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ORIGINAL ARTICLE

Long-term prognosis of hearing loss in patients with unilateral Ménière's disease

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Abstract

Conclusion: The finding of deteriorated hearing loss at the initial visit at middle to high frequencies is a factor of poor hearing prognosis in Ménière's disease. Early intervention with instructions for lifestyle changes may lead to good outcomes in hearing. *Objective:* An attempt was made to examine long-term changes in hearing loss in unilateral Ménière's disease and factors associated with prognosis of hearing loss retrospectively. *Methods:* Based on their last hearing level of the affected ear, 36 patients were subdivided into two groups: the poor prognosis of hearing (PPH) group and the good prognosis of hearing (GPH) group. *Results:* In the PPH group, the hearing levels at the initial visit at middle and high frequencies were significantly worse than those in the GPH group. Moreover, the hearing loss progressed during the first 2 years of the disease, and stayed flat to approximately 50 dB at the later stage. Conversely, the hearing loss at the onset in the GPH group showed no further progression over the first 2 years, and remained constant to approximately 35 dB at the later stage. In addition, the mean intervals from the onset to the initial visit in the PPH group were significantly longer than those in the GPH group.

Keywords: *Endolymphatic hydrops, vertigo attack, early intervention*

Introduction

Ménière's disease is an intractable inner ear disease that is characterized by episodes of recurrent vertigo with hearing loss, tinnitus, and aural fullness. Its pathophysiology is recognized to be idiopathic endolymphatic hydrops [1,2].

Previous studies reported that in most patients with Ménière's disease, the frequency of vertigo attacks decreased gradually after the onset until it reached a steady-state phase free of vertigo [3,4]. On the other hand, it was also reported that sensorineural hearing loss in Ménière's disease deteriorated progressively until it reached a moderate or severe level [5–7]. Since the vertigo attacks decrease or disappear in the long-term course, the goal of treatment for Ménière's disease should be to prevent the progression of hearing loss that causes a reduction in the quality of life in the patients [4,8,9].

In the present study, an attempt was made to retrospectively examine long-term changes in hearing loss during follow-up in patients with unilateral Ménière's disease. We divided the patients into two groups based on their last hearing level of the affected ear: (1) the poor prognosis of hearing (PPH) group and (2) the good prognosis of hearing (GPH) group, and then investigated the factors associated with the prognosis of hearing loss.

Material and methods

A total of 36 patients (14 males and 22 females; 24–75 years old; mean age 47.6 ± 13.3 years) with unilateral definitive Ménière's disease according to

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the 1995 guidelines of the American Academy of Otorhinolaryngology and Head and Neck Surgery (AAO-HNS) were included in the present study (Table I) [10]. They were referred to University Hospital from private ENT clinics, because of their intractable Ménière's attacks.

The onset of Ménière's disease was determined by detailed interview at the initial visit to the hospital. After the initial visit, hearing levels were evaluated and the frequency of vertigo attacks was requested at each follow-up visit. The mean follow-up period was 49.2 ± 34.3 months after the initial visit to University Hospital. In 17 patients, their referrals reported the audiograms containing hearing level at the onset of the disease. This retrospective study was approved by the Committee for Medical Ethics of Tokushima University Hospital.

According to a grading system to assess the severity of symptoms in patients with Ménière's disease proposed by the Intractable Vestibular Disorder Committee of the Ministry of Health and Welfare of Japan, the patients were subdivided into two groups based on their last hearing level of the affected ear: (1) the poor prognosis of hearing (PPH) group, whose hearing levels were over 40 dB in all 125, 250, 500, 1000, 2000, 4000, and 8000 Hz; (2) the good prognosis of hearing (GPH) group, whose hearing levels were lower than 40 dB in at least one of 125, 250, 500, 1000, 2000, 4000, and 8000 Hz [11].

The averaged hearing levels were calculated every 3 months and changes in hearing levels at every 3-months period after the onset of the disease were evaluated. The hearing levels at the low (125–500 Hz), middle (500–2000 Hz), and high (2000–8000 Hz) frequencies were then analyzed separately. The progression of hearing levels during vertigo attack was estimated by the formula of the averaged hearing level of 125–8000 Hz at the 3-month period when patients experienced vertigo attacks minus the averaged level of the pre-vertigo attack period. The progression of hearing levels after vertigo attack was estimated by subtracting the averaged hearing level between 125 and 8000 Hz at the 3-month period when patients experienced vertigo attacks from that of the post-vertigo attack period.

Table I. Patients with unilateral Ménière's disease at the initial visit.

Characteristic	PPH group (n = 19)	GPH group (n = 17)
Mean age (years)	47.1 ± 13.3	46.8 ± 16.4
Sex		
Male (%)	6 (32)	8 (47)
Female (%)	13 (68)	9 (53)
Mean hearing level at low frequencies (dB)	48.0 ± 19.8	40.3 ± 14.1
Mean hearing level at middle frequencies (dB)	41.8 ± 18.7*	28.9 ± 11.3
Mean hearing level at high frequencies (dB)	45.6 ± 15.2*	32.4 ± 11.9

Values are shown as mean ± SD. GPH, good prognosis of hearing; PPH, poor prognosis of hearing.
*$p < 0.05$.

After the initial visit, the patients received betahistine and/or anti-emetic during a vertigo attack, and isosorbide, an osmotic diuretic, for at least 3 months after a vertigo attack. They also received instructions to avoid mental and physical stress in their lifestyle. Patients who underwent surgery such as endolymphatic sac surgery and intratympanic injection with gentamicin were excluded from the present study.

The Mann–Whitney test was used for statistical analysis and $p < 0.05$ was considered statistically significant.

Results

The mean hearing levels at the initial visit to the hospital in Ménière's patients with PPH were 48.0 ± 19.8 dB at low frequency, 41.8 ± 18.7 dB at middle frequency, and 45.6 ± 15.2 dB at high frequency, while in those with GPH, they were 40.3 ± 14.1 dB, 28.9 ± 11.3 dB, and 32.4 ± 11.9 dB, respectively (Table I). Thus the mean hearing levels at the initial visit in patients with PPH at middle and high frequencies, but not at low frequencies, were significantly worse than those in patients with GPH.

In patients with PPH, the mean hearing levels at the onset of the disease that were reported in their referrals at low, middle, and high frequencies were 39.3 ± 12.7 dB, 31.2 ± 8.5 dB, and 38.8 ± 8.6 dB, respectively. Two years after the onset, they deteriorated rapidly to 53.5 ± 22.2 dB, 50.9 ± 21.7 dB, and 51.1 ± 16.3 dB before stabilizing, respectively, at 44.4 ± 5.9 dB, 50.6 ± 7.9 dB, and 59.4 ± 6.7 dB 8 years later (Figure 1). On the other hand, in patients with GPH, the mean hearing levels at the onset of the disease that were reported in their referrals at the same frequencies were 39.8 ± 15.1 dB, 26.5 ± 10.7 dB, and 30.3 ± 11.3 dB, respectively. Two years after the onset, they deteriorated slightly to 44.3 ± 13.4 dB, 33.8 ± 10.2 dB, and 35.7 ± 11.9 dB, and then became stable at 35.8 ± 27.1 dB, 34.2 ± 24.6 dB, and 34.2 ± 5.9 dB, respectively, 8 years later (Figure 2).

The mean progression of hearing levels during vertigo attacks was significantly larger than that after vertigo in patients with both PPH and GPH (Figure 3). The mean progressions of hearing levels during vertigo attack in patients with PGH and GPH

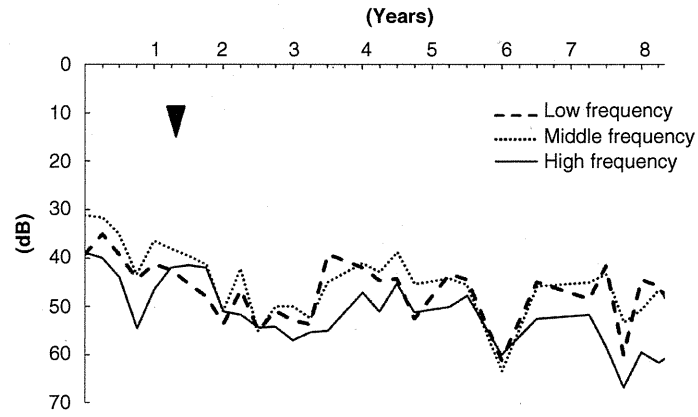

Figure 1. Time course of the mean hearing levels at low, middle, and high frequencies in Ménière's patients with poor prognosis of hearing after the onset of the disease. Arrowhead indicates their mean interval from the onset to the initial visit.

