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ORIGINAL COMMUNICATION

Causes and characteristics of horizontal positional nystagmus

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Abstract Direction changing horizontal positional nystagmus can be observed in a variety of central and peripheral vestibular disorders. We tested sixty subjects with horizontal positional nystagmus and vertigo on the Epley Omniax® rotator. Monocular video recordings were performed with the right or left ear down, in the supine and prone positions. Nystagmus slow-phase velocity (SPV) was plotted as a function of time. Thirty-one subjects diagnosed with horizontal canalolithiasis had paroxysmal horizontal geotropic nystagmus with the affected ear down (onset 0.8 ± 1 s, range 0–4.9 s, duration 11.7–47.9 s, peak SPV 79 ± 67°/s). The SPV peaked at 5–20 s and declined to 0 by 60 s; at 40 s from onset, the average SPV was 1.8 % of the peak. Nine subjects diagnosed with cupulolithiasis had persistent apogeotropic horizontal nystagmus (onset 0.7 ± 1.4 s, range 0–4.3 s). Peak SPV was 54.2 ± 31.8°/s and 26.6 ± 12.2°/s with unaffected and affected ears down, respectively. At 40 s, the average SPV had decayed to only 81 % (unaffected ear down) and 65 % (affected ear down) of the peak. Twenty subjects were diagnosed with disorders other than benign positional vertigo (BPV) [vestibular migraine (VM), Ménière's Disease, vestibular schwannoma, unilateral or bilateral peripheral vestibular loss]. Subjects with VM ($n = 13$) had persistent geotropic or apogeotropic horizontal nystagmus. On average, at 40 s from nystagmus onset, the SPV was 61 % of the peak. Two patients with Ménière's Disease had persistent apogeotropic horizontal nystagmus; the peak SPV at 40 s ranged between 28.6 and 49.5 % of the peak. Symptomatic horizontal positional nystagmus can be observed in canalolithiasis, cupulolithiasis and diverse central and peripheral vestibulopathies; its temporal and intensity profile could be helpful in the separation of these entities.

Keywords Canalithiasis · Cupulolithiasis · Horizontal canal · Vestibular migraine · BPV

Introduction

Benign positional vertigo (BPV) is a common and correctable cause of episodic vertigo triggered by otoconia dislodged from the otolith membranes of the utricle into the semicircular canals. Movement of otoconia activates semicircular canal (SCC) receptors and produces a unique pattern of nystagmus that is specific to the affected canal [1, 2]. BPV can result from canalolithiasis where the otoconia are freely floating in the duct of the semicircular canal or cupulolithiasis where the otoconia are adherent to the cupula. Typically, a patient with canalolithiasis will experience a paroxysm of vertigo which is induced by head-tilt and resolves rapidly. In contrast, cupulolithiasis is characterized by persistent vertigo and nystagmus which does not subside until the patient moves away from the provocative position [3]. BPV most commonly affects the posterior canal, accounting for 60–90 % of patients; horizontal canal BPV is less common and accounts for 5–30 % of all patients [4],while anterior canal BPV is exceedingly

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rare, accounting for only 1–2.3 % [5, 6]. Typically, horizontal canalolithiasis is characterized by horizontal nystagmus beating towards the ground (geotropic nystagmus) with either ear down. Cupulolithiasis of the horizontal canal is typified by horizontal nystagmus beating away from the ground towards the uppermost ear (apogeotropic nystagmus). As dictated by Ewald's law, ampullopetal flow of endolymph in the lateral canal (excitation) evokes a stronger response than an ampullofugal flow (inhibition); therefore, canalolithiasis is expected to produce more pronounced nystagmus with the affected ear down and cupulolithiasis to produce more intense nystagmus when the unaffected ear is lowermost. Previous investigators have suggested additional methods to lateralize horizontal canal BPV [7–9]. The "bow and lean test" compares the direction of nystagmus upon leaning forwards with arching backwards; typically, canalolithiasis is reported to evoke nystagmus beating to the unaffected ear upon leaning backwards and towards the affected ear upon bowing forwards and the converse is reported in cupulolithiasis [4, 10]. "Pseudo spontaneous nystagmus" (PSN) refers to nystagmus observed in the upright position, which is not due to a fixed unilateral vestibular loss and is attributed to minor ampullofugal flow of otoconia. When the patient sits upright, PSN beats towards the healthy side, as does supine nystagmus in canalolithiasis [11].

Analysis and comparison of the spatiotemporal characteristics of horizontal positional nystagmus observed in BPV, other peripheral and central vestibular disorders has not been undertaken thus far. Here we report the nystagmus patterns of 60 patients who were found to have horizontal positional nystagmus and positional vertigo when assessed on the Epley Omniax® rotator. We sought to compare the characteristics of positional nystagmus found in canalolithiasis, cupulolithiasis and disorders other than BPV.

