TEMPORARY THRESHOLD SHIFT AND EFFERENT ACTION

Reg. No. 8407

Prabhakar Kumar Sinha

A Dissertation submitted in part fulfilment for the degree of Master of Science (Speech & Hearing) of the University of Mysore

ALL INDIA INSTITUTE OF SPEECH & HEARING
MYSORE-570006

1985

Dedicated

to

my beloved PARENTS

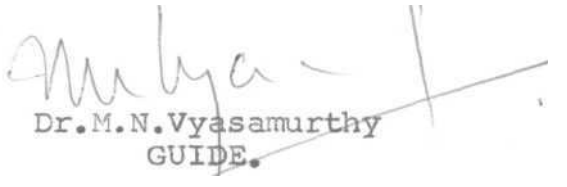
CERTIFICATE

This is to certify that the Dissertation entitled 'TEMPORARY THRESHOLD SHIFT AND EFFERENT ACTION' is the bonafide work on part fulfilment for the Degree of Master of Science (Speech and Hearing) of the student with Register No.

(Dr. Nitya Seelan)
Director
All India Institute
of Speech & Hearing
Mysore - 570 006.

CERTIFICATE

This is to certify that this Dissertation entitled "Temporary Threshold Shift and Efferent Action" has been prepared under my supervision and guidance.



Dr.M.N.Vyasamurthy
GUIDE.

DECLARATION

I hereby declare that this dissertation entitled 'TEMPORARY THRESHOLD SHIFT AND EFFERENT ACTION' is the result of my own study under the guidance of Dr. M.N.VYASAMURTHY, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any University for any other diploma or degree.

Mysore

Date: March, 1985 Reg. No. 8407

ACKNOWLEDGEMENT

I, with a deep sense of gratitude, acknowledge the kind help received from Dr. M.N.VYASAMURTHY. I owe to him all that I know in 'Audiology'. I remain ever grateful to him and I am indebted to him for his masterly and constructive guidance throughout the work. I strongly believe that without his wisdom and assistance this work would not have been completed.

My thanks are due to Dr.Nitya Seelan, Director, AIISH for having permitted me to carry out the study. I am equally thankful to Dr.S.Nikam, for giving me permission to use the instruments for my study.

My special thanks go to Mr.J.Dayalan, Lecturer in Audiology whose advices were of great relevance throughout the study.

I am thankful to Dr.T.R.Rama Prasad whose suggestions were of special use to me.

My innumerable thanks are due to Ms.Yasmeen who helped me in the statistical analysis of the data. I am thankful to Ms.Gayathri and Ms.Nazneen who served as a source of inspiration for me during the study.

I am thankful to Mr.Rajinder Kaul, Mr.Biswas, Mr.Sanjay, Mr.Ashok and my other friends who extended their cooperation during the study.

My sincere thanks go to all those individuals who participated in the experimental study with great enthusiasm and patience.

I am thankful to Ms.Rajalakshmi Gopal for her excellent and blemishless typing.

LIST OF TABLES

LIST OF FIGURES

CONTENTS

INTRODUCTION

The term, 'Auditory fatigue' or 'temporary threshold shift' (TTS) is commonly used to the transitory change in hearing sensitivity induced by a fatiguing stimulus. It is well established that when the ear is exposed to continuous intense auditory stimulation, temporary dysfunction of hair cells would result. Histological studies have established temporary changes in the structure of the hair cells when the ear is exposed to intense auditory stimulus for longer periods.

Of recent, Pratt et al (1978) have found that the TTS of about 10-20 dB, following noise exposure, in human subjects is probably due to effect on the synapse between the hair cells and nerve fibres i.e. they have attributed the TTS of 10 to 20 dB to the "reduced synaptic efficacy". They have arrived at the above conclusion as 10-20 dB TTS was not accompanied by the reduced amplitude of cochlear microphonic (CM). Since there was no reduction in the cochlear microphonic, they have concluded that the function of the hair cells was not affected.

Thus Pratt et al (1978) have shown that if the TTS in a subject is about 20 dB, 'reduced synaptic efficacy' may be responsible for the TTS and that dysfunction of the hair cells may be ruled out.

In view of the above facts, it may be assumed that the temporary dysfunction of hair cells results whenever TTS of greater than 20 dB is observed and that the TTS of 20 dB or less may be due to the reduced synaptic efficacy.

Regarding the action of the efferent system innervating the outer hair cells, Vyasamurthy (1982, 1985) reports that the action of the efferent system innervating the outer hair cells is to increase the loudness of the adapting stimulus when the adapting stimulus is presented continuously, i.e., he suggests that the efferent system innervating the outer hair cells is responsible for 'loudness gain' during auditory adaptation. He cites the following studies in support of it: Spoendlin 1975, Cody and Johnstone 1982, Gerkin 1984, Fex et al 1982, Comis and Whitfield 1968, Hoffamann et al 1983, Pickles 1982 and Stopp et al 1983.

Cody and Johnstone (1982) have established that the action of the efferent system innervating the outer hair cells increases the sensitivity of the auditory neuron. The increase in the AP (action potential) response during bilateral stimulation (i.e. in presence of efferent action) may be explained in terms of recycling of the released neuro—transmitter. Fex et al (1982) have reported that the action

of the efferent system innervating the outer hair cells is recycling of the released neurotransmitter.

In the light of the evidences (Pratt et al, 1982; Vyasamurthy, 1982, 1985; Cody and Johnstone, 1982; Fex et al, 1982), it is likely that the action of the efferent system innervating the outer hair cells had increased the synaptic efficacy through 'recycling' of the released neuro-transmitter in the study reported by Cody and Johnstone (1982). The increase in the AP response during bilateral stimulation (condition 2 in Cody and Johnstone's study) may be due to the improvement in the synaptic efficacy brought about by the action of the efferent system innervating the outer hair cells through 'recycling' of the released neuro-transmitter.

Need for the present study:-

The present study has been undertaken to verify whether the action of the efferent system innervating the outer hair cells increases the synaptic efficacy in human subjects. The study reported by Cody and Johnstone (1982) was (in guinea pigs.

Furthermore, the present study was designed to see whether the efferent action has any role in changing the temporary dysfunction of the hair cells.

The following null hypotheses have been proposed:

i. There is no significant difference in the TTSo (TTS_2) obtained in the experiments I and II.

ii. There is no significant difference in the TTSo (TTS_2) obtained in the experiments III and IV.

iii. There is no significant difference in the TTSo (TTS_2) obtained in the experiments V and VI.

Definitions of the Terms Used:

Temporary Threshold Shift (TTS): Refers to any post stimulatory shift in threshold.

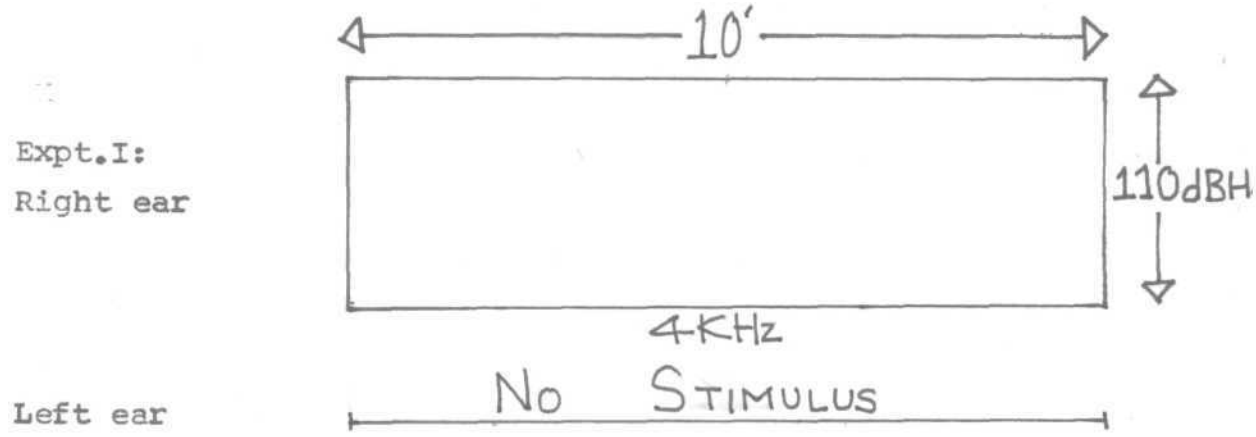
Fatiguing Stimulus: The acoustic stimulus used to produce auditory shift in threshold.

TTSo: Temporary threshold shift immediately after the termination of the fatiguing, stimulus.

TTS_2: Temporary threshold shift after the recovery time of 2 minutes.

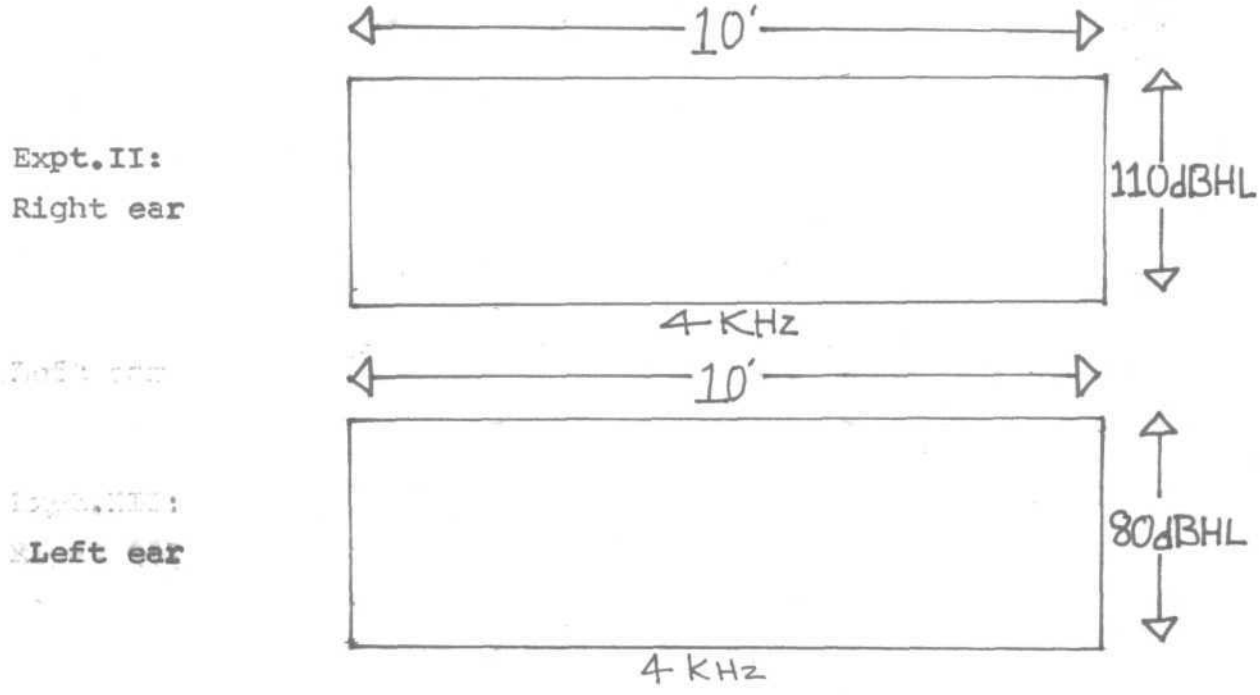
ESIOHCs: Efferent system innervating the outer hair cells.

Schematic Diagrams of the Experiments I, II, III, IV, V and VI are given below. See the methodology for details: 05



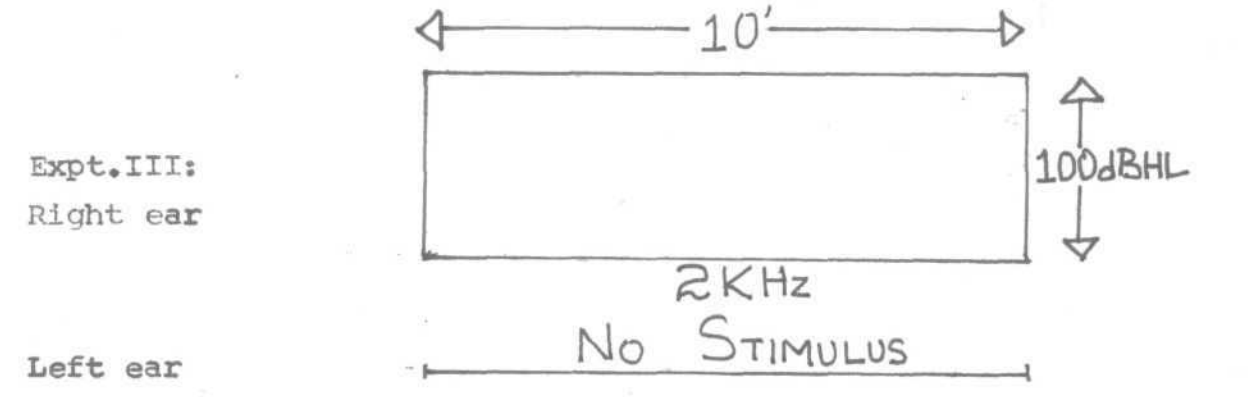
Expt.I:
Right ear

Left ear



Expt.II:
Right ear

Left ear

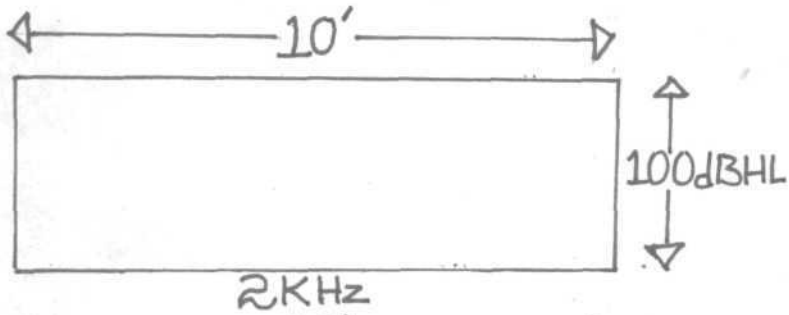


Expt.III:
Right ear

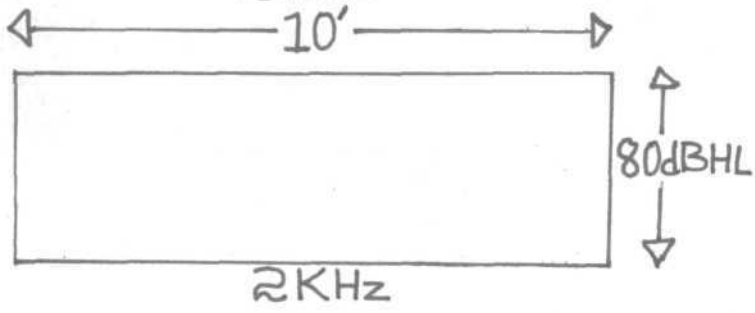
Left ear

Expt.IV:

