

# Visual vertigo: symptom assessment, spatial orientation and postural control

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## Summary

Certain patients with balance disorders report a ‘visual vertigo’ in which their symptoms are provoked or aggravated by specific visual contexts (e.g. supermarkets, driving or movement of objects). In order to determine the causes of visual vertigo (VV), we assessed symptoms, anxiety and the influence of disorienting visual stimuli in 21 such patients. In 17 out of 21 patients, a peripheral vestibular disorder was diagnosed. Sixteen bilateral labyrinthine-defective subjects (LDS) and 25 normal subjects served as controls. Questionnaire assessment showed that the levels of trait anxiety and childhood motion sickness in the three subject groups were not significantly different. Reporting of autonomic symptoms and somatic anxiety was higher than normal in both patient groups but not significantly different between LDS and VV patients. Handicap levels were not different in the two patient groups, but the reporting of vestibular symptoms was higher in the VV than in the LDS group. The experimental stimuli required subjects to set the subjective visual vertical in three visual conditions: total darkness, in front of a tilted luminous frame (rod and frame test) and in front of a large disc rotating in the frontal plane (rod and disc test). Body sway was also

measured in four visual conditions: eyes closed, eyes open, facing the tilted frame and during disc rotation. In psychophysical and postural tests, both LDS and VV patients showed: (i) a significant increase in the tilt of the visual vertical both with the static tilted frame and with the rotating disc; and (ii) an increased postural deviation whilst facing the tilted frame and the rotating disc. The ratio between sway path with eyes closed and eyes open (i.e. the stabilizing effect of vision) was increased in the LDS, but not in VV patients, compared with normal subjects. In contrast, the ratio between sway path during disc rotation and sway path during eyes open (i.e. the destabilizing effect of a moving visual stimulus) was increased in the VV patients but not in LDS. Taken together, these data show that VV patients have abnormally large perceptual and postural responses to disorienting visual environments. VV is not related to trait anxiety or a past history of motion sickness. The results indicate that VV emerges in vestibular patients if they have increased visual dependence and difficulty in resolving conflict between visual and vestibulo-proprioceptive inputs. It is argued that treating these patients with visual motion desensitization, e.g. repeated optokinetic stimulation, should be beneficial.

**Keywords:** vertigo; visual; posture; vestibular; visual dependency

**Abbreviations:** COP = centre of foot pressure; LDS = labyrinthine-defective subjects; VV = visual vertigo

## Introduction

We have investigated why some patients with balance disorders report a prominent increase in their symptoms in certain visual surroundings. These include situations such as driving (motorist disorientation syndrome; Page and Gresty,

1985), being subjected to repetitive visual patterns, walking in supermarket aisles and viewing moving scenes [visual vertigo (VV); Bronstein, 1995].

Vertigo, dizziness and unsteadiness are frequently

encountered symptoms in neurology, ENT and general practice (Perkin *et al.*, 1997; Yardley *et al.*, 1998). If the clinician witnesses an episode of spontaneous or positional vertigo, the chances of making a correct diagnosis are high, e.g. vestibular neuritis, benign paroxysmal positional vertigo. However, in the patient with long-standing dizziness, there may not be hard clinical/imaging findings, strict diagnostic criteria or pathological definition. In addition, in patients with protracted symptoms, psychogenic components are often contributory (Baloh, 1996; Brandt, 1996; Furman and Jacob, 1997). For these reasons, quantitative assessment of vertigo and anxiety symptoms should be implemented in research in balance disorders (Yardley *et al.*, 1992a, b).

A factor which interferes with our ability to understand the patient with long-standing balance symptoms is the variable degree of recovery of the actual vestibular lesion. In a 5-year follow-up study, approximately half of the patients with vestibular neuritis still showed abnormal caloric responses and one-third reported vestibular symptoms (Okinaka *et al.*, 1993). Other factors are the considerable discrepancy between symptom recovery and normalization of vestibular test results (Okinaka *et al.*, 1993; Kanayama *et al.*, 1995) and the multifactorial nature of the process of vestibular compensation. Indeed, symptom resolution in peripheral vestibular patients is influenced by adaptive changes in visual motion perception (Wist *et al.*, 1983; Grunfeld *et al.*, 2000), psychological factors (Brandt, 1996; Yardley and Hallam, 1996; Grunfeld *et al.*, 2000), the degree of recovery in vestibular function (Bronstein *et al.*, 1995; Okinaka *et al.*, 1993; Allum and Ledin, 1999) and the presence of additional CNS lesions (Rudge and Chambers, 1982).

We now investigate another factor which may perpetuate symptoms in patients with balance disorders (Bronstein, 1995), namely the presence of increased visual dependence (Witkin and Asch, 1948; Witkin, 1959). The question was prompted by the observation that patients often report onset or worsening of symptoms in visual surroundings with increased visual motion (Hood, 1980; Page and Gresty, 1985; Jacob *et al.*, 1989; Bronstein, 1995; Baloh, 1996). Whilst visually or driving-induced symptoms may be due to anxiety or panic disorders, there is evidence that a vestibular disorder can be the underlying cause (Page and Gresty, 1985; Jacob *et al.*, 1989, 1996; Bronstein, 1995).

