

ORIGINAL RESEARCH

Galvanic vestibular-evoked myogenic potentials in evaluating damaged sites of vestibular neuritis

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Funding information

:Funding was received by a grant from Far Eastern Memorial Hospital (to Chih-Ming Chang): Grant number FEMH-2015-C-048.

Abstract

Objectives: To test the possibility of pure otolithic organ deficits and validate the histopathological evidence of retrovestibular neural impairment in vestibular neuritis (VN), the authors adopted a topographic survey combining cervical vestibular-evoked myogenic potential (cVEMP) and ocular vestibular-evoked myogenic potential (oVEMP) using various stimulation modes and caloric tests.

Methods: Forty patients with VN were enrolled in this study. All patients underwent pure tone audiometry, acoustic cVEMP, galvanic cVEMP, vibratory oVEMP, galvanic oVEMP, and caloric tests. Different combinations of vestibular tests were further compared and analyzed.

Results: According to vestibular test results in affected VN ears, the proportion (10%) of pure saccular dysfunction was significantly less than that (52.5%) of saccular nerve deficit. The proportion (2.5%) of pure utricular dysfunction was significantly less than that (37.5%) of utricular nerve deficit. The percentage (82.5%) of VN involving the ampullar vestibulo-ocular reflex (VOR) pathway was significantly higher than that (40%) involving the utriculo-ocular reflex (UOR) pathway. The superior, inferior, and total VN percentages were 37.5%, 17.5%, and 45%, respectively. The proportion of inferior VN was significantly less than that of VN involving the superior vestibular nerve.

Conclusion: There were significantly fewer cases of pure otolithic organ dysfunction than vestibular nerve involvement in VN patients. The damage to the ampullar VOR pathway was more significant than that to the UOR pathway, and both pathways might be independent of each other. In addition, the incidence of isolated inferior VN was significantly less than that of VN involving the superior vestibular nerve.

Level of Evidence: Level 3.

KEYWORDS

cervical vestibular-evoked myogenic potential, galvanic vestibular stimulation, ocular vestibular-evoked myogenic potential, vestibular neuritis

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1 | INTRODUCTION

The syndrome of acute-onset prolonged peripheral vertigo without associated hearing or central neurologic symptoms in an otherwise healthy adult is generally termed vestibular neuritis (VN).^{1,2} The acute symptoms of VN commonly persist in the first few days, improve gradually within several weeks, and completely subside after vestibular compensation within 3–6 months.¹ Although the pathophysiology remains uncertain, the etiology is usually considered a viral infection.³ The histopathologic changes in the vestibular system, including degeneration of the vestibular nerve branches and rarely the endorgan neuroepithelium, are thought to be most compatible with the changes in known viral disorders of the inner ear.^{3,4}

The vestibular nerve includes the superior and inferior divisions. The superior vestibular nerve innervates the utricle, hook region of the saccule, and horizontal and superior semicircular canals (SCCs). In contrast, the inferior vestibular nerve innervates the shank region of the saccule and posterior SCC. VN can affect the superior and inferior vestibular nerves together or individually.^{5–7} In epidemiology, the incidence is much higher in superior VN than in inferior VN.^{6,7} This phenomenon could be explained by nerve susceptibility to viral infection,^{3,4,6,8} and vulnerability to nerve swelling and compromised vascular supply in the narrower and longer bony canal of the superior vestibular nerve.^{6,9,10} Traditionally, patients are diagnosed with VN based on an abnormal caloric response in the involved ear^{1,4,6,11} and history of sudden-onset unilateral vestibular dysfunction lasting for days or weeks without cochlear or central nervous system impairments. However, the caloric test only reflects the functions of the horizontal SCC and the subsequent superior vestibular nerve,⁵ and the incidence of inferior VN might be substantially underestimated.

Clinically, the application of vestibular-evoked myogenic potentials (VEMPs) has been widespread in recent years.^{12,13} According to different recording sites, cervical VEMP (cVEMP) and ocular VEMP (oVEMP) are mainly utilized for probing the reflex pathway integrity of the ipsilateral sacculo-collic reflex (SCR) via the inferior vestibular nerve and crossed utriculo-ocular reflex (UOR) via the superior vestibular nerve, respectively.^{14,15} The combined use of cVEMP, oVEMP, and the caloric test may compensate for the shortcomings of the traditional criteria of VN mentioned above.