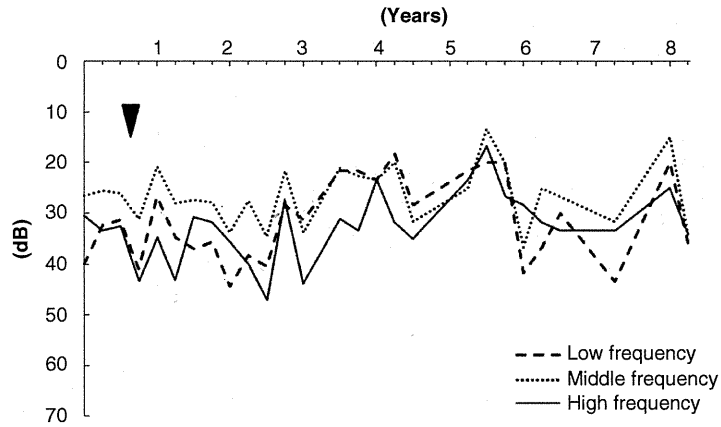

Figure 2. Time course of the mean hearing levels at low, middle, and high frequencies in Ménière's patients with good prognosis of hearing after the onset of the disease. Arrowhead indicates their mean interval from the onset to the initial visit.

were 3.3 ± 11.4 dB and 2.6 ± 11.9 dB, respectively. After vertigo attacks, the mean progression of hearing levels in patients with PPH was 0.48 ± 11.6 dB, whereas that of those with GPH was –3.6 ± 10.5 dB, indicating an improvement of hearing levels after vertigo attacks.

The percentage of patients who suffered from vertigo attacks every 3 months decreased rapidly

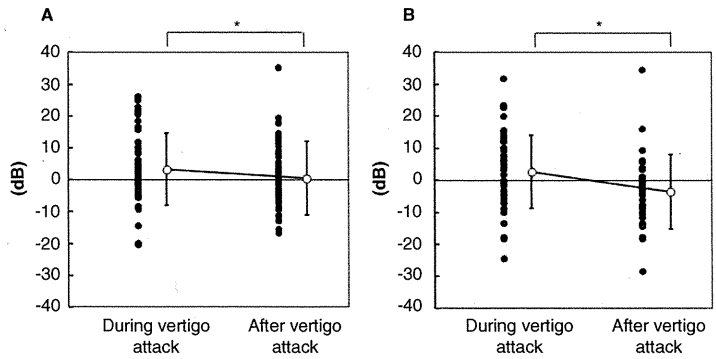

Figure 3. Changes in hearing levels during and after vertigo attack in Ménière's patients with poor prognosis of hearing (A) and good prognosis of hearing (B). Mean ± SD. *$p < 0.05$.

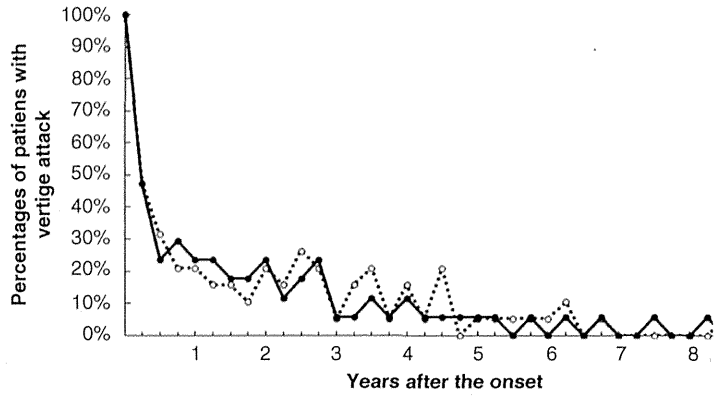

Figure 4. Time course of the percentages of Ménière's patients who suffered from a vertigo attack during every 3-month period. Filled circles, patients with poor prognosis of hearing; open circles, patients with good prognosis of hearing.

during the first 3 years after the onset of the disease in both PPH and GPH groups, and then 8 years later stabilized at less than 20% (Figure 4). The mean interval from the onset to the initial visit in patients with PPH (15.5 ± 18.7 months) was significantly longer as compared with that of those with GPH (7.6 ± 11.7 months) (Figure 5).

Discussion

In the present study, the mean hearing levels at the initial visit to the hospital in Ménière's patients with PPH were significantly worse than those with GPH at middle and high frequencies but not at low frequencies. It is suggested that the deteriorated hearing loss at the initial visit at middle to high frequencies is a factor of poor prognosis of the hearing loss in patients with Ménière's disease, because the hearing loss at low frequencies is fluctuating and reversible at the early stage of the disease [5] and that at high frequencies tends to be irreversible and progressive [6,7].

The present study also showed that in patients with PPH, the hearing loss progressed during the first 2 years after the onset of the disease, and stayed flat to approximately 50 dB at the later stage of Ménière's disease. Conversely, the hearing loss at the onset in patients with GPH showed no further progression over the first 2 years, and became constant to approximately 35 dB at the later stage of the disease. These findings suggest that the rapid progression of hearing loss during the first 2 years leads to poor hearing outcomes in patients with Ménière's disease. Indeed, many studies found that the hearing loss in Ménière's disease deteriorated over time within the first several years of the disease and was followed by a stabilization of moderate to severe hearing loss [4,12,13]. Thomas and Harrison reported that the progression of hearing loss was more

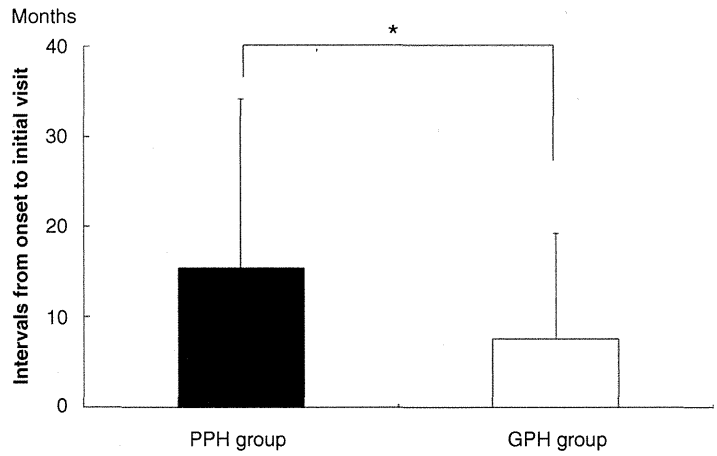

Figure 5. The mean intervals from the onset to the initial visit in Ménière's patients with poor prognosis of hearing (PPH) and good prognosis of hearing (GPH). Mean ± SD. *$p < 0.05$.

common during the first 5 years [12], whereas Stahle reported it to occur within the first 2 years [13].

During follow-up, the mean hearing levels worsened by about 3 dB during the vertigo attacks in Ménière's patients with both PPH and GPH. However, the deteriorated hearing loss of 3 dB improved after the vertigo attack in patients with GPH but not in those with PPH. It is possible that the progression of hearing loss during vertigo attacks became irreversible in patients with PPH who suffered from repeated vertigo attacks. This is because an intractable vertigo attack was suggested to be a factor for poor hearing prognosis, based on the findings that hearing loss took place in the early course of Ménière's disease before the relief in vertigo attack [3,4]. However, the possibility is unlikely, because the present study showed that the percentages of patients who suffered from a vertigo attack every 3 months decreased rapidly during the first 3 years after onset of the disease and that there were no differences in these percentages between patients with PPH and those with GPH. Previous studies also reported that the frequency of vertigo attacks decreased over time in most patients with Ménière's disease [14]. Perez-Garrigues et al. also reported that the frequency of vertigo attacks showed a rapid decline during the first 8 years of the disease [3]. Thus, the frequency of vertigo attack seems to have little effect on hearing prognosis of Ménière's disease. It is suggested that hydrops itself, but not the rupture of hydrops, leads to hair cell degeneration, because a vertigo attack in Ménière's disease is induced by its rupture [15].

Another possibility of the irreversible progression of hearing levels during the vertigo attack in patients with PPH is the higher susceptibility of their inner ear to endolymphatic hydrops. Recently, Moon, et al. reported that a high SP/AP ratio on electrocochleogram was a predictor of poor hearing outcomes in patients with Ménière's disease and suggested that excessive endolymphatic hydrops associated with a high SP/AP ratio led to hair cell degeneration and malfunction [16]. Therefore, the inner ear of patients with PPH may be more susceptible to endolymphatic hydrops than that of those with GPH.

