ORIGINAL ARTICLE

Multiple Sclerosis and Postural Control: The Role of Spasticity

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ABSTRACT. Sosnoff JJ, Shin S, Motl RW. Multiple sclerosis and postural control: the role of spasticity. Arch Phys Med Rehabil 2010;91:93-9.

Objectives: To examine the association between spasticity and postural control in subjects with multiple sclerosis (MS).

Design: Cross-sectional.

Setting: Motor control laboratory.

Participants: Subjects with MS (n=16, 2 male) and age and sex-matched subjects (n=16) participated in the investigation. All subjects with MS had Expanded Disability Status Scale scores between 0 and 4.5 and modified Ashworth scale scores between 1 and 3.

Interventions: Not applicable.

Main Outcome Measures: Postural control was measured with a force platform that quantifies ground reaction forces and moments in mediolateral and anteroposterior directions. Postural control was indexed with anterior-posterior sway range, medial-lateral sway range, 95% elliptical area of the deviations of center of pressure (COP), velocity of COP sway, and the frequency at which 95% of spectral profile was contained. Participants with MS further underwent assessment of the soleus Hoffman reflex (H-reflex) as an index of spasticity.

Results: Cluster analysis on H-reflex data identified groups of MS participants with high spasticity (n=7) and low spasticity (n=9). There were no differences in age, duration of MS, and disease severity between MS groups. There were no differences in anterior-posterior sway range between any of the groups. The high spasticity group had greater COP area, velocity, and mediolateral sway compared with the low spasticity and control group, and the low spasticity group had postural control values between the high spasticity and control groups.

Conclusions: The pattern of results suggests that spasticity contributes to postural deficits observed in MS.

Key Words: H-reflex; Rehabilitation.

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MULTIPLE SCLEROSIS is the most prevalent chronic disabling neurologic disease among adults worldwide and in the United States. The National Multiple Sclerosis Society has estimated that there are approximately 400,000 cases of MS in the United States with an incidence of nearly 200 new cases each week. The majority of people with MS are diagnosed between 20 and 50 years of age, and women are affected 2 to 3 times as often as men.[1]

MS involves intermittent bursts of focal inflammation in the CNS.[2] This results in the demyelination and transection of axons in the brain, optic nerves, and spinal cord. The resulting axonal damage leads to conduction delay and conduction block of electrical potentials along neuronal pathways throughout the CNS.[3] The dispersion of axonal dysfunction throughout the CNS is associated with the unpredictable and heterogeneous symptoms of MS.

Spasticity is a common symptom of MS that results from neuronal damage in the long fiber tracts of the CNS. Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes and exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex.[4] Recent evidence from the Patient Registry of the North American Research Committee on MS indicates that 84% of patients with MS reported spasticity.[1] Of those who reported spasticity, 31% reported that the spasticity was minimal, 19% mild (occasional), 17% moderate (frequently affects activities), 13% severe (need to modify daily activities), and 4% total (prevents daily activities).

There are multiple methods to quantify spasticity including the tendon tap, the H-reflex, scales of muscle tone, and the modified Ashworth scale.[5] All techniques indicate a specific aspect of spasticity. For instance, the H-reflex is a low-threshold spinal reflex that results from the electrical stimulation of the peripheral nerve. It is believed to be indicative of alpha motor neuron excitability. People with spasticity have increased alpha motor neuron excitability and increased H-reflex amplitude.[5]

There is evidence indicating that spasticity is associated with mobility impairments in MS. For example, evidence from the North American Research Committee on MS Patient Registry indicates that spasticity is associated with mobility impairments as measured by the Patient-Determined Disease Steps scale in persons with MS.[1] Those with progressively worse spasticity transition from normal mobility to mild/moderate gait disability, early and late cane use, and then bilateral support and wheelchair dependence.

The maintenance of upright stance or postural control requires the integration of multiple sensorimotor processes (visual, vestibular, proprioception) to generate coordinated movements that maintain the center of mass within the limits of stability.[6,7] Any adverse alteration in these processes results in impaired postural control. Unfortunately, subjects suffering from MS have impairments in some if not all of these pro-

List of Abbreviations

ANOVA	analysis of variance
AP	anterior-posterior
CNS	central nervous system
COP	center of pressure
EDSS	Expanded Disability Status Scale
EMG	electromyography
H-reflex	Hoffman Reflex
ML	mediolateral
MS	multiple sclerosis

cesses. As such, it is not surprising that subjects with MS have poorer postural control indicated by greater amounts of postural sway compared with healthy controls.[8-12] For instance, Rougier et al[12] showed that subjects with MS had greater postural sway than normal controls. The authors speculated that the deficits in postural control stem from either sensory information processing and/or motor impairments such as spasticity.

