EFFECTS OF 4 kHz DIP ON AUDITORY BRAINSTEM RESPONSE

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

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MAY 1999

CERTIFICATE

This is to certify that this Dissertation entitled : ***EFFECTS OF 4 kHz DIP ON AUDITORY BRAINSTEM RESPONSE*** *is the bonafide work in part fulfilment for the degree of Master of science (Speech and Hearing) of the student with Register* ***No.M9707.***

Mysore
May, 1999

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CERTIFICATE

This is to certify that this Dissertation entitled: *EFFECTS OF 4 kHz DIP ON AUDITORY* ***BRAINSTEM RESPONSE*** has been prepared under my supervision and guidance.

Mysore
May, 1999

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DECLARATION

This Dissertation entitled : *EFFECTS OF 4 kHz DIP ON AUDITORY BRAINSTEM RESPONSE* is the result of my own study under the guidance of *Ms. Vanaja C.S.* Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other diploma or degree.

Mysore
May, 1999

Reg. No.M9707

Aniruddh Sir,

Vanaja Maa'm

Maa - Baba

I express my deep regards and heartfelt gratitude towards my teacher and guide Mrs. Vanaja C.S. Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore for all the patient listening to my disorganised ideas. Maa 'm your knowledge and wisdom makes me bow and feel proud to be your student.

I am grateful to Dr.(Miss) S.Nikam, Director, All India Institute of Speech and Hearing, Mysore for permitting me to carry out the study.

Each day I live, each step I climb has the foundation of your loving care. Maa and Baba with your blessings, I promise to scale the heights you dreamt for me. I must thank the other three members of my family dada, didi and Robinda for making me live a dream, my life.

Krithika for all yon are, words are mute. Your understanding, even being around makes me feel cozy amidst disasters.

My dear classmates, far and near, I thank you all for metamorphosizing me into a person-real. Each of you hold special places in my heart for each of you have contributed in my remake.

My loving juniors, Tyagi, Siddarth, Rampo, Jose, Praxi, Ajith and Vijay this line is to acknowledge the innumerable encouragements you guys extended when I was down with failures. You all will form the sweetest of my AIISH memory.

Hia, Kavitha, Chaya, Piyali and Siva, I wish your prayers may always be there for me. They boost the confidence in me when I need it most. Thank you all.

Akka, I must pity your eyes for reading through my hand writing and must praise your fingers to change them into a glazy print.

TABLE OF CONTENTS

INTRODUCTION

Noise, a slow poison to the ear like any other pollutant is unavoidably present in the everyday environment of the civilized man and is increasing with every minute added. The factories, vehicles and other machineries along with their comforts and ease push us to a noisy world everyday.

An exposure to high level noise causes a deterioration of the hearing sensitivity. The auditory effects of exposure to loud noise are typically reported as a change or shift in thresholds either temporary (TTS) or permanent (PTS). A TTS results due to some anatomical changes occurring as the initial impact of exposure to high level of noise. These changes slowly disappear and the condition . of the auditory system recovers after the noise is terminated. But, in case of severe damage due to very high intensity noise or prolonged stimulation, the anatomical conditions do not improve. The changes are reflected as a permanent shift in the auditory threshold (PTS). The anatomical changes due to a noise exposure can vary from microtrauma to Cillia, their actin and myosin (Strlioff and Flock, 1984; Nielsen and Slepecky, 1986) to other vascular, metabolic, ischemic and ionic disorders (Jerger and Jerger, 1981; Bohne, 1976). The anatomicnl changes that are seen soon after a high level noiso exposure for a short duration fire slight distortion of the OIIC, which take on an irregular shape instead of normally regular cylindrical shape. The next step is an outward bending and occasional fusion of the sensory hairs of the IHCs. Frequently the afferent fibers and their

endings below the IHCs are greatly swollen and occasionally even burst (Spoendlin, 1976). Temporal bone analysis has shown that Noise Induced Hearing Loss (NIHL) or acoustic trauma may cause a damage ranging from mild swelling of OHCs to absence of the organ of corti. OHCs are first affected with the effect on IHCs resulting from continued and/or more intense exposure. Secondary degeneration of ganglionic cells and nerve fibres occur where all hair cells have been eliminated but not where partial hair cell loss exists and is more pronounced in the basal region (Hood and Berlin, 1986).

Physiologically these hair cell damages due to noise exposure results in broadening of the psychophysical as well as the neural tuning curves in the exposed frequency region. Thus the thresholds elevate and the region of basilar membrane acts as a low pass filter (Henderson and Salvi, 1998). These anatomical changes are also reflected in the reduction of hearing sensitivity. As the severity of damage increases with increase in exposure time and/or intensity the amount of deterioration in hearing sensitivity also increases. In terms of physiological changes in intensity coding, abnormal loudness growth was reported by 1 Inllpike nnd 1 lood (1 960), Salvi, ct al. (1992) measured the evoked response amplitudes and found them to be depressed in the region of greatest hearing loss and increased in the lower frequency region. In terms of temporal coding, temporal integration was found to be disrupted (Henderson, 1969; Soleck and Gerken, 1990; Watson and Gengel, 1969; Wright, 1968). These changes are found to be more pronounced in the basal part specially in the region corresponding to 3-6 kHz range in the cochlea. This portion of ear is particularly vulnerable to permanent hearing loss

due to noise (Burns, 1973). As it progresses in severity, the hearing loss extends to other frequencies. Initially the puretone audiogram, shows a gradual deterioration in 3 to 6 kHz region. Most often 4 kHz frequency is the one that is affected maximally with neighbouring frequencies retaining normal thresholds. This gives a pattern of 'dip' or notch at 4 kHz frequency. This is popularly known as the 4 kHz dip audiogram. This is considered to be a hallmark for NIHL. This 4 kHz, dip is observed irrespective of the particular noise environment (Burns, 1978). This increased susceptibility of 3-6 kHz region, particularly the 4 kHz can be due to the following reasons :

(i) The 4 kHz area of the basilar membrane which is usually affected by noise lies in the basal turn of cochlea, This bears the initial impact of sound waves stimulating the inner ear, particularly those of higher frequency which travel directly across the middle ear space by para ossicular conduction. At this point the basilar membrane is more firmly fixed, thus subjected to more torsion and so more liable to undergo degenerative changes. A weakness of the bony capsule corresponding with the critical 4 kHz dip has been demonstrated (Kelemen, 1962).

