

Somatosensory Influence on Postural Response to Galvanic Vestibular Stimulation

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Summary

We investigated how postural responses to galvanic vestibular stimulation were affected by standing on a translating support surface and by somatosensory loss due to diabetic neuropathy. We tested the hypothesis that an unstable surface and somatosensory loss can result in an increase of vestibulospinal sensitivity. Bipolar galvanic vestibular stimulation was applied to subjects who were standing on a force platform, either on a hard, stationary surface or during a backward platform translation (9 cm, 4.2 cm/s). The intensity of the galvanic stimulus was varied from 0.25 to 1mA. The amplitude of the peak body CoP displacement in response to the galvanic stimulus was plotted as a function of stimulus intensity for each individual. A larger increase in CoP displacement to a given increase in galvanic current was interpreted as an increase of vestibulospinal sensitivity. Subjects with somatosensory loss in the feet due to diabetes showed higher vestibulospinal sensitivity than healthy subjects when tested on a stationary support surface. Control subjects and patients with somatosensory loss standing on translating surface also showed increased galvanic response gains compared to stance on a stationary surface. The severity of the somatosensory loss in the feet correlated with the increased postural sensitivity to galvanic vestibular stimulation. These results showed that postural responses to galvanic vestibular stimulus were modified by somatosensory information from the surface. Somatosensory loss due to diabetic neuropathy and alteration of somatosensory input during stance on translating support surface resulted in increased vestibulospinal sensitivity.

Key words

Somatosensory • Vestibular • Posture • Sensory integration

Introduction

The influence of vestibular-somatosensory interaction in human postural control results in different postural responses to galvanic vestibular stimulation depending on the state of the somatosensory system. For example, healthy subjects standing on a translating

surface showed increased responses to galvanic vestibular stimulation (Inglis *et al.* 1995). Postural responses to galvanic current are also larger when subjects simultaneously make voluntary movements (Severac-Cauquiel and Day 1998). Thus, responsiveness to vestibular signals appears to go up whenever somatosensory information from surface contract

regarding body orientation in space is modified or uncertain. This hyperactive responsiveness to vestibular stimulation disappears when somatosensory information is sufficient for postural orientation or when postural stability is assured by an external support (Briton *et al.* 1993).

A model of vestibular-somatosensory interaction proposes that healthy subjects primarily use proprioceptive cues for motion perception and postural control when they are supported by a stable surface (Mergner and Rosemeier 1998). In this model, vestibular information is used to determine the state of the support surface, and, if the support surface is unstable, vestibular information is used to control balance.

An observation of increased sensitivity to galvanic stimulation in subjects with neuropathy cannot reveal the underlying source of the increase. If healthy subjects show similar increases in galvanic sensitivity when standing on translating surfaces, it is likely that the change in sensitivity observed in subjects with chronic somatosensory loss is related to a similar adaptive change in 'weighting' of sensory information for postural control (Nashner *et al.* 1982, Horak and MacPherson 1996, Maurer *et al.* 2006). Thus, an increase in responses to galvanic stimulation could reflect either an increase in vestibular response bias such that sway increases similarly at all stimulus intensity or an increase in response sensitivity or "gain".

Our previous studies showed that a step of galvanic vestibular stimuli, combined with surface translations, changed mainly the final equilibrium position of the postural response to platform translation (Inglis *et al.* 1995, Hlavačka *et al.* 1999). The effect of galvanic current was largest when vestibular stimulation was initiated 500 ms before, or at the same time, as the platform translation (Hlavačka *et al.* 1999). Sensory interaction between the vestibular error signal induced by the galvanic stimulation and the platform translation was observed such that subjects leaned significantly farther than the sum of the responses to galvanic and support perturbations presented independently. Other studies agree that galvanic stimulation during an ongoing movement has an even a larger effect than during quiet stance (Severac-Cauquiel and Day 1998).

