IPSILATERAL ADAPTATION AND CHANGES IN THRESHOLD

Reg. No. 8407

AN INDEPENDENT PROJECT WORK SUBMITTED IN PART
FULFILMENT FOR FIRST YEAR M.Sc.,
(SPEECH AND HEARING) TO THE
UNIVERSITY OF MYSORE

ALL INDIA INSTITUTE OF SPEECH & HEARING
MYSORE-570006

Dedicated to:

" R" family, each of whom
has taught me many things
in life.

CERTIFICATE

This is to certify that the Independent Project entitled "IPSILATERAL ADAPTATION AND CHANGES IN THRESHOLD" is the bonafide work in part fulfilment for First Year M.Sc., (Speech and Hearing) of the student with Reg.No. 8407

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CERTIFICATE

This is to certify that the Independent Project entitled "Ipsilateral Adaptation and Changes in Threshold" has been prepared under my supervision and guidance.

GUIDE

DECLARATION

This Independent Project entitled "IPSILATERAL ADAPTATION AND CHANGES IN THRESHOLD" is the result of my own study undertaken under the guidance of Dr.M.N.Vyasamurthy, Lecturer in Audiology, All India Institute of Speech & Hearing, Mysore and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore

Dated: May 1985 Register No. 8407.

ACKNOWLEDGEMENT

I am grateful to

Dr.M.N.Vyasamurthy, my teacher and guide, who heard, explained and cleared all my doubts at all stages of my project with an amount of patience, only he has.

Dr.M.Nithya seelan, Director, All India Institute of Speech and Hearing, Mysore, for giving me this opportunity to work.

Dr.(Miss) S. Nikam, Professor and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore for permitting me to go ahead with the project.

Ms.Malini, M.S., Mr. Balakrishnan, E.P. and Mr. Dayalan, for the timely help rendered.

All my subjects, for making themselves available for the repeated testing.

Ms.Rajalakshmi R Gopal, for taking over the reins, once the write-up was done and converting those scribbled sheets of paper into this form.

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INTRODUCTION

INTRODUCTION

All our senses tend to become less responsive to stimuli after a certain duration of stimulation.

Adaptation is a phenomenon which characterizes all sensory systems. Its operational definition has customarily been in terms of a shift in some aspects of the intensive dimension of subjective experience, often in the threshold, brought about by previous stimulation of a sense organ by the same type of stimulus to determine the threshold. (Small, 1963).

"Auditory adaptation in its most general sense could be taken to refer to any change in the functional state of the auditory system brought about by an acoustic stimulus".

"Such a change in the auditory system's functional state manifest itself in a variety of ways. Indeed, it is possible to cite atleast five psycho-physical measures which undergo modification as a consequence of acoustic stimulation of sufficient magnitude. Along intensive dimension, the absolute threshold of hearing, the masked threshold, and at suprathreshold levels, the loudness of a sound, have all been shown to change. In addition shift in the pitch of an acoustic stimulus occur and, in the case of dichotically presented stimulus, a change in the apparent location results from prior presentation of an appropriate acoustic stimulus." (Small, 1963).

So, as noted above "Not all shifts in threshold are in the direction of decreased sensitivity. Under some conditions an enhancement of detectability may be observed (Ward, 1973). Hughes (1954) called this increased responsiveness as "immediate sensitization."

Sensitization or facilitation may be defined as the improvement in the threshold of hearing as a result of continued auditory stimulation.

Various investigators have studied the phenomenon of sensitization in the past (Hughes, 1954; Hirsh and Bilger, 1955; Kopra, 1954; Lightfoot, 1955; Jerger, 1955, 1956; Spieth and Trittipol, 1958; Ward, Glorig and Sklar, 1958, 1959; Moore, 1968; Comis and Whitfield, 1968; Noffsinger and Tillman, 1970; Noffsinger and Olsen, 1970; Spoendlin, 1975; Cody and Johnstone, 1982; Fex, et al, 1982; Vyasamurthy, 1977; Pickles, 1982; Hoffmanne et al, 1983; Stopp et al., 1983; and Gerken, 1984, etc).

Moore (1970) reported two different types of sensitization by name "sustained sensitization" following exposure to low intensity stimulation and which may be related to the density of functional receptor elements in the region stimulated and a transitory type that required exposure to moderately intense stimulation and which apparently occured only when two regions of differing sensitivity were stimulated simultaneously in the auditory system; sustained sensitization appeared in both the ipsilateral and contralateral ears, transitory sensitization occured only in ipsilateral ear.

Using a new method (Vyasamurthy, 1977) of measuring adaptation, data were collected on normal hearing adults. The new method makes use of the magnitude of the acoustic reflex as a measure of loudness perceived. The obtained data enabled Vyasamurthy to propose a revised model of adaptation.

The revised model of adaptation answers most of the controversies prevailing in the area of auditory adaptation. It provides possible answers to the following: (i) Asymptotic adaptation; (ii) Perstimulatory adaptation and levelling off of adaptation; (iii) The discrepancy observed by Weiler and Glass(1979) while verifying Small's model (1963) using Monaural Heterophonic technique and (iv) The controversy whether adaptation is real or not.

Vyasamurthy (1985) cites the following studies in support of loudness gain: The assumption that the action of the ESIOHCs is to increase the loudness of the post-adapted test-tone is supported by many studies - Spoendlin, 1975; Cody and Johnstone, 1982; Gerken, 1984; Fex, et al, 1982; Comis and Whitfield, 1968; Hoffmanne, et al, 1983; and Stopp, et al, 1983.

The present study was aimed at studying sensitization in the test ear, when the same ear is continuously exposed to a puretone for 7 minutes, at three levels viz. 20 dB SL, 40 dB SL and 60 dB SL. The study was also designed to study the effect of frequency on sensitization.

Hypothesis of the study:

The present study was undertaken to verify the following null hypothesis:

"There is no significant difference between the thresholds obtained in the test ear in the Conditions A and B".

Condition -A: Threshold for pulsed tone obtained in the test ear in the presence of a pure tone at 20 dB SL/40 dB SL/ 60 dB SL ipsilaterally (See Figure-1).

Condition-B: Threshold for pulsed tone obtained in the test ear at the end of 7 minutes when the same ear is being continuously exposed beyond 7 minutes to a pure tone at 20 dB SL/40 dB SL/ 60 dB SL (See Figure-l).

Where F_1 was 500 Hz, l000Hz, 2000Hz and 4000Hz in each subject and X dB SL = 20 dB for group A, 40 dB for group B and 60 dB for group C.

Brief Plan of the study:

15 subjects were divided into 3 groups viz. A, B, and C of 5 subjects each.

Group-A was tested at 20 dB SL.

Group-B was tested at 40 dB SL.

Group-C was tested at 60 dB SL.

