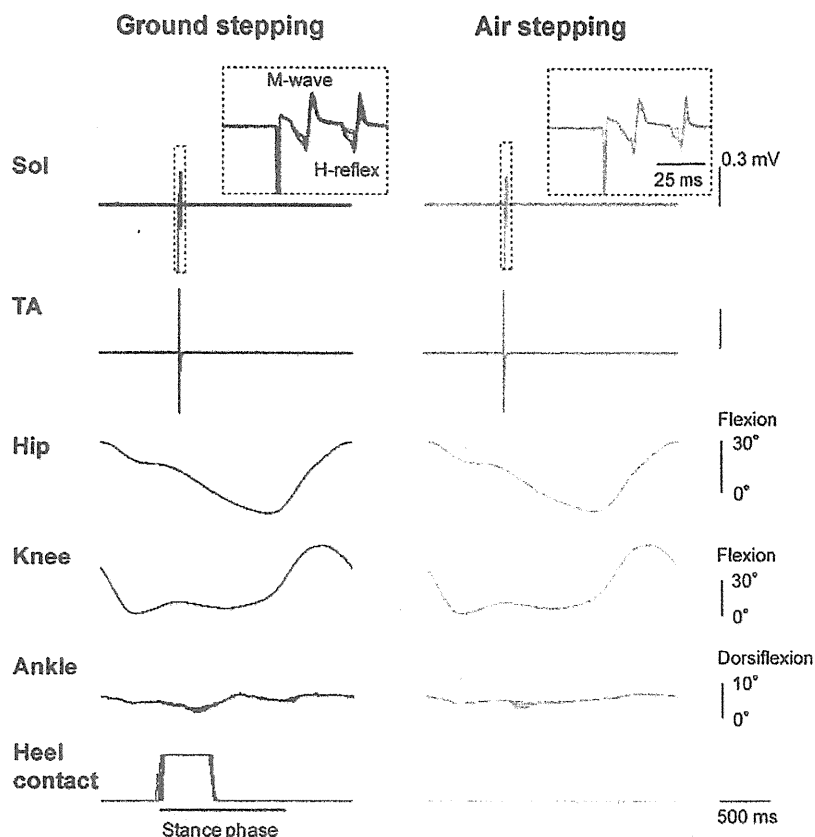


Fig. 2 Typical example of superimposed waveforms (10 sweeps) from the electromyographs (EMGs) of the soleus (Sol) and tibialis anterior (TA) muscles, angles of hip, knee, and ankle joints, and heel contact during ground and air stepping in a healthy subject. The electrical stimuli were applied at the early stance phase. The evoked responses are enlarged at each stepping condition



similar pattern in both loading conditions because of foot orthosis, but the angular variation in the stance phase increased slightly during ground stepping. No EMG activity was observed in the Sol and TA muscles at both loading conditions during passive stepping in this subject. Stimuli to evoke the H-reflex were applied at the early stance phase, and the evoked H-reflex waveforms are shown in the enlarged display. Due to the minimal variability in joint trajectories and muscle activities during stepping, the H-reflexes with a constant M-wave size could be easily evoked.

Figure 3 shows raw waveforms of the Mmax and H-reflex responses at two standing conditions and at six phases of two stepping conditions from a healthy subject. The stimulus intensity for the H-reflex was adjusted to evoke an M-wave size of 10% Mmax. Five sweeps for the Mmax and ten sweeps for the H-reflex are superimposed at each condition in this figure. There was phase-dependent modulation of the H-reflex excitability in both passive stepping conditions, showing that the Sol H-reflex at the early swing phase was markedly suppressed. In the present study, the H-reflex at the swing phase was completely suppressed in half of the subjects, while a small H-reflex response at the swing phase was observed in the remaining subjects.

Figure 4 represents the mean values of M-wave, H-reflex, and background EMG levels during passive stepping for all healthy subjects. The mean background EMG level of the Sol muscle, normalized to Mmax, was 0.09% Mmax for air standing and 0.08–0.09% Mmax through the six phases of air stepping. During ground standing, the background EMG level in the Sol was 0.0046 ± 0.0018 mV, which corresponding to 0.13% Mmax. Small muscle activation in the Sol was observed at the late-stance phase of ground stepping in four of ten subjects. The mean Sol background levels were 0.08–0.11% Mmax through the six phases of ground stepping. Although Sol background EMG levels tended to be larger during ground stepping than during air stepping, two-way ANOVA tests (2 loading conditions \times 6 phases) showed that the main effect of loading was not significant ($F_{1,9} = 5.08$, $P = 0.051$). No significant effects of the phase and loading \times phase interaction were observed in the Sol background EMG. Similarly, background EMG levels in the TA were not significantly different in loading conditions and among the step phases. In the present study, the Mmax size was not measured in the TA muscle. Instead, the EMG level during maximum voluntary contraction was recorded. The TA background levels, normalized by the EMG level during maximum voluntary

Fig. 3 Typical superimposed maximum M-wave (Mmax, 5 sweeps) and H-reflex (10 sweeps) waveforms with an M-wave size of 10% Mmax at standing and at six phases of passive stepping in a healthy subject

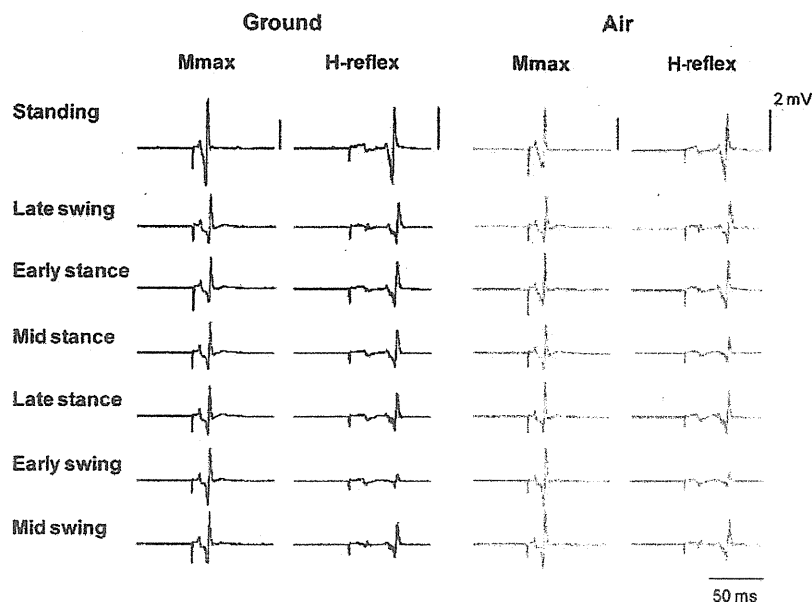
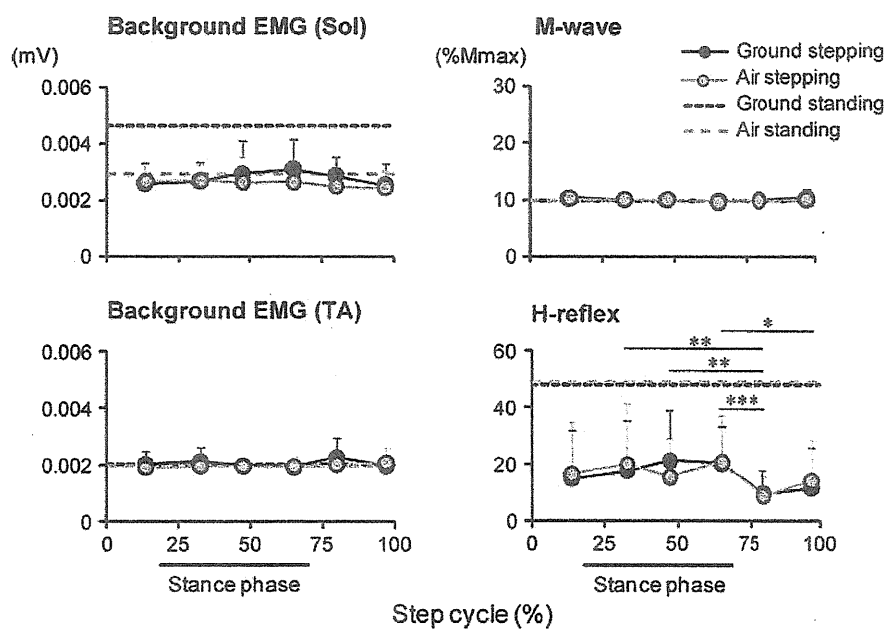


Fig. 4 Mean M-wave, H-reflex, and background EMG activity in the soleus (Sol) and tibialis anterior (TA) muscles at each phase during passive ground and air stepping in all healthy subjects. Significant difference between step phases during passive stepping, * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. The values for two unloading standing conditions are shown by *black* (ground standing) and *gray* (air standing) *dashed lines*



contraction, were less than 1% for both the standing conditions and at each phase for both stepping conditions.

In standing condition, the mean sizes of the H-reflex with M-wave corresponding to 10% of Mmax were not significantly different between ground and air standing (ground standing $48.3 \pm 22.4\%$ Mmax, air standing $49.1 \pm 25.8\%$ Mmax). Although the M-wave size was constant throughout the recording, the H-reflexes evoked at each phase during passive stepping were significantly inhibited compared to those at standing. Two-way ANOVA for the H-reflex during stepping revealed a significant main effect for phase ($F_{5,45} = 3.61$, $P < 0.05$), but

not for loading ($F_{1,9} = 0.07$, $P > 0.05$), and no loading \times phase interaction was observed ($F_{5,45} = 1.63$, $P > 0.05$). These results suggest that the H-reflex excitability was modulated in a phase-dependent manner during passive stepping, but no difference between ground and air stepping conditions. Post hoc test demonstrated that the Sol H-reflex was significantly inhibited at the early swing phase compared to the three stance phases ($P < 0.01$), and at the mid-swing phase compared to the late-stance phase ($P < 0.05$).

