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Vestibular Evoked Myogenic Potentials Tests in Patients with Sudden Hearing Loss

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Abstract

Objective: To investigate the clinical value of two kinds of vestibular evoked myogenic potentials in patients with sudden hearing loss.

Methods: 82 patients were divided into two groups, accompanied with vertigo group and without vertigo group. All the cases were examined pure tone hearing thresholds, middle ear analysis, video nystagmus electrical diagram, caloric tests and vestibular evoked myogenic potentials elicited from the sternocleidomastoid and extraocular muscle respectively. 30 healthy subjects were selected as the control group.

Results: In 30 healthy subjects, the average latency of p13 and n23 of the cVEMPs were 13.13 ± 2.89 ms and 23.51 ± 3.25 ms respectively and the bilateral amplitude asymmetry rate was from 0.05 to 0.31. The average latency of n10 about the oVEMPs was 10.13 ± 0.48 ms. The average amplitude of n10-p15-wave was 5.58 ± 0.65μV. Among the 35 cases of sudden deafness with vertigo patients, 27 patients were normal in cVEMPs and oVEMPs examinations, five cases were abnormal in oVEMPs, and five cases were abnormal in cVEMPs. The latency and the amplifier of oVEMPs and cVEMPs were within the normal range in the 47 patients with sudden hearing loss without vertigo. The chi-square with SPSS17.0 software was analyzed between the two groups. The chi-square values were all 5.647, and the p-values were the same 0.017, the difference was statistically significant at the 95% confidence interval.

Conclusion: OVEMPs and cVEMPs can evaluate the vestibular nerve function of patients with sudden hearing loss accompanied with vertigo.

Keywords: Sudden hearing loss; Vertigo; Vestibular evoked myogenic potentials

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Introduction

Sudden hearing loss is a common disease of ENT, mostly happens unilaterally. Otolaryngology-Head and Neck Surgery Branch of Chinese Medical Association revised the diagnostic criteria for sudden hearing loss in 2015: Sudden sensorineural hearing loss onset may occur without apparent cause, at least in the adjacent two frequency hearing loss ≥ 20 dBHL and reaches the peak in 72 hrs. It is often accompanied with tinnitus, ear clogging, dizziness, nausea, vomiting and so on. Sudden deafness accompanied with vertigo is usually considered to be caused by vestibular involvement. In recent years studies have shown that the cervical-vestibular evoked myogenic potentials response to balloon and vestibular nerve function ocular-vestibular evoked myogenic potentials can respond to the upper vestibular nerve. The combination of the two tests should be able to respond to the vestibular nerves function in patients with sudden deafness. We have analyzed the cVEMPs and oVEMPs of 82 patients with sudden deafness. The results are analyzed and summarized.

Materials and Methods

Subjects

Diagnostic criteria for sudden deafness based on the Chinese Journal of Otorhinolaryngology Head and Neck Surgery, 2015, according to the vertigo symptoms; patients were divided into two groups. Accompanied by sudden paroxysmal rotative vertigo subjects were selected into sudden deafness with vertigo group, subjects without vertigo were selected into sudden deafness group. All subjects are unilateral. Between the 35 subjects of sudden deafness with vertigo, 16 left ears and 19 right ears, 21 males and 14 females, aged 23 to 49 years old. Between the 47 subjects without vertigo, 23 left ears and 24 right ears; 26 males and 21 females, aged 21 to 46 years old. The symptoms of sudden deafness and vertigo almost simultaneously onset, without other nerve involvement symptoms,

vertigo continued 1 hour to 5 days. 32 subjects accompanied by nausea, 16 subjects accompanied by vomiting. Except for subjects with vertigo recurrent, 30 healthy volunteers with normal hearing and no vertigo were selected as the normal control group.

All subjects had the necessary physical examination, neurological examination, necessary laboratory tests, temporal bone CT scan and pure tone hearing threshold, middle ear analysis, video electroretinogram, caloric tests, cervical-vestibular evoked myogenic potentials and ocular-vestibular evoked myogenic potentials. Ensure all subjects were without diseases like benign paroxysmal positional vertigo, labyrinthitis, vestibular neuritis, drug toxicity vertigo, vertebrobasilar insufficiency and intracranial space-occupying lesions and so on.

Stimuli and recording techniques of cVEMPs

A commercial electromyographic (EMG) system (Japan MEB-9200K) was used for cVEMP testing Subjects sat. A disc-shaped recording electrode placed at the midpoint of the sternocleidomastoid muscle belly, a reference electrode placed at sternocleidomastoid muscle clavicle, the ground electrode placed at mid-forehead skin surface. The skin surface impedance is less than 5 kilohms. Using the air guide click sound stimulation, the intensity was 105dBnHL, stimulation frequency was 3 times per second, bandpass filter range was 500Hz ~ 2000Hz, superimposed 200 times. Subjects maintained a tonically-contracting sternocleidomastoid muscle during stimulation and recording. Sound stimuli were delivered monaurally and recorded bilateral. Amplitude and latency of p13 and n23 were recorded. The lower vestibular nerve function was evaluated by compared with the normal subjects [1].