Each group was tested at all the four frequencies viz. 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

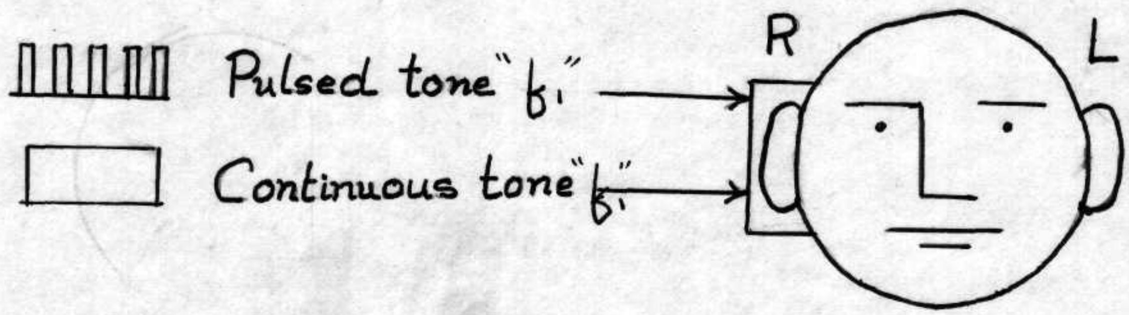
The threshold for pulsed tone was obtained in the test ear in the presence of continuous tone in the same ear at specified sensation - levels for each group viz, 20 dB SL for Group-A, 40 dB SL for Group-B and 60 dB SL for Group-C.

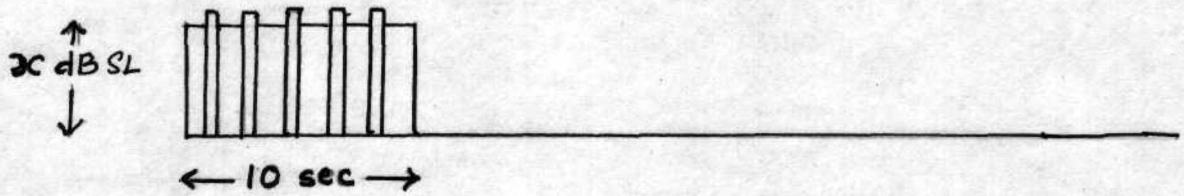
Then the test ear was exposed to continuous tone at the same sensation level (20/40/60 dB SL) for 7 minutes.

The threshold for pulsed tone was obtained in the test ear at the end of 7 minutes, (The continuous tone was not withdrawn after 7 minutes).

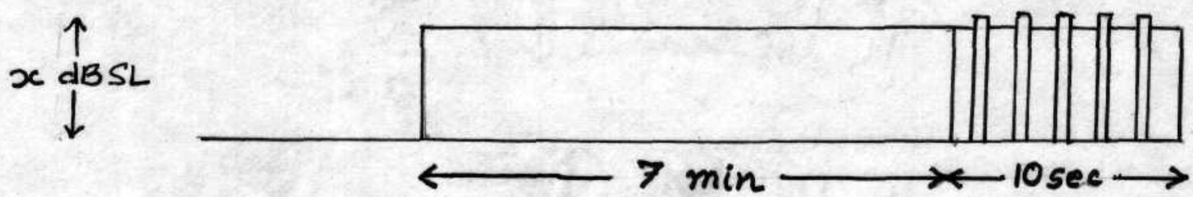
Sensitization was determined by subtracting the threshold obtained at the end of continuous stimulation for 7 minutes from threshold obtained prior to continuous stimulation.

Fig-1





Condition - A



Condition - B

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The first investigation of auditory adaptation of any sort was that of Dobve (1859), who noted during the course of a study of binaural beats, that if one ear was exposed for some time to a tuning fork, then binaural presentation of the same frequency would result the perception of a tone only at unexposed ear.

Fluegel in 1920 reported a series of experiments in which he systematically determined the effect of various parameters on both perstimulatory and post-stimulatory adaptation, carefully done studies which generally is limited only by fact that tuning fork alone was used as the major source of adapter and test tone. He discovered different relations subsequently confirmed by modern experimentation.

Fluegel (1920) even tried to determine the effect of central factor. The result implies a complicated central mechanism not a simple peripheral comparator.

Auditory adaptation is the change in the functional state of the auditory system brought by an acoustic stimulus or merely a reduction in apparent magnitude or an increase in true threshold. (Eliott end Fraser, 1970).

Ward, (1973) distinguishes the phenomena commonly included under adaptation in two different ways. . . . whether they are observed during or after exposure to the acoustic stimulus (concomittant or residual) and whether they require one ear (monaural) or two (binaural) for their measurement.

From the survey of literature and from the new data offered, Scharf concludes the following way:

1. A sound presented alone adapts only if it is below 30 dB SL.
2. High frequency pure tones adapt more than low frequency tones or / than noise, whether broad - band or narrow - band.
3. Steady sounds adapts more than modulated sounds and if the sound amplification is modulated sufficiently, adaptation may disappear altogether as when 2 tones beat together.
4. People differ widely with respect to the degree of adaptation they experience, although most people hear the loudness of a high frequency low-level decline by at least half within one minute, others report no change in loudness and still others report that the tone disappears.
5. No relation has been found however between the degree to which a person adapts and individual characteristics such as threshold, age and sex, although there is some evidence that children under 15 years adapt less than adults.
6. Free field testing may produce less adaptation than earphone listening.
7. Loudness adaptation may also be used by presenting a steady sound in 1 ear intermittent sound in the other.

8. The loudness of the steady sound decreases markedly over 2 or 3 minutes even at high levels where its loudness does not change when presented alone. This form of adaptation is ascribed to the overriding of the normal variation in excitatory input from a high level steady sound by the much greater variation from the intermittent sound, the role of interaural interaction and of lateralization. In this adaption is obscured especially since the intermittent sound may induce some adaptation when in the same ear as the steady sound.

Scharf himself states that some of the researchers disagree strongly with some of his interpretation of the data.

"Following stimulation, the auditory system can manifest increased sensitivity, decreased sensitivity, oscillation between increased and decreased sensitivity or no change in sensitivity". (Noffsinger and Tillman, 1970).

"Not all shifts in threshold are in the direction of decreased sensitivity. Under some conditions, an enhancement of detectability may be observed". (Ward, 1973).

Hughes (1954) called this increased responsiveness as immediate sensitization. He used this term to describe pure tone threshold sensitivity that was better than it had been before another pure tone stimulated the ear and that appeared as the first notable deviation from the post exposure threshold. Hughes demonstrated this phenomenon by employing low frequency stimulus tones at moderately intense levels at 80-100 dB. He found that immediate sensitization appeared only when the frequency of the test tone was lower than that of the exposure tone. The time course for these events feature immediate threshold sensitivity that grew to maximum size at about 30 seconds post exposure and then gradually disappeared by one minute. Hughes, found this interesting, since sensitizatlon for other exposure conditions usually occured as part of a multiphasic recovery process in which the sensitized threshold were preceded, succeeded, desensitized thresholds i.e. occured as part of an R-l; bounce,R-2 sequence (see Hirsh and Ward, 1952; Hirsh and Bilger, 1955; Hughes, 1954, Copra, 1954; Lightfoot, 1955; Jerger, 1955, 1956; Spieth and Trittipoe, 1958; Ward, Glorig and Sklar, 1958, 1959; Moore, 1968). Hughes found the phenomenon of immediate sensitization sufficiently unique to characterize it as perhaps resulting from some specific activity related to the auditory processing of low frequency signals (Noffsinger and Tillman, 1970).

In sensitization, greater sensitivity as measured by means of absolute threshold, from 1-2 minutes after exposure to the fatiguing stimulus than it did prior to any stimulation (Hirsh and Ward, 1952; Hughes, 1954). This phenomena has also been confirmed neurophysiologically.

Threshold for a tone can be affected in 3 major ways by exposing the ear to another tone.

1. Isolated sensitization.
2. Multiphasic behaviour (sensitization and desensitization - the bounce effect).
3. Isolated desensitization.

These changes seem dependent on at least following variables.

