DPOAE in Recruiting and Non-Recruiting Ears : An Aid to Differential Diagnosis of Sensory and Neural Loss

Register NO.M9904

This Independent Project submitted as part fulfilment for the First Year M.Sc, (Speech and Hearing), submitted to the University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MYSORE 570006

MAY 2000

To

Baba, Dadi,
Nani
&
Bade Tauji

CERTIFICATE

This is to certify that this Independent Project entitled : *DPOAE in Recruiting **and** Non-Recruiting Ears : An Aid to Differential Diagnosis of Sensory and Neural Loss* is the bonafide work in part fulfilment for the degree of Master of Science (Speech and Hearing) of the student with Register No.M9904.

Mysore
May, 2000

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CERTIFICATE

This is to certify that this Independent Project entitled : ***DPOAE in Recruiting and Non-Recruiting Ears : An Aid to Differential Diagnosis of Sensory and Neural Loss*** **has been** prepared under my supervision and guidance.

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May:2000

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DECLARATION

This Independent Project entitled: ***DPOAE in Recruiting and Non-Recruiting Ears : An Aid to Differential Diagnosis of Sensory and Neural*** *Loss* is the result of my own study under the guidance of Mr.Animesh Barman, Lecturer in Audiology, Deparmtent 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, 2000

Reg. No M9904

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I can fly higher than Eagle.
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INTRODUCTION

Hearing is one of the most important sensory functions of the body. It is one of the window through which we communicate with the environment, the interaction through which one moves from the level of existence to higher living. Thus, it is quite important that this sensory function be preserved with care. But, like any other biological functions it too is prone to damage.

For years together, the inner ear had been assigned a rather passive role as a hearing receptor. However, with discovery of otoacoustic emissions, the cochlea is considered now to be a highly sophisticated organ with bi-directional transduction properties.

Otoacoustic emission, first hinted at by Gold (1948), are known to be microvibrations of the outer hair cells in the cochlea which propagates towards the foot plate of the stapes and is transmitted to the external auditory meatus by the ossicles where in it may be picked up by a high sensitivity microphone (Kemp, 1978; Kemp et al. 1986). Otoacoustic emissions (OAEs) may be broadly classified into two types (Norton and Stover, 1994).

(i) Spontaneous otoacoustic emissions (SOAEs) which are low level, tonal signals measured in the external ear canal in the absence of any known stimulus.

(ii) Evoked otoacoustic emission (EOAEs) are those signals which are produced by acoustic stimulation of the cochlea. They are of three types:

(a) Transient evoked OAEs

These responses are commonly elicited by the use of brief acoustic stimuli

(b) **Distortion product OAEs**

These are the acoustic form of the difference tones that are produced by the cochlea during simultaneous stimulation with two continuous puretones of Fl and F2 with F2 greater than Fl.

(c) **Stimulus frequency OAEs**

These are the responses of cochlea to a continuous sweep frequency puretone.

The force responsible for OAE generation is the electro-motility of the outer hair cells. This outer hair cell electromotility is what sets the outer hair cells in to oscillations at audible frequencies. These oscillations are magnified by the middle ear system and transmitted into the air as sound. So, by sealing a receiver microphone probe in to the ear canal, sounds made by the cochlea can be recorded (Robinette, et al. 1997).

Among all the various types of OAEs measurements, DPOAEs, and TEOAEs have come up as the most promising and popular tools.

Probst et al. (1993) compared results of TEOAE and DPOAEs in normal hearing and hearing impaired population. They found a high correspondence between the two and both are largely derived from similar mechanisms. DPOAEs are found to be present more often than TEOAEs when hearing loss across the frequency is greater than 30 dB HL suggesting that TEOAEs are more preferable for screening purpose and DPOAEs for monitoring cochlear changes clinically.

It is important to note that OAE generation is preneural and independent of both afferent and efferent ennervation (Norton, 1992), i.e. if a lesion is central to outer hair cells (OHCs), OAEs could be present with behavioral and neural responses depressed. Based on this Robinette (1999) stated that EOAEs can be used in the differential diagnosis of cochlear vs. retrocochlear hearing disorders.

Patuzzi (1993) put forward a categorization of cochlear and retrocochlear lesions based on otoacoustic emissions as

(i) Motor loss (Associated with dysfunction of OHC and vibration of the organ of corti)

(ii) Sensory loss (associated with the dysfunction of IHC and the primary afferent neurons)

(iii) Mixed loss (when dysfunction of both motor and sensory component is present).

Loudness recruitment phenomenon is considered as a hallmark of cochlear pathology. Subjective or objective loudness recruitment

measure is a most easily accessible and commonly used measure to differentially diagnose retrocochlear pathology from cochlear pathology.

Theories initially put forward to explain the phenomenon of recruitment, were based on the assumption that nerve fibers were responsible for this phenomenon (Evans, 1976; Kiang, et al. 1970).

Lurie (1940) explained loudness recruitment in terms of the differential functions of the inner hair cells and outer hair cells. Simmon and Dixon (1966) explained recruitment based on two operational mechanism (i) place principle (ii) summmation principle. The place principle explained the role of OHCs and the summation principle explained the role of nerve fibers in the phenomenon of recruitment. Tonndorf (1981) proposed a "center clipping" theory of loudness recruitment.

More recent studies (evoked potential) suggested the central auditory pathway may also be involved in loudness recruitment (Salvi, et al. 1991).

However, there is no clear cut view about the physiological basis of recruitment

Auditory brainstem response (ABR) is an objective method which has already established itself as a proficient diagnostic tool amongst auditory assessment procedure. Thus the question which remains is, how lucrative are the investigations of DPOAEs and its clinical

application in terms of differential diagnosis of recruiting vs. non-recruiting ear. This may be answered by the several advantages of DPOAEoverABR.

(i) ABR is not specific to cochlear physiology whereas DPOAEs give information solely of the sensory elements of the cochlea.

(ii) ABR does not tap OHC physiology in detail which is one of the major advantage of OAE.

(iii) DPOAEs can also detect noise exposure through reduced emission amplitude with frequency specificity which is not possible for ABR (Samurzynski et al. 1990; Kemp et al. 1986).

(iv) Compared to ABR, DPOAE testing procedure takes lesser time.

(v) Preparing the patient for ABR testing takes a long time, whereas it is minimal in DPOAE testing.

(vi) Lastly, wave interpretation is highly subjective with respect to the tester with a high value of intra-subject variability, whereas DPOAE interpretation is much less subjective with in individual ears over time and across testers (Rhode, et al. 1992).

Hence this study was taken up to probe -

(a) If there is significant difference in DPOAE between recruiting ears and non-recruiting ears.

(b) To explain possible site of recruitment based on DPOAE findings.

(c) To see whether DPOAE can be used to differentially diagnose retrocochlear pathology from cochlear pathology.

REVIEW OF LITERATURE

Loudness recruitment which is a common clinical symptom of cochlear hearing loss, refers to the abnormally rapid growth of loudness with increasing stimulus levels (Hallpike and Hood, 1959).

The phenomenon of recruitment was first reported by Edmund Prince Fowler in 1928, who defined it as "loudness recruitment is the greater change (or the difference) in the increment of loudness, in relation to stimulus increase observed in ears with a partial neural loss of hearing as compared with normal ears or ears with only impedance lesions".

There are various hypothesis on the exact mechanism involved in the phenomenon of recruitment. Theories put forward initially were based on the assumption that nerve fibers were responsible for the phenomenon of recruitment.

Steven (1936) proposed that presumably the coding of loudness involves some integration of neural activity across the population of nerve fibers. Since loudness recruitment is a symptom of cochlear hearing loss, these might be some change in the way individual nerve or the population of nerve fibers respond to changes in intensity.

