# SENSITIZATION FOR CONTRALATERAL AUDITORY STIMULATION

Reg. No. 8406

An independent project work submitted as part fulfilment for First Year M.Sc, (Speech and Hearing) to the University of Mysore.

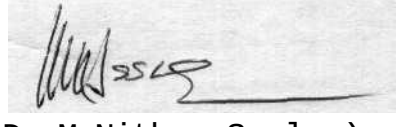
ALL INDIA INSTITUTE OF SPEECH AND HEARING

MYSORE - 570006.

In loving memory of 'Papu'

# C E R T I F I C A T E

This is to certify that the independent project entitled "SENSITIZATION FOR CONTRALATERAL AUDITORY STIMULATION" is the bonafide work done in part fulfillment for First Year M.Sc., (SPEECH AND HEARING) of the student with Registration Number: 8406



(Dr.M.Nithya Seelan)
Director
ALL INDIA INSTITUTE OF
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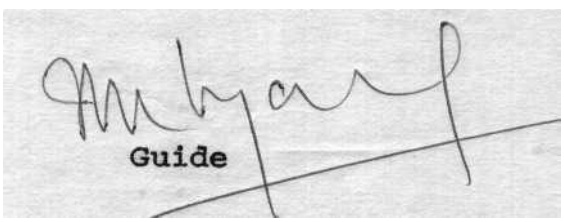
# C E R T I F I C A T E

This is to certify that the independent project entitled "SENSITIZATION FOR CONTRALATERAL AUDITORY STIMULATION" has been prepared under my supervision and guidance.



# D E C L A R A T I O N

This independent project entitled "SENSITIZATION FOR CONTRALATERAL AUDITORY STIMULATION" is the result of my own work undertaken under the guidence of Dr.M.N.Vyasamurthy, Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore

Date:29.4.85 Register No. 8406

**ACKNOWLEDGEMENTS.**

I express my deep gratitude to Dr.M.N.Vyasa-murthy, Lecturer in Audiology for his valuable guidance.

I am thankful to Dr.M.Nithya Seelan, Director, All India Institute of Speech and Hearing for providing me an opportunity to carryout the present study.

I extend my thanks to Dr (Miss) S.Nikam, Professor and Head of the Department of Audiology, for making available the instruments and materials connected with the study.

My sincere thanks to all my friends for their co-operation and timely help and especially those who underwent the experiment sparing their precious time.

I am grateful to Mr.Venkatesh and Mr.Prasada Rao for their help and patience which brought the project neatly typed and bound in the final form.

# C O N T E N T S

# C H A P T E R - 1

## INTRODUCTION

Adaptation is a phenomenon which characterizes all sensory systems. It is 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 as used to determine the threshold. (Small, 1963).

In vision, the adaptation effects are dark adaptation and light adaptation i.e., the increase or decrease in the threshold sensitivity occuring as a result of continued stimulation of eye by light. For some systems the sensation may disappear completely. Gustatory and olfatory senses are examples. In case of the sense of audition, there is merely a reduction in apparent magnitude or an increased threshold. (Small, 1963).

All our senses tend to become less responsive to stimuli after a certain durations of stimulation. Adrian (1928) and his colleagues have studied the phenomenon in sensory nerves and in end organs. They used the therm 'Adaptation' to describe the gradual setting down of neural activity as the stimulus is continued.

**SENSITIZATION OR FACILITATION:**

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

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

"Sensitization seems to be best produced by exposure intensities between 70 and 100 db SPL and is more pronounced for exposure frequencies below 1000 Hz than above (Hughes, 1954). The maximum sensitization occurs at the exposure frequency itself, but an effect can be seen earlier for test frequencies below the exposure frequency than for those above it (Noffsinger and Olsen, 1970). There also appears to be greater sensitization to a continuous test tone (Hughes, 1954) than to an interrupted one(Noffsinger and Tillman, 1970). Finally, sensitization is not restricted to the ear exposed (Hughes, 1954)" (Ward, 1973).

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

In essence, the revised model assumes that there are three types of adapted neural units viz., stable (a) and unstable ($a_1$ and $a_2$) 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 characterstic 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., 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.

**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 Jhonstone (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 neurones 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

(> 60db SPL) were greater in the presence of continuous tone (3KHz 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 "enhancement".

(4) FEX etal (1982) have concluded that the efferent terminals to the OHCs may participate in the recycling of the released neuro-transmitter using aspartate amino transferase (A A Tase). Interestingly, they have found the AA Tase like immuno reactivity in the Medical system of efferents but not in the lateral system.

(5) COMIS and WHITFIELD (1968) report that the acetyl-choline (neuro transmitter of ESIOHCs) is an exicitatory neurotransmitter.

(6) HOFFMANNETAL (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 inhibi-tory.

(8) STOPP ETAL (1983) suggest that the efferent system may increase the dynamic range of the neurones" (Vyasa-murthy, 1985).

The present study was aimed at studying sensitization in the test ear when the contralater-al ear is continuously exposed to a pure tone for 7 minutes, at 50 db HL (ANSI, 1969). Also, the study was designed to study the effect of frequency on 'Sensitization'.

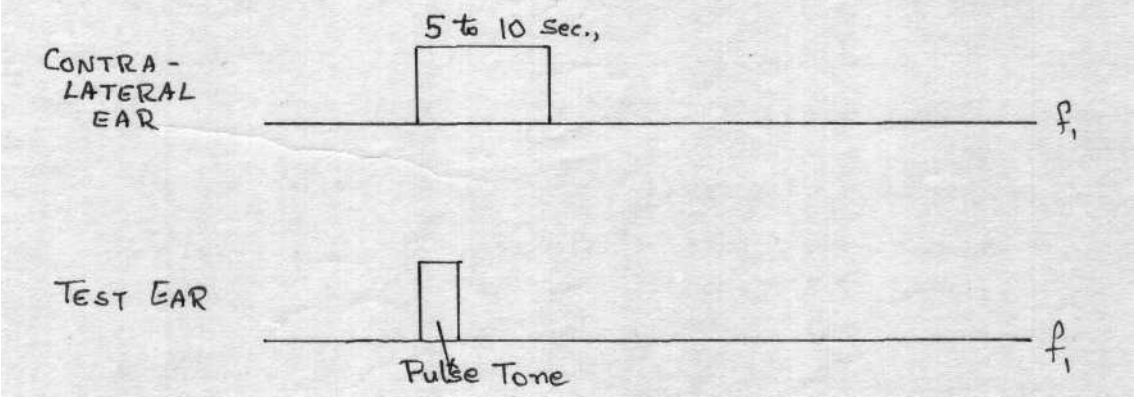
**HYPOTHESIS OF THE STUDY:**

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.

