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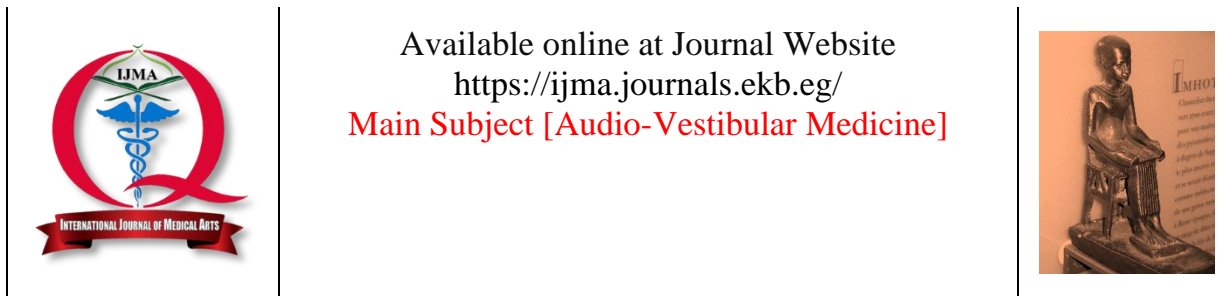
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Original Article

Cervical Vestibular Evoked Myogenic Potentials in Vestibular Neuritis

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ABSTRACT

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Background and Aim: Vestibular evoked myogenic potentials [VEMP] are electromyographic responses to acoustic stimuli to assess the otolith function and integrity of inferior vestibular nerve. It is an easy test to perform and non-invasive. This study was designed to study and compare AC and BC cVEMPs in patients with vestibular neuritis.

Patients and methods: This observational case control study was conducted on 40 subjects in the age range of 20-60 years selected from Audio vestibular clinic of Al Zahraa university hospital. Twenty patients diagnosed with Vestibular neuritis according to a standard clinical criterion, and the other twenty subjects were normal healthy subjects with no complaint of dizziness or history of vestibular disorders

Results: In this work, about 25% of study group had abnormal AC cVEMPs while 35.0% had abnormal BC cVEMPs.

Conclusion: Both AC and BC evoked cVEMPs should be considered as complementary test along with other conventional vestibular function tests in patients with vestibular neuritis.

Keywords: AC cVEMPs, BC cVEMP, Saccular function testing, Dizzy patients.



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INTRODUCTION

Vestibular neuritis [VN] is described as a degenerative neuropathy of the vestibular nerve trunks [1]. It is one of the most common reasons for vertigo [2]. VN is distinguished by sudden onset of rotatory vertigo, nausea, vomiting, & nystagmus [3]. It is usually severe for few days & progressively subsides within 2-3 weeks [4].

Reason for VN is not totally understood, but several hypotheses are elucidating its pathophysiology [5]. The most possible hypothesis is reactivation of dormant neurotropic virus [6]. Other mechanisms may be involved, are auto-immune or microvascular ischemia affecting the vestibule [7].

Superior division of vestibular nerve has been identified as being affected by VN, however after vestibular evoked myogenic potentials [VEMPs] testing has been introduced, it became evident that both vestibular nerve divisions could be impacted, either together or independently [8]. When both divisions are impacted, spinal ganglion is regarded to be impacted [9]. Depending on location of lesion, VEMPs response may be normal, abnormal or even absent totally [10].

Diagnosis of inferior vestibular neuritis is difficult since typical symptoms of vestibular neuritis are absent in this condition [11]. As a result, isolated inferior vestibular neuritis may be mistakenly attributed to central pathology unless inferior vestibular function is not thoroughly evaluated [12].

Cervical VEMPs [cVEMPs] is the only objective test for integrity of saccule & inferior vestibular nerve, through reaction of reflex muscle [sternocleidomastoid] in response to high intensity acoustic stimulation [9].

Different types of Stimuli have been used in cVEMPs testing. They include air & bone-conducted [AC & BC] tone bursts, clicks, forehead taps, & galvanic stimulation [13].

Using AC sound, **Chihara et al.** [14] and **Chou et al.** [15], noted absence of cVEMPs from affected ears, but these researches were small & presented inconsistent proof of inferior nerve insight to AC evoked reflexes [16]. By Using BC stimulation, **Brantberg et al.** [17], proved that forehead & mastoid taps generated more cVEMPs abnormalities than AC stimulation.

