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Research Article

Vestibular Activation by Sound in Human

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Abstract: It has been established that the seismic sensitivity of the human vestibular system exceeds that of the cochlea for low-frequencies. The objective of this work is to determine spurious low frequency air bone gap (slABG) observed in some patients with severe sensorineuronal hearing loss (sSNHL) is due to vestibular otolith organ perception. Study design was Cross-sectional study. Twenty normal volunteers and twenty age-matched patients with sSNHL were included in this study. The participants were undergone comprehensive audiologic tests, cervical vestibular-evoked myogenic potentials (cVEMPs), and thin-sliced computerized tomography scans (CT) at the audiology department of Hamadan University of Medical Sciences, Iran. The observed sIABG in the patients group was disappeared at frequencies over 1000Hz. Mean value for sIABG was 38.7 dB, 29.3 dB, 20.4 dB and 13.9 dB for frequencies of 250 500 750 and 1000 Hz, respectively. We observed twenty three ears with sSNHL showed slABG>10 dB and abnormal cVEMPs (affected). Seventeen ears without slABG>10dB had normal cVEMPs (unaffected). Normal group had normal cVEMPs. None of the patients showed Tullio sign or noise- pressure-induced nystagmus, or third window type symptoms during exam and testing periods. All CT were normal. Statistical analysis of the peaks latency and amplitude of cVEMPs among three groups revealed significant differences (P = 0.039, one-way ANOVA test, Tukey HSD). Higher threshold for bone-conduction at low-frequencies in the ears with SSNHL may be due to stimulation of the otolith organs. We assumed that the observed slABG is not due to vibrational sensation.

Keywords: Vestibular Evoked Myogenic Potentials, Sensorineural Hearing Loss, Vestibule.

INTRODUCTION

Behavioral and physiological studies indicate that the sacculus functions in part as a hearing organ in several non-mammalian and mammal species (fish, rays, toads, birds, guinea pigs, cats squirrel monkeys and human) [1-3]. There are direct evidences of vestibular sensation to low-frequency sounds [4-6]. All the vestibular endorgans (three canals and two maculae) respond to sound. Among the five endorgans, the saccular macula shows the lowest thresholds. The vestibular and cochlear sensory organs have similar mechanical receptors and transducers but their specific ranges of sensitivity lie widely apart and tuning seems to be a special cochlear property. The best frequencies is not exceed 1000 Hz to sound and 500 Hz to vibration [7]. The natural stimuli (voice intonation in speech, singing, crowd actions at a concert or sporting event, self-monitoring of vocalisations and instrumental sounds) can stimulate and be perceived by vestibular system [8-10].

Saccular stimulation by sound can induce a possible sensation to improve better hearing in clamor locations. In hard of hearing conditions, saccule has a facilitating role for cochlea and can contribute to the detection of high intensity low-frequency tone. It may contribute to the hearing of this frequency band [6]. The acoustic sensitivity of the saccule to low frequency component is effective in neural activities [5] and there is a vestibular-auditory interaction [7].

Vestibular hearing as an auditory sensitivity of the saccule in the human ear is revealed by cVEMPs. The range of vestibular hearing happens to coincide with the range of our voice pitch [8]. People with normal saccular function or safe vestibular hearing have intact projections to cochlear nucleus, lateral lemniscus, and to inferior colliculus. It is effective in the improvement of the neural synchronization and can be effective in better interaction of bottom-up processing [7]. Vestibular hearing can contribute to frequency discrimination of loud tones and can improve speech perception [8]. It may be effective in top-down processing of loud sounds and can be valuable for speeh processing and perception/production system [9].

The sensation of the sound at low frequencies may be present in patients with total deafness and normal

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vestibular function (predominantly saccule [10]. This improvement disappears when saccular function is lost [11]. The auditory sensitivity of the saccule augments bone-conducted low frequency hearing thresholds in deaf patients [12].

During routine audiologic testing, one encounters patient with some degree of hearing loss whom demonstrates some degree of slABG in the lowfrequencies range. This gap has been attributed to possible vibrational sensation produced by strong vibration of the testing vibrator. However the gap is not seen in the entire patient with hearing loss. In this work, we investigated the possibility of the vestibular sensation as a cause of the low frequency slABG in patients with some degree of hearing loss.

