Int. Adv. Otol. 2013; 9:(1) 96-100

ORIGINAL ARTICLE

Vestibular Evoked Myogenic Potentials in Splenius Capitis Muscle

Feray Gulec, Nese Celebisoy, Timur Kose

Department of Neurology, Tepecik Training and Research Hospital, Izmir, Turkey. (FG)
Department of Neurolog, Ege University Medical School Hospital, Izmir, Turkey. (NC)
Department of Bioistatistics and Medical Informatic, Ege University Medical School Hospital, Izmir, Turkey (TK)

Objectives: Vestibular evoked myogenic potentials (VEMPs) are biphasic responses recorded from tense anterior neck muscles [sternocleidomastoids (SCM)] with loud sound stimulus. The aim of this study was to record VEMPs from the splenius capitis muscles (SPC).

Method: Seventy-four healthy controls were studied. Recordings were performed from the ipsilateral SCM, contralateral and ipsilateral SPC muscles when the subjects were seated and turned their head contralaterally to the ear being tested. The recording sites were named as I, II and III respectively.

Results: The responses were recorded in 100 % at site I, in 98.6 % at site II, in 97.9 % at site III. The earliest responses were detected at sites II and I, followed by site III. The potential amplitude was determined by the muscle tension. The responses recorded both from flexor and extensor muscles had positive/negative polarity.

Conclusions: Extensor neck muscles can also be used for VEMP studies and the responses can be recorded even from mildly contracted muscles which make it easier to study in patients with poor cooperation.

Significance: Positive/negative polarity of the muscles recorded from all sites may indicate that sacculo-collic connections are mainly inhibitory in nature.

Submitted : 28 September 2012 *Revised : 12 December 2012* *Accepted : 02 January 2013*

Introduction

Cervical vestibular evoked myogenic potential (VEMP) is a short-latency myogenic response recorded from the sternocleidomastoid muscle (SCM) in response to saccular stimulation. The currently understood VEMP was first described by Colebatch and Halmagyi and Colebatch et al. [1, 2]. Since then, VEMPs have become a standard clinical test of otolith function [3, 4, 5, 6, 7, 8]. At present, they are of clinical importance for estimating the severity of peripheral vestibular damage due to different pathological processes such as Ménière's disease, vestibular neuritis, and vestibular schwannoma [9, 10, 11, 12, 13]. In addition, VEMP testing constitutes an electrophysiological method that can be used to detect subclinical lesions in central vestibular pathways [14, 15, 16].

Briefly, the VEMP is a biphasic response elicited by loud clicks or tone bursts recorded from the tonically contracted SCM muscle, being the only resource available to assess the function of the saccule and the lower portion of the vestibular nerve [5, 6, 7, 8]. However, it is difficult to record VEMPs in children, elderly patients and in patients with poor cooperation as maximal contraction of the SCM is needed for a stable response. The aim of this study was to record VEMPs from the splenius capitis muscles (SPC) to see if weaker muscular contraction will be sufficient to get the responses.

Methods

The study was conducted in the specialized Neuro-otology Clinic in Ege University Department of

Corresponding address:
Feray Gulec
Department of Neurology, Tepecik Training and Research Hospital, İzmir, Turkey.
Tel: 90 232 469 69 69
Fax: 90 232 433 07 56
email:gulecferay@gmail.com

Neurology. The study protocol was approved by Ege University local ethics committee. Informed consent was obtained from all the participants. Seventy-four healthy controls [41 females, 33 males; age ranging from 19 to 54 years (mean±SD: 32,8±8.8 years)] were studied. All individuals underwent audiometric testing, showing no hearing loss.

VEMPs were recorded by using a Synergy device (Medelec; Oxford Instruments Medical Inc, UK). To record the surface EMG activity, a reference electrode was placed on the upper third of sternum and the ground electrode around the wrist. Recordings were performed from three different sites; the ipsilateral SCM was addressed as site I, contrateral SPC as site II and ipsilateral SPC as site III. For site I, the active electrode was on the upper half of the SCM ipsilateral to the stimulation. For sites II and III the active electrode positions were on the contralateral and ipsilateral SPC respectively (the midpoint between the mastoid process and the spinous process of C7). Patients were seated on an armchair and were asked to turn their head contralaterally to the ear being tested all through the recordings from the three sites. Two stimulation consequences consisting of 250 sound stimuli were given. The acoustic stimuli were clicks at an intensity of 110 dBnHL (normal hearing level) of 0.1 millisecond duration, delivered at a frequency of 5 Hz through a headphone unilaterally to each ear. The EMG signal was bandpass filtered from 10 to 1000 Hz and averaged during a 100-millisecond interval. The initial positive/negative polarity of the waveform with peaks was termed p13 and n23 on the basis of respective latencies. The latencies of peaks p13 and n23 and peak-to-peak amplitude of p13–n23 were measured and taken into consideration.