This is consistent with BC evoked cVEMPs being mediated at least in part by superior vestibular nerve afferents. On the other hand, **Curthoys** [18] declared that in VN, BC stimulation generated normal cVEMPs reaction. This was explained by regardless of stimulus modality, otolith-collic projections originate primarily from saccule [19]. This discrepancy of findings of preceding researches, led us to study the effect of type of stimulus on cVEMPs test results.

THE AIM OF THE WORK

This study aimed to study and compare AC and BC cVEMPs in patients with vestibular neuritis.

PATIENTS AND METHODS

This observational case control research included 40 subjects. Their age ranged from twenty to sixty years old, recruited from Audio vestibular clinic of Al Zahraa university hospital. Written consents were obtained from all participants. They were categorized to 2 groups:

[1] Control group: comprised of twenty normal healthy subjects with no complaint of dizziness or history of vestibular disorders.

[2] Study group: comprised of 20 patients identified with vestibular neuritis [they did not receive any vestibulo-suppressant medications for at least 48 hours before examination] according to clinical criteria of **Taylor et al.** [7]. They gave history of vertigo [at least one attack of rotational vertigo that increases significantly with head movement], nausea/vomiting and imbalance. On vestibular assessment, patients showed spontaneous horizontal rotational nystagmus toward lesion side, deviation in the opposite direction to nystagmus and unilateral caloric weakness.

All participants in this study had no hearing complaints, normal hearing sensitivity in the frequency ranges of two hundred fifty – eight thousand Hz as shown in pure tone audiometry. Middle ear functions were normal as evidenced by tympanometry and acoustic reflexes threshold. Also, they had no history of chronic diseases. cVEMPs testing for all participants was carried out using Interacoustics Eclipse [EP25, Inc., Middlefart, Denmark].

- Electrode montage: skin was cleaned to verify that impedance was less than five k Ω . Positive electrode was located on upper 3rd of stimulated side's SCM, negative and ground electrodes were placed on sternum and forehead respectively.
- Instructions to patients: they were instructed to turn their head to opposite side of stimulation with slight head flexion to enhance muscle contraction.
- AC and BC 500 Hz tone burst stimuli with intensity of 95 dBnHL & 70 dBnHL respectively, presented at a rate of 5/ second, with a total sweep of 200 and analysis time of 50 milliseconds were used [11].
- Wave analysis: during the study, equipment system was observing EMG levels. To compensate for possibility of uneven SCM compression on both sides, EMG scaling was done by the device to allow for an accurate contrast among right & left sides. Latencies of cVEMPs waves were recognized, & rectified amplitudes of each wave were measured. To ensure reproducibility, at least 2 consecutive averages were obtained from each side for both AC & BC cVEMPs. Amplitude & latencies were averaged over at least 2 moves. Next formula was used to calculate asymmetric Ratio: one hundred $[(AR-AL)/(AR+AL)]$ Formalized paraphrase [AR denotes amplitude of P13-N23 on right side, while AL denotes amplitude of P13-N23 on left side] [13].

Statistical Analysis: Data were collected, modified, coded, & joined into IBM SPSS version twenty-three [Statistical Package for Social Science]. For qualitative data, frequency & percent were measured, while mean, standard

deviations, & ranges were determined for quantitative data. Significant P-value was <0.05.

RESULTS

In this work, there was no statistically significant difference between Rt and Lt ears as regard AC and BC cVEMPs in both groups [Tables 1 & 2].

Table [3] shows no significant difference between AC & BC cVEMPs measures in control group.

Table [4] shows that the differences between AC & BC cVEMPs in study group are not statistically significant.

As the differences between Rt & Lt ears in both groups were not statistically significant, we used 40 ears for statistical analysis in [tables 5, 6].

Table [5] shows no significant differences between both groups as regard AC cVEMPs measures.

Table [6] shows no significant difference between both groups as regard BC cVEMPs.

Table [7] shows that the differences between affected ears & non- affected ears of VN group as regard AC cVEMPs P13& N23 latencies are significant.

Table [8] shows that there is statistically significant difference between intact ears & affected ears as regard BC cVEMPs P13& N23 latencies.

Table [9] shows that there are more abnormalities in BC cVEMPs than AC cVEMPs.

