steadiness: A/P postural sway (max anterior COP – max posterior COP, PS), and COP path length (PL). All measures were normalized to foot length.

Statistics

One-way analysis of variance (ANOVA) with Tukey post-hoc tests was used to determine between-group differences. Repeated measures ANOVA with group (Y, O, O-PN) as a between-subjects factor and visual feedback condition (VIS, NVIS) as a within-subjects factor, was used to determine the effect of visual feedback and group by visual condition interactions. Bivariate correlations were computed to assess relations between steadiness and postural control. Exact P-values are provided for statistical significance where appropriate. All results are noted as mean $\pm$ SD in text, and mean $\pm$ SEM in figures. SPSS (IBM, Chicago, IL) version 20 was used.

Results

Muscle strength

The MVC force (Figure 1A) for the PF muscles was similar ($P = 0.27$) for O-PN and O, and less than Y ($P < 0.001$). Similarly, the MVC force for the DF (Figure 1B) muscles was similar ($P = 0.95$) for O-PN and O and was less than Y ($P < 0.001$).

Force fluctuations

Visual feedback and force steadiness—For the older PN patients the CV of force (Figure 2) for the DF muscles was greater ($P = 0.002$) for VIS than NVIS and the CV of force for the PF muscles was also greater ($P = 0.03$) for VIS than NVIS. For the older healthy adults the CV of force for the DF muscles was greater ($P = 0.03$) for VIS than NVIS and the CV of force for the PF muscles was also greater for VIS than NVIS ($P = 0.001$). For young adults the CV of force for the DF muscles was not different ($P = 0.22$) between VIS and NVIS but the CV of force for the PF muscles was greater ($P = 0.05$) for VIS than NVIS.

Differences in force steadiness between groups—For the DF muscles, the CV of force (Figure 2) with VIS tended to be greater for O-PN than Y ($P = 0.07$), was similar for O-PN and O groups ($P = 0.70$), and was not different between O and Y groups ($P = 0.23$).

For NVIS, there were no differences between O-PN, O, and Y groups ($P > 0.10$). For the PF muscles, the CV of force with VIS tended to be greater for the O-PN vs. O group ($P = 0.08$), was greater for the O-PN vs. Y group ($P = 0.003$), and similar between the O and Y group ($P = 0.17$). For NVIS, the CV of force was greater for the O-PN than the O group ($P = 0.001$) and Y group ($P < 0.001$), and similar between the O and Y groups ($P = 0.82$).

Group differences in steadiness pooled across visual conditions—The CV of force was also expressed as an average of the VIS and NVIS conditions to produce an overall steadiness value for an individual.

Postural fluctuations

Visual feedback effects—For the O-PN group, PS (min-max A/P) (Figure 3) was 64% greater ($P < 0.001$) without visual feedback compared to with visual feedback. Similarly, PL (Figure 3) was 74% greater ($P < 0.001$) for without visual feedback compared to with visual feedback (3.43 ± 0.445 cm/foot length). For the O group, PS was 22% greater ($P < 0.001$) without visual feedback compared to with visual feedback. Similarly, PL was 29% greater without visual feedback compared to with visual feedback ($P = 0.001$). For the Y group, PS was similar ($P = 0.16$) for both visual feedback conditions, and path length was 9% greater ($P = 0.01$) without visual feedback than with visual feedback.

Group differences in postural stability—Postural sway (Figure 3): With visual feedback, there were no differences in PS between the O-PN, O, and Y groups ($P = 0.7$ to 0.9). Without visual feedback, PS was significantly greater for the O-PN group compared to O ($P = 0.01$) and Y ($P = 0.01$), with no differences between O and Y ($P = 0.68$). Path length (Figure 3): With visual feedback, PL was similar for O-PN and O ($P = 0.16$) and greater for the O-PN group compared to the Y group ($P = 0.01$). Without visual feedback, PL was greater for the O-PN group than the O group ($P < 0.001$) and the Y group ($P < 0.001$) but not different between the O group and the Y group ($P = 0.35$).

Force steadiness/postural stability correlations

Peripheral neuropathy patients—Neither of the postural stability outcome measures were correlated with dorsiflexor or plantarflexor MVC force ($P > 0.05$).