1. The frequency of the frequency relationship between the test and exposing stimuli.
2. The intensity of exposure stimulus.
3. The duration of exposure stimulus.
4. The condition applying during the exposure period, eg. whether the subject was required to track threshold during the exposure tone (Tr) or not (DN Tr).

To study these Noffsinger and Olsen (1970) examined the threshold sensitivity for train of 250 msec. test pulses (250, 1000, 4000 Hz) following exposure tones of various types. Associated with each test tone were 2 minute exposure tone of same frequency, half the frequency and twice the frequency as well as 2 additional tones one of whose frequency was considerably higher and one considerably lower than that of the test tone. Each exposure tone was presented at 4 intensity levels namely 20, 60, 85 and 105 dB. Both DN Tr and Tr procedures were employed.

The results of the experiment showed following facts:

1. Isolated auditory sensitization is a real phenomena. It can be demonstrated for both high and low frequency tones. Duration

of much sensitization ranged from 20-100 seconds. Sensitization that occurs later in the post exposure time course, usually following R-1 was also demonstrated in some experimental conditions. It usually attains maximum magnitude at about 1 minute post exposure, has a duration of 16-30 seconds and generally is of smaller magnitudes than more immediate sensitization.

2. If an ear is stimulated by a pure tone whose strength is gradually increased, the first noticeable post stimulation change in threshold for another pure tone in some instances is sensitization. Such sensitization will increase in magnitude and/or duration to a critical point and then decline with further increase in exposure tone strength. Following even stronger stimulation, desensitization will become apparent in post exposure thresholds, first as an initial threshold shift that rapidly declined (R-1) and may yield to sensitized threshold then as a multiphasic process containing R-1, a bounce and a second period of desensitization (R-2) and finally as a long lasting period of desensitization that is most aptly described as R-2 alone.

3. The sequence of post exposure events described above is initiated at lower exposure levels following tones whose frequency in lower or equal to that of the tilt tone than following those with higher frequencies. Given this distinction, decrease in the frequency, differential between the test and exposure tones has an effect similar to that produced by increasing the exposure tone intensity.

4. Continued threshold tracking of the test tone during exposure tone period usually produces more post exposure desensitization than is produced when the exposure tone is presented alone.

Sensitization and desensitization reflect the state of atleast partially separate physiological mechanism that are affected in different ways and for different periods of time by prolonged stimulation. One reasonable hypothesis is that sensitization mirrors a presynaptic electrical or electromechanical hyper-excitability, i.e. hyperpolarization and desensitization reflects a reduced post synaptic receptive capability.

There have also been some studies that indicated an enhanced sensitivity of the auditory system following exposure to low intensity stimuli (5-20 dB SL) short duration (5 ms - 10 sec) short recovery time (5 ms - 1.0 sec) (zwislocki, Pirroda and Rubin, 1959; Rubin, 1960).

This phenomena was termed facilitation by Rubin to distinguish it from sensitization as described by Hughes (1954) which is elicited by relatively long exposure duration and more intensive stimulation.

From Hughes report (1954) it is known that a greater amount of transitory sensitization occured at 500 Hz than at higher frequencies (eg. 1000 Hz). Hughes suggested that the reason may be the higher frequencies were more effective in producing a positive TTS and that this may have been interacting with sensitization so as to produce an apparent lessing of the amount of sensitization.

At 1000 Hz the ipsilateral ear showed the typical transitory sensitization recovery function, with secondary rise above the reference threshold level.

Thomas J.Moore (1940) reported 2 different types of sensitization.

1. A sustained type that was elicited following exposure to low intensity stimulation and which may be related to the density of functional receptor elements in the region stimulated.

2. A transitory type that required exposure to moderately intense stimulation and which apparently occured only when 2 regions of different sensitivity were stimulated simultaneously in the auditory system; sustained sensitization in both the ipsilateral and contralateral ears, transitory sensitization occured only in the ipsilateral ear.

In experiments involving an increase of the intensity of the exposure tone, on successive runs, an effect can be seen earlier for test frequencies below the exposure frequencies than for those above it. Noffsinger and Tillman (1970) stimulated human ears by 3 minutes 65-90 dB SPL continuous tones and post exposure thresholds for tones of lesser frequency were examined. In most cases such procedure allowed demonstration of auditory sensitization that was not preceded or succeeded by desensitization and then ran its course in the first post exposure minute. Such sensitization was noted at 200 Hz following certain 500 Hz exposure tones and at 2000 Hz following certain 3000 Hz exposure tones.

There appears to be greater sensitization to a continuous test tone (Hughes, 1954) than to an interrupted one (Noffsinger and Tillman, 1970).

Sensitization is not restricted to the ear exposed. Hughes, (1954) using a special apparatus to produce an interaural attenuation of 85 dB found nearly as much sensitization at 500Hz after stimulation by a 500 Hz, 85 dB SPL tone in the contralateral ear as after ipsilateral stimulation. Noffsinger and Tillman (1970) have also demonstrated this.

The transitory type of sensitization found occured only in the ipsilateral ear and only after moderate to moderately intense stimulus. This type of sensitizationiis more fully documented in both the psychophysiological (Hirsh and Ward, 1952) and neurophysiological (Rosenblith, Gollanbose and Hirsh, 1950; Hughes and Rosenblith, 1957) literature.

"Using a new method (Vyasamurthy, 1977) of measuring adaptation, data were collected on normal hearing adults. The new method makes use of the magnitude of the acoustic reflex as a measure of loudness perceived. The obtained data enabled the author to propose a revised model of adaptation. The details regarding the new method and the revised model are available elsewhere (Vyasamurthy, 1982, 1984a, 1984b).

In essence, the revised model assumes that there are three types of adapted neural units viz, stable (a) and unstable (a_1 and a) adapted neural units, 'a' units may originate from the place of maximal stimulation of the basilar membrane or they may originate from the neural units of the characteristic frequency (frequency of the adapting stimulus). a_1. and a_2 units may originate from the actions of the efferent system innervating the inner hair cells (ESIIHCs) and the efferent system innervating the outer hair cells (ESIOHCs) respectively, 'a' and a_1 units decrease the loudness of the post adapted test tone, where as a_2 units increase the loudness of the post adapted test tone i.e. 'a' and a_1 units are responsible for loudness loss and a_2 units are responsible for loudness gain. The efferent action/s ceases, the moment, the post adapted test tone at an intensity higher than the adapting Intensity is presented to the adapting ear.

The revised model of adaptation answers most of the controversies which are prevailing in the area of auditory adaptation. It provides possible answers to the following: (1) asymptotic adaptation, (2) perstimulatory adaptation and levelling off of adaptation (3) the discrepancy observed by Weiler and Glass (1979) while verifying small's model (1963) using Monaural heterophonic technique and (4) the controversy whether adaptation is real or not real.

LOUDNESS GAIN:

The assumption that the action of the ESIOHCs is to increase the loudness of the post adapted test tone, is supported by many 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 "enhancement". 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 neurotransmitter using Aspartate amino transferase (AATase). Interestingly, they have found the AATase like immuno reactivity in the

Medial system of efferents but not in the lateral system. (5) Comis and Whitfield (1968) report that the acetylcholine (neuro-transmitter of ESIOHCs) is an excitatory neurotransmitter. (6) Hoffmanne et al., (1983) have detected enkephalin like peptides (putative neuro-active substances) 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.

LOUDNESS LOSS:

The assumption that the ESIIHCs (and 'a' units) is responsible for decreasing the loudness of the post adapted test tone, is supported by many studied: (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 neurotransmitter of the efferent auditory system which produces inhibition (Pickles, 1982).