Methods

Between January 2009 and January 2013, 60 patients (47 women and 13 men) aged 64.5 ± 16.8 years (range 16–95) presenting with positional vertigo and horizontal positional nystagmus were recruited from a clinic dedicated to the investigation and treatment of "intractable BPV". All patients were symptomatic at the time of examination and their chief complaint was positional vertigo. Referrals were received from neurologists and otolaryngologists. Patients with asymptomatic positional nystagmus were excluded. All patients gave informed consent to testing and were studied with local ethics committee approval. A history was elicited from all patients and a neuro-otological assessment was performed. Assessment on the Epley Omniax® rotator (Vesticon, Portland, Oregon, USA) included Dix Hallpike tests, and side-lying tests during which the subject was rolled from supine to the right ear down or from supine to the left ear down positions. A majority of subjects were tested in the nose up (supine) and nose down (prone) positions as well. When horizontal canal BPV was diagnosed, upon identification of the affected ear, all subjects were treated with 360° rotations to the unaffected ear in 90° steps. Patients with a history of motion sensitivity were pre-medicated with intramuscular prochloperazine (12.5 mg) and sublingual ondansetron (8 mg). Following treatment, patients were instructed to sleep exclusively on the unaffected side for 1 week and contact the clinic for an update on the status of their symptoms.

The Epley Omniax® rotator, a motorized chair with real time video oculography was used for assessment and treatment. The chair is mounted on a two-axis rotator that allows alignment of the patient in the plane of any given canal. Eye position was recorded using an infrared video camera with a frame rate of 30 Hz [6]. All testing was conducted (in the dark) with visual fixation removed. For all subjects testing began in the sitting up position with recording of spontaneous nystagmus; afterwards, the subject was lowered to the supine position then rolled to the left or right ear down position, depending on which one was more symptomatic. For each position tested, video recordings began when the chair reached the target position; therefore, we treated recording onset as time = 0. Three subjects whose eye movement recordings were not analysable due to repeated blinking were excluded. One subject who had nonspecific positional disequilibrium and horizontal positional nystagmus, whose diagnosis was uncertain, was also excluded.

Subjects diagnosed with horizontal canal BPV were subclassified into groups with geotropic horizontal nystagmus (canalolithiasis) or apogeotropic nystagmus (cupulolithiasis). A further group of patients with horizontal positional nystagmus and positional vertigo not attributed to BPV were also characterized. Video data from the Epley Omniax® rotator were extracted and stored as *.avi* files. The onset, offset and duration of nystagmus was measured when clearly visible and uninterrupted by blinks. Custom made LabVIEW® pupil tracking software (written by HM and CT) was used to measure the horizontal and vertical slow-phase velocities (SPV). Since all patients recruited for this study had predominately horizontal positional nystagmus with less intense vertical components, we only analysed nystagmus characteristics in the horizontal plane. The gradient of these lines was used to calculate the slow-phase velocities for the entire recording. The asymmetry ratio of the peak SPV for the affected and unaffected ear was calculated with the Jongkees' formula. A polynomial curve was fitted to the velocity trace and the peak of this curve

was detected. The order of the curve fit was adjusted manually (fifth–eighth order) to maximise the accuracy of the fit. Peak slow-phase velocities for the roll test (affected vs. unaffected ear down) were statistically compared using Wilcoxon's paired test. Descriptive statistics are expressed as the mean ± SD and range.

Results

Characteristics of horizontal canalolithiasis

Thirty-one subjects were diagnosed with horizontal canalolithiasis based on (a) the presence of short-lived spells of positional vertigo (<1 min) and paroxysmal positional nystagmus, which was geotropic and changed direction with either ear down, and (b) complete symptom resolution in response to barbecue manoeuvres.

In all subjects, upon lying with the affected ear down, a paroxysm of horizontal nystagmus was observed almost immediately (average onset latency = 0.8 ± 1.1 s based on 27 subjects) and lasted between 11.7 and 47.9 s ($n = 26$). The SPV when plotted as a function of time could be fitted into a fifth–seventh order polynomial curve (Fig. 1) which peaked between 5–20 s of the onset and had always declined to zero by 60 s (range of nystagmus duration: 11.7–47.9 s). At 40 s from onset, the average SPV was 1.8 % of the peak (Fig. 2a). Only four patients had nystagmus at 40 s (SPV range at 40 s: 3.9–17.7°/s). The average peak SPV was 79.2 ± 67.0°/s (range 12.9–320.0°/s). Upon lying the subject on the unaffected side, a less intense paroxysm of horizontal nystagmus with a similar profile was observed (peak SPV 19.3 ± 15.8°/s; range 2.7–40.2°/s). The asymmetry ratio of the peak SPV was 54.4 ± 32.5 % (range 1.5–90.2 %).

