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ORIGINAL ARTICLE

Dynamics of canal response to head-shaking test in benign paroxysmal positional vertigo

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Abstract

Conclusions. Time constant and maximum slow phase velocity (SPV) of head-shaking nystagmus (HSN) demonstrated a differential canal response to head shaking in 24% of patients with posterior canal benign paroxysmal positional vertigo (BPPV). We suggest that vestibular lithiasis has a limited contribution to the mechanism that generates HSN. *Objective.* To determine the canal response to head shaking in BPPV. *Patients and methods.* This was a case-control study including 104 individuals with BPPV. The diagnosis was based on the presence of vertigo and nystagmus during the positional test. Subjects were examined by the horizontal and vertical head-shaking test. Eye movements were recorded on a video camera to analyze the nystagmus. The head was shaken passively in the horizontal and sagittal planes, respectively, for horizontal and vertical HSN at a frequency of 2 Hz. HSN was considered when six consecutive beats of nystagmus with an SPV of at least 2°/s were detected. Main outcome measures were the presence of horizontal and vertical HSN, maximum SPV of HSN, time constant of HSN, and canal paresis. *Results.* Maximum SPV of vertical HSN was higher in BPPV patients with posterior canal BPPV ($n=10$) than in controls ($p=0.04$). Moreover, the time constant of vertical HSN was significantly lower for posterior canal BPPV when compared with controls ($p<0.02$).

Keywords: *Head-shaking nystagmus, case-control studies, BPPV, vestibular testing, video-oculographic examination, vestibular system*

Introduction

Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder and its diagnosis and treatment are based on the features of positional nystagmus [1,2]. It is defined by spells of positioning vertigo and nystagmus of short duration that are elicited by turning of the head in the plane of the affected semicircular canal [3]. There are three clinical variants: posterior, horizontal, and anterior canal BPPV, the most frequent being the involvement of the posterior canal [4,5]. The diagnosis of posterior canal BPPV is based on the observation of a torsional and up-beating nystagmus during the Dix-Hallpike (DH) test [1,6].

According to the hypothesis for explaining the positional nystagmus, the vestibular lithiasis can affect any of the three semicircular canals. High density particles could be either free-floating in the canal (canalithiasis) or they could be found adhered to the cupula of the crista ampullaris (cupulolithiasis). These mechanisms can explain the features of the positional nystagmus (geotropic or apogeotropic, latency and duration) [7–12].

The head-shaking test was initially described by Vogel [13]. This passive high frequency rotation of the head is usually performed in the horizontal and sagittal planes. The head-shaking test can evoke a transient nystagmus, which is usually horizontal, monophasic, and beats towards the labyrinth with more caloric excitability. Sometimes, the nystagmus can be biphasic, which means that the nystagmus changes direction as it decays. In 1951, Moritz [14] and later, Kamei et al. [15], proposed horizontal head-shaking nystagmus (HSN) as a diagnostic tool for vestibular dysfunction, but later experiences have demonstrated that its sensitivity can change

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according to different vestibular disorders, recording methods, and evaluation criteria. The sensitivity of the test can be increased with video-oculography or scleral coil techniques and it decreases with Frenzel glasses, electronystagmography, or direct observation [16].

There are several mechanisms to explain the generation of HSN [17,18]. (i) An asymmetric peripheral input and a central velocity storage mechanism. The biphasic response was obtained in patients with complete unilateral arreflexia. The first phase was explained by the second Ewald's law resulting from vestibular asymmetry and the velocity storage mechanism, whereas the secondary phase of biphasic HSN is produced by the second Ewald law and the vestibular adaptation of the preserved side [17]. (ii) Directional asymmetry of the central velocity storage mechanism. (iii) Asymmetric peripheral dynamics, such as high viscosity, that affect endolymph movement [17,19]. (iv) Asymmetric proprioceptive inputs from neck muscles, as has been shown for vibration-induced ocular torsion and nystagmus [20].

Our working hypothesis is that vestibular lithiasis may affect the endolymph dynamics and the canal response to head shaking by setting up an asymmetric response and produce an ipsilateral monophasic HSN. To test this hypothesis, we compared the ocular response to head shaking in patients with BPPV with a control group.