Salvi et al. (1983) proposed a potential model for recruitment based on a proportionality between the intensity, the discharge rate of auditory nerve fibers, and the perception of loudness. The mechanism for explaining loudness recruitment involves a steepening of the slope of the function which relates the neural discharge rate to intensity, i.e. in

pathological ear, a small change in stimulus intensity produces on abnormally large increment in the neural firing rate which in turn is "coded" as a large change in loudness.

Another explanation of loudness recruitment was suggested by Kiang et al. (1970) and Evans (1976). It is based on the rate at which new units are activated in the auditory nerve depending upon the shapes of the neural tuning curves. A tone of low intensity will activate only a few units with characteristic frequencies (CFs) near the frequency of the stimulus. As stimulus intensity increases more units are activated, particularly those with CFs above the stimulus frequency, i.e. a high CF unit can be activated by frequencies located in the tail of the tuning curve, but only at high intensities. Thus, the difference in threshold between frequencies in the tip and tail of the tuning curve influences the rate at which new units are activated with increasing intensity.

The other group of thoughts ascribed the phenomenon of recruitment to the hair cells.

Lurie (1940) explained loudness recruitment in terms of the differential functions of the inner hair cells (IHCs) and the outer hair cells (OHCs) of the organ of corti. He believed that the OHCs responded to the sounds of low intensity only and that the IHCs responded to higher intensity sounds. If the more sensitive OHCs are damaged/defective, then the puretone threshold would be raised to the extent consistent with the OHCs damage. With increase in intensity, the IHCs are excited, resulting in a loudness sensation which eventually equals that in impaired

ears. Absence of recruitment would result from diffuse damage affecting both IHCs and OHCs whereas decruitment would result from damage primarily to IHCs.

According to Simmons and Dixon (1966) two operational mechanisms exists for explaining recruitment. These mechanisms are:

(i) Place principle

(ii) Summation principle.

(i) Place principle

This principle is explained on the basis of hair cell phenomenon. According to this principle, nerve fibers excited by OHCs require a less intense stimulus than do the fibers excited by IHCs (Harris, 1953). When the more sensitive OHCs (or related structures) are damaged, auditory threshold is elevated. When the intensity of a sound is increased and excites undamaged IHCs, the resulting loudness sensation eventually equals the undamaged ear.

(ii) Summation principle.

Loudness perception depends upon the total number of nerve fibers excited (Harris, 1953). More intense sounds excite a larger area of the cochlea and ultimately more nerve fibers. An important feature of this code is its distribution. Within the cochlea: as intensity increases, most of the additional energy is distributed toward the basal end; low frequencies spread further man high frequencies.

Tonndorf (1981) proposed a "center-clipping" theory of loudness recruitment. According to this theory, the cilia of the hair cells in the organ of corti lose their stiffness because of cochlear dysfunction, thereby becoming decoupled from the tectorial membrane. The decoupling occurs as the cilia pass through their centre points as they move from side to side during the shearing action. At the moment of decoupling, there is an amplitude loss in the response waveform. This amplitude reduction has a fixed magnitude. As the intensity increases, the proportion of amplitude loss with respect to the total amplitude becomes smaller and smaller and eventually disappears. Tonndorf attributed this for the phenomenon of recruitment.

Brownell (1990) described the action of the OHCs as amplifiers for the IHCs. The OHCs do not merely respond passively to the sound stimuli; they amplify it. i.e. they inject additional energy into the system. He pointed out that the OHCs provide a large amount of amplification for weak signals and only a small amount of amplification for intense signals i.e. they are level dependent amplifiers.

Berlin et al. (1996) said that OHCs are embedded in the tectorial membrane and the IHCs are merely or just touching the tectorial membrane. All the signals going to the brain come through the IHCs. He believes that the motion of the OHCs modulates the gap between the tectorial membrane and stereocilia of the IHCs. He said because the fluid flow resistance varies as the third power of the gap spacing, only a small motion would produce required 40 dB change in the sensitivity. If the OHCs are damaged or missing orparalyzed, it will take something

like 50 dB SPL (40 dB HL) to cause the IHCs to fire and 40 dB bearing loss will be seen. In case of loud sounds, when large motion is available, the IHCs will fire whether the OHCs move or not i.e. at high intensity signals, the IHCs should fire normally. This manifests as recruitment.

More recent studies (evoked potential) suggest that central auditory pathway may also be involved in loudness recruitment One mechanism that could potentially lead to enhanced evoked response amplitudes in the central auditory system is an alteration of the balance of excitation and inhibition in the central auditory neurons. Many units in the cochlear neucleus and inferior colliculi have single tone inhibitory side bands located above and below the excitatory response area of the tuning curve; auditory nerve fibers do not have such inhibitory response areas. Sound that activate the inhibitory side bands of central nerves system (CNS) neuron may limit the maximum discharge rates of that neuron at high intensity (Salvi, et al. 1991).

Until recently, when bone conduction was found to be reduced, a case would be classified only as sensorineural or was more commonly called as nerve deafness. With the development of improved tests based on clearer understanding of auditory pathology, it is now possible in some cases to determine whether the damage is primarily in sensory or in the neural mechanism. The designation "sensory" and "neural" are becoming more meaningful as the knowledge of earpamology improves.

Gorga et al. (1993a) studied distortion product responses from normal hearing and hearing-impaired. The results of this study indicated that under clinical conditions DPOAE measurements can distinguish

normals from hearing-impaired subjects for higher frequencies once the loss exceeds 20 dB HL (Gorga et al. 1993b; Ricci et al. 1996; Suckfull et al. 1996).

Kim et al. (1996) compared DP-amplitude in sensorineural hearing loss vs. normal. They concluded that the conditions of DPOAE test were strongly dependent upon frequency, not only regarding the test performance but also on optimum DPOAE amplitude used for differentiating hearing-impaired from normals (Ohmls et al. 1990).

Kimberly and Nelson (1989) correlated DPOAE emission with auditory threshold in normal as well as in sensorineural hearing loss cases. The results suggested that distortion product emission measurement can predict frequency specific auditory thresholds. Similar results were reported by Lonsbury-Martin and Martin (1990), Harris (1990), Samurzynski et al. (1990), Harris and Probst (1991) and Avan and Bonfils (1993).

Nelson and Kimberley (1992) studied DPOAE input-output function at frequency regions between 707 Hz and 5050 Hz. Seven distinctly different shapes/patterns of DP emission growth curves were observed. Low level irregular shaped segments were more frequent in normal hearing ears, suggestive of normal low level active non-linearities from the OHC subsystem. High level, steeply sloped were frequent in hearing-impaired ears, suggestive of residual non-linearities from the cochlear partition without functional OHC. DP threshold were able to predict auditory sensitivity with some precision.

Probst and Harris (1993), Kimberly et al. (1994); Stover et al. (1996) attempted optimizing the clinical utility of distortion product otoacoustic emission measurement They examined the effect of primary stimulus level on the ability of DP emission measurement in both normal and sensorineural hearing loss. The results confirmed that high level stimulation might under predict hearing loss. The moderate level stimuli LI = 60 dB SPL or L2=50 dB SPL were recommended to be optimal in sensitivity for detecting hearing loss.

A study specifically on ototoxic hearing loss was done by Machekan and Dellg (1997) who compared distortion product emission generation between receiving frequent gentamycin therapy and control subjects. The resulting input/output function showed that though 4 out of 15 patients showed normal *(Jess* than 10 dB HL) a significantly elevated stimuli level was required to generate their DP emission at 4 kHz. This indicated the sensitivity of DPOAE over puretone audiometry as a clinical tool in predicting the earliest form of cochlear damage.

Theoretically sensorineural hearing loss can be divided into 3 groups.