AN ACTIVE MECHANISM:

Of recent, many investigators (Kemp, 1978, 1979; ZwislockI, 1980, Zurek, 1981; Zwislocki and Kletsky, 1982; Neely and Kim, 1983, Davis, 1983) have suggested that there is an active mechanism in the cochlea. Siegal and Kim (1982) state that the active mechanism is controlled by the central nervous system through the activity of the efferent synapses 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 spontaneous acoustic emissions. While discussing the functions of the efferent auditory system/s, Crane (1983) comments: "OHC afferents are part of the servo-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 sources of efferent excitation".

ELECTRICAL STIMULATION OF COCB:

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 evidence for inhibitory role of the efferent innervation of the cochlea pertains to IHC system. . . . . function of ESIOHCs is yet to be known. Presnece of efferent innervation of the verstibular receptors suggests a general role for all labyrinthine efferent pathways such as the enhancement of sensitivity of the various receptors". (Bodian, 1983).

"Electrical stimulation of COCB increases the damping of the cochlear partition", (Siegel and Kim, 1982, Mountain, 1980).

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). This increase in the damping in the BM (Basilar membrane) (i.e. when 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).

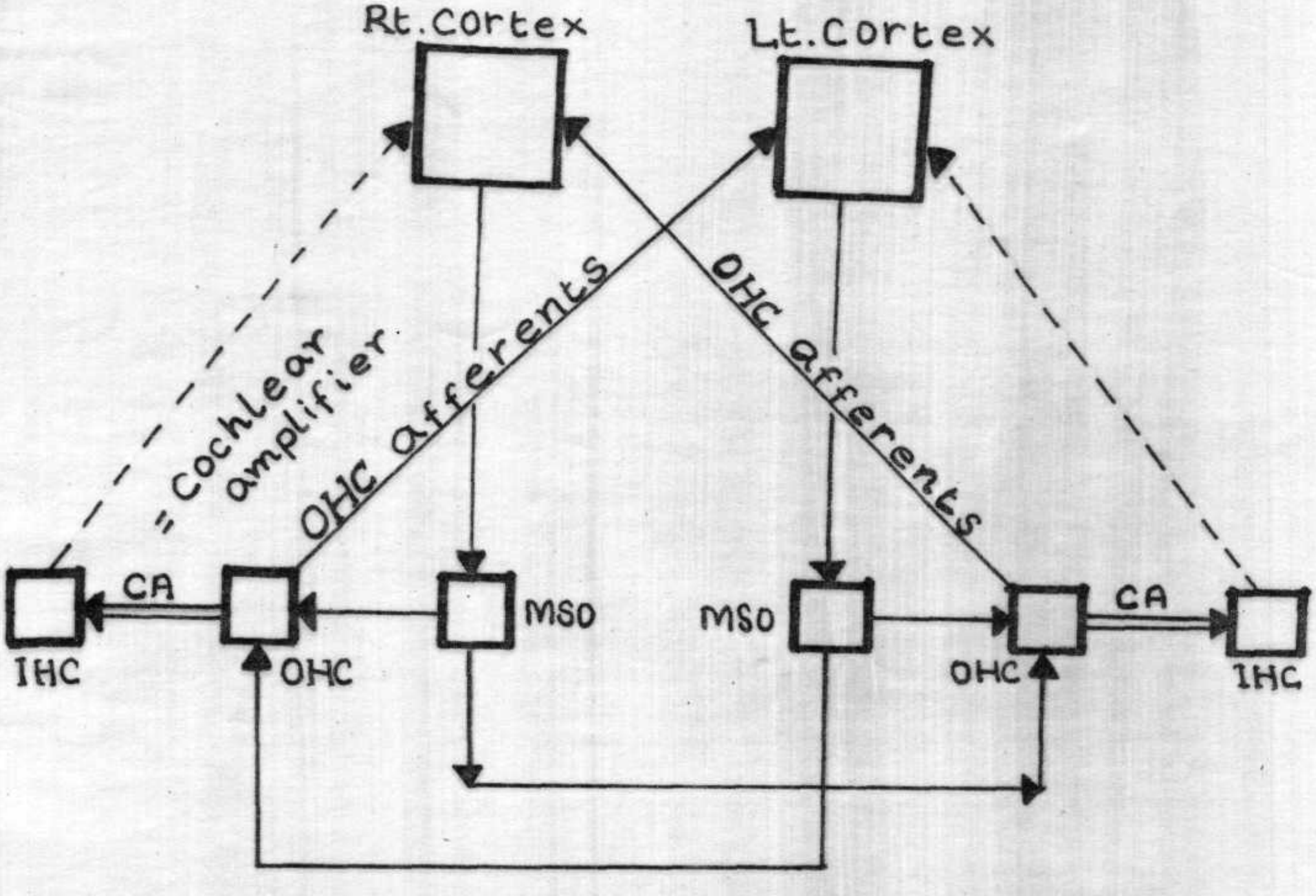
MODELS OF THE EFFERENT MECHANISMS:

By putting all the above pieces of information, the following neural models (Fig. 1 and 2) of the efferent mechanisms during auditory adaptation have been proposed.

The model (Fig.3) suggests 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 (Fig.4) 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 efferents to the IHCs synapse with the afferent dendrites of IHCs. (Note: In Fig.3 dashed line means not important for loudness gain; cochlear amplifier (CA) refers to active mechanism - see Davis, 1983).

He concludes in the following way:

In the light of the recent developments in auditory physiology, the neural models of the efferent mechanisms during auditory adaptation have been proposed. The two efferent auditory systems - MSO and LSO, may be responsible for loudness gain and loudness loss, during auditory adaptation, respectively", (Vyasamurthy, 1980).