The association between spasticity and postural control is based on the notion that postural control results in part from the modulation of ankle stiffness.[13] This view maintains that subjects with spasticity are less able to modulate their ankle stiffness because of greater higher reflex modulation and, consequently, have poorer postural control (ie, greater postural sway). Although there is limited empirical evidence supporting the notion that spasticity is associated with poor postural control in persons with MS, Angulo-Kinzler,[14] Koceja,[15] and colleagues have shown a relationship between H-reflex amplitude and postural control. Specifically, it was shown that older adults (ie, age >65y) who showed higher reflex activity (ie, greater H-reflex amplitude) in the ankle musculature when standing showed greater postural sway. As such, it is logical to speculate that subjects with MS and spasticity as indexed with H-reflex will have greater postural sway.

This study involved a pilot investigation of the association between spasticity and postural control in subjects suffering from MS. We hypothesized that subjects with MS with elevated spasticity would have greater amounts of postural sway compared with subjects with MS with less spasticity and healthy controls.

METHODS

Participants

Sixteen subjects with MS (2 males) and 16 age- and sex-matched controls participated in the investigation. All subjects with MS had Expanded Disability Status Scale scores between 0 and 4.5 and modified Ashworth Scale scores between 1 and 3. The MS group included 16 subjects with either relapsing-remitting (n=14), primary progressive (n=1), or secondary progressive (n=1) MS. On average, it had been 7.8 years since the diagnosis of MS, with a range between 0.5 and 22 years. All subjects currently using oral or intrathecal antispastic medications were excluded. The MS group had a mean age of 44.4 years and ranged from 20 to 60 years old, whereas the control group had a mean age of 44.3 years and ranged from 20 to 60 years old.

Measures

The H-reflex served as an electrophysiologic index of spasticity and was measured in the soleus muscle of the right leg with the participant with MS in a comfortable semireclined position. The H-reflex was evoked by stimulating the tibial nerve in the popliteal fossa through a monopolar stimulating electrode with an anode placed superior to the patella. The stimulus was a single, 1-millisecond rectangular pulse delivered every 10 seconds. The H-reflex was measured by using bipolar electrodes placed 2cm apart along the ipsilateral soleus muscle and standard EMG. The EMG signal was band-pass filtered, amplified by 1000, and sampled at 2500Hz. We generated an H-reflex recruitment curve by progressively increasing the intensity of the stimulation in an effort to locate the largest obtainable H wave and M wave measured as peak-to-peak amplitude of the nonrectified wave. The maximal H wave and maximal M wave were then measured as an average of 5 subsequent recordings of the largest obtainable M wave and M wave, respectively. The maximal H-reflex was expressed as the maximal H wave/maximal M wave ratio.

All participants underwent postural control testing. Participants stood on dual AMTI force platforms[a] with each foot on an individual force platform shoulder width apart. Each platform records postural dynamics with 3 force components: the ML force (Fx), the anteroposterior force (Fy), and the vertical force (Fz) and 3 moment components: Mx, My, and Mz, which are the moments taken about the respective axes. The signals were amplified with a gain of 4000 through a 6-channel AMTI model Sga6-4 AMPLIFIER.[a] The platform excitation voltage was set to 10V. All 6 channels were factory calibrated. Data were collected with a sample frequency of 100Hz with a low pass filter of 10.5Hz. Four 30-second trials were completed.

The posture analysis was based on the motion of the COP. This component of postural data may be considered a reflection of the system's neuromuscular response to the imbalances of the body's center of gravity.[7] The COP in the AP and ML direction was determined according to the following equations:

$$COP_{AP} = \frac{(Fz1 * COPx1) + (Fz2 * COPx2)}{Fz1 + Fz2}$$

where COP_{AP} is the overall center of pressure along the AP axis, Fz1 is the vertical force from force plate 1, Fz2 is the vertical force from force plate 2, COPx1 is the COP parallel to AP axis calculated from force plate 1, and COPx2 is the COP parallel to AP axis calculated from force plate 2 and

$$COP_{ML} = \frac{(Fz1 * COPy1) + (Fz2 * COPy2)}{Fz1 + Fz2}$$

where COP_{ML} is equal to the overall COP along the ML axis, Fz1 is the vertical force from force plate 1, Fz2 is the vertical force from force plate 2, COPy1 is the COP parallel to ML axis calculated from force plate1, and COPy2 is the COP parallel to the ML axis calculated from force plate 2.

