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Natural course of positional vertigo in patients with apogeotropic variant of horizontal canal benign paroxysmal positional vertigo

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Abstract

Objective: The purpose of this study was to assess the natural course of positional vertigo in patients with the apogeotropic variant of horizontal canal type of benign paroxysmal positional vertigo (AH-BPPV), which is reported to be more refractory to physiotherapy than the geotropic variant of horizontal canal type of BPPV (GH-BPPV).
Methods: 14 patients with AH-BPPV treated without physiotherapy were asked to visit the hospital every 2 weeks. At every follow-up visit, they were interviewed and positional nystagmus was assessed. After the disappearance of positional nystagmus, patients were asked about the time of cessation of the positional vertigo. Thus, the primary outcomes were evaluated by the self-reported onset and remission of positional vertigo. The time course of remission of positional vertigo was then calculated.
Results: The average and median period from the onset to natural remission of positional vertigo in patients with AH-BPPV was 13 and 7 days, respectively.
Conclusion: We have already reported that the average and median period from the onset to natural remission of positional vertigo in patients with GH-BPPV was 16 and 7 days, respectively (Imai et al., 2005 [8]). Thus, the natural course of AH-BPPV is not as refractory as that of GH-BPPV.

Keywords: Benign paroxysmal positional vertigo; Apogeotropic variant; Horizontal canal; Natural course; Cupulolithiasis

1. Introduction

In recent years, two types of horizontal canal benign paroxysmal positional vertigo (H-BPPV) have been recognized. One type shows geotropic positional nystagmus when the head is turned to the side while lying down (GH-BPPV) [1]. The other type shows apogeotropic positional nystagmus (AH-BPPV) [2,3]. The pathophysiology of GH-BPPV is thought to be canalolithiasis in the horizontal semicircular canal (HSCC) and physiotherapy such as canalith repositioning therapy was effective in patients with GH-BPPV [4–7]. Moreover, we reported that GH-BPPV disappeared naturally within about 2 weeks without physiotherapy [8]. On the other hand, the pathophysiology of AH-BPPV is still controversial, with cupulolithiasis in the HSCC being reported as the most plausible cause of AH-BPPV [2,3]. On the other hand, it was reported that outcomes of physiotherapy in patients with AH-BPPV were poorer as compared with those with GH-BPPV [6,7].

In the present study, in order to examine if AH-BPPV was more refractory than GH-BPPV without physiotherapy, we assessed the natural course of remission of positional vertigo in patients with AH-BPPV.

2. Methods

The present study included 14 consecutive patients who were diagnosed as AH-BPPV in the Department of

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Table 1
The data of all patients. m: male, f: female, CP: canal paresis, R-CP: canal paresis exist in right ear, L-CP: canal paresis exist in left ear, N.P.: not performed, SPEV (R): slow phase eye velocity of positional nystagmus when their head was right lateral position in supine, SPEV (L): slow phase eye velocity of positional nystagmus when their head was left lateral position in supine.

ID	Sex	Age (years)	Affected side	Visit (days)	Remit (days)	Caloric	SPEV (R) (°/s)	SPEV (L) (°/s)
A	m	59	Right	1	2	N.P	8.9	41.8
B	f	28	Left	0	3	R-CP	6.6	6.4
C	f	71	Right	1	3	No CP	3.5	53.5
D	f	85	Right	1	3	N.P.	25.5	28.4
E	m	26	Left	2	3	No CP	6.0	3.1
F	m	72	Left	1	4	N.P	83.6	65.5
G	f	57	Right	1	6	N.P.	9.7	50.1
H	f	61	Right	1	7	N.P.	1.9	2.7
I	m	61	Right	4	7	No CP	23.0	61.9
J	f	72	Left	7	23	N.P.	9.9	2.3
K	m	37	Left	10	26	L-CP	51.8	25.8
L	f	60	Right	12	27	No CP	4.6	15.4
M	m	73	Right	22	32	N.P.	1.1	7.6
N	m	70	Left	7	35	R-CP	1.7	1.3

Otolaryngology, Kansai-Rosai Hospital between April 2001 and November 2003 (7 males and 7 females; 26–85 years old: mean age, 59 years, Table 1). All patients who complained of dizziness and/or vertigo were tested by lateral head rotation in supine position. The positional nystagmus was recorded by an infrared CCD camera (*RealEyes*, Micromedical Technologies) in all subjects. Maximum slow phase eye velocity (max SPEV) of the positional nystagmus was analyzed using our own video-oculography system [9]. AH-BPPV was diagnosed on the following criteria; (i) a history of brief episodes of positional vertigo, (ii) observation of an apogeotropic direction-changing positional nystagmus mainly with a horizontal component triggered by lateral head rotations in supine position; the apogeotropic variant was indicated by an intense apogeotropic nystagmus beating away from the ground in lateral head position on both sides, reaching a maximum immediately after change in head position and lasting more than 1 min, and (iii) absence of an identifiable central nervous system disorder able to explain the positional vertigo following neurological and neurophysiological examinations. The affected ear was determined as the head turning side that created less intense nystagmus [3]. Patients with nystagmus, of which direction changed from apogeotropic to geotropic or from geotropic to apogeotropic, were excluded from this study.

The patients were asked about the onset time of positional vertigo through a detailed interview. After they were given the details of the physiotherapy [4], their informed consent to non-medicinal treatment was obtained. Thus, all patients received no drug and were advised to visit the hospital every 2 weeks after the initial visit. At every visit, they were interviewed and examined by lateral head rotation in supine position. After the disappearance of positional nystagmus, patients were required to indicate the time at which the positional vertigo disappeared through detailed interview. Thus, the primary outcomes were assessed by self-reported onset and remission of positional vertigo, while the time course of remission of positional vertigo after the onset was calculated by Kaplan–Meier method. Patients were then examined 4 weeks after the disappearance of positional nystagmus and remained free of positional vertigo and nystagmus.

Patients with AH-BPPV were divided into two groups: the early remission group including patients A–I whose remission period was within 7 days, and the delayed remission group made of patients J–N whose remission period was more than 3 weeks (Table 1). Differences in age, sex, affected side, result of caloric test and max SPEV of positional nystagmus at the first visit to the hospital between the two groups were analyzed using chi-square test.

3. Results

The residual rate of positional vertigo was 64% in the patients with AH-BPPV at 1 week after the onset and 14% at 1 month without physiotherapy (Fig. 1). The averaged period and median period from the onset to natural remission of positional vertigo was 13 ± 13 days and 7 days, respectively.

There were no significant differences in age, sex, affected side, result of caloric test and max SPEV of positional nystagmus at the first visit to the hospital between the early and delayed remission groups (Table 1).

4. Discussion

In the present study, we assessed the natural course of positional vertigo in patients with AH-BPPV (7 males and 7 females; mean age, 59 years) without physiotherapy and showed that the average and median period from the onset to natural remission of their positional vertigo was 13 and 7 days, respectively. We have already reported in patients with GH-BPPV (15 males and 21 females; mean age, 57 years)

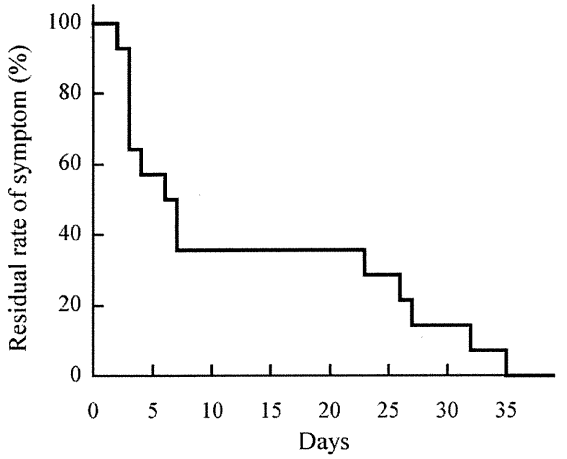

Fig. 1. Time course of positional vertigo after the onset without physiotherapy in patients with AH-BPPV was calculated using Kaplan–Meier method.

that the average and median period from the onset to natural remission of positional vertigo was 16 and 7 days, respectively [8]. Thus, the natural course of AH-BPPV is not as refractory as that of GH-BPPV.