For the postural sway with visual feedback outcome variable, there were no correlations with steadiness measures for either muscle group: CV of force for DF VIS ($P = 0.52$), DF NVIS ($P = 0.91$), PF VIS ($P = 0.86$), or PF NVIS ($P = 0.35$, Figure 4A). Postural sway without visual feedback, however, was correlated with only the PF measures - both the CV of force for PF VIS ($R = 0.43$, $P = 0.04$) and PF NVIS ($R = 0.40$, $P = 0.05$, Figure 4B) - but not with CV of force for DF VIS ($P = 0.83$) and DF NVIS ($P = 0.93$).

A similar pattern emerged for the path length outcome variable. Path length with visual feedback was not correlated with any steadiness measure: CV of force for DF VIS ($P = 0.56$), DF NVIS ($P = 0.72$), PF VIS ($P = 0.09$), or PF NVIS ($P = 0.16$, Figure 4C). However, path length without visual feedback was correlated with the plantarflexor steadiness outcomes: PF VIS ($R = 0.51$, $P = 0.01$) and PF NVIS ($R = 0.54$, $P = 0.01$, Figure 4D), but

not with the dorsiflexor steadiness outcomes: CV of force in DF VIS ($P = 0.30$) and DF NVIS ($P = 0.89$).

Older healthy adults—As with the PN patients, there were no correlations between postural stability outcome measures and MVC force for either muscle group ($P > 0.05$).

The postural sway with vision outcome variable was not correlated with the CV of force in DF VIS ($P = 0.86$), DF NVIS ($P = 0.21$), or PF VIS ($P = 0.15$), but was moderately correlated with PF NVIS ($R = 0.42$, $P = 0.02$). Likewise, postural sway without vision was not correlated with CV of force in DF VIS ($P = 0.31$), DF NVIS ($P = 0.23$), or PF VIS ($P = 0.12$), but was moderately correlated with PF NVIS ($R = 0.47$, $P = 0.01$).

The path length with vision outcome variable was not correlated with CV of force in DF VIS ($P = 0.11$) or DF NVIS ($P = 0.08$), while moderate correlations did exist with PF VIS ($R = 0.42$, $P = 0.02$), and PF NVIS ($R = 0.58$, $P < 0.01$). Path length without vision was not correlated with CV of force in DF VIS ($P = 0.31$), DF NVIS ($P = 0.23$), or PF VIS ($P = 0.12$), but was moderately correlated with PF NVIS ($R = 0.47$, $P = 0.01$).

Young healthy adults—Unlike the two older groups, there was a negative correlation between postural sway with vision with both DF MVC ($R = -0.58$, $P = 0.05$) and PF MVC ($R = -0.62$, $P = 0.03$), but no correlations between postural sway without vision and DF MVC ($P = 0.63$) or PF MVC ($P = 0.23$). The path length outcomes were not correlated with any muscle group steadiness outcome ($P > 0.05$).

For the young group, there were generally no correlations between postural stability outcomes and force steadiness. For postural sway with vision there were no correlations with the steadiness measures: CV of force in DF VIS ($P = 0.53$), DF NVIS ($P = 0.08$), PF VIS ($P = 0.24$), or PF NVIS ($P = 0.48$). Similarly, there were no correlations between postural sway without vision and CV of force in DF VIS ($P = 0.75$), DF NVIS ($P = 0.98$), PF VIS ($P = 0.21$), or PF NVIS ($P = 0.67$).

For the path length variable with vision, there were no correlations with CV of force in DF VIS ($P = 0.73$), DF NVIS ($P = 0.38$), PF VIS ($P = 0.22$), or PF NVIS ($P = 0.59$). For path length with no vision, there were no correlations with CV of force in DF VIS ($P = 0.45$), DF NVIS ($P = 0.65$), PF VIS ($P = 0.77$), or PF NVIS ($P = 0.34$).

Several sensory tests were employed to characterize the effect of peripheral neuropathy on lower limb sensation in the O-PN group. The Michigan Neuropathy Screening Instrument score averaged 4.21 ± 1.85 and the physical examination score averaged 4.64 ± 2.48 . These scores align with previous studies with neuropathy patients (e.g. 5.24 ± 1.4 , 3.03 ± 1.3 , respectively) (Brown et al., 2004; Kluding et al., 2012) and meet the physical examination score definition of peripheral neuropathy (< 2.0) (Al-Geffari, 2012). For a subset of the O-PN subjects, Pressure Specified Sensory Device (PSSD) testing was also performed. At the plantar pulp of the great toe, heel, and dorsum of the foot, measured sensitivity values averaged 86.0 ± 10.7 , 78.9 ± 11.9 , and 72.1 ± 10.2 g/mm², respectively, pooled across the feet. Normative PSSD values in healthy subjects for identical areas are 3.9 ± 4.3 , 8.0 ± 8.0 , and 4.6 ± 4.9 g/mm², and values ranging from 30 to 53 have previously been reported in

