The Journal of Laryngology & Otology
February 2003, Vol. **117,** pp. 104–108

Vestibular-evoked myogenic potential in patients with unilateral vestibular neuritis: abnormal VEMP and its recovery

Kentaro Ochi, M.D., Toru Ohashi, M.D., Shoji Watanabe, M.D.

Abstract

The incidence of inferior vestibular nerve disorders in patients suffering from unilateral vestibular neuritis and the recovery of these disorders were evaluated by monitoring the vestibular-evoked myogenic potential (VEMP). Eight patients ranged from 21 to 73 years that suffered from unilateral vestibular neuritis underwent VEMP and caloric testing. Abnormal VEMP was observed in two of the eight patients with unilateral vestibular neuritis. Two patients were diagnosed as having an inferior vestibular nerve disorder. One of these patients showed recovery of the inferior vestibular nerve function as assessed by the VEMP. Disorders of the inferior vestibular nerve function and their recovery was confirmed by our current results. The time course of recoveries of the superior and inferior vestibular nerve systems were similar in the two patients.

Key words: Vestibular Neuronitis; Vestibular Function Tests

Introduction

Vestibular neuritis is characterized by a sudden onset of vertigo without associated auditory or neurological symptoms. The lesion responsible for vestibular neuritis is thought to affect the superior vestibular nerve system, and is characterized by canal paresis (CP) upon caloric stimulation. It has been reported that the lesion sometimes affects the inferior vestibular nerve system.[1] In contrast, Darlot *et al*.[2] reported that vestibular neuritis spares the inferior vestibular nerve system. The involvement of the inferior vestibular nerve system in patients with vestibular neuritis therefore remains controversial. Although recovery from acute vestibulopathology has been reported, only a recovery from the superior vestibular nerve disorder was referred to.[3–5] This may be related to the absence of methods for evaluating the function of the inferior vestibular nerve system in humans. Recently, the vestibular-evoked myogenic potential (VEMP) has been reported as a potential tool for detecting sacculus-inferior vestibular nerve disorders.[6–12] It has been suggested that the vibration of the footplate stimulates the sacculus, which results in generation of a nerve impulse that is conducted to the sternocleidomastoid (SCM) muscle through a lateral vestibulospinal neural projection.[9] In the study reported in this paper, we evaluated the vestibular function in patients suffering from vestibular neuritis using VEMP as well as caloric testing. In addition, the time courses of recovery of the vestibular function in patients who had abnormal VEMPs were examined.

Patients and methods

Patients

Patients suffering from unilateral vestibular neuritis were investigated. The VEMP and caloric responses of eight patients (three male and five female) aged 21–73 years were monitored and evaluated. They were treated at St. Marianna University Toyoko Hospital. The diagnosis of vestibular neuritis was based on the clinical history and neuro-otological examinations. The chief symptoms is the acute onset of prolonged and severe rotatory vertigo, associated with spontaneous horizontal–rotatory nystagmus, postural imbalance, and nausea in the absence of a simultaneous onset of tinnitus, hearing loss, or other neurological symptoms.[13] Caloric tests were performed to determine ipsilateral hypo- or non-responsiveness.[13] Imaging studies (magnetic resonance imaging (MRI) and/or computed tomography (CT)) were performed to exclude vestibular schwannoma or other intracranial lesions.

VEMP recordings

Clicks with a duration of 0.1 ms and a maximum in their power spectrum at 4 kHz were applied. Rarefaction clicks were presented at a rate of five per second through a headphone, and click intensities were referred to the perceptual threshold for

From the Department of Otolaryngology, St. Marianna University School of Medicine, Kawasaki, Japan.
Accepted for publication: 21 October 2002.

normal subjects in our institution at 0 dB nHL (30 dB peak-equivalent SPL). Electromyographic (EMG) activity was recorded from the upper half of the SCM muscle using bilateral surface electrodes, with a reference electrode on the upper edge of the sternum and a ground electrode on the forehead. Care was taken to place the bilateral electrodes symmetrically. During each recording session, the subject was instructed to rotate the head towards the contralateral side of the tested ear in order to keep the SCM muscle under tension. The EMG signal was amplified (×50 000) and bandpass filtered (5–1500 Hz) using a Synax 1100 (GE Marquette Medical Systems Japan, Tokyo, Japan), and then averaged on the computer using the MacLab software package (AD Instruments, Sydney, Australia). The analysis window was 64 ms wide and the sampling rate was 40×10^3 per second. Usually the responses to 50 stimuli were averaged, but occasionally more stimuli were used to confirm the stability of the response waveform.