In the present study, patients with GPH visited the hospital and were treated significantly earlier than those with PPH, suggesting that early intervention is effective in preventing the progression of hearing loss in patients with Ménière's disease. Patients received isosorbide, an osmotic diuretic, for at least 3 months after vertigo attack, because a Japanese randomized controlled trial showed that isosorbide was more effective in suppressing vertiginous symptoms than betahistine, but had no effect on hearing loss in patients with Ménière's disease [17]. Contradicting this observation, the Cochrane Library indicates that no good evidence regarding the effect of diuretics on Ménière's disease has been previously reported [18]. Therefore, the good prognosis of hearing associated with the early intervention may not be due to the treatment with osmotic diuretic. On the other hand, in the present study, patients with Ménière's disease also received the instruction to operate changes in their lifestyle, especially to avoid mental and physical stress, because Ménière's disease is recognized as a stress-induced disease [7]. Indeed, it has been reported that counseling is more effective in improving the hearing level in patients with Ménière's disease than drug treatment [19], suggesting that it decreases levels of stress hormones such as vasopressin, which increases the extent of endolymphatic hydrops [20]. Therefore, it is suggested that early intervention with instructions for changes in lifestyle leads to good outcomes of hearing in patients with Ménière's disease.

Conclusion

In the present study, we showed that the deteriorated hearing loss at the initial visit at middle and high frequencies is a factor for poor hearing prognosis in patients with Ménière's disease. The hearing loss further progressed over the first 2 years, and stayed flat at approximately 50 dB later, indicating poor outcomes of hearing. In Ménière's patients with PPH, the hearing level worsened by about 3 dB during vertigo attacks, after which the loss was irreversible. Because the frequency of vertigo attacks in patients with PPH was not different from that of those with GPH, it is suggested that the inner ear in patients with PPH is more susceptible to endolymphatic hydrops. Conversely, the hearing loss in patients with GPH did not progress after the onset, probably because the mean interval from the onset to the initial visit to the hospital in patients with GPH was shorter than that of those with GPH, indicating the need for early intervention with instructions for changes in lifestyle. This may lead to reducing the extent of endolymphatic hydrops and to good outcomes of hearing in patients with Ménière's disease.

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Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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BMJ Open

Light cupula: the pathophysiological basis of persistent geotropic positional nystagmus

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ABSTRACT

Objective: To clarify the pathophysiological basis of persistent geotropic positional nystagmus (PGN) in patients with the horizontal canal type of benign paroxysmal positional vertigo (H-BPPV), the time constant (TC) of nystagmus and the relationship between its slow phase eye velocity (SPV) and the angle of head rotation in supine were defined.

Methods: Geotropic or apogeotropic positional nystagmus was recorded by video-oculography and analysed three-dimensionally.

Results: Geotropic positional nystagmuses in patients with H-BPPV were classified as transient geotropic positional nystagmus with a TC of ≤35 s or PGN with a TC of >35 s. Alternatively, the TC of persistent apogeotropic positional nystagmus (AN) in patients with H-BPPV was >35 s. The direction of the SPV of patients with PGN was opposite to that of patients with AN at each head position across the range of neutral head positions. The relationship between the SPV of patients with PGN and the angle of head rotation was linearly symmetrical against that of patients with AN with respect to a line drawn on the neutral head position.

Conclusions: Since its TC was >35 s, it is suggested that PGN is induced by cupula deviation in response to gravity at each head position. It is also suggested that the direction of cupula deviation in patients with PGN is opposite to that of patients with AN across the neutral head positional range with no nystagmus where the long axis of cupula is in alignment with the axis of gravity. Since the pathophysiological basis of AN is considered a heavy cupula, it is suggested that PGN is conversely induced by a light cupula.

Strengths and limitations of this study

- In this study, the benign paroxysmal positional nystagmus was precisely analysed in three dimensions.
- This is the first study to clarify the relationship between slow phase eye velocity of persistent geotropic positional nystagmus and the angle of head rotation.
- The light cupula theory in this study was discussed under the assumption that apogeotropic positional nystagmus is caused by a heavy cupula.

INTRODUCTION

Benign paroxysmal positional vertigo (BPPV) is the most common peripheral vestibular disease, and it is usually caused by involvement of the posterior and/or horizontal semicircular canal (HSCC).[1] In particular, horizontal positional nystagmus in the supine position of patients with the HSCC type of BPPV (H-BPPV) consists of the geotropic type (fast phase towards the ground) as well as the apogeotropic type (fast phase away from the ground).[2] Geotropic positional nystagmus is induced by canalolithiasis—free-floating debris—in HSCC patients when the head is rotated to either side in a supine position.[3] Thus, transient geotropic positional nystagmus (TGN) disappears rapidly when the head position is maintained. However, in HSCC patients, apogeotropic positional nystagmus is induced by a deviation of the cupula and attached debris, known as cupulolithiasis, in response to the head position.[4] Thus, persistent apogeotropic positional nystagmus (AN) lasts longer than TGN even when the head position is maintained.[5]

Recently, another type of geotropic nystagmus—persistent geotropic positional nystagmus (PGN)—has been reported in patients with H-BPPV.[6] In the present study, an attempt was made to clarify the pathophysiology of PGN in patients with H-BPPV. We examine the time constant (TC) of this type of nystagmus and the relationship between its slow phase eye velocity (SPV) and the angle of head rotation in a supine position.

METHODS

Patients

This study includes a total of 107 patients with H-BPPV who visited the Department of Otorhinolaryngology—Head and Neck

Surgery of the Osaka University Hospital between 15 February 2011 and 31 May 2013, reporting positional vertigo, showing geotropic or apogeotropic positional nystagmus and having no central lesion identified by MRI. Written informed consent was obtained from all patients before the study, which was performed in line with the Helsinki II Declaration. The study is also reported in accordance with the guidelines of standards for the reporting of diagnostic accuracy studies (STARD). The patients who could not consent to this study, those who had additional positional nystagmus of the posterior canal type of BPPV (P-BPPV) and those who showed transition from apogeotropic nystagmus to geotropic nystagmus were excluded (figure 1).

The patients lay in the supine position and their heads were rotated quickly or stepwise. Their positional nystagmus was then recorded by video-oculography (720×480dot, 30 Hz) using *RealEyes* (Micromedical Technologies). The angle of the head in the supine position and the rotational velocity around three-dimensional axes of *WAA-006* sensors (ATR-Promotions) were also recorded with a sensor of linear acceleration in three dimensions.

Analysis of head position

To record the angle of head rotation, *WAA-006* sensors were attached to *RealEyes* goggles and the sensors' X, Y and Z axes were aligned with the patient's nasooccipital, interaural and dorsoventral axes, respectively. The head position was calculated by applying the atan2 function in *Excel* software (Microsoft) to the *WAA-006* data (atan2(z, y)). *WAA-006* sensors can record the information of the computer timer. Using this information, the data of *WAA-006* sensors were synchronised to the video of eye movement.

Analysis of positional nystagmus and SPV of positional nystagmus

Positional nystagmus was recorded on a Windows computer with the goggles. The digital movie of positional nystagmus was converted to 720×480dot Jpeg images and analysed using an algorithm.[7] In this study, eye movements were three-dimensionally described by rotation vectors, which characterise the eye positions around a single axis. An eye position can be reached by rotating the eye from the reference position around a single axis. The eye position is represented by a vector around the axis, the length of which is proportional to the angle of rotation. The reference position was defined as the eye position when the patient was looking straight ahead with the head in an upright position. Straight ahead was defined as looking at a target located horizontally in front of the eye.[8] The method of analysing the eye rotation vector and its accuracy have already been described elsewhere.[7 9] The spatial coordinates of the pupil centre and an iris freckle were reconstructed in three dimensions and were defined as follows: the X axis, parallel to the nasooccipital axis (positive forward); the Y axis, parallel to the interaural axis (positive left) and the Z axis, normal to the X–Y plane (positive upwards). The X, Y and Z components mainly reflect the roll, pitch and yaw components, respectively. The direction of rotation was described from the patient's point of view. Regarding the X component, 'right torsional' indicates that the superior pole of the eyeball rotates towards the right ear and "left torsional" indicates rotation towards the left ear. The rotation vector $\mathbf{r}$, describing a rotation of θ around the axis $\mathbf{n}$, was given by the formula $\mathbf{r}=\mathbf{n}\tan(\theta/2)$, with $\mathbf{n}$ being the unit vector, whose direction represents its axis. We used the Euler angle parameter that was given as $2\times\tan^{-1}$ (magnitude of rotation vector) to represent the eye position as an axis angle.[10 11] Using the following formula: $\boldsymbol{\omega}=2\times(d\mathbf{r}/dt+\mathbf{r}\times d\mathbf{r}/dt)/(1+\mathbf{r}^2)$, we calculated the eye velocity $\boldsymbol{\omega}$ around the X, Y and Z axes.[8] We then extracted the SPV of the nystagmus by a fuzzy set-based approach.[12 13]

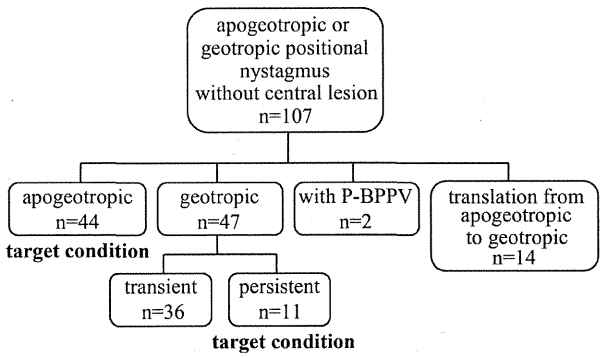

Figure 1 Classification of patients (P-BPPV, posterior canal type of benign paroxysmal positional vertigo).