Our recent study provided evidence for an increase in the sensitivity of the postural responses to vestibular galvanic stimulation when somatosensory information from the surface is altered, either by neuropathy or by standing on a compliant surface (Horak

and Hlavačka 2001). These findings are consistent with previous studies showing increases in ankle EMG and CoP sway responses to galvanic stimulation when healthy subjects stood on moving surfaces with different velocities (Inglis *et al.* 1995) and decreases in responses when subjects are supported or sitting (Briton *et al.* 1993, Fitzpatrick *et al.* 1994). The other study also suggested that the vestibulospinal responsiveness increased in subjects with pathological neuropathy to allow functional sensory substitution and that this increase in sensitivity can occur immediately when somatosensory information from the postural support surface is disrupted (Horak and Hlavačka 2001).

In order to better understand vestibular and somatosensory interaction in human stance control, postural responses to four different intensities of bipolar galvanic stimulation in subjects with diabetic peripheral neuropathy and age-matched control subjects were analyzed when standing on translating and stationary surfaces. We hypothesized that larger body leans will be induced by galvanic stimulation when somatosensory information from support surface is altered by the onset of support translation. Furthermore, we predict a minimal sensory interaction after the offset of platform translation.

Methods

Eight subjects (6 males and 2 females) with diabetic peripheral neuropathy (mean age 58±11 years, range 38-70 years) and eight age-matched healthy volunteers (6 males and 2 females; mean age 59±12 years, range 38-72 years), gave informed consent to participate in these studies. Mean duration of diagnosis with diabetes mellitus was 16±9 years. Before testing, each subject underwent a physical examination that excluded subjects with signs of central nervous system or vestibular dysfunction.

The severity of peripheral neuropathy in the subjects with diabetes was established by a clinical sensory assessment index for proprioception and vibration, superficial plantar pressure sensation (Semmes-Weinstein monofilaments), and sensory and motor nerve conduction velocity tests. Subjects with 'moderate' somatosensory loss had measurable but prolonged sural nerve conduction and impaired vibration/proprioception and plantar Semmes-Weinstein clinical test results. Subjects with severe somatosensory loss had absent sensory nerve conduction and very abnormal vibration/proprioception and plantar Semmes-Weinstein

clinical test results.

Subjects stood on two computer-controlled force platforms with their eyes closed and their head turned toward the right shoulder so that galvanic stimulation would produce mainly anterior sway (Lund and Broberg 1983). Their arms were folded and feet placed 20 cm apart.

For galvanic trials, a constant current isolation unit was used to pass ramp-and-hold current impulses (50-ms ramp and 6-s hold) to 9-cm² pieces of carbon rubber placed over the subjects' mastoid processes. In this bipolar, binaural stimulation, four current intensities for each subject were used (0.25, 0.5, 0.75 and 1 mA). With the head turned toward the right shoulder, the anode was on left ear and with the head turned toward the left shoulder the anode was on right ear so anterior sway only was obtained from galvanic stimulation.

The experiment consisted of 6-second trials in 10 different conditions. The two control conditions without galvanic stimulation consisted of a quiet stance condition and a 9-cm, 4.2 cm/s backward platform translation. In the eight experimental conditions, anterior galvanic stimulation with four current intensities was paired either with a fixed platform or with a backward platform translation. In all trials with galvanic stimulation, current was applied after a 100-ms baseline period and lasted for the duration of the 6-second trial. The platform translation started 500 ms after the onset of galvanic stimulation to obtain the maximal effect of galvanic stimulation on postural responses to platform translation (Hlavačka *et al.* 1999, Inglis *et al.* 1995). The 10 different conditions were randomized to control for prediction, habituation and fatigue and repeated 3 times each.

The center of pressure (CoP) under each foot was sampled at 250 Hz and low-pass filtered at 20 Hz. The whole body CoP in the sagittal plane was calculated from the sum of the CoP from the two force plates under each foot, normalized by the proportion of weight over each plate (Henry *et al.* 1998). All subjects realigned CoP consistently to their initial positions after each trial, and these initial CoP positions were assigned to a value of 0. The CoP was averaged for three like-trials for each subject, and figures show group averages across all subjects.