Right ear

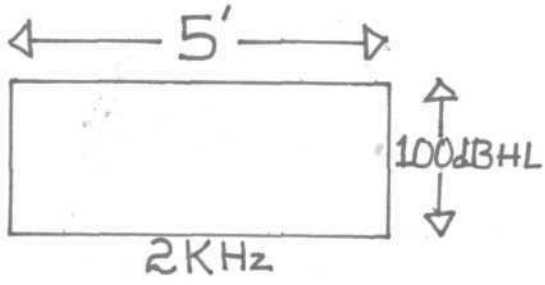


Left ear



Expt.V:

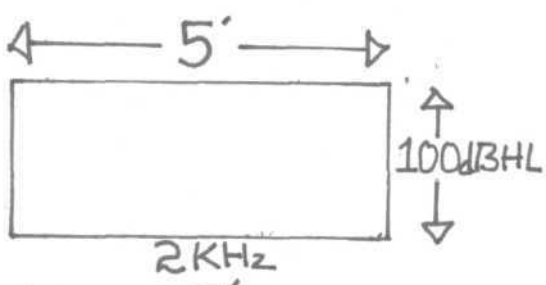
Right ear



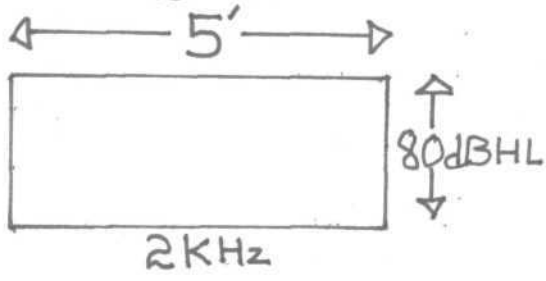
Left ear

Expt.VI:

Right ear



Left ear



REVIEW OF LITERATURE

The review of literature deals with the following aspects:

I Binaural and Monaural stimulation in TTS

II Effects of noise on Cochlear Potentials

III Mechanisms Involved in post stimulatory reductions of Cochlear Potentials

IV Patterns of hair cells damage

V Damage of supporting cells due to acoustic stimulation in the cochlea.

VI Revised Model of adaptation and loudness gain

VII An active mechanism

VIII Models of the efferent mechanisms

IX Cochlear microphonic and TTS in man.

I. Binaural and Mon-aural Stimulation in TTS:

Hirsch (1958) studied TTS following mon-aural and binaural exposures under 3 experimental conditions to ascertain whether or not TTS depends upon whether one ear or both ears were exposed to sound. The results showed that "The TTS for 1 KHz tone is the same whether the ear was tested alone, or both ears simultaneously".

A similar study was done by Ward (1965), he in his study compared the TTS following mon-aural and binaural exposures to three different high intensity stimuli. The maximum effect occured at 2 KHz where the binaural exposure gave less TTS as compared to mon-aural exposure. Ward explained this reduction in TTS in terms of feedback loop and he further reports that "with the increased input when the second ear is stimulated, the total activity of the reflex centre also increases in middle ear muscle activity".

Melnick (1967) that more TTS occured when the exposure signal was 180^{o} out of phase, in his experiment in the effect of two inter aural phase conditions for binaural exposures on threshold shift.

Guiot (1969) showed that stimulation of the left ear had a definite influence upon the TTS measured on the right ear.

Further he says that if any summation effects were to occur, a reduction of sensitivity should have resulted rather than an increase as was actually recorded. A reasonable interpretation of this outcome can be formulated if one admits that a central inhibitory process, in conjuction with fatigue to be intervened in the production of TTS. Inhibition, considered as associating with fatigue to form a response system, should be expected to be affected by same external stimulation, that is to be inhibited when operant. The phenomenon of disinhibition can be revealed by a reduction in TTS. In the same perspective, the disinhibition effects of certain nonauditory stimuli as reported by Rawden Smith (1936) could be cited.

Thus, TTS can be shown to demonstrate peripheral and neural effects. Randolph and Gardner (1973) in their study of an interaural phase effect in Binaural TTS, showed that if particular neural units in an afferent pathway are constantly stimulated and ultimately fatigued, the post exposure threshold resulting from restimulation of the same neural units would be shifted. Further, Randolph and Garden (1973) points out that sequentially occuring tonal exposure to test stimuli of like interaural phase might then be expected to produce more TTS than would sequentially occuring stimuli of opposite interaural phase. Since the peripheral

exposure and test events at the individual peripheral receptors may be considered identical, such differences, of course, could only be attributed to adaptation or alteration of neural responsiveness and to peripheral factors.

Dichotic exposure to certain acoustic stimuli at high intensity levels results in reduced post exposure TTS relative to monotic exposure to the same stimuli (Ward, 1965; Melnick, 1967; Karlorich, Lutermann and Abbs, 1972).

Karlovich and Wiley (1974) assumed that the reductions in TTS observed resulted from involvement of the acoustic reflex. The increased effectiveness of the reflex activating stimuli having more rapid repetition rates was not completely clear. But they speculated however that some type of adaptation or 'reflex decay' may be involved in which the acoustic reflex response to continuous or slowly pulsed stimuli diminishes over time more so than the response to stimuli with faster repetition rates.

Shivshankar (1976) has reported that there is no significant difference in TTS between mon-aural and binaural exposure to high frequency tones, especially for 3 KHz at TTS_3. He has concluded that the action of homolateral Olivo -

cochlear bundle which might inhibit the responses of the higher centres, as crossed Olivo cochlear bundle does not play a role in the adaptation mechanism at high frequency Dayal (1972).

Nazneen (1984) has reported that TTS observed in the right ear is significantly greater than the TTS observed in the left ear. So she suggests that the ear difference does exist in TTS for binaural stimulation using 2 KHz tone. Further she reports that the right ear shows more TTS more than the left ear - is an indication that the action of the efferent auditory system during binaural stimulation is more intense in the right ear than in the left ear.

II. Effects of noise on cochlear potentials:

a. Changes in Summating Potentials (SP)

Durrant (1976) has given a detailed report about the change of summating potential due to noise exposure. He has reported that SP DIF (recorded by differential electrodes) is much more sensitive to effect of noise than CM and AP, which suggests that marked changes can be produced in the SP even in cases where the CM is hardly affected. He reports that there is a trend toward less depression in the SP from the lower to the upper turn. In the first turn, SP is usually more depressed than CM. In the third turn, the opposite is nearly always observed, at least in the initial phase of recovery. The second-turn data are immediate. Only the negative SP was investigated using tone-burst stimuli presented at levels within the linear range of the CM input-output function.

Legouix and Pierson (1981) did a study on guinea pigs and found that there is a relation between the voltage of the negative SP for a given stimulus and the susceptibility to fatigue i.e. individualistic variation in amplitude of SP. They report that these variations are not known whether this is due to some cochlear pathology or normal ear features - the cochlear which show the largest negative SP are more susceptible to fatigue.

"When SP and CM are depressed by noise, it is no longer possible to create additional fatigue, at least with the same stimulus as was used for the initial exposure. This stage seems to correspond to the asymptotic threshold shift. When the negative SP is depressed or has disappeared after exposure, fatiguability is minimal" (Legouix and Pierson, 1981).

b. Variation in Endocochlear Potential (EP) :

"It is well known that in normal conditions, EP recorded by a microelectrode penetrating into scala media can be decreased during the presentation of high-intensity tones or noises. This fact was first demonstrated by Von Bekesy in 1950 (Von Bekesy, 1960) and was studied by several authors (Tasaki et al, 1954; Misrahy et al, 1958b; Honrubia and Ward, 1966; Legouix and Pierson, 1977). The change in the dc level occurs immediately upon sound onset and increases gradually during the presentation. At the end of sound, EPreturns progressively to its previous level. In contrast, Misrahy et al (1958b) reported that vhen the dc shift is recorded from the perilymph its time course is the same as that of the stimulus. In their own experiments (Legouix and Pierson, 1977), they found that recording the dc shift from the perilymph with differential electrodes led

to variable results. In general, a shift analogous to the summating potential was recorded. If the duration of the tone was increased, the dc shift was maintained but a larger shift was often superimposed, which could be related to a change in EP transmitted to the differential electrodes. The probable reason these large shifts in dc potential are not always observed is that the resistance of the cochlear partition may differ in the various animals. As a result of prolonged exposures, so far no permanent change in EP could be demonstrated (Benitez et al, 1972). However, Johnstone and Sellick (1972) reported that EP could be potentiated following exposure to an intense pure tone" (Legouix and Pierson, 1981).

c. Changes in compound Action Potential (CAP):

Legouix and Pierson (1974) observed that CAP recording from the apical regions is not convenient, So it is recorded with a pair of differential electrodes placed in the basal turn of the cochlea or with one electrode in contact with the round window and other connected to the body of animals.

Davis et al (1953) suggest that CAP is too labile to provide a good index of cochlear changes. Handerson and MØller (1975) states that the measurement of AP over-estimates the cochlear damage.

Smoorenburg and Van Hensden (1979) studied the CAP latency due to noise exposure at threshold level and suprathreshold level before and after exposure. They found that there was no change at threshold level but there was reduction in the latency at suprathreshold level. Further they state that this wave form modification may be related to inactivation of a particular group of fibres or to the desynchronization of unit potentials.

"A convenient way of measuring the changes of response amplitude is drawn input - output curves. These curves classically show two branches. The first, with a low slape, is often considered to represent the responses of a particular group of more sensitive fibres. The second branch, with a steeper slope, would represent the responses of another group of less sensitive fibres. However, other interpretations of this nonmonotonic function, involving a single group of fibres, have been presented (Davis, 1973? Evans, 1975; Legouix et al, 1978). Generally, after noise exposure, the affect the most sensitive responses and leave the others intact. It follows that the intensity function is modified in such a way that N, magnitude grows more rapidly with intensity above a certain level. This type of curve has been compared to a recruitment phenomenon (Portmann et al, 1973)". (Legouix and Pierson, 1981).

Zwicker and Schorn (1978): (cited by Legouix and Pierson, 1981), measured psychophysical tuning curve in noise - induced hearing loss cases they found tuning is decreased especially in the high frequency range. However, when the loss exceeds 60 dB, the measurements are obscured by the occurance of difference tones, so that the curve cannot be considered as representing the tuning.

d. Changes in Cochlear Microphonics (CM):

i) Different types of CM Reduction:-

Legouix and Pierson (1981) write that the depression of cochlear microphonics after noise exposure is a clear sign of a traumatic effect on the sensory cells and that the deterioration of these cells may result from various mechanisms that follow different time courses and some are reversible and others are not. Further they write that some research has made use of a single electrode in contact with the round - window membrane, and the results give information about the basal turn only. Differential electrodes provide the possibility of recording the CM at various locations along the basilar membrane. Tasaki and Fernandez (1952) (cited by Legouix and Pierson, 1981) report that CM is attenuated about 6 dB per millimeter

around the electrodes (in the guinea pig). Further, Tasaki and Fernandez (1952) point out that when this small portion of the organ of corti has been damaged, the same electrodes can record responses from adjacent areas 2 mm away with an attenuation of 6-12 dB - it could record from more distant areas with greater attenuations.

Legouix and Pierson (1981) suggest that it is probable that these exist several sorts of CM reduction (depressions), which can be related to different kinds of alterations - mechanical or biochemical for instance and some others related to the destruction of the cell. Further, they report: "CM could not display any change until it disappeared after injury to hair cells, in contrast with AP, which is sensitive to fatigue and shows complete recovery and according to recent studies CM shows reversible changes that correspond with the behavioral TTS."

Benitez et al (1972) nave measured CM in the three cochlear turns of chinchilla after fatiguing by 500 Hz tone. They found that the third turn was more affected rather than the other turns.

ii) Modifications of CM Intensity Function Produced by Intense Noise Exposures:-

Legouix and Pierson (1981) suggest that to evaluate the effects of intense noises on the cochlea, a complete study of space - time and intensity patterns should be carried out - so a convenient way of exploring the changes in the CM responses is to trace the input - output functions (e.g., CM voltage against sound - pressure level). This function displays quantitative differences according to the place in the cochlea where CM is recorded. Generally, that function is approximately linear at low sound pressures and bends at higher levels. Further they explain that at each location along the cochlea, the bending occurs at a lower level for frequencies above the characteristic frequency of the recorded locus and that the origin of above nonlinearity is still a matter of debate.

They write further that after exposure the input-output function is altered. Tondorf and Brogan (1952) reported a greater reduction of the response at high levels so that the curve was truncated. In some particular cases, Lawrence (1958) observed a nonmonotonic input-output function following exposure. In plotting the characteristic of transfer, they noted that the curve was rapidly modified and displayed a greater summetry (Legouix and Pierson, 1978; Legouix et al, 1980).

Legouix and Pierson (1981) suggest that the changes in the input-output function may lead to some interpretation about the nature of the lesion in the cochlea. This function shifted toward a lower sensitivity, could reflect some impairment in the conduction of the acoustic energy within the cochlea and the other changes, on the contrary, point toward some disorder in the transducing process in the hair cells.

iii. CM as an Index for the Localization of Cochlear Damage:

Legouix and Pierson (1981) write that there are some investigators who have shown a clear correlation between the decrease in magnitude of CM response and the number of hair cells that are damaged (e.g. Eldredge et al., 1973). They further suggest that when the stimulus is a pure tone, the maximum reduction occurs in the region corresponding to the localization of the maximum of vibratory displacement along the cochlear partition, however, the interpretations of the results is not straight forward because the sensitivity at the various turns is not similar.

