AUDITORY FATIGUE: CHANGES IN AUDITORY BRAINSTEM RESPONSE

Register No. M 9922

An Independent Project submitted as part fulfillment for the I year M.Sc. (Speech and Hearing) to University of Mysore

ALL INDIA INSTITUTE OF SPEECH AND HEARING, MYSORE-570 006
MAY 2000

Certificate

This is to certify that the independent project entitled "Auditory Fatigue: Changes in Auditory Brainstem Response"is a bonafide work done in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student with Register No. M 9922.

Mysore

May 2000

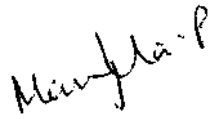
Director

All India Institute of Speech & Hearing

Mysore - 570 006

Certificate

This is to certify that the independent project entitled "Auditory Fatigue: Changes in Auditory Brainstem Response" has been prepared under my supervision and guidance.



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Declaration

I hereby declare that this independent project entitled "Auditory Fatigue: Changes in Auditory Brainstem Response" is the result of my own study under the guidance of Mrs. P. Manjula, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in any other University for any other Diploma or Degree.

Mysore *Register No. M 9922*

May 2000

DEDICATION

SRI VENKATESWARA

SWAMIVARIKI

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INTRODUCTION

Auditory fatigue can be broadly defined as a temporary loss of auditory sensitivity due to previous auditory stimulation (Ward, 1963).

Noise induced auditory fatigue is commonly measured as NITTS (Noise induced temporary threshold shift). It is a time linked process and grows with duration of exposure and disappears as a function of time since exposure. The time course for TTS recovery is dependent upon the type, duration and intensity of the acoustic exposure.

The mechanism underlying TTS, its location in the auditory system and the relationship between TTS and PTS (permanent threshold shift) are not fully known and are still subject to further studies (Failkowska et al., 1983).

The literature on physiological processes involved in TTS can be classified under three categories:

I. The evidences that links the TTS to the cochlea:

- The localized TTS process is correlated with gross pattern of movement of the basilar membrane (Bekesy, 1949).
- Noticeable loss of hair cells in the cochlea (Benitez et al., 1972).

- Depression of the cochlear potentials such as choclear microphonics (Benitez et al., 1972)
- Abnormal loudness growth or recruitment (Hickling, 1967).
- Widening of the tuning curves (Klein and Mills, 1981).
- Significant reduction in amplitude of the transient evoked otoacoustic emissions [TEOAE] (Kvarner et al., 1995).
- Subjective tinnitus (Smith and Loeb, 1970)
- Reduction in the auditory temporal summation (Mills et al., 1970).
- Temporary alteration in spontaneous otoacoustic emissions (SOAEs) (Norton et al., 1989, Furst et al., 1991)

II. The evidences that links the TTS to the central phenomenon:

- Greater loss of evoked responses from the central auditory system (gross and single electrode recordings) as compared with decrement of the click evoked action potentials. (Babighian et al., 1975; Salvi et al., 1975; Salvi, 1976)
- Large threshold shift at cochlear nucleus compared to behavioural TTS. (Saunders and Rhyne, 1970).
- Depression of spontaneous potentials at cochlear nucleus, superior olive and inferior colliculus following noise exposure (Starr and Livingston, 1963).
- Elevation of the threshold in the contralateral ear (Starr, 1965).
- Increased TTS when the subjects were engaged in the mental task during the time of exposure to fatigue sitmuli (Wernick and Tobias, 1963).

III. Failkowska, et al. (1983); Klein and Mills, (1981); Pratt et al. (1975, 1978) findings indicate that the auditory fatigue alters the synaptic mechanism between hair cells and the auditory nerve fibers and then transferred to the auditory nerves.

These desparate findings suggest that the physiological basis for the TTS appears to be complex. The present study was undertaken to investigate the effects of exposure to fatigue stimulus on the neural responses, using Auditory Brainstem Response (ABR) to arrive at indications regarding the site in the auditory system.

REVIEW OF LITERATURE

There have been a number of interesting investigations, both experimental and clinical, on the physiological processes involved in the noise induced auditory fatigue or noise induced temporary threshold shift (NITTS). These investigations are reviewed under two main categories, namely animal and human studies.

