

Modeling of human balance control with somatosensory deficit in lower leg

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Abstract. *We analyzed postural responses to galvanic vestibular stimulation in subjects with lower leg sensory deficit and age-matched control subjects. The aim was to determine whether subjects with somatosensory deficit show a sensory substitution with compensatory increase in sensitivity to vestibular stimulation.*

The pulse of galvanic current with duration 6s and intensity to 1 mA was applied to standing subjects so that forward body tilt was induced. Body lean was measured by force platform as center of foot pressure (CoP). Body tilt increased proportionately with increasing galvanic vestibular stimulation intensity for all subjects. Subjects with peripheral neuropathy showed larger forward CoP displacement in response to galvanic stimulation than controls. The largest differences between subjects with somatosensory deficit and controls were at the highest galvanic intensities, indicating an increased gain of vestibular loop. Simulations from a proposed model of postural control including vestibular and somatosensory feedback suggests that the increase in body lean in response to galvanic current in subjects with somatosensory deficit could be reproduced only if central vestibular gain was increased when peripheral somatosensory gain was decreased.

Key words: human posture, vestibular stimulation, sensory substitution, posture model.

1. Introduction

Sensory postural control during stance with eyes closed is based only on vestibular and somatosensory inputs. Recent studies suggest that changes in somatosensory information from the support surface can change the magnitude of responses to vestibular stimulation by electrical current [1], [2]. An increase in postural sway in response to vestibular stimulation in subjects with somatosensory deficit does not necessarily mean that the gain or sensitivity of the vestibular response has increased. In addition to an increase in postural instability in subjects with somatosensory loss, may be a compensatory increase in vestibular sensitivity as a sensory substitute. To show a compensatory increase of gain in vestibular loop, we compared changes in extent of postural lean in response to galvanic vestibular stimulation in subjects with lower leg sensory deficit and healthy control subjects. A feedback control model was used to determine whether the sensitivity to vestibular stimulation in subjects with somatosensory deficit reflects a decrease in somatosensory feedback or whether an increase in central vestibular gain is likely.

2. Subjects and Methods

Seven subjects (6 males and 2 females) with diabetic peripheral neuropathy (age range 38 - 70 years) and 7 age-matched healthy volunteers (5 males and 2 females; range 38 - 72 years) participated in test. Mean duration of diagnosis with diabetes mellitus was 16 years.

Subjects stood on force platform with eyes closed and head turned toward the right shoulder so that galvanic stimulation would produce forward sway. For galvanic trials, a constant current impulse with duration 6s was used to pass to 9 cm² pieces of carbon rubber electrodes

placed over the subjects' mastoid processes. In this binaural stimulation, four current intensities for each subject were used (0.25, 0.5, 0.75 and 1 mA).

The experiment consisted of 6-second trials in five conditions: the quiet stance as control condition and the anterior galvanic stimulation with the four current intensities. 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 5 different conditions were randomized and repeated 3 times.

The center of pressure (CoP) was sampled at 250 Hz and was averaged for three like-trials for each subject. The average amplitude of CoP was computed for each trial 1.5-2.5 s after galvanic stimulation onset. The sensitivity (gain) of galvanic vestibular response was estimated for each subject and condition using the slope of the linear regressions between the CoP final position response as a function of stimulus intensity. Paired t-tests were used to determine the effects of galvanic stimulation.

3. Results

Healthy subjects increased anterior postural tilt with increasing intensity of galvanic vestibular stimulation. Figure 1A shows the CoP responses to 4 intensities of galvanic stimulation and no stimulation averaged across the control subjects. In response to the stimulation initial backward CoP (-1 cm) occurred and followed by sustained forward body leans resulting in forward CoP positions of 0.36 ± 0.13 cm, 0.52 ± 0.14 cm, 1.1 ± 0.17 cm and 1.2 ± 0.12 cm responses in response to 0.25, 0.50, 0.75 and 1.0 mA of galvanic stimulation, respectively.