Patients and methods

Subjects

A prospective case-control study was carried out in a series of individuals with BPPV. The case group consisted of 104 consecutive adults (37 males, 67 females) with a clinical diagnosis of BPPV. Clinical diagnosis was carried out by positional testing: DH test and head-hanging maneuver, for anterior and posterior canal, respectively, and lateral turning of the head in supine position for the lateral canal. The control group consisted of a sample of 50 age- and sex-matched individuals who did not have a history of hearing loss or vertigo and agreed to participate in the study. Physical examination was performed in all individuals (patients and controls) and included otoscopy, 256 and 512 Hz Rinne and Weber tests, pure tone audiometry (250–8000 Hz), and a basic neurotologic examination (oculomotor, saccades, head-impulse test, cranial nerve examination, and Romberg, Barany and Fukuda tests). Individuals with a hearing loss >30 dB in any frequency tested or an abnormal neurotologic examination were excluded from the control group.

Experimental protocol

The video-oculographic (VOG) examination included spontaneous nystagmus (SN) in the primary position of the eye, horizontal and vertical HSN, positional testing (DH test for the lateral canal and head-hanging maneuver for the anterior canal), and water bithermal caloric testing. The criteria for diagnosis of BPPV during the positional testing comprised the observation of: (1) up-beating and torsional nystagmus for the posterior canal during the DH test with reversal of nystagmus on rising from the DH position; (2) horizontal direction-changing nystagmus for the lateral canal during the DH test or the roll test – the affected side was decided on the basis of the higher maximum SPV and vertigo severity; (3) down-beating nystagmus during the DH test or the head-hanging maneuver. All the individuals with down-beating nystagmus were evaluated by a gadolinium-enhanced cranial MRI to rule out a structural central nervous system (CNS) disease.

A caloric test by using a Variotherm Plus model water irrigator (Atmos, Berlin, Germany) was performed on each subject, with a water flow of 250 ml/20 s at 30°C and 44°C with an interval of 10 min between successive irrigations. All irrigations were performed with the head and trunk elevated 30° above horizontal. The percentage of canal paresis and directional preponderance was calculated using the Jongkees index formula on the difference of maximal slow-phase velocity [21]. The values for the normal limits of canal paresis and directional preponderance in our laboratory were 28%.

Eye movement recording

Horizontal and vertical eye movements were recorded by a charge-coupled device (CCD) camera adapted to goggles (SMI, Berlin, Germany) in complete darkness. The camera was adapted in the contralateral eye to the side of testing to minimize the movement of the camera during the DH test. Video image analysis software was used to analyze the eye response. Recording time was 60 s for SN or HSN and 120 s for the caloric response. Maximum slow phase velocity (SPV) was determined by the software and SPV values obtained in the caloric test were compared with normal values for our laboratory. The default parameters for the discrete analysis of the nystagmus were: minimum acceleration 500°/s^2, minimum velocity of the fast phase 50°/s, minimum amplitude of the fast phase 1°, minimum amplitude of the slow phase 1°, and maximum slow phase duration 50 ms.

Spontaneous nystagmus was considered significant if six consecutive beats with an SPV of 2°/s were

recorded. The HSN test was performed with the patient in a sitting position with the head anteflexed to 30°. The head was then shaken passively in the earth horizontal and sagittal planes, respectively, for horizontal and vertical HSN, by the examiner at 45° for 30 cycles with a frequency of 2 Hz. After the head movement stopped, nystagmus was recorded for at least 1 min. HSN was considered present when it fulfilled the following criteria: (i) at least six consecutive beats of nystagmus; (ii) a latency from the end of head-shaking of no more than 5 s; (iii) an SPV of at least 2°/s. HSN was quantified according to its maximal SPV and qualitatively categorized into monophasic or biphasic. The cases of biphasic HSN were classified according to their first phase. The time constant of the HSN was calculated using a least-squares linear fit of the relationship between the log of slow-phase velocity and time [17].

Treatment

Patients were treated by the positional treatment according to the canal involved. The particle repositioning maneuver (PRM) or Epley's modified maneuver was used for posterior and anterior canals [1]; Lempert's maneuver was used for the lateral canal [5]. Written information was made available to the patients and informed consent was obtained for all of them after explaining the purpose of the study. The ethical and research committee of the hospital approved this research study.

