ORIGINAL ARTICLE

Subjective Visual Vertical and Horizontal

Effect of the Preset Angle

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Objectives: (1) To study the subjective visual vertical (SVV) and subjective visual horizontal (SVH) in patients with long-standing unilateral peripheral vestibular dysfunction (PVD) and unilateral Ménière's disease (MD) compared with controls. (2) To study the relationship between the direction of deviation of the linear marker (preset angle) and measures of SVV and SVH values.

Design: Prospective case-control study.

Setting: Outpatient clinic in a tertiary neuro-otology department.

Patients: Seventeen healthy volunteers (mean age, 35.5 years), 9 patients with PVD (mean age, 43.1 years), and 10 patients with MD (mean age, 50.7 years) were included in the analysis.

Interventions: All subjects had a detailed neuro-otological evaluation. Twelve replicate readings of SVV and SVH were taken for each subject, with random preset angles, 6 in the clockwise and 6 in the counterclockwise direction.

Main Outcome Measure: The SVV and SVH values were correlated with clinical features and the direction of the preset angle.

Results: The 2 subjects with PVD who had abnormal mean SVV and SVH values had symptoms of dysequilibrium and otolithic involvement. The 5 patients in the MD group who had abnormal mean SVV and SVH values had either recent acute vertiginous attacks or total canal paresis on the affected side. A previously unreported finding, to our knowledge, is that the SVV value depends on the direction of the preset angle in all subject groups, more so in the PVD and MD groups compared with controls. The SVV is inclined toward the direction of the preset angle. A weaker relation is seen between the SVH and preset angle.

Conclusion: The preset angle should be considered when comparing SVV and SVH values.

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THE SUBJECTIVE VISUAL VERtical (SVV) and subjective visual horizontal (SVH) values evaluate function of the otolithic pathways. A recent study[1] has indicated that these values may also be influenced by input from the semicircular canals. The SVV and SVH deviate toward the side of the lesion in acute unilateral peripheral vestibular lesions, such as vestibular neuritis, but normalize over a period of weeks to years.[2-4] One of the main limitations of these tests is their low sensitivity in chronic peripheral vestibular diseases. The mean SVV and mean SVH values are computed by averaging multiple readings of SVV and SVH values, respectively, for different clockwise (CW) and counterclockwise (CCW) start positions of the linear marker, but there is no agreed-on protocol for setting the start position of the linear marker.[5,6]

A previous study[7] indicated that when the head is inclined in the roll plane, SVV values could be influenced by the start position of the linear marker. Other studies[8,9] noted no significant difference between SVV measures performed with an initial right or left tilt of the marker, but the possibility of a relationship between the preset angle and the SVV value has yet to be explored. Because the SVV and SVH values are often compared in longitudinal studies at different times in the same subject and in cross-sectional studies between subjects and controls, it is important to question whether these values could be biased by the preset angle. Furthermore, it will be useful to explore the possibility of determining an optimal protocol to measure SVV and SVH with respect to the preset angle, to optimize their sensitivity and specificity. The present study aims to determine values of SVV and SVH

in patients with unilateral peripheral vestibular dysfunction (PVD) and unilateral Ménière's disease (MD) and correlate them with clinical features and with the start direction of the linear marker. The pilot study was designed to focus the direction of further research, which is aimed toward producing an optimal protocol for measuring SVV and SVH values.

METHODS

CRITERIA

This prospective study was based at a tertiary level neuro-otology department. Patients with PVD and definite MD attending outpatient clinics were considered for enrollment. Diagnosis of definite MD was based on the criteria of the committee of hearing and equilibrium.[10] Diagnosis of PVD was based on the following criteria:

1. History of sudden vertigo resolving within days to weeks.
2. Audiometric tests showing normal hearing and middle ear function.
3. Unilateral canal paresis on standard Fitzgerald-Hallpike caloric testing[11] as measured by the duration parameter using the formula of Jongkees et al[12] of more than 8% in the absence of optic fixation.[13]
4. Direct current electronystagmography (ENG) showing the presence of unidirectional spontaneous nystagmus on gaze testing with enhancement of the response on removal of optic fixation.

Patients were required to meet at least criteria 1 to 3 to be included in this group.

