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RESEARCH ARTICLE

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Phobic postural vertigo

Body sway during visually induced roll vection

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Abstract Patients with phobic postural vertigo (PPV) often report a particularly increased unsteadiness when looking at moving visual scenes. Therefore, the differential effects of large-field visual motion stimulation in roll plane on body sway during upright stance were analyzed in 23 patients with PPV, who had been selected for the integrity of their vestibular and balance systems, and in 17 healthy subjects. Visual motion stimulation induced a sensation of apparent body motion (roll vection) in all patients and normal subjects. Normal subjects showed an increased lateral sway path with a lateral shift of the center of pressure (COP) in stimulus direction (mean 1.67 cm, SD 1.63). The patients also exhibited an increase in sway path during visual motion stimulation; however, their body sway differed from that of normals in that there was no lateral displacement of COP (mean 0.19 cm, SD 0.73). The lateral displacement of COP and the increase in RMS of body sway during visual motion stimulation were significantly greater in normals than in the patients ($p<0.05$). The patients' increased body sway without COP deviation does not imply an increased risk of falling. Two explanations are conceivable for this increased body sway without body deviation in patients with PPV: (a) the patients rely more on proprioceptive and vestibular rather than on visual cues to regulate upright stance; or (b) they depend on visual, vestibular, and proprioceptive information, but the threshold at which they initiate a compensatory body sway opposite in direction to a perceived body deviation is lower than in normal subjects. The data support the second explanation.

Keywords Phobic postural vertigo · Posturography · Roll vection · Body sway · Visual motion stimulation · Human

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Introduction

Phobic postural vertigo (PPV; Brandt and Dieterich 1986; Brandt 1996) is a frequent condition in our dizziness unit, which is distinct from panic disorder with and without agoraphobia (Jacob et al. 1997), acrophobia, space "phobia" (Marks 1981), and "mal de debarquement" syndrome (Murphy 1993). PPV is characterized by a dissociation of subjective postural instability and objectively maintained balance skills. The afflicted patients complain of dizziness and disturbances of balance while standing or walking and of momentary perceptions of illusory body perturbations. Unsteadiness occurs in the form of attacks or as a continuous fluctuating symptom. Symptoms, which may or may not be associated with anxiety, occur spontaneously but are often also elicited by certain perceptual stimuli (e.g., crossing bridges) or social situations (department stores, restaurants, etc.). The subjective disturbance of balance in patients with PPV is marked and leads to rapid conditioning and avoidance behavior. However, falls are not part of this disorder (Brandt 1996). Routine otoneurological and balance tests (electro-oculographic testing of spontaneous and gaze-evoked nystagmus, rotational responses in the dark, pursuit, vestibulo-ocular reflex suppression, and caloric irrigation; neuro-ophthalmologic examination; Romberg's test; tandem walking) of patients with PPV do not reveal any pathology.

In an earlier posturographic study on 12 patients with PPV, we found a significantly increased fore/aft and lateral body sway activity that was most pronounced in the 3.5- to 8-Hz frequency band (Krafczyk et al. 1999). This was interpreted to be a consequence of the patients' anxious control of balance which augments coactivation of antigravity muscles, a strategy applied by normal subjects when performing demanding balancing tasks (Krafczyk et al. 1999; De Luca and Mambrito 1987; Smith 1981). In a subsequent study on 17 patients with PPV, demanding balance tasks were used to test whether the increased body sway activity actually impairs postural balance and increases the risk of falling. In the most diffi-

cult condition tested (tandem stance on foam rubber with the eyes closed), all measured sway parameters did not differ between patients and controls. This indicates that the more difficult the balance task, the better the balance performance was in these patients (Querner et al. 2000).

