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Imbalance in Multiple Sclerosis: A Result of Slowed Spinal Somatosensory Conduction

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Abstract

Balance problems and falls are common in people with multiple sclerosis (MS) but their cause and nature are not well understood. It is known that MS affects many areas of the central nervous system that can impact postural responses to maintain balance, including the cerebellum and the spinal cord. Cerebellar balance disorders are associated with normal latencies but reduced scaling of postural responses. We therefore examined the latency and scaling of automatic postural responses, and their relationship to somatosensory evoked potentials (SSEPs), in 10 people with MS and imbalance and 10 age-, sex-matched, healthy controls. The latency and scaling of postural responses to backward surface translations of 5 different velocities and amplitudes, and the latency of spinal and supraspinal somatosensory conduction, were examined. Subjects with MS had large, but very delayed automatic postural response latencies compared to controls ($161\text{ms} \pm 31$ vs 102 ± 21 , $p < 0.01$) and these postural response latencies correlated with the latencies of their spinal SSEPs ($r=0.73$, $p < 0.01$). Subjects with MS also had normal or excessive scaling of postural response amplitude to perturbation velocity and amplitude. Longer latency postural responses were associated with less velocity scaling and more amplitude scaling. Balance deficits in people with MS appear to be caused by slowed spinal somatosensory conduction and not by cerebellar involvement. People with MS appear to compensate for their slowed spinal somatosensory conduction by increasing the amplitude scaling and the magnitude of their postural responses.

Keywords

Multiple sclerosis; balance; proprioception; cerebellum; posturography; somatosensory evoked potentials

Introduction

Many people with MS report having poor balance. Impaired balance can result in falls and injury, and reduced activity participation. Although the incidence of falls in people with MS is not well documented, clinicians report that most of their patients with MS have balance difficulties. One study found that 54% of a group of 50 patients with MS reported 1 or more falls in the previous 2 months and 32% reported 2 or more falls in the previous 2 months (Cataneo et al. 2002). In this study, people who fell 2 or more times in the previous 2 months were found to have poorer balance than nonfallers. The authors of this study concluded that, “falls in the MS population commonly result from the accumulation of multiple impairments.”

Given the wide distribution of CNS lesions in people with MS, poor balance control in this population may be caused by impairments in cognitive, visual, vestibular, motor, sensory and/or coordination functions, and the contributions of each of these factors may vary among individuals (Speers et al. 2002). However, it is also possible that certain lesion locations and resultant impairments are the primary contributors to imbalance in this population.

It is a commonly held belief that cerebellar lesions are the primary cause of imbalance in MS because the gait of patients with MS can appear ataxic and because cerebellar dysfunction is known to cause ataxia and imbalance when it occurs in association with other diseases and disorders (Thach 2004). The MS Information Sourcebook produced by the National MS society describes balance problems in MS as “typically resulting in a swaying and “drunken” type of gait known as ataxia,” and, the first reason given by the National MS society website for loss of balance in people with MS is “damage in the brain, usually in the cerebellum”. However, to date, no studies clearly make an association between cerebellar dysfunction, or other specific areas of central nervous system lesions, and balance deficits in people with MS.

Balance disorders can be evaluated by quantifying automatic postural responses to surface displacements and by comparing these responses to those of people without balance difficulties and to those of people with various specific neurologic disorders (Horak 1997). Subjects without balance disorders respond to backwards surface translations by quickly contracting their gastrocnemius muscles to prevent falling forward at the ankles. This automatic postural response normally occurs 70–100 milliseconds (ms) after the platform starts to move, keeping the person’s center of mass within their base of support (Horak, Macpherson 1996). In addition, subjects without balance disorders scale the magnitude of their postural response based on somatosensory detection of displacement velocity (Horak et al 1989). They can also scale the magnitude of their postural response to predictable amplitudes of displacement, but this requires expectation based on prior experience of a sequence of trials with consistent displacement amplitudes (Horak 1989, Horak 1990). Postural responses can only be scaled to displacement amplitude when displacements of different amplitudes are presented in predictable blocked sets because the response must be initiated before the platform starts to move. Thus, velocity scaling is reactive, in response to proprioceptive coding of stimulus velocity during the trial whereas amplitude scaling is predictive, based on memory from prior experience.