Three kinds of stimuli, including air-conducted sound (ACS), bone-conducted vibration (BCV), and galvanic vestibular stimulation (GVS), are used to provoke VEMP responses. In terms of stimulation site, ACS and BCV stimuli mainly elicit responses from the otolithic organs; however, GVS stimuli directly provoke vestibular afferents and fire the subsequent neural pathways. Consequently, combining GVS-VEMPs with ACS-VEMPs or BCV-VEMPs can differentiate otolithic lesions from retrootolithic neural disorders.^{16–18} By adding GVS-VEMPs into our inner ear test battery, this study aimed to test the possibility of pure otolithic organ deficits and retrovestibular neural impairments.

2 | MATERIALS AND METHODS

2.1 | Patients

Forty patients diagnosed with VN were retrospectively enrolled in the study. All patients underwent history taking, audiovestibular evaluation, and neurological examination. The VN diagnosis was based on a single episode of acute-onset peripheral vertigo with spontaneous and persistent nystagmus lasting more than 24 h without accompanying auditory or central nervous system symptoms. Those with atypical disease presentation and atypical nystagmus were excluded. The diseased ears were determined according to the fast phase direction of nystagmus, which beat away from the affected side during the acute stage. A battery of inner ear tests, including pure tone audiometry, ACS-cVEMP, GVS-cVEMP, BCV-oVEMP, GVS-oVEMP, and caloric tests, was performed. The Research Ethics Review Committee of Far Eastern Memorial Hospital approved this study.

2.2 | Pure tone audiometry

Pure tone audiometry was obtained to evaluate the mean hearing level, calculated by averaging the hearing thresholds at four frequencies (0.5, 1, 2, and 3 kHz). Hearing loss was defined as a mean hearing level > 25 dB.

2.3 | ACS-cVEMP test

In the ACS-cVEMP test, an active surface electrode was placed on the upper half of the sternocleidomastoid (SCM) muscle with the negative electrode placed at the sternoclavicular junction. The ground electrode was positioned on the suprasternal notch. During VEMP acquisition, the electromyography (EMG) activity of the SCM muscle was measured using an evoked potential system (Cadwell Industries, Inc., Kennewick, USA). According to the ipsilateral SCR pathway of cVEMP, right SCM muscle potential was recorded as the response to the right air-conducted sound, and vice versa. EMG signals were bandpass filtered between 20 and 2000 Hz and amplified. Monaural clicks were administered at the ipsilateral ear through a headphone with a repetition rate of 5 Hz, a stimulus intensity of 105 dB nHL, and a stimulus duration of 0.5 ms. All subjects were instructed to elevate their heads in the supine position to exert the most significant efforts of SCM muscle during the recording. The reactions to 128 click stimuli were averaged for each run, and two reproducible runs were averaged to yield the final ACS-cVEMP response.

2.4 | GVS-cVEMP test

In the GVS-cVEMP test, the settings for EMG recording conditions were the same as those designated for the ACS-cVEMP test. The cathode and anode electrodes for delivering galvanic stimuli were placed on the mastoid process of the test side and the forehead, respectively. The subjects were galvanically stimulated with a

repetition rate of 5 Hz, a stimulus intensity of 5 mA, and a stimulus duration of 1 ms, with and without SCM muscle contraction in a supine position. To ensure the absence of electrical artifacts in the original GVS-cVEMP waveform, we adopted the method in which the response recorded without muscle contraction was subtracted from that recorded with SCM muscle contraction. The reactions to 128 galvanic stimuli were averaged for each run, and two reproducible runs were averaged to provide the final GVS-cVEMP response.

2.5 | BCV-oVEMP test

In the BCV-oVEMP test, the active electrode was positioned inferior to the contralateral eye with the negative electrode positioned 1–2 cm below the corresponding active electrode. The ground electrode was also placed on the suprasternal notch. According to the crossed VOR pathway of oVEMP, left extraocular muscle potential was recorded as the response of the right ear to the forehead bone-conducted vibration, and vice versa. The EMG signals were bandpass filtered between 1 and 1000 Hz and amplified. BCV stimuli with a repetition rate of 5 Hz and a stimulus duration of 0.5 ms were administered at the Fpz site using a hand-held electromechanical vibrator (V201 vibrator; Ling Dynamic Systems, Royston, UK). A customized combination of amplifiers generated the input signal, and the driving voltage was adjusted to create a peak force of 12 Newtons. The examiner's hand supported most of the vibrator's weight, and the axis of the connected bakelite cap was perpendicular to the subject's skull. All subjects were instructed to look upward at a fixed small target more than 2 m from the eyes, with a vertical visual angle of approximately 30–35° above horizontal. The 50 reactions were averaged for each run, and two reproducible runs were averaged to generate the final BCV-oVEMP response.

