Neuroscience Letters 591 (2015) 182–186

Contents lists available at ScienceDirect

Neuroscience Letters

journal homepage: www.elsevier.com/locate/neulet

Research article

Anticipatory and compensatory postural adjustments in individuals with multiple sclerosis in response to external perturbations

Alexander S. Aruin*, Neeta Kanekar, Yun-Ju Lee

University of Illinois at Chicago, Chicago, IL 60612, USA

HIGHLIGHTS

- Anticipatory and compensatory postural control was studied in individuals with multiple sclerosis (MS).
- External perturbations were used to perturb standing balance.
- Anticipatory postural adjustments (APAs) were delayed in individuals with MS.
- Enhancement of APAs may add to balance rehabilitation of individuals with MS.

ARTICLE INFO

Article history:
Received 4 November 2014
Received in revised form 29 January 2015
Accepted 3 February 2015
Available online 21 February 2015

Keywords:
Multiple sclerosis
Impairment
Postural control
Anticipatory
Compensatory
External perturbations
Rehabilitation

ABSTRACT

Deficit in balance control is a common and often an initial disabling symptom of multiple sclerosis (MS). The aim of the study was to investigate the organization of anticipatory and compensatory postural adjustments in individuals with MS dealing with external perturbations. Ten individuals with MS and ten age-and-gender matched healthy controls were exposed to external perturbations applied at the shoulder level. The perturbations were either predictable or unpredictable as subjects stood with eyes open or closed. Electrical activity of six leg and trunk muscles as well as displacements of the center of pressure (COP) were recorded and quantified within the time intervals typical of anticipatory (APAs) and compensatory (CPAs) postural adjustments. Individuals with MS demonstrated delayed anticipatory onsets of muscle activity and smaller anticipatory COP displacements as compared to healthy control subjects. The deficiency of the APAs was associated with increased displacements of the COP during the balance restoration phase. The results demonstrate the underlying impairment in anticipatory postural control of individuals with MS. The study outcome provides a background for development of rehabilitation strategies focused on balance restoration in people with MS.

1. Introduction

Multiple sclerosis (MS) is a chronic autoimmune demyelinating disease affecting the central nervous system (CNS) [6]. Postural imbalance is often described as one of the initial symptoms of MS [7,10,22] and one of the most disabling MS symptoms that affects about 75% of patients during the course of the disease [24,25]. Poor balance control is a significant contributing factor to the increased risk of falling in individuals with MS [7,8,11,36] and is also linked to lower engagement in physical activity [26]. Moreover, poor balance control in individuals with MS is associated with impaired anticipatory postural adjustments [15]. It was also shown that inefficient anticipatory postural adjustments could result in accidental falls [37]. Likewise, fear of falling is also associated with an increased risk of falls in MS [11,29] and over 80% of people with fear also report activity restrictions [29]. Additionally, people with MS identify fatigue as one of the primary reasons for falling [28].

The stability of human vertical posture is affected by the high location of the center of mass, small support area, and multiple joints between the feet and the center of mass. Moreover, when a standing person performs a quick movement or interacts with external objects, the mechanical coupling of body segments leads to postural perturbations that may be detrimental for the fragile balance. While maintaining vertical posture, the CNS uses two main types of adjustments in the activity of the trunk and leg muscles when dealing with body perturbations. Anticipatory postural adjustments (APAs) control the position of the center of mass (COM) of the body by activating the trunk and leg muscles prior

* Corresponding author at: Department of Physical Therapy (MC 898), University of Illinois at Chicago, 1919 W. Taylor St. Chicago, IL 60612, USA.
Tel.: +1 312 355 0904.
E-mail address: aaruin@uic.edu (A.S. Aruin).

http://dx.doi.org/10.1016/j.neulet.2015.02.050
0304-3940/

to a forthcoming body perturbation, thus minimizing the danger of losing equilibrium [reviewed in [23]]. Compensatory postural adjustments (CPAs) are initiated by the sensory feedback signals and serve as a mechanism of restoration of the position of the COM after a perturbation has already occurred [1].