Figure 5 shows the M-wave and H-reflex (H-M) recruitment curves at air standing and at the stance and

Fig. 5 Examples of H-M recruitment curves at standing and at the swing and stance phases of passive air stepping from three healthy subjects. Each plot shows the mean value of three responses at each stimulus intensity

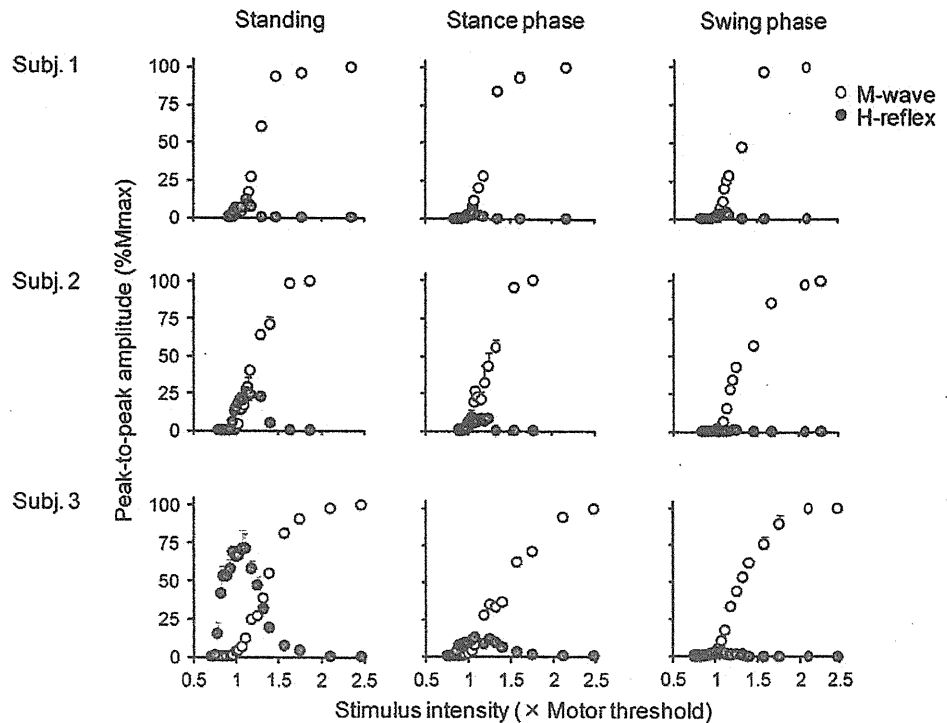
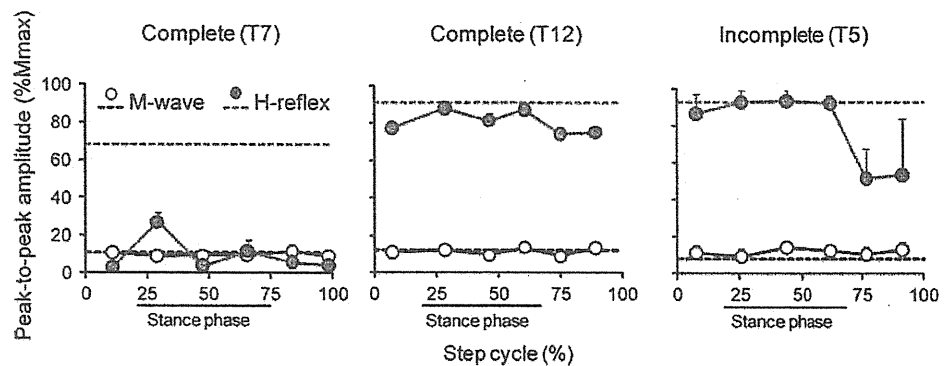


Fig. 6 Mean M-wave and H-reflex amplitudes during passive ground stepping in three spinal cord-injured (SCI) patients. The black and gray dashed lines represent the mean amplitudes of the M-wave and H-reflex at ground standing, respectively



swing phases of air stepping from three healthy subjects. The angle of the hip joint was similar at the instant when these H-M recruitment curves were measured. Although there were marked differences in the maximal H-reflex amplitude at the standing posture among the three subjects, the maximal H-reflex amplitude decreased from the standing posture to the stance and to the swing phase in all subjects. In addition, it was evident that the H-reflex recruitment curves at the swing phase of passive stepping were reduced across a wide range of stimulus intensities.

Furthermore, in the present study, we recorded H-reflex during ground stepping for two complete and one incomplete SCI patients. These patients showed elevated H-reflex amplitudes at ground standing (mean value in three SCI patients: $84.3 \pm 12.0\%$ Mmax, Fig. 6). During ground stepping, spastic muscle activities were rarely observed for

a short time at the beginning of stepping and after the electrical stimulation in two SCI patients. Therefore, we applied the simulation when no spastic EMG activation was observed. Although the extent of H-reflex inhibition during ground stepping differed among the three patients (Fig. 6), the mean H-reflex amplitude at three (early-, mid-, and late-) swing phases was smaller than that at three stance phases (mean values at the stance and swing phases for all SCI patients: 64.2 ± 43.5 and $47.9 \pm 38.3\%$ Mmax, respectively).

Discussion

The aim of this study was to clarify the effect of peripheral sensory inputs on the Sol H-reflex excitability during

walking. In the present study, passive stepping was conducted using a DGO in healthy subjects and SCI patients who were asked to keep their lower-limb muscles relaxed during stepping. During passive stepping, the H-reflexes were inhibited at the swing phase compared to the stance phase, which showed phase-dependent modulation. No significant difference was observed in the H-reflex excitability between different body weight unloading conditions for passive stepping.

Mechanism for H-reflex modulation during passive stepping

A constant M-wave size normalized to Mmax is typically used as an index of stimulus intensity to excite the group Ia afferents for H-reflex experiments to dynamic movement in humans. However, there is a possibility that the Mmax amplitude itself differs among the step phases due to changes in muscle geometry with respect to the surface electrodes and stimulus efficacy to nerves (Simonsen and Dyhre-Poulsen 1999). Therefore, in the present study, the Mmax size was recorded at each phase of stepping, and the H-reflex was evoked at a stimulus intensity in which the M-wave amplitude was 10% Mmax (Fig. 3). Compared to the stance phase, the Sol H-reflex with a constant M-wave was markedly reduced in the swing phase of the passive stepping (Fig. 4). Moreover, the H-reflex amplitudes across a wide range of stimulus intensities were inhibited from the standing posture to the stance phase to the swing phase (Fig. 5), indicating that any change in the H-reflex amplitude during passive stepping was not due to a change in the stimulus efficacy to the posterior tibial nerve.

The present study attempted to reduce the effect of voluntary command on the Sol H-reflex during walking by applying DGO to the healthy subjects. Indeed, no TA or Sol EMG activity was observed during ground and air stepping, except the Sol EMG activity at the latter-stance phase of ground stepping (Figs. 2, 4). Thus, with substantially reduced descending drive to the stepping task in healthy subjects, the results obtained during passive stepping would provide the effect of peripheral sensory inputs on the H-reflex excitability. Moreover, we investigated the H-reflex modulation during passive stepping in the SCI patients whose commands from the supraspinal center were completely or incompletely interrupted. Three SCI subjects who showed exaggerated H-reflex excitability at standing posture revealed that the Sol H-reflex was inhibited at the swing phase of passive stepping (Fig. 6). Recently, Phadke et al. (2007) investigated the effects of different walking environments (treadmill with body weight support and manual assistance by trainers as well as overground walking with an assistive device and brace) on the Sol H-reflex in two phases of mid-stance and mid-swing in

incomplete SCI patients. They found that the H-reflexes during mid-swing phase were smaller than those during mid-stance phase for both walking environments in incomplete SCI patients. While they investigated the Sol H-reflex during active voluntary stepping for incomplete SCI, we also found a similar tendency for H-reflex modulation in six phases during passive stepping in SCI patients.

From previous studies, it is known that passive movements around the hip or knee joint significantly inhibit the Sol H-reflex. For example, reflex inhibition appears at the flexion phase of hip and knee joints, and peaks close to full flexion during passive pedaling (Cheng et al. 1995). Knikou et al. (2007) reported that the passive flexion of the hip significantly depressed the Sol H-reflex excitability when SCI patients were in the supine position. Moreover, afferent signals from the quadriceps muscles in decerebrate cats inhibit the Sol H-reflex during locomotion (Misiasek and Pearson 1997). These results suggest that the inhibition of the H-reflex arises from movement-elicited sensory receptors discharging at the hip and knee joints. The findings of the present study also demonstrated that the H-reflex inhibition increased at the early- and mid-swing phases when the hip and/or knee joints were flexed. Therefore, the source underlying the phase-modulation of the H-reflex during passive stepping can be attributed to the sensory inputs, which likely arise from muscle spindle primary endings when the joints are flexed. The modulation pattern of the Sol H-reflex during passive stepping was similar to that observed during normal walking (Capaday and Stein 1987; Simonsen and Dyhre-Poulsen 1999; Ethier et al. 2003). Therefore, it is considered that the modulation pattern in the Sol H-reflex excitability during normal walking might be largely formed by peripheral sensory inputs associated with the lower-limb movements.

It is widely thought that phase-modulation of the Sol H-reflex during normal walking is associated with presynaptic inhibition of the Ia terminal (Capaday and Stein 1987; Crenna and Frigo 1987; Zehr 2002). Because no EMG activity in the TA muscle was observed during stepping in the present study, it appears that postsynaptic effect from reciprocal inhibition might play a minor role in the Sol H-reflex inhibition. In addition, it has been reported that the Sol H-reflex was still inhibited during passive movement with voluntary tonic contraction of the Sol muscle (Brooke et al. 1995; Misiasek et al. 1995). When excitability in the motoneuron pool is stabilized by tonic contraction during passive movement, the postsynaptic effects on the H-reflex inhibition are likely to be minimized. Thus, it appears that the major source of the H-reflex modulation during passive stepping can be attributed to the presynaptic inhibition. However, the postsynaptic effect on the H-reflex modulation cannot be excluded in the present study. Because

passive stepping was performed without the background EMG, any change in the subliminal fringe of the motoneuron during passive stepping was unknown. The change in the resting membrane potential of the motoneuron dependent on a step cycle might be partly involved in the H-reflex modulation during passive stepping.

Methodological limitations

In the present study, passive stepping was used to investigate the role of sensory inputs on the H-reflex modulation during walking. Although it appears that the modulation observed in the present study was generated by processes within the spinal cord, a few methodological limitations should be noted for the interpretation of these results. By instructing the subjects to relax during passive stepping, EMG activities in the lower limb muscles were not observed. However, the nearly complete disappearance of the EMG activity may not be a sufficient criterion to make the claim that there was no influence of descending drive on the H-reflex excitability. Also, because the sensory information that the subjects received during passive stepping with body weight unloading was different from that during normal walking, even a minor effect from the cortex due to the novel sensory perceptions during passive stepping may be related to modulations of the H-reflex. Furthermore, a possible implication of descending drive on the H-reflex excitability cannot be completely excluded, even in complete SCI patients, as it has been reported that a small percentage of individuals designated as complete SCI converted to incomplete SCI within 1 year after injury (Marino et al. 1999). Thus, the effect of the supraspinal input on the H-reflex modulation might not have been entirely eliminated in the present study.

