

## Otoacoustic Emissions in Sudden Sensorineural Hearing Loss: Changes of Measures with Treatment

\*ShadmanNemati<sup>1</sup>, Seyyed-EbrahimNaghavi<sup>2</sup>, EhsanKazemnejad<sup>3</sup>, RoozbehBanan<sup>4</sup>

### Abstract

#### Introduction:

To identify changes in OAEs parameters in treatment course of idiopathic sudden sensorineural hearing loss (iSSNHL).

#### Materials and Methods:

In a prospective study from August 2005 to January 2009, 26 patients with iSSNHL underwent conventional audiometry/tympanometry and two types of OAEs (TEOAEs and DPOAEs) before and after the completion of standard drug therapy. The changes in pre- and post-treatment parameters were compared with each other and with normal-contralateral ears.

#### Results:

In TEOAEs, the mean overall correlation (reproducibility) and the mean overall strength in involved ears were  $10.96 \pm 23.36$  and  $0.99 \pm 3.45$  dB, respectively, before the treatment, which reached  $22.88 \pm 36.55$  and  $1.85 \pm 5.3$ , respectively, after the treatment ( $P > 0.05$ ). Significant difference between "correlation score" (average of correlations at 3-4 involved frequencies) before and after treatment was found:  $6.52 \pm 18.19$  vs.  $21.67 \pm 37.8$  ( $P < 0.034$ ). The difference between pre- and post-treatment overall correlation and correlation score in the "response group" were significant ( $P < 0.031$ ). In DPOAEs of the involved ears, the mean DP1 level and the DP1 signal-to-noise ratio changes were not significant with the treatment ( $P > 0.05$ ).

#### Conclusion:

Evoked OAEs, especially TEOAEs, are objective, rapid, and sensitive tools in the treatment course of iSSNHL.

#### Keywords:

Idiopathic sudden sensorineural hearing loss, Otoacoustic emissions, Response, Treatment

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<sup>1</sup>Department of otorhinolaryngology, Guilan University of Medical Sciences, Rasht, Iran

<sup>2</sup>Department of otorhinolaryngology, Guilan University of Medical Sciences, Rasht, Iran

<sup>3</sup>Department of biostatistics, Guilan University of Medical Sciences, Rasht, Iran

<sup>4</sup>Ear, Nose, Throat, Head and Neck surgery Research Center, Guilan University of Medical Sciences, Rasht, Iran

\*Corresponding author:

Ear, Nose, Throat, Head and Neck surgery Research Center, Amiralmomenin Hospital, Rasht, Guilan, Iran

E-mail: [nemati@gums.ac.ir](mailto:nemati@gums.ac.ir), Tel: +989111370488

### **Introduction**

David Kemp in 1978 described some types of sound waves of cochlear origin which can be detected with a microphone in the external auditory canal. Since then, many studies have been performed in the clinical applicability of these "emissions" named "otoacoustic emissions (OAEs)". Out of the various types of OAEs, transient evoked OAEs (TEOAEs) and distortion product OAEs (DPOAEs) may be detected in nearly all subjects with normal cochlear and middle ear function. While TEOAEs will be absent in sensorineural hearing losses with less severities, DPOAEs are absent in sensorineural hearing loss exceeding 50 dB Hearing Level, but are measurable in inflammatory conditions causing HL secondary to cochlear nerve involvement (1-3). OAEs are objective and non-invasive testing of the cochlear outer hair cell (OHC) function and have a direct relationship to hearing threshold sensitivity. With high reproducibility, high test-retest stability, and with temporal and spectral properties unique to each individual, OAEs are performed conveniently and rapidly and are more sensitive in comparison with routine audiometric tests. They can be applied in difficult-to-test cases and inorganic hearing losses and are able to show "subclinical" events in the cochlea (1,4-9).

Many researchers have shown that evoked OAEs can successfully separate normally hearing and hearing impaired populations. Normative measurements have been studied, but more studies should be performed on the clinical applications of OAEs and on optimizing current protocols, especially in hearing-impaired populations. (3,10,11). Sudden sensorineural hearing loss (SSNHL) is the loss of hearing more than 30 dB in three contiguous frequencies that occurs in less than three days. It is fairly uncommon and has an overall incidence of 5-20 per 100,000 individuals per year. SSNHL is a controversial topic in

otolaryngology, with more than 100 different etiologies, yet its etiology remains unknown: "idiopathic" SSNHL (4,12,13). There are increasing evidences in the literature that in some cases SSNHL only has psychogenic causes (14-16). Since TEOAEs and DPOAEs seem to reflect the activity of the OHCs, it is reasonable to hypothesize that in most idiopathic SSNHL cases, OHC function deteriorates when the hearing threshold is raised, and it recovers as hearing improves. In this study we tried to identify the changes in measures of these 'objective' tests during the recovery process of iSSNHL.