Calculating TC

All patients in this study showed rightward and leftward horizontal nystagmus because the nystagmus was either geotropic or apogeotropic. When calculating TC, we used the rightward positional nystagmus when the SPV of rightward nystagmus was larger than that of leftward nystagmus, and vice versa. We based our calculations on a 30 s period from the time that the SPV reached its maximum value. When the direction of nystagmus along the Z component was reversed within the 30 s period, we used the data from the time that the SPV reached the maximum value to the time just before the sign of the Z component of the SPV changed. Using the least squares method, the SPV against time in three components was approximated exponentially. Finally, TC was calculated as the reciprocal of the coefficient of time.[14]

Approximating a formula describing the relationship between SPV and head rotation angle in supine subjects

We excluded the data reflecting the vestibulo-ocular reflex during head movement from the data of apogeotropic or geotropic nystagmus with a long TC and used the remaining data to plot the Z component of the SPV on the Y axis

and the head position on the X axis. Using the least squares method, the plotted data were approximated by the following formula: $\alpha\sin(x-\theta)$ $(x\geq\theta)$, $\beta\sin(x-\theta)$ $(x<\theta)$. The data of this function between −90° and 90° were approximated by quartic function, because this function is non-differentiable at point $x=\theta$ but is continuous.

RESULTS

A total of 107 eligible patients with H-BPPV (22 men and 85 women, median age 68 years, age range 36–88 years) who reported positional vertigo and showed geotropic or apogeotropic positional nystagmus were included in this study (figure 1). We excluded 2 patients who had an additional positional nystagmus of type P-BPPV, and 14 patients who showed a transition from apogeotropic nystagmus to geotropic nystagmus. As shown in figure 1, of the 107 patients, apogeotropic positional nystagmus was seen in 44 patients with H-BPPV (10 men and 34 women, median age 70.5 years, age range 36–88 years) and geotropic positional nystagmus in 47 patients with H-BPPV (10 men and 37 women, median age 67 years, age range 39–82 years). The three-dimensional eye position and SPV of PGN in patient A (a 44-year-old woman) is shown in figures 2A and 3A. A left-torsional and leftward horizontal nystagmus was induced by head rotation to the left and a right-torsional and rightward horizontal nystagmus was induced by head rotation to the right (figure 2A). The left-torsional and leftward horizontal nystagmus at the left-ear-down head position declined very gradually and was calculated by the following formulas:

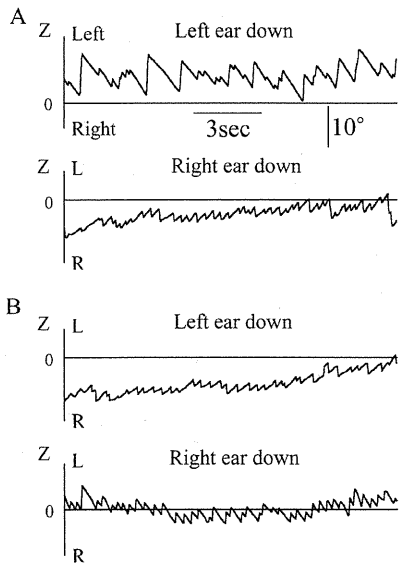

Figure 2 Eye position of positional nystagmus in the right-ear-down and left-ear-down head positions in patients with horizontal canal type of benign paroxysmal positional vertigo. (A) Persistent geotropic positional nystagmus in patient A and (B) persistent apogeotropic positional nystagmus in patient B.

$3.9\exp((t_1-t)/121)$ in the X component, $1.9\exp((t_1-t)/121)$ in the Y component and $-9.0\exp((t_1-t)/121)$ in the Z component (t_1 represents the time when SPV was at a maximum), with a TC of 121 s. The maximum SPV of left-torsional and leftward horizontal nystagmus at the left-ear-down head position was higher than that of the right-torsional and rightward horizontal nystagmus induced in the right-ear-down head position (figure 3A). The three-dimensional eye position and SPV of AN in patient B (a 74-year-old woman) are shown in figures 2B and 3B. A left-torsional and leftward horizontal nystagmus was induced by head rotation to the right while a right-torsional and rightward horizontal nystagmus was induced by head rotation to the left (figure 2B). The left-torsional and leftward horizontal nystagmus at the right-ear-down head position declined very gradually and was calculated by the following formulas: $1.8\exp((t_4-t)/2897)$ in the X component, $-0.3\exp((t_4-t)/2897)$ in the Y component and $-5.3\exp((t_4-t)/2897)$ in the Z component (t_4 represents the time when SPV was maximum), with a TC of 2897 s. The maximum SPV of the left-torsional and leftward horizontal nystagmus in the right-ear-down head position was higher than that of the right-torsional and rightward horizontal nystagmus in the left-ear-down head position (figure 3B).

The TC of patients with TGN ranged from 4.9 to 32.2 s (median: 11.7 s). The distribution of TCs of 11 patients with PGN and of 44 patients with AN is shown in figure 4. The TCs of patients with PGN ranged from 66 to 3600 s (median: 168 s). The TCs of patients with AN ranged from 35.3 to 3600 s (median: 127 s). Changes in the SPV induced by stepwise rotation in patient A with PGN are shown in figure 5A. Rightward nystagmus was induced when the head was rotated 61° to the right and gradually disappeared with the stepwise rotation of the head to the left. Leftward nystagmus was then induced by further stepwise rotation of the head to the left. The SPV of rightward and leftward nystagmus changed stepwise in accordance with the stepwise rotation of the head. However, no nystamus was induced when the head was rotated 24° to the left to a neutral head position. Changes in the SPV induced by stepwise rotation in patient B with AN are shown in figure 5B. Rightward nystagmus was induced when the head was rotated 57° to the left and gradually disappeared with stepwise rotation of the head to the right. Leftward nystagmus was then induced by further stepwise rotation to the right. The SPV of rightward and leftward nystagmus changed stepwise in accordance with the stepwise rotation of the head. However, no nystagmus was induced when the head was rotated 27° to the left to a neutral head position. The relationship between the angle of head rotation and the SPV induced by stepwise head rotation in patient A with PGN (shown in figure 5A) was plotted in XY coordinates, with the SPV plotted on the Y axis against the angle of head rotation on the X axis (figure 6A). The relationship was approximated by the following formula:

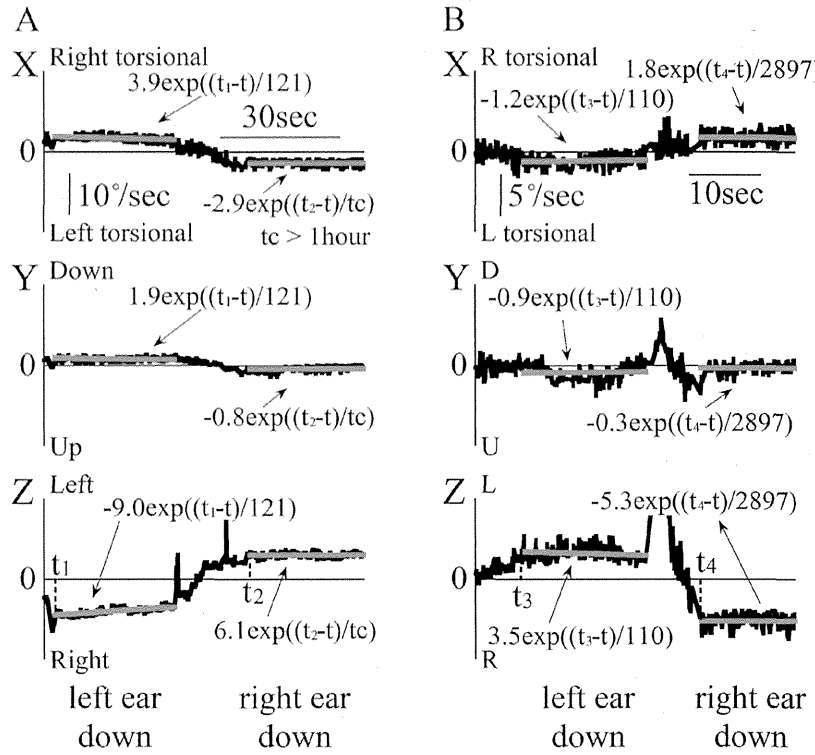

Figure 3 Three-dimensional X, Y and Z components of eye velocity of positional nystagmus in right-ear-down and left-ear-down head positions in patients with horizontal semicircular canal type of benign paroxysmal positional vertigo. (A) Persistent geotropic positional nystagmus lasted with long TCs (time constant) of 121 s and >1 h in patient A and (B) AN lasted with long TCs of 110 and 2897 s in patient B.