APAs are an essential mechanism of balance control that ensures adequate postural preparation prior to task performance or in dealing with the external environment. Impairments of APAs have been reported in the elderly [16] and various neurological populations such as in individuals with stroke [35], cerebral palsy [12], Parkinson's disease [20], individuals with Down syndrome [2], as well as in individuals with MS [17,18]. However, the majority of APA studies in these populations utilized voluntary movement of an upper limb or load release from extended arms. However, in the arm lifting or load releasing paradigms most commonly used to study APAs, motor action and perturbation are interrelated as slow movements do not induce APAs [13]. Therefore, the diminished APAs seen in patients could be either a consequence of the disease or the result of slowness of performance of the arm movements seen for example in patients with Parkinson's disease or elderly individuals.

In order to have a clear understanding of how MS affects the organization of APAs, it is important to distinguish the influence of voluntary movement performance and postural preparation. In the recent past, external perturbations were used to investigate anticipatory postural control. The studies involving external perturbations such as load catching [3,34] and pendulum impact paradigms [32,33] revealed that APAs could be seen in preparation to perturbations that were externally induced with no voluntary movement being performed by the subjects themselves. Thus as long as the perturbation is predictable (either external or internal in origin), APAs are observed with patterns that are adequate for counteracting the expected impact. Moreover, external predictable perturbations provide a definite advantage of delivering consistent postural disturbances that are independent of the variability intrinsic to the voluntary movement performance. In addition, using an external perturbation paradigm eliminates the influence occurring from the interaction between voluntary movement and postural control. Therefore, to avoid the possible interaction with a focal voluntary movement (such as during arm raising tasks) and to investigate the direct effect of MS on the anticipatory postural control by itself, using an external predictable perturbation (with its magnitude consistent across the subjects), will allow minimizing the confounding effects of the voluntary movement itself. This study was thus focused on investigating the differences in anticipatory and compensatory postural adjustments between individuals with MS and control subjects exposed to external perturbations. We hypothesized that in individuals with MS: (1) the latency of the APAs will be delayed, (2) the anticipatory displacement of the center of pressure (COP) will be smaller and (3) compensatory COP displacement will be larger when compared to healthy controls.

2. Material and methods

2.1. Participants

Ten individuals (8 females and 2 males) with remitting-relapsed MS (MS group, mean age 52 ± 13 years, height 169.8 ± 10.3 cm, mean weight 68 ± 12 kg, mean EDSS score 2.3 ± 0.9) and ten age and gender matched healthy subjects (HC group, mean age 51 ± 14 years, mean height 167.8 ± 10.0 cm, mean weight 73 ± 12 kg) participated in the study. The inclusion criteria for the individuals with MS were: normal or corrected to normal vision, an EDSS [19] score of 5 or less, and the ability to stand independently without any aid or orthosis for at least 3 min. Patients with a history of shoulder subluxation or dislocation, any kind of pain, or unable to perform the experimental tasks were excluded. The experiments were approved by the UIC Institutional Review Board.