Previous studies have shown that people with neurologic disorders associated with poor balance have typical patterns of abnormal postural responses. People with cerebellar disorders have normal postural response latencies, larger than normal response magnitudes and normal reactive scaling to displacement velocities but, they do not scale the magnitude of their responses based on prediction of perturbation amplitudes (Horak 1994, Timmann 1997). In contrast, people with peripheral neuropathy caused by diabetes have delayed postural response latencies (by about 40 ms), reduced reactive velocity scaling but normal predictive amplitude scaling (Inglis et al.1994).

The aim of this study was to gain a better understanding of the causes of imbalance in people with MS. To achieve this we examined postural responses in people with MS and in healthy controls. In addition, in order to assess the contribution of slowed CNS conduction in MS to balance impairments, the relationships between somatosensory evoked potential (SSEP) latencies and postural responses were also examined. Since measures of postural performance reflect the effects of neural deficits and the nervous system's attempts to compensate for these deficits, we also examined the relationships between delayed postural response latencies and postural response scaling.

A better understanding of the cause(s) of impaired balance in MS may facilitate development and application of interventions that will improve balance, or compensate for deficits, in people with MS and thus minimize the functional impact of balance impairments in this population. Additionally, a valid and reliable measure of balance in this population may be used as functional outcome measure in trials of symptomatic interventions intended to improve nerve conduction in people with MS.

Materials and Methods

Subjects

The subjects were 10 patients with MS aged 39–52 years, with complaints of balance difficulties, and mild to moderate levels of disability (Table 1). None of the subjects had been treated with any disease modifying agents. Ten healthy people aged 25–51 years without neurologic or orthopedic limitations served as controls. There was no significant difference in age or weight between the MS and control groups ($p>0.05$). All subjects received informed consent for protocols approved by the Institutional Review Board and had a complete clinical neurological evaluation by one of the authors (RH).

Protocol

Balance Platform Measures—This study examined, in free-standing subjects, the timing and amplitude of automatic postural responses to backward displacement of the support surface and the scaling of the amplitude of these responses to changes in the velocity and amplitude of the displacement (Figure 1A). Previous publications have described, in detail, the apparatus and methods used to impose displacements and quantify the resulting responses (Diener et al. 1988, Horak 1986, Horak et al. 1989). The subjects stood on two computer-servocontrolled, custom-made, hydraulic platforms that translated backward together causing forward body sway (Horak 1986). Subjects stood with arms folded across the waist, eyes open, and feet a comfortable 6–9 cm apart at the heels.

Responses to a range of platform displacement amplitudes and velocities were examined. Five different displacement amplitudes (1.2, 3.6, 6.0, 8.4, and 12 cm), all at a velocity of 15 cm/second, were presented in blocks of 7 repetitions, each with increasing amplitude, for a total of 35 trials. This block presentation was used to allow for predictive amplitude scaling based on experience from prior trials (Horak et al. 1989). Five different displacement velocities (10, 15, 20, 25 and 35 cm/second), all of 6 cm amplitude, were presented 5 times each in random order, for a total of 25 trials. This random presentation was used because velocity scaling of postural responses is reactive to the initial rate of movement of the support surface (Horak et al. 1989). The time between displacements was randomly varied by the experimenter (5–20 sec) and was delayed until the subjects obtained the same initial position of the whole body center of pressure as observed on an oscilloscope.

Surface EMG activity was recorded using two-2.5 cm² surface electrodes placed 2 cm apart on the right and left medial gastrocnemius muscles. Amplified EMG signals were band-pass

filtered (70–2,000Hz), rectified and stored for off-line analysis. Although no attempt was made to calibrate EMGs on an absolute scale, amplifier gains were fixed throughout all experimental sessions for all subjects. The latency of the postural response was the time from the onset of surface displacement to the first measurable increase in activity greater than 2 SD from baseline that was sustained at least for at least 50 ms. Each EMG latency was measured with 2 ms accuracy with reference to platform displacement onset.