MATERIALS AND METHODS

This case–control study involved twenty healthy subjects, which were consisted of audiology students and hospital staff, compared to twenty cases with congenital or early-age acquired sSNHL. Total of eighty ears were evaluated, and testing was performed bilaterally. All the patients with sSNHL were presented to the audiology department of Hamadan university of medical sciences (Iran, Hamadan), from December 2011 through April 2012. The study was approved by the Hamadan university ethics committee.

Patients with history of ear infections and middle ear diseases and ear surgeries, sign and/or symptoms of superior semicircular canal dehiscence during clinical and audiological examinations (fistula sign and/or Tullio phenomenon) or on thin-cut CT images of the temporal bone were excluded.

After initial full auditory and vestibular examinations and history taking by our otolaryngologist, full auditory and vestibular test batteries consisted of pure tone audiometry and tympanometry (Madsen, OB-822) impedance acousticmetry (Maico, MI. 34), cVEMPs (Labat Epicplus) and CT scans were performed on of the patients.

During the audiometric evaluation of the patients with sSNHL possibility of vibrotactile responses for bone-conducted low-frequencies was reduced by using of insert (ER-3A) earphones [13]. In addition, we gave instructions to patients about bone-conducted hearing sensation versus feeling of vibrotactile stimulation. Since, the greater inter-aural attenuation provides by ER-3A insert earphones in lower frequencies, the need for contralateral masking is eliminated at 250, 500 and 1000 H_{Z} [13]. The use of contralateral masking was indicated whenever the results of unmasked boneconduction audiometry were suggested the presence of an air-bone gap in the test ear of 15 dB or greater [13]. The absence of Tullio phenomenon during audiometry and tympanometry interpreted as a less likelihood of possibility of third window syndrome [14].

CVEMPs

Patients were placed in the supine position on a gurney within a sound-treated room. They were instructed to turn and hold their heads as far as possible toward the side contralateral to the stimulated ear (thereby activating the ipsilateral sternocleidomastoid muscle). At that point, the overall electromyogenic activity of the was set as the reference level of the tonic contraction (usually 100-400 µV). Patients were asked to maintain contraction at this level throughout the test session (approximately 30 s). The active electrode was placed over the middle portion of the ipsilateral muscle body [4]. The reference and the ground electrodes were placed over the upper sternum and on the midline forehead, respectively. Impedance was less than 20 k Ω . Auditory stimuli consisted of tone burst (500 Hz, 120 dB peakSPL, rise/fall time=1 ms, plateau=2 ms), presented to the ear ipsilateral to the contracted muscle, band pass filtered (20 Hz to 2 kHz), and a grandaverage of the 200 responses was recorded [14]. The cVEMPs results for the control group were used as normative data. Latencies longer than the calculated upper limit were interpreted as abnormal.

Data Analyses

All analysis was done by means of the statistics software $SPSS_{17}$. Kolmogorov-Smirnov test was used for evaluation of normal test distribution. One-way ANOVA was used to compare findings among the groups. Tukey's least significant difference (Tukey HSD) test was chosen as the post hoc test. The P-value of < 0.05 was considered to be statistically significance.

RESULTS

We evaluated twenty healthy subjects, including 10 females and 10 males (20-29 years old, mean age of 26 years). Patient group was consisted of twenty cases 8 females and 12 males with sSNHL (23-39 years old, mean age of 32 years).

All of the patients and subjects had normal head and neck exam, normal otoscopic exam. Thinsliced CT scans of the temporal bone revealed normal middle and inner ear anatomy. No radiological sign of thin tegmen, dehiscence of any of the semicircular canals or large vestibular aqueduct was noticed. All of the patients had congenital sSNHL except on case with early childhood acquired damage.

Tympanometery revealed normal pressure and volume in all the tested ears. No sign of pressure- or sound-induced nystagums or vertigo. All of the tested ears had negative fistula test and/or Tullio phenomenon.

Twenty patients with sSNHL examined in this study. In total we observed 23 ears with slABG > 10 dB and 17 ears without slABG > 10dB. Eight patients had binaural damage and bilateral slABG more than 10 dB, seven patients with bilateral sSNHL had unilateral

slABG > 10 dB, and five patients had bilateral sSNHL without slABG > 10dB. Figure-1 illustrates audiometric finding of a patient with slABG and normal cVEMPs morphology and latencies.