$y=8.9\sin(x+149.0)$ $(x\geq 31.0°)$, $4.1\sin(x+149.0)$ $(x<31.0°)$, where x is the Euler angle. Values of x between −90° and 90° were approximated by the quartic function $y=2\times10^{-8}x^4+4\times10^{-7}x^3-0.000x^2-0.068x+2.257$, where x is the Euler angle, or $y=0.236x^4+0.073x^3-2.357x^2-3.918x+2.257$, where x is the angle in radians.

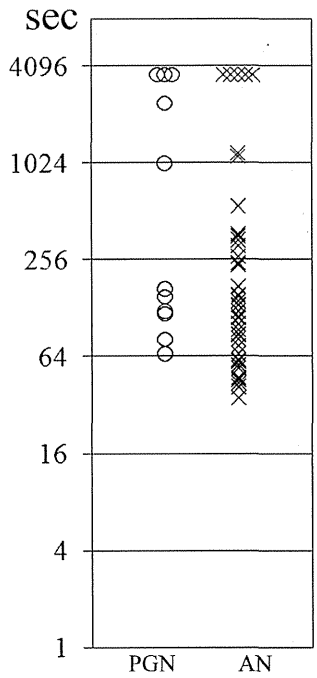

Figure 4 Distribution of time constants (TCs) of positional nystagmus in 11 patients with persistent geotropic positional nystagmus (PGN) and 44 patients with AN. TCs of all 44 patients with AN was more than 35 s. TCs of 11 patients with PGN was more than 35 s. When TC was more than 1 hour, we set the TC value at 3600 s.○: TC of PGN, ×: TC of AN.

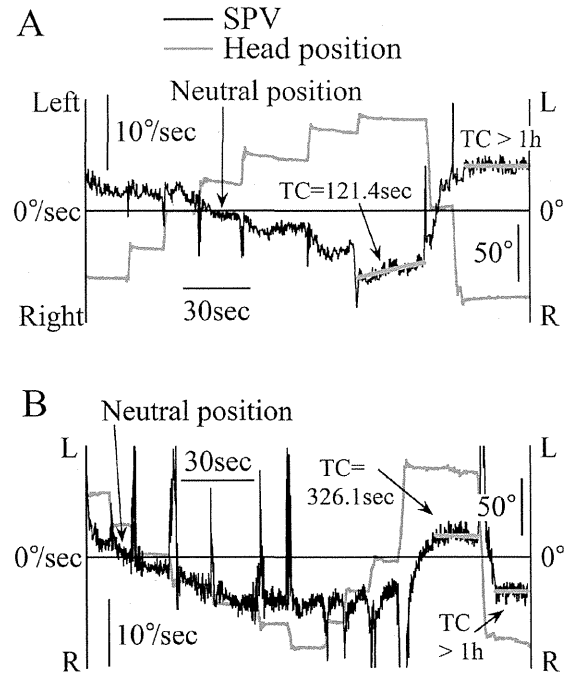

Figure 5 Changes in the slow phase eye velocity (SPV) of positional nystagmus induced by stepwise rotation of the head in patients with horizontal canal type of benign paroxysmal positional vertigo. (A) Persistent geotropic positional nystagmus in patient A and (B) AN in patient B (TCs, time constant).

The relationship between the angle of head rotation and the SPV induced by stepwise head rotation in patient B with AN (shown in figure 5B) was plotted in XY coordinates, with the SPV plotted on the Y axis against the angle of head rotation on the X axis (figure 6B). The relationship was approximated by the following formula: $y=3.6\sin(x-22.0)$ $(x\geq 22.0°)$, $7.0\sin(x-22.0)$ $(x<22.0°)$, where x is the Euler angle. Values of x between −90° and 90° were approximated by quartic function $y=2\times10^{-8}x^4-6\times10^{-6}x^3+2\times10^{-8}x^2+0.100x-2.545$, where x is the Euler angle, or $y=0.165x^4-1.128x^3+7\times10^{-5}x^2+5.747x-2.545$, where x represents the angle in radians.

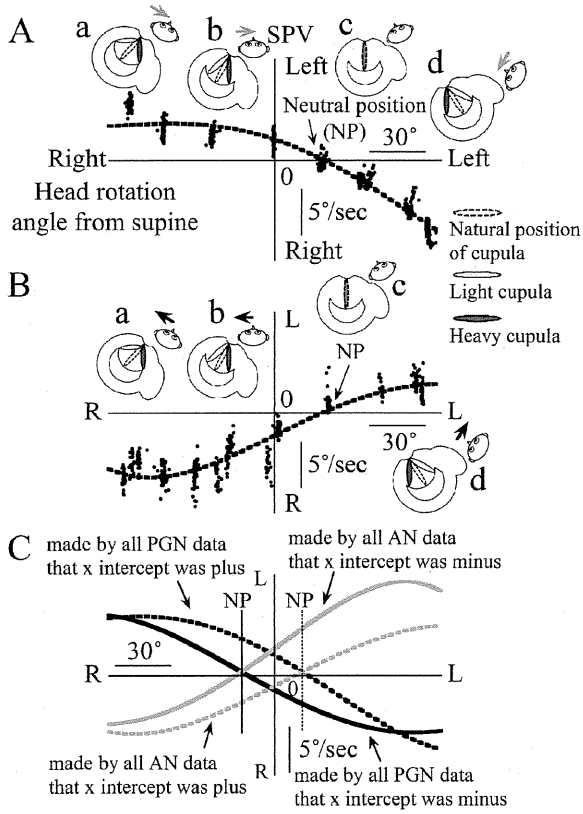

Figure 6 The relationship between the angle of head rotation and the slow phase eye velocity (SPV) of positional nystagmus in patients with horizontal canal type of benign paroxysmal positional vertigo. (A) The relationship between the angle of head rotation and the SPV of persistent geotropic positional nystagmus (PGN) induced by stepwise rotation of the head in patient A as shown in figure 5A was plotted in XY coordinates on the Y axis against the angle of head rotation on the X axis, (B) The relationship between the angle of head rotation and the SPV of AN induced by stepwise rotation of the head in patient B as shown in figure 5B was plotted in XY coordinates on the Y axis against the angle of head rotation on the X axis. Inserted figures: light cupula and heavy cupula in the left horizontal semicircular canal. a: Head turning to the right, b: in the supine position, c: in the neutral head position and d: head turning to the left. Arrows near face: the direction of positional nystagmus. (C) The approximated relationship between the angle of head rotation and the SPV of PGN at the leftward neutral head position (black dotted line) and at the rightward neutral head position (black solid line). The approximated relationship between the angle of head rotation and the SPV of AN in the leftward neutral head position (grey dotted line) and in the rightward neutral head position (grey solid line).

In 7 of the 11 patients with PGN, the relationship had positive x-intercepts, which indicates the absence of nystagmus at the neutral head position when the head had been rotated 15–20° to the left. The relationship was approximated by the formulas $y=4\times10^{-8}x^4-8\times10^{-6}x^3-0.001x^2-0.208x+4.040$, where x is the Euler angle, and $y=0.481x^4+1.457x^3-3.519x^2-11.95x+4.040$, where x represents the angle in radians. The approximated relationship between the angle of head rotation and the SPV of PGN in these seven patients is shown by a black dotted line in figure 6C. However, in the other four patients, the relationship had negative x-intercepts, indicating the absence of nystagmus at the neutral head position when the head had been rotated 15–20° to the right. The relationship was approximated by the formulas $y=-2\times10^{-8}x^4-8\times10^{-6}x^3-0.000x^2-0.196x-2.936$, where x is the Euler angle, and $y=-0.214x^4+1.592x^3+1.901x^2-11.28x-2.936$, where x represents the angle in radians. The approximated relationship between the angle of head rotation and the SPV of PGN in these four patients is shown by a black solid line in figure 6C.

