CONTRALATERAL SUPPRESSION OF DISTORTION PRODUCT OTO-ACOUSTIC EMISSIONS INPUT-OUTPUT FUNCTION IN INDIVIDUALS WITH AUDITORY NEUROPATHY SPECTRUM DISORDER

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This dissertation is submitted in part fulfilment for the degree of

Masters of Science (Audiology)

University of Mysore



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May, 2019

CERTIFICATE

This is to certify that this dissertation entitled "**Contralateral suppression of Distortion Product Oto-acoustic Emissions input-output function in individuals with Auditory Neuropathy Spectrum Disorder**" is a bonafide work submitted as a part for the fulfillment for the degree of Master of Science (Audiology) of the student Registration Number: 17AUD022. This has been carried out under the guidance of the faculty of this institute and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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CERTIFICATE

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This is to certify that this dissertation entitled "**Contralateral suppression of Distortion Product Oto-acoustic Emissions input-output function in individuals with Auditory Neuropathy Spectrum Disorder**" is the result of my own study under the guidance of Dr. Prashanth Prabhu P., Assistant Professor, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier to any other University for the award of any other Diploma or Degree.

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Dedicated to Ma!

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Abstract

Auditory Neuropathy Spectrum Disorder (ANSD) is an electrophysiological diagnosis that is reached when test results indicate the presence of evoked Otoacoustic Emissions (OAEs), cochlear microphonics, absent or abnormal Evoked Auditory Brainstem Responses, absent Stapedial reflex, and variable behavioral hearing thresholds. Even with varying peripheral thresholds, OAEs are usually preserved or of higher amplitude in them, mostly a result of impaired functioning of the efferent suppression. The aim of this study was to determine the contralateral suppression of Distortion Product Oto-acoustic Emissions input-output function in individuals with Auditory Neuropathy spectrum disorder. The method involved two groups of participants with 15 individuals each, normal hearing individuals as the control group and individuals with ANSD as the clinical group. DPOAE I/O function was carried out with and without noise for both the groups to estimate the effect of noise and to compare the slope and area of DPOAE input output function. Results revealed that there was no suppression effect seen in individuals with ANSD owning to fact that there are some efferent system dysfunctions similar to the previous literature. In addition, it was found that there was no significant effect of frequency on the slope and area of DPOAE input output function.

Chapter 1

Introduction

ANSD is an electrophysiological diagnosis that is reached when test results indicate the presence of Evoked Otoacoustic Emissions (OAE's), cochlear microphonics, absent or abnormal Evoked Auditory Brainstem Responses, absent stapedial reflex and variable behavioral hearing thresholds (Starr et al., 1996). In India, the prevalence of ANSD is 5.3% of the pediatric population in tertiary care hospitals, and 14% in those diagnosed with severe to profound hearing loss (Mittal et al., 2012). The prevalence of ANSD in children with hearing impairment going to school is reported to be about 2.47 % (Bhat, Kumar, & Sinha, 2007).

Even though a lot of research has been dedicated to finding the specific etiology of ANSD, the results remain more or less inconclusive. It has been reported by Manchaiah et al., (2011) that the largest proportion of ANSD is due to genetic factors that can be syndromic, mitochondrial related or non-syndromic. Mostly, ANSD is attributed to dysfunction at the inner hair cells, the junction of the spiral ganglion cells and/or the auditory nerve (Amatuzzi, Liberman, & Northrop, 2011; Nachman, 2012; Rohr, 2011). But, since OAEs are usually present and robust in these individuals despite the variation in etiologies, otoacoustic emissions (OAEs) are routinely used clinically to assess cases of ANSD, having proven to be a valuable diagnostic tool (Berlin et al., 2010).