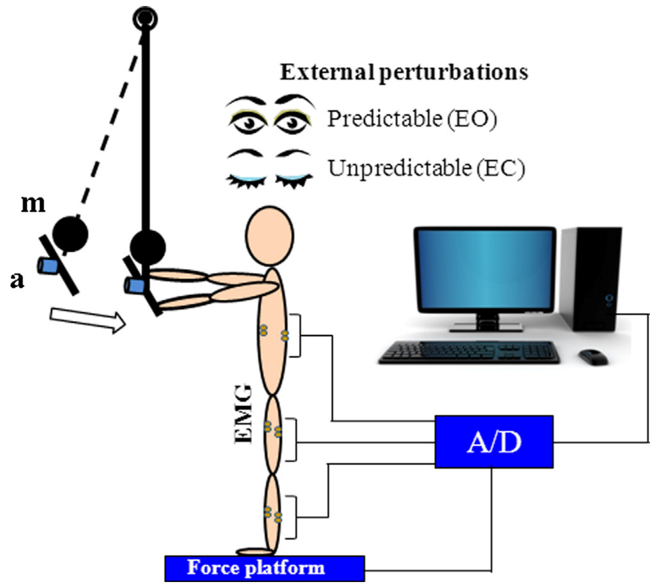

Fig. 1. Schematic representation of the experimental set up. Subjects were exposed to a predictable (eyes open) and unpredictable (eyes closed) perturbation. EMG activity and ground reaction forces were recorded. *m* – mass of the pendulum, a – accelerometer, A/D – analog-to-digital converter.

2.2. Experimental set-up and procedure

The subjects were instructed to maintain upright stance while standing barefoot on the force platform with their feet shoulder width apart. The pendulum impact paradigm was used to perturb the subjects. A load (mass, m = 3% of the subjects' body weight) was attached to the pendulum next to its distal end. The subjects were required to receive each pendulum impact with their hands, while their arms, wrists, and fingers were extended at the shoulder level (Fig. 1), and to maintain their balance after the perturbation. Both the groups received a series of perturbations while their eyes were open (EO) and with eyes closed (EC). When vision was available the perturbations were thus predictable and hence elicited both, APAs and CPAs. When vision was not available, the perturbations were unpredictable and only CPAs were generated. Two to three practice trials were given to both the groups in each experimental condition. For safety purposes, the subjects wore a harness (NeuroCom, USA) with two straps attached to the ceiling. Ten trials were performed, each five seconds in duration. All participants were allowed to have rest periods as needed.

2.3. Instrumentation

Disposable self-adhesive electrodes (Red Dot 3M) were used to record the electrical activity (EMG) of the following right muscles: tibialis anterior (TA), gastrocnemius lateralis (GASL), vastus lateralis (VL), biceps femoris (BF), rectus abdominis (RA), and erector spinae (ES). Based upon recommendations reported in previous literature [4], electrodes were attached to the muscle belly of each of the above muscles after cleaning the skin area with alcohol wipes. A ground electrode was attached to the anterior aspect of the leg over the tibial bone. EMG signals were collected, filtered, and amplified (10–500 Hz, gain 2000) with the EMG system (Myopac, RUN Technologies, USA). A force platform (model OR-5, AMTI, USA) was used to record the ground reaction forces and moments of forces.

