Acta Otolaryngol (Stockh) 1997; 117: 1–16

Deviation of the Subjective Vertical in Long-standing Unilateral Vestibular Loss

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Tabak S, Collewijn H, Boumans LJJM. *Deviation of the subjective vertical in long-standing unilateral vestibular loss.* Acta Otolaryngol (Stockh) 1997; 117: 1–16.

We evaluated changes in the subjectively perceived gravitational vertical as an index of imbalance in the function of the right and left otolith organs. In addition to normal subjects ($n = 25$), we measured patients with a longstanding (mean 4.5 year ± 3.2 SD; range 0.5–11.5 years) unilateral vestibular loss after surgery for acoustic neuroma ($n = 32$), patients with partial unilateral vestibular loss ($n = 7$) and patients with bilateral vestibular hyporeflexia ($n = 8$). Normal subjects could accurately align a vertical luminous bar to the gravitational vertical in an otherwise completely dark room (mean setting $-0.14° \pm 1.11$ SD). Patients with leftsided (complete; $n = 13$) or rightsided (complete; $n = 19$ and partial; $n = 7$) unilateral vestibular loss made mean angular settings at $2.55° \pm 1.57$ (SD) leftward and $2.22°$ (± 1.96 SD) rightward, respectively. These means differed highly significantly from the normal mean ($p < 0.00001$). In the time interval investigated (0.5–11.5 years) the magnitude of the tilt angle showed no correlation with the time elapsed since the operation. The mean setting by patients with clinically bilateral vestibular loss ($-1.17° \pm 1.96$ SD; $n = 8$) did not significantly differ from the control group. The systematic tilts of the subjective vertical in patients with a unilateral vestibular impairment were correlated with their imbalance in canal-ocular reflexes, as reflected by drift during head-oscillation at 2 Hz ($r^2 = 0.44$) and asymmetries in VOR-gain for head-steps ($r^2 = 0.48–0.67$). These correlations were largely determined by the signs of the asymmetries; correlation between the absolute values of the VOR gain asymmetries and subjective vertical angles proved to be virtually absent. We conclude that the setting of the subjective vertical is a very sensitive tool in detecting a left–right imbalance in otolith function, and that small but significant deviations towards the defective side may persist for many years (probably permanently) after unilateral lesions of the labyrinth or the vestibular nerve. *Key words*: *unilateral and bilateral vestibular loss, human, acoustic neuroma, ocular torsion, otolith, utricle.*

INTRODUCTION

The perception and control of the orientation of the head relative to the direction of gravity is vital in maintaining a normal posture and equilibrium. Obviously, the otolith organs provide an important sensory input to these functions. The evaluation of otolith function, however, has been complicated and often unreliable, until recently. Historically, a link between the otoliths and the torsional position of the eyes has been long demonstrated, and for many decades assessing otolith function from the magnitude of ocular counter-rolling (OCR) has been attempted. Diamond and Markham (1) reviewed this work and concluded that this approach is unfruitful in diagnosing unilateral deficits in otolith function. Attempts to correlate the side of the reduced OCR or asymmetry with the side of injury produced contradictory results, and a significant number of cases of known unilateral vestibular nerve section showed neither asymmetry nor reduced magnitude of the OCR. As a refinement, Diamond and Markham (1) recorded OCR continuously during ongoing roll at a constant, low velocity. Even under these conditions the asymmetry of the binocular OCR profiles in patients with unilateral vestibular nerve section appeared to be inconsistent, although the binocular OCR profiles of these patients were abnormal, compared to the normal subjects, when consistency, conjugacy, smoothness, and symmetry were examined. Such tests of the OCR probe the modulation of ocular torsional position in relation to changes in the relative orientation between head and gravity; they do not address changes in the basic torsional orientation of the eye. In other words, they probe gain, not offset of otolith–ocular reflexes. As the gain of OCR is very low (about 0.1) even in normal conditions, this probe is indeed likely to be not very sensitive.

