SENSITIZATION AT FREQUENCY LOWER THAT THE STIMULUS FREQUENCY

Register No. 8502

An Independent project submitted as part fulfilment for

First year M.Sc. (Speech and Hearing)
to the University of Mysore.

All India Institute of Speech & Hearing
MYSORE-570006.

MAY—1986

CERTIFICATE

This is to certify that the Independent Project entitled "Sensitization at Frequency Lower Than the stimulus Frequency" is the bonafide work done in part fulfilment for First Year M.sc, (Speech and Hearing) of the student with Register No. 8502

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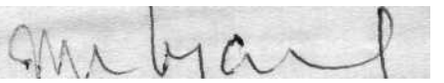
CERTIFICATE

This is to certify that the Independent Project entitled "Sensitization at Frequency Lower Than the Stimulus Frequency" has been prepared under my supervision and guidance.



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DECLARATION

This Independent Project entitled "Sensitization at Frequency Lower than the Stimulus Frequency" 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 - 570 006, and has not been submitted earlier at any other University for any other Diploma or degree.

Mysore Register No. 8502

Dated April 1986.

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I would like to thank my friends and classmates each of whom was of encouragement in one way or the other.

To Rajalakshmi R Gopal, for agreeing to bring it to this form at such a short notice.

And to all those who have helped me.

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INTRODUCTION

It has been consistently found in research studies that when the auditory system is continuously stimulated with a signal, a temporary threshold shift (TTS) occurs. This post exposure performance exhausts the permutations available; i.e. following stimulation, the auditory system can manifest increased sensitivity, decreased sensitivity, oscillation between increased and decreased sensitivity or no change in sensitivity.

Adaptation refers to "Any change in the functional state of the auditory system brought about by an acoustic stimulus. Such a change in the auditory systems functional state may manifest itself in a variety of ways. Along the intensive dimension, the absolute threshold of hearing has been shown to change" (Small, 1963). This variability in threshold, depends, atleast, in part on the type of stimulus used to excite the ear and on the post exposure time at which responsiveness is determined.

Sensitization is a term which is used generally to describe improvement in threshold consequent to auditory stimulation. Under certain conditions, stimulation increases the sensitivity of the neural system. This phenomenon referred to, as "sensitization" can be observed under certain conditions to affect not

only pure tone thresholds (Hughes 1954) and thresholds of action nerve potentials (Hughes and Rosenblith, 1957) but also thresholds of Acoustic Reflex (Simmons, 1960).

Increased responsiveness of the neural system after application of a tetanizing stimulus has been termed post tetanic potentiation (PTP) by Eccles (1953).

Specific to increase in behavioural threshold, the phenomenon of sensitization has been studied by various investigators in the past (Hughes, 1954, Noffsinger and Tillman, 1969, Noffsinger and Olsen, 1969, RajaniKanth, 1985, Ragini, 1985 and Sridhara, 1985). However, the phenomenon of sensitization has been proved by various other methods such as increase in ART (Chabot, 1977) and changes in physiological potentials (Benitz, 1972). (Cody and Jhonstone, 1982).

Hughes (1954) used the term immediate sensitization 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 noticeable deviation from the pre—exposure threshold." Hughes demonstrated this phenomenon by employing low frequency stimulating tones at moderately intense levels (80-100 dB SPL) for one minute. 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 featured an immediate threshold sensitization that grew to a maximum size at about 30 sec. post exposure and then

gradually disappeared by one minute. Hughes found immediate sensitization interesting since sensitisation for other exposure conditions usually occured as part of a multiphasic recovery process, in which the sensitized thresholds were preceded and succeded by desensitized threshold i.e. occured as part of a R-l and R-2 sequence. Hughes found the phenomenon of Immediate sensitization sufficiently unique to characterized it as perhaps resulting from some specific activity related to the auditory processing of low frequency signals.

Noffsinger and Tillman (1969) wanted to replicate and considerably expand Hughe's study. So they used 3 intensity levels 40, 65 and 90 dB and 200, 500, and 4000 Hz signals. In expanding, they used (1) Larger subject population and (2) Trials using higher frequency stimulus. Noffsinger and Tillman found, in contrast to Hughe's study that sensitization that is the only notable deviation from preexposure performance is not restricted to low frequency condition. They concluded that "Immediate sensitization is a real auditory phenomenon that can be elicited from a group of normal hearing subjects." Many of the trials in their experiments allowed exhibition of sensitization which was the first and only notable deviation from the preexposure level of threshold sensitivity. In addition, this phenomenon was not restricted to situations employing low frequent exposure and test stimuli. Since it could be elicited by stimulating the ear with a 3000Hz tone and examining threshold for 2000Hz pulses.

They further found that (1) sensitization is greater and appears sooner in the post stimulation time course for the low frequency conditions than for the high frequency ones (2) sensitization magnitude increases as a function of exposure intensity for both low and high tone conditions.

In another study carried out to find out the effect of ipsilateral adaptation and changes in threshold, Rajanikanth (1985) found that while using pure tones of 500, 1000, 2000 and 4000 Hz and at 20, 40 and 60 dB SL's, magnitude of sensitization was nearly same at all the frequencies tested.i.e. the frequency of the adapting stimulus had no effect on the magnitude of sensitization. He also found that the magnitude of sensitization at 60 dB SL was more than that for either 20 or 40 dB SL.

There was improvement in thresholds in the ipsilateral ear for frequencies, 500, 1000, 2000 and 4000 Hz after the ear was stimulated with narrow-band noise (sridhara, 1985). sridhara also found that the magnitude of sensitizationwas not significantly affected by the test frequency. However, the level of stimulus presentation did have an effect on the magnitude of sensitization, in that more sensitivity (7 dB) was observed with 60 dB SL presentation of Narrow Band Noise (NBN) rather than for 20 dB SL (4 dB) or 40 dB SL (2.5 dB).

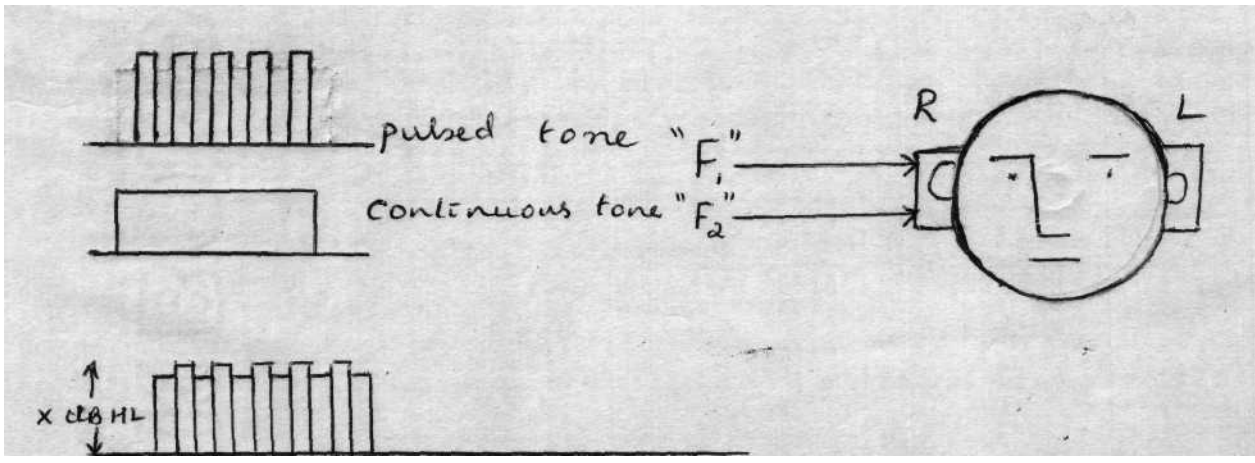
To find out if stimulation of one ear with a pure tone, can bring about sensitization in the contralateral ear as shown by improved behavioural thresholds, Ragini (1985) carried out a study on normals. The results of her study showed "Sensitization" in the contralateral ear consequent to auditory stimulation of the test ear. The present study aims to find out sensitization at frequencies lower than the stimulus frequency, when the duration of the continuous stimulation is 7 minutes.

Hypothesis of the study:

The following null hypothesis was formulated for the present study.