diabetic neuropathy patients (Ferreira, Rodrigues, & Fels, 2004). Two point discrimination was performed in a subset of fourteen O-PN at the point of the great toe, though only five O-PN subjects had the ability to feel the points for the test. The average distance at which these five O-PN subjects could discern two points of pressure was 1.03 ± 0.32 cm (average of both feet) compared with values from another study of healthy older subjects of approximately 0.5 cm (Periyasamy, Manivannan, & Narayanamurthy, 2008). Peripheral neuropathy patients in other studies have previously demonstrated a two point discrimination distance of 2.5 cm (Periyasamy et al., 2008). While this is greater than the average distance noted in our subjects, it must be reinforced that the majority of our subjects could not feel the points of pressure at all, indicating substantial decline in cutaneous sensation. Fine touch sensation was also assessed in the most affected foot of a subset of 14 O-PN subjects by applying a 10g Semmes-Weinstein monofilament to 10 sites on the foot. They exhibited a $47 \pm 34\%$ error rate, as compared to a $0.7 \pm 1.4\%$ error rate reported elsewhere for healthy subjects (Dimitrakoudis & Bril, 2002). The error rate of our PN patients aligns with the error rate of $56 \pm 34\%$ described previously in a neuropathic population (Olaleye, Perkins, & Bril, 2001). No correlations existed between measures of cutaneous sensation and force output or postural stability ($P > 0.05$).

Discussion

This study sought to determine 1) the impact of peripheral neuropathy on the production of low forces in the ankle muscles of older adults, and 2) the extent to which impaired control of the ankle muscles is correlated with the postural instability observed in older adults with peripheral neuropathy. Participants with peripheral neuropathy exhibited greater motor output variability during low force isometric contractions. When visual feedback did not contribute to the postural task in participants with peripheral neuropathy, plantarflexor but not dorsiflexor force variability was generally correlated with postural stability. These findings demonstrate that individuals with a reduced ability to control plantarflexor force fluctuations also exhibited the greatest postural instability, suggesting that plantarflexor motor variability contributes to the balance impairment of older adults with peripheral neuropathy.

Force fluctuations—Motor output variability in the plantarflexors was clearly impaired for the peripheral neuropathy patients compared with similarly-aged older adults (Figure 2A). This was evident not only when visual feedback was available, but also for the no-vision condition when proprioceptive and somatosensory feedback were the only sources of afferent feedback. This finding is novel as very little information is available on force variability in the isolated ankle muscles of peripheral neuropathy patients. Previous work showed impaired passive ankle tracking ability in peripheral neuropathy patients, suggesting that the degradation of proprioceptors also is implicated along with the loss of sensory function from cutaneous pressure receptors (van Deursen, Sanchez, Ulbrecht, & Cavanagh, 1998). However, the study did not directly investigate the quality of motor output.

Previous studies have described greater amplitude of force fluctuations for older compared with young adults when visual feedback contributes to the task, and similar values when

visual feedback is removed and central visuomotor processing is not required (Tracy et al., 2007). This visuomotor-dependent age effect on force steadiness has been attributed to impaired visuomotor processing in older adults (Tracy, 2008). Indeed, the coefficient of variation of force output is reduced by 19% in plantarflexors and dorsiflexors when visuomotor processing is removed, which is likely due to a lesser influence of oscillations associated with descending commands. (Tracy, 2007b) These findings support the notion that while visuomotor processing is diminished with age proprioceptive processing remains relatively intact, thereby allowing force modulation to actually be improved by removal of visual feedback. In the present investigation, we found that without visual feedback the force fluctuations for the older peripheral neuropathy patients remained elevated above the values for older and young adults. This novel finding suggests that peripheral neuropathy degrades force output to a greater degree than the typical aging effect for both VIS and NVIS conditions. Degraded force production without vision for peripheral neuropathy patients is presumably due to decreased quality of moment-to-moment afferent feedback relaying mechanical sensory stimuli in the lower limb to the central nervous system (van Deursen & Simoneau, 1999). Such proprioceptive degradation was previously demonstrated in a study that involved passive contralateral ankle position matching in young healthy subjects, peripheral neuropathy patients, and age-matched controls (van Deursen et al., 1998). In a similar study of peripheral neuropathy patients and older adults, cutaneous mechanoreceptors were also demonstrated to have a greater mechanical threshold (Mizobuchi et al., 2002). The reduced acuity of cutaneous and muscle sensory function could affect peripheral neuropathy patients' ability to detect, and therefore modulate, force output - both with and without vision. Although sensory impairment is the dominant and early presentation in the clinical course of peripheral neuropathy, there also remains the possibility that the degraded output was in part due to impaired fidelity of efferent transmission to the muscle via degraded motor pathways. To our knowledge, these are the first data that describe impaired low-force isometric steadiness in the plantarflexors in older adults with peripheral neuropathy.

