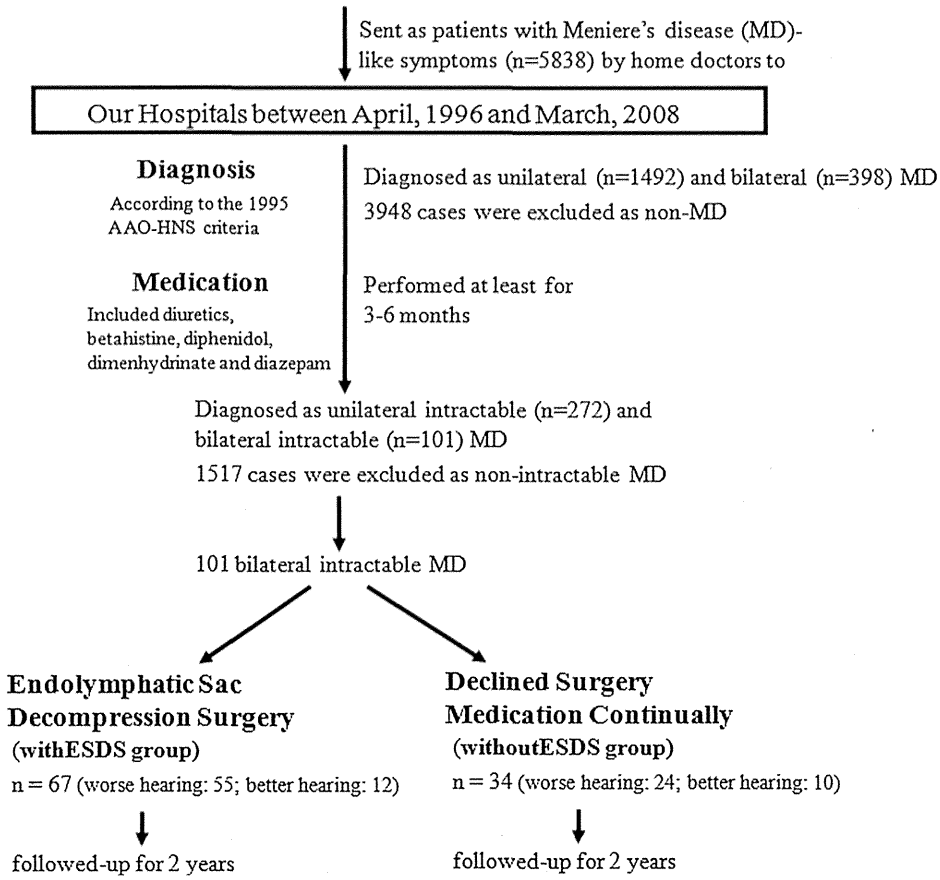

FIG. 1. Flowchart of patient enrollment in the present study. Patients were eligible for enrollment if they had received a definitive clinical diagnosis of unilateral or bilateral Ménière's disease (MD) according to the 1995 AAO-HNS criteria. From April 1996 to March 2008, we offered endolymphatic sac decompression surgery (ESDS) to 101 patients with intractable bilateral MD and performed ESDS in 67 of these patients (withESDS group). We treated the remaining 34 patients, who declined ESDS, with the best available medical therapies (withoutESDS group). All patients were regularly followed up for at least 2 years.

Patients and Treatments

This study was conducted from April 1996 to March 2008. In total, 5838 successive patients who were 20 years or older and exhibited MD-like symptoms were prescreened for eligibility at the vertigo and dizziness department of Osaka University Hospital to determine whether they should be clinically diagnosed as having unilateral (n = 1492) or bilateral (n = 398) MD according to the 1995 American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) criteria (9). The patients were diagnosed according to their clinical symptoms (fluctuating/progressive hearing loss and recurrent vertigo). In total, 3948 patients with diseases other than MD that cause vertigo and dizziness were excluded. Intractable MD was clinically diagnosed in 373 patients (unilateral, n = 272; bilateral, n = 101) who had undergone medical and/or psychological treatment for their clinical symptoms (progressive hearing deterioration of >10 dB with/without recurrent vertigo at least once a month) for at least 3 to 6 months but in whom treatment had failed according to the definition outlined during the 2008 Lancet seminar about MD (10). Patients with unilateral MD or nonintractable MD were also excluded from the study.

A flowchart of the patient selection process is shown in Figure 1. The selection criteria used in this study were originally used in a previous study examining the surgical effects of ESDS (7). Some method used in the latter study was modified as follows. As the follow-up system used in Osaka, Japan, is stricter than that used in Kyoto, Japan, we excluded patients from Kyoto and treated additional patients with intractable MD at our hospital. We diagnosed 101 symptomatic ears (in 101 patients) that exhibited progressive hearing loss with/without recurrent vertigo with intractable MD and offered ESDS to these 101 patients. We subsequently performed ESDS in 67 of the 101 patients (6–8). These patients comprised the withESDS group. We treated the remaining 34 patients, who declined ESDS, with the best available medical therapies (11). These patients comprised the withoutESDS group. All patients were regularly followed up for at least 2 years.

The patients' background data are shown in Table 1. The disease duration, frequency of vertigo episodes, and disease stage were determined for the treated ear. The disease duration was defined as the period from the day of MD symptom onset to the day on which treatment was started (i.e., the day of surgery or the day when surgery was declined). ESDS is a very common treatment strategy for patients with intractable MD (10), and family doctors in Osaka often tell their patients to go to our hospital to undergo ESDS. Thus, a perfect blinded and randomized controlled trial would have been difficult to perform because it would have required us to not perform ESDS in some patients who would otherwise have chosen to undergo the procedure.

TABLE 1. *Backgrounds of the patients with intractable bilateral Ménière's disease*

	Age (yr)	Sex (m/f)	Dur (ipsi-side, mo)	Vf (ipsi-side, a/mo)	Stage (ipsi-side, I/II/III/IV)	Side (wHE:bHE)	wHL (dB)	bHL (dB)
ESDS (n = 67)	52.7 ± 7.4	m31/f36	68.1 ± 25.4	1.8 ± 1.1	0/7/33/27	55:12	68.2 ± 15.7	60.0 ± 18.4
non-ESDS (n = 34)	55.2 ± 8.5	m15/f19	72.0 ± 22.6	1.4 ± 1.5	0/3/19/12	24:10	60.1 ± 13.5	56.5 ± 20.2
	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.	N.S.

Age, age in years (yr) at surgery or at the point when surgery was declined; Sex, m/f; Dur (ipsi-side), duration of disease in months (mo) on the ipsilateral side before surgery or at the point when surgery was declined; Vf (ipsi-side), mean number of vertigo episodes, i.e., the number of vertigo attacks suffered per month (a/mo) on the ipsilateral side during the 6 months before surgery or the point when surgery was declined; Stage (ipsi-side), stages I, II, III, and IV, indicates that the mean result for the worst audiogram for the ipsilateral side obtained during the 6 months before surgery or the point when surgery was declined was less than 25, 25 to 40, 41 to 70, and greater than 70 dB, respectively; Side, number of cases in which the ipsilateral ear exhibited worse (wHE) or better (bHE) hearing function than the contralateral ear; wHL, mean hearing level in dB in the ear that displayed worse preoperative hearing function; bHL, mean hearing level in dB in the ear that displayed better preoperative hearing function. There were no significant differences in the patients' background data between the withESDS and the withoutESDS groups (N.S.).

Surgical Procedures

The technical details of the ESDS procedure are as follows (6–8). Simple mastoidectomy was performed, clearly exposing the endolymphatic sac in the region between the sigmoid sinus and the inferior margin of the posterior semicircular canal. The endolymphatic sac was then opened via an L-shaped incision along the posterior and distal margins of the lateral wall, and the sac was filled with 20 mg of prednisolone. While the prednisolone was dissolving, we prepared a bundle of absorbable gelatin film with fan- and stick-shaped ends. These bundles were attached to one another using biochemical adhesive applied to the stick-shaped end. The fan-shaped end was then inserted into the sac. Small pieces of absorbable gelatin sponge soaked in a high concentration of dexamethasone (32 mg/4 mL) were placed inside and outside the sac lumen, which expanded with the bundle. The dexamethasone-soaked sponges placed outside the sac were coated with adhesive so that the dexamethasone was slowly delivered into the sac as long as possible. The stick-shaped end extending out of the sac was fixed to the front edge of the mastoid cavity using the same adhesive so that the incision in the sac remained open for as long as possible. The mastoid cavity was filled with relatively large pieces of absorbable gelatin sponge dipped in a steroid-antibiotic solution, after which the wound was closed with skin sutures.

Functional Examinations

Episodes of vertigo that lasted for more than 20 minutes were regarded as MD-induced vertigo attacks according to the 1995 AAO-HNS criteria (9). The frequency of vertigo attacks among the patients who underwent ESDS (withESDS group) was calculated relative to the number of vertigo attacks they experienced during the 6 months before they underwent the procedure. The frequency of vertigo attacks in the nonsurgical control group (withoutESDS group) was calculated relative to the number of vertigo attacks they suffered during the 6 months before the day on which they declined ESDS. The frequency of vertigo attacks during the second follow-up year was calculated based on the number of vertigo attacks suffered during the 6-month period from 18 to 24 posttreatment months. When no vertigo attacks occurred during the second follow-up year, it was considered that the patient's vertigo was under "complete" control. "Better" control was defined as a relative score between 0.0 and 0.8, "worse" control was defined as a relative score of 1.2 or higher, and all other scores were defined as "no change." We excluded posttreatment vertigo attacks that originated from the contralateral side when determining the effect of treatment on vertigo.

Hearing function was measured using a pure-tone audiometer and evaluated based on four-tone average values calculated using the following formula: (a + b + c + d)/4 (where a, b, c, and d are the hearing levels at 0.5, 1.0, 2.0, and 4.0 kHz, respectively) as described in the modified 1995 AAO-HNS criteria (7,8). Pretreatment disease stage was assessed based on hearing function. Stages I, II, III, and IV indicate that the mean result for the worst audiogram obtained during the 6 months before treatment was less than 25, 25 to 40, 41 to 70, and more than 70 dB, respectively (9). The worst hearing level detected during the 6 months before treatment was adopted as the pretreatment hearing level, and the worst hearing level detected during the period from 18 to 24 posttreatment months was adopted as the hearing level for the second follow-up year. Improvements of more than 10 dB between the hearing levels detected before and after treatment were regarded as indicating "improvement," reductions in hearing levels of -10 dB or worse were considered to represent "deterioration," and intermediate values were classified as "unchanged." We focused on the posttreatment hearing levels of the ipsilateral (the ear subjected to ESDS) and contralateral ears separately when assessing the effects of treatment on hearing.