Mean value for air slABG per frequency was 38.7, 29.3, 20.4 and 13.9 dB for frequencies of 250, 500, 750 H_Z and 1000 H_Z , respectively (table 1). The observed slABG in the patients group was disappeared at frequencies over 1000Hz. All the ears except one (22 ears) with slABG showed near normal cVEMPs morphology. Ears without slABG (17 ears) and one ear with slABG did not revealed any recordable cVEMPs. The mean latency values for p13 and n23 and peak-to-

peak amplitude are observable in table-2 and a normal graph of cVEMPs in a healthy ear .is visible in figure-2. Comparable to normative data collected from control group prolonged latency and lower amplitude was recorded in five patients with bilateral slABG and seven patients with unilateral slABG. One of the ears of a patient with bilateral slABG did not show cVEMPs response. The cVEMPs asymmetry ratio findings indicated smaller cVEMPs amplitude in the ears with slABG < 10dB comparison to the ears without the gap. Statistical analysis of the peaks latency and amplitude among the groups revealed significant differences (p = 0.039, one-way ANOVA test, Tukey HSD).

Table 1: Air-and Bone-conducted hearing thresholds (250, 500, 750, and 1000 $_{\rm HZ}$) in twenty-three unaffected ears of the case-group with severe low-frequency sensorineural hearing loss

Case number	Air conducted hearing threshold	Bone conducted hearing threshold	
	250 500 750 1000	250 500 750 1000	
1	90856565	40503045	
2	75859085	35557575	
3	70708085	30456065	
4	85858585	50556575	
5	75858585	45657075	
6	90858585	60556560	
7	80908585	40607075	
8	70707075	35654560	
9	75758585	25405065	
10	70758585	40557075	
11	90858590	60556575	
12	80857070	45504550	
13	75859090	30456070	
14	80757575	40456065	
15	90908585	40557065	
16	80858585	55657575	
17	75858585	45657075	
18	85857070	45505060	
19	70707575	40456065	
20	80858585	55657075	
21	75858585	45657075	
22	85859090	40456075	
23	70656565	35505555	

 Table 2: The mean latency and peak to peak amplitude of cervical vestibular evoked myogenic potentials

 (cVEMPs) results in control groups and affected ears of the case-group with severe low-frequency sensorineural hearing loss (slSNHL)

Group	Ν	latency of p13 (ms)	latency of n23 (ms)	peak to peak amplitude(µv)
Healthy controls	40	12.7 ± 1.0	22.1 ± 2.2	45.9 ± 3.8
Affected ears	17	16.8 ± 1.4	29.7 ± 2.2	14.1±5.7
Unaffected ears	23	12.4 ± 1.9	21.9 ± 1.8	43.4 ± 7.1

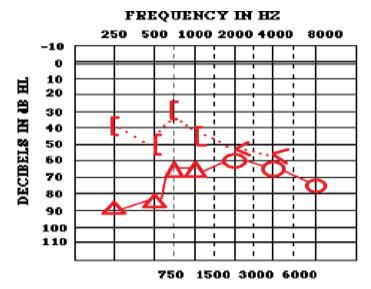


Fig. 1: Spurious Low Frequency Air-Bone Gap (slABG) in an ear with Severe Sensori-Neuronal Hearing Loss (sSNHL)

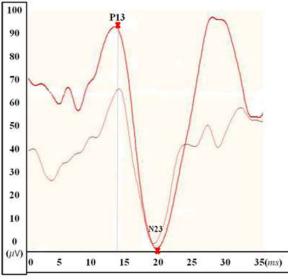


Fig. 2: A normal graph of cVEMPs in a healthy ear

DISCUSSION

Our study in a subset of patient with sever sSNHL with low-frequency range slABG showed close association of presence of the gap with functionality of otolith organ revealed by cVEMPs recording. The possible hypothesis such as tactle perception, third window or vestibular hearing, which may explain this association, will be discussed in details in this section.