Animal studies:

Most of the studies were done either on chinchillas or cats. Saunders and Rhyne (1970) have measured the threshold shifts at the cochlear nucleus in cats using the Frequency Following Response (FFR) after inducing mild TTS. They have found the threshold shifts at cochlear nucleus to be relatively large compared to the behavioural TTS values obtained. This implies that exposures to noise were capable of reducing the sensitivity of units in the cochlear nucleus.

Benitez, Eldredge and Templer (1972) measured a subset of auditory potentials of chinchilla during the slow recovery from asymptotic threshold shift. They found that endocochlear potentials remained unchanged, while cochlear microphonics showed large loss in sensitivity. Action potentials were not recordable even at high signal levels. At the same time, an evoked response recorded from the scalp (response thought to be from the inferior calliculus) was

present at elevated thresholds which were similar to behavioural threshold shifts. They also noticed just detectable loss of hair cells anatomically. This suggested that the primary dysfunction for TTS was in the hair cells plus failure to synchronize primary neural responses.

Contrary to the above, Babighian et al. (1975); Salvi, Henderson and Hamemik (1975) evaluated evoked responses from the central nervous system (gross and single-electrode recordings) and compared the decrement of these potentials to click evoked action potential recordings. Considerably greater losses for central versus action potential recordings led each research team independently to hypothesize the existence of a central nervous system component in the auditory fatigue.

To account for the above, Salvi (1976) suggested that the conditions for central auditor}[1] involvement appeared to depend on the nature of the acoustic exposure. Asymptotic TTS may primarily affect the cochlea and low level, short duration (i.e., 95 dBSPL for 15 min.) exposures may primarily affect the retrocochlear structures.

Durrant (1976) has reported that the Summating Potential (SP), a peripheral or cochlear potential was also reduced more than the Action Potential (AP) or Cochlear Microphonics (CM), when the ear was exposed to pure tones at high levels. It was also reported that cochlear microphonics has been shown to

be less sensitive to electrical and mechanical modifications of the cochlea than the SP (Durrant and Gans, 1975).

Gans (1980) measured AP, SP and CM, in rats, in response either to clicks or pure tones prior to and following three minutes exposure to pure tones at the level 5 dB less than that which produced maximum CM. There was a greater voltage reduction for SP than for the AP and the CM showed essentially no decrement. This suggested that SP might be a better indicator of noise induced auditory fatigue. Based on the above observations, he stated that central contributions to auditory fatigue, if present, are considerably smaller than those suggested by the data of Babighian et al. (1975) and Salvi et al. (1975). Further, he stated that theories of central auditory fatigue may be based on incorrect interpretations of previously published data obtained from cochlear and neural recordings.

From the animal studies, it could be inferred that the involvement of central auditory structures depends on the nature of the acoustic exposure.

Human Studies:

Behavioural Methods:

The issue of central influences on auditory fatigue was raised by Wemick and Tobias, in 1963, who reported that mental activity in the form of mental arithmetic during a pure tone exposure resulted more auditory fatigue than the same during reverie. Similar findings have been reported in two other investigations (Capps and Collins, 1965; Collins and Capps, 1965), when original conditions were replicated.

When the conditions were changed slightly, however, manipulation of the level of mental activity ceased to result in differences in the amount of auditory fatigue (Bell and Stern, 1964; Collins and Capps, 1965; Riach and Sheposh, 1964; Ward and Sweet, 1963). Capps and Collins (1965) attributed that this failure was due to the type of mental task used.

Fricke (1966) exposed subjects to 100 dB, 110 dB or 120 dB white noise for 15 minutes after which they listened either for interruptions in noise or to a story to which they were supposed to attend. Results showed greater TTS only for noise plus story condition at 110 dB. However, differences were generally not significant as a function of the attention demanding story.

Price and Oatman (1967) replicated the study of Wemick and Tobias and produced similar data. They found that if subjects could resume post exposure

threshold tracking without being required to do something else at the same time, the differences between experimental groups disappeared. Thus central auditory fatigue seem to be an artifact.

Smith and Loeb (1968) conducted four experiments on normal hearing men concerning several activating or attention demanding conditions upon temporary threshold shifts (TTS). Two arithmetic tasks failed to produce significant differences in TTS. Consistently greater TTS was seen when subjects were exposed while tracking a 1 KHz tone than while exposed during reverie. D-amphetamine and secobarbital did not differ from a placebo in their effects on TTS. This suggested that the differences previously obtained under certain experimental conditions are not due to changes in the general level of arousal but to something more specific. Melnick (1968) found less TTS at 1400 Hz following a two minute exposure to a 1000 Hz tone at 110 dBSPL under the same tracking at 250 Hz tone in non-test ear conditions. The confusing results were due to the procedural differences in these studies.