- Cochlear lesion
- Retrocochlear lesion without cochlear dysfunction
- Retrocochlear lesion with minor cochlear element.

But clinically it is difficult to localize such a lesion at the hair cell level because there have been no clinical tools with which to identify

the exact site (Park and Lee, 1998). Bright et al. (1995) concluded from his study that OAEs can help to determine the site of lesion and distinguish between subcategories of cochlear pathology.

Recruitment is the outstanding feature of sensory rather than neural hearing. However, there may be some degree recruitment in neural hearing loss particularly if the loss originally started as sensory and progressed to involve the nerve endings (Sataloff and Sataloff, 1996).

Researcher's viewed the OHCs role in sound transduction as one compressing the large dynamic range of input levels for the imitated response range of the IHCs. Based on this understanding, one could argue that OHC dysfunction and altered. DPOAE measures would correlate with recruitment rather than puretone thresholds (Allen, 1995).

According to the literature, OAEs can sometimes be produced in patients with profound Sensorineural hearing loss (SNHL) Lutman et al. (1989) reported a patient with a profound SNHL with a presence of click evoked OAES. Prieve et al. (1991) and Katona et al. (1993) reported similar cases. Monroe et al. (1996) identified an 11 year old girl with profound hearing loss with a presence of TEOAEs and DPOAEs, who was diagnosed juvenile pilocystic astrocytoma of the pons. Konradsson (1996) reported 4 children with severe to profound SNHL who showed clear bilateral TEOAEs which could indicate the neural type of hearing loss.

Park and Lee (1998) studied the potential of DPOAE in differential diagnosis of hearing loss. 232 ears of severe to profound

SNHL were measured out of which in 16 ears (8 patients) normally recordable DPOAEs were found. The results were confirmed through retest after intervals; positive responses of TEOAEs were additionally tested. The concluded that the nerve deafness and hair cell deafness may be partially distinguishable based on DPOAEs.

Robinette (1999) stressed the role of clinical measurement of evoked otoacoustic emissions in the differential diagnosis of cochlear versus retrocochlear hearing disorders. He illustrated examples of vita nerve tumors, ideopamic sudden hearing loss, sudden hearing loss related to multiple sclerosis, a child with profound hearing loss and an adult under consideration for a cochlear implant. In toto he reported, EOAE helped to confirm the hearing loss due to cochlear origin of one patient and the retrocochlear origin of the other fourpatients. Presence of EOAEs in a patient going for a cochlear implant indicated that the lesion was above the level of cochlea. In such case surgery not only would have destroyed a satisfactorily functioning of cochlea but the cochlear implant would have been doomed to failure in view of the apparently marked retrocochlear injury.

Lonsbury-Martin et al. (1990) stated that normal DPOAE functioning in the presence of significant hearing loss, indicates a locus of damage central to the region of OHCs. DPOAE has the ability to facilitate in the distinction between the sensory and neural component of a cochlear based disorders.

Patuzzi (1993) put forward a categorization of cochlear and retrocochlear lesions based on otoacoustic emission. He referred the

process associated with OHC and vibration of the organ of corti at motor process and those associated with IHC and the primary afferent neurons as sensory processes. Lesions within the cochlea could be categorized as either motor, sensory or mixed if they contained components of each Patuzzi stated that OAE test would only be sensitive to the motor component of any lesion.

Kaga et al. (1996) reported two patients who showed absence of ABR but broad compound action potentials on electrocochleograms and almost normal OAEs together with absence of caloric response and preservation of per rotatory nystagmus for both ears. The auditory examination disclosed mild threshold elevation in puretone audiometry and markedly poor scores in speech audiometry and good scores in auditory comprehension test. They were diagnosed as having auditory nerve disease of unknown cause.

Auditory neuropathy is a hearing disorder characterised by severely abnormal ABR waveform, beginning at wave one, suggesting that the hearing disorder arises from a cochlear or VIIIth nerve pathology rather than from some more central lesion (Starr, et al. 1996).

Harris (1990) stated that in some cases of auditory neuropathy, though ABR thresholds are markedly poor, puretone thresholds are comparitively better may be only mild to moderate SN HL. This indicates that in these cases, if there is a cochlear damage it is not extensive enough to prevent some relatively low threshold cochlear afferent activity across arrange of frequency locations. On the other hand to reduce the number

of synchronized neurons that contribute to the ABR, the deterioration has to be quite significant These conditions could arise from scattered IHC loss. Given the findings of relatively normal OAEs, the OHCs are minimally involved.

According to Berlin (1999) auditory neuropathy is operationally defined when one sees normal otoacoustic emissions (OAEs) with absent electrocochleograms and/or absent ABR. The pathophysiology varies from patient to patient, sometimes encompassing systemic peripheral neuropathies, other times with symptoms suggesting a lack of inner hair cell or primary neuron function.

The distinction between IHC damage and neural damage appears to be important because rehabilitation methods such as hearing aids and cochlear implantation should be selected.

On the basis of all the studies reported above, it can be inferred that

(i) distortion product emissions are sensitive in distinguishing normals from the sensorineural hearing impaired, and

(ii) within sensorineural hearing-impaired, they are sensitive to distinguish sensory hearing loss from neural hearing loss.

None of the study, we came across, studied the effect of recruitment on the OAEs. So present study was carried out to investigate if DPOAEs could shed some light on the much discussed phenomenon of recruitment. DPOAE was selected as a measure for the study because it can be administered in cases with greater degree of hearing loss.

METHODOLOGY

This study was taken up with an aim of comparing DPOAEs in recruiting and non-recruiting ears, so as to explain the possible origin of recruitment and if it can be used as an aim to differentialy diagnose RCP from CP.

The methodology used was as follows :

I **Subjects**

They were basically divided into two groups

(a)Coatrol group - 30 ear with hearing within normal limits, age ranged from 18 to 25 years.

(b)Experimental group - 18 recruiting ears and 18 non-recruiting ears with sensorineural hearing loss of varying degree (mild, moderate, or moderately severe). Age range was 15 years - 50 years.

Subject Selection Criteria:

(a) Control Group

Ail the subjects had puretone hearing thresholds in the frequency range 250 Hz to 8000 Hz, less than 15 dB HL. This was ascertained using a calibrated two channel audiometer (GSI-61).

These subjects also had normal middle ear function. This was ascertained by using immittance audiometer. They had "A-type" tympanograms with reflexes at normal level.

None of the volunteers reported to have any otological symptoms (hearing loss, tinnitus, giddiness, etc), or otological or history of exposure to noise, ototoxicity etc.

(b) Experimental Group

All the individuals selected for the study had sensorineural hearing loss with normal middle ear function. This was ascertained by using an audiometer and immittance meter.

They were categorized into two groups depending on presence or absence of recruitment.

Criteria:

Individuals were classified as having recruitment or not based on one of the two conditions given below:

Ears were decided to have recruitment if -

(i) Metz recruitment test is positive i.e. the difference between the puretone thresholds and acoustic reflex thresholds is less than 65 dB and/or.

(ii) **The** dynamic range for speech is reduced i.e. the arithmetic difference between the Speech recognition threshold (SRT) and the Uncomfortable level (UCL) is less than 100 dB.

- (Martin, 1991).

Non-recruiting ears

- Those ears which did not meet the above criteria were considered as non-recruiting ears.

A group of subjects were found to have DP thresholds below or near the behavioural thresholds where ABR could not be recorded at 90 dB nHL. They were categorical as a separate group having auditory neuropathy. Some of the ears with auditory neuropathy did exhibit recruitment and other did not.

II INSTRUMENTATION

The following equipments were used,

(a) **Puretone audiometer**

A **two** channel clinical diagnostic audiometer (GSI-61) was used to assess the behavioural thresholds of all the subjects. The audiometer was calibrated prior to the study as per the recommendations of the manufacturer.