2.6 | GVS-oVEMP test

In the GVS-oVEMP test, the EMG recording conditions were the same as those mentioned above for the BCV-oVEMP test. The cathode and anode electrodes for giving galvanic stimuli were positioned on the mastoid process of the test side and the forehead, respectively. The subjects were instructed to gaze up or down. They were galvanically stimulated with a repetition rate of 5 Hz, a stimulus intensity of 5 mA, and a stimulus duration of 1 ms in a sitting position. To eliminate electric artifacts, we used the method in which the average of 128 reactions obtained while gazing down was subtracted from that obtained while gazing up for each run. Two reproducible runs were averaged to yield the final GVS-oVEMP response.

2.7 | VEMP outcome assessment

A p13 latency of ACS-cVEMP > 14.7 ms (mean + 2SD) and GVS-cVEMP > 12.4 ms were regarded as delayed in our laboratory. In contrast, a nl latency of BCV-oVEMP > 13.0 ms and that of GVS-

oVEMP > 10.1 ms were also regarded as delayed. Furthermore, the asymmetry ratio of VEMP, defined as the amplitude difference between both ears divided by the amplitude sum of both ears, was considered abnormal if the value was >0.33. Delay, asymmetric (reduced or augmented), and absent responses were defined as abnormal VEMP responses.

2.8 | Caloric test

We performed bithermal caloric testing using cold (30°C) or warm water (44°C) irrigation for patients in the supine position. Their heads were raised at an angle of 30 degrees from the horizontal, and video nystagmography recorded nystagmus. The patient was diagnosed with canal paresis if the maximal difference in slow phase velocities between both ears was >25% of the sum of the slow phase velocities. If the caloric test failed to elicit a response, external ear canal irrigation using ice water was performed to confirm caloric areflexia.

2.9 | Statistical analysis

The abnormal rates of pure tone audiometry, VEMPs elicited by various stimuli, and caloric tests between affected and unaffected ears were compared using McNemar's test. VEMP parameters, including p13, n23, nl, and pl latencies and p13-n23 and nl-pl amplitudes, were compared using Student's *t* test. Comparisons between different vestibular test combinations were calculated using McNemar's test, χ^2 test, or Fisher's exact test. A *p* value <.05 was considered statistically significant.

3 | RESULTS

Forty patients with unilateral VN, including 19 (47.5%) men and 21 (52.5%) women, were enrolled in the study. Their ages ranged from 16 to 76 years, with a mean of 51.0 years. Twenty-three cases of VN occurred on the left side, and 17 cases occurred on the right side.

3.1 | Pure tone audiometry

The abnormal rate of pure tone audiometry was 45% in affected ears and 42.5% in unaffected ears, revealing no significant difference between affected and unaffected ears (*p* = 1.000, McNemar's test; Table 1).

3.2 | Cervical VEMP test

The abnormal rate of ACS-cVEMPs in affected ears was 62.5%, including six delayed responses, five decreased responses, and 14 absent responses, which was significantly higher than that (15%) in unaffected ears (*p* < 0.001, McNemar's test; Table 1; Figure 1). The

TABLE 1 Comparisons of inner ear tests between affected and unaffected ears in VN patients