Hickling (1967) studied hearing test pattern in noise induced temporary hearing loss and found a positive result for recruitment. The reason suggested was hair cell malfunction. Small changes in auditory adaptation, narrowing of the fixed frequency Bekesy continues trace may originate in some more central lesion responsible for a fraction of total loss.

Later, Grauer and Dunn, in 1978, performed two experiments to study per-stimulatory auditory central fatigue. They supported central fatigue in a per-stimulatory paradigm. Again, Dunn and Grauer (1981) studied attention factors in contralateral threshold shift and found that only when subject's attention was drawn towards the fatiguing tone did the thresholds rise, otherwise the thresholds dropped.

In the per-stimulation study, subjects must attend to stated auditory dimension of the primary stimulus during the presentation of the fatiguing stimulus; while in post-stimulatory paradigm there is no such attention demand, here fee subject is making an attempt to ignore the fatiguing stimulus. Thus, there is a potential difference between the two types of fatigue, implying that a mechanism which is capable of producing a contralateral fatigue effect is also affected by manipulations of attentional variables.

Narasimhan (1988) studied the influence of central factors on TTS. The subjects were exposed to fatigue stimuli under the states of reverie and solving an arithmetic problem. The results showed that there was no role of central factors in the amount of post exposure threshold shifts and concluded that TTS was a peripheral phenomenon.

The findings of behavioural studies on humans are equivocal in nature. Some researchers believe that there is a central component in fatigue, on the other hand, some researchers believe, it is purely a peripheral phenomenon.

Oto Acoustic Emission (OAE) Studies:

Norton et al. (1989) observed reduced amplitudes and frequencies of SOAEs (spontaneous otoacoustic emissions) following exposure to intense acoustic stimuli. Further, they suggested that following a brief noise exposure, changes occur in cochlear partition, especially in the outer hair cells, which causes both TTS and alteration in SOAEs.

Furst et al. (1992) measured the effects of noise exposure (100 dBSPL white noise for 10 minutes) on the threshold microstructure near an SOAE and on the amplitude and frequency of SOAE. Results indicate a temporary reduction in SOAE frequency and amplitude, and alters reversibly the threshold microstructure in the vicinity of the SOAE. They also found that the threshold at the SOAE frequency is most sensitive to noise exposure. This is modeled by reduction in the cochlear partition amplification.

Kvarner et al. (1995) recorded the amplitude of transient evoked otoacoustic emission (TEOAE) in 13 healthy employee's both before and after exposure to an industrial noise level of 85-90 dBA for 7 hours. A significant

reduction of the TEOAE amplitude was found, however, there was no correlation between TTS and TEOAE amplitude reduction.

From the above findings, it could be interpreted that the fatigue stimulus primarily affects the cochlear partition amplification.

Electrophysiological Studies:

Electrophysiological measurements from humans with TTS are limited in number. Mills, Gergel, Watson and Miller (1970) studied the temporary changes of the auditory system due to exposure to noise for one or two days. They reported that auditory evoked cortical response thresholds were similar to behaviour thresholds and amplitude-intensity functions were reported to be unchanged following 24 hour of noise exposure. They also found that the time constant of temporal integration was reduced at 750 Hz, recruitment, reduced amplitudes of Bekesy tracings. Frequency discrimination was unaffected. The above findings suggested that TTS is a cochlear phenomenon.

Sohmer and Pratt (1975) observed changes in latency and amplitude of components in the human auditory-evoked brain stem response (ABR) after exposing subjects to white noise (117 dBSPL for 15 min.) which produced 15 dB TTS. The first wave (whole nerve action potential) of ABR was most affected showing increased latency and decreased amplitude than later waves following

noise exposure. So this could be the evidence that the behavioural shift is more correlated with a peripheral (cochlear) electrophysiological effect.

PTatt, Sohmer and Barazani (1978) recorded cochlear microphonic potentials (CM) by means of surface electrodes before, during and after white noise exposure (100 dBSPL for 10-20 minutes), which induced TTS of about 10 dB. The results showed that the behavioural shift was not accompanied by a change in amplitude of CM.