(b) Immittance audiometer

An immittance audiometer (GSI-33) was used to assess the middle ear function of the subjects. The audiometer was calibrated as per recommendations of the manufacturer.

(c) Otoacoustic emission analyzer

Madsen celesta 503 cochlear emission analyzer was used to obtain DP emission. It is a computer based OAE measuring system. The system allows for the user specifications to be used in testing for a number of parameters. With reference to the study, following parameters, were set.

(i) Display type

The display type controls the pattern of measurement. Since DP threshold was to be established display was set to input/output function curve of DPOAE. This setting plots growth of distortion product responses at a single frequency for different input levels for two primary tones.

(ii) Frequencies (fl and f2)

Testing was carried out at 4 sets of frequencies from 500 Hz to 4000 Hz. The primaries.fo and 2fl-f2 are as follows :

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

(iii) DP frequency

It refers to the frequency of emission. It was set to 2fl-f2, because the intermodulation distortion product at 2fl-f2 is most prominent (Kemp. 1979).

(iv) f2-fl ratio

The ratio of stimulus frequencies (primaries) at which the distortion product occurs has been determined by several previous studies. The maximum distortion is produced at f2/f 1 ratio of about 1.22 (Harris et al. 1989). So this ratio was taken up in the study. Also celesta 503 has a default ratio of 1.22.

(v) **Intensity level (LI** and **L2)**

This refers to the intensities of the stimulus frequencies. It has been clearly established that very high levels of stimulus gives rise to a "non-local response" i.e. distortion product does not correspond to a specific area on the basilar membrane (Avans and Bonfils, 1993) and also saturation occurs when higher level of intensity is used (Humes, 1983; Weber and Mellert, 1975).

Keeping these observations in mind 70 dB SPL was considered as the starting level.

There are controversies regarding the intensity level of the two primaries which gives maximum DPOAE, while some studies show that a difference in LI and L2 elicits greater DPOAE amplitude (Gaskill, 1990). Other studies either question those results entirely (Rasmussen, 1993) or suggest that the differences in intensity of primaries has different roles at different frequencies (Hanser, 1991). Thus for this study the intensities of both the primaries in each test condition were kept equals i.e. L1-L2.

(vi) S/N ratio:

It is one of the criteria for determining when to stopaveraging. The signal to noise ratio (i.e. the ratio of DPOAE level to the noise level) of +3 dB was taken as criteria to consider the presence or absence of DP-emission.

(vii) Accepted Sweeps

The instrument plotted the average DP emissions level and noise floor after the completion of 260 sweeps at a particular intensity. If the instrument was able to detect the emission before 260 sweeps it stopped averaging and gave the measurements.

III TEST ENVIRONMENT

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

The test room was comfortable enough in terms of temperature and lighting. The subjects were provided with a comfortable chair to sit on during the test. Since this is an objective test, the subjects were not required to perform any task.

IV TEST PROCEDURES

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

a) Puretone audiometry

Thresholds were obtained at octave frequencies from 500 Hz to 4000 Hz for both air conduction and bone conduction using modified Hughson-Westlake procedure in a sound treated room to reach a proper diagnosis.

(b) Speech audiometry

Speech recognition threshold: The lowest hearing level at which the subject correctly recognized the speech stimuli 50% of the

time was considered as his SRT. Bisyllabic words were used for the same (Rajashekhar, 1976).

Uncomfortable level : That sound pressure level at which speech becomes uncomfortably loud was considered as the subjects uncomfortable level (UCL). Stimulus used was speech (continuous discourse).

(c) Immittance testing

Tympanometry and acoustic reflexometry was done to assess middle ear conditions in normals and individuals with sensorineural hearing loss and helped to categorize the ear as recruiting or nonrecruiting.

(d) Distortion product otoacoustic emission measurement

Was carried out on both the control and experimental group in a sound treated room where background noise levels were kept minimum.

(i) **Preparation of the subject**

A suitable probe tip was fitted on to the probe and inserted into the ear canal of the test ear. Subjects were instructed to sit back and relax and reduce his body movement as much possible.

(ii) Probe fit

This is a procedure to check adequate fitting of the probe into the ear canal. This was carried out, automatically by the instrument. A

transient stimulus was presented to the ear and the measured response was displayed as a spectrum and a waveform- A correct probe fit would give a waveform as in fig. I (b). Fig.l(a) shows the probe fitting screen. If such waveform was not obtained probe was refitted with a different ear tip till proper waveform is obtained.

Procedures involved in emission measurement were as follows:

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

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

- The instrument plotted the input/output function curve with each set of primary frequencies fl and f2.

- If the S/N ratio falls below + 3 dB SPL for two consecutive intensity levels then the testing was terminated at that frequency.

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

- Distortion product emission were obtained both in normaland in ears with sensorineural hearing loss using above mentioned procedure.

DPOAE response obtained from an individual are shown in Fig.2 and 3. Fig.2 Depicts the amplitude spectrum graph and Fig.3 the input-output graph.

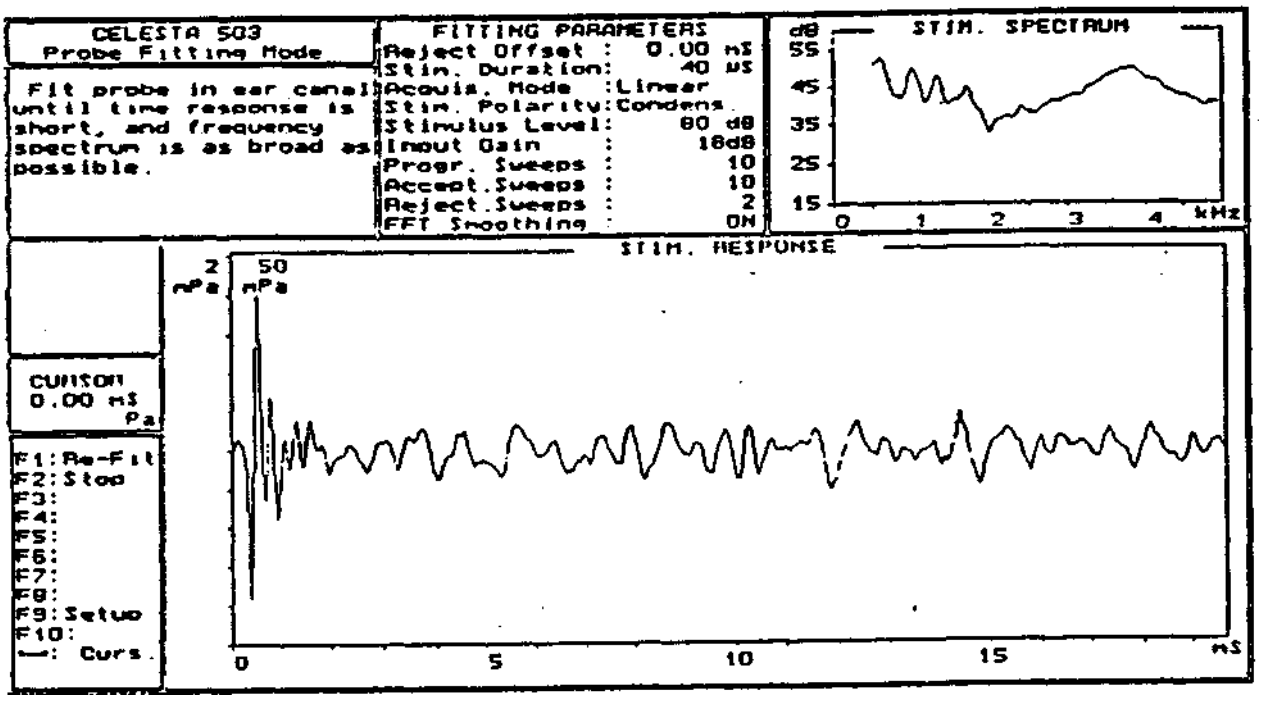

Fig.1(a). Probe Fitting Screen.