The recording strategy was as follows: (1) stimulus intensity of 95 dB; (2) stimulus intensities of 100 dB and 105 dB; (3) stimulus intensity of 95 dB, to check the reproducibility; and (4) decreasing intensities down to the threshold in 5-dB steps. Threshold was determined as the lowest value that evoked a visually detectable response on a computer monitor. The first positive-negative wave was defined as pInII. The pInII ratio was calculated as $100(A_U–A_A)/(A_U+A_A)$, where A_U is the pInII amplitude on the unaffected side and A_A is the pInII amplitude on the affected side. The interaural threshold difference and pInII ratio were used for evaluation of the severity and recovery of the response. The normal ranges of the interaural threshold difference and absolute values of pInII ratios at 95 dB and 105 dB were determined from the means and standard deviations from a previous report[11]: 1.67 ± 2.43, 12.6 ± 8.1, and 13.6 ± 12.1, respectively (all $n = 18$), these three quantities were defined as abnormal when they were greater than two standard deviations above their respective means (i.e., when they were greater than 6.53 dB, 28.8, and 37.8, respectively).

Caloric testing

An electrode was placed laterally in the outer canthus of each eye. A ground electrode was located on the forehead. Horizontal eye movements were recorded to obtain the slow-phase velocity of nystagmus. To bring the horizontal canal into the vertical plane, the head of the supine patient was tilted forward by 30°. The caloric stimulus was achieved by irrigating the external ear with 50 ml of water at 20°C for 20 seconds. An interval of approximately 10 minutes was allowed between each infusion. The caloric test was performed in all patients with the eyes open in complete darkness. Verbal mental arithmetic stimuli were provided to maintain mental alertness. The mean maximum intensity of the slow-phase eye velocity was determined during the period 50–55 seconds after the onset of water irrigation. As for the pInII ratio, this was calculated as a percentage according to $100(V_U–V_A)/(V_U+V_A)$, where V_U is the maximum slow-phase eye velocity on the unaffected side and V_A is the maximum slow-phase eye velocity on the affected side. In patients with spontaneous nystagmus, this was first subtracted when calculating the slow-phase eye velocity of caloric-induced nystagmus. When the CP percentage was larger than 20 per cent, the ear was defined as presenting CP (abnormal caloric response).

Results

Audiological examinations, imaging studies, and definition of the affected ear

The pure-tone threshold was measured at 125 Hz, 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, and 8 kHz using audiometer (AA-61BN; Rion, Tokyo, Japan). Normal hearing was indicated when the hearing threshold at each of these seven frequencies was within 20 dB of normal threshold. The pure-tone audiogram showed bilateral normal configuration in five patients (patient Nos. 1, 3, 4, 5, 7). The residual three patients have symmetrical, high-frequency dominant (down sloping) sensorineural hearing loss (patient No. 2, 2 kHz: 25 dB, 4 kHz: 30 dB, 8 kHz: 45 dB; patient No. 6, 4 kHz: 25 dB, 8 kHz: 40 dB, patient No. 8, 8 kHz: 25 dB) consistent with presbyacusis. Imaging studies presented no abnormal findings in any of the patients. All eight patients had spontaneous horizontal rotatory nystagmus and CP at the initial stage. The direction of the spontaneous nystagmus (slow phase) coincided with the side of CP in all patients. We defined the CP side as the affected one.

TABLE I
DATA SUMMARY OF EIGHT PATIENTS

Patient No.	Age	Sex	Side	Follow-up duration (month)	VEMP
1	37	F	L	2	Normal
2	61	M	R	2	Normal
3	28	M	R	3	Normal
4	21	F	R	6	Normal
5	56	F	R	14	Absent
6	61	M	L	15	Absent
7	44	F	R	18	Normal
8	73	F	R	20	Normal

https://doi.org/10.1258/002221503762624521 Published online by Cambridge University Press

TABLE II
TIME COURSE OF RECOVERY IN CASE 6

		Initial stage	After 7 months	After 15 months
	Threshold difference (dB)	Over 10*	10*	0
VEMP	PInII ratio (95 dB)	100*	100*	11.6
	PInII ratio (105 dB)	100*	31.8*	4.5
Caloric testing	CP %	50.0*	21.7*	1.4

Click intensities are expressed relative to normal hearing level. Asterisks indicate values that are outside the normal range.