Table [1]: Comparison between right and left ears of control group as regard AC and BC cVEMPs (P13& N23) latency and (P13 N23) amplitude

		Control group		Test value	P-value	Sig.
		Right	Left			
AC cVEMPs						
P13 latency	Mean \pm SD	15.51 \pm 1.73	15.63 \pm 1.35	-0.234•	0.816	NS
N23 latency	Mean \pm SD	25.58 \pm 1.71	25.66 \pm 1.70	-0.157•	0.876	NS
P13 N23 Amplitude	Mean \pm SD	43.32 \pm 12.53	44.33 \pm 10.76	-0.274•	0.786	NS
BC cVEMPs						
P13 latency	Mean \pm SD	15.11 \pm 1.81	15.25 \pm 1.92	-0.238•	0.813	NS
N23 latency	Mean \pm SD	25.40 \pm 2.28	25.53 \pm 2.32	-0.177•	0.861	NS
P13N23 Amplitude	Mean \pm SD	44.25 \pm 10.67	43.39 \pm 8.80	0.279•	0.782	NS

Table [2]: Comparing of right & left ears of study group as regard AC and BC cVEMPs [P13& N23] latency and [P13 N23] amplitude

		Study group		Test value	P-value	Sig.
		Right	Left			
AC cVEMPs						
P13 latency	Mean ± SD	15.88 ± 1.33	16.07 ± 1.7	-0.406•	0.687	NS
N23 latency	Mean ± SD	25.79 ± 1.35	25.99 ± 1.38	-0.466•	0.644	NS
P13 N23 Amplitude	Mean ± SD	41.61 ± 11.51	43.81 ± 12.11	-0.589•	0.560	NS
BC cVEMPs						
P13 latency	Mean ± SD	15.09 ± 1.93	15.95 ± 2.02	-1.376•	0.177	NS
N23 latency	Mean ± SD	25.35 ± 1.64	25.49 ± 2.11	-0.235•	0.815	NS
P13N23 Amplitude	Mean ± SD	41.37 ± 11.18	43.2 ± 13.44	-0.468•	0.643	NS

Table [3]: Comparison between AC and BC cVEMPs as regard P13& N23 latency, [P13 N23] amplitude and asymmetric ratios in control group

		Control group		Test value	P-value	Sig.
		AC cVEMPs	BC cVEMPs			
P13 latency	Mean ± SD	15.57 ± 1.53	15.18 ± 1.85	1.549•	0.129	NS
N23 latency	Mean ± SD	25.62 ± 1.68	25.46 ± 2.27	0.530•	0.599	NS
P13 N23 Amplitude	Mean ± SD	43.83 ± 11.54	43.82 ± 9.66	0.006•	0.996	NS
Asymmetry	Median [IQR]	18.5 [13.5 – 40.5]	21 [14 – 38]	-0.428≠	0.669	NS

Table [4]: Comparison between AC and BC cVEMPs as regard [P13& N23] latency, P13 N23 amplitude and asymmetric ratios in study group

		Study group		Test value	P-value	Sig.
		AC cVEMPs	BC cVEMPs			
P13 latency	Mean ± SD	15.97 ± 1.51	15.52 ± 2	1.314•	0.197	NS
N23 latency	Mean ± SD	25.89 ± 1.35	25.42 ± 1.87	1.522•	0.136	NS
P13 N23 Amplitude	Mean ± SD	42.71 ± 11.71	42.29 ± 12.24	0.413•	0.682	NS
Asymmetry	Median [IQR]	26 [16 – 40]	24.5 [16 – 38]	-1.556•	0.120	NS

Table [5]: Comparison between VN group & control subjects as regard AC cVEMPs P13& N23 latency, [P13 N23] Amplitude and asymmetric ratios

AC cVEMPs		Study group	Control group	Test value	P-value	Sig.
		No. = 20	No. = 20			
P13 latency	Mean ± SD	15.97 ± 1.51	15.57 ± 1.53	1.191•	0.237	NS
N23 latency	Mean ± SD	25.89 ± 1.35	25.62 ± 1.68	0.799•	0.427	NS
P13 N23 Amplitude	Mean ± SD	42.71 ± 11.71	43.83 ± 11.54	-0.430•	0.669	NS
Asymmetry	Median [IQR]	26 [16 – 40]	18.5 [13.5 – 40.5]	-0.502≠	0.616	NS

