Asymmetry of the Subjective Visual Vertical in Patients With Unilateral Peripheral Vestibular Deficit

Souad Haijoub1 and Michel Lacour2,3

1Physiotherapist (Independent Researcher), Paris, France
2Aix-Marseille University, Marseille, France
3Neurosciences Department, Centre National de la Recherche Scientifique, Paris, France

Background and Objectives: Perception of verticality is clinically assessed using the subjective visual vertical (SVV), a test of the otolith system that consists of aligning a bar on the gravitational vertical in darkness. Patients with acute unilateral vestibulopathy (AUVP) show a systematic SVV bias toward the affected side, whichever the side of line orientation. Whether SVV estimates are symmetrical has not been investigated.

Subjects and Methods: This study included 10 patients with AUVP (vestibular neuritis) and 10 with BPPV (posterior semicircular canal). SVV measurements were made at two preset angles of line orientation (15° and 30°) toward the ipsilateral and contralateral sides, relative to the affected side.

Results: The results showed asymmetrical SVV estimates in the AUVP group, with significantly greater SVV errors for ipsilateral than contralateral line orientation, as well as for the preset angle of 30° compared to 15°. SVV estimates were significantly lower in patients with BPPV who also exhibited SVV asymmetry. SVV estimates remained unchanged just after the maneuver and were normalized some days later or after supplementary maneuvers.

Conclusions: SVV asymmetry should be routinely considered in the clinic. We recommend individually assessing ipsilateral and contralateral SVV and using at least two preset angles. This allows for a better assessment and diagnosis of otolith organ imbalance that can trigger chronic instability and dizziness. The contribution of neck afferents related to head position in space seems to be the main source of SVV asymmetry.

Keywords: Space perception; Otolithic test; Subjective visual vertical; Vestibular pathology; Acute unilateral vestibular hypofunction; Benign paroxismal positional vertigo.
ed by the central integration of otolith signals encoding head orientation in space with respect to gravity, body proprioception encoding body orientation in space, and neck proprioception encoding the relative position of head on body [11]. The Mittelstaedt’s model of upright orientation is based on the vectorial summation of the idirotropic vector (Z longitudinal axis) and the gravity vector (otolith organs: [12]). Bayesian models incorporate head orientation in space and eye in head position in a common reference frame to determine upright orientation. The cortical network involved in the perception of orientation lies at the temporo-parietal junction, in the posterior insular vestibular cortex [11,13-15]. Perception of upright is a high level neural process, alteration of which has been reported in various balance system disorders: Parkinson’s disease, vestibular neuritis, vestibular schwannoma, Pisa syndrome, aging, hydrocephalus, chronic neck pains, acute phase of Meniere’s disease, and multiple sclerosis [16].

The perception of orientation relative to gravity can be assessed by psychophysical tasks. The SVV test uses the adjustment protocol of a visual line in total darkness (or in the absence of visual orientation cues) to report perceived earth-vertical orientation. In healthy participants, the errors typically remain within 2° of earth vertical while SVV deviations to the affected side were found in patients with vestibular nerve section and vestibular neuritis [4,17-19]. The rate of SVV deviation progressively decreased over weeks or months due to the vestibular compensation process [18,20-23]. The SVV test is used in clinical routine to detect otolith imbalance [24-26]. Interestingly, patients with benign paroxismal positional vertigo (BPPV) also showed SVV deviation to the affected side [16,27] that could result from reduced otolith receptor function, as the consequence of the decreased density of the otoconia macular mass on the affected side [28].

Different parameters can affect the perception of verticality when the adjustment protocol of a visual line relative to the true gravitational vertical is used. An attraction bias to the line presentation side has been reported in both healthy subjects [3,29,30] and acute unilateral vestibulopathy (AUVP) patients [29,31], but the SVV estimate was not influenced by the deviation of the preset angle [30]. The viewing distance [32], the torsional position of the eyes [20], and the head/body lateral orientation [10] also influence the SVV estimate. The dynamic SVV test performed in the presence of a moving visual surround showed asymmetrical estimates of verticality in patients with unilateral peripheral vestibular loss, depending on the direction of the optokinetic flow [18]. To our knowledge, the static SVV estimates never reported such asymmetry. The reason is that clinicians always measured the mean bias estimated from data collected independently of the line orientation side and of the preset angle. The aim of the study was to determine whether AUVP and BPPV patients also exhibit asymmetrical values with the static SVV psychophysical test. This question has clinical and functional implications for otolith system diagnosis and vestibular rehabilitation therapy as well.