The average amplitude of CoP was computed for each trial 1-2 sec after platform translation onset. Figure 1 shows an example of the CoP responses to galvanic stimulation of 5 different intensities in a subject

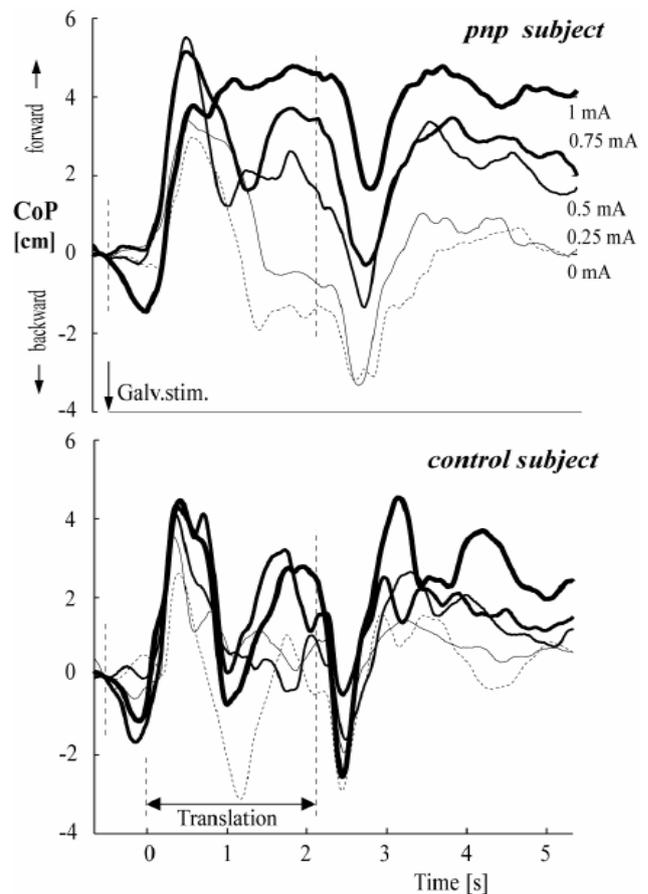


Fig. 1. Postural responses - CoP position (average from 3 trials) as a function of time for 5 stimulus intensities including no stimulation from a representative subject with neuropathy (upper panel) and a representative control subject (lower panel). Dotted vertical lines show the onset of the 6 sec galvanic stimulation and the platform translation period.

with PNP and in a control subject. The sensitivity (gain) of vestibulospinal response was estimated for each subject and condition using the slope of the linear regressions between the CoP final position responses as a function of stimulus intensity. The values of the first points for regression were assigned to a value of 0. A two-way, repeated measures ANOVA was used to determine the effects of galvanic stimulation and surface conditions on CoP amplitudes, otherwise, paired t-tests were used to determine the effects of galvanic stimulation within a group.

Results

The CoP responses to 4 intensities of galvanic stimulation and no stimulation paired with platform translation for one neuropathy subject and one control subject are presented in Figure 1. Initial dorsiflexor

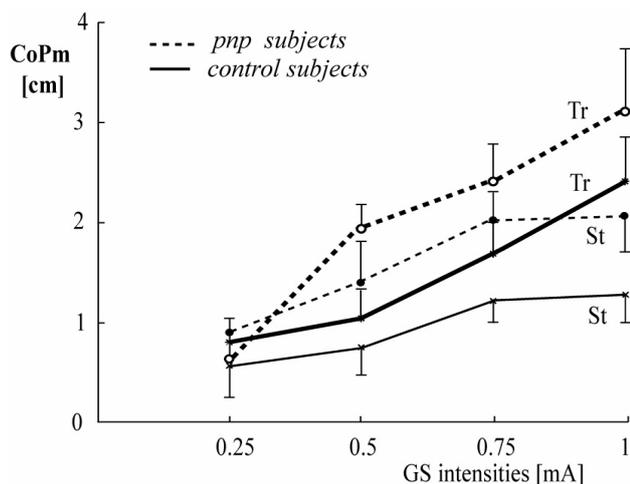


Fig. 2. Comparison of control and neuropathy group means and SEM of CoP forward lean at period 1-2 sec after platform translation onset during 4 different intensities of galvanic stimulation. Subjects stood on the stationary support surface (St) or on the translating support surface (Tr).

activation in response to the stimulation initially resulted in backward CoP shift, followed by sustained forward body leans resulting in forward CoP positions in response to 0, 0.25, 0.5, 0.75 and 1mA of galvanic stimulation.