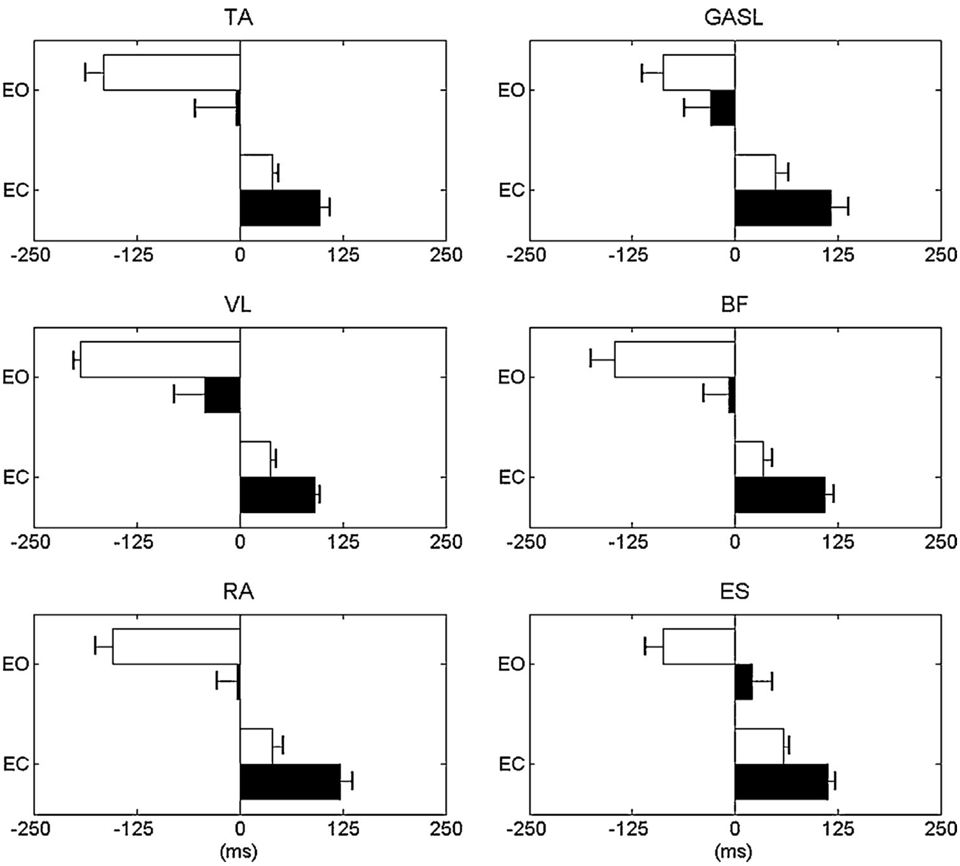

Fig. 2. Onsets of EMG activity in individuals with MS (black boxes) and control subjects (empty boxes) in conditions with eyes open (EO) or closed (EC). Means and standard errors are shown.

The signal obtained from an accelerometer (Model 208CO3, PCB Piezotronics, Inc., USA) attached to the pendulum was used to register the moment of the pendulum impact. All the signals were sampled at 1000 Hz frequency with a 16-bit resolution.

2.4. Data processing

The data were analyzed off-line using MATLAB (MathWorks, Natick, MA) programs. Five to seven trials were used for further analysis. EMG signals were rectified and filtered with a 100 Hz low-pass, 2nd order, zero-lag Butterworth filter, while the ground reaction forces, and moments were filtered with a 40 Hz low-pass, 2nd order, zero-lag Butterworth filter. The ‘time-zero’ ($T_0 = 0$, moment of pendulum impact) was calculated as a point in time at which the accelerometer signal exceeded 5% of the maximum. Data in the range from −600 ms (before T_0) to +1000 ms (after T_0) were selected for further analysis. Individual trials were aligned according to T_0 and this was used as a common reference point for all the signals.

The muscle onset (beginning of activation/inhibition of a muscle) for each trial was detected in a time window from −250 ms to +250 ms in relation to T_0 by a combination of computer algorithm and visual inspection of the trials. The muscle onset for a specific muscle was defined as the instant lasting for at least 50 ms when its EMG amplitude was greater (activation) or smaller (inhibition) than the mean ± 2SD of its baseline value, measured from −500 to −400 ms. The muscle onsets for each muscle were then averaged across the trials for each subject.

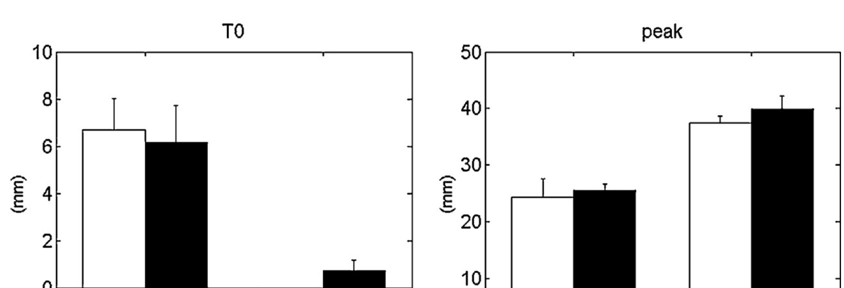

Fig. 3. COP displacements in individuals with MS (black boxes) and control subjects (empty boxes) in conditions with eyes open (EO) or closed (EC). Means and standard errors are shown. The positive values represent the backward COP displacement.