		Pure tone audiometry			Caloric test		
		Unaffected ears			Unaffected ears		
		Normal	Abnormal	Total	Normal	Abnormal	Total
Affected ears	Normal	21	1	22 (55%)	4	3	7 (17.5%)
	Abnormal	2	16	18 (45%)	30	3	33 (82.5%)
	Total	23 (57.5%)	17 (42.5%)		34 (85%)	6 (15%)	
	<i>p</i>	1.000			<.001		
		ACS-cVEMP			GVS-cVEMP		
		Unaffected ears			Unaffected ears		
		Normal	Abnormal	Total	Normal	Abnormal	Total
Affected ears	Normal	15	0	15 (37.5%)	19	0	19 (47.5%)
	Abnormal	19	6	25 (62.5%)	20	1	21 (52.5%)
	Total	34 (85%)	6 (15%)		39 (97.5%)	1 (2.5%)	
	<i>p</i>	<.001			<.001		
		BCV-oVEMP			GVS-oVEMP		
		Unaffected ears			Unaffected ears		
		Normal	Abnormal	Total	Normal	Abnormal	Total
Affected ears	Normal	22	2	24 (60%)	23	2	25 (62.5%)
	Abnormal	12	4	16 (40%)	13	2	15 (37.5%)
	Total	34 (85%)	6 (15%)		36 (90%)	4 (10%)	
	<i>p</i>	.013			.007		

Note: McNemar’s test calculated the *p* value.

Abbreviations: ACS, air-conducted sound; BCV, bone-conducted vibration; GVS, galvanic vestibular stimulation; cVEMP, cervical vestibular-evoked myogenic potential; oVEMP, ocular vestibular-evoked myogenic potential; VN, vestibular neuritis.

abnormal rate of GVS-cVEMPs in affected ears was 52.5%, including four delayed responses, 13 decreased responses, and four absent responses, which was also significantly higher than that (2.5%) in unaffected ears (*p* < 0.001, McNemar’s test; Table 1; Figure 1).

3.3 | Ocular VEMP test

The abnormal rate of BCV-oVEMPs in affected ears was 40%, including two delayed responses, seven decreased responses, and seven absent responses, which was significantly higher than that (15%) in unaffected ears (*p* = .013, McNemar’s test; Table 1). The abnormal rate of GVS-cVEMPs in affected ears was 37.5%, including two delayed responses, six decreased responses, and seven absent responses, which significantly exceeded that (10%) in unaffected ears (*p* = .007, McNemar’s test; Table 1).

3.4 | Caloric test

Thirty-three (82.5%) affected ears showed abnormal caloric responses, including canal paresis in 13 ears and areflexia in 20 ears. Six (15%) unaffected ears exhibited abnormal caloric responses,

including canal paresis in four ears and areflexia in two ears. The abnormal rate differed significantly between affected and unaffected ears (*p* < .001, McNemar’s test; Table 1).

3.5 | Topographical analysis of SCR pathway

Those with normal GVS-cVEMPs but abnormal ACS-cVEMPs were considered as having pure saccular dysfunction (Figure 2A; Table 2), while those with abnormal GVS-cVEMPs were regarded as having inferior VN involving saccular afferents (Figure 2B,C; Table 2). The saccule might be either spared (Figure 2B) or involved (Figure 2C) in the latter group.

The proportion of pure saccule dysfunction was 10% in the affected ears, significantly less than that of inferior VN involving saccular afferents (52.5%) (*p* = .001, McNemar’s test; Table 2).

3.6 | Topographical analysis of UOR pathway

Patients with normal GVS-oVEMPs but abnormal BCV-oVEMPs were considered as having pure utricular dysfunction (Figure 2D; Table 2), whereas those with abnormal GVS-oVEMPs were regarded as having