Statistical analysis

Variables studied were SN, horizontal or vertical HSN, positional nystagmus, canal paresis, and directional preponderance. All were categorized as present or absent in both groups and the odds ratio (OR) with 95% confidence intervals were calculated for each variable. Significant differences were determined by the χ^2 method with Yates' correction. Fisher's exact test was used if any value obtained in the 2×2 table was <5. Maximum SPV and time constant of horizontal and vertical HSN were compared between patients and controls by ANOVA (analysis of variance) and non-parametric t tests. Regression analysis was used to relate maximum SPV of HSN and positional nystagmus and time constant of HSN with canal paresis. $p<0.05$ was considered significant.

Results

The mean age of the individuals in the BPPV group was 56.9 ± 14.8 (mean $\pm$ standard deviation, range 20–81). Fifty-nine patients had a posterior canal variant, 23 cases presented a lateral canal affected (17 geotropic, 6 apogeotropic direction-changing nystagmus), and 22 cases an anterior canal variant. The mean age of the 50 individuals (25 men, 25 women) in the control group was 55.0 ± 11.5 (range 34–74).

Spontaneous nystagmus (SN)

An SN was found in 22/104 (21%) individuals with BPPV (8 left, 7 right, 6 up, 1 down). The percentages of individuals with SN were not statistically different in the two groups (OR = 1.97 (95% confidence intervals, 0.7–5.9), $p = 0.25$). In the control group, SN was observed in 6/50 (12%) individuals. Subjects with SN were excluded from both groups to facilitate HSN evaluation.

Horizontal HSN

Table I presents the results of the head-shaking test, the caloric response, and the canals' occurrence in patients with BPPV. The head-shaking test produced a monophasic response in 15/20 patients and biphasic HSN in 5 cases (Figure 1a). In controls, monophasic HSN was found in 10/12 individuals. Horizontal head-shaking test elicited nystagmus in 20/82 (24%) patients with BPPV and in 12/44 (27%) cases in the control group. There was no difference in the frequency of horizontal HSN between patients and the control group (OR = 0.86 (0.35–2.15), $p = 0.88$).

To analyze the effect of the canal affected in the head-shaking response, we compared if the

Table I. Horizontal and vertical head-shaking nystagmus (HSN) frequency in patients with benign paroxysmal positional vertigo (BPPV) and a control group*.

BPPV variant	Spontaneous nystagmus	Horizontal HSN	Vertical HSN	Canal paresis	Directional preponderance
Posterior canal	13/59 (22%)	13/46 (28%)	11/46 (24%)	10/59 (17%)	13/59 (22%)
Horizontal canal	3/23 (13%)	4/20 (20%)	3/20 (15%)	2/23 (9%)	14/23 (60%)
Anterior canal	6/22 (27%)	3/16 (19%)	1/16 (6%)	4/22 (18%)	3/22 (15%)
Total	22/104 (21%)	20/82 (24%)	15/82 (18%)	16/104 (15%)	30/104 (29%)
Control group	6/50 (12%)	12/44 (27%)	5/44 (11%)	2/50 (4%)	6/50 (12%)

*Patients with spontaneous nystamus were excluded to evaluate HSN.

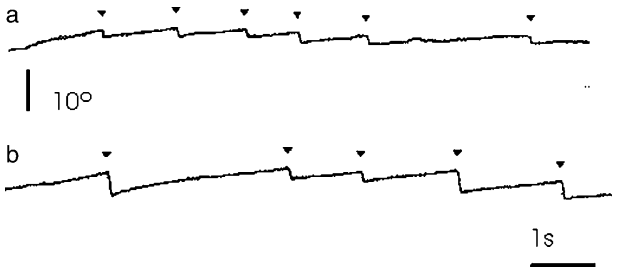

Figure 1. (a) Eye response (left horizontal nystagmus) to horizontal head shaking in a patient with a left posterior canal BPPV. (b) The same eye response was obtained after vertical head shaking.

rotational plane of the head-shaking test (horizontal or vertical) generates a different response in patients with vertical (anterior or posterior canals) or horizontal BPPV. Horizontal head-shaking test produced a horizontal ipsilateral beating nystagmus in 4/20 (20%) patients with horizontal canal BPPV, whereas it elicited nystagmus in 12/44 (27%) in the control group (OR = 0.67 (0.15–2.76), $p = 0.75$).

Vertical HSN

Vertical head-shaking test produced a nystagmus in 15/82 individuals with BPPV (18%) and in 5/44 cases in the control group (11%). There was no difference in the frequency of vertical HSN between patients and the control group (OR = 1.75 (0.54–6.0), $p = 0.44$.