Displacements of the center of pressure (COP) in the anterior–posterior direction were calculated using the following equation:

$$\mathrm{COP} = \frac{Mx - (Fy \times dz)}{Fz}$$

where Mx is the moment in sagittal plane, Fz and Fy are the vertical and the anterior–posterior components of the ground reaction force, and dz is the distance from the origin of the platform to the surface (0.038 m).

All signals were then analyzed using MATLAB programs. Aligned trials were averaged for each subject. The displacement of COP at T_0 which is anticipatory in nature and the peak displacement (maximum displacement after T_0) that is compensatory in nature were calculated. As the perturbations were symmetrical, they were associated with anterior–posterior displacements of the COP and negligible displacements in the medial–lateral direction. Therefore, COP displacements in the anterior–posterior direction will be reported.

2.5. Statistical analysis

Muscle onsets and COP displacement (at T_0 and peak displacement) were evaluated using two way mixed design ANOVA with one between-subject factor of Group (2 levels: MS, HC) and one within-subject factor of condition (2 levels: EO, EC). The statistical significance was set at $p < .05$ in all the tests. Statistical analysis was performed in SPSS 17 for Windows XP (SPSS Inc., Chicago, USA).

3. Results

With vision available, onset of muscle activity was observed T_0 in both groups. However, anticipatory activity of the trunk and leg muscles occurred closer to the moment of perturbation in individuals with MS as compared to control subjects. The onset of APA activity was as follows: TA (MS: −4.87 ± 49.98 ms, HC: −164.96 ± 21.76 ms); GASL (MS: −29.86 ± 29.80 ms, HC: −87.07 ± 24.93 ms); VL (MS: −42.34 ± 119.12 ms, HC: −193.27 ± 23.88 ms); BF (MS: −7.77 ± 30.61 ms, HC: −146.20 ± 29.04 ms); RA (MS: −3.48 ± 69.39 ms, HC: −153.31 ± 67.31 ms); and ES (MS: 21.11 ± 23.51 ms, HC: −86.94 ± 21.03 ms) (Fig. 2). When vision was not available, muscles became active in both the groups only after the T_0. The onset of muscle activity during the CPA (balance restoration phase) was as follows: TA (MS: 96.95 ± 37.16 ms, HC: 39.47 ± 15.56 ms); GASL (MS: 117.23 ± 19.84 ms, HC: 48.62 ± 16.60 ms); VL (MS: 91.11 ± 17.27 ms, HC: 37.92 ± 19.22 ms); BF (MS: 109.45 ± 9.91 ms, HC: 34.22 ± 9.40 ms); RA (MS: 121.58 ± 54.00 ms, HC: 40.21 ± 17.37 ms) and ES (MS: 113.07 ± 8.73 ms, HC: 58.78 ± 7.81 ms). Overall, all muscles were initiated statistically significantly later in individuals with MS than in the HC group (Table 1). There were also statistically significant vision and group interactions in TA, VL and RA muscles and vision and group interaction was close to the level of statistical significance in ES.

Fig. 3 depicts the displacements of COP at T_0 and the peak displacements after T_0. When vision was available, the anticipatory COP displacement (at T_0) in the backward direction was seen in both the groups. Moreover, individuals with MS demonstrated smaller COP displacements (6.15 ± 1.58 mm) than control subjects (6.68 ± 1.35 mm). Smaller anticipatory COP displacements at T_0 seen in individuals with MS resulted in larger peak COP displacements after the perturbation: (25.54 ± 1.09 mm) as compared with control subjects (24.28 ± 3.23 mm). The observed trend, however, was not statistically significant.

Table 1
The results of repeated measure ANOVAs for the muscle onsets and COP displacements.

