DISTORTION PRODUCT OTOACOUSTIC EMISSION FINDINGS IN CONDUCTIVE HEARING LOSS

Reg.No.M9821

Independent Project as a part fulfilment of first year M. Sc., (Speech and Hearing), submitted to the University of Mysore, Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MYSORE 570 006

MAY 1999

To
anna
&
amma,

वागर्थाविव संपृक्तौ वागर्थ प्रतिपत्तये ।
जगतः पितरौ वंदे पार्वतीपरमेश्वरौ ।।

अखंडमण्डलाकारम्
व्याप्तं येन चराचरम् ।
तत्पदं दर्शितं येन
तस्मै श्रीगुरवे नमः ।।

(Salutations to "SRI GURU", the teacher, who is omnipresent and source of knowledge and philosophies).

CERTIFICATE

This is to certify that this Independent Project entitled : **DISTORTION PRODUCT OTOACOUSTIC EMISSION FINDINGS IN CONDUCTIVE HEARING LOSS** is the bonafide work in part fulfilment for the degree of Master of science (Speech and Hearing) of the student with Register No.M9821

Mysore
May, 1999

Dr. (Miss) S. Nikam
Director
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CERTIFICATE

This is to certify that this Independent Project entitled : **DISTORTION PRODUCT OTOACOUSTIC EMISSION FINDINGS IN CONDUCTIVE HEARING LOSS** has been prepared under my supervision and guidance.

Mysore
May, 1999

Animesh Barman
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DECLARATION

This Independent Project entitled : **DISTORTION PRODUCT OTOACOUSTIC EMISSION FINDINGS IN CONDUCTIVE HEARING LOSS** is the result of my own study under the guidance of Mr.Animesh Barman, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other diploma or degree.

Mysore
May:1999 Reg.No.M9821

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Shrilatha, Mamtha, Hemi, Kirana - my cousins, your affection and love makes my life more pleasant and I always look forward for the times to spend with you for sharing my sorrows and joys.

Rajalakshmi Akka, thanks a lot for neat typing and giving a shape to this project.

Last, but not the least my dearest and nearest **friends**

I am running short of words to express my feelings and love towards you. It would be a meek attempt of mine to express ray gratitude and thank you through this small chit of paper, which does not even express **a** smallest fraction of my feelings. *Thanks* for being my friends and letting me to be there for you.

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INTRODUCTION

Ear has been recognized as the organ of hearing from past several centuries which receives the sounds and enables the individuals to have an effective communication and deal with external environment. But the concept of cochlea as a passive organ that converts the mechanical vibrations into neural discharges has been altered or disregarded with the discovery of OAEs.

OAEs are sounds generated within the normal cochlea, either spontaneously or in response to acoustic stimulation. These were first measured by Kemp in 1978 from the Institute of Laryngology and Otology (ILO). Although these sounds are of very low intensity, they are loud enough and can be detected at the eardrum by a miniaturized sensitive microphone.

OAEs are thought to reflect the activity of active biological mechanisms within the cochlea. The results of a considerable number of experiments and theoretical studies of OAE carried out since their discovery indicate that emissions are produced as a normal by product of micro mechanical actions of the cochlear amplifier which is situated in the outer hair cells.

OAEs can be of two types :

(i) ***Spontaneous Otoacoustic Emission (SOAE's)*** *:* Emissions which are more or less continuous narrow band signals emitted in the absence of external acoustic stimulation. These occur in about 50% of all ears with normal hearing and generally concentrated in the region of 1-3 kHz. Recorded by obtaining a satisfactory seal of sensitive miniature microphone into ear canal.

(ii) ***Evoked Otoacoustic Emissions (EOAE's)*** : Emissions in response to the presentation of acoustic stimulation to the ear. A sound generating source must also be sealed into the ear canal along with microphone.

Evoked OAE's may be of following types :

(a) ***Transient Evoked Otoacoustic Emission (TEOAE)*** *:* Elicited by transient acoustic stimulus such as click or tone burst. This is seen in about 98% of ears with normal hearing.

b) ***Stimulus Frequency Otoacoustic Emission (SFOAE) :*** **Elicited** by a single continuous sweep frequency puretone. The emission resembles the puretone in terms of frequency. Because of complexity in generation and lack of temporal and spectral separation it is not used frequently.

c) ***Distortion Product Otoacoustic Emission (DPOAE)*** - Elicited by two continuous puretones (f1 and f2) called as primaries, separated in frequency by a prescribed difference. In humans the most prominent distortion is found to be at 2f1-f2 and required cochlear region can be explored by selecting f1 and f2. This is seen in 100% of ears with normal hearing.

Acoustic distortion products (ADP) are technologically the easiest types of emissions to measure, being relatively artifact free. The threshold (the lowest level of the primaries at which ADP can be distinguished from the noise floor) as the magnitude of ADP depends on the frequency ratio (f1/f2) and relative levels (L1/L2) of two puretones.

Acoustic distortion products can be measured commonly using two methods :

(1) **DPOAE Audiogram (DP Gram)** :

Frequency pattern of a ear's ability to generate acoustic distortion product is established by measuring emission amplitude as a function of geometric mean of two primary frequencies.

(2) **Input-Output Function** :

Input-output function is determined at geometric mean frequencies (that are usually related to the conventional audiogram) by varying primary tone level in 5 or 10 dB steps for a range of dB SPL and DP threshold is obtained (The minimum intensity level of the primary frequencies at which the amplitude of emission is above certain level of noise floor).

This input/output function measures several other features as maximum amplitude, dynamic range, slope which relates the rate at which the emission grows as a function of increased primary tone level. DPOAE has a wide dynamic range of 40 dB SPL which is one of the major advantage. Hence from clinical perspective, DPOAE audiogram, appears to reflect the frequency configuration of the standard audiogram (Martin et al. 1990).

Acoustic distortion products are generally found in an ear with the behavioural threshold up to 50-55 dB HL (Harris, 1990).

Advantages of DPOAE Measurement:

Test is objective in nature, and does not require patient co-operation for it to be administered.

It does not require cumbersome procedures such as electrode placement and measurement of impedance at the electrodes.

It does not require air tight seal.

Requires less time when compared to BSERA testing but longer than that required for tympanometric measurements.

It is highly frequency specific.

Compared to other evoked emissions reasonably wide dynamic range of DPOAE in terms of growth response amplitude as a function of stimulus level is found and permits a complete evaluation of cochlear function at both threshold and suprathreshold level.

Clinical Application of DPOAEs :

As the origin of OAE is believed to be the outer hair cells **(Davis,** 1983; Zwicker, 1984) they provide an indirect evidence of the hair cell physiology, permitting fine analysis of inner ear properties.