Vertical head-shaking test produced a nystagmus in 12/62 (19%) cases of vertical canal BPPV (Figure 1b) and it was observed as frequently in patients with anterior or posterior canal BPPV as in the control group (OR = 1.87 (0.55–6.72, $p = 0.40$).

Maximum SPV of HSN

Figure 2 shows the response to horizontal head shaking in a subject with BPPV. The maximum SPV profiles of horizontal HSN fit to a log function. Table II presents the features of HSN in BPPV patients according to the canal variant. The amplitude of HSN reflects the initial maximum SPV of HSN; the SPV of the horizontal component of horizontal HSN obtained were 11.6 ± 8.1 ($n = 11$), 14.3 ± 11.7 ($n = 3$), and 9.3 ± 5.3 ($n = 5$; mean $\pm$ standard deviation), for posterior, anterior, and horizontal canal BPPV, respectively.

In the posterior canal variant, the SPV of the horizontal component of horizontal HSN ($n = 11$) was not significantly different from SPV values obtained in the controls ($n = 12$, $t = 1.05$, $p = 0.31$). However, the SPV of the vertical component of vertical HSN was higher in BPPV patients with posterior canal BPPV ($n = 10$) than in control subjects ($n = 5$, $t = 2.29$, $p = 0.04$).

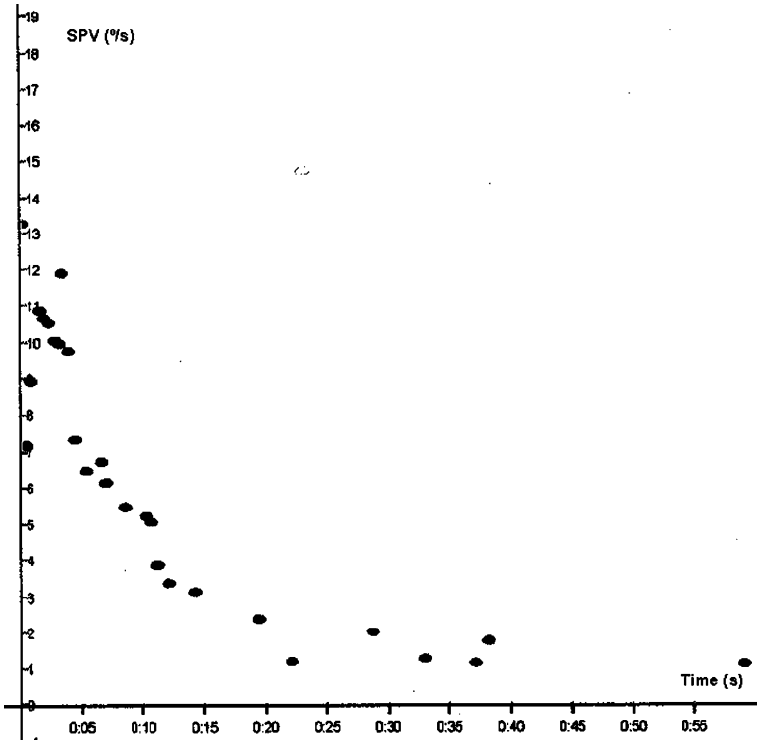

Figure 2. Horizontal head-shaking nystagmus of a subject with posterior canal BPPV. Individual slow phases, uncorrected for spontaneous nystagmus are plotted.

Table II. Features of horizontal and vertical head-shaking nystagmus (HSN) in the BPPV ($n=86$) and control groups ($n=50$).