(ii) Resonance in the external auditory meatus increases the amplitude of sound waves between 2 kHz and 3.5 kHz by about 20 dB compared with waves at other frequencies (Onchi, 1951).

(iii) Hydrodynamic causes were demonstrated by Von Bekesy (1953) to be responsible for the maximal sensitivity of the ear between 1 and 4 kHz. Stimulation in any specific frequency range

activates regions of the basilar membrane responsive to higher frequency specificity (Davis et al. 1953; Hood, 1950; Littler, I966).

(iv) Summation of' TTS curves resulting from several puretones recorded in the same diagram, is found to occur at or above 4 kHz (Albcrti, 1979).

The changes in the auditory system are reflected upon various audiometric tests both behavioral and physiological or electrophysiological.

The effects of initial minimal loss in the 4 KHz region on electrophysiological tests such as ECochG and ABR remains subtle. ABR peaks are believed to be generated from distinct higher nuclei in the CAP and hence uneffected by the peripheral changes. Cochlear pathology was believed to prolong all the peaks equally as the site of lesion is before the generation site of I peak. As a result I-V LPL supposedly a measure of central conduction time was believed to be constant in cochlear pathology. But for the stimulation of nerves, cochlea is required and there exists a tonotopic organization along the length of the basilar membrane. As the travelling wave has to travel from the basal part to the apical part to stimulate it, there is a small time difference between the stimulation of the two ends of the basilar membrane. This concept of cochlear travel time is hypothesized to induce a differential change in L-I function of subjects with sensori-neural hearing loss of different audiometric

configurations (Goldstein and Kiang, 1965; Borg, 1981; Don, Eggermont and Brakmann, 1979; Coats and Martin, 1977; Keith and Grevillc, 1987).

Bauch and Olsen (1986) studied the ABRs in 4 kHz losses. They reported that ABRs were normal till the hearing loss was restricted to 4 kHz only and was not more than mild in degree. When the loss extended to 3 kHz and hearing loss at 4 kHz was more than 40 dB, the latency of wave V was prolonged. Similar results were reported by Xu et al. (1998). Investigators also report of prolonged I wave latency with unchanged I-V 1PL in cases with high frequency hearing loss (Aran, 1971; Elberling, 1974; Montandon et al. 1975; Odenthal and Eggermont, 1974; Yoshie, and Ohashi, 1969).

Keith and Greville (1987) reported that in their subjects with 3-4 kHz notched loss a prolonged V wave and an earlier I wave was observed. This resulted in an increase in the I-V IPL. They found prolonged wave I in subjects with sloping high frequency loss consistent with the reports of previous authors.

However, there have been very few studies on the effects of this 4 kHz dip on the ABR waveforms. In the studies reviewed a clear discrepancy is evident regarding the changes in latency and wave form nature in cases with 4 kHz notched audiogxam. Hence this study was designed to observe the subtle effects of 4 kHz dip on the ABR waveform parameters, checking the possibility of developing a criteria for identification of cases with 4 kHz dip based on the observed changes.

REVIEW OF LITERATURE

A prolonged exposure to noise above the risk level causes a damage to the auditory system. The most vulnerable part to this damage is the 3 kHz - 6 kHz region of the auditory system (Bums, 1973). This is reflected as a decrement in hearing sensitivity in this region. A puretone audiogram most often shows a selective increase in the threshold for 4 kHz, puretone and hence popularly known as "4 kHz dip". This decrement in hearing sensitivity being less in degree and cochlear *in* etiology in its initial stages does not produce much observable changes in the ABR wave forms (Xu et al. 1998). Click evoked ABR which lack frequency specificity has not been used as a tool to detect such changes in the auditory system. However, a few studies have been conducted to observe the subtle changes in the electrophysiological responses caused by hearing loss due to noise exposure. As these early changes are sensory in nature only EcochG and ABR studies have been reviewed.

Studies on EcochG

Investigators have used EcochG to study the cochlear microphonics (CM) and the action potentials (AP) in subjects with the NIHL or other steep sloping hearing loss, due to cochlear pathology. Effects of these hearing losses on the threshold, latencies and amplitude of the cochlear and auditory nerve potentials have been studied.

Pugh et al. (1974) conducted a study on non-human primates to see the effect of noise exposure on the electrophysiological responses. They recorded the behavioural and electrophysiological thresholds (for CM and AP) for 1 kHz, 2 kHz, 4 kHz, 8 kHz and 10 kHz tone bursts before and after an exposure to octave band noise centered at 8 kHz at 114 dB SPL. They found behavioural threshold shifts for all frequencies above 500 Hz. Maximum shift of 30 dB was seen at 10 kHz followed by 22 dB at 8 kHz immediately after the exposure for 30 minutes. It was observed that in general threshold shift for the N_1 potential was greater for higher frequencies (>4 kHz). Amount of shift for electrophysiological thresholds was slightly lesser was than that for behavioural thresholds. The I/O function for N_1 was observed to be more steeper in the post exposure recordings for high frequency especially at 8 kHz. The amplitude for N_1 was also depressed immediately after the exposure.

Coats and Martin (1977) also reported of elevation in the thresholds for AP in their human subjects with cochlear pathology This AP threshold for clicks showed a good correlation with the behavioural thresholds in the mid frequency (1-4 kHz) region.

To investigate the effect of noise on the latency and amplitude of the cochlear potentials. Sohmer and Pratt (1975) recorded EcochG to 60 dB SL clicks from ten normal hearing adult subjects before, during and after an exposure to white noise at 90 dB SL for a total duration of 15 minutes. The recording during the noise exposure was done after an initial exposure of 10 minutes. They observed a behavioural TTS of 15 dB lasting for 30-60 minutes. This

TTS was found to be accompanied by a decrease in amplitude and increase in latency of the N_1 potential. The decrease in N_1 potential was apparent in the recording during the exposure (after 10 minutes exposure). They attributed this to the decrease in the number of fiber activated and/or to a decrease in the neural synchrony of firing. In the recovery phase, latency of N_1 potential recovered faster than the amplitude. Prolongation of latency of AP in subjects with high frequency hearing loss at supra threshold levels has also been reported by Coats and Martin (1977) and this prolongation correlated well with the amount of hearing loss at 4 kHz to 8 kHz region. In another study on TTS by Bcnitcz et al. (1 972), shifts in the CM latency were reported along with cochlear hair cell damage.