GH-BPPV is thought to be induced by canalolithiasis made of the otoconial debris, which move under the influence of gravity within the distal long arm of HSCC when the head rotates in the supine position [1]. On the other hand, AH-BPPV is thought to be due to cupulolithiasis made of the otoconial debris attached to the cupula of HSCC and the gravity-sensitive cupula bends when the head rotates in the supine position [2,3,10]. Similarly to the posterior canal type of benign paroxysmal positional vertigo [11], the management of GH- and AH-BPPV is based on physiotherapy, having the same purpose of removing the debris from the location in the canal toward the utricle, even considering that the debris may be variously positioned in the lumen of the canal [6,7]. The physiotherapy of GH-BPPV is based on removing the debris from HSCC along its distal long arm into the utricle by various methods including 180° rotation [12], barbecue rotation [4], and forced prolonged position [5]; whereas the physiotherapy of AH-BPPV consists of detaching the debris from the cupula by various methods including therapeutic head shaking [13], modified Semont maneuver [14], Gufoni maneuver [6], and Vanucchi–Asprella maneuvers [15], and then removing the debris from HSCC along its distal long arm into the utricle by the same methods used for the physiotherapy of GH-BPPV. It was reported that outcomes of the physiotherapy in patients with AH-BPPV were poorer compared to those with GH-BPPV [6,7], probably because the physiotherapy of AH-BPPV contains two steps.

But, as shown in the present study, the average period from the onset to natural remission of positional vertigo in patients with AH-BPPV was slightly shorter than that in patients with GH-BPPV. Indeed, in patients with GH-BPPV even without physiotherapy, the head movement of daily life moves canalolithiasis along the distal long arm of HSCC to the utricle. On the other hand, among patients with AH-BPPV even without physiotherapy, we hypothesized that the head movement of daily life makes the cupulolithiasis fall off from the cupula into the proximal short arm of HSCC in patients A–I and into its distal long arm in patients J–N. Thus, the short trajectory of the debris from the proximal short arm of HSCC into the utricle after its detachment from the cupula may be accomplished within 7 days in patients A–I, whereas the long trajectory of the debris from the distal long arm of HSCC into the utricle may take more than 3 weeks in patients J–N.

Because AH-BPPV in the early remission group disappeared within a week, it is possible that some other patients with AH-BPPV might have been cured naturally before the visit to the hospital behind the present study. This bias that cannot be estimated may slightly prolong the natural course of AH-BPPV.

In the present study, we showed that the average and median period from the onset to natural remission of positional vertigo in patients with AH-BPPV was 13 and 7 days, respectively. We concluded that despite the poorer physiotherapy outcomes in AH-BPPV patients, the natural remission of their disease was not as refractory in comparison with that of GH-BPPV patients.

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ORIGINAL ARTICLE

Vestibular and cochlear neuritis in patients with Ramsay Hunt syndrome: a Gd-enhanced MRI study

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Abstract

Conclusion: It is suggested that vertigo in patients with Ramsay Hunt syndrome is mostly induced by superior vestibular neuritis consecutive to the reactivation of varicella-zoster virus (VZV) infection from the geniculate ganglion through the faciovestibular anastomosis. Refractory hearing loss in patients with Ramsay Hunt syndrome may be due to cochlear neuritis following the spread of VZV. *Objectives:* An attempt was made to selectively identify vestibulocochlear nerves in the internal auditory canal (IAC) on gadolinium (Gd)-enhanced MRI in patients with Ramsay Hunt syndrome. *Methods:* Fourteen patients with Ramsay Hunt syndrome presenting with facial palsy, herpes zoster oticus, vertigo, and/or sensorineural hearing loss were scanned on 1.5 T MRI enhanced with Gd. Perpendicular section images of the IAC were reconstructed to identify the facial, superior, and inferior vestibular nerves and the cochlear nerves separately. *Results:* All except one of the patients with Ramsay Hunt syndrome with vertigo showed both canal paresis on the caloric test and Gd enhancement of the superior vestibular nerve in the IAC on MRI. Among 10 patients with hearing loss, 3 patients with severe to moderate sensorineural hearing loss showed Gd enhancement of the cochlear nerve in the IAC on MRI.

Keywords: *Facial palsy, varicella zoster virus, vestibular nerves, vestibulocochlear nerves*

Introduction

Ramsay Hunt syndrome is characterized by herpes zoster oticus, peripheral facial palsy, and eighth cranial nerve symptoms including vertigo and hearing loss [1]. Ramsay Hunt syndrome is caused by the reactivation of varicella-zoster virus (VZV) infected latently in the geniculate ganglion of the seventh cranial nerve. Reactivated VZV in the geniculate ganglion induces the inflammation of the facial nerve, resulting in facial palsy [2]. Since gadolinium-DTPA (Gd) accumulates in the inflamed tissue where there is a breakdown of the blood–nerve barrier, Gd-enhanced magnetic resonance imaging (MRI) can visualize the inflammation of the cranial nerves. Therefore, Gd-enhanced MRI studies have reported a high frequency of enhancement in the regions of the internal auditory canal (IAC) and/or the intratemporal segments of the facial nerve in patients with Ramsay Hunt syndrome [3].

Because the vestibulocochlear nerves are in proximity to the geniculate ganglion, they are also inflamed by transneural infection from reactivated VZV, leading to the vestibulocochlear symptoms in patients with Ramsay Hunt syndrome [4]. However, the enhancement of the eighth cranial nerves independent from the facial nerve was seldom reported in Gd-enhanced MRI studies in patients with Ramsay Hunt syndrome.

In the present study, an attempt was made to investigate the enhancement of the vestibulocochlear nerves in the IAC on Gd-enhanced MRI in patients

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Table I. Details of the patients in the study (n = 14).

Case no.	Sex	Age (years)	Affected side	HB grade	ENoG	Vertigo	CP	Hearing loss (dB)	FN	SVN	IVN	CN
1	F	72	Left	VI	100%	+	+	88	+	+	+	+
2	F	59	Left	VI	24%	+	+	37	+	+	–	+
3	M	66	Right	VI	100%	+	+	33	+	+	–	+
4	F	45	Right	VI	82%	+	+	23	+	+	–	–
5	M	62	Left	V	52%	+	+	28	–	+	–	–
6	M	41	Left	V	22%	+	+	27	–	+	–	–
7	M	43	Right	VI	100%	+	+	23	–	+	–	–
8	F	60	Left	IV	56%	+	+	20	–	+	–	–
9	F	78	Right	VI	75%	+	+	–	–	+	–	–
10	M	77	Right	V	83%	+	+	–	–	+	–	–
11	M	72	Right	VI	83%	+	+	–	–	+	–	–
12	F	83	Right	V	75%	+	+	–	–	–	–	–
13	M	58	Right	VI	100%	–	–	52	–	–	–	–
14	M	61	Right	IV	21%	–	–	28	–	–	–	–

CN, cochlear nerve; CP, canal paresis on the caloric test; ENoG, electroneurography; FN, facial nerve; HB grade, House-Brackmann grading; Hearing loss, the averaged right–left differences in hearing levels at 2000, 4000, and 8000 Hz; IVN, inferior vestibular nerve; SVN, superior vestibular nerve; +, positive enhancement of Gd-MRI; –, negative enhancement of Gd-MRI.

with Ramsay Hunt syndrome. For this purpose, MRI images perpendicular to the IAC were reconstructed to identify the facial, superior, and inferior vestibular nerves and the cochlear nerves separately. Correlations between their enhancement and canal paresis (CP) on the caloric test and/or sensorineural hearing loss on pure tone audiometry were also examined.