	Group		Vision		Group × Vision	
EMG onset	*F*	*p*	*F*	*p*	*F*	*p*
GASL (1,18)	8.93	0.01	72.37	<0.001	0.09	0.77
TA (1,18)	10.97	<0.01	48.02	<0.001	5.39	0.03
VL (1,18)	25.74	<0.001	90.68	<0.001	6.51	0.02
BF (1,18)	23.11	<0.001	41.55	<0.001	3.57	0.08
RA (1,18)	33.29	<0.001	100.58	<0.001	8.49	<0.01
ES (1,18)	22.98	<0.001	79.34	<0.001	4.16	0.06
COP (1,18)						
T0	0.09	0.76	44.5	<0.001	0.93	0.37
Peak	0.81	0.38	42.16	<0.001	0.09	0.76

When anticipatory postural adjustments were not utilized (EC condition) the COP displacements at T_0 were much smaller in both the groups. It is also notable that the COP at T_0 shifted forward in control subjects while it still was backwards in individuals with MS. In the EC condition, peaks of COP displacements in both the groups were much larger as compared to condition when APAs were utilized (EO). Moreover, the peak of the COP displacements in individuals with MS was larger than in control subjects. The observed trend, however, was not statistically significant.

4. Discussion

The focus of this study was on the investigation of APAs and CPAs in individuals with MS exposed to the externally induced body perturbations. Importantly, individuals with MS were able to generate APAs to control posture. These results suggest that MS itself, does not seem to affect anticipatory recruitment of postural muscles. This finding is in line with previous literature on APAs in individuals with MS involving self-initiated perturbations [17,18]. Moreover, for the first time we report that individuals with MS while being exposed to external perturbations demonstrated the delayed anticipatory onsets of muscle activity compared to control subjects. Furthermore, the study outcome confirmed that individuals with MS are able to utilize APAs for postural control: when the perturbation was predictable, muscles were recruited prior to the perturbation, as opposed to being activated after the impact when the perturbation was unpredictable.

Thus, it appears that deficits in postural control in MS may not be due to an absence of APAs, which have otherwise been found to be sometimes absent in individuals with neurological conditions such as Parkinson's disease [5]. Moreover, a significant delay in anticipatory postural adjustments was reported in the elderly individuals with postural muscles being recruited closer to the activation of prime mover muscles [14,21,31] or after prime mover activation [38]. We recently demonstrated that in preparation to an external predictable perturbation, anticipatory muscle activity was significantly delayed in older adults as compared to young adults, such that the muscles were either activated or inhibited very close to the moment of perturbation [16].

As a consequence of the delayed anticipatory activation of muscles, a larger displacement of the COP was produced during the balance restoration phase (after T_0). It is important to note that the greater is the magnitude of peak displacement during the compensatory postural control, the larger is the postural instability. Thus, large COP displacements were reported in the elderly [27] and in individuals with neurological disorders [30] indicating body instability. As such, it is reasonable to believe that the increased displacement of COP seen in individuals with MS represents their increased instability while restoring balance after being exposed to an external perturbation. These results provide support to the proposed hypotheses. Moreover, the findings from the current study highlight the critical role played by anticipatory postural mechanisms in preparation to balance perturbations in MS. This outcome taken together with the literature data on the gait and balance problems in individuals with MS even in the absence of disability [9,10] suggest the potentials of enhancing anticipatory postural adjustments in balance rehabilitation of individuals with MS.

5. Conclusions

Anticipatory postural adjustments are delayed in individuals with MS reflecting impairment of postural control. The outcome of the study creates a basis for further investigations of postural control in people with MS. It also provides a background for development of strategies for balance rehabilitation of individuals with MS.

Acknowledgements

This work was supported by the National Multiple Sclerosis Society grant PP1871. We thank the individuals with MS and control subjects for their exceptional cooperation. We also thank Vennila Krishnan for help in data collection.