### **Materials and Methods**

This prospective study was performed in two university Hospitals at Isfahan University of Medical Sciences and Guilan University of Medical Sciences from August 2005 to January 2009. Out of over 60 patients with SSNHL who had been referred to our clinics, after excluding patients with known causes of SNHL (such as Meniere's disease, acoustic trauma, and multiple sclerosis), and also patients with more than two weeks from the onset of sudden deafness and those who were treated for SSNHL before referral to us, we enrolled 28 cases for our study. All patients underwent physical examinations, and such audiological tests as pure tone audiometry (PTA) and tympanometry (Amplaid 728 clinical, Amplaid 314 clinical), DPOAE and TEOAE (Capella, MADSEN clinical version 2.10, 2001). All of the tests were performed in identical conditions and by the same (well-trained) operator. Patients with abnormal tympanograms (two cases) were excluded from the study. The TEOAEs were obtained with stimuli consisting of non-filtered clicks of 80 microsecond duration and 80-90 dB SPL level. The click rate was 55 per second, and a total of 2000-5000 sweeps were averaged using a passband of 500-6000 Hz recordings utilizing fast-screen

mode. The TEOAE waveform was analyzed in 500-1000-Hz-width frequency bands, and the signal-to-noise ratio and the reproducibility of signals (correlations)-in percent-and emission strength-in db-were obtained in 5 different contiguous frequency bands (750 Hz to 4500 Hz). For DPOAEs, primary tones  $f_1$  and  $f_2$  were presented at 70dB and 60-dB Sound Pressure Levels (SPL). The  $f_2/f_1$  ratio was kept at approximately 1.2 (ranging from 1.21 to 1.23) and the frequency of  $f_2$  was changed in 1/4-octave steps from 500 Hz to 8000 Hz. The levels of the DPOAEs at  $2f_1-f_2$  were recorded. In nine different frequencies (ranging between 500 and 8000 Hz), DP-gram showed DP1 level (dB) and DP1 signal-to-noise ratio/dB. For all the patients, necessary tests for the disease were performed, and they were then treated with oral steroids (prednisone 1mg/kg oral daily for 10 days and then tapered) and acyclovir (800mg qid for 7 days). PTA and Speech Discrimination Score (SDS) were performed every 3-5 days during the treatment, and post-treatment PTA, SDS, TEOAE, and DPOAE were performed two weeks after termination. According to the treatment response, the patients were classified into three groups: the complete- or good-response group ( $\geq 30$ dB recovery in affected frequencies in PTA or  $\geq 30\%$  increase in SDS), the partial- or moderate-response group ( $\geq 10$ dB and  $\leq 30$  dB recovery in affected frequencies or  $\geq 10\%$  and  $< 30\%$  increase in SDS), and the poor- or no-response group ( $\leq 9$  dB recovery in PTA or  $\leq 9\%$  increase in SDS) (3,4). Then we analyzed the data (various parameters of pre- and post-treatment DPOAE and TEOAE) from affected ears in the three study groups and in comparison with those of contralateral non-affected ears as controls. The data were analyzed by Chi-square test, Levene's test for equality of variances, T-test, one-way ANOVA, and Wilcoxon Signed Ranked test using SPSS-

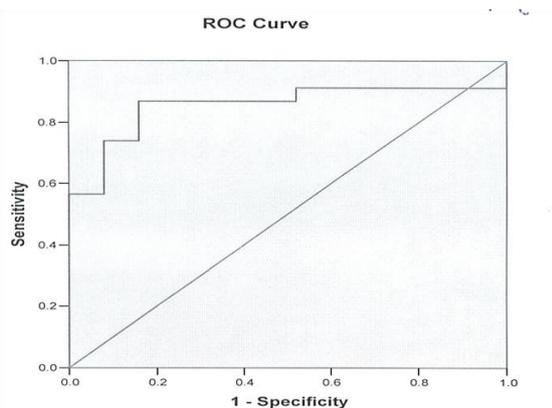
16 software, and the level of significance was considered 0.05.