To quantify participants' postural control, the amount of motion of the COP was indexed with several measures. First, a 95% confidence ellipse that has been shown to be a robust indicator of the amount of postural motion[16] was calculated. Additionally, the sway range in the AP and ML direction was indexed. The velocity of the sway was also calculated.

Spectral analysis, which allows for the decomposition of a signal into its frequency components, was also conducted. Specifically, the fast Fourier transform was calculated by using the FFT command in Matlab 14.0,[b] which uses Welch's averaged periodogram method. A 512-point 50% overlapping Hanning window was used, with a sampling frequency of 100Hz, resulting in a 0.39-Hz bin width. The power in each bin represented the amplitude of the COP oscillations that occur at the frequency specified by the bin. To characterize the effect of spasticity on the spectral profile of postural control, 95% power spectrum from the frequency was derived.[7]

Procedures

All participants received a brief verbal explanation of the experimental protocol and were given an opportunity to ask questions. The participants subsequently provided written informed consent. The participants with MS underwent assessment of the H-reflex, and all participants underwent assessment of postural control. Participants did not receive monetary remuneration.

Table 1: Age, MS Duration, EDSS Score, and H-Reflex/M Wave Ratio as a Function of Group

Variable	High Spasticity Group (n=9)	Low Spasticity Group (n=7)	Control Group (n=16)
Age (y)	43.8±3.9	45.0±4.4	44.3±2.9
Female/male	7/2	7	14/2
MS duration (y)	9.2±2.2	5.9±2.5	NA
EDSS score	3.6±0.6	2.9±0.7	NA
H-reflex/M wave M wave ratio	.80±.06*	0.43±.07*	NA

NOTE. Values are mean ± SD except for female/male ratio.
Abbreviation: NA, not applicable.
*$P<.05$.

Data Analysis

The data were initially examined for normality violations, outliers, and errors. Cluster analysis was performed on H-reflex data as a means of generating 2 groups of subjects with MS who had high (n=9) or low (n=7) spasticity. The dependent variables of sway area, sway velocity, AP, and ML sway range were averaged across the 4 trials and then entered into univariate ANOVAs with group (control, low spasticity, high spasticity) as the between-subject factor. Main effects were decomposed by using post hoc analyses with a correction of alpha. Effect sizes associated with F ratios were expressed as eta squared (η^2).

RESULTS

Descriptive Characteristics

The descriptive characteristics of the high spasticity, low spasticity, and control groups are provided in table 1 as mean scores ± SD. The independent samples *t* tests did not identify any significant differences in the demographic variables of age, duration of MS, and EDSS scores between high spasticity and low spasticity groups.

Spasticity: Maximal H wave/Maximal M wave Ratio

The maximal H wave/maximal M wave ratio data are provided in table 1. The univariate ANOVA on maximal H wave/maximal M wave ratio values identified a statistically significant effect of group ($F_{1,14}=15.84$; $P<.01$; $\eta^2=.53$). Per design, the low spasticity group (maximal H wave/maximal M wave ratio=.44) had significantly lower H-reflex values as an electrophysiologic index of spasticity than the high spasticity group (maximal H wave/maximal M wave ratio=.80).

Postural Sway

Figure 1 shows the COP trajectory of a control subject, an individual with MS with low spasticity, and an individual with MS with high spasticity. It is clear in the figure that both subjects with MS have greater sway than the healthy control and that the greatest difference in postural sway is between the healthy control and the individual with high spasticity. To quantify these differences, various postural sway measures were determined and examined as a function of groups. Statistical analysis of the postural sway area identified a main effect of group ($F_{2,30}=10.89$; $P<.05$; $\eta^2=.41$). Post hoc analysis revealed that the high spasticity group had greater sway (238.9mm^2) compared with the low spasticity (100.9mm^2) and control (38.1mm^2) groups ($P<.05$, $\eta^2=.48$, .35) (fig 2). There was a trend for the low spasticity group to have greater sway

than the control group, but traditional levels of significance were not reached ($P=.13$, $\eta^2=.23$).

Figure 3 depicts sway velocity as a function of group. The 1-way ANOVA revealed a main effect for group ($F_{2,30}=13.0$; $P<.05$; $\eta^2=.47$). Post hoc analysis revealed that the control group (3.04mm/s) had lower sway velocity compared with the high spasticity (10.9mm/s) and low spasticity (6.58mm/s) groups ($P<.05$, $\eta^2=.54$, .41). There was a trend for the high spasticity group to have greater sway velocity than the low spasticity group, but again traditional levels of significance were not reached ($P=.06$, $\eta^2=.34$).