In recent years, however, it has become clear that a primary effect of unilateral labyrinth lesions consists of a substantial change (offset) of the basic torsional angle of the eyes, with rotation of the upper poles of the eyes towards the lesioned side; this ocular tilt is accompanied by a similarly directed tilt of the subjective vertical. Early observations of changes in the subjective vertical due to peripheral and central vestibular lesions were reported by Friedmann (2). Halmagyi et al. (3) described a case of transient abnormalities of posture after acute destruction of the left labyrinth, consisting of leftward ocular torsion (25°, as assessed by fundus photography), leftward head tilting (10°), and a right-over-left skew deviation (7°). The above-described symptomatology is known as the "ocular tilt reaction": a head–eye postural synkinesis including ocular torsion, head tilt, and changes in visual perception of the vertical, all

 ISSN 0001-6489

directed to the side of the lesion. Dai et al. (4) tested the ability to perceive roll tilt in 33 patients before and from 1 week to 6 months after unilateral vestibular neurectomy, by rotating the subjects on a fixed-chair centrifuge in a completely dark room and having them set a small, illuminated bar to the perceived gravitational horizontal. (Both the subjective vertical and horizontal are being used in this type of experiments, with identical results.) Normal subjects ($n = 31$) accurately aligned the bar with respect to the gravito-inertial resultant vector. In contrast, patients had asymmetrical perceptions of resultant vectors to the right and to the left 1 week after the unilateral vestibular loss. Even at rest there was an asymmetry in the baseline settings; i.e. in order to see the bar as gravitationally horizontal, they set the bar down to the side of the lesion. A progressive decrease in perceptual asymmetry followed, rapidly in the first 3 weeks, and more slowly in the next 6 months. These findings were further amplified by Curthoys et al. (5), who established with fundus photography that in 23 patients, measured 1 week after unilateral vestibular neurectomy, both eyes were torted towards the side of the operation (average 9.5°). There was a significant reduction of this torsion in the 16 weeks following the operation, after which time residual torsion was on the order of 2.8°. The change in torsion was paralleled by a change in the orientation of the subjective gravitational horizontal: a high correlation ($r = 0.95$) was found between the direction and magnitude of the change in torsional eye position and the direction and magnitude of the change in the perceived visual horizontal one week after the operation, with the change in perception being roughly similar to (and statistically even slightly larger than) the change in torsion.

Thus, it is apparent that absolute changes in the orientation of eye torsion and subjective vertical are a much more sensitive and specific index of imbalance in otolith signals than asymmetries in the OCR responses to tilt stimuli. The robust correspondence between the rotations of the subjective vertical and of the eye (5) suggests that they are either controlled by the same common factor or that the subjective vertical is predominantly retinotopically determined. Whichever of these may be the case, determination of the subjective vertical seems much more practical as a test of otolith imbalance than fundus photography. Normal subjects can align a luminous rod in a dark room with the objective vertical or horizontal with an accuracy better than 2° (4, 6). Thus, this test, which requires only simple equipment, has an absolute reference. In contrast, fundus photography requires special equipment and expertise, while small torsional deviations may remain undetected because of the anatomical variability in retinal landmarks.

In our previous work (7, 8 and in preparation) we measured three dynamic vestibular responses to high frequency, passive head movements: (1) VOR gain; (2) VOR phase lag; and (3) directional trend of gaze during head oscillation, in normal and vestibular patients. These tests addressed semicircular canal functions. In the present article we describe the changes in the subjective vertical for a number of these patients, as a probe of the static vestibular otolith functions. The results of our study extend the results of the authors cited above; while the latter concentrated on the effects occurring within months after acute lesions, our patients were measured years after the lesion. The mean settings in our patient group with unilateral vestibular loss were systematically biased towards the side of the lesion. Patients with bilateral vestibular hyporeflexia did not significantly differ from the control group.

MATERIAL AND METHODS

Protocol

To evaluate changes in the subjective vertical, subjects were seated on a chair in complete darkness and rested their head on chin and forehead rests in an upright position, to the best estimate of the experimenter. A dim luminous bar (length 81 cm, width 1 cm) was back-projected on a large translucent screen, 145 cm in front of the subject. This was the only visible object. The tilt of the line could be adjusted by the subject by rotating a disk, connected to a potentiometer, to a subjectively vertical position. The voltage emerging from the potentiometer reflected the objective tilt of the line and was recorded when the subject had finished his setting. The initial orientation of the line was randomized before each of 16 successive trials. Mean and SD of the 16 settings were determined for each subject. The true vertical orientation will be denoted as 0°; leftward and rightward tilts will be represented by negative and positive angles.