Twenty-nine patients had their eye movements recorded in the supine position during the course of testing. Twenty-seven subjects had supine nystagmus (average peak SPV 20.2 ± 21.6°/s), two had no recordable nystagmus. Of the 27 demonstrating nystagmus in the supine position, 20 had horizontal nystagmus beating to the unaffected ear but seven had horizontal nystagmus beating to the affected ear. (i.e. contrary to bow and lean criteria). Ten of 13 subjects tested in the prone position had horizontal nystagmus with an average peak SPV of 30.1 ± 35.2°/s and three had no recordable nystagmus. Seven had nystagmus beating to the affected ear and three patients had nystagmus beating to the unaffected ear.

Twenty-nine patients were diagnosed on the basis of Ewald's law where the side with more vigorous nystagmus on visual inspection was considered to be the affected side. In three patients, the nystagmus appeared symmetrical; therefore, the affected side was determined by the direction

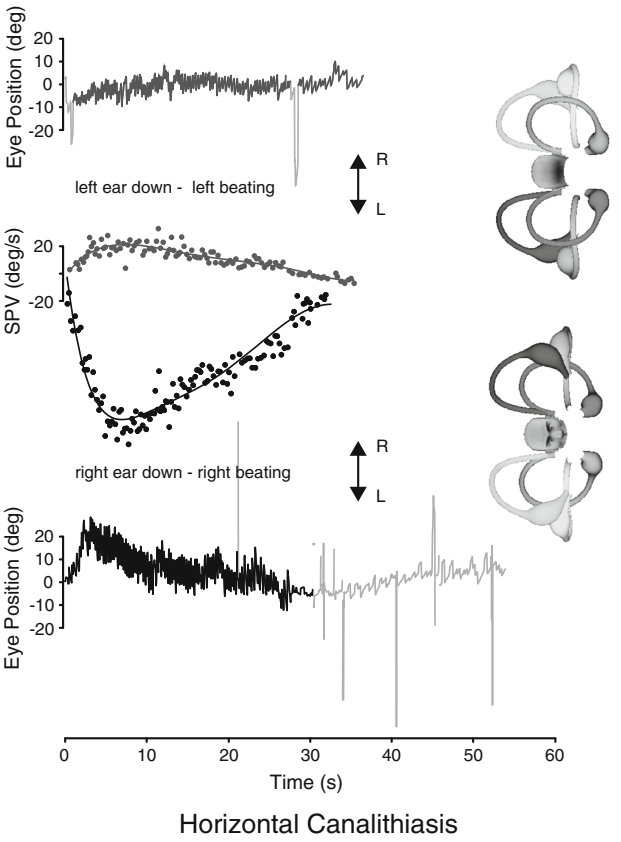

Fig. 1 Comparison of *left* and *right* roll test in a subject with *right-sided* horizontal canalolithiasis. Testing the subject in the right roll position reveals a violent paroxysm of right beating nystagmus with no latency and a high peak slow-phase velocity. The SPV had returned to baseline by 40 s. The *left roll* test produces a paroxysm of nystagmus with almost no latency, a short duration and a smaller peak SPV

of the supine nystagmus alone. Eighteen of the 27 patients in whom supine and/or prone nystagmus was sought, also fulfilled the bow and lean criteria. Upon using the Wilcoxon signed rank test, the peak SPV with the affected ear down was significantly greater than with the unaffected ear down ($p < 0.001$). On comparing prone with supine nystagmus there was no significant difference in peak SPV ($p = 1.0$).

All patients were treated with barbecue manoeuvres towards the unaffected ear, aimed at shifting the canaliths ampullofugaly. Repeat treatment was only offered when the symptoms persisted 1 week after repositioning. Twenty-one patients were successfully treated with one treatment only; seven patients were treated with two sessions; four patients required up to four treatments for horizontal canalolithiasis. All patients were given the contact details of the clinic and instructed to make a phone call at the end of 1 week in the event of persistent symptoms. During a 4-year period, five patients had recurrences of BPV in another canal, three patients had recurrences of BPV in the same canal.