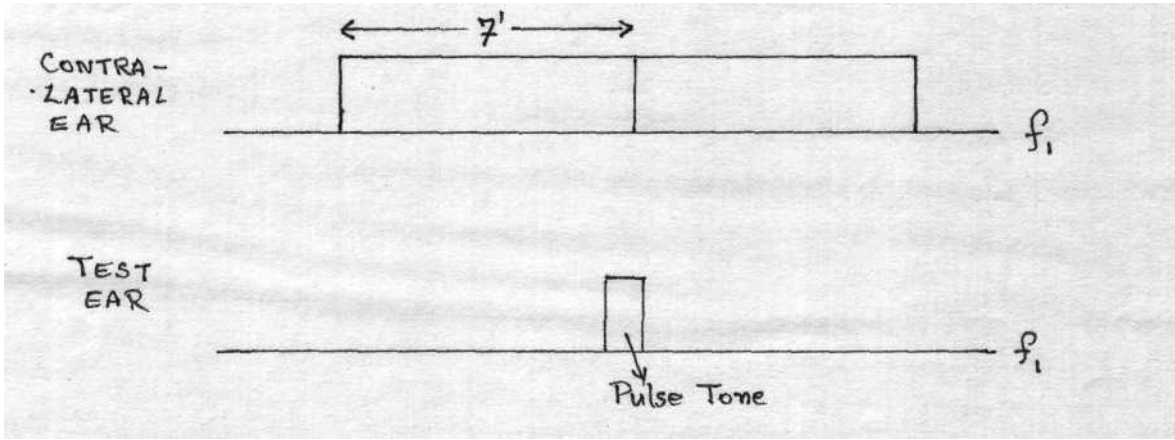
**CONDITION A:** Threshold for pulse tone obtained in the test ear in the presence of a pure tone at 50 dB HL (ANSI, 1969) in the contralateral ear (See fig. 1)

Figure 1:



**CONDITION B**: Threshold for pulse tone obtained in the test ear at the end of 7 minutes when the contra-later al ear is being continuously exposed beyond 7 minutes to a pure tone at 50dB HL (ANSI, 1969). (See fig. 2)

**FIGURE 2**




where f1 is 500 or 1000 or 2000 or 4000 Hz

**BRIEF PLAN OF THE STUDY:**

32 subjects were divided into 4 groups viz., ABCD. A, B, C and D groups were tested using 500, 1000, 2000 and 4000 Hz tones respectively. The threshold for pulse tone was obtained in the test ear in the presense of continuous tone in the contralateral ear at 50 dB HL (ANSI, 1969)

Then the contralateral ear was exposed to continuous tone at 50 dB HL (ANSI, 1969) 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 the threshold obtained prior to the continuous stimulation.

**DEFINITIONS OF THE TERMS USED:**

CONTRALATERAL EAR -- The ear in which the pure tone at 50 dB HL (ANSI, 1969) is presented continuously for 7 minutes and beyond.

TEST EAR -- The ear in which the thresholds for the pulse tone are obtained before and after the contralateral ear is exposed continuously to a pure tone.

NOTE: Pulse test tone frequency was same as the frequency of the continuous tone.

# C H A P T E R - II.

## REVIEW OF LITERATURE

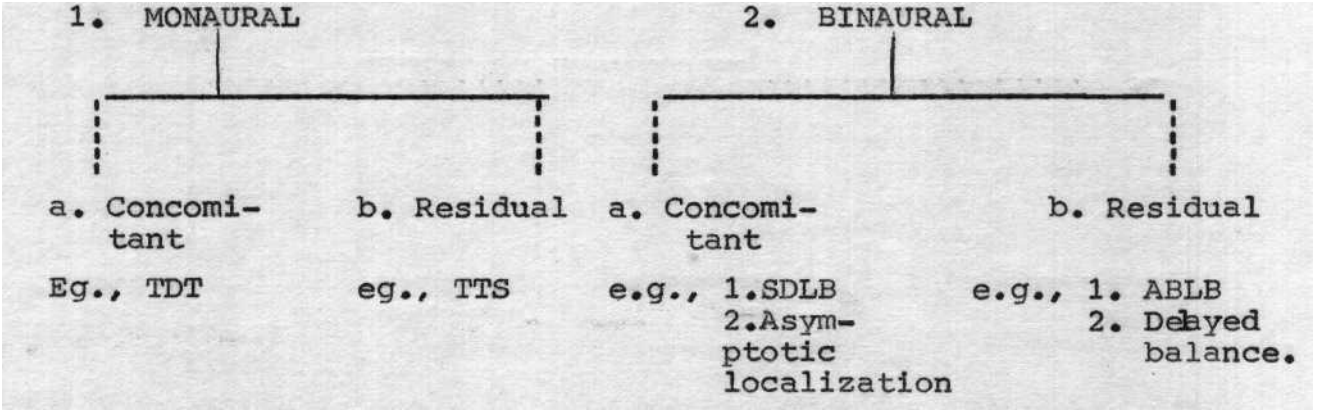
" Loudness adaptation means a decrease in the loudhess of a steady sound over time, according to YOUNG and SACHS (1973)it is "decrease in response magnitude "to a "constant stimulus level" (SCHARF, 1983).

The phenomena included under adaptation are distinguished in two different ways: concomitant or perstimu— latory adaptation and residual adaptation

Concomitant or perstimulatory adaptation is a shift in the laterlization of a diotic tone following a period of monotic adaptation to a steady sound (WARD, 1973).

Residual adaptation is when the adaptation is measured after withdrawl of the stimulus.

Adaptation varies depending upon the method used. Different methods give different amounts of adaptation. SDLB method developed by Hood gives more adaptation compared to other methods. The monaural techniques which do not use comparision stimuli show negligible adaptation. To avoid this confusion Ward has classified adaptation based on the technique used.




LAWRENCE et. al., (1949) observed that 9 observers with little training said 82% of the time that the loudness of an unchanging tone had decreased when it was 15dB above threshold; they said 66% of the time that loudness had increased when the tone was 70 dB above threshold. One interpretation of these results is that the loudness of a tone at a moderate level may increase slightly.

MIRABELLA et. al (1967) reported that 72 observers showed 2 to 5 dB of adaptation for a 3500 Hz tone over a 10 minutes period at 70 dB SPL and reverse adaptation (increased loudness) at 90 dB, in the "Tracking method".

SCHARF (1983) reports the effect of frequency is small and statistically insignificant. But at 10 and 15 dB SL, adaptation at 4 KHz does seem to exceed adaptation at the lower frequencies. AT 20 dB SL adaptation is small at all 4 frequencies,and at 30 dB SL adaptation is negative at all frequencies, meaning that observers reported a small

increase in loudness over the 2 minutes listening period. Thus, a clear effect of frequency on adaptation may be measurable most easily in the neighbour-hood of 10 dB SL.