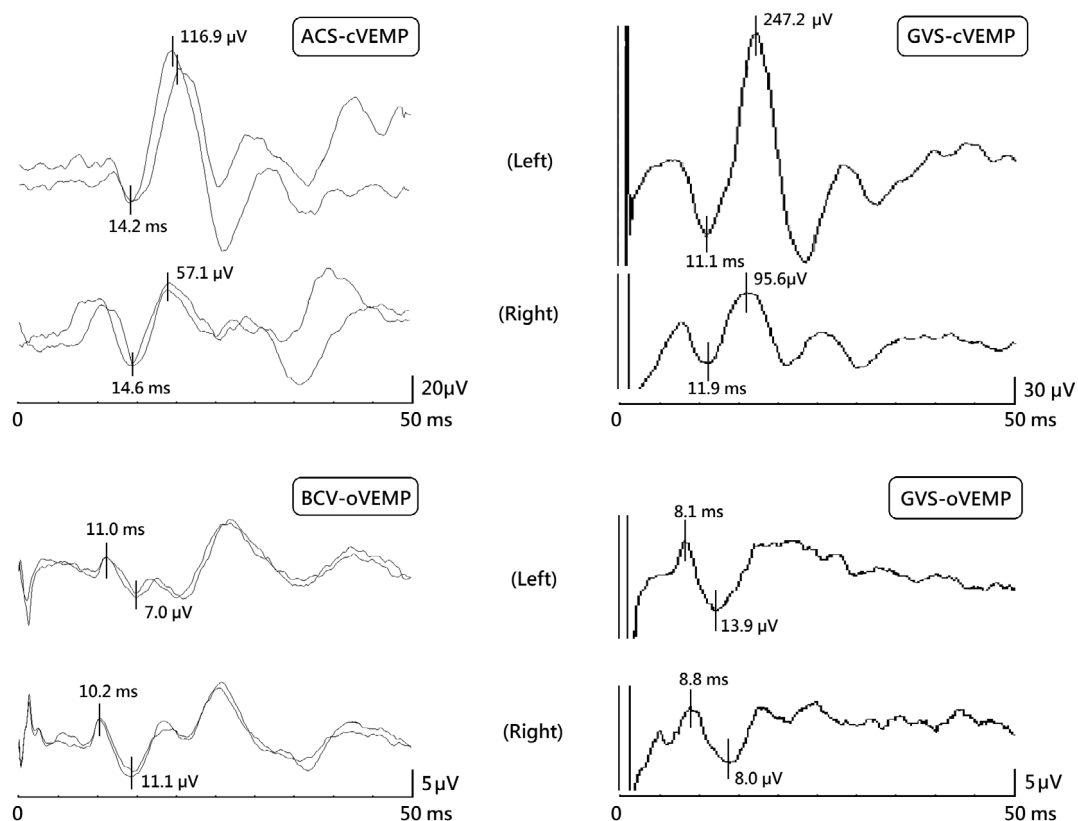


FIGURE 1 Cervical and ocular vestibular-evoked myogenic potentials (cVEMP/oVEMP) evoked by air-conducted sound (ACS), bone-conducted vibration (BCV), and galvanic vestibular stimulation (GVS) modes in a 34-year-old male patient with right vestibular neuritis. The results showed right reduced ACS-cVEMP and GVS-cVEMP responses

superior VN involving utricular afferents (Figure 2E,F; Table 2). The latter group might have either utricular dysfunction (Figure 2F) or not (Figure 2E).

In the affected ears, the proportion of pure utricle dysfunction was 2.5%, significantly less than that of superior VN involving the utricular afferents (37.5%) ($p < .001$, McNemar's test; Table 2).

3.7 | SCR pathway vs. UOR pathway

In the affected ears, the proportion of inferior VN involving saccular afferents (Figure 2B,C) was 52.5%, whereas that of superior VN involving utricular afferents (Figure 2E,F) was 37.5%. There was no significant difference between these two groups ($p = .345$, McNemar's test; Table 2).

3.8 | Horizontal Ampullary superior VN vs. Utricular superior VN

Patients with abnormal caloric responses were regarded as having horizontal ampullary superior VN (Figure 2G-I), which meant superior VN involving horizontal SCC and/or ampullary afferents. Those with

abnormal BCV-oVEMPs were regarded as having utricular superior VN, which meant superior VN involving utricle and/or utricular afferents (Figure 2D-F).

The proportion of horizontal ampullary superior VN was 82.5% in the affected ears, significantly more than that (40%) of utricular superior VN ($p = .029$, Fisher's exact test; Table 3). In addition, all patients (100%) with abnormal BCV-oVEMPs also had abnormal caloric responses.

3.9 | Superior VN vs. inferior VN vs. total VN

Patients with abnormal caloric and/or BCV-oVEMP responses combined with normal ACS-cVEMPs represented superior VN, while those with abnormal ACS-cVEMPs combined with normal caloric and BCV-oVEMP responses represented inferior VN. Those with both superior and inferior vestibular nerve involvement represented the total VN.

The proportions of superior, inferior, and total VN were 37.5%, 17.5%, and 45%, respectively. The proportion (17.5%) of inferior VN was significantly less than that (37.5%) of superior VN ($p = .033$, Fisher's exact test; Table 3) and that (82.5%) of superior VN plus total VN ($p < .001$, Fisher's exact test; Table 3).