Statistical Analysis

The data are shown as ratios of the total number of cases to the total number of treated cases and were statistically analyzed using a 2 × 2 contingency table-based method and the SPSS version 14.0 software (SPSS, Inc., Chicago, IL, USA). Correlations were assessed using the χ^2 test or Fisher's exact test. All reported p values are two-sided, with values of $p < 0.05$ being considered to indicate significance.

All of the statistical analyses performed in the present study were conducted by Dr. Michiko Shuto, a registered statistician who is independent of our organization. The number of patients in the present study was confirmed to be sufficient for statistical analyses.

RESULTS

In total, 101 patients received a definitive clinical diagnosis of intractable bilateral MD. There were no significant differences in the patients' background data between the withESDS group (n = 67) and withoutESDS control group (n = 34) (Table 1).

According to examinations performed from 18 to 24 posttreatment months based on the 1995 AAO-HNS criteria (9), the vertigo of 22 (64.7%) of the 34 patients in the withoutESDS group and 60 (89.6%) of the 67 patients in

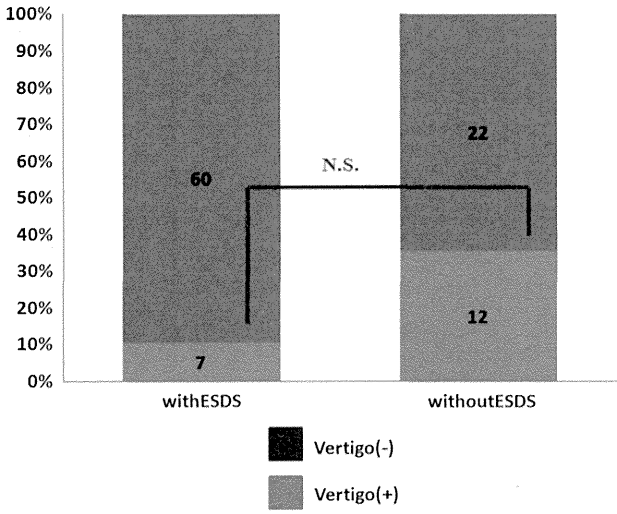

FIG. 2. Graph of 2-year vertigo results in patients with intractable Ménière's disease. All bar graphs show vertigo data for the second posttreatment year. The vertigo-negative columns indicate the percentage of patients who did not suffer any vertigo attacks during this period, and the vertigo-positive columns indicate the percentage of patients who suffered recurrent vertigo during this period. The results for the withESDS group were not significantly better than those for the withoutESDS group (N.S.).

the withESDS group was completely resolved ($p = 0.055$, Fisher's exact test) (Fig. 2).

Of the 24 patients in the withoutESDS group and 55 patients in the withESDS group in whom the ipsilateral ear (the ear subjected to ESDS) exhibited worse hearing function than the contralateral ear, the hearing level of the former ear was preserved in 13 (54.2%) and 52 (94.5%) patients, respectively ($p = 0.007$, Fisher's exact test) (Fig. 3A). Of the 10 patients in the withoutESDS group and 12 patients in the withESDS group in whom the ipsilateral ear exhibited better hearing function than the contralateral ear, the hearing level of the former ear was preserved in 2 (20.0%) and 11 (91.7%) patients, respectively ($p = 0.035$, Fisher's exact test) (Fig. 3B).

Of the 24 patients in the withoutESDS group and 55 patients in the withESDS group in whom the contralateral ear exhibited better hearing function than the ipsilateral ear, the hearing level of the former ear was preserved in 22 (91.7%) patients and 52 (94.5%) patients, respectively ($p = 0.663$, Fisher's exact test) (Fig. 4A). Of the 10 patients in the withoutESDS group and 12 patients in the withESDS group in whom the contralateral ear exhibited worse hearing function than the ipsilateral ear, the hearing level of the former ear was preserved in 8 (80.0%) and 12 (91.7%) patients, respectively ($p = 0.637$, Fisher's exact test) (Fig. 4B).

DISCUSSION

According to previous reports, 10% to 40% cases of unilateral MD gradually become bilateral (2–4). Neurotologists often encounter intractable cases of bilateral MD involving recurrent vertigo attacks and/or progressive sensorineural hearing loss. The surgical options for

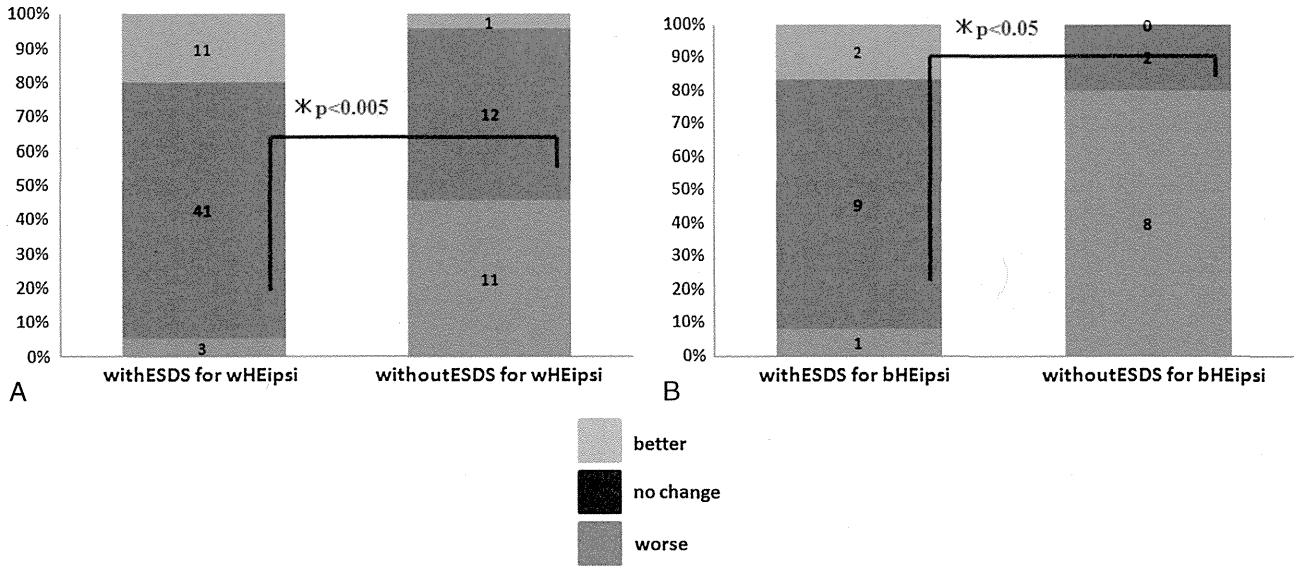

FIG. 3. Graph of the hearing levels of the ipsilateral ears of patients with intractable Ménière's disease at 2 posttreatment years. All bar graphs show the hearing levels recorded during the second posttreatment year for ipsilateral ears that exhibited worse (*wHEipsi*) (*A*) or better (*wHEcontra*) (*B*) preoperative hearing levels than the corresponding contralateral ears. The better columns show the percentage of ears that exhibited hearing level improvements of more than 10 dB, the worse columns show the percentage of ears that displayed hearing level deterioration of more than 10 dB, and the no change columns show the percentage of ears for which intermediate hearing level changes were detected. Regardless of whether the ipsilateral ear exhibited worse or better preoperative hearing function than the contralateral ear, significantly better hearing preservation (better + no change) was achieved in the withESDS group compared with the withoutESDS group during the second posttreatment year (*).

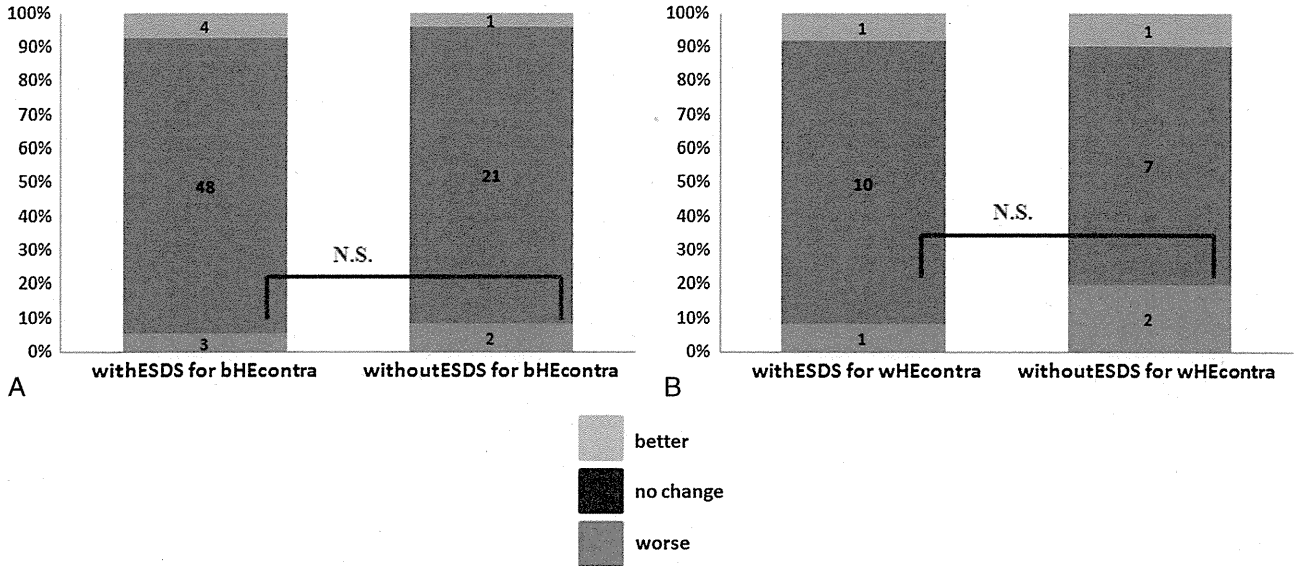

FIG. 4. Graph of the hearing levels of the contralateral ears of patients with intractable Ménière's disease at 2 posttreatment years. All bar graphs show the hearing levels recorded during the second posttreatment year for contralateral ears that exhibited better (*bHEcontra*) (*A*) or worse (*wHEcontra*) (*B*) preoperative hearing levels than the corresponding ipsilateral ears. The better columns show the percentage of ears that exhibited hearing level improvements of more than 10 dB, the worse columns show the percentage of ears that displayed hearing level deterioration of more than 10 dB, and the no change columns show the percentage of ears for which intermediate hearing level changes were detected. Regardless of whether the contralateral ear had worse or better pretreatment hearing function than the ipsilateral ear, the frequency of hearing preservation (better + no change) did not differ significantly between the withESDS and withoutESDS groups during the second posttreatment year (N.S.).

such patients should be considered carefully because surgery can damage the ipsilateral ear, and the hearing function of the contralateral ear is often suboptimal (12,13). There have been few well-designed clinical studies, that is, studies involving control groups or long-term observation, into the efficacy of surgery with respect to vertigo control and hearing preservation in patients with bilateral MD. We have described our 2-year surgical and nonsurgical results in the present article to provide evidence that will facilitate decision-making regarding whether ESDS should be performed for patients with bilateral MD.