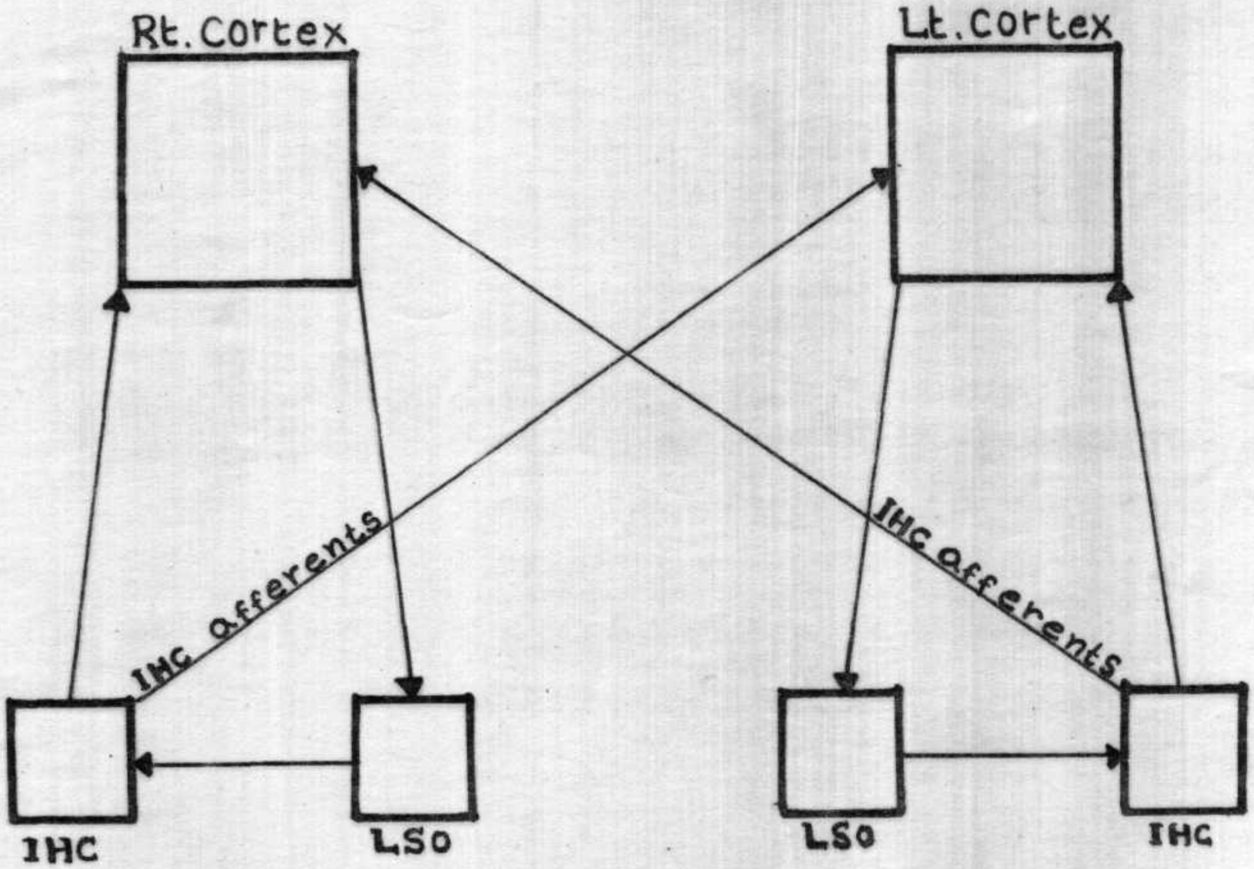


Model of the Efferent Mechanisms

(Loudness gain)

during auditory adaptation.

FIG 3.



Model of the Efferent Mechanisms

(Loudness Loss)

during auditory adaptation

FIG 4.

METHODOLOGY

METHODOLOGY

Subjects:

15 normal hearing subjects (20 dB HL ANSI 1969), 6 males and 9 females with age range of 17 to 25 years were selected for this study. None of the subjects had any history of ear-ache or any other complaints.

Equipment used:-

A dual channel diagnostic audiometer Beltone 200-C (Figure-2) with TDH-39 earphone placed in MX-41/AR cushion was used for testing. This audiometer allowed testing of frequencies from 125 Hz to 8000 Hz and Hearing level range from -10 to 110 dB HL for pure tones.

Rise time - 0.02 - 0.1 sec.

Decay time - 0.005 - 0.1 sec.

'On' time - 0.3 sec ± 10%.

'Off' time - 0.3 sec ± 10%.

Stop Watch:

An 'Omega' stop watch with second hand and minute hand was used for testing. Calibration procedure used: The audiometer used in this study was calibrated both before and after the study according to the guidelines given by Wilber (1978). The output and linearity of both frequency and intensity was found to be within permissible limits (ANSI 1969). All these measurements were done in a sound treated room of All India Institute of Speech and Hearing, Mysore.

Environment:

The audiometric tests were performed in a sound treated two-room situation. The subject was seated comfortably on a chair in such a way that the control panel of the audiometer was out of his/her line of vision. Also throughout the testing period the lighting in the subjects room was kept bright and in the tester's room was dull so that the subject could not make out the tester's finer eye-movements (if any) clearly.

Instructions:

"I am going to test you in the following way:-

- I shall take your threshold for pulse tone at a particular frequency.
- I shall again take your threshold for pulse tone, but this time in presence of a continuous tone of the same frequency. Respond only for the pulse tones.
- Next I will present the continuous tone for 7 minutes continuously.
- At the end of 7 minutes, I will again find your threshold for pulse tone in the presence of the continuous tone.
- I will test your Right ear only".

Since the subjects chosen were students of All India Institute of Speech and Hearing, they did not have any problem in understanding the instructions. Nevertheless the subjects were asked to repeat the instructions the in order to confirm that they had understood the instructions properly.

Procedure:

The 15 subjects were divided into 3 groups A, B and C of 5 subjects each.

Only the Right ear of all the subject was tested (i.e. Right ear was the test ear in all the subjects).

All the subjects were tested at 4 frequencies viz. 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz.

Only one frequency was tested per day for each subject and in this way all the subjects were tested on 4 consecutive days. This procedure was adopted to rule out the possibility of any residual effect due to adaptation.

During testing, after giving instruction and making sure that the subject was seated comfortably, the following procedure was followed in all subjects.

1. The threshold of pulse tone was got using Hughson-Westlake procedure.

2. The subject was exposed to 20 dB SL/40 dB SL/60 dB SL continuous tone depending on whether he/she was in group-A, group-B or group-C respectively. This continuous tone was of the same frequency as that of the pulse-tone. In presence of this continuous tone, the threshold of the pulse-tone was got again. This was called "Condition-A" (Shown in Fig.l).

3. Next the subject was exposed to the continuous tone at an intensity equal to that used in step2, for 7 minutes.

4. Threshold for pulsed tone was obtained at the end of 7 minutes in the presence of the continuous tone (The continuous tone was not withdrawn after 7 minutes) in the test ear. This was called "Condition-B" (Figure-1).

Sensitization was calculated by subtracting the pulse tone threshold obtained in "Condition-B" from the pulse-tone threshold obtained in "Condition-A".

The obtained data were collected in the following data sheet.

Serial No.	Sensation level at which tested.	Frequency at which tested.	Pulse-tone threshold	Pulse-tone threshold in presence of continu-ous tone "Condition-A"	Post-adaptive pulse-tone threshold "Condition-B"	Improvement in threshold "A - B"