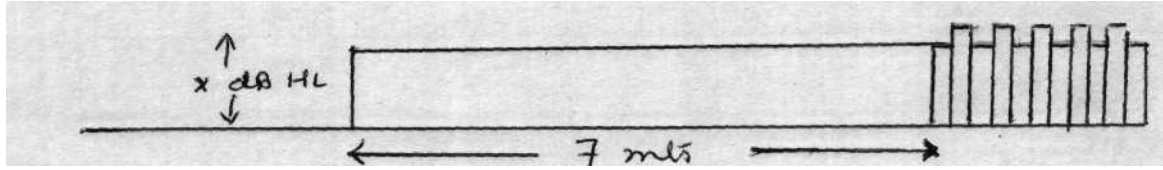
"There is no significant difference between the thresholds obtained in the conditions A and B".

Condition-A: Threshold for pulsed tone obtained in the presence of a continuous pure tone. The pulsed tone being one octave lower in frequency than the continuous pure tone. The level of the continuous tone being 40/ 60/ 80 dB HL.



Condition-B: Threshold for pulsed tone obtained again when the ear is being stimulated beyond 7 minutes by the continuous tone the pulsed tone being one octave lower in frequency than the continuous pure tone.Level of continuous tone being 40/60/80 dB HL.

Condition-B



Where

X = 40/60/80 dB HL

and

F_1= 500/1000/2000 Hz

F_2 = 1000/2000/4000 Hz

Brief plan of the study:

15 subjects with normal hearing (ANSI, 1969) were selected and they constituted 3 groups of 5 subjects each.

Group-A : Was tested at 40 dB HL

Group-B : Was tested at 60 dB HL

Group-C : Was tested at 80 dB HL

Each group was tested using the three adapting frequencies viz. 1000, 2000 and 4000 Hz, separately. The test frequencies

for the three adapting frequencies (1000, 2000 and 4000 Hz) were 500, 1000 and 2000 respectively. In other words, the test frequency, for measuring the thresholds after the ear was adopted, was always one octave below the adapting frequency. The duration of adapting stimulus was more than 7 minutes. At the end of 7 minutes, the threshold for the pulsed tone (one octave below the adapting frequency) was determined in the presence of the adapting stimulus. That is, the adapting stimulus was not with drawn at the end of 7 minutes, while measuring the threshold for the pulsed tone (see the figures 1 and 2 for clarity).

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REVIEW OF LITERATURE

Studies on Loudness adaptation have been carried out, over many years. As early as 19th century, researchers such as Dove demonstrated the existence of Adaptation. 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 and Fraser, 1970).

Davis (1961)says - In the neuro-physiological term adaptation may be described as "peripheral or local change in the sensitivity of sensory cells". Auditory adaptation can be classified as:

1. Perstimulatory |(Ward, 1973). Q. Post Stimulatory

or |

2. Concomitant | Residual

1. Simple |

| (Scharf, 1973).

2. Induced |

- Perstimulatory adaptation is - Adaptation measured using simul- -Residual adaptation in taneous dichotic loudness balance method. adaptation measured after the process of

- Concomitant adaptation is - Adaptation measured during the adoptrate process of adaptation.

- post stimulatory adoptation is adaptation measured using ABLB test

These two types of adaptation can further be divided into Monaural or Binaural.

Simple adaptationrefers to "the reduction in loudness, measured over a period of time, of a single auditory stimulus". Here, there is no comparing stimulus.

On the other hand, the adaptation measured using inter-aural matching procedure with a steady sound in one ear and a pulsatile sound in another or same the ear is termed "Induced Adaptation".

Adaptation has been traditionally measured by using:

I. Psychophysical methods.

II. Neurophysiological methods.

I. Let's first deal with psychophysical methods. They are, according to Scharf, 1982.

(a) Measurements without recourse to interaural loudness matches.

(b) Measurements of adaptation based on interaural loudness matches.

(c) Measurements of adaptation based on lateralization judgements

(a) Measurements without recourse to interaural loudness matches:

Researchers in the past have tried to measure adaptation with this method. In one such study by Lawrence et al., in 1949 nine untrained observers were required to say whether the sound had increased or decreased in loudness, while the loudness re-mained unchanged, increased or decreased in loudness.

When this 1KHz tone was 15 dB above threshold, the observer said that loudness of an unchanging tone had decreased, 82% of the times. They also said that loudness had increased, 66% of the times, when the tone was actually unchanged and was 70 dB about threshold. The observers judged the loudness of a 15 dB tone as unchanged (i.e. half the judgements were that the sound had increased in loudness and half that it had decreased when the tone actually increased at a rate of nearly 3 dB/min.) At 70 dB SL, the tone had to be decreased at the rate of 0.5 dB/min. for the loudness to be judged as constant one interpretation of these results is that the loudness of a soft tone decreases over time but that of a tone at a moderate level may increase slightly.

In another study Harris and Pittler (1960) gradually increased or decreased the intensity of the tone while the observer tried to keep loudness constant by compensatory tracking. The observers maintained nearly constant intensity for a 1KHz tone at 40 phons for upto 1 minute. Had loudness been decreasing owing to adaptation, observers would have been expected to err by overcompensating for a tone physically decreasing in intensity and undercompensating for a tone physically Increasing in intensity.

b) Measurement of loudness based on interaural loudness matches:

There is a large amount of discrepancy between the monaural and binaural loudness matching techniques. The supposedly neutral

comparison sound brought out merely as a yardstick, has often a marked effect on the loudness of the test sound, a depressing effect that increases over time. The simultaneous measures tell us about induced loudness adaptation, the delayed dichotic measures tell us about simple adaptation.

In Simultaneous dichotic measures, the comparison sound is presented to the ear contralateral to that receiving the steady sound while the steady tone is still on, the comparison sound in presented either once or repeatedly.

In delayed dichotic loudness balances, the loudness of a brief sound in one ear is matched to a long-duration sound after termination of the long duration sound.

Scharf is of the opinion that contemporary literature reveals an absence of loudness adaptation by delayed dichotic loudness balances as well as from monaual studies. The adaptation in simultaneous dichotic loudness balances is ascribed to interaction between the comparison tone and is not assumed to demonstrate a decline in loudness that would have taken place simply as a function of time without the intervention of the contralateral comparison sound. This is because (1) If interaction is eliminated by using monaural studies or if it is reduced by masking the frequency of a comparison tone which is presented only once different from the test frequency (Bray,1973). Then adaptation disappears (2} If interaction is increased by

lengthening the duration of simultaneous comparison sound or by presenting it intermittently throughout the period of the adapting sound adaptation may be increased to as much as 30 dB.

c) Measurement of adaptation using lateralization procedure:

Adaptation measured using loudness matching and lateralization do not yield the same results. When true sounds of similar frequency are given to the two ears at same intensity levels, the 2 tones are heard as a single tone at or near the center of the head. It follows that if one ear is exposed to a steady sound whose loudness decreases due to adaptation, the introduction of an equally intense sound in the unadapted ear should result in lateralization towards the ear where loudness is greater. But it has been shown that (1) median—plane localization does not require equal loudness at the two ears (2) Even if loudness equality is required for median - plane localization, it does not necessarily follow that prolonged stimulation results in loudness adaptation, prolonged exposure could just as well fatigue or result in adaptation of lateralizatlon mechanism.

Scharf suggests that adaptation of the lateralization mechanism, like induced loudness adaptation, occurs when a steady tone to one ear is accompanied by an intermittent tone to the other ear. The two phenomena probably depend on a common mechanism.

II. Neurophysiological data on auditory adaptation:

Few data are available on/low neural responses within the auditory system depend on time beyond durations of 1 or 2 secs. It has often been demonstrated that a sound evokes an initial burst of rapid firing in a single eighth-nerve fiber and that within the first 50 msec, the rate decreases to a more or less steady value. But just how steady that value remains over the next several minutes is not clear.

Young and Sachs (1973) showed that discharge rate changes as a function of time at low as well as at high stimulus levels. SPL's from 28-89 dB, the response to a 60 sec. tone near 2KHz decreased rapidly during the first few seconds of stimulation and then slowly throughout the remainder of the 60 sec.period.

The effect of level on neurophysiological responses in the cochlear nucleus is reported by tenkate et al., (1977). In cat, the spike rate in units of DCN decreased with duration in response to a steady 100 sec. tone or white noise. The decline in firing rate as a function of duration increased with stimulus level. The initial spike rate increased with level, but after 100 sec, the firing rate after 100 sec. was faster to a low level tone than to a tone 80 dB more intense. This is contrary to the psychophysical data on humans. Humans show a decrease in adaptation with increasing level until by 30 dB SL simple loudness adaptation is hardly measurable.

