Suppression of eye movements improves balance

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Summary
The aim of this study was to investigate the possible interaction of vestibulo-ocular and vestibulo-spinal functions. Spontaneous eye movements and anterior–posterior and lateral body sway were recorded simultaneously in 10 patients with vestibular neuritis (Experiment 1) and in 11 healthy subjects (Experiment 2) while all subjects wore a mask that allowed fixation of a head-fixed target. For the healthy subjects, there was no significant difference in postural sway for the conditions of eyes open in darkness and fixation of the head-fixed target. For the patients, the question was whether transient suppression of the spontaneous nystagmus by fixating the target affected excessive body sway or whether modulation of nystagmus and postural sway were largely independent. The mean peak slow-phase velocity of the spontaneous nystagmus decreased from 13.5 ± 5.6 to 4.3 ± 2.4°/s during fixation. The suppression of nystagmus also reduced postural sway while standing on foam rubber. Mean value decreased from 25.2 ± 7.6 to 16.2 ± 7.7 mm (right–left root mean square values; ANOVA, \( P = 0.003 \)). Since a head-fixed target was used to suppress spontaneous eye movements, the data cannot be explained by any stabilizing effect of afferent visual cues. Instead, ocular motor efference copy signals or reafferences may have contributed to the postural instability of patients with vestibular neuritis, which would explain the reduction of postural sway during fixation suppression of the nystagmus. Thus, ocular motor signals rather than afferent visual cues about retinal slip are used for visual control of postural sway, at least in this experimental paradigm.

Keywords: vestibular neuritis; postural control; spontaneous nystagmus; posturography

Abbreviations: AP = anterior–posterior; LED = light-emitting diode; RL = right–left; RMS = root mean square; SPV = slow-phase velocity; VOG = video-oculography

Introduction
Ocular motor and postural controls are sensorimotor functions that largely operate independently. Both use input from the vestibular system, which converges with visual and somatosensory signals and is subsequently processed in multisensory vestibular structures that are incorporated in vestibulo-ocular and vestibulo-spinal neuronal circuits.

The stabilizing effect of vision on posture is well known to clinicians and has long been the object of scientific investigation (Travis, 1945; Edwards, 1946; Nashner, 1970; Lee and Lishman, 1974). Our current understanding of visual stabilization of postural sway is based on the detection of retinal slip due to involuntary head movements relative to the seen environment. However, several neurophysiological studies suggest that extraocular proprioception contributes to the coding of eye, head and body position in relation to posture and environmental conditions (e.g. Roll et al., 1989) and that eye movements influence postural sway (Brandt et al., 1986; Rushton et al., 1989). There is a conceptual model of the postural system (Wolsley et al., 1996a, b) which incorporates a gain control unit for the visuo-postural loop with inputs from the ocular-cervical proprioceptive system. Studies on the influence of motion parallax in the control of body sway (Bronstein and Buckwell, 1997; Guerraz et al., 2000) discuss the existence of two modes of visual detection of body sway: afferent (retinal slip) and efferent (extraretinal or eye movement-based).

In the light of these studies and the close anatomical interrelationship of ocular motor and postural control, we investigated the interaction between the vestibulo-ocular and vestibulo-spinal functions. Patients with acute unilateral partial labyrinthine failure due to vestibular neuritis were examined for spontaneous nystagmus and postural imbalance. The major question was whether transient suppression of nystagmus by means of a mask that allows fixation of a head-fixed stationary target and does not provide any useful visual cues for postural control had an influence on excessive
body sway or whether modulation of nystagmus and postural sway were largely independent.

Methods
All participants gave their informed consent to participation in the study. The experiments were performed in accordance with the Declaration of Helsinki, adopted at the 52nd World Medical Association General Assembly 2000 in Edinburgh.