Recovery of the superior- and/or inferior-vestibular nerve function after acute vestibulopathies

The results of VEMP in patients suffering from vestibular neuritis at the initial stage are shown in Table I. Two cases presented VEMP abnormality while normal VEMPs were obtained in the other six affected and all eight unaffected ears. Two patients (Nos. 1 and 2) with subjective full recovery at two months after the onset could not be followed up because they did not return to the hospital. Four patients (Nos. 5–8) were followed over one year, and two (Nos. 6 and 8) of these presented full recovery of the superior vestibular nerve function in terms of their caloric response. Two patients who exhibited abnormal VEMP initially presented no VEMP response even at the highest stimulus intensity used in our institute (i.e. 105 dB). One of these patients (No. 5) presented recovery in neither the VEMP nor the caloric testing for 14 months after the onset. In contrast, the other patient (No. 6) showed a gradual recovery of VEMP during the follow-up period. This patient also presented a recovery in CP, as described below.

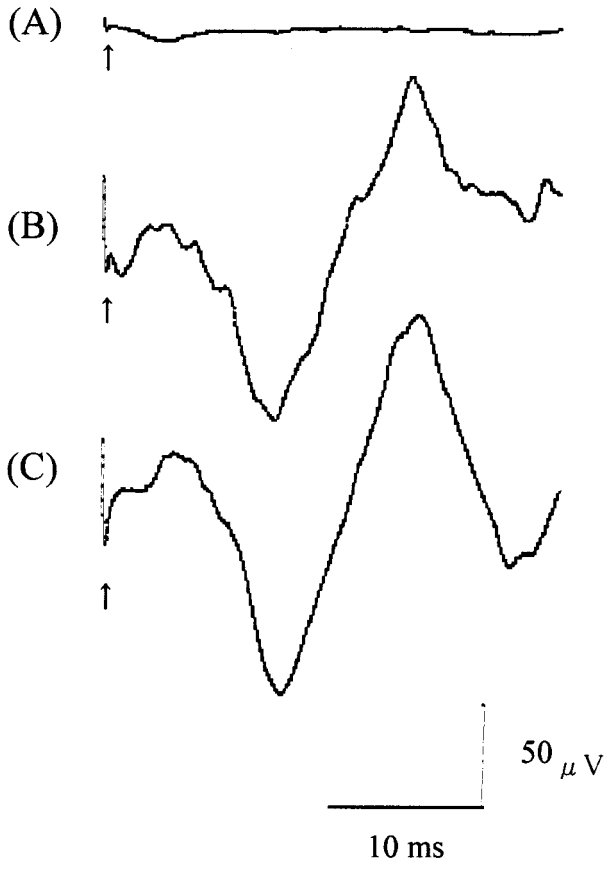

FIG. 1
VEMP waveforms were recorded at the initial stage in the right ear (A), 10 months after the onset in the right ear (B), and in the left ear (C). The stimulus intensity is 105 dB. Arrows indicate the onset of each click. The first 30 ms after the onset of the click stimulus is plotted.

Case report (Patient No. 6)

A 61-year-old male with severe vertigo presented to the out-patient clinic of St. Marianna University Toyoko Hospital. He had right-directed (slow-phase) nystagmus. He had no cochlear symptoms such as hearing impairment or tinnitus. His pure-tone audiogram was normal. The caloric test revealed a right-side CP. No VEMP response was obtained from his right ear even at 105 dB, while a normal response was observed in his left ear. MRI revealed no abnormality. His vertigo was diagnosed as a right-side vestibular neuritis. After seven months the VEMP could be detected at a stimulus intensity of 100 dB and the slow-phase eye velocity upon caloric testing was improved. After 15 months there was no left-right difference in both the VEMP threshold and the caloric response. The pInII ratios both at 95 dB and at 105 dB were within the normal range. Table II shows the time course of recovery, and Figure 1 illustrates the VEMP response waveforms.

Discussion

Murofushi *et al.*[1] reported that 16 (34 per cent) of 47 patients with vestibular neuritis presented an absence of VEMP on the affected side, while all the patients presented normal VEMP on the unaffected side. Darlot *et al.*[2] reported the usefulness of off-vertical axis rotation by examining 54 patients suffering from a unilateral vestibular neuritis, including five patients (nine per cent) presenting signs of probable otolith effects. In contrast, Chen *et al.*[14] reported that seven (88 per cent) of eight patients had bilateral normal VEMPs. The remaining patient had absent VEMP responses bilaterally, and they concluded that vestibular neuritis mainly affects the superior division of the vestibular nerve. Similarly, by analysing the three-dimensional properties of the vestibuloocular reflex in 16 patients, Fetter and Dichgans[15] reported that vestibular neuritis spares the inferior division of the vestibular nerve. Schucknecht and Kitamura[16] observed intact inferior division of the vestibular nerve in four temporal bones from patients that had suffered from vestibular neuritis, while detecting atrophy of the superior division of the vestibular nerve. They also suggested