Subjects with peripheral neuropathy showed larger forward CoP lean than healthy control subjects in response to both galvanic stimulation and to surface translation. The largest differences between subjects with neuropathy and control subjects were at the highest galvanic intensities.

Figure 2 compares the control and neuropathy subjects' mean \pm SEM of CoP responses to the 4 intensities of galvanic stimulation averaged across all subjects. On average over the 1-2s periods after platform translation onset, the neuropathy subjects leaned further forward than normal subjects. This increase of the mean CoP shift forward in neuropathy subjects was similar for stance on the stationary and translating support surface. ANOVA repeat measure (2 groups \times 2 surfaces \times 4 intensities) comparison of these CoP means showed a significant effect of surfaces ($F=6.17$; $p=0.026$), a significant effect of galvanic intensities ($F=23.29$; $p<0.001$) and no significant effect of group. Notice that the difference in responses between the stationary and translating surface were similar for both groups with the largest differences at the highest stimulus intensities.

Figure 3 compares the slopes of the CoP/galvanic intensity relations for each neuropathy subject (5 with severe and 3 with mild neuropathy) with the mean of CoP for the age-matched control subjects.

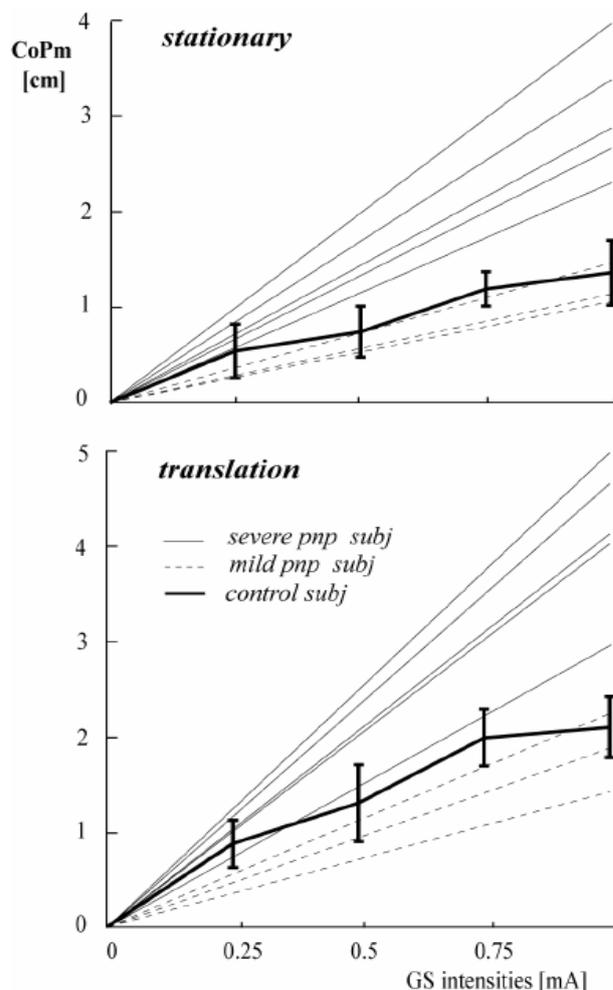


Fig. 3. Slopes of linear regressions of CoP response for individual neuropathy subjects (lines) compared with group mean CoP forward lean for the control subjects (thin solid line), Vestibulospinal sensitivity was quantified by the slope of the linear regression of mean CoP position as a function of stimulus intensity. The neuropathy slopes are all aligned to zero (0).

The subjects with severe neuropathy (thin solid lines on Fig. 3) had slopes of CoP/galvanic responses, which were significantly greater than the mean of control subjects. In contrast, the 3 neuropathy subjects with mild neuropathy showed similar responses to galvanic stimulation as the control subjects. This increase of the slopes in neuropathy subjects was similar for stance on the stationary and translating support surfaces.

The mean slopes of the relations between CoP response and galvanic stimulus intensity during stance on hard support surface were significantly (t -test, $p=0.039$) larger in subjects with peripheral neuropathy (2.28 ± 0.37 SEM) than in the age-matched control subjects (1.34 ± 0.25 SEM, Fig. 4).