Various studies have been carried out which ascertains the applicability of DPOAEs clinically in screening the hearing impairment as well as to estimate the hearing acuity. DPOAEs have been used in infant screening for sensori-neural hearing impaired ears (Brass and Kemp, 1994; Lafreniere, et al. 1991). DPOAEs are used to detect the cochlear status which is damaged due to various conditions as ototoxicity, noise induced hearing loss, meniere's disease and other various cochlear pathologies (Lonsbury-Martin and Martin, 1990; Martin, 1990; Kimberley, et al. 1994a; Kimberley, et al. 1994b). Hearing acuity is also estimated through DPOAE's in sensorineural loss individual (Kimberley and Nelson, 1989; Harris, 1990; Smurzynski, et al. 1990; Gorga et al. 1993a). It is used in monitoring the cochlear status regularly (Probst and Harris, 1997; Zorowka, et al. 1993). It gives information solely on the sensory elements of the cochlea which cannot be tapped by any other testing as ABR (Smurzynski et al. 1990; Kemp et al. 1986; Leonard et al. 1990). They may indicate hearing difficulties which may go undetected by conventional audiometry (Gaskil and Brown, 1990). It is used for differential diagnosis of the retrocochlear disorders (Durrant, 1992; Telischi, et al. 1995; Cane et al., 1994).

As the DPOAEs are recorded in the ear canal, effective reverse transmission is needed to transmit the OAEs from the inner ear to the ear canal (Margolis and Trine, 1997). Hence, middle ear conditions directly influence OAE measurements. Although the middle ear can transmit sound bidirectionally, the forward transmission characteristics and backward transmission characteristics are different. Various middle ear disorders affect the transmission differently.

Hence DPOAEs are found to be significantly affected in conductive hearing loss individuals, depending upon the pathology such as middle ear negative pressure (Owens, et al. 1992), Otitis media (Owens,et al. 1992; Margolis and Trine, 1997), middle ear effusion (Chang et al. 1993; Amedee, 1995; Bray, 1989), Eardrum abnormalities (Weiderhold, 1990; Kemp, 1980; Margolis and Trine, 1997) and Otosclerosis (Rossi, et al. 1988; and Ralli, et al. 1996).

Only few studies have been conducted to evaluate the effect of middle ear pathologies on OAEs which are inconclusive when compared to studies done to examine the DPOAE findings in sensorineural hearing loss individuals. This may be because the DPOAEs are more sensitive to the micromechanical activity of outer hair cells and hence more effective in determining sensorineural loss. Few studies have been done to find out the DP emission in various conductive pathologies where they report of reduced emissions. But no quantitative result has been reported in the literature. No study have been done to find relationship between the DP thresholds and the behavioral thresholds to check the affect of various middle ear pathology on DPOAE's. Hence the aim of the present study is :

1) To find the DPOAEs in various degrees of conductive loss.
2) To estimate the hearing acuity with the DP thresholds obtained in conductive hearing loss cases.
3) To compare the relation of DP thresholds with behavioural thresholds in conductive loss population when compared to normal population.

REVIEW

Otoacoustic emissions are defined as "sound generated within the cochlea, by the outer hair cells, which can be detected at the tympanic membrane" (Norton and Stover, 1994).

Discovery of OAE is attributed to Kemp, at the Institute of Laryngology and Otology, London in 1978 and observed that on presenting brief broad spectrum sound stimuli to the ear, the ear emitted another sound of similar spectra but of very small intensity. Initially these were thought to be echoes of the stimulus and were labelled as Kemp's echoes. However, over the years, it has been confirmed that these sounds are not echoes, but emitted from the ear. These were called otoacoustic emissions. They reflect some aspect of active processes involved in the transduction of the auditory stimulus.

As DPOAE's are elicited by f1 and f2 stimulus, forward transmission of the stimulus and fl and f2 should be effective. The stimulus will be converted to mechanical energy at tympanic membrane and transmitted through the ossicles to the cochlea. Cochlea by its nonlinear processes produces OAEs. Hence the DPOAE's depend upon the stimulus reaching the cochlea which by its active nonlinear process generates OAE's.

In transmitting the emissions from the cochlea to the outer ear, the ear drum act like cone of a loudspeaker by transducing the mechanical energy of the ossicles to airborne acoustic energy. Backward transmission seems to be less efficient than forward transmission by 12-16 dB (Kemp, 1980).

The presence of DP was first postulated by Hall (1972). However for a number of years, it was not realized that these distortions could be picked up at the tympanic membrane (Kemp, 1984). Such distortion product emissions obtained in the ear canal consisted of acoustic energy at specific frequencies that were detectable above the noise floor in power spectrum of the signal.

Parameters Affecting Distortion Product Otoacoustic Emissions

a) f2 f1 Ratio : There have been various studies regarding the optimal ratio of f2 and f1 to yield maximum distortion product. Harris (1989), Lonsbury-Martin et al. (1990a, 1990b) have reported that a ratio of 1.22 of primaries yield the maximum distortion product. But a ratio of 1.15, according to Brown and Norton (1994) gives the most sensitive distortion product threshold. Beyond an optimal ratio, the intensity of the DPOAE falls at a very rapid rate (Gaskil et al. 1990).

b) Intensity of Primaries (L1 and L2) : Various studies indicate the effect of intensity level of the primaries on DPOAEs. Lower levels of the primaries elicit a local response and thus gives a frequency specific information. At higher levels, the response is more complex, non-local (Avans and Bonftls, 1993). The DPOAEs elicited from low stimulus levels are dominated by active cochlear mechanical processes, whereas the high stimulus level DPOAEs may be dominated by passive cochlear mechanics (Whitehead, et al. 1992a, 1992b). Hence the saturation occurs at high levels, Humes (1983); Weber and Millert (1975). Avans and Bonfils (1993) also reported that DPOAEs elicited from high stimulus levels (72 dB SPL) are not as sensitive to a decrease in hearing threshold, possibly owing to a broadening of the cochlear tuning.

c) **The Difference Between** L1 **and** L2 : A few studies report that maximum DP amplitude occurs when L1> L2 (Gaskil et al. 1990). The emission level is said to be better when L1-L2 =10 dB HL or 15 dB HL (Sun et al. 1996). Others report that the differences in the intensity of primaries has different roles at different frequencies (Hauser, 1991). The amplitude of DPOAE has a variability of 10 dB HL to 20 dB HL depending on the relative levels of L1 and L2 and the frequencies f1 and f2 of the primaries. If using, an equal level stimuli (L1 = L2) the DP emission, stimuli/growth functions is linear with a slope near about to a stimulus level of 60 dB SPL to 70 dB SPL, after which saturation may be observed. DPOAEs amplitude is found to be highest when equi-level primaries are used (Kemp, 1978).

Irrespective of the controversies regarding the best parameters for obtaining DPOAE, it is well established that it promises to be an excellent clinical tool for audiological evaluations, as elevated behavioural threshold corresponds to a reduced DP amplitude if the stimulus parameters are kept constant (Harris, et al. 1989). It can thus differentiate between normal hearing and hearing loss patients (Martin, 1990; Smurzynski, 1990).

DPOAEs in Normal Population

As the common application of DPOAE's is for detecting an abnormal reduction of DPOAE amplitude; normative of DP amplitude and DP threshold is needed. Several studies have been conducted. The lowest DPOAE thresholds of 5 dB SPL, obtained in normals was given by Wilson (1980), Scholth (1982), Burns et al. (1984) and Wier et al. (1988). Lonsbury-Martin et al. (1990b) averaged DPOAE input output growth function of 44 normal ears. The function were generally less steep for lower geometric mean frequencies (1 kHz to 2 kHz) with a slope of less than 0.8 whereas higher geometric mean frequencies showed a steeper input output function around 0.8 to 0.95. This study putforth normal threshold to be 35-45 dB SPL for emissions between 1 to 8 kHz.

