COMPARISON OF DPOAE AND TEOAE IN THE SUBJECTS WITH SENSORINEURAL HEARING LOSS

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A dissertation submitted as part fulfilment of Final Year M.Sc., (Speech and Hearing), Mysore.

All India Institute of Speech and Hearing, Mysore

May 1997

DEDICATED TO

MY FAMILY AND

MY SHADOW OF INSPIRATION

KEMP, PROBST, HARRIS AND LONSBURY-MARTIN

CERTIFICATE

This is to certify that this Dissertation entitled COMPARISON OF DPOAE AND TEOAE IN SUBJECTS WITH SENSORINEURAL 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.M9515.

Mysore May, 1997

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This is to certify that this Dissertation entitled COMPARISON OF DPOAE AND TEOAE IN SUBJECTS WITH SENSORINEURAL HEARING LOSS has been prepared under my supervision and guidance.

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DECLARATION

This Dissertation entitled COMPARISON OF DPOAE AND TEOAE IN SUBJECTS WITH SENSORINEURAL HEARING LOSS is the result of my own study under the guidance of Mrs.Vanaja C.S. Lecturer in Audiology, Department of 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, 1997

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ACKNOWLEDGEMENT

I would like to thank Mrs.Vanaja, C.S. Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore for her invaluable guidance and support. Madam, as a guide and lecturer you are par excellence. Need I say say any more?

I thank Dr.Nikam, Director, All India Institute of Speech and Hearing, Mysore, for granting me permission and the opportunity to undertake this study.

Thanks are also due to Mr.Animesh, Mr.Sunil, Ms.Manjula, Ms. Rohini, Ms. Revathi, Mr. Rajendra Swamy, Mr.Rajeev for helping me to carry out the study successfully.

A million thanks to all my subjects, without whose cooperation I could not have conducted the study.

"Friends who care are rare" and so needless say than to Rajesh, Jay, Su, Anu, Shanth, Archu and Sam.

Sunil and Arvin, I am richer for having known you. Thank for colouring my life.

Prachi, Smitha, Chandu, Vandana, Kaveri, Beula, Arushi, Pambe, Archu, Radhe, Sonia, Piyali, Anshu, Jaya and little Beryl. Darlings, a million thanx to you all for giving me warmth and affection throughout the year and helping me in and out in everything.

Neha, my beauty you have been a friend cum sister to me and this eternal bond was very precious to me.

To all my friends at AIISH. Thanx for everything we have shared and I have enjoyed.

Pubali, Asha, Bini, Makku, Vinu no matter what, you have always been there and that's what counts. I'll always tressure our friendship. My deary Sudha, Dhanesh and Suma, I'll always love you the same.

To my parents, if it hadn't been for you, I wouldn't have been. You made me what I am to day and the credit goes to you.

Chinku and Biju - Life means a lot to me because of you both. You know I LOVE YOU.

Thanks to Rajalakshmi Akka for her immense timely hectic neat typing work. God Bless You.

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INTRODUCTION

One of the most exciting advances in our understanding of hearing during recent years has concerned the discovery of oto-acoustic emissions (OAEs). OAEs describe the responses that the cochlea emits in the form of acoustic energy. The recognition that the cochlea not only receives sound, but also produces acoustic energy has been a major factor in modifying recent thinking concerning cochlear function.

Over 50 years age, Gold (1948) proposed the hypothesis that the sharp frequency selectivity exhibited by the cochlea has resulted from feedback а system consisting of а mechanical to electrical transduction process coupled to an electrical-mechanical transduction process. Gold's postulate of a reverse transduction process in the form of an electromechanical conversion mechanism suggested the sensibility of detecting this process in the form of sound in the ear canal. However Gold's hypothesis was never taken seriously until Kemp (1978) demonstrated that energy was indeed emitted by the cochlea and that it was recordable as vibrations in the ear canal using specialized methods and equipment.

The discovery of OAEs was important for both theoretical and practical reasons. The presence of evoked sound pressure

oscillation in the ear canal provided direct evidence of the existence of active mechanical mechanisms in the cochlea. Awareness of OAE also suggested a role for the OHCs in the stimulus - transduction process given the knowledge that these receptors don't process a prominent afferent nerve fibre innervation (Spoendlin, 1979). Finally, the discovery of OAEs permitted the proposition of testable hypothesis to account for several psychoacoustical phenomenon including the microstructures of behavioural sensitivity and loudness enhancement which were incomprehensible in terms of conventional models of the auditory system (Kemp, 1979 b).

OAEs can be broadly classified into two types (1) Spontaneous OAEs which occur in the absence of any external stimulation (2) Evoked OAEs which occur during or after external acoustic stimulation. There are several subclasses of EOAEs based primarily on the stimuli used to evoke them. They include

 (i) TEOAEs - These are frequency dispersive emissions occurring in response to a transient acoustic stimuli such as click or a tone burst.

- (ii) DPOAEs these are generated in response to two continuous puretones closely separated in frequency by a prescribed difference (in Hz) and presented simultaneously to the ear.
- (iii) SFOAEs These occur as a synchronous response to a continuous tonal stimulus and are at the same frequency as the stimulus.

The OAE, clinical tool, provides several as а advantages, hitherto not possible using other contemporary tools for the purpose (Martinet al., 1990). First this test is objective in nature, doesn't require patient co-operation for it to be administered. Thus it may be conveniently used for measuring the hearing acuity of young children including neonates. While the time taken to test a patient using DPOAE varies with the exact procedure used for measurement; but longer than that required for tympanometric measurements. Major advantage of DPOAE is it gives highly precise frequency specific information (Anova et al., 1993). Since DPOAE are emitted at a known frequency, related to stimuli, it helps in determining the exact place on the basilar membrane which responds to 2 known stimuli. It is difficult to obtain frequency specific information using BSERA.

Similar to DPOAE, TEOAE is also present in 98% of the ears of normal hearing individual (Bonfils et al., 1990). In addition, they have highly individual and repeatable spectra, suggesting their applicability in long term monitoring of an individuals cochlear status.

In short clinical implementation of EOAE are -

- Screening for peripheral auditory system dysfunction in newborn babies and infants.
- 2. Separating the cochlea and neural components of sensorineural hearing loss.
- 3. Monitoring the effects of noxious agents such as ototoxic drugs and intense sound on cochlea.
- 4. Assessing fluctuating hearing loss with or without therapeutic regiment.

Need for the study

The origin of the OAE is believed to be the hair cells, specifically the outer hair cells (OHC) (Davis, 1983; Zwickes, 1984). Many pathologies causing hearing loss, such as NIHL, ototoxicity etc. are known to selectively damage the OHC. Hence, in the these cases a measure of DPOAE and TEOAE may indicate the severity of damage of OHC directly (Wier et al., 1988; Probst, et al., 1993). This has opened up possibilities of using the OAEs to measure the place and extent of damage of OHC on the basilar membrane, such as in patients exposed to noise.

One of the clinical application of EOAEs is screening for peripheral auditory dysfunction. It has been reported in literature that TEOAEs cannot be recorded if the hearing loss is more than 30 dB HL (Robinette, 1992). Initially Kemp reported that TEOAEs were absent when the hearing loss was greater than 30 dB HL. However, later Norton and Stoves (1994) observed the TEOAEs could be present in patient with hearing loss up to 50 dB HL. Harris (1990) reported that DPOAEs were absent in ears with hearing loss greater than 50 dB HL. In some cases DPOAE could be detected with hearing loss up 70 dB HL (Suckfull, et al., 1996). However a very few studies have compared DPOAE and TEOAE in the same subjects. So this study was deviced with the aim to compare the DPOAE and TEOAE results in subjects with sensorineural hearingloss.

The present study also aimed at checking the efficiency of DPOAE/TEOAE in differentiating hearing-impaired

individuals from those with normal hearing when different DP-NF criteria were used for detection of presence of emission.

A review of that criteria used to literature shows determine the presence/absence of DPOAE is not same in all the studies. Lonsbury-Martin et al. (1990); Harris (1990)have reported that amplitude of distortion product should be above noise floor for detection of dB DPOAE. at lest 3 However Probst et al. (1993) used 4 dB criteria for detecting the presence of DPOAE. Gorga et al. (1992) observed DP/NF difference of 8-12 dB differentiates between subjects with normal hearing and hearing loss.

Thus the following null hypothesis were tested in this study ;

- There is no correlation between puretone threshold and amplitude of DPOAE in subjects with sensorineural hearing loss.
- There is no correlation between puretone threshold and amplitude of TEOAE in subjects with sensorineural hearing loss.
- 3. There is no change in incidence of DPOAE in subjects with sensorineural hearing loss when different DP-NF criterion were used for detecting the presence of DPOAE.

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- 4. There is no change in incidence of TEOAE in subjects with sensorineural hearing loss when different TEOAE-NF criterion were used for detecting the presence of TEOAE.
- 5. There is no correlation between amplitude of TEOAE and DPOAE in subjects with sensorineural hearing loss.

REVIEW OF LITERATURE

Ever since OAEs were first defined by Kemp (1978, 1979), there has been growing interest in their clinical application to the problem of identifying the presence of hearing loss. There are increasing number of papers describing Distortion Product Otoacoustic Emissions (DPOAEs) and TEOAE from impaired ears (eg.Bonfils and Uziel, 1989; Kemp, et al., Probst et al., 1987; Bray and Kemp, 1987; Collect, 1991; Harris, 1990; Martin et al., 1990a; Martin et al., 1990b; Lonsbury-Martin and Martin, 1990; Nelson and Kimberley, 1992). In general, the results of these investigators show that EOAE are present in the vast majority of normal hearing subjects and are absent in subjects with hearing loss once it is exceeds 30 to 50 dB HL. It has been argued that within an individual ear, OAEs are present from region of normal hearing but absent from regions of hearing loss (Kemp, et al, 1990; Martin et al., 1994; Martin, et al., 1990b).

Both TEOAE and DPOAE represent an objective measurement of the active micromechanical function of outer hair cells of the inner ear. OAE can appear spontaneously. For clinical purposes they are evoked either by transient stimulus (TEOAE) or are measured during bitonal stimulation as the so called

intermodulation products (DPOAE). Both TEOAE and DPOAE measurements are now becoming part of clinical routine.

A. TEOAE AND DPOAE IN NORMAL HEARING SUBJECTS

1. PREVALENCE/OCCURRENCE

TEOAE

TEOAEs are measurable in essentially all individuals with normal middle ears and normal cochlea (Kemp, 1978; Johnson and Elberling, 1982; Grauderi, 1985; Alexander and Brown, 1986; Probst, 1986). The existence of emissions in all normal ears makes it a sensitive tool to detect even minor changes in the hearing status (Norton and Nedy, 1987; Bonfils and Piron, 1988).

Though 100% occurence has been found in adults, it is slightly lower in neonates and infants Bonfils et al., (1990) measured TEOAEs in neonate ranging from 2 hours to 4 days, 98% of the tested ears had emissions. There was no significant difference in the occurence between one and four days postpartum, but the occurence increased within the first 24 hours. Kok, Van Zanten and Brocars (1992) and Vohr, et al., (1993) observed a 100% increase in ears with emissions when the ears were first tested 3 to 51 hours after birth and repeated at least 24 hours later. Engdahl et al., (1994) observed TEOAEs in 96% of the tested ears of 3 to 4 days old infants. Delaying testing until after the first postnatal day resulted in a 13% higher pass rate.

Gender difference has not been found in the occurence of TEOAEs (Kok, et al., 1982).

Johnsen and Elberling (1982) and Coren and Habestain (1990) reported that the interear variability of TEOAE was the same as intersubject variability. Therefore TEOAEs of each ear are statistically relatively independent.

DPOAE

There is growing evidence that DPOAEs are a property of all normally hearing human ears, Kemp et al., (1986) reported DPOAEs in all 14 normal ears they examined. With appropriate stimulus parameters, DPOAEs can be measured in nearly all normally hearing ears (eg. 98% of 113 ears, Hauser and Probst, 1989, 100% of 44 ears; Lonsbury-Martin, et al., 1990; 100% of 10 ears Zwicker and Harris, 1990). These findings indicate that DPOAEs can be recorded in well over 90% of normal ears. The frequency range within which acoustic distortion products are reliably detected is between 1 and 8 KHz with respect to the geometric mean of fl and f2 stimuli (Lonsbury-Martin, 1990).

2. AMPLITUDE

TEOAE

The amplitudes of TEOAEs depend on stimulus level as well as on the number and frequencies of innate dominant emissions. Moreover, emission amplitudes are also dependent upon the frequency response of both the middle ear and the recording system.

A straight forward peak-to-peak amplitude of a TEOAE is useful only in the cases in which TEOAE are dominated by a single frequency. Amplitudes are usually expressed in technical jargonas sound pressure level (Kemp, 1978; Zwicker, 1983a) or power spectra (Probst, et al., 1990).

DPOAE

The amplitude of DPOAE is dependent on several factors like level of primaries, frequencies of primaries, ratio of F2/F1 and innate properties of each ear. Similar to other OAEs, DPOAEs amplitudes increase if coinciding with SOAEs (Wilson, 1980c; Wit et al., 1981; Schlotlz, 1982; Furst, et al., 1988; Wier et al., 1988. Harris (1990) demonstrated that largest amplitudes occurred with F2/F1 ratios between 1.18-1.2. F2/F1 ratio of 1.22 is recommended for eliciting best DPs between 1-4 KHz.

3. LATENCY

TEOAE

TEOAEs appear in human ear canal with a specific latency that depends on the frequency of the emission. For example, high frequency stimulation elicits TEOAEs with shorter latencies than those evoked by low frequencies (Lonsbury-Martin, 1990).

