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Changes of visual vertical perception: A long-term sign of unilateral and bilateral vestibular loss

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Abstract

This study investigates how unilateral and bilateral vestibular deafferentation modifies visual vertical perception in the presence of dynamic and static visual cues. We tested 40 Menière's patients before and after (from 1 week to 1 year) a curative unilateral vestibular neurotomy (UVN), and 4 patients with bilateral vestibular loss. Patients' performances were compared with those of 24 healthy subjects. The perception of the dynamic visual vertical (DVV) was investigated during optokinetic stimulations around the line of sight at various angular velocities. The static visual vertical (SVV) was recorded with a stationary visual pattern. In the acute stage after UVN, Menière's patients exhibited drastic impairment of DVV, which was tilted towards the lesioned side, whatever the direction of the optokinetic stimulation. In addition, the SVV was systematically tilted towards the lesioned side. The optokinetic-induced tilt of the vertical was asymmetrically organized around the new SVV with a significant decrease for contralesional stimulations and no change for ipsilesional stimulations, whatever the postoperative time. The SVV regained normal values 1 year postoperatively. For the patients with bilateral vestibular loss, the optokinetic-induced tilt of the visual vertical was drastically increased and symmetrically organized around an unmodified SVV aligned with the gravitational vertical. This study constitutes the first description of the recovery time-course of DVV perception after unilateral vestibular loss. Data reveal a long-term impairment of the DVV perception after unilateral vestibular loss, suggesting an asymmetrical processing of visual information and a permanent increased weight of dynamic visual cues after bilateral vestibular loss.

Keywords: Menière's disease patients; Unilateral vestibular neurotomy; Vestibular compensation; Roll optokinetic stimulation; Spatial frames of reference; Spatial cognition

1. Introduction

The vertical is a fundamental reference given by the direction of the gravitational vector and determining motor behavior on Earth. By representing the vertical, the brain can organize a proper erected posture with respect to the ground. Indeed, the control of body orientation and stabilization in space as well as locomotion depends on antigravity commands devoted to maintaining the body axis aligned with the vertical. To achieve such control, the brain has to build up a central representation of the vertical. There is much evidence that this representation is based on the integration of vestibular, visual, and somatosensory cues (for reviews see Berthoz, 1991; Bronstein, 1999; Mittelstaedt, 1999), and that it involves multimodal cortical areas (Brandt & Dieterich, 1999; Brandt, Dieterich, & Danek, 1994; Lopez, Lacour et al., 2005). In healthy humans, the role of vestibular cues in the perception of the vertical has been investigated mainly in roll-tilted subjects that committed perceptual errors whose direction depends on the amount of body tilt (Aubert, 1861; Dichgans, Diener, & Brandt, 1974; Guerraz,

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Poquin, & Ohlmann, 1998; Kaptein & Van Gisbergen, 2004, 2005; Mittelstaedt, 1983, 1991; Müller, 1916; Udo de Haes, 1970; Van Beuzekom & Van Gisbergen, 2000). These numerous studies have pointed to the preponderant, although not exclusive, role of the static gravitational/otolithic inputs on the perceived verticality. The specific role of dynamic vestibular cues has been evidenced through galvanic and rotational stimulations that led to a contralateral tilt of the perceived visual vertical (Mars, Popov, & Vercher, 2001; Pavlou, Wijnberg, Faldon, & Bronstein, 2003; Zink, Bucher, Weiss, Brandt, & Dieterich, 1998). The influence of the vestibular cues on verticality perception has also been investigated by several studies conducted on patients with peripheral and central vestibular disorders, especially in those with a brainstem lesion involving the vestibular nuclei complex (Brandt & Dieterich, 1987, 1992; Bronstein, Pérennou, Guerraz, Playford, & Rudge, 2003). Additionally, the role of visual cues in perception of verticality has been studied by manipulating static and dynamic visual cues. Tilt of static visual frame of reference with respect to the true gravitational vertical is known to bias the perception of the *static visual vertical* (SVV; the terminology "static visual vertical" refers to the perceived visual vertical in the presence of an immobile visual surrounding). In particular, the SVV perception recorded using the rod and frame test is deviated in the direction of the visual frame tilt (Guerraz et al., 1998, 2001; Lopez, Lacour, Magnan, & Borel, 2006; Witkin & Asch, 1948). Rotation of a visual background around the line of sight causes changes in the perceived visual vertical, a perceptual effect discovered by Dichgans, Held, Young, and Brandt (1972). Throughout the article, we will use the terminology "*dynamic visual vertical*" (DVV, as proposed by Brandt & Dieterich, 1987, 1992) to name the visually induced tilt of the subjective visual vertical in the direction of the circular optokinetic stimulation. This marked perceptual effect has been extensively described with circular optokinetic stimulations at steady velocity (Dichgans et al., 1972, 1974; Guerraz et al., 1998, 2001; Hughes, Brecher, & Fishkin, 1972; Isableu, Ohlmann, Crémieux, & Amblard, 1998), with sinusoidal oscillations of the visual surrounding (Mauritz, Dichgans, & Hufschmidt, 1977), and it has been reported for different visual characteristics of the rotating background (Held, Dichgans, & Bauer, 1975).

Vestibular loss is a good model for evaluating how vestibular and visual cues combine in building up a central representation of verticality. The consequences of impaired vestibular function on the perceived visual vertical have been studied mostly for patients with unilateral vestibular loss. Typically, in the absence of visual cues, the SVV was dramatically tilted towards the lesioned side (Bisdorff, Wolsley, Anastasopoulos, Bronstein, & Gresty, 1996; Böhmer, Mast, & Jarchow, 1996; Curthoys, Dai, & Halmagyi, 1991; Friedmann, 1970; Halmagyi, Gresty, & Gibson, 1979; Riordan-Eva, Harcourt, Faldon, Brookes, & Gresty, 1997; Vibert & Häusler, 2000). When vertical static visual references were available, the SVV deviation was significantly reduced, but it remained directed towards the lesioned side for patients tested in the acute period after vestibular loss (Borel, Harlay, Magnan, & Lacour, 2001; Lopez et al., 2006). Furthermore, tilted static visual references (rod and frame test) led to greater SVV deviation when the frame was tilted towards the lesioned ear than towards the healthy ear (Hafström, Fransson, Karlberg, & Magnusson, 2004; Lopez et al., 2006). These authors postulated unilateral vestibular-defective patients process static visual references asymmetrically. Little is known, however, about the effect of dynamic visual cues on the perception of the vertical after unilateral vestibular loss. An asymmetrical tilt of the DVV was reported for patients with vestibular brainstem lesions (Brandt & Dieterich, 1987, 1992) and for patients operated on for vestibular schwannoma (Goto et al., 2003). In the latter study, it was postulated that the asymmetry of the DVV resulted from the impaired SVV. However, no study investigated the recovery time-course of the DVV. In addition, the comparative effect of unilateral versus bilateral vestibular loss on DVV perception is still debated. To our knowledge, only two studies focused on the DVV perception with rotating background for bilateral vestibular-defective patients (Bronstein, Yardley, Moore, & Cleeves, 1996; Guerraz et al., 2001). They showed an increased visual dependence, evidenced by an increased magnitude of the DVV tilt.

In the present experiment, we analyzed how vestibular deafferentation modifies the visual vertical perception in the presence of dynamic and static visual cues. First, we analyzed the consequences of complete unilateral loss of vestibular information on the perception of the vertical. For this, we investigated DVV perception of Menière's patients who underwent a curative unilateral vestibular neurotomy (UVN). Dynamic visual cues were provided using circular optokinetic stimulations around the line of sight. To analyze the changes of the apparent upright as precisely as possible, we used a large range of visual stimulation velocities. To test whether the DVV depends on the direction of stimulation, i.e. the lesioned versus the healthy side, we used clockwise and counterclockwise rotations of the visual background. The second issue was to determine the recovery time-course of the DVV deficits and to compare it with that of the SVV. For this, we examined patients from the acute postoperative stage (1 week) up to the compensated stage (first postoperative year). Finally, we compared the weight of visual cues on DVV perception after bilateral and unilateral vestibular loss. All perceptive responses of the patients were compared with those of healthy control subjects.

2. Methods

2.1. Participants

The group of patients with unilateral vestibular loss included 40 patients suffering from Menière's disease. Patients were 20 men and 20 women aged 22–74 years (mean ± standard deviation: 48 ± 13 years). Each patient described the classical triadic syndrome of hearing loss, tinnitus and recurrent vertigo. They all underwent a neuro-otological investigation that revealed a pure unilateral vestibular deficit. Unilateral vestibular loss determined by the bithermal caloric irrigation with cold (30 °C) and warm (44 °C) water averaged 46 ± 16% (range 22–86%). Hearing loss averaged 45 ± 19 dB (range 5–70 dB) in the affected ear. The history of the symptoms ranged from 1 to 22 years (mean 6 ± 6 years). Attacks of vertigo were reported by the subjects as being the most incapacitating sign of their disease, causing them to stop their professional activity. Since the patients became resistant to antivertigo substances, they underwent unilateral neurotomy to abolish intractable vertigo without affecting hearing acuity. The surgical procedure was a retrosigmoid vestibular neurotomy (see Magnan, Bremond, Chays, Gignac, & Florence, 1991) on the right side for 23 patients and

on the left side for the 17 others. This procedure consists in a complete section of the vestibular nerve. The section is performed medially to the Ganglion of Scarpa when the vestibular nerve is one single structure. The caloric test showed a total lack of responses on the operated side acutely after UVN as well as later on. All patients were able to stand straight and motionless 1 week after surgery and they received no drug that could influence their performance. Patients with additional motor or visuomotor disorders were excluded from the study.

The group of patients with bilateral vestibular loss included four patients: one man and three women aged 26–76 years (mean 58 ± 22 years). Bilateral vestibular areflexia was established by the absence of response to bithermal caloric irrigation of both ears and to rotational testing in darkness. For one patient, ototoxicity by aminoglycoside antibiotic as treatment for tuberculosis was proven to have caused the vestibular loss. For the three others, the vestibular loss was classified as idiopathic. All patients had normal neurological examination and MRI was negative. Hearing loss was <20 dB in the two ears for three patients and one patient was totally deaf in one ear. Patients were tested during the compensated stage of the bilateral vestibular loss since the duration of the vestibular areflexia ranged from 3 to 10 years.