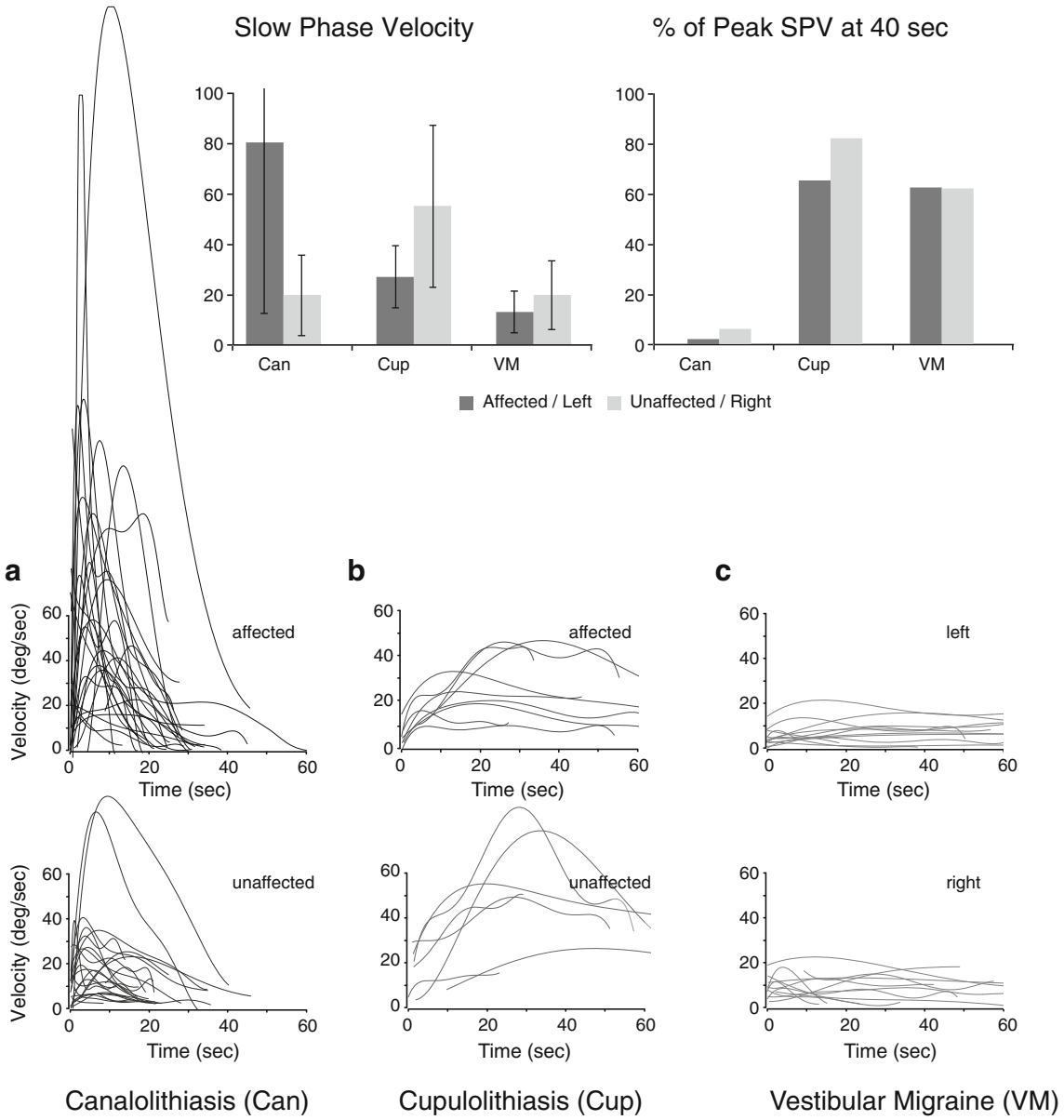

Fig. 2 The slow-phase velocity profiles of canalolithiasis, cupulolithiasis and VM. The *bar charts* compare average peak SPV and the % SPV at 40 s for all three conditions. The SPV of canalolithiasis show the crescendo–decrescendo velocity profiles and marked asymmetry with affected and unaffected ears down. Cupulolithiasis shows a variable SPV profile with a slower decay of SPV than for canalolithiasis. A majority of subjects diagnosed with VM, had symmetrical, flat SPVs with either ear down

Reversal of geotropic to apogeotropic nystagmus

Eleven patients, upon completion of one or more barbecue manoeuvres, demonstrated reversal of the direction of horizontal nystagmus from the geotropic to the apogeotropic form. This finding was attributed to the canaliths entering the short arm of the horizontal canal; therefore, treatments were ceased and the subjects were instructed to sleep on the unaffected side for $\sim$1 week and reassessed either by a follow-up appointment or phone consultation to ensure the symptoms had abated. The high frequency of conversion from geotropic to apogeotropic was attributed to the multiple barbecue manoeuvres performed on any given subject. None of these subjects had ongoing positional vertigo after 1 week.