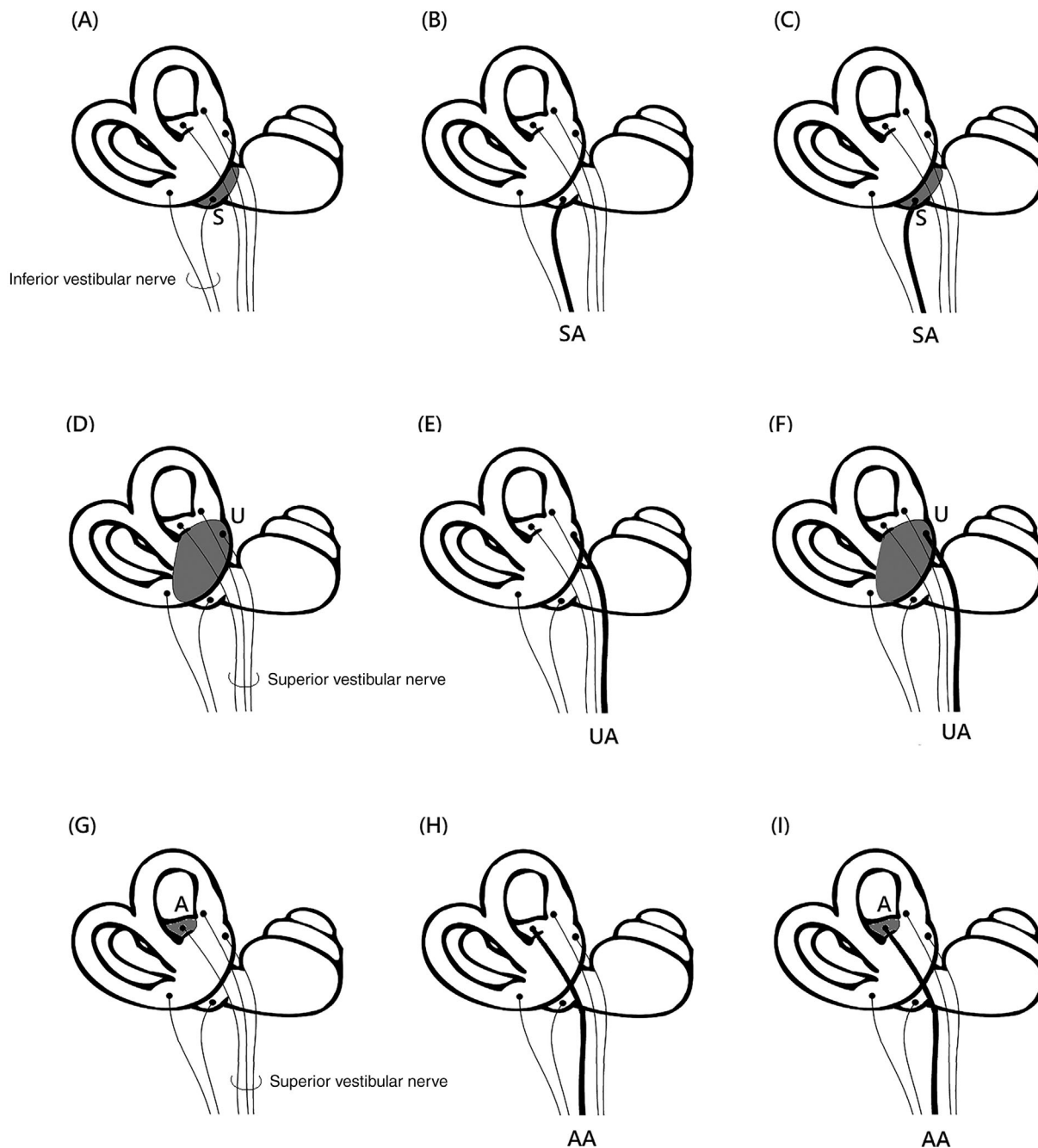


FIGURE 2 The involvements of the vestibular system, such as saccule (S), saccular afferents (SA) of the inferior vestibular nerve, utricle (U), utricular afferents (UA) of the superior vestibular nerve, horizontal semicircular canal ampulla (A), and ampullar afferent (AA) of the superior vestibular nerve. (A) Pure saccular dysfunction; (B) pure saccular afferent deficit; (C) deficits involving both saccule and saccular afferents; (D) pure utricular dysfunction; (E) pure utricular afferent deficit; (F) deficits involving both utricle and utricular afferents; (G) pure horizontal ampullar dysfunction; (H) pure ampullar afferents deficit; (I) deficits involving both ampulla and ampullar afferents