Performances of unilateral and bilateral vestibular defective patients were compared with those of 24 healthy control subjects selected on the basis of normal vestibular, visual and oculomotor functions. Controls included 12 men and 12 women aged 21–56 years (mean 40 ± 12 years). Informed consent was obtained from each subject before participation. The experimental protocols were approved by the local Ethics Committee and followed the recommendations of the Declaration of Helsinki.

2.2. Sessions

Menière's patients were examined 1 day before UVN ($D-1$) when not experiencing vertigo, and postoperatively throughout the recovery process (1 week, $D+7$; 2 weeks, $D+15$; 1 month, $D+30$; 2–3 months, $D+75$; 4–5 months, $D+135$; 1–2 years, $D>365$ after surgery). Since familiarity with the task might have modified the performance, we verified that responses were stable over time in a group of 10 control subjects tested at the same time intervals.

2.3. Experimental device

Examinations were done with subjects standing upright with the head unrestrained in front of a large disk (1.5 m in diameter) positioned 1.3 m in front of them. The subjects were surrounded by a protective handrail in order to recover stable upright posture should they experience imbalance or fear of falling. The disk was covered with a pseudorandom-dot visual pattern. Dots were 1 cm in diameter subtending 0.44° of the visual field with a density of 1175 dots/m^2. The disk was motorized and could be rotated clockwise (CW) and counterclockwise (CCW) with constant angular velocities (5, 10, 20, 40, 80 and 120° s^{-1}). A black circular target (2.8° of the visual field) was placed at the center of the disk, which was aligned with the direction of gaze of each subject. Visual vertical perception was assessed using a red laser line (Framiral, Cannes, France) subtending 8.8° of the visual field and projected at the center of the disk. The orientation of the laser line was adapted using a pair of joysticks held in each hand of the subject. CCW rotations of the laser line were performed by continuous or successive presses with the subject's left thumb on the left joystick. CW rotations were performed with the right thumb on the right joystick. The orientation of the visual vertical was continuously sampled by a computer.

2.4. Experimental procedures

Each experimental session was composed of tests performed with a stationary disk and during disk rotations allowing us to record the SVV and DVV, respectively. The subjects were instructed to look at the central target of the visual pattern. They wore goggles narrowing the visual field to the intended visual scene (40° in the horizontal and vertical planes). In this situation, all vertical and horizontal visual coordinates were excluded from the visual field. All measures were performed binocularly.

For the SVV measurement, recordings were done in light, in front of the stationary visual pattern, and consisted of four consecutive trials. The starting position of the laser line was positioned by the experimenter either CCW (two trials) or CW (two trials) with respect to the true gravitational vertical. The subjects were instructed to align the laser line with their perception of verticality.

For the DVV measurement, recordings were performed during optokinetic stimulations around the line of sight at each stimulation velocity and for each direction (ipsilesional and contralesional, or CCW and CW). Optokinetic stimulation consisted of 40 s periods of disc rotation. Recording started 10 s after the initiation of disc rotation and lasted 30 s. This protocol was aimed at excluding from the data the period of latency before the changes of the perceived DVV take place. During optokinetic stimulation, the subjects were instructed to adapt the position of the laser line so as to perceive it as vertical throughout the stimulation. Sequences of optokinetic stimulations were randomized across sessions and for each subject. They were triggered after a few minutes of rest to eliminate possible perturbations due to transition from one stimulation to another.

2.5. Data acquisition and processing

Mean SVV was expressed in degrees and calculated by averaging the four trials with the stationary disk, for each subject. For the DVV perception, the laser line position was sampled at 20 Hz. We calculated the mean position of the laser line during the last 30 s period of optokinetic stimulation. To compare the performance of Menière's patients before UVN and that of bilateral vestibular-defective patients with respect to that of the controls, we used the following convention: a 0° angle indicates the perceived vertical is exactly aligned with the true gravitational vertical, a positive sign refers to CW deviation of the perceived vertical, and a negative sign to CCW deviation. To compare the postoperative performance of Menière's patients with respect to the preoperative performance, the convention was the following: a positive sign refers to contralesional deviation of the perceived vertical and a negative sign to ipsilesional deviation. Moreover, to specify the nature of DVV changes, we computed the DVV with respect to the SVV. This computation is designed to free the dynamic perception from static changes, leading to a DVV centered on the SVV. We called this new parameter the "*relative dynamic visual vertical*" (RDVV). The RDVV was calculated by subtracting the SVV value from the DVV value. For each subject, the RDVV was analyzed for each stimulation velocity and for each direction of stimulation.

To quantify how body tilt affected the judgment of the perceived visual vertical, we recorded head and trunk tilts for five patients with unilateral vestibular loss and three patients with bilateral vestibular loss. For this, we used a video motion analyzer (Statis System, ABS, Marseille France) (see Borel et al., 2001). One camera (MICAM VHR 1000) was positioned 4 m behind the subject. The camera recognized white spherical markers (8 mm in diameter) reflecting the ultraviolet light from three neon tubes. Four markers were placed medially on the head (vertex and occiput) and trunk (C7 and L2). Marker position was sampled at 40 Hz. Markers were detected through shape recognition, and marker centroid coordinates were computed to elaborate 2D marker trajectories. In our experimental set-up, the overall accuracy of marker angular position was 0.1°. The markers defined a morphological model that gave head and trunk position and allowed us to compute orientation in the roll plane. The orientation in the roll plane was defined as the mean angular position of the head and trunk with respect to the gravitational vertical.

2.6. Statistical analysis

Statistical analyses were carried out using SAS procedures (SAS/STAT, Version 9, 2004). To characterize the preoperative status of the Menière's patients with respect to the control population, each dependent variable described above was analyzed using SAS Proc ANOVA with group (patients versus controls) as the between-subjects factor, and with direction of stimulation (CCW and CW) and stimulation velocity (5, 10, 20, 40, 80, 120° s^{-1}) as within-subjects factors. Similar statistical analyses were conducted to compare the performance of bilateral vestibular-defective patients to those of the control population. Moreover, we used the SAS Proc Mixed repeated measurement procedure to evaluate the postoperative-changes of the perceptive parameters over time (Littel, Milliken, Stroup, & Wolfinger, 1996). This analysis incorporated three within-subjects factors: session ($D-1, D+7, D+15, D+30, D+75, D+135, D>365$), direction of stimulation (ipsilesional and contralesional for patients), and stimulation velocity (5, 10, 20, 40, 80, 120° s^{-1}). Pearson correlations were calculated between

each dependent variable and each parameter of the clinical evaluation of the preoperative status of the patients. Finally, to measure the real strength of the association between the perceived visual vertical and the optokinetic stimulation velocity, controlling for the body (head and trunk) tilt, we used the partial correlation technique (Agresti & Finlay, 1997). Results were considered statistically significant for $P<0.05$.

3. Results

3.1. Comparison of the perception of the visual vertical for controls and Menière's patients before unilateral vestibular neurotomy

Comparison of perceptive performance of controls and Menière's patients before surgery showed no difference between the two groups for all the perceptive parameters. Concerning DVV perception, the group effect was not significant [$F(1,49)=0.48$, $P=0.49$]. The ANOVA evidenced significant effects of direction of stimulation [$F(1,49)=1044$, $P<0.0001$] and stimulation velocity [$F(10,490)=3.75$, $P<0.0001$]. The effects of direction of stimulation and stimulation velocity on DVV perception are illustrated in Fig. 1A. In controls as in patients, the tilt of the DVV increased with the stimulation velocity up to $20^\circ\,s^{-1}$. For the controls, the maximal amplitude of tilt averaged $7.1\pm2^\circ$ and $7.1\pm1.4^\circ$ for CCW and CW optokinetic stimulations, respectively. For the patients, the maximal amplitude of tilt averaged $9.2\pm2^\circ$ and $7.2\pm2^\circ$ for CCW and CW optokinetic stimulations, respectively.

In addition, regarding SVV perception, the group effect was not significant [$F(1,49)=0.00$, $P=0.99$]. The SVV did not differ from the true gravitational vertical for controls (mean, $0.1\pm0.4^\circ$; corrected one sample t-test, $P=0.90$) and for the Menière's patients (mean, $-1.1\pm0.9^\circ$; corrected one sample t-test, $P=0.08$) (Fig. 1A).

Finally, since DVV and SVV did not differ between controls and patients, RDVV did not differ between the two groups (Fig. 1B). Together, patients' preoperative data revealed symmetrical perceptive responses with respect to SVV centered on the gravitational vertical.

To specify how the clinical status of the patients relates to DVV perception, each parameter of the preoperative clinical evaluation was correlated with DVV perception. No correlation was found between DVV perception and history of the disease, vestibular loss or hearing loss, whatever the direction of stimulation and the stimulation velocity.