In a previous study, some patients with VV due to underlying peripheral or central vestibular disorders showed abnormally large postural responses to optokinetic stimulation (Bronstein, 1995). It was postulated that the combination of a vestibular disorder and enhanced visual dependence could explain why some patients experience visual triggering of their symptoms. The concept of visual dependence derives from the fact that spatial orientation is based on both gravito-inertial (vestibulo-proprioceptive) and visual cues, and that normal human subjects make variable and idiosyncratic use of such cues for spatial orientation (Witkin and Asch, 1948; Witkin, 1959; Guerraz *et al.*, 1998) and postural control (Isableu *et al.*, 1998). Essentially, in the presence of conflict

between sensory modalities, visual-dependent subjects rely more on visual cues whereas visual-independent subjects rely more on vestibulo-proprioceptive cues. It is likely that such perceptual preferences observed in normal subjects are also present, if not enhanced, in balance disorder patients. In fact, in postural studies of unselected patients with vestibular lesions, some patients are clearly more susceptible than others to conditions of distorted visual feedback, e.g. a sway-coupled visual surround (Shepard *et al.*, 1993; Shumway-Cook *et al.*, 1996).

A major deficiency of previous studies, however, is that, apart from conventional vestibular tests, few additional data were collected (Redfern and Furman, 1994; Bronstein, 1995; Peterka and Benolken, 1995). This limitation is important in patients because of the dissociation observed between vestibular test results, perceptual responses and questionnaire assessment of symptoms (Stephen *et al.*, 1991; Kanayama *et al.*, 1995). For instance, patients may be subjectively disoriented by visual stimuli but this might not necessarily be reflected in a postural task. It was therefore felt that what was required was to measure symptom load, perceptual responses and postural control in the same group of patients with VV. Since the hypothesis is that VV patients may be abnormally dependent on vision, a group of bilateral labyrinthine-defective subjects (LDS), expected to be particularly visually dependent, were also used as controls. The possible influence of psychological factors was also assessed with validated questionnaires. This is necessary because some symptoms in patients with balance disorders overlap with those in patients with psychogenic conditions (Brandt, 1996; Bronstein *et al.*, 1997; Furman and Jacob, 1997), possibly leading to the dismissal of patients with genuine vestibular disorders. Finally, the presence of pre-existing motion sickness susceptibility as a contributory factor for the emergence of VV was also quantified. This was done because of the possibility that protracted vestibular symptoms may be more common in patients with a propensity to motion sickness (Baloh, 1997; Golding, 1998) and because visual dependence and motion sickness trait appear to be correlated (Long *et al.*, 1975; Yardley, 1990).

## Material and methods

### *Experimental subjects*

#### *Visual vertigo (VV) patients*

The VV group included 21 patients who, in response to open-ended questions (i.e. what triggers or makes your dizziness worse?), reported triggering or worsening of vertigo, dizziness, unsteadiness or spatial disorientation by certain visual surroundings. These visual surroundings included movements of crowds or traffic, moving images at the cinema, walking in supermarket aisles, eye movements or driving on open roads/motorways. These patients had been referred for assessment of a possible vestibular disorder, usually by neurologists. Patients with clinical or imaging evidence of

CNS disease were excluded, except one with an incidental, irrelevant finding of *en plaque* parietal meningioma on MRI. Mean age was 41 years, range 25–60. There were 11 females and 10 males. Our aim was to study 20 patients, but one could not complete the postural tests so one further patient was added.

All patients had a neurological and neuro-otological clinical examination, and caloric testing with visual fixation according to Fitzgerald and Hallpike (Fitzgerald and Hallpike, 1942); when the nystagmic response ceased, the eyes were observed with Frenzel's glasses. Spontaneous and gaze-evoked nystagmus, rotational responses in the dark (velocity steps 60°/s; sinusoidal oscillation 0.2–0.4 Hz peak velocity 40°/s), optokinetic, pursuit and vestibulo-ocular reflex suppression functions were examined with horizontal DC electronystagmography. Normal data are reported elsewhere (Stell *et al.*, 1989; Francis *et al.*, 1992; Lopez *et al.*, 1992). Pure tone audiograms were obtained in all patients. Additional neuro-otological, ocular-motor and imaging investigations were carried out according to clinical indication.

Based on the history and/or findings, a current or past peripheral vestibular disorder was diagnosed as the most likely explanation for symptom onset in 17 out of 21 patients. The diagnosis was based on the clinical history of a single, long-duration, rotational vertigo attack suggestive of vestibular neuritis ( $n = 4$ ), repetitive, brief, positional, rotational vertigo suggestive of benign paroxysmal positional vertigo ( $n = 2$ ) or recurrent episodes of vertigo (benign recurrent vertigo,  $n = 5$ , basilar migraine,  $n = 2$ ). Other diagnoses were Ménière's disease ( $n = 2$ ), post-traumatic vestibulopathy ( $n = 1$ ) and subtotal bilateral vestibular failure with mild idiopathic peripheral polyneuropathy ( $n = 1$ ). None of these patients was observed in an acute vertiginous phase. Two patients had congenital squints; in one of these, the visuo-vestibular symptoms developed after corrective squint surgery. Five patients had migraine, but the association with the vestibular symptoms was less clear than in the patients with basilar migraine (Baloh, 1997). Nine patients suffered from the 'motorist disorientation syndrome' (Page and Gresty, 1985) usually in combination with other VV symptoms; in two, there were no additional vestibular symptoms or findings. In nine patients, anxiety, phobia or depression were noted by the neuro-otologist, but no formal interview by a psychiatrist was available. The four patients who had no evidence of vestibular disease were: a patient with motorist syndrome plus migraine, one with motorist syndrome and height vertigo (this was the only patient in whom a psychogenic component was clinically dominant), one with motorist syndrome alone and one with pronounced motion sickness and a past history of migraine.

Vestibular tests were completely normal in 11 patients. Canal paresis and directional preponderances were present in four patients each. One patient had combined canal paresis and directional preponderance, whilst another had subtotal bilateral reduction in caloric/rotational responses. Four patients had unilateral sensory neural hearing loss.