### Results

From the 26 patients with iSSNHL (16 male, 10 female, mean age=40.54±15.25 years), the left ear was involved in 16 and the right ear in 10 cases. There was no case with bilateral involvement, but in two cases, the uninvolved ears were not normal because of previous ear surgery (Case 15) and temporal bone fracture (Case 21). Seven cases had profound hearing loss (hearing thresholds  $> 70$  dB), 6 cases had severe hearing loss (Hearing thresholds  $> 55$  and  $< 70$  dB), 10 cases had moderate hearing loss, and 3 cases had mild HL (hearing thresholds  $< 40$ , but  $> 20$  dB). After the completion of the treatment, 12 cases showed complete response, 8 cases partial response, and 6 cases poor response according to audiometries. There were no statistical difference in these three groups as for age and sex. In TEOAEs (Table 1), the mean overall correlation (reproducibility) and overall strength before the treatment in involved ears were  $10.96 \pm 23.36$  and  $0.99 \pm 3.45$  dB respectively, while measured  $57.52 \pm 41.39$  and  $10.26 \pm 6.8$  dB respectively in the normal ears ( $P < 0.01$ ). After the treatment, these values in the affected ears changed to  $22.88 \pm 36.55$  and  $1.85 \pm 5.3$  ( $P > 0.05$ ), respectively. In DPOAEs, the mean DP1 level and DP1 signal-to-noise ratio before the treatment were  $-19.2 \pm 9.49$  dB and  $-2.28 \pm 5.26$  respectively in the involved ears, and  $-0.8 \pm 7.9$  dB and  $8.51 \pm 5.69$  in normal ears respectively ( $P < 0.01$ ). After the treatment, these values in the affected ears changed as follows: DP1 level =  $-15.68 \pm 11.25$  dB and DP1 signal-to-noise ratio =  $0.41 \pm 5.29$  ( $P > 0.05$ ). Based on the definition of iSSNHL, we defined some "new" parameters in TEOAE and DPOAE (e.g. "correlation score", "emission strength score" and "emission strength score") by averaging the values of 3 to 4

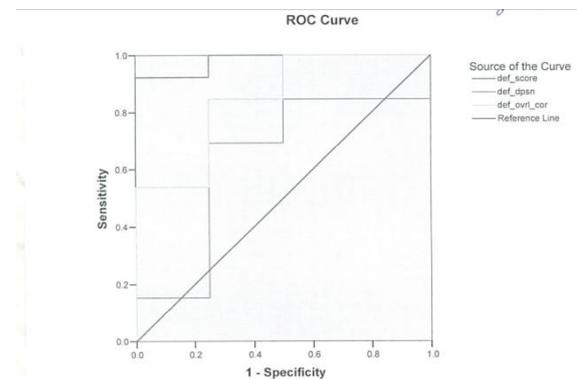
contiguous, involved frequency bands. Therefore, we found significant difference between the “correlation scores” before and after the treatment:  $6.52 \pm 18.19$  vs.  $21.67 \pm 37.8$ , ( $P < 0.034$ ). However, no significant difference was found in the “emission strength scores” before and after the treatment ( $P = 0.44$ ). We enrolled all the patients who responded to treatment (i.e. complete and partial response) in one group: response group ( $n = 20$  cases), and not responding patients in the other group ( $n = 6$ ). The difference between pre- and post-treatment parameters of the affected ears in the “response group” was significant for the correlation score ( $P < 0.007$ ) and the overall correlation ( $P < 0.031$ ), but there was no statistically significant difference in other parameters such as DP1 signal-to-noise ratio ( $P < 0.075$ ) or in the overall strength, the emission strength scores, and the DP1 levels. Further, none of these parameters showed any statistically significant changes in the “no response group”.

Using the Receiver Operating Characteristics Curve (ROC curve), we found some cut-off points in the pre-treatment “overall correlation” and “correlation scores” indicating abnormality (Fig.1).



**Fig 1:** Receiver operating characteristics curve (ROC curve) indicates “correlation score” below 11 as abnormal (sensitivity=87%, specificity 68%, ( $P < 0.001$ ).