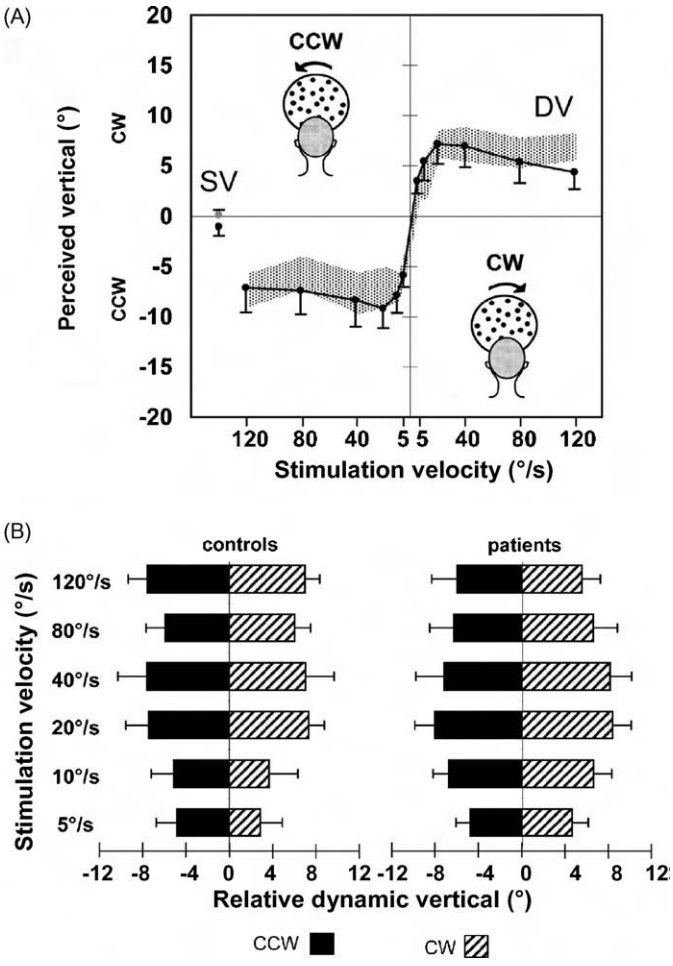

Fig. 1. Perceived visual vertical for Menière's patients tested 1 week before unilateral vestibular neurotomy and for controls. (A) Mean dynamic vertical (DVV) recorded for counterclockwise (CCW) and clockwise (CW) optokinetic stimulations in the group of Menière's patients plotted as function of the stimulation velocity. The gray area shows mean DVV (±95% confidence intervals to the mean) for the controls. The mean static vertical (SVV) is represented on the left of the curves for patients (filled symbol) and for controls (gray symbol). Positive and negative signs refer to CW and CCW deviations of the perceived vertical, respectively. Vertical bars represent the 95% confidence intervals to the mean. (B) Mean relative dynamic vertical (RDVV) recorded for CCW (filled histograms) and CW (hatched histograms) optokinetic stimulations for controls (left) and for patients (right). Horizontal bars represent the 95% confidence intervals to the mean. Note that the RDVV was symmetrical for CCW and CW stimulations in controls as well as in patients.

3.2. Effects of unilateral vestibular loss on the perception of the visual vertical

3.2.1. Dynamic visual vertical

ANOVAs with mixed models conducted on patients tested before and after UVN evidenced significant effects of session [$F(6,78)=90.29$, $P<0.0001$], direction of stimulation [$F(1,39)=910$, $P<0.0001$] and stimulation velocity [$F(10,390)=2.26$, $P<0.05$]. In addition, a significant interaction of session × direction of stimulation was observed [$F(6,78)=9.42$, $P<0.0001$]. This effect indicated that DVV was differently affected over time for ipsilesional and contralesional stimulations. Therefore, subsequent analyses are reported separately for each direction of stimulation. Fig. 2 illustrates the postoperative changes of DVV perception over time for contralesional and ipsilesional stimulations. The DVV was drastically impaired after UVN and performance was differently altered according to the direction of stimulation. Table 1 reports detailed analyses contrasting performance between patients tested before UVN and at each postoperative session for contralesional and ipsilesional stimulations.

ANOVA performed for contralesional optokinetic stimulations indicated that session [$F(6,78)=89.22$, $P<0.0001$] and stimulation velocity [$F(5,195)=2.63$, $P<0.05$] constitute the

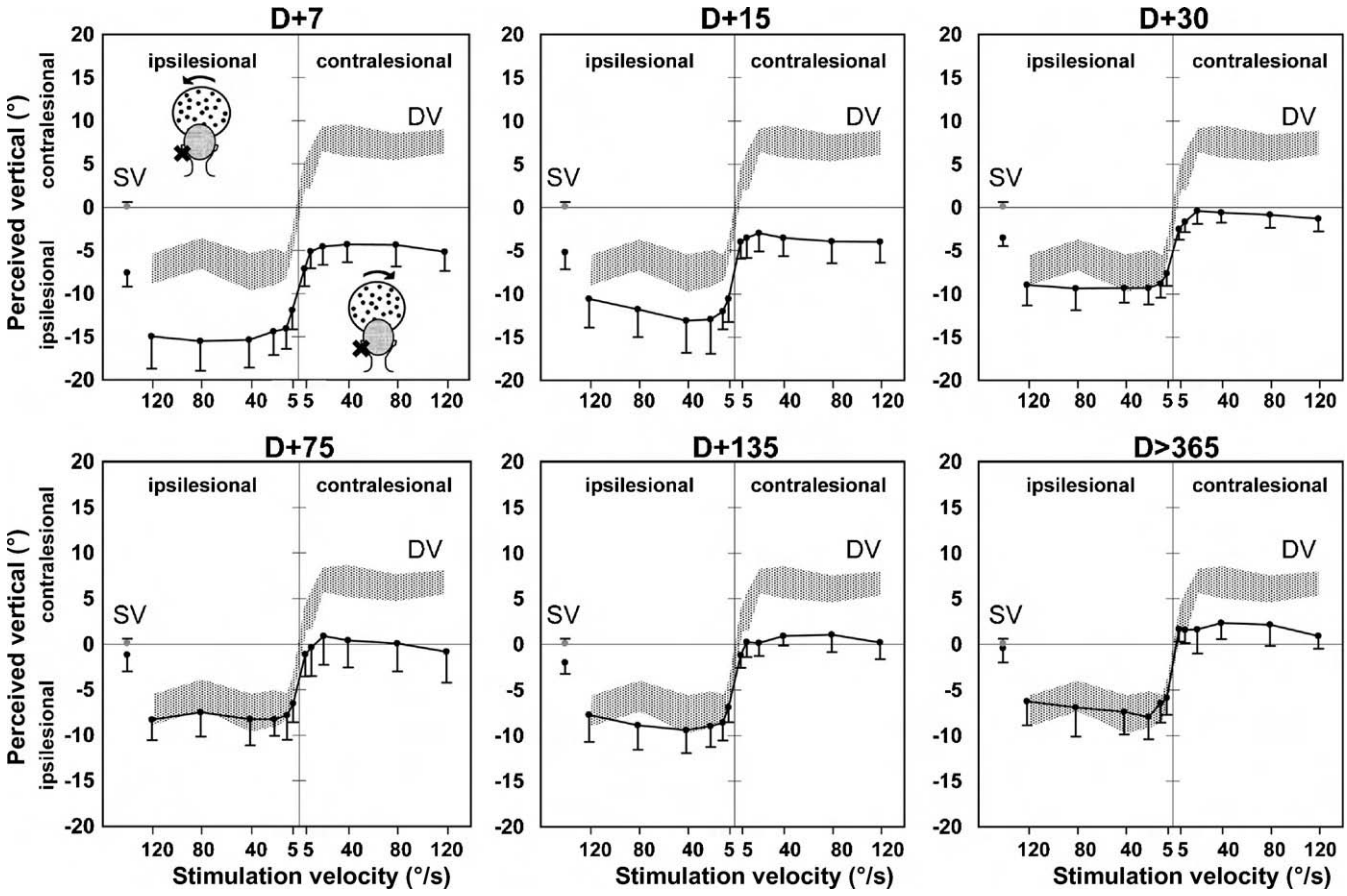

Fig. 2. Effect of unilateral vestibular loss on the perceived visual vertical. Mean DVV (curves) and SVV (symbols on the left) recorded for ipsilesional and contralesional optokinetic stimulations in the whole population of Menière's patients tested longitudinally after UVN (1 week, $D+7$; 2 weeks, $D+15$; 1 month, $D+30$; 2–3 months, $D+75$; 4–5 months, $D+135$; 1–2 years, $D>365$ after surgery). Same conventions as in Fig. 1A.

main effects providing the sources of variation after UVN. Interestingly, UVN induced changes in DVV to the point of changing the direction of the tilt. Indeed, during contralesional stimulation, the vertical was tilted towards the lesioned side. Such changes were maximal at $D+7$. Results indicated that recovery was already initiated as soon as $D+15$ [$F(1,78)=5.33$, $P<0.05$]. However, DVV perception remained significantly affected up to the first postoperative year.

Table 1
Recovery time-course of the dynamic visual vertical after unilateral vestibular neurotomy

	Ipsilesional stimulation	Contralesional stimulation
$D+7$	$F(1,78)=107.97$, $P<0.0001^*$	$F(1,78)=467.17$, $P<0.0001^*$
$D+15$	$F(1,78)=26.04$, $P<0.0001^*$	$F(1,78)=214.55$, $P<0.0001^*$
$D+30$	$F(1,78)=3.54$, $P=0.06$	$F(1,78)=172.58$, $P<0.0001^*$
$D+75$	$F(1,78)=0.02$, $P=0.90$	$F(1,78)=54.89$, $P<0.0001^*$
$D+135$	$F(1,78)=1.09$, $P=0.30$	$F(1,78)=85.63$, $P<0.0001^*$
$D>365$	$F(1,78)=1.02$, $P=0.31$	$F(1,78)=36.80$, $P<0.0001^*$

Statistical analysis conducted on Menière's patients contrasting DVV perception before UVN and at each postoperative session, for ipsilesional and contralesional stimulations. Local significant differences ($P=0.05$) are marked by asterisk. Note that the DVV was restored within 1 month for ipsilesional stimulations, whereas it remained uncompensated up to 1 year after UVN for contralesional stimulations.

ANOVA on DVV perception for ipsilesional optokinetic stimulations also evidenced significant effect of session [$F(6,78)=27.23$, $P<0.0001$]. Nevertheless, the recovery time-course of the DVV differed drastically from that described for the contralesional stimulations. Detailed analysis revealed that during ipsilesional stimulation, the tilt of DVV towards the operated side was significantly increased at $D+7$ and $D+15$ as compared to preoperative data. Recovery was initiated at $D+15$. Total compensation was observed at and after the first postoperative month; there was no difference between $D-1$ and later postoperative sessions (Table 1).