SCHARF (1983) concluded from the survey of literature and from the new data offered, that, high frequency pure tonesadapt more than low frequency tones or than noises (whether broad-band or narrow-band). A sound presented alone adapts only if it is below 30 dB SL. Steady sounds adapt more than modulated sounds, and if the sound amplitude is modulated sufficiently adaptation may disappear altogether. No relation has been found 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.

By presenting a steady sound in one ear and an intermittent sound in the other, loudness adaptation can be induced. 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 alove. The role of interaural interaction and of lateralization in this adaptation is obscure, especially since the intermittent sound may induce some adaptation when in the same ear as the steady sound.

Following auditory 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).

Many studies have shown that when the ear is exposed to a continuous stimulus the threshold sensitivity does not always decrease under some conditions an enhancement of detectability may be observed.

VYASAMURTHY 1982, 1985 has proposed a revised model of adaptation. The essence of the revised model is as follows:

> Using a new method (VYASAMURTHY, 1977) of measuring adaptation, VYASAMURTHY collected data 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 assumes that there are three types of adapted neural units viz., stable (a) and unstable ($a_1$ and $a_2$) 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 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

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**LOUDNESS GRIN:** 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 counteracts the effect produced by the noise. Further, they have found that the sensitivity of the auditory neurous 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 (3KHz at 70 dB SPL). He has termed the

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**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 studies: (1) SPOENDLIN (1975) has established that the efferents to the inner hair cells (IHCs) synape 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).

**EVIDENCE FOR THRESHOLD IMPROVEMENT:**

Under certain conditions, for example, moderate expose intensity (60 - 100 dB SPL), low exposure frequency (500 Hz), the initial portion of the recovery function exhibits the auditory sensitization.

HUGHES (1954) called this increased responsiveness asimulate 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 pre-exposure threshold.

HUGHES is reported to have 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 on immediate threshold sensitization that grew to maximum size at about 30 sec. post exposure and then gradually disappeared by one minute.

In sensitization, greater sensitivity was observed 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 phenomenon has also been confirmed neurophysiologically.

There have also been some studies which indicate an enhanced sensitivity of the audiotory system following exposure to low intensity stimuli (5 — 20 dB SL) short duration (5 m.sec. - 10 sec) short recovery time (5 m.sec. - 1.0 sec.) (ZWISLOCKI, PIRRODA and RUBIN 1959, RUBIN 1960). This phenomenon 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 intense stimulation.

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

1. isolated sensitization
2. multiphasic behaviour (sensitization and desensitization - the bounce effect).
3. isolated desencitization.

These changes seem dependent on at least the following variables.

1. The frequency of and the frequency relationship between the test and exposing stimuli.
2. the intensity of the exposure stimulus
3. the duration of the exposure stimulus and
4. the condition applying during the exposure period example: 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 m sec. test pulses (250, 1000, 4000 Hz) following exposure tones of the 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 pre sented at four intensity levels namely 20, 60, 85 and 105 dB. Both DNTr and Tr procedures were employed.

The results of the experiment showed following facts:

1. isolated auditory sensitization is a real phenomenon, it can be demonstrated for both high and low frequency tones. Duration of such sensitization ranged from 20 to 100 sec. sensitisation that occurs later in the post-exposure time course usually following R-l 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 magnitude 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 sensi—tization. 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 the post exposure thresholds, first as an initial threshold shift that rapidly declines (R-l) and may yield to sensitized threshold then as a multiphasic process containing R-l, a bounce and a second period of desensitization (R-2) and finally as a long lasting period of desensi—tization that is most aptly described as R-2 above.

3. The sequence of post-exposure events described above is initiated at lower exposure levels following tones whose frequency is lower or equal to that of the tilt tone than following those with higher frequencies. Given this distinction, decreasing 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 the exposure tone period usually produces more post—exposure desensitization than is produced when the exposure tone is presented above.

Sensitization and desensitization reflect the state of at least 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 pre-synaptic electrical or electro - chemical hyper excitability i.e., hyper-polarization and desensitization reflects a reduced post synaptic receptive capacity.

**EVIDENCE FOR LOUDNESS GAIN:**

From HUGHES report (1954) it is known that a greater amount of transitory sensitization occured at 500 Hz than at higher frequencies (1000 Hz). HUGHES suggested that,the reason may be the higher frequencies were more effective in producing a positive temporary threshold shift and that this may have been interacting with the sensitization so as to produce an apparent lessening of the amount of sensitization. At 1000 Hz of the ispilateral ear showed the typical transitory sensitization recovery function with secondaryrise above the reference threshold level.

THOMAS J MOORE (1970) reported two 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 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 the ipsilateral ear.

In experimets 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 three minutes 65-90 dB SPL continuous tones and post exposure thresholds for tones of lesser frequency were examined. In most cases such procedures allowed demonstration of auditory sensitization that was not preceded or succeeded by desensitization was noted at 200 Hz following certain 500 Hz exposure tones and at 2000 Hz following certain 3000 Hz exposure tones.

There appears to greater sensitization to a continuous test tone (HUGHES 1954) than to an interrupted one (NOFFSINGER & 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 500 Hz after stimulation by a 500 Hz at 85 dB SPL tone in the contralateral ear as after ipsilateral stimulation.

NOFFSINGER and TILLMAN (1970) have also demonstrated this.

GUMLICH (1971) had 30 observers press a button the length of time necessary for the perceived duration of the button press to match the loudness of 1/3 octave band of noise. Results suggested a large increase in loudness over the set minute, as compared to a 2 - sec.noise, followed by the equivalent of a 12 dB drop during the next 55 minutes, since even after 60 min., the continuous noise was judged louder than a brief, 2 sec., noise of the same level. GUMLICH did not demonstrate loudness adaptation.

The effect of level on neurophysiological responses in the cochlear nucleus is seen in a report by ten, KATE et.al., (1977). For both kind of stimulus steady 100 sec., tone white noise, the decline in the firing rate as a function of duration increased with stimulus level whereas the initial spike rate increased with level, the firing rate after 100 sec., was faster to a low-level tone than to a tone 80 dB more intense. These findings are just the reverse of the psychophysical data from humans who show a decrease in adaptation with increasing level until, by 30 dB SL, simple loudness adaptation is hardly measurable.

**AN ACTIVE MECHANISM:**

Of recent, many investigators (KEMP, 1978, 1979; ZWISLOCKI, 1980, ZUREK, 1981; ZWISLOCKI and KLESSKY, 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 controlld 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 tunning curves.