Subjects

Approval by the local ethical committee and informed consent from the subjects were obtained for all experiments. We recorded 25 healthy subjects who had no known ocular or vestibular pathologies. Absence of vestibular pathology was confirmed by testing them with our reactive torque helmet (7, 8), using steps and high-frequency oscillation. Subjects were instructed to abstain from alcohol and any drugs in the 24 h preceding the measurements. The labyrinthine defective (LD) patients consisted of several groups

Group A. Patients with, according to routine clinical testing (torsion swing and bithermal caloric stimulation) and surgical history, total unilateral vestibular loss ($n = 32$; 13 left-sided, 19 right-sided). All of these patients had undergone surgery for an acoustic neuroma (vestibular Schwannoma) several years beforehand (mean 4.5 years, ± 3.2 SD; range 0.5–11.5 years). The size of tumour was known for 27 patients: <2 cm ($n = 15$); 2–4 cm ($n = 8$); >4 cm ($n = 4$). All surgeries were, in principle, radical; only one patient showed a recurrence. Patients in this group had only few vestibular symptoms; i.e. they had feelings of disorientation or dizziness only after rapid head or body movements. Post-operative evaluation of eye movements (ENG) was performed in 27 of these patients; only 3 of them showed mild deficits of smooth pursuit, optokinetic nystagmus or saccades.

Group B. Patients with, according to routine clinical testing, partial unilateral vestibular loss ($n = 7$). The vestibular loss happened to be in all of these patients on the right side; five of these patients had an acoustic neuroma but had not yet been operated on. The case histories of the two other patients were a traumatic fall on the mastoid bone and lesion of the labyrinth after stapedectomy. No patient was on any medication.

Group C. Patients with, according to routine clinical testing, bilateral strong vestibular hyporeflexia ($n = 8$). Three of these patients had severe, sustained, invalidating vestibular symptoms in daily life, such as oscillopsia or severe unsteadiness when walking, necessitating the use of a wheelchair or walking frame. One of these three patients had been prescribed Gentamicin in the past for meningitis; the second had undergone middle ear surgery during which the left labyrinth was damaged (the right labyrinth also showed a severe hyporeflexia on routine clinical testing); the third had a bilateral hyporeflexia with unknown aetiology. The diagnosis of the five remaining, not severely invalidated, patients was in most cases bilateral Ménière; these patients were not on any medication at the time of the measurements. None of the patients of this group had a labyrinth predominance of more than 8%.

RESULTS

The normal subjects were quite accurate in aligning the luminous bar correctly with the true vertical. The frequency distribution (in 1° bins) of the mean settings of each subject is shown in Fig. 1; the inter-subject means and SDs are shown in Fig. 2. The overall mean of all normal subjects was $-0.14° \pm 1.11$ SD The majority of these was accurate within $\pm 1°$, and all were accurate within $\pm 2°$, in agreement with previous literature (see Introduction). It is important to note that the intra-subject SDs for the means of 16

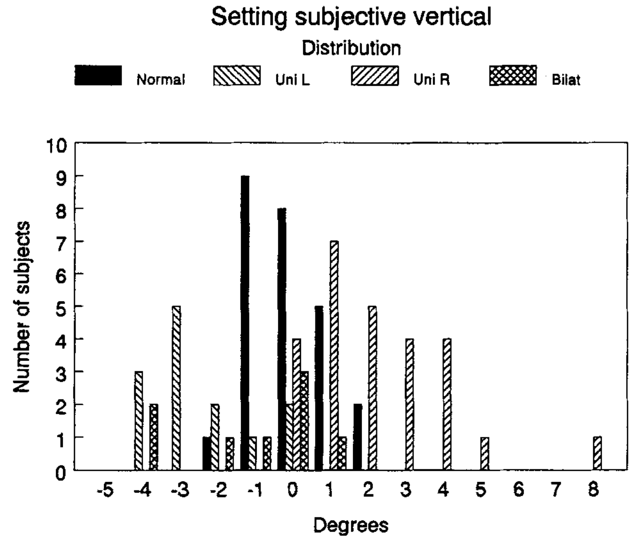

Fig. 1. Frequency distribution of mean settings of subjective vertical for normal subjects, patients with unilateral left-sided (Uni L) or right-sided (Uni R), or bilateral (Bilat) vestibular lesions. Bin width: 1°.