The slope of CoP responses to galvanic current intensity during surface translations increased similarly in

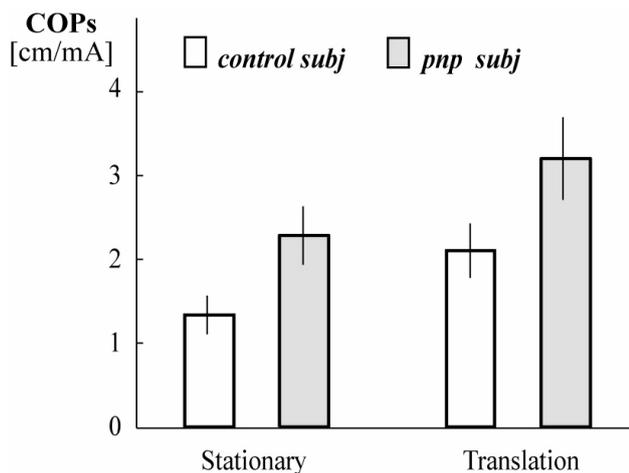


Fig. 4. The group mean and SE of slopes of CoP response to galvanic stimulation intensity on the hard, stationary and translating surface for the control group and the neuropathy subjects.

subjects with neuropathy and in healthy control subjects. Repeated measures ANOVA of mean slopes (2 groups x 2 surface) showed a significant difference between controls and neuropathy subjects ($F=6.07$; $p=0.027$) and between surfaces ($F=7.9$, $p=0.014$). The increase in vestibulospinal sensitivity (slope) due to translation was similar for both groups; the slope of CoP versus galvanic intensity increased 0.64 [cm/mA] for the control subjects and 0.9 [cm/mA] for the subjects with neuropathy.

The group average CoP responses to 4 intensities of galvanic stimulation and no stimulation paired with the offset of platform translation for neuropathy subjects and control subjects are presented in Figure 5.

The stop of backward platform translation initially resulted in a backward CoP shift, followed by sustained forward body lean, resulting in a forward CoP position. The subjects with peripheral neuropathy showed a similar pattern CoP response to the offset of surface translation. Sensory interaction between altered somatosensory input from support surface deceleration and continuous galvanic stimulation was absent.

Discussion

The present study provides evidence for an increase in the sensitivity of the vestibular-evoked postural responses from galvanic stimulation when somatosensory information from the surface is altered, either by chronic neuropathy or by standing on moving surfaces. These results are consistent with previous studies showing increases in CoP sway responses to

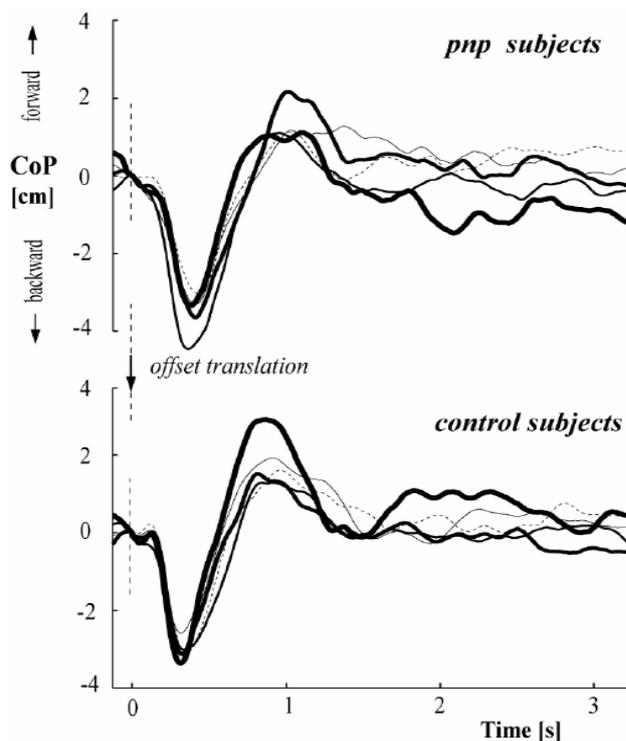


Fig. 5. The group average of CoP responses to 4 intensities of galvanic stimulation and no stimulation paired with the offset of platform translation for neuropathy subjects and control subjects. To extract initial CoP shift induced by continual galvanic stimulation, the CoP positions of postural responses at time of platform translation offset were assigned to a value of 0.

galvanic stimulation when healthy subjects stood on soft surfaces (Horak and Hlavačka 2001) or moving surfaces (Inglis *et al.* 1995) or during unstable support surface (Fitzpatrick *et al.* 1994). The results showed that this increase in responsiveness to galvanic stimulation represents a change in sensitivity of the vestibulospinal response rather than a generalized increase in postural instability.