DPOAE in Sensorineural Hearing Loss

DPOAE is found to be more resistant to cochlear damage, and may be seen in the ears with behavioral thresholds up to 50 dB HL (Harris, 1990). It may thus be more useful in monitoring cochlear changes clinically than the other OAEs. Research suggest that the effect of ototoxicity on the cochlea can be monitored as it leads to a reduction in DPOAE amplitude.

Harris (1990) concluded that if hearing at predetermined frequency was better than 15 dB HL, DPOAE were always detected. However emission were absent/attentuated if behavioral threshold exceeded 50 dB HL in study of both normal and patients with unspecified SN hearing loss. According to Martin et al. (1990) ability of DPOAE to assess sensory component of sensorineural disorder may contribute to eventual understanding of the complicated pathogenesis of many cochlear disorders. Gorga, et al. 1993 a, b; Ricci et al. 1996; Suckfull, et al. 1996; Lonsbury-Martin and Martin, 1990; Smurzynsky et al. 1990 also reported similar findings.

DPOAEs in Conductive Hearing Loss Cases

As the otoacoustic emissions are transmitted from the cochlea to the ear canal via the middle ear the transmission properties of the middle ear directly influence OAE characteristics. Even the emission eliciting stimulus has been affected by middle ear transmission characteristics. Hence changes in OAE characteristics due to middle ear dysfunction result from changes in both forward and backward transmission variably (Margolis and Trine, 1997).

As the ear canal volume effects the intensity and other characteristics of the response, deep insertion is desirable to maximize the amplitude of the response. A loss in the backward transmission due to middle ear dysfunction would reduce the emission level measured in the ear canal, various middle ear disorders have found to effect the forward and backward transmission of sound energy variably, depending upon the pathology.

Because OAEs are also sensitive to the external and middle ear factors it can be potentially used as a clinical tool in -

(i) screening for middle ear dysfunction in newborns, infants and schoolage children,

(ii) to evaluate the middle ear dysfunction and estimating the hearing acuity.

TEOAEs have been used for screening programs frequently. Chang et al. (1993) used OAEs for screening neonates and reported that debris in the external auditory canal can attenuate the OAE signal in spite of normal middle ear and cochlear status. Nozza, et al. (1997) included otoscopy, immittance measurement and puretone hearing screening together with TEOAEs for screening the hearing-impairment and middle ear disorders in schoolage children. The screening criterias adopted produced good results with increased sensitivity and specificity (Parker & Banford, 1996).

OAEs can be used to evaluate the various middle ear disorders and estimate the hearing acuity. Qui, et al. (1997) used impedance audiometry, DPOAE and ABR in evaluating the effect of glomus tumour on the auditory system as well as their pathologic extent. Hunter and Margolis (1997) together with the multifrequency tympanometry, otoreflectance and the video otoscopy, used OAEs to detect cholesteotoma, chronic otitis media with effusion and illustrated the use of OAEs as a diagnostic tool when combined with other instruments.

Many studies have been conducted to evaluate the various middle ear pathological conditions using OAE's alone.

Variations in the air pressure in the middle ear and external ear affects the otoacoustic emission amplitude as well as the recording in the external auditory canal. Osterhamrnel, et al. (1993), reported that at 1 kHz, the effect of positive and negative pressure in the external ear symmetrically reduced DPOAE amplitude by about 8 dB for 100 dapa and 11 dB for 200 dapa. The effect was smallest at 2 kHz, and at higher frequencies, negative pressure decreases DPOAE amplitude and positive pressure causes slight increase in amplitude. Robinson and Haughton (1991) said that positive pressure has more affect than negative pressure and the response was reduced in both across the frequency range. A positive pressure applied to the ear canal influence the middle ear transmission in the same way as a negative pressure of equal magnitude applied to the middle ear.

Trine, et al. (1993) reported that in the patients with negative tympanometric peak pressures ranging from -100 to -310 dapa, TEOAEs amplitude as well as reproducibility was better with the ear canal pressure adjusted to compensate for the negative middle ear pressure when compared to its amplitude measured at ambient ear canal pressure.

Kemp et al. (1990) reported that middle ear negative pressure or middle ear fluid has a definite attenuation of energy below 2 kHz and enhancement above 3 kHz for DPOAEs.

Zhang and Abbas (1997) reported that positive pressure affected low frequency stimuli, but had little effect for high frequency stimuli. But negative pressure affected transmission across all frequencies tested.

Schmuziger, et al. (1996) showed that TEOAE and DPOAE levels increased when air-borne gap was reduced by an average of 8 dB after negative middle ear pressures returned from -400 dapa to a normal state. Negative middle ear pressure affected DPOAE's more in the 1 kHz than in the higher frequencies. But TEOAE's and air borne gap were more uniformly affected across the entire frequency range.

Plinkert and Plot (1994) conducted a study on hypoventilation of the middle ear. Results showed that negative middle ear pressure significantly attenuated the amplitude of low frequency OAE (less than 2 kHz), but for high frequencies the emissions were stable. This contradicts the results obtained for high frequencies by Kemp et al. (1990) and Zhang and Abbas (1997).

Owen et al. (1992) recorded TEOAE's and DPOAE's in patients with various amounts of middle ear effusion. They were unable to record TEOAE's in any case where there was effusion. DPOAE's were observed only in the low frequencies. DPOAE's were not observed in patients with large volumes of middle ear fluid. Chang et al. (1993) supports the study telling that in serous middle ear effusion with absent tympanic membrane movement, OAE responses were consistent with the low frequency attenuating effect of middle ear fluid. Amedee (1995) says that type of effusion in the middle ear does affect the presence or absence of TEOAE. Although otitis media often eliminates OAE responses, it is possible to record emissions in some patients with middle ear effusion (Margolis and Trine, 1997). Bray (1989) after examining the effect of fluid loading on the tympanic membrane through the introduction of three droplets of water, said that emission was almost completely absent in all cases.

Vanstenis, et al. (1995) used TEOAE's in the evaluation of hearing acuity in children with otitis media with effusion before and after the ventilation tube insertion in the operation theatre itself under general anasthesia. None of the ears showed any increase in OAE's. It may be due to -

1) Some effusion present in the middle ear after surgical procedure which diminished the inability of the oscicular chain and caused the hearing loss.

2) Effect of anasthetic gas on middle ear dysfunction owing to increased middle ear pressure due to diffusion of anasthetic gas into the middle ear.

3) Temporary threshold shift due to high noise levels caused by suctioning.

4) Mechanical effects on the middle ear processes that govern reverse transmission from the cochlea to the outer ear canal.

But at follow-up visits, they recorded OAE's in 65% of the ears. Richardson, et al. (1996) also supports the above study, where they were able to record OAE's in 50% of ears immediately after grommet insertion, but reduced in amplitude. Hence they conclude that OAE's can be used in outpatient setting effectively to check hearing acuity.

Wiederhold (1990) measured DPOAE's in anasthesized cats before and after ear abnormalities were created, i.e. mass loading of the tympanic membrane and ear drum perforation. DPOAE's in the mid frequency region (3 kHz) were reduced. This mainly affected the transmission of the emission from the inner ear to the ear canal more than the forward transmission. The difference in forward and backward transmission, estimated by Kemp (1980) to be 12 to 16 dB was increased by the ear drum abnormality,. Widerhold suggested that a scarred tympanic membrane could have similar effect.