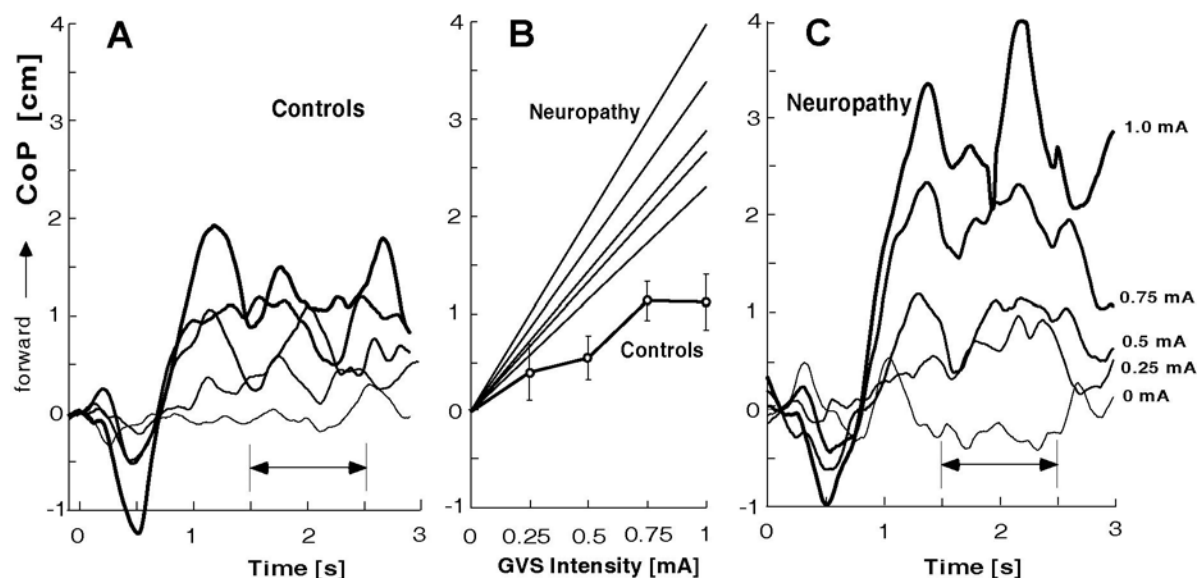


Fig.1. The CoP responses at each intensity of galvanic vestibular stimulation averaged across the control subjects (A) and all the subjects with peripheral neuropathy (B). The slopes of the CoP/galvanic intensity relations for 5 severe neuropathy subjects with the average slope for the control subjects are compared (C).

Subjects with peripheral neuropathy showed larger than normal forward CoP lean in response to galvanic stimulation and the largest differences between subjects with neuropathy and control subjects were at the highest galvanic intensities. Figure 1B shows the CoP responses at each intensity of galvanic stimulation averaged across all the subjects with peripheral neuropathy. Comparison the control and neuropathy subjects' mean \pm SE of CoP responses to the 4 intensities of galvanic stimulation averaged across all subjects over the 1.5-2.5 s period showed that the neuropathy subjects leaned farther forward than normal subjects by 0.25 cm,

0.38 cm, 0.50 cm and 1.3 cm, for the 0.25, 0.5, 0.75 and 1.0 mA galvanic stimulation respectively.

Figure 1C compares the slopes of the CoP/galvanic intensity relations for five neuropathy subjects with the average slope for the age-matched control subjects. The slopes of the relations between CoP response and galvanic stimulus intensity were significantly larger in subjects with peripheral neuropathy (2.3 ± 0.98) than in the age-matched control subjects (1.2 ± 0.21 ; t -value=2.4; $p=0.03$). The subjects with severe neuropathy had a mean slope of 2.9 ± 0.25 which was very different from control subjects (t -value=4.9; $p=.0007$).

4. Discussion

The present study provides evidence for an increase in the sensitivity of the vestibular-evoked postural responses from galvanic vestibular stimulation when somatosensory information from the surface is altered by chronic neuropathy. However, this study demonstrates that this increase in responsiveness to galvanic stimulation represents a change in sensitivity of the vestibular loop rather than a generalized increase in postural instability.