Previous studies have shown that somatosensory information, including proprioceptive, joint and cutaneous, is the primary sense responsible for postural orientation and equilibrium in quiet stance, at least when subjects stand on hard, stationary surfaces (Peterka 2002, Mergner and Rosemeier 1998, Horak and MacPherson 1996). Evidence for the dominance of somatosensory information for posture is the significant increase in sway excursion and sway velocity when somatosensory information from the feet is reduced by ischemia, visual scene motion or cooling (Horak *et al.* 1990, Magnusson *et al.* 1990, Diener *et al.* 1984, Adamcová and Hlavačka 2004). Sway in stance on a stationary, hard surface is also larger than normal in subjects with somatosensory loss

due to diabetic peripheral neuropathy (Boucher *et al.* 1995, Dickstein *et al.* 2003, Simoneau *et al.* 1995, Uccioli *et al.* 1995). In addition, previous studies showed that postural sway in subjects with somatosensory loss was significantly larger than normal on a firm surface but not on the sway-referenced surface, suggesting that sway-referencing disrupts somatosensory information for postural control already disrupted by neuropathy (Horak *et al.* 2002).

It is likely that normal subjects are using somatosensory feedback during galvanic vestibular stimulation to limit the size of their tilt since normal subjects with eyes closed do not fall, even to very large galvanic currents (Day *et al.* 1997). Loss of somatosensory feedback due to neuropathy partly eliminates this sensory feedback on body sway resulting in increased amplitude of sway (Day and Cole 2002). We favour the interpretation that galvanic stimulation asymmetrically activates vestibular signals which alters the internal perception of vertical orientation to which the body responds by changing body vertical alignment in space in order to match the actual, with the perceived, internal reference for vertical (Smetanin *et al.* 1988, Inglis *et al.* 1995, Hlavačka *et al.* 1995). Subjects with loss of somatosensory information to signal actual body position lean their body farther than control subjects by aligning with the new vestibular vertical without the stabilising effect from the somatosensory feedback loop (Gurfinkel *et al.* 1995).

Subjects with neuropathy may also be more sensitive than healthy subjects to vestibular error signals because they interpret the surface as “unstable” and thus, re-weight sensory orientation away from surface somatosensory signals (Maurer *et al.* 2006). Another explanation is that the increase in body lean in response

to galvanic current represents only a decreased somatosensory loop gain due to neuropathy. The data suggests that both interpretations are partly true. This kind of interpretation was suggested earlier using a comparison of the postural responses to galvanic stimulation on hard and soft surface in neuropathy subjects with the results of a model simulation of the effects of partial somatosensory loss (Horak and Hlavačka 2001). A simple feedback control model was adapted (Hlavačka *et al.* 1996) that included parallel feedback by vestibular and somatosensory loops to maintain body alignment in stance. Model simulations showed that the experimental CoP data could be reproduced only if central vestibular gain was increased as well as peripheral somatosensory gain was decreased. Reduction of somatosensory gain, alone, resulted in a smaller than observed increase in amplitude of CoP lean compared to control subjects and did not explain change of CoP responses as seen in the experimental data.

The findings showed that subjects with neuropathy, like control subjects, can increase their central vestibular gain in the condition of standing on translating support. This increase in gain is possible because many experimental studies suggest that vestibulospinal system gain is normally very low in quiet stance on a hard, stationary surface (Fitzpatrick *et al.* 1994, Horak and MacPherson 1996). Adaptive vestibulospinal gain change may occur often in daily life as we attempt to orient our posture for an unstable surface conditions as well as a compensatory mechanism for loss of somatosensory information.

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