Thus, it can be concluded from these studies that the effects of noise induced hearing loss or high frequency hearing loss due to other cochler pathologies has a three fold effect on CM or AP. They are - (i) Elevation of threshold, (ii) Increase in latency, (iii) Decrease in amplitude.

Studies on ABR

Though ABR had mainly been a tool to assess the neural functioning and differential diagnosis between cochlear vs. retrocochlear pathologies, different cochlear pathologies also have been reported to affect ABR differently (Keith and Greville, 1987; Xu et al. 1998; Oates and Stapells, 1992; Rosenhamer et al. 1981). These studies report of significant deviations in the ABR waveforms

in cases of high frequency loss due to cochlear pathology. These deviations have been in terms of absolute latency, amplitude and interpreak intervals of different peaks.

A majority of the investigators have studied the effect of sloping high frequency cochlear hearing loss on the latency-intensity function (L-I function) of the V peak (Gorga et al. 1985; Oates and Stapells, 1992; Rosenhamer et al. 1981; Bauch and Olsen, 1986; Keith and Greville, 1987). Studying the L-l function for the wave V in 103 patients (194 ears) with various degrees of cochlear impairment, Oates & Stapells (1992) found that the latency of wave V increases as hearing threshold at 4 kHz increases. Also the slope of the function for wave V latency vs. hearing loss was more at low intensity. The L-I function was found to be steeper at lower intensities as compared to the higher levels. This relationship between 4 kHz hearing threshold and the wave V latency has been agreed upon by many investigators (Coats, 1978; Coats and Martin, 1977; Jerger and Mauldin, 1978; Moller and Blegvad, 1976; Rosenhamer et al. 1981). Arslan, Prosser and Rosignoli (1988) studied the wave V latency *in* 308 subjects with cochlear hearing loss. Analysis was carried out to study of correlation of wave V latencies with the hearing loss from 2 to 4 kHz (an average of thresholds at 2 kHz and 4 kHz) and audiometric profile. Both these factors i.e. average threshold of 2 KHz **and** 4 KHz and **the** audiometric profile show a highly significant positive correlation with the latency. Bauch and Olsen (1986) reviewed the puretone hearing sensitivity at 2 kHz, 3 kHz and 4 kHz and ABR results for 458 patients with cochlear hearing loss in an effort to evaluate the influence of the

peripheral hearing loss on wave V latency. They found that cochlear pathology resulting in peripheral hearing loss influences the latency of wave V or distorts the waveform for many patients particularly when contribution from the basal portion of the cochlea is diminished. However, they added that the region corresponding to 2 kHz also exerts an influence on wave V latency or overall waveform. They concluded from the study that the threshold sensitivity at all frequencies from 2 kHz to 4 kHz influenced ABR latency or waveform. Hearing sensitivity at 3 kHz was found to influence responses more than sensitivity at 4 kHz level. Also when the audiomctric contours raising between 2 kHz and 4 kHz arc more likely to yield normal ABR. Jerger and Johnson (1988) in their study on a large series of patients with sensory hearing impairment, reported that latency is stable for hearing loss upto 60 dB HL, and then it increases linearly to a maximum of about 0.4 msec, through 90 dB HL, with most pronounced latency change in patients with hearing loss more than 70 dB 1 1L.

The L-I function has repeatedly been reported to reveal a minimal latency increase in Wave V at high stimulus intensity levels despite moderate to severe sensory loss (Coats and Martin, 1977; Jerger and Mauldin, 1978; Brackmann and Selters, 1977; Sohmer et al. 1981). In fact, the ABR wave I-V latency interval is relatively unaltered by the pure sensory hearing loss, so the minimal shift with hearing loss is not due to changes in wave V latency but, rather due to prolongation latency of ABR wave I (EcochG AP) (Aran, 1971; Elberling, 1974; Montandon, et al. 1975; Odenthal and Eggermont, 1974; Yoshie and Ohashi, 1969).

The contribution of different frequency regions of cochlea to different waves of ABR has also been studied under experimental conditions using masking noises. Klein in 1979 studied the location of generation of wave I and V using the masking paradigm using a 4 kHz tone pip as stimulus. He observed that wave I was more sensitive to high frequency maskers than wave V, latency of wave I was severely affected by 5-8 kHz high pass masking noise whereas wave V showed a marked increase in latency with 5 kHz low pass noise. On the other hand a low pass noise with cutoff frequency slightly above 4 kHz had no effect on the wave I latency or amplitude in adult subjects.

Again this notion that wave T is maximally dominated by the activity in the basal part of the cochlea is supported by the findings of Goldstein and Kiang (1958), Xu et al. (1998), Struzebecher et al. (1985). They reported that detectability of wave I decreases with increase in hearing loss.

There is lack of consensus regarding the effect of high frequency sensory hearing-impairment on the inter waves (I-V) latency interval of ABR. A few investigators report a. significant decrease in the inter-wave latency difference in patients with high frequency sensory impairment (Coats and Martin, 1977; Keith and Greville, 1987; Struzebecher et al. 1985). Coats and Martin (1977) in their study using simultaneous recording of EcochG and BSERA found that the inter wave latency difference between wave N_1 and wave V was reduced in subjects with high frequency hearing loss.

In contrast, some investigators demonstrated no change in wave I-V latency values with high frequency sensory loss (Rosenhammer et al 1981 Abramovich and Billings, 1981 Eggermont et al. 1980). Rosenhamer el a1. (1981) recorded the I-V and III-V IPL in 110 cochlear hearing loss with various configuration and with various etiologies. In 77 subject with high frequency hearing loss, the changes *in* I-V and III-V IPL were found to be in significant and did not correlates with hearing loss at 4 kHz. They attributed the difference between their finding and that of Coats and Martin's (1977) that the previous (Coats and Martin's) study considered the N_1-V IPL in case I peak in ABR waveform was absent. And this N_1 was recorded from the EAM.

The hypothesis that the wave latencies are determined not only by the neural generators but by the area of activation in the cochlea too was affirmed by Gorga et al. (1985). They reported I/V interpeak latency to be just below the normal range in a subject with high frequency conductive hearing loss (mild to moderate degree of loss above 500 Hz). Interestingly, they observed a steep L-I function for the wave-V. They attributed this to be basal spread of excitation at higher intensities. Conductive loss acting as an attenuator reduced the effective amplitude of the high frequency components of the stimulus reaching the cochlea thus reducing the basal spread and in turn prolonged the wave V at lower intensities.