SPSS 20 for Windows was used for the statistical analysis. Hypothesis tests were performed at the α: 0.05 significance level (means $p<0.05$ were accepted as significant). The Shapiro-Wilk test was performed to check if the data were normally distributed. As none of the parameters were normally distributed, nonparametric methods were used. Friedman test was used to determine if the parameters tested were significantly different in three recording sites. For the parameters showing a statistically significant difference comparison between two sites was performed by using Wilcoxon signed ranks test. For all three sites studied comparison of the right and left sided p13, n23 latencies and amplitudes was also done by the Wilcoxon signed ranks test.

Table 1. Median p13, n23 latencies and p13/n23 amplitudes of the responses recorded from three different sites.

	Site I		Site II		Site III	
	R	L	R	L	R	L
p13 latency (ms)						
med:	13,3	13,3	13,1	13,1	13,2	13,3
min:	11,2	11,2	11,2	11,2	11,2	11,4
max:	18,3	16,6	20,1	19,5	20,1	19,5
n23 latency (ms)						
med:	22,3	21,8	21,7	21,1	22,5	21,7
min:	19,2	19,8	19,2	19,3	19,2	19,8
max:	25,7	24,5	24,7	24,2	25,7	24,5
p13/n23 amplitude (μv)						
med:	115,7	125,3	105,9	129,6	102,8	112,4
min:	71,6	80	71,6	55,2	71,6	55,2
max:	563,2	506,7	294	381	294	381

Site I: Ipsilateral sternocleidomastoid muscle
Site II: Contralateral splenius capitis muscle
Site III: Ipsilateral splenius capitis muscle
R: Right
L: Left

Results

In healthy individuals VEMPs were recorded 100 % at site I, 98.6 % at site II and 97.9% at site III. Median p13, n23 latencies and p13/n23 amplitudes of the responses recorded from three different sites is given in Table 1. The responses recorded from two healthy controls from the three sites are given in Figure 1a and b.

Comparison of the p13 latencies between sites revealed that a statistically significant difference was present for both the right and left sides (p: 0.001). When the two sites were compared with each other it was found that p latencies recorded at sites I and III were not statistically different both for the right (p: 0.17) and left (p: 0.07) sides. However, both right sided (p: 0.014) and left sided (p: 0.007) p latencies from sites I and II were significantly different from each other. The same was true for the comparison of the right (p: 0.002) and left sided (p: 0.002) p latencies recorded both from sites II and III, p latency recorded from site II being slightly but significantly shorter than I and III.

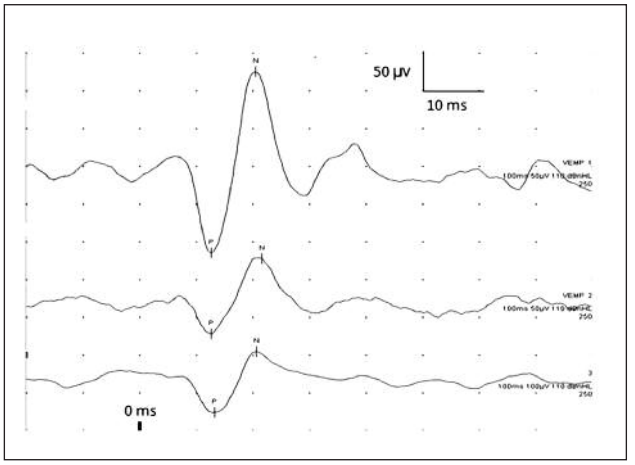
Comparison of the n23 latencies between sites also revealed that a statistically significant difference was present for both the right and left sides (p< 0.001). When the two sites were compared with each other it was found that n latencies recorded at sites I and III were not statistically different both for the right (p: 0.39) and left sides (p: 1). However, both right sided (p: 0.001) and left sided (p: 0.002) n latencies from sites I and II were significantly different from each other. The same was true for the comparison of the right (p: 0.001) and left sided (p: 0.003) n latencies recorded from sites II and III, n latency recorded from site II being slightly but significantly shorter than I and III.

Comparison of the p13/n23 amplitudes between sites revealed that a statistically significant difference was present (p< 0.001). When the two sites were compared with each other it was found that right sided (p: 0.002) and left sided (p<0.001) p13/n23 amplitudes recorded at sites I and II were significantly different from each other. The same was true for the comparison of the right (p<0.001) and left sided (p:<0.001) amplitudes recorded at sites I and III and II and III (p: 0.003 for the right, p: 0.009 for the left side). Amplitudes recorded from site I were the larger and site III were the smaller.