Subjects	H HSN			V HSN		
BPPV type	SPV	Direction	Time	SPV	Direction	Time
Posterior canal						
1 R	13.7	R	53	19.3	R	34
*2 R	3.8	R	42	3.4	L	16
3 L	9.6	L	18	9.7	L	47
4 R+L	6.3	R	44	–	–	–
5 R	–	–	–	5.5	R	41
6 L	10.3	L	7	5.8	L	49
*7 L	10.2	R	7	5.4	L	12
8 R	4.8	R	19	8.8	L	17
9 R	4.6	D	44	19.9	D	25
10 R+L	18.3	L	17	–	–	–
*11 R	31.8	L	10	19.2	L	22
*12 R	13.7	R	30	17.9	U	51
Mean±SD	11.6±8.1		26.5±16.8	11.5±6.8		31.4±14.9
Anterior canal						
13 R	5.2	L+D	55	5.0	L	20
14 R+L	10.3	R	36	–	–	–
15 R	27.5	R	9	–	–	–
Mean±SD	14.3±11.7		33.3±23.1	5.0		20
Horizontal canal						
16 R	–	–	–	11.4	R	33
17 R	17.6	R	18	–	–	–
18 R	12.5	R	15	7.9	U	46
19 R	2.3	R	36	–	–	–
20 R	5.3	R	53	–	–	–
*21 R	8.7	R	8	–	–	–
22 L	–	–	–	8.8	D	40
Mean±SD	9.3±5.3		26.0±15.3	9.4±1.8		39.7±6.5
Control						
1	18.9	D	15	–	–	–
2	4.4	L	33	–	–	–
3	2.5	R	18	2.3	R	37
4	3.4	R	28	–	–	–
5	10.1	L	55	–	–	–
6	–	–	–	2.5	R	24
7	7.8	L	43	–	–	–
8	7.0	D	27	–	–	–
9	8.3	R	15	–	–	–
10	8.7	L	21	–	–	–
11	16.7	L	21	–	–	–
12	10.3	L	20	–	–	–
13	5.8	L	15	–	–	–
14	–	–	–	7.5	L	56
15	–	–	–	11.0	R	30
16	–	–	–	2.1	R+D	55
Mean±SD	8.6±4.9		25.9±12.4	5.1±4.0		40.4±14.5

H, horizontal; V, vertical; SPV, slow phase velocity; R, right; L, left; U, up; D, down.
*Patients with biphasic horizontal HSN.

Time constant of HSN

Table III summarizes the time course of horizontal and vertical HSN and the caloric response in each subject. No differences were found in the time constant of horizontal HSN (HTc) between patients with posterior canal BPPV and controls ($t=-0.16$, $p=0.88$). However, the time constant of vertical HSN (VTc) was significantly reduced in individuals with posterior canal BPPV ($n=10$) when they were compared with the control group ($t=2.29$, $p=0.04$).

Bithermal caloric test

In the BPPV group, the caloric test showed a horizontal canal paresis in 14/86 of cases (16%; 8 ipsilateral, 6 contralateral), and a directional preponderance in 16/104 (15%; 8 ipsilateral, 8

Table III. Time constant of SPV for horizontal component of horizontal HSN (HTc) and vertical component of vertical HSN (VTc) in the BPPV and control groups.

Subjects	H HSN			V HSN	
BPPV type	HTc	Direction	CP	VTc	Direction
Posterior canal					
1 R	17.2	R	8	9.79	R
*2 R	26.8	R	9	11.1	L
3 L	6.8	L	51	17.6	L
4 R+L	20.4	R	2	–	–
5 R	–	–	−3	20.5	R
6 L	2.6	L	−66	23.7	L
*7 L	2.6	R	18	6.1	L
8 R	10.3	R	−5	6.7	L
9 R	24.6	D	−20	7.12	D
10 R+L	4.9	L	11	–	–
*11	2.5	L	−46	6.34	L
*12	9.8	R	−11	15.1	U
Mean±SD	11.7±9.1		20.8±21.3	11.4±7.4	
Anterior canal					
13 R	28.4	L+D	−23	10.3	L
14 R+L	13.5	R	0	–	–
15 R	2.3	R	−37	–	–
Mean±SD	14.7±13.1		30±9.9	10.3	
Horizontal canal					
16 R	–	–	35	11.6	R
17 R	5.3	R	–7	–	–
18 R	5.1	R	13	18.9	U
19 R	36.8	R	15	–	–
20 R	27.1	R	−24	–	–
*21 R	3.2	R	−5	–	–
22 L	–	–	NA	15.7	D
Mean±SD	15.5±15.4		16.5±11.3	15.4±3.7	
Control					
1	6.0	R	10	–	–
2	18.9	L	3	–	–
3	16.7	R	−3	37.8	R
4	19.5	R	−28	–	–
5	20.3	L	0	–	–
6	–	–	7	22.31	R
7	17.83	L	−19	–	–
8	11.82	D	10	–	–
9	6.0	R	−29	–	–
10	8.3	L	4	–	–
11	6.3	L	−21	–	–
12	7.3	L	10	–	–
13	7.2	L	−4	–	–
14	–	–	5	23.7	L
15	–	–	1	10.6	R
16	–	–	7	63.1	R+D
Mean±SD	12.1±5.9		10.1±9.2	31.5±20.1	

CP, canal paresis; H, horizontal; V, vertical; SPV, slow phase velocity; R, right; L, left; U, up; D, down.
*Patients with biphasic horizontal HSN.

contralateral). In the control group, there were two individuals (4.5%) with a borderline canal paresis (29% in both cases) and six individuals with directional preponderance. The percentage of subjects with canal paresis was significantly different between BPPV and control groups (OR = 4.36 (0.96–19.78), $p = 0.03$, Fisher test). In addition, the directional preponderance was observed more frequently in BPPV than in controls (OR = 2.97 (1.07–8.68), $p = 0.03$).