OAEs are usually preserved or of higher amplitude in them, mostly a result of the impaired functioning of the efferent suppression (Berlin et al., 2003; Teagle et al., 2013). Studies have shown that Otoacoustic emissions (OAEs) are routinely used clinically to assess cases of ANSD, having proven to be a valuable diagnostic tool (Berlin et al.,

2010). Other cochlear tests like cochlear microphonics can also be used to diagnose ANSD (Berlin et al., 2003, Starr et al., 2000). A long ringing cochlear microphonics is usually observed in ANSD. Also OAEs are a pre-neural phenomenon that occurs at the level of the cochlea. Distortion Product OAEs (DPOAEs) are dependent on the level of presentation of these tones, and an I/O or input-output function can be obtained by keeping the stimulus frequency and frequency ratio constant. Through this, audiometric thresholds can be estimated in clients who may be incapable of responding to behavioral tests. The relation between these two measures becomes stronger depending on the frequency of the signal [reviewed in (Rasetshwane, Neely, Kopun, & Gorga, 2013; Rasetshwane et al., 2015)] and when repeated estimates are combined in a multivariate analysis. The input-output function slope obtained for different input levels is directly reliant on cochlear health and therefore gives a picture of the supra-threshold nonlinear characteristics of the cochlea.

1.1 Need for the study

Traditionally, cochlear functioning is reported to be normal in those suffering from ANSD. However, there are studies which report that the properties of OAE are different in individuals with ANSD compared to normal controls. A study by Kumar, Avilala, Mohan, and Barman (2012), compared Spontaneous Evoked Otoacoustic Emissions (SOAEs) and found that SOAEs show a different spectral distribution (<1.5 kHz) in the clinical group versus the normal control group (>1.5 kHz), along with greater numbers of multiple SOAEs. These changes were attributed to subtle impairment in the medial olivocochlear system. Another study by Narne, Prabhu and Chatni (2014) reported higher amplitude TEOAEs, with slightly shorter latencies for lower frequency signals than the normal control group in the clinical group, which were also thought to be caused by damage to the efferent system.

Thus, the above studies suggest that there could be a subtle cochlear impairment in individuals with ANSD. So, suppression of OAE in ANSD is usually absent, or suppression phenomenon is not observed. There are limited studies to explore the differences in cochlear non-linearity if any in individuals with ANSD. DPOAE Inputoutput growth function of the slope and areas of each frequency of normals (control group) were compared with individuals with ANSD (Karnad & Prabhu, 2018). They reported significant difference (p<0.05) in slope and area at lower frequencies compared with high frequencies (Karnad & Prabhu, 2018).

Previous studies have attempted to determine the effect of contralateral noise on DPOAE I/P function in neonates. Significant DPOAE amplitude suppression effects were observed for neonates while the DPOAE I/O slope was also affected (Campos, Hatzopoulos, Kochanek, Sliwa, Skarzynski & Carvallo, 2011). However, there aren't any prior studies on recordings of DPOAE input-output growth function with broadband noise on those suffering from ANSD. Thus, the present study attempts to investigate the differences in DPOAE input-output function (regarding slope and area) comparing individuals with normal hearing and ANSD, with and without noise to see the effect of contralateral supression effects on input-output growth function. It is also attempted to study these differences, if any, across different frequencies. Hence, the study would help in exploring and understanding the pathophysiology of ANSD and the effect of the efferent system in suppression of OAE's for ANSD.

1.2 Aim of the study

Evaluation of distortion product otoacoustic emissions input-output function in those with auditory neuropathy spectrum disorder in the presence and absence of broadband noise at different frequencies.

1.3 Objectives of the study

- To compare the slope and area of DPOAE input/output (DP I/O) in individuals with normal hearing and ANSD with and without contralateral broadband noise
- To study the effect of frequency on DP I/O slope and area of DP I/O with and without noise in both the groups.

Chapter 2

Review of literature

2.1 Auditory Neuropathy Spectrum Disorder

ANSD is a heterogeneous disorder, and its causational factors vary from congenital to acquired cases. In the early 1970's, symptoms of ANSD were reported by Hinchcliffe, Osuntokun, and Adeuja. However, it wasn't until the early 1990's the term was used by a group of authors, Starr, Picton, Sininger, Hood, and Berlin in 1996. Since the OAEs were present, the cochlea was normal, and the disorder was assumed to be of the functioning of the auditory nerve due to a "neuropathy" which could have occurred on its own or due to any other pathological condition.