Finally, no correlation was found between the patients' preoperative clinical status (history of the disease, vestibular loss and hearing loss) and DVV perception, for each postoperative session, whatever the direction of stimulation and the stimulation velocity.

3.2.2. Static visual vertical

Fig. 2 illustrates the modifications of the mean SVV for each postoperative session. ANOVA indicated that perception of SVV varied as a function of the experimental session [$F(6,78)=14.84$, $P<0.0001$]. After UVN, the SVV was systematically tilted towards the operated side and averaged $-7.6\pm1.6^\circ$ 1 week postlesion. Restoration of the perceived SVV was long: it

regained the gravitational vertical only 1 year after UVN (mean, $-0.4 \pm 1.6^{\circ}$; corrected one sample *t*-test, $P = 0.90$).

3.2.3. Relative dynamic visual vertical

Previous results indicated a concomitant tilt of the SVV and the DVV. To specify whether the changes in DVV constituted a simple shift of the dynamic perception around a new static reference or whether postoperative changes revealed more complex mechanisms, we used the RDVV parameter. The ANOVA conducted on patients tested before and after UVN indicated an overall significant effect of session [$F(6,78) = 9.38$, $P < 0.0001$], direction of stimulation [$F(1,39) = 1102$, $P < 0.0001$] and stimulation velocity [$F(10,390) = 2.74$, $P < 0.005$]. The significant interaction of session × direction of stimulation [$F(6,78) = 11.40$, $P < 0.0001$] indicated that RDVV was differently affected by the neurotomy for contralesional and ipsilesional stimulations. Fig. 3 shows the comparisons of the mean RDVV perception for each postoperative session, direction of stimulation, and stimulation velocity. Table 2 reports detailed analyses contrasting performance between patients tested before UVN and at each postoperative session. Again, subsequent analyses are reported separately for contralesional and ipsilesional stimulations.

Regarding contralesional stimulations, ANOVA indicated an overall significant effect of session [$F(6,78) = 23.20$, $P < 0.0001$] and stimulation velocity [$F(5,195) = 2.77$, $P < 0.05$]. RDVV was dramatically reduced acutely after UVN. The decrease averaged 89, 62, 63, 58, 49 and 54% with respect to the preoperative data for stimulation velocities of 5, 10, 20, 40, 80 and 120° s^{-1}, respectively. Interestingly, the decreased RDVV observed 1 week after UVN was never improved thereafter (Fig. 3). Evidence of the absence of recovery is that RDVV recorded 1 year after UVN did not differ significantly from that recorded at $D + 7$ [$F(1,78) = 0.32$, $P = 0.57$].

Table 2
Recovery time-course of the relative dynamic visual vertical after unilateral vestibular neurotomy

	Ipsilesional stimulation	Contralesional stimulation
$D + 7$	$F(1,78) = 0.11$, $P = 0.74$	$F(1,78) = 72.48$, $P < 0.0001^{*}$
$D + 15$	$F(1,78) = 0.01$, $P = 0.94$	$F(1,78) = 67.56$, $P < 0.0001^{*}$
$D + 30$	$F(1,78) = 3.64$, $P = 0.06$	$F(1,78) = 72.28$, $P < 0.0001^{*}$
$D + 75$	$F(1,78) = 0.00$, $P = 0.99$	$F(1,78) = 55.63$, $P < 0.0001^{*}$
$D + 135$	$F(1,78) = 0.00$, $P = 0.81$	$F(1,78) = 60.94$, $P < 0.0001^{*}$
$D > 365$	$F(1,78) = 0.00$, $P = 0.76$	$F(1,78) = 52.50$, $P < 0.0001^{*}$

Statistical analysis conducted on Menière's patients contrasting RDVV perception before UVN and at each postoperative session, for ipsilesional and contralesional stimulations. Local significant differences ($P = 0.05$) are marked by asterisk. Note that the RDVV was unaffected for ipsilesional stimulations, whereas it was permanently affected for contralesional stimulations.

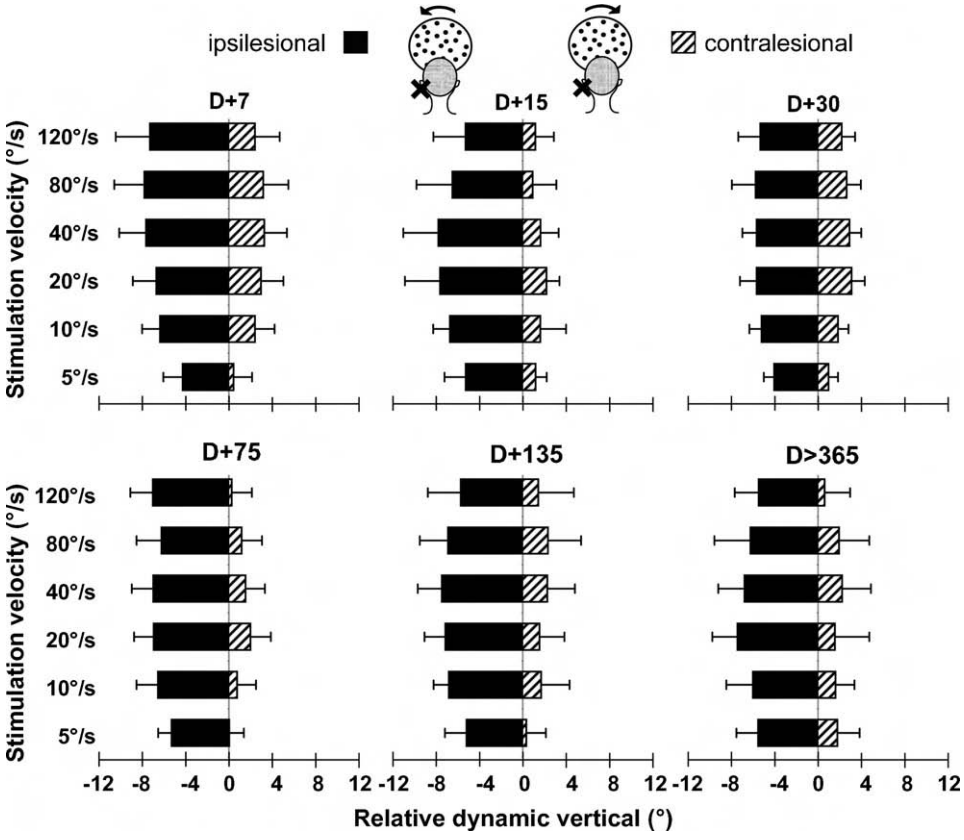

Fig. 3. Effect of unilateral vestibular loss on the relative dynamic visual vertical. Mean RDVV recorded for ipsilesional (filled histograms) and contralesional (hatched histograms) optokinetic stimulations in Menière's patients tested longitudinally after UVN (1 week, $D + 7$; 2 weeks, $D + 15$; 1 month, $D + 30$; 2–3 months, $D + 75$; 4–5 months, $D + 135$; 1–2 years, $D > 365$ after surgery). Horizontal bars represent the 95% confidence intervals to the mean. Note that the RDVV remained asymmetrical throughout the sessions.

Opposite results were obtained for ipsilesional stimulations. Surprisingly, the RDVV was never affected by the unilateral vestibular lesion [$F(6,78) = 1.17$, $P = 0.33$], even at $D + 7$ after UVN. The only significant source of variation of the RDVV was the stimulation velocity [$F(5,195) = 2.71$, $P < 0.05$]. As seen in Table 2, the RDVV remained similar to that obtained before surgery whatever the postoperative time, up to $D > 365$.

3.2.4. Body tilt

Head tilt and trunk tilt were quantified for five patients. Particularly, at $D + 7$, head tilt did not exceed 6–7° towards the lesioned side for ipsilesional stimulations. To quantify how head and trunk tilts affected the judgment of visual vertical, head tilt and trunk tilt were statistically controlled using partial correlations. With this method, we could statistically dissociate the optokinetic stimulation velocity effect on body (head and trunk) tilt and body tilt effect on the perceived visual vertical. For this sample of patients, the partial correlations between the stimulation velocity and the perceived visual vertical remained relatively high and significant at least at 5% even after having controlled for head tilt and trunk tilt. For the different patients, the partial correlation coefficient ranges were the following: $D - 1$ [0.55–0.88],