Relationship between HSN and canal paresis

Six patients with vertical BPPV had a positive HSN and canal paresis, whereas 52 patients with vertical BPPV did not have HSN or canal paresis (OR = 3.90

(0.94–16.4), $p=0.04$). The overall frequency of HSN in vertical canal BPPV with canal paresis was 43%. For the 23 cases with horizontal canal BPPV variant, there was no case with HSN and canal paresis and 10 individuals did not have canal paresis or HSN (OR = 0, $p=0.48$). However, there was no relationship between the frequency of HSN and canal paresis in the control group (OR = 2.82 (0–115), $p=0.47$, for horizontal HSN; OR not calculable for vertical HSN).

The time constant for horizontal HSN was not related to canal paresis in either posterior or horizontal canal BPPV ($r=0.22$ for posterior canal and $r=0.42$ for horizontal canal) or in the control group ($r=-0.12$, all $p>0.05$). Hence, the time constant for vertical HSN did not show a significant correlation with canal paresis in posterior canal BPPV ($r=0.17$, $p=0.651$). The number of cases with anterior canal and HSN was too low for a reliable regression analysis.

Discussion

The dynamic properties of HSN have been examined in patients with BPPV. It is thought that HSN reflects a directional imbalance of the VOR in the 2–3 Hz frequency range. During high acceleration head rotations, the excited side mainly generates the VOR, either because the nonlinear pathway of the inhibited side is driven into inhibitory cut-off [22], or by the existence of a directional asymmetry [19]. The nystagmus appears after head oscillation has stopped if the directionally asymmetric response has been stored during head shaking to be discharged thereafter. Although biphasic HSN is considered the basic response [17], it is the least frequent in our BPPV and control group and in most series [18,23,24]. If the first phase of HSN depends on the second Ewald law and on the velocity storage system, it could be deduced that small asymmetries would not be sufficient to produce the first phase of HSN. In this study, we found that horizontal HSN was observed in 27% of healthy individuals and vertical HSN in 11% of cases, respectively.

Our study is the first to investigate the dynamics of canal response to head shaking in BPPV and it demonstrates that HSN is not more frequent in patients with BPPV than in healthy subjects. However, the maximum SPV of HSN was significantly higher in patients with posterior canal BPPV than in controls. In addition, the mean time constants of vertical HSN (VTc) were reduced for posterior canal patients when they were compared with the control subjects. However, the mean time constant for horizontal HSN (HTc) did not differ in posterior canal BPPV from controls. These findings suggest an impairment of vertical canal function in posterior canal BPPV.

Hence, the asymmetric peripheral input in the plane of the canal affected during the positional test (the inertial effect of debris in the canal), has a significant effect on the mechanism of generation of HSN in posterior canal BPPV. The unexpected finding of our study is that HTc for horizontal canal BPPV did not differ between patients and controls. This could be because the BPPV stimulus is too weak or its frequency too low to charge the velocity integrator in horizontal canal BPPV. In addition, a limited unilateral vestibular lesion of low degree affecting the semicircular canals could produce adaptive changes in the velocity storage system by the action of the commissural pathways in such a way that the canal dynamics would remain as if there were a single central labyrinth, that would equalize the VOR in the two rotational directions of the canal plane [25]. It has been shown that dynamic properties of positional nystagmus are different among posterior, anterior, and horizontal canal BPPV [26,27]. So, the second Ewald law effect should be small in horizontal canal BPPV because the asymmetric BPPV stimulus is probably too weak (either because of the low inertial effect of canalith or vertical canals are away from the plane of head shaking) to charge the velocity integrator and generate the HSN.

The major limitation of the study is the low number of BPPV patients with HSN. Only 21 patients had HSN (20 horizontal and 15 vertical), 13 were posterior canal, 4 horizontal canal, and 3 anterior canal variants. These findings limit the study of the relationship of the time constants of HSN with the degree of canal paresis in anterior ($n=3$) and horizontal canal BPPV ($n=6$).