Control subjects with no history of hearing loss, imbalance, or tinnitus were recruited from hospital staff. Subjects were excluded if they had used vestibular sedatives or alcohol in the previous 24 hours, or if there was a history of neurological or psychiatric disorder, uncorrected refractive errors, ocular palsy, or squint. Ethics approval for the study was given by the National Hospital for Neurology and Neurosurgery and the Institute of Neurology joint research ethics committee. Informed consent was received prior to participation in the study. Details of the clinical neuro-otological evaluation and audiovestibular tests results, including pure-tone audiogram using the recommended procedure of the British Society of Audiology,[14] tympanometry using a 226-Hz probe, stapedial reflex thresholds (ipsilateral and contralateral recordings at 0.5, 1, 2, and 4 kHz), direct current ENG and bithermal caloric testing using the Fitzgerald-Hallpike[11] technique and the duration criteria for nystagmus[15] were recorded. An Easygraph recorder (Gould Instruments, Hainault, England) recorded the direct current ENG traces, which included recording of gaze with and without optic fixation; smooth pursuit at 0.2, 0.3, and 0.4 Hz across 30° right and left of the midposition of gaze; optokinetic responses to a full-field striped curtain rotated at 40°/s; sinusoidal rotation at 0.2 Hz; and impulsive step acceleration/deceleration of 140°/s^2 and a fixed chair velocity of 60°/s. Canal paresis and directional preponderance were calculated using the duration criteria of nystagmus in the formula of Jongkees et al.[12] A value of more than 8% was taken as indicating clinically significant canal paresis.[13]

The SVV and SVH were recorded in a totally darkened room in the absence of visual clues. The equipment included a liquid crystal display unit, laser projector, and a remote control (Micromedical Technologies, Chatham, Illinois). The subject was seated upright in a chair, and head supports were used to maintain a vertical position of the head. The laser projector projected a luminous linear marker 1.6 m long and 2 mm wide on the facing wall (1.6 m distant) at a random angle (20°-45°) to the gravitational vertical or horizontal. Participants were allowed to keep their eyes open during the offset of the linear marker and were asked to align the marker to the gravitational vertical or horizontal using a remote control. The SVV and SVH readings were taken from the liquid crystal display unit for 6 CW and 6 CCW start positions of the linear marker, giving a total of 12 values for each task. Familiarization trials were allowed, and no time limits were set for the adjustments.

STATISICAL ANALYSIS

The true gravitational SVV and SVH were taken as zero degrees. Values of SVV and SVH in the CW direction from zero were designated as positive and those in the CCW direction as negative. The mean SVV was defined as the mean of 12 SVV readings (in degrees) from the gravitational vertical. The mean SVH was similarly defined. The start position of the linear marker was denoted by the term *preset angle*. Values of SVV and SVH were analyzed for correlation with audiovestibular findings and with the direction of their respective preset angle. The mean SVV and mean SVH were also computed separately for positive and negative preset angles in each subject group. The mean SVV-CW was defined as the mean value of SVV for the 6 CW or positive positions of the preset angle. The mean SVV-CCW was similarly defined as the mean value of SVV for the 6 CCW or negative positions of the preset angle. The reference ranges of the SVV-CW and SVV-CCW were computed using their (mean ± 2 SDs) values in the control group. The term *unidirectional SVV* (uSVV) was derived from a combination of the results of both SVV-CW and SVV-CCW when these values were computed separately. The term *uSVV* denotes a categorical (binary) variable, with 2 possible values: normal and abnormal. The uSVV value was considered normal when both the SVV-CW and SVV-CCW values were normal, and it was considered abnormal when either the SVV-CW or SVV-CCW value was abnormal. The uSVV was used to calculate the sensitivity and specificity values. The terms *mean SVH- CW*, *mean SVH-CCW*, and *unidirectional SVH* (uSVH) were similarly defined. Sensitivity and specificity of SVV, SVH, uSVV, and uSVH were computed by combining the PVD and MD groups together.

$$\text{Sensitivity of SVV} = \frac{\text{Total No. of Subjects With Abnormal Mean SVV in (PVD+MD) Groups}}{\text{Total No. of Subjects in (PVD+MD) Groups}},$$

$$\text{Specificity of SVV} = \frac{\text{No. of Control Subjects With Normal Mean SVV}}{\text{No. of Subjects in the Control Group}},$$

$$\text{Sensitivity of uSVV} = \frac{\text{Total No. of Subjects With Either Abnormal SVV-CW or SVV-CCW in (PVD+MD) Groups}}{\text{Total No. of Subjects in (PVD+MD) Groups}},$$

$$\text{Specificity of uSVV} = \frac{\text{No. of Control Subjects With Normal SVV-CW and SVV-CCW}}{\text{No. of Subjects in the Control Group}}.$$

The effect of direction of the preset angle on the SVV and SVH was assessed by fitting a random effects analy-