The above findings indicated that the affected site is central to the site of generation of CM. From the previous study, it was proposed that the site affected is peripheral to the generation conducted action potentials. Thus the synapse between hair cells and the auditory nerve fibers is most likely to be the affected site.

Klein and Mills (1981) measured tuning curves and amplitude-intensity functions on wave I and wave V of the auditory evoked brainstem response and psychophysical tuning curves with noise induced temporary threshold shift of about 28 dB. In some subjects, amplitudes of wave I and wave V decreased following the noise exposure with wave I typically being more affected, while in other subjects, little or no change in amplitude occurred. All tuning curves had wider response areas following noise exposure, In a given subject, changes in

psychophysical and wave V tuning curves were similar and were less than changes measured for wave I.

Failkowska et al. (1983) observed a significant increase in latency and decrease in auditory compound action potentials (ACAP) in the ABR and Electrocochleography (ECOG) after exposing subjects to a octave band noise centered around 2.8 kHz at a sound level of 108 dBSPL. The duration of exposure was not mentioned The increase in wave III and wave V latency determined by the increase in the latency of wave I and ACAP. With this the unaltered interpeak latencies implied that auditory fatigue possibly alters the synaptic mechanism within the cochlea and was then transferred to the auditory nerves. This supported the earlier inference made by Pratt et al. (1978).

Electrophysiological studies on humans reveal that the auditory fatigue primarily changes the hair cells in cochlea and or synapse between hair cells and auditory nerve.

METHODOLOGY

Subjects : Twenty normal hearing adults, ten males and ten females, age ranging from 18 to 25 years served as subjects.

Selection criteria:

> Pure tone thresholds of less than 25 dB HL in frequency range of 250 Hz to 8000 Hz, at octave intervals. [As recommended by ANSI 1969/ISO 1978].

> 'A' type tympanogram and reflexes present on immittance screening.

> No significant history of noise exposure.

> Negative history of tinnitus and giddiness.

> Able to relax and sit without any extraneous movements throughout the entire test, i.e., for about 30 minutes.

Equipment:

The electrophysiological test unit used to record ABR waveforms was Nicolet Bravo, auditory evoked potential system, version 3.0. Headset with TDH-39 P earphones encased in MX-41/AR ear cushions were used to present the auditory stimuli.

> A calibrated two channel clinical audiometer, Madsen OB822, with TDH -39 P earphones encased in MX-41/AR ear cushions and audiocups was used to present the fatigue stimulus.

TEST ENVIRONMENT:

> The testing was carried out in an air-conditioned sound treated room with optimum lighting.

> The subjects were made to sit comfortably on a chair during the test session.

TEST PROCEDURE :

> The subjects were first screened for pure tone thresholds, Tympanogram and reflexes in both ears to confirm that the subjects meet the selection criteria.

> ABR recording was done before and two times immediately after the exposure to fatiguing stimulus.

> The subjects were instructed to 'sit comfortably and relax' on a chair facing away from the instrument. They were also instructed to avoid extraneous movements of head, neck and jaw for the duration of test

> Three silver chloride disc type electrodes were used for the recording of ABR. Continuity of the electrode wire was confirmed before placement.

- ABRs were recorded from disc-electrodes between the ipsilateral mastroid (Inverting) and forehead (Non-inverting).
- Before placing the electrodes, the skin surface was cleaned thoroughly by scrubbing with skin preparing liquid using cotton.
- Adequate amount of conducting gel was used before fixing the electrodes in their appropriate positions, the electrodes were secured in place by an adhesive plaster.
- Electrode impedance check was done to ensure that the impedance was less than 5 kiloohms and inter-electrode impedance was less than 2 kiloohms.
- Head set with earphones (Blue on left and red on right ear) was placed taking care not to dislodge the electrodes. Placement of earphones was such that the earphone diaphragm was in alignment with the ear canal, so that accurate stimulus intensity levels were delivered to the ear.

> The protocol used to record ABR waveforms are as follows.

a) Stimulus parameters

Type: Broad Band Clicks:

Broad band dicks was used to obtain a better waveform as click produces better synchronization.

Rate: 20.1/sec.

Repetition rate of 20.1/sec. was used to obtain ABR waveform with good marphoiogy at a faster recording time, which is an important factor in auditory fatigue.