In another study by Gisselson and Sorensen (1959), the researchers found that brief change in the sensitivity due to low intensity auditory stimulation, which they termed adaptation seen in psychoacoustic experiments, could not be recorded in the cochlear microphonic in the guinea pig. For this reason they felt that the cause of this adaptation should be sought more centrally. In a further experiment Sorensen studied auditory adaptation in nerve action potential in the guinea pigs. He found a reduction in the amplitude of the click responses when the clicks were masked by white noise of moderate intensity, and he also recorded the recovery time for the second of a pair of clicks. He concluded that a "depression could not be provoked by stimulation of the contralateral ear, for which reason central inhibition could be excluded". However, Petty et al (1970), used successive and simultaneous presentation of heterophonic stimuli in order to determine whether adaptation is a central or a peripheral phenomenon. They observed decrements with the typical simultaneous dichotic balance procedures which they feel probably reflect slowly developing changes in binaural interaction and consequently are central rather than peripheral.

Sensitivity of single auditory nerve fibers to pure tone stimuli can be reduced by anoxia, ototoxic drugs exposure to high intensity tones (Kiang, 1970).

Furukawa and Matsuura (1978) are of the opinion that adaptation takes place at the synapses between the hair cell and afferent nerve fibers.

Thus various sites of adaptation have been proposed by various researchers. An important region in the auditory system which plays a major role in adaptation is olivo cochlear bundle. This is one of the major efferent pathways running from superior olivary complex to hair cells of cochlea. Because of efferent pathway the activity of the lower levels of the nervous system can be influenced by the complex responses of the highest. A suggested possibility is that the centrifugal pathways could modify the sensory input during processes such as attention (Pickles, 1982). However, declining of attention over a period of time as the cause for adaptation has been refuted (scharf, 1982).

The fibers of the olivo cochlear bundle enter the cochlea and branch off to enter cochlear nucleus. Within the cochlea, the fibers terminate in two ways. Some fibers terminate with large granulated synaptic terminals around the lower ends of the outer hair cells. They appear to envelope both the base of the outer hair cells and the afferent terminals. They therefore appear to be able to control not only the state hair cells but possibly also synaptic transmission to the afferent pathway. These are mainly crossed fibers (COCB).

A rather great proportion of the fibers (UOCB) end in the region of the IHC. They make dendritic synapses with afferent terminals on the base of IHC but seldom do they make contact with the IHCsthemselves. Projection to the IHC's comes mainly from lateral superior olive (LSO) but those to OHC comes from MSO. This separation into 2 systems, one to the region of the IHC and one to the OHC, may well be associated with a functional separation associated with the different roles of the IHC and OHC in transduction.

Activation of crossed olivo cochlear bundle (COCB) hyper-polarized cells and therefore elicits greater cochlear micro-phonic (CM). CM comes mainly from OHC which receives fibers from COCB. Stimulation of the UOCB reduces the N1 potential of the cochlea but has no effect on the cochlear microphonics.

Leibrandt (1965) studied the role of OCB in adaptation. He recorded whole nerve responses at the round window of guineapigs to a series of tone bursts. Adaptation to successive stimuli was noted in the round window response. However, on injection of "procaine" absence of adaptation was noted in 6 of 10 animals. In others arrest of respiration occured or the adaptation remained unchanged. He concluded that the absence of adaptation was secondary to the blockage of the efferent bundles to the cochlea. Researchers have found that stimulation of the crossed olivo cochlear bundle produces a suppression of the AP response from the round window.

Dayal (1974) studied adaptation to successive auditory stimuli in the nerve action potential. The frequencies investigated ranged from 4KHz - 7KHz at 60-70 dB SPL. Changes in adaptation were studied before and after sectioning of the COCB. No effect was seen at the AP recorded from the round window.

Adaptation is avoided when stimuli evoke on responses in units of the auditory system at levels beyond the cochlear nuclear continuously. Scharf assumes that on-responses occur when the level of excitation increases sufficiently either because of an increase in stimulus intensity over a small group of fibers or because of variation in pattern of excitation across fibers.Changes in stimulus intensity at low levels are needed to evoke on responses. Otherwise the excitation remains relatively fixed over a small group of units.

Variation in excitation patterns occurs at higher levels where the pattern evoked by a tone in wide spread and unstable as a large number of fibers fire out of phase.

The general rule may be that sensory systems adapt to steady, prolonged stimulation that is concentrated on a constant set of receptor units. Fluctuations in the level of stimulation reduce or eliminate adaptation. Fluctuations may be in the stimulus or in the sensory system with respect to loudness, the

question is just how and where in the auditory system temporal variation are imposed on the level of excitation ao as to avoid adaptation under most listening condition.

Sensitization:

Lawrence (1949) and Hughes (1954), Mirabella et al., (1969), Olsen and Tillman (1970), Noffsinger and Tillman (1970), Fex (1982), Gerken (1984), Pickles (1982), Cody and Johnstone (1982) have all reported loudness gain after continuous stimulation of the auditory system. Evidence for "sensitization" comes from both psychophysical and neurophysiological studies.

As sited earlier Lawrence et al., (1949) in a study with 9 normal subjects concluded that loudness of a tone at moderate intensity may increase slightly. Mirabella et al., (1967) reported that 72 observers showed reverse adaptation for a 3.5KHz tone at 90 dB in the tracking method.

Qerken (1973) studied the effects of 3KHz tone bursts of 2 ms. duration in terms of the evoked response obtained from the medial geniculate nucleus in cats. Increased stimulus intensity produced increased evoked response amplitude. In addition of a 3KHz, 70 dB SPL continuous tone made a significant alteration in one of the amplitude intensity function. In the presence of the continuous tone the evoked response amplitude was greater. Gerken terms facilitation of this sort

by a sustained sound, "enhancement". The relationship between enhancement, stimulus parameters and parameters of the continuous tone was found to be complex. Gerken also hypothesizes that the enhancement produced in medial geniculate evoked responses by a sustained sound is related to the stimulation hypersensitivity produced by the sustained sound in the cochlear nucleus.

Gerken defines "hypersensitivity" as any phenomenon representing sustained alteration from the resting state of the auditory system in the unanesthetized animal, meaning that some measurable aspect of the central auditory system has changed so that the system is more excitable or sensitive than in its resting state. But then, there is still controversy regarding, whether adaptation is central or peripheral. Petty et al., (1970) are of the opinion that adaptation is a central phenomenon.

Noffsinger ana Olsen (1970) are of the opinion that sensitization and desensitization reflect the state of atleast partially separate physiological mechanisms that are affected in different ways and for different periods of timely 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.

Noffsinger and Tillman (1970) studied sensitization and conclude that (1) Sensitization to a continuous tone is greater than to that to an interrupted tone (2) Sensitization is not restricted to the ear exposed although transitory sensitization is.

Bodian (1983) says that it must be kept in mind that evidences for inhibitory role of the efferent innervation of the cochlea pertains to the inner hair cell system. Function of ESIOHC is yet to be known presence of efferent innervation of the vestibular receptors suggests a general role for all labyrinthine efferent pathways such as the enhancement of sensitivity of the various receptors.

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 (1982). The released neurotransmitter may be "Aspartate Amino Transferase" or even encephalin like neuro-active substance which contributes to sensitization.

Vyasamurthy (1977) used the magnitude of acoustic reflex as a measure of perceived loudness. He used this technique to measure adaptation and recovery from adaptation. Data was collected on normal hearing adult subjects using this technique. Having obtained this data, the researcher proposed a revised

model of adaptation, which among many other things, also provides explanation for sensitization of loudness gain. The revised model of adaptation considers both the peripheral and central organs in explaining the phenomenon of adaptation.

To understand the revised model of adaptation it's necessary to know a few anatomical details of some of the structures of the auditory system. As mentioned earlier the inervation of OHC and IHC by the efferent pathway is different. This probably implies functional differentiation of IHCs and OHCs in transduction (Pickles 1982).