The plantarflexion torque of each foot on the platform and EMG activity of the left and right medial gastrocnemius were measured starting 200 ms before, and ending 2000 ms after, the onset of surface translation. Plantarflexion torque under each foot was calculated from information from four vertical strain gauges embedded under each hydraulic platform. The slopes of the first 100 ms of rate-of-change of surface reactive torque were measured for each foot and represent the magnitude of the postural response (see Horak et al. 1989 for details)

Complete balance platform data for 8 of the 10 subjects with MS and all of the control subjects was available for analysis.

SSEPs

Somatosensory evoked potentials (SSEPs) were examined bilaterally from both hands and feet in subjects with MS. Total central afferent conduction time was measured for the right and left sides and divided into spinal (ACT_{sp}) and supraspinal (ACT_{sup}) components. The procedures for measuring SSEPs are presented schematically in Figure 1B.

SSEPs were evoked by stimulation of the posterior tibial nerve in the leg and the median nerve in the arm. Peripheral conduction time was assessed by measuring the conduction time from the periphery to the low thoracic (T12) spinal cord (N20 potential) for the posterior tibial nerve, and to Erb's Point (N9 potential) for the median nerve. Total central conduction time was obtained by recording at the cortex (P37) and recording at cervical spine (P27 for the posterior tibial nerve and P13 for the median nerve) was used to divide total afferent conduction time (ACT) into spinal and supraspinal components. SSEP abnormalities were defined in terms of delayed latencies, or as absent when well-preserved waveforms could not be recorded. Measures were obtained from both sides of the body and compared with data from control subjects using the same method in the same laboratory.

Data analysis

The latencies of the postural response for all trials were averaged for each subject. Linear regressions were used to calculate correlations between postural response latencies and spinal and supraspinal SSEP latencies, with right and left sides considered to be independent.

The slopes of the regression between the average postural response magnitude (rate of change of average torque) across increasing amplitudes and velocities of surface displacements were calculated for each subject to represent scaling of postural responses. Predictive scaling was quantified as the slope of the magnitude of the postural response versus translation amplitudes, and reactive scaling was quantified as the slope of the magnitude of the postural response versus translation velocities. The slopes of these regressions provided a measure of the “gain” of the postural response and elevations of the regressions (the y-intercept) indicated the degree of relative “hypermetria” of the response. Each subject's linear least squares fit regression was plotted, with the intersubject variability of these regressions indicated by the standard deviation of slopes, r-values, and y-intercepts. In plots of control and MS group linear regressions, the mean and standard deviation of torque values are shown for each group. The lines represent the average linear regression for all subjects in a group. Group linear regressions were tested for significance (slope significantly different from 0) using a one-sided t tests ($p < 0.05$). The

level of significance for each regression of initial rate of change of torque with displacement velocity or amplitude was reported in which $r = 0.715$ was significant at $p < 0.01$ and $r = 0.87$ was significant at $p < 0.005$ (Shavelson 1988). Differences in the slopes and elevations (y-intercepts) of the linear regressions between the control and MS groups were compared with a one-way analysis of variance (ANOVA) with significance set at the $p < 0.05$ level. A linear regression across MS subject's data was used to determine whether there was a relationship between the latency of postural response and the ability to perform reactive or predictive scaling.

Results

Relationship between postural response latencies and SSEP latencies

Postural response latencies were significantly longer in the MS subjects than in control subjects (161 ± 31 vs 102 ± 21 ms, $p < 0.01$). Table 2 shows that many MS subjects had different postural response latencies in the right and left legs (difference range 3–37 ms; $p = 0.005$), unlike control subjects who had symmetric postural response latencies (difference range 0 – 10 ms; $p > 0.05$).