It has been referred with various names since the initial establishment of this disorder, however the terms used to diagnose as being varied widely, as some preferred the term Dys-synchrony ((Berlin et al., 2002), while some used Neuropathy. Hence the standard term for diagnosis of this disorder varied until 2008, where it was established that it would be referred to as Auditory Neuropathy Spectrum Disorder in a panel in Italy for the development of guidelines for the management and identification of infants with Auditory Neuropathy. It was referred as spectrum due to its wide variations in general occurrence.

2.2 Prevalence of Auditory Neuropathy Spectrum Disorder

In hearing impaired NICU children, the prevalence rate increased up to 40% (Rea & Gibson, 2003). In India alone, 2.27% (Bhat et al., 2007) were diagnosed with ANSD who were school going children from grade one to eight. In terms of the occurrence of

the disorder with respect to gender, approximately equal distribution was found between males and females, the prevalence being 55% and 45% respectively (Sininger & Starr, 2001). But a study by Kumar and Jayaram 2006, reported that female to male ratio being 2:1. In a pediatric population in tertiary care hospitals the prevalence is reported to be 5.3%, and as high as 14% in those diagnosed with severe to profound hearing loss (Mittal et al., 2012). Such findings were obtained in another study by Vignesh, Jaya, and Muraleedharan (2016), where 5.06 % (N = 11) were diagnosed with ANSD from a population of 217 children with sensory neural hearing loss.

2.3 Signs and symptoms

Besides poorer speech understanding, individuals with ANSD also have difficulty processing information restricted to one modality (auditory). When more than one modality is used, say auditory and visual modality their understanding of the context seems to improve. The speech comprehension significantly improves if speech strategies are used either by the listener or the speaker. A large portion of individuals diagnosed with ANSD show vestibular symptoms, however it not always necessarily present in all of them. Some individuals have even the peripheral system involved, although even a normal audiogram configuration is observed in ANSD.

2.5 Oto Acoustic Emissions

Oto-acoustic emissions (OAE) are an objective measure to evaluate the functioning of outer hair cells of the cochlea. Since cochlear functioning is normal, individuals with ANSD will have the presence of OAE. In a study conducted by Norton and Widen (1990) in patients with ANSD, they found robust OAEs but may not be abnormally large as their patient group was predominantly children. This could be due to efferent auditory system damage (Sininger & Starr, 2001).

The disappearance of OAEs over a period of time is also observed by Starr et al. (2000) which could be attributed to middle ear disease or the use of amplification devices. Similar findings were found in Tallat's study where the disappearance of OAE was claimed due to the spread of the pathology to the outer hair cells. According to a study done by Narne, Prabhu and Chatni (2014) on TEOAE's in ANSD, higher amplitude TEOAEs with slightly shorter latencies for lower frequency signals than the normal control group was found in the clinical group, which were also thought to be caused by damage to the efferent system.

2.6 Efferent Suppression of OAE's

There are always more LOC fibres than MOC fibres in humans. Furthermore, LOC fibers predominantly project to the same side (3:1) and MOC fibers predominantly project to the opposite side (2:1). Suppression can be recorded using three ways-Ipsilateral Suppression, Contralateral Suppression, and Binaural Suppression. The ipsilateral effects may be due to both OCB and intra-cochlear mechanical processes. The contralateral effect has been attributed to the activation of OCB. Contralateral suppression of OAEs was first discovered by Collet et al 1990. Individuals with ANSD usually show no or minimal suppression of TEOAEs for binaural, ipsilateral, and contralateral suppressor stimuli. Similar results were found in TEOAE's for individuals with ANSD according to the study done by Hood, Berlin, Boredelon, Rose in 2003. James (2010) reported that two out of five neonates with a confirmed diagnosis of ANSD showed absent DPOAE suppression effect.