Table [6]: Comparison between VN group & control subjects as regard BC cVEMPs [P13& N23] latency, P13 N23 amplitude and asymmetric ratio

BC cVEMPs		Study group	Control group	Test value	P-value	Sig.
		No. = 40	No. = 40			
P13 latency	Mean ± SD	15.52 ± 2	15.18 ± 1.85	0.783•	0.436	NS
N23 latency	Mean ± SD	25.42 ± 1.87	25.46 ± 2.27	-0.091•	0.927	NS
P13N23 Amplitude	Mean ± SD	42.29 ± 12.24	43.82 ± 9.66	-0.622•	0.536	NS
Asymmetry	Median [IQR]	24.5 [16 – 38]	21 [14 – 38]	-0.176≠	0.860	NS

Table [7]: Comparison between Affected ears [diseased side] and non- affected ears [intact sides] as regard AC cVEMPs [P13& N23] latency and [P13N23] amplitude in study group

		AC cVEMPs		Test value	P-value	Sig.
		Not affected [Intact ears]	Affected			
		No. = 20	No. = 20			
P13 latency	Mean ± SD	15.49 ± 0.84	16.46 ± 1.86	-2.115•	0.041	S
N23 latency	Mean ± SD	25.4 ± 1.07	26.38 ± 1.45	-2.436•	0.020	S
P13 N23 Amplitude	Mean ± SD	45.33 ± 10.31	40.09 ± 12.68	1.435•	0.160	NS

Table [8]: Comparison between Affected ears [diseased sides] and non- affected ears [intact sides] as regard BC cVEMPs P13& N23 latency and [P13N23] amplitude in study group

		BC cVEMPs		Test value	P-value	Sig.
		Not affected [intact]	Affected			
		No. = 20	No. = 20			
P13 latency	Mean ± SD	14.77 ± 1.61	16.26 ± 2.1	-2.519•	0.016	S
N23 latency	Mean ± SD	24.82 ± 1.68	26.03 ± 1.89	-2.141•	0.039	S
P13N23 Amplitude	Mean ± SD	43.92 ± 11.4	40.65 ± 13.11	0.842•	0.405	NS

Table [9]: Distribution of normal and abnormal response of AC and BC cVEMPs in study group

Study group [no. = 20]		
AC cVEMPs results	Normal	15 [75.0%]
	Abnormal	5 [25.0%]
BC cVEMPs results	Normal	13 [65.0%]
	Abnormal	7 [35.0%]

DISCUSSION

Statistical analysis was done on the results and the study showed that there is no significant difference of AC and BC cVEMPs measures between right and left ears of both groups [tables 1&2]. Also, we reported no significant difference between AC & BC cVEMPs in both groups [tables 3&4]. As a result, we used 40 ears for statistical analysis in tables 5&6 and declared that the differences between study and control groups as regard latency of P13& N23, P13N23 amplitude and asymmetric ratios were not of significant difference [table 5&6]. These outcomes were in agreement with outcomes of **Curthoys et al.** [20], **Govender et al.** [16], and **Oh et al.** [21], who reported that most VN patients shows normal cVEMPs.

This can be explained by VN mostly affects fibers in superior division of vestibular nerve more severely than fibers in inferior division [22]. This could be due to the anatomical variations between the two divisions. When compared to inferior division, superior vestibular nerve has more length, reduced diameter, & increased bony trabeculae of its housing bony canal [23]. In

agree with this postulation is the result of **Sirige et al.** [24] who get normal cVEMPs in 3 studied cases previously diagnosed with vestibular neuritis. Furthermore, these findings are consistent with findings of **Welgampola et al.** [25], who declared that both AC & BC cVEMPs are more likely to exert their impacts via similar pathways, resulting in similar patterns of response in VN.

On comparing affected [diseased] and non-affected[intact] ears in study group as regard AC cVEMPs and BC cVEMPs, important variation was showed with delayed P13 & N23 latencies and there was no significant difference as regard P13N23 complex amplitude [table 7&8]. This outcome is in agreement with **Govender et al.** [20], who detected delayed latencies of affected ears when compared to the latencies of the nonaffected ears. **Sirige et al.** [24] provided objective proof that inferior vestibular neuritis exists a subtype of vestibular neuritis. His work showed normal caloric testing outcomes and abnormal cVEMPs outcomes and thus constructing firm diagnosis of inferior vestibular neuritis.