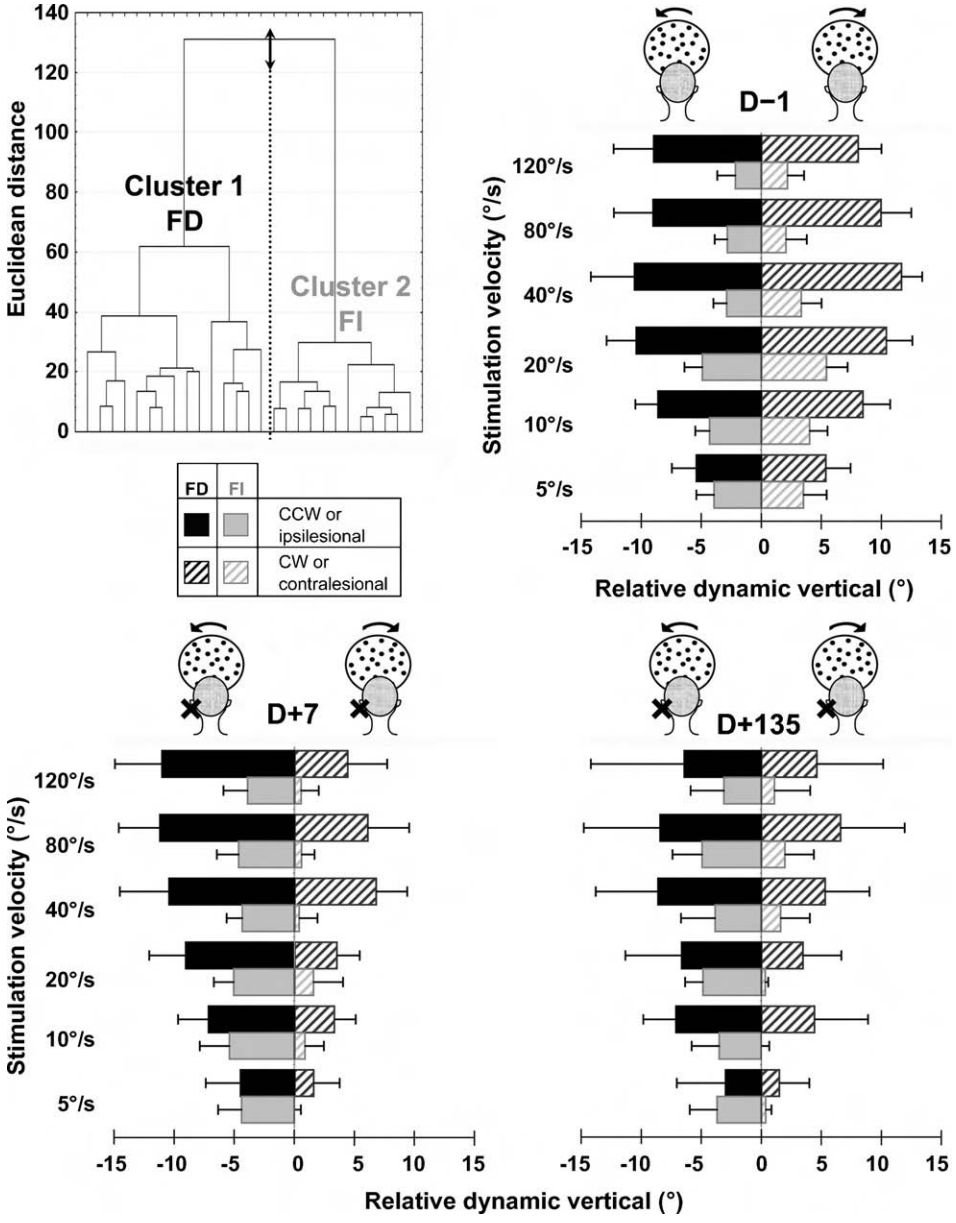

Fig. 4. Visual field dependence-independence in Menière's patients. Top left figure represents the hierarchical tree obtained from the ascending hierarchical classification on the basis of the relative dynamic visual vertical measured for clockwise and the counterclockwise optokinetic stimulations during the first experimental session. Each vertical line on the *X*-axis represents one patient of the population. *Y*-axis represents the Euclidean distance, that is, the dissimilarity index, between participants. Patients were grouped into clusters using Ward's aggregation method. The double arrow and the vertical dotted line represent the level of the breakdown to partition the patients into two clusters displaying close performances. The three histograms represent the mean relative dynamic visual vertical recorded for ipsilesional (filled histograms) and contralesional (hatched histograms) optokinetic stimulations in visual field dependent (FD) and visual field independent (FI) patients tested longitudinally before ($D - 1$) and after UVN (1 week, $D + 7$ and 4–5 months, $D + 135$). Same conventions as in Fig. 3.

$D+7$ [0.51–0.94], $D+30$ [0.70–0.93], $D+135$ [0.91–0.93], $D>365$ [0.53–0.66]. Consequently, the perceived visual vertical is clearly correlated with the stimulation velocity.

3.2.5. Visual field dependence-independence

Finally, we accounted for the role of the visual field dependence-independence in the perception of the dynamic visual vertical. Clustering took into account simultaneously the RDVV measured for the clockwise and the counterclockwise optokinetic stimulations (see Lopez et al., 2006 for a similar statistical procedure). Clustering showed that Menière's patients tested before UVN could be split into two well identified subpopulations. The patient population included 56% of field-dependent (FD) subjects, exhibiting large tilt of the vertical during optokinetic stimulations, and 44% of field-independent (FI) patients with weaker deviations of the visual vertical (Fig. 4). It was not possible to link the preoperative visual field dependence-independence of the Menière's patients either to personal characteristics or to clinical parameters. There was no significant statistical difference between the mean age (FD, 48 ± 14 years; FI, 45 ± 12 years; $P=0.55$), vestibular loss (FD, $46 \pm 13\%$; FI, $45 \pm 18\%$; $P=0.78$), hearing loss (FD, 44 ± 23 dB; FI, 43 ± 12 dB; $P=0.91$) and history of the disease (FD, 4 ± 3 years; FI, 8 ± 7 years; $P=0.11$) of the two subpopulations of patients.

The ANOVA indicated an overall significant effect of subpopulation [$F(1,27)=127.38$, $P<0.0001$] and a highly significant session × direction of stimulation interaction for FD [$F(6,34)=8.41$, $P<0.0001$] and FI [$F(5,19)=13.90$, $P<0.0001$] patients. Local analyses revealed that, regarding contralesional stimulation, the RDVV was drastically reduced at $D+7$ in both FD ($P<0.0001$) and FI ($P<0.0001$) patients with respect to $D-1$. The RDVV remained reduced at $D+135$ for the two subpopulations: FD ($P<0.0001$) and FI ($P<0.0001$). As described before, opposite results were obtained for ipsilesional stimulation for both subpopulations ($P>0.10$). These results obviously show that the effects of the unilateral vestibular loss do not depend on the visual field dependence versus independence and are largely observed in all the Menière's patients.

3.3. Effects of bilateral vestibular loss on the perception of the visual vertical

Bilateral vestibular loss induced drastic changes in perception of the vertical during optokinetic stimulations. Interestingly, the nature of changes differed from that observed after unilateral vestibular loss. Fig. 5 shows mean modifications of the perceptive responses for DVV, SV and RDVV.

3.3.1. Dynamic visual vertical

The ANOVA revealed that mean DVV perception was significantly different for the bilateral vestibular-defective patients and for the controls [$F(1,307)=5.68$, $P<0.05$]. As a result of bilateral vestibular loss, the tilt of the DVV was higher for patients than for controls. Contrary to data reported for unilateral vestibular patients, the increase was observed whatever the direction of stimulation (Fig. 5A).

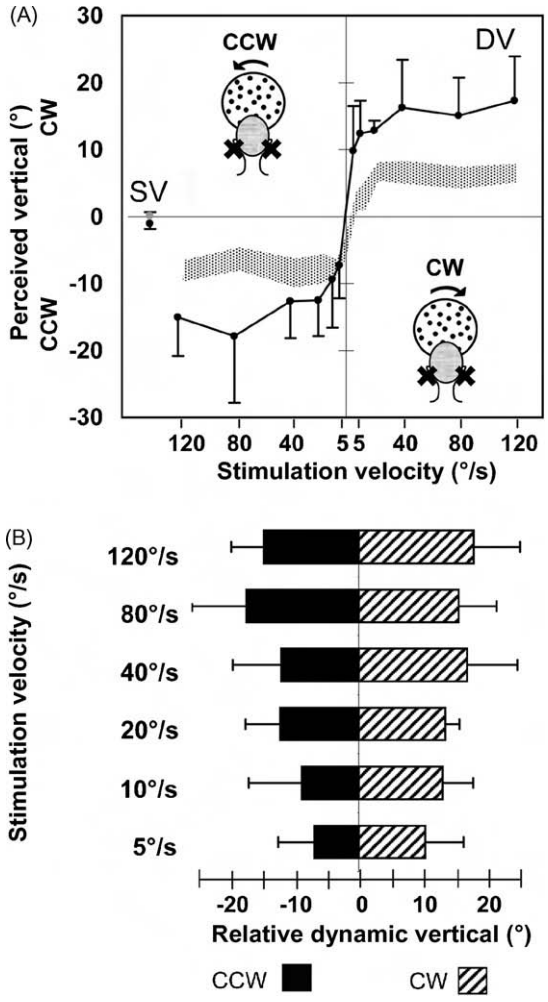

Fig. 5. Perceived visual vertical in bilateral vestibular-defective patients. (A) Mean DVV for CCW and CW optokinetic stimulations in the bilateral vestibular-defective patients plotted as a function of the stimulation velocity. The gray area shows mean DVV (±95% confidence intervals to the mean) for the controls. The mean SVV is represented on the left of the curves for patients (filled symbol) and controls (gray symbol). Same conventions as in Fig. 1A. (B) Mean RDVV recorded for CCW (filled histograms) and CW (hatched histograms) optokinetic stimulations. Same conventions as in Fig. 1B. Note that the RDVV was symmetrical for CCW and CW optokinetic stimulations.

3.3.2. Static visual vertical

For the SV, there was no effect of group [$F(1,26)=1.11$, $P=0.30$], indicating that perception of the SV was not impaired for bilateral vestibular-defective patients. Mean SV averaged $-0.5 \pm 0.8^{\circ}$ and did not differ from the true gravitational vertical (corrected one sample t-test: $P=0.54$) (Fig. 5A).

3.3.3. Relative dynamic visual vertical

The ANOVA performed on RDVV perception indicated a significant effect of group [$F(1,307)=97.49$, $P<0.0001$] and stimulation velocity [$F(5,307)=6.25$, $P<0.0001$]. These effects can be seen in Fig. 5B, which illustrates a large increase of RDVV for bilateral vestibular-defective patients. Detailed analyses showed that mean increase averaged 128% ($P<0.005$), 103% ($P<0.005$), 77% ($P<0.005$), 87% ($P<0.0001$), 174% ($P<0.0001$) and 129% ($P<0.0001$) with respect to the con-

trol data for stimulation velocities of 5, 10, 20, 40, 80 and 120° s^{-1}, respectively (averaged for both directions of stimulation). Finally, note that RDVV was increased for CCW and CW stimulations, and for each stimulation velocity.

3.3.4. Body tilt

Postural tilt of the head and trunk were recorded for three among the four bilateral vestibular-defective patients. For the different patients and the different velocities of the rotating disk, head tilt ranged from 1.5° to 7.0° (4.5 ± 1.7°: mean ± S.D.) and trunk tilt ranged from 0° to 2.0° (0.5 ± 1°). To quantify how head and trunk tilts affected the judgment of visual vertical for bilateral vestibular-defective patients, a statistical analysis was performed in which head tilt and trunk tilt were controlled using the partial correlation technique. The data showed a real optokinetic effect on the perceived visual vertical since partial correlation remained high (range 0.55–0.88) between the perceived visual vertical and the optokinetic stimulation velocity, controlling for the head tilt and the trunk tilt.