**Subjects and Methods**

**Subjects**

The study population comprised 10 patients with AUVP (vestibular neuritis) and 10 patients with BPPV.

Neurotological examination of the AUVP patients was done in one tertiary referral center (Centre d’Explorations Fonctionnelles Otoneurologiques, Paris: Dr. Michel Toupet) and in one ENT Department (Hôpital Lariboisière, Paris: Dr. Charlotte Hautefort). All AUVP patients exhibited the five main inclusion criteria [33]: acute onset of spinning vertigo, horizontal rotatory spontaneous nystagmus beating to the intact side, positive head impulse test (HIT) on the weaker side, nausea, and postural imbalance. Spontaneous nystagmus was observed with and without visual fixation using video nystagmography goggles (Framiral, Grasse, France). The neurotological examination included the caloric test, the HIT, and the head shaking test. The caloric test (30° and 44°) showed diminished responses on the hypofunction side. The pathological weaker side was also determined when using the angular vestibulocular reflex (aVOR) gain during passive video HIT (vHIT Ulmer; Synapsis, Marseille, France). The inclusion criteria were aVOR gain below 0.70 and when the presence of overt/covert saccades. The three pairs of semicircular canals were tested in order to document the vestibular deficit and to determine which part of the vestibular nerve (superior branch, inferior branch, or both branches) was impaired. Among the 10 AUVP patients, 9 had pathological HIT responses to horizontal canal test and vertical anterior canal test (superior branch) while one had pathological HIT responses to horizontal, anterior and posterior canal tests. The ocular and colic vestibular evoked myogenic potentials (oVEMPs and cVEMPs) were not systematically performed. One patient among the 5 patients tested showed absent oVEMPs and cVEMPs on the hypofunction side and normal responses on the healthy side while the remaining four patients showed abolished oVEMPs only on the hypofunction side. The patients’ history was done by one author (S.H.) who also preformed the SVV test.

The study population comprised 7 women and 3 men with a mean age of 51.5 years (range: 24–72 years), and included patients whose initial visit took place on average 8.9 days after symptoms onset (range: 5–21 days). The AUVP affected
SVV test

The SVV test was performed with the Framiral system (Grasse, France) which uses the adjustment protocol to assess the perceived vertical. The SVV protocol consisted of projecting a red fluorescent line, 30 cm long, on a white screen located 1.5 m in front of the patient. All patients were tested in standing position and wore goggles that limited their visual field to ±15° from central vision. This restriction device has the advantage of doing the SVV test in a non-dark room and of totally eliminating all visual orientation cues. The patients were asked to keep their head straight, to focus their attention to the line, and to adjust the line orientation to their own perception of verticality.

The line was pseudo-randomly oriented to the clockwise and the counterclockwise directions with preset angles of 15° and 30° from the vertical. The line was rotated with a velocity of 2° per second, and the patients were asked to indicate verbally when they perceived the line orientation as being vertical. To compare the patients with right and left unilateral vestibular deficits, and by convention, the SVV estimates were positively quoted when they were on the same side as the vestibular loss. Ten measurements were done, beginning with a first line deviation of 15° of preset angle on the left side (ipsilateral side for a patient with a left vestibular deficit, contralateral side for a patient with a right vestibular deficit) followed by the same deviation on the right side. A couple of line deviation to 30° of preset angle was then performed on both sides, and the same deviations with 15° and 30° were repeated on both sides. The patients were instructed to close their eyes between each trial, i.e., in the time period during which the software automatically changed the line orientation to the next position. The SVV estimates for the ipsilateral and contralateral line orientations were automatically recorded. The whole SVV test duration was around 5 minutes.

In the BPPV patients, the SVV was first evaluated before the diagnostic maneuver, and re-evaluated 15–30 minutes after the liberatory maneuver, and 3–4 days later for verification.

Statistical analysis

The mean value and the variance of the SVV estimates were calculated separately in the AUVP and the BPPV patients. The SVV values were first calculated for the whole of the data, independently of the line orientation side (ipsilateral vs. contralateral) and of the amplitude of the preset angle (15° and 30°). The SVV estimates were thereafter calculated as a function of the side (ipsilateral vs. contralateral) and amplitude (15° vs. 30°) of the preset angle.