Measurement of TEOAE latencies in absolute terms is difficult due to contamination of the beginning of the TEOAE by the "stimulus tail" and it is to be understood that determining the beginning of a response in multi frequency event such as TEOAE is methodologically difficult (Anderson, 1980; Norton and Neely, 1987). One of the widely used method is the 'group delay' (Johnsen and Elberling, 1982). This 'group delay' measures the point in time when the energy of the TEOAE reaches a maximum relative to specific frequencies, the actual beginning of a frequency component is undetectable.

The TEOAE-latency values reported in literature (Kemp, 1978; Rutter, 1980; Wilson, 1980; Schoth, 1982; Norton and Neely, 1987) range from 10-16 ms for frequencies around 1 KHz.

DPOAE

The latency of DPOAEs can be defined by phase measurements. A systematic relationship between phase and Distortion Product F2/F1 ratios were noted by Wilson (1980c) and Kemp and Brown (1983) in that latency was much shorter with high rather than with low ratios. Wilson (1980) reported a latency of about 1/2 a cycle or <2ms for f2/f1 ratios of 1.3 and 2 1/2 cycle or around 3 ms for ratios of 1.1 Kemp (1986) found that phase are nearly constant with slow frequency sweeps of the primaries at a fixed F2/F1 ratio of about 1.3, thus indicating a very short group latency. Group latencies increased upto 8 ms with decreasing F2/F1

ratios. In addition frequency appeared to decrease slightly with increasing stimulus level.

4. DETECTION THRESHOLD

TEOAE

Detection thresholds of TEOAEs are often lower than their corresponding psychoacoustic thresholds (Kemp, 1978; Wit and Ritsma, 1979; Zwicker, 1981 a, b; Johnsen and Elberling, 1982 a, b; Probst et al., 1986; Bonfils et al., 1988 a). These observations are consistent with the notion of a mechanical, preneural origin of TEOAEs (Kemp, 1978).

The visual detection threshold, however is influenced by the frequency content of the TEOAEs. The presence of highly tuned emission generators, such as synchronised SOAEs, imply longer duration. TEOAEs and purer waveforms, presumably leading to lower detection threshold (Lonsbury-Martin, 1990). Additionally, less energy is needed to phaselock SOAEs than to evoke emissions not already present (Wit and Riksma, 1983 a).

In a study examining TEOAEs in older individuals, age was shown to influence TEOAE-detection thresholds (Bonfils et al., 1988 a). They observed that, when related to the subjective click threshold, the detection thresholds, when associated with normal hearing levels had similar values of around 0 dB HL, upto the age of 40 years, and around -5 dB SL upto 30 years of age. For older individuals TEOAE thresholds increased linearly at a rate of about 8 dB HL/decade. A significant reduction in prominent emission frequencies was measured only in individuals above 50 years of age. These concluded that factors other than a broader tuning of responses may contribute to the observed age-related increase in TEOAE threshold.

DPOAE

Detection 'thresholds' for DPOAEs depend almost entirely on the noise floor and the sensitivity of the measurement equipment. Lonsbury-Martin, et al., (1990 a) reported detection thresholds that were 3 dB above the noise floor at about 35-45 dB SPL, for DPOAEs between 1-8 KHz. However much lower 'thresholds' down to 5 dB SPL, have been determined when measuring near or at strong fixed places of emission frequencies (Wilson, 1980c, Schloth, 1982; Burns et al., 1984; Wier et al., 1988).

5. RESPONSE/GROWTH RELATIONSHIP

TEOAE

Because of methodological difficulties, the details of the response/growth or input/output functions reported in the literature differ considerably. As the first I/O function for TEOAE was reported by Kemp (1978), who related the amplitude to the square root of the stimulus level. Α constant growth of TEOAE is seen between 10-20 dB HL stimulus level and a pronounced saturation above this level was later observed by other investigators (Wit and Ritzma, 19791 Kemp and Chum, 1980b; Wilson, 1980 a); Schloth, 1982; Zwicker, 1983 a). Major difference between individual ears were emphasized in many of these studies (Wilson, 1980 b; Wit et al. 1981; Zwicker, 1983 a).

Indeed, Zwicher (1983 a) discovered that I/O functions, with the above mentioned characteristics concerning linear growth and pronounce saturation, were mainly exhibited by ears without SOAEs. Thus it is possible that at stimulus levels, spontaneous emissions interfere nonlinearly with TEOAEs so that liner growth for example, would be uncommon in ears with SOAEs.

There is however, general agreement that non-linear growth of TEOAE occurs for stimulus levels >20-30 dB SL. The linear growth 'function' for TEOAEs is maximized by equipment that make use of 'linear cancellation' technique for recording TEOAE (Kemp et al., 1990).

DPOAE

Wilson (1980c) and Schloth (1982) each measured I/O functions for 3 ears. Both investigators examined DPOAEs at strong emission frequencies and made comparisons to psychoacoustic measurements. Whereas Wilson (1980c) obtained widely differing functions for several F2/F1, ratios, Schloth (1982) measured a slope of one when both primaries were at the same level. No clear differences between psychoacoustic and otoacoustic findings were noted by these workers. Later, averaged I/O functions from 44 normally hearing ears were reported by Lonsbury-Martin et al., (1990 a). With respect to geometric mean frequency of the primaries, the functions were generally steep, at the lower frequency of 1 to 2 Hz (slope <0.8) than at the higher frequency of 3-8 KHz (slope = 0.8 - 0.95) where the I/O slope didn't quite reach to unity one.

6. EXTERNAL INFLUENCES

TEOAE

The expression of TEOAE may be influenced by many factors which has been studied since the past decade. This is because the TEOAE was recognized as a potentially useful clinical tool and the search was on for the optimum testing conditions.

Some recent studies have concentrated on instrumentation related factors which effect the TEOAE response (Zwicker, 1990; Lutman et al. 1994; Thonton, et al., 1994). Zwicke (1990) described how the acoustical impedance of the probe could influence both the amplitude and waveform of the emission. Lutman et al., (1994) coroborated the above study and demonstrated that the acoustic characteristics of the probe could modify the measured response. Further, if the probe acted as a reactive load, oscillation at a particular frequency was seen. This could be confused with a TEOAE response even though no significant oscillatory behaviour would occur without the reactive load provided by the probe.

Thornton, Kimm, Kennedy and Cafarelli-Dees (1994) identified the instrumentation related factors which affect the TEOAE response. Of these, the form of stimulus, the characteristics of the microphone, amplifiers and filter were identified as the major ones. Data were collected from 64 neonates (3 days post partum) on ILO88 (Otodynamics Ltd., U.K.) and on POEOMS system (Institute of Hearing Research, Nothingham, U.K). The ILO88 TEOAES consistently had larger high frequency components and higher correlations between repeat recordings.

The influence of various stimulies parameters and their efficacy in evoking emission has also been studied (Wit et al., 1979; Zwicker, 1983; Elberling, et al., 1985; Norton and Neely, 1987; Thornton, 1993). Wit et al., (1979) investigated the influenced of tone burst frequency on the emission amplitude. At the same stimulus level stimuli of higher frequency generated much smaller emissions. They found similar results with filtered clicks (Wit et al., 1981). Elberling (1985) evaluated TEOAEs in response to various tonal stimuli in normal hearing adults (48-90 years). They noted that changing the stimulus frequency had only a minor effect on the power spectra. The click was a better stimulus than the tone burst since it gave wider frequency information.

Probst et al., (1986) studied the efficacy of different stimulus types in eliciting OAE. The click (0.1 ms pulse) and tone burst (0.5, 1, 1.5, 3 KHz) were used. Two patterns were observed (i) 18% of the ears showed short broad band click evoked OAE emissions with less than 20 ms latency (ii) 82% of the ears had emissions lasting greater than 20 ms. They also found the click to be a better stimulus than tone burst.

Zwicker (1983) studied the relationship between stimulus intensity and the emission. The two were proportional upto 20 dB SL above after which the emission level saturated. Wit et al. (1979) noted that at low stimulus levels, the relation between stimulus level and response level is approximately linear. Norton and Neely (1987) investigated the relation between tone burst frequency (0.5 to 2 KHz) and intensity. The saturation was noted at all curve the frequencies. The level at which saturation occurred was lower at higher frequencies. In addition, the spectra of the TEOAE resembled those of the evoking stimuli.

In an attempt to hasten the testing procedures high repetition rates have been experimented on (Thornton, 1993; Elberling, 1994). Thornton (1993) noted the effect of

varying click repetition rate (338 to 840/s). A higher repetition rate did not contaminate the response. It also reduced the test time to a few seconds in adults 4 neonates.

Stimulation of the contralateal ear also has aneffect on the OAE emission. The results of various studies on contralateral acoustic stimulation has been summed by up Collet et al., (1994) as follows :

- 1. Alteration (mainly a decrease) of emission amplitude.
- 2. Alteration of the response spectrum (upward shift in the frequencies) especially with spontaneous emissions.
- 3. Alteration of phase.
- 4. The effect depends on the intensity of the contralateral stimulus.
- 5. The effect is inversely proportional to the intensity of ipsilateral stimulation.
- 6. The amount of suppression increases with the band width of the noise, especially for noises centred around 1-2 KHz. Wide band noise had greater suppressive effects than narrow band noise.

It is of utmost importance to identify the patient related variables which may affect the response. Some of the variables that have been studied and their results are summarized here :

- Changes in the posture of the head and the body influenced the TEOAE emission (Antonelli and Grndore, 1986; Wilson, 1980). However, contradictory results were reported by Johnsen et al., (1982).
- Mean TEOAE amptitude was larger for women by 2.8 dB (Robinette, 1992)
- As the age increased the response amplitudes decreased in adults (Kemp, 1980; Collet, et al., 1990; Kemp, et al., 1990).
- Presence of spontaneous OAE increased the amplitude of TEOAE (Meric, et al., 1993).
- Middle ear dynamics influence TEOAE emission i.e. TEOAEs are most distinctly detected at the middle ear resonant frequency in normal subjects (Wadwa, 1993).
- Ear canal pressure (negative or positive) reduced the TEOAE responses (Robinette, 1991).
- Ototoxic drug acetyl salicylate induced SN hearing loss where the threshold got elevated and the OAE response pattern was altered (Johnsen, et al., 1980).
- Exposure to noise reduced TEOAE amplitude in frequency range of 2 to 4 KHz (Holtz, 1993).

- Auditory attention had no significant influence on emission levels (Meric, et al., 1993).
- General anasethesia produced a reduction in TEOAE amplitudes in normal hearing subjects (Hauser et al., 1929).

DPOAE

Similar to TEOAE, DPOAE is also effected by many factors related to stimulus, instrumentation and subjects.

Lonsbury and Martin et al., (1991) studied the influence of aging on the generation of DPOAEs and reported that when compared to emissions in young ears, DPOAEs accurately tracked the systematic deterioration of HF hearing in aging individual.

Osterhammel, et al., (1993) studied the influence of ME transmission on DPOAEs and concluded that amplitudes of the DPS depend on optimal transmission through the ME and that measurement of DPOAEs should always be preceded by determination of ME pressure. Gaskill, et al., (1990) investigated the dependence of DPOAE level on stimulus parameters and found that the frequency ratio F2/F1 at which DPOAE level is maximal varies only slightly across the frequencies and subjects. As the level of one stimulus is increased relative to the other, DPOAEs grow, saturate and in most cases show a bend over. Maximum distortion was seen when L1 exceeded L2.

Giomfrone et al., (1993) studied some effects of tonal fatiguing on DPOAE and reported that in DP; growth modifications takes place within a period of 5-7 min. and depends on frequency of fatiguing stimulus and on the closeness between SOAE and DP place.

The influence of aspirin on DPOAEs was examined by Wier et al., (1988). In these studies, aspirin ingestion clearly resulted in less amplitude reduction of DPOAEs than the increments observed in SOAEs in the same ears. Additionally, the reductions in DPOAE amplitude were less pronounced or even absent at higher primary tone levels. Thus the dissociation of the response to aspirin between SOAEs and DPOAEs supports the notion of а different generation mechanisms for each emission type.

Suppression measurements were reported by Brown and Kemp (Brown and Kemp, 1983, 1984; Kemp and Brown, 1983 a) and the general forms of the suppression-tuning curves were similar to those noted for other OAE classes. For 2F1-F2, the maximum of suppression was always in the frequency range between the primaries and not around the dp frequency, thus, indicating that the generation of DPOAEs occurs primarily as a frequency place between the primaries. However, in most instances, more complex tuning curves with several minima were measured. Additionally, Brown and Kemp (1983, 1984) noted reasonably similar suppression tuning curves between human and gerbil ears.

B. TEOAE AND DPOAE FINDINGS IN CLINICAL POPULATION

For clinical use, it is particularly important to establish reliable relationships between TEOAEs and hearing disorders in patient populations. They are currently under investigation as an objective tool for evaluating hearing, particularly in the screening of hearing and threshold estimation in difficult to test subjects.

In the presence of hearing loss, TEOAEs has been shown to decrease in incidence as hearing thresholds increase (Kemp, 1978; Kemp, et al., 1986; Tanaka, 1987; Bonfils, et

al., 1988; Stevens, 1988; Gartner and Moutin, 1989, Tanaka, 1990).

Generally, if the hearing loss exceeds 40-50 dB, an emission cannot be evoked to a transient stimulus. Kemp et al., (1986) reported that the upper limit is 30 dB HL for a 80 dB SPL, 80 /us click. At higher stimulus levels the limit appears to be 50 dB HL (Lonsbury-Martin, et al., 1990).