successive settings of individual normal subjects were much smaller (mean 0.20°) than the inter-subject SD of the means. Thus, successive settings by these subjects were quite reproducible, reflecting a high degree of confidence of the individual means, while the inter-subject distribution of the mean settings probably reflects small, but consistent individual biases. The settings by the subjects with long-standing unilateral vestibular lesions differed very systematically from those of the normal subjects. The results for the "partial" lesions followed the same pattern and were

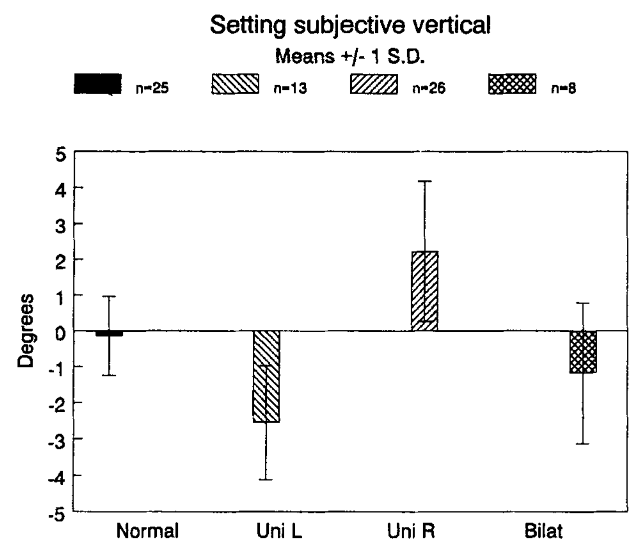

Fig. 2. Means and standard deviations of mean settings of the various subject groups shown in Fig. 1.

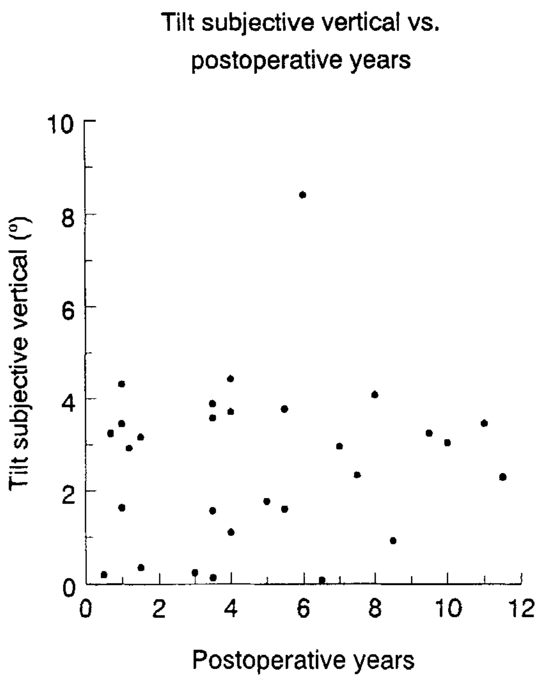

Fig. 3. Tilt (° absolute value) of the subjective vertical, plotted as a function of the number of post-operative years in 30 patients operated for a unilateral acoustic neuroma. The graph demonstrates the absence of any correlation.