Table 1. SVV and SVH Values in Controls and Patient Groups[a]

Type of Value	Controls	PVD Group	MD Group
SVV	0.16 (0.96) [−1.97 to 1.67]	0.14 (1.65) [−1.99 to 2.78]	−0.45 (2.32) [−4.15 to 3.43]
SVH	−0.17 (1.44) [−3.15 to 2.29]	−0.46 (2.63) [−5.13 to 2.58]	−0.98 (2.66) [−3.97 to 3.50]

Abbreviations: MD, Ménière's disease; PVD, peripheral vestibular dysfunction; SVH, subjective visual horizontal; SVV, subjective visual vertical.
[a]Data are given as mean (SD) [range].

sis of variance using Stata statistical software (release 8.0; StataCorp, College Station, Texas). This model takes into account individual replicate readings within subjects and the random variability among subjects. A group factor was included in the model, along with interaction terms between group and preset angle, to investigate whether the effect of the preset angle on SVV and SVH varied between groups. For the purpose of analysis, the preset angle was considered as a categorical (binary) variable with 2 possible values: positive and negative. In the model, the difference in the means of SVV-CW and SVV-CCW was used to assess the relationship between the preset angle and the SVV or SVH. This difference was designated as the *angle effect*. The effect of the preset angle on the SVV and SVH was also assessed by using a frequency table, comparing individual SVV and SVH values with their respective preset angles. Values of SVV and SVH corresponding to zero were excluded. The χ^2 test was used to ascertain significance. Solely for the purpose of computing a mean SVV and mean SVH value, patients with PVD and MD were evaluated considering their right ear as affected to prevent SVV and SVH values of right- and left-sided lesions from negating each other during averaging. Thus, a deviation of SVV and SVH toward the ear with a lesion was designated as positive and away from that ear as negative. In all other analyses (ie, fitted model, calculation of SVV-CW, SVV-CCW, and corresponding SVH values), individual values of SVV and SVH and the preset angle were denoted according to the direction of deviation (CW deviations were denoted as positive, and CCW deviations were denoted as negative).

RESULTS

Of the 46 subjects recruited for the study, 36 satisfied the eligibility criteria and were included in the analysis (17 healthy controls, 9 patients with PVD, and 10 patients with definite MD). The mean ages (range) of the groups were as follows: control group, 35.5 years (20-59 years); PVD group, 43.1 years (29-63 years); and MD group, 50.7 years (35-77 years). The ratio of men to women in the control group was 12:5; in the PVD group, 0:9; and in the MD group, 3:7. Findings from audiovestibular tests were in the reference range for all controls. All patients with PVD had longstanding dizziness (mean duration of dizziness, 6.1 years [range, 1.0-21.0 years]) with partly compensated symptoms and a canal paresis of 9% to 60%. In the MD group, duration of symptoms ranged from 13 months to 17 years (mean, 7.8 years) and the number of acute MD episodes in the preceding year varied from none to more than 10. All the affected ears in the MD group had a moderate to profound sensorineural hearing loss.

MEAN SVV AND SVH VALUES IN CONTROLS AND PATIENT GROUPS

The mean SVV and mean SVH values in the 3 groups are shown in **Table 1**, and the corresponding scatterplot is depicted in the **Figure**. Based on the mean values in controls (mean ± 2 SDs), a mean SVV deviation of less than −1.8° or more than 2.1° and a mean SVH deviation of less than −3.0° or more than 2.7° was considered abnormal in this study. Because the mean age in the control group was lower than those in both the patient groups, the mean SVV and mean SVH were computed separately for control subjects 40 years or older (n=5) and those younger than 40 years (n=12). The mean SVV value for subjects 40 years or older was −0.03° (0.44°) and that for those younger than 40 years was 0.24° (1.12°); the difference in values was not statistically significant ($P=.56$). Similarly, the mean SVH value for subjects 40 years or older was −0.51° (1.49°) and that for those younger than 40 years was −0.04° (1.47°); the difference in values was not statistically significant ($P=.61$). Similarly, there was no significant difference between men (n=12) and women (n=5) in the values of mean SVV and mean SVH ($P=.14$ for mean SVV and $P=.51$ for mean SVH). One subject in the control group had a value of mean SVV outside the reference range (−1.97°), and another control subject had a value of mean SVH outside the reference range (−3.15°).