Material and methods

Fourteen patients with Ramsay Hunt syndrome who showed peripheral facial palsy, ipsilateral herpes zoster oticus, and vertigo/hearing loss (eight males and six females; 41–83 years old; mean age 62.6 years) participated in the present study (Table I). Seven cases had the simultaneous development of facial palsy and vertigo and/or hearing loss, six cases had vertigo and/or hearing loss preceding the development of facial palsy, and one case had facial palsy preceding the development of vertigo and hearing loss. The House-Brackmann (HB) grading of their initial facial palsy ranged from IV to VI. The electroneurography (ENoG) findings ranged from 21% to 100% at 10–14 days after the onset. Twelve and 10 patients complained of vertigo and hearing loss, respectively. Eight patients had both vertigo and hearing loss. Twelve dizzy patients all showed CP on the caloric test performed 16.9 ± 10.6 days after the onset. On the basis of the initial pure tone audiograms, 10 patients showed sensorineural hearing loss. We evaluated the right–left differences in hearing level, because hearing impairment from other causes such as aging and noise exposure were excluded (Figure 1). Within a week after the onset, patients were treated with systemic steroids (methylprednisolone: 250 mg for 3 days, 125 mg for 3 days, 80 mg for 3 days, i.v.) and antiviral agents (valaciclovir: 3000 mg for 7 days, p.o.).

ENoG was performed with the recording surface electrodes on the nasolabial fold. Bipolar rectangular pulses of 0.2 ms duration with stepwise increase of the stimulating current from 35 mA to 50 mA were given by a bipolar stimulator placed on the skin over the stylomastoid foramen, and the maximal compound action potentials were recorded. ENoG value was calculated using the following formula: ENoG

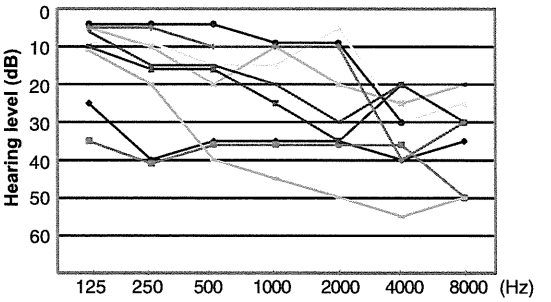

Figure 1. Differences in hearing levels between affected and healthy ears in patients with Ramsay Hunt syndrome with sensorineural hearing loss on the initial pure tone audiogram (n = 9, except case 1 with deafness). Their hearing recovered to normal range or the same level as the unaffected ear, except in case 1 with deafness (not shown) and cases 2 and 3 (■, ◇) with moderate hearing loss. Case 4, • case 5, –; case 6, *; case 7, x; case 8, ▲; case 13, –; case 14, +.

(%) = 100 – (amplitude on the affected side)/(amplitude on the healthy side) × 100.

For the caloric test, cold-water (15°C, 5 ml) irrigation was used and caloric nystagmus was recorded by electronystagmography. The maximum slow-phase eye velocity was measured and the caloric response was calculated by addition or subtraction of the averaged slow-phase eye velocity of spontaneous nystagmus. A caloric response of less than 20°/s was determined as CP.

Patients were then scanned on a 1.5 T MR unit (Signa1.5THDxt, GE) to obtain three-dimensional T1-weighted fast field-echo images at 25.4 ± 15.8 days after the onset. The imaging parameters were as follows: repetition time 10.5 ms, echo time 3.2 ms, flip angle 20°, 1 mm slice thickness, 200 mm field of view, and 256 × 224 (512 reconstruction) matrix. The contrast medium (Gd-DTPA) was administered intravenously at a dose of 0.1 nmol/kg. Then, images perpendicular to the IAC were reconstructed to identify the facial, superior, and inferior vestibular nerves and the cochlear nerves separately. The MRIs were evaluated by two radiologists independently without knowledge of the clinical data. The presence of abnormal contrast enhancement of the facial nerve, superior and inferior vestibular nerve, and cochlear nerve in the IAC was assessed on the basis of visual inspection of the precontrast and postcontrast images.

Fisher's exact probability test was used for statistical analysis and $p < 0.05$ was considered significant.

Results

Twelve patients with Ramsay Hunt syndrome with vertigo all showed CP on the caloric test. Eleven of them showed enhancement of the superior vestibular nerve in the IAC on Gd-enhanced MRI. Thus, there was a significant association between vertigo with CP and enhancement of the superior vestibular nerve in Ramsay Hunt syndrome patients with vertigo ($p < 0.01$) (Table I). A patient with vertigo (case 1) showed the enhancement of both superior and inferior vestibular nerves in the IAC on MRI. Vertigo and/or dizziness disappeared in nine patients, but residual dizziness continued in three patients (cases 1, 2, and 3).

Ten patients with Ramsay Hunt syndrome showed sensorineural hearing loss: profound in one, moderate in three, and mild high-frequency hearing loss in six. Figure 1 shows the differences in hearing levels between the affected and healthy ears at each patient's first visit. Three patients, case 1 with profound and cases 2 and 3 with moderate sensorineural hearing losses, showed enhanced cochlear nerves in the IAC on Gd-enhanced MRI (Table I) and their hearing loss was irreversible. By contrast, moderate hearing loss in case 13 and mild high-frequency hearing loss in six patients with no cochlear nerve enhancement on MRI recovered completely within 2 months after the onset.

All 14 patients with Ramsay Hunt syndrome showed facial palsy ranging from HB grade IV to grade VI. Only four of eight patients with total facial palsy (HB grade VI) showed enhancement of the facial nerve in the IAC on Gd-enhanced MRI (Table I). The facial palsy recovered to HB grade I in two, and one patient each to grade II and III. In the remaining 10 patients with no enhancement of the facial nerve on MRI, facial palsy recovered to HB grade I in eight, grade II in one, and grade III in one patient.

Case 1 showed Gd enhancement of all four nerves: facial, superior, and inferior vestibular nerves and cochlear nerves in the IAC on MRI (Figure 2a). Although her facial palsy recovered to HB grade III and vertigo disappeared, residual dizziness and profound sensorineural hearing loss continued a year after the onset. Case 2 and case 3 (Figure 2b) showed Gd enhancement of three nerves – facial, superior vestibular, and cochlear nerves in the IAC on MRI. Although their facial palsy recovered to HB grade I and II, respectively, and vertigo disappeared, residual dizziness and moderate sensorineural hearing loss continued a year after the onset.

Case 4 showed Gd enhancement of two nerves, the facial and superior vestibular nerves in the IAC on MRI (Figure 2c). Her facial palsy recovered completely, and vertigo/dizziness and hearing loss disappeared within 2 months after the onset. Seven patients including case 8 (Figure 2d) showed Gd enhancement of the superior vestibular nerve only. Their facial palsy recovered and vertigo/dizziness disappeared. Three patients showed no Gd enhancement of any seventh and eighth cranial nerves in the IAC on MRI, with the recovery of their facial palsy, and the disappearance of their vertigo/dizziness and/or hearing loss.