4. Discussion

This study investigated how unilateral and bilateral loss of vestibular inputs combines with dynamic visual cues to modify the perception of the visual vertical. Perceptive responses were analyzed for Menière's patients before a UVN and during the recovery time-course, and for bilateral vestibular-defective patients tested during the compensated stage. They were compared with those of healthy control participants.

4.1. Effects of unilateral vestibular loss on dynamic and static visual vertical perception

Our data show that DVV perception is drastically impaired after UVN. Evidence of impaired DVV perception is that the perceived visual vertical was strongly tilted towards the lesioned side during ipsilesional rotation of the visual field. Moreover, the perceived visual vertical was also tilted towards the lesioned side during contralesional stimulation. Consequently, UVN resulted in a dramatic asymmetry of DVV perception with respect to the gravitational vertical.

The increased tilt of the perceived visual vertical after UVN for ipsilesional stimulation agrees with that reported for neurological patients with brainstem lesions (Brandt & Dieterich, 1987) and for patients operated on for vestibular schwannoma (Goto et al., 2003). By contrast, only some of the patients with brainstem lesions showed a reversal of the direction of tilt of the perceived visual vertical for contralesional stimulation (Brandt & Dieterich, 1987).

The present study provides the first description of the recovery time-course of DVV perception after unilateral vestibular loss. The recovery time-course differed according to the direction of the optokinetic stimulation. Recovery was initiated at $D+15$. Total compensation was achieved 1 month after neurotomy for stimulation directed towards the lesioned side. However, despite progressive compensation, normal DVV perception was not totally regained up to 1 year postoperatively for stimulation directed towards the intact side. These results suggest either that periods longer than 1 year are needed for compensation of the DVV perception or that DVV impairment is part of dynamic functions that, in humans, constitute a permanent feature of unilateral vestibular loss (see Curthoys & Halmagyi, 1995). Using similar experimental conditions, we previously reported asymmetrical torsional optokinetic nystagmus remained uncompensated 3 months after UVN (Lopez, Borel, Magnan, & Lacour, 2005). An asymmetrical pattern of vestibular functions lasting several years has been reported after UVN for perception of the subjective postural vertical (Aoki, Ito, Burchill, Brookes, & Gresty, 1999), horizontal vestibulo-ocular reflex (Halmagyi et al., 1990) and dynamic ocular counterrolling (Diamond & Markham, 1983). Finally, we also reported impairments of walking performance during goal-directed locomotion up to 3 months after UVN (Borel et al., 2004).

In addition, the present data indicate that DVV perception of patients before UVN did not differ from that of controls. DVV perception was symmetrical for CCW and CW stimulations in patients preoperatively and in controls. Controls' performances are consistent with those reported in previous studies (Dichgans et al., 1972, 1974; Held et al., 1975; Hughes et al., 1972).

Concomitantly, UVN impaired perceptive responses in front of a static visual background. After neurotomy, the SVV was drastically deviated towards the lesioned side, similar to DVV. However, the recovery time-course of the SVV differed from that reported for the DVV, since SVV regained the gravitational vertical 1 year after UVN. That the static vertical or horizontal is deviated postoperatively has been extensively reported for unilateral vestibular-lesioned patients (Bisdorff et al., 1996; Böhmer et al., 1996; Curthoys et al., 1991; Friedmann, 1970; Halmagyi et al., 1979; Riordan-Eva et al., 1997; Vibert & Häusler, 2000). The deviation was mainly assumed to originate from the imbalance in otolithic functions (Gresty, 1996; Strupp et al., 2003).

4.2. Effects of unilateral vestibular loss on relative dynamic visual vertical perception

The RDVV allowed us to conclude that the impaired dynamic perceptive responses were not due to a simple shift of the DVV perception around a new SVV as a reference, but that they revealed more complex mechanisms. Indeed, our data indicate a postoperative asymmetry of the RDVV, suggesting an asymmetry of dynamic perceptive responses with respect to the new SVV. Evidence of asymmetry of dynamic perceptive changes after UVN is that the RDVV was strongly decreased for contralesional stimulations whereas it remained unaffected for ipsilesional stimulations. To our knowledge, such an asymmetry has never been reported. Regarding patients operated on for vestibular schwannoma, Goto et al. (2003) reported a symmetrical deviation of the DVV around the SVV, and suggested that their patients relied on their biased SVV as a reference. These discrepancies may be accounted for by the kind of pathology (Menière's disease versus schwannoma).

Why is the RDVV altered only for optokinetic stimulations towards the intact side? Considering that, in natural conditions,

visual background rotation towards the intact side occurs during a head-roll rotation towards the lesioned side, we hypothesize that the decrease in RDVV we observed may correlate with the decreased sensitivity to roll-tilt stimuli directed towards the lesioned side (Dai, Curthoys, & Halmagyi, 1989). Consequently, the postoperative decrease in RDVV for contralesional optokinetic stimulations could be taken as the behavioral evidence of neurophysiological alteration within central vestibular structures. Electrophysiological studies have emphasized the drastic imbalance of neuronal resting activity of the vestibular nuclei after the vestibular inputs had been unilaterally suppressed (Precht, Shimazu, & Markham, 1966; Ried, Maioli, & Precht, 1984; Ris, de Waele, Serafin, Vidal, & Godaux, 1995; Smith & Curthoys, 1988, 1989). In particular, the neurons in the ipsilesional vestibular nuclei, which mediate optokinetic stimulations towards the intact side, display a large decrease in their resting activity after UVN and a loss of dynamic sensitivity to head rotation (Precht et al., 1966; Smith & Curthoys, 1988; Xerri, Gianni, Manzoni, & Pompeiano, 1983).

Our results indicate that the decrease in RDVV for contralesional optokinetic stimulation remained unchanged up to 1 year after UVN. This is in line with previous observations from our group in Menière's disease patients showing long-term oculomotor and perceptive deficits for contralesional visual stimulations. In a previous study with similar experimental conditions, we reported that 3 months after UVN, torsional optokinetic nystagmus induced by rotation of a visual background around the line of sight also remained reduced for contralesional optokinetic stimulations, whereas it was compensated as soon as the first postoperative month for ipsilesional optokinetic stimulations (Lopez, Borel et al., 2005). Moreover, using the rod and frame test, we reported perceptive deficits up to 3 months postlesion when the frame was tilted towards the contralesional side (Lopez et al., 2006). Altogether, these behavioral observations suggest that a decreased sensitivity to contralesional optokinetic stimulation as well as to static tilt of the visual references towards the healthy side is a main feature and a long-term effect of the unilateral vestibular loss. These long-term behavioral deficits could be accounted for by long-term neurophysiological changes described in the vestibular nuclei complex in animal models. For instance, the number of responsive neurons and neuronal sensitivity in vestibular nuclei are affected up to 1 year after vestibular loss (Smith & Curthoys, 1988), although vestibular neuron activity on the lesioned side can be partially recovered (Pompeiano, Xerri, Gianni, & Manzoni, 1984; Ried et al., 1984; Ris et al., 1995; Smith & Curthoys, 1989; Xerri et al., 1983).

In other respects, it should be noted that despite the fact that subjects were instructed to stand straight and motionless, the head position in roll relative to the gravitational vector could have influenced the perceived vertical. This is not trivial because roll vection does not only induce tilts of the perceived vertical, but also head and body tilts (Clément, Jacquin, & Berthoz, 1985; Guerraz et al., 2001; Mauritz et al., 1977), and head and trunk tilts in turn affect the perceived verticality (Dichgans et al., 1974; Guerraz et al., 1998; Udo de Haes, 1970). In the present experiment, we have observed slight body deviations in the direction of the optokinetic stimulation, which were certainly limited because of the circular target at the centre of the visual surrounding. To dissociate the optokinetic effect on body tilt (head and trunk) and body tilt effect on the perceived vertical, we used a statistical control (partial correlations) showing that the perceived vertical is mainly correlated with the optokinetic stimulation. Previously interpreted as a realignment of the standard erect posture to the subject's central representation of the verticality (see Dichgans & Brandt, 1978; Dichgans et al., 1972), the optokinetic-induced body tilt appears to be a postural effect of the roll vection rather than a prominent factor influencing the perceived visual vertical. In addition, we have observed an overall ipsilesional tilt of the head, predominantly acutely after the UVN, as previously reported by many authors (e.g. Borel et al., 2001; Halmagyi et al., 1979). However, we showed the asymmetrical effect of the optokinetic stimulations on the perceived visual vertical cannot be attributable to the patients' roll head tilt.

4.3. Effects of bilateral vestibular loss on visual vertical perception

In this study, the weight of dynamic visual cues was increased during optokinetic stimulation in bilateral vestibular-defective patients. Indeed, deviation of DVV perception was significantly larger than that of control subjects, whatever the direction of stimulation. In addition, perception of the SVV was not impaired for these patients, therefore leading to an increased and symmetrical RDVV for CCW and CW optokinetic stimulations.

That the tilt of DVV is increased after bilateral vestibular loss agrees with previous studies (Bronstein et al., 1996; Guerraz et al., 2001). Such an increase has been shown for optokinetic stimulation up to $60^\circ\,s^{-1}$. In the present study, the increase in DVV and RDVV also occurred for higher stimulation velocities (up to $120^\circ\,s^{-1}$). Our data therefore indicate an increased weight of dynamic visual cues for a large range of stimulation velocity. These findings are in line with those of studies dealing with postural stabilization in a tilting room (Bles, Vianney de Jong, & de Wit, 1983) and with gaze stabilization during optokinetic stimulations (Huygen, Verhagen, Theunissen, & Nicolasen, 1989), which reported an increased visual dependence after bilateral vestibular loss.