Keith and Grcvillc (1987) in their extensive study on effects of audiometric configurations on ABR studied the latencies of wave

I and V and the I-V IPI. in 47 ears with high frequency 8 ears with low frequency, 44 ears with that frequency and 27 ears with notched (4 kH7.) loss configuration They high lighted their findings based on both, **the** configuration as well as the stimulus intensity. The wove 1-V IPL tended to be shorter in sloping high frequency was and in raising configerations, unchanged in flat configuration, and in cases with notched configuration the I-V IPL was found to be prolonged. Differences in the wave I-V latency interval were greatest for lower stimulus intensities (below 50-60 dB HL) consistent with the data of Coats and Martin (1977). Thus, they opined that the I-V interval is not a pure measure of central conduction time.

However, Xu et al. (1998) *in* their study of location of the pathological processes the NIHL using EOAE and ABR reported that with mild NIHL only at 4 kHz, the ABR parameters, i.e. I-V IPL and I/V amplitude ratio were within normal range. But when hearing loss extended to 3 kHz and the degree of hearing loss increased, the wave V latency increased and also wave I became less detectable.

Bauch and Olsen (1986) also reported that when 2 kHz, sensitivity is normal and 3 kHz and 4 kHz thresholds are 35 dB HL or better, ABR results are normal for cochlear hearing loss patients at least 94% of the time.

There can be several factors contributing to these discrepancies. They can be the stimulus factors such as use of high intensity levels (80 to 90 dB nHL) for click by Bauch and Olsen (1986) and Xu et al. (1998) as compared to the inclusion of lower

intensities by Keith and Gieville (1 987) to obtain a L-i function. Other acquisition factor like the electrode placement as pointed by Rosenhamer et al. (1981 a) to attribute differences between their findings and Coats and Martin's (1977) can also be the contributing factors. Other authors like Coats and Martin (1977) and Ornitz and Walter (1975) also acknowledge the contribution of these stimulus and subject related factors to these discrepancies.

The amplitude of the ABR peaks (I and V) have also been studied in relation to the high frequency cochlear hearing ioss. The amplitude of I peak of ABR was found to be decreased relative to the amplitude of V peak these reducing the F*/V* amplitude ratio in case *of* high frequency cochlear hearing loss. Also this decrease cutoff loss (Musiek, 1984; Xu, et al. 1998). However, Xu et al. (1998) reported that the 1/V amplitude ratio did not decrease until the hearing loss extended to 3 kHz from 4 kHz and was more than mild in degree.

Klein (1986) in his study using masking paradigm found the wave I amplitude reduced with presentation of a high pass masking noise.He speculated amplitude of wave I as a sensitive measure to identify disruption of cochlear and/or the corresponding neurons as in the case of NlHLs.

Several investigators (Borg,1981; Coats and Martin, 1 977; Don et al. 1979; Galambos and Hccox, 1977; Gorga et al. 1985; Ozdamar and Hallos, 1976; Paantev et al 1 985; Sohmer et al 1981; Yamada et al. 1 979; Keith and Greville, 1 987) have cited the concept

of travelling time along the basilar membrane to explain the latency variation of the ABR waves or cochlear potentials in high frequency or low frequency region. In brief, according, to this concept, wave V is generated normally by activity in the base of the cochlea (the high frequency region). In general, for normal hearers, high intensity energy at 4000 Hz, activates a wider portion of the basilar membrane and approximates the most basal end of the cochlea. High frequency hearing-impairment alters the normal cochlear generator sites for ABR wave I, but not the basilar-membrane sites for wave Y With high intensity stimulation, which exceeds the degree of hearing loss, wave V continues to be generated in part by basal cochlear activity as well as more apical activity and latency is reasonably normal. The travel time from the 10,000 Hz fo the 500 Hz region of the cochlea is approximately 2 ms (Borg, 1981 a).

Thus, with loss the different frequency region (basal or apical) the wave I and wave V are differentially effected at least in the lower frequency. However the reported effects on the parameters of ABR, viz. I-V IPL or absolute latency of I and V wave and their relative amplitude is not *in* consensus.

METHODOLOGY

This study aimed at investigating the effects of a 4 kHz dip on (he ABR waveform. For this, hearing loss at 4 kHz was simulated in normal hearing subjects using puretone masking paradigm. A pilot study was conducted to ascertain the frequency specificity of this masking procedure.

Pilot Study

SUBJECTS

5 normal homing young adults wi(h purclonc micliomctric thresholds below 20 dB III- in the frequency range of 250 Hz 8 kHz. were included for the study. The subjects had no middle ear pathology on immittance screening. There was no significant audiological/otological history.

PROCEDURE

Using a double channel clinical audiometer (GSI-10) audiometric thresholds for puretone were obtained in the frequency range of 250 Hz. -8 kHz.

Selecting ipsilateral masking set up (tones selected in the two channels directed to the same ear), a 4 kHz puretone was used as the masker. Thresholds at 3 kHz,, 4 kHz and 6 kHz were rechecked in the presence of the 4 kHz masker.

. The results of the pilot study showed a significant shift in 4 kHz frequency with minimal or no shift in the neighbouring 3 kHz or 6 kHz threshold. Hence this masking paradigm was used to simulate a frequency specific loss in the subjects for the main study.

MAIN STUDY

SUBJECTS

30 young adults were taken selection criteria being the same as that in the pilot study.

INSTRUMENTS

The following instruments were used for the study :

A calibrated audiometer Madsen OB 822 with TDH-39P cushioned in MX-41 circumaural ear cushions.
GSI-33 (Version 1) middle ear analyser
Nicolet spirit evoked potential system (version 1.5) with TDH-39P earphones.

PROCEDURE

Subjects were screened for hearing loss using Madsen OB 822 clinical audiometer at all the octave frequencies from 250 Hz - 8 kHz. This was followed by immittance screening using GSI-33 middle ear analyser (version 1) to rule out any conductive pathology.

- Two ABR recordings were carried out for each of the subjects.

1) Control recording using broadband clicks of 60 dB nHL (as in Appendix I) at the rate of 11.1 /sec.

2) Experimental recording using 60 dB nHL broad band clicks in presence of 4 kHz puretone ipsilaterally presented at 70 dB SL.