In the revised model of adaptation, Vyasamurthy proposes that there are units in the auditory system specifically responsible for loudness gain. He terms them "a_2" units in contrast to the "a" and "a_1" units which are responsible for loudness loss, "a" units are stable "a_1 and a_2" units are unstable, "a" units may originate from the afferent neural units of characteristics frequency. a1 units may originate from the efferent system innervating the inner hair cell (ESIIHC) and a_2 units which are responsible for loudness gain may originate from the efferent system innervating the outer hair cell (ESIOHC). The efferent system to the IHC comes mainly from LSO and that to the OHC comes mainly from MSO. Thus, a_1 and a_2 units may originate from the actions of the LSOES and MSOES respectively. The loudness gain is attributed

to MSOES. However, this system stimulation the IHCs for low intensity sounds. The model assumes a mechanical coupling between the outer and inner hair cells, which modifies the input to the inner hair cells. The role of OHC including the MSOES for stimulating the IHCs for low intensity sounds has been cited by Davis,(1983) Mouatain,(1985)among many others as reported by Vyasamurthy (1985). The "cochlear amplifier" is responsible for the greater sensitivity and sharp tuning curves expressed by the IHC efferents. This active mechanism is an electromechanical amplification, whose reduction would result in a diminished input to the receptor cells and hence the discharge rates of the auditory nerve fibers of characteristic frequency would result. This reduction in electromechanical amplification could bs brought about by exposure to sound. Thus, the active mechanism may be responsible for the greater sensitivity and sharp tuning expressed by the IHC afferents. The active mechanism may also be responsible for the production of "a" units - which operate during adaptation. In explaining what happens during sensitization, in the light of the revised model, Vyasamurthy proposes that - "the mechanical input to the inner hair cells of lower characteristic frequency may increase during auditory adaptation". This proposal in supported by Hugheg's finding that "sensitization" is found at frequencies lower than the adapting frequency,i.e. to say, there is an increase in the rate of firing of neurons whose characteristic frequency is lower than the stimulus frequency, as a consequence of increased

active mechanism. The active mechanism probably shifts towards the apical end which is responsible for lower frequency.

However, the increased active mechanism which probably is responsible for the increased firing rate, is not evidenced in terms of the AP response. Infact, AP response reduces after the ear is adapted. However, since AP response reflects the synchronized firing of the neurons mainly from the basal and, it is likely that the increased rate of firing of the neurons at the apical end, may not be represented in the AP response.

There is yet another explanation for the loudness gain proposed by Vyasamurthy. The OHC afferents may be reporting back the state of stiffness of the stereo cilia to efferent excitation. Since the input (OHC afferents) is important for the servo-system (OHC afferents and efferents), the MSOES may maintain the synaptic efficacy by recycling the released neuro-transmitter using the aspartate-amino transferase. Excessive activity of the MSOES which probably results from the changes in the input may also contribute to the loudness gain. Bodian (1983), Code and Jhonstone (1982) have shown that the role of the MSOES is loudness gain.

Loudness Loss: LL may arise from IHC afferents whose characte-ristic frequency is same as the frequency of the adapting stimulus These CF units are thought to be responsible for the "a" units. "a1", units arising from LSOES may also be responsible for loudness

The evidence for this is (1) afferents to the IHCs synapse with the afferent dendrites (no connection with the cell body) (2) Stimulation of the uncrossed OCB (which mainly supplies IHCs) reduces N_1 potential (Sohmer, 1966) (3) The neurotransmitter of the LSOES is ankephaline which is an inhibitory transmitter. (Eyebolin and Pujor, 1984) (4) Code and. Johnstone (1982) demonstrate that the ipsilateral sensitivity loss induced by an intense pure tone could be reduced by acoustic stimulus of the same frequency delivered simultaneously to the opposite ear. Probably the LSOES inhibit the IHC afferents from firing during binaural acoustic stimulation. The neural units being "So" inhibited may be expected to have avoided the adaptation process and hence their contribution to N_1 response during the post exposure period could be responsible for the reduced ipsilateral sensitivity loss (The contralateral stimulus may prevent the loudness gain in the adapting ear by interrupting the active mechanism - this factor also may be responsible for the reduced ipsilateral sensitivity loss) (5) Stimulation of the UOCB inhibits the activity of auditory nerve fibers (COM's, 1962).

The revised model of adaptation has been verified by 3 investigators studying sensitization. All the three studies (Ragini, Rajanikanth, and Sreedhara, 1985) supported the revised model.

Ragini's study concentrated on sensitization for contralateral auditory stimulation. She found that the ear opposite to the adapted ear exhibits sensitization at 500 Hz, l000Hz, 2000Hz and

4000Hz. Adaptation was carried on at 50 dB HL for 7 minutes. She used 32 subjects who were divided into 4 groups. Group-A was tested only at 500Hz, Group-B was tasted at 1KHz, Group-C was tested at 2KHz and Group-D was tested 4 KHz. Ragini concludes that "the fact that the ear opposite to the adapted ear exhibits sensitization is an evidence that some facilitatory process may be operating in the ear opposite to the adapted ear". This facilitatory process may be viewed interms of synaptic efficacy brought about by the efferent system innervating the outer hair cells (ESIOHC). The loudness gain in the contralateral ear, according to Vyasamurthy (1982) is due to efferent action, i.e. "a2" units will be produced in the ear opposite to the adapted ear and this presumably is responsible for greater adaptation observed in the SDLB technique, when adaptive stimulus of 80 dB is used.

Sridhara (1985) studied sensitization in ipsilateral ear on exposure to continuous narrow band noise. He used 15 normal hearing subjects divided into 3 groups. The first group was exposed to NBN at 20 dB SL, the second group was exposed to 40 dB SL and 3rd group was adapted at 60 dB SL. The duration of exposure was 7 minutes for all the three groups. All the 3 groups were tested at 500Hz, l000Hz, 2000Hz and 4000Hz pulsed pure tones, while the same ear was being exposed to NBN centered round 500Hz, l000Hz, 2000Hz and 4000Hz respectively. He tested only the right ear.

Sridhara found sensitization at all the four frequencies tested . In his study the magnitude of sensitization increased as the level of the adapting stimulus increased. The mean values of sensitization at different levels of stimuli are 4dB for 20 dB SL, 6.5 dB for 40 dB SL and 7 dB for 60 dB SL.

In another study carried out on the effect of ipsilateral adaptation and changes in threshold, Rajanikanth (1985) tested 15 normal hearing subjects who were divided into 3 groups based on at what intensity their ears were adapted. Group-A was exposed to 20 dB SL, Group-B to 40 dB SL and Group-C to 60 dB SL. The frequencies tested were 500Hz, l000Hz, 2000Hz and 4000Hz. Like Sridhara, Rajanikanth also found that the"frequency of the stimulus had no effect on the magnitude of sensitization. However, the level of adaptation did have positive correlation with the magnitude of sensitization, in that sensitization at 60 dB SL was more than that for either 20 dB SL or 40 dB SL.

It has been speculated (Vyasamurthy, 1985) that when an auditory stimulus is presented continuously to the ear, the locus of the active mechanism may shift apically, which in turn may be responsible for the "sensitization" at frequencies lower than the adapting frequency. Many investigators have reported that the function of the active mechanism (comprising of OHCs and their afferents and efferents) is to increase the sensitivity

of IHCs for lower intensity sounds. That is, it is reported that the MSOES may increase the stiffness of the stereo cilia of the OHCs to increase the mechanical input to the IHCs. Thus, the "sensitization" may result from the increased mechanical input to the IHCs whose characteristic frequency is lower than the adapting frequency.