Both studies on postural sway were performed under static conditions without body perturbations or moving visual stimuli. However, patients with PPV often report a particularly increased unsteadiness when looking at moving visual scenes such as flowing traffic or moving crowds of people. Therefore, in the current posturographic study we evaluated the differential effects of large-field visual motion stimulation on upright stance in patients with PPV who did not have a history or signs of vestibular dysfunction. The aim of this patient selection was to avoid the possibility that a body sway pattern due to a residual or minor organic vestibular dysfunction could interfere with a body sway pattern that reflected a change in strategy. In normal subjects, large, rotating visual scenes induce a continuous sensation of self-motion in the roll plane opposite in direction to pattern motion (roll vection) with a tilt of perceived visual vertical (Dichgans et al. 1972). Roll vection is also accompanied by an increased lateral body sway and a measurable body tilt in the direction of pattern motion.

We addressed the question of whether the patients' complaints of a subjective instability when exposed to moving visual stimuli corresponded to an actual lateral body sway that exceeded that of normal subjects.

Materials and methods

Twenty-three patients (16 men, 7 women; aged 25–67 years, mean age 40 years) with PPV and 17 healthy subjects (12 men, 5 women; aged 26–47 years, mean age 32 years) without any history of disorders of balance, posture, or gait gave their informed consent to participate in the study. The diagnosis of PPV was based on the criteria described elsewhere (Brandt and Dieterich 1986; Brandt 1996). Additional inclusion criteria for the current study were that patients had to: (a) be symptomatic on the day of testing; (b) have had symptoms for more than 3 months; (c) have no history of a former vestibular disorder; (d) show no pathology on neuro-ophthalmologic examination and electro-oculographic testing, as well as no signs of polyneuropathy in the clinical examination; (e) have been medication-free for more than 1 month; and (f) have no history of alcohol abuse.

Lateral body sway was measured during upright stance using a platform that transduces changes in force exerted on the foot support (Tönnies, Freiburg). Sway was recorded in segments of 30 s duration for off-line analysis with a sampling frequency of 40 Hz. Subjects were instructed to remain upright and to refrain from any voluntary movements during the recording. While standing on the platform (feet next to each other, splayed at an angle of 30°), subjects looked into a hemispheric dome (60 cm in diameter), whose inner surface was covered with randomly distributed colored dots. Two seconds after the beginning of a first body-sway registration period, the hemispheric dome started to rotate clockwise around the line of sight at 40°/s. This large-field roll motion stimulation was continued for 1 min. Two seconds before the dome rotation stopped, a second registration period of body sway was started. After the registration, patients and controls were asked whether a sensation of self-rotation (i.e., circular vection) had occurred during visual motion stimulation.

Experiment 1

In both groups, patients with PPV and normal subjects, changes in the center of pressure (COP, in centimeters) were measured in lateral (*x*) direction at the beginning of, during, and after visual motion stimulation. After off-set elimination sway path (SP; i.e., total length of the path described by the COP in 1 min, representing the regulatory activity of the balancing system; Hufschmidt et al. 1980) and root mean square of lateral body sway (RMS; i.e., mean amplitude of rectified COP movements, representing a reciprocal parameter for the effectiveness of balancing; Brandt et al. 1981) were analyzed at the end (18–22 s) and at the beginning (0–4 s) of each registration period. Furthermore, absolute displacement of the COP due to the visual motion stimulus was calculated in the *x*-direction as the difference between means of COP values at the end (18–22 s) and at the beginning (0–4 s) of each registration period.

Experiment 2

Based on the results obtained from the above series, a second series was designed in which nine other normal subjects (5 men, 4 women; aged 22–48 years, mean age 31 years) after one naive registration were asked to try to consciously avoid any body tilt despite visual motion stimulation in roll. This was performed five times over a time span of 3 weeks during stimulation periods of about 1 min while postural performance was registered.