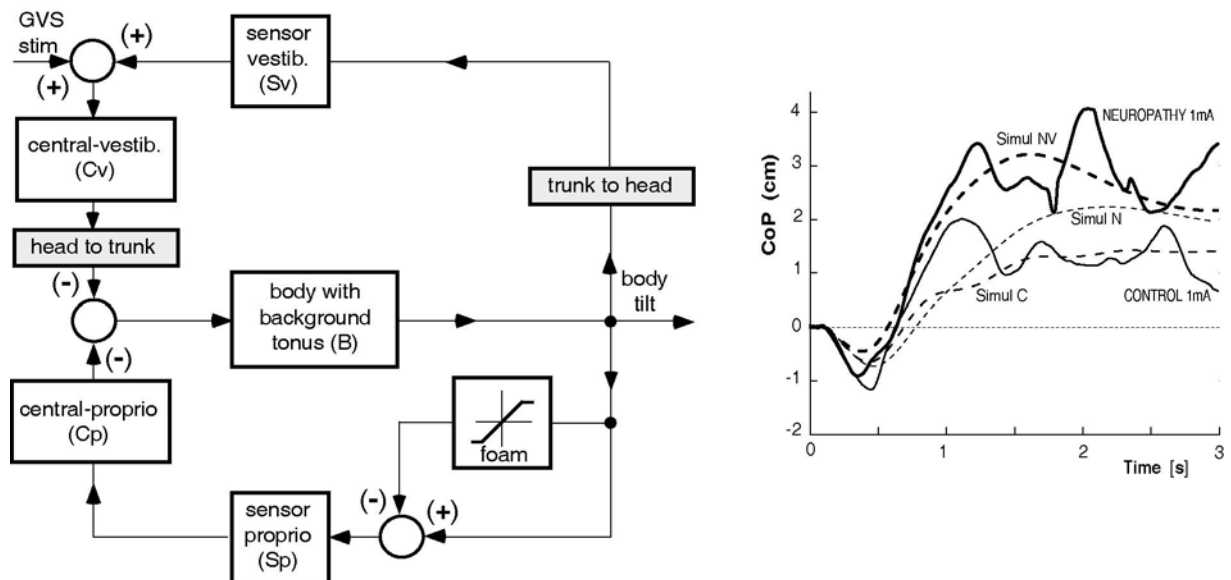


Fig.2. Block diagram of control model of vestibular and somatosensory interaction for body orientation in stance. Vestibular loop including spatial transformation from the trunk to head coordinate and reverse from the head to trunk coordination (marked gray). Stance on foam is simulated by a nonlinear saturation function. Galvanic stimulation sums with vestibular sensor output. Model simulations for control (SimulC) and neuropathy subjects (simulN) are compared with the experimental results CONTROL 1mA and NEUROPATHY 1mA on the right side.

To understand a mechanisms for alteration in vestibular loop sensitivity we compared our results with results of a model simulation of the effects of partial somatosensory loss using a simple linear control model that includes parallel use of vestibular and somatosensory feedback to maintain body alignment in stance. A block diagram of the model is in Fig. 2.

The model was adapted from [3] with the addition of central processing of vestibular and proprioceptive information and the effects of standing on compliant foam to somatosensory feedback. The block of surface foam is represented by a non-linear, saturation function which minimize ankle angle feedback during small amounts (<1 deg) of body sway. This model is based on the following assumptions: 1) Sensory interactions act on a stable, third order musculoskeletal system representing a standing body with normal, background muscle activity; 2) Two parallel feedback loops, somatosensory and vestibular, for supraspinal

control of muscle activity are divided into a peripheral sensory block and a central nervous system block; 3) The integration of somatosensory and vestibular feedback loops consist of simple addition; 4) Linear transfer functions are used for deviations of the body in a small range around the center of equilibrium.

Based on measurements of postural responses to galvanic vestibular stimulation [3] we assumed a transfer function $C_v = (-0.4s+1)/(s+1)$ for the central vestibular block. For the central proprioceptive block, we assumed ideal transfer characteristics $C_p=1$. Transfer functions of body dynamics were estimated as third order system $B(AP) = 2/(0.004s^3 + 0.03s^2 + 0.6s + 1)$ for anteroposterior sway.

The model simulation applied a similar pattern of galvanic stimulation to the vestibular loop as in our study and a somatosensory system gain of 1 for control subjects and a gain of 0.4 for subjects with severe somatosensory deficit. Increased vestibular sensitivity was realized by increasing gain in the transfer function $C_v = (-0.4s+2)/(s+1)$.

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 (see Fig. 2 simul NV). Reduction of somatosensory gain, alone (simul N), resulted in a smaller than observed increase in amplitude of CoP forward lean compared to control subjects and did not change the initial rate of change of CoP as seen in the experimental data. Figure 2 – simul NV shows a good match of the model simulation with the group averaged control and neuropathy CoP data when central vestibulospinal gain was increased from 1.0 to 2.0. It also shows a poor match to group neuropathy data when only the gain of the somatosensory loop was decreased without increasing the central vestibular loop gain (simul N).

Our results suggest that vestibular loop gain increases in subjects with chronic lower leg sensory loss to allow functional sensory substitution following somatosensory deficit in human balance control.

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