The relationship between the angle of head rotation and the SPV of 44 patients with AN with a TC of >35 s was approximated by quartic function. In 23 of the 44 patients, the relationship had positive x-intercepts, which indicates the absence of nystagmus at the neural head position when the head had been rotated 15–20° to the left. The relationship was approximated by the formulas $y=9\times10^{-9}x^4-9\times10^{-6}x^3+0.000x^2+0.189x-2.163$, where x represents the Euler angle, and $y=0.097x^4-1.655x^3+0.360x^2+10.84x-2.163$, where x represents the angle in radians. The approximated relationship between the angle of head rotation and the SPV of AN in these 23 patients is shown by a grey dotted line in figure 6C. In the other 21 patients, the relationship had negative x-intercepts, indicating an absence of nystagmus at the neutral head position when the head had been rotated 15–20° to the right. The relationship was approximated by the formulas $y=-5\times10^{-8}x^4-1\times10^{-5}x^3+0.000x^2+0.268x+5.376$, where x is the Euler angle, and $y=-0.556x^4-2.790x^3+0.622x^2+15.35x+5.376$, where x represents the angle in radians. The approximated relationship between the angle of head rotation and the SPV of these 21 patients with AN is shown by a grey solid line in figure 6C. The dashed black lines show the approximated relationship between the angle of head rotation and the SPV of patients with PGN with a TC of >35 s, which was linearly symmetrical against the dashed grey line showing the approximated relationship between the

angle of head rotation and the SPV of patients with AN with respect to a line on positive x-intercepts of the neutral head position (figure 6C). The solid black line shows the approximated relationship between the angle of head rotation and the SPV of patients with PGN with a TC of >35 s, which was linearly symmetrical against the solid grey line showing the approximated relationship between the angle of head rotation and the SPV of patients with AN with respect to a line on negative x-intercepts of the neutral head position (figure 6C).

DISCUSSION

In this study, three-dimensional analysis of positional nystagmus showed that the TCs of PGN and AN were longer than those of TGN in patients with H-BPPV. The distributions of TCs of geotropic positional nystagmus in patients with H-BPPV were segregated into two groups: patients with PGN with TCs of >35 s and patients with TGN with TCs of ≤35 s. In contrast, the TCs of patients with AN were distributed over 35 s (figure 4). These findings suggest that PGN and AN have a common pathophysiology underlying positional nystagmus which is different from that of TGN. Since TGN is induced by canalolithiasis in HSCC and AN is induced by cupulolithiasis in HSCC, it is also suggested that a lesioned HSCC cupula induces PGN in patients with H-BPPV.

In this study, the relationship between the angle of head rotation and the SPV of positional nystagmus was quantitatively examined in patients with H-BPPV. PGN and AN were induced by stepwise rotation of the head (figure 5). Moreover, in PGN and AN, no nystagmus was induced in the neutral head position (figures 5 and 6A, B). These findings support the hypothesis that PGN is induced by a lesioned cupula in HSCC, as with the cupulolithiasis of HSCC in patients with AN. However, the direction of PGN was opposite to that of AN. Indeed, at the right-ear-down head position, right-beating nystagmus was induced in patients with PGN, while left-beating nystagmus was induced in patients with AN. In the left-ear-down head position, left-beating nystagmus was induced in patients with PGN, while right-beating nystagmus was induced in patients with AN (figures 2 and 3). Therefore, it is suggested that the response of the lesioned HSCC cupula to the head position in patients with PGN was opposite to that in those with AN.

The approximated relationship between the angle of head rotation and the SPV of PGN induced by a stepwise rotation of the head was linearly symmetrical with that of AN with respect to a line plotting the neutral head position in patients with H-BPPV (figure 6). Moreover, the approximated relationship between the angle of head rotation and the SPV of 4 patients with PGN was linearly symmetrical with the SPV of 21 patients with AN with respect to a line plotting the neutral head position where the head was rotated approximately 18° to the right (figure 6C). The approximated relationship between the angle of head rotation and the SPV of 7 patients with PGN was linearly symmetrical with that of 23 patients with AN with respect to a line plotting the neutral head position where the head was rotated approximately 18° to the left (figure 6C). Since the abnormally heavy cupulolithiasis deviates in response to gravity in patients with AN, these findings suggest that the lesioned cupula deviates in an opposite direction in response to gravity in patients with PGN (inserted figures in figure 6A, B). Moreover, in the neutral head position where the head was rotated approximately 18° to either the right or the left, the long axis of the HSCC cupula was in alignment with the gravitational axis, and no deviation of either the heavy or the lesioned cupula was induced (inserted 'c' in figure 6A, B). Altogether, these findings suggest that a lesioned HSCC cupula acts as a light cupula that deviates to the opposite direction of a heavy cupula in response to gravity and induces PGN in a neutral head position in patients with H-BPPV. Previous studies hypothesised that the characteristics of PGN are due to a light cupula.[6 15 16] In this study, however, quantitative three-dimensional analysis of the response of patients with PGN to changes in head position demonstrated that lesioned HSCC cupulae deviate similarly to light cupulae in response to gravity and induced PGN in patients with H-BPPV. Bergenius and Ichijo have also speculated that the attachment of debris of lower density makes a cupula in HSCC lighter than the endolymph.[6 15 16] Alternatively, a substance in the blood, such as alcohol, which has lower density than the endolymph, could diffuse into the cupula earlier than the endolymph by way of its proximity to capillaries, rendering the cupula lighter than the endolymph.[16] The attachment of debris of lower density is a feasible explanation because usually only one side is affected.

Since the cupula is laterally tilted in the supine position (inserted 'b' in figure 6A, B), a slight head rotation to the affected side while in this position can align the axis of light and heavy cupulae (inserted 'c' in figure 6A, B). Four patients with PGN and 21 patients with AN reported a neutral head position when the head was rotated to the right. The right side in these patients was affected. However, the left side was affected in 7 patients with PGN (including patient A) and 23 patients with AN (including patient B) who reported a neutral head position when the head was rotated to the left. Based on Ewald's second Law,[15] the flow of endolymph towards the ampulla of HSCC results in greater stimulation than flow away from the ampulla. When the left side is affected, a head rotation to the left away from *the perceived neutral head position* (inserted 'c' in figure 6A, B) results in a deviation of light cupulae in the ampullopetal direction (inserted 'd' in figure 6A) and heavy cupulae in the ampullofugal direction (inserted 'd' in figure 6B). As shown in figure 6A, B, in patient A, who presents with a light left cupula, head rotation to the left (affected side) from *the neutral head position* caused a greater SPV than head rotation to the right (healthy side) (8.9sin(x+149) vs 4.1sin(x+149)). In

patient B, who presented with a heavy left cupula, head rotation to the left (affected side) from *the neutral head position* caused a lesser SPV than head rotation to the right (healthy side) ($3.6\sin(x-22.0)$ vs $7.0\sin(x-22.0)$).

Geotropic positional nystagmus was subjectively subdivided into two groups—TGN and PGN—based on the length of the sustained period of positional nystagmus. In this study, we showed that the TC of the SPV of positional nystagmus is an objective index of transient or persistent positional nystagmus and proposed that a TC of <35 s indicates a transient type of PGN while a TC of >35 s indicates the persistent type.

In conclusion, in this study, we showed that the direction of the SPV of PGN with a TC of >35 s was opposite to that of AN with a TC of >35 s in response to the same neutral head position where no nystagmus was induced, and that the relationship between the SPV of PGN and the angle of head rotation was linearly symmetrical to that of AN with respect to a line plotting the neutral head position. These findings suggest that the direction of cupula deviation in PGN is opposite to that of AN across the neutral head position, where the long axis of the cupula is in alignment with the gravitational axis. Since AN was induced by a heavy cupula, we concluded that the basic pathophysiological mechanism of PGN is a light cupula in patients with H-BPPV. This light cupula theory is important for the explanation of morbidity associated with H-BPPV.

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Light cupula: the pathophysiological basis of persistent geotropic positional nystagmus

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原　著

少量注水法に対応するエアーカロリック検査の刺激条件の検討

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The temperature of aural air stimulation equivalent to aural stimulation with water at 20℃ in the caloric test.

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In the present study, we examined the temperature of aural air stimulation that was equivalent to aural stimulation with water at 20℃ in the caloric test. In 10 ears of 5 healthy volunteers, the maximum slow phase eye velocities (MSPEVs) of nystagmus induced by aural stimulation with air at 22℃, 46℃ and 16℃ were the same as those with water at 30℃, 44℃ and 20℃, respectively. These findings suggest that caloric stimulation with air at 16℃ is equivalent to that with water at 20℃ that is commonly used in the caloric test in Japan. The MSPEVs of nystagmus induced by aural stimulation with air at 16℃ were over 20°/sec in all ears except one, in which the MSPEV was 19.7°/sec. The criteria of canal paresis where the MSPEV of caloric nystagmus induced by aural stimulation with water at 20℃ is less than 20°/sec can be used in the caloric test with aural air stimulation at 16℃.