During routing hearing assessment, it is not unusual to have patient with sever hearing loss that shows low-tone threshold discrepancy between air- and bone-conduction hearing threshold. This type of audiogram which is called "Receptive audiograms" with a rapidly sloping curve display bone thresholds which are exceptionally good up to 500 Hz and definitely better than the corresponding air thresholds (generally from 30 to 50 dB) [11]. This air- bone gap disappears over frequencies higher than 500 Hz. This striking discrepancy between air and bone thresholds over the lower frequencies is generally present in cases where vestibular excitability is within normal limits. In those cases when vestibular excitability is lost there is no discrepancy between the bone and air thresholds over the entire auditory range [11]. Bocca and Perani, 1960 has shown that in total deafness, bone conduction as a sensation of sound is entirely lost [3].

Traditionally it has been assumed the patients with receptive audiograms are responding to the vibrational property of the bone vibrator and not to the sound itself. Of interest is that slABG is not an universal finding in patient with sSNHL. In our study, prior to data collection time, we extensively educated the patients about the tactile perception of the bone vibrator and true sensation of the sound hearing. We ran multiple tests to check the reliability of the responses and confirm their understanding of the difference between vibrational sensation and sound perception. So, we do not believe that the observed gap and low-frequency sensation would be due to perception of the vibration. In addition, our data clearly revealed that not all of the patients with SNHL has the ABG. More interestingly some patients with bilateral sSNHL showed only unilateral slABG which correlated closely with the presence of the cVEMPs responses.

Our patients with severe sSNHL, and with absence of slABG in low frequencies, regardless of the uni- or bi-lateral nature of the sSNHL, did not have recordable cVEMPs. On the other hand, we were able to record cVEMPs in patients with sSNHL and low-tone slABG. The strong link between presence of cVEMPs and presence of the slABG also can argue against the tactile perception assumption but true threshold for bone conduction and responsiveness of the otolith organ to low-tone high intensity sound.

We assumed that this low-tone perception by this group of patient specially at 250-1000 Hz range might be due to perception of the low-tone sound by intact otolith organs and not vibrational sensation by the patients. Recent studies of otolith and vestibular endorgans to sound clearly revealed such a low-tone sound sensitivities of otolith organ recorded at the single neuron level as well as the far filed electrophysiological recordings [14,16]. Auditory sensitivity of the sacculus has been demonstrated among mammals, including guinea pigs [17,18], cats [19,20], and squirrel monkeys [21].

Earlier studies using air conducted tone pips as stimuli have pointed out that the myogenic inion response amplitude and the air-conducted cVEMPs response amplitude is greatest at the tone pip frequencies of 250 and 500 Hz [22, 23]. More recently Sheykholeslami et al, using Bone- cVEMPs has been reported a frequency range up to 1000 Hz in human with best frequency range of 200-400 Hz [4, 24].

The low-tone air-bone gap has been recently reevaluated after discovery of the third window phenomenon [25, 26]. The third window condition is present in a patient with anatomically discrete lesions at different locations such as semicircular canals, bony vestibule, or the cochlea. Some of these abnormalities are semicircular canal dehiscence (SCD), enlarge vestibular aqueduct syndrome (EVAS), DFN-3 (Xlinked deafness with stapes gusher) and Paget disease [27]. Various names such as pseudoconductive loss, inner ear conductive loss, cochlear conductive loss, etc, have been used for the observed air bone gap (ABG). In this work, we used of pseudoconductive loss for the observed ABG. A numbers of clues and functional tests are available to the clinician for making an accurate diagnosis. These conditions have been extensively studied in the literature. The exact mechanism of the ABG in these disorders has not been satisfactorily

explored. Putative explanations have included: (a) a decrease in stapes mobility due to increased perilymphatic or endolymphatic pressure [28], (b) an unspecified disturbance of cochlear mechanics [29], (c) a selective facilitation of bone- conducted hearing because of the large VA acting as a third window [30]; and (d) an impairment of air-conducted hearing combined with facilitation of bone-conducted hearing because of the large VA acting as a third window in the inner Clinical, audiologic ear [31]. and electrophysiological features of these disorders have been expensively studied. Clinically some of these patients have sound- or pressure induced nystagmus or sensation of movement with balance problem. Audiological studies of these patients shown to have low-frequency ABG and normal acoustic reflexes. cVEMPs recording from these patients revealed low response threshold with higher peak-to-peak amplitude.