Margolis and Trine (1997) observed that small ear drum perforation affected DPOAE's differently than mass loading. The change in DPOAE amplitude was identical to that predicted by the change in the forward transmission. Thus, the change appeared to be due entirely to a change in the effective stimulus reaching the inner ear and not to a change in the transmission of the emission from the inner ear to the ear canal. In their study effects of perforations of different sizes and different locations were not explored. Hence they say one should be careful in generalizing the results to patients with ear drum perforation.

Rossi, et al. (1988) recorded tone burst evoked OAE's from 8 patients with unilateral otosclerosis for air conduction and bone conduction 1 kHz tone bursts. Air conducted stimuli presented at 30 dB HL did not evoke measurable emissions. Bone conducted stimuli, did elicit measurable responses.

Ralli, et al. (1996) tested DPOAEs in 45 patients with otosclerosis and 18 subjects who had otosclerosis but had undergone stapedectomy. In 53% of cases DP emissions were clearly evident, but differed greatly and average amplitude was lower than in normals. There was no signal at all, at the lower and middle frequencies. 58% who had undergone stapedectomy showed valid DP emissions. Lower and mid frequency amplitudes were significantly found to be better.

The review shows that DP emissions were reduced in middle ear pathology. They were reduced with negative as well as positive pressure in the middle ear with contradictory results in different studies. They were reduced in tympanic membrane abnormalities and otosclerotic patients and were completely absent in few ears with middle ear effusion. Hence the present study was carried out to determine the effect of middle ear pathology on DPOAE's and their relation with their behavioural thresholds.

METHODOLOGY

This study was to find out the effect on distortion product otoacoustic emissions in different degrees of conductive hearing loss cases in comparison with the normal hearing individuals.

Methodology used was as follows :

I. SUBJECTS

a) ***No. of subjects***

(i) Control group :- 30 ears of age ranging from 18-25 years with normal hearing,

(ii) Experimental group :- 30 ears of age ranging from 15-50 years with conductive hearing loss of varying degrees (mild, moderate and moderately severe).

b) ***Subject selection criteria***

(i) Control group : Puretone thresholds of lesser than 25 dB HL (ANSI, 1969) in 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were taken. They had normal tympanometric findings with normal reflexes and with no history of neurological symptoms, ototoxicity, exposure to noise and otological history.

(ii) Experimental group : Individuals with abnormal tympanometric findings and reflexes and who has been confirmed to have middle ear pathology by ENT specialist were taken. They were categorized into three groups depending upon the degree of hearing loss.

Mild - 26-40 dB HL

Moderate - 41-55 dB HL

Moderately severe 56-70 dB HL

The subjects with no history of sensorineural symptoms like giddiness, tolerance problem and nauseaet. were taken.

^INSTRUMENTATION

a) **Puretone audiometer** - A calibrated double channelled diagnostic audiometer was used to do air conduction, bone conduction and speech audiometry to arrive at the diagnosis.

b) **Immittance meter** - A calibrated immittance meter GSI-33, version-2 was used to assess the middle ear function.

c) **Otoacoustic emission analyzer** - Madsen Celesta 503 cochlear emissions analyzer was used to obtain DP emission. This is a computer based OAE measuring system.

The system allows for the user specification to be used in testing for a number of parameters. Following parameters were taken for DPOAE measurement:

i) **Intensity Level (L1 and L2)** : This refers to the intensities of the stimulus frequencies. The starting intensity level was 70 dB SPL i.e. L1 and L2 = 70 dB SPL. Very high levels of stimulus give rise to non-local response i.e. distortion product does not correspond to a specific area on the basilar membrane (Avans and Bonfils, 1993) and saturation also occurs when higher levels of intensity is used (Humes, 1983; Weber and Mellert, 1975).

Hence 70 dB SPL was considered as starting level.

(ii) **Frequencies (**f1 **and f2)** : Testing was carried out at 4 sets of frequencies from 500 Hz to 4000 Hz.

f1(Hz)	f2 (Hz)	fo(Hz)	2f1-f2(Hz)
452	553	500	351
910	1112	1006	708
1819	2223	2011	1415
3651	4462	4036	2840

(iii) **DP frequency** - It refers to the frequency of emission. It would be set to 2f1-f2. Because the inter-modulation distortion product at 2f1-f2 is most prominent (Kemp, 1979).

(iv) **f2/**f1 **ratio** : f2/f1 ratio was 1.22 as DPOAEs had maximum amplitude for this ratio in a study by Harris et al. (1989). Celesta 503 also has a default ratio of 1.22.

(v) **Points per octave':** It refers to the number of points tested per octave. The test was carried out at one point per octave.

(vi) **Display type** - The display type controls the pattern of measurement. Since DP threshold was to be established, display was set to "Input/Output display" to obtain a input output function curve of DPOAE. This setting plots growth of distortion product responses at a single frequency for different input levels of two primary tones.

(vii) S/N **ratio**: S/N ratio refers to the criteria to determine a particular emission or to determine when to stop averaging a frequency. S/N ratio of+3 dB was taken as a criteria to stop averaging at that intensity.

(viii)Accepted sweeps - The instrument plotted the average DP emissions level and noise floor after the completion of 250 sweeps at a particular intensity. If the instrument was able to detect the emission before 250 sweeps it stopped averaging and gave the measurement.

III. TEST ENVIRONMENT

Puretone testing was carried out in a sound treated room where the ambient noise level was within the specified limits, ANSI (1977). DP emission measurement was carried in a sound treated room with controlled background noise levels.

The test room was comfortable enough in terms of temperature and lighting. The subjects were made to sit on chair comfortably during test.

IV. TEST PROCEDURE

The subjects who satisfied the selection criteria were taken for the study.

a) **Pure tone testing** : Thresholds were obtained at octave frequencies from 500 Hz to 4000 Hz for both air conduction and bone conduction using modified Hughson-Westlake Procedure in a sound treated room as recommended by ANSI (1977).

b) **Immittance testing**: Tympanometry and acoustic reflexometry was done to assess middle ear condition in normals and individuals with middle ear pathology.

c) **Distortion product otoacoustic emission measurement** was carried out on both the control and experimental group in a sound treated room where background noise levels were kept minimum.

ci) Preparation of **the** subject: A suitable probe tip was fitted on to the probe and inserted into the ear canal of the test ear. Subject was instructed to sit back and relax and reduce his body movement as much as possible.

cii) **Probe fit** - This is a procedure to check adequate fitting of the probe into the ear canal. This was carried out, automatically by the instrument. A transient stimulus was presented to the ear and the measured response was displayed as a spectrum and a waveform. A correct probe fit would give a waveform as in fig-1. If such a