### *Labyrinthine-defective subjects*

Sixteen patients with caloric and rotational responses, either absent or reduced to <10% of the mean normal level (Rinne *et al.*, 1998), were tested. The diagnoses were idiopathic bilateral vestibular failure (Rinne *et al.*, 1998) ( $n = 11$ ), gentamicin ototoxicity ( $n = 3$ ), post-meningitic ( $n = 1$ ) and sarcoidosis ( $n = 1$ ; reported in von Brevern *et al.*, 1997). The mean age was 53 (range 33–84 years); there were five females and 11 males.

### *Normal controls*

Twenty-five subjects with no history of labyrinthine or neurological disease were tested as normal controls. Mean age was 46 (range 23–78 years); there were eight females and 17 males. All subjects consented to the study, approved by the ethical committee of The National Hospital for Neurology and Neurosurgery, London, UK, according to the Helsinki declaration.

### *Questionnaire measures*

All subjects completed the following validated scales.

The Situational Vertigo Questionnaire (adapted from Jacob *et al.*, 1989) yields a normalized score of 0–4 summing the severity of symptoms induced by disorienting environments, especially those with visual-vestibular conflict.

The Vertigo Symptom Scale (Yardley *et al.*, 1992a, b) yields two normalized scores, ranging from 0 to 4, summing number and frequency during the past year of symptoms of (i) vertigo and imbalance (e.g. 'feeling that things are spinning or moving around', 'feeling unsteady, about to lose balance') and (ii) autonomic and somatic anxiety symptoms (e.g. 'heart pounding or fluttering', 'excessive sweating').

The Childhood Motion Sickness Questionnaire (Reason, 1968) yields weighted scores ranging from 0 to 10, based on frequency of vomiting and nausea induced by various types of transport before the age of 12 years.

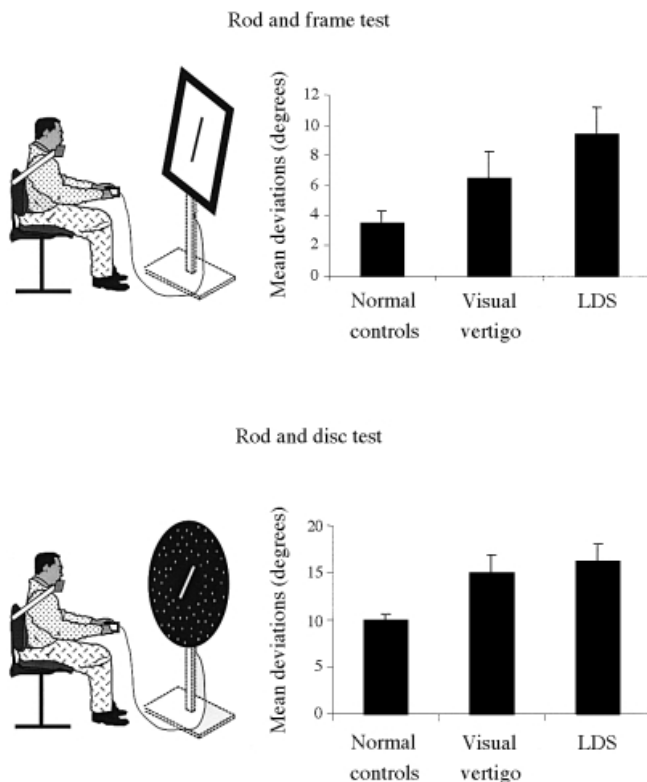
The Spielberg Trait Anxiety Inventory (Spielberger *et al.*, 1970) assesses how often respondents typically feel anxiety (e.g. 'I lack self-confidence', 'I worry too much'). Summed scores range from 20 (minimum anxiety) to 80 (maximum anxiety).

Patients also completed the Vertigo Handicap Questionnaire (Yardley and Putman, 1992), which yields a score of 0–80 indicating the extent of handicap due to vertigo symptoms.

### *Experimental apparatus and techniques*

#### *Rod and frame apparatus (Fig. 1, top left)*

The frame was a square of 90 × 90 cm, with a frame width of 2.4 cm; viewed at 80 cm from the subject's eyes, it subtended an angle of 60°. It could be upright or tilted 28° in a clockwise or counterclockwise direction around the



**Fig. 1** Experimental set-up and mean deviation (and standard error) of the subjective visual vertical, under static frame tilt (upper panels) and disc rotation (lower panels), for normal control, visual vertigo and labyrinthine-defective subjects (LDS). Statistics are shown in Table 2.

subject's line of sight. A rod ( $40 \times 0.5$  cm) was placed coaxially within the frame, subtending an angle of  $30^\circ$ . Both the frame and rod were fluorescent in order to be the only visible objects in the otherwise dark experimental room. The rod and frame could be masked independently. The angular position of the rod could be remotely controlled by the subject and experimenter, but the frame by the experimenter only.

#### *Rod and disc apparatus (Fig. 1, bottom left)*

The disc was black (90 cm diameter), covered with fluorescent irregular dots; when viewed at 80 cm from the subject's eyes, it subtended an angle of  $60^\circ$ . It could be rotated at  $30^\circ/s$  in a clockwise or counterclockwise direction around the subject's line of sight. At its centre there was a black disc, covering the fluorescent central dots, with an adjustable fluorescent rod ( $30^\circ$  visual angle). The rod could be removed for postural recordings.

#### *Subjective visual vertical*

Subjects were seated upright facing the device at 80 cm. The head was held in place by a chin-rest. Both the frame and the disc were adjusted in height to eye level. Settings of the

rod in complete darkness (i.e. no frame, no disc) were done first, and then during the static visual disturbance (frame tilted  $\pm 28^\circ$ ) and the kinetic disturbance (disc rotating at  $\pm 30^\circ/s$ ). The two visual conditions as well as the two directions of disturbance were presented in counterbalanced order. The starting position of the rod was tilted either to the left or right ( $\sim 40^\circ$ ). There were four trials per condition and per subject.