Considering the findings mentioned above, studies on the I/O functions in individuals with ANSD becomes essential. Also due to limited literature, investigation and exploration with respect to contralateral suppression of I/O function in individuals with are essential. It would help in identifying subtle pathology at the cochlear level in these individuals, and it may help to better understand the efferent auditory system functioning in them.

Chapter 3

Methods

3.1 Participants

A total of 30 participants (15 individuals in each group) were considered for the study. The first group consisted of individuals with ANSD aging from 13 to 50 years, (mean age of 24 years, SD. 7.94). In addition, the second group consisted of a comparison group of normal hearing young adults aging from 13-50 (mean age of 21, SD. 2.07 years) were considered.

3.2 Participant Selection criteria

3.2.1 Clinical Group

Clinical group participants were selected based on the diagnostic criteria suggested by Berlin et al. (2003) and Starr et al. (2000) as mentioned below:

- Presence of Oto-acoustic emissions
- Poor morphology or absent auditory brainstem response
- Passed the neurological examination done by the clinical neurologist.
- They had normal otologic findings on examination of the ear canal by an experienced otologist
- They had normal immittance findings

3.2.2 Control Group

15 individuals with normal hearing sensitivity with following criteria were selected

• Individuals who had a pure tone average of less than 15 dB HL.

- None of the participants reported of having any middle ear disorder/pathology.
- No history of otological complaints, noise exposure, ototoxic medications, diabetes/hypertension.

3.2 Procedure

Behavioral thresholds were obtained using a modified version of Hughson-Westlake procedure, where the intensity was reduced by 10 dB whenever the subjects heard the sound and an increase of 5 dB when they stopped hearing the sound. This was repeated until a threshold (intensity at which the subject heard the sound two out of three times) was obtained. Both air conduction and bone conductions thresholds were obtained in this way.

Spondee words (to find speech recognition threshold), and phonemically balanced words (to find speech discrimination score) were used to perform speech audiometry. Middle ear function analysis was done using a calibrated tympanometer (GSI Tympstar V 2.0). Then Auditory Brainstem Evaluation was done using Intelligent Hearing System (IHS) or Biologic EP in which the stimulus was presented through an ER-3A insert receiver using the standard test protocol.

3.4.1 Obtaining the DP I/O function

OAE Measurements through a calibrated Otodynamics ILO V6 Echoport system was done. Distortion Product Otoacoustic Emissions were obtained for two tones, F1 and F2 (primaries), their ratio being 1.22, with intensities of 65 dB SPL and 55 dB SPL (L1 and L2) respectively. The input-output function was then obtained for tones of frequencies 1 kHz, 1.5 kHz, 2 kHz, 3 kHz, 4 kHz, and 6 kHz, holding the frequency ratio between the test tones constant at 1.22, for different intensities. An average of three responses was taken for each individual response. The intensities were set according to

the stimulus paradigm found to be optimal for clinical testing (Kummer, Janssen, Hulin, & Arnold, 2000; (Janssen, Niedermeyer, & Arnold, 2006) where primary tone stimulus is L1=(0.4*L2) +39dB SPL, as the L2 decreases in 5 dB steps. Once the OAE's inputoutput functions were recorded in the absence of noise, the contralateral broadband noise of 40 dB SPL was introduced (using an insert receiver from an audiometer) without altering the placement of the OAE tip and DPOAE input output growth function was recorded again for each specific frequency alternatively.

3.5 Analyses

The comparison of the DPOAE Input-Output function was done using two variables, the first being slope of the I/O function and the second being the area under the curve. The slope was calculated using the linear trend model. The DPOAE I/O data were fitted with linear functions for the stimulus range from 75 to 45 dB SPL. Once a linear fit was obtained, the slope was estimated at 2 points of the x coordinate equal with x2=75 dB SPL and x1=45 dB SPL. Given the corresponding points of the DPOAE amplitude as y2 and y1, the slope of the fitted linear function was defined as: b = (y2-y1)/(x2-x1).