HSN reflects a high frequency gain asymmetry in medial vestibular nuclei. This mechanism might be the same as that proposed by Halmagyi et al. for the development of isolated directional preponderance of caloric nystagmus [21]. In the absence of spontaneous nystagmus, a left–right difference in the sensitivity of the horizontal canal mechanoreceptors or a difference in the resting activity of type I medial vestibular nucleus neurons cannot explain a directional preponderance. Furthermore, the resting activity of left and right medial vestibular nucleus must be the same if the patient does not have spontaneous nystagmus. According to these authors, an isolated directional preponderance could reflect an asymmetry in dynamic gain between medial vestibular nucleus neurons on either side (asymmetry to rotational stimulation without spontaneous nystagmus or canal paresis). As directional preponderance, HSN might reflect an asymmetry

in dynamic sensitivity between left and right medial vestibular nucleus neurons, suggesting an ongoing or uncompensated vestibular lesion [24].

Palla et al. analyzed the influence of gravity on SPV of horizontal HSN in patients with unilateral vestibular hypofunction and found that HSN is best elicited when patients are lying on their affected ear [28]. This suggests a gravity-dependent mechanism that indicates an interaction between otolith and horizontal semicircular canal signals. BPPV is a gravity-dependent condition and position in bed seems to be involved in the pathophysiology of BPPV, since many patients experience attacks when they are moving in bed. It has been shown that the side affected by BPPV correlates with the preferred position in bed in patients with posterior canal BPPV [29] and it is possible that BPPV predominantly involves the right ear because most persons prefer to sleep in the right lateral supine position [30].

To elicit HSN, the brainstem network that transiently accumulated the vestibular velocity information, the velocity storage system [31], must be operative to receive and accumulate the input that predominantly comes from the dominant ear [32,33]. We have found HSN in subjects with BPPV, so the central velocity storage system must be at least partially functioning in these individuals. We suggest that despite the position of vertical canals 45° away from the plane of head shaking, the mass of free-floating otoconial debris associated with canalolithiasis is enough to change the canal response to vertical head shaking in posterior canal BPPV and it makes a significant contribution to the generation of HSN by reducing its time constant.

Conclusion

Time constant and maximum SPV of HSN demonstrated a differential canal response to head shaking in posterior canal BPPV when they were compared with controls. We suggest that vestibular lithiasis has a limited contribution to the mechanism that generates HSN.

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References

[1] Epley J. The canalith repositioning procedure for treatment of benign paroxysmal positional vertigo. Otolaryngol Head Neck Surg 1992;107:399–404.

[2] Parnes L, Price-Jones R. Particle repositioning maneuver for benign paroxysmal positional vertigo. Ann Otol Rhinol Laryngol 1993;102:325–31.

[3] Brandt T. Positional and positioning vertigo and nystagmus. J Neurol Sci 1990;95:3–28.

[4] Honrubia V, Baloh RW, Harris MR, Jacobson KM. Paroxysmal positional vertigo syndrome. Am J Otol 1999;20:465–70.

[5] Lempert T, Tiel-Wilck K. A positional maneuver for treatment of horizontal-canal benign paroxysmal positional vertigo. Laryngoscope 1996;106:476–8.

[6] Dix MT, Hallpike CS. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. Ann Otol Rhinol Laryngol 1952;61:987–1016.

[7] Baloh RW, Jacobson K, Honrubia V. Horizontal semicircular canal variant of benign positional vertigo. Neurology 1993; 43:2542–9.

[8] Baloh RW, Sakala SM, Honrubia V. Benign paroxysmal positional nystagmus. Am J Otolaryngol 1979;1:1–6.

[9] Brandt T, Steddin S. Current view of the mechanism of benign paroxysmal positional vertigo: cupulolithiasis or canalolithiasis? J Vestib Res 1993;3:373–82.

[10] Korres S, Balatsouras DG, Kaberos A, Economou C, Kandiloros D, Ferekidis E. Occurrence of semicircular canal involvement in benign paroxysmal positional vertigo. Otol Neurotol 2002;23:926–32.

[11] McClure JA. Horizonatal canal BPV. J Otolaryngol 1985;14: 30–5.