Subjects
Experiment 1
Ten patients with acute unilateral vestibular failure due to vestibular neuritis (mean age 58 years, range 31–80 years; two females) were included in the study. Diagnosis of patients was based on a history of acute onset of severe prolonged rotational vertigo and nausea, clinical and neuro-ophthalmological examinations (horizontal-rotatory spontaneous nystagmus towards the unaffected ear without evidence of a central vestibular lesion, pathological bedside testing of high-frequency vestibulo-ocular reflex, postural imbalance with ipsiversive Romberg fall) and electronystagmography with caloric irrigation (hypo- or unresponsiveness of the affected horizontal semicircular canal). Patients were tested 2–6 days after the onset of symptoms. All subjects still exhibited horizontal nystagmus with their eyes open in darkness.

Experiment 2
Eleven healthy subjects without any history of neurological disorders (mean age 26 years, range 25–36 years; two females) were tested with the same equipment as the patients.

Posturography
Anterior–posterior and lateral body sway were measured during upright stance (feet splayed at an angle of 30°, arms hanging) on a stabilometer platform (Type 9261 A; Kistler, Winterthur, Switzerland), which transduces changes in the force exerted on the foot support. Changes of the centre of foot pressure (COP) were measured in the anterior–posterior and right–left directions. Subjects were instructed to remain upright and to refrain from any voluntary movements during the recording. In order to make balancing more difficult, a slab of foam rubber (height 10 cm, specific weight 40 g/dm³) was placed under the rigid foot support. This reduces the reliability of somatosensory signals and therefore enhances the particular weight of afferent visual and vestibular cues. The subjects were prevented from falling while standing by a protective fence at hip height.
Parameters and testing procedure
Sway was recorded in segments of 20 s duration for off-line analysis (sampling frequency 40 Hz; low-pass filter cut-off frequency 20 Hz, 60 dB/decade). After offset elimination, sway velocity and root mean square (RMS) were calculated for the $X$ (right–left) and $Y$ (anterior–posterior) directions (Hufschmidt et al., 1980; Brandt et al., 1981).

Video-oculography
Two-dimensional monocular video-oculography (VOG) was performed using an infrared camera system integrated in a mask that prevented any perception of ambient light or visual orientation (Mack, Pfaffenhofen, Germany). Sampling frequency for horizontal and vertical eye movements was 50 Hz. Range was ±40° for horizontal and ±20° for vertical eye movements. Maximal signal deviation from actual eye position was 3% for horizontal and 4% for vertical movements (noise ±0.2%), as described previously (Pereira et al., 2000). Calibration was made by ±40° horizontal and ±20° vertical saccades; the cross-talk between horizontal and vertical channels was <1%. Mean slow-phase velocity (SPV) of each nystagmus beat was measured. The average of the five fastest SPV periods was used for further analysis. The mask ensured that there was complete darkness even with the eyes open. A mask-integrated, light-emitting diode (LED), located just below the camera lens, could be turned on for fixation. The light source could be dimmed enough that attempted fixation of the still visible target caused only minimal suppression of spontaneous nystagmus.

Procedure

Experiment 1
Patients first performed a routine clinical posturography with eyes open and eyes closed without mask on firm ground and on foam rubber. They were then tested in random order while wearing the VOG mask and standing on: (i) firm ground with eyes open while fixating a head-fixed stationary LED; (ii) firm ground with eyes open in complete darkness; (iii) firm ground with eyes open while attempting to fixate the dimmed LED; (iv) foam rubber while fixating the mask-integrated LED; (v) foam rubber in darkness; and (vi) foam rubber while attempting to fixate the dimmed LED.

Experiment 2
Only two conditions were tested in healthy subjects: (i) standing on foam rubber while fixating the mask-integrated LED; and (ii) standing on foam rubber with eyes open in complete darkness (mask). Figure 1 illustrates the experimental setup.