Characteristics of horizontal cupulolithiasis

Nine subjects were diagnosed with horizontal cupulolithiasis on the basis of persistent positional vertigo and direction-changing apogeotropic horizontal nystagmus. None had associated aural symptoms, focal neurological

symptoms or headaches that were temporally related to the vertigo. Although three patients had a past history of migraine headaches, they responded completely or partially to barbecue manoeuvres and were, therefore, thought to represent cupulolithiasis rather than VM. Seven (who did not recover after a single manoeuvre) underwent magnetic resonance imaging of the brain to exclude a central cause.

Typically in the roll position, persistent apogeotropic nystagmus was observed almost immediately after onset (0.7 ± 1.4 s, $n = 9$). Recordings lasted between 22.5 and 110 s. Testing the subjects with the unaffected ear down showed nystagmus with an average peak SPV of 54.2 ± 31.8°/s. On rolling the subject onto the affected side, a less intense paroxysm of apogeotropic nystagmus (peak SPV 26.6 ± 12.2°/s) was observed (Fig. 3). Eight subjects had supine nystagmus with average peak SPV of 18.1 ± 10.8°/s. Four of the nine subjects had nystagmus in the prone position, with an average peak SPV of 31.3 ± 41.0°/s. The SPV profiles for cupulolithiasis were diverse (Fig. 2b). Three subjects had clearly identifiable peak SPV at 20, 30 and 35 s after onset. SPV plotted as a function of time could be fitted into a sixth–eighth order polynomial curve. At 40 s the SPV had decayed to only 65.9 % of the peak SPV on the affected side and to 81 % of the peak SPV on the unaffected side. The average asymmetry ratio for the peak SPV was 42.2 ± 24.7 %.

On paired Wilcoxon tests, the peak SPV with the affected ear down was significantly lower than with the unaffected ear down $p = 0.008$. All nine patients were diagnosed on the basis of Ewald's law using SPV measures; seven had clinically identifiable SPV asymmetry. Seven also fulfilled the bow and lean criteria. In one patient, the converse of the bow and lean test was observed; in one patient the bow and lean test was not performed.

All patients were treated with high velocity 360° barbecue manoeuvres (in 90° increments) towards the unaffected ear with mastoid vibration applied to the affected ear. Two patients had resolution of symptoms after a single treatment. One patient was treated on two sessions, two on three, one patient on four. Two patients underwent seven treatments and one underwent a total of 15 sessions. Three (of nine) patients experienced complete symptom resolution following treatment on the Epley Omniax® rotator. One further patient was treated with repeated head shaking in the yaw plane (for a period of 1–2 min at hourly intervals) over an 8 h period, and experienced complete symptom resolution within 1 week. Three patients had partial symptom resolution with residual nystagmus on