[12] Nuti D, Vannucchi P, Pagnini P. Benign paroxysmal positional vertigo of the horizontal canal: a form of canalolithiasis with variable clinical features. J Vestib Res 1996;6: 173–84.

[13] Vogel K. Uber den nachweis des latenten spontannystagmys. Z Laryngol Rhinol 1932;22:202–7.

[14] Moritz W. Auswertungen des Kopfschüttelnystagmus. Z Laryngol Rhinol 1951;30:269–75.

[15] Kamei T, Kimura K, Kaneko H, Noro H. [Re-evaluation of the head-shaking test as a method of nystagmus provocation. Part 1. Its nystagmus-eliciting effect.] Jpn J Otolaryngol 1964;67:1530–4 (in Japanese).

[16] Yuen K, Fujimoto M, Masuda Y. Observation of head-shaking nystagmus with an infrared Frenzel's glass. Acta Otolaryngol Suppl 1995;519:191–2.

[17] Hain TC, Fetter M, Zee DS. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. Am J Otolaryngol 1987;8:36–47.

[18] Kasarkas A, Smith H, Galiana H. Head-shaking nystagmus (HSN): the theoretical explanation and the experimental proof. Acta Otolaryngol (Stockh) 2000;120:177–81.

[19] Demer JL. Hypothetical mechanism of head-shaking nystagmus (HSN) in man: asymmetrical velocity storage. Soc Neurosci Abstr 1985;11:1038.

[20] Karlberg M, Aw ST, Black RA, Todd MJ, MacDougall HG, Halmagyi GM. Vibration-induced ocular torsion and nystagmus after unilateral vestibular deafferentation. Brain 2003;126:956–64.

[21] Halmagyi GM, Cremer PD, Anderson J, Murofushi T, Curthoys IS. Isolated directional preponderance of caloric nystagmus, I: clinical significance. Am J Otol 2000;21:559–67.

[22] Lasker DM, Hullar TE, Minor LB. Horizontal vestibulo-ocular reflex evoked by high-acceleration rotations in the squirrel monkey. III. Responses after labyrinthectomy. J Neurophyisol 2000;83:2482–96.

[23] Asawavichianginda S, Fujimoto M, Mai M, Desroches H, Rutka J. Significance of head-shaking nystagmus in the

evaluation of the dizzy patient. Acta Otolaryngol (Stockh) 1999;540:27–33.

[24] Perez P, Llorente JL, Gomez JR, Del Campo A, Lopez A, Suarez C. Functional significance of peripheral head-shaking nystagmus. Layngoscope 2004;114:1078–84.

[25] Maire R, van Meller G. Dynamic asymmetry of the vestibulo-ocular reflex in unilateral peripheral vestibular and cochleovestibular loss. Laryngoscope 2000;110: 256–63.

[26] Aw ST, Todd MJ, Aw GE, McGarvie LA, Halmagyi GM. Benign positional nystagmus. A study of its three-dimensional spatio-temporal characteristics. Neurology 2005;64: 1897–905.

[27] Lopez-Escamez JA, Molina MI, Gámiz M, Fernandez-Perez AJ, Gomez M, Palma MJ, et al. Multiple positional nystagmus suggests multiple canal involvement in bening paroxysmal positional vertigo. Acta Otolaryngol (Stockh) 2005;125:954–61.

[28] Palla A, Marti S, Straumann D. Head-shaking nystagmus depends on gravity. J Assoc Res Otolaryngol 2005;6:1–8.

[29] Lopez-Escamez JA, Gamiz MJ, Fiñana MG, Perez AF, Canet IS. Position in bed is associated with left or right location in benign paroxysmal positonal vertigo of the posterior semicircular canal. Am J Otolaryngol 2002;23: 263–6.

[30] Von Brevern M, Seelig T, Neuhauser H, Lempert T. Benign paroxysmal positional vertigo predominantly affects the right laryrinth. J Neurol Neurosurg Psychiatry 2004;75: 1487–8.

[31] Raphan T, Matsuo V, Cohen B. Velocity storage in the vestibulo-ocular reflex arc (VOR). Exp Brain Res 1979;35: 229–48.

[32] Fetter M, Zee DS, Koenig E, Dichgans J. Head-shaking nystagmus during vestibular compensation in human and rhesus monkeys. Acta Otolaryngol (Stockh) 1990;110:175–81

[33] Main TC, Zee DS. Velocity storage in labyrinthine disorders. Ann NY Acad Sci 1992;656:297–304.