In this work about 25% of study group had abnormal AC cVEMPs while 35% had abnormal BC cVEMPs [table 9]. These outcomes were in agreement with **Brantberg et al.** [17], who demonstrated that BC evoked cVEMPs showed more abnormalities than AC stimulation. This may be explained by the BC cVEMPs may be mediated by afferents within the superior vestibular nerve [utricle effect] [26].

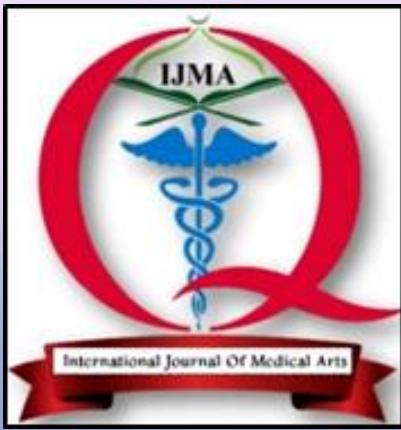
While AC evoked cVEMPs is sensitive to many pathologies [25], BC can be used when there is no response to AC stimuli and in patients suffering from conductive hearing loss [27].

Conflict of interest and financial disclosure: None

REFERENCES

1. Lee JY, Park JS, Kim MB. Clinical Characteristics of Acute Vestibular Neuritis According to Involvement Site. *Otol Neurotol.* 2020 Jan;41[1]:143. doi: 10.1097/MAO.0000000000002517.
2. Zaper D, Adamec I, Gabelić T, Krbot M, Isgum V, Hajnsek S, Habek M. Vestibular neuronitis: patofiziologija, dijagnoza i liječenje [Vestibular neuronitis: pathophysiology, diagnosis and treatment]. *Lijec Vjesn.* 2012 Nov-Dec;134[11-12]:340-5. Croatian. PMID: 23401980.
3. Kim YH, Kim KS, Kim KJ, Choi H, Choi JS, Hwang IK. Recurrence of vertigo in patients with vestibular neuritis. *Acta Otolaryngol.* 2011 Nov;131[11]:1172-7. doi: 10.3109/00016489.2011.593551.
4. Lee H, Kim JS, Chung EJ, Yi HA, Chung IS, Lee SR, Shin JY. Infarction in the territory of anterior inferior cerebellar artery: spectrum of audiovestibular loss. *Stroke.* 2009 Dec;40[12]:3745-51. doi: 10.1161/STROKEAHA.109.564682.
5. Roehm PC, Camarena V, Nayak S, Gardner JB, Wilson A, Mohr I, Chao MV. Cultured vestibular ganglion neurons demonstrate latent HSV1 reactivation. *Laryngoscope.* 2011 Oct;121[10]:2268-75. doi: 10.1002/lary.22035.
6. Jeong SH, Kim HJ, Kim JS. Vestibular neuritis. *Semin Neurol.* 2013 Jul;33[3]:185-94. doi: 10.1055/s-0033-1354598.
7. Taylor RL, McGarvie LA, Reid N, Young AS, Halmagyi GM, Welgampola MS. Vestibular neuritis affects both superior and inferior vestibular nerves. *Neurology.* 2016 Oct 18;87[16]:1704-1712. doi: 10.1212/WNL.0000000000003223.
8. Curthoys IS, Grant JW, Burgess AM, Pastras CJ, Brown DJ, Manzari L. Otolithic Receptor Mechanisms for Vestibular-Evoked Myogenic Potentials: A Review. *Front Neurol.* 2018 May 25;9:366. doi: 10.3389/fneur.2018.00366.
9. Madzharova K, Beshkova A. Application of the VEMP test for diagnosing patients with vestibular neuritis. *Int Bull Otorhinolaryngol.* 2020 Jun 30;16[1]:16-20. doi: 10.14748/orl.v15i1.6685.
10. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry.* 1994 Feb;57[2]:190-7. doi: 10.1136/jnnp.57.2.190.
11. Kim JS, Kim HJ. Inferior vestibular neuritis. *J Neurol.* 2012 Aug;259[8]:1553-60. doi: 10.1007/s00415-011-6375-4.
12. Halmagyi GM, Aw ST, Karlberg M, Curthoys IS, Todd MJ. Inferior vestibular neuritis. *Ann N Y Acad Sci.* 2002 Apr;956:306-13. doi: 10.1111/j.1749-6632.2002.tb02829.x.
13. Murofushi T. Clinical application of vestibular evoked myogenic potential [VEMP]. *Auris Nasus Larynx.* 2016 Aug 1;43[4]:367-76. doi: 10.1007/978-4-431-85908-6.
14. Chihara Y, Iwasaki S, Ushio M, Murofushi T. Vestibular-evoked extraocular potentials by air-conducted sound: another clinical test for vestibular function. *Clin Neurophysiol.* 2007 Dec;118[12]:2745-51. doi: 10.1016/j.clinph.2007.08.005.
15. Chou CH, Wang SJ, Young YH. Feasibility of the simultaneous ocular and cervical vestibular-evoked myogenic potentials in unilateral vestibular hypofunction. *Clin*