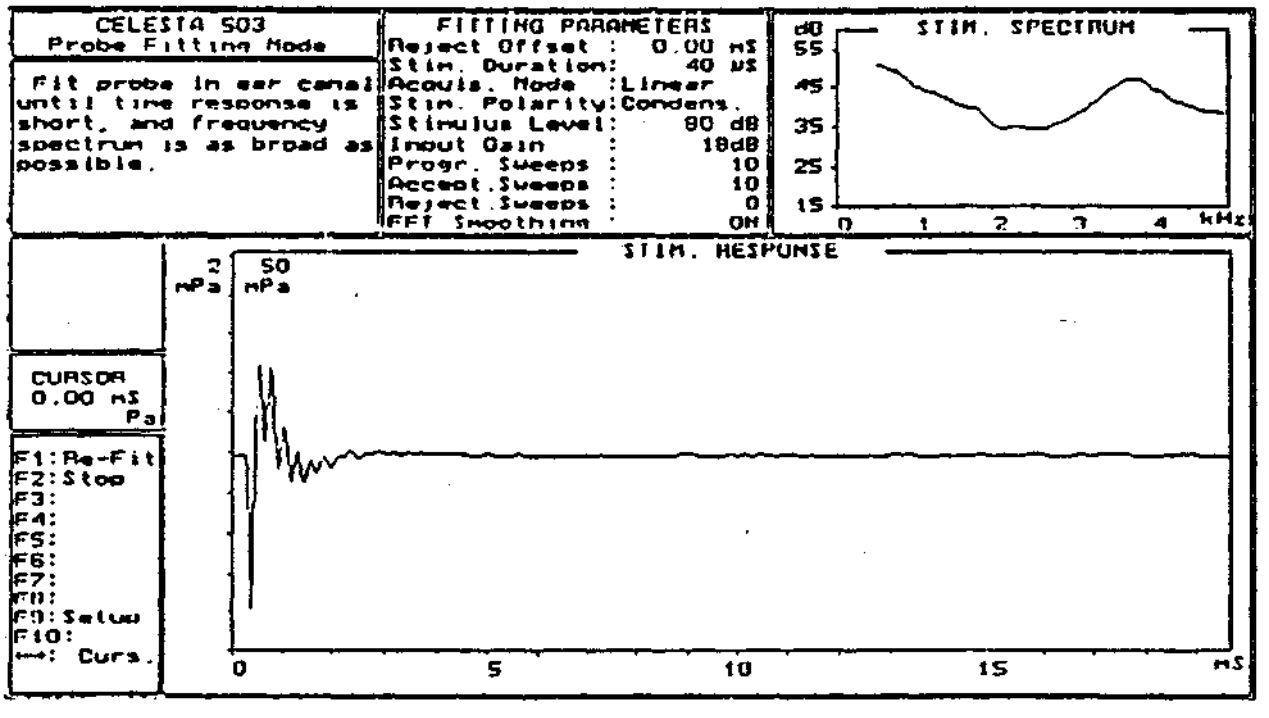

Fig 1(b). Good Probe Fit

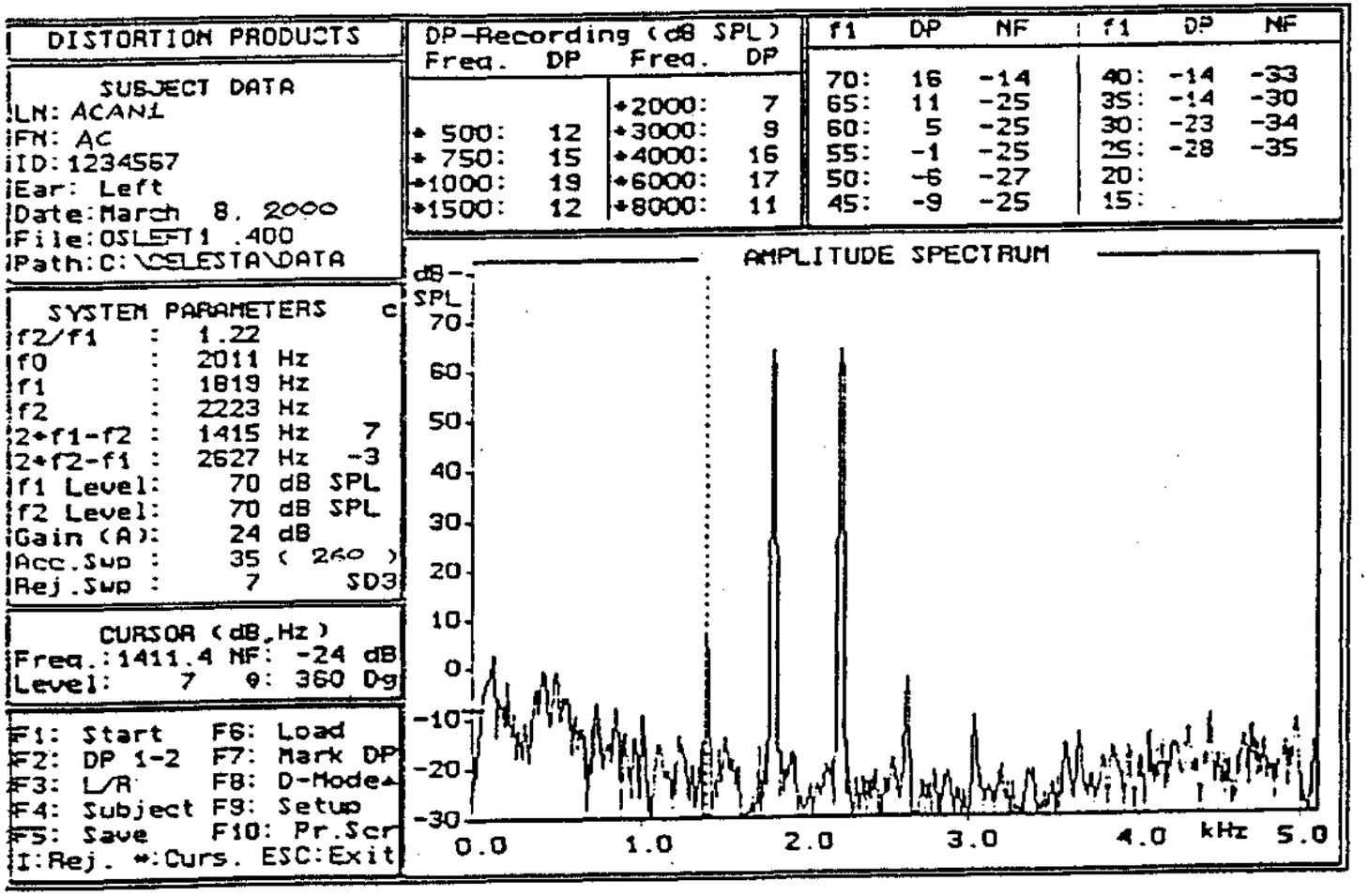

Fig. 2.. Amplitude Spectrum with Cursor.