Discussion

In the present study, 12 patients with Ramsay Hunt syndrome with vertigo all showed CP in the caloric test. Such a high frequency of CP on the caloric test was also reported previously in patients with Ramsay Hunt syndrome [4,5]. All except one patient showed the enhancement of the superior vestibular nerve in the IAC on Gd-enhanced MRI. Thus, there was a significant association between vertigo with CP and enhancement of the superior vestibular nerve in Ramsay Hunt syndrome patients. Since the caloric test primarily stimulates the lateral semicircular canal, it can be used to assess the superior vestibular nerve, which innervates the lateral and anterior semicircular

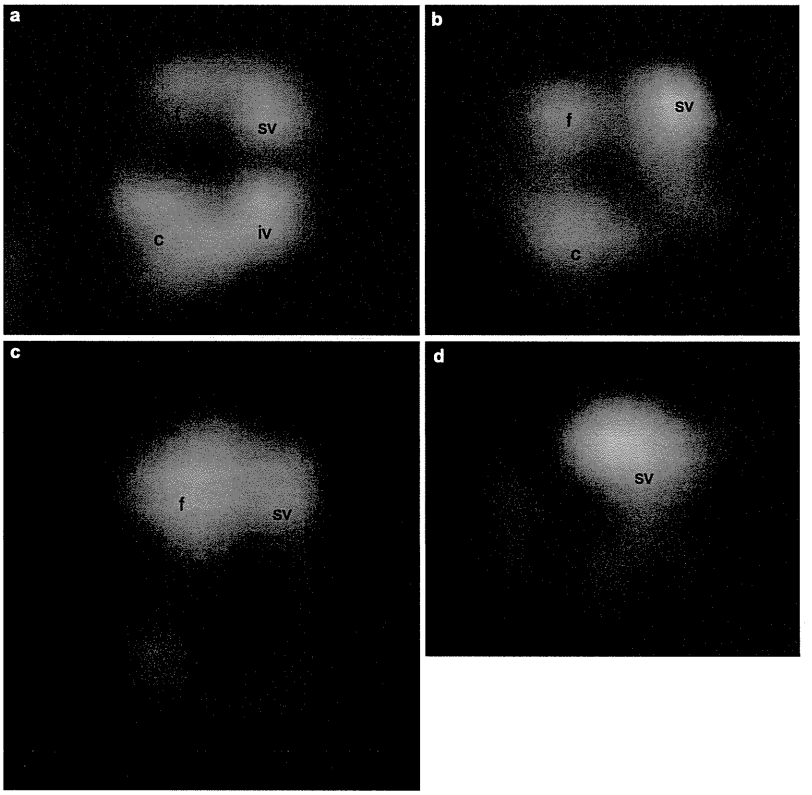

Figure 2. (a) Case 1: facial palsy of HB grade VI, CP, and deafness. The patient had residual dizziness and deafness that continued a year after the onset. Facial (f), superior (sv), and inferior vestibular (iv), and cochlear (c) nerves were enhanced by Gd in the internal auditory canal (IAC) on MRI. (b) Case 3: facial palsy of HB grade VI, CP, and flat type of mild sensorineural hearing loss (◇ in Figure 1). The patient had residual dizziness and moderate sensorineural hearing loss that continued a year after the onset. f, sv, and c nerves were enhanced by Gd in the IAC on MRI. (c) Case 4: facial palsy of HB grade VI, CP, and mild high-frequency sensorineural hearing loss (• in Figure 1). Her facial palsy fully recovered, and vertigo/dizziness and hearing loss disappeared within 2 months of the onset. f and sv nerves were enhanced by Gd in the IAC on MRI. (d) Case 8: facial palsy of HB grade IV, CP, and mild high-frequency sensorineural hearing loss (▲; in Figure 1). Her facial palsy fully recovered, and vertigo/dizziness and hearing loss disappeared within 2 months of the onset. Only the sv nerve was enhanced by Gd in the IAC on MRI.

canals and utricle. Therefore, it is suggested that the origin of vertigo in patients with Ramsay Hunt syndrome is the lesion of the superior vestibular nerve. Moreover, both superior and inferior vestibular nerves were enhanced in case 1, who showed vertigo. A previous study reported the disappearance of caloric response and vestibular evoked myogenic response (VEMP) in dizzy patients with Ramsay Hunt syndrome [5]. Taken together, the above observation also suggested that in some cases lesions to both superior and inferior vestibular nerves induce vertigo, because the inferior vestibular nerve innervates the saccule that is the origin of VEMP. There is another possibility that the lesion of the superior vestibular nerve induces vertigo and affects VEMP, because the superior vestibular nerve innervates the anterosuperior part of the sacculus in addition to the lateral and anterior semicircular canals and the utricle.

Gd does not normally cross the blood–nerve barrier. However, it is postulated that inflammation induced by reactivated VZV breaks down the blood–nerve barrier and increases the permeability of Gd from the blood to the nerve [3]. This is the possible mechanism of Gd enhancement of the affected facial nerve on MRI. Since the facial nerve is connected to the superior vestibular nerve by the faciovestibular anastomosis [6], Gd enhancement of the superior vestibular nerve on MRI in Ramsay Hunt syndrome patients with vertigo suggests superior vestibular neuritis due to reactivated VZV from the geniculate ganglion through the anastomosis. Subsequently, the adjoining inferior vestibular nerve may be infected by VZV. However, the superior nerve is more susceptible to inflammation than the inferior nerve, because the lateral bony channel of the superior vestibular nerve is longer and contains more bony spicules [7].

Seven of the 10 Ramsay Hunt syndrome patients with sensorineural hearing loss who showed no enhancement of the cochlear nerve had a complete recovery and their right–left differences in hearing loss were mostly mild and limited to high frequencies. This finding is in line with a previous report that showed a good prognosis of sensorineural hearing loss that mainly affected high frequencies in patients with Ramsay Hunt syndrome [8,9]. In the remaining three patients with Gd enhancement of the cochlear nerve in the IAC, profound hearing loss in case 1 and moderate hearing loss in cases 2 and 3 were irreversible. The close association between the enhancement of the cochlear nerve and refractory hearing loss in patients with Ramsay Hunt syndrome suggests that the origin of refractory sensorineural hearing loss might be the cochlear neuritis. Therefore, moderate hearing loss in case 13 recovered completely, probably because his cochlear nerve was not enhanced in MRI. Retrocochlear involvement in patients with Ramsay Hunt syndrome has also been reported [4,9]. Since the cochlear nerve is connected to the inferior vestibular nerve by the Oort's anastomosis [6], reactivated VZV from the geniculate ganglion may spread to the vestibular and cochlear nerves through the faciovestibular and Oort's anastomoses, as seen in case 1 who showed Gd enhancement of the superior and inferior vestibular nerves and the cochlear nerve in the IAC on MRI. However, the mechanisms of moderate hearing loss in cases 2 and 3 with enhancement of cochlear nerve and mild hearing loss in other patients who show no enhancement of cochlear nerve are unknown.

A previous report has shown Gd enhancement of the facial nerve in the IAC and the geniculate ganglion a week after the onset of facial palsy in patients with herpetic facial palsy, suggesting that the peak of inflammatory response to VZV occurs at around 10–14 days after the onset [10,11]. In contrast, in the present study, Gd enhancement of the facial nerve in the IAC was observed in only four patients with Ramsay Hunt syndrome on MRI obtained more than 20 days after the onset of the palsy. In addition, there was no correlation between Gd enhancement of facial nerve in the IAC on MRI and the prognosis of facial palsy in patients with Ramsay Hunt syndrome, which is in line with previous studies [3,12].

In conclusion, our data demonstrate that vertigo in patients with Ramsay Hunt syndrome is mostly induced by superior vestibular neuritis infected by reactivated VZV from the geniculate ganglion through the faciovestibular anastomosis, as demonstrated in all except one dizzy patient with Ramsay Hunt syndrome, showing both CP on the caloric test and Gd enhancement of the superior vestibular nerve in the IAC on MRI. Refractory hearing loss in patients with Ramsay Hunt syndrome may be due to cochlear neuritis involved in the spread of VZV, as shown in three patients with severe or moderate sensorineural hearing loss and Gd enhancement of the cochlear nerve in the IAC on MRI. Further studies including a quantitative analysis of enhancement on MRI are needed to prove the above hypothesis.