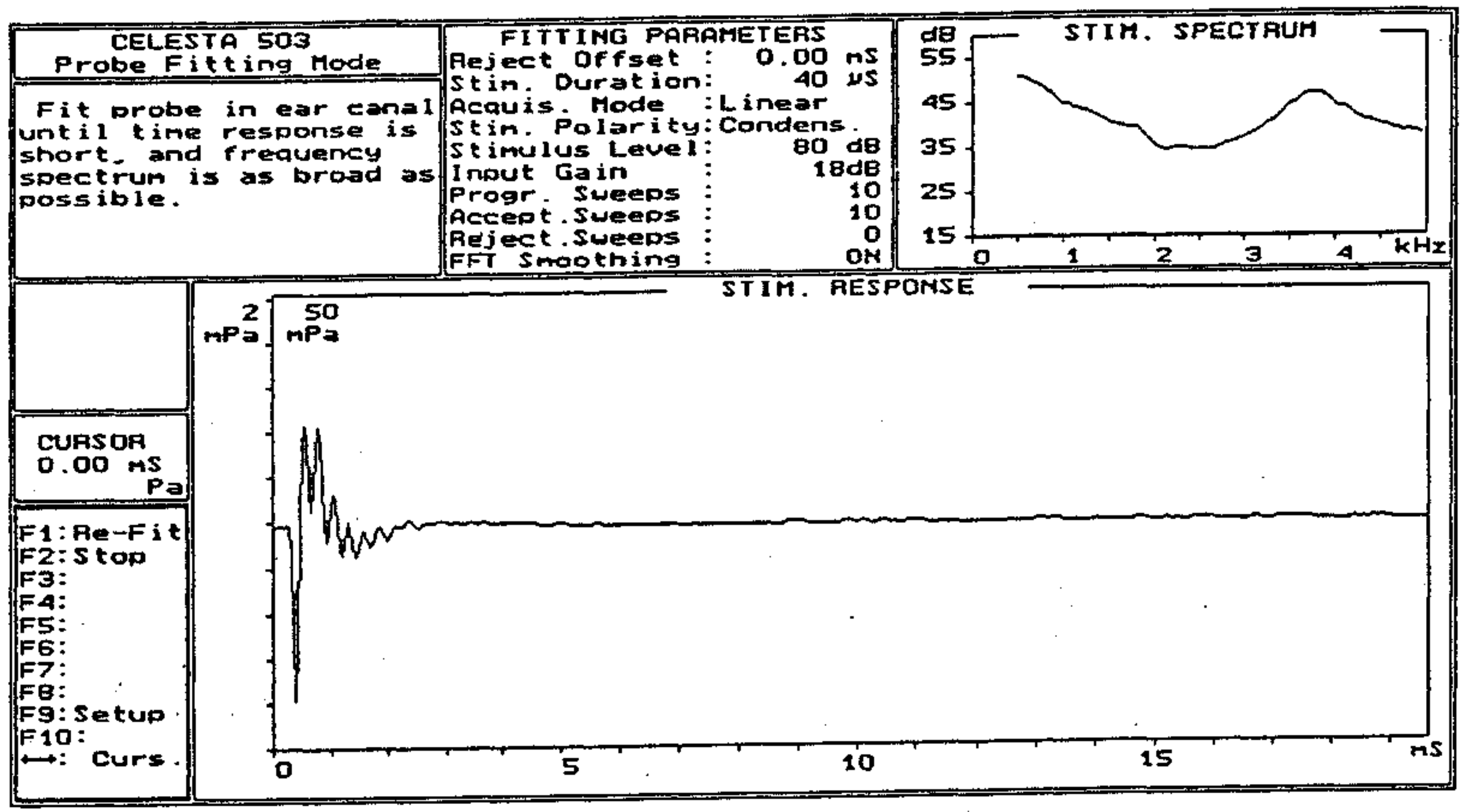

CELESTA 503
Probe Fitting Mode
Fit probe in ear canal until time response is short, and frequency spectrum is as broad as possible.
FITTING PARAMETERS
Reject Offset : 0.00 mS
Stim. Duration: 40 µS
Acquis. Mode :Linear
Stim. Polarity:Condens.
Stimulus Level: 80 dB
Input Gain : 18dB
Progr. Sweeps : 10
Accept.Sweeps : 10
Reject.Sweeps : 0
FFT Smoothing : ON
STIM. SPECTRUM
dB
55
45
35
25
15
0
1
2
3
4
kHz
STIM. RESPONSE
2 mPa
50 mPa
0
5
10
15
mS
CURSOR
0.00 mS
Pa
F1:Re-Fit
F2:Stop
F3:
F4:
F5:
F6:
F7:
F8:
F9:Setup
F10:
←→: Curs.

fig 1

waveform was not obtained as in fig-l the probe was taken out. With different size of the tip it was refitted. The check fit phase was redone to obtain correct fit. Once the probe was fitted, it was not removed till the completion of the test.

Procedures involved in emission measurement were as follows :

Two puretone stimuli, both at 70 dB SPL were presented initially.

The intensities were attenuated from 70 dB SPL jn steps of 5 dB SPL till the intensity, where no emission was obtained keeping L1 and L2 equal at every step.

The instrument plotted an input output function curve with each set of primary frequencies f1 and f2.

The testing was stopped at each intensity level if,

* S/N ratio exceeds or equal to +/- 3 dB during average.
* 250 stimuli was accepted or if the instrument detects DP emission within certain sweeps and gives the measurement.

If the S/N ratio fall below + 3 dB SPL the testing was terminated at that frequency.

Distortion product emission were obtained both for normal hearing and conductive hearing loss ears in the above mentioned procedure.

The minimum intensity level of the primary at which the S/N ratio is +3 dB was considered as distortion product threshold.

The study aims at assessing distortion product otoacoustic emissions in conductive hearing loss cases. Hence the thresholds in conductive loss ears were compared with the DP threshold in normal ears to assess the effects of conductive pathology on DPOAE's.

DP threshold may be a better indicator of hearing sensitivity than of its amplitude when high level primaries are used to elicit DPOAE's (Stover and Norton, 1993; Stover et al. 1995) as long as the noise floor is consistent enough across subjects to allow useful information (Nelson & Kimberley, 1992). Hence DP amplitude was not analyzed.

Analysis

The 't' test was used to compare the mean DP thresholds in normals and pathological population to determine if a significant difference existed between the two groups. The Kearl Pearson's product moment correlation was used to find out the correlation between normals DP thresholds and abnormal DP thresholds. The 't' test was used to check the significant difference between the correlation co-efficients.

For few statistical purposes the behavioural threshold which showed DP emissions were taken. The percentage of ears which showed the response as in table X5 in results and discussion were only taken to know the direction of relation. For rest of the data descriptive analysis W45 adopted.

RESULTS AND DISCUSSION

The DP thresholds were obtained from normal and conductive loss cases and were analysed using various statistical procedures as mentioned in the methodology.