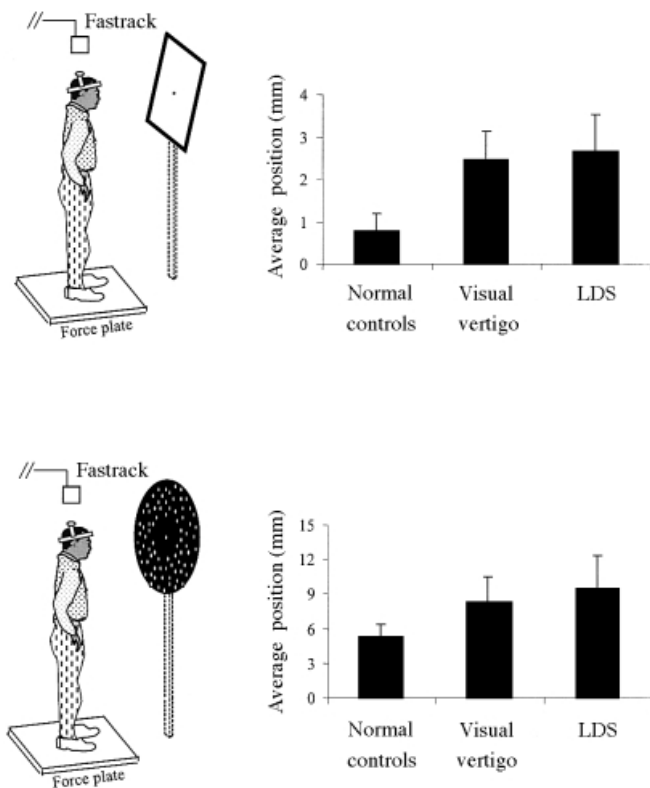
Subjects were required to adjust the rod without time constraints to the gravitational vertical with a hand-held potentiometer. Performance in subjective visual vertical adjustments was expressed as the deviation from gravitational vertical ( $0^\circ$ ) measured in degrees. Deviations to the left (counterclockwise) were counted as negative and deviations to the right (clockwise) as positive.

#### *Postural sway*

The subjects stood bare-foot on a force platform, secured by a harness. Their feet were placed on footprints symmetrically drawn at an angle of  $30^\circ$  with heels 2.5 cm apart. Postural sway was recorded in the lateral and anteroposterior direction using a force platform for the centre of foot pressure (COP). An additional head-mounted search coil (Polhemus Fastrak) was used to measure head sway in 10 LDS, 16 normal controls and 17 VV patients. These signals were digitally sampled at 50 Hz. During the different visual conditions, subjects were instructed to stand still and be relaxed with their hands at their side.

Four different visual conditions were used. (i) Eyes open: subjects were instructed to fixate for 45 s a dot at a distance of 80 cm from the eyes with the surrounding upright frame in full light illumination. (ii) Eyes closed: subjects closed their eyes for 45 s. (iii) Static disturbance (frame condition): the visual scene consisted of the luminescent frame placed at 80 cm from the eyes in the otherwise dark room. At the beginning of the trial, subjects were instructed to close their eyes for 15 s, during which the frame was oriented at  $28^\circ$  clockwise or counterclockwise; these first 15 s served as a baseline. Then, the subjects were asked to open their eyes and fixate a luminescent dot placed in the centre of the tilted frame for 45 s (Fig. 2, top left). (iv) Kinetic disturbance (disc condition): subjects were instructed to fixate a luminescent dot in the centre of the stationary luminescent disc placed at 80 cm from the eyes for 15 s in an otherwise dark room; these first 15 s served as a baseline. Then, the disc started to rotate either clockwise or counterclockwise, reaching, in 2.5 s, a constant velocity of  $30^\circ/s$ , sustained for 45 s (Fig. 2, bottom left). The four visual conditions were presented to each subject. Eyes open and eyes closed conditions were presented in a counterbalanced order prior to the two conditions of visual disturbances. Then the two conditions of visual disturbance were presented also in a counterbalanced order.

From COP and head recordings, two parameters of sway were computed: average deviation in the lateral direction,



**Fig. 2** Experimental set-up and mean lateral deviation (and standard error) of the centre of foot pressure (COP) and the head in normal control, visual vertigo and labyrinthine-defective subjects (LDS). The displacements induced by the static tilted frame (upper panels) and the rotating disc (lower panels) are shown. Statistics are presented in Table 3.

during frame and disc stimulation; and total sway path length, which combines lateral and antero-posterior body sway. Average deviation in the lateral direction was evaluated as the shift of the COP and of the head during the 45 s of visual disturbance (tilted frame or disc rotation) relative to the preceding 15 s baseline. This parameter is an indicator of postural orientation. Total sway path is the length of the path described by the COP or the head position signal, defined as the sum of the distances between sequential points sampled during the analysis period. This parameter is an indicator of postural instability. From these raw sway path data, two quotients were computed: the Romberg quotient (RQ = eyes closed sway/eyes open sway), which reflects the amount of postural stability provided by stationary visual surroundings; and a visual-kinetic quotient (V-KQ = rotating disc sway/eyes open sway), which quantifies the destabilizing effect of a moving visual stimulus. The subjective adjustments preceded the postural records, in order to allow patients to familiarize themselves with the potentially destabilizing visual stimuli. Subjects rested for 15 min between subjective adjustment and postural records.