4 | DISCUSSION

VN was mainly diagnosed by clinical presentations and abnormal caloric response in the past^{1,4,6,11}; however, the caloric test only assesses the horizontal ampullar VOR pathway and fails to evaluate lesions involving other vestibular reflex pathways. Recently, cVEMP and oVEMP have been widely adopted to evaluate the function of otolithic organs and their subsequent neural pathways in various inner

ear diseases. To differentiate between superior and inferior VN, cVEMP and oVEMP tests were adopted to provide vestibular-specific information in previous investigations.^{5,19-21} These studies demonstrated that oVEMP and caloric tests could be abnormal in the event of superior VN.^{19,20} In contrast, inferior VN may have an abnormal cVEMP response without the impairment of oVEMP and caloric tests.^{5,19,21} Our results showed that caloric tests, ACS-cVEMPs, BCV-oVEMPs, GVS-cVEMPs, and GVS-oVEMP all exhibited significantly

		GVS-cVEMP		
		Normal	Abnormal	Total
Abnormal ACS-cVEMP + normal GVS-cVEMP	No	15	21	36 (90%)
	Yes	4	0	4 (10%)
	Total	19 (47.5%)	21 (52.5%)	
	<i>p</i>	.001		

		GVS-oVEMP		
		Normal	Abnormal	Total
Abnormal BCV-oVEMP + normal GVS-oVEMP	No	24	15	39 (97.5%)
	Yes	1	0	1 (2.5%)
	Total	25 (62.5%)	15 (37.5%)	
	<i>p</i>	<.001		

		GVS-oVEMP		
		Normal	Abnormal	Total
GVS-cVEMP	Normal	8	11	19 (47.5%)
	Abnormal	17	4	21 (52.5%)
	Total	25 (62.5%)	15 (37.5%)	
	<i>p</i>	.345		

TABLE 2 Comparisons between different VEMP combinations in affected ears of VN patients

Note: McNemar's test calculated the *p* value.

Abbreviations: ACS, air-conducted sound; BCV, bone-conducted vibration; GVS, galvanic vestibular stimulation; cVEMP, cervical vestibular-evoked myogenic potential; oVEMP, ocular vestibular-evoked myogenic potential; VN, vestibular neuritis.

TABLE 3 Comparisons between different inner ear test combinations in affected ears of VN patients

		BCV-oVEMP (represent utricular superior VN)		
		Normal	Abnormal	Total
Caloric response (represent horizontal ampullary superior VN)	Normal	7	0	7 (17.5%)
	Abnormal	17	16	33 (82.5%)
	Total	24 (60%)	16 (40%)	
	<i>p</i>	.029		

		Normal ACS-cVEMP + abnormal BCV-oVEMP and/or caloric response (represent superior VN)		
		No	Yes	Total
Abnormal ACS-cVEMP + normal BCV-oVEMP + normal caloric response (represent inferior VN)	No	18	15	33 (82.5%)
	Yes	7	0	7 (17.5%)
	Total	25 (62.5%)	15 (37.5%)	
	<i>p</i>	.033		

		Abnormal BCV-oVEMP and/or caloric response (represent superior + total VN)		
		No	Yes	Total
Abnormal ACS-cVEMP + normal BCV-oVEMP + normal caloric response (represent inferior VN)	No	0	33	33 (82.5%)
	Yes	7	0	7 (17.5%)
	Total	7 (17.5%)	33 (82.5%)	
	<i>p</i>	<.001		

Note: Fisher's Exact test calculated the *p* value.

Abbreviations: ACS, air-conducted sound; BCV, bone-conducted vibration; GVS, galvanic vestibular stimulation; cVEMP, cervical vestibular-evoked myogenic potential; oVEMP, ocular vestibular-evoked myogenic potential; VN, vestibular neuritis.

higher abnormal rates in affected ears, which meant that all the inner ear tests might have diagnostic value for VN.

