SENSITIZATION FOR CONTRALATERAL AUDITORY STIMULATION

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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 duration of stimulation. Adrian (1928) and his colleagues have studied the phenomenon in sensory nerves and in end organs. They used the term "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 (Noff singer 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 magnitude 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, 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 characteristic frequency (frequency of the adapting stimulus). a, 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,' 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 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 (A) 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) reported 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 01ivo-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 **increase** 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). He has termed the facilitation by sustained tone "enhancement". He has also speculated that the efferent action might be responsible for "enhancement".

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

(5) COMIS and WHITFIELD (1968) reported that the acetylcholine (neuro transmitter of ESIOHCs) in an exicitatory neurotransmitter.

(6) HOFFMANN et. al (1983) have detected enkephalin like peptides (putative neuro-active substances) in the efferent terminals of OHCs.

(7) PICKLES (1982) reported that the centrifugal fibres to the cochlear nucleus are both excitatory and inhibitory.

(8) STOPP et.al (1983) suggested that the efferent system may increase the dynamic range of the neurones (Vyasamurthy, 1985).

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

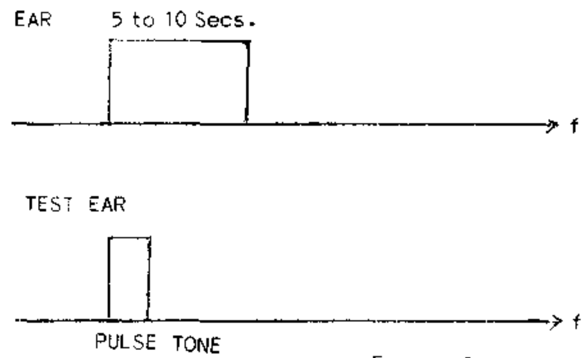
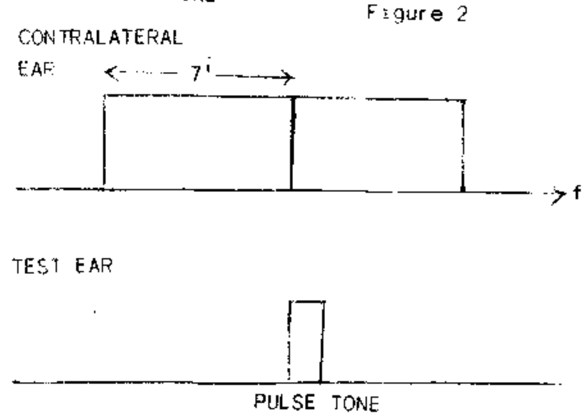


Figure 2



Where f1 is 500 or 1000 or 2000 or 4000 Hz.

Methodology:

subjects:

32 adults normal hearing subjects within the age range of 17 years to 23 years were selected. The criteria for the selection of subjects was thay 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.

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.

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 a 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.

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 A		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 200 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 400 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

From the Tables it is obvious that the thresholds in the test ear obtained after continuous stimulation in the non-test ear (i.e., contralateral ear) show sensitization. The improvement in threshold at all these frequencies (500 Hz, 1000 Hz, 2000 Hz and 4000 Hz) is 5 dB and the frequencies of the test stimulus have no effect on sensitization.

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

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 tc 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 80 dB is used (in SDLB technique) he proposed there would be loudness gain and loudness loss in the ear opposite to the adapted ear.

The combined action of the efferent system in the compaision ear is expected when an 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 adapting ear.

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

$L\ L^*_{80}$ = Loudness Loss at 80 dB in the companision ear.

According to the 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' (VYASA-MURTHY, 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 (VYASA-MURTHY, 1982).

The results of the present study clearly show that the ear opposite to the adapted ear exhibits sensitization. In none of the subjects tested the threshold in the ear opposite to the adapted ear did not become worse in condition B.

The fact that the ear opposite to the adapted ear exhibits sensitization in an evidence that some facilitatory process may be operating in the ear opposite to the adapted ear. This facilitatory process may be viewed in terms of synaptic efficacy brought about by the efferent system innervating the outer hair cells (M S 0 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 neuroactive 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).

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