Stover and Norton (1992) reported good correlation between psychophysical thresholds and TEOAEs for the same stimuli. Responses to suprathreshold stimuli decreased as hearing sensitivity decreased. However in mild to moderate losses, TEOAEs may appear to be within normal limits for high level stimuli. If one is interested in sensitivity, one may need to measure emission at several stimulus levels and determine the emission threshold. If interested only in the cochlear reserve or integrity, one may use a single high level stimulus.

There are exceptions to the above results and cases have been reported with normal hearing showing absence of TEOAE and with hearing loss showing TEOAE responses. Lutman et al., (1989) reported a eleven year old child with profound SN hearing losses showing TEOAE. They hypothesised the lesion to be retrocochlear pathology, but did not conduct anv confirmatory tests. Prieve, et al., (1993) reported findings of TEOAE in a 33 year old woman with severe to profound sensorineural hearing loss. They assumed a group of surveying OHC in some region of the cochlea with corresponding IHC being intact to be the source of the emission. They also added that hearing loss may be due to neural damage. They concluded that TEOAE are a true indicator of site of lesion.

Collet et al., (1989) reported statistically significant correlation between TEOAE threshold and hearing loss at 1 KHz. They concluded that the presence of TEOAE indicates middle frequency functional integrity of the OHC of the organ of corti. Absence of TEOAE is hard to interpret. Stover and Norton (1992) studied the relationship between the audiogram and tone burst evoked OAEs at the octave frequencies at 80 dB SPL in a young adult with sensorineural hearing loss. The amplitude was measured. It provided a good snap shot of the audiogram configuration. The click evoked OAE contained energy from 1000 to 3500 Hz.

Attempts have also been made to correlate TEOAE with configuration of audiogram where audiogram shows frequency

bands of normal hearing emissions are usually evoked at those frequency by a click stimulus. With high frequency loss emissions are usually obtained up to the frequency of first loss. There is strong evidence for a high degree of frequency specificity in OAE.

investigated whether a simple Lind and Landa (1989) technique with a simple repeated recording at fixed stimulus intensity could give information enabling differentiation between high frequency and low/medium frequency hearing losses. The latency was measured. They concluded that it can be used to evaluate the presence of low/medium frequency hearing loss exceeding 40 dB HL. Collet et al., (1992) reported that the TEOAE spectrum and sensorineural hearing loss are significantly positively correlated. They added that however, it is not possible to establish an audiogram by spectrum analysis.

Johnson (1993) studied the relationship between audiogram configuration, pure tone average and the recurrence of TEOAE. When the audiogram was flat not a single subject with thresholds below 40 dB HL showed absence of TEOAE, and not a single subject who showed absence of TEOAE had thresholds below 30 dB HL. In sloping audiogram pattern the

thresholds at 1 and 2 KHz were considered important for generating TEOAE.

Robinette (1992) studied TEOAE parameters in case of flow and high frequency cochlear hearing loss. In pure SN hearing loss there was a linear relationship between the TEOAE threshold and the mean audiometric threshold for frequency between 1 KHz and 4 KHz (Bonfils, et al., 1986). Hence TEOAE thresholds could give information on the auditory threshold for mid frequencies (1-4 KHz). Nevertheless, this audiometric interest is strongly limited by the disparity of TEOAEs when the puretone thresholds for these frequency is greater than 30 dB HL.

It is felt by many researchers the TEOAEs can be used to detect unilateral losses, especially in infants where behavioural observation audiometry (BOA) does not indicate a unilateral loss. Tanaka et al., (1987) reported the inter-aural amplitude difference in TEOAE to be a useful indicator in unilateral hearing loss (Cochlear pathology) rather than the threshold value itself. They also noted a high positive correlation between interaural differences in unilateral functional hearing loss. Tanaka, et al., (1990) reported the mean inter-aural difference to be 35 dB HL in unilateral profound hearing loss. For example, a 10 years

old boy who had sudden deafness in the left ear after mumps had a mean audiometric threshold of 11.3 dB in the right ear and 70 dB in the left ear. The OAE threshold was 10 dBn HL in the right ear and 50 dBn HL in the left ear. Similar inter-aural difference were found in unilateral cases of Meniere's disease and cerebropontine angle tumours.

In cases of middle ear pathology TEOAEs may not be measurable because they are not effectively transmitted by the middle ear. Generally, if the air-borne gap for puretone thresholds exceeds 30-35 dB TEOAEs cannot be measured (Norton and Stover, 1994).

In otosclerosis TEOAEs have never been observed when the mean audiometric thresholds for 0.5 KHz and 1 KHz were greater than 30 dB HL. Bonfils and Troloun (1989) observed that after stapes surgery, TEOAEs appeared in cases whose audiometric threshods were less than 30 dB HL.

In serous otitis media, TEOAEs were recorded only when auditory thresholds were lower than 30-35 dB HL (Bonfils, Uziel and Nancy, 1988). When TEOAEs were recordable, the emission spectrum gave additional information, that is only high frequencies (above 1.5 - 2 KHz) were present. This frequency pattern seemed specific to E.T dysfunction.

Engdahl et al., (1994) studied the reproducibility and short-term variability of TEOAE (intra-subject) in term of amplitude and the possible effect of diurnal middle ear pressure variations noted. TEOAE amplitudes were observed to vary with naturally occurring middle ear pressure changes. Hence they suggested combining tympanometry with TEOAE recording, thus making it possible to make measurements at peak acoustic admittance. This is especially important when monitoring small changes in cochlear function by means of TEOAES.

Ever since the cochlea was identified as the source of OAEs, it is considered a reliable predictor of SN hearing loss especially cochlear pathology (Kemp, 1978; Kemp, et al., 1980; Harris, et al., 1982; Slover, 1982; Bonfils et al., 1989; Norton, et al., 1990).

Bonfils, et al., (1989) studied the clinical applicability of TEOAEs as objective indicators of cochlear pathology in the range of 14 to 74 years. They found it to be a reliable technique for the objective study of normal micromechanical activity within the cochlea and for the

detection of subtle changes in cochlear diseases. Thus findings were supported by Norton, et al., 1990). Norton, et al., (1990), opined that TEOAEs, can be used as a screening tool for cochlear dysfunction across individuals and to monitor changes in cochlear status oval time within an ear.

Tanaka et al., (1990) found the sensitivity of the test to be 96% for cochlear losses. They reported that TEOAEs were useful in predicting susceptibility to noise induced hearing loss. The scatter plot shows the relationship between psychoacoustic threshold and OAE threshold in 15 ears of noise-induced hearing Loss.

Kemp (1982) measured TEOAE in young adult after exposure of 80 dB SPL broad band noise for 1 hour. The amplitude of TEOAE response was inversely related to the degree of temporary threshold shift (TTS) and increase non-linearly with time post exposure. Similar results were reported by Norton and Mayers, (1990). The correlation was found to be In ears with notch type hearing loss that represented 0.85. the initial stages of NIHL, OAE were detected at one or two octave lower that of the neural dip frequency. Thus, the OAE threshold was not a crucial parameter. But the duration of OAE within 20 msec. after stimulus onset was prolonged according to the increases of stimulus intensity and this appeared more prominent in the ears with dip type hearing loss.

The findings regarding emission in Meniere's disease are equivocal. Elevated detection emission thresholds have long been reported by Johnson and Elberling, 1982; Rossi, Solero and Rolando, 1989. Bonfils, et al., (1988) suggested that TEOAEs could be used clinically for staging menieres disease Norris, et by recording glycerol induced changes. al., (1990) reported absence of emissions in endolymphatic hydrops induced in chinchillas. Rupture of the Reissner's membrane caused the emissions to disappear. But this may be due to trauma caused to the organ of corti while inducing membrane Tanaka, Suzuki rupture. and Tsueno (1990) reported improvement in TEOAE thresholds with glycerol administration. In contrast to these studies, Harris, et al., (1992) reported that TEOAE responses are not affected in menieres disease. They studied patients with menieres disease in the age range of 20-70 years. Clicks and tone bursts were used to elicit emissions. With clicks, emission were recorded in 26 out of the affected ear, and in 29/31 in the 31 subjects in unaffected ear. With tone bursts as stimuli, emissions were recorded in 28/31 subjects in the affected ear and 30/31 in the unaffected ear.

Tanaka and Suzuki (1990) found interaural amplitude differences in a case of unilateral cerebellopontine angle tumour. Robinette (1992) evaluated 61 acoustic neuroma patients pre-operatively and measured TEOAEs in 31 of them. For 19 of these patients, TEOAEs were expected because hearing thresholds for most frequencies were within the normal range. But 12 patients who had mild to moderate hearing losses showed TEOAEs. He concluded that TEOAEs were positive suggesting in retrocochlear pathology for 20% of these patients.

Johnsen and Elberling (1980) observed thresholds and altered response patterns when a SN hearing loss was inducted by ingesting acetyl salicylate. The TEOAEs reappeared after the drug intake was stopped and hearing had returned to normal.

However the clinical applicability of TEOAEs to detect ototoxicity seems to be limited by the frequency range of TEOAEs. In ototoxicity the inner ear damage predominantly affects the basal turn of the cochlea (that is high frequency). As TEOAEs are optimally suited to observe mid frequency activity of the cochlea (1-4 Hz) they don't seem adapted to an early detection of ototoxicity, which could be achieved with more efficiency by high frequency audiometry.

Central auditory disorders has not been researched extensively, mainly since OAEs are known to originate from the cochlea and the role of the CNS is presumed to be negligible. Bonfils, et al., (1990) observed that evoked OAE are always present in infants with lesion involving the central nervous system. Lafreneir, et al., (1991) attempted to characterize the emission from neonatal and infant subjects at risk for hearing loss TEOAEs and DPOAEs were of low amplitude or absent in subjects with suspected central hearing loss.

The feasibility of using TEOAEs as an inpatients check of hearing status in children recovering from bacterial meningitis in the age range of 3 to 16 years was studied by Fornum, et al., (1993). They found a 100% specificity. All those who failed the test had subsequent hearing loss.

TEOAEs may also be valuable in identifying functional hearing loss. Robinette, (1992) found TEOAE useful in the 12 cases that he studied. He described a 41 years old man with a bilateral severe SN hearing loss speech reception. Thresholds were 35 dB bilaterally and word recognition scores

were normal. His behavioural responses to stimuli were delayed. TEOAEs were present bilaterally and following careful reconstruction, normal behavioural thresholds were obtained.

incidence The population of severe and profound congenital SN hearing loss is between per one and two thousand (Pecham, 1980; Davis and Wood, 1992). The importance of early detection of affected infants and their habilitation is widely acknowledged. The speech intelligibility of infants fitted with hearing aids before the age of six months has been reported to be superior to the of infants fitted after this age (Markides, 1986) and oral language production abilities are improved by early intervention (Raun-Kahawan and Davis, 1992). There is also evidence that a lack of experience during early infancy can result in a permanent loss of hearing sensitivity (Fisch, 1990). Therefore there is an urgent need to consider screening of all infants for auditory impairment. Results from some large clinical trials (Maxon, Norton, White and Breheus, 1991; Volur, et al., 1994) indicate that TAOAEs can be a rapid, sensitive tool for detecting hearing loss in both full term and at risk new borns.

The use of OAEs for screening hearing function in neonates and infants has been suggested by many investigators (White, et al., 1980; Kemp, et al., 1981; Tanaka, et al., 1986, 1989; Bonfils, et al., 1991; Baldwin, 1992; Fortum, et al., 1993, Meredith, et al., 1994; Englahl, et al., 1994). The problems associated with behavioural tests, high risk registers and ABR response testing give impetus to the research on the feasibility of OAE for infant screening.

Hemtes, et al., (1994) studied the feasibility of OEA detection followed by ABR as a universal neonatal screening test for hearing-impairment. The procedure was feasible in 95% of the babies born in a hospital. It required two testers working in 12 hours shifts to screen all babies using both the tests. The specificity of OAE was 70%, only 8.5% of the babies (who had bilateral failures) required ABR confirmation.

Recently the emphasis has shifted to longitudinal data to determine specificity and clinical applicability. The Rhode Island hearing assessment showed promising results (Volus et al., 1990) of the TEOAE as a screening tool after creening over 12000 infants over 5 years. Meredit, et al., 1994) screened high risk neonates for 5 years between 1988 and 1993. They found an overall failure rate of 27.7%. The

failure rate for low birth weight babies was higher (45.9%) than for babies in other at risk categories. Sensitivity was 100% and the specificity was 72.3% (There was a high false-positive rate. This higher failure rate in premature infants was earlier noted by Stevens (1990) and Uziel, et al., (1991).

To summarize, TEOAE are now widely used as objective, efficient and non-invasive method for screening auditory function in infants.

DPOAE

A review of literature shows that only a few results of DPOAEs in pathological ears have been published. Kemp, et al., (1986) reported 3 examples of DPOAE measurements in ear with high frequency hearing losses. In these cases, the DPOAE amplitudes were significantly smaller than normal values at frequencies where hearing thresholds were better than 30 dB HL. However despite mild hearing loses, DPOAEs were still present, in most instances. Additionally, the relationships between the DPOAE and hearing loss frequencies were not always straight forward. Similar results were also reported by Harris, et al., (1990), Smurzynski, et al, 1990; Nelson, et al., 1992).