Benitz et al (1972) measured electrophysiological correlates of temporary threshold shifts in chinchilla. They recorded CM from the first, second and third turns.

They used as a test tone a 200 Hz tone burst, in the control record, such a stimulus produced input-output functions that are approximately equivalent, so a comparison could be made between the alteration of curves representing these input-output functions. They exposed to an octave band of noise centered at 500 Hz and at 95 dB SPL for 48 hours produced a decrease of the intensity function that was maximum for the third turn, the reduction in CM amplitude was graded and less severe from apex to base.

Legouix and Pierson (1981) report that narrow bands of noise (1/3 of an octave) produce effects similar to those of a pure tone at the same centre frequency.

Eldredge and Covell (1958) found that narrow-band noises were more effective in producing CM reduction than pure tones presented at an SPL equal to the over-all SPL of the noise.

Legouix and Pierson (1981) suggest that when two pure tones are applied simultaneously two different situations may occur depending upon the frequency separation of the two tones. If the two frequencies are sufficiently separated to activate different regions of the cochlea,

so that there is no interaction, the result is expected to be equal to the sum of two single exposures to the two different tones. Pye (1974) did a study on guinea pig cochlea for anatomical alterations exposed to two widely separated but nonsimultaneous frequencies (2 and 20 KHz). There was time interval left between first and second exposures so that destruction of the sensory resulting from the first exposure would be almost complete before the second exposure. He found the damaged cells on the basilar membrane were situated in the two separated regions, and no particular effect could be attributed to the double exposure as compared to a single exposure.

Poche et al, (1969) found (in some histological results concerning the effects of impulsive noises on guinea pigs) that the most sensitive region in the cochlea is the second turn, as would be expected in the case of a hearing gap around 4 KHz. Legouix and Pierson (1981) mention that there are several explanations for the acute susceptibility in the 4 KHz region - one of them is that ear canal, especially in humans, has a resonance for this frequency - these differ among the various species according to the anatomy of the external ear.

iv. Effect of the_time characteristics_of_the_exposure:

Legouix and Pierson (1981) write that the CM depression

produced by intense noises seems to be directly related to the sound-pressure level and to the logarithm of the duration of the exposure. Eldredge et al (1959, 1961) have suggested that the reduction in the CM was the same so long as the sound contains the same energy.

Legouix and Pierson (1981) reveal that it is clear that the trade between intensity and duration can be justified only if the same mechanism alters the hair cells at all intensities, but this does not appear to be the case. The cochlear mechanisms are highly nonlinear in the intensity domain, and different processes might occur when the stimulation level is modified. Further they say that a critical intensity must be exceeded to produce a loss at any duration and at very short durations, the influence of intensity seems to be greater than that of duration. After a certain duration, they state that the CM reduction induced by the noise is maximum and does not grow with time and that this effect seems well correlated with behavioral TTS.

e. Variations in Auditory Neural Responses:

Salvi et al (1979) did a study on chinchillas. They exposed an octave band of noise centered at 4 KHz and having an SPL of 86 dB during 5 days and measured the changes in the threshold of auditory nerve fibres. They compared the

auditory nerve fibre threshold to behavioral threshold after a recovery period of approximately 6 months, then they found that the fibre thresholds were elevated upto 70 dB for units with a CF between 4 and 14 KHz. These were smaller shifts seen in behavioral threshold and hence the results showed that damage occured in a cochlear region tuned about 1 octave above the centre frequency of the noise exposure.

Smoorenburg and Van Hendsen (1979) did experiments on cats and recorded single - unit tuning curves in the anteroventral cochlear nucleus (AVCN). They suggested that it was possible in this structure to hold a unit for a period of time sufficient to make the measurements, which is not possible in the cochlear nerve unit. They found that the characteristics of response are known to be similar in the AVCN and in the cochlear nerve, so that comparisons are justified.

Smoorenberg and Van Hendsen (1979) reported another feature of single unit responses in their locking to the stimulus. They found in AVON that the phase locking was preserved but the response time was modified. As an increase of 0.2 msec. was observed for a unit with a CF

at 1.5 KHz, and of 0.5 msec. for a unit with CF at 3.0 KHz. But the increase in cochlear time did not agree with the decrease in tuning of system which, on theoretical grounds should be accompanied by a shorter response time. Similarly, Harrison and Evans (1979) observed ia Kenamycin treated guinea pigs that the phase - locking ability of fibres from damaged cochlea was not modified.

II. Mechanisms involved in post stimulatory reductions of cochlear potentials

a. Role of nonlinearity of CM:-

Legouix and Pierson (1981) write that there is general agreement that cochlear fatigue and trauma are likely to occur when the ear is overloaded, and more specifically, when the intensity is such that the CM amplitude does not grow linearity with intensity, the bending of the input-output curve indicates the threshold of overload. Near this level, distortion occurs and manifests itself by the presence of overtones (harmonics). They further suggest that the level at which the intensity function departs from linearity corresponds to the possibility of overload of the cochlea.

Lawrence and Yatis, 1956a, b; Opheim and Flottorp, 1955; Humes, 1978; studied the determination of a threshold of overload in human subjects in order to predict the fragility of the hearing organ. Lawrence and Blandard (1954) propose that a lower threshold of aural overload tended to show greater damage in the organ of corti after noise exposure. Drescher and Eldredge (1974); in a study of cochlear fatigue in guinea pig and chinchilla, found that any intensity level variation above the point at which the CM input-outout function departs from linearity represents a stress to the cochlea that results in a loss of CM sensitivity.

Lawrence and Yantis (1956a, b, 1957) report that in the cochlear pathology subjects, the overload threshold was higher than for normal subjects. Wever and Lawrence (1955) explained the above fact by the "injured hair cells hypothesis" which assumes that the hair cells in the area of maximal stimulation are no longer responding, and therefore intensity must be raised so as to stimulate hair cells in adjacent regions whose output then reaches the threshold of non-linearity.

Legouix and Pierson (1981) mention that the origin of the non linearity is still a matter of debate. Further they state that the vibrations in the middle ear are linear up to the highest intensities. They suggest that there may be several processes in the cochlea can introduce a non-linearity in the responses.

"The mechanical vibrations at the basilar membrane were reported to be linear by some authors. (Von Bekesy, 1960; Johnstone and Boyle, 1967; Wilson and Johnstone, 1972), however, other results suggested an important non-linearity (Rhode 1971). Some data indicate that the processes of transduction in the hair cells can introduce a non-linearity in the electrical responses of the cochlea (Legouix and Chocholle, 1957; Dallos et al, 1969)". (Legouix and Pierson, 1981)

Legouix and Pierson (1978), Legouix et al (1978) studied guinea pigs to investigate the relations between the CM input-output function and cochlear fatigue. They used low-frequency tone bursts (100 Msec) and measured individual

diffections, positive and negative, and plotted them against the acoustic pressure in order to obtain a transfer characteristic of CM generator. This characteristic displayed a saturating non-linearity and also an asymmetry ground the ordinates. They noted that the CM recorded with differential electrodes represents the average output from a relatively large number of hair cells and is probably not an exact representation of the unit CM, except perhaps at very low frequencies. This asymmetry of the characteristic differed among the various individuals ana that those presenting a large negative asymmetry were very labile and diminished after a fatigue. They proposed that the symmetry of the CM response appears to be more rapidly modified than the overall amplitude.

Legouix and Pierson (1981) reported that the asymmetry can be modified by various physiological factors for example asphaxia provokes a rapid decrease of CM amplitude while the asymmetry shows a transient increase. They found a phase of dominating positivity after return to normal breathing, when the negativity was increased, the fatigue was more prominent.

"During the recovery period, when the asymmetry was positive or when the negativity was minimal, the fatigue was sometimes replaced by a facilitation this effect probably represents a recovery process that is accelerated by the tone. It likely has some relation to the oveshoot that follows a period of anoxia". (Logouix and Pierson, 1981)

b. Relations of Cochlear Fatigue to the Phenomenon of Interference:

Interference is defined as the suppressing effect of a tone (interfering tone) on the CM produced by another tone (probe tone). Engebretson and Eldrdge (1968) state that this classical phenomenon has been related to the same asymmetric non linearity of the CM generator that produces the negative summating potential.

Legouix and Pierson (1981) state that the decrease of CM is present only during the presentation of the interfering tone and disappears when it is turned off, if the intensity of the interfering tone is raised, a level is reached it which the CM reduction remains after cessation of the interfering tone, this reduction is not different from fatigue. They further suggest that the interference is frequency dependent." When the amplitude of the CM evoked by a probe tone is plotted against the frequency of the interfering tone, the curve shows a single peak of reduction when the interfering frequency is near the best frequency of the locus that is recorded (Legouix et al 1973)". (Legouix and Pierson, 1981)

Legouix et al (1980) recorded CM in the first turn of the guinea pig cochlea, and the magnitude of the CM response to a fixed probe tone was measured as a function of the frequency of the interfering tone presented at a constant SPL,

the probe frequency was set at 7.5 KHz, 50 dB SPL and the interfering frequencies were presented at 60 dB. They found that the amplitude of the CM response to a probe tone showed one dip at a frequency slightly above the best frequency and a tail toward the low frequencies. They delivered the stimuli through a closed acoustic system and monitored by a sound probe whose tip was at 3 mm from the tympanic membrane, when the interfering tone intensity was above 90 dB, a lasting reduction of CM was observed. Again they measured the CM amplitude 5 sec after terminating the interfering tone and was used as a measure of fatigue. So, they further reveal that the dependence of this fatigue on the frequency of the interfering (or fatiguing) tone was similar to that in interference hence it suggests that the assymetric non-linearity which is involved in interference participates also in the mechanism of fatigue.

c. Mechanical process:-

Legouix and Pierson (1981) write that the primary factor in the destructive action of noise on the organ of corti is the excessive amplitude of vibrations of the cochlear partition. Spoendlin (1971) demonstrated in special cases with very high peak pressures there were

some structural ruptures. Legouix and Pierson (1981) further report that this type of mechanical lesion is more likely with impulse noises, whose features include a short rise time, a short duration, and a high peak amplitude (e.a. firearms noise). They state that if the intensity is sufficient, one single presentation may be followed by TTS or PTS. Eldredge et al., 1961; Dancer and Franke, 1977 (cited by Legouix and Pierson, 1981) report that because of the short duration, the important factor determining the damage seems to be the peak amplitude. Legouix and Pierson (1981) explain:

"with high amplitudes, the ear structures are driven to excessive excursions and it is very likely that a membrane may be ruptured. For instance gun fire often produces a rupture of the tympanic membrane, in general, the effects of impulse noise on humans are indistinguishable from those of continuous noise. Both types produce a notch in audiogram around 4 KHz. In guinea pigs, histological studies have confirmed that destruction of the hair cells (with toy caps) are maximal in a narrow region in the second turn".

Poche et al (1969) also suggest similar destructions can be produced by a 4 KHz pure tone at the narrow region in the second turn.

Pugh et al (1974) found the decrease of CAP recorded in monkeys continued for a few minutes after cessation of the noise. Later they observed a rebound after 20 hours. Legouix and Pierson (1981) state that the above features

have been observed also with continuous tones, but in this case, it may signal the occurance of a particular mechanical action associated with large peak amplitudes.

Luz and Hodge (1971) reported when the lesion increases gradually during successive presentation of impulse noise, a structural fatigue may be involved. They further state that this type of mechanical effect is probably related to other biochemical changes elicited by the exposure.

d. Biochemical Processes:

Legouix and Pierson (1981) reported that biochemical disturbances appear more probable than mechanical destruction when the noise is of relatively moderate intensity but of long duration. They suggest further that some metabolic components could be exhausted after a while, which could entail structural and irreversible damage.

Fernandez (1955) reported that the metabolism of the organ of corti has been found to be of the same order as that of the neural tissues and is very dependent upon the oxygen supply. Thalmann (1975) suggests that EP is more directly dependent upon oxygen than the membrane potential of the hair cells, which appear to derive more energy from

anaerobic reactions. Lawrence et al 1967; Hawkins (1971) report: "Noise would produce a constriction of the small vessels that supply the stria vascularis and the organ of corti". Hawkins (1971) observed vasoconstrictions in the supra-strial portion of the spiral ligament in the guinea pig and chinchilla.

Hawkins (1976) observed in monkey who sacrificed after noise exposure vasoconstriction in the strial capillaries but not in those of the spiral ligament. Further he suggested that the great fragibility of the chinchilla cochlea that has been described by many authors might be related to constriction of cochlear capillaries. Hawkins (1971) gives his explanation that an increase of the blood flow to the cochlea would explain the rebound of CAP and CM magnitudes that is sometimes observed after exposure.

In contrast, Perlman and Kimura (1962) did not observe any change in the capillaries in the apical part of the guinea pig cochlea during the presentation of low-frequency tones. Duvall et al (1974) suggested that damage to hair cells occur before the changes in stria vascularis, and Hawkins (1976) found strial edema and loss of suprastrial cells in chinchilla while no alteration in the hair cells was detectable.

Misrahy et al (1958b) reported that the oxygen availability may be an important factor since oxygen consumption is increased during the presentation of high intensity noises. Tondorf and Brogan, 1955; Pierson and Legouix 1976) suggested that the CM losses are accelarated in animals breathing gas mixtures containing lower percentage of oxygen than air.

Drescher (1976) emphasized with the increase in temperature the decline of CM voltage during noise exposure, which indicates that accelerating the oxidative reactions has an adverse effect on the cochlear responses to excessive stimulation.

Legouix and Pierson (1981) reported: "Mutabolic reactions involve alteration of C - partial pressure, changes of PH, accumulation or exhaustion of metabolities, changes in Acetylcholinesterage activity, changes in ARM content of various structures".