Considering the small size of our two groups of patients and the distribution of the SVV in each group that did not follow
SVV Asymmetry in Vestibular Pathology

a normal Gaussian law, non-parametric tests were used for the statistical analysis of the data. Comparisons were done with the Mann–Whitney U test and the Wilcoxon signed rank test (StateView II; SAS Software Inc., Cary, NC, USA). Results were considered significant for \( p<0.05 \).

Results

SVV in AUVP

The SVV bias was always on the side of the weaker side in the AUVP patients. The mean SVV estimate was calculated by pooling all the data recorded with ipsilateral and contralateral line orientations with respect to the hypofunction side, at the two preset angles of 15° and 30°. The mean bias in the AUVP group was 7.91°±4.2° (Fig. 1). Clinicians refer to this mean value to determine whether the patient's SVV was pathological or not, i.e., within or outside the normality range (±2°).

When the data were pooled as a function of the side of line orientation (ipsilateral vs. contralateral), the results showed significantly different SVV estimates. The mean SVV bias was higher with ipsilateral line orientations (9.49°±3.9°) compared to contralateral line orientations (7.03°±3.8°; \( p<0.01 \)), and higher compared to the mean bias including the ipsilateral and contralateral values (\( p<0.05 \)).

Table 2 illustrates the raw data as the mean (±standard deviation) of the SVV estimates for the ipsilateral, contralateral, and ipsilateral+contralateral 15° and 30° preset angles.

Supplementary data recorded in three chronic AUVP patients still complaining of instability and dizziness 1 year after onset of their symptoms also showed SVV asymmetrical values (Table 2). At 30° of preset angle, for instance, the mean

![Fig. 1. Subjective visual vertical (SVV) depends on the side of line orientation in the AUVP patients. The mean SVV estimates (±SD) are plotted as a function of the side of the line orientation with respect to the affected side: ipsilateral side and contralateral side. The assessment of the SVV in clinical routine pools together all the ipsilateral and contralateral measurements is shown for comparison. The horizontal grey area represents the normal range of the SVV in healthy subjects. Asymmetrical SVV values are seen with significantly higher estimates for line orientation to the ipsilateral side. *\( p<0.05 \); **\( p<0.01 \). AUVP, acute unilateral vestibulopathy.]

![Fig. 2. Subjective visual vertical (SVV) depends on the side and preset angle of line orientation in the AUVP patients. The boxplots show the median with the 1st and 3rd quartiles, and whiskers indicate the minimum and maximum SVV values. Significantly higher SVV values were recorded for 30° deviation of the line to the ipsilateral side compared to the contralateral side and the ipsilateral line deviation at 15°. The horizontal grey area indicates the non-pathological SVV range. **\( p<0.01 \). AUVP, acute unilateral vestibulopathy.]

Fig. 2. Subjective visual vertical (SVV) depends on the side and preset angle of line orientation in the AUVP patients. The boxplots show the median with the 1st and 3rd quartiles, and whiskers indicate the minimum and maximum SVV values. Significantly higher SVV values were recorded for 30° deviation of the line to the ipsilateral side compared to the contralateral side and the ipsilateral line deviation at 15°. The horizontal grey area indicates the non-pathological SVV range. **\( p<0.01 \). AUVP, acute unilateral vestibulopathy.
values were 4.2°±0.8° and 2.2°±1.1° for ipsilateral and contralateral line orientations, respectively (p<0.001). These values were significantly lower than those recorded acutely after AUVP (p<0.001).

SVV in BPPV patients

The data collected in the BPPV patients with altered SVV showed a pattern of SVV deviations very similar to that observed in the AUVP patients. The main difference was the smaller SVV bias found in the VPPB BPPV patients, comparable to those found in chronic AUVP patients.

Significantly higher mean SVV errors were observed with 30° of preset angle compared to 15° of preset angle for ipsilateral line orientations (5.8°±2.8° vs. 4.0°±2.2°; p<0.05), and between 30° of preset angle to the ipsilateral side compared to the contralateral side (5.8°±2.8° vs. 3.4°±2.3°; p<0.01) (Fig. 3).