Other examples of DPOAEs in patients were reported by Lonsbury-Martin and Martin and their associates (Lonsbury-Martin and Martin, 1990; Martin, et al., 1990b). "These workers showed that acoustic distortion products objectively detected a 20 dB HL noise induced impairment in one individual and a 10 dB improvement, followed a glycerol test, in the hearing of another subject suspected of having menieres disese. Additional examples of usefulness of the frequency specific DPOAEs to track the boundary between normal and abnormal hearing in more severe cases of noise damage and in individuals with heriditary hearing loss, due to either autosomal dominant or recessive factors, have also been presented by these investigators (Lonsbury-Martin and Martin, 1990; Martin, et al., 1990b; Ohlms, et al., 1990, 1991). Moreover other examples were presented in these reports and illustrating the distinction between acoustic neuromas, that cause damage to sensory cells through harmful pressure on either the vascular or efferent supply to the truly effect only retrograde cochlea, and tumours that symptoms. Further manifestations of several possible subtypes of MD involving either cochlear hair cells or peripheral elements central to the OHCs were also provided.

Finally, the ability of DPOAEs to track the dynamic changes in OHC condition, as is sometimes observed in sudden idiopathic SN hearing loss may be illustrated as well.

In another study of DPOAES, Harris (1990) reported systematic results in 20 patients with high frequency loss. Specifically at high frequencies, DPOAE amplitudes and I/O functions were significantly different when compared to DPOAEs measured in normal subjects. He concluded that if hearing thresholds at predetermined frequencies were better than 15 dB HL DPOAEs were always detected. However emissions were absent or attenuated if behavioral thresholds exceeded 50dB HL. Additionally considerable variation was noted in the range of between these 2 threshold values.

Martin et al., (1990b) measured DPOAE in a noise-damaged individual with an asymmetrical high frequency hearing loss. They found that when the primaries were located in a frequency region associated with normal hearing threshold DPOAE could be measured. But when hearing thresholds were elevated above 40 dB HL no DPOAE was recorded.

In conclusion DPOAEs have significant potential for clinical application, although their relationship to hearing

threshold is not entirely clear, as yet, in all types of sensorineural disease. Moreover, even when the dysfunction is clearly limited to OHCs, as in the early stages of NIHL, some discrepancies between DPOAE and hearing thresholds occurs across individual (Martin, et al., 1990 b). Although some initial works have been performed in terms of identifying candidate values for the multitudes of parameters that need to be considered when eliciting DPOAEs, additional studies must be performed to establish variables that are most relevant to clinical testing. However the major advantage of DPOAEs his in their relatively, frequency specific measurement of evoked emissions that provides an objective complement to the conventional, pure tone audiogram.

METHODOLOGY

This study was taken up with an aim to compare the differences in the results of DPOAE and TEOAE in adult subjects with sensorineural hearing loss.

SUBJECTS

The subjects were adults registered at All India Institute of Speech and Hearing, Mysore with the complaint of hearing problem. A total of 30 patients (49 ears) presented with pure sensorineural hearing loss of different etiologies, were taken up for this study. These patients (21 male and 17 females) were in the age range of 18-35 years. Any middle ear pathology was ruled out otoscopic immittance by evaluvations. Special test such as tone decay test, suprathreshold adaptation tes, reflex decay test,auditory brainstem response were administered whenever indicated. Informed consent was obtained from all the subjects participating in the study.

The puretone average of subjects ranged from 16 to 90 dB HL.Goodmann's classification modified by Clark(1981) was used to categorise the subjects into different groups based on the puretone average thresholds. The abnormal hearing consisted of a variety of etiologies and pure tone audiogram patterns majority of it being flat hearing loss at 500 Hz, 1KHz, 2KHz frequencies (Flat = 40 ears, high frequency loss -4 ears, low frequency loss in 5 ears). All the subjects were classified under Goodmann's classification.

	Minimal	Mild	Moderate	Moderate - severe	Severe
No.of ears	6	12	8	10	13

INSTRUMENTS

The following equipments were used.

A. Pure tone audiometer

A two channel clinical audiometer (OB 922) with TDH 39 housed in MX/41 earphone and radio AV 71 BC vibrator was used to assess the behavioral thresholds of all the subjects. The audiometer was calibrated prior to the study as per the recommendations of the manufacturer (Appendix-I) B. Immittance audiometer

Immittance evaluation was carried out was using GSI-33 middle ear analyzer version 2. The audiometer was calibrated as per recommendations of the manufacturer (Appendix II).

C. Otoacoustic Emission Analyser

The distortion product otoacoustic emission and transient evoked otoacoustic emissions were also measured using a Bio-logic Scout Plus System with ER-10C Probe (Software version 1.22) in standard default operational mode. The 'Display type' controls the pattern of the measurement. It was set to 'DP gram'. This setting plots the DPOAE on a frequency vs. intensity graph, similar to an audiogram. The range of frequencies being tested was set to 500 Hz to 8 KHz. The acoustic stimuli presented were two primary tones with a frequency ratio f2/fl fixed at 1.20. Equal intensity of L1 and L22 of 70 dB SPL in the external ear canal was chosen. DPOAE was measured at frequencies given in Table-1. The frequencies Fl and F2, the corresponding distortion product 2fl-f2 are Geometric mean are given in Table-1.

fl (KHz)	f2 (KHz)	2f-f2 (KHz)	GM
415	488	342	450
855	1024	864	936
1660	2002	1318	1823
3296	3955	2637	3610
6665	8008	5322	7306

Table-1 Fl, F2 and Corresponding DP 2fl-f2 used for DPOAE measurements.

Since during puretone audiometry, the test was carried out at one point per octave, the same was done here. SN ratio was set to +30 dB. Over all noise level was set to -30 dB. Token buffer size was set to 2048, which is also the default testing. Noise side bands was set to '0' which indicated that the noise was measured only at the frequency of the DPOAE. The measured signal was considered as significantly different from the background noise if it was at least 3 dB above the average noise level.

The stimulus were 100 µs. rectangular pulses with a presentation rate of 4/second. The stimulus level varied from 64-100 mpa with a mean of 70 mPa. 8 samples per buffer and 128 sweep sets were recorded. The spectrum level range was 40 dBpSPL and the spectrum frequency range was 08 KHz. The stimuli were presented in blocks of four where 3 stimuli of one polarity were added to a 4th stimulus of opposite

polarity 3 times the amplitude so that the stimulus artifact was minimized.

The ER-10C probe with appropriate ear tip size was used. Each response was band pass filtered from 5656.900 Hz (low pass filter frequency) to 2000.000 Hz (High pass filter frequency) in order to reject artifacts. The artifact rejection threshold was 0.977 mPa. The responses were stored after completion of 256 averages.

TEST ENVIRONMENT

in air conditioned All measurments were made sound treated rooms where the ambient noise level was within permissible limits according to ANSI 1969. The noise level was measured with a sound level meter (Bruel and Kjaer 2209). The test room had adequate lighting and was at a comfortable temperature. The subjects were provided with a comfortable chair to sit on during the test.

TEST PROCEDURE

Initially the subjects were screened in a verbal interview for a history of otological disease, noise

exposure, ototoxic drug use, metabolic diseases associated with hearing loss and a family history of hearing-impaired. Pure tone test was carried out and subjects with sensori neural hearing loss theshold within 16 dB HL - 90dB HL were taken for this study.

They were tested for tympanograms and reflexes in both ears using (GSI-33) Middle ear analyser Version 2. Only subjects who had A-type tympanograms were included in the study.

DPOAE testing

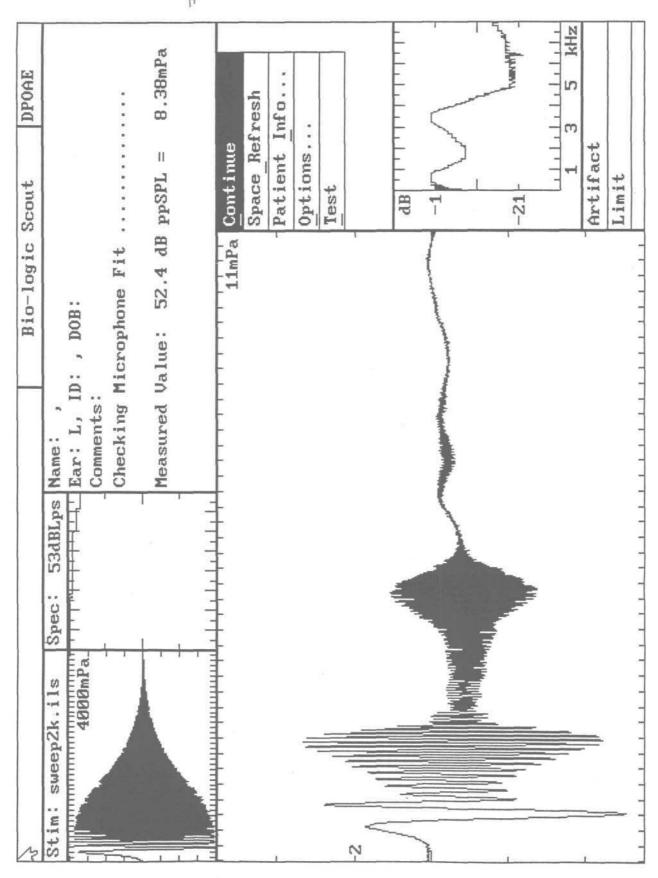
- (1) Check fit : In this phase, a transient stimulus (frequency sweep) is presented to the ears and the measured response is displayed as a spectrum and а waveform. A correct probe fit would give a waveform as shown in the figure-3.1. If such a waveform was not obtained, the probe was taken out, checked for debris and refitted. The 'check fit' phase was redone untill correct fit was obtained.
- (2) Calibration : After obtaining a correct fit, the stimulus sources were calibrated. In this phase the system response of each of the two loudspeakers necessary to

provide the stimulus was measured and displayed. If the response of the two overlapped, then it was calibrated. (Figure 3.2).

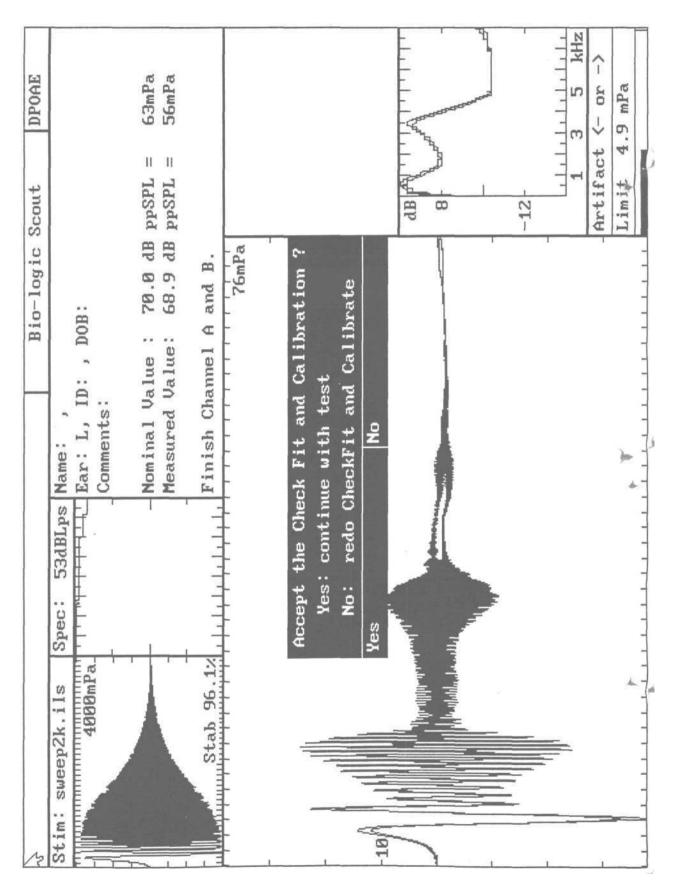
(3) Measurement : In this main phase of the test, the stimuli were presented and the Distortion Product emissions measured. The measures were done in accordance with the parameters, described earlier. The measures may then be displayed either as a DP-Gram in which the distortion product amplitude was shown as a function of frequency (Figure-3.3) or as a spectrum, where the final spectra of each test frequency may be viewed individually (Figure 3.4). Alternatively, the results may be studied as a 'Report' which includes a DP-gram and the values for various stimulus and DPOAE parameters (Figure 3.5).