In the present study, vertigo attacks were completely controlled in 89.6% of the ESDS group and 64.7% of the nonsurgical control group. Thus, it can be stated that ESDS has a tendency to control vertigo in patients with bilateral MD. However, the frequency of vertigo attacks did not differ significantly between the two groups, which makes sense because a previous study found that the vertigo attacks suffered by patients with bilateral MD are not more severe than those experienced by patients with unilateral MD (14). In addition, previous studies have shown that the vertigo control achieved in patients with bilateral MD was as good as that achieved in patients with unilateral MD during the second postoperative year (7,8,15–19); thus, clinicians should not hesitate to use ESDS to suppress intractable vertigo, even in patients with bilateral MD.

The hearing level of the treated ear was preserved in more than 90% of patients in the ESDS group, regardless of whether the treated ear exhibited better or worse preoperative hearing function than the contralateral ear, which was a significantly better outcome than that obtained in the nonsurgical control group. Our previous data showed that hearing levels had improved by approximately 50% in patients with unilateral MD by the second postoperative year (7,8,15–19). The 20% hearing improvement achieved in the patients with bilateral MD in the present study was obviously not as high; however, in terms of hearing preservation, ESDS might effectively control progressive sensorineural hearing loss in both ears in patients with bilateral MD. Postoperatively, however, we detected a 5.5% deterioration in hearing function in the ears that exhibited the worse preoperative hearing level and an 8.3% hearing deterioration in the ears that displayed the better preoperative hearing level in the patients who underwent ESDS; thus, adequate preoperative informed consent should be obtained. As bilateral hearing impairment might also eventually influence the patient's mental state, clinicians should also prepare patients for the possibility that they might require mental health care and/or hearing supportive devices in the future (20,21).

Our study has some limitations. First, based on the long controversial history of surgical treatment for intractable MD (22,23), it is well understood that the inclusion of a nonsurgical control group is necessary when evaluating surgical outcomes because of the potential for spontaneous symptom resolution. However, ESDS is a very common treatment strategy for patients with intractable MD, as described during the Lancet seminar about the condition (10), and family doctors in Osaka often tell their patients to go to our hospital to undergo ESDS. Thus, a perfect blinded and randomized controlled trial would have

been difficult to perform because it would have required us to not perform ESDS in some patients who would otherwise have chosen to undergo the procedure. We therefore used a control group containing 34 patients (only those who declined to undergo ESDS) in the present study. Second, ideally an ESDS group that was not treated with steroids would have been used as a control group for evaluating the effects of intra–endolymphatic sac steroid treatment, as reported previously (7). However, all of the patients who underwent ESDS without steroids were followed up in Kyoto, and all of these patients were excluded from the study because of the stricter follow-up system used in Osaka compared with that used in Kyoto. Thus, randomized controlled trials involving an additional control group should be performed in the future.

CONCLUSION

The present findings suggest that ESDS combined with local corticosteroid treatment can control progressive hearing loss in both ears in patients with definitively diagnosed intractable bilateral MD at least during the first 2 postoperative years.

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ORIGINAL ARTICLE

Differential diagnosis of vertigo and dizziness in the emergency department

YOSHIYUKI OZONO[1,2], TADASHI KITAHARA[1,2], MUNEHISA FUKUSHIMA[2], TAKAHIRO MICHIBA[1], RYUSUKE IMAI[1], YOUICHIROU TOMIYAMA[1], SUETAKA NISHIIKE[1], HIDENORI INOHARA[2] & HISAKI MORITA[1]

[1]*Departments of Otolaryngology and Emergency, Osaka Rosai Hospital, Japan and* [2]*Department of Otolaryngology, Osaka University, Graduate School of Medicine, Osaka, Japan*

Abstract

Conclusions: To establish a system of differential diagnosis for vertigo/dizziness at the Emergency Department (ED), careful history-taking of complications and examinations of nystagmus should be helpful and therefore prepared by ED staff. *Objectives:* Vertigo/dizziness could come from various kinds of organs for equilibrium, sometimes resulting in an emergency due to the central origin. In the present study, we checked patients' background data at the ED in advance of a definitive diagnosis at the Department of Otolaryngology and examined the significance of the correlation between the data and the diagnosis. *Methods:* We studied a series of 120 patients with vertigo/dizziness, who visited the Departments of Emergency and Otolaryngology between April 2011 and March 2012. At the ED, we first checked patients' backgrounds and carried out neurologic and neuro-otologic examinations. At the Department of Otolaryngology, we finally diagnosed all the patients according to the criteria and classified the origins of vertigo/dizziness into central and non-central diseases. *Results:* The ratio of patients with disease of central origin was 12.5% and that for non-central origin was 87.5%. The risk factors for cerebrovascular disease such as hypertension, heart disease, and diabetes were also the risk factors for central vertigo/dizziness by the chi-squared test. To predict a central origin for vertigo/dizziness, only gaze nystagmus was the significant factor by multivariate regression analysis.

Keywords: *Multivariate regression analysis, complications, nystagmus, CCD Frenzel goggles*

Introduction

Vertigo and/or dizziness could be derived from not only the vestibular peripheral end organs but also various kinds of organs for equilibrium, sometimes resulting in an emergency due to the central origin. This suggests that it is not so easy to make a differential diagnosis and to determine appropriate treatment for vertigo/dizziness at the Emergency Department (ED) [1]. In the present study, to establish a safe and accessible system for the treatment with patients with vertigo/dizziness at the ED, we checked patients' background data at the ED in advance of a definitive diagnosis at the Department of Otolaryngology and examined the significance of correlation between the data and the diagnosis. As vertigo/dizziness specialists, we would like to use the data from the present study to educate ED staff members, including young residents.

Material and methods

The present study was approved by the Ethics Committee of Osaka Rosai Hospital. Informed consent was obtained from each patient with vertigo/dizziness after each treatment in case of emergency.

Correspondence: Tadashi Kitahara MD PhD, Department of Otolaryngology, Osaka University, Graduate School of Medicine, 2-2 Yamada-oka, Suita-city, Osaka 565-0871, Japan. Tel: +81 6 6879 3951. Fax: +81 6 6879 3959. E-mail: tkitahara@ent.med.osaka-u.ac.jp

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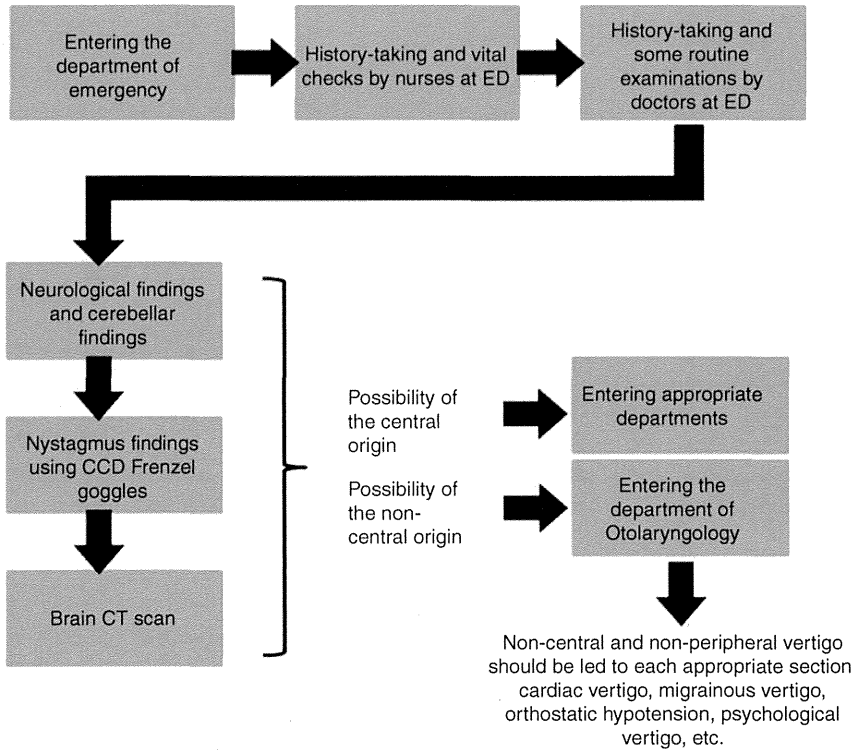

Figure 1. Flow chart of the treatment for patients with vertigo/dizziness at the emergency department.

Patients

We studied a series of 120 patients with vertigo/dizziness who visited the Departments of Emergency and Otolaryngology at Osaka Rosai Hospital between April 2011 and March 2012. In our hospital, only patients with a past history of seeing our doctors were entitled to undergo medical checks and treatments at the ED, i.e. not the first emergency unit.

We treated these patients according to the flow chart used in our hospital (Figure 1). At the ED, we first checked patients' backgrounds and carried out a couple of neurologic and neuro-otologic examinations. At the Department of Otolaryngology, we finally diagnosed all the patients according to all the findings above and the relevant criteria [2] and classified the patients into those with disorders of central origin and those of non-central origin (Table I).