After the successful CRM, patients were asked to rest a few minutes before they were subjected to a second SVV test. The SVV bias remained was generally unchanged during this second test but the standard deviation of the estimates was increased, suggesting a less accurate precision of the perceived vertical after the CRM. The mean values recorded at 15° of preset angle on the ipsilateral side were 4.0°±2.2° before CRM and 4.16±3.2 after CRM, 5.8°±2.8° and 5.2°±3.9° for the pre- and post-CRMs, respectively, at 30° of preset angle on the same side. The SVV estimates returning within the ±2° normal range were found later on, when the patients were re-subjected to a control SVV test. Seven among the ten BPPV patients showed non-pathological SVV values when retested 3 days after the first CRM whereas supplementary maneuvers were needed in the remaining three patients to regain SVV values in the non-pathological range (2 CRMs in one patient and 3 CRMs in two patients).

Fig. 4 illustrates the mean SVV estimates from a 49-year-old man with a BPPV of the right posterior semicircular canal who was examined before and after the first successful CRM, and 3 days after. The other BPPV patients showed similar results after the first maneuver but they needed 2 or 3 supplementary maneuvers to regain non-pathological SVV values.

Table 2. Modifications of the SVV in the acute and chronic unilateral vestibular patients

<table>
<thead>
<tr>
<th>Side and preset angle</th>
<th>SVV (°)</th>
<th>Acute unilateral vestibular patients (n=10)</th>
<th>Chronic unilateral vestibular patients (n=3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral 15°</td>
<td>8.3±2.9</td>
<td>3.1±1.5</td>
<td></td>
</tr>
<tr>
<td>Ipsilateral 30°</td>
<td>11.1±4.1*</td>
<td>4.2±0.8†</td>
<td></td>
</tr>
<tr>
<td>Contralateral 15°</td>
<td>7.0±3.5</td>
<td></td>
<td>2.2±1.4</td>
</tr>
<tr>
<td>Contralateral 30°</td>
<td>7.4±4.0</td>
<td></td>
<td>2.2±1.1</td>
</tr>
<tr>
<td>Ipsilateral+contralateral 15°</td>
<td>7.7±3.4</td>
<td>2.5±1.4</td>
<td></td>
</tr>
<tr>
<td>Ipsilateral+contralateral 30°</td>
<td>9.4±4.5</td>
<td>3.2±1.3</td>
<td></td>
</tr>
</tbody>
</table>

Values are presented as mean ± standard deviation. *p=0.006 (vs. ipsilateral 15°) and p=0.003 (vs. contralateral 30°); †p=0.05 (vs. ipsilateral 15°) and p=0.001 (vs. contralateral 30°). SVV, subjective visual vertical

Discussion

Taken together, the study showed that AUVP patients 1) had systematic SVV deviations to the hypofunction side, a result in agreement with the literature, and 2) showed asymmetrical SVV deviations depending on both the side of the line orientation and the preset angle deviation, a new and original result. Our data extends the SVV asymmetry to our BPPV patients who exhibited deviations to their affected side. What is the cause of the static SVV asymmetry, and what are
SVV Asymmetry in Vestibular Pathology

Fig. 4. Effect of the CRM on the SVV estimates. Data was recorded in the case of a 49-year-old man with BPPV of the right semicircular posterior canal. The SVV was measured during the first examination of the patient, before and after the CRM (Epley’s maneuver). The SVV estimates were not improved despite the successful maneuver when the second test was done a short time after (30–60 minutes), but the SVV was normalized 3 days later in this patient. Data was recorded for ipsilateral and contralateral 15° line orientations. The horizontal grey area indicates the non-pathological SVV range. ***p<0.001. CRM, canalith repositioning maneuver; SVV, subjective visual vertical; BPPV, benign paroxysmal positional vertigo.

The dynamic SVV recorded with a moving visual background elicits an optokinetic stimulation with long lasting asymmetrical effects after unilateral vestibular loss [18]. The optokinetic-induced circular vection produced lower dynam-ic SVV changes when the optokinetic flow was directed to the intact side compared to optokinetic flow directed to the diseased side. The low rotational velocity of the visual line (2°/s) used in our protocol could induce similar effects. The visual (entrainment) effect [40] consists of the entrainment of the ocular torsion by a visual line rotating around the line of sight in the direction of the line rotation. The entrainment of the ocular torsion is low in gain, but repeatable and reliable among subjects, with however considerable individual differences between subjects. If present, a counterclockwise line rotation for a preset angle on the right hypofunction side, for example, should produce an ocular torsion entrainment in the opposite direction with respect to the cyclotorsion resulting from the otolith imbalance. And the SVV bias should be reduced (and vice versa for a clockwise rotation and preset angle oriented on the intact side). Our data showed opposite results: the static SVV was significantly increased with preset angles on the weaker side and the line moving to the intact side. This second argument still does not support the contribution of eye position signals to the SVV asymmetry.