When the p13, n23 latencies and amplitudes recorded from the right side were compared with the latencies and amplitudes recorded from the left side at three different sites no statistically significant difference could be found (p>0.05).

Discussion

The cervical VEMP is thus a manifestation of the vestibulo-collic reflex. It is a short-latency reflex evoked by intense auditory clicks [5, 6, 7, 8]. The conventional method for recording the VEMP involves measuring EMG activity by surface electrodes placed over the tonically-activated SCM muscles. This muscle is innervated by the ipsilateral accessory nerve. It originates from the manubrium of the sternum and medial clavicle and inserts into the mastoid process and occipital prominence behind the ear. Unilateral activation flexes the head and rotates it towards the opposite side. Bilateral activation flexes the head and neck. SPC, the other muscle used in this study, originates from the lower nuchal ligament and spinous processes and supraspinous ligaments from thoracal 1 to 3.The



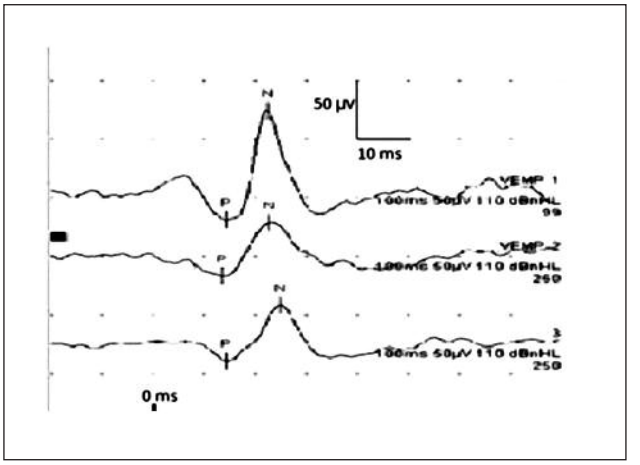


Table 1. Median p13, n23 latencies and p13/n23 amplitudes of the responses recorded from three different sites.

fibers of the muscle are directed upward and laterally and are inserted, under cover of the SCM, into the mastoid process and into the lateral third of the superior nuchal line. Bilaterally the muscles extend the head and neck, unilaterally it flexes the head and neck laterally and rotates the head to the same side. It is innervated by the dorsal rami of spinal nerves C3-C6. The ipsilateral SCM and contralateral SPC muscles are agonists with respect to rotation of the head.

There are only two previous studies about VEMPs from posterior neck muscles [17, 18]. Wu et al. recorded VEMPs from the activated ipsilateral SPC showing a negative/positive polarity of wave I/II and in only half of the patients a response could be gathered from the contralateral SPC. Referring to the study of Di Lazzaro et al. about the initial positive peak reflecting an inhibition of underlying motor unit activity in tonically activated SCM, the authors concluded that ipsilateral VEMPs from SCM were inhibitory whereas ipsilateral VEMPs from SPC were excitatory responses [19]. The latencies of the ipsilateral SPC responses were shorter and the amplitudes were smaller [17]. Sakakura et al. also made recordings from ipsi and contralateral SPC [18]. The subjects were not asked to contract the neck muscles. A negative/positive potential with the peak latency of 13.3ms has been found and the result was accepted to be compatible with the findings of Uchino et al. in cats, who have reported that the sacculocollic connections to the ipsilateral neck flexors were inhibitory whereas the connections to the ipsilateral neck extensors were excitatory [20]. VEMPs were recorded in 74.2% of the healthy young subjects from the neck extensors. The figure was 92% for the SCM. However, VEMPs on the neck extensors were reported to require little muscular tension [18].

In our study VEMPs were recorded in 100 % of the subjects from the ipsilateral SCM, in 98.6 % from the contralateral SPC and in 97.9% from the ipsilateral SPC. The figures for the posterior neck muscles were much higher than the results of the two previous studies reporting rates around 50% and 74% [17,18]. During the recordings from all three sites the patients were asked to turn their head contralaterally to the ear being tested while seated so the ipsilateral SCM and contralateral SPC were active whereas the activity of the ipsilateral SPC was minor. When the p13 and n23 latencies of the responses recorded from the ipsilateral SCM and ipsilateral SPC were compared a statistically significant difference could not be found. However, the latency of the responses recorded from the contralateral SPC was slightly but significantly shorter than the responses recorded from the abovementioned two sites. When the amplitudes were taken into consideration a statistically significant difference was noted for all three sites studied; amplitudes recorded from the maximally active ipsilateral SCM being the largest and amplitudes recorded from the ipsilateral SPC being the smallest. We couldn't find a reversal of the polarity of the response during the recordings from the extensor muscles as reported previously [17,18]. According to our findings sacculocollic connections to both flexor and extensor neck muscles seem to be inhibitory in nature. A slightly shorter latency of the ipsilateral SPC potential was concordant with the finding of Wu et al. which may be due to shorter length of neural pathways or fewer number of synapses [17]. The amplitudes recorded from the same muscle were the smallest as the muscle was not active during the recordings. Responses from the ipsilateral SCM, the most active muscle was the biggest followed by the responses from the contraletaral SPC as expected.

