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Vestibular evoked myogenic potentials in Bell's palsy

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Abstract

The aim of the present study was to evaluate vestibular nerve involvement in patients with Bell's palsy with ocular and cervical vestibular evoked myogenic potentials (oVEMP and cVEMP). Ten patients who were diagnosed with Bell's palsy and ten healthy controls were included. All patients underwent VEMP recordings within six days after their initial presentation. Patients with Bell's palsy had greater oVEMP asymmetry ratio comparing to healthy controls (-38,4±28,7% vs -1,3±19.3%, p=0.005). As well N10 latencies of the oVEMP response were prolonged comparing to healthy controls (11.575 vs 9.72 ms). There was no difference in cVEMP asymmetry ratio or latencies between groups. We found no correlation between House-Brackmann grading scale and oVEMP asymmetry ratio (r=0.003, p=0,994). There are three possible explanations for increased oVEMP amplitudes on the affected side: 1) oVEMP response on the ipsilateral eye could be contaminated by facial nerve activity (blink reflex); 2) the amplitude of n10-p33 could be affected through the stapedial reflex; and 3) increased oVEMP amplitude could be the consequence of the vestibular nerve dysfunction itself, with prolonged latencies of the N10 oVEMP further supporting this explanation. The results of this study indicate possible involvement of the superior branch of the vestibular nerve in patients with Bell's palsy.

Key words: Bell's palsy, ocular and cervical vestibular evoked myogenic potentials

Introduction

Several studies have indicated asymptomatic involvement of vestibular nerve in cases of idiopathic facial nerve palsy (Bell's palsy), ranging from 22%-46% (1,2). The basis for such observation is the close proximity of the vestibular and facial nerves, which have a common course in the internal auditory canal (IAC). Some studies have even suggested that some of the superior and inferior vestibular nerve bundles may receive fibers from the facial nerve. (3)

Vestibular evoked myogenic potentials (VEMP) are myogenic short latency responses evoked by sound or bone conducted impulses. Aforementioned diagnostic method is in use for less than 30 years. Two potentials can be recorded: cervical VEMPs (cVEMP) which evaluate the integrity of vestibulospinal pathway, and therefore inferior vestibular nerve; while ocular VEMPs (oVEMP) are a manifestation of vestibuloocular reflex, and therefore evaluate the integrity of superior vestibular nerve (4).

The aim of the present study was to evaluate vestibular nerve involvement in patients with Bell's palsy with oVEMP and cVEMP.

Patients and methods

Patients who were diagnosed with Bell's palsy in the Emergency department of our institution from June 2012 until June 2013 were included. Age and gender adjusted healthy controls were recruited from the database of Laboratory for cognitive and experimental neurophysiology. All patients and controls signed

informed consent approved by the Ethical committee of the University Hospital Center Zagreb.

All patients underwent detailed neurological and ear, nose and throat examination, House-Brackmann grading scale was determined and neuroradiological tests (brain CT or MRI) were performed when indicated.

All patients underwent VEMP recordings within six days after their initial presentation. Methods of recording and analysis of recorded data were designed according to previously described details (4).

During the experiment participants sat in a comfortable chair. Patients were instructed to slightly move their head away from the back of the chair and push against an elastic band that was strapped over their forehead in order to activate sternocleidomastoid muscle (SCM). The contraction of the muscle was maintained due to the cooperation of patients in maintaining the same position during the test. Participants were also instructed to direct their gaze to the ceiling in order to activate ocular muscles (OM). The evoked response from the SCM was recorded by an active surface electrode placed on the belly of the stimulated SCM and was referred to a surface electrode placed on the tendon of the same SCM. The evoked response from the OM was recorded from two surface electrodes situated 2 cm below the contralateral eye. The active electrode was situated closer to the eye and referred to the reference 1 cm below.

The stimuli were delivered by a pair of headphones in series of 50 trails to one ear at a time and repeated two times for each ear in order to provide reproducibility. The presented stimuli were acoustic clicks, 1 ms in duration at the intensity level of 130 dB SPL. The stimulation rate was 1 Hz.