Background data

Patients' background data were obtained by medical and paramedical staff at the ED. Twenty-one factors were included in the exhaustive check list: age (years), sex (male/female), season of onset (spring/summer/autumn/winter), time of onset (morning/afternoon/evening/midnight), use of ambulance (yes/no), admission on foot (yes/no), rotatory vertigo (yes/no), head movement-induced (yes/no), vomiting (yes/no), headache (yes/no), accompanying hearing loss (yes/no), number of complications of hypertension/heart disease/diabetes (0/1/2/3), blood pressure (mmHg), pulse rate (/min), O_2 saturation (%), other cranial nerve disorders (yes/no), cerebellar symptoms (yes/no), gaze nystagmus (yes/no), spontaneous nystagmus (yes/no), CT findings (yes/no), and hospitalization (yes/no). CT scan is routinely performed at the first visit, because MRI is usually performed within a couple of days in our hospital. Regardless, both CT and MRI scans were performed in all 120 patients to enable differential diagnosis between patients with disorders of central origin and those with non-central origin.

Examinations

Neurologic examinations were performed at the ED, included checking 12 cranial nerve functions in brief. Before the start of this study, ED staff members, including young residents, received a lecture on how to examine and record nystagmus with CCD Frenzel goggles by specialists registered in the Japan

Table I. A series of 120 cases with vertigo/dizziness seen at the emergency department.

Group	Disease	*n*
(A) Central vertigo		15
	Cerebellar infarction	6
	Brainstem infarction	5
	Cerebral hemorrhage	4
(B) Non-central vertigo		105
	Benign paroxysmal positional vertigo	48
	Vestibular neuritis	21
	Meniere's disease	11
	Sudden sensorineural hearing loss with vertigo	5
	Ramsay-Hunt syndrome	5
	Orthostatic hypotension	4
	Migrainous vertigo	2
	Cholesteatoma with inner ear fistula	1
	Others	8

We diagnosed 120 patients with vertigo/dizziness and classified them into (A) 15 cases with central origin and (B) 105 cases with non-central origin according to the relevant criteria.

Society for Equilibrium Research to perform a standard level of diagnosis of nystagmus at the ED [3]. First of all, we tried to educate ED staff to check precise characteristics of gaze and spontaneous and positional/positioning nystagmus. However, the staff found this difficult in the first year of our trial for ED system reform. Therefore, we told them only to check gaze and spontaneous nystagmus and judge as (yes/no). When at least three successive beats of nystagmus were rhythmically observed, the nystagmus was classed as significant [4,5].

Neuro-otologic examinations were performed at the Department of Otolaryngology, included checking cochlear and vestibular functions using the audiometer and caloric test, respectively. Hearing function was measured by a pure-tone audiometer and was evaluated based on the five-tone average formulated by (a + b + c + d + e)/5 (where a, b, c, d, and e are hearing levels at 0.25, 0.5, 1, 2, and 4 kHz, respectively). Unilateral hearing loss was judged when the left–right differences of the five-tone average were more than 15 dB. Bilateral hearing loss was judged when the average was more than 30 dB [6]. Vestibular function was measured by a caloric test. For the caloric test, the external auditory canal was irrigated in turn with cold water at 30°C and hot water at 44°C (20 ml) for 10 s. The duration of the induced nystagmus was recorded using chronographs in a dark, open-eyes situation. Unilateral canal weakness was judged when left–right differences of the duration were more than 20% [6].

Statistical analysis

All the data in the present study were treated statistically with the use of SPSS version 14.0 (Chicago, IL, USA). Statistical analysis of the chi-squared test was adopted to compare the exhaustive checklist of 21 factors of the patients' background between patients with vertigo/dizziness of central origin and those of non-central origin (Table II). Univariate regression analysis was used to examine the correlation between central origin of vertigo/dizziness and 15 factors of the patients' background suitable for this analysis (Table III). Furthermore, multivariate regression analysis was used to determine which factor was the most essential (Table IV). A p value < 0.05 was considered as significant for the chi-squared test and multivariate regression analysis, and variables with p values < 0.2 were included in the univariate regression analysis.

Results

The ratio of patients with vertigo/dizziness of central origin was 12.5% (15/120), which included cerebellar infarction, brainstem infarction, and cerebral hemorrhage (Table IA). The ratio of those with non-central origin was 87.5% (105/120), which included benign paroxysmal positional vertigo (BPPV), vestibular neuritis, Meniere's disease, etc. (Table IB). Other non-central vertigo origins included BPPV suspected (n = 3), Meniere's disease suspected (n = 3) and psychological vertigo/dizziness suspected (n = 2).

First of all, we examined the exhaustive checklist of 21 factors listed in the Materials and methods section, to determine whether there was a correlation with the central vertigo/dizziness diseases using chi-squared test (Table II). Male gender, complications of hypertension/heart disease/diabetes, blood pressure, other cranial nerve disorders, cerebellar symptoms, gaze nystagmus, and hospitalization had a significant correlation with the central origin of vertigo/dizziness. Cranial nerve symptoms ($n = 5$), cerebellar symptoms ($n = 1$), gaze nystagmus ($n = 19$), CT findings ($n = 1$), and MRI findings ($n = 1$) were detected in non-central vertigo, because of facial nerve paresis in Ramsay-Hunt syndrome, saccadic smooth pursuit in aging, gaze nystagmus at the acute stage of peripheral vertigo, and hydrocephalus findings in BPPV patients using CT/MRI, respectively.

Then, we examined 16 selected factors suitable for univariate regression analysis to determine if there was

Table II. Factors of patients' background linked to the central origin of vertigo/dizziness.

Factor	Central vertigo (n = 15)	Non-central vertigo (n = 105)	p value
Age (years)	66.5 ± 7.7	61.6 ± 15.0	0.22
Sex (male/female)	10/5	38/67	0.02
Season of onset*	1:8, 2:4, 3:2, 4:1	1:28, 2:31, 3:19, 4:27	0.15
Time of onset†	1:9, 2:5, 3:0, 4:1	1:68, 2:18, 3:6, 4:13	0.38
Use of ambulance (yes/no)	10 (67)	66 (63)	0.77
Admisssion on foot (yes/no)	3 (20)	37 (35)	0.21
Rotatory vertigo (yes/no)	8 (53)	66 (63)	0.48
Head movement-induced (yes/no)	5 (33)	49 (47)	0.33
Vomiting (yes/no)	5 (33)	50 (48)	0.30
Headache (yes/no)	6 (40)	22 (21)	0.10
Accompanying deafness (yes/no)	1 (7)	17 (16)	0.33
Hypertension/heart disease/diabetes‡	0:0, 1:0, 2:4, 3:11	0:48, 1:39, 2:17, 3:1	< 0.0001
Blood pressure (mmHg)	166.1 ± 17.3	141.2 ± 24.9	0.0003
Pulse rate (/min)	72.2 ± 10.8	75.3 ± 15.8	0.06
O_2 saturation (%)	98.1 ± 1.3	98.7 ± 1.2	0.46
Other cranial nerve symptoms (yes/no)	3 (20)	5 (5)	0.03
Cerebellar symptoms (yes/no)	3 (20)	1 (1)	0.0001
Gaze nystagmus (yes/no)	14 (93)	19 (18)	< 0.0001
Spontaneous nystagmus (yes/no)	14 (93)	75 (71)	0.07
Brain CT findings (yes/no)	1 (7)	1 (1)	0.11
Hospitalization (yes/no)	12 (80)	46 (44)	0.0087
Pure-tone audiogram (yes/no)	2 (13)	20 (19)	NA
Brain MRI findings (yes/no)	15 (100)	1 (1)	NA

Values in parentheses are percentages. Chi-squared test revealed the factors of patients' background with significant correlation to the central origin of vertigo/dizziness (shown in bold type).
NA, not applicable (because not performed at ER in the 1st visit).
*1, spring; 2, summer; 3, autumn; 4, winter.
†1, morning; 2, afternoon; 3, evening; 4, midnight.
‡Number of complications.

a correlation with the central vertigo/dizziness diseases (Table III). Male gender, other cranial nerve disorders, cerebellar symptoms, gaze nystagmus, and hospitalization were significant factors to predict a central origin for vertigo/dizziness. Furthermore, multivariate regression analysis revealed that gaze nystagmus was the only factor to have a significant correlation with the central vertigo (Table IV).

Discussion

In the present study, the ratio of vertigo/dizziness patients with central origin was 12.5%, relatively higher than in previous reports, which had a large range from 2 to 20% [7–11]. We suppose that this ratio of the central origin depends on the system employed in the ED in each hospital. In our hospital, the ED is not the first emergency unit but receives only patients with a past history to see our doctors. However, these patients with vertigo/dizziness were taken care of at ED without exception. Therefore, in comparison with the vertigo/dizziness section in the Department of Otolaryngology, many more patients with vertigo/dizziness of non-peripheral origin could be involved in the present study.

First of all, we exhaustively examined patients' background factors to determine whether there was a correlation with central vertigo/dizziness diseases using the chi-squared test. Male gender, complications of hypertension/heart disease/diabetes, blood pressure, other cranial nerve disorders, cerebellar symptoms, gaze nystagmus, and hospitalization had a significant correlation with the central origin of vertigo/dizziness. In our hospital, the ED is not the first emergency unit but receives only patients with a past history to see our doctors. Therefore, these

Table III. Univariate regression analysis of patients' background factors linked to the central origin of vertigo/dizziness.

Factors	*p* value	Odds ratio	95% CI
Age (years)	0.22	0.03	−0.07–0.01
Sex (male)	0.03	0.63	0.08–1.24
Use of ambulance	0.77	0.08	−0.47–0.70
Admission on foot	0.25	−0.39	−1.15–0.22
Rotatory vertigo	0.48	−0.20	−0.74–0.36
Head movement-induced	0.34	−0.28	−0.89–0.27
Vomiting	0.30	−0.30	−0.91–0.25
Headache	0.11	0.46	−0.13–1.02
Accompanying deafness	0.35	−0.50	−1.96–0.36
Other cranial nerve symptoms	0.04	0.80	−0.03–1.57
Cerebellar symptoms	0.01	1.63	0.56–3.15
Gaze nystagmus	< 0.0001	2.07	1.23–3.54
Spontaneous nystagmus	0.10	0.86	0.03–2.32
Brain CT findings	0.16	1.00	−0.63–2.64
Hospitalization	0.02	0.82	0.21–1.58

Univariate regression analysis showed that the factors of male sex, other cranial nerve disorders, cerebellar symptoms, gaze nystagmus and hospitalization were correlated with the central origin of vertigo/dizziness (highlighted in bold type). CI, confidence interval.

patients with vertigo/dizziness at the ED possibly had a couple of internal complications. In previous reports, patients with central vertigo included various kinds of complications, implying cerebrovascular diseases [12–15]. Actually, approximately 60% of cases had complications of risk factors of central diseases such as hypertension, heart disease, and diabetes in the present study. Therefore, this high rate of complications could lead to the significant correlation with central vertigo. Since factors other than complications were also raised by univariate regression analysis, they will be discussed in the next paragraph.