Comparisons of reflex modulation during passive stepping and normal walking

In the present study, half of the subjects showed almost complete suppression at the swing phases of both stepping conditions, while the remaining subjects showed small H-reflex responses. Such inter-individual differences in the H-reflex inhibition have also been observed during normal walking (Simonsen et al. 2002). However, the amount of change in the H-reflex amplitude through the step cycle during passive stepping appeared to be less than that observed during normal walking. During the stance phase of normal walking, the Sol H-reflex increases progressively, nearly in parallel with the Sol background EMG (Capaday and Stein 1986). Ethier et al. (2003) demonstrated that, in most subjects, the H-reflex amplitude at late-stance during normal walking was larger than that during standing. However, the present study found that the

H-reflex excitability at the stance phase in both loading conditions during passive stepping was significantly lower than that during standing. The lower H-reflex excitability in the stance phase during passive stepping when compared with normal walking might be attributed to little or no Sol EMG activity. As for the swing phase of normal walking, the H-reflex is almost completely suppressed due to the relevance of reciprocal inhibition, while TA is active (Ethier et al. 2003). Schneider et al. (2000) also observed that during the swing phase in a knee-locked walking task, the H-reflex inhibition was correlated with the TA EMG activity, and suggested that the modulation pattern during human walking follows the centrally produced reciprocal inhibition. In the present study, the H-reflexes were not completely suppressed at the swing phase of passive stepping in half of the subjects, presumably due to the absence of TA EMG activity. Thus, the EMG activity of the Sol and TA muscles that occurs normally during walking might quantitatively contribute to phase-modulation of the H-reflex. In addition, the difference in stepping speed between normal walking and this passive stepping condition might also be involved in the amount of the H-reflex inhibition during the swing phase because attenuation in the Sol H-reflex would depend on the velocity of the joint movement (McIlroy et al. 1992; Cheng et al. 1995).

Effect of load-related sensory inputs on reflex modulation during passive stepping

Inputs from load-related receptors during normal locomotion are well recognized to be significant for neural control (Harkema et al. 1997; Van de Crommert et al. 1998; Dietz and Duysens 2000; Dietz et al. 2002). Dietz et al. (2002) showed that afferent inputs from the hip joint in combination with those from the load receptors play crucial roles in generation of locomotor activity in SCI patients. However, the extent to which the Sol H-reflex is modulated by the load-related sensory inputs during walking is unclear. In the present study, there was no foot contact with the treadmill during air stepping (Fig. 2). Knikou and Conway (2001) observed that during sitting, the Sol H-reflex is inhibited by pressure (15–80 N) applied to the foot sole in healthy subjects and SCI patients. Therefore, we hypothesized that the Sol H-reflex during passive stepping would be inhibited by body weight loading. However, no significant difference was observed in the H-reflex between air and ground stepping in the present study (Fig. 4), i.e., no effect of the load-related sensory information was observed on the Sol H-reflex excitability during passive stepping. It is well known that the Sol H-reflex excitability is inhibited in a task-dependent manner from lying to sitting, to standing, and to walking (Capaday and Stein 1987; Crenna

and Frigo 1987; Brooke et al. 1991; Zehr 2002; Stein and Thompson 2006). Similarly, the difference of the load-related effect on the H-reflex excitability between sitting and passive stepping could be explained by task dependency. Alternatively, the different loading effects on the H-reflex between sitting and stepping may be attributed to the differences in pressure to the foot sole. While tonic pressure was applied to the metatarsal region of the foot sole in the sitting condition (Knikou and Conway 2001), during walking, the focus of the pressure on the foot sole in stance phase moved from the heel toward the toe, and the amount of foot pressure was also changed through the stance phase (Nakajima et al. 2008). Thus, the difference in the sensory feedback provided by the pressure on the foot sole might possibly account for the inconsistent results for the loading effects on the H-reflex excitability between two different tasks.

Regarding the load effect on the transmission in other afferent pathways, Faist et al. (2006) reported that Ib inhibition from the stimulation of the gastrocnemius nerve to the Sol H-reflex was reduced by loading of the leg, regardless of motor tasks. In the present study, although the load-related afferent inputs were involved during ground stepping, there were no significant differences in modulation of the Sol H-reflex between ground and air stepping. However, it was observed that slight difference of the H-reflex amplitude between two stepping conditions was observed only in the mid-stance phase (Fig. 4). During passive stepping on a treadmill, foot sole pressure was the greatest in this phase (Nakajima et al. 2008). Regarding the effect of loading on Ib inhibition during stepping, additional investigations may be required.

As for the effect of load on the cutaneous reflex pathway, Bastiaanse et al. (2000) previously suggested that load-related afferent inputs were involved in the regulation of the cutaneous reflex in the lower limb muscles evoked by sural nerve stimulation during normal walking. In addition, using the same DGO as in the present study, we have recently observed strong facilitation of the cutaneous reflex in the TA muscle during the late-stance to early swing phase of passive ground stepping, but not during passive air stepping (Nakajima et al. 2008). These results suggest that the load-related sensory inputs play a key role in modulation of cutaneous reflexes during walking. In contrast, no significant difference was observed in the H-reflex between ground and air stepping in the present study. Also in a recent study by Knikou et al. (2009), it was reported that Sol H-reflex modulation remained constant across 0, 25, and 50% body weight support levels during treadmill walking for healthy subjects. Thus, it appears that the effects of load-related sensory inputs to the reflex responses during stepping are different between the cutaneous and the H-reflex pathways.

Acknowledgments This study was partially supported by the Grant-in-Aid for Young Scientists (B) (No. 19700460) from the Japanese Ministry of Education, Culture, Sports, Science and Technology (MEXT) and Global COE program on “Cybernetics: fusion of human, machine, and information systems” from the Japan Society for the Promotion of Science.

References

- Bastiaanse CM, Duysens J, Dietz V (2000) Modulation of cutaneous reflexes by load receptor input during human walking. *Exp Brain Res* 135:189–198
- Brooke JD, Collins DF, Boucher S, McIlroy WE (1991) Modulation of human short latency reflexes between standing and walking. *Brain Res* 548:172–178
- Brooke JD, Misiaszek JE, Cheng J (1993) Locomotor-like rotation of either hip or knee inhibits soleus H reflexes in humans. *Somatosens Mot Res* 10:357–364
- Brooke JD, Cheng J, Misiaszek JE, Lafferty K (1995) Amplitude modulation of the soleus H reflex in the human during active and passive stepping movements. *J Neurophysiol* 73:102–111
- Capaday C, Stein RB (1986) Amplitude modulation of the soleus H-reflex in the human during walking and standing. *J Neurosci* 6:1308–1313
- Capaday C, Stein RB (1987) Difference in the amplitude of the human soleus H reflex during walking and running. *J Physiol* 392:513–522
- Cheng J, Brooke JD, Misiaszek JE, Staines WR (1995) The relationship between the kinematics of passive movement, the stretch of extensor muscles of the leg and the change induced in the gain of the soleus H reflex in humans. *Brain Res* 672:89–96
- Colombo G, Joerg M, Schreier R, Dietz V (2000) Treadmill training of paraplegic patients using a robotic orthosis. *J Rehabil Res Dev* 37:693–700
- Crenna P, Frigo C (1987) Excitability of the soleus H-reflex arc during walking and stepping in man. *Exp Brain Res* 66:49–60
- Dietz V, Duysens J (2000) Significance of load receptor input during locomotion: a review. *Gait Posture* 11:102–110
- Dietz V, Müller R, Colombo G (2002) Locomotor activity in spinal man: significance of afferent input from joint and load receptors. *Brain* 125:2626–2634
- Duysens J, Pearson KG (1980) Inhibition of flexor burst generation by loading extensor muscles in walking cats. *Brain Res* 187:321–332
- Duysens J, Clarac F, Cruse H (2000) Load-regulating mechanisms in gait and posture: comparative aspects. *Physiol Rev* 80:83–133
- Ethier C, Imbeault MA, Ung V, Capaday C (2003) On the soleus H-reflex modulation pattern during walking. *Exp Brain Res* 151:420–425
- Faist M, Hofer C, Hodapp M, Dietz V, Berger W, Duysens J (2006) In humans Ib facilitation depends on locomotion while suppression of Ib inhibition requires loading. *Brain Res* 1076:87–92
- Garrett M, Kerr T, Caulfield B (1999) Phase-dependent inhibition of H-reflexes during walking in humans is independent of reduction in knee angular velocity. *J Neurophysiol* 82:747–753
- Grey MJ, Nielsen JB, Mazzaro N, Sinkjær T (2007) Positive force feedback in human walking. *J Physiol* 581:99–105
- Harkema SJ, Hurley SL, Patel UK, Requejo PS, Dobkin BH, Edgerton VR (1997) Human lumbosacral spinal cord interprets loading during stepping. *J Neurophysiol* 77:797–811
- Knikou M, Conway BA (2001) Modulation of soleus H-reflex following ipsilateral mechanical loading of the sole of the foot in normal and complete spinal cord injured humans. *Neurosci Lett* 303:107–110