Table 1 Means and standard deviations for sway path (*SP*), root mean square (RMS) of body sway, and position of the center of pressure (*COP*) during normal upright stance in 23 patients with phobic postural vertigo and 17 normal subjects at the beginning (0–4 s of registration) of clockwise visual motion stimulation (*VS*) and during (18–22 s of registration) motion stimulation that induced roll vection. Differences of absolute values during and at the beginning of visual motion stimulation are computed for the patients and the control group and *p*-values resulting from a comparison between both groups by *t*-test are indicated

		Normal subjects		Patients		*p*-value
		Mean	SD	Mean	SD	
SP (m/min)	Start of VS	0.83	0.71	0.58	0.29	
	During VS	1.56	1.41	2.06	2.64	
	Difference	0.73	1.69	1.48	2.62	0.31
RMS (m)	start of VS	0.31	0.34	0.23	0.17	
	During VS	1.89	1.59	0.93	0.85	
	Difference	1.58	0.87	0.69	1.63	0.032
COP (cm)	start of VS	0.08	0.21	0.05	0.17	
	During VS	1.75	1.65	0.24	0.74	
	Difference	1.67	1.63	0.19	0.73	0.0004

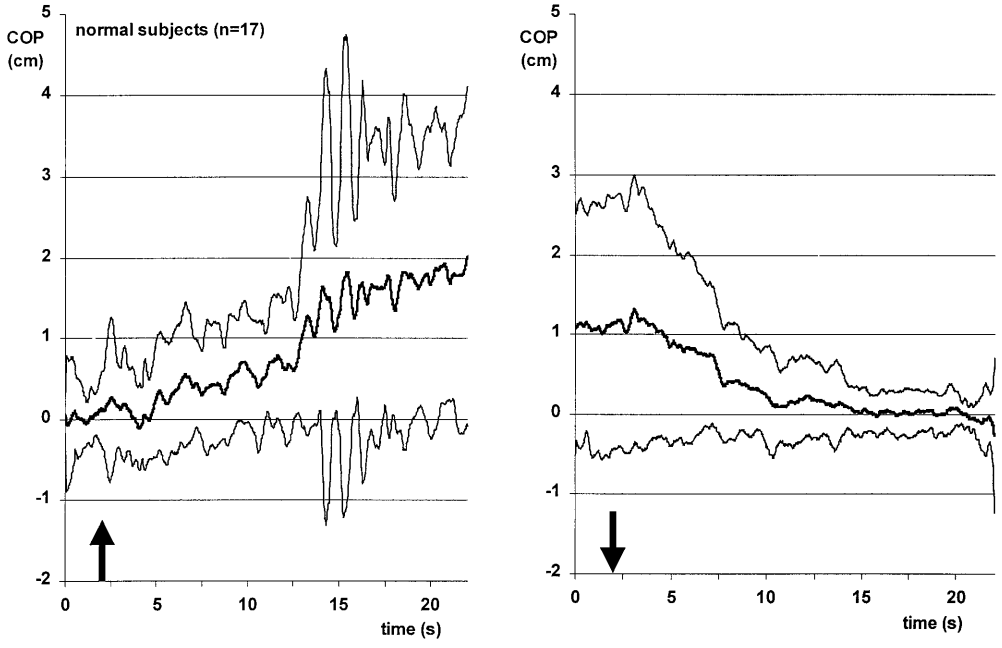

Fig. 1 Lateral shift of center of pressure (*COP*, in centimeters) while in normal upright stance during (*left*) roll visual motion stimulation (roll motion stimulus clockwise, roll vection counterclockwise, deviation of COP to the right) and after (*right*) the end of visual motion stimulation as means (*thick line*) and standard deviations (*thin lines*) for 17 normal subjects. *Arrows* mark beginning and end of stimulation. Note the lateral deviation of COP