In the PVD group, 1 subject of 9 had a mean abnormal SVV deviation *toward* the direction of her 9% canal paresis (2.78°). Her mean SVH value was within reference range, and she complained of "tilt illusion." She described brief episodes of the visual surround appearing as "tilted" in the direction opposite to her canal paresis, causing imbalance. Another subject showed an abnormal mean SVH deviation *opposite* to the direction of her 13% left canal paresis (−5.13°), but her mean SVV value was within reference range. She had a history of drop attacks and demonstrated a right directional preponderance and right vestibular nystagmus on ENG, in the direction of the SVH deviation. Thus, a total of 2 of 9 subjects in the PVD group (22%) in this study showed an abnormal mean SVV or mean SVH value. In the MD group, 3 of 10 subjects (30%) had abnormal values of both SVV and SVH, 1 subject had an abnormal value of SVV, and 1 subject had an abnormal value of SVH. A total of 5 of 10 subjects (50%) had an abnormal mean SVV or mean SVH value. The deviations were ipsiversive or contra-

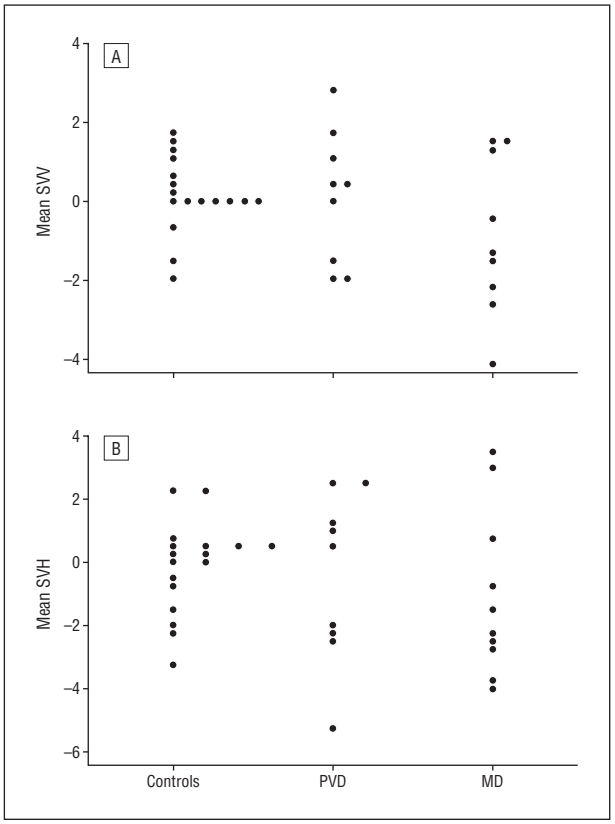

Figure. Scatterplots of the mean values in the 3 subject groups (controls, peripheral vestibular dysfunction [PVD], and Ménière's disease [MD]). A, The mean subjective visual vertical (SVV) values. B, The mean subjective visual horizontal (SHV) values.

versive, and all of these 5 subjects either had a 100% canal paresis on the affected side or had experienced an acute MD episode in the preceding 3 weeks.

CORRELATION OF SVV AND SVH VALUES WITH CLINICAL FEATURES

There was no significant correlation between the degree of canal paresis and mean SVV or mean SVH value in either the PVD or the MD groups (Pearson correlation coefficient; $P = .80$ for SVV and $P = .60$ for SVH in the PVD group; $P = .15$ for SVV and $P = .91$ for SVH in the MD group). Similarly, there was no significant correlation between the duration of dizziness and mean SVV or mean SVH value in both PVD and MD groups (Pearson correlation coefficient; $P = .80$ for SVV and $P = .60$ for SVH in the PVD group; $P = .08$ for SVV and $P = .20$ for SVH in the MD group). There was also no significant correlation between the puretone mean threshold in the affected ear and the mean SVV or mean SVH value in the MD group (Pearson correlation coefficient; $P = .42$ for SVV and $P = .30$ for SVH).

SVV VALUE VS PRESET ANGLE

Table 2 shows the fitted model of the effect of the direction of the preset angle and subject group on the SVV measurement and the significance values. A group factor was included in the model to investigate whether the effect of the preset angle on SVV varied between the subject groups. The model indicates a significant difference between the mean SVV-CW and the mean SVV-CCW (the angle effect) value in the control group (difference in means, 1.30; $P < .001$). The angle effect in the PVD group is significantly greater than that of the control group (difference in angle effect between the PVD and control groups, 2.53; $P < .001$). A similar observation is made for the control group vs MD group (difference in angle effect between the MD and control groups, 3.58; $P < .001$). **Table 3** depicts the frequency table for positive and negative SVV values compared with their respective positive and negative preset angles in the 3 subject groups. If the preset angle is positive, then the SVV value is more likely to be positive, and if the preset angle is negative, then the SVV value is more likely to be negative, in all 3 subject groups; that is, the SVV value is inclined toward the direction of the preset angle (χ^2 test; $P < .001$ in all 3 groups).