Stimuli and recording techniques of oVEMPs

A commercial electromyographic (EMG) system (Japan MEB-9200K) was used for oVEMP testing. The subjects lay in the supine position. The standard was placed at 70 cm from the top of the head and 25 degrees backward positioning. A disc-shaped recording electrode placed at the middle of the lower side of the pupil 10 mm. The reference electrode placed at the skin 20 mm lower than recording electrode. The ground electrode placed at middle of forehead. The skin surface impedance is less than 5 kilohms. Using the air guide 500Hz click sound stimulation, the intensity was 105dBnHL. Sound stimuli were delivered monaurally and recorded bilateral. Stimulus frequency was 3 times per second, bandpass filter range was 20Hz ~ 500Hz, superimposed 50 times. The subjects must keep their head unmoved and eyes were kept on the visual standard during the test. A significant negative waveform named n10 was recorded about 10 seconds after stimulus onset. The latency and amplitude of n10 were calculated and comparative analyzed with normal subjects [2].

Statistical Analysis and Results

According to WHO classification of hearing loss: normal hearing is less than 25dBHL, mild deafness is 26-40dBHL, moderate deafness is 41-60dBHL, severe deafness is 61-80dBHL, extremely severe deafness is more than 80dBHL.

CVEMPs and oVEMPs were both evoked in 30 healthy subjects. The statistical results were assessed by mean addition and subtraction standard deviation. The latency of p13 and n23 of cVEMPs was 13.13 ± 2.89 ms and 23.51 ± 3.25 ms. The amplitude asymmetry is 0.05 ~ 0.31. The latency of the n10 waveform of oVEMPs is 10.13 ± 0.48 ms and the mean amplitude of the n10-p15 waveform is 5.58 ± 0.65 μV. The amplitude asymmetry between the two sides is 0.05 ~ 0.35.

Between 35 subjects of sudden deafness with vertigo, 6 subjects were mild deafness, 21 were moderate deafness, 5 were severe deafness and 3 were extremely severe deafness. There were 27 subjects whose latency and amplitude of cVEMPs and oVEMPs were normal. There were 5 subjects with abnormal cVEMPs (2 with extended p13 latency, 1 with extended n23 latency, 1 with extended p13 and n23 latency); There were 5 subjects with abnormal oVEMPs (3 with n10 extended latency, 2 were not evoked). There were 2 subjects whose results were both abnormal (1 contralateral n10 latency and ipsilateral p13 latency extended, 1was not evoked).

Between 47 subjects of sudden deafness without vertigo, 6 subjects were mild deafness, 29 were moderate deafness, 8 were severe deafness, 4 were extremely severe deafness. OVEMPs and cVEMPs waveforms and latency were in the normal range compared with the control group.

The chi-square with SPSS17.0 software was analyzed between the two groups. The chi-square values were all 5.647, and the p-values were the same 0.017, the difference was statistically significant at the 95% confidence interval.

Discussion

Sudden deafness is a kind of sensorineural deafness without apparent cause. The cause of sudden deafness with vertigo is more complex and the clinical hearing loss is usually worse, the probability of hearing recovery is also significantly lower than those without vertigo [3]. The probable cause is damage of the cochlea because of vascular factors or viral infection. The vertigo symptoms appear when the vestibular organ is also affected.

The incidence of deafness and dizziness occur almost accompanied. Studies have shown that patients with sudden deafness with vertigo are different from Meniere's disease, it mostly caused by the involvement of the vestibular nerve afferent receptors, and the symptoms of vertigo rapidly improved through the quickly established of vestibular central compensatory mechanism [4]. The function of the vestibular nerve is essential for the diagnosis and differential diagnosis of sudden deafness with vertigo.

Studies have confirmed that cVEMPs receptors located in the balloon, nerve impulses through the vestibular nerve conduction and the vestibular nerve lateral nucleus to the medulla oblongata, then spread to the ipsilateral sternocleidomastoid muscle production. OVEMPs receptors are located in the oval capsule, the sound stimulation through the synaptic connection, the nerve impulses send from the vestibular nerve up to the vestibular nerve medial nucleus, and then to the high brain stem (midbrain or pons), the oculomotor nerve to the contralateral oblique and inferior rectus produce vestibular induced myogenic potential. To patients with sudden deafness, the tests of cVEMPs and oVEMPs, video electroacoustic examination and caloric tests can be more effective response to vestibular nerve function [5], It is important to patients of sudden deafness with vertigo in the diagnosis and differential diagnosis.

In this study, 82 subjects of sudden deafness were tested with cVEMPs and oVEMPs. Abnormal results of cVEMPs and oVEMPs can be 25.7% (9/35) detected in patients with vertigo which has statistically significant compared with patients without vertigo. So it has a certain clinical significance to test vestibular evoked myogenic potential to sudden deafness patients with vertigo. The abnormal rate of the VEMP is high in patients with moderate to extremely severe

deafness. Further observation is needed for determine the specificity of the diagnosis of vertigo. More cases were needed to prove the significance of latency and amplitude before and after treatment.

From the abnormal rate of cVEMPs and oVEMPs, we can conclude that involvements of the upper vestibular nerve and lower vestibular nerve of this group of sudden deafness patients with vertigo were roughly the same, and it was not statistically significant. From the correlation analysis of cVEMPs and oVEMPs with pure tone and middle ear analysis, the chi-square test was not statistically significant; we can conclude that vestibular myogenic evoked potentials were not related to the degree of hearing loss and the functional status of the middle ear. Further study is needed to confirm weather VEMPs are related to cochlear electrograms, video electroencephalography, caloric tests and other vestibular function tests.

Sudden deafness is common in ENT, but the pathogenesis of those with vertigo is more complex. To the initial onset of vertigo patients, it's difficult to identify the sudden deafness accompanied with vertigo or Meniere's disease Caused by dizziness associated with hearing loss. Patients with sudden deafness accompanied with vertigo have no specific findings with cVEMPs and oVEMPs tests. VEMPs tests can provide evidence for the lesion location of the vestibular and provide objective information for further clinical observation.

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