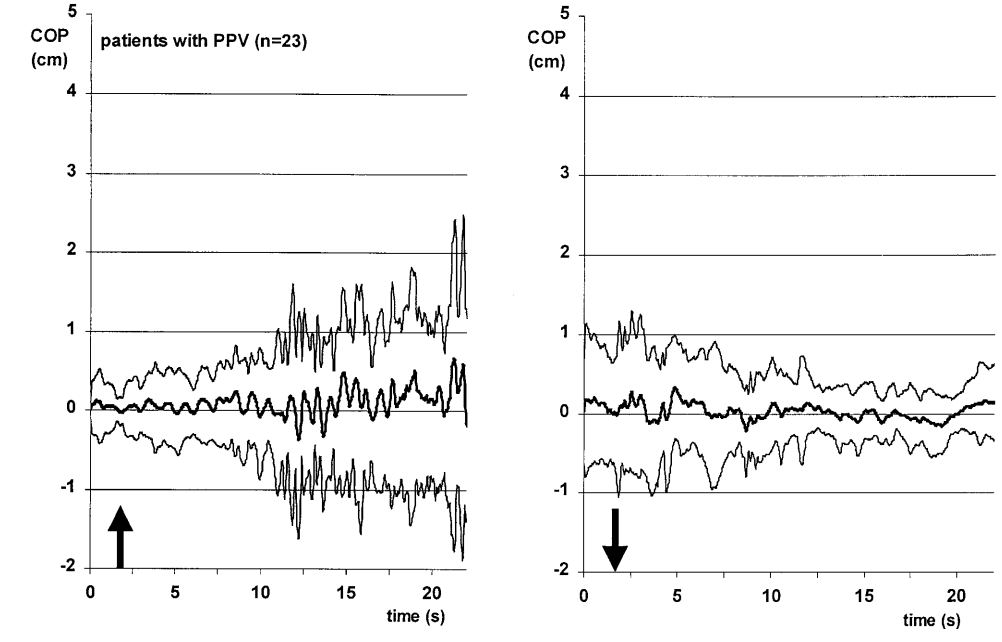

Fig. 2 Lateral shift of the center of pressure (COP in centimeters) while in normal upright stance during (*left*) and after (*right*) visual motion stimulation (roll motion stimulus clockwise, roll vection counterclockwise) as means (*thick line*) and standard deviations (*thin lines*) for 23 patients with phobic postural vertigo (*PPV*). *Arrows* mark beginning and end of stimulation. Note that in contrast to normals (Fig. 1) a lateral deviation of COP is largely absent in the patients

Results

Experiment 1

After 20 s of visual motion stimulation normal subjects exhibited an increased lateral body sway as compared to normal upright stance at the beginning of dome rotation (for absolute values of SP and RMS during and before visual motion stimulation and mean differences, see Table 1) and a mean lateral displacement of COP of 1.67 cm (SD 1.63) in the direction of stimulus roll motion (Table 1, Fig. 1, left; superimposed individual tracings, see Fig. 3, right). This displacement of COP began several seconds after stimulus onset and was associated with the perception of apparent self-rotation (roll vection). After the motion stimulation was terminated, lateral body sway and COP returned to normal pre-stimulus values within about 20 s (Fig. 1, right).

All patients with PPV also experienced roll vection during roll motion stimulation. Their lateral body sway also increased during visual motion stimulation, mainly with respect to SP. For absolute values in SP and RMS of body sway during and before visual motion stimulation, and for mean differences, see Table 1. However, there was no direction-specific lateral displacement of COP (mean 0.19 cm, SD 0.73) in the patients (Table 1, Fig. 2, left; individual tracings superimposed in Fig. 3, left). After the stimulus was terminated, sway parameters of