Statistical analysis
Sway velocity, RMS values and SPV were tested for normal distribution. A repeated measures ANOVA (analysis of variation; Table 1) was performed on the patient data (Experiment 1) with three within-subjects levels (Ground: firm ground versus foam rubber; Vision: space-fixed target versus head-fixed bright LED versus darkness versus head-fixed dim LED; RLAP: right–left versus anterior–posterior). The ANOVA had three within-subjects levels (Ground = firm ground versus foam rubber; Vision = space-fixed target versus head-fixed bright LED versus darkness versus head-fixed dim LED; RLAP = right–left versus anterior–posterior). Significant interactions are also shown (Vision × Ground, Vision × RLAP, RLAP × Vision × Ground).

Results

Experiment 1: patients with vestibular neuritis
Of the 10 patients, eight had vestibular neuritis on the left side and showed horizontal-rotatory nystagmus to the right; the two patients with vestibular neuritis on the right side had nystagmus to the left. Mean peak SPV of horizontal nystagmus in complete darkness for all patients was 13.5 ± 5.6°/s. Fixation of the mask-integrated LED reduced mean peak SPV to 4.3 ± 2.4°/s ($P = 0.016$). Fixation of the dimmed LED reduced SPV to 10.0 ± 3.5°/s (not significant).

When input from the visual (darkness) and somatosensory (foam rubber) system was reduced, all patients showed excessive postural sway. The mean RMS values were 25.2 ± 7.6 mm (right–left, RL) and 26.7 ± 7.3 mm.
(anterior–posterior; AP). Post hoc inspection of the data using the Bonferroni method revealed that sway on foam rubber while fixating a head-stationary LED was significantly less than in darkness ($P = 0.003$) or when attempting to fixate a dim LED ($P = 0.019$). For fixation of the bright LED, mean RMS values were $16.2 \pm 7.7$ mm (RL) and $16.4 \pm 5.5$ mm (AP; $P = 0.003$). The difference between the darkness and the dim LED conditions [mean RMS values $21.4 \pm 8.1$ (RL) and $27.6 \pm 8.7$ (AP)] was not significant. Figure 2 also shows fixation of a space-stationary target to demonstrate postural sway with optimal suppression of nystagmus and visual control. The reduction of postural sway with suppression of vestibular nystagmus was only present on foam rubber. On firm ground, there was no significant difference between the four visual conditions (Fig. 2). Analysis of sway velocity gave similar results to the RMS values described above, the absolute values of which are presented in Table 2.

There was a clear correlation between body sway and mean peak SPV in patients with vestibular neuritis ($r = 0.64$, $P = 0.0001$). Body sway on foam rubber (RMS values) is plotted against mean peak SPV of spontaneous nystagmus for the three conditions of eyes open in darkness, fixation of a head-fixed LED and attempted fixation of a dimmed LED in Fig. 3.

**Experiment 2: young healthy subjects**

Healthy subjects were tested on foam rubber under two conditions: eyes open in darkness and fixation of a head-bound LED. In darkness, RMS values were $6.2 \pm 2.8$ mm (RL) and $10.4 \pm 4.6$ mm (AP). The sway parameters showed no significant difference for the fixation condition. RMS values increased slightly (non-significantly) to $6.6 \pm 2.2$ mm (RL) and $12.6 \pm 7.8$ mm (AP).

**Discussion**

The data show a clear association between the suppression of spontaneous nystagmus and the reduction of postural sway in patients with vestibular neuritis. Since a head-fixed target was used to suppress spontaneous nystagmus, the data cannot simply be explained by the stabilizing effect of any afferent visual cue. In the following we first discuss other clinical and experimental evidence indicating that eye movements and postural sway interact and then try to identify the particular