Legouix and Pierson (1981) suggest that the biochemical changes that occur in the organ of corti under the influence of noises is the alteration of the ionic content of the fluids and of the hair cells. Misrahy et al (1958b) suggested that the acoustic vibration could induce modifications in the permeability of the reticular membrane, leaving potassium ions to leak out from scala media and block the hair cells and nerve endings. Tasaki and Fernandez (1952) observed

when potassium content of perilymph is increased, cochlear responses are reversibly reduced. Legouix and Pierson (1977) report that a small quantity of Kcl in the perilymph not only decreases CM responses but also increases the fatiguability of the responses to high-intensity stimuli.

Legouix and Pierson (1981) reveal that the electron microscopic studies have shown that zonule occludes, the tight junctions between sensory and supporting cells of the organ of corti, prevent leakage of endolymphatic components toward the hair cells (e.q. Beagley, 1965). Duvall et al (1969) reported that the leakage of endolymph can result from ruptures of the reticular lamina. Bohne (1976) suggested that noise exposure can quickly produce cellular degeneration in the organ of corti and will temporarily disrupt the continuity of the reticular lamina so that endolymph can escape toward the hair cells.

e. Hydrodynamic Processes:

Legouix and Pierson (1981) mention that the hydrodynamic processes associated with the cochlear travelling wave appear to be responsible for some of the effects produced by high intensity noises. They observed the poststimulatory depre-ssion of CM that follows the presentation of a very high -

intensity tone for a few seconds. "This particular type of fatigue was initially described by Davis in 1938 (Davis, 1951) and has been fermed hysteresis". (Legouix and Pierson, 1981)

Legouix and Pierson (1981) further explain that the depression of CM is followed by a relatively slow recovery, suggesting a sort of inertia in some structure of the ear, it appears independently of any reflex action of the ossicular muscles and is clearly a cochlear phenomenon. Burgeat and Burgeat - Menguy (1964) propose that it is a biochemical mechanism and considered as similar to other types of fatigue.

Legouix and Pierson (1974) conducted a study on guinea pig to record CM by differential electrodes. Electrode was introduced in the first and third turns of cochlea, a sustained low-level tone was used, upon which was super-imposed another high-intensity tone. They found that the high-intensity tone produced a depression (hysteresis) in all animals and occured only when the superimposed tone reached 100 dB SPL and thereafter increased with level. They found the duration of the recovery was proportional to intensity of the stressing tone and its duration. They report further that during the presentation of a high - intensity tone, the dc variation, after an onset transient, displayed a progressive negativity that usually passed

through a maximum and at the end of presentation of the tone, the negativity decreased slowly, while the CM response to a test tone recovered its original size.

They write further that the various characteristics of hysteresis suggest that it is related to some hydrodynamic effects. Bekesy, 1960 and Tondorf, 1957 (cited by Legouix and Pierson, 1981) report that the traveling wave elicited by high-intensity tones is accompanied by complex movements of the fluids. "They manifest themselves in the fluid by eddies that may account for a unidirectional movement of the cochlear partition and can explain the occurance of SP (Tondorg, 1970)". (Legouix and Pierson, 1981).

Burgeat et al (1963) reported on increase in hydrostatic pressure in the semicircular canals of the guinea pig during the presentation of high intensity noises. Legouix and Pierson (1973) observed fluid movements with a microscope and found that fluid movements associated with membrane displacement were present with low frequencies at high intensity. Further they report that an outward flow of perilymph occured when small holes were drilled in the walls of the scala vestibuli, at the same time an outward displacement of the stapes and of the ossicular chain was visible and indicated a hyperpressure in the

scala vestibuli. This pressure was transmitted to the vestibular spaces, since the fluid escaped also through holes drilled in the semicircular canals and the fluid pressure in the scala tympani was decreased in such a way that when a hole was made in the wall of the scal tympani, air was aspirated and replaced the fluid. At the low frequencies and high frequencies they found that the perilymph seemed to escape entirely from the cochlea to be replaced by air. They have shown in the cochlear partition, after making a relatively large hole in the third turn, was moved toward the scala vestibuli and at the end of the tone, the fluid moved back into the cochlea and cochlear partition resumed its resting position. Legouix and Pierson (1981) proposed that the fluid displacement during hysteresis have a relatively large amplitude, which raises questions about their physiological consequences. They write that it is not clear whether or not these mechanical processes are detrimental to the cochlear structures and case that they may protect against acoustic trauma, since the vibratory movements are probably decreased during hysteresis. The fluid displacements (suggest Legouix and Pierson, 1981) may also account for the vestibular effects that are classically observed during exposure to high-intensity noises.

IV. **Patterns of Hair Cells Damage:**

It is well established that the intense sounds damage the hair cells of the cochlea.

Stockwell, Ades and Engstrom (1969) found that the sounds at 150 dB produced more hair cell damage than did the sounds of 130 dB and that the damage caused by the more intense sound was also less narrowly localized. He has observed that the increased sound intensity caused damage to spread primarily toward the base. It is concievable (writes Engstrom, 1969) that distance along the organ of corti rather than exposure frequency determines the radial distribution of damage. His further observations are: lower frequencies caused maximum damage near the apex than did higher frequencies, so the proportionately greater effect of the low frequencies on the more distal hair cell row could be due to the fact that cells in those rows near the apex are relatively more susceptible to damage than those nearer the base, however, the damage curves suggest this is not the case.

Dolan, et al (1975) have discussed about the damage of OHCs and IHCs after noise exposure. But their data don't permit for definite answer. But evidence was found

in the study on cat, there was almost complete destruction of OHCs, but a large number of IHCs in the apical turn were still present. Post exposure testing indicated complete loss of hearing although some IHCs remained in the apical and basal turns.

Beagely (cited by stockwell, Ades and Engstrom, 1969) has stated that OHC1 (first row of OHCs) shows greatest damage in guinea pig ears when exposed to a 500 Hz tone. But stockwell et al (1969) have observed that OHC1 was most severely damaged in ears exposed to 4000 Hz. In contrary to Beagely's results, they have observed that 0HC1 showed the least damage of any OHC row after exposure to 500 Hz.

Stockwell, et al (1969) exposed ears to 4000 Hz and observed that the IHCs were damaged almost as severely as OHC and the most severely damaged OHC row was the innermost, OHC1. Ears exposed to lower frequencies on the other hand, showed damage that was increasingly confined to OHC. In these groups damage was least severe in OHC1, greater in OHC2, and still greater in OHC3.

V. **Damage of Supporting Cells due to Acoustic Stimulation in the cochlea:**

Liberman and Kiang (1978) have observed that the regions of cochlea where a full complement of hair cells remained, all supporting structures of the organ of corti were typically present and erect - even in regions where some hair cells had been destroyed, the supporting structures frequently appeared almost normal. Further they report as follows: in regions wnere only IHCs remained, it was possible to see loss of Dieter's cell and/or outer pillar cells, with partial collapse of the tunnel of corti. In certain regions of some traumatized ears, the organ of corti had completely disappeared only a layer of low cuboidal cells lined at the surface of the Basilar Membrane - loss of organ of corti was typically accompanied by severe depletion of the layer of tympanic mesothelial cells that lie under the basilar membrane.

Engstrom and Engstrom (1979) also found that the pillar cells in the first cochlear turn are nearly as susceptible to noise exposures as are outer hair cells and believed this to be related to the cochlear damage in industrial noise.

VI. Revised Model of Adaptation and Loudness Gain:

Using a new method (Vyasamurthy, 1977) of measuring adaptation data were collected on normal hearing adults. Further Vyasamurthy (1982) has proposed a revised model for Loudness Coding During Auditory Adaptation in ORMLA (Objective Residual Monaural Loudness Adaptation). He reports that the revised model of adaptation answers most the controversies which are prevailing in the area of auditory adaptation. It provides possible answers to the following:

a) asymptotic adaptation;

b) perstimulatory adaptation and levelling off of adaptation;

c) the discrepancy observed by Weiler and Glass (1979) while verifying Small's model (1963) using monaural heterophonic technique and

d) the controversy whether, adaptation is real or not real.

He has presented the essence of the revised model for loudness coding during auditory adaptation in the following steps:

1. The adapted neural units are of three types:

i) Stable adapted neural units (a)

ii) Unstable adapted neural units (a_1)

iii) Unstable adapted neural units (a_2)

2. 'a' units originate from the place of maximum stimulation of the basilar membrane.

3. The source of 'a ' units is the neural units innervating the Inner Hair Cells (NIIH) and they are produced by the action of the efferent system innervating the inner hair cells.

4. The source of 'a_2' units is the Neural Units innervating the Outer Hair Cells (NIOH) and they are produced by the action of the efferent system innervating the outer hair cells.

5. a_2 units perform a reverse function of a_1 units and also that of 'a' units. In other words, a_2 units (responsible for negative adaptation) will be contributing to the loudness during the adaptation process. a_1 and a_2 units may be linked to 'R_2' and 'R_1' processes (respectively) reported by Hirsh and Bilger (Ward, 1963) in connection with 'bounce' phenomenon observed within 2 minutes of recovery time during TTS experiments. Thus, the revised model corroborates Hirsh and Bilger's hypothesis that 'bounce' phenomenon is a neural phenomenon.

6. a_1 and a_2 units regain their original pre-adapted state whenever a test tone at an intensity higher than the adapting intensity is presented during the post adapted state. In other words, the moment the post-adapted test tone at an intensity higher than the adapting intensity is presented, the efferent action ceases.

7. For higher stimulus intensities, loudness of the post adapted test tone at the adapting intensity is determined by the total number of activated - neural units from the following:

i) Place of maximal stimulation of the basilar membrane.

ii) NIIH and

iii) NIOH

However, the contribution of the sensory units (activated by the action of the efferent system innervating the outer hair cells) for loudness of the post adapted test tone will be insignificant.

8. For low or moderate intensity stimulus, the loudness of the post adapted test tone at adapting intensity is determined by the un-adapted neural units and NIOH (sensory units of outer hair cells activated by the action of the efferent system innervating the outer hair cells).

9. **Possible Mechanisms of Loudness Adaptation:**

When a stimulus (1 KHz) of moderate intensity is continuously presented to one of the ears of a normal hearing subject, the adaptation process (i.e., a general and continuous decrease in the number of activated neural units) begins in the place of maximal stimulation of the basilar membrane. This process goes on as long as the adapting stimulus is continued; hence, stable adapted neural units (a) will be increasing. Simultaneously with the adaptation process, the number of sensory units innervating the outer hair cells, (NIOH) activated by the efferent system (innervating the outer hair cells) gradually increases as long as the adapting stimulus/is continued i.e. a2 units will be increasing. This process is known as 'negative adaptation' (or reverse adaptation). As the adapting stimulus is continued, more and more number of sensory units innervating the outer hair cells (NIOH) begin to fire nerve impulses. Initially, the rate of decrease in the number of activated neural units will be taster than the rate of increase in the number of activated (through the efferent action) sensory units innervating the outer hair cells. After some time, the rate of increase in the activated sensory units innervating the outer hair cells will 'catch-up' with the rate of decrease in the number of activated units of the 'simple category'. As a consequence of this, the total number of nerve impulses contributing to the loudness of the adapting stimulus (or test tone at the adapting intensity) remains fairly constant (contribution of the nerve impulses from the place of maximal stimulation of the basilar membrane will be decreasing, but the contribution of nerve impulses from the NIOH will be increasing). This may asymptotic loudness adaptation can be explained.

Incidently, the above description, reveals a 'vital' information about 'inhibition' process. 'Inhibition' process in the auditory system is poorly understood. The revised model appears to be a 'break-through' in psycho-acoustic and electro-physiological research as far as understanding the mechanisms of 'inhibition' process in the auditory systems is concerned. Since a2 units are responsible for inhibiting the adaptation. Process, phenomenon of negative adaptation can be viewed as 'inhibition process". The efferent system innervating the outer hair cells, is responsible for a2 units. Since the efferent system is known for its 'inhibitory' function, the negative adaptation brought about by the efferent system (innervating the outer hair cells) may be regarded as 'inhibition of adaptation process.

10. Xh depends on (a) units only
(Xh = adaptation measured at an intensity higher than the adapting intensity).

11. Xb depends on a, a1 and a2
(Xb = adaptation measured at the base-line condition or adaptation measured at the adapting intensity or simple adaptation).

12. XL depends on a_1, a_1l and a_2l
(XL) = adaptation measured at an intensity lower than the adapting intensity).
a_1 - adapted neural units at a level lower than the base-line condition.
a_1l - Positive unstable adaptable/adapted neural units at a level lower than the base-line condition.
a_2l - -ve unstable/adapted neural units at a level lower than the base line condition.

13. The effect of 'a' units can either be enhanced by a, units and/or decreased or cancelled by a2 units. In other words, the loudness loss brought about by 'a' units can be aggravated by a, units and/or the loudness can be minimized or completely cancelled or loudness can be increased by the action of a2 units.

14. The revised model can be used to explain the different results obtained in this study.

3) $Xh > Xb < 0$;

$a > a+a_1 - a_2 < 0$

The necessary conditions are:

i) $a_1 < a_2$

ii) $a+a_1 < a2$

b) $Xh > Xb = 0$;

$a > a+a_1 - a = 0$

The necessary conditions are:

i) $a_1 < a_2$ and

ii) $a+a_1 = a_2$

c) $Xh > Xb > 0$;

$a > a+a_1-a_2 > 0$

The necessary conditions are:

i) $a_1 < a_2$

ii) $a+a_1 > a_2$

iii) $a > 0$

d) $Xh = Xb = 0$;

$a = a+a_1-a_2 = 0$

The necessary conditions are:

i) $a = 0$(This was not observed)

ii) $a_1 = a_2$

e) $Xh < Xb = 0$;

$a < a+a_1-a_2 = 0$

The necessary conditions are:

i) $a_1 > a_2$ (This was also not observed in any subject)

ii) $a+a_1 = a_2$

f) $Xh < xb > 0$;

$a < a+a_1-a_2 > 0$

The necessary conditions are:

i) $a_1 > a_2$

ii) $a+a_1 > a_2$

iii) $a > 0$

g) $X_L < Xb > 0$;

$al + a_1 l - a_2 l < a+a_1-a_2 > 0$

The necessary condition is

i) $al+ a_1 l - a_2 l < a + a_1 - a_2$

15. Xb (SDLB) >Xb (MH) is due to 'binaural interaction'. The 'binaural interaction' can be defined as the efferent action of monaural stimulation on the periphery of the contralateral auditory system.