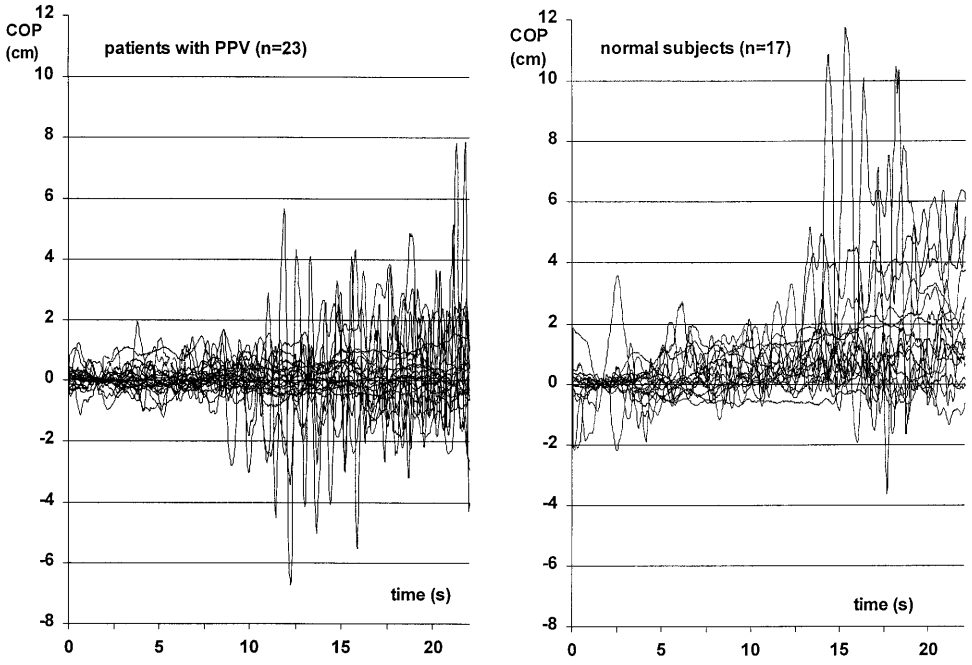

Fig. 3 Individual tracings superimposed for 23 patients with phobic postural vertigo (*PPV, left*) and for 17 normal subjects (*right*) during visual motion stimulation (roll motion stimulus clockwise, roll vection counterclockwise). Positive COP values indicate a deviation of COP to the *right*