The area under the curve was to be determined as the difference between the noise floor and the DP amplitude at all the 5 dB stimulus level steps from 65 dB SPL to 35 dB SPL. If the responses were below the noise floor, they were excluded from further analysis. The cumulative amplitude of the DP responses above the noise floor was multiplied by 5 and reported in $dBSPL^2$ (area²). The square root of area² (i.e. area) was used for the analyses. This procedure for calculating the area was proposed by Gates and Rubel (2002).

3.6 Statistical Analyses

The data were normally distributed based on tests of normality, hence parametric tests were chosen for further analysis. Descriptive statistics for determining Mean, Median and Standard Deviation were done for both Area and Slope measurements, for each test of the six test frequencies with and without noise. Dependent t-tests, independent t-tests and repeated measures ANOVA were used as inferential statistical measures.

Chapter 4

Results and Discussion

The results of this study are discussed under the following subheadings -

4.1 Comparison of slope and area of DPOAE input-output function with and without noise for both the groups separately.

4.2 Comparison of slope and area of DPOAE input-output function at each frequency with and without contralateral noise between the two groups.

4.3 Comparison of slope and area of DPOAE input-output function across frequencies in both the groups with and without noise.

4.1 Comparison of slope and area of DPOAE input-output function with and without noise for both the groups separately.

Descriptive statistics were performed for the slope of DPOAE I/O function. Mean Standard Deviation (SD) was determined at all the six test frequencies for both normal group (Figure 4.1) and ANSD group (Figure 4.2) in two conditions - with contralateral noise and no noise condition. The data showed that the slope values were reduced with noise in individuals with normal hearing sensitivity. However, the slope values were similar with and without noise in individuals with ANSD.

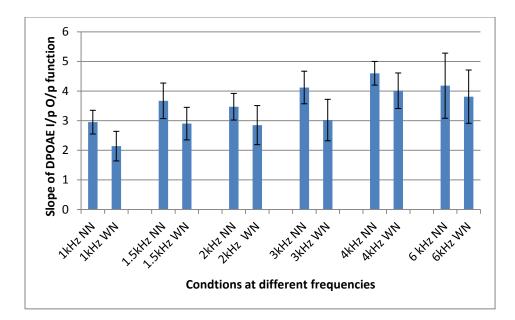


Figure 4.1 Mean and standard deviation of DPOAE I/p O/p function of the slope determined with and without noise at six test frequencies in individuals with normal hearing group (NN = No noise, WN = With noise).

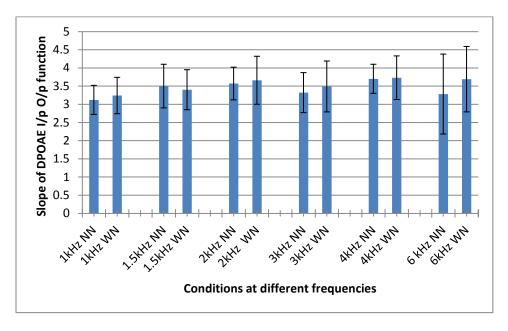


Figure 4.2 Mean and standard deviation of DPOAE I/p O/p function of the slope determined with and without noise at six test frequencies in individuals with ANSD group (NN = No noise, WN = With noise)..

Descriptive statistics were performed for the area of DPOAE I/O function. Mean Standard Deviation (SD) was determined at all the six test frequencies for both normal group (Figure 4.3) and ANSD group (Figure 4.4) in two conditions - with contralateral noise and no noise condition. The data showed that the area values increased with noise in individuals with normal hearing sensitivity. However, the slope values were similar with and without noise in individuals with ANSD.

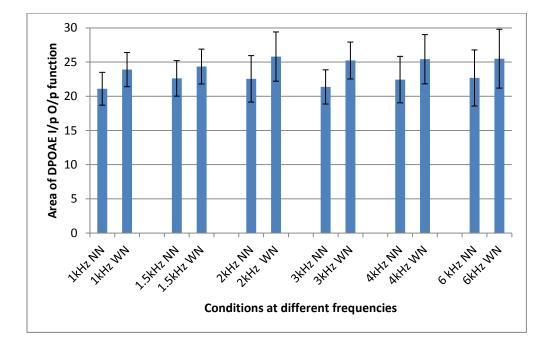


Figure 4.3 Mean and standard deviation of DPOAE I/p O/p function of the area determined with and without noise at six test frequencies in individuals with normal hearing group (NN = No noise, WN = With noise).