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ORIGINAL ARTICLE

Effects of sleep position on time course in remission of positional vertigo in patients with benign paroxysmal positional vertigo

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Abstract

Conclusion: The findings suggest that it is easy for otoconial debris dislodged from the utricle to fall into the posterior semicircular canal (PSCC) or the horizontal semicircular canal (HSCC) of the undermost ear during sleep, but not to exit from the uppermost ear in patients with benign paroxysmal positional vertigo (BPPV). *Objective:* The aims of the present study were two-fold. (1) To examine the association between the preferred side of head-lying during sleep and the side of the affected ear in patients with both posterior canal BPPV (P-BPPV) and horizontal canal BPPV (H-BPPV). (2) To see whether that position affects the time course in remission of their positional vertigo. *Methods:* One hundred and sixteen patients with P-BPPV and 40 patients with H-BPPV who showed a habitual preference for right or left side sleeping position were included in this study. *Results:* The side of the affected ear was significantly associated with the head-lying side during sleep in patients with P-BPPV and was closely but not significantly associated with it in patients with H-BPPV. However, the head-lying side during sleep did not affect the remission rate of their positional vertigo.

Keywords: *Canalolithiasis, posterior canal BPPV, horizontal canal BPPV, head-lying side during sleep*

Introduction

Benign paroxysmal positional vertigo (BPPV) is a common vestibular disease characterized by brief episodes of vertigo triggered by changes in head position. BPPV most commonly affects the posterior semicircular canal (PSCC) [1], but another type of BPPV has been reported in recent years, in which the horizontal semicircular canal (HSCC) is affected [2–4]. Recently, the pathophysiology of BPPV has been recognized to be canalolithiasis in most patients with both PSCC (P-BPPV type) and HSCC (H-BPPV type). Canalolithiasis, an otoconial debris that is dislodged from the utricle falls and becomes trapped into the semicircular canal [5,6]. The otoconial debris acts like a plunger in the semicircular canal to induce the movement of endolymph during changes in head position, resulting in nystagmus and vertigo [7].

It was reported that habitual head-lying side during sleep was associated with the side of the affected canal in patients with P-BPPV [8–10], suggesting that otoconial debris dislodged from the utricle more likely enters into the PSCC of the undermost ear. Recently, such association was reported in patients with H-BPPV [11]. In the present study, we first confirmed the association in patients with both P-BPPV and H-BPPV. Moreover, the association led to the working hypothesis that the remission of positional vertigo would be facilitated in patients with BPPV who prefer the affected ear-up head position during sleep, because this position is more likely to facilitate the exit of the otoconial debris from the semicircular canal of the uppermost ear. Thus, the second aim of the present study was to investigate effects of the preferred side of head-lying during sleep on the time course in remission of their positional vertigo.

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Material and methods

From April 2009 to March 2011, we diagnosed P-BPPV in 184 patients (127 women and 57 men; 15–86 years old; mean age 59.7 ± 15.6 years) and H-BPPV in 66 patients (36 women and 30 men; 27–93 years old; mean age 59.1 ± 16.0 years). One hundred and sixteen patients with P-BPPV and 40 patients with H-BPPV who showed a habitual preference for right or left side sleeping position were included in this study. BPPV was diagnosed on the basis of the following criteria. (1) History of brief episodes of positional vertigo. (2) Absence of an identifiable central nervous system (CNS) disorder able to explain the positional vertigo in neurological examination and neuroradiological studies of the brain. (3) No spontaneous nystagmus. (4) The presence of a direction-changing torsional positional nystagmus triggered by the Dix-Hallpike test was indicative of P-BPPV, while a direction-changing geotropic horizontal positional nystagmus triggered by supine roll test indicated H-BPPV.

In patients with P-BPPV, the affected ear was determined by the presence of a torsional nystagmus beating towards the affected and undermost ear in the Dix-Hallpike test, while in those with H-BPPV, a more intense geotropic nystagmus was induced when the head was turned to the side of the affected ear in the supine roll test.

Patients with P-BPPV were treated by modified Epley maneuver without mastoid oscillation [12], while those with H-BPPV were not treated because the efficacy of the Lempert maneuver for the treatment of patients with H-BPPV was limited [13]. Patients were asked to come to the hospital every week and relapses were investigated by the Dix-Hallpike test or supine roll test. After the disappearance of positional nystagmus, the patients were interviewed to determine the timing of the remission. Thus, the primary outcomes were self-reported remission of positional vertigo and the rate of residual patients with positional vertigo was then calculated by the Kaplan–Meier method.

Binomial test and Kaplan–Meier method with log rank test were used for statistical analysis and $p < 0.05$ was considered significant.

Results

Sixty-nine patients (37.5%) with P-BPPV showed a habitual preference for a right-sided sleeping position, while 47 (25.5%) showed a left-sided preference. Sixty-two patients (53.4%) with P-BPPV showed a positive Dix-Hallpike test on the right undermost ear and 54 (46.6%) on the left undermost ear. These findings were indicative of a significant association between the side of the affected ear and the head-lying side during sleep in patients with P-BPPV ($p < 0.001$) (Table I).

Table I. Positive Dix-Hallpike test side and habitual sleeping position in patients with P-BPPV.

	Dix-Hallpike test		
Habitual sleeping position	Right	Left	Total
Right	51	18	69
Left	11	36	47
Total	62	54	116

The side of the affected ear was significantly associated with the head-lying side during sleep: binomial test, $p < 0.001$.

Twenty-nine (72.5%) patients with H-BPPV showed a habitual preference for a right-sided sleeping position, while 11 (27.5%) showed a left-sided preference. Seventeen patients (42.5%) with H-BPPV showed a more intense nystagmus when the head was turned to the right in the supine roll test and 23 (57.5%) showed the same kind of nystagmus when the head was turned to the opposite side. These data indicate that the side of the affected ear tended to be associated with the head-lying position during sleep in patients with H-BPPV ($p = 0.08$) (Table II).

The residual rate of positional vertigo after modified Epley maneuver in patients with P-BPPV who preferred to lie in the affected ear-up head position during sleep was not different from that in patients who preferred to lie in the affected ear-down position during sleep ($p = 0.98$) (Figure 1). The residual rate of positional vertigo in patients with H-BPPV who preferred to lie in the affected ear-up head position during sleep was also not different from that in patients who preferred to lie in the affected ear-down during position sleep ($p = 0.34$) (Figure 2).

Discussion

In the present study, the side of the affected ear was significantly associated with the preferred head-lying

Table II. Supine roll test side and habitual sleeping position in patients with H-BPPV.

	Supine roll test		
Habitual sleeping position	Right	Left	Total
Right	16	13	29
Left	1	10	11
Total	17	23	40

The side of the affected ear had a tendency to associate with the head-lying side during sleep: binomial test, $p = 0.08$.

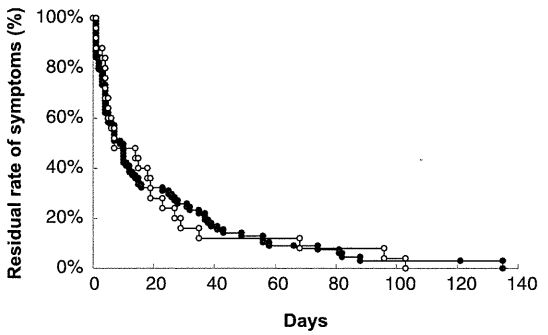

Figure 1. Residual rate of positional vertigo in patients with P-BPPV after modified Epley maneuver. Open circle, patients who prefer to lie with the affected ear up during sleep; filled circle, patients who prefer to lie with the affected ear down during sleep. $p = 0.98$.

side during sleep in patients with P-BPPV, and was closely but not significantly associated with it in patients with H-BPPV. The association in patients with P-BPPV and H-BPPV was in line with previous studies [8–11]. Korres et al. proposed that under the influence of gravity, otoconial debris dislodged from the utricle is likely to enter into the PSCC of the undermost ear during sleep and develop P-BPPV. The hypothesis can explain the findings that a prolonged bed-rest after major surgery or central nervous system disease may trigger the development of P-BPPV [14,15]. It also explains the association between the preferred head-lying side and the affected ear in patients with H-BPPV, as shown in the present study. Because both the short arm of the PSCC and the long arm of the HSCC hang down from the utricle in the supine position during sleep [1], it is suggested that it is easy for otoconial debris dislodged from the utricle to fall into the PSCC or the HSCC of the undermost ear during sleep. Because the opening of the short arm of the PSCC is inferior to that of the long arm of the HSCC of the head-lying side ear, the association between the preferred head-lying side and the affected ear in patients with P-BPPV is closer than that in patients with H-BPPV.