Peripheral somatosensory conduction times (N20) were normal in all subjects in this study. In the MS subjects, spinal SSEP latencies were absent in 6 of the 20 legs tested. In the MS subjects in whom spinal SSEPs could be recorded, these were significantly longer than laboratory normal values (17.2 ± 8.1 vs 7.9 ± 1.8 , $p < 0.01$). In contrast, supraspinal SSEP latencies in the MS subjects were not significantly longer than normal (11.0 ± 2.9 vs 9.5 ± 3.0 , $p > 0.05$). Postural response latencies in MS subjects were significantly correlated with slowed spinal somatosensory conduction ($r = 0.73$, $p < 0.01$) but not with supraspinal somatosensory conduction times ($r = 0.1$, $p > 0.05$; Figure 2).

Scaling postural response magnitude

Subjects with MS demonstrated significantly more (steeper) scaling to predicted translation amplitudes than controls. As can be seen in Figure 3A, the slope of the linear regression between the initial rate of change of surface reactive torque and translation amplitude was significantly larger for the MS than for the control group (8.4 ± 3.5 vs 4.9 ± 1.0 ; $p = 0.02$). In fact, 11 of the 20 legs of the MS patients showed larger than the largest scaling values in the control subjects (see Table 2).

Reactive scaling of postural response magnitude to increasing velocities of surface translations was often lower in subjects with MS than controls. Seven of the 20 legs of MS subjects showed velocity scaling values lower than the lowest control subject value (see Table 2). There was a significant difference in velocity scaling between the MS and control subjects' lower velocity scaling legs ($p = 0.005$) but not between the average scaling for both legs or for the higher scaling legs ($p > 0.05$). The slope of the linear regression between the initial rate of change of surface reactive torque and translation velocity was 2.8 ± 2.2 in the MS subjects versus 4.4 ± 1.1 in the controls ($p = 0.08$; Fig. 3B).

The magnitude of postural responses was significantly larger (hypermetric) in subjects with MS than in control subjects during both amplitude and velocity scaling ($p < 0.001$) (Fig. 3A and B for group means and Table 2 for each subject's average postural response magnitude across all conditions).

Relationships between postural response latencies and scaling

MS subjects with the longest latencies of postural responses (and with the longest spinal SSEP latencies) showed the steepest scaling of postural responses to predicted translation amplitudes. Figure 4A shows the relationship between the latency of gastrocnemius EMG for each MS leg

tested versus their associated predictive amplitude scaling value. This relationship was significantly different from zero ($r = 0.47$, $p = 0.05$).

In contrast to the positive relationship between postural response latencies and predictive scaling, reactive velocity scaling was negatively correlated with postural response latencies. Figure 4B shows the relationship between the latency of gastrocnemius EMG for each MS leg tested versus their associated reactive velocity scaling value. This relationship was significantly different from zero ($r = -0.5$, $p = 0.03$).

DISCUSSION

The results of this study indicate that people with imbalance due to MS have extremely delayed postural responses. Their postural responses to surface translations have the longest average latency reported in the literature, except for one case report of a person with total body somatosensory loss due to dorsal root afferent cell body degeneration (Bloem et al 2002; Horak, 2001). In the subjects with MS in this study, the delay in postural response onset correlated with the degree of prolongation in spinal cord somatosensory conduction, consistent with previous studies demonstrating that postural responses to surface translations are likely triggered by primary and/or secondary afferents from muscle spindles (Schiepatti and Nardone, 1999; Stapley et al, 2002). Thus, because of slowed spinal somatosensory conduction, people with MS receive somatosensory information about postural displacements late, and they then respond to this information late.