Maximal force—Compared with the young subjects, plantarflexor strength was reduced by 57% for the PN patients and 56% for the old adults (Figure 1A). This finding was mirrored in the dorsiflexor muscles, with greater values for young subjects but little difference between these samples of older adults and older neuropathy subjects (Figure 1B). For the plantarflexors, the lower values for older versus young adults reflects the well-documented effect of aging on strength (Tracy et al., 2007). The lack of significant difference between our older adults and peripheral neuropathy subjects is likely due to the greater proportion of women in the older healthy group (25 women, 9 men) compared with the more balanced distribution of the sexes in the peripheral neuropathy group (17 women, 15 men). More women in the healthy older group certainly produced a lower mean value for strength than if the sex distribution was balanced. This notion is supported by the finding that within each group the mean MVC values were lower for women than men ($P < 0.05$). Also, the differences in strength between young, older, and peripheral neuropathy groups were not different between men and women (sex by group interaction $P > 0.05$). Therefore, the peripheral neuropathy subjects in all likelihood exhibited the reduced strength that would be expected in such a clinical population, in agreement with other reports (Martinelli et al.,

2013). The general lack of correlation between plantarflexor or dorsiflexor muscle strength and postural stability for these older adults suggests that for these groups of subjects strength loss was not a major contributor to the sway fluctuations or maximal excursion of postural sway during quiet standing.

Postural stability—When visual feedback was available, postural stability was similar between peripheral neuropathy patients, older adults, and young adults (Figure 3A). These similarities have been observed previously (Yamamoto et al., 2001) and speak to the dominance of visual feedback in the mix of afferent inflow to the central nervous system during postural tasks. Without the benefit of visual feedback, postural stability for peripheral neuropathy patients was significantly impaired compared with the other groups - a 1.7-fold increase in variability for PN patients compared with a 1.25-fold increase for older adults - suggesting a substantial degradation in peripheral sensory input for the neuropathy patients and a greater reliance on visual feedback when available (Figure 3B). The postural instability observed in our peripheral neuropathy subjects agrees with previous investigations (Simoneau, Ulbrecht, Derr, Becker, & Cavanagh, 1994). In Simoneau's earlier study of peripheral neuropathy and postural control (Simoneau, 1992), older adults with diabetic neuropathy exhibited a 21% greater anterior-posterior sway range than age-matched diabetic patients without peripheral neuropathy and 27% greater AP sway range than age-matched control subjects. These findings were replicated by Katoulis, et al. who showed that postural stability was diminished by 21% in diabetic neuropathy patients compared with age-matched diabetics without peripheral neuropathy and age-matched non-diabetics (Katoulis et al., 1997). The greater postural sway in the peripheral neuropathy subjects compared with the older and young adults may be due, in part, to the decreased ability to sense plantar sensation demonstrated in the present study. Our findings regarding impaired postural control, in conjunction with our findings of reduced plantar sensation, agree with a study of diabetic neuropathy patients with impaired Semmes-Weinstein filament sensibility, who displayed greater postural instability than diabetic neuropathy patients with normal sensation and healthy controls (Simmons, Richardson, & Pozos, 1997).

Interestingly, findings from the present study and those regarding reduced lower limb sensation align with a postural stability strategy posited by Cavanagh, *et al.* in which neuropathic subjects may employ greater amounts of sway in order to boost the amount of afferent feedback and maintain postural stability in the face of decreased proprioceptive and somatosensory feedback from the periphery (Cavanagh, Simoneau, & Ulbrecht, 1993). Our results may lend support to this notion, in that postural sway was increased without visual feedback to a greater degree for peripheral neuropathy subjects compared to older adults, potentially suggesting a strategy for exploring the boundaries of sway to obtain greater peripheral feedback for maintenance of posture.