We examined 16 selected factors suitable for univariate regression analysis to determine if there was a correlation with the central vertigo/dizziness diseases. Male gender, other cranial nerve disorders, cerebellar symptoms, gaze nystagmus, and hospitalization were significant factors to predict the central origin for vertigo/dizziness. Furthermore, multivariate regression analysis revealed that gaze nystagmus was the only factor to have a significant correlation with the central vertigo. Regardless of the origin of vertigo/dizziness, the factors male sex, other cranial nerve disorders, cerebellar symptoms, and hospitalization were strongly linked to gaze nystagmus in data for the present study. Gaze nystagmus could also be observed at the acute stage in patients with peripheral vertigo, such as vestibular neuritis and Meniere's disease, not limited within central vertigo. However, we understand that gaze nystagmus could be very important when it is observed in direction, in comparison with spontaneous nystagmus, in changes with head position and in changes with time. Before the start of this study, ED staff members including young residents received a lecture on how to check gaze and spontaneous nystagmus with CCD Frenzel goggles to perform a standard level of diagnosis of nystagmus at the ED [3–5]. Therefore, although there were still limitations to check various kinds of complicated nystagmus for now, this result for gaze nystagmus, we believe, might be reliable and available for the medical training of ED staff.

Table IV. Multivariate regression analysis of patients' background factors linked to central origin of vertigo/dizziness.

Factor	*p* value	Odds ratio	95% CI
Sex (male)	0.054	0.731	0.012–1.528
Other cranial nerve symptoms	0.447	0.445	−0.711–1.664
Cerebellar symptoms	0.185	0.909	−0.342–2.558
Gaze nystagmus	0.001	1.947	0.963–3.491
Hospitalization	0.742	0.167	−0.813–1.260

Multivariate regression analysis showed that the most essential factor was gaze nystagmus (highlighted in bold type). CI, confidence interval.

In conclusion, to establish a safe and accessible system for the treatment of patients with vertigo/dizziness at the ED, careful history-taking of internal complications and examinations of nystagmus should be helpful and prepared by ED staff. Therefore, at the same time, it is also essential to establish an educational system for staff members [3], especially young medical interns or residents, to learn various kinds of vestibular examinations including CCD Frenzel observation for nystagmus at the ED [4,5].

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MAIN ARTICLE

Clinical study of tympanostomy tube placement for patients with intractable Ménière's disease

Y OGAWA, K OTSUKA, A HAGIWARA, A INAGAKI, S SHIMIZU, N NAGAI, S ITANI, Y SAITO, M SUZUKI

Department of Otolaryngology, Tokyo Medical University, Japan

Abstract

Objective: To evaluate the effectiveness of tympanostomy tube placement in controlling symptoms of intractable Ménière's disease.

Methods: Fifteen patients with intractable Ménière's disease underwent tympanostomy tube placement in the affected ear. Post-operative changes in vertigo attacks and hearing level were recorded, and were evaluated according to American Academy of Otolaryngology–Head and Neck Surgery criteria.

Results: At 12 months after treatment, 3 patients (20 per cent) showed complete control of vertigo, 7 (47 per cent) showed substantial control and 2 (13 per cent) showed limited control; 3 patients (20 per cent) required other treatment. At 24 months after treatment, 7 patients (47 per cent) showed complete control of vertigo, 3 (20 per cent) showed substantial control and 1 (7 per cent) showed limited control; 1 patient required other treatment 15 months after tympanostomy tube placement.

Conclusion: There is no definite pathophysiological explanation for the effect of tympanostomy tube placement in reducing vertigo attacks. This treatment is not effective for all patients with intractable Ménière's disease. However, tympanostomy tube placement might be an additional surgical therapeutic option to consider prior to contemplating other, more invasive treatments.

Key words: Middle Ear Ventilation; Vertigo; Dizziness; Meniere's Syndrome; Endolymphatic Hydrops

Introduction

Ménière's disease is an inner-ear disorder characterised by recurrent spontaneous vertigo, hearing loss, tinnitus and ear fullness. It is a condition of cochleovestibular dysfunction and is defined as the idiopathic syndrome of endolymphatic hydrops. In the course of the disease, damage to the vestibular and cochlear organs occurs, which hampers any therapeutic procedure. Vertigo attacks accompanied by nausea and vomiting, and fluctuating sensorineural hearing loss, affect the patient's daily activities.

The common medical therapy is a low-sodium diet, diuretic therapy, corticosteroids and vasodilator therapy. If a patient continues to have episodic vertigo in spite of an adequate trial of medical therapy, surgical management, including transtympanic middle-ear overpressure treatment (using the Meniett® device), intratympanic gentamicin administration, transmastoid endolymphatic sac surgery, transmastoid labyrinthectomy and retrosigmoid vestibular nerve section, should be considered.

Tympanostomy tube placement is one of the surgical management methods for intractable Ménière's disease and is a treatment in which residual disability is less.[1,2] It is considered preferable to the Meniett, intratympanic gentamicin administration and transmastoid endolymphatic sac surgery procedures. Although tympanostomy tube placement has been performed for intractable Ménière's disease at many facilities, only a few studies have been published on the treatment.

We studied patients with tympanostomy tube placement and examined the therapeutic effects after one and two years, comparing our results with previous reports.

Materials and methods

Fifteen patients (5 women and 10 men, aged 26 to 77 years (mean age, 52.4 years)) with intractable Ménière's disease were studied. The patients were followed up for at least two years after the placement of tympanostomy tubes for intractable Ménière's disease.

Diagnosis of definitive Ménière's disease was based on the history of the disease and findings of neuro-otological examinations, which assessed the coexistence of recurrent episodic vertigo and fluctuating cochlear symptoms including hearing loss, tinnitus and aural pressure. Diagnosis was made according to

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the American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS) foundation 1995 guidelines proposed by the Committee on Hearing and Equilibrium.[3] All patients underwent audiological and otoneurological examinations before insertion of the tympanostomy tube in order to exclude other inner-ear disorders and retrolabyrinthine disorders.

All patients had previously received medical management therapy for at least six months before undergoing placement of a tympanostomy tube. The insertion of a tympanostomy tube was suggested to them as a first-line surgical attempt to prevent the occurrence of vertigo attacks or reduce the severity of the attacks. The tympanostomy tube was placed in the anterior-inferior part of the tympanic membrane under topical anaesthesia; anaesthesia was achieved with lidocaine drops in the external auditory canal using iontophoresis.

The treatment outcomes of hearing and equilibrium were evaluated according to the AAO-HNS criteria.[3] We evaluated these outcomes at one and two years after treatment. The frequency of definitive vertigo attacks experienced during the 6 months before treatment was compared with the number of attacks experienced between 6 months and 12 months (1 year) and between 18 and 24 months (2 years) after treatment. In order to express the effect of treatment on vertigo attacks, a numeric value was calculated; that is, the average number of definitive attacks per month after tube placement was divided by the number that occurred prior to tube placement (as per the AAO-HNS guidelines[3]). Control of vertigo, as determined by the numeric value, was categorised as follows: 0 = A, complete control; 1–41 = B, substantial control; 41–80 = C, limited control; 81–120 = D, insignificant control; >120 = E, worse (poor) control; F, secondary treatment initiated because of vertigo-related disability.

Hearing change was also evaluated using AAO-HNS criteria.[3] These criteria consider the thresholds of 0.5, 1, 2 and 3 kHz, but threshold levels of 3 kHz are not usually measured in Japan. Therefore, we considered the average hearing thresholds of 0.25, 0.5, 1 and 2 kHz. The patients' poorest hearing levels using a four-frequency (0.25, 0.5, 1 and 2 kHz) pure tone average were assessed before and after treatment (short-term, 6–12 months; long-term, 18–24 months). Hearing change was defined as: improved (gain of more than 10 dB), unchanged (±10 dB) or worse (loss of more than 10 dB).

Results

Vertigo attacks

At 12 months after treatment, 3 patients (20 per cent) showed complete control of vertigo, 7 (47 per cent) showed substantial control and 2 (13 per cent) showed limited control. Three patients (20 per cent) required other treatment (Figure 1): one patient underwent transmastoid endolymphatic sac surgery and afterwards intratympanic gentamicin administration, and the other two patients underwent intratympanic gentamicin administration, which led to control of the vertigo.

At 24 months after treatment, 7 patients (47 per cent) showed complete control of vertigo, 3 (20 per cent)

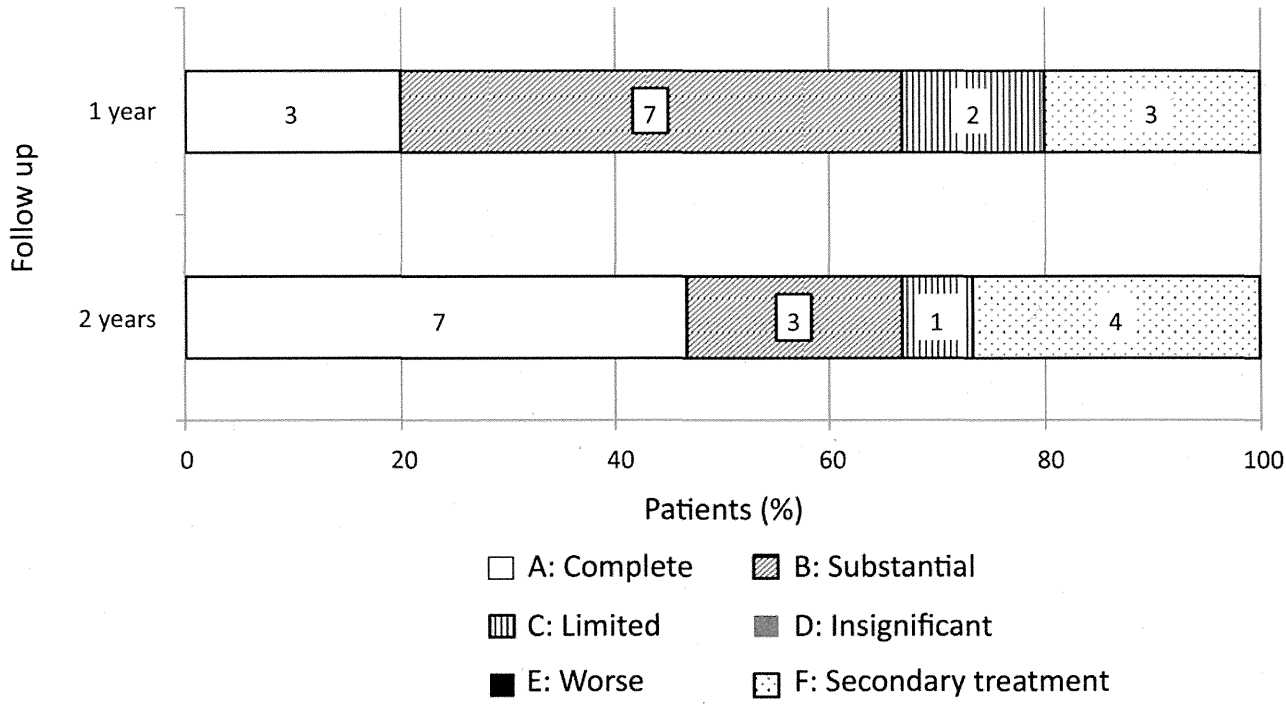

FIG. 1

Control of vertigo attacks following tympanostomy tube placement: comparison of effectiveness at one and two years' follow up.

showed substantial control and 1 (7 per cent) showed limited control (Figure 1). One patient required intratympanic gentamicin administration at 15 months after tympanostomy tube placement.