Table 2. Fitted Model of the Relationship Between the Direction of PA and the SVV Value for the 3 Subject Groups[a]

SVV Value vs PA	Angle Effect for SVV Value	SE (95% CI)
Control group	1.30	0.27 (0.78-1.82)
PVD group vs control group	2.53	0.45 (1.64-3.42)
MD group vs control group	3.58	0.44 (2.72-4.43)

Abbreviations: CI, confidence interval; MD, Ménière's disease; PA, preset angle; PVD, peripheral vestibular dysfunction; SVV, subjective visual vertical.
[a] The model indicates the comparison between controls vs PVD group and between controls vs MD group. $P < .001$ for all comparisons.

SVH VALUE VS PRESET ANGLE

Table 4 shows the fitted model of the effect of the direction of the preset angle and subject group on the SVH value, and the significance values. A group factor was included in the model to investigate whether the effect of the preset angle on the SVH value varied between groups. In the control subjects, there was a significant difference between the mean SVH-CW and mean SVH-CCW value; that is, the angle effect was significant (difference in means, –0.92; $P < .001$). The difference in angle effect between the PVD group and the control group was not significant (difference in angle effect between the PVD and control groups, –0.57; $P = .17$). The angle effect in the MD group was significantly greater than that in the control group (difference in angle effect between the MD and control groups, 1.48; $P < .001$). **Table 5** demonstrates the frequency table for positive and negative SVH values compared with their respective positive and negative preset angles in the 3 subject groups. A significant relationship was seen only in the control group, wherein the positive preset angle was likely to be associated with a negative SVH value and vice versa ($P < .001$).

uSVV AND uSVH VALUES

The mean values of SVV-CW and SVV-CCW and the respective values for SVH in the control group are shown

Table 3. Relationship Between SVV Value and PA in the 3 Subject Groups[a]

	Controls (n = 17)			PVD Group (n = 9)			MD Group (n = 10)		
	PA for SVV Value			PA for SVV Value			PA for SVV Value		
SVV Value	Positive	Negative	Total	Positive	Negative	Total	Positive	Negative	Total
Positive	73	33	**106**	41	13	**54**	46	18	**64**
Negative	27	66	**93**	12	40	**52**	13	40	**53**
Zero	2	3	**5**	1	1	**2**	1	2	**3**
Total	**102**	**102**	**204**	**54**	**54**	**108**	**60**	**60**	**120**

Abbreviations: MD, Ménière's disease; PA, preset angle; PVD, peripheral vestibular dysfunction; SVV, subjective visual vertical.
[a]The frequency categorization table indicates the number of patients with positive and negative SVV values for their respective positive and negative PAs. In each of the 3 subject groups, the SVV is more likely to be positive when the PA is positive and more likely to be negative when the PA is negative. The SVV values corresponding to zero have been excluded from the analysis. The *P* value is significant in each group ($P < .001$).

Table 4. Fitted Model of the Relationship Between the Direction of the PA to the SVH Value[a]

SVH vs PA	Angle Effect for SVH Value	SE (95% CI)	*P* Value
Control group	–0.92	0.24 (–1.39 to –0.44)	<.001
PVD group vs control group	–0.57	0.41 (–1.38 to 0.24)	.17
MD group vs control group	1.48	0.40 (0.70 to 2.26)	<.001

Abbreviations: CI, confidence interval; MD, Ménière's disease; PA, preset angle; PVD, peripheral vestibular dysfunction; SVH, subjective visual horizontal.
[a]The model includes a group factor and indicates the comparison between controls vs the PVD group and between controls vs the MD group.

in **Table 6**. Values outside the (mean ± 2 SDs) range were considered abnormal. There was no significant difference between the mean values of the CW and CCW preset angles for SVV and for SVH ($P = .50$ for both). The number of subjects with abnormal values for SVV-CW, SVV-CCW, uSVV, and SVV and the corresponding values for SVH are shown in **Table 7**. Compared with 1 of 17 controls who had an abnormal value for uSVV (ie, either SVV-CW or SVV-CCW), a total of 7 of 9 subjects in the PVD group (78%) and 9 of 10 subjects in the MD group (90%) had abnormal values for uSVV. Six percent of control subjects, 44% of subjects in the PVD group, and 40% of subjects in the MD group had abnormal values for uSVH. The sensitivity and specificity of uSVV and uSVH values compared with those of SVV and SVH values are shown in **Table 8**. When the PVD and MD groups are combined, the sensitivity of uSVV values reaches 84.2% compared with a sensitivity of 26. 3% for SVV values. Similarly, the sensitivity of uSVH values increases to 42.1% (from 26. 3% sensitivity for SVH values).