Subjects with MS also demonstrated differences in the delay in postural response latencies between their two legs. This asymmetry is consistent with the asymmetry in their spinal SSEPs and with the apparently random distribution of spinal cord lesions in MS. People with MS probably depend on the leg with the better somatosensory conduction, and therefore the shorter postural response latency, for functional balance. Despite the long latency of postural responses, the magnitude of postural torque responses were larger than normal in people with MS, suggesting that muscle weakness, due to the impaired efferent motor conduction known to occur in MS (Reich et al, 2007), did not contribute to imbalance when responding to these small postural perturbations with feet in place. In addition, preliminary unpublished data from our lab failed to show a correlation between delayed motor evoked potentials (MEP) and postural response latencies in subjects with MS. In fact, MS subjects' postural responses appear to be larger than normal, possibly to compensate for their prolonged latency. By the time the postural response occurs (sometimes over 100 ms late) it must be larger than normal to effectively return the body's center of mass to the center of the base of support and thus prevent the person from falling. Delayed postural responses may result in a fall because, by the time the patient initiates a balance response their center of mass may be too far outside of their base of support for available postural strategies to effectively maintain balance. The larger postural responses seen in subjects with MS are similar to, but not as large as, the hypermetric postural responses in see in subjects with cerebellar ataxia and likely contribute to the clinical impression of ataxia and subjective perception of imbalance in people with MS (see figures 3A and 3B with data from 10 subjects with cerebellar disorders (Horak et al, 1994)).

In addition to increasing the magnitude of their responses, people with MS and balance problems also appear to compensate for their prolonged postural response latencies by increasing the use of prediction to scale their responses to the amplitude of an upcoming external perturbation. Amplitude scaling is predictive because the nervous system responds before the amplitude is known. Amplitude scaling does not occur when perturbation amplitudes are randomized (Horak FB et al 1989). Enhanced predictive scaling may help to prevent falls and reduce the need for stepping for postural correction. The enhanced predictive scaling found in the subjects with MS in this study is in contrast to people with cerebellar disorders who

characteristically lack predictive scaling (Horak et al. 1994; see Figure 3A). This suggests that the imbalance in the people with MS in this study was not related to cerebellar dysfunction and that these subjects had sufficient cerebellar function to allow for amplified predictive, amplitude scaling.

Although the people with MS and imbalance in this study had good predictive amplitude scaling, they often showed reduced reactive velocity scaling with at least one leg. Poor velocity scaling would be expected if the delayed postural latencies were caused by slowed central somatosensory conduction because scaling of postural responses to perturbation velocity is thought to depend on encoding of perturbation velocity from muscle spindles (Horak and Macpherson 1996, Schieppatti M. Nardone A. 1999). Subjects with slowed conduction in large diameter peripheral sensory nerves due to diabetic neuropathy also have reduced velocity scaling and delayed postural responses (Inglis et al. 1994). However, these delays are considerably smaller (average 24 ms) than in the subjects with MS in the current study (average 59 ms).

In summary, the pattern of postural responses observed in subjects with MS and imbalance are characterized by very prolonged latency, correlation of this prolongation with spinal SSEP conduction, and enhancement of predictive response scaling. This pattern is unlike that of subjects with cerebellar disorders and more closely resembles that of subjects with peripheral neuropathy (See Table 3; Dickstein et al. 2003, Horak 1994, Inglis et al. 1994). Increased predictive postural response scaling, as found in the subjects with MS in this study, is in notable contrast to patients with cerebellar disorders who characteristically fail to predictively scale postural responses. Predictive scaling allows one to adapt and improve performance with repeated presentation of similar balance challenges. Similar to people with diabetic peripheral neuropathy, people with MS and imbalance have very delayed postural response latencies and decreased postural response velocity scaling. However, the delay in postural response latencies is longer in subjects with MS than in subjects with diabetic peripheral neuropathy. In contrast to people with diabetic neuropathy, the subjects with MS in this study had increased, not reduced, postural response magnitudes. This suggests that people with MS have worse disturbances in afferent conduction but better efferent motor function than people with diabetic peripheral neuropathy. Overall, these findings strongly suggest that the primary cause of imbalance in MS is not cerebellar but rather a result of slowed afferent proprioceptive conduction in the spinal cord.