https://doi.org/10.1258/002221503762624521 Published online by Cambridge University Press

that the clinical and pathological features of vestibular neuritis are consistent with a viral aetiology.[16] Based on post-mortem examinations of the brain and temporal bones, Baloh *et al.*[17] also suggested that an isolated viral infection of Scarpa's ganglia is responsible for the well-documented cases of vestibular neuritis. Recently, reactivation of a latent viral infection has been suspected to cause vestibular neuritis.[18,19] Schulz *et al.*[18] examined the distribution of latent herpes simplex type 1 (HSV-1) in temporal ganglia in 18 human temporal bones of adults using a nested polymerase chain reaction (PCR). HSV-1 specific DNA was detected in at least one of the investigated ganglia in all of these temporal bones: 10/18 (56 per cent) of the geniculate, 11/18 (61 per cent) of the vestibular and nine out of 18 (50 per cent) of the spiral ganglia samples were positive. Arbusow *et al.*[19] hypothesized that selective affliction of the superior vestibular nerve is the result of migration of HSV-1 from the geniculate ganglion along the faciovestibular anastomosis to the ganglion cells of the superior vestibular nerve. They investigated 35 human temporal bones for HSV-1 using nested PCR, but were unable to confirm their hypothesis. The relation between vestibular neuritis and reactivation of latent viral infection through the geniculate ganglion along the faciovestibular anastomosis remains controversial. In our study, two (25 per cent) of the eight patients with vestibular neuritis presented with an apparent VEMP abnormality. This incidence of inferior vestibular nerve disorders in our patients with vestibular neuritis is between that of the reports of Murofushi *et al.*[1] (34 per cent) and Darlot *et al.*[2] (nine per cent). Taking all the available data together, we suggest that vestibular neuritis mainly affects the superior division of the vestibular nerve, with effects in the inferior division of the vestibular nerve being less frequent.

Aschan and Stahle[20] have found that after an attack of vertigo, some involved ears may subsequently exhibit recovery of depressed caloric sensitivity. Strupp *et al.*[4] reported complete recovery of labyrinth function in 21 of 60 patients and partial recovery in 23 patients (giving a total of 73 per cent of patients), while the remaining 16 exhibited a persisting deficit upon caloric irrigation 30 days after the onset of symptoms. Bergenius and Perols[5] presented a long-term follow-up study and showed that 10 (56 per cent) of the 18 patients examined had normal caloric responses seven to eight years after the onset of symptoms. In most of the cases that had recovered during the seven-year follow-up period, this recovery had occurred within 12 months after the onset of symptoms. Okinaka *et al.*[3] found unilateral vestibular hypofunction in 50 percent of patients after five or 10 years. The incidence of superior vestibular nerve recovery in 50 per cent of our patients is consistent with the reports of Okinaka *et al.*[3] and Bergenius and Perols[5] and lower than that reported by Strupp *et al.*[4] As mentioned above, functional recovery of the superior vestibular nerve occurred in more than half of patients suffering from vestibular neuritis. Baloh *et al.*[17] suggested that the inflammation resolves without leading to any permanent damage in patients whose caloric responses return to normal.

In addition, in our study a gradual recovery in inferior vestibular nerve function was observed in one of the two patients who had an inferior vestibular nerve disorder. The time courses of recovery of the superior and inferior divisions of the vestibular nerve were similar in each case: one patient showed recovery of the functions of both superior and inferior vestibular nerves, while the other patient showed recovery in neither vestibular nerve. We have previously reported the recovery of inferior vestibular neural system disorders by monitoring the VEMP in two patients suffering from acute sensorineural hearing loss with severe vertigo.[12] There we suggested that the time course of recoveries of the superior and inferior vestibular neural systems, and of the cochlear neural system, were somewhat different in the two cases reported. The discrepancy between that study and the present one may be due to the small number of patients used to assess the recovery pattern of superior and inferior nerve function; it is necessary to investigate more cases to resolve this issue.

Conclusions

This study reports on disorders of the inferior vestibular nerve function and their recovery in patients with unilateral vestibular neuritis. It is suggested that the vestibular neuritis mainly affects the superior division of the vestibular nerve, with effects in the inferior division of the vestibular nerve being less frequent. The time course of recoveries of the superior and inferior vestibular nerve systems were similar in both of our patients.