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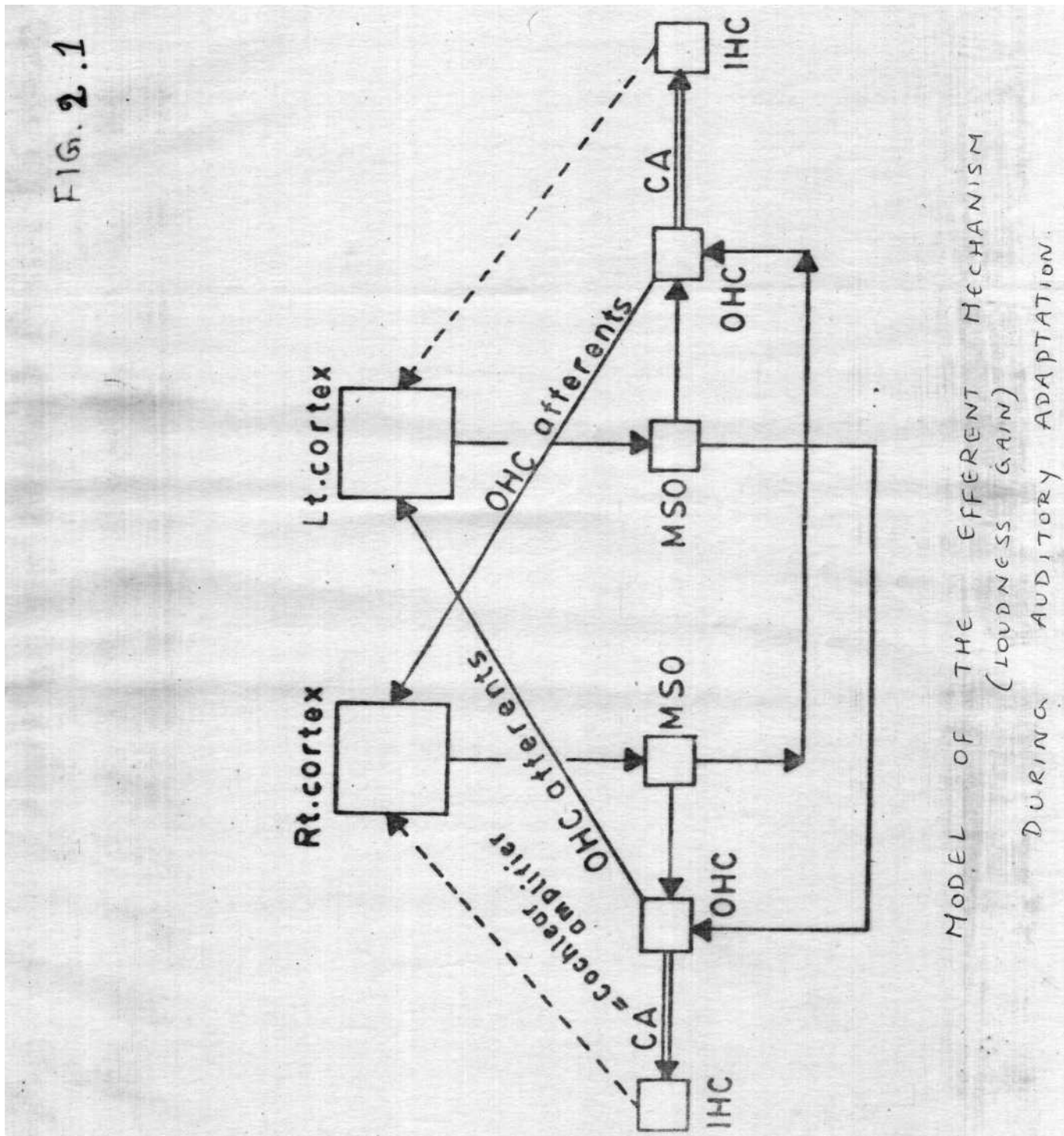
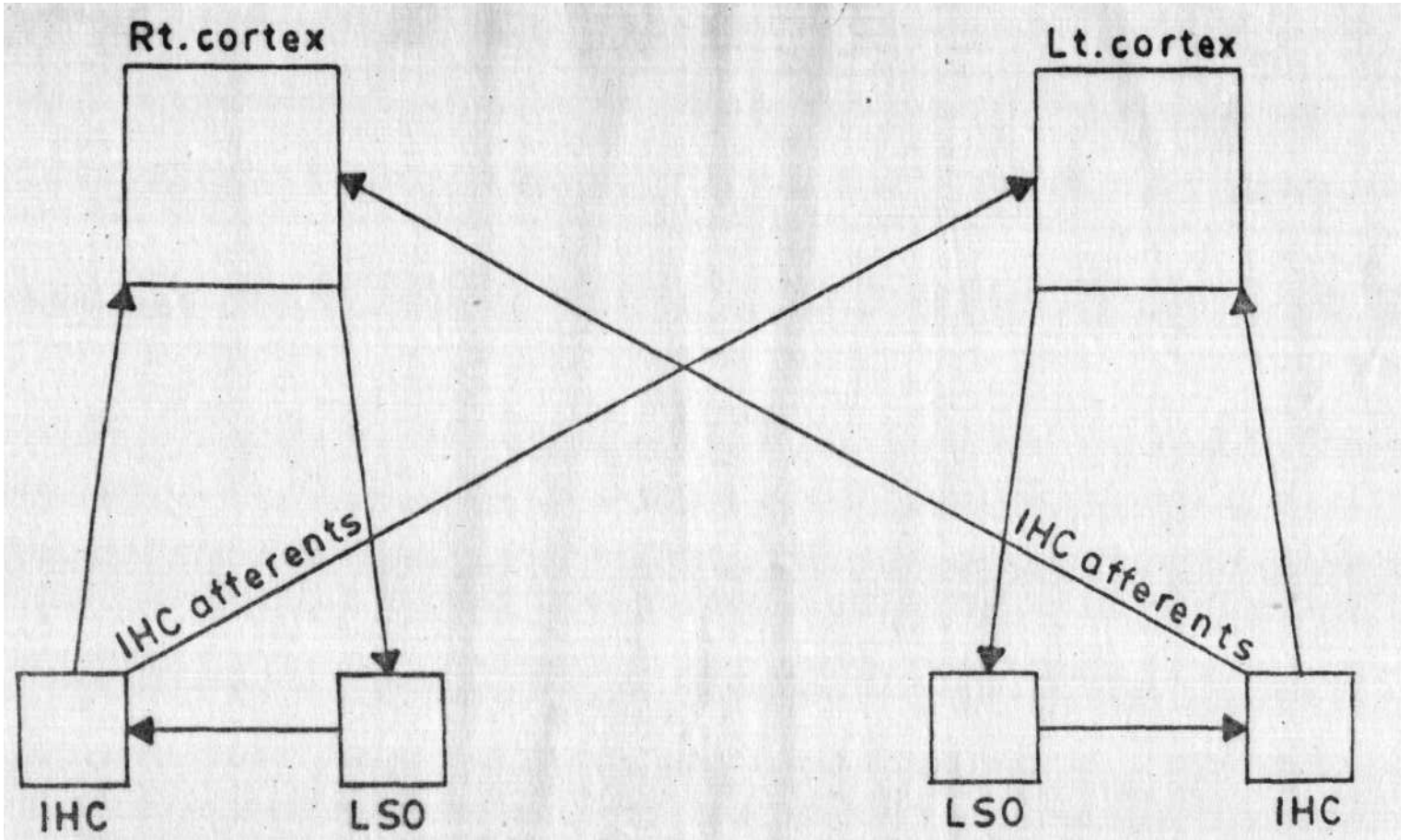
FIG. 2.1



MODEL OF THE EFFERENT MECHANISM (LOUDNESS GAIN) DURING AUDITORY ADAPTATION.



MODEL OF THE EFFERENT MECHANISM
(LOUDNESS LOSS)
DURING AUDITORY ADAPTATION

METHODOLOGY

The present study was carried out to study sensitization at frequency lower than the stimulus frequency. The stimulus frequency wag 'on' for a period of 7 minutes and the pre and post exposure thresholds were measured in the presence of the stimulus frequency. The stimulus frequencies were 1KHz, 2KHz and 4KHz and thresholds were measured at 500Hz, 1KHz and 2KHz respectively, i.e. one octave below the stimulus frequency.

Subjects:- 15 subjects (8 females and 7 males) between 18 and 25 years of age (mean age 19.13 years) served as the subjects for this study. All the subjects had a hearing threshold level of less than or equal to 25 dB (ANSI, 1969). None of the subjects had significant otologic history.

Equipment used:

A dual channel diagnostic audiometer, Beltone 200-C with TDH-39 earphones housed in MX-41/AR cushion was used for the study. The provision of this audiometer, of use, in this study were (1) frequency range from 125Hz to 8000Hz (2) hearing level range from -10 to 110 dB HL (3) provision for simultaneous pre-sentation of a pulsed tone through the channel and a continuous tone through the other channel to the same ear.

The instrument was calibrated periodically during the study in accordance with the instuctions provided by the manual. The output and linearity of both frequency and intensity were calibrated.

Test Environment: A two-room setting which was sound treated room was the test environment. The subject could not see the control panel in the tester's room; due to lighting which also permitted the tester to observe the subject's responses.

Instructions to the patients:

"You shall first hear a pulsed tone and a continuous tone in the same ear. You are required to respond only to the pulsed tone. The continuous tone will then continue for 7 minutes, at the end of seven minutes. You will hear the pulsed tone again in the presence of the continuous tone. Respond to the pulsed tone. Indicate through your left finger if you hear in left ear and through your right finger if you hear in your right ear".