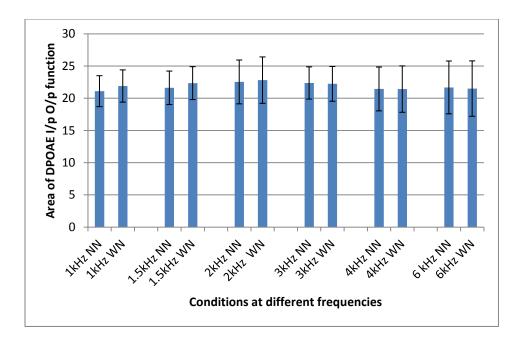


Figure 4.4 Mean and standard deviation of DPOAE I/p O/p function of the area determined with and without noise at six test frequencies in individuals with ANSD group (NN = No noise, WN = With noise).

Shapiro Wilks test of normality was administered, it was found to be normally distributed (p>0.05) and hence appropriate parametric tests were chosen for statistical analyses. Dependent t-tests were performed to compare the data obtained in two conditions with noise and without noise for both the groups. The results of dependent t-test for slope of DPOAE I/p O/p function in both the groups are shown in Table 4.1. The results of dependent t-test for area of DPOAE I/p O/p function in both the groups are shown in Table 4.2. The results of the dependent t-test showed that the slope of DPOAE I/p O/p function was significantly lower (p<0.05) and area of DPOAE I/p O/p function was significantly higher with contralateral noise in individuals with normal hearing. In addition, it was also found that there was no significant difference in slope and area of DPOAE I/p O/p function with and without noise in individuals with ANSD.

Table 4.1

The results of dependent t-test for the slope of DPOAE I/p O/p function in both the

Group	Frequencies	t value	Degrees of freedom	p value
Normal	1 kHz	3.08	29	.008
hearing	1.5 kHz	4.79	29	.041
individuals	2 kHz	3.24	29	.042
	3 kHz	4.37	29	.031
	4 kHz	4.43	29	.012
	6 kHz	3.61	29	.006
	1 kHz	0.61	29	.544
	1.5 kHz	1.30	29	.203
Individuals	2 kHz	0.20	29	.836
with ANSD	3 kHz	1.21	29	.233
	4 kHz	0.36	29	.720
	6 kHz	1.41	29	.167

Table 4.2

The results of dependent t-test for the area of DPOAE I/p O/p function in both the groups

Group	Frequencies	t value	Degrees of freedom	p value
Normal	1 kHz	2.98	29	.031
hearing	1.5 kHz	3.62	29	.002
individuals	2 kHz	3.45	29	.003
	3 kHz	2.90	29	.037
	4 kHz	3.33	29	.021
	6 kHz	4.65	29	.001
	1 kHz	0.84	29	.405
	1.5 kHz	0.52	29	.604
Individuals	2 kHz	0.79	29	.431
with ANSD	3 kHz	2.00	29	.054
	4 kHz	1.33	29	.193
	6 kHz	1.30	29	.203

4.2 Comparison of slope and area of DPOAE input-output function at each frequency with and without contralateral noise between the two groups.

Independent sample t-tests were administered to compare the slope and area of DPOAE I/p O/p function between the two groups at all the test frequencies. The results of independent t-tests for slope of DPOAE I/p O/p function between the two groups are shown in Table 4.3. The results of independent t-tests for area of DPOAE I/p O/p function between the two groups are shown in Table 4.4. The results showed that there was no significant difference (p>0.05) in slope and area of DPOAE I/p O/p function between the two groups in without noise conditions at all the test frequencies. However, there was a significant difference (p<0.05) in the slope and the area of DPOAE I/p O/p function between the two groups with contralateral noise in all the test frequencies.