- Neurophysiol. 2009 Sep;120[9]:1699-705. doi: 10.1016/j.clinph.2009.07.036.
16. Govender S, Rosengren SM, Colebatch JG. Vestibular neuritis has selective effects on air- and bone-conducted cervical and ocular vestibular evoked myogenic potentials. *Clin Neurophysiol.* 2011 Jun;122(6):1246-55. doi: 10.1016/j.clinph.2010.12.040.
 17. Brantberg K, Tribukait A, Fransson PA. Vestibular evoked myogenic potentials in response to skull taps for patients with vestibular neuritis. *J Vestib Res.* 2003;13(2-3):121-30. PMID: 14757915.
 18. Curthoys IS. A balanced view of the evidence leads to sound conclusions. A reply to J.G. Colebatch "Sound conclusions?". *Clin Neurophysiol.* 2010 Jun;121(6):977-8. doi: 10.1016/j.clinph.2010.01.025.
 19. Curthoys IS. A critical review of the neurophysiological evidence underlying clinical vestibular testing using sound, vibration and galvanic stimuli. *Clin Neurophysiol.* 2010 Feb;121(2):132-44. doi: 10.1016/j.clinph.2009.09.027.
 20. Curthoys IS, Iwasaki S, Chihara Y, Ushio M, McGarvie LA, Burgess AM. The ocular vestibular-evoked myogenic potential to air-conducted sound; probable superior vestibular nerve origin. *Clin Neurophysiol.* 2011 Mar;122(3):611-616. doi: 10.1016/j.clinph.2010.07.018.
 21. Oh SY, Kim JS, Yang TH, Shin BS, Jeong SK. Cervical and ocular vestibular-evoked myogenic potentials in vestibular neuritis: comparison between air- and bone-conducted stimulation. *J Neurol.* 2013 Aug; 260[8]:2102-9. doi: 10.1007/s00415-013-6953-8.
 22. Fetter M, Dichgans J. Vestibular neuritis spares the inferior division of the vestibular nerve. *Brain.* 1996 Jun;119 [Pt 3]:755-63. doi: 10.1093/brain/119.3.755.
 23. Gianoli G, Goebel J, Mowry S, Poomipannit P. Anatomic differences in the lateral vestibular nerve channels and their implications in vestibular neuritis. *Otol Neurotol.* 2005 May;26[3]:489-94. doi: 10.1097/01.mao.0000169787.99835.9f.
 24. Sirige S, Kumar SR, Chaitanya VK, Reddy VK. Usefulness of vestibular evoked myogenic potentials in diagnosis of peripheral vestibular disorders. *Int J Otorhinolaryngol Head Neck Surg.* 2021 Nov;7[11]:1724-1728.
 25. Welgampola MS, Myrie OA, Minor LB, Carey JP. Vestibular-evoked myogenic potential thresholds normalize on plugging superior canal dehiscence. *Neurology.* 2008 Feb 5;70[6]:464-72. doi: 10.1212/01.wnl.0000299084.76250.4a.
 26. Rosengren SM, Todd NP, Colebatch JG. Vestibular evoked myogenic potentials evoked by brief interaural head acceleration: properties and possible origin. *J Appl Physiol* [1985]. 2009 Sep;107[3]:841-52. doi: 10.1152/jappphysiol.00296.2009.
 27. Govender S, Dennis DL, Colebatch JG. Vestibular evoked myogenic potentials [VEMPs] evoked by air- and bone-conducted stimuli in vestibular neuritis. *Clin Neurophysiol.* 2015 Oct;126[10]:2004-13. doi: 10.1016/j.clinph.2014.12.029.



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