CRANE (1983) suggests that the hyperactivity of the active mechanism map 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, WIDER-HOLD and KIANGL (1970) have reported that the electrical stimulation of C O C B results in the desensitization of the 'tips' of the tuning curves. Further, PICKLES (1982) has concluded that the electrical stimulation of C O C B reduces the response of the auditory nerve fibres to sound. VYASAMURTHY (1985) points out that this contro-versial issue can be easily resolved if we recall the Observations of BODIAN (1983); SIEGAL AND KIM (1982); and MOUNTAIN (1980) "It must be kept in mind that evidences for inhibitory role of the efferent innervation of the cochlea pertains to IHC system . . . . . function of ESIOHCs is yet to be known, presence of efferent innervation of the vestibular receptors suggests a general role for all labyrinthine efferent pathways such as the enhancement of sensitivity of the various receptors" (BODIAN, 1983).

"Electrical stimulation of COCB increased the damping of the cochlear partition" (SIEGAL and KIM, 1982) MOUNTAIN(1980).

Further, VYASAMURTHY (1981) writes "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 CRANES (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 of the 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 deceease 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 as suggested by FEX et.al., (1982)".

VYASAMURTHY (1985) has proposed a model for the efferent mechanisms by putting all the above pieces of information, the following neural models (Fig. 2.1 & 2.2) of the efferent mechanism during auditory adaptation have been proposed.

The model (fig.2.1) suggests that the efferent system passing through the medial superior olive (MSo)

FIG. 2.1

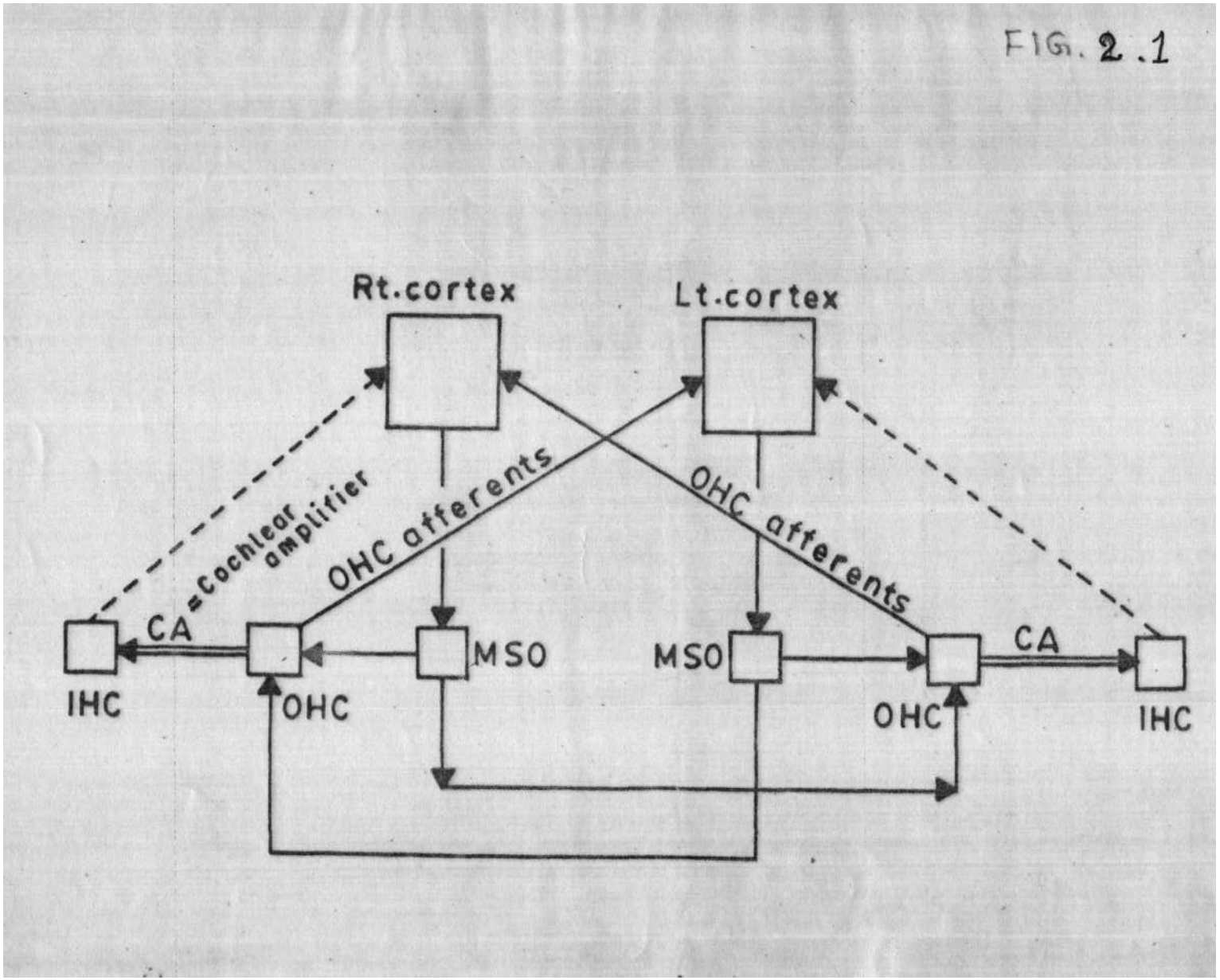
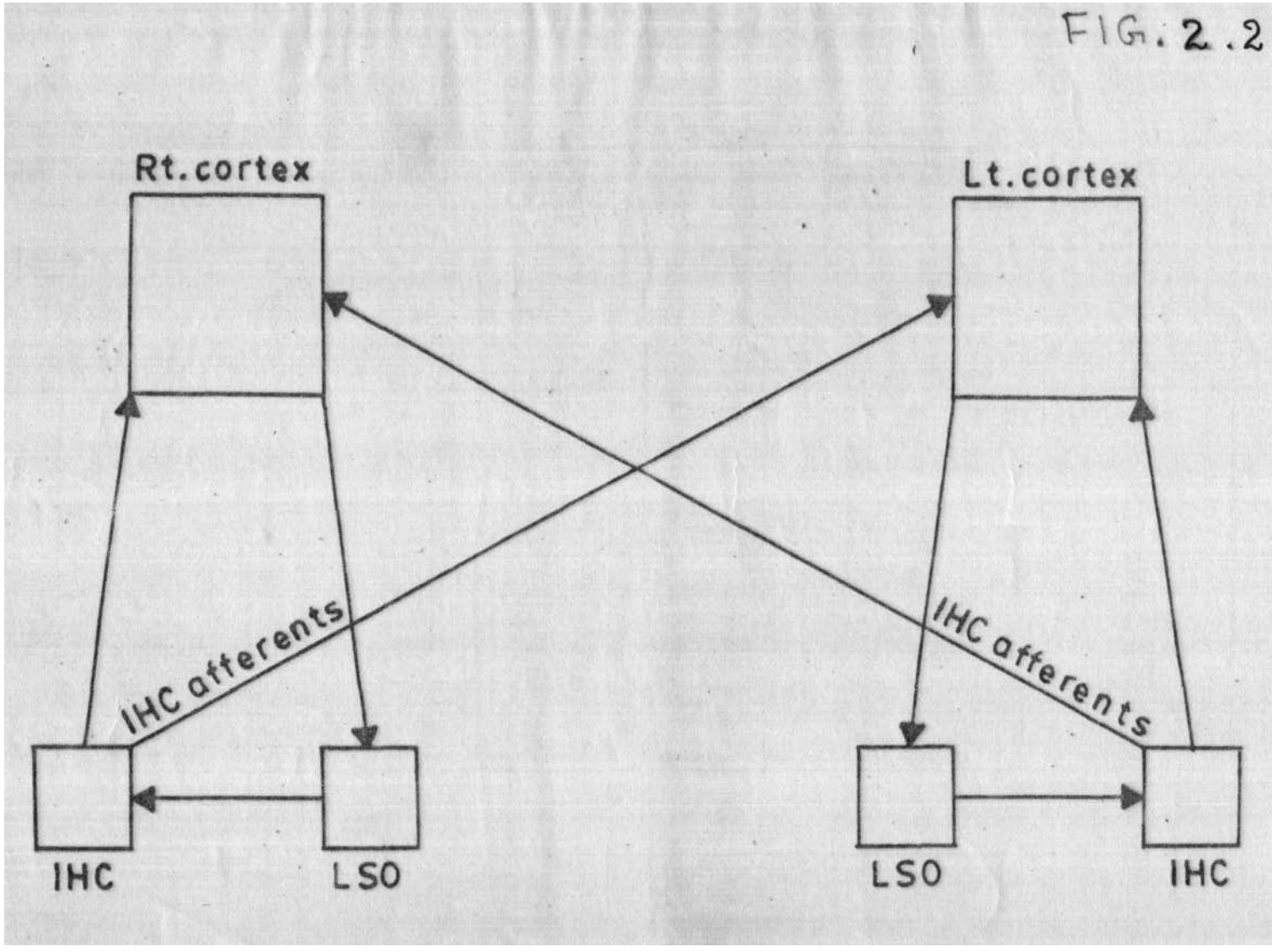