### Vibration perception

Vibration perception thresholds at the ankles were measured as described (Bergin *et al.*, 1995) in most subjects. This was done in order to control for possible subtle differences in somatosensory function which may account for differences in the amount of postural sway.

### Statistical analysis

Statistical analysis was based on MANOVA (multi-way analysis of variance) for questionnaire data. For subjective visual vertical and postural data, MANCOVA (multi-way analysis of covariance) was used, with age as a covariable, as it was noted that there were age-related effects. Age effects will be reported only if significant ( $P < 0.05$ ) or close to significant. *Post hoc* mean comparisons were carried out with the Tukey HSD (honestly SD) test. All tests were carried out with SPSS. Additional statistics are described in Results.

## Results

### Questionnaire data

Completed questionnaire scores were subjected to MANOVA, which revealed a significant difference between groups (Pillai's trace = 4.17,  $P < 0.001$ ). Descriptive data and statistics are shown in Table 1. Both patient groups (VV and LDS) had higher levels of visually induced, autonomic and somatic anxiety symptoms than normal controls, but the levels of these symptoms did not differ in the two patient groups. However, the VV subjects had higher levels of vertigo than the controls or the LDS; the LDS group did not differ significantly from the control group. There were no between-group differences in childhood motion sickness susceptibility or trait anxiety, and the patient groups did not differ in handicap levels (Table 1).

### Subjective visual vertical

Since no differences between leftwards and rightwards visual stimuli were observed for the subjective visual vertical and the postural tasks, the results were normalized by reversing data with leftwards stimuli.

Subjective adjustments were subjected to MANCOVA, which showed a significant difference between groups (Pillai's trace = 2.4,  $P < 0.05$ ). Descriptive data and statistics are shown in Table 2. Differences between groups were observed during static frame tilt and disc rotation, but not when the vertical rod adjustments were performed in darkness. In darkness, the subjective visual vertical was close to the gravitational vertical in the three groups of subjects. In the presence of the tilted frame, the subjective visual vertical was deviated in the direction of the frame in the three groups (Fig. 1, top). Rod deviation was smaller in the control group compared with the other two patient groups, but no difference

**Table 1** Comparison of questionnaire scores (mean and SD) in the visual vertigo, labyrinthine-defective and control groups, with values of F and P for univariate ANOVAs

	Control	VV	LDS	F*	P
SVQ	0.19 (0.24)	1.59 (0.98) †	1.14 (0.68) †	21.28	<0.001
VSS-V	0.08 (0.17)	1.05 (0.85) †,‡	0.44 (0.55)	15.94	<0.001
VSS-A	0.27 (0.22)	1.17 (0.64) †	0.80 (0.46) †	18.57	<0.001
CMSQ-V	0.59 (1.19)	0.69 (1.51)	0.91 (1.41)	0.25	>0.1
CMSQ-N	1.60 (1.81)	1.98 (2.09)	1.34 (1.66)	0.48	>0.1
STAI	35.33 (9.32)	38.29 (9.52)	37.73 (9.47)	0.53	>0.1
VHQ	–	41.50 (13.69)	45.75 (14.77)	0.89	>0.1

CMSQ-N = Childhood Motion Sickness Questionnaire (nausea); CMSQ-V = Childhood Motion Sickness Questionnaire (vomiting); STAI = Spielberg Trait Anxiety Inventory; SVQ = Situational Vertigo Questionnaire; VSS-A = Vertigo Symptom Scale (autonomic and somatic anxiety component); VSS-V = Vertigo Symptom Scale (vertigo and imbalance component); VHQ = Vertigo Handicap Questionnaire. \*The value for the VHQ is *t* for the independent *t* test. †Significantly different from the control group (Tukey's honestly SD). ‡Significantly different from the LDS group (Tukey's honestly SD).

**Table 2** Comparison of subjective visual vertical values in the visual vertigo, labyrinthine-defective and normal control groups, with values of F and P for univariate ANCOVAs

	Control	VV	LDS	F	P
SVV darkness	0.49 (1.1)	0.39 (0.8)	-0.15 (1.8)	0.79	>0.1
RFE	3.49 (3.8)	6.51 (7.8) †	9.37 (6.9) †	4.49	<0.05
RDE	9.78 (4.2)	15.1 (8.4) †	16.2 (7.4) †	5.8	<0.01

Data are mean (standard deviation) expressed in degrees of deviation from the gravitational vertical. RFE, RDE = rod and frame, rod and disc effect; SVV = subjective visual vertical. †Significantly different from the control group (Tukey's honestly SD).

was observed between the LDS and the VV patients. During disc rotation, the subjective visual vertical deviated in the direction of motion, in all three groups (Fig. 1, bottom). The mean deviation of the subjective visual vertical in the direction of disc rotation was greater in the two patient groups than in the normal control subjects, but there was no difference between the two patient groups. Values of the subjective visual vertical during frame tilt were significantly correlated (Spearman coefficient) with those during disc rotation, both in LDS ( $r = 0.59$ ,  $P < 0.05$ ) and VV patients ( $r = 0.45$ ,  $P < 0.05$ ) but not in normal subjects ( $r = 0.23$ ,  $P > 0.05$ ). Frame and disc effects increased as a function of age [ $F(1,57) = 16.2$ ,  $P < 0.01$ ;  $F(1,57) = 3.8$ ,  $P = 0.056$ , respectively].

### Postural sway

Average deviation and sway path quotients, subjected to MANCOVA with age as a covariable, were significantly different between subject groups for COP (Pillai's trace = 3.5,  $P < 0.01$ ) and head sway (Pillai's trace = 2.4,  $P < 0.05$ ). Descriptive data and statistics are shown in Table 3.