Recordings were performed using a Brain Products Brain Vision Recorder and the analysis of the recorded data was performed using a Brain Products Brain Vision Analyzer. Signals were filtered with a band pass filter from 5 Hz to 1000 Hz. For analytical purposes the signals were divided into segments with duration of 120ms (20 ms before the stimulus and 100 ms after the stimulus) and averaged for each set of 50 trials. Averaged responses from each set were used to calculate a grand average, used for further analysis.

Normalized baseline values were used as SCM amplitude data instead of the absolute amplitude value, because absolute amplitude of the evoked response depends on the amplitude of the muscle activity (muscle contraction) and is not a reliable measure. The normalized baseline amplitude value is calculated by dividing the absolute peak-to-peak amplitude (P13-N23) with the mean value of rectified muscle activity prior to stimulus.

The amplitude asymmetry ratio (AR) was calculated using the following formula: AR = ((healthy side – affected side)/(healthy side + affected side) x 100).

Statistical analysis was performed using IBM SPSS 19.0 (Chicago, IL).

Independent sample t-test was used to calculate difference between two independent samples with normal distribution and paired sample t-test was used to calculate difference between two paired samples. With the aim of calculating correlation between asymmetry ratio and Median House-Brackmann grading scale, Pearson correlation was performed. P values less than 0.05 were considered statistically significant.

Results

Ten patients with Bell's palsy (4 females and 6 males; median age 33.5 years, range 26-80) and 10 healthy controls (4 females and 6 males; median age 33 years, range 27-42) were included in this study. There was no statistically significant difference between groups regarding age and gender. Median House-Brackmann grading scale was 3.5 (range 2-5). In 5 patients diagnostic imaging was performed (brain CT in 2 and brain MRI in 3 patients) and results were normal.

Of all patients, only one had clinical signs of vestibular nerve involvement on examination. This 26-year-old female had unidirectional horizontal nystagmus with rotational component and a fast phase contralateral to the affected side, grade 1. Brain MRI was normal and nystagmus spontaneously resolved after 2 days.

Patients with Bell's palsy had greater oVEMP asymmetry ratio comparing to healthy controls (-38,4±28,7% vs -1,3±19.3%, p=0.005). As well N10 latencies of the oVEMP response were prolonged comparing to healthy controls (11.575 vs 9.72 ms). There was no difference in cVEMP asymmetry ratio or latencies between groups.

We found no correlation between House-Brackmann grading scale and oVEMP asymmetry ratio (r=0.003, p=0,994).

Discussion

The results of this study have shown that VEMP can detect subclinical affection of ipsilateral vestibular nerve in patients with Bell's palsy. This subclinical affection seams to be limited to the superior branch of the vestibular nerve, as only oVEMP differences were observed. This could be explained by anatomical studies showing connections between the superior vestibular and the facial nerve bundles, while the inferior vestibular has connection with the cochlear nerve bundles within the internal auditory canal. (3)

There are only few studies investigating VEMP in small series of patients with Bell's palsy. Initial studies using cVEMP only have shown that Bell's palsy does not affect the cVEMP results. (5) Similarly, 5 patients with unilateral Bell's palsy showed almost symmetrical oVEMPs, and there were no significant differences in the latencies and amplitudes of the oVEMP responses evoked by acoustic click and bone-conducted vibration between these patients and healthy subjects, suggesting that facial nerve activity had little effect on the oVEMP waveform. (6) Contrary to these studies, the present study has surprisingly shown increased amplitudes of the oVEMP response on the affected side. There are three possible explanations for this: 1) oVEMP is a manifestation of the crossed vestibulo-ocular reflex, so oVEMP response on the ipsilateral eye could be contaminated by facial nerve activity (blink reflex); 2) in Bell's palsy stapedial reflex is often reduced or absent, therefore the amplitude of n10-p33 could be affected through the stapedial reflex, similarily as p13-n23 amplitude of cVEMP is affected (7); and 3) increased oVEMP amplitude could be the consequence of the vestibular nerve dysfunction itself. We have recently shown increased cVEMP amplitudes in combination with prolonged latencies in patients with multiple sclerosis, demonstrating the possibility of this association. (8) Furthermore, prolonged

latencies of the N10 oVEMP further support the affection of the vestibular nerve in patients with Bell's palsy.