- Knikou M, Schmit BD, Chaudhuri D, Kay E, Rymer WZ (2007) Soleus H-reflex excitability changes in response to sinusoidal hip stretches in the injured human spinal cord. *Neurosci Lett* 423:18–23
- Knikou M, Angeli CA, Ferreira CK, Harkema SJ (2009) Soleus H-reflex modulation during body weight support treadmill walking in spinal cord intact and injured subjects. *Exp Brain Res* 193:397–407
- Lavoie BA, Devanne H, Capaday C (1999) Differential control of reciprocal inhibition during walking versus postural and voluntary motor tasks in humans. *J Neurophysiol* 78:429–438
- Marino RJ, Ditunno JF, Donovan WH, Maynard F (1999) Neurologic recovery after traumatic spinal cord injury: data from the model spinal cord injury systems. *Arch Phys Med Rehabil* 80:1391–1396
- McIlroy WE, Collins DF, Brooke JD (1992) Movement features and H-reflex modulation. II. Passive rotation, movement velocity and single leg movement. *Brain Res* 582:85–93
- Misiaszek JE, Pearson KG (1997) Stretch of quadriceps inhibits the soleus H reflex during locomotion in decerebrate cats. *J Neurophysiol* 78:2975–2984
- Misiaszek JE, Brooke JD, Lafferty KB, Cheng J, Staines WR (1995) Long-lasting inhibition of the human soleus H reflex pathway after passive movement. *Brain Res* 677:69–81
- Nakajima T, Kamibayashi K, Takahashi M, Komiyama T, Akai M, Nakazawa K (2008) Load-related modulation of cutaneous reflexes in the tibialis anterior muscle during passive walking in humans. *Eur J Neurosci* 27:1566–1576
- Pearson KG, Collins DF (1993) Reversal of the influence of group Ib afferents from plantaris on activity in medial gastrocnemius muscle during locomotor activity. *J Neurophysiol* 70:1009–1017
- Phadke CP, Wu SS, Thompson FJ, Berhman AL (2007) Comparison of soleus H-reflex modulation after incomplete spinal cord injury in 2 walking environments: treadmill with body weight support and overground. *Arch Phys Med Rehabil* 88:1606–1613
- Querry RG, Pacheco F, Annaswamy T, Goetz L, Winchester PK, Tansey KE (2008) Synchronous stimulation and monitoring of soleus H reflex during robotic body weight-supported ambulation in subjects with spinal cord injury. *J Rehabil Res Dev* 45:175–186
- Schneider C, Lavoie BA, Capaday C (2000) On the origin of the soleus H-reflex modulation pattern during human walking and its task-dependent differences. *J Neurophysiol* 83:2881–2890
- Simonsen EB, Dyhre-Poulsen P (1999) Amplitude of the human soleus H reflex during walking and running. *J Physiol* 515:929–939
- Simonsen EB, Dyhre-Poulsen P, Alkjær T, Aagaard P, Magnusson SP (2002) Interindividual differences in H reflex modulation during normal walking. *Exp Brain Res* 142:108–115
- Stein RB, Thompson AK (2006) Muscle reflexes in motion: how, what, and why? *Exerc Sport Sci Rev* 34:145–153
- Van de Crommert HW, Mulder T, Duysens J (1998) Neural control of locomotion: sensory control of the central pattern generator and its relation to treadmill training. *Gait Posture* 7:251–263
- Zehr EP (2002) Considerations for use of the Hoffmann reflex in exercise studies. *Eur J Appl Physiol* 86:455–468



Contents lists available at ScienceDirect

Human Movement Science

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



Temporal correlations in center of body mass fluctuations during standing and walking

Masaki O. Abe^{a,*}, Kei Masani^{b,c}, Daichi Nozaki^d, Masami Akai^e,
Kimitaka Nakazawa^f

^a Action Lab, Department of Biology, Northeastern University, 134 Mugar Life Science Building, 360 Huntington Avenue, Boston, MA 02115, United States

^b Rehabilitation Engineering Laboratory, Institute of Biomaterials and Biomedical Engineering, University of Toronto, 164 College Street, Toronto, ON, Canada M5S 3G9

^c Rehabilitation Engineering Laboratory, Lyndhurst Centre, Toronto Rehabilitation Institute, 520 Sutherland Drive, Toronto, ON, Canada M4G 3V9

^d Physical Education Laboratory, Graduate School of Education, University of Tokyo, 7-3-1 Hongo, Bunkyo-ku, Tokyo 113-0033, Japan

^e Hospital of National Rehabilitation Center for Persons with Disabilities, 4-1 Namiki, Tokorozawa, Saitama 359-8555, Japan

^f Department of Life Sciences, Graduate School of Arts and Sciences, University of Tokyo, 3-8-1 Komaba, Meguro-ku, Tokyo 153-8902, Japan

ARTICLE INFO

Article history:

Available online 6 July 2010

PsycINFO classification:

2330

Keywords:

Balance
Walking
Standing
Center of body mass
Temporal correlation

ABSTRACT

Body fluctuations during both quiet standing and walking exhibit temporal correlations that reflect mechanisms of balance control. However, knowledge about the relationship between the temporal structures observed during standing and walking is limited. The goal of the present study was (1) to investigate temporal correlations of the center of body mass acceleration (ACC) in standing and walking, and (2) to test the hypothesis that the degree of the temporal correlation for the two tasks is similar and correlated across participants. Seventeen young, healthy participants stood and walked for 10 min on a treadmill equipped with two force platforms. The temporal correlations of the ACC in the anteroposterior (ACC_{AP}), mediolateral (ACC_{ML}), and two-dimensional (ACC_{2D}) directions were evaluated using the scaling index (α) as calculated with Detrended Fluctuation Analysis. The scaling indices of ACC fluctuations during standing and walking were categorized as stationary signals which are temporally correlated ($0.5 < \alpha < 1.0$). Further, there were significant, positive correlations for ACC_{AP} and ACC_{2D} between the scaling indices during standing and walking. The results suggest that there are common characteristics in the balance control system for standing and walking.

* Corresponding author. Tel.: +1 617 373 5093; fax: +1 617 373 3724.
E-mail address: m.abe@neu.edu (M.O. Abe).

which may be associated with temporal correlations in COM acceleration.

© 2010 Elsevier B.V. All rights reserved.

1. Introduction

Much research has been devoted to the control mechanisms of standing and walking, which are fundamental behaviors in daily life. Studies of these activities are motivated by scientific interest and the need to find solutions to the problem of motor disabilities that develop as a result of disease and aging. One possible approach to finding a solution is to analyze the characteristics of body fluctuations in standing and walking. The control systems of standing and walking consist of various sensory-motor integrations at multiple levels of the nervous system, and the dynamic interaction between the nervous system and the musculo-skeletal system. Therefore, the study of changes in movement, such as body sway, can be useful in the detection of characteristics of the underlying system.

Many studies have focused on center of pressure (COP) fluctuations during quiet standing, in order to investigate upright postural control. The COP is proportional to the resultant ankle torque, which primarily controls the center of body mass (COM) during quiet standing and is partly regulated by the neural system (Loram, Maganaris, & Lakie, 2005a, 2005b; Masani, Popovic, Nakazawa, Kouzaki, & Nozaki, 2003; Masani, Vette, & Popovic, 2006; Peterka, 2000, 2002). Thus, by investigating COP dynamics, one may gain insight into the neural control of balance. Previous studies have reported that COP fluctuations are not random in time, but have temporal correlations (Collins & De Luca, 1993, 1994, 1995; Collins, De Luca, Burrows, & Lipsitz, 1995; Duarte & Zatsiorsky, 2000, 2001). Collins and colleagues (Collins & De Luca, 1993, 1994, 1995; Collins et al., 1995) believe that such temporal correlations reflect open-loop control in the short-time scale (within one second) and closed-loop control in the long time scale (over one second). Peterka (2000) suggested that this characteristic could be produced by appropriate selection of control parameters in a very simple feedback model that represents body dynamics as an inverted pendulum. Although these studies suggest different mechanisms for the emergence of temporal correlations, the common understanding between these studies is that the neural controller that maintains the COM over the base of support can modulate the temporal correlation. In fact, the degree of the correlation is influenced by aging and disease, which degrade the neural system for balance control (Collins et al., 1995; Laughton et al., 2003; Maurer, Mergner, & Peterka, 2004; Priplata, Niemi, Harry, Lipsitz, & Collins, 2003; Priplata et al., 2006).

Inter-stride fluctuations during walking have been used to investigate the control system of walking, with particular focus on falling in the elderly (Gabell & Nayak, 1984; Hausdorff, Rios, & Edelberg, 2001; Maki, 1997; Masani, Kouzaki, & Fukunaga, 2002; Pailhouse & Bonnard, 1992; Yamasaki, Sasaki, & Torii, 1991). Hausdorff and colleagues (Hausdorff, Peng, Ladin, Wei, & Goldberger, 1995; Hausdorff et al., 1996) demonstrated that the fluctuation of stride intervals shows temporal correlations, and that such time dependent dynamics are influenced by aging and disease (Hausdorff et al., 1997). There are some debates about the emerging process of the temporal correlation in stride intervals; Hausdorff et al. (1995) showed that the central pattern generator (CPG) model with memory function could generate a persistent temporal structure across stride intervals. West and Scafetta (2003) developed the super CPG (SCPG) model to explain the changes in stride interval temporal correlations in fast and slow walking speeds, as well as walking paced by a metronome. While these researchers focused on the neural mechanism that generates a given rhythm, Gates, Su, and Dingwell (2007) demonstrated that interaction among simple neural controllers, the musculo-skeletal system, and noise input could generate temporal correlations. It is unknown, however, if balance control in walking relates to the emergence of the temporal correlation observed in stride interval fluctuation. Furthermore, it has not been determined if the body fluctuations during walking show the temporal correlations that have been reported in body fluctuations while standing.

As a logical development, the next question would be whether body fluctuations in walking have temporal correlations similar to those in standing, and if so, how the structure in the body fluctuations during standing and walking are related. While the COM dynamics and CNS control strategies used in walking are generally considered to be different from that of standing (Winter, 1995), the control of posture and gait share the same neural (Mori, 1987, 1989; Mori, Nakajima, Mori, & Matsuyama, 2004; Morton & Bastian, 2003, 2004) and musculo-skeletal systems. In addition, both tasks have a common goal; the COM position must be controlled to prevent falling. We hypothesize that the temporal correlations in body fluctuations during standing and walking could be similar and correlate with each other among a group of people, i.e., those who have a strong correlation in walking would have a similarly strong correlation in standing.

A few studies have compared the stability between standing and walking. Shimada et al. (2003) investigated the response to a perturbation applied to the body during standing and walking, and demonstrated that the responses between the motor tasks were unrelated. Kang and Dingwell (2006) investigated the trunk motion during standing and walking, and analyzed its stability using local dynamic stability analysis. They measured the trajectory of the trunk in state space in standing and walking. They then estimated the rate of the divergence of the trajectory to the neighboring one, which describes local dynamic stability, i.e., faster divergence indicates greater instability. They found that the parameters of the divergence curves between standing and walking had no correlation, suggesting that the mechanisms governing standing and walking stability are different. Since temporal correlations during standing and walking may be affected by the stability of the control system of each task, their results could provide evidence against the above-mentioned hypothesis. However, as far as we know, no study has directly compared temporal correlations of body fluctuations among strides during walking with body fluctuations during standing.

In order to directly compare temporal correlations in body sway during standing and walking, we used “COM acceleration” (ACC) as a measure of body fluctuation since ACC is one of the representative and commonly measurable parameters of body behavior during the motor tasks. Therefore, the purposes of this study were to (1) investigate the temporal correlations for ACC in standing and walking, and to (2) test the hypothesis that the degree of temporal correlation in ACC time-series during standing and walking are related. A preliminary account of the results was published in abstract form (Abe, Nakazawa, Masani, & Akai, 2004).