Force steadiness – postural stability correlations—The line of pull of gravity on the body's center of mass is just ahead of the center of rotation of the ankle joint, such that during standing the plantarflexors are activated at low levels to produce a controlled, counteracting ankle torque that limits anterior excursions of the center of pressure and body mass around the base of support. In this scheme, impaired output (greater variability) of

plantarflexor torque could contribute to the postural instability observed in the elderly (Horak, Shupert, & Mirka, 1989). Furthermore, both the fluctuations in ankle torque during standing and the plantarflexor force fluctuations observed during isolated muscle group steadiness tasks are similarly dominated by low frequencies (0–3 Hz) (Masani et al., 2003), supporting the idea that muscle force unsteadiness and postural instability could be correlated.

A very small body of literature has recently emerged on the relation between postural steadiness and ankle force production. Oshita and Yano (2012) assessed submaximal force steadiness during isolated plantarflexor motor tasks and center of mass displacement during standing in young men (Oshita & Yano, 2012). In contrast to our young subjects, they found a correlation between the amplitude of anterior-posterior center of mass displacement and the amplitude of isometric force fluctuations at 10% MVC. This relationship can be mechanistically predicted as alterations in the center of pressure during quiet stance activate a variety of muscles in the lower limb, including the soleus, to maintain upright stability (Masani et al., 2003). This relationship is also reflected in less sophisticated assessments of balance. Single-leg standing balance time, for example, is moderately negatively correlated with force fluctuations at 20% MVC (Oshita & Yano, 2010).

For the older adults with peripheral neuropathy, the current study suggest moderate correlations between postural stability (with the eyes closed) and no-vision force steadiness in the plantarflexor muscles, with no correlations involving the dorsiflexors (Figure 4). This is in slight contrast with the non-neuropathic older adults, who exhibited some moderate correlations, albeit inconsistently, in both visual conditions during postural assessment. Between-group inspection of these correlations suggests an increased reliance on vision during postural maintenance for the older peripheral neuropathy subjects, presumably due to decreased afferent input from the lower limb (Boyd, Wanek, Gray, & Topp, 2010). Impaired plantar pressure sensitivity could reasonably be expected to contribute to a greater reliance on visual input during quiet standing. This notion of a lack of pressure sensitivity is bolstered by the decreased amplitude of sensory nerve action potentials observed in older subjects with peripheral neuropathy (Kokotis & Papadimas, 2012) which may, in turn, contribute to output variability (Masani, Vette, Kawashima, & Popovic, 2008). In addition to decreased amplitude of afferent signaling, increased delays in sensory input, processing and output could lead to impaired force production at the ankle thereby leading to potentially decreased postural stability (Masani et al., 2008). This notion is logical from the perspective of peripheral neuropathy because decreased afferent signal strength secondary to a reduced abundance of sensory neurons (Narayanaswamy et al., 2012) may lead to an impaired likelihood of action potential initiation at target neurons further upstream. This decreased likelihood of action potential initiation effectively increases the time between sensory stimulus and motor action - a greater stimulus would be required to elicit a central motor response. Also, Proprioceptive input may play a role in postural stability under the open loop system model. While controlling for vision and vestibular signaling, Fitzpatrick and McCloskey had subjects control an inverted pendulum equivalent to their own body mass with the ankle dorsiflexors and plantarflexors, and concluded that even very small amounts of ankle rotation provided important feedback regarding body sway (Fitzpatrick, Rogers, & McCloskey, 1994).

A study performed by Masani *et al.* also suggests that proprioceptive afferent input at the ankle contributes to muscle activation for postural control (Masani, Sayenko, & Vette, 2013). During free standing, sEMG activity at the soleus and ankle torque was significantly greater than that found at rest, as expected. When anterior shank movement was prevented by a mechanical restraint, soleus sEMG activity and ankle force output were similar to that found at rest. These findings demonstrate that the mechanical action at the ankle during postural sway contributes to activation of muscles to maintain posture by muscle spindle mediated reflex action. For peripheral neuropathy patients, diminished proprioceptive acuity would likely negatively impact postural stability and force output which could, in turn, account for the differences noted between older neuropathic adults, healthy older adults, and young adults.

Recent investigations suggest that restoration of postural stability may be possible with ankle-specific motor training. Four weeks of steadiness training consisting of 60-s constant-force tasks at 10% and 20% MVC, was focused on just the plantarflexors (Oshita & Yano, 2011). The standard deviation of the center of mass velocity was reduced by ~15% during quiet standing trials as a result of the training. It is possible that this mode of training, or other forms of training that require precise control of the ankle muscles (Christou, Yang, & Rosengren, 2003), could improve the postural stability of older adults with peripheral neuropathy (Tracy, Byrnes, & Enoka, 2004). Generally, this training effect supports the notion of a relation between ankle motor output and postural steadiness.