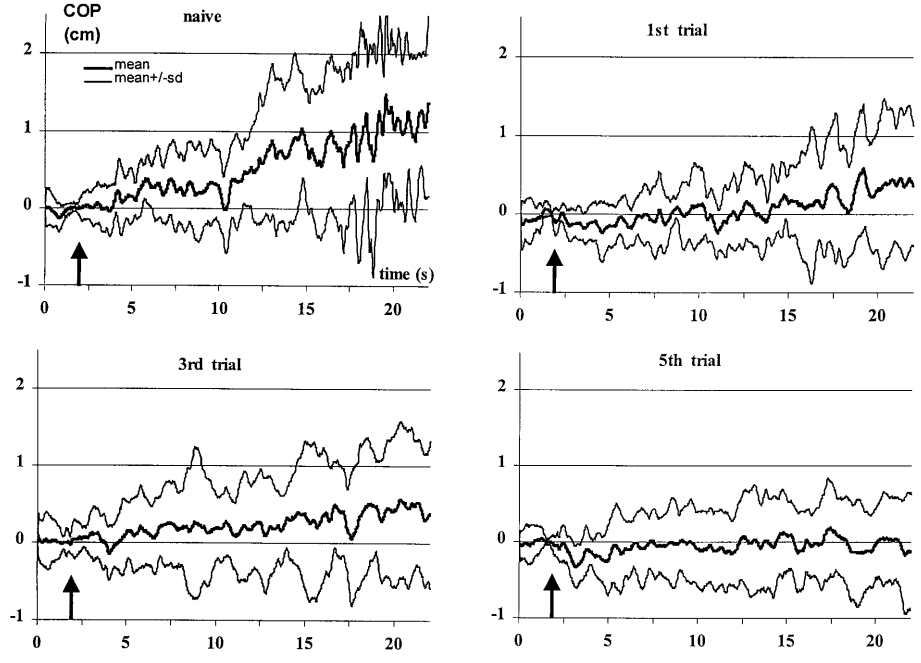

Fig. 4 Lateral shift of the center of pressure (COP in centimeters) in nine normal subjects who, after the first naive registration, were asked to avoid any body deviation during visual motion stimulation (roll motion stimulus clockwise, roll vection counterclockwise, deviation of COP to the *right*) while in normal upright stance in five subsequent trials performed within a time period of 3 weeks (shown for trials 1, 3, and 5) as means (*thick line*) and standard deviations (*thin lines*). *Arrows* mark beginning of stimulation. With an increasing number of trials, subjects were increasingly able to avoid lateral deviation of COP

the patients returned to normal pre-stimulus values within about 20 s (COP see Fig. 2, right).

Thus, lateral displacement of COP due to visual motion stimulation was significantly smaller in the patients than in the normal controls ($p<0.05$; see Table 1). This was also reflected in significantly smaller RMS values in the patients during visual motion stimulation. A stimulus-induced increase in SP was seen in both groups, and it tended to be higher in the patients (not significant).

Since patients with PPV were on the average 8 years older than the normal subjects, lateral SP and lateral RMS of body sway were compared for the subgroup of older patients (aged 38–67 years, $n=11$) and for the subgroup of younger patients (aged 25 to 37, $n=12$). This was done for the balancing condition of normal stance on foam rubber (height 10 cm, specific weight 0.04 g/cm^3, placed under the rigid foot support) with the eyes open. SP and RMS of body sway did not differ significantly between older and younger patients (SP, $p=0.9$; rms of body sway, $p=0.26$): In the older patients, mean SP was 0.62 m/min (SD 0.15) and mean RMS of body sway was 5.42 m (SD 4.34). In the younger patients, mean SP was 0.61 m/min (SD 0.22) and mean RMS of body sway was 3.9 m (SD 1.36). Therefore, it is unlikely that the overall

Table 2 Means and standard deviations for position of the center of pressure (*COP*), sway path (*SP*), and root mean square (RMS) of body sway during normal upright stance in nine normal subjects at the beginning (0–4 s of registration) of clockwise visual motion stimulation (*VS*) and during (18–22 s of registration) motion stimulation. After the first naive registration, subjects were asked to avoid any body deviation during visual motion stimulation while in normal upright stance in five subsequent trials performed within a time period of 3 weeks. Note that displacement of COP subsequently decreased over the number of trials and was completely suppressed by the 5th trial. Negative values indicate a shift of COP opposite to stimulus motion, i.e., to the *left*, positive values, a shift of COP in the direction of stimulus motion, i.e., to the *right*

	Lateral COP (cm)				Lateral SP (m/min)				Lateral rms (m)			
	Start of VS		During VS		Start of VS		During VS		Start of VS		During VS	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Naive	–0.01	0.11	1.08	0.72	0.52	0.11	2.05	1.31	0.19	0.06	1.36	0.61
1st trial	–0.08	0.10	0.34	0.76	0.50	0.16	0.72	0.28	0.18	0.10	0.76	0.41
2nd trial	–0.06	0.14	0.48	0.50	0.53	0.13	1.01	0.62	0.21	0.13	0.65	0.43
3rd trial	0.03	0.11	0.44	0.84	0.56	0.25	0.66	0.23	0.22	0.12	0.77	0.58
4th trial	–0.02	0.11	0.54	0.59	0.45	0.09	0.65	0.33	0.18	0.08	0.68	0.54
5th trial	–0.08	0.12	–0.05	0.56	0.50	0.10	0.48	0.16	0.21	0.12	0.51	0.28

results were substantially affected by differences in age between the patients and the control group.