2. Methods

2.1. Participants

Seventeen healthy subjects (9 males and 8 females), aged 21–34 years, participated in this study. The mean (\pm standard deviation: *SD*) height and weight of all participants was 1.72 ± 0.07 m and 64.5 ± 8.4 kg, respectively. None of the participants had a history of motor disorder. Informed consent was obtained from all participants prior to their participation in this study. The experimental procedure used was approved by the local ethics committee.

2.2. Apparatus

To obtain ACC during standing and walking, we recorded ground reaction forces (GRF) using a treadmill equipped with two force platforms (ADAL3D, Techmachine, Andrézieux-Bouthéon, France). GRF data in mediolateral (ML), anteroposterior (AP), and vertical (VL) directions from each force platform were recorded. The natural frequency of this apparatus was over 120 Hz and the linearity was ensured by the manufacturer to range from 0 to 3000 N for the vertical components and 500 N for the horizontal components. Belli, Bui, Berger, Geyssant, and Lacour (2001) examined the same type of treadmill in detail and concluded that the treadmill would be accurate for analyzing human gait behavior.

2.3. Protocol

Participants performed two tasks: a standing task and a walking task. In the standing task, participants were asked to stand still without moving their arms or feet for 10 min on the treadmill. The heel-to-heel distance between feet was about 6 cm. In the walking task, the participants were asked to walk for 12 min on the treadmill with the belt speed set at 1.1 m/s. All participants reported that the speed was natural, and that they performed the task easily without feeling excessive effort and fatigue. Before the recording session, participants performed a five-minute practice session. There were five-minute breaks between the pre-training and the experimental trials in walking tasks, and 10-min breaks between the standing and walking tasks. The length of each break was decided based on the participants' objective comments. They were able to execute the next task comfortably after the break.

2.4. Data analysis

The GRF data was recorded at 100 Hz using a 16-bit AD converter (WE7000, Yokogawa, Tokyo, Japan) and stored on a personal computer. All data were digitally low-pass filtered with a zero-phase-lag 10th order FIR filter with a Blackman window at a cut-off frequency of 20 Hz. This filtering removed noise artifacts during push-off, and enabled the moment of heel contact to be accurately identified. For the walking task, the last 10 min of data (out of 12 min) were used in the subsequent analysis. The standing and walking data in the AP and ML directions for 10 min were used to calculate COM acceleration in AP (A_{AP}) and ML (A_{ML}) directions as follows:

$$\begin{aligned} A_{AP}(t) &= F_{AP}(t)/m \\ A_{ML}(t) &= F_{ML}(t)/m \end{aligned} \quad (1)$$

where m is the mass of the body, and F_{AP} and F_{ML} are GRF data in AP and ML directions, respectively. Note that we only focused on balance control in horizontal plane and therefore did not analyze the vertical force in the present study, since vertical movement during standing is not prominent. The ACC absolute value in each direction (ACC_{AP} and ACC_{ML} for AP and ML directions, respectively) and the magnitude of the two-dimensional ACC vector (ACC_{2D}) were calculated for each trial as follows:

$$\begin{aligned} ACC_{AP}(t) &= \sqrt{A_{AP}(t)^2} \\ ACC_{ML}(t) &= \sqrt{A_{ML}(t)^2} \\ ACC_{2D}(t) &= \sqrt{A_{AP}(t)^2 + A_{ML}(t)^2} \end{aligned} \quad (2)$$

First, we assessed stride-to-stride fluctuations in the walking data by partitioning the time-series into individual strides. The stride interval was defined as the moment from one heel contact to the next ipsilateral heel contact. Heel contact was identified as the time when the vertical GRF reached 10% of participants' body mass. For all participants, the stride interval was about 1 s (1.09 ± 0.04 s, mean \pm SD) and contained roughly 500–600 data points. For each stride interval, the average magnitudes of ACC_{AP} , ACC_{ML} , and ACC_{2D} were calculated as in Eq. (2). This created a new time-series for each variable that consisted of these average ACC magnitudes, in which the number of data points was equal to the number of stride intervals. Then based on these stride intervals, the entire sequence of ACCs in the standing task was divided into bins in which the data length was defined by the average stride interval for each participant. Fig. 1 represents typical examples of the ACC series for standing and walking. The number of data points was equal in the standing and walking tasks. Table 1 shows a summary of average and standard deviations for the ACC time-series.

To investigate temporal correlations in ACC, we used Detrended Fluctuation Analysis (DFA) (Peng, Havlin, Stanley, & Goldberger, 1995; Peng et al., 1993). DFA evaluates degree of correlation in a time-series by a parameter referred to as the scaling index (α). The scaling index is quantified by calculating the slope of the line relating $\log F(n)$ to $\log n$, where n and $F(n)$ represent the window size and the variance of the time-series within each window (of size n), respectively (Fig. 2). In DFA, the data

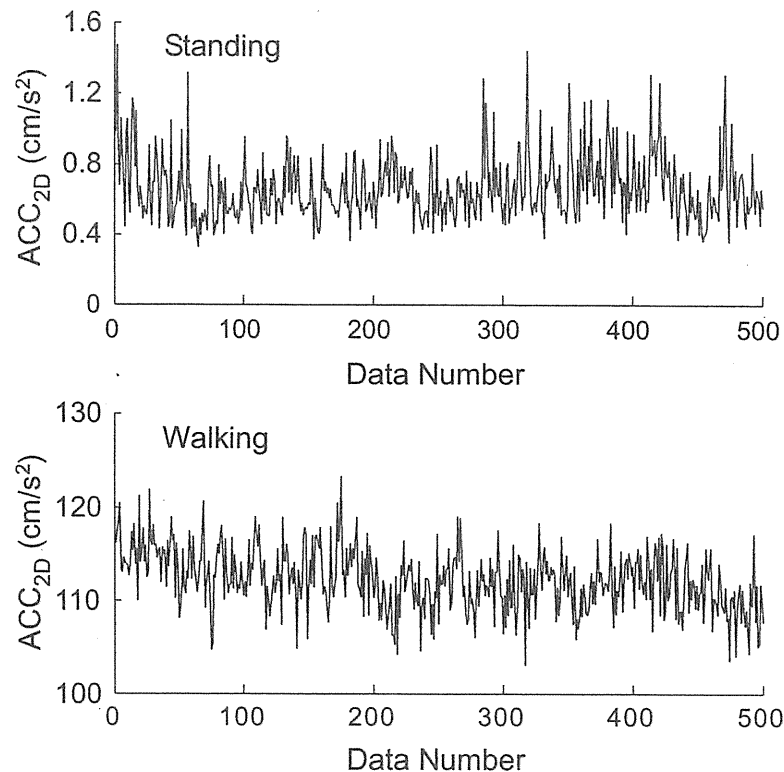


Fig. 1. Typical examples of the fluctuations of ACC_{2D} in standing (top) and walking (bottom).

Table 1
Mean and standard deviation of ACC.

	Mean (cm/s^2)		Standard deviation (cm/s^2)	
	Standing	Walking	Standing	Walking
ACC_{AP}	0.576 ± 0.126	75.7 ± 9.6	0.235 ± 0.070	4.09 ± 0.67
ACC_{ML}	0.330 ± 0.077	58.0 ± 7.7	0.161 ± 0.048	2.87 ± 0.58
ACC_{2D}	0.752 ± 0.136	99.7 ± 10.1	0.261 ± 0.071	3.51 ± 0.59

was normalized with mean = 0 and $SD = 50$, and integrated for the calculation of slope. If the scaling index (α) is less than 1, the time-series is categorized as a stationary signal (Eke, Herman, Kocsis, & Kozak, 2002). In addition, if there are no correlations between past and future fluctuations in the time-series, as in *white noise*, the scaling index will be $\alpha = 0.5$. When the signal is temporally correlated, the scaling index ranges from 0.5 to 1.0 (Eke et al., 2002). Note that $\alpha > 0.5$ in DFA does not necessarily indicate “long-range” correlations, as time-series having short-range correlations also can show $\alpha > 0.5$ in DFA (Maraun, Rust, & Timmer, 2004; Wagenmakers, Farrell, & Ratcliff, 2004, 2005). In this study, we used the scaling index simply as an estimation of the degree of time correlation. In stationary signals ($0.5 < \alpha < 1.0$), the scaling index is statistically equivalent to the degree of the decay of autocorrelation and power spectrum. Peng et al. (1993) demonstrated that the measurement of scaling index could dramatically reduce the influence of artifact noise compared to the other analyses.

The range of the slope calculation was from the 8th step to the 28th step ($8 < n < 28$) in which the slopes were straight. To validate our method for calculating the scaling index, we created twenty time-series with $\alpha = 0.5, 0.6, 0.7, 0.8, 0.9, 1.0$ (data length of 512) by using a previously validated method (Peng et al., 1993), and calculated the scaling indices using the above-mentioned method. The mean of the scaling indices for the simulated time-series was within 5% of the theoretical value as a result.

Although we recorded 10 min of data in standing and walking in order to compare their scaling index by the same time scale and same data number, these tasks might be affected by boredom

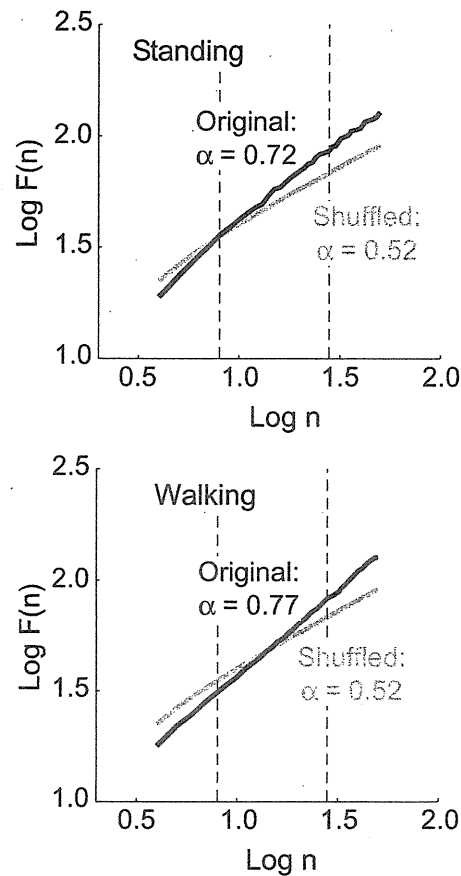


Fig. 2. Ensemble plots of detrended fluctuation analysis for ACC_{2D} . Black lines show the results of original data and gray lines show the results of shuffled surrogate data. The dotted lines represent the range for the slope calculation. The slopes, i.e., the scaling indices (α) are shown for each plot.

and fatigue. In particular, the standing task might be unnaturally long compared to standing in daily life. To check the influence of time effects over the scaling indices, the 10 min of data were divided into two bins (2×5 min) and scaling indices for these bins were compared in each direction by using a paired *t*-test. The test showed no significant difference between scaling indices in the two time bins for both tasks in three directions ($p > .05$ in all cases), suggesting that the effect of physical/mental fatigue on the scaling index of body fluctuation in standing and walking was not particularly critical in this study. Thus, we used the ACC series for 10 min in the subsequent analysis.