1. **Distortion product thresholds**

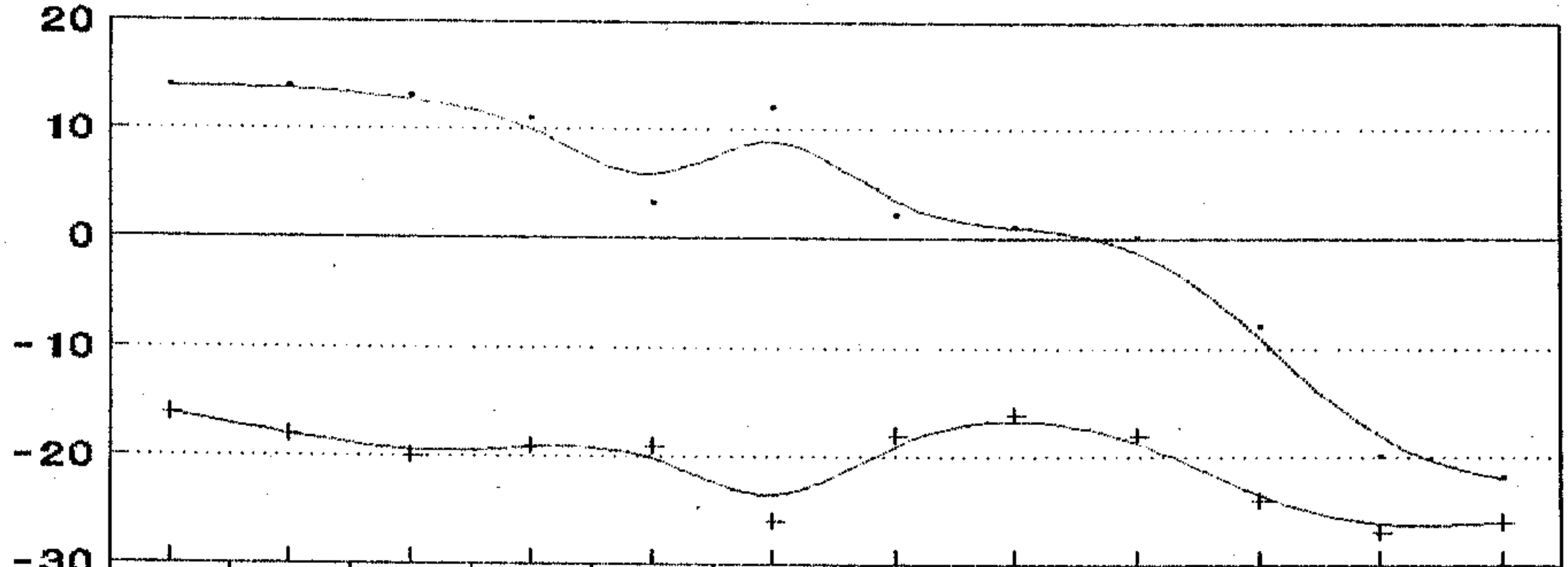
a) Normals : The distortion product threshods for 30 normal ears were obtained for geometric mean frequencies of approximately 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. The mean values, standard deviation and range of the DP detection threshold at each frequency was obtained.

Frequency (Hz)	Puretone threshold (dBHL)	Mean DP threshold (dBSPL)	SD	Range (dB SPL)
500	11.83	49.83	9.04	30-65
1000	14.5	40	12.01	20-65
2000	7.3	32	9.05	10-55
4000	9.3	31	12.08	15-70

Table X1 :Mean puretone thresholds and DP thresholds and their standard deviation, range at different frequencies in normals.

The above table shows the average DP thresholds of 49.83, 40, 32 and 31 dB SPL and standard deviation of 9.04, 12.01,9.05 and 12.08 at 500, 1000, 2000 and 4000 Hz respectively. The-range

Input-output graph [Normals]
at 2 kHz



DP	14	14	13	11	3	12	2	1	0	-8	-20	-22
Noise	-16	-18	-20	-19	-19	-26	-18	-16	-18	-24	-27	-26

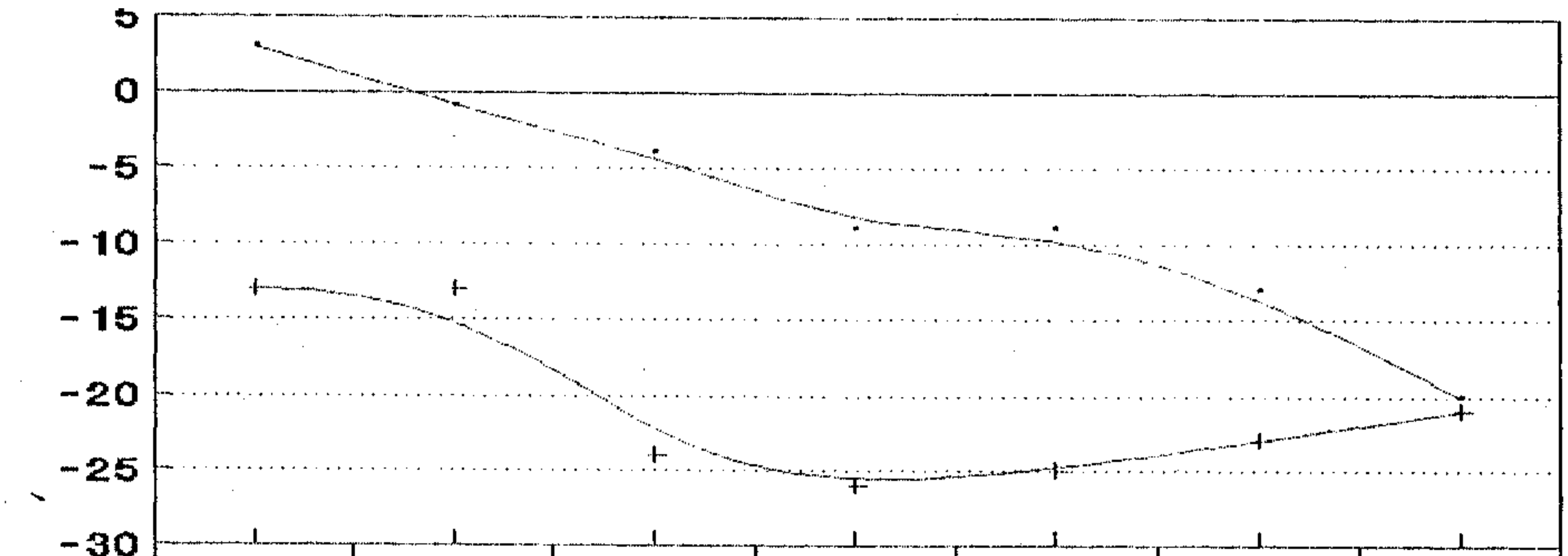
was wider at all frequencies. At 500 Hz it was 30-65 dB SPL, 1000 Hz 20-65 dB SPL and 2000 Hz 10-55 dB SPL and 4000 Hz 15-70 dB SPL. These are supported by the findings of Lonsbury-Martin et al. 1990; who report a threshold of 35 dB SPL to 45 dB SPL between 1000 Hz to 8000 Hz. As the frequency increased the average DP thresholds also decreased from 48.83 dB SPL at 500 Hz to 31 dB SPL at 4000 Hz. The lowest DP threshold also reduced from 30 dB SPL at 500 Hz to 10 dB SPL at 2000 Hz and 15 dB SPL at 4000 Hz. This might be due to the increased noise floor at low frequencies which effects the emission detection. Lonsbury-Martin et al. (1997) reported that both acoustic noise from the environment and physiological noise from the subject makes the measurement of DP emissions at DP frequencies (lesser than 1 kHz) difficult.

b) **Pathological Ears** : The distortion product thresholds for 30 ears with various kinds of middle ear pathologies were measured at **all** frequencies. The mean values, standard deviations and the range for DP threshold at respective frequencies was calculated.

Frequency (Hz)	Mean Puretone threshold (dBHL)	Mean DP threshold (dB SPL)	SD	Range (dB SPL)
500	40	65	8.017	50-70+
1000	39.61	62.3	8.32	40-70+
2000	35.9	57.75	8.02	45-70+
4000	34.75	59.75	8.18	40-70+

Table X2 :Mean puretone thresholds and DP thresholds of conductive loss population together with standard deviation and the range at different frequencies.