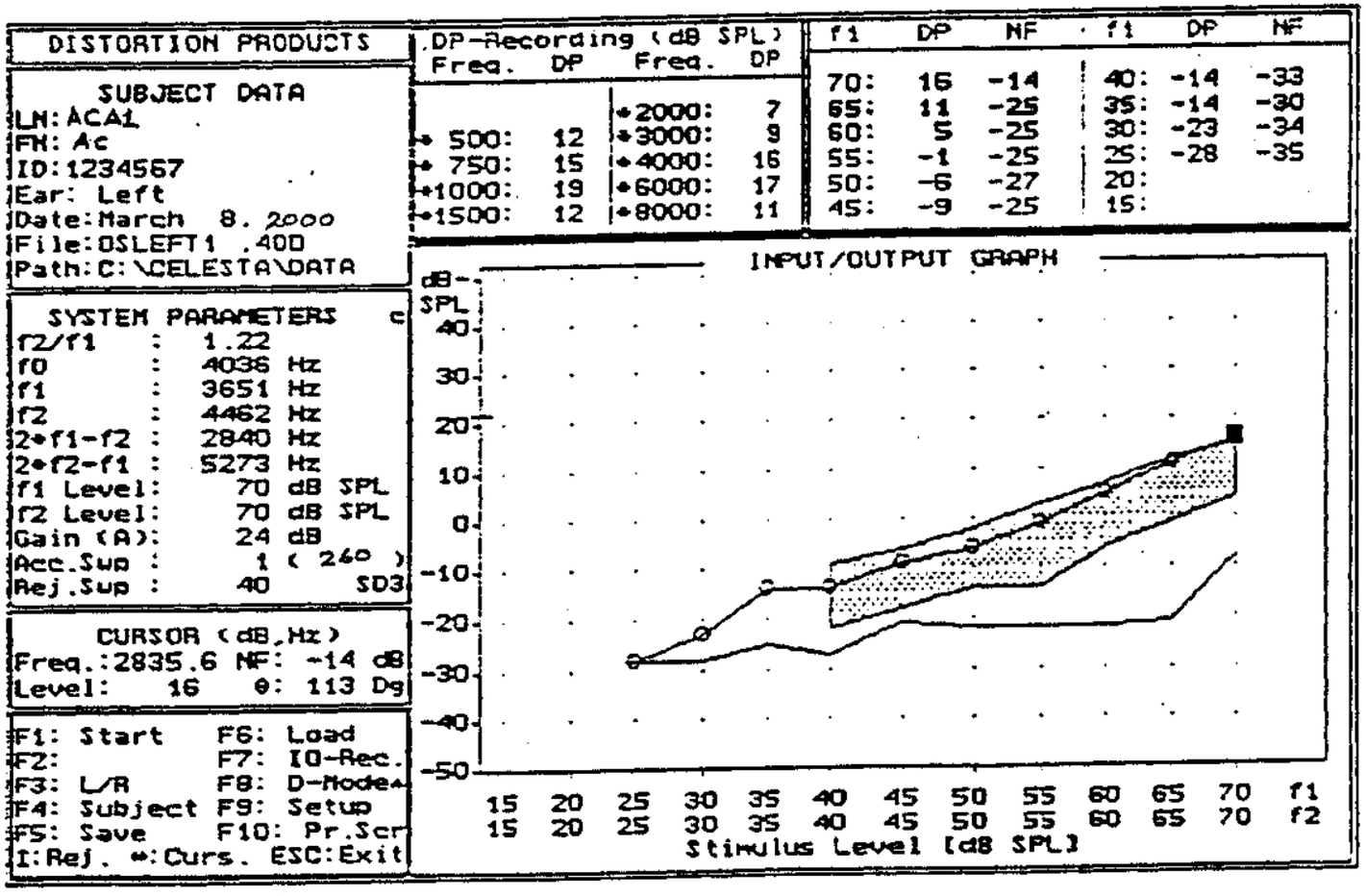

Fig. 3. *Example of an Input / Output Measurement.*

During the course of study some of the cases selected (6) were found to have DP thresholds near or well below their behavioural thresholds. Auditory brainstem evoked responses(ABR) were obtained for these cases in order to

* confirm their behavioural thresholds.
* to rule out functional hearing loss.

Based on the results of puretone audiometry, speech audiometry, immittance, ABR and OAEs, these cases were later diagnosed as having auditory neuropathy.

Instrumentation used for ABR was as follows:

- A computer based system was used.
- Electrode placement

The electrode placement was as follows:

Position	Function	Connection to electrode box
Forehead (F2)	Non-inverting	Fz
Mastoid region of the test ear.	Inverting	A1
Mastoid region of the non-test ear.	Common	A

Click stimulus at 90 dB nHL was used with the repetition rate of 11.1/sec, as to get better synchrony of auditory nerve.

Analysis

Analysis of the data of the clinical group was carried out in two different ways:

Method-1: DPOAE threshold was considered at 70 dB SPL in the instances where DPOAE response was not obtained at maximum stimulus level and then statistical analysis was done.

Method-2: The instances where DPOAE response was absent at maximum stimulus level (70 dB SPL) were excluded for statistical analysis.

This was done to avoid errors due to over estimation or under estimation of the data.

In both the methods the 't* test (unpaired) was used to compare the mean of the difference of DP threshold and behavioural threshold also called as OAE-audiometric threshold gap (O-T gap) (Kemp 1997) in normal vs. each pathological group i.e. recruiting SN hearing loss, non-recruiting SN hearing loss and auditory neuropathy group. Significant difference of the means among the pathological groups was also calculated.

When significant difference of the mean was calculated for auditory neuropathy group with recruiting SN hearing loss group and non-recruiting SN hearing loss, the auditory neuropathy cases who

showed either recruitment or no recruitment based on subjective or objective method mentioned above were excluded from the other two groups.

In order to find the relation between the DP threshold and behavioural thresholds in each group, the Karl Pearson's product moment correlation coefficient was worked out for each group, i.e., recruiting SN hearing loss group, non-recruiting SN hearing loss and auditory neuropathy group.

RESULTS

This study was taken up with an aim to investigate, how successful DPOAEs are in distinguishing recruiting ears from non-recruiting ears.

The values obtained from the input-output function of normals, and different sensorineural hearing loss groups were analysed using various statistical procedures.

***OAE-Audiometric Threshold Gap (O-T Gap)**

The O-T gap was calculated for each of the groups by deducting DP-threshold from the behavioural thresholds for all the octave frequencies - 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

When O-T gap was compared within the different groups, it was noticed that, in comparison to normals, sensorineural hearing loss group had smaller O-T gap. Within SN hearing loss group, auditory neuropathy group was found to have smallest O-T gap. This could be justified saying that, in auditory neuropathy cases, DP threshold was found to be near or below the audiometric threshold.

Mean values of the O-T gap of the normal and SN hearing loss groups were tested for the significant difference at 0.01 level and 0.05 level with the help of 't'-test (unpaired).

The results are presented in the following tables :

	Frequency / Measure	500 Hz	1kHz	2kHz	4kHz
Normals	Mean	34.63	26.16	34.00	30.80
	SD	8.65	8.37	10.03	11.96
Non-recruiting SNHL	Mean Ml	26.76	20.00	25.29	23.52
	Mean M2	10.62	14.64	21.5	19.60
	SD Ml	14.75	13.69	13.04	12.71
	SD M2	23.82	14.46	23.43	13.45
l-values	Ml	2.591*	1.686	2.38*	1.93
	M2	2.82*	2.21*	1.84*	2.50*

* P>0.05; ** P>0.01

Table-1: Mean and standard deviation in normals and non-recruiting ears along with t-values (Ml - Method; M2 - Method 2).

In both, method I and Method 2, mean O-T gap of nonrecruiting SN hearing loss group was reduced when compared to O-T gap of the control group.

Significant difference for the above two groups was found between the means of the O-T gap at 0.05 level in both the methods except at 1 kHz in method 1.