TEOAE testing

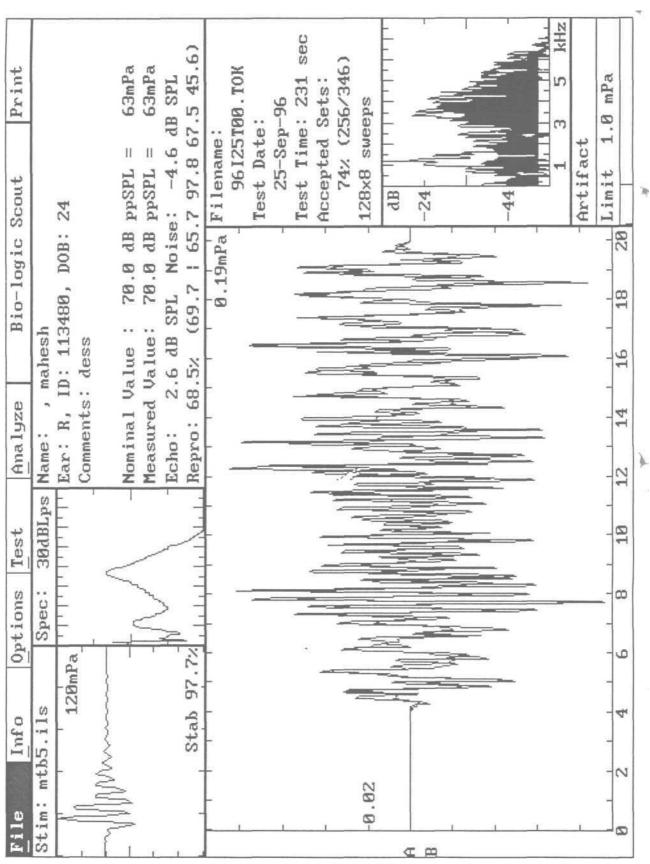
- (i) Check fit : A transient broad frequency stimulus was presented to the ear and the measured response was displayed. The fit of the probe in the ear canal was adjusted to obtain the flattest possible spectrum. (Fig: 3.6)
- (ii) Calibration : The attenuator was automatically adjusted to achieve the target output level. (Fig: 3.7)



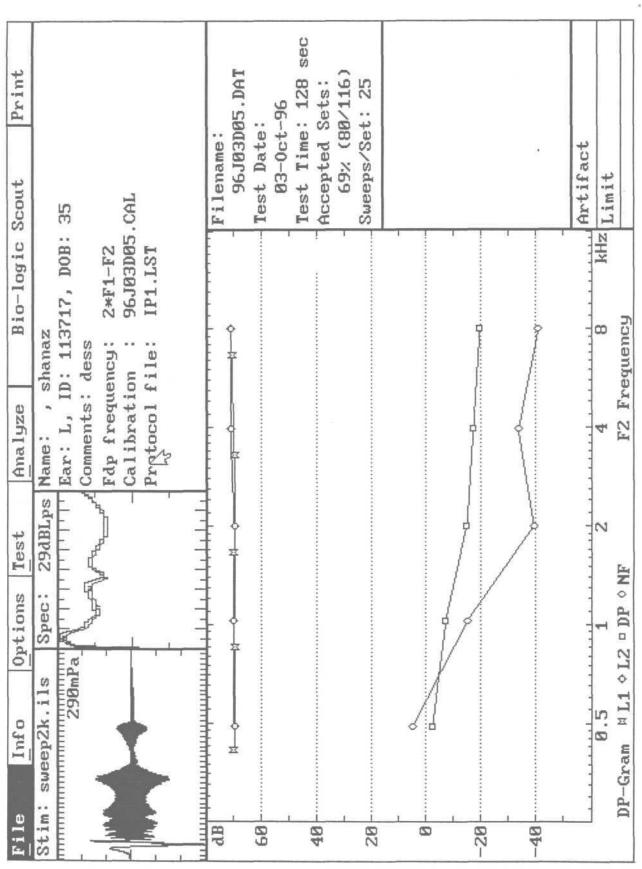
7161:2.1



F19: 32



719 : 3.3



7197: 3.4

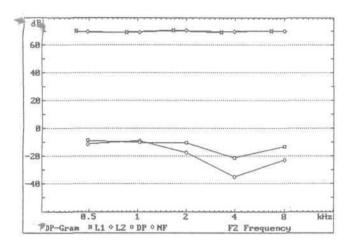
	1660 Hz, F2/F1 = 1.21 2002 Hz, Time = 8.2 s 1823 Hz, Fdp = $2 \times F1 - F2 = 1318$ Hz d Ualues: L2 DP NF DP-NF 71.2 9.1 -25.3 34.4 dB 71.2 9.1 -25.3 34.4 dB B=Back, F7=Swap, <-,->, Esc=Stop 96124D01.DAT Test Date: 24-Sep-96 76cepted Sets:
	Time = 8.2 s Fdp = 2*F1-F2 = 1318 Hz P NF DP-NF 1.1 -25.3 = 34.4 dB F7=Swap, <-,->, Esc=Stop F7=Swap, <-,->, Esc=Stop Filename: 96124D01.DAT Test Date: 24-96 Test Time: 240 Accepted Sets:
	Fdp = 2*F1-F2 = 1318 Hz P NF DP-NF .1 -25.3 34.4 dB F7=Swap, <-,->, Esc=Stop F7=Swap, <-,->, Esc=Stop Test Date: 24-Sep-96 Test Time: 240 Accepted Sets:
	P NF DP-NF .1 -25.3 34.4 dB F7=Swap, <-,->, Esc=Stop Filename: 96124D01.DAT Test Date: 24-Sep-96 Accepted Sets:
L1 67.6 	L2 DP NF DP-NF 71.2 9.1 -25.3 34.4 dB B=Back, F7=Swap, <-,->, Esc=Stop 96124D01.DAT Test Date: 24-Sep-96 Test Time: 240 Accepted Sets:
	71.2 9.1 -25.3 34.4 dB B=Back, F7=Swap, <-,->, Esc=Stop Filename: 96124D01.DAT Test Date: 24-96 Test Time: 240 Accepted Sets:
	B=Back, F7=Swap, <-,->, Esc=Stop Filename: 96124D01.DAT Test Date: 24-Sep-96 Test Time: 240 Accepted Sets:
	E
31-7	
	Test Time: 240 sec Accepted Sets:
	Accepted Sets:
31-M	26% (58/228)
	Sweeps/Set: 25
	40
	OF
	-40
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MINAALA NGA AAAAM AMA	WWW WWIIIIII
	XHZ XX

BIO-LOGIC OTOACOUSTIC EMISSIONS (OAE) REPORT

BIO-LOGIC SYSTEMS CORP. ONE BIO-LOGIC PLAZA MUNDELEIN, IL 60060 (800)323-8326

Patient: , asha Birthdate: 30 Comment: dess

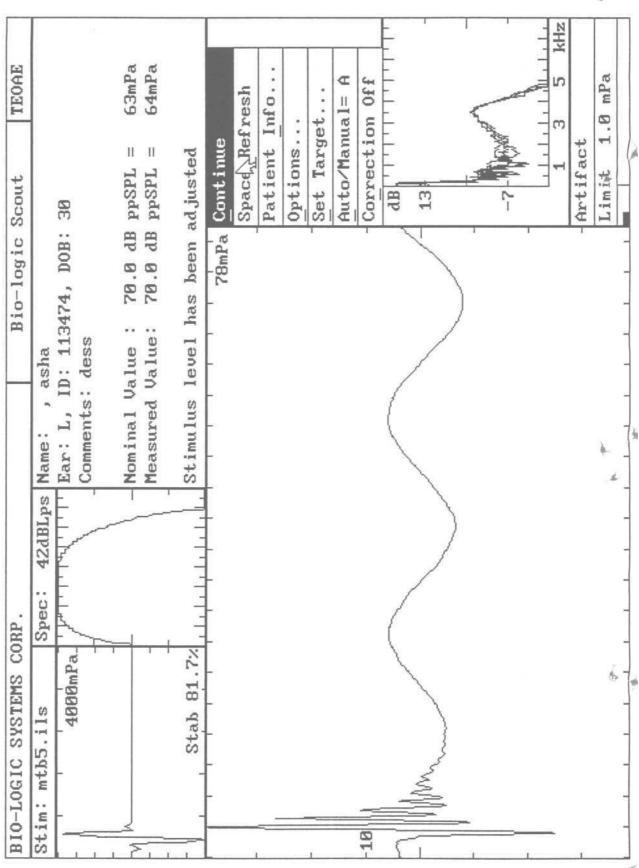
Ear: Left ID: 113474



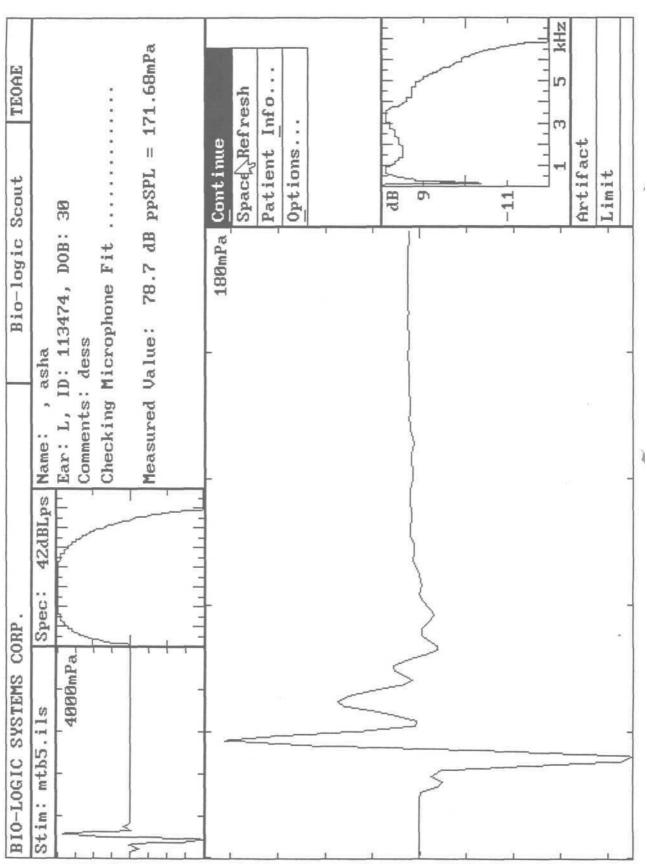
Testdate: 25-Sep-96 Filename: 96I25D01.DAT

L1(dB)	L2(dB)	Fl(Hz)	F2(Hz)	GM(Hz)	DP(dB)	NF(dB)	DP-NF(dB)
70.1	69.7	415	488	450	-8.3	-11.1	2.8
69.5	69.5	855	1025	936	-9.6	-8.6	-1.0
70.6	70.7	1660	2002	1823	-10.3	-17.4	7.1
69.4	70.0	3296	3955	3610	-21.3	-35.1	13.8
70.1	70.1	6665	8008	7306	-13.1	-22.7	9.6

Fig : 3.5



9. 2 515



+161: 2.4

|--|

8.2 · bit

(iii) Measurement : The stimulus was presented automatically. The stimulus spectrum, the response over time and the response spectrum were displayed on the screen during the test. The responses were stored after the test was completed. Fig: 3.8

STATISTICS

The Karl-Pearson's product moment method was used to calculate the correlation between different measured.

- 1) DPOAE vs TEOAE
- 2) DPOAE amp vs. TEOAE amplitude
- 3) DPOAE (Df-Nf) amp. vs specific frequencies thresholds.
- 4) TEOAE amp. vs. specific frequencies thresholds
- 5) DPOAE amp. vs. puretone threshold.

RESULTS AND DISCUSSION

Fourty-nine ears of the 38 subjects included in the study had pure tone average thresholds of 16 dB HL or above upto a maximum pure tone average of 90 dB HL. It was observed that as the hearing loss increased the amplitude of DPOAE and TEOAE decreased. The data was analyzed to test the null hypothesis.

Pearson's correlation coefficient of the DPOAE amplitude (0.41- 66.65 KHz) and the auditory thresholds measured at 0.5, 1, 2, 4, 8 KHz were computed. The results are shown in Table 4.1

PTT *		DP amplitude				
	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz	
500 Hz 1000 Hz 2000 Hz 4000 Hz 8000 Hz	-0.26 -0.39 -0.40 -0.42 -0.43	-0.55 -0.65 -0.57 -0.55 -0.58	-0.49 -0.69 -0.71 -0.66 -0.64	-0.60 -0.70 -0.67 -0.61 -0.56	-0.19 -0.34 -0.36 -0.40 -0.46	
PTA	-0.38	-0.60	-0.68	-0.70	-0.34	

Table 4.1: Pearson's correlation coefficient of DPOAE amplitude and auditory thresholds

PTT - Pure tone threshold PTA - Pure tone average theshold.

It may be noted that DP amplitudes and pure tone

thresholds show a good correlation at 1 KHz, 2 KHz, 4 KHz

with the highest correlation noted at one particular frequency. However, there was poor correlation between DP amplitude and pure tone threshold at 500 Hz and 8 KHz. Martin et al. (1990) have also reported poor correlation between DP amplitude and pure tone thresholds at 500 Hz. (1993) observed that DPOAE amplitude decrease Gorga, et al. as hearing loss increases in high frequencies (2K, 4K, 8 KHz). This trend was not clear at lower frequencies (500 Hz). Investigations by Suckfull et al. (1996) showed good correlation between DP amplitude and puretone thresholds at high frequencies. The difference for 8 KHz results obtained in the present study could be due to variations in the methodology.

The DP amplitude at each frequency showed strongest correlation with pure tone thresholds of one particular frequency. The correlation coefficient for the adjacent frequencies decreased gradually. DP amplitude at 1 KHz, 2 KHz and 8 KHz correlated best with the pure tone threshold of corresponding frequency i.e., at these three frequencies DPOAE gave frequency specific information. However DP amplitude at 4 KHz showed strongest correaltion with pure tone threshold at 1 KHz and DP amplitude at 500 Hz correlated best with pure tone threshold at 8 KHz. Suckfull et al.

(1996) have also reported that for all auditory threhsold frequency maximum correlation exist at one particular frequency. But they did not specify the frequency at which there was maximum correlation.

A majority of earlier investigators have reported that frequency specific information can be obtained using DPOAE at least at high frequencies (Harris, 1990; Gorga, et al. 1993)

In many of these stuidies DPOAE detection threshold was correlated with pure tone threshold (Lonsbury-Martin, 1990); Lichard, 1990). In the present study DPOAE amplitude for high level stimulation was studied. Avan (1993) reported that low level of primaries elicit a local response and they give frequency specific information. At higher level, the response is more complex and non-local. It is a well known fact that two distinct mechanism may be involved in the generation of DPOAE. One mechanism may generate DPOAE inrsponse to low level stimuli and primaries at nearby frequencies with similar latencies where as the other process may depend upon a short latency mechanisms, in the presence of the higher level stimuli and larger F2/F1 ratio (Kemp, 1986). Probably better results would have been observed if detection thresholds were studied in the present study.