Polariiy: Rarefaction

Rarefaction was *used* as *it enhances the amplitude of wave I.*

Number of Stimuli: 1600

1600 to optimize the time spent versus merphoiogy of the waveform.

intensity: 60 dBnHL

60 dBnHL to *ascertain an intensity high enough to maximize neural discharge. Also a level lower than this will lead to the absence of i peak.*

Transducer: Earphones

Earphones TDH-39 P with supra-aural ear cushions MX41/AR to deliver the sound through air-conduction.

b) Acquisition parameters

Number of channels: One

ABR was recorded using Fz-M, montage.

High frequency cut-off: 3000 Hz

As there is no appreciable ABR spectral energy above 3000 Hz.

Low frequency cut-off: 100 Hz

100 Hz as majority of the spectral energy of the early waves I and ill is above 100 Hz. And to avoid contamination by 60 Hz electrical and myogenic activities.

Analysis time: 10 msec.

- Ipsilateral ABR waveforms (Pre) were recorded for clicks presented monoaurallly at 60 dBn HL. The ear, i.e., right or left, was selected randomly. Recording was done only when rejection rate was less than 20% of the number stimuli.
- Then subjects were exposed to fatiguing stimulus of 3 kHz pure tone at 100 dBHL for 10 minutes in the ear in which pre-exposure recording of ABR was done. During this, the electrodes were left in their place.
- 3 kHz pure tone was selected as the fatigue stimulus because it has been reported to cause more damage than bands of noise (Ward, 1963).
- Immediately after the exposure two successive ABR waveforms (Post 1, Post 2) were recorded from the same ear. Each ABR recording took about one-and-a-half minute.
- Data on the following for each waveform of each subject was tabulated and statistically analysed.

    1. Absolute latencies for three major peaks; I, IE and V.
    2. Interpret latencies between I-III, IV-V & I-V.
    3. Absolute amplitude for peaks I & V.
    4. V/I amplitude ratio.

- ABR waveforms (Pre, Post 1, Post 2) of a subject exposed to fatigue stimulus is given in Figure 1.

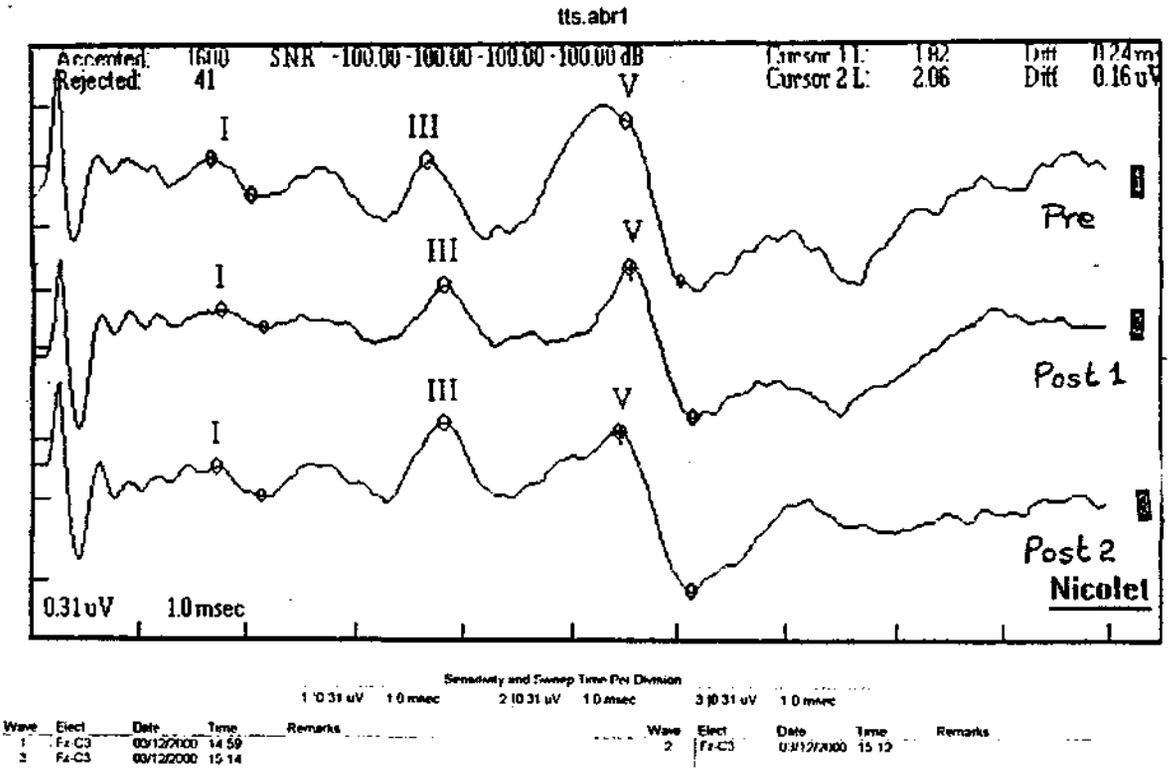

Fig 1: Pre, Post 1 & 2 ABR of a subject exposed to fatigue stimulus.