References

[1] A.V. Alexandrov, A.A. Frolov, F.B. Horak, P. Carlson-Kuhta, S. Park, Feedback equilibrium control during human standing, Biol. Cybern. 93 (2005) 309–322.
[2] A. Aruin, G. Almeida, A coactivation strategy in anticipatory postural adjustment in persons with Down syndrome, Motor Control 2 (1997) 178–197.
[3] A.S. Aruin, T. Shiratori, M.L. Latash, The role of action in postural preparation for loading and unloading in standing subjects, Exp. Brain Res. 138 (2001) 458–466.
[4] J.V. Basmajian, R. Blumenstein, Electrode placement in EMG biofeedback, Williams & Wilkins, Baltimore, 1980, 88 pp.
[5] D. Bazalgette, M. Zattara, N. Bathien, S. Bouisset, P. Rondot, Postural adjustments associated with rapid voluntary arm movements in patients with Parkinson's disease, Adv. Neurol. 45 (1987) 371–374.
[6] C. Bjartmar, B.D. Trapp, Axonal and neuronal degeneration in multiple sclerosis: mechanisms and functional consequences, Curr. Opin. Neurol. 14 (2001) 271–278.
[7] M.H. Cameron, S. Lord, Postural control in multiple sclerosis: implications for fall prevention, Curr. Neurol. Neurosci. Rep. 10 (2010) 407–412.
[8] D. Cattaneo, C. De Nuzzo, T. Fascia, M. Macalli, I. Pisoni, R. Cardini, Risks of falls in subjects with multiple sclerosis, Arch. Phys. Med. Rehabil. 83 (2002) 864–867.
[9] M.L. Daley, R.L. Swank, Quantitative posturography: use in multiple sclerosis, IEEE Trans. Biomed. Eng. 28 (1981) 668–671.
[10] O. Findling, J. Sellner, N. Meier, J.H. Allum, D. Vibert, C. Lienert, H.P. Mattle, Trunk sway in mildly disabled multiple sclerosis patients with and without balance impairment, Exp. Brain Res. 213 (2011) 363–370.
[11] M.L. Finlayson, E.W. Peterson, C.C. Cho, Risk factors for falling among people aged 45 to 90 years with multiple sclerosis, Arch. Phys. Med. Rehabil. 87 (2006) 1274–1279, quiz1287.
[12] G.L. Girolami, T. Shiratori, A.S. Aruin, Anticipatory postural adjustments in children with hemiplegia and diplegia, J. Electromyogr. Kinesiol. 21 (2011) 988–997.
[13] F.B. Horak, P. Esselman, M.E. Anderson, M.K. Lynch, The effects of movement velocity, mass displaced, and task certainty on associated postural adjustments made by normal and hemiplegic individuals, J. Neurol. Neurosurg. Psychiatry 47 (1984) 1020–1028.
[14] B. Inglin, M. Woollacott, Age-related changes in anticipatory postural adjustments associated with arm movements, J. Gerontol. 43 (1988) M105–113.
[15] J.V. Jacobs, S.L. Kasser, Balance impairment in people with multiple sclerosis: preliminary evidence for the balance evaluation systems test, Gait Posture 36 (2012) 414–418.
[16] N. Kanekar, A.S. Aruin, The effect of aging on anticipatory postural control, Exp. Brain Res. 232 (2014) 1127–1136.
[17] V. Krishnan, N. Kanekar, A.S. Aruin, Anticipatory postural adjustments in individuals with multiple sclerosis, Neurosci. Lett. 506 (2012) 256–260.
[18] V. Krishnan, N. Kanekar, A.S. Aruin, Feedforward postural control in individuals with multiple sclerosis during load release, Gait Posture 36 (2012) 225–230.
[19] J.F. Kurtzke, Rating neurologic impairment in multiple sclerosis: an expanded disability status scale (EDSS), Neurology 33 (1983) 1444–1452.
[20] M.L. Latash, A.S. Aruin, I. Neyman, J.J. Nicholas, Anticipatory postural adjustments during self inflicted and predictable perturbations in Parkinson's disease, J. Neurol. Neurosurg. Psychiatry 58 (1995) 326–334.