The most distinct findings of this study are that people with MS and imbalance have very delayed postural response latencies that correlate with delayed spinal SSEP conduction. It is most likely that demyelination of the posterior columns of the spinal cord is the direct cause of the SSEP conduction delays, and that this causes delayed postural responses and imbalance in most people with MS. Given that postural EMG response latency (measured as the latency of gastrocnemius (GAS) EMG response to backward surface displacement (forward body sway)) correlates with spinal SSEP conduction velocity in people with MS, suggests that postural EMG response latency could be used as a functional measure of CNS conduction velocity and demyelination in people with MS.

Postural EMG response latency has a number of advantages over other tools currently used to examine the severity of CNS demyelination and disease impact in patients with MS including SSEP latencies and MRI. Although SSEP latencies can indicate the degree and location of CNS demyelination in MS, they are limited by a low ceiling effect. As found in this and other studies, when conduction is severely slowed, absence of all waves after the Erb's point potential is not uncommon (Oken 1986). The absence of long latency responses indicates severe slowing of conduction but fails to further quantify this slowing. In this study, long latency SSEPs were absent in a number of subjects with MS, but there were no subjects in whom GAS EMG

latencies in response to postural translations could not be measured. EMG latencies to postural perturbations can be measured for any subject who can stand unsupported for as little as a few seconds. Unlike MRI, which provides largely qualitative information about disease progression and activity in MS, postural response latency may provide a more continuous, quantifiable, and functional measure of disease activity, either over time or in response to interventions intended to reduce inflammation or enhance CNS conduction.

Based on the findings of this study, we suggest that clinical examination of patients with imbalance associated with MS include measurement of postural responses. If delayed postural responses are found, rehabilitation therapies directed toward interventions, such as use of canes (Ashton-Miller et al. 1996, Dickstein et al. 2001), and sensory substitution with intact vestibular (Horak and Hlavacka 2001) and visual function, (if the patient has intact vestibular or visual function) may help compensate for the delayed transmission and response (Herndon et al, 2000). In addition, patients should be encouraged to take extra care when walking on unstable or moving surfaces or in crowded areas, as these conditions are likely to contribute to falls in people with delayed postural responses. These changes may help reduce the morbidity, mortality, and activity and participation restrictions in this vulnerable population

This study has a number of limitations. The sample size was small and the subjects were not randomly selected. The subjects with MS were patients with MS selected for subjective complaints of imbalance but no measure of the severity of their experience of imbalance or falls was performed. Future studies of balance in MS would benefit from a larger sample size drawn from multiple sites, detailed examination of MRIs, inclusion of subjects with MS with and without subjective complaints of imbalance, examination of functional degree of imbalance including incidence of falls and evaluation of the relationship between clinical balance measures and the latency, magnitude and scaling of APRs.

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Figure 1.

Postural response and SSEP measurement methods

Figure 1A. Balance Platform. The subject stands with each foot on a force plate of the movable balance platform. The platform is translated backward, which results in forward displacement of the body over the feet. Measured responses to this support surface translation include the plantarflexion torque exerted by each foot and the latency of EMG activation of the gastrocnemius muscles, the prime movers to restore equilibrium.

Figure 1B. Location of SSEPs recorded from stimulation of the posterior tibial nerve in the leg or the median nerve in the arm; potentials associated with posterior tibial nerve stimulation are shown in italics. Intervals corresponding to total afferent conduction time (ACT) and its spinal (ACT_{sp}) and supraspinal (ACT_{sup}) components are also shown.

$$\begin{aligned} \text{ACT} &= \text{P37} - \text{N20} \\ \text{ACT}_{\text{sup}} &= \text{N19} - \text{P13} \\ \text{ACT}_{\text{sp}} &= \text{ACT} - \text{ACT}_{\text{sup}} \end{aligned}$$

Figure 2.

Relationships between postural response latencies and SSEP latencies.

Figure 2A and 2B. Relationship between postural response latency (GAS EMG) to backward displacement and spinal SSEP conduction times (shown in Figure 2A) and supraspinal SSEP conduction times (shown in Figure 2B). The x's on Figure 2A represent postural response latencies for subjects with absent spinal SSEPs. These subject's values were not included in the linear regression.