We performed a “shuffled” surrogate test (Scheinkman & LeBaron, 1989) for the scaling indices. While this test cannot distinguish white noise from linear-filtered white noise, we used it in order to confirm that the scaling index was generated from the temporal structure of the ACC series and not merely from the amplitude distribution (Theiler, Eubank, Longtin, Galdrikian, & Farmer, 1992). Twenty shuffled data sets from each of the ACC data were made by randomly shuffling the temporal order of the original data, and the averaged scaling index for the shuffled data set was compared with that of the original data by a paired *t*-test at each task and direction.

A comparison of the scaling indices of ACC in each condition was done by repeated measures two-way (Direction \times Task) ANOVA with a Bonferroni post hoc test. The cross correlation between the scaling indices in standing and walking tasks in each direction was tested using Pearson’s correlation coefficient. The significant levels of all statistical tests including the above analyses were set to $p < .05$.

3. Results

Fig. 2 shows the averaged DFA results for the original ACC_{2D} data and the shuffled data in standing and walking tasks for all participants. As shown in this figure, the scaling index of the original data was

much larger than 0.5 in both the standing and walking tasks, while the shuffled data set showed the scaling indices close to 0.5. Table 2 summarizes the results of the scaling indices of each ACC component for the standing and walking tasks. The scaling indices for the standing and walking tasks ranged from 0.524 to 0.915 and from 0.602 to 1.006, respectively. The ANOVA showed a significant interaction between conditions, $F(2, 32) = 5.267, p = .011$. The post hoc test for each direction showed a significant difference in the scaling indices for ACC_{ML} between standing and walking tasks ($p = .037$). There were no significant differences for ACC_{AP} and ACC_{2D} between the tasks ($p > .05$). Table 2 also shows the scaling indices for shuffled data. Paired t -tests between scaling indices in the original and shuffled data showed that the scaling indices of the original data were significantly larger than that of shuffled data for both tasks in all directions ($p < .05$).

Fig. 3 shows the relationships of the scaling indices for each ACC parameter between the standing and walking tasks across all participants. In all figures, the horizontal axis represents the scaling indices of the standing task and the vertical axis represents the scaling index of the walking task. There were strongly significant correlations between the scaling indices from the standing and walking tasks for ACC_{AP} ($r = .593, p = .012$) and ACC_{2D} ($r = .612, p = .009$). For ACC_{ML} , there was no significant correlation between the scaling indices during standing and walking ($r = -.081, p = .758$).

Previous studies for walking focused on the long-time correlation of the stride interval. Thus, we also compared scaling indices of ACC in walking with scaling indices of stride interval in walking. The averaged scaling index of the stride interval was 0.780 ± 0.112 . There were no significant correlations between the scaling indices for stride interval and for ACC during the walking task in each direction ($r = .358, p = .159$ in ACC_{AP} , $r = -.128, p = .624$ in ACC_{ML} , and $r = .335, p = .189$ in ACC_{2D}).

4. Discussion

4.1. Temporal correlations in ACC fluctuations during standing and walking

The scaling indices of ACC for each direction ranged from 0.60 to 1.01 for walking and 0.52 to 0.92 for standing (Table 2 and Fig. 3). Except for one scaling index for ACC_{ML} in walking, these values were less than one, meaning that the fluctuations of these signals were stationary (Eke et al., 2002). This suggests that participants in this study controlled the center of body mass in both standing and walking so that the amplitude of the acceleration was maintained at a certain level. In addition, all parameters of ACC_{AP} , ACC_{ML} , and ACC_{2D} were significantly larger from the values of the surrogate data, i.e., ≈ 0.5 , which indicates that the ACC signals were temporally correlated. The results of the surrogate testing indicate that the time correlations were due to the temporal ordering of the ACC fluctuations, and were not related to the amplitude distribution.

Temporal correlations in the body fluctuation during standing have been analyzed using COP (Collins & De Luca, 1993, 1994, 1995; Collins et al., 1995; Duarte & Zatsiorsky, 2000, 2001) and body displacement (Priplata et al., 2003, 2006). Duarte and Zatsiorsky (2001) examined COP using DFA and found that the scaling index of COP fluctuation during standing was $\alpha = 0.98 \pm 0.17$ and $\alpha = 1.01 \pm 0.03$ in AP and ML directions, respectively. Since their measurements were different from ACC, it is impossible to compare the values in their research directly to the results in the present study. It is true, however, that temporal correlations are commonly observed in body fluctuations measured using ACC as well as COP.

Table 2

Scaling indices of ACC in standing and walking for all participants. The right column “Mean (shuffled)” shows the result of shuffled surrogate test.

	Maximum–minimum		Mean		Mean (shuffled)	
	Standing	Walking	Standing	Walking	Standing	Walking
ACC_{AP}	0.87–0.52	0.80–0.60	0.67 ± 0.09	0.69 ± 0.05	0.51 ± 0.01	0.52 ± 0.01
ACC_{ML}	0.90–0.62	1.01–0.74	0.74 ± 0.08	0.84 ± 0.07	0.52 ± 0.01	0.52 ± 0.01
ACC_{2D}	0.92–0.52	0.89–0.69	0.72 ± 0.10	0.77 ± 0.05	0.52 ± 0.01	0.52 ± 0.01

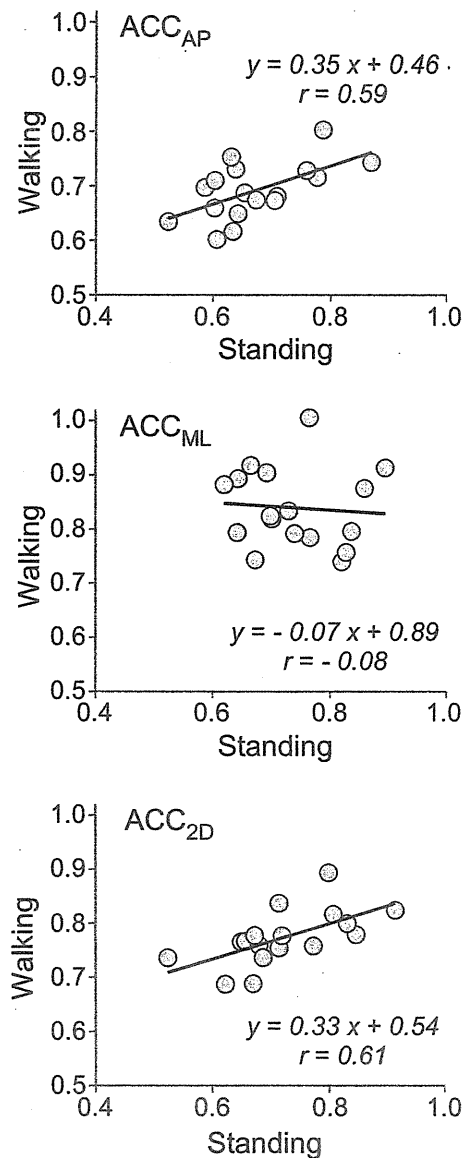


Fig. 3. Relations between scaling indices of ACC in standing (horizontal axis) and walking (vertical axis). The top, middle, and bottom figures show the results for ACC_{AP}, ACC_{ML}, and ACC_{2D}, respectively.

Temporal correlations in body sway during walking have been shown using the stride time interval of gait (Hausdorff, Ashkenazy et al., 2001; Hausdorff et al., 1997, 1995, 1996; Hausdorff, Zeman, Peng, & Goldberger, 1999). Hausdorff et al. (1995) reported the scaling exponent as $\alpha = 0.76 \pm 0.11$ for young participants. This value is close to the value for ACC in this study. In fact, the scaling index for the stride interval in this study also showed a similar value (0.78 ± 0.11). The scaling indices of both ACC and stride interval during walking, however, were not significantly correlated across participants. This suggests that temporal correlations of ACCs during walking were not simply a by-product of fluctuations in stride intervals.

Two possible causes of the temporal correlations are considered: neural effects related to balance control and mechanical effects of the musculo-skeletal system. The temporal correlations can reflect neural processes for balance control in which the equilibrium of the body during standing and walking is maintained by referring to past states of the body. In this context, the degree of temporal correlation reflects the underlying neural mechanism for maintaining postural stability. On the other hand, the temporal correlations may be caused by the filtering properties of the musculo-skeletal system during standing and walking (Gates et al., 2007; Peterka, 2000). Even if motor commands generated by the

neural system have no temporal correlations, these outputs can be low-pass filtered by the musculo-skeletal system, based on its visco-elastic and inertial properties. Such neuro-mechanical influences, and the interaction between them, can affect the degree of correlation in ACC, although this study cannot identify the relative contributions.

Temporal correlations in ACC fluctuations during walking might be influenced by the use of a treadmill. While the treadmill with twin force platforms afforded us precise and stable recordings of ACC in walking, the constant belt speed might decrease variability compared to overground walking (Dingwell, Cusumano, Cavanagh, & Sternad, 2001). Although the scaling indices for stride interval in walking, as described above, showed similar levels with those of previous studies that recorded overground walking, further tests would be needed to determine the relationship between ACCs in treadmill and overground walking.

4.2. Comparison of the degree of temporal correlation between standing and walking

While scaling indices of ACC in standing and walking showed inter-subject variability, they were correlated for ACC_{AP} and ACC_{2D} measures (Fig. 3). This result indicates that an individual, who shows strong temporal correlations when standing, also shows strong temporal correlations when walking, and *vice versa*. This result is interesting because, in general, balance control and body segmental dynamics in standing and walking are considered different (Winter, 1995). The positive relationship between the temporal correlations in standing and walking has two potential explanations. First, the temporal characteristics of balance control in standing and walking might be similar across participants. Second, the intrinsic properties of the participants' musculo-skeletal systems may have similar effects on the temporal correlations of ACC during standing and walking.

Previous studies suggested that there is little relation between balance controls in standing and walking (Kang & Dingwell, 2006; Shimada et al., 2003). The difference between previous investigations and this study might be due to the adopted methods. In the present study, periodic components of walking data were removed by using the averaged data at each stride, and the temporal correlations among strides was compared with that of standing data in the same time scale. Focusing on the body fluctuations among strides in walking allowed a direct comparison between time-dependent characteristics of standing and walking in the same time scales.