Pearson's correlation coefficient were also computed for DPOAE/noise , the auditory threshold measured at specific frequencies and pure tone average thresholds as shown in Table 4.2

PTT *	DP/Noise				
	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz
500 Hz 1000 Hz 2000 Hz 4000 Hz 8000 Hz	-0.08 -0.22 -0.18 -0.20 -0.24	-0.45 -0.50 -0.45 -0.42 -0.45	-0.53 -0.65 -0.59 -0.54 -0.66	-0.52 -0.62 -0.62 -0.55 -0.48	-0.20 -0.25 -0.22 -0.27 -0.31
PTA	-0.12	-0.50	-0.65	-0.61	-0.36

Table 4.2: Pearson's correlation coefficient of DPOAE/noise and auditory thresholds.

PTT - Pure tone threshold

PTA - Pure tone average threshold

Moderate correlation was seen for DP/noise at 1 KHz, 2 KHz and4 KHz and pure tone threshold and low correlation was observed at 500 Hz, and 8 Khz. Overall correlation was poor for DP/noise than that observed for DP amplitude. However Gorga et al. (1993) reported that in their study DPOAE/noise provided better performance than DP amplitude, the significant difference of performance being very small. This contradictory result may be accounted by the difference in thestimulus level of the 2 studies. [L1=65 dB SPL; L2=50 dBSPL(Gorga et al 1993) Ll=L2=70dBSPL in the present study.]

PTT	TEOAE Amplitude (KHz)					
	Echo	E-N	Reproducibility			
500 Hz 1000 Hz 2000 Hz 4000 Hz 8000 Hz PTA	-0.42 -0.67 -0.71 -0.70 -0.68 -0.67	-0.44 -0.62 -0.67 -0.65 -0.64 -0.64	-0.44 -0.64 -0.70 -0.70 -0.69 -0.66n			

Table 4.3: Pearson's correlation coefficient of TEOAE and auditory thresholds.

PTT - Pure tone threshold PTA - Pure tone average threshold E-N - Echo - noise

Table 4.3 correlates TEOAE amplitude, TEOAE/noise and reproducibility with the auditory threshold. Higher correlation was observed between puretone and TEOAE at 2 KHz. In contrast to DPOAE, TEOAE did not show a sharp maximum for correlation with one particular frequency of the auditory threshold. This indicates that TEOAEs are not as frequency specific as DPOAEs. These results are also supported by Suckfull et al. (1996). TEOAE amplitude and TEOAE noise showed the strongest correlation at 2 KHz audiometric Reproducibility of threshold. TEOAE showed highest correlation with 2 frequencies i.e. 2 KHz and 4 KHz among TEOAE amplitude (Echo) TEOAE/noise and reproducibility pure tone average threshold best correlated with TEOAE amplitude. It may also be noted that the correlation was better with higher frequencies (1 Khz - 8KHz). This could be because click were used as stimuli. It has been established that with the clicks only the higher frequency range of the cochlea tends itself to effective assessment and probably lies in somewhere around 1500 Hz and above (Weber, 1976).

Identification of hearing loss using DPOAE and TEOAE

Several investigators have used different criterion values of DP/noise to differentiated between the normal hearing ears and those of hearing-impaired ears. Lonsbury-Martin (1990), Harris (1990); Smurzynski et al. (1990) employed a criteria of 3 dB for DP/noise Probst et al. (1993) employed 4 dB criteria and Gorga et al. (1993) employed 8-15 dB forDP/noise to differentiate between normal and impaired ears. Since majority of the studies used 3 dB criteria the results of the present study was analysed employing 3 dB criteria.

Frequency	F 0.011-	1 1711-	0 1/11-	4 1211-	01211-
Hearing loss	500Hz	1 KHz	2 KHz	4 KHz	8KHz
Minimal Mild Moderate Moderately- Severe Severe	100% 50% 57.2% 60% 61.5%	100% 75% 85.8% 50% 38%	100% 100% 100% 90% 61.5%	100% 41.6% 100% 70% 92.3%	100% 100% 100% 100% 92.3%

Table 4.4: Occurrence of DP/Noise with 3 dB criteria

From Table 4.4 it may be noted that as the degree of hearing loss increased the occurrence of DP decreased. DP was present in all the subjects with minimal sensorineural hearing loss which was true for all frequencies with less hearing loss. A superior occurrence of DPOAE was seen at 8 Khz followed by 2 KHz, 4 KHz,1 KHz and the lowest incidence was at 500 Hz. 100% incidence was maintained at higher frequencies (2 KHz, 4 KHz and 8 KHz) for moderate hearing loss patients. As the degree of hearing loss increased the incidence of DPOAE decreased. The presence of DPOAE was more at higher frequencies.

In severe hearing loss patients a high incidence of DP was observed at higher frequencies (4KHz and 8KHz) which was incidence at 500 Hz followed by the and 1 KHz. This contradicted the other preliminary studies reported in literature Harris (1990) opined absence of DPOAE inears with hearing loss greater than 10 dB HL. Suckfull et al. (1996) reported DPOAE in same ears witha hearing loss of upto 70 dB 3 dB criteria failed to differentiate HL. Employing a hearing impaired subjects from subjects with normal hearing. So a more stringent criteria of 8 dB and 12 dB were used.

Frequency	E 0 0 U z	1 KHz	0 1711-	4 KHz	8KHz
Hearing loss	500Hz	I KHZ	2 KHz		
Minimal Mild Moderate Moderately- Severe	66.6% 33.3% 37.5% 60%	100% 50% 50% 40%	100% 83.3% 87.5% 60%	100% 91.6% 100% 60%	78.3% 83.3% 87.5& 80%
Severe	53.8%	15.3%	15.3%	69.2%	61.5%

Table 4.5 : Occurrence of DP/Noise with 8 dB criteria.

Frequency	F 0.011-	1 1211-	0 1711-	4 1711-	01711-	
Hearing loss	500Hz	1 KHz	2 KHz	4 KHz	8KHz	
Minimal Mild Moderate Moderately- Severe Severe	66.6% 25% 25% 40% 23%	100% 50% 37.5% 30% 7.7%	100% 83.3% 87.5% 30% 7.7%	100%% 91.6% 100% 60% 46.1%	66.6% 66.6% 75% 60% 46.1%	

Table 4.6 : Occurrence of DP/Noise with 12 dB criteria.

It may be observed from Table 4.5 that the overall occurrence reduced with 8 dB criteria, however even with this criteria higher occurrence was noted for higher frequencies 2K, 4K, 8 KHz). Overall the maximum occurrence was at 4 Khz followedd by 8 Khz and 2 KHz. More than 50% of thesevere hearing loss subjects exhibited DP in all frequencies except 1 KHz and 2 KHz.

From Table 4.6 it was clear that at 4 KHz the occurence was highest for all the degrees of hearing loss. Even in cases with severe hearing loss 46.1% the subjects (6 out of 13 subjects) exhibited DP when 12 dB criteria was used. As discussed earlier Gorga et al. (1993) opined that DP/noise of 12 dB at 4 Khz correctly identified at least 90% of the hearing-impaired ears. Bonfils et al. (1994) found that DP incidence varied as a function of frequency and stimulation level of primaries. The mean DP incidence for subjects with a hearing loss below 30 dB HL varied between 82.3% and 77.7%. When the primary stimulation level was varied from 72 dB SPL to 62 dB SPL. The mean DP incidence for patients with a hearing threshold greater than or equal to 30 dB HL dropped from 47% to 67% when the stimulus level was varied from 72 to 62 dB SPL. The best results are obtained with primary stimulation level around 52dB SPL. As there is prevalence of DPOAEs in response to primary stimulation level below 50 dB HL in clinical practice it cannot be recommended Lisastover, et al. (1996) found DPOAE amplitude in response to moderate level of primaries (L1/L2 = 60/50) had greatest predictive value but they did not examine high level stimuli.

Gorga et al. (1993) employed the criterion values of 8 to 15 dB for DPOAE/noise to accurately distinguish normal hearing ears from those of hearing-impaired ears at the best performed frequencies (4 KHz and 8 Khz). It appeared that DP

and noise amplitude were comparable in the lower frequencies, regardless of the auditory thresholds. So it followed that DP/noise would increase and would show greater separation between normals and impaired ears as frequency increases similar to DP amplitude observations. They also proposed that DP/noise of 12 dB at 4KHz correctly identified at least 90% of the hearing-imparied ears.

Collecively, superior occurence was maintained at 4 Khz followed by 8KHz, 2 KHz 1 KHz and the lowest occurrence being observed at 500 Hz. These results agreed with the results of (1993). Bonfils al. (1994) Gorga et al. et that as frequency increases, performance also improves with the best performance being observed at 4 KHz and performance was only slightly poorer at 8 KHz. Thus in the present study DP was present in 3 subjects at 500 Hz, one subject at 1 KHz; one subject at 2 KHz and six subjects at 4 KHz and 8 KHz out of 13 severe hearing impaired patients even with a stricter criteria of 12 dB for DP/noise.

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Degree	3 dB	8 dB	12 dB
Minimal	100%	78.3%	66.6%
Mild	58.3%	41.6%	33%
Moderate	37.5%	25%	0%
Moderately-severe	10%	10%	0%
Severe	0%	0%	0%

Table 4.7: Occurrence of TEOAE/noise in % with different criteria

Table 4.7 shows the mean TEOAE noise occurrence employing the 3 different criterion. the Table it may From be noted that when 3 dB criteria was employed, only minimal degree of hearingloss secured 100% TEOE occurrence. TEOAE kept reducing as a function of degree of hearingloss, lowest being observed with severe hearing loss cases. None of the subject with severe hearing loss exhibited TEOAE. Only 1 hearing loss exhibited TEOAE. patient with moderately severe It has been repoted in literature that TEOAE was absent whenever theloss was greater than 25 dB HL (Probst, et al.1986), 35 dB HL (Bonfils et al. 1988) and45 dB HL (Collet 1984). Collet et al. (1989) reported that TEOAE was et al. absent when hearing loss exceeded 40 dB HL at 1 KHz. The occurrence of TEOAE may also depend upon the intensity level. It is also said that TEOAE gives better results than DPOAE at higher stimulus presentation level. Thus the results of the present study support the findings of the earlier studies.

Degree	Reproducibility			
Minimal	100%			
Mild	50%			
Moderate	37.5%			
Moderately-severe	10%			
Severe	0%			

Table 4.8: Percentage of subjects above 50% reproducibility

From the Table 4.8. it was observed that а reproducibility of 100% was maintained in minimal degree of hearing loss. But reproducibility decreased as a function of degree of hearing loss. Only 50% of the cases exhibited a reproducibility of 50% and above when hearing loss was mild. Only one subject with moderately severe hearing loss had reproducibility of 50%. Gorga et al. (1993) maintained that percent reproducibility resulted in the best performance among TEOAE echo, TEOAE/noise and reproducibility with the output amplitude at different frequencies. In the present study with TEOAE/noise producibility showed and similar performance in identifying subjects with hearing loss.

Isolated examples of emissions being measured in presence of profound sensorineural hearing loss also has been reported (Lutman, Mason, Shepard and Gibbin, 1990). Lawrence et al. (1996) observed TEOAE present in a child with bilateral profound loss. 3 This case as well as cases previously reported in patients with severe hearing loss is a rare but not an impossible event. Such a condition might be due to retrocochlear pathology, neural damage or inner hair cell damage. In the case reported by Lutman et al. (1989) Electrocochleography noted measurable compound potential documenting a good cochlear functions. In the case by Welzl-Muller et al. (1993) the successful stimulationo of the cochlear nerve ruled neural damage out and therefore suggested that the inner hair cell were damaged. Other reports of TEOAE present in subjects with severe hearing loss (Rossi, et al.1989), Tanaka (1987, 1988, 1989) are difficult to assess because artifacts can also be repeatable. kemp et al. (1986) illustrated, some hearing-impaired persons may have what appears to be low energy OAEs and found that cross correlation of repeated wave forms from hearing impaired patients were typically less than 50% suggesting no emission was actually present. Patuzzi (1993) opined that any processes associated with the inner hair cell, the primary afferent synapse and dendrites would not affect the cochlear mechanics directly and so wouldn't show up in OAE tests.

In the present study, TEOAE was not present when the hearing loss exceeded 60dB. But many isolated results were observed in the occurrence of DPOAE.

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In a correlation analysis, the Pearson's correlation coefficient of DPOAE amplitudes (0.41 - 66.65 KHz), DPOAE/noise and TEOAE amplitude, TEOAE/noise, reproducibility at different distortion product frequencies were computed.

	500 Hz	1 KHz	2 KHz	4 KHz	8 KHz	
TEOAE echo	0.55	0.69	0.85	0.60	0.60	DP amp
Reproducibility	0.58	0.71	0.85	0.70	0.63	DP amp
TEOAE/Noise	0.42	0.65	0.70	0.55	0.41	DP/Noise

Table 4.9: Pearson's correlation coefficient of DPOAE and TEOAE

From Table 4.9 it was observed that the strongest correlation between DP amplitude and TEOAE echo was noted at 2 KHz followed by 1 KHz and the lowest correlation was found at 500 Hz. Similar results were computed for reproducibility When the correlation analysis was and DP amplitude also. computed between DPOAE/noise and TEOAE/noise, again similar results were obtained with superior correlation at 2 KHz followed by 1 KHz and minimum correlation observed at low frequency and high frequency (500Hz and 8 KHz). So overall highest correlation was found between DPOAE at 2 KHz and all parameters of TEOAE. TEOAE reproducibility correlation among TEOAE echo, reproducibility, and TEOAE/noise with the output amplitude at different frequencies. The results of study by Gorga et al. (1994), Smurzyuski et al. 1992; Prieve et al.