16. Xb (at 60 dB) in SDLB = Xb (at 80 dB) in SDLB = 20 dB (the data reported by Weiler et al, 1972). is due to a phenomenon which may be named the "eclipse - phenomenon". The 'eclipse phenomenon' is the phenomenon in which the increase in the amount of adaptation which should result in the adapting ear, due to the increase in the intensity of the adapting stimulus (i.e. increasing the intensity from 60 dB to 80 dB SPL) is 'eclipsed' by the combined actions of the two efferent systems (the efferent system innervating the outer hair cells and the efferent system innervating the inner hair cells) in the comparison ear. The action of the efferent system innervating the outer hair cells is to increase the loudness of the post adapted test tone in the comparison ear and the action of the efferent system innervating the inner hair cells is to decrease the loudness of the post adapted test tone in the comparison ear. When the adapting stimulus is at 80 dB SPL in the adapting ear, there will be combined actions of the efferent systems in the comparison ear which results in *Ltba = *Ltbb = Ltbb (* = comparison ear)
Ltba = Loudness of a test tone at the adapting intensity after adaptation.

Ltbb = Loudness of a test tone at the adapting intensity before adaptation.

17. Xh < Xb > 0 at 60 dB SPL adapting intensity in SDLB, is due to the fact that Xb will be greater as a result of 'Loudness gain' in the comparison ear (binaural interaction), whereas, this 'loudness gain', will be absent during Xh measurements (when the post adapted test tone is presented at an intensity higher than the adapting intensity the efferent action ceases.

18. Xh < X b > 0 at 80 dB SPL adapting intensity in SDLB, is due to the combined efferent actions in the comparison ear and the adapting ear, together with the cessation of efferent actions during Xh measurements.

19. 'Binaural interaction' is effective only when the adapting stimulus and the comparison stimulus are of the same frequency. In support of this, the data reported by Morgan and Davis (1973) can be recalled can be recalled (they found that 500 Hz tone at 70 dB SPL for 12 minutes did not show adaptation for SDLB technique (hetereophonic technique).

20. The fact that the efferent action (negative adaptation) persists even after the withdrawl of the adapting stimulus (Xh > Xb < 0) is probably an important clue to the mechanism of 'tinnitus'. The pathophysiology of tinnitus is yet to be known. The discovery that the efferent action (action of the efferent system innervating the OHCs) activates the sensory inputs even in the absence of the acoustic stimulus is a clear evidence for understanding the pathophysiology of tinnitus. Tinnitus, arising out of exposure to continuous auditory stimulation is likely to be due to the action of the efferent system innervating the outer hair cells. A disturbance in the efferent system innervating the outer hair cells, may be responsible for the 'tinnitus' observed in, atleast, some pathological cases.

Further Vyasamurthy (1985) reports the interpretation of the revised model in the light of the recent developments in auditory physiology and proposes neural models of the efferent mechanisms during auditory adaptation.

In support of the assumption that the action of the ESIOHCs is to increase the loudness of the post adapted test tone, he cites the following studies: (1) Spoendlin (1975) reports that the efferents to the outer hair cells (OHCs) synapse with the hair cells and that the enormous efferent nerve supply to the OHCs would tally with a concept of a more monitoring role of the OHC system. (2) Cody and Johnstone (1982) have demonstrated that the acoustically activated activity of the crossed Olivo cochlear bundle (COCB) may modify the response of the OHCs to acoustic trauma i.e., the efferent action counter acts the effect produced by the noise. Further, they have found that the sensitivity of the auditory neurons increases due to the action of the COCB. (3) Gerken (1984) has demonstrated in conscious cats that the evoked response amplitude for 3 KHz tone bursts (> 60 dB SPL) were greater in the presence of continuous tone (3 KHz at 70 dB SPL), He has termed the facilitation by sustained tone "enchancement", He has also speculated that the efferent action might be responsible for the "enhancement". (4) Fex et al, (1982) have concluded that the efferent terminals to the OHCs may participate in the recycling of the released neuro-transmitter using

Aspartate Amino transferase (AA Tase). Interestingly, they have found the AA Tase like immuno reactivity in the Medial system of efferents but not in the lateral system. (5) Comis and Whitfield (1968) report that the acetylcholine (neurotransmitter of ESIOHCs) is an excitatory neurotransmitter. (6) Hoffmann et al (1983) have detected enkephalin like peptides (putative neuro-active substance) in the efferent terminals of OHCs. (7) Pickles (1982) reports that the centrifugal fibres to the cochlear nucleus are both excitatory and inhibitory. (8) Stopp et al (1983) suggest that the efferent system may increase the dynamic range of the neurons.

In support of the assumption that the ESIIHCs (and 'a' units) is responsible for decreasing the loudness of the post adapted test tone, he cites the following studies:

(1) Spoendlin (1975) has established that the efferents to the inner hair cells (IHCs) synapse with the afferent dendrites.(2) Sohmer (1966) reports that the electrical stimulation of uncrossed olivo-cochlear bundle (UOCB) reduces the N_1 potential of the cochlea. (3) It has been established that nor-adrenaline is an inhibitory neuro-transmitter of the efferent auditory system which produces inhibition (Pickles, 1982).

Regarding the electrical stimulation of COCB Vyasamurthy (1985) writes:

"Although many studies, as mentioned earlier, show that the ESIOHCs is responsible for loudness gain, Widerhold and Kiangl (1970) have reported that the electrical stimulation of COCB results in the desensitization of the 'tips' of the tuning curves. Further, Pickles (1982) has concluded that the electrical stimulation of COCB reduces the response of the auditory nerve fibres to sound. This controversial issue can be easily resolved if we recall the observations of Bodian (1983); Siegal and Kim (1982); and Mountain (1980)." It must be kept in mind that evidences for inhibitory role of the efferent innervation of the cochlea pertains to IHC system — function of ESIOHCs is yet to be known. Presence of efferent innervation of the vestibular receptors suggests a general role for all labyrinthine efferent pathway such as the enhancement of sensitivity of the various receptors (Bodian, 1983)".

(1985)

Further, Vyasamurthy/points out "Electrical stimulation of COCB increases the damping of cochlear partition (Seigel and Kim, 1982; Mountain, 1980)".

Vyasamurthy (1985) comments: "it may not be a correct assumption that the electrical stimulation of COCB and the acoustic stimulation of COCB produce similar effects. We should have - Crane's (1983) view of OHC afferents and OHC efferents acting as a servo-system, in mind, when COCB is electrically stimulated. Naturally, we can expect the servo-system to be disturbed when COCB is electrically stimulated. Indeed, the damping of the basilar membrane increases (or negative damping decreases i.e., whan COCB is electrically stimulated) might be responsible for the desensitization of the 'tips' of the tuning curves and also for the decrease in N_1 response. The acoustic stimulation of COCB may be expected to result in the increase of the sensitivity of OHC afferents through the recycling of the released neuro-transmitter (acetylcholine?) as suggested by Fex et al (1982)".

VII. An Active Mechanism:

There are two groups of efferent system

i) Efferent system innervating the OHCs

ii) Efferent system innervating the IHCs Weiss (1982) studied Bidirectional properties in vertebrate hair cells i.e. Mechano-electric and electro-mechanical. Weiss (1982) states that the stimulation of the efferent system leads to an electrical effect and consequently a mechanical effect on the OHC which in turn modifies the mechanical input to the IHCs. He has mentioned the following as evidences:-

a) Spontaneous acoustic emissions from the cochlea.

b) Changes in the distortion products as a result of efferent stimulation.

c) Presence of Action filaments in the stereocilia.

Davis (1983) has proposed that there is an active process in cochlear mechanics. The OHCs responds to sounds of intensities less than 60 dB. The OHCs when stimulated have an effect on IHCs. Thus, indirectly the IHCs too are stimulated at low intensities. At high intensities (greater than 60 dB), the IHCs are directly stimulated. Thus the IHCs are stimulated at low intensities and at high intensities. At higher intensities they are stimulated by the

passive system. At low intensities (less than 60 dB), an active process is present.

Other investigators (Kemp, 1978, 1979; Zwislocki, 1980; Zyrek, 1981; Zwislocki and Klessy, 1982; Meeley and Kim, 1983) have also suggested that there is an active mechanism in the cochlea. Siegel and Kim (1982) state that the active mechanism is controlled by the Central Nervous system through the activity of the efferent synopses on the OHCs. Many investigators are of the opinion that the active mechanism is responsible for the greater sensitivity and sharp tuning expressed by the 'tips' of the neural tuning curves.

Crane (1983) suggests that the hyperactivity of the active mechanism may be responsible for the spontantaneous acoustic emissions. While discussing the functions of the efferent auditory systems. Crane (1983) comments: "OHC afferents are part of the serve - control system (for instance, reporting back the state of OHC responses to efferent excitation) the speed of a servo-system can generally be increased if position information is available from the mechanism under control - another possibility is that OHC afferents reflect a crude estimate of the acoustic level at the OHCs and that they rather than the IHC afferents are the source of the efferent excitation.

VIII. Models of the Efferent Mechanisms:

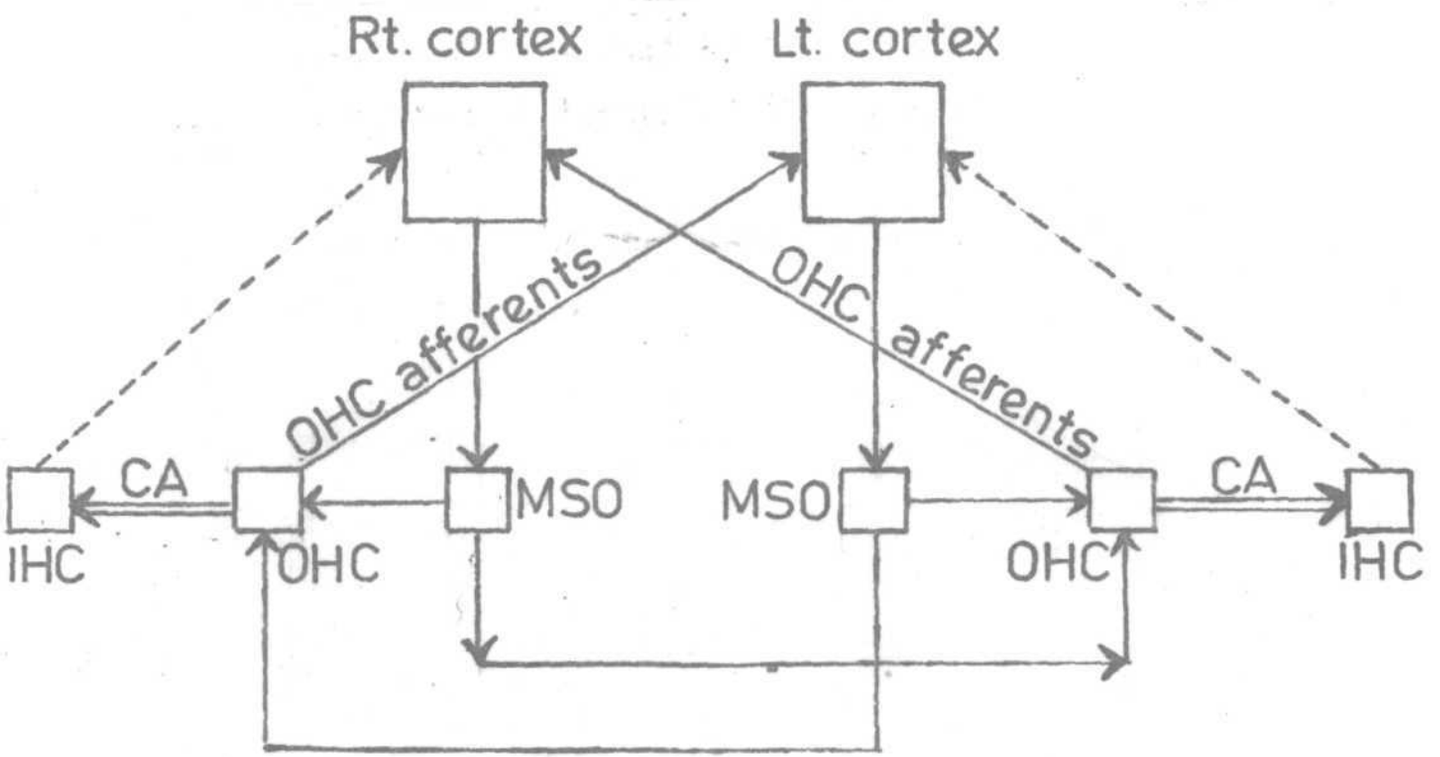
By putting all the above pieces of information, Vyasamurthy (1985) proposed the following neural models (Figure 1 and 2) of the efferent mechanisms during auditory adaptation. He explains:

" The model (Figure 1) suggestes that the efferent system passing through the Medial Superior Olive (MSO) is responsible for the loudness gain (recycling of the released neurotransmitter) and the model (Figure 2) suggests that the efferent system passing through the lateral superior olive (LSO) is responsible for loudness loss ('a' units are also responsible for loudness loss). The efferent system passing through LSO may be expected to release nor-adrenaline to inhibit the responses of the neurons innervating the IHCs as the etferents to the IHCs synapse withthe afferent dendrites of the IHCs".

Note: In figure-1 dashed line means not important for loudness gain; Cochlear Amplifier (CA) refers to active mechanism

-See Davis, 1983.