This study has shown that VEMPs can also be recorded from the SPC muscles using surface electrodes even when the muscles are not active in around 98% of the young healthy subjects. Recordings from the posterior neck muscles don't seem to necessitate strong muscle tension which makes the method easier to use in children and in patients with poor cooperation. However, the nature of the sacculocollic connections being either inhibitory or excitatory is still a matter of debate. Further studies on different muscles seem to be necessary to clarify the issue.

References

1. Colebatch JG, Halmagyi GM. Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. Neurology 1992; 42: 1635-1636.

2. Colebatch JG, Halmagyi GM, Skuse NF. Myogenic potentials generated by a click-evoked vestibulocollic reflex. J Neurol Neurosurg Psychiatry 1994; 57: 190-197.

3. Honaker JA, Samy RN. Vestibular evoked myogenic potentials. Curr Opin Otolaryngol Head Neck Surg 2007; 15: 330-334.

4. Eleftheriadou A, Koudounarakis E. Vestibular-evoked myogenic potentials eliciting: an overview. Eur Arch Otorhinolaryngol 2011; 268: 331-339.

5. Welgampola MS, Colebatch JG. Characteristics and clinical applications of vestibular evoked myogenic potentials. Neurology 2005; 64: 1682-1688.

6. Rauch SD. Vestibular evoked myogenic potentials. Curr Opin Otolaryngol Head Neck Surg 2006; 14: 299-304.

7. Welgampola MS. Evoked potential testing in neuro-otology. Curr Opin Neurol 2008; 21: 29-35.

8. Rosengren SM, Welgampola MS, Colebatch JG. Vestibular evoked myogenic potentials: past, present and future. Clin Neurophysiol 2010; 121: 636-651.

9. Murofushi T, Halmagyi GM, Yavor RA, Colebatch JG. Absent vestibular evoked myogenic potentials in vestibular neurolabyrinthitis: an indicator of inferior vestibular nerve involvement? Arch Otolaryngol Head Neck Surg 1996; 122: 845-848.

10. Murofushi T, Matsuzaki M, Mizuno M. Vestibular evoked myogenic potentials in patients with acoustic neuromas. Arch Otolaryngol Head Neck Surg 1998; 124:509–512.

11. Chen CW, Young YH, Wu CH. Vestibular neuritis: three-dimensional videonystagmography and vestibular evoked myogenic potential results. Acta Otolaryngol 2000; 120:845-848.

12. Young YH, Huang TW, Cheng PW. Assessing the stage of Meniere's disease using vestibular evoked myogenic potentials. Arch Otolaryngol Head Neck Surg 2003; 129:815-818.

13. Akkuzu G, Akkuzu B, Ozluoglu LN. Vestibular evoked myogenic potentials in benign paroxysmal positional vertigo and Meniere's disease. Eur Arch Otorhinolaryngol 2006; 263:510-517.

14. Shimizu K, Murofushi T, Sakurai M, Halmagyi GM. Vestibular evoked myogenic potentials in multiple sclerosis. J Neurol Neurosurg Psychiatry 2000; 69:276-277.

15. Itoh A, Kim YS, Yoshioka K, Kanaya M, Enomoto H, Hiraiwa F, et al. Clinical study of vestibular-evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. Acta Otolaryngol 2001; 545: 116-119.

16. Bandini F, Beronio A, Ghiglione E, Solaro C, Parodi RC, Mazzella L. The diagnostic value of vestibular evoked myogenic potentials in multiple sclerosis: a comparative study with MRI and visually evoked potentials. J Neurol 2004; 251: 617-621.

17. Wu CH, Young YH, Murofushi T. Tone burst evoked myogenic potentials in human neck flexor and extensor. Acta Otolaryngol 1999; 119: 741-744.

18. Sakakura K, Takahashi K, Takayasu Y, Chikamatsu K, Furuya N. Novel method for recording vestibular evoked myogenic potential: minimally invasive recording on neck extansor muscles. Laryngoscope 2005; 115: 1768-1773.

19. Di Lazzaro V, Quartarone A, Higuchi K, Rothwell JC. Short-latency trigemino-cervical reflexes in man. Exp Brain Res 1995; 102: 474-482.

20. Uchino Y, Sato H, Sasaki M, Imagawa M, Ikegami H, Isu N, Graf W . Sacculocollic reflex arcs in cats. J Neurophysiol 1997; 77: 3003-3012.