COMMENT

The main finding of this study indicates that SVV and, to a lesser extent, SVH values are influenced by the direction of the preset angle. The SVV value is biased toward the direction of the preset angle, and this relation is stronger in the PVD and MD groups compared with the control group. To our knowledge. this finding has not been previously reported. The SVH value in the control group is biased in the direction opposite to the preset angle. The uSVV value of subjects in the PVD and MD groups may be abnormal, although the SVV values are within reference range (Table 7). There is no increase in the number of control subjects with abnormal uSVV values compared with the number of control subjects with abnormal SVV values. Similar to the uSVV results, the uSVH values in the PVD and MD groups may be abnormal, despite the SVH values being within reference range, but in a smaller number of subjects (Table 7). As with uSVV values, there is no increase in the number of control subjects with an abnormal uSVH value compared with the number with abnormal SVH values. The study also confirms that mean SVV and SVH are not sensitive tests for chronic PVD and MD. The mean SVV and SVH values do not correlate with the degree of canal paresis or duration of symptoms in either pathological group. The SVV and SVH values do not correlate with the hearing threshold in subjects with MD.

SVV AND SVH VALUES IN PVD AND MD GROUPS

Absolute values of mean SVV and SVH in the controls in the present study are in agreement with earlier reports.[4,16-19] The 2 control subjects, one with an abnormal value of SVV and another with an abnormal value of SVH, had values outside the reference range and may represent the extremes of the bell-shaped normal distribution curve. A smaller percentage of subjects in the PVD group had abnormal mean SVV and SVH values in the present study (2 of 9 subjects [22%]) than reported earlier. This could be explained by the longer duration of symptoms in this group and the fact that subjects with vestibular neurectomy, who may have persistently abnormal SVV and SVH values, were not included in the present study.[18] Abnormal mean SVV or SVH measures were seen in 2 subjects in the PVD group. These subjects had symptoms suggestive of otolithic dysfunction and persistent dysequilibrium; the latter has been postulated owing to slower compensation from otolithic dysfunction.[20] Because both subjects had only a modest canal paresis, this may indicate either a recovery of

Table 5. Relationship Between SVH Value and PA in the 3 Subject Groups[a]

	Controls (n = 17)[b]			PVD (n = 9)[c]			MD (n = 10)[d]		
	PA for SVH Value			PA for SVH Value			PA for SVH Value		
SVH	Positive	Negative	Total	Positive	Negative	Total	Positive	Negative	Total
Positive	31	61	**92**	40	41	**81**	42	35	**77**
Negative	71	37	**108**	15	12	**27**	17	22	**39**
Zero	0	4	**4**	0	0	**0**	1	3	**5**
Total	**102**	**102**	**204**	**55**	**53**	**108**	**60**	**60**	**120**

Abbreviations: MD, Ménière's disease; PA, preset angle; PVD, peripheral vestibular dysfunction ; SVH, subjective visual horizontal.
[a] The frequency categorization table indicates the number of patients with positive and negative SVH values for their respective positive and negative PAs. The SVH values corresponding to zero have been excluded from the analysis.
[b] In the control group, the SVH value is more likely to be positive when the PA is negative, and it is more likely to be negative when the PA is positive. The *P* value is significant in this group ($P < .001$).
[c] In the PVD group there was no significant relationship ($P = .58$).
[d] In the MD group there was no significant relationship ($P = .32$).

semicircular canal function or a vestibular insult predominantly involving the otoliths and/or the vertical canals.[1] A contraversive deviation of the SVH measure was noted in 1 subject, contrary to findings reported previously.[4,18] Interestingly, this subject had a directional preponderance in the same direction on ENG, and this finding may be postulated as being caused by overcompensation.

Mean SVV and SVH values were congruous when within reference range, in agreement with earlier reports,[3,21] but not so when either value was abnormal. Dissociation between the SVV and SVH values has been previously reported when these tests are done in the roll tilt position.[22] The percentage of subjects in the MD group showing an abnormal SVV or SVH value deviation (50%) is in agreement with an earlier study, as is contraversive SVH value deviation.[6] The latter is postulated to be caused by an increased resting activity of otolithic afferents, an analogue of the irritative nystagmus seen in MD. In the MD group, abnormal deviation of the SVV and SVH values occurred in association with a preceding acute attack, as reported previously,[3] or a total canal paresis on the affected side.