Figure 3.

Scaling of postural responses in MS and control subjects from this study and cerebellar subjects from our previous study (Horak et al. 1994).

Figure 3A. Predictive amplitude scaling of rate of initial postural response to block presentation of increasing platform amplitudes, for MS (circles), control (squares) and cerebellar (triangles) subjects.

Figure 3B. Reactive velocity scaling of rate of initial postural response to randomly presented platform velocities, for MS (circles), control (dark squares) and cerebellar (triangles) subjects. The slope values shown represent the group means and SEMs.

Figure 4.

Relationships of postural response latencies and scaling

Figure 4A. Relationship of postural response latency (GAS EMG) to the amplitude scaling slope for each individual MS subject (values for both legs are included)

Figure 4B. Relationship of postural response latency (GAS EMG) to the velocity scaling slope for each individual MS subject (values for both legs are included).

Table 1

Subject characteristics

Subject Characteristics	MS	Control
Age in years - mean (range)	44.6 (36–52)	45.2 (25–51)
Sex	8 F, 2 M	7 F, 3 M
EDSS: range	1–4	N/A
Duration of disease in years: mean (range)	8.7 (1–22)	N/A

Table 2

Summary of postural and SSEP values for both sides in each subject with MS and for the control and cerebellar group means (from Horak et al. 1989).

Subject	Side	Gas	EMG Latency (ms)	Avg Response Amplitude (Nm/ms)	ACTsp (ms)	ACTsup (ms)	Predictive Amplitude Scaling (nM/cm)	Reactive Velocity Scaling (nM/m/s)
1	L	168*		196	No P27	7.2	11.5	-1.2
	R	178		182	No P27	9.6	10.6	0.7
2	L	110		196	8.9	9.2	10.0	4.0
	R	106*		176	10.0	11.8	10.8	4.4
3	L	179*		214	No P27	9.4	8.8	3.0
	R	194		143	No P27	9.8	12.3	4.9
4	L	160		175	18.0	15.5	4.6	2.0
	R	152*		181	No response	18.8	10.2	1.5
5	L	209*		128	35.7	9.5	12.0	-1.7
	R	212		188	Not tolerated	Not tolerated	12.0	3.7
6	L	131		208	7.8	8.1	3.3	5.0
	R	127*		186	10.3	8.3	3.4	4.3
7	L	191		185	16.8	10.1	NR	NR
	R	185*		182	22.1	11.4	NR	NR
8	L	180		199	13.6	10.4	No data	5.5
	R	150*		138	18.4	15.8	No data	2.0
9	L	125*		218	12.2	12.0	1.9	3.7
	R	162		198	15.0	11.2	6.8	6.4
10	L	166		181	29.6	10.2	5.5	1.5
	R	144*		180	21.7	10.6	9.9	1.6
MS Mean (SD)		161 (31)		183 (25)	17.2 (8.1)	11.0 (2.9)	8.4 (3.5)	2.8 (2.2)
Control mean (SD)		102 (21)		132 (36)	7.9 (1.8)	9.5(3.0)	4.9 (1.0) range 2.6–6.9	4.4 (1.1) range 2.2 –6.6
Cerebellar mean (SD)		106 (20)		350 (35)	Not tested	Not tested	-1.4 (4.4)	3.9 (2.4)

* longer latency leg for each subject

Table 3

Comparison of balance measures and SSEPs in people with MS, cerebellar disorders and diabetic peripheral neuropathy

	MULTIPLE SCLEROSIS	CEREBELLAR	NEUROPATHY
BALANCE MEASURES			
Latency	Very delayed	Normal	Delayed
Magnitude	Increased	Increased	Decreased
Amplitude scaling	Normal/increased	Decreased	Normal
Velocity scaling	Reduced	Normal	Reduced
SSEPs			
Supraspinal	Slightly delayed	Unknown	Normal
Spinal	Very delayed	Normal	Normal
Peripheral	Normal	Normal	Delayed