Experiment 2

When nine normal subjects were repeatedly tested during visual roll motion stimulation while attempting to avoid postural deviation, the mean lateral displacement of COP after 20 s of stimulation decreased from 1.09 to 0.03 cm on the fifth trial (Fig. 4; for absolute values, see Table 2; negative values indicate a shift of COP opposite to stimulus motion, i.e., to the left, positive values a shift of COP in the direction of stimulus motion, i.e., to the right). In contrast to the patients with PPV, suppression of COP deviation in normal subjects was not associated with increased body sway (Table 2). While trying to avoid postural deviation during roll motion stimulation, normal subjects reported that they perceived dome rotation rather than self-motion, i.e., they seemed to suppress the sensation of roll vection.

Discussion

Patients with PPV and normal subjects exhibited an increase in body sway during visual motion stimulation. However, the patients' posturographic data differed from that of normals in that there was no direction-specific lateral shift of COP during roll vection in the patients. Body sway of patients with PPV during visual motion stimulation therefore was characterized by increased lateral to-and-fro movements around a stable COP. This finding confirms a hypothesis presented in a previous study on patients with PPV (Krafczyk et al. 1999): the patients' sole increase in a high-frequency/low-amplitude sway does not cause postural instability or falls, since the center of pressure is kept well within the area of foot support. According to the recent findings, this is true even under difficult balancing conditions with conflicting sensory input such as upright stance during visual motion stimulation. As shown by findings in the elderly, who have a higher risk of falling (Maki et al. 1994; Williams et al. 1997), body instability generally occurs with lower-frequency/large-amplitude sway, which was not found in the patients with PPV.

A discussion of the differential effects of large-field visual motion stimulation in the roll plane in patients with PPV must take into account the fact that the experimental stimulus is not identical to stimulus conditions normally occurring in natural environments.

The increased body sway without body deviation in the patients with PPV during roll vection allows two alternative explanations:

1. Patients with PPV rely more on proprioceptive and vestibular cues than on visual cues to regulate upright stance. At least under stimulus conditions that create a mismatch between multisensory inputs, the misleading visual, moving stimulus may be largely disregarded by the patients in favor of the more reliable, static proprioceptive and vestibular signals. If one assumed that patients with PPV were less dependent on visual cues when controlling balance, a disturbing visual stimulus would have only little effect on the stabilization of upright stance and neither a significant direction-specific body deviation nor an apparent self-motion would be expected.
2. Patients with PPV may be oversensitive to any afferent information that signals impending destabilization, i.e., their threshold to initiate a compensatory body sway opposite in direction to a perceived body deviation is lower than that of normal subjects. Then, minimal perturbations of any sensory quality could cause immediate reactive movements that would typically result in a low-amplitude/high-frequency sway, as found in a previous study on patients with PPV (Krafczyk et al. 1999). If this explanation were true, patients with PPV would depend equally on visual, vestibular, and proprioceptive information. Their sensation of roll vection during experimental visual motion stimulation supports the second explanation.

The first alternative was favored by Jacob and coworkers (1997) as an explanation for postural regulation in a group of patients with a different condition, i.e., panic disorder with agoraphobia. In these patients they found the lowest equilibrium scores (i.e., worst balancing performance) during upright stance under conditions in which proprioceptive feedback was minimized. They postulated that their patients rely less on visual and more on proprioceptive information, a balance control strategy called "surface dependence." Unlike patients with PPV (Querner et al. 2000), patients with panic disorder with agoraphobia exhibited increased body sway under the more difficult balance conditions.

In an earlier study on patients with anxiety disorders associated with space and motion discomfort, Jacob and coworkers (1995) investigated postural responses to oscillating optic flow stimuli and found that they entrained to the stimulus frequency, suggesting "visual dependence." A direct comparison of our data with Jacob's data is difficult due to the different experimental conditions and the distinct patient groups tested. Nevertheless, Jacob's patients, like our patients with PPV, did not disregard the disturbing visual stimulus, but they used visual information for balance control.