Figure 1



Model of the Efferent mechanisms during auditory adaptation (Loudness gain)

(Vyasamurthy, 1985)

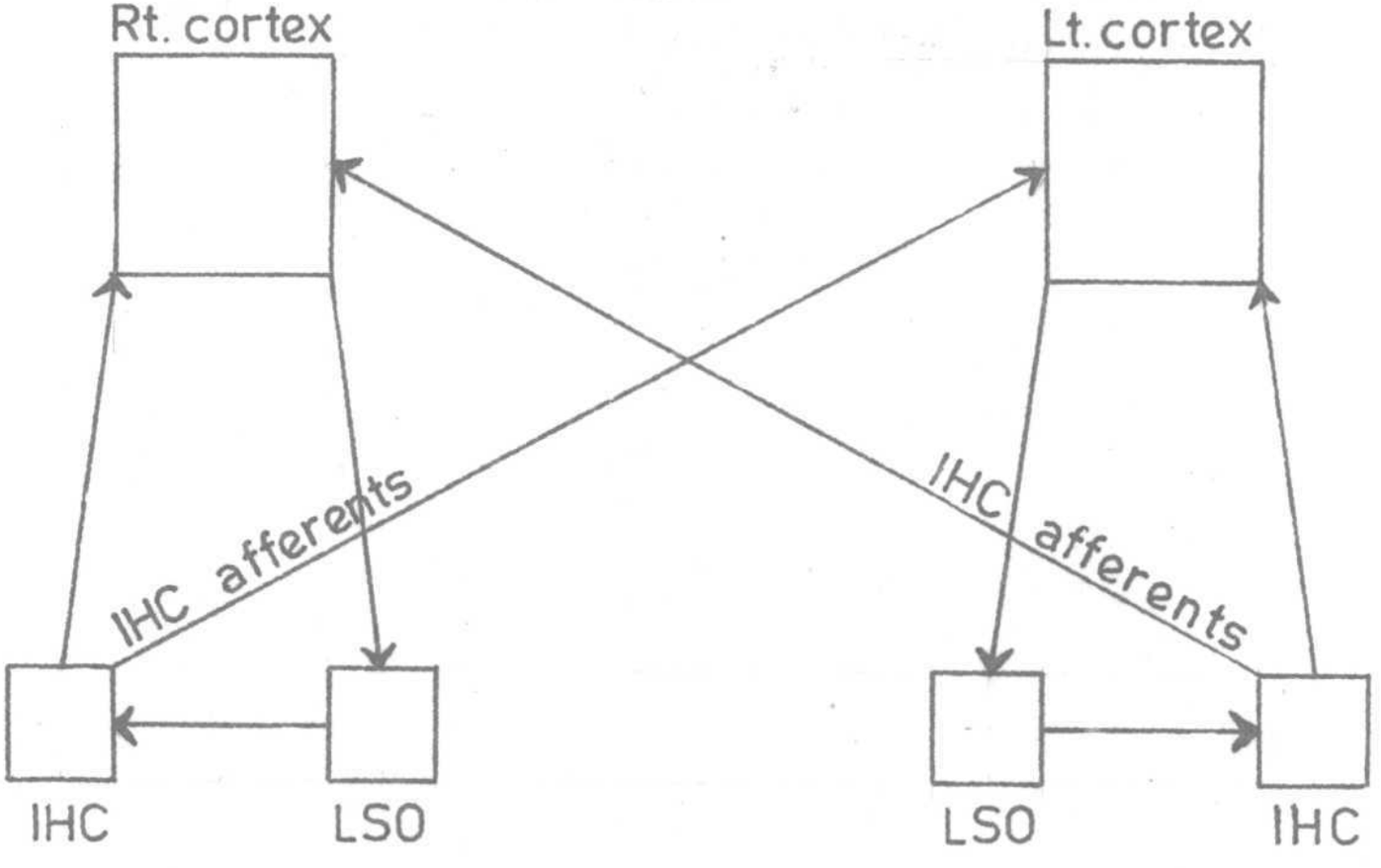


Figure 2

Model of the Efferent mechanisms during auditory adaptation (Loudness Loss)

(Vyasamurthy, 1985)

IX. Cochlear microphonic and TTS in man:-

"Cochlear microphonic potentials (CM) were recorded, by means of surface electrodes, before during and after white-noise-induced temporary threshold shifts (TTS) in human volunteers. The behavioural threshold shift was not accompanied by a change in amplitude of CM. These findings indicate that in humans, the site affected by the noise exposure and which probably gives rise to the TTS is central to the site of generation of CM. In a previous study, the compound action potential generated in the auditory nerve was found to be of lower amplitude and longer latency during TTS, and it is thus proposed that the site affected is peripheral to the generation of conducted action potentials. The synapse between hair cells and the auditory nerve fibres is the most likely candidate to be the affected side". (Pratt et al; 1978)

METHODOLOGY

Subjects:

The study consists of twenty one males and ten females ranging in age from 17 years to 25 years. The subjects were selected on the basis of the following criteria:

No history of

i. ear discharge;
ii. ear ache;
iii. tinnitus;
iv. giddiness;
v. headache;
vi. brain damage and
vii. exposure to loud sounds.

All the subjects had hearing sensitivity within 20 dB HL (ANSI 1969) in the frequencies - 250 Hz, 500 Hz, 1KHz, 2 KHz, 4 KHz and 8 KHz.

Instrument used:

Beltone 200-C audiometer with TDH-39 earphones and circum aural cushion MX-41/AR was used. The audiometer was calibrated according to the specifications given by ANSI 1969.

Calibration:

Calibration of the audiometer was maintained using Bruel and Kjaer calibration unit. It consists of artificial ear (type 4152), Sound Level Meter (2203) and Octave Filter Set (1603). Calibration was done in a sound treated room.

Periodic checking was made to keep the unit in calibration throughout the period of study.

Test Environment:

The study was carried out in an acoustically sound treated room at All India Institute of Speech and Hearing. The ambient noise levels present in the test room were below the proposed maximum allowable noise levels.

Procedure:

All the subjects were screened at 20 dB HL (ANSI, 1969) for the frequencies 250 Hz, 500 Hz, 1 KHz, 2 KHz, 4 KHz and 8 KHz in both the ears.

The subjects were divided into 3 groups viz. A, B, C. A, B, C groups had 17 subjects, 7 subjects and 7 subjects respectively. Bach group had to undergo two experiments.

Totally six experiments were done. Experiments I and II were done using group A. Experiment III and IV were done using group B. Experiments V and VI were done using group C.

Experiment-I:

Thresholds were established for 4 KHz and 8 KHz separately for the group A.

The seventeen subjects were then exposed to 4 KHz tone at 110 dB HL in right ear (Monaural) for ten minutes.

Threshold, was then determined in the same ear immediately after cessation of the stimulus, using 8 KHz as the test tone.

Temporary Threshold Shift immediately after the termination of the fatiguing stimulus (TTSo) was computed.

i.e. TTSo = Threshold at 8 KHz tone immediately after termination of the fatiguing stimulus - Threshold at 8 KHz before the ear was fatigued.

Temporary Threshold Shift after the recovery time of 2 minutes (TTS_2) was also determined by finding the

threshold at 8 KHz, 2-minutes after the termination of the fatiguing stimulus.

TTS_2 = Threshold at 8 KHz, 2 minutes after the termination of the fatiguing stimulus - threshold at 8 KHz before the ear was fatigued.

Experiment-II:

Thresholds were established for 4 KHz and 8 KHz for the group A.

A rest period of at least 24 hours was allowed between the experiments I and II. The 17 subjects were exposed to binaural stimulation (4 KHz tone at 110 dB HL to right ear and 4 KHz tone at 80 dB HL to left ear) for 10 minutes.

TTSo and TTS_2 were determined in right ear using 8 KHz as the test tone, in the same manner as it is described in experiment I.

Experiment-III:

Thresholds were established for 2 KHz and 4 KHz separately for the group B.

The seven subjects were then exposed to 2 KHz tone at 100 dB HL in right ear (monaural stimulation) for 10 minutes.

Threshold, was then determined in the same ear immediately after cessation of the stimulus, using 4 KHz as the test tone.

Temporary Threshold Shift immediately, after the termination of the fatiguing stimulus (TTSo) was computed.

i.e. TTSo = Threshold at 4 KHz tone immediately after termination of the fatiguing stimulus - threshold at 4 KHz before the ear was fatigued.

Temporary Threshold Shift after the recovery time of 2 minutes (TTS_2) was also determined by finding the threshold at 4 KHz, 2 minutes after the termination of the fatiguing stimulus.

TTS_2 = Threshold at 4 KHz, 2 minutes after the termination of the fatiguing stimulus - threshold at 4 KHz before the ear was fatigued.

Experiment-IV:

Thresholds were established for 2 KHz and 4 KHz for the group B.

A rest period of at least 24 hours was allowed between the experiments III and IV. The 7 subjects were exposed to binaural stimulation (2 KHz tone at 100 dB HL to right ear and 2 KHz tone at 80 dB HL to left ear) for 10 minutes.

TTSo and TTS_2 were determined in right ear using 4 KHz as the test tone, in the same manner as it is described in experiment III.

Experiment-V:

Thresholds were established for 2 KHz and 4 KHz separately for the group C.

The seven subjects were then exposed to 2 KHz tone at 100 dB HL in right ear (monaural stimulation) for 5 minutes.

Threshold, was then determined in the same ear immediately after cessation of the stimulus, using 4 KHz as the test tone.

Temporary Threshold Shift immediately after the termination of the fatiguing stimulus (TTSo) was computed.

i.e. TTSo = Threshold at 4 KHz tone immediately after termination of the fatiguing stimulus - Threshold at 4 KHz before the ear was fatigued.

Temporary Threshold Shift after the recovery time of 2 minutes (TTS_2) was also determined by finding the threshold at 4 KHz, 2 minutes after the termination of the fatiguing stimulus.

TTS_2 = Threshold at 4 KHz, 2 minutes after the termination of the fatiguing stimulus - threshold at 4 KHz before the ear was fatigued.

Experiment VI:

Thresholds were established for 2 KHz and 4 KHz for the group C.

A rest period of at least 24 hours was allowed between the experiments V and VI. The 7 subjects were exposed to binaural stimulation (2 KHz tone at 100 dB HL to right ear and 2 KHz tone at 80 dB HL to left ear) for 5 minutes.

TTSo and TTS- were determined in right ear using 4 KHz as the test tone, in the same manner as it is described in experiment V.

RESULTS AND DISCUSSIONS

Table-1 shows the temporary threshold shift (TTSo, TTS_2) at 8 KHz obtained in experiment-I.

Temporary threshold shifts (TTSo, TTS_2) at 8 KHz obtained in experiment-II are given in Table-2.

Table-3 presents the temporary threshold shifts (TTSo, TTS_2) at 4 KHz obtained in experiment-III.

Temporary threshold shifts (TTSo, TTS_2) at 4 KHz obtained in experiment-IV are presented in Table-4.

Table-5 shows the temporary threshold shifts (TTSo, TTS_2) at 4 KHz obtained in experiment-V.

Temporary threshold shifts (TTSo, TTS_2) at 4 KHz obtained in experiment-VI are given in Table-6.

Experimental Paradigms are shown in Table-7.

The mean TTSo values of experiments I and II show that the presence of contralateral stimulus and the absence of contralateral stimulus does not produce much difference in TTSo values.

TTS- values of experiments I and II also show not much difference.

The mean TTSo of experiments III and IV show that the presence of contralateral stimulus and the absence of contralateral stimulus does not produce much difference in TTSo values.

TTS- values of experiments III and IV show much difference.

The mean TTSo of experiments V and VI show that the presence of contralateral stimulus and the absence of contralateral stimulus does produce significant difference in TTSo values.

TTS_2 values of experiments V and VI show not much difference.

Table-8 shows significance of difference between the mean TTS values obtained in experiments I and II; experiments III and IV; and experiments V and VI.

It is clear from Table-8, there is significant difference between mean TTSo values obtained in experiments V and VI. Significant difference between TTS_2 values obtained in the experiments III and IV has also been observed.

TTSo values obtained in experiments I and II do not show significant difference, in the same way TTSo values obtained in experiments III and IV also do not show significant difference. On the contrary, TTSo values obtained in experiments V and VI show significant difference.

Comparison of TTSo obtained in experiments I and II with the TTSo values obtained in experiments III and IV shows that the TTSo values of experiments I and II are greater than those of experiments III and IV. This shows that the high frequency stimulus produces more TTS than a relative low frequency stimulus i.e. in this study 4 KHz fatiguing stimulus produced more TTS than 2 KHz stimulus.

This result agrees with the earlier reports that the high frequency fatiguing stimulus produces more TTS than the low frequency stimulus.

Comparison of TTSo values obtained in experiments III and IV with the TTSo values obtained in experiments V and VI shows that there is significant difference between the TTSo values obtained in experiments V and VI, whereas there is no difference between TTSo values obtained in experiments III and IV. These results can be explained in terms of efferent action.

Pratt et al (1978) have reported that TTS of 20 dB in normal hearing subjects may be due to 'reduced synaptic efficacy'. TTSo of experiment VI was significantly less than the TTSo of experiment V i.e. in the presence of contralateral stimulation (efferent action - see Cody and

Johnstone, 1982) the TTS obtained was less. In other words in experiment VI there was efferent action (due to contra-lateral stimulation) and there was no efferent action in experiment V (Monaural stimulation). From the results of the present study (especially the results of experiments V and VI) it is clear that due to efferent action the TTS reduces - this is in agreement with the findings of Cody and Johnstone (1982). Cody and Johnstone (1982) have established (see table-9) that the TTS reduces in the presence of contralateral stimulation.

A question arises regarding the mechanism by which the efferent action reduces the TTS. It is quite probable that the efferent action increases the synaptic efficacy, as a result, the TTS reduces. The same explanation may hold good in the study reported by Cody and Johnstone (1982). Thus the explanation for the reduction in TTS in experiment VI is likely due to increase in the synaptic efficacy as a result of efferent action.