For the ABR recordings, subjects were seated on a comfortable chair. Electrode sites were cleaned using skin preparing gel. Silver coated disc type electrodes were placed using adhesive tapes. For better conduction, conducting paste was used. Impedance for electrodes were essentially 5 kOhms, interelectrode difference not exceeding 3 kOhms.

Broadband clicks used as stimulus for ABR were generated by inbuilt generator in the Nicolet Spirit instrument. The 4 kHz masking tone was presented in the same ear as the clicks by choosing the 'external' option under the noise selection options in the instrument. The puretone was generated by Madsen OB 822 clinical audiometer which was connected to Nicolet spirit. The test protocol for ABR recording was as follows:

(i) Signal

Type	-	Broadband clicks
Rate	-	11.1 /sec
Intensity	-	60 dB nHL
Polarity	-	Rarefaction
Duration	-	100 /μs
No.ofstimuli	-	1600

(ii) Transducers - Headphones (TDH-39P)

(iii) No. of channels - two

(iv) Montages - $Cz - M_1$ and Cz- M2

(v) Time window - 10 msec.

(vi) Sensitivity - 50 uV

(vii) Filter setting - 100 Hz - 3 kHz

From the recorded waveform the absolute latencies of the I, III and V peak and the V/I amplitude ratio was calculated. The I-V, I-III and III-V IPL were derived from the absolute latency measures.

RESULTS AND DISCUSSION

A pilot study was done to check the efficiency of the purctonc masker in producing a hearing loss at a specific frequency. The results of study are shown in Graph 1. Average shift of 35 dB HL at 4 KHz 7dB HL at 3 Khz and 9 dB HL at 0 KHzwas observed. This served as an indication that a purctonc could be used to produce a threshold shift at a specific frequency.

For the main study, ABR waveforms were recorded in two different conditions i.e. the experimental and control conditions. The representative waveform is shown in Fig. 1. These waveforms were analysed to study the latencies of I, III and V peak and peak to peak amplitude for I and V peak. The interpeak latency (IPL) difference for I-V, I-III and III-V and the V/J amplitude ratio were derived from the absolute latencies and amplitudes respectively.

Statistical analysis was carried out using NCSS software. Descriptive statistics which included mean, SD and range was calculated for all the measures - i.e. absolute latency, interpeak latency differences and V/I amplitude ratio in experimental and control conditions. Paired T-test was administered to check if there is significant difference between means obtained in the two conditions. The statistical summary is shown in Table 1 and Graph 2.

It is evident from table 1 that the latencies of wave III and wave V increased in the presence of 4 kHz puretone i.e. in the

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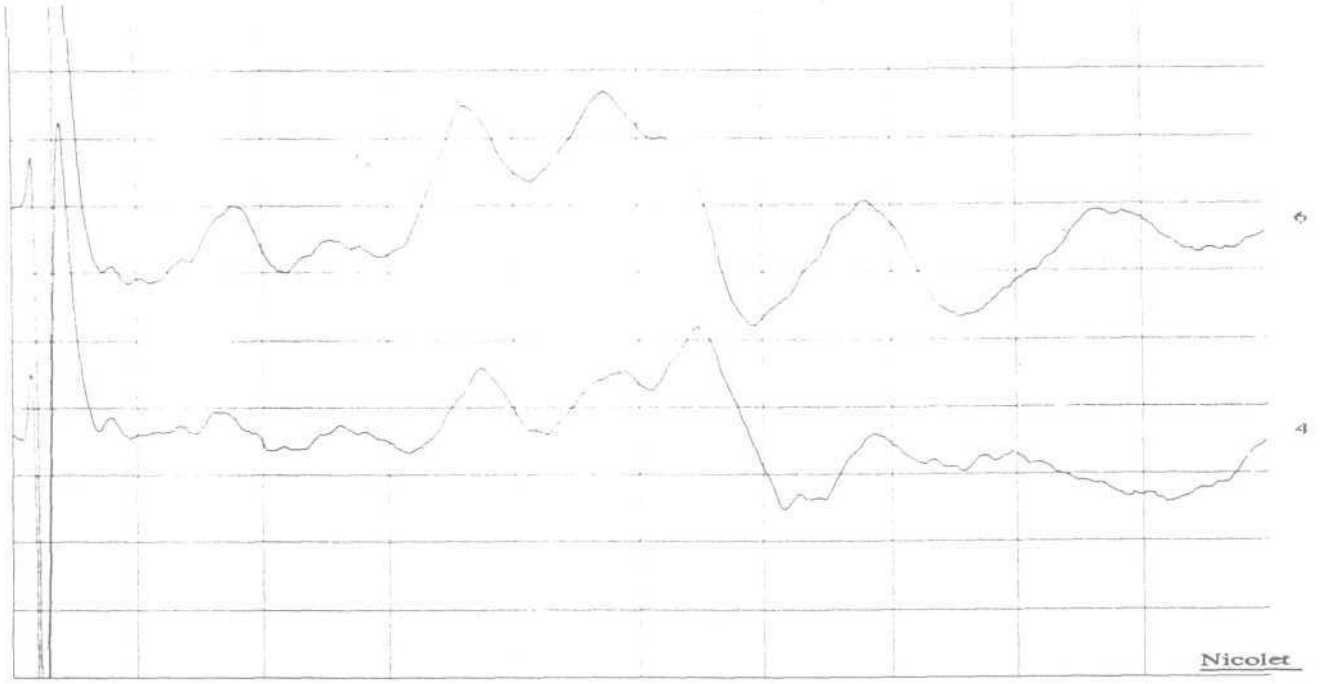
chan, jayaradha