RESULTS

The ABR data (latencies and amplitudes) was obtained before and after exposure to fatigue stimulus. These were tabulated and statistically analysed.

Mean, standard deviation and range for the absolute latencies, interpeak latencies, absolute amplitude and amplitude ratio for the peaks in three different conditions **(Pre, Post 1, Post 2)** were tabulated, as in table 1, 2 and 3.

Table 1 : **Mean (M), standard deviation** (SD), and **range** (R) of the absolute latencies (msec) of I, III & V peak, in pre-, post 1, post 2-exposures.

		I			III		V		
	M	SD	R	**M**	SD	R	**M**	SD	**R**
Pre	1.85	0.11	1.6-1.98	3.84	0.18	3.52-4.16	5.7	0.15	5.42-6.0
Post 1	1.89	0.14	1.68-2.16	3.91	0.17	3.56-4.28	5.72	0.15	5.48-6.18
Post 2	1.87	0.15	1.64-2.16	3.85	0.15	3.6-4.1	5.67	0.18	5.36-6.04

Table 1 shows that the latency shift (immediately following the noise exposure i.e., **Post 1)** was smallest for the V peak (i.e., 0.02 msec) and greatest for III peak (i.e., 0.07). Post 2 values shows a recovery in terms of latencies.

Table 2 : Mean (M) standard deviation (SD), and range (R) of the interpeak latencies (msec) of I - III,III - V & I - V, in pre, post 1-, post 2- exposures.

I	I-III			III-V			I-V		
	M	SD	R	M	SD	R	M	SD	R
Pre	1.98	0.15	1.72-2.2	1.85	0.11	1.68-2.02	3.84	0.16	3.58-4.18
Post 1	2.0	0.17	1.72-2.28	1.80	0.11	1.64-2.02	3.81	0.15	3.5-4.14
Post 2	1.98	0.13	1.72-2.16	1.81	0.11	1.64-2.02	3.79	0.16	3.4-4.1

Table 2 reveals that the shift in the interpeak latencies for I-III, following noise exposure (Post 1), was prolonged by 0.02 msec and reduced for III - V & I - V by about 0.05 msec, 0.03 msec respectively. Interpeak latencies (I - HI, III - V) showed a recovery in post 2 condition

Table 3 : Mean *(tA),* standard deviation (SD), and range (R) of the absolute amplitudes (p-V) of I & V peak and V/I amplitude ratio in pre-, post 1-, post 2- exposures.

	I			V			V/I		
	M	SD	R	M	SD	R	M	SD	R
Pre	0.19	0.08	0.07-0.57	0.42	0.15	.25-0.83	2.62	1.37	0.7-5.3
Post l	0.12	0.06	0.06-0.23	0.40	0.19	.21-0.80	4.68	2.29	1.26-8.8
Post 2	0.17	0.09	0.07-0.58	0.41	0.14	.23-0.71	3.23	1.67	1-6.2

From table 3, it can be noticed that greater reduction in I peak amplitude was noticed following noise exposure compared to V peak reduction, i.e., the I peak amplitude reduced by about 0.07µV where as V peak amplitude reduced only by 0.02 µV. The amplitude ratio (V/I) increased following noise exposure i.e., from 1.37 µV to 2.29 µV. However, it is noteworthy that the I peak was absent in the two ears (out of 20 ears) following noise exposure compared to pre-exposure condition. The recovery in the amplitudes was noticed in the post 2-wave.

The standard deviation and range measures on the absolute latencies, interpeak latencies, absolute amplitudes and amplitude ratio reflect that they do vary in pre-, post 1 and post 2 conditions.