Also in 1995, Bronstein reported an increase in SP values during visual motion stimulation in patients with "visual vertigo" syndrome, a heterogeneous group of patients complaining of dizziness or off-balance symptoms triggered by specific visual environments. In his study, patients were examined for changes in SP, but not for a direction-specific body deviation during visual motion stimulation. Their increased body sway was ascribed to organic deficits, since these patients suffered from peripheral vestibular disorders, cerebellar degeneration, or brainstem lesions (Bronstein 1995). The patients with PPV in our current study, however, were selected for the integrity of their vestibular and balance systems. They also showed an increased lateral body sway but – in contrast to normals – no body deviation during visual motion stimulation. Thus, an increased body sway alone does not necessarily indicate a pathologic sensomotoric function.

If patients with PPV relied less on visual cues to regulate upright stance, the increased SP during visual motion stimulation found in our study would remain unexplained. Moreover, one would expect that patients who disregard visual motion stimulation would not experience roll vection. Therefore, we tend to favor the second interpretation of our results, i.e., that patients with PPV have a lower threshold for counteracting subtle body motions, which they perceive as disturbing. Anxious control of balance in patients with PPV could lead to this "unnecessary" increase in SP. A significant increase in body sway activity during normal upright stance was found in an earlier study on patients with PPV (Krafczyk et al. 1999), although postural balance was not impaired. This was interpreted as a change in strategy of postural control associated with increased coactivation of antigravity muscles due to the patients' anxious expectation of body destabilization. Similarly, a stiffening strategy of stance has been described in normal subjects, who when standing on a high surface try to control the COP within a smaller area by generating faster movements of COP through smaller amplitude displacements (Carpenter et al. 1999). The association of an increase in anxiety and arousal with adaptations of postural sway has also been shown by Maki and McIlroy (1996) in normal subjects performing stressful cognitive tasks during quiet unperturbed stance. The assumption of an anxious, conscious control of stance by the patients tested in our study seems to correspond to the obsessive-compulsive personality ascertained by psychiatric evaluation of a majority of patients with PPV (Kapfhammer et al. 1997). A strategy of increased coactivation of antigravity muscles in patients with PPV could reduce body deviation during upright stance even under visual motion stimulation that induces roll vection.

In the second series of experiments, we tested in six subsequent trials performed over a period of 3 weeks whether normal subjects adopt a strategy comparable with that used by patients with PPV when asked to consciously avoid any body tilt while exposed to visual motion stimulation during upright stance. The displacement of COP subsequently decreased over the number of trials performed until the COP shift was completely suppressed by about the 5th trial. The critical difference from the patients' data was that this suppression of COP shift was not associated with an increase in SP during visual motion stimulation. All normal subjects reported perceiving dome rotation rather than self-motion, i.e., they seemed to increasingly suppress the sensation of roll vection, which they described as completely absent in the last two trials. This means that they were obviously able to shift the "sensorial weight" from visual to proprioceptive information.

Similarly, Hufschmid and coworkers (1980) used posturography to examine the influence of visual roll motion stimulation on upright stance in five normal subjects. Contrary to our experiment, their subjects performed repeated trials on a single day without any specific instructions. Hufschmid and coworkers also reported a continuous decrease in displacement of COP and in sway parameters after several subsequent trials. Unfortunately, they do not mention in which trials subjects reported experiencing roll vection during visual motion stimulation. The authors interpret their data as due to habituation and conclude that in normal subjects the "sensory weight" of visual cues in postural regulation is adaptable and decreases with long-lasting conflicting stimuli.

In conclusion, patients with PPV seem to counteract perturbing roll vection due to large-field visual motion stimulation at a lower threshold than normal controls. This results in an increased body sway without displacement of COP and without an increased risk of falling. Normal subjects seem to be able to disregard a visual motion stimulus in the roll plane with repeated exposure, so that the sensation of roll vection, the displacement of COP, and the increase in body sway disappear.

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