As in this study we did not test stapedial reflex, we cannot determine it's influence on oVEMP amplitudes. Another problem is that there is a significant variations in methodology and interpretation of VEMP results, which may limit comparisons of VEMP data across studies. In this study we have used acoustic clicks and we have followed recently published International guidelines for the clinical application of cervical vestibular evoked myogenic potentials, which should allow reproducibility of the results. (9)

Another limitation of the study is that MRI was not perform in all patients, thus we cannot be absolutely sure that Bell's palsy was not due to a lesion. However recently published guidelines on diagnosis and management of Bell's palsy by the American Academy of Otolaryngology—Head and Neck Surgery Foundation stated that clinicians should not routinely perform diagnostic imaging for patients with new onset Bell's palsy, which we followed in our study. (10)

In conclusion, the results of this study indicate possible involvement of the superior branch of the vestibular nerve in patients with Bell's palsy. Further studies combining caloric testing, stapedial reflex testing and VEMP on a larger number of patients should be performed in order to give definitive conclusions.

References

1. Rauchbach E, May M, Stroud MH. (1975) Vestibular involvement in Bell's palsy. Laryngoscope 85:1396-8.

2. Uri N, Schuchman G. (1986) Vestibular abnormalities in patients with Bell's palsy. J Laryngo Oto 100:1125-8.
3. Ozdoğmuş O, Sezen O, Kubilay U, Saka E, Duman U, San T, Cavdar S. (2004) Connections between the facial, vestibular and cochlear nerve bundles within the internal auditory canal. J Anat 205:65-75.
4. Rosengren SM, Welgampola MS, Colebatch JG. (2010) Vestibular evoked myogenic potentials: past, present and future. Clin Neurophysiol 121:636-651
5. Ochi K, Ohashi T, Kinoshita H. (2012) Acoustic tensor tympani response and vestibular-evoked myogenic potential. Laryngoscope 112:2225-9.
6. Chihara Y, Iwasaki S, Ushio M, Fujimoto C, Kashio A, Kondo K, Ito K, Asakage T, Yamasoba T, Kaga K, Murofushi T. (2009) Ocular vestibular-evoked myogenic potentials (oVEMPs) require extraocular muscles but not facial or cochlear nerve activity. Clin Neurophysiol 120:581-7.
7. Takegoshi H, Murofushi T. (2003) Effect of white noise on vestibular evoked myogenic potentials. Hear Res 176:59-64.
8. Gabelić T, Krbot M, Šefer AB, Išgum V, Adamec I, Habek M. Ocular and cervical vestibular evoked myogenic potentials in patients with multiple sclerosis. J Clin Neurophysiol 2013;30:86-91.
9. Papathanasiou ES, Murofushi T, Akin FW, Colebatch JG. (2014) International guidelines for the clinical application of cervical vestibular evoked myogenic potentials: An expert consensus report. Clin Neurophysiol 125;658-666.
10. Baugh RF, Basura GJ, Ishii LE, Schwartz SR, Drumheller CM, Burkholder R, Deckard NA, Dawson C, Driscoll C, Gillespie MB, Gurgel RK, Halperin J,

Khalid AN, Kumar KA, Micco A, Munsell D, Rosenbaum S, Vaughan W. (2013) Clinical practice guideline: Bell's Palsy executive summary. Otolaryngol Head Neck Surg 149;656-63.

Figures

Figure 1. Superimposed tracings from left (red line) and right (black line) oVEMP response in the patient with right Bell's palsy. Note the increased amplitude and prolonged latency of the right oVEMP response.