VN is generally considered a disorder of vestibular afferents and rarely involves the neuroepithelium of vestibular endorgans.^{3,11} The main finding of the histopathological study is degeneration of all or a portion of the vestibular nerves and Scarpa's ganglions. ACS- or BCV-VEMPs alone cannot distinguish between pure endorgan dysfunction and vestibular nerve deficit. In the past, we have added galvanic VEMP tests to investigate vestibular afferent involvement in idiopathic sudden sensorineural hearing loss²² and Meniere's disease.¹⁸ Therefore, we intended to clarify the VN's specific lesion site by adding GVS-VEMPs to the other VEMP tests in this study. Our results showed that pure saccular dysfunction in the SCR pathway and pure utricular dysfunction in the UOR pathway were significantly less common than lesions involving vestibular afferents in VN patients, consistent with the histopathological findings. There was no significant difference in the abnormal rate of GVS-cVEMPs and GVS-oVEMPs, indicating similar degrees of involvement of superior vestibular fibers from the UOR pathway and inferior vestibular fibers from the SCR pathway. It is important to note that the finding only reflected the condition of otolithic afferents while ignoring that of ampullar afferents, and future studies with more histopathological evidence are needed to support our speculation.

In the present study, the caloric test had a significantly higher abnormal rate than the BCV-oVEMP test (82.5% vs. 40%), and all patients with abnormal BCV-oVEMPs also had abnormal caloric responses. From these results, some inferences can be drawn. First, the horizontal ampullar VOR pathway of the caloric test and the UOR pathway of the oVEMP test may be independent. Two pathways have different vestibular endorgans and transmit signals via different nerve fibers of the same superior vestibular nerve. Second, damage to the superior VN might mainly occur in the caloric pathway rather than the oVEMP pathway. In other words, the horizontal ampullar VOR pathway might be more vulnerable than the adjacent UOR pathway.

We used abnormal caloric and/or BCV-oVEMP responses combined with normal ACS-cVEMPs to represent superior VN. Abnormal ACS-cVEMPs combined with normal caloric and BCV-oVEMP responses represented inferior VN. The results showed that inferior VN was significantly fewer than superior VN and the combination of superior VN plus total VN, consistent with the current view that most VN has superior vestibular nerve involvement.^{6,10}

Although we tried to utilize caloric and VEMP tests to delineate the diseased territories of the vestibular system in detail, some limitations still needed to be mentioned. The caloric, cVEMP, and oVEMP tests could not identify VN's rare subtypes that only involve the superior SCC and/or subsequent ampullar afferents or the posterior SCC and/or its ampullar afferents. Patients of these subtypes have normal caloric, cVEMP, and oVEMP responses. In addition, they may only have minor symptoms²³ and torsional nystagmus, which might be mistaken as upbeat or downbeat nystagmus. Hence, these patients could be missed or erroneously ascribed to central vertigo. The lack of video head impulse test (vHIT) was a drawback of the present study. To

accurately investigate the single SCC and subsequent neural pathway in each direction and to obtain a more precise definition of VN lesions,^{24,25} the vHIT may be more helpful rather than the caloric test.

5 | CONCLUSION

The combination of VEMP and caloric tests offered additional insight into understanding the impairment sites better. The results suggested that pure otolithic organ dysfunction was significantly less common than vestibular nerve involvement. The damage to the horizontal ampullar VOR pathway was more significant than that to the UOR pathway, and both pathways may be independent of each other. In addition, the percentage of isolated inferior VN was significantly lower than that of VN involving the superior vestibular nerve.

None of the authors has potential sources of conflict of interest, financial, non-financial interests, or otherwise to be declared.

CONFLICT OF INTEREST

None of the authors has potential sources of conflict of interest, financial, non-financial interests, or otherwise to be declared.

AUTHOR CONTRIBUTIONS

Chih-Ming Chang and Po-Wen Cheng designed and performed the experiment; Chih-Ming Chang, Wu-Chia Lo, Yi-Ho Young, Li-Jen Liao, Po-Hsuan Wu, Ping-Chia Cheng, and Po-Wen Cheng collected, analyzed data, and wrote the manuscript; Chih-Ming Chang and Po-Wen Cheng provided data interpretation and critical revision. Po-Wen Cheng supervised the manuscript. All authors read and approved the final manuscript.

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How to cite this article: Chang C-M, Lo W-C, Young Y-H, et al. Galvanic vestibular-evoked myogenic potentials in evaluating damaged sites of vestibular neuritis. *Laryngoscope Investigative Otolaryngology*. 2022;7(2):506-514. doi:10.1002/lio2.745