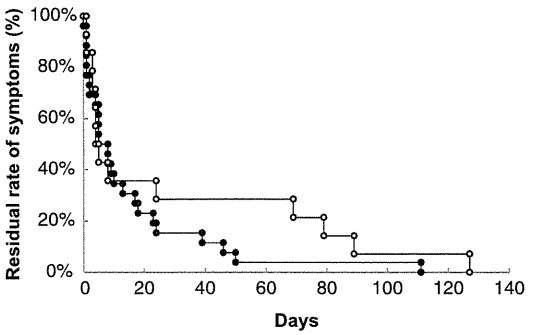

Figure 2. Residual rate of positional vertigo in patients with H-BPPV without any maneuver. Open circle, patients who prefer to lie with the affected ear up during sleep; filled circle, patients who prefer to lie with the affected ear down during sleep. $p = 0.34$.

Next, we investigated the effects of the preferred side of head-lying during sleep on the time course in remission of positional vertigo in patients with P-BPPV or H-BPPV, because the otoconial debris seemed to exit from the PSCC or the HSCC of the uppermost ear during sleep. However, the head-lying side during sleep did not affect the remission rate of positional vertigo in patients with P-BPPV or H-BPPV. Although it is easy for otoconial debris to exit from the PSCC or the HSCC through a series of head position changes in the canalith repositioning maneuver, it is hard for the debris to slip out of the uppermost ear by gravity during sleep. This is probably because otoconial debris is easy to move by kinetic positional change, but not by a static head position [16].

Conclusion

The findings of the present study suggest that the side of affected ear is associated with the head-lying side during sleep in patients with both P-BPPV and H-BPPV. However, the time course in remission of positional vertigo is not affected by the head-lying side during sleep in these patients.

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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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ORIGINAL ARTICLE

Effects of repeated optic flow stimulation on gait termination in humans

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Abstract

Conclusions: Because the basic strategies to stop walking are stored as motor programs, visual stimulation may have little influence on body deviation during gait termination and its time course. Walking velocity, however, demonstrated dynamic flexible changes, which may subserve the stable process of gait termination under variable circumstantial changes such as optic flow. *Objective:* The aim of this study was to examine the effect of repeated optic flow on body deviation and walking velocity during gait termination, which may be more complicated than continuous standing or walking. *Methods:* Twenty-three healthy subjects were instructed to start walking upon an acoustic cue and to stop walking when the scenery changed in a virtual reality environment. Subjects underwent eight control trials without optic flow and three sets of optic flow conditions including four trials each of optic horizontal and rotational movement randomly. *Results:* Repeated optic flow caused no significant change of body deviation or the time course of the gait termination process in comparison with that in the control. The walking velocity at the start of the termination process showed short-term flexibility that denoted a gradual increase over the trial for within-set and long-term flexibility that denoted a gradual decrease for between-set.

Keywords: *Postural balance, adaptation, walking velocity, virtual environment*

Introduction

For all vertebrate animals, locomotion is indispensable for survival. For humans in particular, whose bodies are maintained in erect bipedalism and who are surrounded by a complex environment, a complicated mechanism is necessary to maintain stable walking. Human locomotion has been well documented, including gait changes due to normal aging and different pathologies [1,2].

Most of the published literature focuses on steady-state locomotion. However, locomotion is not limited to steady-state body transportation. It needs to be adapted immediately to a permanently changing environment, especially for turning and gait termination [3,4], the latter of which is an important transitional task in everyday life [5]. Gait termination puts special demands on postural control, as a transition occurs from a dynamic situation (walking) to a quasi-static situation (standing) [6].

In everyday life, we are often exposed to the movement of scenery during gait termination, such as motor vehicles, trains, and digital signage [7]. It is known that movement of the visual surroundings induces head and body displacements in the same direction as that of the visual stimulus [8]. However, to the best of our knowledge, there have been no reports on the correlation between optic flow and gait termination that requires complex postural adjustments. In this study, we investigated the effects of

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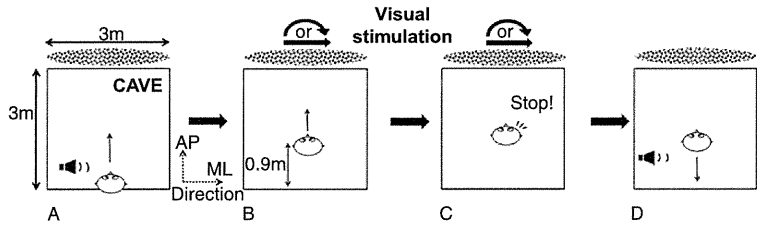

Figure 1. Scheme for one trial in the experiment. (A) Subjects start walking as soon as they receive an acoustic cue. (B) Control: random-dot pattern (RDP) vanishes when subjects pass the 0.9 m point. Optic flow: RDP starts moving horizontally or rotationally when subjects pass the 0.9 m point. (C) Subjects stop walking as soon as they notice the start of optic flow. (D) Subjects walk backwards as soon as they receive the third acoustic cue.

optic flow on body deviation and walking velocity during gait termination.

Material and methods

Twenty-three healthy subjects (19 males and 4 females, aged in their twenties) completed the study. Informed consent was obtained from all subjects, and all experimental procedures were approved by the Ethics Committee of Osaka University, School of Medicine and the Ethics Committee for Human and Animal Research of the Human Stress Signal Research Center at the National Institute of Advanced Science and Technology.

We used the VR system, a projection-based system that surrounds the subject with four screens each measuring 9 m^2 (CAVE; Electronic Visualization Laboratory, University of Illinois, Chicago, IL, USA) [9,10]. Subjects wore a helmet loaded with an infrared CCD camera with two-way mirrors enabling them to see normally, and polarized glasses to resolve the stereoscopic imagery. An electromagnetic tracking system attached to the helmet determined the location and angle of the user's head orientation. Data from the CAVE were collected at a sampling rate of 60 Hz.

The experiment began with baseline trials (control), in which, first, a random-dot pattern (RDP, diameter of 3 m on the screen and view angle of around 70°) was projected on a dark screen. Subjects were instructed to start walking when they received an acoustic cue that rang for 4 s after a first cue, and to stop with their feet side by side immediately after recognizing that the projected image had disappeared, leaving only darkness on the front screen, which automatically occurred when they had walked more than 0.9 m (Figure 1). Baseline trials were continuously repeated eight times. These baseline trials were followed by two experimental trials: horizontal movement (HM) and rotational movement (RM). The conditions of HM and RM were almost the same as for the control, except for the change of projected image that occurred when subjects had walked more than 0.9 m. During the HM condition, the RDP started moving horizontally at 1.3 m/s to the right, and during the RM condition, the RDP started rotating clockwise at 29°/s. Trials in HM and RM conditions were repeated four times each in a mixed random order as the first set. The second set and the third set were each resumed after a 3 min rest (Figure 2). We defined the anteroposterior (AP) axis as being perpendicular to the front screen and the mediolateral (ML) axis as being perpendicular to the AP axis (Figure 1). We defined the gait termination as the period between the times when visual stimulation started and when subjects stopped walking, namely, the walking rate of acceleration was within ± 0.005 m/s^2 after the highest walking velocity. We defined the difference between maximum and minimum coordinate values of the head on each axis during gait termination as 'body deviation' and the walking velocity at the start point of optic flow as 'walking velocity' (Figure 3).