CORRELATION OF SVV AND SVH VALUES WITH CLINICAL FEATURES

As in earlier reports, there was no correlation between the mean value of SVV or mean value of SVH tilt and the duration of symptoms in either the MD group or the PVD group in the present study.[17] Abnormal SVV tilts are known to normalize over weeks to years after an acute vestibular insult. Because all subjects in the PVD and MD groups in the present study had symptoms of more than 1 year's duration, the early compensation of SVV and SVH may have been missed in the present study.[20] There was no significant linear correlation between mean SVV or mean SVH values and severity of canal paresis in either the MD group or PVD group (see the "Results" section for *P* values), although 2 subjects with MD and abnormal values for SVV and SVH deviation had 100% canal paresis, confirming earlier reports.[4,6] This would be in keeping with phylogenetic preservation of the vertical semicircular canal and otolithic function. The present study did not find any correlation between "worse" puretone threshold and mean value for SVV or mean value for SVH tilt in subjects in the MD group, as reported previously,[23] supporting differential involvement of cochlear and vestibular function in MD.

Table 6. Reference Range Values of SVV-CW, SVV-CCW, SVV, SVH-CW, SVH-CCW, and SVH in Controls

Variable, Mean (SD)	Reference Range
SVV-CW, 0.81 (1.30)	3.4 to −1.8
SVV-CCW, −0.48 (1.25)	2.0 to −3.0
SVV, 0.16 (0.96)	2.1 to −1.8
SVH-CW, −0.63 (1.87)	3.1 to −4.3
SVH-CCW, 0.28 (1.57)	3.4 to −2.9
SVH, −0.17 (1.44)	2.7 to −3.0

Abbreviations: CCW; counterclockwise; CW, clockwise; SVH, subjective visual horizontal; SVV, subjective visual vertical.

RELATIONSHIP OF SVV AND SVH VALUES WITH THE PRESET ANGLE

The present study found that the SVV value depends on the direction of the preset angle in all 3 groups, more so in the PVD and MD groups compared with the controls. There is a bias of the SVV tilt toward the initial direction of the linear marker. It is therefore important to consider the value of the preset angle when comparing SVV values among subjects, among different test settings, and during serial measurements in the same subject.

The relationship between SVV value and preset angle could be explained by the effect of visual roll motion produced by the rotating linear marker because the subjects had their eyes open during the line offset. This effect is postulated to be mediated by central visuovestibular interaction, and the otoliths may have a role in limiting the tilt effects of visual motion.[24] This effect is stronger in labyrinthine defective subjects owing to their visual

Table 7. Patients With Abnormal Values for SVV-CW, SVV-CCW, SVV, uSVV, SVH-CW, SVH-CCW, SVH, and uSVH in the 3 Subject Groups

Subject Group (No.)	No. of Subjects With Abnormal Value for							
	SVV-CW	SVV-CCW	uSVV	SVV	SVH-CW	SVH-CCW	uSVH	SVH
Controls (17)	1	0	1	1	1	0	1	1
PVD (9)	5	4	7	1	2	2	4	1
MD (10)	5	6	9	4	2	3	4	4

Abbreviations: CCW; counterclockwise; CW, clockwise; SVH, subjective visual horizontal; SVV, subjective visual vertical; uSVH, unidirectional SVH; uSVV, unidirectional SVV.

Table 8. Sensitivity and Specificity of SVV and SVH Values Compared With uSVV and uSVH Values[a]

Variable	SVV	uSVV	SVH	uSVH
Sensitivity	26.3	84.2	26.3	42.1
Specificity	94.1	94.1	94.1	94.1

Abbreviations: SVH, subjective visual horizontal; SVV, subjective visual vertical; uSVH, unidirectional SVH; uSVV, unidirectional SVV.

[a]The peripheral vestibular dysfunction and Ménière's disease groups are combined (n = 9 + 10 = 19) to make 1 group. Data are given as percentages.

overdependence and lack of vestibular information compared with controls.[25] A stimulus rotating around the nasooccipital axis has been shown to produce ocular torsion,[26] and this may contribute to the SVV value deviation. Ocular torsion measurements were not performed in the present study.