Key words: caloric test, canal paresis caloric nystagmus, aural air stimulation

はじめに

カロリック（温度刺激）検査は，左右耳の半規管を個別に刺激し，誘発された温度眼振から左右別の半規管機能を評価する重要な前庭機能検査である[1)]。外耳道に温水または冷水を注水することにより温度刺激を与え，外側半規管に対流による内リンパ流動を引き起こし，温度眼振を誘発する。

カロリック検査では，一般に注水刺激が用いられるが，最近では外耳道に温風または冷風を通風して温度刺激を与えるエアーカロリック検査も普及しつつある。臨床検査技師等に関する法律では，臨床検査技師は外耳道に注水することができないが，エアー刺激であれば行えるためである。注水刺激に比べてエアー刺激はより長い刺激時間が必要という欠点があるが，鼓膜穿孔があっても

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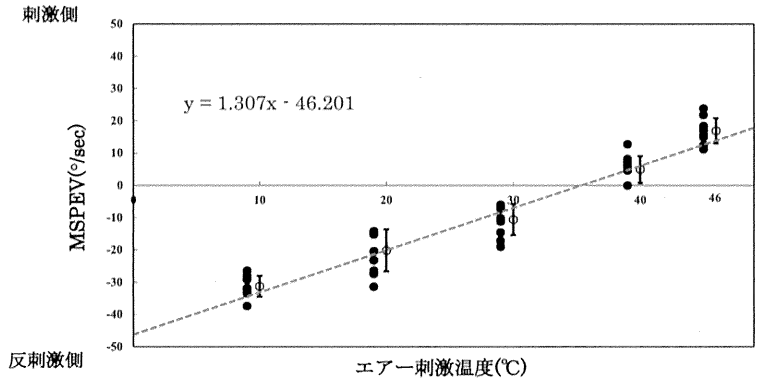

図1 エアー刺激の温度（10℃，20℃，30℃，40℃，46℃）と誘発された温度眼振のMSPEVとの関係。平均値±標準偏差。

感染の危険性がない点が長所である。

カロリック検査は，30℃と44℃の注水による冷温交互法が標準的である[2]。これに対して，2002年に日本めまい平衡医学会から提案されたエアーカロリック検査基準化案では，冷温交互刺激で用いる30℃と44℃の注水に対応するエアー刺激として，温度が26℃以下と46℃以上で6～8 lの通風，60秒刺激と提案された[3]。一方，日本では，カロリック検査として20℃の冷水を用いる少量注水法が広く用いられている[4]。しかし，少量注水法で用いる20℃の冷水刺激に対応するエアー刺激については提案されていない。本研究では，日本で普及している定性的な少量注水法をエアーカロリック検査で行うため，20℃の冷水刺激に対応するエアー刺激の温度について検討を行った。

対象と方法

耳疾患の既往および聴覚・平衡障害のない健常成人5名（年齢：26～35歳，男性：4名，女性：1名）の10耳に対して，エアー刺激と注水刺激により外耳道に温度刺激を与えてカロリック検査を行った。なお全ての被検者に対し，本研究内容を充分に説明し同意を得た。エアー刺激はエアーカロリック装置（第一医科株式会社製　FAC-700）を用い，エアー刺激の流量と刺激時間は，日本めまい平衡医学会のエアーカロリック検査基準化案[3]に従って行った。ノズルにはストッパーを付けて外耳道口から1 cm挿入し，外耳道後上壁を刺激した。エアー刺激の温度は10℃，20℃，30℃，40℃，46℃で行った。誘発された温度眼振を電気眼振計（ENG）により記録し，最大緩徐相速度（maximum slow phase eye velocity, MSPEV）を測定した。エアー刺激の温度と誘発された温度眼振のMSPEVとの関係は，刺激耳向きの眼振のMSPEVを正，非刺激耳向きの眼振のMSPEVを負とし，相関回帰分析法により近似線を求めた。

冷温交互法では，被検者を仰臥位にして外側半規管が鉛直位になるように頭部を30°前屈させ，18 G鈍針を装着した注射器で30℃と44℃の水20 mlを10秒間で外耳道後壁に向けて注入した[1]。少量注水法では，被検者を仰臥位にして頭部を枕で30°前屈させ，次に枕の上で側位をとらせた。先端に軟性チューブをつけた18 G鈍針を装着した注射器で20℃の水5 mlを10秒間で外耳道後壁に向けて注水し，さらに10秒間，側位を保ち，注水開始20秒後に正面頭位に戻した[5]。誘発された温度眼振を暗所開眼下のENGにより記録し，温度眼振のMSPEVを測定した。

30℃，44℃注水の冷温交互法，20℃の少量注水法によるカロリック検査と同じMSPEVの温度眼振を誘発できるエアー刺激の温度は，近似線からそれぞれ22℃，46℃，16℃であると考えられたため，同じ健常成人5名10耳に対して22℃，46℃，16℃のエアー刺激を流量6 l/min，刺激時間60秒で与え，誘発された温度眼振のMSPEVを測定した。同一耳において30℃，44℃注水の冷温交互法，20℃の少量注水法の注水刺激で誘発された温度眼振のMSPEVと，対応する22℃，46℃，16℃のエアー刺激で誘発された温度眼振のMSPEVとの相関を，ピアソンの相関係数の検定を用いて評

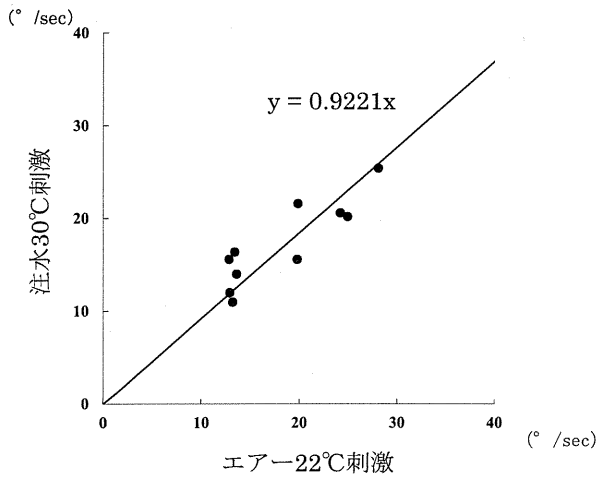

図2 エアー22℃刺激により誘発される温度眼振のMSPEVと注水30℃刺激により誘発される温度眼振のMSPEVとの関係。

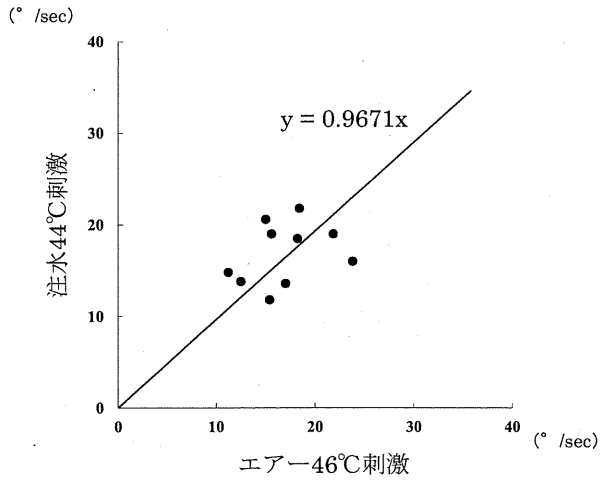

図3 エアー46℃刺激により誘発される温度眼振のMSPEVと注水44℃刺激により誘発される温度眼振のMSPEVとの関係。

価した。また，10耳の注水刺激とエアー刺激によるCP%の平均値の差は，対応のあるt-検定で評価した。なお，同一耳に対するカロリック検査は，5分以上の間隔をあけて行った。

本研究は平成16年度に実施され，その結果をその後に再解析した。

結　果

健常成人5名10耳に対する10℃，20℃，30℃，40℃，46℃のエアー刺激により誘発された温度眼振のMSPEVは，エアー刺激温度と直線的な相関

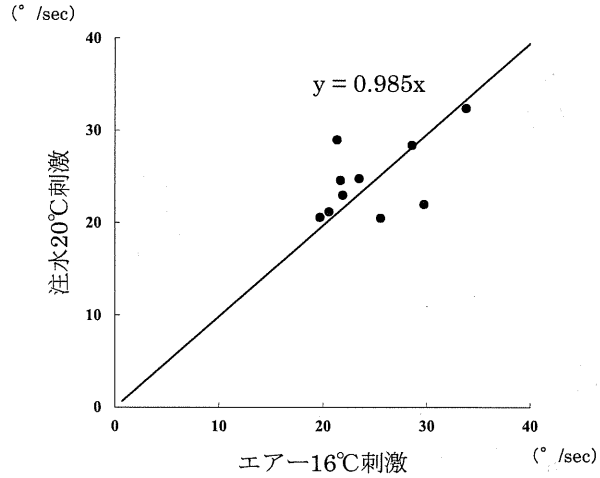

図4 エアー16℃刺激により誘発される温度眼振のMSPEVと注水20℃刺激により誘発される温度眼振のMSPEVとの関係。

を認めた（図1）。相関回帰分析法により近似線を引くと，MSPEV＝0°/secとなる温度は37.3℃であった。次に，同じ健常成人5名10耳に対し，30℃，44℃注水の冷温交互法，20℃の少量注水法で注水刺激を行い，誘発された温度眼振の平均MSPEVは，30℃で－17.2±4.6°/sec，44℃で16.9±3.3°/sec，20℃で－24.7±5.5°/secであった。図1の近似線から，－17.8°/sec，16.9°/sec，－24.7°/secのMSPEVの温度眼振を誘発できるエアー刺激の温度を求めると，22℃，46℃，16℃であった。すなわち，30℃，44℃，20℃の注水刺激と同じMSPEVの温度眼振を誘発できるエアー刺激の温度はそれぞれ，22℃，46℃，16℃であると考えられた。