Procedure:

15 subjects were divided into 3 Groups A, B and C. The criteria for grouping was the intensity of the stimulus frequency

Group-A : was exposed to 40 dB HL

Group-B : Was exposed to 60 dB HL

Group-C : Was exposed to 80 dB HL

Each subject was tested at all the three frequencies; 500Hz, 1KHz, 2KHz with the stimulus frequencies being 1KHz, 2KHz and 4KHz respectively.

Subjects belonging to Groups B and C were not tested the same day in both the ears to rule out the effect of cross-over.

The threshold for pulsed tone was obtained in the presence of a continuous tone which was one octave higher in frequency. This is threshold of "Condition-A".

The same ear was adapted using 40/60/80 dB HL continuous tone depending on the group for 7 minutes. At the end of 7 minutes , threshold for pulsed tone was once again obtained without withdrawing the continuous tone. This is threshold of "Condition-B".

Sensitization was determined by subtracting the threshold obtained in condition-B from threshold obtained in condition-A

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RESULTS AND DISCUSSION

The following Tables shows the results of the study in terms of the amount of sensitization in dB for the three groups. The means and standard deviations of these three groups have also been indicated.

Graphical representation of the improvement has also been shown.

The Table-1 depicts the amounts of sensitization at 500Hz, (stimulating frequency l000Hz) l000Hz (stimulating frequency 2000Hz) and 2000Hz (stimulating frequency 4000Hz) for both the ears. Table-1 represents sensitization at the above mentioned frequencies with the stimulating intersity at a level of 40 OB HL. Table-2 represents sensitization with the stimulus intensity at 60 dB HL and Table-3 represents sensitization at the stimulus at of 80 dB HL.

It is clear from the tables and graphs that there is sensitization at frequencies below the stimulus frequency, when the mean values are considered.

There were, however subjects who showed no sensitization (i.e. threshold of Condition-A Threshold of Condition-B=0) or even adaptation (i.e. threshold of condition-A - threshold of condition B>O) in this study. Their number, however was statistatcally insignificant.

There is improvement in threshold at all frequencies (500Hz, 1KHz and 2KHz) and at all intensities (40, 60 and 80 dB HL).

The magnitude of sensitization, however, does not depend on the frequency or intensity of the stimulus.

"The Wilcoxon matched pairs signed ranks test" was used to find out whether there is significant difference between the thresholds obtained before and after adaptation. From the data analysis, it is concluded that significant difference does exist at the 0.05 level of significance.

Sensitization studies have been reported by many investigators Hughes (1954), Noffsinger and Olsen (1970), Vyasamurthy (1982), Cody and Johnstone (1982), Rajanikanth (1985).

However, none of the above studies has made use of the methodology which has been used here. Hence the results of this study cannot be compared with the results of other studies.

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

Sensitization is observed in both the right and left ear as reflected in the mean threshold improvement found after continuous auditory stimulation.

However, adaptation was found for two subjects in Group-A at 500Hz in the right ear.

In group-B, adaptation was observed in one subject at 500Hz in the right ear and at 1KHz and 2KHz in the left ear.

In Group-C, adaptation was observed in one subject at 2KHz in the left ear.

In the rest of the subjects, sensitization has been observed at all the frequencies in both the left and right ear.

It is commonly believed that with continuous auditory stimulation reduces the sensitivity of the auditory system. In this study, we have found that, continuous auditory stimulation with a pure tone enhances the ear's sensitivity to pure tones of frequency lower than the stimulus frequency thus supporting. Hughes' (1954) study which also found that maximum sensitization occurs at frequencies lower than the stimulus frequency.

The explanation for this phenomenon can be got while viewing loudness gain in the light of the revised model of adaptation (Vyasamurthy, 1982). Vyasamurthy proposes production of "a_2" units during adaptation which arises from MSOES. The a_2 units are produced in the adapted ear.

The neural model of the efferent mechanism for loudness gain proposed by Vyasamurthy, 1982, views facilitatory process in terms of synaptic efficacy brought about, by the BSIOHCs.

Fex et al., (1982) attribute loudness gain to "Encephalin" which is a neuroactive, substance released by the efferent system. They also hypothesize that the ESIOHCs may participate in recycling of released neuro-transmitters, this AAT (Aspertate Amino transferase) activation.

Hence, the above explanation has been offered for the sensitization observed in the present study also.

The null-hypothesis formulated at the beginning of the study will be rejected or the results indicate that there is a 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-1: Showing sensitization of GroupA exposed to 40 dB HL

Subject	Right ear threshold improvement			Left ear threshold improvement		
	500Hz	lOOOHz	2000Hz	500Hz	lOOOHz	2000Hz
A_1	5	5	0	5	5	5
A_2	-5	5	10	0	0	5
A_2	5	0	0	5	0	0
A_4	-5	5	0	10	0	5
A_5	0	5	5	5	5	0
Mean	0	4	3	5	2	3
$S.D_N$	4.47	2	4	3.16	2.44	2.44
$S.D_{N-1}$	5	2.33	4.47	3.53	2.73	2.73

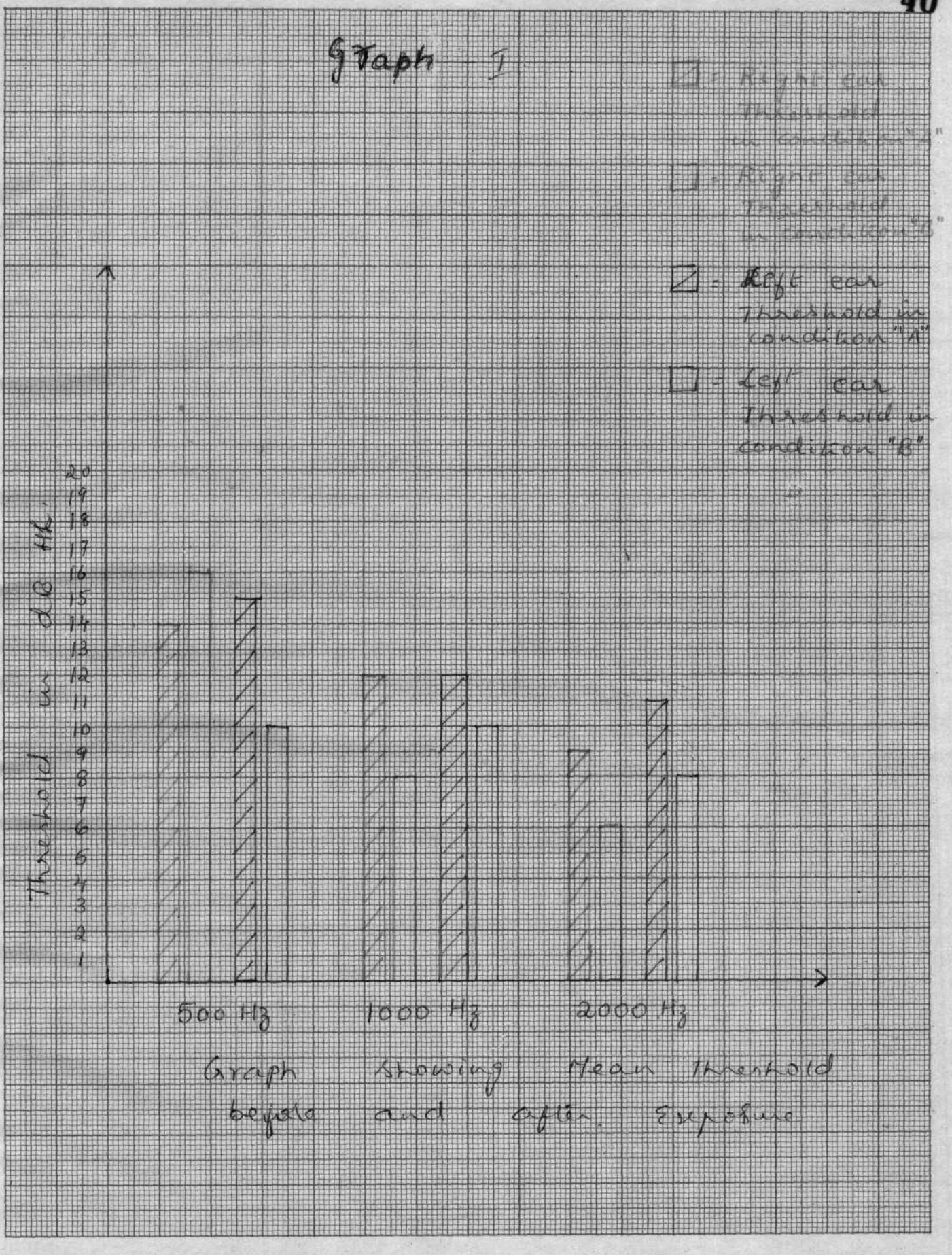
Table-2: Showing sensitization of Group-B exposed to 60 dB HL