Rt.cortex
Lt.cortex
= Cochlear amplifier
OHC afferents
OHC afferents
CA
CA
MSO
MSO
IHC
OHC
OHC
IHC

FIG. 2.2


Rt.cortex
Lt.cortex
IHC afferents
IHC afferents
IHC
LSO
LSO
IHC

is responsible for the loudness gain (recycling of the released neurotransmitter) and the model (fig.2.2) suggests that the efferent system passing through the lateral superior olive (LSO)is responsible for loudness loss (a units are also responsible for loudness loss). The efferent system passing through LSO may be expected to release nor-adrenaline to inhibit the responses of the neurons innervating the IHCs as the efferent to the IHCs synapse with the afferent dendrites of IHCS. (Note: In 2.1 dotted line means not important for loudness gain; cochlear amplifier (CA) refers to active mechanism - see DAVIS, 1983).

# C H A P T E R - III

## METHODOLOGY

The present study was aimed at studying sensitization in the test ear when the contralateral ear is continuously exposed to a puretone for 7 minutes at 50 dB HL (ANSI, 1969).

Also the study was designed to study the effect of frequency on 'Sensitization'.

**SUBJECTS:**

32 adult normal hearing subjects within the age range of 17 yrs to 23 yrs were selected. The criteria for the selection of subjects was that they should have hearing threshold within 10 & 20 dB.

**INSTRUMENTATION:**

A dual channel clinical Audiometer, Beltone 200 C with TDH-49 ear phones, enclosed in MX 41/AR ear cushions was used for testing.

Fig. 3.1 test illustrates the operational availabilities in Beltone 200 C.