In our patients group, we did not encounter any of the maintained findings such as sound- pressureinduced nystagmus, abnormal cVEMPs (higher amplitude or lower threshold) and normal CT of temporal bone. We speculated that our findings is suggestive of a different condition and is of vestibular organ origin, particularly otolith organs.

Interest in vestibular contribution to hearing has been debated for long time. It has been suggested that the perception of the 'volume' of sounds depends on the `cochleo-vestibular' system and, in addition, that the perception of low-frequency BC sounds is mediated through the vestibular endings in the saccule [4, 11, 32]. Later on work of Ribaric et al. showed reproducible and measurable bioelectric responses when the mastoid of profoundly deaf subjects who had a normally functioning vestibular apparatus was stimulated with 100-Hz sinusoidal vibration [32]. Recent work in nonhuman mammals, e.g. cats [19], considered the possibility that acoustic sensitivity of the saccule may indicate the `conservation of a mechanism'. These authors hypothesized that the saccular afferents in cats may give rise to a response in middle ear muscles, such as the stapedius, which might serve an `anti-masking' function of low frequency on high-frequency tones since the saccular tuning curve partially, but not completely, overlaps the stapedius tuning curve in cats. Such experimental works suggest that profoundly deaf subjects with a normally functioning vestibular system might obtain useful information from sound when stimulated adequately [20]. More recently, Todd et al. reported a short-latency and low-threshold response to bone conducted acoustic stimulations. They concluded that given the low threshold of vestibular acoustic sensitivity it is possible that this mode may make a contribution to the detection of and affective responses to loud low frequency sound. A short latency vestibular evoked potential (VsEP) produced by bone-conducted acoustic stimulation [33]. In addition same author was investigated contribution of vestibular receptors to the

late auditory evoked potentials of cortical origin vestibular receptors contribute to cortical auditory evoked potentials [34]. Their study revealed that in passing through the vestibular threshold while recording cortical mid- and late- latency responses, systematic changes were observed in the morphology of the potentials, a fronto-central negativity, which appeared at about 42 ms, referred to as an N42, prior to the AEP N1, and in the intensity dependence of their amplitude and latency. These changes were absent in a patient without functioning vestibular receptors. Clinical implication of our research work is not clear at this time. However, it has been suggested that single channel electrical stimulation of sacculus might able to patient to perceive the sound. Trivelli et al. reported in deaf patients saccule has a compensatory roel for cochlea [35]. The new studies show in healthy adults saccule has an auditory function for low-frequency/high intensity sound [9], in noisy competing situations can cooperate in speech perception [8], improves pitch detection and music perception [7], and in other word all human hearing is not cochlear [6]. Auditory sensitivity of the saccule augments bone-conducted low frequency hearing thresholds in deaf patients, which may be mediated through vestibular endings in the saccule [11]. In this regard, many perceptive audiograms with a rapidly sloping curve display bone thresholds which are exceptionally good up to 500Hz and definitely better corresponding the air thresholds. than This improvement for bone-conducted low frequency disappears over frequencies higher than 500Hz [11].

In conclusion, we evaluated a subset of patient with sensorineuronal hearing loss whom showed presence of cVEMPs and saccular hypersensitivity to low-frequency sound when they demonstrated low to mid- frequency air-bone gap. In contrast to patient with third window phenomenon, our patients did not have any evidence of bony labyrinth defect on thin-sliced CT of temporal bone nor high amplitude, low threshold cVEMPs. we believe that the phenomenon observed here is not due to vibrational perception observed correlation of ABG with presence of cVEMPs in patients with severe sSNHL in this study is in agreement with the hypothesis that the perception of low-frequency sounds may be mediated through vestibular otolith organ. Further studies need to address the possibility of contribution of otolith organ to low-frequency hearing and other reflexes depending on low-frequency high intensity input such as stapedial reflexes.

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Abbreviations

CVEMPs: Cervical Vestibular Evoked Myogenic Potentials; slABG: Spurious Low Frequency Air-Bone

Gap; sSNHL: Severe Sensori-Neuronal Hearing Loss; CT: computerized tomography scans.

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