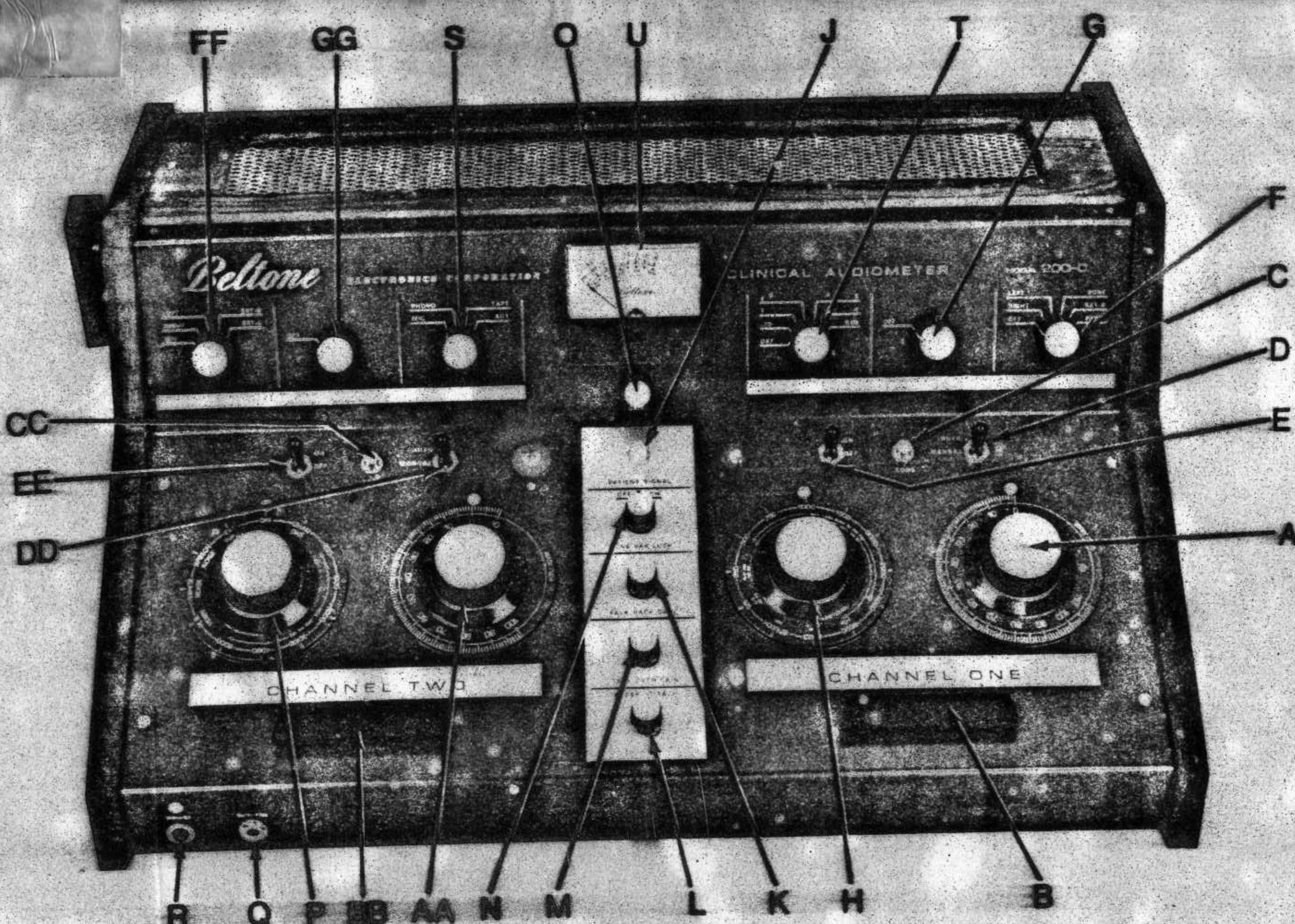


FIGURE 2.1 ILLUSTRATION, OPERATING CONTROLS

FIGURE - 2.

BELTONE 200-C

Expansion of the short forms given in Figure-2.

(A),	(A-A)	Output Attenuators.
(B),	(B-B)	Tone Interrupter.
(C),	(C-C)	Tone 'on' Lamp.
(F),	(F-F)	Output selector.
(J)		Patient signal lamp.
(E),	(E-E)	Tone Reversing switch.
(D),	(D-D)	Automatic/Manual Switch
(G),	(G-G)	Monitor Control.
(T)		SISI (Short Increment Sensitivity Index)
(S)	Speech	output.
(V)		VU (Volume Unit). ^
(N)		Tone Bar Lock.
(L)		Talk-Over switch.
(M)		Talk-Over Gain.
(K)		Talk-Back Gain.

(Ref: Beltone 200-C : Installation and Service Manual)

RESULTS AND DISCUSSION

RESULTS AND DISCUSSION

Tables-1, 2, 3 and 4 reveal the improvement in thresholds (sensitization) in dB for GroupA, Group-B, Group-C and mean and standard deviation of these three groups respectively.

The same has also been represented graphically in Graphs I, II, and III for the 3 levels of presentation namely 20, 40 and 60 dB SL.

From the tables and graphs it is obvious that there is improvement (sensitization) in the test ear (ipsilateral ear) after continuous stimulation by pure tones, i.e. on continuous ipsilateral stimulation of the test ear, the thresholds become better.

It is also clear from the tables that there is improvement in thresholds at all levels (20, 40 and 60 dB SL) and at all frequencies (500, 1000, 2000 and 4000 Hz).

The results of the present study reveal that the magnitude of sensitization at all frequencies tested show very little difference, i.e. the frequency of the adapting stimulus has no effect on magnitude of sensitization.

"The Wilcoxon matched-pairs signal-ranks test" (Siegel, 1956) was used to find out whether there is significant difference between the thresholds obtained in Condition-A and Condition-B.

The analysis of the data shows that there is significant difference at all levels of all frequencies at 0.01 level of significance.

And the magnitude of sensitization at 60 dB SL is more than that for either 20 or 40 dB SL.

The phenomenon of sensitization has been reported by many investigators; but the survey of literature shows that there is no study which has made use of the methodology adopted in the present study. Hence it may not be correct to compare the magnitude of the sensitization obtained in the present study with those reported by other investigators.

The present study shows that when an ear is adapted for 7 minutes or more using continuous pure-tone, the ipsilataral ear shows improvement in thresholds of hearing or shows sensitization.

In none of the subjects there was deterioration in threshold and in all of them there was improvement. It is commonly believed that continuous auditory stimulation decreases the sensitivity of the ear; but the present study is contrary to this belief. This shows that continuous auditory stimulation activates some facilitatory process.

In the revised model of adaptation (Vyasamurthy, 1982),loudness gain is expected in the ipsilateral ear due to efferent action. In this study, he speculates that 'a_2' units will be produced in the adapted ear (ipsilateral ear).

The revised model is based on the assumption that the efferent system innervating the outer hair cells ESIOHCs (or MSO system) is responsible for loudness gain in the ipsilateral ear.

The neural model of the efferent mechanism for loudness gain has also been proposed (Vyasamurthy, 1982) (See Figures 3 and 4). According to this model, the facilitatory process may be viewed in terms of synaptic efficacy brought about by the ESIOHCs.

Fex, et al (1982) have suggested that the ESIOHCs may participate in recycling of released neuro-transmitters through AATase (Aspertate Amino Transferase) activation.

Additionally the release of "Enkephalin" like neuroactive substance (Fex, et al 1982) by the efferent system may also contribute to the sensitization observed in the present study.

So, based on the above results the null-hypothesis formulated at the beginning of the study may be rejected. It can be aid, that there is significant difference between the thresholds obtained in the test ear in the condition A and B.

The results of the present study thus support the revised model of adaptation.

Table-l : Showing improvement in thresholds in dB for Group-A (Adapting Stimulus Level:20 dB SL)

Subjects Group-A	Frequencies 500 Hz	1000 Hz	2000Hz	4000 Hz
A.1	10	5	5	5
A.2	0	5	5	5
A.3	10	10	5	10
A.4	0	5	5	5
A.5	5	5	5	5
Mean	5.0	6	5	6
6n	4.47	2.0	0	2.0
6n-l	5.0	2.24	0	2.25

Table-2: Showing improvement in thresholds in dB for Group-B (Adapting Stimulus Level: 40 dB SL)

Subjects	Frequencies			
Group-B	500 Hz	1000 Hz	2000 Hz	4000 Hz
B.1	5	5	5	5
B.2	10	10	5	5
B.3	5	5	5	5
B.4	0	0	0	5
B.5	5	5	5	5
Mean	5	5	4.0	5
n	3.16	3.16	2.0	0
n-1	3.54	3.54	2.24	0