Table 4.3

Conditions	Frequencies	t value	Degrees of freedom	p value
Without Noise	1 kHz	1.12	58	.267
	1.5 kHz	0.07	58	.940
	2 kHz	0.75	58	.452
	3 kHz	1.41	58	.162
	4 kHz	0.98	58	.331
	6 kHz	0.90	58	.369
With Noise	1 kHz	2.36	58	.040
	1.5 kHz	2.59	58	.034
	2 kHz	2.87	58	.021
	3 kHz	3.26	58	.009
	4 kHz	2.95	58	.019
	6 kHz	3.23	58	.004

The results of independent t-test for slope of DPOAE I/p O/p function between the groups

Table 4.4

Conditions	Frequencies	t value	Degrees of freedom	p value
Without Noise	1 kHz	1.06	58	.143
	1.5 kHz	1.00	58	.150
	2 kHz	1.49	58	.142
	3 kHz	0.19	58	.844
	4 kHz	1.11	58	.272
	6 kHz	0.26	58	.796
With Noise	1 kHz	2.31	58	.041
	1.5 kHz	2.19	58	.047
	2 kHz	2.14	58	.048
	3 kHz	2.91	58	.031
	4 kHz	3.52	58	.001
	6 kHz	2.50	58	.015

The results of independent t-test for area of DPOAE I/p O/p function between the groups

4.3 Comparison of slope and area of DPOAE input-output function across frequencies in both the groups with and without noise.

Repeated measures ANOVA were administered to determine the effect of frequency on slope and area values for DPOAE I/O function in both the groups with and without noise. The results of repeated measures ANOVA for slope and area of DPOAE I/O function are shown in table 4.5 and 4.6 respectively. The results revealed that there was no significant effect (p>0.05) of frequency on both slope and area of DPOAE I/O function in both the groups with and without noise.

Table 4.5

The results of Repeated measures ANOVA for slope of DPOAE I/p O/p function in both

Groups	Conditions	F value	Degrees of freedom	p value
Normal	With noise	0.56	5, 28	0.59
Hearing	Without noise	2.21	5, 28	0.65
ANSD	With noise	1.28	5, 28	0.63
	Without noise	2.31	5, 28	0.54

the groups with and without noise

Table 4.6

The results of Repeated measures ANOVA for area of DPOAE I/p O/p function in both

the groups with and without noise

Groups	Conditions	F value	Degrees of freedom	p value
Normal	With noise	0.58	5, 28	0.59
Hearing	Without noise	0.89	5, 28	0.65
ANSD	With noise	2.3	5, 28	0.63
	Without noise	1.82	5, 28	0.54

The results of the study showed that there was no significant difference in slope and area of DPOAE Input-output function with and without noise in individuals with ANSD. In addition, it was found that there was no significant difference across frequency in individuals with normal hearing and ANSD with and without noise. Thus, null hypothesis 1 and 2 are partially accepted and null hypothesis 3 is accepted.

Norton and Widen (1990) reported robust OAEs in individuals with ANSD. The amplitude of TEOAEs in individuals with ANSD is abnormally higher compared to individuals with normal hearing (Hood & Berlin 2001). Also, Kumar, Avilala, Mohan and Barman (2012), found that SOAEs showed a different spectral distribution (<1.5

kHz) in the individuals with ANSD group versus the normal hearing individuals control group (>1.5 kHz), along with greater numbers of multiple SOAEs. All of the above attributions were due to the subtle changes in the functioning of the cochlea and its medial olivocochlear system. Another study by Narne, Prabhu and Chatni (2014) reported higher amplitude TEOAEs, with slightly shorter latencies for lower frequency signals than the normal control group in the clinical group, which were also thought to be caused by damage to the efferent system.