Frequency Measure			500 Hz	1kHz	2kHz	4kHz
Normals	Mean		34.8	26.16	34.00	30.83
	SD		8.6	8.37	10.03	11.96
Recruiting SNHL	Mean	Ml	19.44	11.11	8.88	8.05
		M2	19.09	10.35	7.91	7.27
	SD	Ml	14.13	11.57	12.89	13.14
		M2	15.13	11.67	12.87	14.72
t-values		Ml	4.17**	4.81**	7.07**	5.80**
		M2	3.25**	4.58**	6.29**	4.76**

* P>0.05; ** P>0.01

Table-2: Mean and standard deviation of normal and recruiting ears along with t-values (Ml -Method 1; M2-Method 2).

From the table *2,* it can be inferred that O-T gap in recruiting ears is reduced when compared to normal ears.

Difference of the mean of the O-T gap of the two groups is highly significant at 0.01 level. Both the methods 1 and 2 show similar results.

Frequency Measure			500 Hz	1kHz	2 kHz	4kHz
Normals	Mean		34.83	26.16	34.00	30.83
	SD		8.65	8.37	10.03	11.96
Auditory neuropathy	Mean	Ml	7.30	4.23	6.92	13.84
		M2	0.0	4.20	6.92	3.0
	SD	Ml	16.40	16.43	15.75	27.47
		M2	24.82	16.43	15.75	23.47
t-values		Ml	5.71**	5.02**	5.44**	5.26**
		M2	4.16**	4.56**	5.71**	3.05**

* P>0.05; ** P>0.01

Table-3: Mean and standard deviation of normal and auditory neuropathy cases along with t-values (Mi-Method 1; M2-Method 2)

Even in table *3,* it can be seen that the means of O-T gap of the two groups, in both the methods are highly significant.

Frequency Measure			500 Hz	1kHz	2 kHz	4 kHz
Non-recruiting SNHL	Mean	Ml	26.76	20.00	25.29	23.52
		M2	10.65	14.60	21.53	19.61
	SD	Ml	14.75	13.69	13.04	12.71
		M2	23.82	19.46	23.48	13.45
Recruiting SNHL	Mean	Ml	19.44	11.11	8.88	8.05
		M2	19.09	1035	7.91	7.27
	SD	Ml	14.13	11.57	12.89	13.14
		M2	15.13	11.67	12.37	14.72
t-values		Ml	1.07	2.06*	3.73**	3.44**
		M2	-0.88	0.70	1.81	2.12*

* P>0.05; ** P>0.01

Table-4: Mean and standard deviation of non-recruiting and recruiting ears along with t-values (Mi-Method 1;M2-Method 2)

When mean of the O-T gap of recruiting and non-recruiting SN hearing loss group was compared for significant difference. Method 1 showed significant difference at 1 kHz at 0.05 level and at 0.01 level in 2 kHz and 4 kHz. No significant difference was found at 500 Hz. Recruiting ear showed less mean O-T gap than non-recruiting group. But method 2 did not show any significant difference between the two groups. This discrepancy could be due to the reduction in sample size.

Frequency Measure			500 Hz	1kHz	2kHz	4kHz
Non-recruiting SNHL	Mean	Ml	26.76	20.00	25.29	23.52
		M2	17.50	1833	34.28	21.00
	SD	Ml	14.75	13.69	13.04	12.71
		M2	8.66	16.39	11.33	11.25
Auditory neuropathy	Mean	Ml	7.30	4.23	6.92	13.34
		M2	0.00	4.23	6.92	3.00
	SD	Ml	16.40	16.43	15.75	27.47
		M2	24.62	16.49	15.75	23.47
t-values		Ml	3.79**	2.76**	3.65**	0.70
		M2	1.88	1.98	447**	2.18*

* P>0.05; ** P>0.01

Table-5: Mean and standard deviation of non-recruiting and auditory neuropathy ears along with t-values (Ml-Method 1; M2-Method 2)

Analysis by both the method indicate that the O-T gap mean of auditory neuropathy group is smaller than that of NR group at all the tested frequencies.

Method 1 indicates a high significant difference between the means of O-T gap of the two groups at all the frequencies except at 4 kHz at 0.01 level and whereas analysis in method 2 indicates that only at high frequencies, 1 kHz and 4 kHz there is a significant difference with 0.01 level for 2 kHz and at 0.05 level for 4 kHz.

Frequency Measure			500 Hz	1kHz	2 kHz	4 kHz
Recruiting SNHL	Mean	Ml	19.44	11.11	8.33	8.05
		M2	17.35	3.33	7.143	4.16
	SD	Ml	14.13	11.57	12.39	13.14
		M2	17.90	12.24	14.39	11.58
Auditory neuropathy	Mean	Ml	7.30	4.23	6.92	13.84
		M2	0.00	4.23	6.92	3.00
	SD	Ml	16.40	16.43	15.75	27.47
		M2	24.62	16.43	15.75	23.47
t-values		Ml	1.29*	1.01*	0.33	0.83*
		M2	1.67*	0.67	3.15	0.13

* P>0.05; ** P>0.01

Table-6: Mean and standard deviation values of recruiting and auditory neuropathy ears along with t-values (Ml-Method 1:M2-Method 2).

Table-6 shows that between the auditory neuropathy group and recruiting SNHL group, there is a significant difference in mean at all frequencies except 2 kHz at 0.05 level, in method 1. In method *2,* only at 500 Hz, a significant 0.05 difference can be seen at 0.05 level for the mean O-T gap.

Correlation:

Karl Pearson's product moment correlation was carried out to see the relationship between behavioural threshold and DP threshold for each group. Results are given in the table-7.

Frequency Measure		500 Hz	1kHz	2kHz	4kHz
Normals	Mean M1	0.266	0.497	0.152	0.125
	M2	0.166	0.49	0.520	0.125
Non-recruiting	Mean M1	0.453	0.650	0.612	0.612
	M2	0.176	0.20	0.49	0.01
Recruiting	Mean M1	0.035	0.632	0.482	0.433
	M2	0.30	0.12	0.13	0.13
Auditory neuropathy	Mean M1	-0252	-0.003	-0.021	-0.048
	M2	-0.54	0.008	-0.06	0.614

Table-7: Correlation values of normal, non-recruiting ears, recruiting ears and auditory neuropathy ears for behavioural and DP thresholds (Mi-Method 1; and M2-Method 2).

In auditory neuropathy group low negative correlation was found between DP threshold and behavioural threshold. In method 2 at 4 kHz low positive correlation was found.

For all the groups except the auditory neuropathy, a positive correlation was found between the DP thresholds and behavioural threshold **i.e. with** increase in the behavioural threshold, an increase in the DP threshold was also observed.

In the auditory neuropathy group, a negative correlation was seen between DP thresholds and behavioural thresholds. It was found that cases with auditory neuropathy group always had DP threshold either near or below their behavioural thresholds.

DISCUSSION

Recruitment, or the abnormally rapid growth of loudness with intensity is a common disturbance of listeners with sensorineural hearing loss.

Till date, recruitment is considered as a characteristic feature of cochlear hearing loss. Fowler (1928), for the first time spoke about recruitment and in 1937 and attributed it to the cochlear dysfunction. This was followed by a series of studies which tried to explain the origin of phenomenon of recruitment (Lurie, 1940; Tonndorf, 1980; Steven, 1936; Salvi, 1983; Kiang, et aL 1970; Evans, 1976; Simmons and Dixon, 1966; Salvi, et al. 1991; Brownell, 1990; Berlin, 1990).

Though more than six decades have passed since Fowler proposed the possible origin of recruitment, still the scientific world has not been able to successfully explain the phenomenon of recruitment and its origin.