Input-output Graph (2kHz)
Pathological population



DP	3	-1	-4	-9	-9	-13	-20
Noise	-13	-13	-24	-26	-25	-23	-21

— DP —+— Noise

The above table shows the average puretone threshold of 40, 39.61, 35.9 and 34.75 dB HL and the mean DP threshold of 65, 62.3, 57.75 and 59.75 dB SPL at 500, 1000, 2000 and 4000 Hz respectively. As the frequency increased the DP thresholds decreased from 65 dB SPL at 500 Hz to 57.75 dB SPL at 2000 Hz and 59.75 dB SPL at 4000 Hz. The range was wider with the lowest DP threshold to be 40 dB SPL at 1000 and 4000 Hz. The highest was not estimated in some cases and was taken as above 70 dB SPL. At all frequencies many ears did not show response even at 70 dB SPL input level and hence the range was wide. The lowest DP threshold was 50 dB SPL, 40 dB SPL, 45 dB SPL, 40 dB SPL at 500, 1000,2000, 4000 Hz. It was seen that the lowest DP threshold at different frequencies decreased from 50 dB SPL at 500 Hz to 40 dB SPL at 1000 and 4000 Hz with 45 dB SPL at 2000 Hz.

This might be due to increased noise floor at low frequencies which effects the DP emission detection. These findings are similar to that obtained in normal population.

The standard deviation of approximately 8 was obtained at all frequencies.

Frequency (Hz)	Mean DP threshold normals (dB SPL)	Mean DP threshold conductive loss (dB SPL)	Significance 'Z' value
500	49.83	65	0.0007
1000	40	62.3	0.0000
2000	32	57.75	0.0000
4000	31	59.75	0.0000

Table X3 : Mean DP thresholds of normals and conductive loss population with their values of significance.

The mean DP thresholds of normals and conductive loss population were tested for significant difference at 0.01 levels where in the significant difference was present at all frequencies with Z values 0.0007 at 500 Hz, 0.0000 at 1000, 2000, 4000 Hz which is significant at 0.01 level. This implies that pathological group has a significantly higher puretone as well as DP thresholds than normal ears at the frequencies being tested. It was seen that as the hearing loss increased (due to ineffective forward transmission) the DP thresholds worsened in spite of normal cochlear status. This is attributed to affected forward transmission which affects the f1 and f2 stimuli reaching theinner ear and backward transimmision which affects the transmission of OAEs. Due to middle ear pathology the emission energy reaching the earcanal was very less overcoming backward transmission loss. Hence the primary stimulus f1 and f2 would be more intense so as to elicit as well as record the emission at the earcanal overcoming the forward and backward transmission loss. These findings support the findings obtained by Owens et al. (1992); Osterhammel, et al. (1993); Amedee, (1995); Chang, et al. (1993); Margolis & Trine (1997).

2. Comparison of DP thresholds of normals with the DP thresholds of various degree of conductive hearing loss.

Frequency (Hz)	Normals		Mild		Moderate	
	PTT dBHL	DPT dBSPL	PTT dBHL	DPT dBSPL	PTT dBHL	DPT dBSPL
500	11.83	49.83	31.25	60	48.75	70
1000	14.5	40.66	27.85	58.57	50	65
2000	7.33	32	27.69	51.53	49.16	62.5
4000	9.33	31.17	31.42	58.57	47	62

PTT : Puretone threshold; DPT : Distortion Product threshold

Table X4 : Mean Puretone and DP thresholds of normals, and different degrees (mild and moderate) of conductive hearing loss at different frequencies.

Table shows the average puretone thresholds of normals together with the average DP thresholds at different frequencies. Average DP thresholds were 49.83,40.66, 32 and 31.17 dB SPL for the normal hearing sensitivity at all frequencies.

In mild conductive loss, average DP thresholds were 60, 58.57, 51.53, 58.57 dB SPL with the average puretone thresholds of 31.25, 27.85, 27.69 and 31.42 dB HL at 500, 1000, 2000 and 4000 Hz. respectively. In moderate category, average DP thresholds were 70, 65, 62.5 and 62 dB SPL with average puretone thresholds of 48.75, 50,49.16,47 dB HL at 500, 1000, 2000 and 4000 Hz. It was seen that as the degree of loss increased from mild to moderate correspondingly the DP thresholds also increased which is statistically significant at 0.01 levels. This shows that the increase in the loss definitely showed an increase in the DP threshold. The same is reported by Schmuziger, et al. (1996).

At moderately severe degree of loss the distortion product emissions were completely absent. This can be attributed to the high degree of hearing loss of greater than 55 dB HL, because of which DP emissions could not be recorded at any frequency. Harris (1990) also suggested that DP emissions are seen only in ears with behavioral thresholds upto 55 dB HL. It can be concluded from the above findings that the degree of behavioural thresholds has a direct effect on the DP thresholds i.e. DP threshold tend to vary with behavioral thresholds accordingly. Thus, it indicates that DP threshold can be used to estimate the behavioral thresholds in conductive loss population.

Table X4 indicates that as the frequency increased the DP thresholds were better in both mild and moderate degree of loss. In mild category the DP thresholds decreased from 60 dB SPL to 51.*53* dB SPL and increased to 58.57 dB SPL at 4000 Hz. In moderate category, from 70 dB SPL to 62 dB SPL in frequency range of 500 Hz to 4000 Hz. This may be attributed to the fact that middle ear pathology affects the low frequency transmission more than the high frequency because the impedance of the tympanic membrane (middle ear) decrease with increase in the frequency. Hence increase in the stiffness of the eardrum (usually associated with various middle ear pathology) has its greatest affect on low frequency transmission. Chang et al. (1993), Schmuziger, et al. (1996) Ralli, et al.(1996) also reported the same findings. Even in normals, the same trend was seen when DP threshold decreased from 49.83 dB SPL to 31.66 dB SPL in the frequency range of 500 to 4000 Hz.

Though there was variation among the DP thresholds at various frequencies, it was not statistically significant at 0.05 levels in both categories of conductive loss.

3. **OAEs and range of DP thresholds in various degrees of conductive hearing loss population.**

Frequency Hz	Mild		Moderate	
	% of ears which showed response	Range of DPT (dB SPL)	% of ears which showed response	Range of DPT (dB SPL)
500	13.3	50-70+	13.3	70+
1000	23.3	40-70+	13.3	60-70+
2000	43.3	45-70+	20.0	55-70+
4000	46.6	40-70+	16.6	55-70+

DPT - Distortion Product Threshold

Table X5 : Percentage of responses and their range at different frequencies in different degrees of conductive loss.

In table X5, the percentage of ears which showed responses are given for both degrees of hearing loss at different frequencies. In mild hearing loss individuals, the responses were consistent. At 500 Hz, only 13.3% ears showed emissions which were reduced in amplitude considerably. 23.3%, 43.3% and 46.6% of ears showed DP emissions at 1000, 2000 and 4000 Hz. In moderate degree of loss, the percentage of ears showing response was further reduced to 13.3% at 500 Hz and 1000 Hz and 20.0%, 16.6% at 2000 and 4000 Hz. This shows that as the frequency increased the percentage of ears which showed responses were increased because the middle ear pathology which is associated with increased stiffness has its more effect on low frequency transmission than on high frequency transmission. In general at low frequencies DP thresholds are affected more than at high frequencies. Because the high frequencies are more stable for various middle ear pathologies, responses were seen more and easily obtained at those frquencies. Studies by *Zhang* and Abbas, 1997; Schmuzriger, et al. 1996; Ralli, et al. 1996 are on support with the above findings that at high frequencies DP threshold are affected less than the low frequencies.