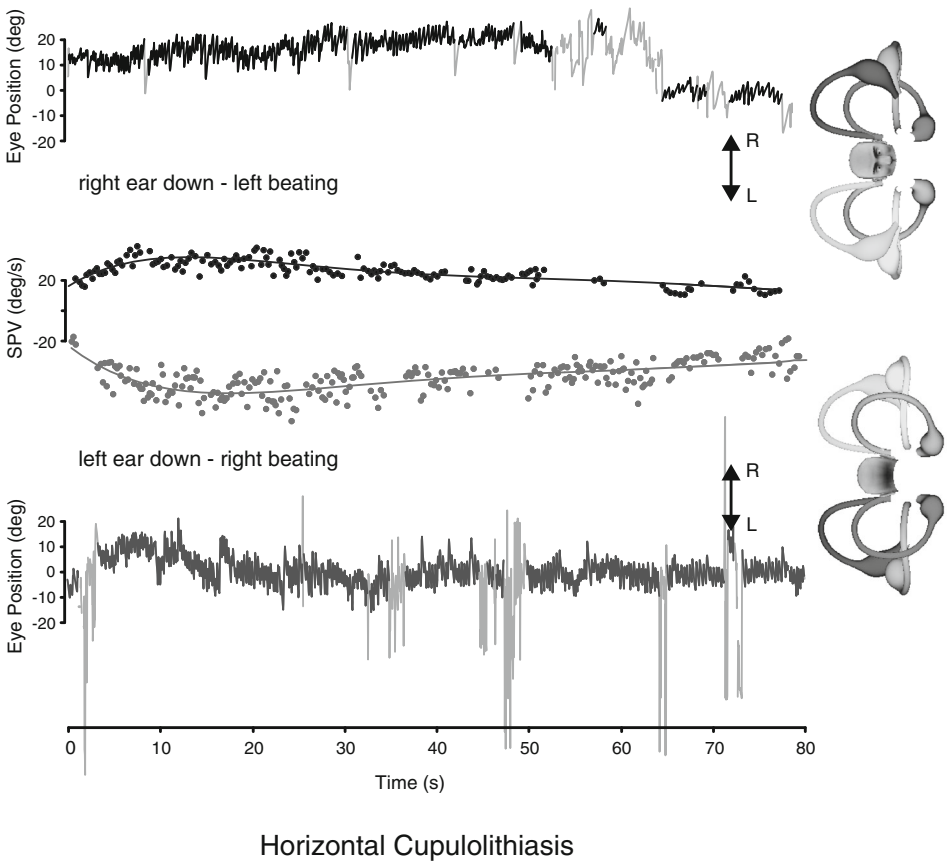

Fig. 3 Comparison of *right* and *left* roll-tests in a subject with *left horizontal* cupulolithiasis. With the affected and unaffected ears down, apogeotropic nystagmus was recorded. The peak SPV was 54.8° and 32.4° with the unaffected and affected ears down. With the unaffected and affected ears down, the SPV had declined to 81.8 and 64.8 % of the peak SPV at 40 s, respectively

testing. However, two patients had no improvement after 15 and seven treatments, respectively; both were trialled on empirical migraine preventative therapy without significant changes in symptoms.

Positional vertigo with atypical direction changing horizontal nystagmus

Twenty subjects with positional vertigo and horizontal direction changing positional nystagmus were diagnosed with disorders other than BPV. These diagnoses were reached on the basis of (a) absence of typical paroxysmal positional nystagmus indicative of canalolithiasis, presence of persistent positional nystagmus and positional vertigo during an examination performed on a symptomatic day; (b) no history of previous recurrent BPV to imply cupulolithiasis; (c) history, examination findings and vestibular function tests indicative of an alternate cause for positional vertigo such as vestibular migraine (VM), endolymphatic hydrops, vestibular schwannoma, unilateral or bilateral vestibulopathy.

According to the Neuhauser criteria [12, 13] 13 patients were diagnosed with VM (three clinically definite and 10 clinically probable). The average SPV on either ear down was symmetrical (peak SPV 12.7 ± 8.1°/s on left vs. 19.4 ± 13.5°/s on right, asymmetry ratios 30.9 ± 24.4 range 0.07–68.9 %). Unlike the crescendo-decrescendo SPV profile observed in canalolithiasis, the SPV profile of most migraineurs was flat (Figs. 2c, 4). At 40 s from onset, the average SPV had declined to 63 and 61 % of its peak value on the left and right side down, respectively. One subject, whose nystagmus profile is illustrated in Fig. 3, was tested ictally. Five of the VM patients had geotropic horizontal nystagmus. Eight subjects had apogeotropic nystagmus. All patients experienced symptom resolution after initiation of migraine prophylaxis.

Two patients who first presented with positional vertigo were found to have clinically definite Ménière's Disease (MD). Both had apogeotropic horizontal nystagmus. In one patient, the peak SPV was symmetrical (4.6°/s left vs. 5.3°/s right), whereas for the second patient it was asymmetrical on the roll test (19.6°/s right ear down vs. 7.6°/s left ear down). Both had electrocochleography with elevated SP/AP ratios consistent with endolymphatic hydrops, with either negative summating potentials or an elevated SP/AP ratio.

One subject, who had apogeotropic nystagmus (R ear down: peak SPV 32°/s > L ear down: peak SPV 3°/s) was found to have a right-sided vestibular schwannoma with impaired audio-vestibular function on the right side. His