A recent study by Hoppenbrouwers et al[7] reported that SVV measurement may be influenced by the preset angle in the absence of visual roll effect. In the study by Hoppenbrouwers et al,[7] the "E effect" (the SVV deviation opposite to the direction of head tilt, occurring when the head is tilted by <60°) was suppressed by aligning the linear marker parallel to the head longitudinal axis for the same degree of head tilt. The visual roll effect was excluded in that study[7] by turning off the linear marker during misalignment and allowing subjects to adapt to the darkness for 5 minutes before starting the test. The authors[7] postulated that the sensitivity of specific regions of the visual cortex to visual stimuli aligned with the cardinal planes of the head (0° or 90°) makes visual pattern recognition easier in the parallel paradigm and overrides the utricular information during head tilt.[7] This phenomenon may be compared with the visual overriding of vestibular external ocular movements.

The SVH value is dependent on the preset angle in the control group, but the SVH values are biased in a direction opposite to the change in preset angle. This effect cannot be explained on the basis of visual roll. The subjects in the PVD group show no significant differences compared with controls (see "Results" section for *P* values), although the SVH value of subjects in the MD group is more dependent on the preset angle compared with controls. The frequency table analysis for SVH values did not yield significant results in the PVD and MD groups (see Table 5 for *P* values), and this may be related to a combination of factors (eg, absence of a significant relationship between the SVH value and preset angle in these groups, influence of the side of the lesion, and a small sample size). Few other studies have considered the effect of the preset angle on the SVV or SVH value. An earlier study[8] noted no significant difference between SVV measures performed with an initial right or left tilt of the marker, but the study methods were not detailed. Another study[9] described an abnormal deviation of SVV values when the linear marker was preset on the side with a vestibular lesion in subjects with PVD. In the present study, a similar valid comparison could not be made because the numbers of right and left lesions in both the PVD and MD groups were small. Studies[19] in subjects with cerebral hemispheric stroke have reported that the E effect is absent when the linear marker is preset on the nonparetic side, and the authors[19] correlated this observation with visuospatial neglect, proposing a lesion close to the corticovestibular areas to account for disturbance of spatial perception in the roll plane.

uSVV AND uSVH VALUES

Subjects in the PVD and MD groups were more likely to have abnormal uSVV value deviations, despite having had mean SVV values within the reference range. This presents a potential avenue for further research to improve the sensitivity of SVV values in chronic vestibular lesions, as has been sought previously using methods such as measuring SVV values in body roll tilt positions,[27] eccentric rotation,[16] and with neck vibration.[18] From the results of this study, it seems that SVH values may not be as useful as SVV values in this respect. From Table 8, it is evident that the uSVV value improves the sensitivity of the test from 26.3% to 84.2%. The uSVH value is less sensitive compared with the uSVV value.

The results of the present study may be biased by the small numbers of participants and the age and sex inequalities of the groups. The mean values of SVV and SVH did not differ significantly between the sexes, or in the subgroup of 5 control subjects 40 years or older, suggesting that age differences among the groups may not cause a significant bias on the results (see the "Mean SVV and SVH Values in Controls and Patient Groups" subsection in the "Results" section). In the experimental design, the start positions of the light bar were set randomly, with possible confounding effects from the sequence of readings and a learning effect. Although all testing was done by a single observer, there was no blind-

ing. Also, with the instrumentation used, participants remarked that making fine adjustments with the remote control was sometimes not easy, despite a defined instrument accuracy of 0.1°. Future directions for research in this area may include using a larger age- and sex- matched subject population, simultaneous measurements of ocular torsion, and functional parameters such as posturography and symptom scales (eg, the Dizziness Handicap Inventory), studying the effect of the magnitude of the preset angle on SVV and SVH values, considering the effect of visual roll, and studying the effect of the preset angle in relation to the side of the lesion. It may be then possible to suggest an optimal protocol for performing the SVV and SVH tests with a view to improving sensitivity in chronic peripheral vestibular lesions.

CONCLUSIONS

The findings of this pilot study allow 3 broad conclusions to be drawn:

- There is currently no standard protocol for performing the SVV and SVH test in relation to the preset angle. The present study indicates that the preset angle should be taken into consideration when comparing SVV and SVH results among different test settings and different conditions.
- The direction of the preset angle can influence values of SVV and the SVH. This relation is most marked for SVV values in subjects with PVD and MD compared with controls.
- The uSVV value improves the sensitivity of the test, and this finding could be explored further to devise an optimal protocol to measure SVV and SVH.

This study indicates factors that may influence the results and interpretation of SVV and SVH values and highlights the need for further research on these simple tests.

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