Subject	Right ear			Left ear		
	500Hz	lOOOHz	2000Hz	500Hz	lOOOHz	2000Hz
B_1	0	0	5	5	0	0
B_2	-5	0	0	0	-5	-5
B_3	5	5	10	0	5	0
B_4	0	0	5	5	0	0
B_5	0	0	0	5	10	5
Mean	0	1	4	3	2	0
$S.D_N$	2.23	2	3.74	2.44	5.09	3.16
$S.D_{N-1}$	2.5	2.23	4.18	2.73	5.70	3.53

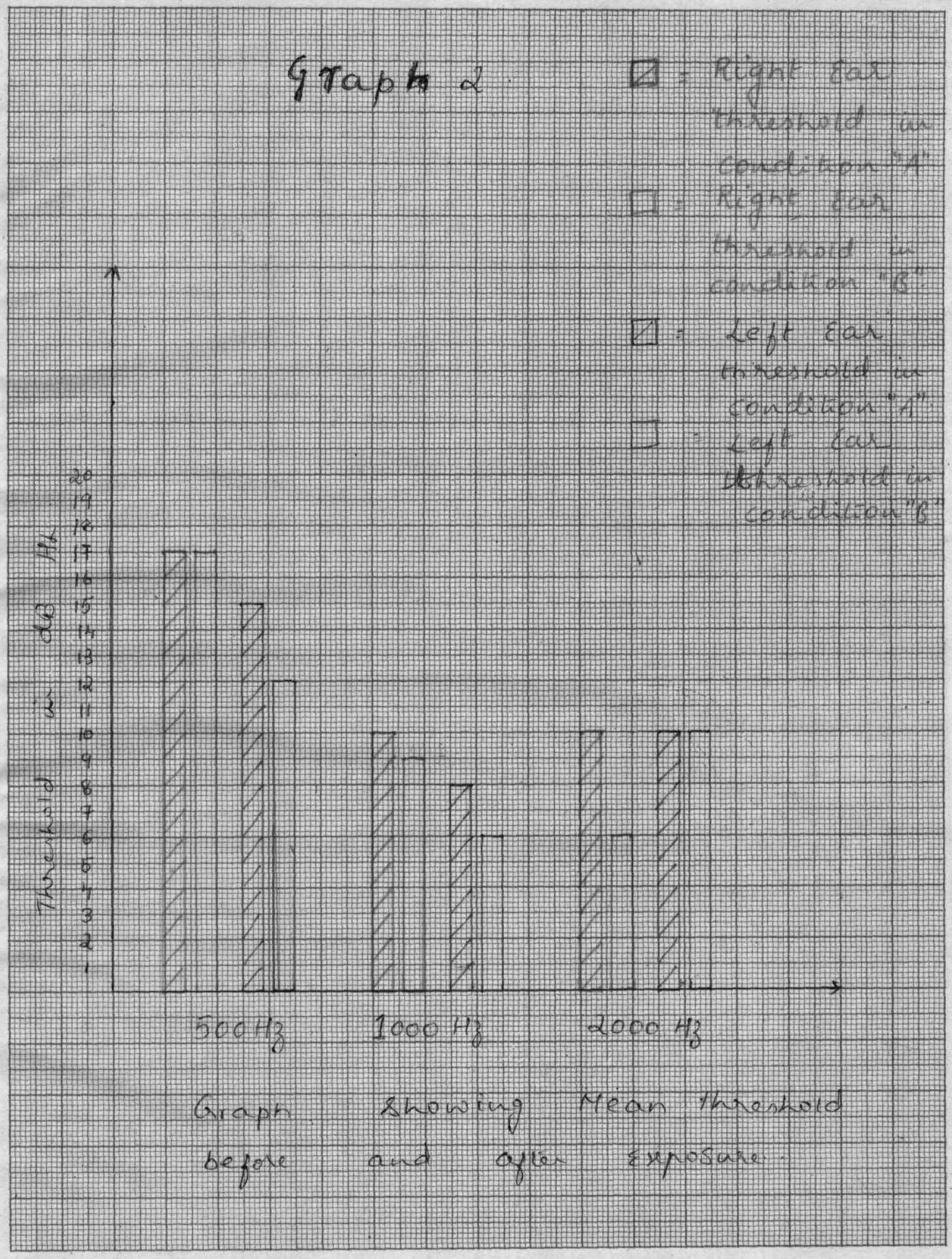
Table-3:Showing sensitization of Group-C exposed to 80 dB HL

Subject	Right ear			Left ear		
	500Hz	lOOOHz	2000Hz	500Hz	lOOOHz	2000Hz
C_1	5	0	5	5	5	-5
C_2	0	0	5	0	0	10
C	5	5	5	5	10	0
C_4	5	0	10	5	5	0
C_5	5	0	10	10	5	0
Mean	4	1	7	5	5	1
$S.D_N$	2	2	2.44	3.16	3.16	4.89
$S\text{-}D_{N-1}$	2.23	2.23	3.93	3.53	3.53	5.47

Graph 1

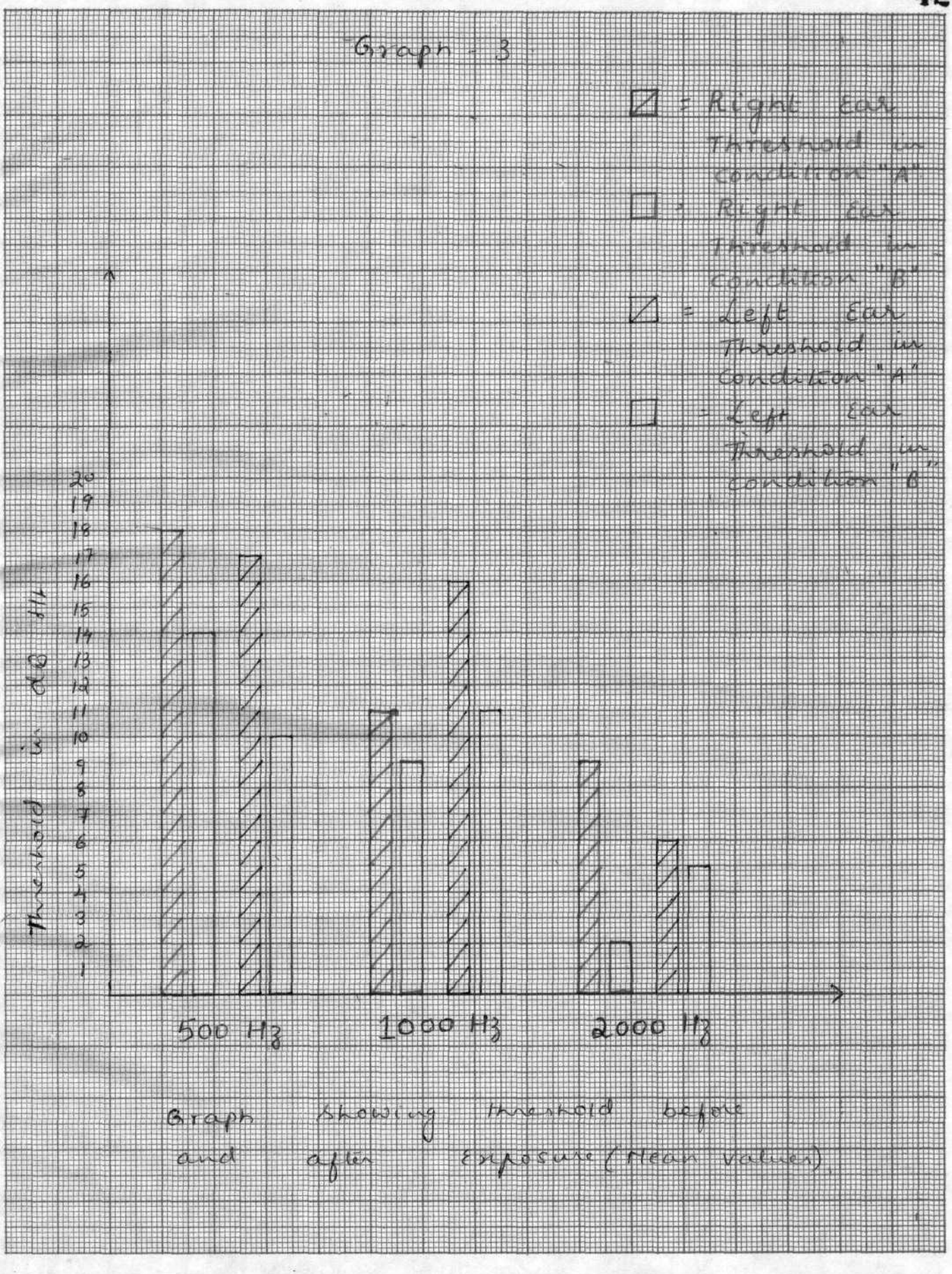


Graph showing Mean Threshold before and after Exposure



Graph showing Mean Threshold before and after Exposure.