It is well reported in the literature that individuals with ANSD showed no or minimal suppression of TEOAEs for binaural, ipsilateral, and contralateral suppressor stimuli (Starr et al, 2001). There are previous studies on contralateral suppression of distortion product otoacoustic emissions in individuals with ANSD (Hood, Berlin, Boredelon, Rose, 2003; Abdala, et al 2000) which suggest an absent suppression due to damage to the efferent auditory nervous system. Thus, in the present study, the absence of suppression in DPOAE input output function could be attributed to abnormal efferent auditory system functioning. Thus, the result of the present study supports efferent auditory system damage in individuals with ANSD and helps to understand the pathophysiological changes in the efferent system of individuals with ANSD.

Chapter 5

Summary and Conclusions

Anatomically and functionally, its damages limits, to the external auditory nerve and inner hair cells sparing the outer hair functioning to be adequate. Hence a test battery of OAE, ABR, acoustic reflexes along with the routine pure tone and speech audiometry becomes vital. Their characteristics vary from having normal to a profound degree of low-frequency hearing loss, very poor speech perception skills, absent acoustical reflexes with abnormal or absent ABR's and normal OAE's to low-level stimuli.

Since the working of outer hair cells is rendered to be normal, the individuals with ANSD are expected to have OAE's and cochlear microphonics. OAEs are usually present and robust in these individuals despite the variation in etiologies, Otoacoustic emissions (OAEs) are routinely used clinically to assess cases of ANSD, having proven to be a valuable diagnostic tool. The DPOAE slope is the growth rate of the DPOAE response, with a lesser amplitude as the stimulus intensity increases (45peSPL - 75peSPL) and it is at the higher intensities that cochlea, behaves non linear compressing the higher intensity sounds. So at higher intensities, the cochlea's supra threshold nonlinearity and functioning can be monitored using DPOAE slope values for varying intensities. (Campos et al., 2011) Also, cochlea's efferent pathway working can be assessed using contralateral suppression with a masker (broadband noise) of a particular intensity to the opposite ear. There is a dearth of literature on the effect of contralateral noise on DPOAE Input-output function especially in individuals with ANSD.

In the present study population group (15 clinically confirmed diagnosis of individuals with ANSD with 15 normal hearing individuals) were compared to evaluate

the efferent auditory system functioning using the measures of slope, area of DPOAE input output function in presence of contralateral noise. The DPOAE's slope was calculated for various frequencies 1 kHz, 1.5 kHz, 2 kHz, 3 kHz, 4 kHz, and 6 kHz with the ratio of 1.2 being constant between F1 and F2 and the input intensities were set high, from 45 dB pe SPL to 75 dB pe SPL which was varied in 5 dB steps. The slope was calculated using the linear model and the area was calculated using the method given by Gates and Rubel (2002). The effect of suppression was estimated using a broadband noise signal at 50 dB as a masker in the contralateral ear and magnitude of the amplitude of OAE's with and without noise were estimated.

Descriptive statistics for determining the mean, standard deviation and median were obtained for both area and slope measurements, for all the six test frequencies. The data were normally distributed (p>0.05), hence parametric tests were chosen for further analysis. A dependent t-test was administered to compare the effect in contralateral noise present and contralateral noise absent condition in both groups separately. Independent t-test was administered for comparing the slope and area between the two groups at all the frequencies (with and without noise). Finally, repeated measures of ANOVA were administered to determine the frequency effect for both slope and area (with and without contralateral noise).

The results of the study showed that there was no significant difference in slope and area of DPOAE Input-output function with and without noise in individuals with ANSD. In addition, it was found that there was no significant difference across frequency in individuals with normal hearing and ANSD with and without noise. Suppression of OAE in individuals with ANSD is usually absent, or suppression phenomenon is not observed. Thus, in the present study, the absence of suppression in DPOAE input-output function could be attributed to abnormal efferent auditory system functioning.

5.1 Advantages of the study

- This study is first of its kind to test for contralateral suppression in DPOAE I/O function.
- Provides more insight into the efferent auditory system functioning in those suffering from ANSD.

5.2 Limitations of the Study

- Further studies could be done on a larger sample, as there is great heterogeneity.
- The findings of the present study are valid only for the adult population and cannot be generalized to children with ANSD.

5.3 Future Directions

- The study can be done on a larger population.
- The study could be carried out in children with ANSD.

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