statistically indistinguishable from the "total" group. Therefore, both groups were pooled in Figs 1 and 2 so that these represent 13 left-sided and 26 right-sided unilateral lesions. For the left-sided lesions the mean setting was $-2.55° \pm 1.57$ (SD); for the right-sided lesions the mean was $2.22° \pm 1.96$ (SD). Both means differed highly significantly from the normal mean ($p < 0.00001$; two-tailed t-test). Thus, the subjective vertical was tilted systematically towards the lesioned side. As shown in Fig. 1, there were overlaps between the "normal" and "unilateral lesion" distributions. However, 8 out of the 13 left-sided lesions and 10 out of the 26 right-sided lesions showed tilts of the subjective vertical exceeding 2°, the maximum value for normal subjects. The unilateral groups showed a larger inter-subject variability than the normal group; this may reflect different degrees of long-term adaptation to the defect, with part of the patients reaching the normal range. Also the intra-subject SD of the settings was somewhat larger than for the normal group: 0.33 vs 0.20°. Thus, reproducibility of successive settings was somewhat lower in the unilateral patients than in the normal patients.

To test whether the deviation of the subjective vertical still diminished systematically as a function of time in the period starting 6 months after the operation, we plotted the tilt of the subjective vertical (degrees absolute value) against the number of post-operative years for 30 subjects who had undergone unilateral surgery for an acoustic neuroma at least 6 months previously. As shown in Fig. 3, and verified by regression calculation, there was no correlation whatsoever between the residual tilt of the subjective vertical and post-operative time for the range 0.5–11.5 years.

In the group of eight bilaterally-deficient subjects, the mean setting was $-1.17° \pm 1.96$ (SD). This mean did not differ significantly from the normal distribution ($p = 0.19$; two-tailed t-test). The lack of a systematic deviation is also evident from Fig. 1, which shows that six out of the eight bilaterally-deficient subjects had means within the normal $0 \pm 2°$ range, and two were outliers at $-4°$. The mean intra-subject SD of successive settings in the "bilateral" group was 0.64°, which is twice as high as in the unilateral group. Thus, there was a clear trend for successive settings by a subject to be less reproducible, as the overall vestibular functionality was lower.

As the unilaterally operated group formed an essentially homogeneous group, which was deficient for both otolith and canal function, it was meaningful to search for correlations between the present findings on the subjective vertical, and two parameters relating to asymmetries of canal-function, determined for the same subjects in separate studies using an inertial torque helmet (7, 8). Unilateral vestibular lesions cause an asymmetry in the vestibulo-ocular response to head rotations: gain is much lower for rotation of the head towards the lesioned side than for rotation towards the intact side.

Firstly, this asymmetry is very evident when brief acceleration pulses are applied to the head, as has been first described by Halmagyi and colleagues (9). Such acceleration impulses (on the order of 1000°/sec^2) induce head velocities exceeding 50°/sec within 100 msec, a period short enough to preclude other than pure vestibular effects. We determined VOR-gain as eye velocity/head velocity at the moments at which head velocity reached 25 and 50/sec for rightward (GR) and leftward (GL) pulses. These velocities were reached about 25 and 50 msec after the onset of head movement; effects of latency were disregarded for the present purpose. To compare gain in the two directions, we define the gain index as: $(GR - GL)/(GR + GL)$. This index has a range between 1 (for $GL = 0$ and $GR = 1$) and -1 (for the opposite gain values). This index is plotted in Fig. 4 as a function of the angle of the subjective vertical, for head velocities of 25°/sec (upper panel) and 50°/sec (lower panel). Both graphs show a reasonable correlation between the two parameters of vestibular asymmetry. The oblique lines show linear regressions of the type $y = a + bx$, calculated separately for each graph. In