Apparently conflicting results exist upon comparison of force and postural control with regard to the effects of vision. While removal of visuomotor targeting improved force production about the ankle in all groups, removal of vision negatively impacted postural control in peripheral neuropathy patients and older adults. Postural control involves many more types of sensory input (e.g. vestibular input) than does isolated ankle force output. The decline of one or more of these sensory systems could place a greater demand on vision as a contributor to postural stability. The inverse nature of the effect of vision on these tasks, however, does not impact the relation of ankle force production to postural stability.

Limitations

We do not have objective information on ankle or foot sensory acuity for the healthy older adults to positively ensure that they did not have subclinical sensory impairment or neuropathic symptoms. Also, vestibular dysfunction, which could produce postural dyscontrol especially when visual feedback is removed (Nashner, 1972), was not objectively determined with a physical test. These limitations are mitigated, however in that; 1) we queried the healthy older adults about any neurological problems and specifically about any problems with their feet, 2) we obtained physician-confirmed diagnosis of neuropathy for the older adults in the peripheral neuropathy group, 3) objective measures of sensory function clearly confirmed the neuropathic sensory status in the peripheral neuropathy group, and 4) we specifically queried the subjects during the physical examination about inner ear/vestibular issues. In future research, this weakness could be mitigated by performance of the Clinical Test of Sensory Integration and Balance or the Sensory Organization Test. Greater statistical power may also allow potential correlations between

steadiness and/or postural stability and our measures of sensation to reach significance. Proprioception at the ankle contributes to postural stability (van Deursen & Simoneau, 1999), thus objective experimental measures of joint movement perception or joint position perception (Simoneau, Derr, Ulbrecht, Becker, & Cavanagh, 1996) would strengthen the results.

This study sought to describe the steadiness of ankle muscle force in older adults with peripheral neuropathy compared with healthy older adults, and to assess the degree to which the ability to minimize force variability at the ankle was correlated with the ability to minimize postural sway during quiet standing. The finding that force steadiness was impaired for the plantarflexors of older adults with peripheral neuropathy is novel, and suggests that the sensory and/or motor effects of peripheral neuropathy produces significant alterations in motor output of the plantarflexor muscles. The impaired motor output of critical ankle torque appears to be related to the ability to maintain the center of mass in a stable fashion over the base of support, which is an important feature of daily functional ability. Future studies should investigate the relation of ankle force variability and postural control to clinical measures of balance. These comparisons may, in turn, better inform clinical practice and rehabilitation techniques aimed to increase stability in patients with peripheral neuropathy.

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Figure 1. Maximal voluntary contraction force (N) for the (A) plantarflexors and (B) dorsiflexors. *P 0.05 for older healthy adults vs. young healthy adults. †P 0.05 for older peripheral neuropathy subjects vs. young healthy adults.

Figure 2.

Coefficient of variation (CV) of force (%) for the (A) dorsiflexors and (B) plantarflexors. †P 0.05 for older peripheral neuropathy subjects vs. young healthy adults. ‡ P 0.05 for older peripheral neuropathy subjects vs. older healthy adults. § P 0.05 for vision vs. without vision.

Figure 3.

Postural steadiness assessed by (A) postural sway (cm/foot length) and (B) path length (cm).

† $P < 0.05$ for older peripheral neuropathy subjects vs. young healthy adults. ‡ $P < 0.05$ for older peripheral neuropathy subjects vs. older healthy adults. § $P < 0.05$ for vision vs. without vision.

Figure 4. Plantarflexor force steadiness correlation to postural steadiness measures in older peripheral neuropathy subjects. (A) postural sway with vision (cm) (B) postural sway without vision (cm) (C) path length with vision (cm/foot length) (D) path length without vision (cm/foot length).

Table 1

Subject Characteristics

	O-PN	O	Y
Age (yr) ^{*†}	72.2 ± 7.9	74.9 ± 7.6	23.2 ± 3.4
Height (m) ^{*‡}	1.7 ± 0.1	1.6 ± 0.1	1.7 ± 0.1
Mass (kg)	82.1 ± 15.7	74.8 ± 15.4	72.3 ± 13.3
BMI (kg/m²)	28.4 ± 5.4	29.1 ± 5.7	25.1 ± 3.7
Fine Touch Sensation Error Rate (%) (n = 14)	47 ± 34		

^{*}P 0.05, O vs. Y

[†]P 0.05, O-PN vs. Y

[‡]P 0.05, O-PN vs. O