Acknowledgements

The authors would like to thank Ms. Yurie Oshiki for her support in correcting and analysing data.

References

1 Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG. Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis. *Arch Otolaryngol Head Neck Surg* 1996;**122**:845–8
2 Darlot C, Toupet M, Denise P. Unilateral vestibular neuritis with otolithic signs and off-vertical axis rotation. *Acta Otolaryngol (Stockh)* 1997;**117**:7–12
3 Okinaka Y, Sekitani T, Okazaki H, Miura M, Tahara T. Progress of caloric response of vestibular neuritis. *Acta Otolaryngol Suppl* 1993;**503**:18–22
4 Strupp M, Jäger L, Müller-lisse U, Arbusow V, Reiser M, Brandt T. High resolution Gd-DTPA MR imaging of the inner ear in 60 patients with idiopathic vestibular neuritis: No evidence for contrast enhancement of the labyrinth of vestibular nerve. *J Vestibular Res* 1998;**8**:427–33
5 Bergenius J, Perols O. Vestibular neuritis: a follow-up study. *Acta Otolaryngol (Stockh)* 1999;**119**:895–9
6 Colebatch JG, Halmagyi GM. Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. *Neurology* 1992;**42**:1635–6
7 Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 1994;**57**:190–7

https://doi.org/10.1258/002221503762624521 Published online by Cambridge University Press

8 Uchino Y, Sato H, Imagawa M, Sasaki M, Ikegami H. The sacculo-neck reflex arc of the decerebrate cat. In: Taguchi K, Igarashi M, Mori S, eds. *Vestibular and Neural Front*, Amsterdam: Elsevier Science Publishers, 1994;517–20
9 Robertson DD, Ireland DJ. Vestibular evoked myogenic potentials. *J Otolaryngol* 1995;**24**:3–8
10 Murofushi T, Curthoys IS, Toppie AN, Colebatch JG, Halmagyi GM. Responses of guinea pig primary vestibular neurons to clicks. *Exp Brain Res* 1995;**103**:174–8
11 Ochi K, Ohashi T, Nishino H. Variance of vestibular-evoked myogenic potentials. *Laryngoscope* 2001;**111**:522–7
12 Ochi K, Ohashi T. Recovery of vestibular-evoked myogenic potential: relationship to other neural disorders in two patients with acute sensorineural hearing loss. *ORL J Otorhinolaryngol Relat Spec* 2002;**64**:346–51
13 Brandt T. *Vertigo: Its Multisensory Syndromes*, 2nd edn. London: Springer Verlag, 1999
14 Chen C-W, Young Y-H, Wu C-H. Vestibular neuritis: three-dimensional videonystagmography and vestibular evoked myogenic potential results. *Acta Otolaryngol* (Stockh) 2000;**120**:845–8
15 Fetter M, Dichgans J. Vestibular neuritis spares the inferior division of the vestibular nerve. *Brain* 1996;**119**:755–63
16 Schuknecht HF, Kitamura K. Vestibular neuritis. *Ann Otol Rhinol Suppl* 1981;**90**:1–19
17 Baloh RW, Lopez I, Ishiyama A, Wackym PA, Honrubia V. Vestibular neuritis: Clinical-pathologic correlation. *Otolaryngol Head Neck Surg* 1996;**114**:586–92
18 Schulz P, Arbusow V, Strupp M, Dieterich M, Rauch E, Brandt T. Highly variable distribution of HSV-1-specific DNA in human geniculate, vestibular and spiral ganglia. *Neurosci Lett* 1998;**252**:139–42
19 Arbusow V, Schulz P, Strupp M, Dieterich M, von Reinhardstoettner A, Rauch E, *et al*. Distribution of herpes simplex type 1 in human geniculate and vestibular ganglia: implication for vestibular neuritis. *Ann Neurol* 1999;**46**:416–9
20 Aschan G, Stahle J. Vestibular neuritis. *J Laryngol Otol* 1956;**70**:497–511

Address for correspondence:
Kentaro Ochi, M.D.,
Department of Otolaryngology,
St. Marianna University Toyoko Hospital, 3-435 Kosugi-cho,
Nakahara-hu, Kawasaki City,
Japan 211-0063.

Fax: +81 (044) 711-3316
E-mail: k2ochi@marianna-u.ac.jp

K Ochi, M.D., takes responsibility for the integrity of the content of the paper.
Competing interests: None declared