The effect of spasticity on AP and ML sway range is shown in figure 4A and B. Figure 4A shows that there was no group difference in anterior-posterior sway length ($F_{2,30}=0.06$; $P>.05$; $\eta^2=.00$). In contrast, examination of medial-lateral sway length revealed a main effect for group ($F_{2,30}=10.6$; $P<.05$; $\eta^2=.42$) (see fig 4B). Post hoc analysis revealed that the high spasticity group (10.28mm) had greater ML sway range than the low spasticity group (6.59mm) and the control group (2.32mm) ($P<.05$, $\eta^2=.49$, .36). There was no significant difference between the low spasticity and control group ($P>.05$).

Figure 5A and B shows the effect of spasticity on AP and ML sway spectral profiles. There was no group difference on the frequency at which 95% proportion of power was contained of the AP sway ($F_{2,30}=.19$; $P>.05$; $\eta^2=.00$). However, examination of ML sway spectral profile yielded a main effect for group ($F_{2,30}=4.91$; $P<.05$; $\eta^2=.21$). Post hoc analysis revealed that the high spasticity group (1.30Hz) had a more dispersed spectral profile compared with the control (0.88Hz) and low spasticity group (0.93Hz) ($P>.05$, $\eta^2=.22$, .17). There was no difference between the control group and the low spasticity group ($P>.05$).

DISCUSSION

Persons with MS exhibit decreased postural control,[8,12] and these postural deficits are paramount in dynamic movement tasks.[9,10] The reduction in postural control, in part, increases the risk of falls in subjects with MS.[17,18] This investigation examined the relation between spasticity and postural control in subjects with MS, and we observed 2 novel findings: (1) subjects with elevated spasticity have significantly greater postural sway compared with healthy controls and subjects with MS with lower levels of spasticity and (2) the increase in postural sway is predominantly in the ML direction.

Spasticity, the velocity-dependent increase in tonic stretch reflexes and exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex,[4] is believed to decrease an individual's ability to modulate ankle stiffness, a primary component of postural control.[13] Importantly, previous research[8,12] has reported postural control dysfunction in MS, but the vast majority did not differentiate participants based on the level of spasticity. Within the current investigation, all of the participants with MS reported some amount of spasticity on self-report and had modified Ashworth scale scores between 1 and 3 because it was an inclusion criterion, but the severity varied across subjects. The subjects with lower levels of spasticity, as indexed by the H-reflex, had better postural control (reduced sway, sway velocity) than the high spasticity group. Moreover, the low spasticity group's postural control was similar to that of the healthy control group on several measures (ie, AP and ML sway, spectral profile). It is important to note there was no difference in age, duration of disease, or severity (EDSS score) between MS groups. This similarity between the groups strengthens the proposal that spasticity in and of itself is a contributing factor in postural dysfunction in subjects with MS.
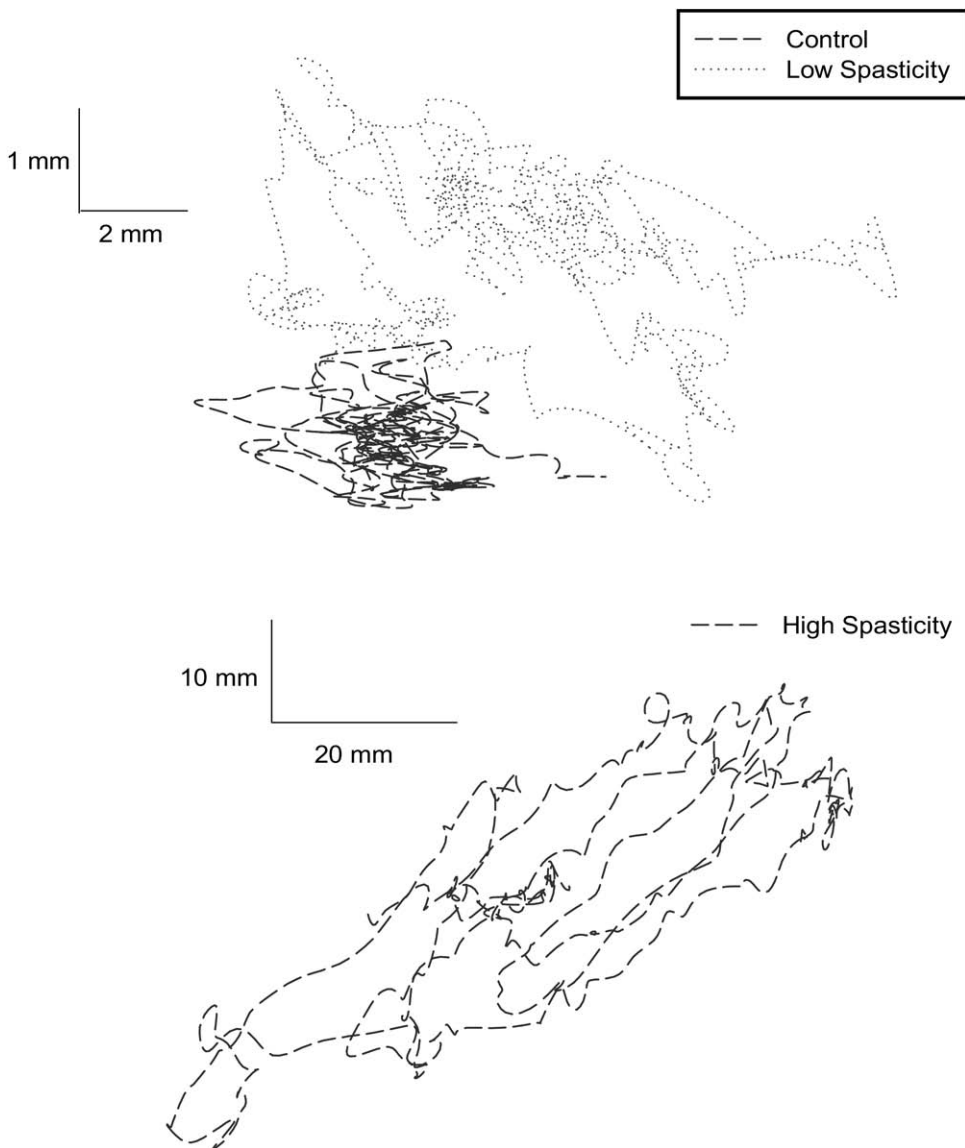
Fig 1. Representative COP for a healthy control subject with MS with low spasticity and a subject with high spasticity.