The failure to observe reduction in TTSo in experiment IV when compared to experiment-Ill, needs explanation. If the efferent action reduces TTS, the TTSo obtained in experiment-IV should have been less than in TTSo obtained in experiment-Ill. But the results of the present study show

that there is no significant difference between TTSo values obtained in experiment III and IV. The explanation for the failure to observe TTSo in experiment-TV may be found in terms of the changes of the function of hair cells. In experiment-III and IV the duration of fatiguing stimulus was 10 minutes in contrast to 5 minutes which was used for experiments-V and VI. Hence in experiments III and IV temporary dysfunction of hair cells can be expected. Actually the present study shows more TTS in experiments III and IV than in experiments-V and VTZ. Since dysfunction of hair cells is responsible for TTS observed in experiments-III and IV, the efferent action (which mainly changes the synaptic efficacy) might not have been effective in reducing the TTS. Thus the failure to observe less TTSo in experiment-TV (in spite of efferent action) may be due to the fact that the efferent action changes the synaptic efficacy but not the dysfunction of hair cells. Had the efferent action changed in dysfunction of hair cells TTSo in experiment-IV should have been less than TTSo of experiment-Ill.

It can be concluded from the present study that the efferent action changes the synaptic efficacy without bringing much changes in the dysfunction of hair cells.

In support of Cody and Johnstone's (1982) observation that the efferent system innervating the outer hair cells increases the sensitivity of neurons a model of efferent mechanism during adaptation, proposed by Vyasamurthy (1985) may be cited. The figure-1 in his model shows that the efferent action passing through medial superior olive (MSO) is responsible for loudness gain. Vyasamurthy (1985) explains in terms of recycling of the released neurotransmitter as suggested by Fex et al (1982). The efferent action may (action of the efferent system innervating the outer hair cells) may increase the synaptic efficacy through the recycling of the released neurotransmitter. TTS of about 20 dB in human subjects may be due to reduced synaptic efficacy - this reduced synaptic efficacy can be set right partially by contralateral stimulation, which produces efferent action in the test ear (in this study right ear) as per the model described in figure-1.

Table-1: TTSo, TTS_2 (at 8 KHz) values obtained in Expt.I for group 'A'

Subject	Fatiguing stimulus		Duration in minutes	Measured at 8 KHz	
	Frequency	Intensity in HL		TTSo	TTS_2
1.	2.	3.	4.	5.	6.
1. Trial-I	4 KHz	110 dB	10	50	55
Trial-II	4 KHz	110 dB	10	30	10
Trial-III	4 KHz	110 dB	10	35	15
2. Trial-I	4 KHz	110 dB	10	30	35
Trial-II	4 KHz	110 dB	10	35	30
3.	4 KHz	110 dB	10	35	35
4. Trial-I	4 KHz	110 dB	10	35	25
Trial-II	4 KHz	110 dB	10	30	20
5.	4 KHz	110 dB	10	50	50
6.	4 KHz	110 dB	10	40	20
7.	4 KHz	110 dB	10	30	15
8.	4 KHz	110 dB	10	35	25
9.	4 KHz	110 dB	10	50	45
10.	4 KHz	110 dB	10	40	30

Contd. Table-1

In continuation of Table-1:

1.	2.	3.	4.	5.	6.
11.Trial-I	4 KHz	110 dB	10	40	35
Trial-II	4 KHz	110 dB	10	45	35
12.	4 KHz	110 dB	10	60	45
13.	4 KHz	110 dB	10	45	40
14.	4 KHz	110 dB	10	40	35
15.	4 KHz	110 dB	10	40	15
16.	4 KHz	110 dB	10	35	20
17.	4 KHz	110 dB	10	45	30

Table-2: TTSo, TTS_2 (at 3 KHz) values obtained in Experiment II for group 'A'

Subject		Fatiguing stimulus		Duration	Measured at 8 KHz	
		Frequency	Intensity in HL	in minutes	TTSo	TTS_2
1.		2.	3.	4.	5.	6.
1	Trial-I	4 KHz	110 dB	10	35	25
	Trial-II	4 KHz	110 dB	10	25	5
	Trial-III	4 KHz	110 dB	10	30	10
2	Trial-I	4 KHz	110 dB	10	30	35
	Trial-II	4 KHz	110 dB	10	35	25
3		4 KHz	110 dB	10	25	30
4	Trial-I	4 KHz	110 dB	10	30	25
	Trial-II	4 KHz	110 dB	10	35	25
5		4 KHz	110 dB	10	50	50
6		4 KHz	110 dB	10	35	20
7		4 KHz	110 dB	10	30	15
8		4 KHz	110 dB	10	35	30

Contd.Table-2

In continuation of Table-2

1.		2.	3.	4.	5.	6.
9		4 KHz	110 dB	10	40	40
10		4 KHz	110 dB	10	45	30
11	Trial-I	4 KHz	110 dB	10	45	35
	Trial-II	4 KHz	110 dB	10	45	35
12		4 KHz	110 dB	10	55	45
13		4 KHz	110 dB	10	45	30
14		4 KHz	110 dB	10	45	30
15		4 KHz	110 dB	10	35	25
16		4 KHz	110 dB	10	45	30
17		4 KHz	110 dB	10	45	40

Table-3: TTSo, TTS_2 (at 4 KHz) values obtained in Experiment III for group 'B'

Subject	Fatiguing stimulus		Duration in minutes	Measured at 4 KHz	
	Frequency	Intensity in HL		TTSo	TTS_2
1	2 KHz	100 dB	10	40	30
2	2 KHz	100 dB	10	50	40
3	2 KHz	100 dB	10	40	35
4	2 KHz	100 dB	10	30	20
5	2 KHz	100 dB	10	35	30
6	2 KHz	100 dB	10	35	30
7	2 KHz	100 dB	10	25	20

Table-4: TTSo, TTS_2 (at 4 KHz) values obtained in Experiment IV for group 'B'

Subject	Fatiguing stimulus		Duration in minutes	Measured at 4 KHz	
	Frequency	Intensity in HL		TTSo	TTS_2
1	2 KHz	100 dB	10	35	30
2	2 KHz	100 dB	10	45	35
3	2 KHz	100 dB	10	40	30
4	2 KHz	100 dB	10	25	15
5	2 KHz	100 dB	10	40	30
6	2 KHz	100 dB	10	35	20
7	2 KHz	100 dB	10	30	15

Table-5: TTSo, TTS_2 (at 4 KHz) values obtained in Experiment-V for group 'C

Subject	Fatiguing stimulus			Measured at 4 KHz	
	Frequency	Intensity in HL	Buration in minutes	TTSo	TTS_2
1	2 KHz	100 dB	5	20	15
2	2 KHz	100 dB	5	25	10
3	2 KHz	100 dB	5	15	10
4	2 KHz	100 dB	5	30	20
5	2 KHz	100 dB	5	25	10
6	2 KHz	100 dB	5	20	20
7	2 KHz	100 dB	5	30	20

Table-6: TTSo, TTS_2 (at 4 KHz) values obtained in Experiment-VI for group 'C'

Subject	Fatiguing Stimulus		Duration in minutes	Measured at 4 KHz	
	Frequency	Intensity in HL		TTSo	TTS_2
1	2 KHz	100 dB	5	15	10
2	2 KHz	100 dB	5	15	15
3	2 KHz	100 dB	5	15	10
4	2 KHz	100 dB	5	20	15
5	2 KHz	100 dB	5	15	10
6	2 KHz	100 dB	5	15	10
7	2 KHz	100 dB	5	20	15

Table-7: Experimental Paradigms

Expt.	Ipsilateral exposure	Contralateral exposure	Mean TTS in dB	
			TTSo	TTS_2
I	4 KHz 110 dB HL D = 10'		40.29 (7.89)	29.85 (11.36)
II	4 KHz 110 dB HL D = 10'	4 KHz 80 dB HL D = 10'	39.36 (8.42)	30.14 (10.40)
III	2 KHz 100 dB HL D = 10'		36.42 (8.01)	29.28 (7.31)
IV	2 KHz 100 dB HL D = 10'	2 KHz 80 dB HL D = 10'	35.71 (6.72)	25.00 (8.16)
V	2 KHz 100 dB HL D = 5'		23.57 (5.56)	15.00 (5)
VI	2 KHz 100 dB HL D = 5'	2 KHz 80 dB HL D = 5'	16.42 (2.44)	12.14 (2.67)

Note: i) Figures in parenthesis indicate standard deviation

ii) D indicates duration of exposure in minutes.

Table-8: Significance of difference (Wilcoxon Matched Pair Signed Ranks Test)

Expt.I and Expt.II	TTSo	T = 19, N = 10, P >0.01	NS
	TTS_2	T = 35, N = 11, P >0.01	NS
Expt.III and Expt.IV	TTSo	T = 6, N = 5, P >0.01	NS
	TTS_2	T = 0, N = 5, P <0.01	S
Expt.V and Expt.VI	TTSo	T = 0, N = 6, P <.0.01	S
	TTS_2	T =-2.5,N = 5, P >0.01	NS

Note: NS = Not significant, P > 0 .01

S = Significant, P < 0.01

Table-9: Experimental Paradigms (Cody and Johnstone - 1982)

n, the number of animals used for each paradigm: the total number used for the study was 34.

Experiment	Ipsilateral exposure	Contralateral exposure	Average maximum ipsilateral N_1 threshold loss (±SD).
Control (n=9)	10 KHz 107 dB SPL	none	38.77 ± 2.77 dB
A (n=10)	10 KHz 107 dB SPL	10 KHz 80 dB SPL	26.10 ± 4.30 dB
B (n=5)	10 KHz 107 dB SPL	7 KHz 80 dB SPL	38.25 ± 2.06 dB
C (n=5)	10 KHz 107 dB SPL	14 KHz 80 dB SPL	37.80 ± 2.48 dB
D (n=5)	10 KHz 107 dB SPL	10 KHz 80 dB SPL	38.80 ± 1.92 dB
	1 mg/kg strychnine		

Significantly different from the control N_1 Threshold loss at the 5% level (Students t-test)

SUMMARY AND CONCLUSIONS

Pratt et al (1978) have shown that if the TTS in a subject is about 20 dB 'reduced synaptic efficacy' may be responsible for the TTS and that the dysfunction of the hair cells may be ruled out. Their conclusion was based on the fact that they did not observe reduction in cochlear microphonic although there was TTS of about 20 dB.

Cody and Johnstone (1982) have established that the action of the efferent system innervating the outer hair cells increases the sensitivity of the auditory neurons. The increase in the AP (action potential) response during bilateral stimulation (i.e. in presence of efferent action) may be explained in terms of recycling of the released neuro-transmitter (Fex et al, 1982).

Vyasamurthy (1982, 1985) has reported that the ESIOHCs is responsible for 'loudness gain' during adaptation. He has cited the following studies in support of it: Spoendlin 1975, Cody and Johnstone 1982, Gerken 1984, Fex et al 1982, Comis and Whitfield 1968, Hoffmann et al 1983, Pickles 1982, Stopp et al 1983.

The present study was aimed at investigating (1) whether the ESIOHCs increases the sensitivity of the auditory neurons

in human subjects as Cody and Johnstone (1982) have reported. (2) whether the efferent action has any role in changing the temporary dysfunction of hair cells caused by fatiguing stimulus.

The Beltone 200-C audiometer with TDH-39 earphone and MX-41/AR circum aural cushion, calibrated according to the specifications given by ANSI 1969 was used for the study. Twenty one males and ten females normal subjects in age ranging from 17 years to 25 years were used in this study. Subjects were divided into 3 groups viz., A, B, C. A, B and C groups had 17, 7 and 7 subjects respectively. Experiments I and II were carried out using group A; experiments III and IV were carried out using group B and experiments V and VI were carried out using group C. TTSo, TTS_2 was determined in all experiments.

The results of the present study show that comparison of TTSo obtained in experiments I and II with the TTSo values obtained in experiments III and IV are greater. This shows that the high frequency fatiguing stimulus produces more TTS than a relatively low frequency stimulus (i.e. 4 KHz stimulus produced more TTS than 2 KHz stimulus).

Comparison of TTSo values obtained in experiments III and IV with the TTSo values obtained in experiments

V and VI shows that there is significant ditference between the TTSo values obtained in experiments V and VI, whereas there is no difference between TTSo values obtained in experiments III and IV. These results can be explained in terms of efferent action.

Pratt et al (1978) have reported that TTS of 20 dB in normal hearing subjects may be due to 'reduced synaptic efficacy'. TTSo of experiment VI was significantly less than the TTSo of experiment V i.e. in the presence of contralateral stimulation (efferent action - See Cody and Johnstone, 1982) the TTS obtained was less. In other words in experiment VI there was efferent action (due to contralateral stimulation) and there was no efferent action in experiment V (Monaural stimulation). From the results of the present study (especially the results of experiments V and VI) it is clear that due to efferent action the TTS reduces. This is in agreement with the findings of Cody and Johnstone (1982). Cody and Johnstone (1982) have established (see table-9) that the TTS reduces in the presence of contralateral stimulation.

Regarding the mechanism of efferent action reducing the TTS, it is quite probable that the efferent action increases the synaptic efficacy, as a result, the TTS reduces. The same explanation may hold good in the study

reported by Cody and Johnstone (1982). Thus the explanation for the reduction in TTS, in experiment VI, is likely due to increase in the synaptic efficacy as a result of efferent action.

The failure to observe reduction in TTSo in experiment IV (when compared to experiment III) may be due to temporary dysfunction of hair cells. Actually the present study shows more TTS in experiments III and TV than in experiments V and VI. Since dysfunction of the hair cells is responsible for TTS observed in experiments III and IV, the efferent action (which mainly changes the synaptic efficacy) might not have been effective in reducing the TTS.

Thus, it can be concluded that the efferent action changes the synaptic efficacy without bringing much changes in the dysfunction of hair cells.

BIBLIOGRAPHY

Benitz. L.D,,Eldredge.D.H,,and Templer. J.W,(1972)- Temporary Threshold Shifts in Chinchilla. Electrophysiological Correlates. J.Acoust. Soc.Am.52. 1115-1123.

Bohne, B.A. (1976)- Mechanisms of Noise damage in the inner ear. In Effects of Noise on Hearing (1) Henderson, R.P., Hamernik, D.S., D.S.Dosanth and J.H.Mills eds), pp 41-68. Raven, New York.