ABR N1



Sensitivity and Sweep Time Per Division

4	0.31 uV	1.0 msec	6	0.31 uV	1.0 msec

Wave	Elect	Date	Time	Remarks
4	Cz–A2	01/12/98	20:25	4000

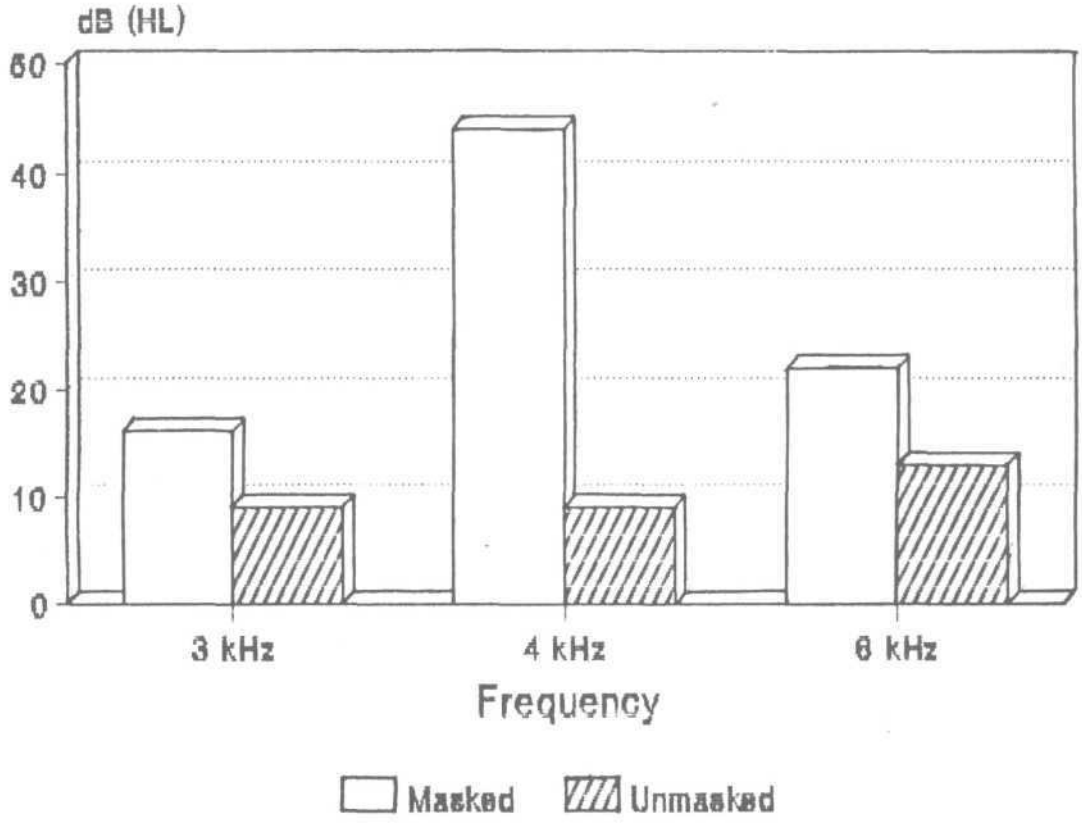
Wave	Elect	Date	Time	Remarks
6	Cz–A2	01/12/98	20:19	no mask

AMP	Elect	Mode	Sns	Lff	Hff	Notch	Artifact	REM	Remarks
4	Cz–A2	Run	50uV	100	3K	Off	On	4	4000
6	Cz–A2	Run	50uV	100	3K	Off	On	6	no mask

ACQ	Comm	Sweep	Time	Delay	Rate	Trigger	Stim	MISC	Type	Ch#	Accept	Reject	Filter	Fsp/SNR	Date	Time	Add	Sub	Inv	Filter	Smooth
4	B	1600	10ms	0ms	11.1	Inter	Gated	4	Sum	2	1600	64	Bessel	3.13	01/12/98	20:25	no	no	no	no	no
6	B	1600	10ms	0ms	11.1	Inter	Gated	6	Sum	2	1600	10	Bessel	7.71	01/12/98	20:19	no	no	no	no	no

STIM	Trans	Type	Pol	Dur	Level	Freq	Pla	Ramp	Env	Noi	NLev	dB	Trans	Type	Pol	Dur	Level	Freq	Pla	Ramp	Env	Noi	NLev	dB
4	Phone	Off								Off		nHL	Phone	Click	Rar	100us	60					Extern	90	nHL
6	Phone	Off								Off		nHL	Phone	Click	Rar	100us	60					Extern	90	nHL

Gr.1:Comparison of Hearing Threeholds
in two conditions



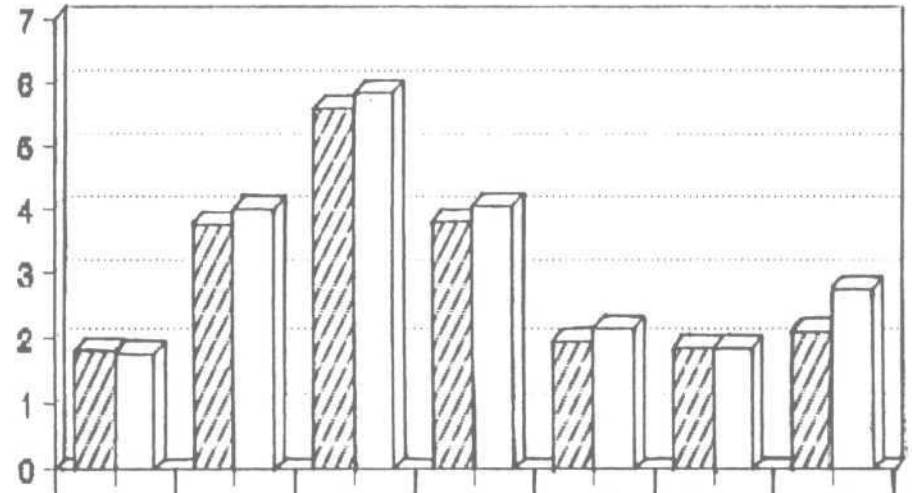
Conditions						
	Control			Experimental		
	Mean (ms)	SD	Range (ms)	Mean (ms)	SD	Range (ms)
I	1.81	0.16	1.58-2.12	1.77	0.15	1.54-2.12
III	3.77	0.18	3.5-4.06	4.00	0.29	3.42-4.5
V	5.59	0.27	5.08-6.1	5.86	0.31	5.26-6.36
I-V	3.81	0.20	3.4-4.22	4.05	0.25	3.58-4.4
I-M	1.95	0.15	1.66-2.22	2.15	0.23	1.82-4.78
I1I-V	1.86	0.18	1.6-2.34	1.88	0.22	1.38-2.36
V/I AMPLITUDE RATIO	2.13	1.31	.72-5.36	2.76	1.69	088-7.5

Table-l:Mean and SD of different parameters in the two conditions.

experimental condition. The latency of wave I was reduced in the experimental condition. As shown in the graph 2, the prolongations latency for wave V and wave III were highly significant (p=0.0768). Similar results i.e. prolongation of wave V has been reported by several investigators in their subjects with high frequency hearing loss (Gorga, et al. 1985; Rosenhamer, 1981; Bauch and Olsen, 1986; Keith and Greville, 1987).