Indeed, the expected value for the maximal H wave/maximal M wave ratio in a healthy subject is approximately 0.5, and this is similar to the value of the maximal H wave/maximal M wave ratio reported for the low spasticity group. This in combination with the observation of minimal differences between the control group and the low spasticity group further indirectly supports the notion that spasticity results in postural dysfunction. Although not surprising, this is the first direct empiric report showing that the level of spasticity is a contributor to postural dysfunction in subjects with MS.

Reports[9,10] have suggested that there are minimal differences in postural control between subjects with MS and those without in quiet stance. These reports have mainly looked at postural sway along the AP direction and have not considered spasticity. Within the current investigation, postural control was examined in both the AP and ML direction. The examination of both directions of sway is based on reports that AP and ML sway are separately controlled and unrelated.[19] Congruent with previous research,[9,10] there were no group differences in AP sway, but the high spasticity group did have greater sway in the

ML direction than the low spasticity and control groups. Although replicating previous research,[19,20] the lack of group differences in AP sway is surprising given that AP sway is believed to result from muscular torque acting at the ankle via plantar flexion. A priori subjects with elevated spasticity recorded in the soleus muscle (a prime mover of the ankle) were expected to have greater AP (ie, forward to backward) sway. It is possible that subjects with elevated spasticity used a postural control strategy that is dominated by movement around the hip or knee and minimized movement around the ankle.

ML sway mainly results from abductor and adductors activity at the hip[19] and was unexpected to be related to spasticity at the ankle. It is possible that the increased ML sway shown by the high spasticity group results as a compensatory strategy resulting from increased "stiffness" at the ankles. Indirect support for this notion comes from the increased distribution of power in the spectral profile of the ML COP trajectory in the high spasticity group compared with the healthy controls. A greater distribution of power indicates a greater number of movements. Moreover, the decreased postural control observed
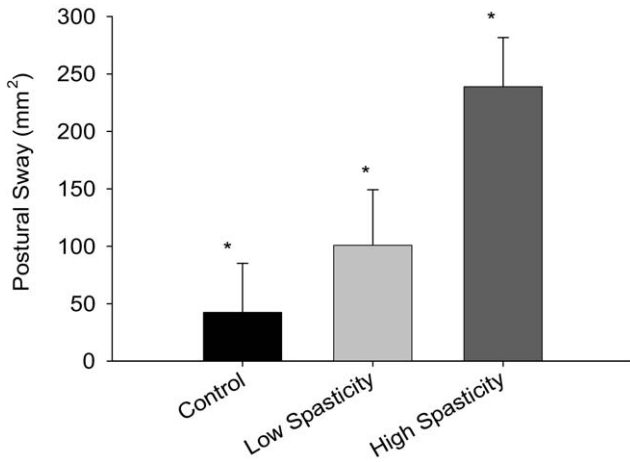
Fig 2. Ninety-five percent elliptical area of COP deviation as a function of group. Values are means ± SE. *Group difference, *P*<.05.