| Table 2 Absolute values (mean ± standard deviation) of sway velocity (SV) and root mean square (RMS) values of patients with acute unilateral vestibular failure due to vestibular neuritis (Experiment 1) and healthy subjects (Experiment 2) for lateral (RL) and anterior–posterior (AP) sway with eyes open and with or without fixation of a head-fixed LED |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                 | RL sway         | AP sway         |                 |                 |
|                                 | SV (m/min)      | RMS (mm)        | SV (m/min)      | RMS (mm)        |
| Experiment 1: patients with vestibular neuritis ($n = 10$) |                 |                 |                 |                 |
| Standing on firm ground         |                 |                 |                 |                 |
| Fixation of bright LED          | $0.7 \pm 0.3$   | $4.9 \pm 3.3$   | $1.1 \pm 0.6$   | $6.4 \pm 3.4$   |
| Darkness                        | $0.8 \pm 0.4$   | $4.8 \pm 3.1$   | $1.1 \pm 0.6$   | $7.4 \pm 4.2$   |
| Fixation of dim LED             | $1.2 \pm 0.4$   | $6.7 \pm 5.9$   | $1.2 \pm 0.9$   | $8.2 \pm 4.9$   |
| Standing on foam rubber         |                 |                 |                 |                 |
| Fixation of bright LED          | $2.0 \pm 0.9$   | $16.2 \pm 7.7$  | $2.7 \pm 1.2$   | $16.4 \pm 5.5$  |
| Darkness                        | $3.6 \pm 1.3$   | $25.2 \pm 7.6$  | $4.6 \pm 1.2$   | $26.7 \pm 7.3$  |
| Fixation of dim LED             | $3.6 \pm 2.0$   | $21.4 \pm 8.1$  | $4.3 \pm 1.9$   | $27.6 \pm 8.7$  |
| Experiment 2: healthy subjects ($n = 11$) |                 |                 |                 |                 |
| Standing on foam rubber         |                 |                 |                 |                 |
| Fixation of bright LED          | $0.9 \pm 0.3$   | $6.6 \pm 2.2$   | $1.7 \pm 0.6$   | $12.6 \pm 7.8$  |
| Darkness                        | $1.0 \pm 0.3$   | $6.2 \pm 2.8$   | $1.6 \pm 0.4$   | $10.4 \pm 4.6$  |
sensory cue which may be used to reduce body sway during suppression of spontaneous eye movements.

**Association and dissociation of eye movements and postural sway in vestibular syndromes**

Spontaneous nystagmus in vestibular neuritis is caused by a tone imbalance of the vestibulo-ocular reflex. Neuronal vestibular pathways also include ascending input to thalamocortical projections for perception (vertigo) as well as descending input to vestibulospinal projections for adjustments of head and body posture. Stimulation or unilateral failure of the peripheral vestibular organ is known to cause perceptual, ocular motor and postural effects that correlate in severity (Brandt and Daroff, 1980). The patients tested in this study showed all signs and symptoms of the vestibular syndrome.

On the other hand, central vestibular pathway lesions may cause syndromes in which perceptual, ocular motor and postural manifestations differ in severity. Examples are (i) cerebellar lesions with central positional nystagmus without associated positional vertigo or postural imbalance (Büttner et al., 1999); (ii) acute ischaemic lesions in the parieto-insular vestibular cortex with contraversive tils of the perceived vertical without associated ocular motor disturbances (Brandt et al., 1994); (iii) skew deviation with paroxysmal room tilt without body tilt (Tiliket et al., 1996); (iv) unilateral pontomesencephalic lesions of the medial longitudinal fascicle with contraversive skew torsion of the eyes and tils of the perceived visual vertical without accompanying head or body tilt (Brandt and Dieterich, 1993).

Thus, perceptual, ocular motor and spinal aspects of vestibular function can operate in either a linked or a separate mode, depending on the site of the lesion or stimulation. Similarly, cat experiments have shown that stimulation of vestibular afferents that originate from the otolith organs activate second-order neurones in the vestibular nuclei, the majority of which project either to vestibulo-spinal or to vestibulo-ocular pathways (Kushiro et al., 2000). In humans, click-evoked vestibular myogenic potentials, which have been shown to originate from the saccule in guinea-pigs, affect vestibulo-spinal but not ocular motor function (Colebatch, 2001).