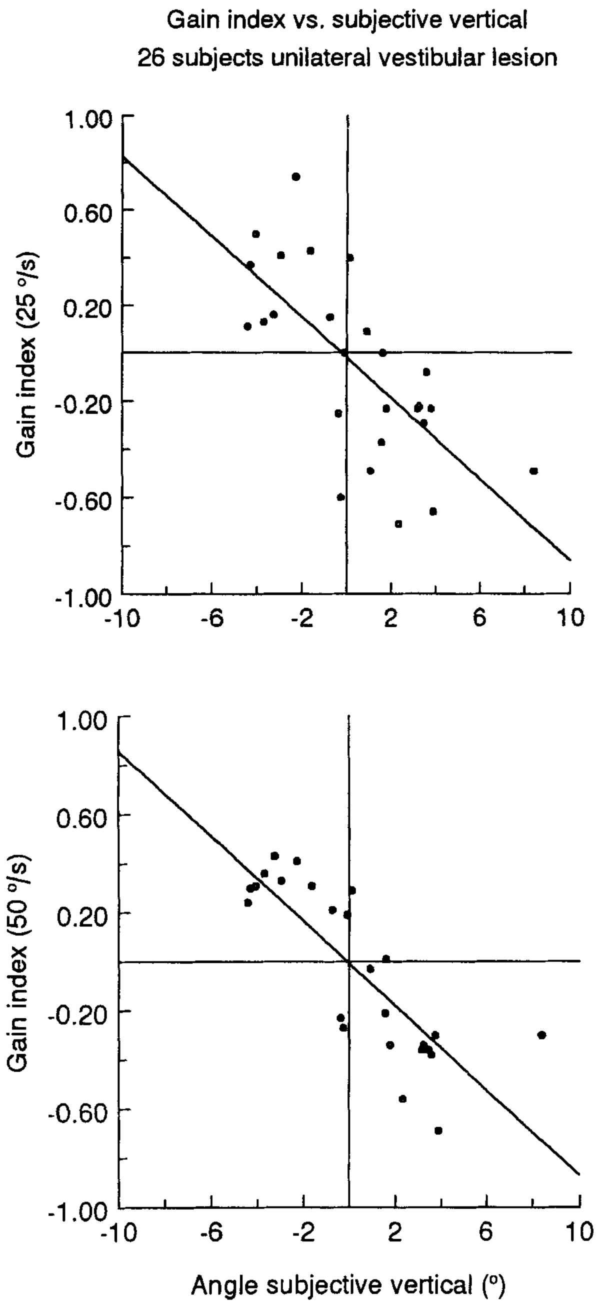

Fig. 4. Asymmetry of the canal-ocular VOR to head-steps, plotted as the gain-index (see text for definition), in relation to the angle of the subjective vertical, as measured in 26 subjects with a unilateral vestibular lesion. The gain-index was determined for the moments at which head velocity reached 25°/sec (upper panel) and 50°/sec (lower panel). The oblique lines show the calculated linear regressions (see text).

both cases, a (the intercept) was not significantly different from zero, and b (the slope; gain index/angle of subjective vertical) equalled approx. 0.085 (0.084 for 25°/sec and 0.086 for 50°/sec). The coefficients of determination (r^2) were 0.48 for 25°/sec and 0.67 for 50°/sec.

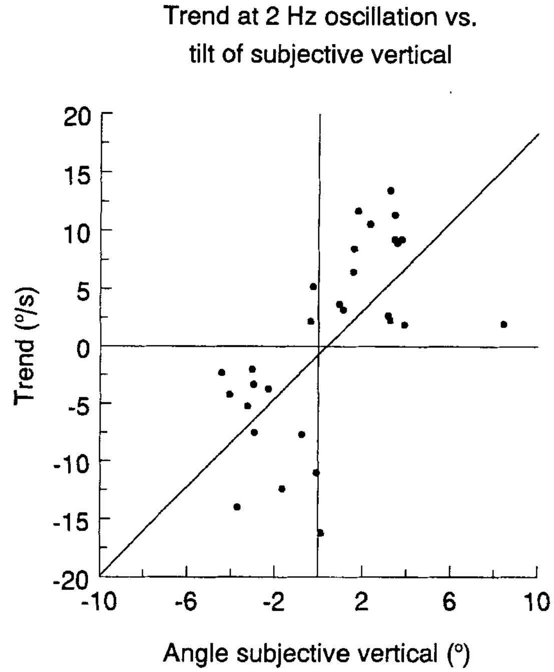

Fig. 5. Asymmetry of the canal-ocular VOR to 2 Hz head oscillation, plotted as the trend (see text for definition), in relation to the angle of the subjective vertical, as measured in 30 subjects with a unilateral vestibular lesion. The oblique line shows the calculated linear regression (see text).