in subjects with MS with higher levels of spasticity is congruent with previous work[14,15] showing that subjects with elevated H-reflex amplitude at the ankle have greater postural sway. As pointed out by one of the reviewers, it has been suggested that ML sway is controlled more reflexively than AP sway. This notion is congruent with the altered reflex pathway in subjects with elevated H-reflex amplitude. Regardless of the mechanism(s) driving increased ML sway, increased ML sway is of paramount importance because of its association with falls.[21] The increased ML sway could be a partial explanation for the increased incidence of falls in subjects with MS.[17,18,22] The current findings also suggest that rehabilitation interventions aimed at reducing fall risk in subjects with MS should focus on exercises that target the control of ML sway, such as strengthening hip abductors and adductors and dynamic balance training, as well as reduce spasticity.

Postural dysfunction in subjects with MS is multifaceted. Adverse alterations in the sensory processing (vision, vestibular, proprioception) that contribute to postural control are common in MS.[23] Obviously, deficits in visual, vestibular, or proprioceptive processing will have detrimental consequences to
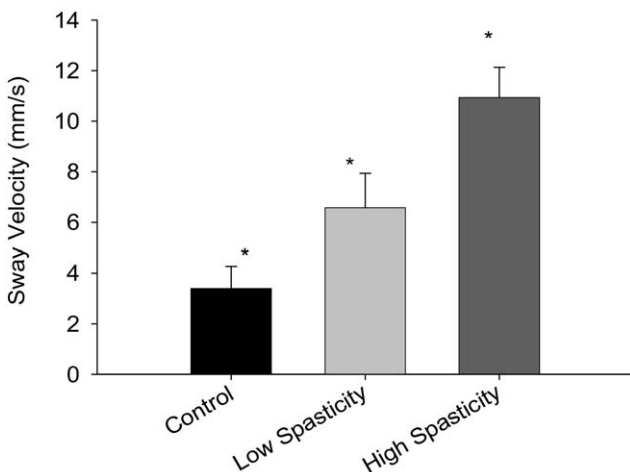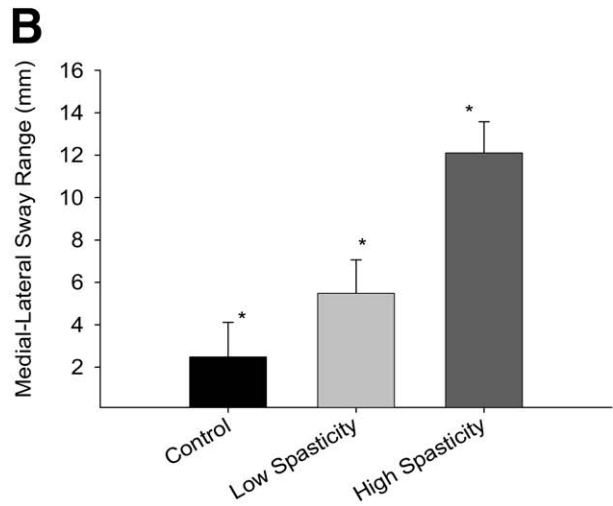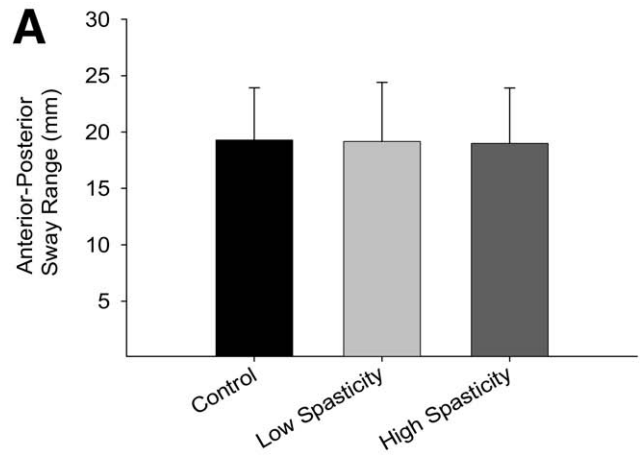

Fig 4. (A) Sway range in the AP direction as a function of group. (B) Sway range in the ML direction as a function of group. Values are means ± SE. *Group difference, *P*<.05.