- To compare the latencies (I, HI, V), interpeak latencies (I-III, III-V, I-V), absolute amplitudes (I, V) and amplitude ratio (V/I) obtained for each wave, paired t-test of significance was done between.
    1) Pre ABR and post 1 ABR
    2) Post 1 ABR and post 2 ABR
    3) Pre ABR and post 2 ABR.

Table 4 : t-values, degrees of freedom, probability of I, IE, V peaks between pre - and Post 1, Post 1 and Post 2, Post 2 and pre - conditions.

	I	III	V
Pre - Post 1	t=1.00;df=37 P>0.05; NS	t = 1.27;df=39 P>0.05; NS	t = 0.38;df=39 P>0.05; NS
Post 1 - Post 2	t = 0.42;df=37 P>0.05; NS	t=1.20;df=39 P>0.05; NS	t = 0.91;df=39 P>0.05; NS
Pre-Post 2	t = 0.487; df= 39 P > 0.05; NS	t = 0.19;dM9 P>0.05; NS	t = 0.60;df=39 P > 0.05; NS

(NS : Not significant, df = degrees of freedom, P = Probability)

Table 5 : t- values, degrees of freedom and probability of I-III, I I I - V & I - V interpeaks between pre - and post post 1 and post 2, post 2 and pre - conditions.

	I-III	III-v	I-V
Pre - post 1	t = 0.39;df=37 P>0.05; NS	t=1.47;df=39 P>0.05; NS	t = 0.61;df=37 P>0.05; NS
Post 1 - post 2	t = 0.41;df=37 P>0.05; NS	t = 0.29;df=39 P>0.05; NS	t = 0.40;df=37 P>0.05; NS
Pre - post 2	t = 0.00;df=39 P > 0.05; NS	t=1.17;df=39 P > 0.05 NS	t=1.00;df=39 P>0.05; NS

(NS : Not significant, df: degrees of freedom, P = Probability.)

Table 6 : t-values degrees of freedom and probability of the absolute amplitudes of I & V peak and V/I amplitude ratio between pre- and post 1, post 1 and post 2, pot 2 and pre - conditions.

	I	V	V/I
Pre - Post 1	t = 3.04; df = 37 P< 0.001; VHS	t = 0.37;df=39 P>0.05; NS	t = 3.43;df=37 P<0.001; VHS
Post 1 - Post 2	t = 2.08; df = 37 P < 0.05; S	t = O.19;df=39 P > 0.05; NS	t = 2.26;df=37 P<0.05; S
Pre - Post 2	t = 0.76;df=39 P > 0.05; NS	t = 0.22;df=39 P>0.05; NS	t=1.27;df=39 P > 0.05; NS

(NS: Not significant S : Significant VHS : very high significant,
df = degree of freedom, P = Probability).

On comparing pre and post 1, post 1 and post 2, pre - and post 2, from tables 4, 5,6, it is evident that-

> The absolute latencies of I, III, V peaks are not significantly different in the three conditions.

> The interpeak latencies (I-III, III-V & I-V) also did not differ significantly in any of the conditions.

> The absolute amplitude of the I peak and amplitude ratio of V/I varied significantly at 0.001 level in between pre and post 1 and 0.05 level between post 1 and post 2 conditions i.e., the amplitudes were significantly more in pre and post 2 waveforms.

DISCUSSION

The results of the study on the effect of temporary threshold shift on ABR are discussed under two headings: latency measures and amplitude measures. The amplitude measures were more affected compared to absolute and interpeak latencies.

Latency measures:

In general, these was no significant difference in absolute and interpeak latencies following noise exposure. A small increase in HI & V peak latencies determined by the increase in the latency of I peak, and unaltered intervals between the successive peaks implies that the noise induced auditory fatigue primarily alters auditory system peripheral to the I peak generation.

The results were in consonance with the findings reported by Failkowska et al. (1983) and Sohmer and Pratt (1975). However, the latency shift in the present study was smaller when compared to the latency shift observed by Failkowska et al. This could be due to the methodological differences. They used octaveband noise (with centre frequency 2.8 kHz) at 108 dBSPL as the fatigue stimulus, while the present study used a 3kHz pure tone at 100 dBHL. Further, the duration of the exposure was not specified by authors, while in the present study, the duration of exposure was 10 minutes.