The percentage of patients who experienced complete control of vertigo obviously increased after two years when compared with the therapeutic effects after one year (Figure 1). Of the seven patients with complete control at the two-year evaluation, two patients showed complete control, three patients showed substantial control and two patients showed limited control at one year.

Changes in hearing

At 12 months after treatment, hearing had improved in 2 patients (13.3 per cent), was unchanged in 8 (53.3 per cent) and was worse in 2 (13.3 per cent) (Figure 2). At 24 months after treatment, hearing had improved in 3 patients (20 per cent), was unchanged in 7 (46.7 per cent) and was worse in 1 (6.7 per cent) (Figure 2). There were no obvious differences between the therapeutic effects at one and two years (Figure 2).

Tympanostomy tube loss

Four patients retained the tympanostomy tube for over two years. The tympanostomy tube fell out within two years in eight of the patients. The tube loss occurred between 6 months and 22 months after insertion (at 6 months in 1 patient, 13 months in 3, 15 months in 1, 18 months in 2 and 22 months in 1). In two patients, vertigo attacks recurred; tube placement was performed again and the vertigo subsided. Another six patients with tube loss suffered no further vertigo attacks and the tubes were not replaced. One patient had vertigo after 2 years; he underwent intratympanic gentamicin administration at 30 months after tube placement. The other patients did not require more invasive surgical therapy. Three of these patients had substantial control of vertigo according to the criteria.

Discussion

The insertion of a tympanostomy tube as a treatment for patients with Ménière's disease was initially proposed by Tumarkin, in 1966.[4] Tumarkin[4] and Lall[5] found that the eustachian tube was often blocked in patients

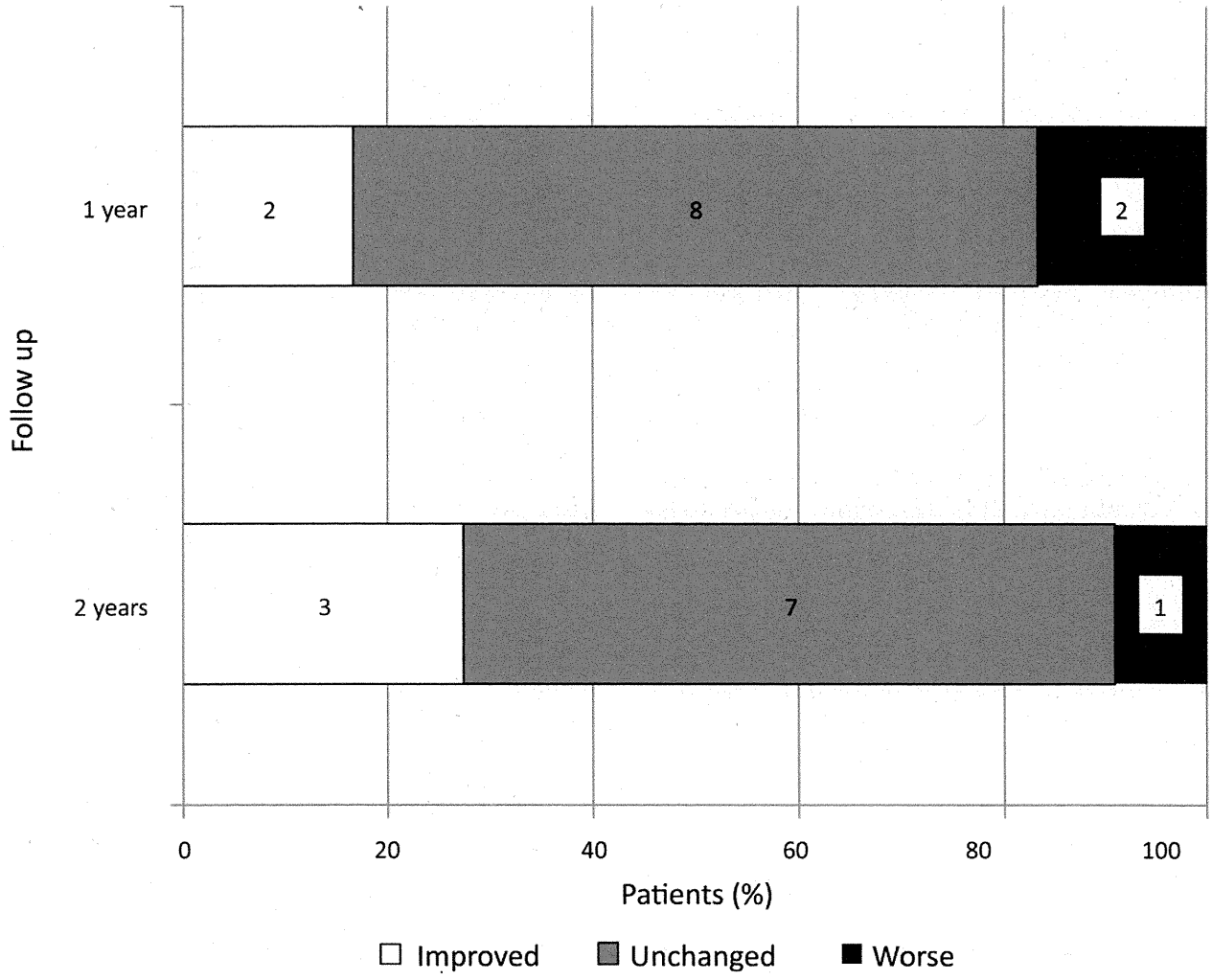

FIG. 2

Changes in hearing level following tympanostomy tube placement: comparison of effectiveness at one and two years' follow up. (Patients that underwent secondary treatment were excluded.)

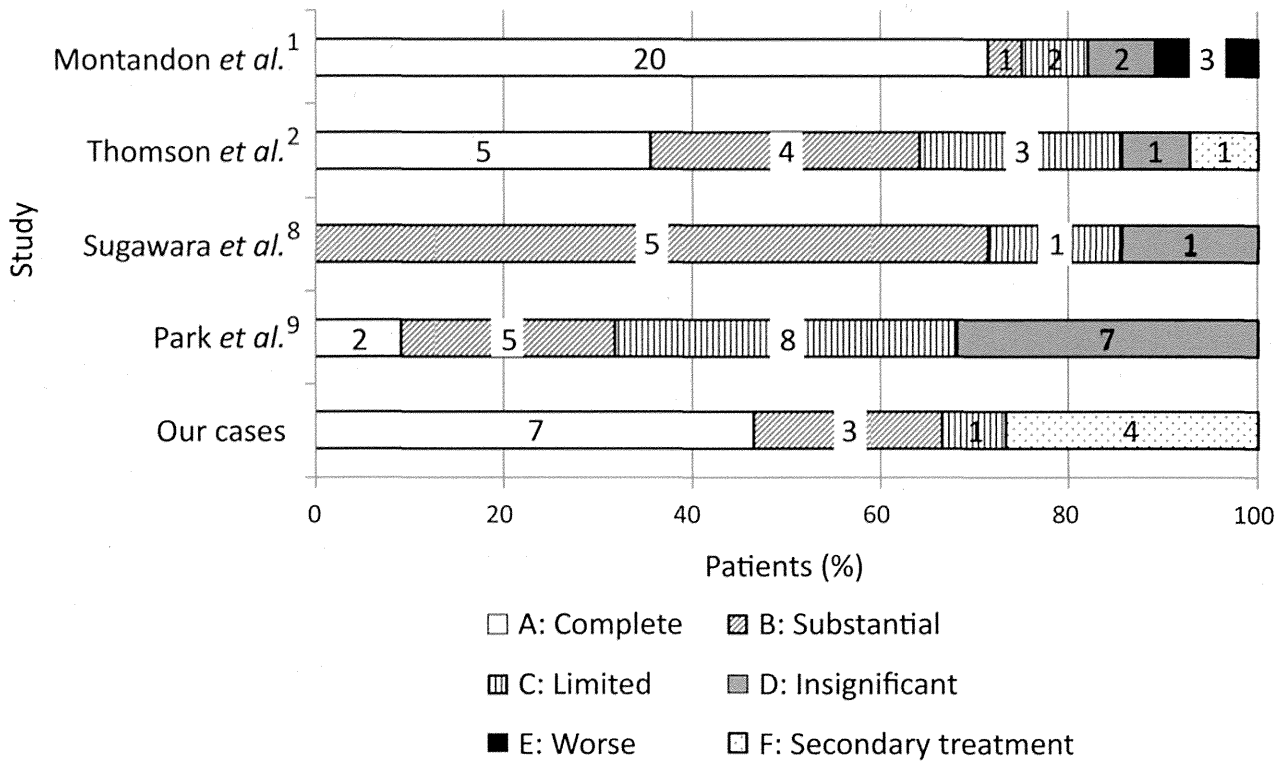

FIG. 3

Therapeutic effect of tympanostomy tube placement on vertigo attacks: comparison with previous reports.

with Ménière's disease. The conclusions were supported by the concept that endolymphatic hydrops in Ménière's disease was correlated with eustachian tube dysfunction. In 1975, Cinnamond[6] disputed this concept by stating that eustachian tube dysfunction was not a consistent feature of Ménière's disease and that the use of a grommet tube to alleviate symptoms was futile. The therapy was also rejected by Hall and Brackmann,[7] in 1977.