Eye movements and postural sway interacted in patients with acquired ocular oscillations, such as down-beating nystagmus. Simultaneous recordings of eye movements and postural sway in these patients showed that lateral gaze or head extension that increased the nystagmus also increased postural sway (BuÈchele et al., 1983). Visual fixation of a mask-integrated LED not only suppressed nicotine-induced nystagmus but also decreased body sway in healthy subjects (Pereira et al., 2001). Voluntary saccades or sinusoidal pursuit eye movements increased anterior–posterior and lateral body sway in normal subjects (Brandt et al., 1986; Hunter and Hoffman, 2001).

**Afferent or efferent cues for visual stabilization of posture?**

The visual stabilization of posture is dependent on several parameters, including the central and peripheral visual fields, illumination, visual acuity, eye–object distance and motion parallax (Paulus et al., 1984, 1989; Bronstein and Buckwell, 1997). The detection of retinal slip due to involuntary head movements relative to the seen environment is believed to be the most important cue for the visual stabilization of posture. Head and eye movements in one direction cause relative motion of the visual scene on the retina in the opposite direction. The data in our experiments cannot be explained by this mechanism, since the stationary target was head-fixed and therefore did not provide any information about head sway relative to the environment. The question arises as to whether the correlation between the amount of nystagmus and the reduction of postural sway is merely an association or a sign of causality between eye movements and postural control.

Three alternative mechanisms should be considered. First, the mere fixation task could induce a change in the postural strategy to achieve optimal balance, e.g. by co-contraction of antigravity muscles. Co-contraction strategies are used in unfamiliar postural tasks and in situations posing a higher risk of falls (Smith, 1981; De Luca and Mambrino, 1987). This mechanism is not supported by the data. Fixation of the head-fixed target in healthy subjects had no effect on postural sway. Similarly, an attempted fixation of the dimmed target by patients, with minor reduction of nystagmus, also caused a minor reduction in postural sway, which was not significant.

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**Fig. 3** Correlation of mean peak slow-phase velocity (SPV) and total postural sway (RMS). There was a correlation of eye and body movements \((r = 0.64, P = 0.0001)\). Symbols represent nystagmus in darkness (black), during fixation of a dim head-fixed LED (grey) and during fixation of a bright head-fixed LED (white).
Secondly, a shift of the particular sensory weight for postural control from the vestibular to the visual system might reduce the relative contribution of vestibulo-spinal signals to multisensory control. In our study, vestibulo-spinal output was destabilizing in patients and stabilizing in healthy subjects. If the relative contribution of vision was increased by fixation of the head-fixed target, we would expect destabilization, since there was a mismatch between the actual head movements and the visual signal of no head movement.

The alternative hypothesis is based on the assumption that information about eye movements rather than afferent visual signals is used to influence body sway. It is known from earlier studies that eye-movement signals are relevant for postural control (Roll et al., 1989; Wolsley et al., 1996a, b; Guerraz et al., 2000). If this were true, the intensity of sway would correlate with the intensity of the nystagmus. This was the case under all conditions tested. It also fits earlier results showing a correlation between eye movements and postural sway (Büchele et al., 1983; Brandt et al., 1986; Hunter and Hoffman, 2001; Pereira et al., 2001). Theoretically, an efference copy of the signal from the tonic ocular motor neural integrator for eye movements in the brainstem could be used. The experiments do not allow us to determine the particular cue or the sensory–motor signal that links eye movements and postural sway. Since spontaneous vestibular nystagmus is an involuntary ocular oscillation, re-afferent input about eye movements may be an alternative signal.

In conclusion, the finding that suppression of spontaneous nystagmus reduces postural sway is compatible with the functional concept that visual stabilization of posture is not only dependent on afferent visual cues but also on ocular motor signals.

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References


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