(1993) comparing DPOAE and TEOAE are in concordance with that of the present study.

Gorga et al. (1993) compared TEOAE and DPOAE in his study and summarized the results as follows:

- (1) Neither TEOAEs and DPOAEs were able to distinguish between normal and hearing impaired subjects at 500Hz, although TEOAEs did perform better than DPOAEs.
- (2) TEOAEs more acuratey distinguished normal and impaired subjects at 1 KHz.
- (3) At 2 KHz TEOAEs and DPOAEs performed comparably.
- (4) DPOAEs were more successful at correctly identifying normal and imparied ears at 4 KHz.
- (5) Measures of OAE amplitude were slightly less accurate than measures that took into account the level of the background noise such as TEOAE/noise, DPOAE/noise and reproducibility.

The better TEOAE test performance at 1 KHz, and 2 KHz reflects the transfer characterstics of the middle ear. Larger amplitude response might be observed because the middle transmits energy both in forward and reverse direction more efficently to these frequencies.

SUMMARY AND CONCLUSION

One of the most excited advances in the understanding of hearing, during recent years has concerened the discovery of OAE. OAEs describe the response that the cochlea emits in the form of acostic energy. The recognition that the cochlea not only receives sounds, but also produces acoustic energy has been a major factor in modifying recent thinking concerning cochlear functions.

EOAE are virtually present in all subjects with normal hearing. It has been reported in literature that the amplitude of DPOAE and TEOAE reduces as the degree of hearing loss increases. These studies indicate that measurement of DPOAE and TEOAE provide valuable information in measurement of hearing.

The present study aimed at checking the efficiency of DPOAE and TEOAE in differentiating hearing impaired individuals from those with normal hearing. Different DP/Noise criteria were used for detection of presence of emission with comparison of DPOAE and TEOAE in some subjects was also undertaken.

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The study was a cross sectional study of 30 young adults (49 ears), in the age range of 18-35 years (mean age 29.6) with sensorineural hearing loss of different etiologies.

The pure tone average ranged from 16.6 to 83.3 dB HL. DP amplitude, DP/noise, TEOAE echo, TEOAE/noise, TEOAE reproducibility, pure tone average thresholds, pure tone thresholds at specific frequencies were the parameters considered for the study. Auditory threshold, tympanometry, TEOAE and DPOAE of these patients were measured. Karl Pearson's correlation was employed to find out the correlation was employed to find out the correlation between TEOAE and DPOAE with auditory thresholds.

The results of the study may be summarized as :

- DP amplitude and pure tone threshold showed a good correlation at 1 KHz, 2 KHz and 4 KHz with the highest correlation at one particular frequency (2 KHz).
- Similar results were obtained for DP/noise but the correlation was poorer than that observed for DP amplitude.

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- 3. Among 3 dB, 8 dB, 12 dB criteria of DPOAE a stringent criteria of 12 dB was found to be better in differentiating, normal hearing ears and hearing impaired ears. However DPOAE was present in 46.1% (6/13 subjects) of the cases with severe hearing loss even with 12 dB criteria.
- 4. TEOAE also showed good correlation with puretone threshold with maximum correlation at 2 KHz.
- 5. Among, TEOAE echo, TEOAE/noise and TEOAE reproducility, TEOAE reporoducibility showed a better correlation with auditory threshold.
- 6. There was no occurrence of TEOAE observed after a loss of 60 dB HL with 3 dB criteria and 40 dB HL with 12 dB criteria.
- There was a good correlation between DP amplitude and TEOAE echo and the maximum correlation was observed at 2 KHz.

To conclude, in the present study comparing DPOAE and TEOAE, TEOAE is found to be more efficient in differentiating normal hearing and sensorineural hearing-impaired ears.

REFERENCES

Antonelli, A., and Grandori, F. (1986). Long term stability, influence of the head position and modelling consideration for evoked otoacoustic emissions. Scandinavian Audiology, Suppl. 25, 97-108.

Avon, P., and Bonfils, P. (1993). Frequency specificity of human distortion product otoacoustic emissions. Audiology, 31(I), 12-26.

Bonfils, P., and Avon, P. (1992). DPOAEs - Values for clinical use. Archieves of Otolaryngology, Head and Neck Surgery, 118, 1069-1976.

Bonfils, P., Avon, P., Francois, N., Trontonn, J., and Nancy, P. (1992). Distortion product in neonates - normative data. Acta. Otolaryngolgol, 112, 739-744.

Bonfils, P., Avon, P., Londero, A., Trotoux, J., and Narcy, P. (1991). Objective low frequency audiometry by distortion product otoaoustic emissions. Archieves of Otolaryngology Head and Neck Surgery, 117(10), 1167-1171.

Bonfils, P., Avon, P., Martine, F., and Marie, P. (1980). Clinical significance of OAEs : A perspective. Ear and Hearing, 11, 155-158.

Bonfils, P., Bertrand, Y., and Uziel, A. (1988). Evoked OAEs : Normative data and presbyacusis. Audiology, 27, 21-35.

Bonfils, P., Dumont, A., Marie, P., Francois, M., Narcy, P. (1990). Evoked Otoacoustic emission in new born hearing screening. Laryngoscope, 100, 186-189.

Bonfils, P., Franmcois, M., Avon, P., Londero, A., Trotoux, J., and Narcy, P. (1992). Spontaneous and evoked otoacoustic emissions in pre-term neonates. Lryngoscope, 102 (2), 182-187.

Bonfils, P., and Uziel, A. (1989). Clinical interest of evoked otoacoustic emissions. Annals of Otology, Rhinology and Laryngology, 98, 326-31.

Bonfils, P., Uziel, A., Piron, and Pryot, P. (1988). Evoked OAEs from adults and infants. Clinical applications. Acta Otolaryngologica, 105, 445-449.

Bright, K.E. (1994). Handson helpful hints for OAE measurement. The Hearing Journal, 47(2), 72-75.

Brown, A.M., Sarah, L.S., and Paul, T.R. (1994). ADP from the ears of term infants and young adults using low stimulus levels. British Journal of Audiology, 28, 273-280.

Brown, A.M., and Gaskill, S.A. (1990). Measurement of acoustic distortion reveals underlying similarities between human and rodent mechanical responses. Journal of Acoustical Society of America, 88(2). 840-849.

Brown, A.M., Gaskill, S.A., Carlyon, R.P., Williams, S.M. (1993). Acoustic distortion as a measure of frequency selectivity. Journal of Acoustical Society of America, 93(6), 3291-3297.

Brown, A.M., and ramp, D. (1984). Supressibility of the 2 F_1-F_2 stimulated acoustic emission in debril and man. Hearing Research, 13, 29-37.

Brownell, W.E. (1990). OHC electromotility and OAEs. Ear and Hearing, 1(2), 82-93.

Came. M.A., Donoghue, G.M., Lutman, M.E. (1992). The feasibility of using OAEs to monitor cochlear function during acoustic neuroma surgery. Scandinavian Audiology, 21(3), 173-177.

Chang, K.W., Vohur, B.R., Norton, S.J., and Otoacoustic Lekas, M.D. (?) (1993). External and middle ear status related to evoked emissions in neonates. Archives of Otolaryngology and head and neck surgery, 119, 276-282.

Cherloff, N.E., Hecox, K.E., and Goldstein, R. (1992). Auditory distortion products measured with averaged auditory evoked potentials. Journal of Speech and Hearing Research, 35, 157-166.

Cianfrone, G., Mattia, M., Cervelliai, M., and Mussachio, A. (1993). Some effects of tonal fatiguing on SOAEs and DPOAEs. British Journal of Audiology, 27(2), 123-130.

Collet, L., Chanal, J.M., Hellal, H., Gartner, M., and Moryon, A. (1989). Validity of bone conduction stimulated ABR, MLR and OAEs. Scandinavian Audiology, 18(1), 43-47.

Collet, L., Gartner, M., Moutlien, A., Kaufmann, I., Disant, F., and Morgon, A. (1989). Evoked OAEs and sensorineural hearing loss. Archives of Otolaryngology and head and neck surgery, 115, 1060-1062.

College, L., Levy, V., Veuillet, E., Trury, E., and Morgon, A.M. (1993). Click evoked otoacoustic emissions and hearing threshold in sensorineural hearing loss. Ear and Hearing, 14, 141-143.

Collet, L., Moulin, A., Gartner, M., and Morgon, A. (1990). Age related change in evoked OAEs. Annals of Otology, Rhinology and Laryngology, 99 (12), 993-997.

Collet, L., Veuillet, E., Moulin, A., Morlet, T., Giraud, A.L., Micheyl, C, Chery-Gross, A. (1992). Contralateral auditory stimulation and otoacoustic emissions a review of basic data in humans. British Journal of Audiology, 298, 213--218.

Collet, L., Veuillet, E., Channal, J.H., and Morgon, A. (1991). Evoked OAEs. Correlates between spectrum analysis and audiogram. Audiology, 30(3), 164-173.

Davis, H. (1993). An active process in cochlear mechanics. Hearing Research, 9, 79-90.

David, K., Brown. (1996). Comparison of DPOAE and ABR travelling wave delay measurements suggests frequency-specific synapse maturation. Annals of Otology, Rhinology, and Laryngology, 17(5), 395-401.

Devries, S.M., and Recker, T.N. (1992). Otoacoustic emissions : Overview of measurement methodologies. Seminars in Hearing, 13(1), 15-21.

Dolhen, P., Hennaux, C, Chantry, P., and Hennebert, D. (1991). The occurrence of evoked OAEs in a normal adult population and neonates. Scandinavian Audiology, 20(3), 203-205.

Elberling, C, Parbo, J., Johnsen, M.J., and Bgi, P. (1985). Evoked otoacoustic emissions, Clinical applications. Acta Otoiaryngologica, 421, 77-85.

Engdahl, B. (1996). TEOAE : Helpful tool in the detection of pseudohypocusis. Scandinavian Audiology, 25(3), 173-178.

Engdahl, B., Arnesan, A.R., Mair, I.W.S. (1994). Reproducibility and short-term variability of transient evoked otoacoustic emissions. Scandinavian Audiology, 23(2), 99-104.

Engdahl, B., Arnesan, A.R., Mair, I.W.S. (1994). Otoacoustic emissions in the first year of life. Scandinavian Audiology, 23 (3), 195-201.

Fabiani, M. (1993). Evoked otoacoustic emissions in the study of adult sensorineural hearing loss. British Journal of Audiology, 27(2), 131-139.

Fortum, H., Farnsworth, A., Davis, A. (1993). The feasibility of evoked OAEs as an in-patient hearing check after meningitis. British Journal of Audiology, 27(4), 227-233.

Furst, M., and Lapid, M.A. (1988). A cochlear model for acoustic emissions. Journal of Acoustical Society of America, 84, 222-229.

Frust, M., Rabinowitz, W.M. and Zwek, P.M. (1988). Ear canal acoustic distortion $2F_1-F_2$ from human ears relation to other emission and perceived combination tones. Journal of Acoustical Society of America, 84(1), 215-221.

Gaskill, S.A., and Brown, A.M. (1990). The behaviour of acoustic distortion product and its relation to auditory sensitivity. Journal of Acoustical Society of America, 88(2), 821-839.

Gebian, G.L., and Ankim, D.O. (1982). Cochlear microphonic evidence for mechanical propagation of distortion products (F_2-F_1) ($2F_1-F_2$). Hearing Research, 6(1), 35-50.

Gobsch, H., Kevanishvilli, Z,, Gamgebeli, Z., Gvelesian, T. (1992). Behavior of delayed evoked OAEs under forward masking paradigm. Scandinavian Audiology, 21(3), 143-149.

Gorga, M.P., Neeley, S.T., Bongman, B.M., Beanchainek, L., Kauinski, J.R., Peters, J., Schulte, L., Jestrad, W. (1993). A comparison of TE and DPOAE in a normal hearing and a hearing-impaired subject. Journal of Acoustical society of America, 94(5), 2639-2648.

Grandori, F. (1985). Non-linear phenomena in click and toneburst evoked otoacoustic emissions from human ears. Audiology, 24, 71-80. Guelke, R.W., and Bum, A.E. (1985). A mechanism for stimulated otoacoustic emissions in the cochlea. Hearing Research, 19(3), 185-191.

Hansen, R., and Probst, R. (1991). Influence of systematic primary tone level variation (L_2-L_{21}) on the acoustic distortion product emission $2F_1-F_2$ in normal human ear. Journal of Acoustical Society of America, 89(1), 282-286.

Harris, F.P. (1990). Distortion product OAE in humans with high frequency sensorineural hearing loss. Journal of Speech and Hearing Research, 33(3), 594-600.

Harris, F.P., and Probst, R. (1991). Reporting click-evoked and distortion product otoacoustic emission results with respect to the pure tone audiogram. Ear and Hearing, 12(6), 399-405.

Johnsen, N.J., and Elberling, C. (1982). Evoked otoacoustic emissions from the human ear. Equipment and response parameters. Scandinavian Audiology, 11, 3-12.

Johnsen, M.J., Bagi, P., and Elberling, C. (1983). Evoked OAEs from the human ear. III findings in neonates. Scandinavian Audiology, 12, 17-24.