Fig 3. Sway velocity as a function of group. Values are means ± SE. *Group difference, *P*<.05.

postural control. It is logical to speculate that the high spasticity group also had greater deficits in sensory processing. Unfortunately, no data were collected to examine this possibility. However, there was no significant difference in disease severity as indexed by EDSS scores between spasticity groups so this possibility is less likely.

Although spasticity is traditionally quantified in the musculature around the ankle, it is quite possible that subjects with MS have spasticity throughout their body. Congruent with this traditional practice, only H-reflex data from the right soleus muscle were collected in the current investigation. It is logical to speculate that subjects with greater spasticity around the ankle also have greater spasticity at the hip. An alternative proposal to the increased ML sway in subjects with higher levels of spasticity could be an inability to modulate musculature around the hip because of spasticity. Unfortunately, no data on spasticity in hip muscles were recorded in the current investigation. Further work is needed to examine this possibility.

Although it is intuitive to speculate based on the current findings that reductions in spasticity will lead to increased postural control, the evidence to support this claim is lacking. There are numerous investigations showing either a reduction
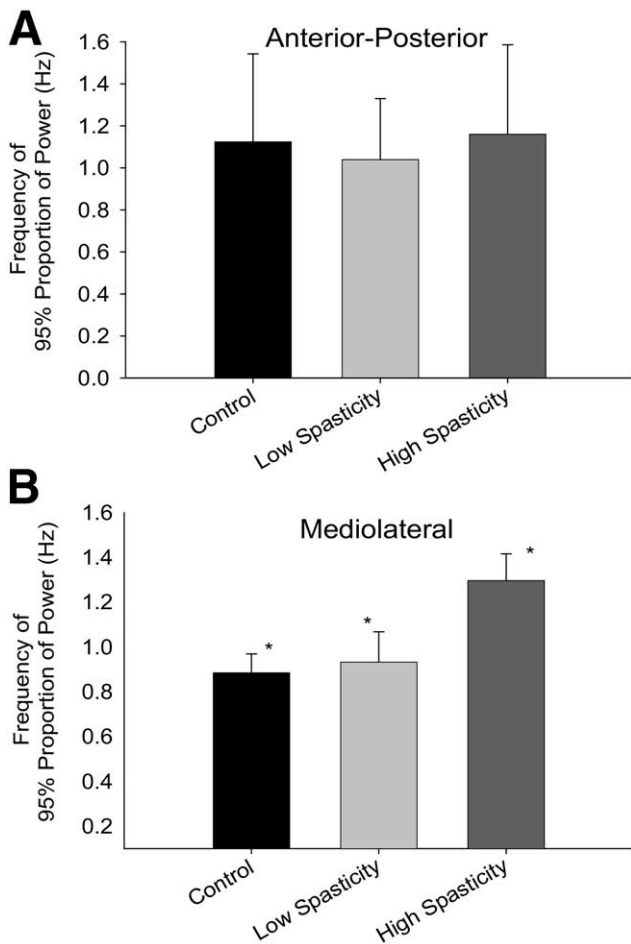
Fig 5. (A) Frequency of 95% proportion of power in the AP direction as a function of group. (B) Frequency of 95% proportion of power in the ML direction as a function of group. Values are means ± SE. *Group difference, $P < .05$.

in spasticity or an increase in postural control via pharmaceutical treatment[23,24] or behavioral modifications.[25,26] To our knowledge, there have been no reports of a reduction in spasticity being associated with improvements in postural control within subjects with MS. However, improvements in postural control and/or gait with reductions in spasticity have been noted in other populations including patients with cerebral palsy.[27] Future work is needed to examine the possibility that acute and chronic reductions in spasticity are indeed associated with improvements in postural control in subjects with MS.

CONCLUSIONS

The current investigation supported previous reports that subjects with MS have postural dysfunction. Two novel findings involving postural control and MS were revealed. First, subjects with MS were found to have increased ML sway compared with healthy controls. Second, subjects with MS with increased levels of spasticity had the greatest amount of ML sway. The association between ML sway and fall risk makes this observation of the utmost importance. Overall, the findings indicate that spasticity plays a significant role in postural deficits observed in multiple sclerosis.