### Average lateral deviation ('postural orientation')

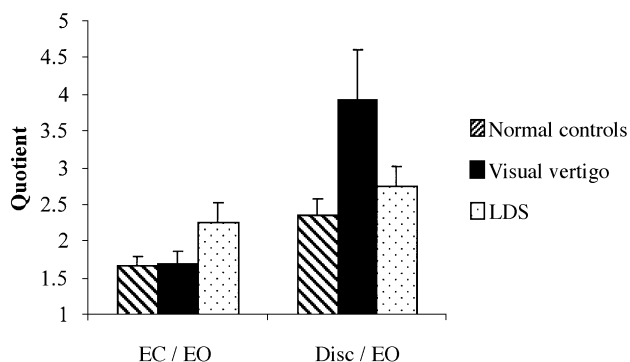
**Tilted frame.** The average deviation of the COP and the head in front of the tilted frame for the three groups of subjects is presented in Fig. 2, top (and Table 3 for statistics). The positive values indicate a postural re-adjustment induced in the direction of the tilted frame, with respect to the baseline position. There was a group effect around the level of significance (COP,  $P = 0.05$ ; head position,  $P = 0.056$ ), but *post hoc* mean comparisons did not reach significance.

**Rotating disc.** The average deviation of the COP and the head in front of the rotating disc for the three groups of subjects is presented in Fig. 2, bottom (and Table 3 for statistics). The rotating disc induced a postural re-adjustment in the direction of rotation in the three subject groups. It can be seen that the mean postural deviation in the direction of motion was larger in the two patient groups than in normal subjects but, due to large inter-individual differences, the factor group was not significant for COP data and was only close to significance for head position data. The deviations

**Table 3** Comparison of postural scores (mean and SD) for both the COP and the head in the visual vertigo, labyrinthine-defective and control subjects, with values of F and P for univariate ANCOVAs

		Control	VV	LDS	F	P
PFE	COP	0.08 (0.19)	0.24 (0.29)	0.27 (0.33)	3.15	0.05
	Head	0.19 (0.31)	0.51 (0.50)	0.57 (0.43)	3.10	0.056
PDE	COP	0.53 (0.51)	0.82 (0.83)	0.96 (1.16)	1.32	>0.1
	Head	1.14 (1.09)	2.24 (1.82)	2.51 (2.21)	2.89	0.068
RQ	COP	1.67 (0.54)	1.69 (0.70)	2.24 (1.06)	2.82	0.068
	Head	1.21 (0.27)	1.47 (0.37)	1.81 (0.63)*	6.06	<0.01
V-KQ	COP	2.36 (1.07)	3.93 (2.59)*	2.74 (1.09)	5.72	<0.01
	Head	1.40 (0.41)	2.27 (1.55)	1.93 (0.71)	2.72	0.078

Data for the postural frame effect (PFE) and disc effect (PDE) are expressed in centimetres of average deviation from the baseline position. Romberg quotient (RQ) is the ratio sway path length with eyes open/eyes closed. Visual-kinetic quotient (V-KQ) is the ratio sway path length during disc rotation/eyes open. \*Significantly different from the control group (Tukey's honestly SD).



**Fig. 3** Romberg quotient (sway path with eyes closed/sway path with eyes open) and visual-kinetic quotient (sway path during disc rotation/sway path with eyes open). EO = eyes open; EC = eyes closed. Mean and standard error are shown. Statistics are presented in Table 3.

of the COP and head position when subjects were facing the rotating disc were not significantly correlated (Spearman coefficient) with those measured in front of the tilted frame. No significant correlation was observed between the postural recordings and the subjective adjustments for the tilted frame or the rotating disc.

*Sway path ('postural stability')*

*Romberg quotient (eyes closed sway/eyes open sway).* Romberg quotient data for the COP and head sway are presented in Fig. 3 and, with statistics, in Table 3. Data with values >1 indicate that vision has a stabilizing effect, i.e. subjects sway less with eyes open than with eyes closed. The Romberg quotient was >1 in the three groups but was larger in the LDS group than in the other groups; univariate tests were close to significance for COP data and significant for head data.

*Visual-kinetic quotient (disc rotation sway/eyes open stationary sway).* Visual-kinetic quotients (V-KQ) are shown in Fig. 3 (and Table 3 for statistics). The values >1 observed in all groups indicate that the rotating disc had a destabilizing effect. This quotient was particularly large in the VV patients as compared with the control and the LDS groups. Univariate analysis indicated that the group effect was highly significant for the COP data, with VV patients significantly more destabilized by the rotating disc than the control subjects. The difference between VV patients and LDS patients was close to significance ( $P = 0.06$ ). Surprisingly, no difference was observed between the LDS group and the normal control subjects. Similar results were observed for the head data, but the factor group only approached significance ( $P = 0.078$ ). Supplementary analysis of postural stability quotients using root mean squares showed similar results to those based on sway path measurements (not shown).

For completeness, raw sway data, both sway path and root mean squares, are presented in Table 4. The general trends in the findings were: (i) sway activity was larger in the LDS than in the other two subject groups, with eyes open and closed; (ii) however, during disc rotation, sway values in the LDS were no longer different from those of VV subjects, and both LDS and VV patients had larger sway than the control subjects.

*Vibration perception thresholds.* Vibration perception thresholds were correlated (Spearman rank correlation coefficient) with age in the three groups, as previously reported for normal subjects (Bergin *et al.*, 1995): LDS ( $n = 14$ ),  $r = 0.45$ ; VV ( $n = 19$ ),  $r = 0.62$ ; controls ( $n = 18$ ),  $r = 0.60$ . With the factor age included as a covariable, no difference between the three groups was observed [univariate test:  $F(2,47) = 1.7$ ,  $P > 0.05$ ].