In 1988, Montandon *et al.*[1] reported reintroducing this treatment clinically; they treated 28 intractable Ménière's disease patients with tympanostomy tubes. The findings showed improvement or a complete remission of vertiginous attacks in 23 patients (82 per cent). In 1998, Thomsen *et al.*[2] compared the effect of 2 surgical modalities in 29 intractable Ménière's disease patients: 15 patients underwent transmastoid endolymphatic sac surgery and 14 underwent tympanostomy tube placement. There were statistically significant reductions in dizzy spells for patients in both groups post-operatively, but there were no statistical differences between the groups. Two of the patients in the sac surgery group developed severe hearing loss. The authors concluded that tympanostomy tube placement should be the first choice of surgical treatment for Ménière's disease patients who have vertiginous symptoms refractory to medical treatment.

Our results showed that tympanostomy tube placement in the affected ear offered complete control of vertigo in three patients and substantial control in seven patients at one year, and complete control in seven patients and substantial control in three patients at two years. Our findings were similar to those of other studies with respect to the control of vertigo at two years after tube placement (Figure 3).[1,2,8,9]

We found that tympanostomy tube placement had an insignificant effect on hearing, in agreement with the literature (Figure 4). A total of five patients (33.3 per cent) required more aggressive treatment (transmastoid endolymphatic sac surgery or intratympanic gentamicin administration): three patients required this treatment within one year, one patient within one to two years, and one patient after more than two years. In comparison with previous reports, the proportion of patients whose hearing became worse or who experienced an insignificant improvement was smaller, and the proportion of patients with complete, substantial or limited improvement in hearing was larger.[1,2,8,9]

Montandon *et al.*[1] reported a recurrence of vertigo attacks in 15 (53.6%) patients whose tube became obstructed or extruded; the attacks disappeared immediately after the reinsertion of a tympanostomy tube. Among our patients, there were eight whose tube extruded in less than two years, and vertigo attacks recurred in two of the eight patients. The number of vertigo attacks was immediately reduced in these two patients after the tympanostomy tube was replaced.

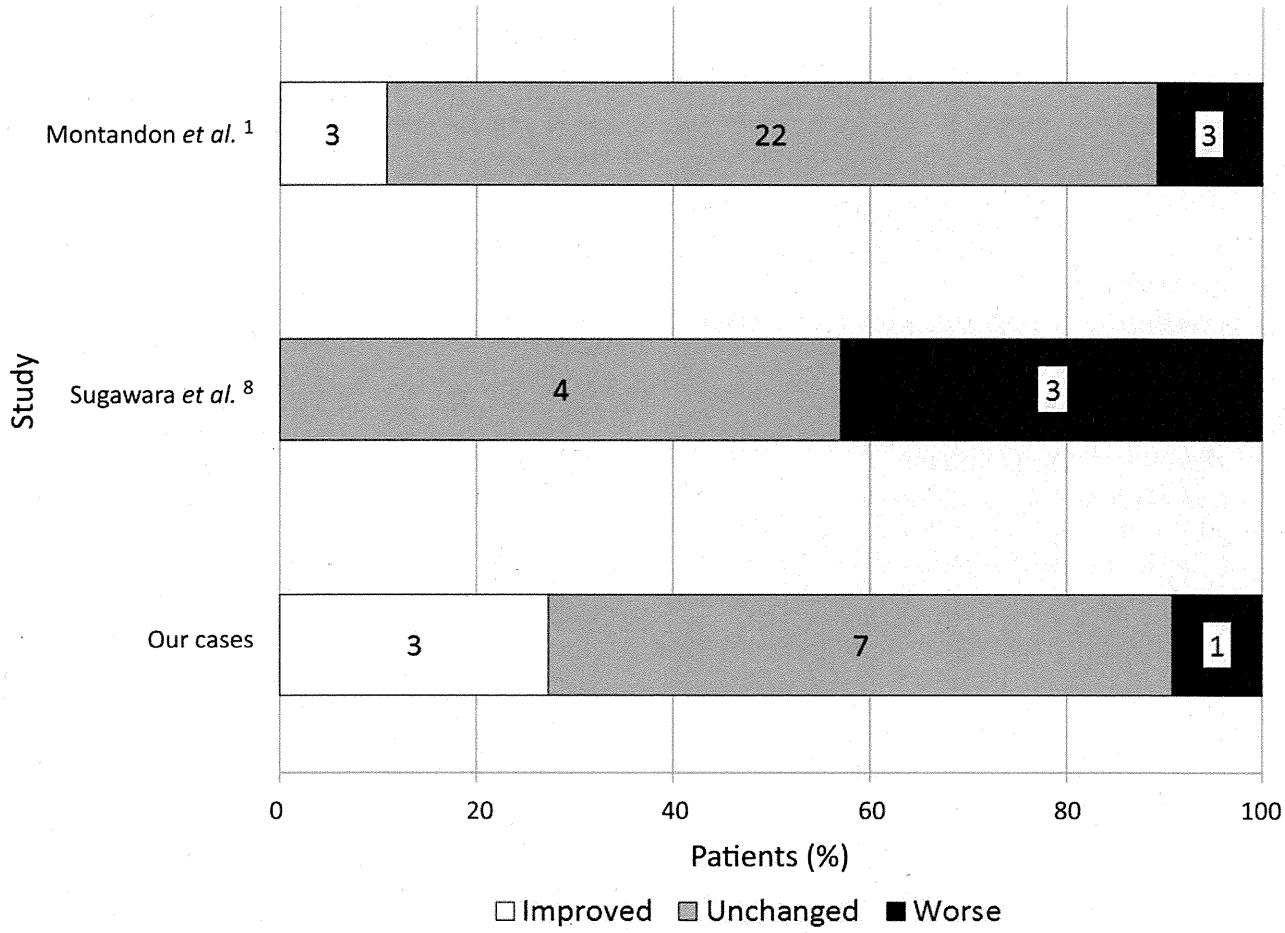

FIG. 4

Therapeutic effect of tympanostomy tube placement on hearing change: comparison with previous reports. (Patients that underwent secondary treatment were excluded.)

There was no recurrence of vertigo attacks in the other six patients, even though the tympanic membrane was closed. It is not clear whether the tympanostomy tube placement was effective or not in these six patients.

The frequency of definitive episodes of vertigo in Ménière's disease has been reported to decrease over time, and many individuals reach a steady-state phase free of vertigo.[10] Pickard[11] described the natural course of Ménière's disease based on a series of 37 patients who received no treatment. Two of the patients in this series had no further attacks following the first consultation, 26 patients (70.3 per cent) ceased to have any further attacks by the end of 12 months and only 5 patients (13.5 per cent) were still suffering from attacks at the end of 2 years. In our study, there were 3 patients in whom the tympanostomy tubes extruded within 6 to 18 months whose evaluation at 1 year showed limited or insignificant control of vertigo. Nevertheless, the evaluation of these patients at two years indicated complete control of vertigo, and it is presumed that they were cured naturally rather than by tube placement.

The exact pathophysiological explanation for the effect of tympanostomy tube placement is still unclear. Park *et al.*[9] investigated whether the reduction in vertigo attacks following ventilation tube insertion was the result of changes in vestibular function. They examined vestibular-evoked potential and sinusoidal harmonic acceleration test findings before and after tympanostomy tube placement in 22 patients with unilateral Ménière's disease. They reported no effects on saccule function or on lateral semicircular canal function after the tube placement. The symptoms of Ménière's disease were believed to result from endolymphatic hydrops.

Kimura and Hutta[12] demonstrated that middle-ear ventilation procedures significantly reduced experimentally induced endolymphatic hydrops in guinea pigs. The authors presumed that the inhibition of hydrops was due to pressure release in the middle ear, and oxygenation of the middle and inner ears. According to our results, tympanostomy tube placement seemed to be effective in reducing vertigo attacks, but actually there were some patients who showed no effects from the tympanostomy tube placement. We cannot discount the possibility that some patients attained complete control of vertigo as a result of natural healing. On the other hand, there

were some patients whose vertigo attacks were obviously reduced by tympanostomy tube placement.

- **Tympanostomy tube placement is a surgical management option for intractable Ménière's disease patients**
- **Few studies have been published on tympanostomy tube placement as a treatment for Ménière's disease**
- **Tympanostomy tube placement has no effect on hearing recovery**
- **Following tube extrusion, vertigo attacks recurred in two patients, but vertigo subsided after repeat tube placement**
- **It is worth trying tympanostomy tube placement in intractable Ménière's disease patients because the treatment is less invasive**

This treatment is not effective in all those who suffer from Ménière's disease. Nevertheless, it is worthwhile trying this technique in intractable Ménière's disease patients because the treatment is less invasive than other surgical procedures such as intratympanic gentamicin administration and transmastoid endolymphatic sac surgery, which also carry the risk of sensorineural hearing loss. In order to use the Meniett device in Japan, private personal importation is necessary and the proceedings are complicated. Tympanostomy tube placement might be an additional surgical option to consider prior to using the Meniett device or undertaking ablative therapy. Tympanostomy tube placement might enable decisions regarding more invasive treatments to be postponed. Tympanostomy tube placement should be the first choice of surgical treatment for Ménière's disease patients, especially elderly patients or those who do not want to undergo more invasive treatments.