Data analysis was performed using MATLAB (MathWorks Inc.) and a mixed-factor repeated measures analysis of variance (ANOVA) was conducted to examine the effect of optic flow on body deviation and walking velocity. Because there was no significantly different effect between horizontal and rotational optic flows on body deviation or walking velocity, we analyzed the average effect of both kinds of optic flow. At first, body deviation was plotted and analyzed chronologically

Control	*Set 1*		*Set 2*		*Set 3*
8 trials	*HM trial1,2,3,4 &(random) RM trial1,2,3,4*	*3 minutes' rest*	*Same as set 1*	*3 minutes' rest*	*Same as set 1*

Figure 2. Time course of the whole study. HM, horizontal movement; RM, rotational movement.

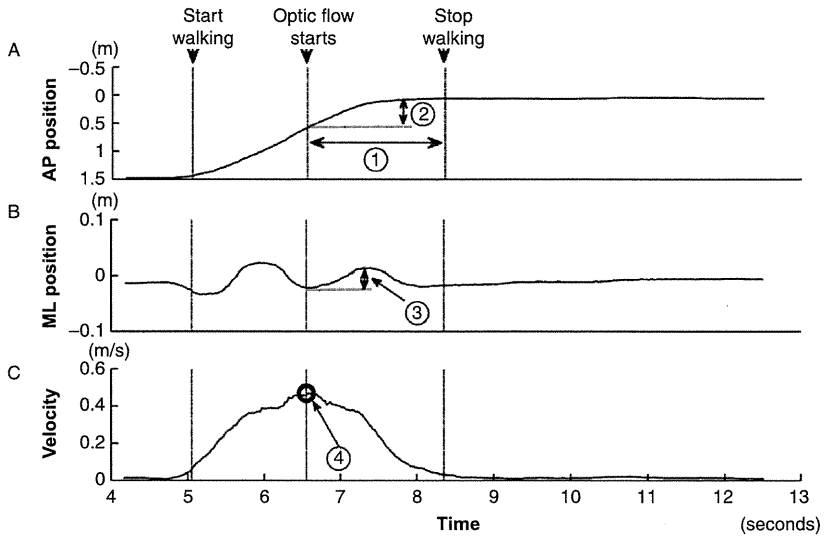

Figure 3. Data for one rotational movement (RM) trial in a representative subject. (A) Anteroposterior (AP) position of body. (B) Mediolateral (ML) position of body. (C) Walking velocity (m/s). Gait termination: period between the times when optic flow starts and when subjects stop walking (1). We defined the difference between maximum and minimum coordinate values of the head on each axis during gait termination as body deviation (2, 3) and the walking velocity at the starting point of optic flow as walking velocity (4).

(Figure 4). Then, it was analyzed by means of trial factor (within-set) and set factor (between-set) (Figure 5). Velocity was analyzed chronologically (Figure 6) and for each condition (Figure 7). Data are expressed as mean ± standard error (SE).

Results

Under the effects of optic flow, subjects terminated their gait stably in the AP axis (Figure 4). ANOVA of body deviation for within-set and between-set demonstrated that the stimulation did not cause significant change of AP deviation for within-set or between-set, or in terms of ML deviation for within-set (Figure 5A, B, C). However, it caused significant flexible change of body deviation in the ML axis for between-set in this study (Figure 5D), but the change was not significantly different from that in the control.

On the other hand, the walking velocity at the start of the termination process dynamically changed in response to the stimulation through the experiment, and the change was significantly different from that in the control (Figure 6).

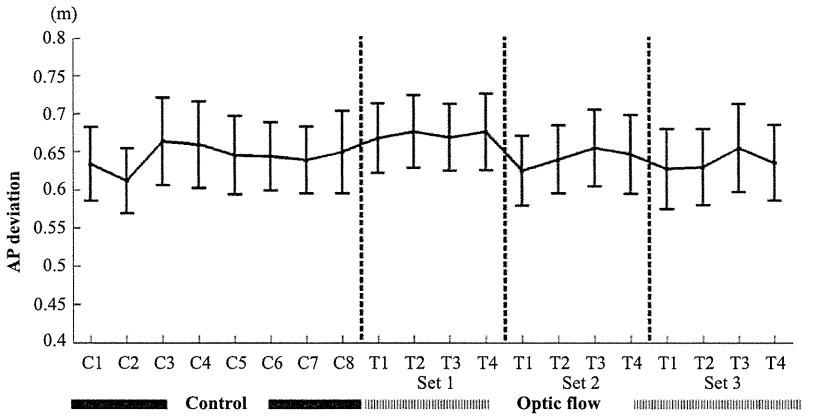

Figure 4. Average changes of body deviation in the anteroposterior (AP) axis throughout the study in chronological order. The data in optic flow were collected from both horizontal movement (HM) and rotational movement (RM) trials. C, control; T, trial of optic flow.

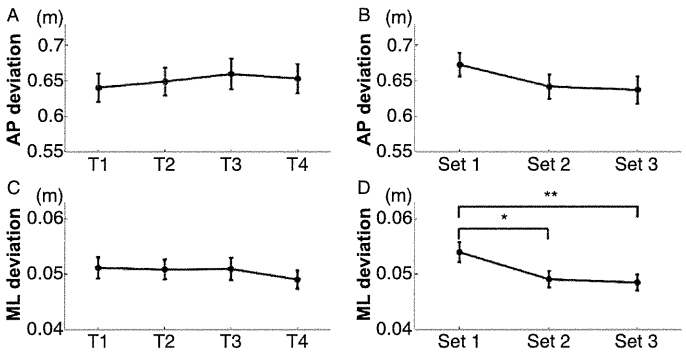

Figure 5. Body deviation during optic flow analyzed in trial and set order. (A) Deviation of the body in the anteroposterior (AP) axis in trial order within-set. (B) Deviation of the body in the AP axis in between-set. (C) Deviation of the body in the mediolateral (ML) axis in trial order within-set. (D) Deviation of the body in the ML axis in between-set. T, trial of optic flow. $*p < 0.05$, $**p < 0.01$.

ANOVA of walking velocity demonstrated that the changes of the velocity were opposite between that for within-set and that for between-set. Although the walking velocity got faster over the trial for within-set, this was considered to be due to short-term flexibility (adaptation) (Figure 7A); it got slower for between-set, which was considered to be due to long-term flexibility (habituation) (Figure 7B). The change of velocity for between-set seemed to be similar to the change of deviation in the ML axis for between-set (Figure 5D).

The time course of gait termination did not change throughout the experiment (data not shown).

Optokinetic nystagmus was always observed in all subjects throughout the HM trials, and it was also observed in all subjects in 79.7% of the RM trials.

Discussion

Because the gait termination process involves a complex mechanism, its analysis is considered to be difficult. Previous reported studies used electromyographic (EMG) evaluation of the thigh and the crus to analyze its mechanism because of the convenience of this approach [4,5]. Evaluation of head deviation should be necessary to investigate the stability of the body during gait termination, but to the best of our knowledge, there have been no reports about this. As mentioned in the Materials and methods section, we defined a new analytical method in this study.

Generally, gait is divided into three parts: gait initiation, stable walking, and gait termination [11]. The velocity at the start of gait termination seems to reflect the preparation period, such as gait initiation and successive stable walking. In this study, we investigated the walking velocity at the start of gait termination to analyze the preparation period of gait termination. The walking velocity is thought to be substantially influenced by the subjects' prediction and adjustment, but no previous studies have reported similar analysis.