Johnsen, N.J., Bagi, P., Parbo, J., and Elberlin ,C. (1988). Evoked OAEs from the human ear. IV. Final results on 100 neonates Scandinavian Audiology, 17, 27-34.

Johnsen, N.J., Parbo, J., and Elberling, C. (1989). Evoked otoacoustic emission from the human ear. V. Developmental changes. Scandinavian Audiology, 18(1), 59-62.

Johnsen, N.J., Parbo, J., and Elberling, C. (1993). Evoked acoustic emissions from the human ear. VI. Findings in cochlear hearing impairment. Scandinavian Audiology, 22(2), 87-96.

Johnsen, N.J., and Elberling, C. (1982). Evoked OAEs from the human ear and influence of posture. Scandinavian Audiology, 11(1), 69-77.

Kemp, D.T. (1978). Stimulated acoustic emissions from within the human auditory system. Journal of Acoustical Society of America, 65, 1386-1391.

Kemp, D.T. (1986). Otoacoustic emissions, travelling waves and cochlear mechanisms. Hearing Research, 22, 95-194.

Kemp, D.T. (1990). Otoaoustic emissions - basic facts and applications. Audiology in Practise, 7(3).

Kemp, D.T., and Chum, R. (1980). Properties of the generator of stimulated acoustic emissions. Hearing Research, 2, 213-232.

Kemp, D.T., Ryan, S., Bray, P. (1990). A guide to the effective use of otoacoustic emissions. Ear and Hearing, 11 (2),93-105.

Kemp, D.T., and Ryan, S. (1993). The use of transient evoked otoacoustic emissions in neonatal hearing screening programs. Seminars in Hearing, 14(1), 15-23.

Kim, D.O. (1980). Cochlear mechanics : Implications of electrophysiological and acoustical observations. Hearing Research, 2, 297-317.

Kim, D.O. (1996). DPOAE test of sensorineural hearing loss : Performance regarding sensitivity, specificity and receiver operating characteristics. Acta Otolaryngologica, 116 (1), 3-11.

Lafreniere, C.C., Jung, M.D., Smurzynski, J., Leonard, G., Kim, D.O. and Sasek, J. (1991). Distortion product and click-evoked OAEs in healthy new borns. Archieves of Otolaryngology and Head and Neck Surgery, 117, 1382-1389.

Laurence (1996). Bilateral evoked otoacoustic emission in a child with bilateral profound hearing loss. Annals of Otology, Rhinology, and Laryngology, 105(4), 286-88.

Lexior, M., and Puel, J. (1987). Development of $2F_1-F_2$ otoacoustic in rat. Hering Research, 29, 265-271.

Lonsbury-Martin, B.L., (1993). Evidence for the influence of aging on DPOAEs in human. Journal of Acoustical Society of America, 89(4), 1749-1759.

Lonsbury-Martin, B.L., Harris, F.O., Hawkins, M.D., Stagner, B.B., and Margin, G.K. (1990). Distortion product emissions in humans : I Basic properties in normally hearing subjects. Annals of Otology, Rhinology and Laryngology, 147, 4-14.

Lonsbury-Martin, B.L., Harris, F.O., Hawkins, M.D., Stagner, B.B., and Margin, G.K. (1990). Distortion product emissions in humans : II Relation to acoustic immittance, stimulus frequency and SOE in normal hearing subjects. Annals of Otology, Rhinology and Laryngology, 147, 15-24.

Lonsbury-Martin, B.L., and Martin, G.K. (1991). Clinical applications of OAE. Journal of Speech and Hearing Research, 34(5), 964-981.

Lind, O., and Randa, J.-(1989). Evoked acoustic emissions in high frequency versus low or medium frequency hearing loss Scandinavian Audiology, 18(1), 21-27.

Lutman, M.E. (1990). Evoked otoacoustic emissions in adults : Implications for screening. Audiology in Practise, 6(3), 6-8.

Lutman, M.E. (1993). Reliable identification of click-evoked OAEs using signal processing techniques. British Journal of Audiology, 27, 103-108.

Lutman, M.E., Mayson, S.N., Sheppard, S., Gibbin, K.P. (1991). Differential diagnostic potential of OAEs : A case study. Audiology, 28, 204-210.

Lutman, M.E., Sheppard, S. (1990). Quality estimation of click evoked otoacoustic emissions. Scandinavian Audiology, 19(1), 3-9.

Martin, G.K., Ohlms, L.A., Franklin, D.J., Harris, F.P., and Lonsbury-Martin, B.L. (1990). Distortion product OAE in Humans III : Influence of SN hearing loss. Annals of Otology, Rhinology and Laryngology, 147-30-42.

Martin, G.K., Probst, R., and Lonsbury-Martin, B.L. (1990). OAEs in Human ears - Normative findings. Ear and Hearing, 11, 106-120.

Meredith, R., Stephens, D., Hongan, S., Cartlidge, P.H.T., Drayton, M. (1994). Screening for hearing loss in an at-risk neonatal population using evoked otoacoustic emissions. Scandinavian Audiology, 23(3), 187-194.

Meric, C, and Collectic, L. (1993). Comparative influence of repeated measurements and of attention on electro otoacoustic emissions. Acta Otolaryngologica, 130(4), 471-477.

Micheyl, C., Collect, L. (1994). Interrelations between psychoacoustical tuning curves and spontaneous and evoked otoacoustic emission. Scandinavian Audiology, 23 (3), 171-178.

Moore, B.L.J. (1982). An introduction to the Psychology of Hearing (Edn.2). London, Academic Press, Chapter 8, Section V, Cochlear Echos, 260-261.

Morgan, D.E., and Canalis, R.F. (1991). Auditory screening in infants. Otolaryngological Clinics of North America, Clinical Audiology, 2, 277-284.

Moulin, A., Collet, L., Veuillet, E., Morgon, A.M. (1993). Inter-relations between transiently evoked otoacoustic emissions, spontaneous otoacoustic emissions and acoustic distortion products in normally hearing subjects. Hearing Research, 65, 216-233.

Naeve, S.L., Margolis, R.H., Levine, S.C., and Fournier, E.M. (1992). Effect of ear canal air pressure on evoked otoacoustic emissions. Journal of Acoustical Society of America, 91, 2091-2095.

Nelson, L.M., and Popelka, R.G. (1993). Clinical significance of probe tone frequency ratio on DPOAE. Scandinavian Audiology, 22(3), 159-164.

Ning-Tine, Schmiedt, R.A. (1995). Fine structure of the 2Fi- F_2 acoustic differential product changes with the primary level. Journal of Acoustical Society of America, 94(5), 2659-2667.

Norton, S.J. (1993). Application of transient evoked otoacoustic emissions to peadiatric populations. Ear and Hearing, 14, 64-73.

Norton, S.J. (1992). Cochlear function and otoacoustic emissions. Seminars in Hearing, 13(1), 1-14.

Norton, S.J., and Neely, S.T. (1987). Tone burst-evoked otoacoustic emissions from normal hearing subjects. Journal of Acoustical Society of America, 81, 1860-1872.

Norton, S.J., and Stover, L.J. (1994). Otoacoustic emissions in Katz, J. Ed. Handbook of clinical Audiology. 4th Edition. William and Wilkins, Baltimore, U.S.A. Norton, S.J., Widen, J.E. (1990). Evoked otoacoustic emissions in normal-hearing infants and children. Emerging Data and issues. Ear and Hearing, 11, 121-127.

Osterhammel, A.O., Nelson, L.H., and Rasmussen, A.N. (1993). DPOAE - The influence of middle ear transmission. Scandinavian Audiology, 22(2), 111-116.

Patak, C.B. (1991). Review of literature of otoacoustic emission. An unpublished Independent Project submitted to University of Mysore.

Patuzzi, R. (1993). OAEs and the categorization of cochlear and retrocochlear lesion. British Journal of audiology, 27(2), 91-97.

Pray, P.J., and Kemp, D.T. (1987). An advanced cochlear echo suitable for infant screening. British Journal of Audiology, 210, 191-204.

Prieve, B.A. (1992). Otoacoustic emissions in infants and children : Basic characteristics and clinical applications. Seminars in Hearing. (Journal ?) 13 (1) Pg. ?

Probst, R., Coats, A.C., Martin, G.K., and Lonsbury-Martin, B.L. (1986). Spontaneous, Click and Tone-burst evoked otoacoustic emissions from normal ears. Hearing Research, 21(3), 261-277.

Probst, R., and Harris, P. (1993). Transiently evoked and distortion product OAEs, comparison of results from normally hearing and hearing-impaired human ears. Archives of Otolaryngoloy and Head and Neck Surgery, 119 (8), 858-860.

Probst, R., Harris, F.P., and Hauser, R. (1993). Clinical monitoring using OAEs. British Journal of Audiology, 27 (2), 85-91.

Probst, R., Harris, F.P., and Housen, R. (1993). Clinical monitoring using OAE. British Journal of Audiology, 27(2), 85-90.

Probst, R., Lonsbury-Martin, B.L., Martin, G.K. (1991). A review of otoacoustic emissions. Journal of Acoustical Society of America, 89 (5), 2027-2067.

Rahul (1996). A comparison of DPOAE in children and adults. An unpublished Independent Project submitted to University of Mysore. Rasmussen, A.N. (1996), The influence of SOAE on the amplitude of transient evoked emissions, Scandinavian Audiology, 25(3), 187-192.

Rasmussen, A.R., Popelka, G.R., Osterhammel, A.P., and Nelson, L.H. (1993). Clinical significance of relative probe tone levels on DPOAE. Scandinavian Audiology, 22(4), 222-234.

Reshef, Haran, I., Attias, J., and Furst M.(1993). Characteristics of click-evoked OAEs in ears with normal hearing and noise-induced hearing loss. British Journal of Audiolcgy, 27(6). 387-397.

Robinette, M.A. (1992). Clinical observation with transient evoked otoacoustic emissions with adults. Seminars in Hearing. 13(1)

Robinett, M.A., and Fraser, G. (1991). Evoked otoacoustic emissions in a patient with a profound sensori-neural hearing loss. Archives of Otolaryngology and Head and Neck Surgery. Pg ?)

Roede, J., Harris, F.P., Probst, R. (1992). Repeatability of DPOAE in normally hearing humans. Audiology, 32 (5), 273-281.

Ruggero, M.A., Rich, N.C., and Fryman (1993). Spontaneous and impulsively evoked OAEs: Indicators of cochlear pathology. Hearing Research, 10, 283-300.

Rutten, W.L.C. (1980). Evoked acoustic emissions from within normal and abnormal human ears : comparison with audiometric and electrocochlographic findings. Hearing Research, 2, 263-271.

Ryan, S., Kemp, D.T., and Hinchcliffe, R. (1991). The influence of contralateral acoustic stimulation on click evoked OAEs in humans. British Journal of Audiology, 25 (6), 391-399.

Suckfull, M. (1996). Evaluation of TEOAE and DPOAE measurements for the assessment of auditory thresholds in sensorineural hearing loss. Acta Otolaryngologica, 116(4), 528-33.

Thornton, A.R.D. (1993). Click evoked otoacoustic emissions :New techniques and applications. British Journal of Audiology, 27, 109-5 (?)

Thornton, A.R.D., and Slaven, A. (1993). The effect of stimulus rate on the contralateral inhibition of evoked otoacoustic emissions. Journal of Acoustical Society of America, 34, 132-136.

Truy, E., Vevillet, E., Collet, L., and Morgon, A. (1993). Characteristics of transient OAEs in patients with sudden idiopathic hearing loss. British Journal of Audiology, 27(6), 379-387

Van dijk, P., and Wit, H.P. (1987). The occurrence of clickevoked otoacoustic emissions (Kemp echoes) in normal hearing ears. Scandinavian Audiology, 16, 62-64.

Wada, H., Ohyama, K., Kobayashi, O., Navhria, S., Kioke (1992). Relationship between evoked otoacoustic emissions and middle ear dynamic characteristics. Audiology, 32 (5), 282-292.

Wilson, J.P. (1980 a). Evidence for a cochlear origin for acoustic re-emissions, threshold fine-structure and tonal tinnitus. Hearing Research, 2, 233-252.

Wit, H.P., and Ritsma, R.J. (1979). Stimulated acoustic emissions from the human ear. Journal of Acoustical Society of America, 66, 911-961.

Wit, H.P., and Ritsma, R.J. (1980). Evoked acoustical responses from the human ear : Some experimental results. Hearing Research, 2, 253-261.

Wit, H.P., Langevoort, J.C., and Ritsman, R.J. (1981). Frequency spectra of cochlear acoustic emissions (Kemp echoes). Journal of Acoustical Society of America, 70, 437-445.

Yasmeen, F. (1997). Age related changes in TEOAEs and relation of amplitude to ear canal volume. An unpublished Independent Project submitted to University of Mysore.

APPENDIX I

Standards for Calibration of Puretone Audiometer

The following standards were used for calibration of the audiometer.

Air conduction (Earphones) - ANSI S3-6-1989.

Bone-conduction (BC vibrator) - ANSI S3-26-1981.

The procedure used was as prescribed by the instruction manual of the audiometer, using a Sound Level Audiometer with octave Filter Set, 1 inch condenser Microphone, Artificial Ear (for headphone calibration) and Artificial mastoid (for bone conduction vibrator calibration).

APPENDIX II

Standards for Calibration of Immittance Audiometer

The immittance audiometer used for the study was calibrated using the following standards.

ANSI S3-7 1973 ANSI S3-39 1987 ANSI S3-6 1969 IEC 645 1979 IEC 126 1973