Results of the previous studies have revealed that the latency of wave V is related to the severity of hearing loss at 4 kHz (Coats, 1978; Coats and Martin, 1977; Jerger and Mauldin, 1978; Moller and Blegvad, 1976; Rosenhamer, etal. 1981; Bauch and Olsen, 1986; Arslan, etal. 1988). Bauch and Olsen (1986) demonstrated that the

Gr.2: Comparison of Mean for parameters in two conditions



	I	III	V	I-V	I-III	III-V	V/I
Control	1.813	3.768	5.694	3.813	1.953	1.869	2.134
Experimental	1.766	3.997	5.881	4.053	2.154	1.881	2.762

	I	III	V	I-V	I-III	III-V	V/I
p-values	0.0768	0.0000	0.0000	0.0000	0.0001	0.6296	0.0632

wave V latency remains uneffected in subjects with less than 40 dB dip at 4 kHz, if the threshold is normal at 2 kHz. Prolongation of latency of wave V was observed when the hearing loss at 4 kHz increased to 50 dB HL to 60 dB HL. Keith and Greville (1987) observed a prolongation of latency of wave V in subjects with 4 kHz, dips. The mean threshold at 4 kHz was 58.3 dB HL in their study. In the present study, mean threshold at 4 kHz was 44 dB HL with a mean threshold of 16 dB HL at 3 kHz and 22 dBHL at 6 KHz in the experimental condition.

Thus the results of the present study supports the findings of Keith and Greville (1987) as prolongation of latency of wave V was seen even when the simulated hearing loss was restricted only to 4 KHz and was less than 50 dB. The reasons for difference in results obtained by Bauch and Olsen (1986) could be due to the use of higher stimulus intensity (85 dB nHL to 90 dB nHL) and a lenient criteria of 6.20 ms. In thier study, only wave V latency beyond 6.20 ms. were considered as abnormal.

The present study replicates the findings of Keith and Greville (1987) that the latency of wave I was shortened in subjects with 4 KHz notched audiogram. In their study as well as in the present study this shortening of wave I latency is indicated by the shorter mean in the control condition compared to that in the experimental condition. However, when this change was tested for significance, it yielded an insignificant p value (p=0.0768).

Early appearance of the wave I accompanied by prolongation of latency of wave V resulted in an increase in the I-V IPL. This increase in IPL was significant even at 0.01 level (p=0.0000).

Earlier reports indicated that there is no change in IPL difference in subjects with sensory hearing loss when compared to that in normal subjects (Aran, 1971; Elberling, 1974; Montandon et al. 1975; Odenthal and Eggermont, 1974; Yoshie and Ohashi, 1969). However, Keith and Greville (1987) observed an increase in I-V IPL difference in subjects with 4 KHz dip. Eggermont et al. (1969) and Abramovich and Billings (1981) also reported of an increase in I-V IPL in their cases with sensory dysfunction. The results of the present study supports Keith and Greville's (1987) conclusion that as the I-V IPL is affected by peripheral functions like cochlear hearing loss or masking. Thus, it is not a true measure of the central conduction time.

I-III and III-V IPLs were measured as differential measure for the prolongation of I-V IPL. The I-III IPL was found to be increased significantly (p=.0001) in the experimental condition, whereas the III-V IPL remained unchanged.

Following the speculation of Klein (1986), and Xu et al.'s (1998) finding that amplitude of wave I is a sensitive indicator of dysfunction in the high frequency region, V/I amplitude ratio was considered as one of the parameters for the study. In the present study also, the V/I - amplitude ratio showed an increase *in* the group mean for the experimental recordings, but the difference fell short of

being statistically significant at .05 level (p=0.064). This is consistent with the report of Xu ct al. (1998) that though the means of I/V amplitude ratio showed reduction in terms of mean for the subjects with 4 kHz hearing loss, the reduction was statistically insignificant. However, in the present study, for 6 of the 30 subjects the I wave disappeared in the experimental recording whereas in Xu et al. 's study. I wave was present in all the subjects with 4 kHz hearing loss.

The present findings of prolonged III and V wave with earlier and smaller I wave thus prolonging the I-V IPL and increasing the V/I amplitude ratio can be explained based on the travelling time concept. This concept assumes that the latency of a particular wave depends on the cochlear region contributing to the response. This has been consistently supported by animal and human research (Eggermont, 1976; Eggermont and Don, 1980; Kiang, et al. 1965; Price, 1978). This concept has been used by many investigators to explain the latency variations of different wave components in their subjects with cochlear pathology (Borg, 1981a, Coats and Martin, 1977; Don, Eggermont and Brakmann, 1979; Galambos and Hecox, 1977; Gorga et al. 1985; Ozdamar and Dallos, 1976; Pantev et al. 1985; Shomer et al. 1987; Yamada et al. 1979). It has been found that latencies of waves I. Ill and V to unmasked clicks are normally dominated by activity in the basal 2 to 3 octaves of the cochlea. But with high pass noise masking, a relatively large contribution to wave V from frequencies as low as 400 Hz has been demonstrated (Conroux et al. 1981; Don and Eggermont, 1978). It has further been shows that wave I latency of the unmasked ABR corresponds to the wave I latency for 4 kHz and 8 kHz tone bursts, whereas the unmasked wave

V latency corresponds to that for 1 and 2 kHz center frequency bands (Eggermont, Don and Brackman, 1980).

Thus, whereas wave V is influenced by contributions initiated from basal to apical regions of the cochlea, wave I is highly dependent on basal cochlear function (Borg, 1981; Keith and Greville, 1987). Hence, the distribution of contributing potentials can be depicted as shown in the fig-2.

Fig.2 Representing possible contributing sites in BM for wave I and V

As suggested by Keith and Greville (1987), the shortening of wave I latency can be attributed to the reduction/absence of contribution from the 4 kHz region which lies in the apical end of the contributing part for wave I as shown in the fig 3. This will reduce the peak latency of ABR wave I as the later contributions are reduced.