Unlike ACC_{AP} and ACC_{2D}, the scaling index for ACC_{ML} in walking was significantly larger than that in standing. In addition, there was no significant correlation between the scaling indices in standing and walking for ML direction. Compared to the AP direction, ACC in the ML direction may be more strongly determined by properties of the musculo-skeletal system. Lateral dynamics in walking is more unstable than in standing, as it takes more time to recover after a lateral perturbation. This could induce large temporal correlations in ACC during walking. The relatively larger difference between standing and walking in this direction might hide the similarity seen in AP and 2D direction. In fact, previous studies demonstrated that the body fluctuations in the ML direction during standing are not sensitive enough to reflect changes induced by aging (Collins & De Luca, 1995; Prieto, Myklebust, Hoffmann, Lovett, & Myklebust, 1996), although the body fluctuations in the ML direction during walking are more sensitive (Dean, Alexander, & Kuo, 2007; Owings & Garabiner, 2004).

5. Conclusions

In summary, we demonstrated that the fluctuations of COM acceleration during standing and walking have a similar degree of temporal correlation ($0.5 < \alpha < 1.0$). There was a positive relationship between the standing and walking ACC temporal correlations in the anteroposterior direction and also in the horizontal plane, but not in the mediolateral direction. This suggests common characteristics in the balance control system in standing and walking, which may be related to the fluctuations in COM acceleration. While neural effects related to balance control and the mechanics of the musculo-skeletal system are presumed to be the causes of the temporal correlation, further studies are needed to identify the relative contribution of the neural and mechanical components in the AP and ML directions.

Acknowledgments

The present study was supported by a Grant from the Japan Society for the Promotion of Science (Grant-in-Aid for Scientific Research (C), 16500370 and 18500430). We thank Mr. John Tan for his assistance with the manuscript preparation. M.O. Abe would like to thank O's meeting members, C. Anderson, and C.J. Hasson for their comments.

References

- Abe, M. O., Nakazawa, K., Masani, K., & Akai, M. (2004). On a common fractal property of body fluctuations in human gait and posture. In *XVth Congress of the International Society of Electrophysiology & Kinesiology* (p. 246). Boston, USA.
- Belli, A., Bui, P., Berger, A., Geyssant, A., & Lacour, J. R. (2001). A treadmill ergometer for three-dimensional ground reaction forces measurement during walking. *Journal of Biomechanics*, *34*, 105–112.
- Collins, J. J., & De Luca, C. J. (1993). Open-loop and closed-loop control of posture: A random-walk analysis of center-of-pressure trajectories. *Experimental Brain Research*, *95*, 308–318.
- Collins, J. J., & De Luca, C. J. (1994). Random walking during quiet standing. *Physical Review Letters*, *73*, 764–767.
- Collins, J. J., & De Luca, C. J. (1995). Upright, correlated random walks: A statistical biomechanics approach to the human postural control system. *Chaos*, *5*, 57–63.
- Collins, J. J., De Luca, C. J., Burrows, A., & Lipsitz, L. A. (1995). Age-related changes in open-loop and closed-loop postural control mechanisms. *Experimental Brain Research*, *104*, 480–492.
- Dean, J. C., Alexander, N. B., & Kuo, A. D. (2007). The effect of lateral stabilization on walking in young and old adults. *IEEE Transactions on Bio-medical Engineering*, *54*, 1919–1926.
- Dingwell, J. B., Cusumano, J. P., Cavanagh, P. R., & Sternad, D. (2001). Local dynamic stability versus kinematic variability of continuous overground and treadmill walking. *Journal of Biomechanical Engineering*, *123*, 27–32.
- Duarte, M., & Zatsiorsky, V. M. (2000). On the fractal properties of natural human standing. *Neuroscience Letters*, *283*, 173–176.
- Duarte, M., & Zatsiorsky, V. M. (2001). Long-range correlations in human standing. *Physics Letters A*, *283*, 124–128.
- Eke, A., Herman, P., Kocsis, L., & Kozak, L. R. (2002). Fractal characterization of complexity in temporal physiological signals. *Physiological Measurement*, *23*, R1–R38.
- Gabell, A., & Nayak, U. S. L. (1984). The effect of age on variability in gait. *Journal of Gerontology*, *39*, 662–666.
- Gates, D. H., Su, J. L., & Dingwell, J. B. (2007). Possible biomechanical origins of the long-range correlations in stride intervals of walking. *Physica A*, *380*, 259–270.
- Hausdorff, J. M., Ashkenazy, Y., Peng, C. K., Ivanov, P. C., Stanley, H. E., & Goldberger, A. L. (2001). When human walking becomes random walking: Fractal analysis and modeling of gait rhythm fluctuations. *Physica A*, *302*, 138–147.
- Hausdorff, J. M., Mitchell, S. L., Firtion, R., Peng, C. K., Cudkovicz, M. E., Wei, J. Y., et al (1997). Altered fractal dynamics of gait: Reduced stride-interval correlations with aging and Huntington's disease. *Journal of Applied Physiology*, *82*, 262–269.
- Hausdorff, J. M., Peng, C. K., Ladin, Z., Wei, J. Y., & Goldberger, A. L. (1995). Is walking a random walk? Evidence for long-range correlations in stride interval of human gait. *Journal of Applied Physiology*, *78*, 349–358.
- Hausdorff, J. M., Purdon, P. L., Peng, C. K., Ladin, Z., Wei, J. Y., & Goldberger, A. L. (1996). Fractal dynamics of human gait: Stability of long-range correlations in stride interval fluctuations. *Journal of Applied Physiology*, *80*, 1448–1457.
- Hausdorff, J. M., Rios, D. A., & Edelberg, H. K. (2001). Gait variability and fall risk in community-living older adults: A 1-year prospective study. *American Journal of Physical Medicine & Rehabilitation*, *82*, 1050–1056.
- Hausdorff, J. M., Zeman, L., Peng, C., & Goldberger, A. L. (1999). Maturation of gait dynamics: Stride-to-stride variability and its temporal organization in children. *Journal of Applied Physiology*, *86*, 1040–1047.
- Kang, H. G., & Dingwell, J. B. (2006). A direct comparison of local dynamic stability during unperturbed standing and walking. *Experimental Brain Research*, *172*, 35–48.
- Laughton, C. A., Slavin, M., Katdare, K., Nolan, L., Bean, J. F., Kerrigan, D. C., et al (2003). Aging, muscle activity, and balance control: Physiologic changes associated with balance impairment. *Gait & Posture*, *18*, 101–108.
- Loram, I. D., Maganaris, C. N., & Lakie, M. (2005a). Active, non-spring-like muscle movements in human postural sway: How might paradoxical changes in muscle length be produced? *Journal of Physiology*, *564*, 281–293.
- Loram, I. D., Maganaris, C. N., & Lakie, M. (2005b). Human postural sway results from frequent, ballistic bias impulses by soleus and gastrocnemius. *Journal of Physiology*, *564*, 295–311.
- Maki, B. E. (1997). Gait changes in older adults: Predictors of falls or indicators of fear? *Journal of the American Geriatrics Society*, *45*, 313–320.
- Maraun, D., Rust, H. W., & Timmer, J. (2004). Tempting long-memory – on the interpretation of DFA results. *Nonlinear Processes in Geophysics*, *11*, 495–503.
- Masani, K., Kouzaki, M., & Fukunaga, T. (2002). Variability of ground reaction forces during treadmill walking. *Journal of Applied Physiology*, *92*, 1885–1890.
- Masani, K., Popovic, M. R., Nakazawa, K., Kouzaki, M., & Nozaki, D. (2003). Importance of body sway velocity information in controlling ankle extensor activities during quiet stance. *Journal of Neurophysiology*, *90*, 3774–3782.
- Masani, K., Vette, A. H., & Popovic, M. R. (2006). Controlling balance during quiet standing: proportional and derivative controller generates preceding motor command to body sway position observed in experiments. *Gait & Posture*, *23*, 164–172.
- Maurer, C., Mergner, T., & Peterka, R. J. (2004). Abnormal resonance behavior of the postural control loop in Parkinson's disease. *Experimental Brain Research*, *157*, 369–376.
- Mori, S. (1987). Integration of posture and locomotion in acute decerebrate cats and in awake, freely moving cats. *Progress in Neurobiology*, *28*, 161–195.
- Mori, S. (1989). Contribution of postural muscle tone to full expression of posture and locomotor movements: multi-faceted analyses of its setting brainstem-spinal cord mechanisms in the cat. *Japanese Journal of Physiology*, *39*, 785–809.

- Mori, S., Nakajima, K., Mori, F., & Matsuyama, K. (2004). Integration of multiple motor segments for the elaboration of locomotion: Role of the fastigial nucleus of the cerebellum. *Progress in Brain Research*, 143, 341–351.
- Morton, S. M., & Bastian, A. J. (2003). Relative contributions of balance and voluntary leg-coordination deficits to cerebellar gait ataxia. *Journal of Neurophysiology*, 89, 1844–1856.
- Morton, S. M., & Bastian, A. J. (2004). Cerebellar control of balance and locomotion. *Neuroscientist*, 10, 247–259.
- Owings, T. M., & Garabiner, M. D. (2004). Variability of step kinematics in young and older adults. *Gait & Posture*, 20, 26–29.
- Pailhouse, J., & Bonnard, M. (1992). Steady-state fluctuations of human walking. *Behavioral Brain Research*, 47, 181–190.
- Peng, C. K., Buldyrev, S. V., Goldberger, A. L., Havlin, S., Simons, M., & Stanley, H. E. (1993). Finite-size effects on long-range correlations: Implications for analyzing DNA sequences. *Physical Review E*, 47, 3730–3733.
- Peng, C. K., Havlin, S., Stanley, H. E., & Goldberger, A. L. (1995). Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. *Chaos*, 5, 82–87.
- Peterka, R. J. (2000). Postural control model interpretation of stabilogram diffusion analysis. *Biological Cybernetics*, 82, 335–343.
- Peterka, R. J. (2002). Sensorimotor integration in human postural control. *Journal of Neurophysiology*, 88, 1097–1118.
- Prieto, T. E., Myklebust, J. B., Hoffmann, R. G., Lovett, E. G., & Myklebust, B. M. (1996). Measures of postural steadiness: Differences between healthy young and elderly adults. *IEEE Transactions on Biomedical Engineering*, 43, 956–966.
- Priplata, A. A., Niemi, J. B., Harry, J. D., Lipsitz, L. A., & Collins, J. J. (2003). Vibrating insoles and balance control in elderly people. *Lancet*, 362, 1123–1124.
- Priplata, A. A., Pattriti, B. L., Niemi, J. B., Hughes, R., Gravelle, D. C., Lipsitz, L. A., et al (2006). Noise-enhanced balance control in patients with diabetes and patients with stroke. *Annals of Neurology*, 59, 4–12.
- Scheinkman, J. A., & LeBaron, B. (1989). Nonlinear dynamics and stock returns. *The Journal of Business*, 62, 311–337.
- Shimada, H., Obuchi, S., Kamide, N., Shiba, Y., Okamoto, M., & Kakurai, S. (2003). Relationship with dynamic balance function during standing and walking. *American Journal of Physical Medicine & Rehabilitation*, 82, 511–516.
- Theiler, J., Eubank, S., Longtin, A., Galdrikian, B., & Farmer, J. D. (1992). Testing for nonlinearity in time series: The method of surrogate data. *Physica D*, 58, 77–94.
- Wagenmakers, E.-J., Farrell, S., & Ratcliff, R. (2004). Estimation and interpretation of $1/f^{\alpha}$ noise in human cognition. *Psychonomic Bulletin & Review*, 11, 579–615.
- Wagenmakers, E.-J., Farrell, S., & Ratcliff, R. (2005). Human cognition and a pile of sand: A discussion on serial correlations and self-organized criticality. *Journal of Experimental Psychology: General*, 134, 108–116.
- West, B. J., & Scafetta, N. (2003). Nonlinear dynamical model of human gait. *Physical Review E*, 67, 1–10051917.
- Winter, D. A. (1995). Human balancing and posture control during standing and walking. *Gait & Posture*, 3, 193–214.
- Yamasaki, M., Sasaki, T., & Torii, M. (1991). Sex difference in the pattern of lower limb movement during treadmill walking. *European Journal of Applied Physiology*, 62, 99–103.