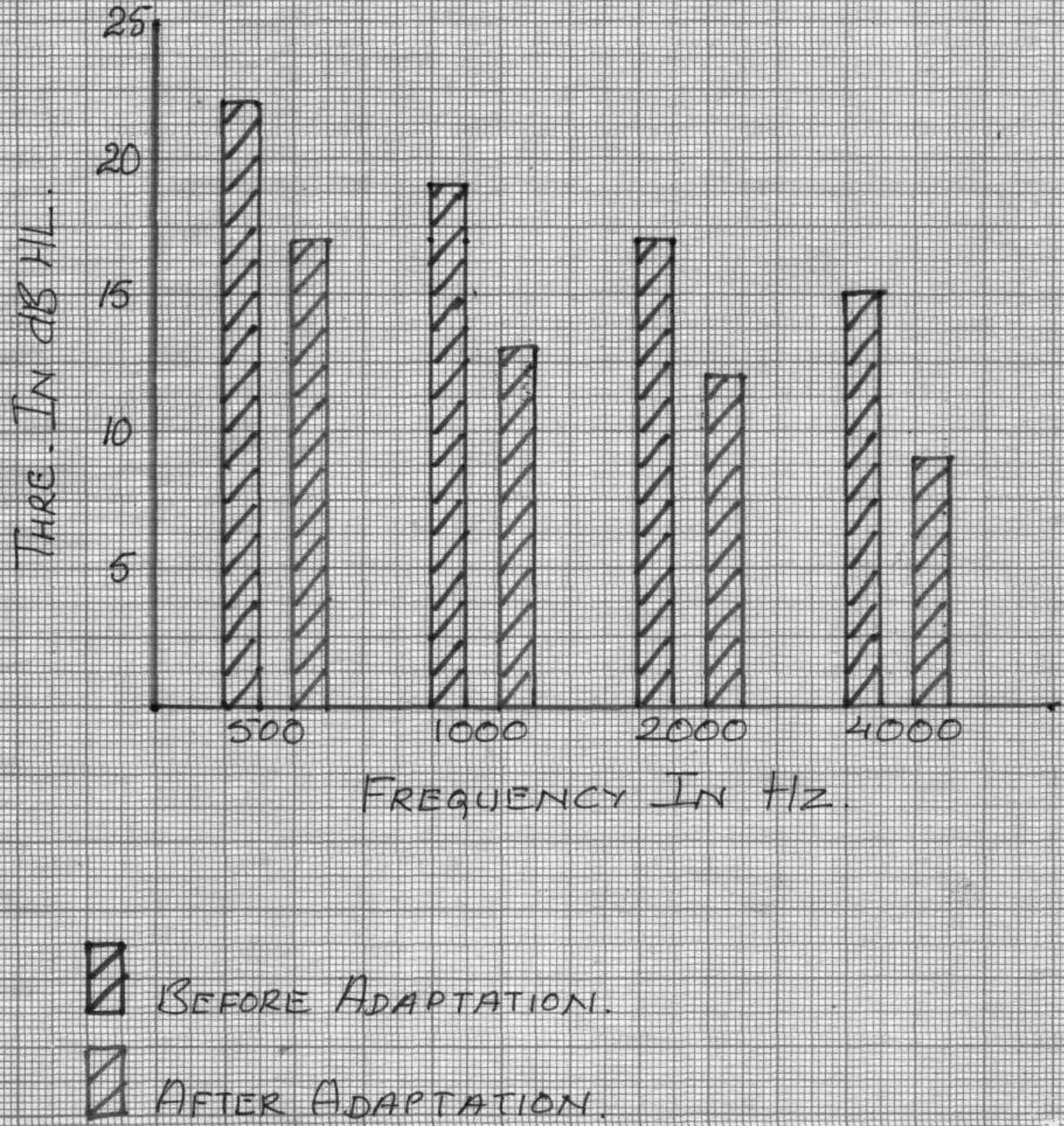
Table-3: Showing improvement in thresholds in dB for Group-C (Adapting Stimulus Level: 60 dB SL)

Subjects	Frequencies			
Group-C	500 Hz	l000Hz	2000Hz	4000Hz
C.1	10	5	5	5
C.2	10	10	5	5
C.3	10	5	10	5
C.4	10	5	10	5
C.5	10	15	5	10
Mean	10.0	8.0	7.0	6.0
6n	0	4.0	2.45	2.0
n-1	0	4.47	2.74	2.24

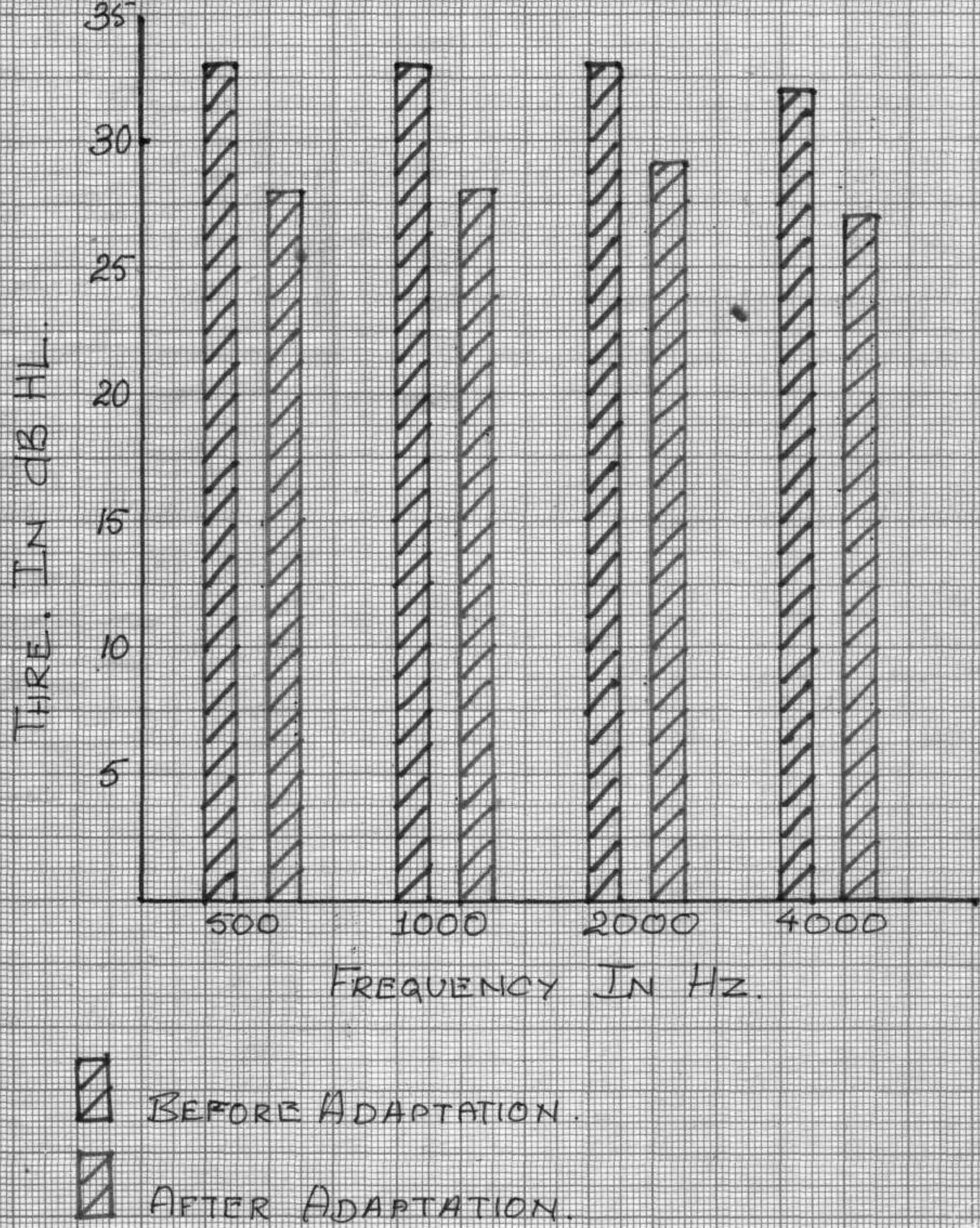
Table-4: Showing mean improvement in thresholds in dB for each group, group Mean and Standard Deviation for all the groups.