Gorga et al. (1993a); Ricci et al. (1996); Suckfull et al. (1996); Kim, et al. (1996); Kimberly and Nelson (1989); Harris (1990); Lonsbury-Martin and Martin (1990) and others concluded that DPOAEs are successful in distinguishing normal ears from sensorineural hearing loss ears i.e. cochlear hearing loss. In the present study significant difference was found between the mean of O-T gap of normals vs. other pathological groups; i.e. recruiting, non-recruiting sensorineural hearing loss groups. Thus this study supports the literature in concluding that

based on DPOAEs, threshold, normal ears can be distinguished from ears with sensorineural hearing loss. No significant difference of O-T gap of recruiting and non-recruiting hearing loss groups was seen in method 2, except at 4 kHz, where the difference of mean was found significant at 0.05 level. In method 1, high significant difference was found to 2 kHz and 4 kHz but low at 1 kHz. At 500 Hz no significance difference was found. However, it was found that the mean O-T gap of recruiting ears were lesser than non-recruiting ears as Allen (1995) stated that DPOAE measures with correlate with recruitment rather than behavioural thresholds.

Patuzzi (1993) reported that in sensory loss, threshold elevation will show little recruitment with normal OAEs. Causes of such a condition can be genetic or malformation of IHCs or primary afferent fibers, ototoxic oto destruction of IHC or afferent fibers synaptic (problems), or degeneration of afferent fibers.

In the recruiting sensorineural hearing loss group inspite of the presence of objective recruitment, DPOAE thresholds could be obtained in 77% of the subjects, as low as 55 dB, indicating the OHCs are functioning. In non-recruiting ears, out of 18 subjects DP-emissions could not be obtained for 4 subjects at the maximum stimulus level (70 dB SPL) indicating OHCs dysfunction present in these ears. But none of these cases were found to have either subjective or objective recruitm nt.

From this we can infer that damage of OHCs are not solely responsible for the phenomenon of recruitment. During the course of

study, we came across cases where behavioural thresholds were in the range of mild-moderate hearing loss and ABR findings showed no response at 90 dB nHL. DP threshold of these cases were found to be near or well below their behavioural thresholds thus indicating auditory nerve pathology. Similar findings have been re|X)rted earlier in literature (Lutman et al. 1969; Prieve, et al. 1991; Monroe et al. 1996; Konradsson, 1996; Park and Lee, 1998;Robinette, 1999). Around50% of these cases had reported of subjective recruitment (reduced uncomfortable level). This further supports the view that recruitment not an OHC level phenomenon. Origin may be of this phenomenon may be at the level of brainstem same as reported by Salvi (1991).

Frequency Measure		500 Hz	1kHz	2kHz	4kHz
Normals	FIT	12	9	6	7
	DPT	47	35	40	37
Non-recruiting SNHL,	FIT	40	37	34	39
	DPT	61	54	56	61
Recurring SNHL	PTT	45	45	48	50
	DPT	65	56	53.9	57
Auditory neuropathy	PTT	47	38	32	34
	DPT	48	45	40	45

Table-8: Behavioural thresholds and DP thresholds of different groups (PTT - Puretone thresholds; DPT -DP thresholds).

The table 8 gives the mean of the behavioural thresholds and DP threshold of each group. On one glance to the above table, it can be

seen that the means of the DP thresholds of the auditory neuropathy group at all the frequencies of near or equal to the DP thresholds of the normal group. Though mean behavioural threshold is higher in auditory neuropathy cases. On the other hand, in recruiting SNHL and non-recruiting SNHL, mean DPT is seen to higher than the normal.

When correlation was found between the behavioural thresholds and DP thresholds of each group. For both, recruiting SNHL and non-recruiting SNHL group, a positive correlation was found.

Kimberly and Nelson (1989) correlated DPOAE emission with auditory thresholds. The results showed a positive correlation with correlation coefficient of 0.86 formalinear relationship between auditory sensitivity and distortion product emission (Gorga et al. 1993a; Nelson and Kimberly, 1992; Probst and Harris, 1993; Kimberly et al. 1994).

In the auditory neuropathy cases, a negative correlation was found between the behavioural thresholds and DP thresholds. This may be because DPOAEs are a pre-neural phenomenon and are not sensitive to neural nerve level lesion (Patuzzi, 1993; Park and Less, 1996; Robinett, 1999; Lonsbury and Martin, 1990; Kaga et al. 1996).

Whenever a negative correlation between the DP thresholds and auditory thresholds or DP thresholds was obtained at normal level diagnosis of neural hearing loss can be made. Hence DPOAE can be used as a successful tool for distinguishing retrocochlear pathology from

Till date, recruitment was considered as a hallmark of cochlear hearing loss, but findings of this study imply that, phenomenon of recruitment is not at all restricted to cochlear hearing loss as even retrocochlear pathology cases report of recruitment. So rather than making recruitment as a basis of diagnosing cochlear pathology, DPOAEs will act as a better tool. Though ABR has established itself as a valid and reliable tool for the differential diagnosis of retrocochlear pathology (RCP) vs. cochlear pathology (CP) but DPOAEs score above ABR in terms of the specificity to cochlear physiology, high sensitivity for cochlear lesion especially OHCs, testing duration etc.

DPOAEs can play an important role in the selection for the candidacy for cochlear implant. Presence of DPOAEs in the subject would not only indicate a satisfactorily functioning cochlea but also failure of cochlear implant in view of the apparent retrocochlear lesion (Robinett, 1999).

SUMMARY AND CONCLUSION

Otoacoustic emission have been a developing clinical tool in the recent past amongst which the distortion product otoacoustic emission look promising as a diagnostic test

Till date, the phenomenon of recruitment has been considered as a hallmark of cochlear hearing loss occurring due to the dysfunction of the OHC.

The present study was taken upto probe :

(a)Whether DPOAE is successful in distinguishing recruiting SNHL from non-recruiting SNHL.

(b)Based on DPOAE findings to explain the possible site of the phenomenon of recruitment.

(c)To see whether DPOAE is a tool to differentially diagnose between RCP and CP.

30 normal ear from 15 subjects, 18 recruiting, 18 non-recruiting SNHL from 18 subjects and 13 ears with auditory neuropathy were included in the study. Behavioural audiometry and DPOAE testing were carried out for all the three groups. ABR was obtained at 90 dB nHL with a repetition rate of 11.1/sec. to confirm auditory neuropathy.

DPOAE testing was done using Madsen Celesta 503 cochlear emission analyser with input-output paradigm selected. Geometric mean frequencies of approximately 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz were tested across intensities from 70 dB SPL to 10 dB SPL.

The results are as follows. The mean DP-mreshold for both recruiting SNHL and non-recruiting. SNHL was found to be higher than that of normals at all the geometric mean freqnencies of approximately 500 Hz, 1 kHz, 2 kHz, and 4 kHz.

At all the frequencies except 500 Hz, a significant difference was found between the mean of the O-T gap of recruiting SNHL and non-recruiting SNHL. But from this observation it cannot be inferred that recruitment is solely a OHC level phenomenon because on a closer look, it was observed that 77% of the recruiting SNHL group had OAE present and in non-recruiting group there were instances when even at maximum stimulus level DP emissions could not be obtained. - lso in the auditory neuropathy group, around 50% of the cases had subjective recruitment. This further supports the view that recruitment may be a phenomenon, which takes place at a higher level, as reported by Salvi et al. (1991).

On comparision of the DPT and PTT of each group, it was found that both recruiting and non-recruiting group showed a positive correlation indicating a sensory level involvement Whereas in the auditory neuropathy group, a negative correlation was found which indicated that the hearing loss is due to the involvement either at the level of IHCs or beyond IHCs.

The presence of recruitment in the 50% of the auditory neuropathy group contradicts the myth that the phenomenon of recruitment is present only in the cases with damaged cochlea. Hence DPOAE is a better tool to differentiate sensory loss from neural loss. In addition, it also aids in the managements of me profound hearing loss cases especially for the cochlear implant candidacy.

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