*Burgeat, M., Burgeat-Menguy.C, and Lehmann, R (1963)- Etudedela transmission despressions acoustiams du milieu aerien aux liquides cochleaires au moyen de sondes microphoniovues. Acoustica 13, No.6, 377-382.

Cody, A.R., Johnstone, B.M (1982) Temporary Threshold shift modified by binaural acoustic stimulation. Hearing Res. 6, 199-205.

Crane, H.D. (1983)- IHC-TM connect disconnect and mechanical interaction among IHCs, OHCs and TM. In Hearing Research and Theory (J.V. Tobias and E.D.Schubert, eds) Vol.11., pp (126-167). Academic Press, New York.

Davis, H. et al (1953)- Acoustic Trauma in the guinea-pig. J.Acoust.Soc.Am. 25, 1180-1189.

Davis, H. (1983)- An active process in cochlear Mechanics. Hearing Res. 9, 79-90.

Dayal, S.V.(1972)- A study of crossed Olivo Cochlear bundle on adaptation of auditory action potentials. Laryngoscope. 82, 693-771.

Dolan, T.R., Ades, H.W., Bredberg, G. and Neff, W.D. (1975)- Inner ear damage and hearing loss after exposure to tones of high intensity. Actaotolaryngol (stock) 80, 343.

Drescher, D.G.and Eldredge, D.H.(1974)- Species differences in cochlear fatigue related to acoustics of outer and middle ears of guinea-pig and chinchilla. J.Acoust.Soc.Am.56, 929-934.

Dreshcher, D.G. (1976) Effects of temperature on Cochlear responses during and after exposure to noise. J.Acoust.Soc.Am. 59, 401-407.

Durrant, J.D. (1976)- Effects of noise on Cochlear Potentials. In effects of Noise on Hearing (D. Henderson, R.P, Hamernik, D.S.Dosanih, and J.H. Mills, eds), pp 179-197. Raven, New York.

Duvall, A.J., Sutherland, C.R., and Rhodes, V.T. (1969). Ultrastructural Changes in the Cochlear duct following mechanical disruption of the organ of corti. Ann.Otol.Rhinol., Laryngol. 78, 342-347.

Duvall, A.J., Ward, W.D., and Lauhala, K.E. (1974)-Stria Ultrastructure and vessel transport in acoustic trauma. Ann.Otol-Rhino-Laryngol. 83,498-514.

Eldredge, D.H., and Covell, W.P. (1958)A laboratory method for the study of acoustic trauma. Laryngoscope 68, 465-477.

Eldredge, D.H., Bilger, R.C., Davis, H., and Covell, W.P (1961) Factor analysis of cochlear injuries and changes in electrophysiological potentials following acoustic trauma in the guinea-pig. J.Acoust.Soc.Am. 33, 152-159.

Eldredge, D.H., Mills, J.H., and Bohne, B.A. (1973)-Anatomical, behavioral and electrophysiological observations on chinchillas after long exposures to noise. Adv.Oto-Rhino-Larynol. 20, 64-81.

Engebretson, A.M.,and Eldredge, D.H. (1968) Model for the nonlinear characteristics of cochlear potentials. J.Acoust.Soc.Am. 44, 548-554.

Engstrom, H. and Engstrom, B (1979) Structural Changes in the cochlea following overstimulation by Noise. Acta oto-laryngologica, Supplement. 360, 75-79.

Fernandez, C (1955) The effect of oxygen lack on cochlear potentials. Ann.Otol.,Rhinol.,Laryngol. 64, 1193-1202.

Fex, J. (1967)- Efferent inhibition in the Cochlea related to hair cell dc activity study of post synaptic activity of the crossed olivo cochlear fibres in the cat. J.Acoust.Soc.Am. 41, 666-675.

Fex, J., Altschuler, R.A., Wonthold, R.J., Parakkal, M.H (1982). Aspartate aminotransterase immunoreactivity in cochlea of guinea pig. Hearing Res. 7, 149-160

Guiot, J.M (1969). Temporary Threshold Shift following pulsed monaural and alternate Binaural exposure. J.Acoust.Soc.Am. 46, 1449-1451.

Harrison, R.V. and Evans, E.F. (1979) Some aspects of temporal coding by single cochlear fibres from regions of cochl ar hair cell degeneration in the guinea-pig. Arch-Oto-Rhino-Laryngol. 224, 71-78.

Hawkins, J.E., Jr. (1971). The role of Vasoconstriction in Noise induced hearing loss. Ann.Otol., Rhinol. Laryngol. 80, 903-913.

Hawkins, J.E., Jr. (1976). Experimental noise deafness: Recollections and ruminations. In Hearing and Davis (S.K.Hirsh, D.H.Eldredge, I.J.Hirsh and S.R.Silverman, eds), pp.73-78. Washington University Press, St.Louis, Missouri.

Hirsch, I.J. (1958) Monaural Temporary Threshold Shift following monaural and binaural exposures. J. Acoust.Soc.Amer., 38, 121-125.

Humes, L.E. (1978) The aural overload test: Twenty years later. J.Speech Hear.Dis. 43, 34-46.

Karlovich, R.S., Abbs, N.H. and Latermann, B.F. (1972). Influence of the acoustic reflex in Temporary Threshold Shift from 1 and 4 Kc/sec. Continuous pure tones. J.Aud.Res. 12(1), 67-70.

Karlovich, R.S. and Wiley, T.J. (1974) Spectral and Temporal Parameters of contralateral signals altering temporary threshold shift. J.Acoust.Soc.Am. 17, 41-50.

Kemp, D.T. (1979).Evidence of Mechanical nonlinearity and frequency selective wave amplification in the cochlea. Arch.Otol.Rhinol.Laryngol. 224, 37-45.

Lawrence, M., and Blanchard, C.L. (1954).Prediction of susceptibility to acoustic trauma by determination of the threshold of distortion. Ind.Med. Surg. 23, 193-200.

Lawrence, M., and Yantis, P.A. (1956a)- Onset and growth of aural harmonics in the over-load ear. J. Acoust.Soc.Am. 28, 852.

Lawrence, M., and Yantix, P.A. (1956b) Thresholds of overload in normal and pathological ears. Arch. Otolaryngol. 63, 67-77.

Lawrence, M., and Yantis, P.A. (1957) Individual differences in functional recovery and structural repair following overstimulation of the guinea-pig ear. Ann.Otol.Rhinol.Laryngol. 66, 595-621.

Lawrence, M (1958).Functional changes in inner ear deafness. Ann.Otol.Rhinol.Laryngol. 67, 802-823.

Lawrence, M., Gonzales, G., and Hawkins, J.E. (1967) Some Physiological factors in noise-induced hearing loss. Ann.Industr.Hyg.Assoc. J. 28, 425-430.

Legouix, J.P.,and Pierson, A (1973) Mechanism of the short-term post stimulatory depression of the Cochlear microphonics (hysteresis). J.Acoust.Soc.Am.54, 16-21.

Legouix, J.P. and Pierson, A (1974) Investigations on the source of whole nerve action potentials recorded from various places in the guinea-pig cochlea. J.Acoust.Soc.Am. 56, 1222-1225.

Legouix, J.P. and Pierson, A (1977) Increased fatigue of cochlear potentials after injection of Kcl solution in the Perilymph. Audiology 16, 453-461.

Legouix, J.P.and Pierson, A. (1978). Nonlinear mechanisms involved in the action of noise and some noxious agents of the inner ear. The proceedings of the third International Congress on Noise as Public Health Problem. American Speech-Lanchage Hearing Association, Rockville, Maryland.

Liberman, M.C. and Kiang, M.Y.S. (1978). Acoustic Trauma in cats: Cochlear Pathology and Auditory - nerve activity. Acta-Oto-Laryngologica, Supplement 358, 5-46.

Legouix, J.P., Teas, D.C., Beagley, H.A. and Remond, M.C.(1978). Relation between the waveform of the cochlear whole nerve action potential and its intensity function. Acta.Oto.Laryngol.85, 177-183.

Legouix, J.P., Pierson, A. and Minot, J.F.(1980). Relations between cochlear fatigue and the asymmetrical non-linearity of the cochlear potentials. Audiology, 19, 263-276.

Legouix, J.P. and Pierson, A.(1981). Electrophysiological correlates of cochlear fatigue and trauma. In Hearing Research and Theory (J.V.Tobias and E.P.Schubert, eds) Vol.1, pp 63-93. Academic Press, New York.

Luz, G.A. and Hodge, D.C.(1971). Recovery from impulse-noise induced TTS in monkeys and men: A descriptive model. J. Acoust.Soc.Am.49, 1770-1777.

Melnick, W(1967). Effects of two interaural phase conditions for binaural exposures on threshold shift. J.Acoust.Soc.Am. 42, 179.

Misrahy, G.A., Shinabarger, E.W. and Arnold, J.E.(1958b). Changes in cochlear endolymphatic oxygen availability, action potential and microphonics during and following asphyxia, hypoxia and exposure to loud sounds. J.Acoust.Soc.Am. 30, 701-704.

Nazneen, G.(1984). Ear Difference in Temporary Threshold Shift for Binaural Stimulation. An Independent Project for Master's Degree, University of Mysore.

Neely, S.T. and Kim, D.O (1983). An active cochlear model showing sharp tuning and high intensity. Hearing Res.9, 123-130.

Opheim, D. and Flottorp, G (1955). The aural harmonics in normal and pathological ears. Acta-Oto-Laryngol. 45, 513-531.

Perlman, H.B. and Kimura, R (1962). Cochlear blood flow in acoustic trauma. Acta-Oto-Laryngol. 54, 99-110.

Poche, L.B., Stockwell, C.W. and Ades, H.W. (1969). Cochlear hair cell damage in guinea-pigs after exposure to impulsive noise. J.Acoust.Soc.Am. 46, 947-951.

Pratt, H., Sohmer, H. and Barazani, N.(1978). Surface-Recorded cochlear Microphonics Potentials during Temporary Threshold shifts in man. Audiology. 17, 204-212.

Pugh, J.E., Horwitz, M.R. and Anderson, D.J. (1974). Cochlear electrical activity in noise-induced hearing loss. Arch.Otolaryngol. 100, 36-100.

Pye, A (1974). Acoustic trauma after double exposure in mammals. Audiology 13, 320-325.

Ramdolph and Gardner (1973),An interaural phase effect in binaural temporary threshold shift. J.Aud.Res. 13(2), 147-151.

Salvi, R.J., Hamernik, R.P. and Henderson, D.(1979).Auditory nerve activity and cochlear morphology after noise exposure. Arch.Oto.Rhino.Laryngol. 224, 117-124.

Shivashankar, N.R (1976). Temporary Threshold Shift following monaural and binaural exposure to high frequency pure tones, A dissertation for Master's degree, Univ. of Mysore.

Siegel, J.H., Kim, D.O. (1982). Efferent neural control of cochlear mechanics: Olivo cochlear bundle stimulation affects cochlear bio-mechanical non linearity. Hg. Res.6, 171-182.

Small, A.M. (1963), Auditory adaptation, in Modern Developments in Audiology, edited by J.Jerger, (Academic Press, New York).

Smoorenburg, G.F. and Van Hensden, E (1979). Effects of actte noise traumata on whole-nerve and single activity. Arch.Otol.Rhino.Laryngol. 224, 117-124.

Spoendlin, H. (1971), Primary structural changes in the organ of corti after acoustic over stimulation. Acta.Oto. Laryngol. 71, 166-176.

Stockwell, C.w., Ades, H.W., and Engstrom, H.(1969). Pattern of hair cell damage after intense auditory stimulation. Ann.Otol.Rhinol.Laryngol. 78, 1144.

Tasaki, I. and Fernandez, C.(1952).Modification of cochlear microphonics and action potentials by Kcl solutions and by direct current. J.Neurophysiol. 15, 497-512.

Thalmann, R.(1975). Biochemistry of the auditory system. In the Nervous System (D.B.Tower, ed.) Vol.3, pp.31-44. Reven, New York.

Tonndorf, J. and Brogan, F.A.(1952).Two Forms of change in Cochlear Microphonics: Parallel shift in stimulus Intensity and Truncation of Gradient curves, USAF Sch.Aviat.Med.Proj. 21-27-001, Rep.6, Columbia University, New York.

Tonndorf, J. and Brogan, F.A. (1955). Combined effect of sound and oxygen deprivation upon cochlear microphonics in guinea-pigs. Ann.Otol.Rhinol. Laryngol. 64, 392-405.

Vyasamurthy, M.N. (1977). An Objective Verification of Small's model of loudness adaptation, paper presented at the IX Annual Conference of Indian Speech and Hearing Association held at Bangalore.

Vyasamurthy, M.N. (1982). Objective Residual Monaural Loudness Adaptation - a new Concept Ph.D. thesis, University of Mysore.

Vyasamurthy, M.N.(1985),Models of the efferent mechanisms during auditory adaptation paper presented at the symposium on "Mechanisms of the efferent auditory system" held at Bombay.

Ward, W.D.(1965). Temporary Threshold Shift following monaural and binaural exposures. J.Acoust.Soc.Am. 38, 121-125.

Weiler, E.M. and Glass, E.B. (1979). Monaural balances and loudness coding during auditory adaptation. Brit.J.Audiol.13, 61-62.

Weiss, T.F. (1982). Bidirectional transduction in vertebrate hair cells: A mechanisms for couping mechanical and electrical process. Hearing Res.7, 353-360.

Wiederhold, M.L., Kiang, N.Y.S.(l970). Effects of electric stimulation of the crossed olivocochlear bundle on single auditory - nerve fibres in the cat. J. Acoust.Soc.Am. 48, 950-965.

Zurek, P.M.(1981). Spontaneous narrow band acoustic signals emitted by human ears. J.Acoust.Soc.Am. 69, 514-523.

Zwislocki, J.J.(1980).Theory of cochlear mechanies. Hearing Research. 2, 171-182.

Zwislocki, J.J. and Kletsky, E.J. (1982).What basilar membrane says about cochlear micro-mechanics. Am.J.Otolaryngol. 3, 48-52.

* Not referred