In this study, even under the effects of optic flow, subjects were able to stop without severe deviation of the body, especially in the AP axis. Change of body

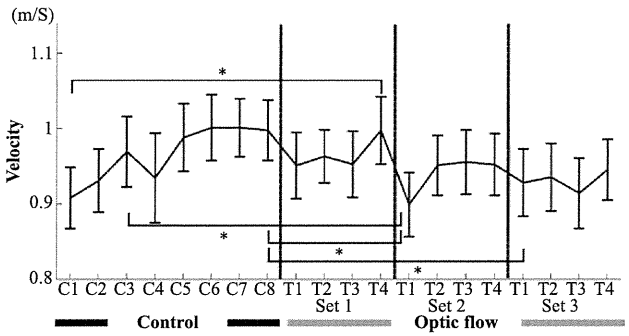

Figure 6. Average changes of walking velocity in the anteroposterior (AP) axis throughout the study in chronological order. The data in optic flow were collected from both horizontal movement (HM) and rotational movement (RM) trials. C, control; T, trial of optic flow. $*p < 0.05$.

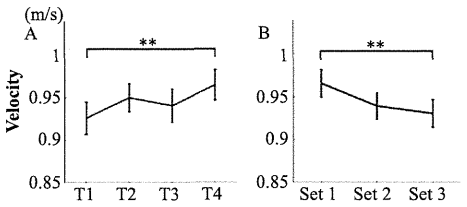

Figure 7. Walking velocity in the anteroposterior (AP) axis analyzed in trial and set order. (A) Walking velocity of the body in trial order in within-set. (B) Walking velocity in between-set. T, trial of optic flow. $^{**}p < 0.01$.

deviation in the ML axis was observed, but it was not significantly different from that in the control. This result is different from a previous report by Fushiki et al. [8], which described that, when subjects were standing, movement of the visual surroundings induced head and body displacements in the same direction as that of the visual stimulus.

The optic flow that we used in this study was also employed in our previous studies [9,10]. A similar strength of optic flow made subjects deviate significantly during standing or walking, so that the strength of the stimulation is considered not to be small. However, it did not cause similar effects during gait termination in the present study. Because optokinetic eye movement was observed during trials, it is supposed that visual input was definitely obtained and processed at the ocular reflex system in the brainstem.

Humans are constantly exposed to the radial optic flow of the surroundings on the front side of the face and/or the AP directional optic flow on the lateral side during daily walking. Previous studies reported that such optic flow had effects on human posture and walking velocity during stable standing or walking [12,13]. In a pilot study, we examined the effects of AP directional optic flow on both lateral sides of the CAVE, and we did not find a significant deviation of the body (data not shown). Because this kind of optic flow requires a quite different set-up from that of HM and RM in this study, we did not include radial or AP optic flow in this study.

One can say that the type of visual stimulation, its duration, and walking distance might also affect the outcome in this study. In our preliminary study, we employed a logo such as Woolmark as a representative of figurative optic flows and found similar results to this study. In the present study, however, we used RDP for the optic flow to avoid figure-specific effects and to provide peripheral visual stimulation, which is thought to be important for optic flow stimulation.

The duration of optic flow between the commencement of optic flow and the end of gait termination was about 2 s in this study. If we increased this optic flow duration to an earlier or later time, it might be difficult to discriminate the effect on gait termination from that on stable walking or standing. Because, in our previous study, the optic flow duration was 3 s and there was significant deviation of posture or gait, the optic flow duration of 2 s in the present study does not seem to be too short. This optic flow duration is appropriate for investigating the effects of optic flow on gait termination.

The walking distance is another possible reason why there was little deviation in the present study. In the present study, the walking distance was within 3 m. In our previous study, subjects deviated in terms of their gait over a similar distance to that in this study, so the distance in this study was not considered to be too short. If we made subjects walk further than 3 m, the maximum walking velocity could be faster and the change of velocity could be greater, but the longer distance was not possible because of the size limitation of CAVE. In this study, the deviation of the body was slight, even if the walking velocity changed dynamically. Therefore, it was supposed that even a greater walking distance may not cause a greater deviation of the body, but another study should be carried out to verify this.

To date, there have been no reports about the effect of optic flow on gait termination. However, our results suggest that, during gait termination, complicated visual information tends to be neglected when all sensory input is integrated in the central nervous system (CNS). It has been described that the basic strategies to stop walking are stored as motor programs and people just rapidly make a decision on which strategy to choose [5,14]. This may be one of the reasons why optic flow had little influence on the deviation of the body during gait termination and its time course. Gait termination is an important process to avoid falling or colliding with an object, so this phenomenon is quite reasonable.

O'Connor et al. [15] studied the effect of optic flow on standing subjects, and observed short-term and long-term flexible changes of postural responses. They defined the short-term changes in within-trial postural responses as adaptation and the long-term changes in between-trial postural responses as habituation. In this study, we also observed short-tem and long-term flexible changes during gait termination with optic flow, and also defined the former as adaptation and the latter as habituation.

Walking velocity when subjects started gait termination rapidly increased through the trial for within-set within a few minutes (adaptation) (Figure 7A). Because such change of velocity was also observed in control trials, it seems that optic flow may have had little effect; instead, subjects' skill may have improved.

It is natural that people become more skilled when they practice the same task repeatedly, and they become able to perform the task more rapidly. On the other hand, the time of the gait termination period did not change. As mentioned above, because gait termination is a kind of programmed routine motion [5], it may take a constant time and rarely be influenced by complicated information from the surrounding circumstances.

On the other hand, the walking velocity at which the subjects started gait termination gradually became lower for between-set (habituation). In contrast with the adaptation, habituation is a change that takes more than several minutes. From first glance at these results, it is possible that the habituation was influenced by repeated optic flows or a few minutes' rest. A reduction of walking velocity was observed when the optic flow started immediately after the control trials without rest, albeit not significantly (Figure 6), suggesting that optic flow definitely influenced this type of velocity change. Although fatigue is one possible reason for the reduction of velocity, if this factor contributed, it might increase the body deviation in AP and ML axes. In this experiment, however, the body deviation in the AP or ML axis did not increase throughout the experiment. Moreover, the body deviation in the ML axis became lower, suggesting that fatigue did not influence subjects' body deviation or walking velocity in this experiment. It is suggested that, during repeated trials, they can predict the optic flow and prepare to stop their forward momentum in advance of it, possibly resulting in the velocity gradually decreasing for between-sets. Because the change of walking velocity and that of ML deviation were similar (habituation) (Figures 5D and 7B), it is also suggested that subjects slowed down effectively in order to terminate their gait stably in the ML axis.

According to the report by O'Connor et al., the direction of the adaptation was similar to that of the habituation [15]. Remarkably, in the present study, the directions of adaptation and habituation were opposite. There have been no previous reports showing this opposite tendency of adaptation and habituation. As we mentioned above, adaptation may be supposed to be a kind of skill improvement (in sport, etc.) and habituation may reflect the stability of motion because habituation correlated with the reduction of ML deviation. It is suggested that these contrary and elastic changes of body velocity may subserve stable gait termination under variable circumstantial changes in human life.

In everyday life, we are often exposed to the movement of scenery during gait termination. In cases where we are exposed to the movement of scenery repeatedly, it is indispensable to become habituated over several minutes in order to stop walking stably. In this study on the effects of optic flow on the gait termination process, the obtained phenomena are more complex than those in previous studies during continuous standing or walking. However, we analyzed the effects of optic flow on gait termination by means of a relatively simple index, such as positional information and velocity information of subjects. Because this experiment is quite easy to reproduce, we should use it to analyze the effects of age on body deviation and walking velocity in a future study. Analyses of the effects of disease, such as in vestibular- or cerebellar-deficient patients, on the adaptation or habituation would also be of great interest.

In conclusion, under the effects of optic flow, subjects terminated their gait stably. This is because the basic strategies to stop walking are stored as motor programs, and complicated visual information may be neglected in the CNS. On the other hand, the velocity of the body at the start of the termination process dynamically changed through the experiment. We observed short-tem (adaptation) and long-term (habituation) flexible changes of velocity. Remarkably, the tendencies of adaptation and habituation were opposite. It is suggested that these flexible changes of the walking velocity subserve the stable process of gait termination under variable circumstantial changes such as optic flow.

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