Experimental results in the four VV patients with no clinical or laboratory evidence of vestibular disease were

**Table 4** Comparison of mean (standard deviation) raw sway path (Swp) and root mean square (r.m.s.) data (in centimetres) for both the COP and head sway in the visual vertigo, labyrinthine-defective and normal control subjects

			Control	VV	LDS	F	P
EO	Swp	COP	31.3 (8.9)	35.8 (13.6)	57.5 (28.1)*,†	9.59	<0.01
		Head	32.2 (7.4)	39.9 (10.4)	49.8 (20)*	5.54	<0.01
	r.m.s.	COP	0.41 (0.13)	0.56 (0.28)	0.64 (0.44)*	3.43	<0.05
		Head	0.56 (0.17)	0.85 (0.40)	1.05 (0.71)*	3.91	<0.05
EC	Swp	COP	52.6 (24)	58.5 (28.7)	137 (114)*,†	8.81	<0.01
		Head	38.7 (9.8)	58.9 (24)	95.4 (61)*,†	8.3	<0.01
	r.m.s.	COP	0.47 (0.17)	0.74 (0.52)	1.14 (1.05)*	5.65	<0.01
		Head	0.60 (0.28)	1.11 (0.75)	1.64 (1.47)*	4.49	<0.05
Disc	Swp	COP	79.6 (64.3)	127.9 (72.2)	167.6 (125)*	4.54	<0.05
		Head	44.8 (14.8)	86.1 (52.5)*	100.6 (67.6)*	5.08	<0.05
	r.m.s.	COP	0.61 (0.29)	1.13 (0.56)*	1.23 (1.12)*	5.29	<0.01
		Head	0.80 (0.40)	1.77 (1.01)*	1.71 (1.31)	5.66	<0.01

Values of *F* and *P* are for univariate ANCOVAs. MANCOVAs indicated significant differences between groups both for the COP (Pillai's trace = 5.1, *P* < 0.01) and head sway path (Pillai's trace = 3.5, *P* < 0.01) and also both for the COP (Pillai's trace = 3.56, *P* < 0.01) and head (Pillai's trace = 3.3, *P* < 0.01) root mean square. EO = eyes open; EC = eyes closed. \*Significantly different from the control group (Tukey's honestly SD). †Significantly different from the VV group (Tukey's honestly SD).

indistinguishable from those of VV patients with vestibular disease.

## Discussion

### Clinical considerations

A past or current peripheral vestibular disorder was the most likely diagnosis in 17 of our 21 VV patients. Audio-vestibular test results often were either normal or mildly abnormal, but it is well established that frank abnormalities may be present only in the acute vertiginous phase (Okinaka *et al.*, 1993; Allum and Ledin, 1999). Our patients were assessed outside the acute episodes, and the visual aggravation of unsteadiness was a chronic feature, observed either in between vertigo attacks or months after a single vertigo episode. In our clinics, these patients have a working diagnosis of poorly compensated peripheral vestibular disorder.

The neuro-otologist also considered that, in nine out of 21 patients, a psychiatric component (anxiety, phobias, depression or panic) could be responsible for either producing or worsening the symptoms. However, a diagnosis of psychogenic dizziness can rarely be made with certainty for many reasons. These include the subjective nature of the symptom of dizziness, the fact that vestibular tests are restricted to the horizontal canals, the different views of clinicians on psychogenic dizziness and the finding that psychiatric symptoms are common in patients with genuine vestibular disease (Eagger *et al.*, 1992; Brandt, 1996; Bronstein *et al.*, 1997; Furman and Jacob, 1997).

In a previous study using a visuo-postural paradigm, it was postulated that VV was due to increased visual dependence in patients with an underlying peripheral or central vestibular disorder (Bronstein, 1995). The increased visual dependence would limit the patient's ability to compensate the vestibular

disorder fully, particularly in situations involving sensory conflict due to excessive visual motion. Several shortcomings of the previous study were addressed by the current one: (i) patients with CNS disease were excluded; (ii) a battery of questionnaires was used to assess possible predisposing causes (e.g. anxiety, motion sickness susceptibility) and symptom load (e.g. vertigo and its associated autonomic components); (iii) visual dependence was measured directly with the rod and frame test, as well as with kinetic visual input (rod and disc test); and (iv) measurements of visual dependence were carried out at both a perceptual and postural level.

### Questionnaire investigation

As expected, the questionnaires revealed significant differences in the vertigo subsection of the Vertigo Symptom Scale (VSS-V) between the different groups of subjects. In this questionnaire, 'vertigo' explicitly encompasses sensations of dizziness, giddiness, light-headedness or unsteadiness. The differences were due to high symptom reporting by VV patients. The LDS group comprised well-compensated patients, recruited from our files as controls rather than currently symptomatic patients.

Symptoms suggestive of somatization, health anxiety and autonomic arousal (VSS-A) were, however, significantly different from normal in both LDS and VV patients. This is expected for the VV group, as these patients are highly symptomatic, but perhaps surprising for the LDS group. It is not clear whether this unexpected finding in LDS indicates a trend for somatization or subtle autonomic dysfunction secondary to the loss of the vestibular drive to autonomic brainstem centres (Yates *et al.*, 1999; Jauregui-Renaud *et al.*, 2000; Radtke *et al.*, 2000).