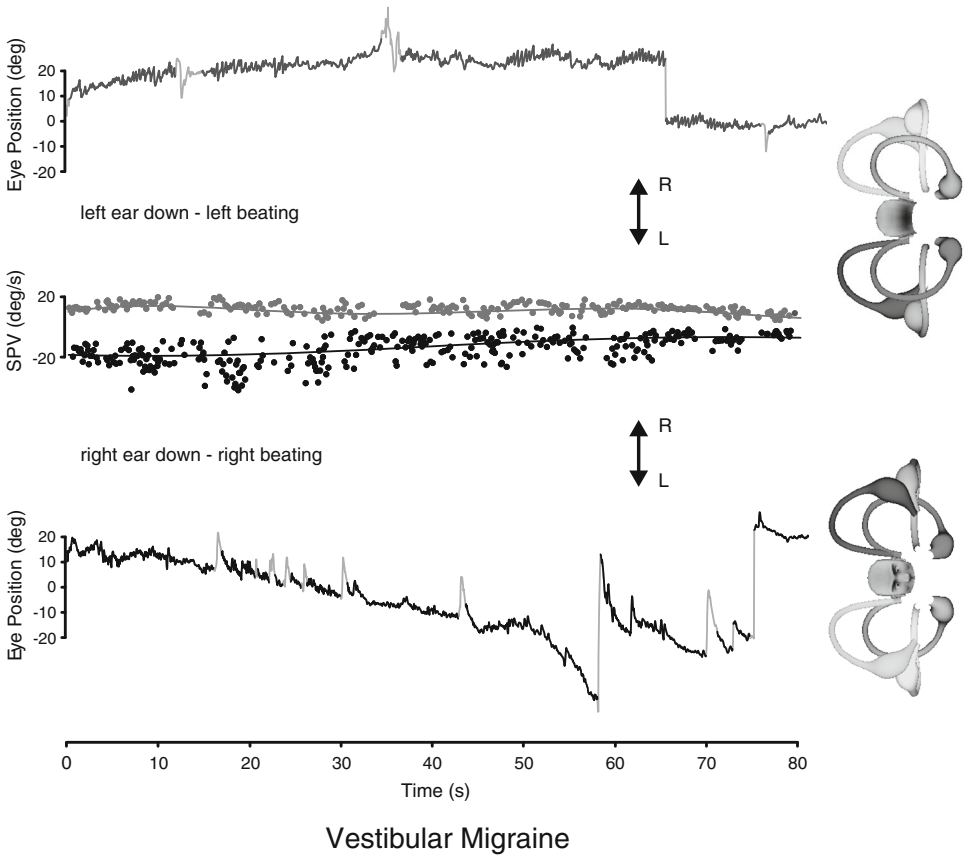

Fig. 4 In a subject with clinically definite VM, the *left* and *right* roll test revealed persistent geotropic horizontal nystagmus with equally flat SPV profiles with either ear down. The peak SPV were 13.0 and 21.0° with the left and right ears down

vertigo was mild, unresponsive to barbecue manoeuvres, lasted several months and abated spontaneously, leaving residual asymptomatic positional nystagmus.

Two patients with unilateral vestibulopathy of uncertain origin had low velocity apogeotropic horizontal nystagmus. One patient with otosclerosis and a left vestibulopathy had geotropic nystagmus with symmetrical SPV. One subject with ANCA positive autoimmune inner ear disease with bilateral vestibular hypofunction, as indicated with absent caloric responses, positive video Head Impulse Test (vHIT) and absent vestibulo-ocular responses to sinusoidal rotation, had persistent geotropic horizontal nystagmus with either ear down. The vestibular function test results of all the above patients are summarized in an supplementary table.

Discussion

We present the first detailed comparison of slow-phase velocity profiles of canalolithiasis, cupulolithiasis and atypical positional nystagmus. As described by previous investigators, horizontal canalolithiasis produced a vigorous paroxysm of geotropic nystagmus when tested with the affected ear down. The onset latency was shorter than that reported in posterior canal BPV and the duration of the paroxysm was longer than described for the posterior canal [1]. The short and relatively symmetrical onset latencies we recorded with the affected and unaffected ears down in canalolithiasis implies a rapid movement of the otoconia in the horizontal canals in response to provocative testing.

Testing the unaffected side revealed a paroxysm of nystagmus with similar SPV profiles but lower peak amplitudes. The wide range of peak SPVs recorded in this study probably represents many factors influencing nystagmus velocity, including the volume of otoconia, the endolymph viscosity and the order testing. In this study, we demonstrated that nearly all subjects diagnosed with canalolithiasis obeyed Ewald's law. Lower rates of accuracy for the bow and lean test may be explained by the sequence in which we conducted tests. Since subjects always underwent roll tests before supine or prone testing, it is possible that the starting position of the otoconia was not the lowest part of the duct of the horizontal canal.

In cupulolithiasis, rolling the subject to the unaffected side elicited nystagmus with a peak SPV double that recorded rolling to the affected ear down. Average peak SPV for the side with the more vigorous nystagmus was smaller for cupulolithiasis than for canalolithiasis. Unlike canalolithiasis, the SPV had not decayed to zero at 40 s (Fig. 3). As observed in canalolithiasis, a wide range of peak SPV were recorded; these may have reflected the volume of otoconia attached to the cupula and endolymph viscosity. Although we would have expected the latency to nystagmus onset to be shortest for cupulolithiasis, onset latencies for canalolithiasis were so short that it was not possible to elicit a meaningful latency difference within the resolution of our methods. Upon comparison of prone and supine SPV, no specific trend for a greater nystagmus intensity in either position was found. This might be explained by variability in the position of the cupula of the horizontal canal in humans, which lies very close to the sagittal plane and is likely to deflect in the ampullopetal or ampullofugal directions in the prone position. Thus, it possible for either excitatory or inhibitory nystagmus to be elicited in the prone position [14].