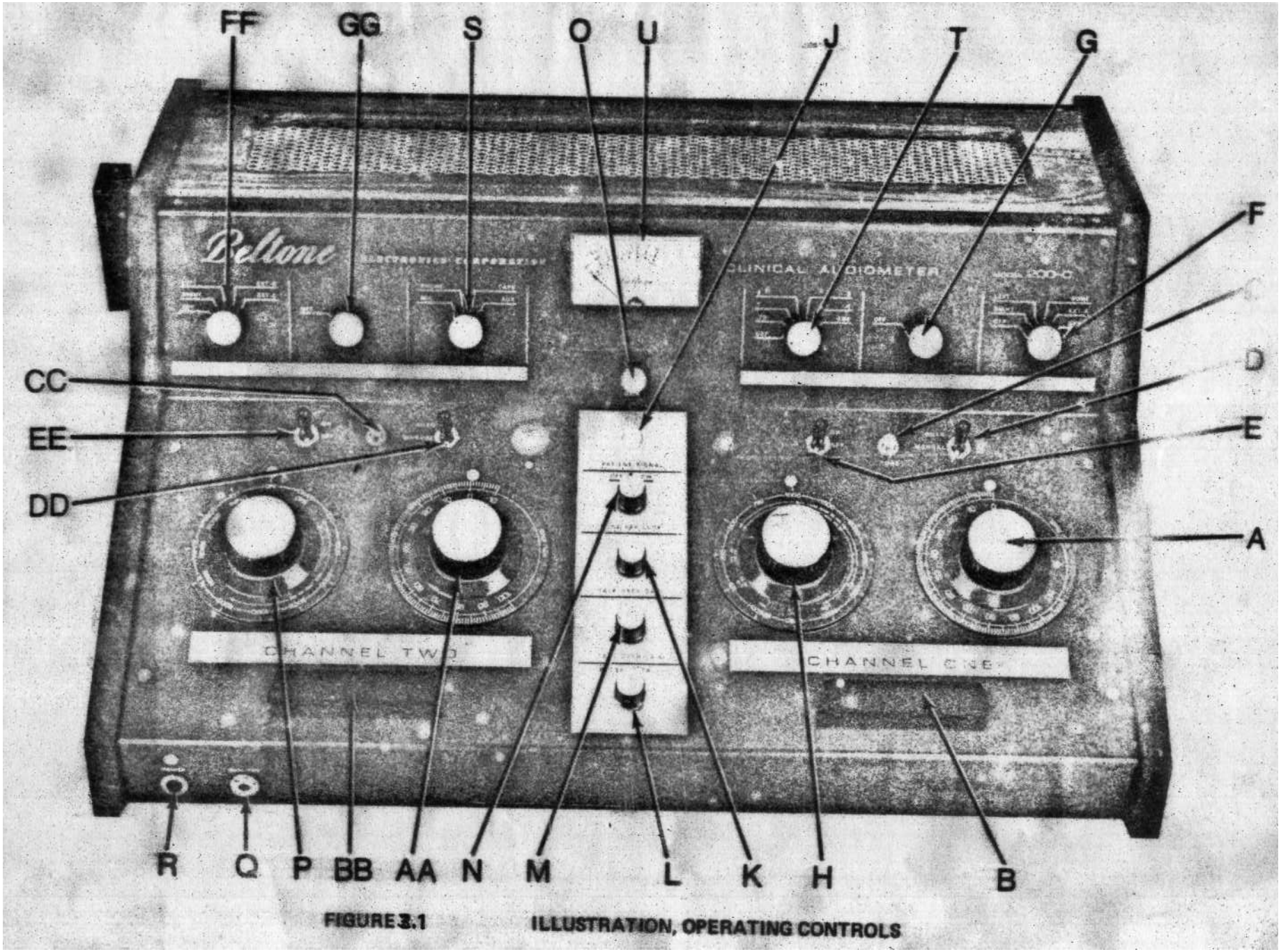



FIGURE 3.1 ILLUSTRATION, OPERATING CONTROLS

This audiometer provides for testing frequencies from 125 Hz to 8000 Hz. The hearing level ranges from 0 dB to 110 dB. Pulse. pure tone may be presented by selecting the automatic position. The tone is presented at the rate of 0.3 sec "on" & 0.3 Sec "off". Pulse tone and continuous tone were presented through the earphones with the following setting —

**FRONT PANEL INDICATORS - CONTROL KNOBS OF BELTONE 200 C.**

| | | | |
|---|---|---|---|
| A | (AA) | ... | out put (Hearing level control) |
| B | (BB) | ... | tone interruptor |
| C | (CC) | ... | Tone 'on' lamp |
| D | (DD) | ... | Automatic (manual switch) |
| E | (EE) | ... | Tone reversing switch |
| F | (FF) | ... | Out put selector |
| G | (GG) | ... | Monitor control |
| H | | ... | Frequency |
| I | | ... | Patient signal lamp |
| K | | ... | Talk back gain |
| L | | ... | Talk over switch |
| M | | ... | Talk over gain |
| N | | ... | Tone bar lock |
| O | | ... | Vu meter selector switch |
| P | | ... | Frequency input |
| Q | | ... | Monitor ear phone |
| R | | ... | Power |
| S | | ... | Speech Unit |
| T | | ... | SISI |
| U | | ... | Vu meter |
| X | | ... | Channel one Vu meter gain control |
| XX | | ... | Channel two Vu meter gain control |

The out put selector of both the channels to the 2 earphones to both the ears.

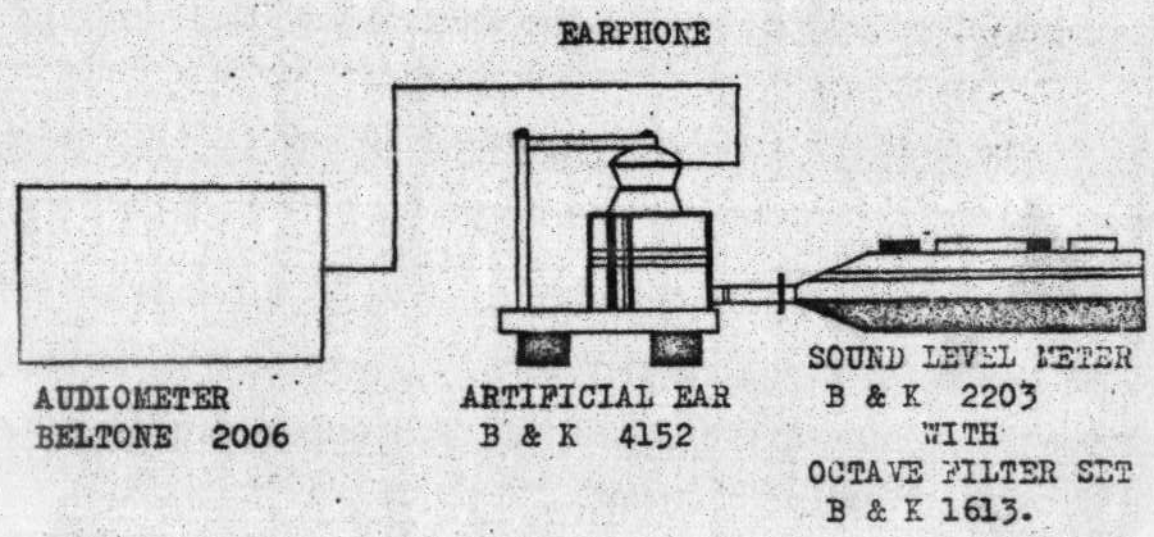
3.3

**CALIBRATION PROCEDURE USED:**

The duall channel clinical audiometer (Beltone 200 c) was claimed by the manufacturer to be calibrated to the ANSI (1969) standards. However, to ensure accuracy in calibration, the audiometer was calibrated periodically during the study according to the guidelines given by WILBER (1978).

Fig. 3.2 illustrates the set up for cabiration.

The audiometer Beltone 200 c was tuned 'ON' and was allowed to warm up. The sound level meter (B&K 2203) was set as follows. The meter switch was turned to 'external filter' and to 'slow'. The signal ear phone (T D H 49 with MX 41/AR ear cushions) of the audiometer was removed from the head band and was placed over the coupler of the artificial ear (B & K 4152). The ear phone was held in place by means of a tension of the artificial ear and was adjusted to 0.5 kg of pressure. After initial placement of the earphone on the coupler, a low frequency tone (250 Hz) was introduced and the ear phone was readjusted until the sound level meter needle read the highest intensity. This is said to ensure best placement according to WILBER (1978).




G. 3.2: Block Diagram of Pure Tone and Narrow Band Noise calibration.

The frequency selector of the audiometer was set to 1000 Hz. The octave filter (B & K 1630) of the sound level matter was set to 1000 Hz. The audiometer was set to right ear phone (selector switch) and the tone was continuously 'on'. The hearing level dial was set to 60 dB for the frequency chosen. The reading on the sound level meter was noted. Similarly other frequencies (250 Hz, 500 Hz, 2000 Hz and 4000 Hz) were checked. The audiometer output intensity was within permissible limits.

To check the linearity of the attenuator of the audiometer, a similar set up was used. The range finder was set to 120 dB. The hearing level dial was set at maximum and out put of the sound level meter was noted. The hearing level dial was dropped in 5 dB steps and the reading on the sound level meter was noted for each 5 dB drop. The reading on the sound level meter that the audiometer linearity was satisfactory.

**ENVIRONMENT:**

The audiometric tests were performed in a sound treated two room situation. The control panel of the audiometer was not visible to the subject. Theambient

noise levels in these rooms were within the maximum permissible noise levels.