Therefore, we can regard the pre-treatment “overall correlation” below 12, and “correlation scores” below 11 as abnormal (sensitivity=82.5% and 87%, specificity=60% and 68%, respectively; ( $P < 0.001$ ). Also, we found that the difference between pre- and post-treatment “correlation scores” and “overall correlation”, in contrast to “DP1 signal-to-noise”, may yield valuable measures for defining “response to treatment in sudden deafness” (Fig2).



**Fig 2:** Receiver operating characteristics curve (ROC curve) indicates that difference between pre-treatment “overall correlation” and its value during treatment course (def\_ovr\_score) as high as 1.5 (61.5% sensitivity and 75% specificity) as an index for “response” ( $P < 0.042$ ). For “correlation score” (def\_score), the difference as high as 3.1, will tell us about “response” with 92% sensitivity and 100% specificity ( $P < 0.005$ ).

In this regard, we can regard the difference between pre-treatment “overall correlation” and its value during the course of treatment as high as 1.5 (61.5% sensitivity and 75% specificity) as an index for “response” ( $P < 0.042$ ). Also, for “correlation scores”, a difference of up to 3.1 will tell us about “response” with 92% sensitivity and 100% specificity ( $P < 0.005$ ).

### Discussion

There are many studies in the literature about the site of lesion and differential diagnoses of idiopathic SSNHL. These studies demonstrate the possibility of developing a clinical method for

noninvasive differential diagnosis of hearing loss by adding the measurement of evoked OAEs growth functions over the range of frequencies to a standard audiometric evaluation(6,17). Also, there are many studies in the literature that demonstrate a prognostic role for OAEs in the ISSNHL (1,3,12,18,19), although there are some studies not agreeing with this (11,20), how are the changes in different parameters of OAEs, and, in principal, what parameters are more suitable, more stable, and more conforming to routine audiometries? In a previous study on ears with long-standing idiopathic sensorineural HL, evoked OAEs could not be recorded in ears with a hearing loss exceeding 35 dB at minimum hearing level of four audiometric frequencies: 500, 1,000, 2,000 and 4,000 Hz (4 MHL). In other words, although four MHLs were greater than 35 dB in most of the ears, evoked OAEs could be detected in about one-half of the ears with idiopathic sudden SNHL (18). Ishida et al published their study on eight SSNHL patients with good hearing improvement, and eight SSNHL patients with poor hearing improvement in an attempt to elucidate the behavior of ear fullness, tinnitus and OAEs in the recovery course of the disease. SSNHL patients with good hearing improvement tended to have OAE responses and the sensations of the ear fullness and tinnitus improved almost simultaneously with hearing level improvement. When hearing recovery was not full, OAEs did not reappear for these frequencies. Patients with poor hearing improvement tended to have absent OAEs and persistent ear fullness and tinnitus (1). Our study is in agreement with this study overall, although in this study the changes of parameters had not been elucidated, and only DPOAE had been performed. In 15 cases of idiopathic SD, Nakamura et al demonstrated that the amplitudes of TEOAEs and DPOAEs increased concurrently with the recovery of the

hearing threshold, and suggested that the function of outer hair cells had deteriorated when the hearing threshold was elevated and their activity recovered as hearing improved to nearly normal levels in cases with good outcome (13). Lalaki et al performed pure-tone audiometry (PTA) and TEOAE recordings in 30 SSNHL patients on the admission day, and at least three measures on the next eight days. The audiometric threshold improvement at each frequency was correlated with the TEOAE parameters on each measure (19). These two studies are in agreement with our results, and in fact, we had performed our study in a better way (e.g. with more cases and more OAE parameters compared with Nakamura's study, and performing both TE and DPOAEs compared with Lalaki's study). In another study, OAEs (both TE- and DPOAE) and PTA were performed on 26 ears of 25 patients suffering from SSNHL from one day to up to 505 days following the drop of hearing. In all the selected patients, one or both ears exhibited a systematic and significant recovery of pure tone threshold in at least one frequency. The correlation between OAE level and actual pure tone threshold was small but significant. Even smaller correlations were observed if the OAE level was related to former hearing loss, whereas the correlation improves if the OAE level is compared to the pure tone threshold measured in a later session. In many cases, the OAEs remain unchanged even if the hearing loss decreases. It was propounded in this study that the reliability of an individual prediction based on the OAE level combined with the threshold after SSNHL and the consequences for the physiologic mechanisms underlying SSNHL remain to be proved in further investigations. These results are in contrast with our current study results, although the study design is different from ours, and the definitions for response to treatment in these cases are questionable (20). In another