It has been reported that the small increase in latency at high stimulus levels may be due to the large contribution to electric responses from the well synchronised, high frequency units in the basal turn of cochlea and to the slight effect of the TTS which extended beyond this region (Failkowska et al., 1983). Also, as clicks stimulate a wider portion of the basilar membrane (i.e., 1-8 kHz region), the localized cochlear pathology has most effect at low intensities when the spread of excitation is lessened. (Keith and Greville, 1987). This could be the reason for the small shift in latencies observed in this study at 60 dBnHL. However, recording of the ABR waveforms could not be done at lower levels than this due to the absence of I peak.

Amplitude measures :

- Reduction in wave I amplitude (0.07)*V) usually exceeded reductions in wave V amplitude (0.02/^{t}V) following the noise exposure. This was reflected as a greater amplitude ratio (V/I) in post 1 condition. This finding *is* similar to observations by Sohmer and Pratt (1975); Klein and Mills (1981), on the ABR recorded from humans after they were exposed to noise. It is also consistent with the findings of Benitez et al. (1972), where action potential from chinchillas exposed to noise was more effected than scalp recorded evoked responses from the inferior colliculus. They further opined that the disruption of the action potential was due to the desynchronization of the contributing neural elements by the temporary noise trauma.

Also, it has been reported that the amplitude reduction in evoked potentials is produced by synaptic inefficiency, maturational delay, fewer neurons generating or fewer fibers conducting the volley, and neural asynchrony or desynchronization of the volley (Jiang, 1998). Therefore the reduction in amplitude of wave I observed in this study may be due to desynchronization secondary to either synaptic inefficiency between hair cells and auditory nerve or due to the cochlear impairment in the basal region due to the fatigue stimulus. (Davis and Hirsh, 1979).

Evidence reported in the literature indicate that wave I of the auditory brainstem responses is influenced by cochlear contributions from a more basal area than is wave V. (Keith and Greville, 1987). Hence, the components of wave I is either absent or of smaller amplitude because of pathology induced by the fatigue stimulus in the very high frequency area of the basilar membrane (i.e., above 4 kHz).

The findings in the present study, i.e., reduction in wave I amplitude could be a reflection of type of fatigue stimulus used. Since the fatigue stimulus was 3 kHz pure tone at 100 dBHL for 10 min, its effects are mainly on the 4-6 kHz region of the basilar membrane. Thus there is a reduction in the wave I amplitude. If, however, a broad band noise was used as a fatigue stimulus, it would have provided more information regarding the involvement of peripheral or central components. On other hand, wave V less dependent on

on basal region, is less affected, except at low intensity levels (Keith and Greville, 1987). In the present study the stimulus level was 60 dBnHL, a moderate level, hence much changes could not seen in the V peak amplitude as compared to I peak amplitude following the noise exposure.

To confirm this, measurment of (t)to Acoustic Emission (OAE) / Cochlear Micro-phonics (CM) would be of great help.

SUMMARY AND CONCLUSIONS

Auditory fatigue can be broadly defined as a temporary loss of auditory sensitivity due to previous auditory stimulation (Ward, 1963). It is commonly measured as noise induced temporary threshold shift (NUTS).

The results of the studies investigating the physiological basis for the TTS appears to be equivocal, i.e., some authors believe that TTS primarily affects the cochlea while others opine, there is an involvement of the central component also in auditory fatigue. Hence, the present study was undertaken to investigate the effects of fatigue stimulus on the neural responses, using ABR, to arrive at indications regarding the site in the auditory system.

To accomplish the mentioned aim, 20 subjects (10 males and 10 females) were exposed to fatigue stimulus of 3 kHz pure tone at 100 dBHL for 10 minutes. Three ABR waveforms (pre, post 1 & post 2) were recorded. Amplitude and latency measurements, for each waveform, of each subject, were tabulated and statistically analyzed. Results indicated that there was a significant reduction in wave I amplitude (at 0.001 level) and significant increase in V/I amplitude ratio (at 0.05 level) following the noise exposure (i.e., post 1). However, there was no significant change in absolute and interpeak latencies. The observed findings could be due to the pathology induced by fatigue stimulus in very high frequency area of the cochlea (i.e.,

above 4 kHz). From the present study, it could be inferred that the effect of fatigue stimulus is on the cochlea.

RECOMMENDATIONS :

1) As broad band noise stimulates the entire basilar membrane, its effect as fatigue stimulus could be studied.
2) Latency-Intensity functions could be studied for better understanding of the physiological process of temporary threshold shift.
3) Combination of oto acoustic emissions and auditory brainstem response results provide more insight on the physiological process of TTS.

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