**INSTRUCTIONS:**

The subject was instructed

"you are going to hear a continuous tone in one ear and pulse tone in the other ear. You should respond only to the pulse tone. The continuous tone will continue for more,than 7 minutes. At the end of the 7 minutes, I will ask you to respond, then, again you should respond only to the pulse tone".

**PROCEDURE:**

32 subjects were divided into 4 groups viz., ABCD. A, B, C and D groups were tested using 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz tones respectively.

The threshold for pulse tone was obtained in the test ear in the presence of continuous tone in the contralateral ear at 50 dB HL (ANSI, 1969)

Then the contralateral ear was exposed to continuous tone at 50 dB HL (ANSI, 1969) for 7 minutes.

The threshold for pulse tone was obtained in

the test ear at the end of 7 minutes, while the continuous tone continued even after 7 minutes.

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

In other words the sensitization was determined by subtracting the thresholds obtained in the condition B from thresholds obtained in the condition A.

# C H A P T E R  IV

## RESULTS AND DISCUSSION

Tables 1, 2, 3 and 4 reveal the sensitization values at 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz respectively. Means and Standard deviations are also presented in the tables

TABLE: 1 Sensitization at 500 Hz

| GROUP A | THRESHOLD OBTAINED IN CONDITION A | THRESHOLD OBTAINED IN CONDITION, B |
|---|---|---|
| 1 | 15 dB | 10 dB |
| 2 | 20 dB | 15 dB |
| 3 | 20 dB | 15 dB |
| 4 | 15 dB | 10 dB |
| 5 | 15 dB | 10 dB |
| 6 | 10 dB | 5 dB |
| 7 | 15 dB | 10 dB |
| 8 | 15 dB | 10 dB |
| | MEAN: 15.625 | MEAN: 10.62 |
| | S.D. 2.9973 | S.D. 2.9973 |

<

**TABLE: 2**

**SENSITIZATION AT 1000 Hz.**

| GROUP B | THRESHOLD OBTAINED IN CONDITION H | THRESHOLD OBTAINED IN CONDITION B |
|---|---|---|
| 1 | 15 dB | 10 dB |
| 2 | 15 dB | 10 dB |
| 3 | 20 dB | 15 dB |
| 4 | 10 dB | 5 dB |
| 5 | 15 dB | 10 dB |
| 6 | 15 dB | 10 dB |
| 7 | 15 dB | 10 dB |
| 8 | 10 dB | 5 dB |
| | MEAN: 14.37 | MEAN: 9.37 |
| | S D : 2.99 | S.D : 2.99 |

**TABLE: 3.**

**SENSITIZATION AT 2000 Hz**

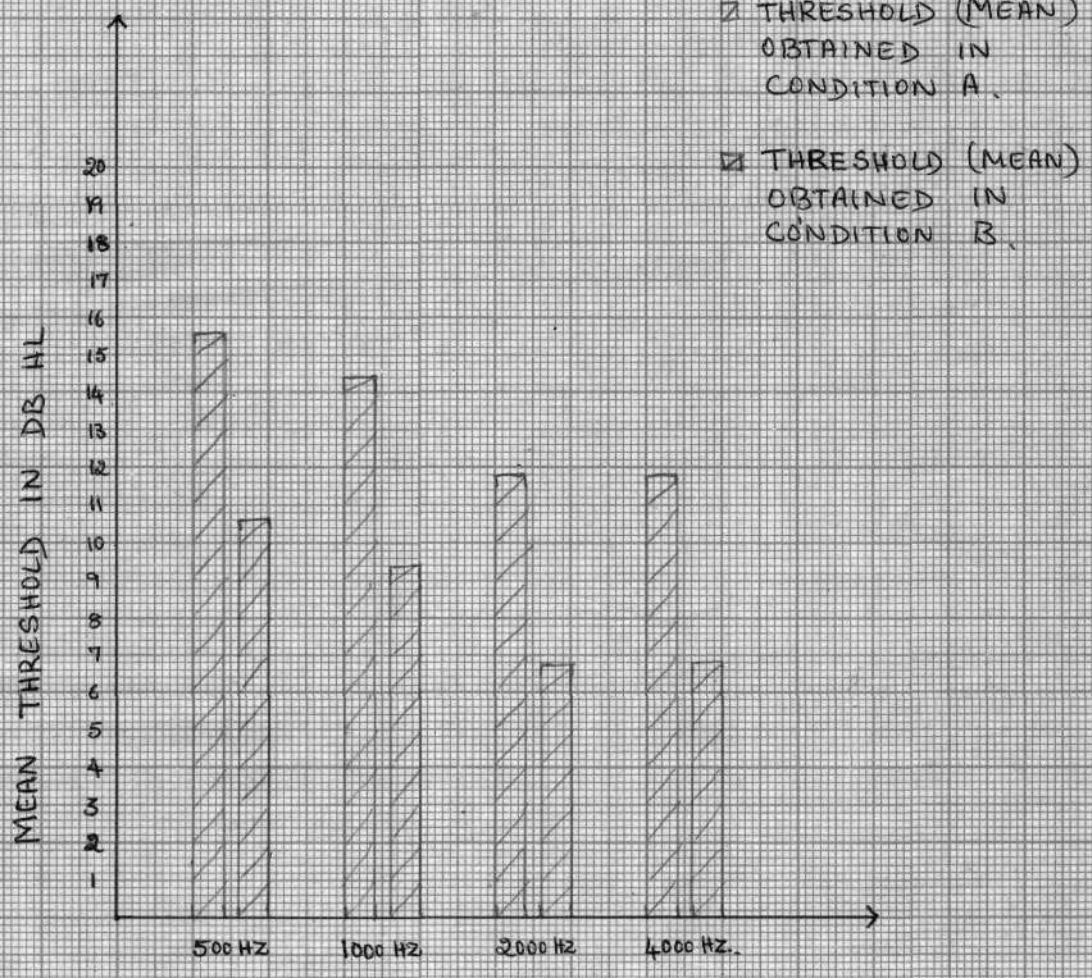
| GROUP C | THRESHOLD OBTAINED IN CONDITION A | THRESHOLD OBTAINED IN CONDITION B |
|---|---|---|
| 1 | 15 dB | 10 dB |
| 2 | 15 dB | 10 dB |
| 3 | 10 dB | 5 dB |
| 4 | 10 dB | 5 dB |
| 5 | 10 dB | 5 dB |
| 6 | 10 dB | 5 dB |
| 7 | 10 dB | 5 dB |
| 8 | 15 dB | 10 dB |
| | MEAN: 11.87 | MEAN: 6.87 |
| | S D : 2.42 | S D : 2.42 |

**TABLE: 4**

**SENSITIZATION AT 4000 Hz**

| GROUP D | THRESHOLD OBTAINED IN CONDITION A | THRESHOLD OBTAINED IN CONDITION B |
|---|---|---|
| 1 | 10 dB | 5 dB |
| 2 | 10 dB | 5 dB |
| 3 | 15 dB | 10 dB |
| 4 | 15 dB | 10 dB |
| 5 | 10 dB | 5 dB |
| 6 | 10 dB | 5 dB |
| 7 | 15 dB | 10 dB |
| 8 | 10 dB | 5 dB |
| | MEAN: 11.87 | Mean: 6.87 |
| | S.D . 2.42 | S.D . 2.42 |

The above results are shown in the graph.