[21] N.B. Man'kovskii, A. Mints, V.P. Lysenyuk, Regulation of the preparatory period for complex voluntary movement in old and extreme old age, Human Physiol. 6 (1980) 46–50.
[22] C.L. Martin, B.A. Phillips, T.J. Kilpatrick, H. Butzkueven, N. Tubridy, E. McDonald, M.P. Galea, Gait and balance impairment in early multiple sclerosis in the absence of clinical disability, Mult. Scler. 12 (2006) 620–628.
[23] J. Massion, Movement, posture and equilibrium: interaction and coordination, Prog. Neurobiol. 38 (1992) 35–56.
[24] C.A. McDonald, et al., Symptoms and signs in the course of disease, in: A. Compston (Ed.), Mcalpine's Multiple Sclerosis, Churchill Livingstone, Philadelphia, 2005.
[25] B. Missaoui, P. Thoumie, How far do patients with sensory ataxia benefit from so-called proprioceptive rehabilitation? Neurophysiol. Clin. 39 (2009) 229–233.
[26] R.W. Motl, E.M. Snook, E. McAuley, R.C. Gliottoni, Symptoms, self-efficacy, and physical activity among individuals with multiple sclerosis, Res. Nurs. Health 29 (2006) 597–606.
[27] H. Nakamura, T. Tsuchida, Y. Mano, The assessment of posture control in the elderly using the displacement of the center of pressure after forward platform translation, J. Electromyogr. Kinesiol. 11 (2001) 395–403.
[28] Y. Nilsagard, E. Denison, L.G. Gunnarsson, K. Bostrom, Factors perceived as being related to accidental falls by persons with multiple sclerosis, Disabil. Rehabil. 31 (2009) 1301–1310.
[29] E.W. Peterson, C.C. Cho, M.L. Finlayson, Fear of falling and associated activity curtailment among middle aged and older adults with multiple sclerosis, Mult. Scler. 13 (2007) 1168–1175.
[30] S.H. Peurala, P. Kononen, K. Pitkanen, J. Sivenius, I.M. Tarkka, Postural instability in patients with chronic stroke, Restor. Neurol. Neurosci. 25 (2007) 101–108.
[31] M.W. Rogers, C.G. Kukulka, G.L. Soderberg, Age-related changes in postural responses preceding rapid self-paced and reaction time arm movements, J. Gerontol. 47 (1992) M159–165.
[32] M.J. Santos, A.S. Aruin, Role of lateral muscles and body orientation in feed forward postural control, Exp. Brain Res. 184 (2008) 547–559.
[33] M.J. Santos, N. Kanekar, A.S. Aruin, The role of anticipatory postural adjustments in compensatory control of posture: 1. Electromyographic analysis, J. Electromyogr. Kinesiol. 20 (2010) 388–397.
[34] T. Shiratori, M.L. Latash, Anticipatory postural adjustments during load catching by standing subjects, Clin. Neurophysiol. 112 (2001) 1250–1265.
[35] H. Slijper, M.L. Latash, N. Rao, A.S. Aruin, Task-specific modulation of anticipatory postural adjustments in individuals with hemiparesis, Clin. Neurophysiol. 113 (2002) 642–655.
[36] J.J. Sosnoff, M.J. Socie, M.K. Boes, B.M. Sandroff, J.H. Pula, Y. Suh, M. Weikert, S. Balantrapu, S. Morrison, R.W. Motl, Mobility, balance and falls in persons with multiple sclerosis, PLoS One 6 (2011) e28021.
[37] K. Uemura, M. Yamada, K. Nagai, N. Ichihashi, Older adults at high risk of falling need more time for anticipatory postural adjustment in the precrossing phase of obstacle negotiation, J. Gerontol. A: Biol. Sci. Med. Sci. 66 (2011) 904–909.
[38] M.H. Woollacott, D.L. Manchester, Anticipatory postural adjustments in older adults: are changes in response characteristics due to changes in strategy? J. Gerontol. 48 (1993) M64–70.