Figures of merit and head orientation in space

The ocular tilt reaction described in AUVP patients combines head tilt, ocular cyclotorsion, skew deviation and tilt of the SVV toward the affected side, and result from the imbalance of the otolith organs [20,24]. According to Wade and Curthoys [34], the SVV bias would be caused by the ocular torsional position. Their conclusion is based on the close correlation (r=0.95) between the amplitude of the cyclotorsion and the SVV bias. We also reported such a correlation in Meniere’s disease patients after a curative unilateral vestibular neurectomy [35]. There is however no relationship of proportionality between these two parameters, and lesions of the thalamo-cortical vestibular pathways [13] and cortical lesions after stroke [36,37] induce SVV deviations without cyclotorsion, recent works clearly demonstrated the lack of causal relationship between SVV and cyclotorsion [11,38,39]. We did not record the torsional position of the AUVP patients in the present study. However, our BPPV patients did not show any cyclotorsion and they exhibited similar SVV biases, a result that does not support the eye cyclotorsion hypothesis.

The dynamic SVV recorded with a moving visual background elicits an optokinetic stimulation with long lasting asymmetrical effects after unilateral vestibular loss [18]. The optokinetic-induced circular vection produced lower dynam-
vestibular dominance at the cortical level is another hypothesis to account for the SVV asymmetry.

Functional implications for vestibular rehabilitation

A new and original result in the present study is the asymmetry of the static SVV. Most of the SVV estimates are based on software that calculates the mean SVV value from one or two preset angles of a line pseudo-randomly oriented to either side. In the absence of oVEMPs and cVEMPs, clinicians refer to this mean value to determine an otolith imbalance in AUVP patients. They base their diagnostic on an SVV estimate that considers neither the amplitude of the preset angle nor the side to which the line is oriented. In such conditions, the mean estimate can be within the normal range (<2°) in spite of pathological SVV values on one side. BPPV patients with pathological SVV estimates do not constitute the majority of the patients treated with the CRM. The asymmetrical SVV values found in our small BPPV population point to an otolith system imbalance that could not be revealed with SVV estimates pooling all the data.

Of key significance for patients, our data show the potential of this new assessment of the SVV taking into account the side and the amplitude of the preset angle. This method avoids underestimation of the otolith dysfunction and its consequences in terms of instability, impairment of the patient’s quality of life, and possible development of chronic dizziness. Pathologies such as BPPV, AUVP, and vestibular migraine have been reported as prior triggers to the onset of persistent postural perceptual dizziness (PPPDP: [44,45]). It is therefore recommended to abandon the mean SVV value and to determine the SVV errors for each side, ipsilaterally and contralaterally with respect to the disease side, and to use at least two different preset angles in the low range 10°–60° for which the E-effect can be modified after unilateral vestibular loss.

Limitations of the study

Our results will have to be confirmed on a broader sample of AUVP and BPPV patients. Unpublished data from 42 AUVP patients included in another paper [23] confirm however the SVV asymmetry found in the present study. All the BPPV patients do not show SVV deviations to their diseased side, and it is necessary to increase the size of this population not only to confirm our data, but also to determine when the patients should be retested and how many repositioning maneuvers have to be done to normalize the SVV. A better understanding of the mechanisms responsible for the SVV asymmetry should include measurements of the eye cyclotorsion, eye countercyclotorsion, and head position in space to determine the contribution of eye position signals and neck afferents.

Conflicts of Interest

The authors have no financial conflicts of interest.

Author Contributions


Acknowledgments

The authors wish to thank Dr. Charlotte Hautefort and Dr. Michel Toupet for the neurotological examination of the patients, and Mr. Steve Edmondson for correcting the English language.

REFERENCES

SVV Asymmetry in Vestibular Pathology