GRAPH 4.1 : SENSITIZATION AT 500HZ, 1000HZ, 2000HZ AND 4000HZ.

From the tables it is obivious that the thresholds in the test ear obtained after continuous stimulation in the non test ear (i.e., contralateral ear) show sensitization i.e., the thresholds of the test ear after continuous stimulation in the non test ear become better.

The improvement in threshold at all these frequencies (500 Hz, 1000 Hz, 2000 Hz and 4000 Hz) is 5 dB.

From the results of the present study it is clear that the magnitude of sensitization is same at all frequencies tested i.e., the frequencies of the test stimulus have no effect on sensitization.

WILCOXON matched pairs signed ranks test was used to find out whether there is significant difference between the threshold obtained in condition A and condition B. The analysis of the data for significance of difference shows that the thresholds obtained in condition A and Condition B significantly differ at all the frequencies tested.

The investigation has been reported by many investigators — HUGHES, 1954; NOFFSINGER and OLSEN, 1970;

GERKEN, 1984; VYASAMURTHY, 1982; STOPP, 1983; FEX et al, 1982; MOORE 1968; CODY & JOHNSTONE, 1982; KEMP, 1978; MOUNTAIN D.C., 1980; SEIGAL and KIM, 1982.

The survey of literature shows 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 sensitization obtained in the present study with the magnitude of sensitization reported by other investigators.

**DISCUSSION:**

The present study shows that when one ear is adapted for 7 minutes or more using continuous pure tone stimulus the contralateral ear shows improvement in threshold of hearing or shows sensitization. This sensitization in the ear opposite to the adapted ear has been observed at all the frequencies tested (500 Hz, 1000 Hz, 2000 Hz and 4000 Hz).

As per the revised model of adaptation (VYASAMURTHY, 1982) loudness gain is expected in the ear opposite to the adapted ear due to efferent action i.e., in his study he reports that '$a_2$' units will be produced in the ear opposite to the adapted ear, and he assumes

that this may be responsible for greater adaptation observed in the SDLB technique, when adaptive stimulus of 60 dB is used, when adaptive stimulus of 80dB is used (in SDLB technique) he propsed there will be loudness gain and loudness loss in the ear opposite to the adapted ear.

The combined action of the efferent system in the comparision ear is expected when 80 dB adaptive stimulus is used in SDLB technique.

He proposes that:

$$L\ L_{80} -- L\ L_{60} = L\ L_{80}^{*}$$

Where $L\ L_{80}$ - Loudness loss at 80 dB in the adapting ear.

$L\ L_{60}$ = Loudness loss at 60 dB in the adapting ear.

$L\ L_{80}^{*}$ = Loudness loss at 80 dB in the comparision ear.

According to above equation the increase in the adaptation which results in the adapting ear by increasing the intensity of the adapting stimulus from 60 dB to 80 dB is equal to the loudness loss produced in the comparision ear through efferent action. The 'levelling off' of

adaptation observed in SDLB technique has been explained in terms of 'Eclipse phenomenon' (VYASAMURTHY, 1982).

Eclipse phenomenon is the one in which the increase in the amount of adaptation which should result in the adapting ear due to the increase in the intensity of the adapting stimulus is eclipsed by the combined action of the two efferent (ESIIHC & ESIOHCS) systems in the comparision ear.

Thus the revised model is based as the assumption the efferent system innervating the outer hair cells (or MSO system) is responsible for loudness gain in the ear opposite to the adapted ear.

The neural model of the efferent mechanism for loudness gain has also been proposed (VYASAMURTHY, 1982).

The results of the present study clearly show that the ear opposite to the adapted ear exhibits sensi—tization. In none of the subjects tested the threshold in the ear opposite to the adapted ear didnot become worse in condition B.

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 (M S O system).

FEX et al (1982) have suggested the efferent system innervating the outer hair cells may participate in recycling of the released neuro transmitter through AAT (Asparatate Amino Transferase) activation.

Additionally the release of eukephalin like peptides (putative heuroactive substances) in the efferent terminals of OHCs may also contribute to the sensitization observed in the present study

The results of the present study thus support the revised model of adaptation (VYASAMURTHY, 1982).

# C H A P T E R - V.

## SUMMARY AND CONCLUSION

The aim of the present study was to collect data on 'sensitization' in the test ear when the contralateral ear is continuously exposed to a puretone for 7 minutes at 50 dB HL (ANSI, 1969).

32 subjects who had hearing thresholds between 10 and 20 dB were divided into 4 groups viz., A, B, C and D. Each group was tested using 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz tones respectively. The thresholds of the test ear for the pulse tone were obtained before and after exposing the contralateral ear to a continuous stimuls. The results showed an improvement of 5 dB in thresholds at all the above frequencies.

The results of the present study show that the ear opposite to the adapted ear exhibits sensitization. This is an evidence that some facilitatory process may be operating in theear 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.

The results of the present study may have some implications:

'Sensitization' observed in the ear contralateral to the adapted ear, may be related to a facilitation process - this facilitation process, as mentioned earlier, maybe viewed interms of synaptic efficacy brought about by the ESIOHCs.

In the revised model of adaptation, it is assumed that when the ear is adapted for more than 7 minutes '$a_2$' units (responsible for loudness gain) would be produced. These units are assumed to be responsible for greater adaptation observed in SDLB technique. Many studies have demonetrated that adaptation obtained in SDLB technique is more than the adaptation obtained in monaural techniques for identical stimulus intensities. The results obtained in the present study reveals that when the ear is adapted continuously for 7 minutes or more, a facilitatory process may be initiated in the ear 'contralateral to the adapted ear may be linked to '$a_2$' units assumed in the revised model of adaptation. The foregoing facts support the assumption that greater adaptation observed in SDLB technique may be due to "loudness gain" in the comparision ear.

The present study also reveals that 'loudness gain' can be possibly studied through threshold measurements. However, further studies are required to find out whether

"sensitization" and "loudness gain" involve the same mechanisms.

**RECOMMENDATIONS:**

1. Magnitude of sensitization may be determined at different levels of intensities.

2. Magnitude of sensitization may be studied using different test and adapting frequencies.

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