It was seen that percentage of ears which showed response decreased as the degree of hearing loss increased from mild to moderate degree. At 500 Hz it remained the same. It decreased from 23.3% to 13.3% at 1000 Hz, 43.3% to 20% at 2000 Hz and a marked reduction at 4000 Hz from 46.67% to 16.6%. This implies that distortion product emissions are directly related to the degree of hearing loss. Thus as the degree of hearing loss increased DP thresholds also increased. Hence DP threshold can be a predictor of behavior threshold in conductive hearing loss population.

The range of DP thresholds for mild and moderate hearing loss was calculated. The lowest DP threshold was 50 dB SPL at 500 Hz, 40 dB SPL at 1000 Hz, 45 dB SPL at 2000 Hz and 40 dB SPL at 4000 Hz, in mild hearing loss cases. The highest was the no response value which was above 70 dB SPL. In moderate degree of loss the lowest DP threshold was 70, 60, 55 and 55 dB SPL at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz respectively. In some ears which showed no emission even at highest input level of 70 dB HL was considered as highest level (no response).

4. **Sensitivity and specificity of distortion product otoacoustic emissions.**

The sensitivity and specificity of the DPOAE as a tool to detect the conductive pathology was obtained from the data.

Frequency (Hz)	Sensitivity (%)	Specificity (%)
500	96.6	73
1000 '	86.6	83.3
2000	83.3	93.3
4000	83	93.3

Table X6: Sensitivity and specificity of DPOAE as a test measure to detect conductive pathology at different frequencies.

Sensitivity was found to be 96.6%, 86.6%, 83.3% and 83% at 500, 1000, 2000 and 4000 Hz. The specificity (percentage with which it ruled out the abnormality was considered) was 63.3%, 833%, 93.3% and 93.3% at 500, 1000, 2000 and 4000 Hz. **It** was seen **that** as the sensitivity of the test increased, the specificity of the test decreased or vice-versa At 500 Hz, the sensitivity was high with 96.6% and specificity was decreased considerably to 73.3%. At 2000 Hz the sensitivity was reduced to 83.3% and specificity increased to 93.3%. The more sensitive a test is the more likely it is to result in false alarms. The reverse is also true; the more specific a test is, the more likely it will be that some subjects who have the disorder will be missed.

5. **Correlation between DP *emission*, threshold and behavioural thresholds.**

Product moment correlation was done to obtain the correlation coefficients for normals and conductive pathology cases.

Frequency (Hz)	Correlation coefficients (r)	
	Normals	Conductive loss
500	0.441	0.668
1000	0.107	0.702
2000	0.396	0.494
4000	0.218	0.513

Table *X7.* Correlation coefficients depicting the correlations between DP threshlold and Behavioral threshold at different frequencies in normals and in conductive loss cases.

In normal population, co-rrelation coefficient (r) was 0.441, 0.107, 0.396 and 0.218 at 500, 1000, 2000 and 4000 Hz. By taking 'r' of 0.5 and above 0.5 to be significant it was seen that none of the frequencies showed significant correlation. At 500 Hz, average DP threshold and behavioural thresholds were corelated better when compared to the other frequencies with a co-efficient of 0.44, followed by 2000 Hz and 4000 Hz. But at 1000 Hz very poor correlation was obtained. This might be due to variation in the puretone threshold with a standard deviation of 12.01 at 1000 Hz.

In conductive loss population correlation co-efficients of 0.668, 0.702, 0.494, 0.513 at 500, 1000, 2000 and 4000 Hz was obtained. By taking 'r' of 0.5 and above 0.5 to be significant there found to be a significant correlation between DP thresholds and behavioural thresholds at all frequencies.

The good correlation was found at 1000 Hz followed by 500, 4000 and 2000 Hz. When compared to other frequencies poor correlation was found at 2000 Hz i.e. 0.494. Correlation between average DP thresholds and average behavioral thresholds was found to range from 0.49 to 0.7 at different frequencies. The findings obtained by Nelson and Kimberley (1992) are also in support of this study who got a correlation of 0.41 to 0.85 in hearing-impaired ears.

There was found to be no significant difference between correlation coefficient obtained from normal and pathological population at 0.05 levels at different frequencies. This suggest that in conductive loss, DP thresholds are correlated to puretone thresholds as in normal population. As the correlation suggests, the DP thresholds increased with behavioural thresholds. This leads to the fact that DP thresholds can be used to estimate the hearing sensitivity in conductive loss population as there is significant correlation obtained in the study.

SUMMARY AND CONCLUSION

Distortion product otoacoustic emissions have been developed as a clinical tool in the recent past which looks promising. Several studies have been conducted to find out DP emission in conductive hearing loss population. Few attempts have been made at correlating DP thresholds with behavioural thresholds in conductive hearing loss individuals. Hence the present study aimed to investigate,

(i) The distortion product emissions in different degrees of conductive hearing loss.

(ii) Correlation of DP thresholds with the behavioural thresholds in comparison with the normals.

30 normal ears (age range 18-25 years) and 30 ears with conductive pathology (age range 15-35 years) were taken for the study. Ears with cochlear symptoms as tolerance problems, giddiness ototoxicity, noise exposure etc. were excluded from the study. Behavioural audiometry and DPOAE testing were carriedout for both groups.

DPOAE testing was done using Madsen Celesta 503, Cochlear Emissions Analyzer. Geometric mean frequencies of approximately 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were tested starting from intensity level of 70 dB SPL reduced in steps of 5 dB SPL.

The results showed the following

a) Normals - Normals had mean DP thresholds of 49.83 dB SPL, 40 dB SPL, 32 dB SPL and 31 dB SPL at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

b) Conductive hearing loss

The mean DP thresholds for this group of subjects were 65 dB SPL, 62.3 dB SPL, 57.75 dB SPL at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

There was a significant difference between mean DP thresholds of normal and pathological population at 0.01 level i.e. DP thresholds were significantly higher in pathlogical population when compared to normals.

Within the group of conductive loss individuals, DP thresholds were increased as the degree of loss increased from mild to moderate.

High frequencies showed better DP thresholds than the low frequencies.

More number of ears showed response at high frequencies rather than the low frequencies with in different categories of hearing loss.

DP thresholds ranged from 43.75 dB SPL (Mean dB SPL) to highest threshold considered as no response (No response obtained at highest input level (70 dB HL) of the present study).

Correlation Study Results

Poor correlation between DP thresholds and behavioural threshold was obtained at different frequencies in normals.

Significant correlation between DP thresholds and behavioral thresholds was obtained at different frequencies for pathological population.

There was no significant difference between the correlation co-efficients of normal and pathological population at 0.05 levels.

Thus it could be concluded that DP thresholds can be used to estimate the hearing sensitivity in conductive pathology ears because of the significant correlation obtained in the study. DP thresholds are found to be increased as the behvioural thresholds increased accordingly overcoming the pathological state and the hearing loss.

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