次に，同じ健常成人5名10耳に対して，22℃，46℃，16℃のエアー刺激によるカロリック検査を行った。22℃のエアー刺激で誘発された温度眼振の平均MSPEVは－18.3±5.9°/secであり，30℃の注水刺激で誘発された温度眼振の平均MSPEVは－17.2±4.6°/secであった。両者の間には，y＝0.9221xの正の相関を認めた（図2）。46℃のエアー刺激で誘発された温度眼振の平均MSPEVは16.9±3.9°/secであり，44℃の注水刺激で誘発された温度眼振の平均MSPEVは平均16.9±3.3°/secであった。両者の間には，y＝0.9671xの正の相関を認めた（図3）。16℃のエアー刺激で誘発された温度眼振の平均MSPEVは－24.6±4.7°/

secであり，20℃の注水刺激で誘発された温度眼振の平均MSPEVは－23.2±5.5°/secであった。両者の間には，y＝0.985 xの正の相関を認めた（図4）。

22℃と46℃のエアー刺激による冷温交互法で求めたCP%の平均は6.49±13.7%であり，30℃と44℃の注水刺激による冷温交互法で求めたCP%の平均は－0.06±7.1%であった。両者に有意差を認めなかった（p＝0.79）。16℃のエアー刺激で誘発された温度眼振のMSPEVは，1耳が19.7°/secであったが，他の9耳は20°/sec以上であった。20℃の注水刺激で誘発された温度眼振のMSPEVは，10耳の全てで20°/sec以上であった。

考　察

カロリック検査は注水による冷温交互法が標準的である[2]。原法であるHallpike法は，30℃および44℃の水250～400 mlを外耳道に40秒間で注水し，温度眼振の持続時間を計測する[1]。本邦では，日本めまい平衡医学会が30℃および44℃の水20 mlを10秒間で注水，または50 mlを20秒間で注水する冷温交互法を提唱している[1]。冷温交互法検査は定量的な結果が得られる点で優れているが，以下のような問題点がある。a）左右の耳の反応の相対的評価であるため，強く反応した耳が必ずしも正常ではないことがある，b）4回の注水を必要とする，c）両側前庭機能低下は評価できない，d）暗所のENG記録では温度眼振の停止時点を決めるのが困難，e）眼振持続時間の代わりに眼振最大緩徐相速度でCP%を求めると値が異なる[6]。そこで，上村ら[4]により，20℃冷水の少量注水で注水精度を高め，ENGによりMSPEVを求めて左右別の半規管機能を定性的に評価する少量注水法が提案され，本邦では普及している。少量注水法は，a）注水は2回，b）温度眼振のMSPEVの大きさで判定する，c）左右耳を個別に評価できる，d）刺激が確実で再現性がよいことから，冷温交互試験より優れている[7]。

近年，注水刺激に代わる温度刺激の方法として，エアーカロリック装置によるエアー刺激が開発された。エアー刺激は，鼓膜穿孔のある耳でも感染の危険なく使用することができ，被検者の注水に対する恐怖がなく，臨床検査技師でも施行できるという長所がある。一方，エアー刺激は注水刺激と比べてより長い刺激時間が必要であり，注水刺激よりも温度眼振の出現が遅い。また，長時間の刺激時間のために外耳道の温度が体温に戻るまで時間を要する[8]。さらに，外耳道に耳漏などの水分が存在すると，温風刺激であっても気化熱により外耳道が冷却され，逆方向の温度眼振が出現する錯倒現象が起こることがある[9][10]。

日本めまい平衡医学会から提案されたエアーカロリック検査基準化案[3]では，冷風刺激のみの場合は15℃以下，流量毎分6～8 l，刺激時間60秒と提案されている。しかし，刺激温度や流量に範囲があり，判定基準がないのが現状である。本研究では，本邦で広く行われている少量注水法の20℃注水刺激に対応するエアー刺激の温度について検討を行った。

健常成人5名10耳において，10℃，20℃，30℃，40℃，46℃で流量6 l/min，刺激時間60秒のエアー刺激により温度眼振を誘発した。誘発された温度眼振のMSPEVは刺激温度と直線的な相関を認め，MSPEV＝0°/secとなる温度は体温とほぼ同じの37.3℃であった。このことから，エアー刺激により外側半規管に内リンパ流動が誘発されていると考えられた。

次に，冷温交互法の注水刺激30℃，44℃と同じMSPEVの温度眼振を誘発できるエアー刺激の温度を図1から求めると，22℃，46℃であった。そこで，同じ健常成人5名10耳に対して，30℃，44℃の注水刺激と22℃，46℃のエアー刺激によるカロリック検査を行い比較した。その結果，30℃注水刺激で誘発される温度眼振のMSPEVと22℃のエアー刺激で誘発される温度眼振のMSPEVとの間には，傾きが1に近い正の相関を認めた。44℃注水刺激で誘発される温度眼振のMSPEVと46℃のエアー刺激で誘発される温度眼振のMSPEVとの間にも，傾きが1に近い正の相関を認めた。エアーカロリック検査基準化案では，冷温交互法で用いる30℃と44℃の注水に対応するエアー刺激は冷風26℃以下と温風46℃以上と提案されているが，本研究結果からは，22℃と46℃のエアー刺激が対応すると考えられた。22℃と46℃のエアー刺激による冷温交互法でのCP%と30℃と44℃の注水刺激による冷温交互法でのCP%には有意差を認めなかったが，22℃と46℃のエアー刺激で誘発される温度眼振のMSPEVの標準偏差は，30℃と44℃

の注水刺激で誘発される温度眼振のMSPEVの標準偏差と比べて大きかった。すなわち，冷温交互法ではエアー刺激の方が注水刺激よりも誘発される温度眼振にばらつきが大きく，冷温交互法は必ずしもエアーカロリック検査に適していないと考えられた。

次に，少量注水法の注水刺激である20℃と同じMSPEVの温度眼振を誘発できるエアー刺激の温度を図1から求めると，16℃であった。そこで，同じ健常成人5名10耳に対して，20℃の冷水刺激と16℃のエアー刺激によるカロリック検査を行った。その結果，20℃の冷水刺激で誘発される温度眼振のMSPEVと16℃のエアー刺激で誘発される温度眼振のMSPEVとの間には，傾きが1に近い正の相関を認めた。このことから，少量注水法の20℃の冷水刺激に対応するエアー刺激の温度は，16℃を用いるべきと考えられた。

また，16℃のエアー刺激で誘発された温度眼振のMSPEVは，1耳が19.7°/secであったが，他の9耳は20°/sec以上であった。少量注水法によるカロリック検査の判定基準はMSPEVが20°/secが正常であり[4)5)]，健常耳に対する16℃のエアー刺激で誘発された温度眼振のMSPEVがほぼ20°/sec以上であったことから，16℃のエアー刺激を用いる少量注水法の半規管麻痺の判定基準は，20℃の冷水刺激を用いる少量注水法の判定基準と同じでよいと考えられた。

さらに，16℃のエアー刺激で誘発される温度眼振のMSPEVの標準偏差は，20℃の冷水刺激で誘発される温度眼振のMSPEVの標準偏差と比べて小さかった。このことから，16℃のエアー刺激はばらつきが小さく，従来の20℃の冷水刺激を用いる少量注水法と同様のCPの判定が可能であると考えられた。

ま と め

20℃の冷水刺激による少量注水法に対応するエアーカロリック検査の刺激条件について検討した。注水刺激の30℃，44℃，20℃に対応するエアー刺激はそれぞれ22℃，46℃，16℃であった。30℃，44℃，20℃の注水刺激で誘発される温度眼振のMSPEVと22℃，46℃，16℃のエアー刺激で誘発される温度眼振のMSPEVとの間には，傾きが1に近い正の相関を認めた。16℃のエアー刺激で誘発された温度眼振のMSPEVは，1耳が19.7°/secであったが，他の9耳は20°/sec以上であったことから，16℃のエアー刺激を用いる少量注水法に対応する半規管麻痺の判定基準は，20℃の冷水刺激を用いる少量注水法の判定基準と同じでよいと考えられた。

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利益相反に該当する事項はない。