Means for different groups.	Frequencies			
	500 Hz	1000HZ	2000Hz	4000Hz
A 20 dB SL	5	6	5	6
B 40 dB SL	5	5	4	5
C 60 dB SL	10	8	7	6
Grand Mean	6.67	6.33	5.33	5.67
6n	3.94	3.40	2.21	1.70
6n-l	4.08	3.52	2.29	1.76

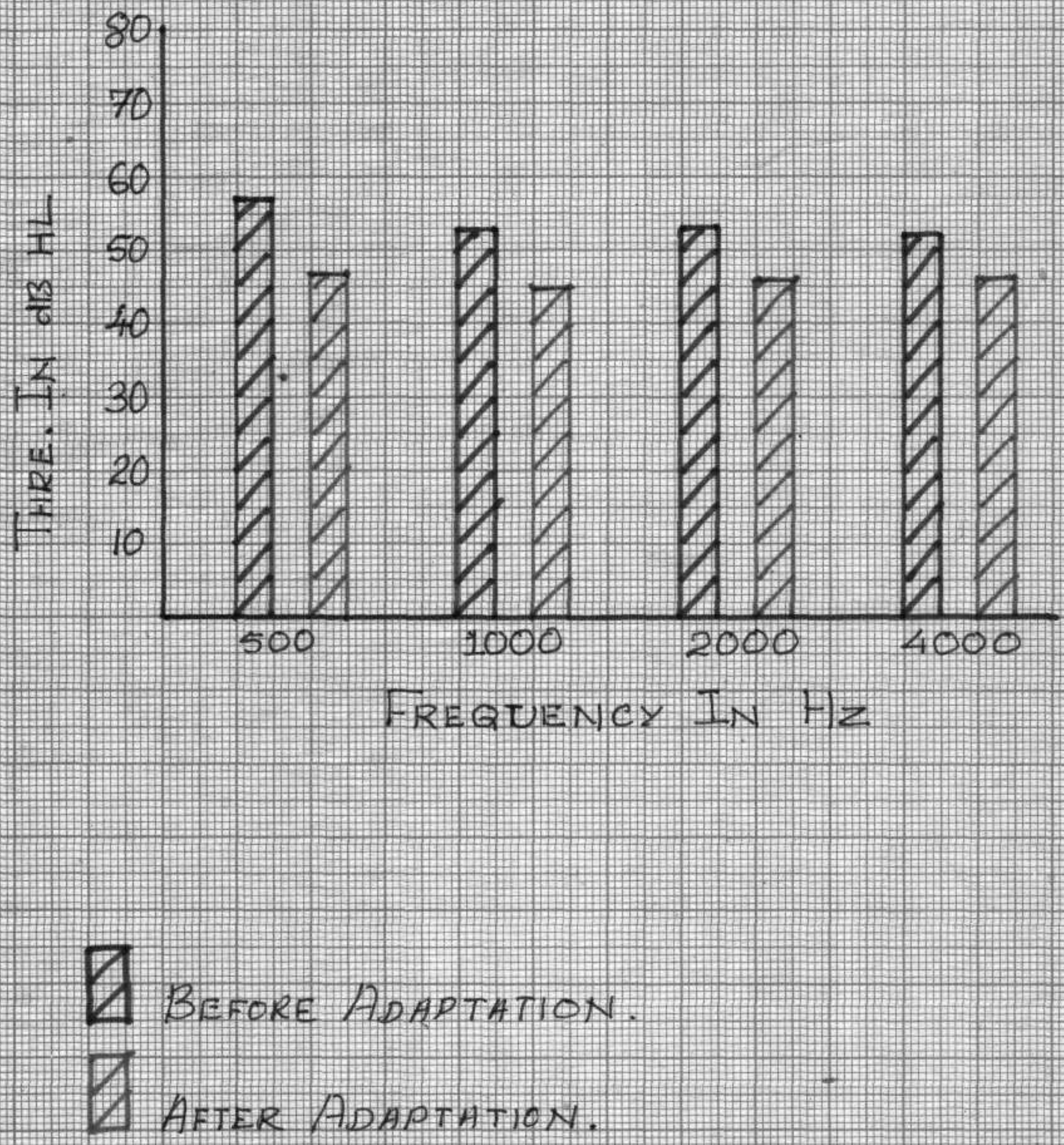
GRAPH I: SHOWING IMPROVEMENT IN THRESHOLDS FOR GROUP A. [20 dB SL]



GRAPH II: SHOWING IMPROVEMENT IN THRESHOLDS FOR GROUP B. [40 dB SL]



GRAPH III: SHOWING IMPROVEMENT IN THRESHOLDS FOR GROUP C [60 dB SL]



SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The phenomenon of sensitization has been studied by various investigators in the past.

The present study was carried out the study the effect of ipsilateral continuous presentation of pure-tones, at different intensity levels, on sensitization in the same ear.

15 normal hearing subjects (6 males and 9 females) in the age range from 17 to 25 years were divided to 3 groups A, B and C of 5 subjects each. Each subject was tested at 4 frequencies (500, 1000, 2000 and 4000 Hz) at one level each i.e. 20 dB SL (Group-A), 40 dB SL (Group-B) and 60 dB SL (Group-C) for 7 minutes continuously. One frequency was tested for a subject per day to rule out residual effect. Thresholds for pulsed tone were determined in the presence of continuous tone, both before and after adapting the ear. Only right ear of all subjects was tested.

Sensitization was calculated by subtracting the pulsed tone threshold in presence of continuous tone after 7 minutes of adaptation (Condition-B) from the pulsed tone threshold obtained in presence of continuous tone before adapting the ear (Condition-A).

From the results obtained (See tables 1, 2, 3, 4 and Graphs, I,II, III) the following conclusions were drawn:

1. There is significant difference between the thresholds obtained

in Condition-A and Condition-B at all levels, Viz.20, 40 dB SL and 60 dB SL for all frequencies at 0.01 level of significance; thus the null-hypothesis has been rejected.

2. The magnitude of sensitization show very little differences among the frequencies tested (500, 1000, 2000 and 4000 Hz); i.e. the frequency of the adapting stimulus has no effect on the magnitude of sensitization.

3. Magnitude of sensitization at 60 dB SL is more than that for either 20 dB SL or 40 dB SL.

Though many other investigators have studied sensitization, the result of this study can not be compared with them as the methodology differs.

Explanation for the above results may be taken from studies of Fex, et al (1982) who suggests that (1) ESIOHCs may participate in recycling of released neuro-transmitters through AATase and (2) the release of "Enkephalin" like substance by the efferent system.

Further support for this comes from the revised model (Vyasamurthy, 1982) which assumes that ESIOHCs is responsible for loudness gain in ipsilateral ear.

Also a neural model of the efferent mechanism is proposed (Vyasamurthy, 1985) according to which facilitatory process may be viewed in terms of synaptic efficacy brought about by the ESIOHCs.

The results of the above study thus supports the revised model of adaptation.

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