References

1. Rizzo MA, Hadjimichael OC, Preiningerova J, Vollmer TL. Prevalence and treatment of spasticity reported by multiple sclerosis patients. Mult Scler 2004;10:589-95.
2. Hemmer B, Nessler S, Zhou D, Kieseier B, Hartung HP. Immunopathogenesis and immunotherapy of multiple sclerosis. Nat Clin Pract Neurol 2006;2:201-11.
3. Bjartmar C, Trapp BD. Axonal and neuronal degeneration in multiple sclerosis: mechanisms and functional consequences. Curr Opin Neurol 2001;14:271-8.
4. Lance JW. The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. Neurology 1980;30:1303-13.
5. Voerman GE, Gregoric M, Hermens HJ. Neurophysiological methods for the assessment of spasticity: the Hoffmann reflex, the tendon reflex, and the stretch reflex. Disabil Rehabil 2005;27:33-68.
6. Peterka RJ. Sensorimotor integration in human postural control. J Neurophysiol 2002;88:1097-118.
7. Winter DA. Biomechanics and motor control of human movement. New York: Wiley; 1990.
8. Corradini ML, Fioretti S, Leo T, Piperno R. Early recognition of postural disorders in multiple sclerosis through movement analysis: a modeling study. IEEE Trans Biomed Eng 1997;44:1029-38.
9. Frzovic D, Morris ME, Vowels L. Clinical tests of standing balance: performance of persons with multiple sclerosis. Arch Phys Med Rehabil 2000;81:215-21.
10. Karst GM, Venema DM, Roehrs TG, Tyler AE. Center of pressure measures during standing tasks in minimally impaired persons with multiple sclerosis. J Neurol Phys Ther 2005;29:170-80.
11. Remelius JG, Hamill J, Kent-Braun J, Van Emmerik RE. Gait initiation in multiple sclerosis. Motor Control 2008;12:93-108.
12. Rougier P, Faucher M, Cantalloube S, Lamotte D, Vinti M, Thoumie P. How proprioceptive impairments affect quiet standing in patients with multiple sclerosis. Somatosens Mot Res 2007;24:41-51.
13. Gatev P, Thomas S, Kepple T, Hallett M. Feedforward ankle strategy of balance during quiet stance in adults. J Physiol 1999;514:915-28.
14. Angulo-Kinzler RM, Mynark RG, Koceja DM. Soleus H-reflex gain in elderly and young adults: modulation due to body position. J Gerontol A Biol Sci Med Sci 1998;3:M120-5.
15. Koceja DM, Markus CA, Trimble MH. Postural modulation of the soleus H reflex in young and old subjects. Electroencephalogr Clin Neurophysiol 1995;97:387-93.
16. Goldie PA, Bach TM, Evans OM. Force platform measures for evaluating postural control: reliability and validity. Arch Phys Med Rehabil 1989;70:510-7.
17. Cattaneo D, De Nuzzo C, Fascia T, Macalli M, Pisoni I, Cardini R. Risks of falls in subjects with multiple sclerosis. Arch Phys Med Rehabil 2002;83:864-7.
18. Finlayson ML, Peterson EW, Cho CC. Risk factors for falling among people aged 45 to 90 years with multiple sclerosis. Arch Phys Med Rehabil 2006;87:1274-9.
19. Winter DA, Prince F, Frank JS, Powell C, Zabjeck KF. Unified theory regarding A/P and M/L in quiet stance. J Neurophysiol 1996;75:2334-43.
20. Okada M, Fujiwara K. Relation between sagittal distribution of the foot pressure in upright stance and relative EMG magnitude in some leg and foot muscles. J Hum Ergol (Tokyo) 1984;13:97-105.
21. Piirtola M, Era P. Force platform measurements as predictors of falls among older people—a review. Gerontology 2006;52:1-16.

22. Peterson EW, Cho CC, Finlayson ML. Fear of falling and associated activity curtailment among middle aged and older adults with multiple sclerosis. Mult Scler 2007;13:1168-75.

23. Clanet MG, Brassat D. The management of multiple sclerosis patients. Curr Opin Neurol 2000;13:263-70.

24. Kesselring J, Beer S. Symptomatic therapy and neurorehabilitation in multiple sclerosis. Lancet Neurol 2005;4:643-52.

25. Giesser B, Beres-Jones J, Budovitch A, Herlihy E, Harkema S. Locomotor training using body weight support on a treadmill improves mobility in persons with multiple sclerosis: a pilot study. Mult Scler 2007;13:224-31.

26. Motl RW, Snook EM, Hinkle ML, McAuley E. Effect of acute leg cycling on the soleus H-reflex and modified Ashworth scale scores in individuals with multiple sclerosis. Neurosci Lett 2006;406:289-92.

27. Brochard S, Remy-Neris O, Filipetti P, Bussel B. Intrathecal baclofen infusion for ambulant children with cerebral palsy. Pediatr Neurol 2009;40:265-70.

Suppliers