In the present study, geotropic horizontal nystagmus was recorded from subjects with both horizontal canalolithiasis and VM. Horizontal positional nystagmus accompanying canalolithiasis had a distinctive velocity profile that enabled its recognition: a crescendo-decrescendo SPV and its rapid decline by 60 s separated canalolithiasis from central positional nystagmus. Additionally, the peak SPV was higher for canalolithiasis when compared with atypical positional nystagmus.

Our group with atypical positional nystagmus was heterogeneous. Those with VM, overall, had symmetrical, persistent, low velocity nystagmus that easily separated them from canalolithiasis when they presented with geotropic positional nystagmus. In contrast, as shown in Figs. 2, 3 and 4, the SPV profiles of VM and horizontal cupulolithiasis were strikingly similar. Although the average SPV were lower for VM there was too much overlap of the peak SPV between cupulolithiasis and VM to enable their effective separation. Several previous authors have reported horizontal positional nystagmus as a physical finding in subjects with VM. Polensk and Tusa [15] found 100 % of patients with acute VM had positional nystagmus and that horizontal positional nystagmus had the highest prevalence. Von Brevern et al. [16] reported both geotropic and apogeotropic horizontal positional nystagmus and vertigo presenting in association with acute VM. Roberts et al. [17] reported a patient with positional vertigo and apogeotropic horizontal nystagmus that was refractory to repositioning manoeuvres who subsequently developed migraine headaches and experienced symptom resolution upon treatment with migraine preventative therapy.

Of the patients within the VM group, only three fulfilled the criteria for clinically definite VM. Three subjects within the cupulolithiasis group had a past history of migraine. Seven of the 13 subjects diagnosed with VM had profound motion sensitivity. One patient in the cupulolithiasis group also had motion sensitivity but responded to a single barbecue manoeuvre. While the classification of these subjects was influenced by their response to migraine preventative therapy vs. repositioning manoeuvres, this

study highlights the uncertainties surrounding the classification of the patient with persistent apogeotropic positional nystagmus. Could cupulolithiasis have triggered secondary migraines thus leading to a diagnosis of "clinically probable VM"? Conversely, could the presence of coincidental migraines and motion sensitivity have interfered with successful repositioning, resulting in "intractable cupulolithiasis"? Further ictal studies in clinically definite VM and further trials of repositioning manoeuvres in subjects with apogeotropic horizontal nystagmus are required to definitively resolve these questions.

Thus far, apogeotropic horizontal positional nystagmus has not been described in subjects with endolymphatic hydrops. Early descriptions of ictal nystagmography in Ménière's Disease report direction changing geotropic positional nystagmus in 25 % of recordings [18]. Bergenius et al. [19] reported persistent geotropic positional nystagmus in six patients with vertigo; they attributed the nystagmus to a "light cupula" resulting from altered homeostasis of proteoglycans synthesized by the cupulae. Both geotropic and apogeotropic horizontal nystagmus are also observed during the "resorption" and "reduction" phase of positional alcohol nystagmus (PAN I and II) when alcohol first diffuses into the cupula making it lighter than endolymph, then leaves the cupula in advance of the endolymph resulting in a relatively heavy cupula [20, 21]. Whether some stages of Ménière's Disease are associated with changes in endolymph specific gravity resulting in derangement of the neutral buoyancy of the cupula remains to be proven. One patient with Ménière's Disease and apogeotropic nystagmus had more intense nystagmus with the affected ear down. This finding, however, is contrary to what is expected with a heavy cupula, where a more intense nystagmus is observed with the unaffected ear down.

Our observations and those of previous authors indicate that apogeotropic horizontal positional nystagmus should raise a wide range of differentials including central positional nystagmus from nodular lesions [22, 23] or VM, endolymphatic hydrops and even vestibular schwannoma [24, 25].

Conclusion

Direction changing horizontal positional nystagmus can be of diverse aetiology. The nystagmus direction, SPV profile and symmetry provide useful diagnostic information that compliments the history and physical examination when separating canalolithiasis, cupulolithiasis and atypical nystagmus secondary to other vestibular disorders. Although not commonly used in clinical practice, recording the slow-phase velocity profile of nystagmus may be useful in subjects with positional vertigo that is refractory to conventional bedside treatments. While typical canalolithiasis is clearly distinguishable from the geotropic horizontal nystagmus of VM, cupulolithiasis is difficult to separate from positional nystagmus and vertigo observed in various central and peripheral vestibular disorders; its diagnosis should be made with these differentials in mind.

Conflicts of interest The authors declare that they have no conflict of interest.

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