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Address for correspondence:
Dr Y Ogawa,
Department of Otolaryngology,
Tokyo Medical University,
6-7-1 Nishishinjuku,
Shinjuku-ku,
Tokyo 160-0023, Japan

E-mail: y-ogawa8@tokyo-med.ac.jp

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原　著

ピッツバーグ睡眠質問票日本版を用いた めまい患者における睡眠障害の検討

許斐　氏元・鈴木　衛・小川　恭生・大塚　康司・萩原　晃
稲垣　太郎・井谷　茂人・斉藤　雄

Assessment of sleep disturbance using the Pittsburgh Sleep Quality Index in patients with dizziness

Ujimoto Konomi, Mamoru Suzuki, Yasuo Ogawa, Koji Otsuka, Akira Hagiwara, Tarou Inagaki, Shigeto Itani, Yu Saito

Department of Otolaryngology, Tokyo Medical University School of Medicine

This study was performed to determine the frequency and degree of sleep disturbance in patients with dizziness using the Pittsburgh Sleep Quality Index, Japanese Version (PSQI-J), and investigate the relationship between dizziness and sleep disturbance. Fifty-two patients (20 male, 32 female) with a chief complaint of dizziness visited the dizziness clinic of the Department of Otolaryngology, Tokyo Medical University, for 3 months in 2013. The patients' age (average ± standard deviation) was 54.4 ± 17.0 years (range, 10–88 years). The average PSQI global score was 7.6 ± 4.2 points, which exceeds the 5.5-point cut-off for insomnia. In total, 67.3% of patients scored >6 points, and 35.8% scored >9 points, indicating definite sleep disturbance. With respect to the demography of disease groups, patients with Meniere's disease scored an average of 7.9 points, those with autonomic imbalance scored 8.8 points, and those with psychogenic dizziness scored 9.7 points; all of these diseases were associated with high PSQI scores. Patients with benign paroxysmal positional vertigo and patients with no abnormal findings showed relatively low scores (6.7 and 5.3 points, respectively). Patients with suspected sleep apnea syndrome, restless leg syndrome, and parasomnias tended to show high scores (>10 points). A high rate and high grade of sleep disturbance were confirmed in patients with dizziness, indicating that sleep quality affects several types of dizziness and vertigo. Understanding sleep disorders is helpful for the diagnosis and treatment of dizziness and provides a new perspective on the etiology of dizziness.

Key words: Pittsburgh Sleep Quality Index, dizziness, vertigo, inner ear, Meniere's disease, orthostatic dysregulation, sleep apnea syndrome

はじめに

めまい症と睡眠障害の関係性を示唆する報告は以前より散見されている[1)~5)]。しかしながら，睡眠障害の原因や評価法は様々であり，睡眠障害の有無や程度を客観的に比較できる尺度は少ない。睡眠時無呼吸症候群（Sleep apnea syndrome；SAS）に対しては，日中の主観的眠気を評価するエップワース眠気尺度（Epworth sleepiness scale；ESS）が一般的に広く使用されている。しかし，これは過眠症患者の症状スクリーニングには適しているが，個々の睡眠障害の原因を分析的に検討するものではない。今回使用したピッツバーグ睡眠質問票（Pittsburgh Sleep Quality Index；PSQI）[6)]は，睡眠障害の評価として広く使用されており，睡眠の質，入眠時間，睡眠時間，睡眠効率，睡眠困難，睡眠薬の使用，日中覚醒困難の7要素の合計得点として算出される（図1参照）。信頼性，妥当性の高い尺度であり，睡眠の質的，量的情報が得られ，個体間や群間での比較が可能となる[7)]。本研究の目的は，めまい患者における睡眠障害の頻度をピッツバーグ睡眠質問票日本版[8)]（Japanese version of PSQI；PSQI-J）を用いて抽出し，めまい発症の背景に睡眠障害が関与する可能性について検討することである。また，めまいの原因疾患別に睡眠障害程度を検討し，睡眠障害の関与が深い疾患の把握を試みた。

対象と方法

1）対象

平成25年4月から3カ月間にめまいを主訴として東京医科大学耳鼻咽喉科めまい外来を受診し，PSQI-Jへの回答が十分であり，精査にてめまいの原因が診断された52例である。

対象の平均年齢は54.4±17.0（平均値±SD）歳（10-88歳）で，内訳は男性20例（50.5±18.9歳），女性32例（56.9±15.4歳）であった。

2）方法

PSQI総合得点（PSQI global score；PSQIG）を算出し[8)]，5点以下を睡眠障害なし，6点以上を睡眠障害ありとし，6～8点を軽度障害，9点以上を高度障害とした。軽度障害とした6～8点は，健常成人のPSQIGが3.78±1.78（平均±SD）より高値であるが，原発性不眠症やうつ病，統合失調症などの精神疾患のPSQIG平均値である9点前後よりも低い範囲とした[8)]。耳鼻咽喉科で疑われるうつ病は軽症が多いという報告があるが[5)]，9点以上はうつ病などの精神疾患と同等以上の明らかな睡眠障害であり，これらを区別して検討した。

疾患群は良性発作性頭位めまい症（BPPV），メニエール病（MD），その他の末梢性めまい，末梢性めまい疑い，自律神経失調症（OD），心因性めまい，所見なし群に分別した。BPPVは確実例のみで，眼振を認めない疑い例は所見なし群とした。MDはメニエール病診療ガイドラインに沿って診断した。末梢性疑い例は，眼振や難聴などめまいと関連する症状や内耳機能低下を伴うものの診断確定に至らない症例とした。OD例については，神経耳科的所見を認めず臨床経過からODを疑う例で，起立後に収縮期血圧21 mmHg以上の低下，脈圧16 mmHg以上の狭小化，心拍数21回／分以上の増加のいずれかに該当し，大国ら[9)]のODに対する問診にて基準を満たした例を確実例，どちらかのみ該当する例を疑い例とした。今回は確実例，疑い例ともOD群として扱った。めまいの原因が複数疑われる例（4例）については，めまい感の直接の原因と考えられる疾患群のなかに振り分けた。

また，PSQIGとめまいの自覚的症状の強さを反映する25項目の質問票である和訳Dizziness Handicap Inventory（DHI）[10)]との相関も検討した。

PSQIにおける睡眠中の他覚的症状についての問い（問10）の結果から，睡眠時無呼吸症（Sleep apnea syndrome；SAS），むずむず足症候群（Restless leg syndrome；RLS），睡眠随伴症が示唆される症例のPSQIGの傾向を検討した。図1で示すように，PSQIでは問10の「同居人がおられますか？」という問いに，「1．どちらもいない」と回答した例を除き，「2．家族／同居人がいるが寝室は別」，「3．家族／同居人と同じ寝室であるが寝床は別」，「4．家族／同居人と同じ寝床」のいずれかを回答した例において，睡眠中の他覚的症状の質問が設定されている。他覚的症状とは，「a．大きないびきをかいていた。」，「b．眠っている間に，しばらく呼吸が止まることがあ

東京医科大学耳鼻咽喉科学教室

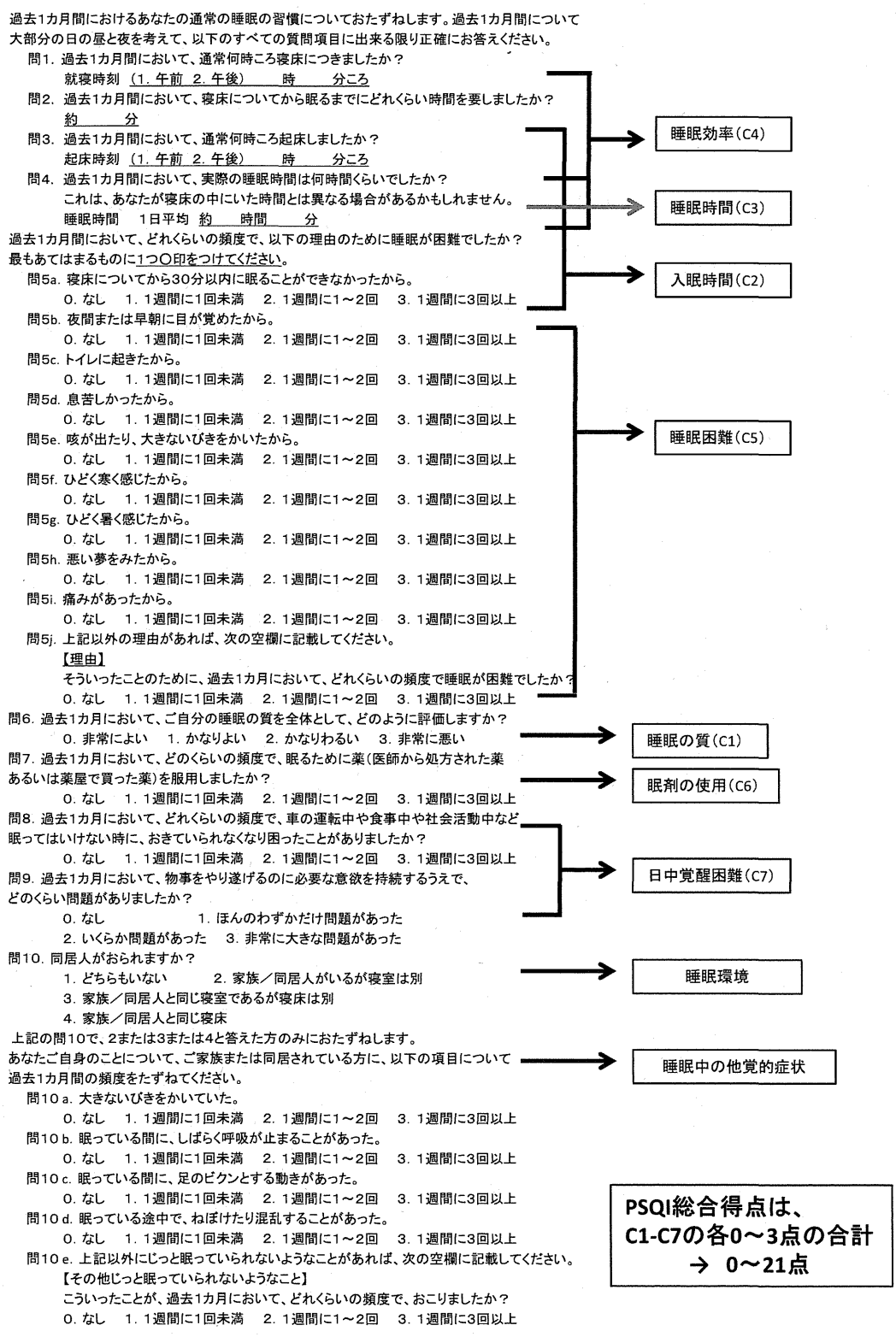

図1 ピッツバーグ睡眠質問票（文献24, 25から引用）

問1から問10まであり，問1～9の中で睡眠の質，入眠時間，睡眠時間，睡眠効率，睡眠困難，睡眠薬の使用，日中覚醒困難の7要素（C1-C7）の合計得点としてPSQI総合得点が算出される。問10は睡眠環境や，無呼吸などの睡眠中の他覚的症状の質問である。