ORIGINAL ARTICLE

Positive effect of balance training with visual feedback on standing balance abilities in people with incomplete spinal cord injury

DG Sayenko¹, MI Alekhina², K Masani¹, AH Vette³, H Obata⁴, MR Popovic^{1,3} and K Nakazawa^{4,5}

¹Rehabilitation Engineering Laboratory, Toronto Rehabilitation Institute, Ontario, Canada; ²Perceptual Motor Behaviour Laboratory, Department of Exercise Sciences, University of Toronto, Ontario, Canada; ³Institute of Biomaterials and Biomedical Engineering, University of Toronto, Ontario, Canada; ⁴Department of Rehabilitation for Movement Functions, Research Institute of National Rehabilitation Center for Persons with Disabilities, Tokorozawa, Japan and ⁵Department of Life Sciences, Graduate School of Arts and Sciences, University of Tokyo, Tokyo, Japan

Objectives: (1) To evaluate the learning potential and performance improvements during standing balance training with visual feedback (VBT) in individuals with incomplete spinal cord injury (SCI) and (2) to determine whether standing static and dynamic stability during training-irrelevant tasks can be improved after the VBT.

Setting: National Rehabilitation Center for Persons with Disabilities, Tokorozawa, Japan.

Methods: Six participants with chronic motor and sensory incomplete SCI who were able to stand for at least 5 min without any form of assistive device performed the VBT, 3 days per week, for a total of 12 sessions. During the training, participants stood on a force platform and were instructed to shift their center of pressure in the indicated directions as represented by a cursor on a monitor. The performance and the rate of learning were monitored throughout the training period. Before and after the program, static and dynamic stability was assessed.

Results: All participants showed substantial improvements in the scores, which varied between 236 ± 94 and $130 \pm 14\%$ of the initial values for different exercises. The balance performance during training-irrelevant tasks was significantly improved: for example, the area inside the stability zone after the training reached $221 \pm 86\%$ of the pre-training values.

Conclusion: Postural control can be enhanced in individuals with incomplete SCI using VBT. All participants showed substantial improvements during standing in both game performance and training-irrelevant tasks after the VBT.

Spinal Cord (2010) 48, 886–893; doi:10.1038/sc.2010.41; published online 20 April 2010

Keywords: spinal cord injury; balance training; biofeedback; motor learning; plasticity

Introduction

The ultimate aim of individuals with spinal cord injury (SCI) is to maximize their independence in all aspects of life, given the limitations imposed by their injury.^{1–3} Recovery of balance ability during standing is, therefore, one of the primary and essential aims of rehabilitative programs in individuals with incomplete SCI. These patients are obliged to develop and re-establish compensatory strategies to maintain balance, including activation of appropriate trunk, neck, and upper limbs muscles in response to internal and external postural disturbances. Conventional therapy in this population focuses on muscle strengthening and improving task-specific balance reactions.⁴ In addition, the importance

of learning to use visual cues and sensory inputs from neurologically intact parts of the body has been emphasized to help maintain safe balance.^{4,5}

Recent advances in technology have resulted in the availability of visual feedback for the retraining of balance function in individuals with neurological disorders, including stroke,^{6,7} cerebellar ataxia,⁷ cerebral palsy,⁸ and Parkinson's disease.⁷ Although further studies are needed to investigate a potential association between positive results obtained from laboratory force plate measures and clinical and functional outcomes,^{6,9} it has been shown that the main positive effect of such training on postural control can be attributed to sensorimotor integration^{5,10–13} as well as the coordination improvement because of the task specificity of training.^{14,15} In the SCI population, benefits of game-based exercises¹⁶ and virtual reality³ have been suggested for dynamic *sitting* balance. These studies have shown their potential for substantial improvements in sitting balance

Correspondence: Dr DG Sayenko, Rehabilitation Engineering Laboratory, Toronto Rehabilitation Institute, Lyndhurst Centre, 520 Sutherland Drive, Toronto, Ontario, Canada M4G 3V9.

E-mail: dimitry.sayenko@utoronto.ca

Received 4 October 2009; revised and accepted 16 March 2010; published online 20 April 2010

through the inclusion of functional approaches in the training.^{3,16} However, the effect of balance training with visual feedback during *standing* in the SCI population has not been reported well. It has been suggested that the standing posture has a number of therapeutic and functional benefits¹⁷ aimed at overcoming physiological problems, such as bladder infections,¹⁷ spasticity,¹⁸ blood pressure homeostasis,¹⁹ and bone demineralization.²⁰ We believe that regaining functionality during self-governed standing will decrease secondary complications and increase independence, and consequently, improve the quality of life of individuals with SCI.

We hypothesized that balance training with visual feedback during standing can improve postural control in individuals with incomplete SCI. The purposes of our study were the following: (1) to evaluate the learning potential and performance improvements during the balance training and determine whether voluntary postural control during different tasks can be improved in individuals with incomplete SCI; (2) to determine whether static and dynamic stability during training-irrelevant tasks can be improved after the balance training; and (3) to suggest mechanisms that may be responsible for a potential improvement in postural control in individuals with incomplete SCI.

Materials and methods

Participants

Six ambulatory participants with motor and sensory incomplete SCI participated in this study (Table 1). Information about each participant's characteristics was based on a self-reported American spinal injury association impairment scale classification, the neurological level of the injury, observed assistive device requirements, and the mobility status at the time when baseline measurements were recorded. The inclusion criteria were the following: (1) at least 12 months post-injury to ensure stability of the participants' neurological condition; (2) ability to stand for at least 5 min without any form of assistive device; and (3) ability to walk 10 m or more with or without the help of parallel sidebars. During the study, the participants did not participate in other rehabilitation or research interventions that might have influenced the outcomes of this study. Each participant gave written informed consent to the experimental procedure, which was approved by the local ethics committee in accordance with the declaration of Helsinki on the use of human subjects in experiments.

Experimental setup and procedure

The training and the data collection were performed with the force plate analysis system 'Stabilan-01' (Rhythm, Taganrog, Russia). The Biodex Unweighing System (Biodex, Shirley, New York, USA) was used in combination with a harness to prevent falls during standing. During the training, participants stood on the force plate and were instructed to look at the monitor, placed at eye level, approximately 1.5 m in front of the force plate. The center of pressure (COP) position signal was used as an input to game-based exercises.

The training was performed 3 days per week for a total of 12 sessions. If a participant was not able to attend a

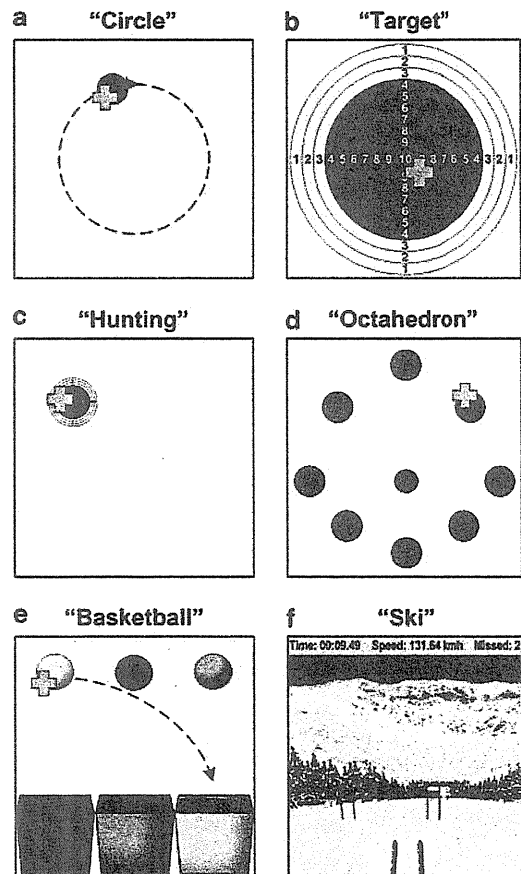


Figure 1 Interface examples of the game-based exercises: (a) 'circle,' (b) 'target,' (c) 'hunting,' (d) 'octahedron,' (e) 'basketball,' and (f) 'ski.' Arrows depict directions for the COP indicator translation (were not shown during the exercises).

Table 1 Characteristics of SCI participants

Participant	Age (years)	Sex	Height (cm)	Weight (kg)	Duration of injury (years)	Level	AIS	Assistive device
1	62	Male	173	65	3	C4	D	Walker
2	50	Male	175	68	4	C4	D	Walker
3	30	Male	180	77	11	T10	C	Wheelchair ^a
4	42	Female	164	62	23	T10	C	Wheelchair ^a
5	27	Male	179	78	6	T11	C	Wheelchair ^a
6	35	Male	178	75	8	T12	C	Wheelchair ^a

Abbreviations: AIS, American spinal injury association (ASIA) impairment scale; SCI, spinal cord injury.

^aParticipants used ankle foot orthoses and canes for walking.