study, Zhang et al investigated the basic characters of DPOAEs in 60 ears of 30 cases with SSNHL before and after treatment. In the recovery course, the amplitude and threshold of DPOAE were improved with the restoration of auditory threshold, but the restoring rate (RR) of auditory threshold was higher, and they concluded that the amplitude and threshold of DPOAE were beyond that of the pure-tone behavioral threshold in SSNHL recovery course, which implies that DPOAE sensitively and directly reflects the function of the cochlea (21). This study is in agreement with ours; although our study contains both DP- and TEOAEs. Perhaps one novelty of our study is calculating "correlation scores" in these cases, which are average of correlations of consecutive affected frequencies. This parameter will be more sensitive in reflecting response to treatment. Also using ROC curves, we offered some cut-off points for defining "abnormality" and "response to treatment" in sudden deafness; however, the sample size in our study is not enough for sensitivity and specificity estimation, and these cut-off points may be used only for future studies with larger sample sizes.

### **Conclusion**

Evoked OAEs, especially TEOAEs, can be used as an objective, sensitive, and specific test in SSNHL, especially in difficult-to-test cases, for monitoring the results of the treatment. We suggest calculating "correlation scores" before and after the completion of treatment in all SSNHL cases.

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**Table 1:** Some demographic, audiometric, and otoacoustic (TEOAE) characteristics of 25 patients with sudden deafness

Case No.	Age (Years)	Sex	Involved ear	Type*	Severity	Response to treatment	Overall cor <sup>**</sup> . pre-treat (%)	Overall cor <sup>**</sup> . post-treat (%)	Cor. Score <sup>***</sup> pre-treat (%)	Cor. Score <sup>***</sup> post-treat (%)
1	15.00	female	left	high tone	profound	no response	8.0	-20.0	-12.6	-13.6
2	26.00	female	left	flat	severe	complete	33.0	97.0	6.6	96.0
3	19.00	male	left	flat	severe	no response	-17.0	-12.0	-19.33	-34.0
4	56.00	female	left	flat	moderate	complete	38.0	48.0	45.0	65.0
5	29.00	male	left	low tone	mild	complete	28.0	86.0	29.0	81.8
6	61.00	male	left	low tone	moderate	complete	-2.0	1.0	5.5	18.5
7	69.00	male	right	low tone	moderate	complete	-12.0	-5.0	10.5	11.5
8	53.00	female	left	low tone	moderate	complete	-6.0	91.0	0.5	89.5
9	30.00	male	left	flat	profound	no response	-3.0	-9.0	1.0	-15.6
10	36.00	female	right	flat	moderate	no response	10.0	4.0	18.2	-4.2
11	52.00	female	left	low tone	moderate	partial	86.0	81.0	11.5	31.0
12	50.00	male	left	low tone	moderate	partial	-9.0	7.0	-15.75	13.25
13	47.00	male	right	high tone	mild	complete	46.0	57.0	52.8	75.6
14	25.00	female	right	flat	severe	no response	22.0	5.0	9.8	8.4
15	19.00	male	left	flat	moderate	partial	25.0	16.0	-6.8	13.6
16	54.00	female	left	flat	profound	complete	18.0	18.0	13.4	29.6
17	44.00	male	right	flat	profound	partial	2.0	22.0	-3.6	3.6
18	49.00	male	left	flat	severe	complete	-18.0	77.0	-7.6	81.0
19	38.00	male	left	flat	moderate	no response	-3.0	-3.0	-8.4	1.6
20	50.00	male	right	high tone	profound	partial	21.0	-8.0	20.0	-18.75
21	24.00	male	right	flat	profound	partial	14.0	1.0	-0.8	-11.4
22	39.00	female	right	flat	moderate	complete	-1.0	2.0	-5.8	1.8
23	52.00	female	right	low tone	severe	complete	-11.0	5.0	-5.6	5.6
24	56.00	male	left	high tone	mild	no response	5.0	0.0	28.3	27.0
25	16.00	male	right	flat	profound	complete	0.0	11.0	-14.0	1.75

\*Audiogram pattern of sensorineural hearing loss

\*\*Cor. = Correlation (reproducibility)

\*\*\*Cor. Score = Correlation Score: this score is calculated by averaging correlations of "involved frequencies"

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