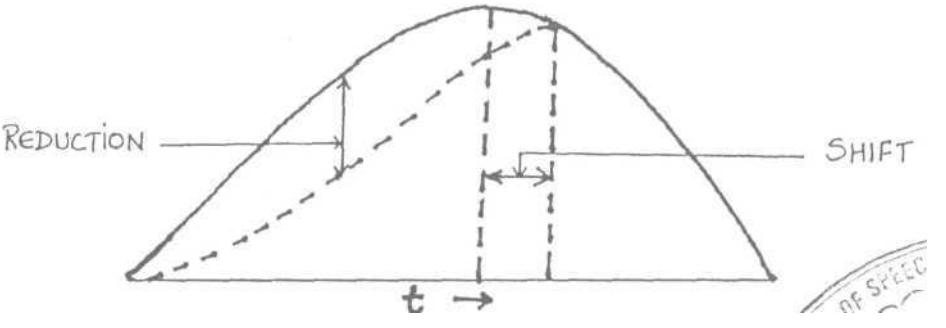


Fig.3 : Showing Shift in Peak Latency due to reduction in early contributors.

Similarly, prolongation of the wave V can be attributed to the reduction in the early potentials for wave V. Increase in the V/I amplitude ratio also suggests a reduction of total potential of wave I as compared to wave V. This may be due to the suppression of maximal contribution for wave I from the cochlea using the 4 kHz masking tone.

Another important finding of this study is the prolongation of wave I-III EPL to significant degree (P=0.000). This suggests that wave III also has a maximal contribution from the apical portions (i.e. frequency region lower than 4 kHz region) of the cochlea. It thus seems that latency of all the ABR waves depends on the contributions from the cochlea.

The second aim of the study was to device a criteria to suspect a subtle 4 kHz dip in the audiogram. For this I-V IPL was taken as the comparing parameter. A range of 1 Std. Dev. across the mean in the control condition was taken as the normal range. Sensitivity and specificity of this criteria were calculated by checking the number of values in the control condition that falls within the ranges for specificity. For sensitivity number of values in the experimental condition that fell above the range or with absent I peak were counted. The sensitivity and specificity measures using I-V IPL as the deciding parameter yielded a sensitivity of 66.66% with a specificity of 73.33%. To differentiate this IPL prolongation from that due retrocochlear lesions, the measures of amplitude ratio can be suggested. In case of retrocochlear pathology, this measure will be decreased whereas it is increased in cases of 4 kHz dip.

Implications of the study

I-V IPL is not a true measure of the central conduction time. It is shown to be affected by cochiear contributions.

Highlights the possible cochlear contributions to ABR waves believed to be generated from higher nuclei in the auditory pathway.

Measures like I-V IPL, latency of wave I and V/I amplitude ratio can be used to detect a 4 kHz dip.

SUMMARY AND CONCLUSION

Prolonged exposure to high intensity noise causes deterioration in hearing sensitivity. This is reflected as a progressive loss in the 3-4 kHz region. Most often 4 kHz is affected first causing a notch nt 4 kHz frequency in ihc nudiogrnm. The loss being less in severity and limited in frequency range affects ABR waveform in very subtle ways.

There has been limited studies investigating the effects of 4 kHz dip on ABR. The results of these studies are equivocal. Hence, this study was designed to investigate the effects of 4 kHz dip on ABR and to suggest screening criteria for predicting a 4 kHz dip based on ABR.

To accomplish the mentioned aims, frequency specific cochlear loss at 4 kHz was simulated in 30 normal hearing subjects. A pilot study done to check the efficiency of the puretone masking paradigm, demonstrated a shift of 35 dB HL at 4 kHz with 7 and 9 dB HL shift in 3 and 6 kHz frequency respectively. For the actual study, two ABR recordings were taken for each subject. One for only clicks at 60 dB nHL and another for clicks at 60 dB nHL in presence of 4 kHz puretone at 70 dB SL to puretone threshold. The absolute latencies of I, III and peak and amplitude of I and V peak were measured and IPL and of I-III, III-V and I-V as well as the V/I amplitude ratio were derived.

On comparisons using paired t-test I-V and I-III IPL and V/I amplitude ratio and the absolute latency of V peak was found to be changed significantly. The means of the measures depicted an increase in the V and III peak, I-V and I-III IPL and V/I amplitude ratio. The absolute latency of I peak was found to be decreased. These findings can be explained based on the cochlear travel time concept, according to which the V peak is influenced by contributions initiated from basal to apical regions of the cochlea, whereas, wave I is highly dependent on basal cochlear function. And a travelling wave stimulates the cochlear base before the apex. Thus a 4 kHz dip being in between the area of maximal contribution of wave I and III or V reduces the early potential of wave III or V and later potentials of wave I. This reduce the absolute latency of wave I while increasing for wave III or V. Also an increased V/I amplitude ratio suggests reduced total potential for wave.

A test criteria was tried to develop based on the I-V IPL criteria to detect the presence of a 4 kHz dip in the audiogram. The sensitivity and specificity based on the data collected in the study comes to be 66.6% and 73.3% respectively for I-V IPL as the detecting criteria.

Thus the following conclusion may be drawn from the present study :

I-V IPL is not & true measure of the central conduction time. It is shown to be arfected by cochlear contributions.

Highlights the possible cochlear contributions to ABR waves believed to be generated from higher nuclei in the auditory pathway.

Measures like I-VIPL, latency of wave I and V/I amplitude ratio can be used to detect a 4 kHz dip.

Limitation of the Study

Because of the unavailability of subjects with exclusive 4 kHz dip in the limited period of 1 year puretone masking paradigm as suggested by Eggertnont et al. (1980) was used to simulate hearing loss at 4 kHz

Further Research Implications

The possibility of different cochlear sites for generation of different ABR waveforms can be investigated through a controlled and detailed study.

Effects of different audiometric configurations through simulated hearing loss can be studied and thus making it possible to predict the audiometric configuration using click evoked ABR.

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APPENDIX

Calibration of nHL

Normal hearing level (nHL) refers to normal threshold for click or brief tone stimuli. Zero dB nHL varies depending on test environment and stimuli used.

A group often normal hearing subjects (5 males,5 females) were taken. The behavioural threshold in SPL for clicks was estimated. The behavioral threshold estimation was done using the same instrument and in the same test environment as the actual ABR testing. Threshold was defined as the lowest level at which 50% of the responses were Observed. Their average behavioral threshold was taken as OdB nHL for that stimulus. The nHL value obtained value for test room was 30 dB SPL.