Graph - 3



Graph showing threshold before and after Exposure (Mean values)

SUMMARY AND CONCLUSIONS

The present study directed its attention towards "Sensitization" at frequency lower than the stimulus frequency.

15 subjects with normal hearing served as subjects. They were divided into 3 groups. GroupA received the adapting tone at 40 dB HL, Group-B at 60 dB HL and Group-C at 80 dB HL. Each group was adapted with l000Hz, 2000Hz and 4000Hz continuous tone for 7 minutes. The thresholds were found out for 500Hz, l000Hz and 2000Hz pulsed tone respectively in the presence of the continuous tone both before and after adaptation.

Both the right and left ears were tested for all subjects and at all frequencies. The test procedure can be termed monaural perstimulatory.

Sensitization was found out by subtracting the pulsed tone threshold in the presence of continuous tone after 7 minutes of adaptation(Condition-B) from the pulsed tone threshold obtained in the presence of continuous tonebefore adapting the ear (Condition-A). The frequency of the pulsed tone was one octave lower than that of the continuous tone.

Results of the present study reveal that (1) sensitization is observed at frequencies lower than the stimulus frequency.
(2) The frequency of the adapting stimulus has no effect on the magnitude of sensitization.
(3) The level of the adapting stimulus also, does not have any effect on the magnitude of sensitization.

In his revised model of adaptation, Vyasamurthy (1982) attributes loudness gain to the efferent system innervating the outer hair cell.

A neural model of the efferent mechanism is also proposed by Vyasamurthy (1985a) in which he explains the facilitatory process in terms of synaptic efficacy brought about by the ESIOHCs.

Explanation to sensitization has been offered by many other studies. According to Noffsinger a "presynaptic" electrical or electrochemical hyperexcitability.

Bodian (1983) says "the presence of efferent innervation of the vestibular receptors suggests a general role for all labyrinthine efferent pathways such as the enhancement of sensitivity of the various receptors". It is plausible that the same holds good with auditory receptors also.

Vyasamurthy (1985 b) is of the opinion that sensitization at frequencie lower than the stimulus frequency may be brought about by the locus of the active mechanism shifting apically which happens when the auditory stimulus is presented continuously to the ear. The active mechanism increases the sensitivity of the inner hair cells for lower intensity sounds.

As has been suggested, the release of encephaline like substance by the ESIOHC, contributes to the loudness gain. It is reported that the MSOES may increase the stiffness of the stereo cilia of the OHCs to increase the mechanical input to the IHCs. Thus the sensitization may result from the increased mechanical input to the IHCs whose characteristic frequency is lower than the adapting frequency.

The results of the present study thus support the view that auditory sensitization is a real phenomenon which represents an electrical or electrochemical hyper excitability having its origin at the central level. The results also support the revised model of adaptation.

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BIBLIOGRAPHY

Abbas, P.J., Effects of stimulus frequency on adaptation in auditory nerve fibers, J.Acoust.Soc.Am. Vol.65, 162-165, (1979).

Beltone 200-C Installation and Service Manual.

Benitz, L.D., et al., Temporary threshold shifts in chinchilla, Electrophysiological correlates, J.Acoust.Soc.Am. 52, 1115-1123, (1972).

Bodian, D., Electronic microscopic atlas of the simian cochlea. Hearing Res. 9, 201-246 (1983).

Chabot, J.L., and Wilson, W.R., The effect of sensitization on the acoustic reflex as a function of frequency J.Aud.Res. Vol.17, 99-104 (1977).

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

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

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

Eggermont, J.J., and Odenthal,D.W., Electrophysiological investigation of the human cochlear adaptation, masking and recruitment. Aud.Vol.12, 191 (1973).

Elliott and Fraser, Fatigue and adaptation, J.V.Tobias, Foundation of modern auditory theory. Academic Press, New York, 1970.

Fex, J., Altschuler, R.H., Wenthold, R.J., and Parakkal, M.H., Aspartate aminotransferase immuno reactivity in cochlea of guinea pig, Hearing Res. 7, 149-160(1982).

Gerken, CM., A systems approach to the relationship between the ear and central auditory mechanisms. Adv.Audiology Vol (Karger Basel) 30-52 (1984).

Jerger, J., (Ed), Modern developments in audiology. First Edition, Academic Press, New York, (1963).

Jerger, J., (Ed), Modern developments in audiology. Second Edition, Academic Press, New York (1973).

Karja, J., Perstimulatory suprathreshold adaptation for pure tones, Acta.Otol.Suppl.241 (1969).
(1949)

Lawrence et al./ Discrimination of a sound changing gradually in intensity Jr.Aviat.Med., 20 (211-220) quoted in chapter "Loudness Adaptation" written by scharf,B in the book hearing research and theory, Vol.2, Ed. by Tobias and Schubert; Academic Press 1983.

Lorente de No, R., Central representation of the eighth nerve in "Ear-Diseases, Deafness and Dizziness" (Goodhill V Ed) Harper and Row, Maryland (1979).

Mirabella, A et al.,(1967) Adaptation of loudness to monaural stimulation J.GemPsychol 76, 251-273. Quoted in chapter "Loudness Adaptation" written by Scharf, B in Hearing Res. and Theory, Vol.2, Ed. by Tobias and Schubert? Academic Press, 1983.

Melnick, W., Auditory sensitization, J.Acoust.Soc.Am.,Vol.46, No.6, 1583-1586(1969)

Moore, T.J., and Welsh, J.R., Forward and backward enhancement of sensitivity in the auditory system, J.Acoust. Soc.Am., Vol.47, No.2, 534-539, (1970).

Noffsinger, P.D., and Tillman,T.W., Post exposure responsiveness in the auditory system - immediate sensitizatlon J.Acoust.So.Am.Vol.47, No.2, 546-551 (1970).

Noffsinger, P.D. and Olsen, Post exposure responsiveness in the auditory system - Immediate sensitization, J.Acoust. Soc.Ame. Vol.47, 552-564 (1970).

Pickles, J.O., An introduction to the physiology of Hearing, Academic Press, New York, (1982).

Petty,J.M et al., A comparison of three methods for measuring auditory adaptation,J.Aud.Res.Vol.9, 352-357 (1969).

Ragini, M., Sensitization for contralateral auditory stimulation, Andise Independent Project submitted to the Univer-sity of Mysore (1985).

Rajanikanth, R, Ipsilateral Adaptation and changes in threshold. An Independent Project submitted to the University of Mysore (1985).

Sacharf, B, Loudness adaptation in Hearing Research and Theory, Ed.by Tobias, J.V. and schmbert. Eg., Vol.2, Academic Press, New York.

Sreedhara, R, Continuoug presentation of Narrow band noise and sensitization - An Independent Project submitted to the University of Mysore (1985).

Siegel, Nonparametric statistics for the behavioral sciences, P.79, (1956).

Small, A.M., Auditory adaptation, in modern developments in audiology Ed. by J.Jerger, Academic Press, New York (1963).

Spoendlin, H., Primarystructural changes in the organ of corti after acoustic over stimulation, Acta.0tol.71, 166-176,(1971).

Vyasamurthy, M.N, An objective verification of smalls model of loudness adaptation. Paper presented at the IX Annual Conference of Indian Speech and Hearing Association held at Bangalore (1977).

Vyasamurthy, M.N.,Objective Residual monaural loudness adaptation - A new concept, Ph.D. thesis, University of Mysore (1982).

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

Vyasamurthy, M.N., A treatise on auditory adaptation, Paper presented at the National Symposium on Acoustics and its Biological effects, held at Madras (1985 b).

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

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

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