An alternative hypothesis for the increased tilt of DVV in bilateral vestibular-defective patients is that the dynamic visual cues (normally indicating that the body is tilting) are no longer contradicted by the otolithic cues (indicating that the body is stationary). Since the graviceptive otolithic cues suppressed by the bilateral lesion did not counteract the visual illusion produced by the rotating background, the circular vection would be strongly enhanced after bilateral vestibular loss. In line with this view, a previous study demonstrated that vection was much more pronounced in bilateral vestibular-defective patients than in control subjects (Johnson, Sunahara, & Landolt, 1999). Another argument suggesting the preponderant role of the graviceptive otolithic cues is that hypogravity conditions such as weightlessness and microgravity phases of parabolic flights enhanced the effects of circular optokinetic stimulations

on circularvection and perceived body tilt (Cheung, Howard, & Money, 1990; Young, Shelhamer, & Modestino, 1986). However, it is important to note that after bilateral vestibular loss, only the graviception originating from the otolithic organs is totally suppressed. Graviception is still present in bilateral vestibular-defective patients through the mechanoreceptors of the muscles, joints, tendons, and skin (especially from the foot soles) [see, e.g. Hlavacka, Mergner, & Krizkova, 1996; Kavounoudias, Roll, & Roll, 2001] and through the abdominal viscera graviceptors (see, e.g. Mittelstaedt, 1992, 1995a,b; Trousselard, Barraud, Nougier, Raphel, & Cian, 2004; Vaitl, Mittelstaedt, & Baisch, 1997). The contribution of these extravestibular graviceptors in sensing the gravity is of particular importance in the bilateral vestibular-defective patients since it is well established that somatosensory cues constitute a prominent source of sensory substitution after vestibular loss (Strupp, Arbusow, Dieterich, Sautier, & Brandt, 1998). In line with this view, previous observations have shown that bilateral vestibular-defective patients were as accurate as healthy subjects in determining their body position in space in static or quasi-static conditions (Bisdorff et al., 1996; Bringoux et al., 2002). Nevertheless, given the marked deviation of the perceived vertical during optokinetic stimulations, it can be proposed that graviception through somatosensory inputs is not sufficient to counteract the effects of the optokinetic stimulation on the visual vertical perception.

4.4. Comparative effects of unilateral and bilateral vestibular loss on visual vertical perception

In the present study, we have shown that loss of vestibular function combines differently with the visual cues to modify the subjective visual vertical for unilateral and bilateral vestibular-defective patients. The data suggest that the adaptive mechanisms differ after unilateral and bilateral vestibular loss. In particular, the weight of dynamic visual cues during optokinetic stimulation depends on the extent of vestibular lesion.

After unilateral vestibular loss, the overall picture of the longitudinal changes in perceived vertical showed static and dynamic symptoms differ in recovery time. Indeed, the global asymmetry of the DVV is caused by the tilt of the SVV, on the one hand, and by the asymmetry of the RDVV, on the other hand. After UVN, central compensation progressively reduces the asymmetry of the static deficits so that the SVV perception regains control values and is aligned with the true gravitational vertical. By contrast, there is a lack of compensation of the dynamic deficits. RDVV remains uncompensated 1 year after UVN with no significant changes compared to 1 week postlesion. Finally, the present data argue against an increased weight of dynamic visual cues after UVN. Indeed, the tilt of the dynamic vertical was decreased for contralesional optokinetic stimulations and unchanged for ipsilesional optokinetic stimulations. These data suggest dynamic visual cues are asymmetrically processed in patients who lack a functioning vestibular apparatus on one side.

After bilateral vestibular loss, there is no reduced optokinetic deviation of the perceived vertical for any direction of the optokinetic stimulation. Clearly, the perceptive effects of a bilateral vestibular loss cannot be described as the sum of the effects of two UVN. In addition, after bilateral vestibular loss, there is no deficit in perception of the static visual vertical, which remains aligned with the gravitational vertical. The adaptive mechanisms involve a reweighting of the dynamic visual cues to compensate for the lack of vestibular information. Our data indicate that the visual cues constitute a prominent and permanent source of sensory substitution in functional recovery after bilateral loss of vestibular inputs.

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References

Agresti, A., & Finlay, B. (1997). *Statistical methods for the social sciences* (3rd ed.). Prentice-Hall Inc..

Aoki, M., Ito, Y., Burchill, P., Brookes, G. B., & Gresty, M. A. (1999). Tilted perception of the subjective 'upright' in unilateral loss of vestibular function. *The American Journal of Otology*, *20*, 741–747.

Aubert, H. (1861). Eine scheinbare bedeutende Drehung von Objecten bei Neigung des Kopfes nach rechts oder links. *Virchows Archiv*, *20*, 381–393.

Berthoz, A. (1991). Reference frames for the perception and control of movement. In J. Paillard (Ed.), *Brain and space* (pp. 82–111). Oxford/New York/Tokyo: Oxford University Press.

Bisdorff, A. R., Wolsley, C. J., Anastasopoulos, D., Bronstein, A. M., & Gresty, M. A. (1996). The perception of body verticality (subjective postural vertical) in peripheral and central vestibular disorders. *Brain*, *119*, 1523–1534.

Bles, W., Vianney de Jong, J. M., & de Wit, G. (1983). Compensation for labyrinthine defects examined by use of a tilting room. *Acta Otolaryngologica*, *95*, 576–579.

Böhmer, A., Mast, F., & Jarchow, T. (1996). Can a unilateral loss of otolithic function be clinically detected by assessment of the subjective visual vertical? *Brain Research Bulletin*, *40*, 423–427.

Borel, L., Harlay, F., Lopez, C., Magnan, J., Chays, A., & Lacour, M. (2004). Walking performance of vestibular-defective patients before and after unilateral vestibular neurotomy. *Behavioural Brain Research*, *150*, 191–200.

Borel, L., Harlay, F., Magnan, J., & Lacour, M. (2001). How changes in vestibular and visual reference frames combine to modify body orientation in space. *Neuroreport*, *12*, 3137–3141.

Brandt, T., & Dieterich, M. (1987). Pathological eye-head coordination in roll: Tonic ocular tilt reaction in mesencephalic and medullary lesions. *Brain*, *110*, 649–666.

Brandt, T., & Dieterich, M. (1992). Cyclorotation of the eyes and subjective visual vertical in vestibular brain stem lesions. *Annals of the New York Academy of Sciences*, *656*, 537–549.

Brandt, T., & Dieterich, M. (1999). The vestibular cortex. Its locations, functions, and disorders. *Annals of the New York Academy of Sciences*, *871*, 293–312.

Brandt, T., Dieterich, M., & Danek, A. (1994). Vestibular cortex lesions affect the perception of verticality. *Annals of Neurology*, *35*, 403–412.

Bringoux, L., Schmerber, S., Nougier, V., Dumas, G., Barraud, P. A., & Raphel, C. (2002). Perception of slow pitch and roll body tilts in bilateral labyrinthine-defective subjects. *Neuropsychologia*, *40*(4), 367–372.

Bronstein, A. M. (1999). The interaction of otolith and proprioceptive information in the perception of verticality: The effects of labyrinthine and CNS disease. *Annals of the New York Academy of Sciences*, *871*, 324–333.

Bronstein, A. M., Pérennou, D. A., Guerraz, M., Playford, D., & Rudge, P. (2003). Dissociation of visual and haptic vertical in two patients with vestibular nuclear lesions. *Neurology*, *61*, 1260–1262.

Bronstein, A. M., Yardley, L., Moore, A. P., & Cleeves, L. (1996). Visually and posturally mediated tilt illusion in Parkinson's disease and in labyrinthine defective subjects. *Neurology*, *47*, 651–656.

Cheung, B. S., Howard, I. P., & Money, K. E. (1990). Visually-induced tilt during parabolic flights. *Experimental Brain Research*, *81*, 391–397.

Clément, G., Jacquin, T., & Berthoz, A. (1985). Habituation of postural readjustments induced by motion of visual scenes. In M. Igarashi & F. O. Black (Eds.), *Vestibular and visual control on posture and locomotor equilibrium* (pp. 99–104). Basel: Karger.

Curthoys, I. S., Dai, M. J., & Halmagyi, G. M. (1991). Human ocular torsional position before and after unilateral vestibular neurectomy. *Experimental Brain Research*, *85*, 218–225.

Curthoys, I. S., & Halmagyi, G. M. (1995). Vestibular compensation: A review of the oculomotor, neural, and clinical consequences of unilateral vestibular loss. *Journal of Vestibular Research*, *5*, 67–107.

Dai, M. J., Curthoys, I. S., & Halmagyi, G. M. (1989). Linear acceleration perception in the roll plane before and after unilateral vestibular neurectomy. *Experimental Brain Research*, *77*, 315–328.

Diamond, S. G., & Markham, C. H. (1983). Ocular counterrolling as an indicator of vestibular otolith function. *Neurology*, *33*, 1460–1469.

Dichgans, J., & Brandt, T. (1978). Visual-vestibular interaction: Effects on self-motion perception and postural control. In R. Held, H. W. Leibowitz, & H. L. Teuber (Eds.), *Handbook of sensory physiology, perception* (pp. 755–804). Springer Verlag.

Dichgans, J., Diener, H. C., & Brandt, T. (1974). Optokinetic-graviceptive interaction in different head positions. *Acta Oto-laryngologica*, *78*, 391–398.

Dichgans, J., Held, R., Young, L. R., & Brandt, T. (1972). Moving visual scenes influence the apparent direction of gravity. *Science*, *178*, 1217–1219.

Friedmann, G. (1970). The judgement of the visual vertical and horizontal with peripheral and central vestibular lesions. *Brain*, *93*, 313–328.

Goto, F., Kobayashi, H., Saito, A., Hayashi, Y., Higashino, K., Kunihiro, T., et al. (2003). Compensatory changes in static and dynamic subjective visual vertical in patients following vestibular schwanoma surgery. *Auris, Nasus, Larynx*, *30*, 29–33.

Gresty, M. A. (1996). Vestibular tests in evolution. I. Otolith testing. In R. W. Baloh & G. M. Halmagyi (Eds.), *Disorders of the vestibular system* (pp. 243–255). New York: Oxford University Press.