The Situational Vertigo Questionnaire (Jacob *et al.*, 1989) primarily assesses space and motion discomfort observed in vestibular, but also in agoraphobic, patients (Jacob *et al.*, 1996). Items investigated include situations provoking symptoms in patients with VV (e.g. walking in supermarket aisles, watching moving scenes, scrolling screens) and driving-related symptoms which appear in the motorist disorientation syndrome (Page and Gresty, 1985). The situational questionnaire showed a strong group effect, with both groups of patients being significantly different from normal. Thus, these data confirm that symptoms in VV patients are not just the occasional discomfort that normal subjects may also experience in visually charged conditions. The data also show that LDS, even in a compensated state, experience a considerable amount of space and motion discomfort. This is not surprising since these and previous data show enhanced effects of visual stimuli on verticality perception and postural control in LDS (Bles *et al.*, 1983; Peterka and Benolken, 1995; Bronstein *et al.*, 1996).

The impact of the symptoms on patients' lives, assessed by the Vertigo Handicap Questionnaire, showed no difference between the VV and LDS groups. This demonstrates that in spite of fairly normal vestibular test results, patients with VV experience considerable levels of handicap and social disability (Yardley and Putnam, 1992; Yardley *et al.*, 1992; Kanayama *et al.*, 1995). It is of note that questionnaire data also established that there were no between-group differences in childhood motion sickness susceptibility or trait anxiety. Thus, it can be concluded that an enduring motion sickness susceptibility or trait anxiety are unlikely to explain the clinical features and visual susceptibility present in the VV patients.

### ***Psychophysical and postural investigations***

One of the aims of the study was to document that patients with VV have an objectively increased response to visually disorienting stimuli. The results show that both LDS and VV patients have larger tilts of the visual vertical under static (frame) and kinetic (disc) visual stimulation. Similarly, an increased postural tilt was observed in the two patient groups with both stimuli; correlation between these variables was not particularly strong, indicating that dissociation between symptoms and findings does occur. Thus, VV patients and LDS are visually dependent for perception and postural control.

The ability to counteract the effect of disorienting visual stimuli such as those used in this study depends on the presence of alternative sources of reliable sensory input (Bles *et al.*, 1983; Bronstein, 1986). In addition, sensory inputs must be centrally re-weighted so that, when facing moving visual surroundings, postural control centres 'listen' more to vestibulo-proprioceptive cues than to visual cues (Talbot and Brookhart, 1980; Bronstein *et al.*, 1990). It is therefore not surprising that LDS showed enhanced perceptual and postural responses to the tilted frame and the rotating disc, since they

lack vestibular input. The majority of patients with VV, however, had little or no abnormality on conventional vestibular testing. Thus, the problem in the VV patients is not lack of alternative sensory input. The problem seems to be one of enhanced visual dependence and an inability to resolve the visually induced sensory conflict by central re-weighting. The absence of CNS findings in the current VV patients makes it difficult to postulate a neurological disorder underlying these problems, so it is more likely that the strong visual dependence reflects an idiosyncratic perceptual style. Such idiosyncratic differences partly explain, for instance, the normal variability to develop motion sickness (Benson, 1999). Unfortunately, however, we have no means of establishing the level of visual dependence prior to the development of visuo-vestibular symptoms in our VV patients. An answer to the question of whether the increased visual dependence observed in the VV patients is pre-morbid, ions triggered by a vestibular insult or a combination of the two may prove elusive.

When the postural data are presented as sway path quotients (eyes open/eyes closed, or Romberg quotient, and stationary disc/rotating disc), new insight is gained. During eye closure, VV patients behave normally whereas LDS, as expected, have a moderate increase in the Romberg quotient. This means that, with eyes closed, VV patients use the available vestibulo-proprioceptive cues to the same extent as normal subjects. In contrast, during disc rotation, the ratio moving disc/stationary disc was only increased in the VV patients but not in the LDS (Fig. 3). This is a significant finding because it indicates that VV patients are destabilized selectively by visual motion, whereas LDS can suppress the conflict induced by disc rotation to near normal levels. This ability is probably an important part in the process of adaptive recovery to bilateral vestibular loss, as shown by Bles and colleagues (Bles *et al.*, 1983). Thus, patients with VV, in spite of the seemingly normal vestibulo-proprioceptive peripheral input, are incapable of resolving the conflict posed by the visual motion stimulus.

### ***Clinical implications***

The treatment effort in VV patients should be aimed at increasing subjective and postural tolerance to disorienting visual stimuli and increasing the patients' use of vestibulo-proprioceptive cues. A study treating unselected vestibular patients with repetitive optokinetic stimulation has shown an improvement in postural stability (Vitte *et al.*, 1994). Unfortunately, visual dependence and subjective improvement were not measured, but clinical experience suggests that patients' symptoms also improve after optokinetic training (A. Semont, personal communication). However, such stimuli are not used routinely in the majority of neuro-otology clinics, and further studies are needed to quantify the efficiency of such treatments and establish their clinical value.

An important differential diagnosis of VV is psychogenic dizziness and an answer to this question requires a separate

study. However, one study has shown that patients with psychogenic dizziness had, unexpectedly, smaller errors than normal subjects and peripheral vestibular patients with the rod and frame test (O'Connor *et al.*, 1989). Similarly, a study with computerized dynamic posturography found a surface-dependent strategy, rather than a visual-dependent strategy, in patients with agoraphobia (Jacob *et al.*, 1997). Thus, on the basis of the limited available evidence from the literature and our own findings, there are reasons to believe that VV is not a psychogenic disorder.

In summary, this study shows that patients with visually induced sensations of dizziness and unsteadiness experience a significant amount of symptoms and handicap. On the basis of questionnaire data, there is no evidence that the symptoms can be explained by trait anxiety or a tendency to motion sickness. Psychophysical and postural experiments indicate that VV patients are visually dependent and have difficulties in resolving sensory conflict induced by visual stimuli. It is suggested that these patients may benefit from incorporating repeated optokinetic stimulation and situations involving visuo-vestibular conflict in currently existing vestibular rehabilitation programmes.

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