Secondly, VOR-gain asymmetry is evident as a cumulative drift of the eye towards the lesioned side during continuous, sinusoidal oscillation of the head. Using our torque helmet (7, 8), we determined the maximum ocular drift velocity, trend, (in°/sec) during head oscillation at 2 Hz. In Fig. 5, trend has been plotted as a function of the angle of the subjective vertical for 30 unilaterally deficient subjects. Again, a positive correlation was present; the oblique line shows the calculated linear regression with a slope (trend/angle of subjective vertical) of 1.92 and intercept not significantly different from zero; r^2 was 0.44. These comparisons show that deviations of the subjective vertical, reflecting imbalance in otolith functions, are significantly, although not very tightly, correlated with parameters reflecting imbalance in canal function.

DISCUSSION

Our results confirm and extend recent investigations (3–5) that addressed the effect of unilateral vestibular loss on torsional eye position and the concomitant changes in the subjective vertical and horizontal. Normal healthy subjects are able to set a visible LED bar to the visually perceived gravitational horizontal (in°) with great accuracy and precision. Our values (mean setting $-0.14° \pm 1.11$) agree very well with those of Dai et al. (2) who found $-0.2° \pm 1.0$. Curthoys et al. (5) reported that patients who underwent a vestibular neurectomy showed very large deviations of the subjective horizontal towards the side of the lesion, 1 week after the operation (mean tilt toward the lesioned side $11.7° \pm 5.6$ SD $n = 23$, $p < 0.001$). These authors found also a high and statistically significant correlation ($r = 0.95$) between ocular torsion and perceived horizontal. In addition, there appeared to be a significant decrease in the tilt of the perceived horizontal from 1 week after operation ($11.8° \pm 8.1$, $n = 8$) to 16 weeks after the operation ($3.8° \pm 3.6$, $n = 8$), but the perceived horizontal at 16 weeks was still significantly different from these patients own pre-operative measures. This reduction in static ocular torsion and its accompanying decrease in the slope of the perceived horizontal seemed to be a manifestation of vestibular compensation.

Our present data show that such compensation may remain incomplete at times much longer after unilateral vestibular neurectomy. The lack of any correlation (Fig. 3) between tilt of the subjective vertical and postoperative time in the range 0.5–11.5 years suggests that compensation essentially reaches an asymptotic value within the first 6 months. Approximately half of our unilateral patients fell outside the maximum normal range ($0 \pm 2°$) even many years after the operation. As only 3 out of 25 normal subjects exceeded the $0 \pm 1°$ range (Fig. 1), the 2° criterion is actually quite strict and any values of the subjective vertical exceeding $\pm 1°$ should be considered as suspect for a vestibular asymmetry, while 2° and more may be considered as definitely abnormal. Figure 1 also shows that none of the unilateral cases showed a tilt of the subjective vertical towards the wrong (healthy) side. Thus, the side-specificity of the test appears to be excellent. Finally, there is a tendency for the reproducibility of successive settings to be lower in subjects that lack the function of one, and especially two labyrinths, compared to normal subjects. This may be expected, as subjects receiving less information are likely to increase the variable error in their settings.

In view of the fact that both the deviation of the subjective vertical and asymmetry in the gain of canal-ocular reflexes are highly side-specific, substantial correlations between these parameters are almost unavoidable in our unilateral patients, whose vestibular lesion was massive and indiscriminate for canals or otoliths. Figures 4 and 5 suggest that much of this correlation was indeed due to this sign-correlation; virtually all data points were located in the lower left and upper right quadrants. Apart from this sign effect, the numeric correlation between the magnitudes of both deviations was very weak. In fact, correlations between absolute values of VOR gain asymmetries and subjective vertical angles proved to be virtually absent. This suggests that, while the sign of vestibular asymmetries is specific for the side of the lesion, the magnitude of the canal- and otolith-related asymmetries varies independently, indicating separate compensation processes for canal and otolith subsystems.

We conclude that the measurement of the subjective vertical, which requires little time, effort and equipment, is a highly sensitive and specific test in assessing asymmetries in otolith function.

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Submitted March 13, 1996; accepted July 31, 1996

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