Guerraz, M., Poquin, D., & Ohlmann, T. (1998). The role of head-centric spatial reference with a static and kinetic visual disturbance. *Perception and Psychophysics*, *60*, 287–295.

Guerraz, M., Yardley, L., Bertholon, P., Pollak, L., Rudge, P., Gresty, M. A., et al. (2001). Visual vertigo: Symptom assessment, spatial orientation and postural control. *Brain*, *124*, 1646–1656.

Hafström, A., Fransson, P. A., Karlberg, M., & Magnusson, M. (2004). Ipsilesional visual field dependency for patients with vestibular schwannoma. *Neuroreport*, *15*, 2201–2204.

Halmagyi, G. M., Curthoys, I. S., Cremer, P. D., Henderson, C. J., Todd, M. J., Staples, M. J., et al. (1990). The human horizontal vestibulo-ocular reflex in response to high-acceleration stimulation before and after unilateral vestibular neurectomy. *Experimental Brain Research*, *81*, 479–490.

Halmagyi, G. M., Gresty, M. A., & Gibson, W. P. (1979). Ocular tilt reaction with peripheral vestibular lesion. *Annals of Neurology*, *6*, 80–83.

Held, R., Dichgans, J., & Bauer, J. (1975). Characteristics of moving visual scenes influencing spatial orientation. *Vision Research*, *15*, 357–365.

Hlavacka, F., Mergner, T., & Krizkova, M. (1996). Control of the body vertical by vestibular and proprioceptive inputs. *Brain Research Bulletin*, *40*, 431–434.

Hughes, P. C., Brecher, G. A., & Fishkin, S. M. (1972). Effects of rotating backgrounds upon the perception of verticality. *Perception and Psychophysics*, *11*, 135–138.

Huygen, P. L., Verhagen, W. I., Theunissen, E. J., & Nicolasen, M. G. (1989). Compensation of total loss of vestibulo-ocular reflex by enhanced optokinetic response. *Acta Oto-laryngologica. Supplementum*, *468*, 359–364.

Isableu, B., Ohlmann, T., Crémieux, J., & Amblard, B. (1998). How dynamic visual field dependence-independence interacts with the visual contribution to postural control. *Human Movement Science*, *17*, 367–391.

Johnson, W. H., Sunahara, F. A., & Landolt, J. P. (1999). Importance of the vestibular system in visually induced nausea and self-vection. *Journal of Vestibular Research*, *9*, 83–87.

Kaptein, R. G., & Van Gisbergen, J. A. (2004). Interpretation of a discontinuity in the sense of verticality at large body tilt. *Journal of Neurophysiology*, *91*, 2205–2214.

Kaptein, R. G., & Van Gisbergen, J. A. (2005). Nature of the transition between two modes of external space perception in tilted subjects. *Journal of Neurophysiology*, *93*, 3356–3369.

Kavounoudias, A., Roll, R., & Roll, J. P. (2001). Foot sole and ankle muscle inputs contribute jointly to human erect posture regulation. *Journal of Physiology*, *532*, 869–878.

Littel, R. C., Milliken, G. A., Stroup, W. W., & Wolfinger, R. D. (1996). *SAS system for mixed models*. Cary, NC: SAS Institute Inc.

Lopez, C., Borel, L., Magnan, J., & Lacour, M. (2005). Torsional optokinetic nystagmus after unilateral vestibular loss: Asymmetry and compensation. *Brain*, *128*, 1511–1524.

Lopez, C., Lacour, M., Ballester, M., Dumitrescu, M., Anton, J. L., Nazarian, B., et al. (2005). Brain activations during subjective visual vertical judgment: A functional magnetic resonance imaging study. *Gait & Posture*, *21*(Suppl. 1), S13.

Lopez, C., Lacour, M., Magnan, J., & Borel, L. (2006). Visual field dependence-independence before and after unilateral vestibular loss. *Neuroreport*, *17*, 797–803.

Magnan, J., Bremond, G., Chays, A., Gignac, D., & Florence, A. (1991). Vestibular neurotomy by retrosigmoid approach: Technique, indications, and results. *The American Journal of Otology*, *12*, 101–104.

Mars, F., Popov, K., & Vercher, J. L. (2001). Supramodal effects of galvanic vestibular stimulation on the subjective vertical. *Neuroreport*, *12*, 2991–2994.

Mauritz, K. H., Dichgans, J., & Hufschmidt, A. (1977). The angle of visual roll motion determines displacement of subjective visual vertical. *Perception and Psychophysics*, *22*, 557–562.

Mittelstaedt, H. (1983). A new solution to the problem of the subjective vertical. *Naturwissenschaften*, *70*, 272–281.

Mittelstaedt, H. (1991). The role of the otoliths in the perception of the orientation of self and world to the vertical. *Zoologische Jahrbücher Abteilung Physiologie*, *95*, 419–425.

Mittelstaedt, H. (1992). Somatic versus vestibular gravity reception in man. *Annals of the New York Academy of Sciences*, *656*, 124–139.

Mittelstaedt, H. (1995a). New diagnostic tests for the function of utricles, saccules and somatic graviceptors. *Acta oto-laryngologica. Supplementum*, *520*, 188–193.

Mittelstaedt, H. (1995b). Evidence of somatic graviception from new and classical investigations. *Acta oto-laryngologica. Supplementum*, *520*, 186–187.

Mittelstaedt, H. (1999). The role of the otoliths in perception of the vertical and in path integration. *Annals of the New York Academy of Sciences*, *871*, 334–344.

Müller, G. E. (1916). Über das Aubertsche Phänomen. *Zeitschrift für Psychologie und Physiologie der Sinnesorgane*, *49*, 109–246.

Pavlou, M., Wijnberg, N., Faldon, M. E., & Bronstein, A. M. (2003). Effect of semicircular canal stimulation on the perception of the visual vertical. *Journal of Neurophysiology*, *90*, 622–630.

Pompeiano, O., Xerri, C., Gianni, S., & Manzoni, D. (1984). Central compensation of vestibular deficits. II. Influences of roll tilt on different-size lateral vestibular neurons after ipsilateral labyrinth deafferentation. *Journal of Neurophysiology*, *52*, 18–38.

Precht, W., Shimazu, H., & Markham, C. H. (1966). A mechanism of central compensation of vestibular function following hemilabyrinthectomy. *Journal of Neurophysiology*, *29*, 996–1010.

Ried, S., Maioli, C., & Precht, W. (1984). Vestibular nuclear neuron activity in chronically hemilabyrinthectomized cats. *Acta Oto-laryngologica*, *98*, 1–13.

Riordan-Eva, P., Harcourt, J. P., Faldon, M., Brookes, G. B., & Gresty, M. A. (1997). Skew deviation following vestibular nerve surgery. *Annals of Neurology*, *41*, 94–99.

Ris, L., de Waele, C., Serafin, M., Vidal, P. P., & Godaux, E. (1995). Neuronal activity in the ipsilateral vestibular nucleus following unilateral labyrinthectomy in the alert guinea pig. *Journal of Neurophysiology*, *74*, 2087–2099.

Smith, P. F., & Curthoys, I. S. (1988). Neuronal activity in the ipsilateral medial vestibular nucleus of the guinea pig following unilateral labyrinthectomy. *Brain Research*, *444*, 308–319.

Smith, P. F., & Curthoys, I. S. (1989). Mechanisms of recovery following unilateral labyrinthectomy: A review. *Brain Research. Brain Research Reviews*, *14*, 155–180.

Strupp, M., Arbusow, V., Dieterich, M., Sautier, W., & Brandt, T. (1998). Perceptual and oculomotor effects of neck muscle vibration in vestibular neuritis. Ipsilateral somatosensory substitution of vestibular function. *Brain*, *121*, 677–685.

Strupp, M., Glasauer, S., Schneider, E., Eggert, T., Glaser, M., Jahn, K., et al. (2003). Anterior canal failure: Ocular torsion without perceptual tilt due do preserved otolith function. *Journal of Neurology, Neurosurgery, and Psychiatry*, *74*, 1336–1338.

Trousselard, M., Barraud, P. A., Nougier, V., Raphel, C., & Cian, C. (2004). Contribution of tactile and interoceptive cues to the perception of the direction of gravity. *Brain Research. Cognitive Brain Research*, *20*, 355–362.

Udo de Haes, H. A. (1970). Stability of apparent vertical and ocular countertorsion as a function of lateral tilt. *Perception and Psychophysics*, *8*, 137–142.

Vaitl, D., Mittelstaedt, H., & Baisch, F. (1997). Shifts in blood volume alter the perception of posture. *International Journal of Psychophysiology*, *27*, 99–105.

Van Beuzekom, A. D., & Van Gisbergen, J. A. (2000). Properties of the internal representation of gravity inferred from spatial-direction and body-tilt estimates. *Journal of Neurophysiology*, *84*, 11–27.

Vibert, D., & Häusler, R. (2000). Long-term evolution of subjective visual vertical after vestibular neurectomy and labyrinthectomy. *Acta Oto-laryngologica*, *120*, 620–622.

Witkin, H. A., & Asch, S. E. (1948). Studies in space orientation. IV. Further experiments on perception of the upright with displaced visual fields. *Journal of Experimental Psychology*, *38*, 762–782.

Xerri, C., Gianni, S., Manzoni, D., & Pompeiano, O. (1983). Central compensation of vestibular deficits. I. Response characteristics of lateral vestibular neurons to roll tilt after ipsilateral labyrinth deafferentation. *Journal of Neurophysiology*, *50*, 428–448.

Young, L. R., Shelhamer, M., & Modestino, S. (1986). M.I.T./Canadian vestibular experiments on the Spacelab-1 mission. 2. Visual vestibular tilt interaction in weightlessness. *Experimental Brain Research*, *64*, 299–307.

Zink, R., Bucher, S. F., Weiss, A., Brandt, T., & Dieterich, M. (1998). Effects of galvanic vestibular stimulation on otolithic and semicircular canal eye movements and perceived vertical. *Electroencephalography and Clinical Neurophysiology*, *107*, 200–205.