AUDITORY BRAIN STEM EVOKED RESPONSE AND EFFERENT ACTION

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TO MY PARENTS

CERTIFICATE

This is to certify that the Dissertation entitled "AUDITORY BRAIN STEM EVOKED RESPONSE AND EFFERENT ACTION" is the bonafide work done in part fulfilment for Final Year M.Sc, Speech and Hearing of the student with Register No.

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CERTIFICATE

This is to certify that the Dissertation entitled : "AUDITORY BRAIN STEM EVOKED RESPONSE AND EFFERENT ACTION" has been prepared under my guidance and supervision.

DECLARATION

This dissertation entitled "AUDITORY BRAIN STEM EVOKED RESPONSE AND EFFERENT ACTION" - is the result of my own study undertaken under the guidance 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.

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INTRODUCTION

INTRODUCTION

"The brain as an object of study, has its own fascinating quality of being an almost unfathomably complex organ of behavior, memory and learning, as well as of creativity and conciousness. Study of the spontaneous activity of the brain has a long history and a well established place in clinical medicine and so does brain electrical activity which is brought about by an experimenter or clinician i.e. it is evoked". (Jewett 1983).

Stem

Auditory Brain/Evoked Response Audioraetry (ABR) became popular in clinical audiology and otology because of reproducibility, ease of administration, low inter and intra subject variability and accuracy in estimating hearing sensitivity (Clemis and McGee 1979; David 1976; Davis and Hirsh 1976; Galambos et al 1977; Morgan et al 1971; Shea et al 1980).

ABR recordings consist of series of seven waves which can be recorded using electrodes in response to a series of stimuli. Usually 1000 or 2000 are used and the response is extracted by means of online averaging. The waves are generally agreed to have the following provenance (Beagley and Sheldrake 1978).

Wave-I from the auditory trunk

Wave-II from the cochlear nucleus

Wave-III from the superior olivary complex

Wave-IV from the nucleus of the lateral lemniscus
Wave-V from the inferior colliculus
Wave-VI from the medial geniculate nucleus
Wave-VII from the primary auditory area.

ABR can be made use of in studying the changes in the cochlear response objectively. It can also be made use of in studying the changes in the Medial Geniculate Body (MGB).

Ward (1973) distinguishes the phenomena commonly included under adaptation in two different ways: Whether they are observed during or after exposure to the acoustic stimulus (concomitant or residual respectively) and whether they require one ear (Monaurel) or two (Binaural) for their measurement.

Many techniques have been developed for measuring adaptation in its development, asymptotic state and recovery. The ABLB technique (Alternate binaural loudness balance), Delayed balance technique, SDLB technique (simultaneous dichotic loudness balance). These techniques are binaural i.e. require two ears for their measurement. The monaural technique . is the MH technique (Monaural heterophonic).

The SDLB technique is the one that has been more frequently used for measuring all of the aspects of adaptation. Adaptation measured using this technique is called perstimulatory adaptation.

Ward (1973) has explained 'perstimulatory adaptation' in the following manner:

"Perstimulatory adaptation therefore appearing only when both ears are stimulated simultaneously, is a complex phenomena involving central interaction of the auditory pathway including, perhaps, efferent action of the non-exposed ear".

In the MH technique, only one ear is required. The adapting stimulus and comparison stimulus are of different frequencies.

Weiler, Linz and Glass (1979) reported that there was no significant correlation between the magnitude of adaptation measured using SDLB and MH techniques.

The available data for SDLB at 60 dBSPL adapting intensity is 20 dB (Mean) (Weiler 1972) and for MH technique it is 17.75 dB, The fact that the SDLB method yields more adaptation than the MH method at identical intensity and at identical adapting period, is an indication for the influence of the comparison ear in the results.

Thus the above findings show that SDLB and MH techniques yield different results although both measure concomittant adaptation. This could be because as suggested by Weiler and

Davis (1975) the monaural technique measures a different auditory change due to adaptation than that measured by simultaneous binaural balances.

The greater amount of adaptation using SDLB method than MH method, has been attributed to binaural interaction. The exact mechanism of binaural interaction is not known.

Vyasamurthy (1982) defines binaural interaction as the efferent action of monaural stimulation on the periphery of the contralateral auditory system.

This definition is based on his study - "Since adaptation of the comparison ear through cross over of the adapting stimulus (80-40=40 dB) can be considered negligible. The influence of comparison ear (CE) can be expected through binaural interaction. The result X_b (SDLB) > X_b(MH) can be expected provided loudness of the post-adapted test tone in the comparison ear > loudness of the pre adapted test tone in the comparison ear ($*L_{tba} > *L_{tbb}$). But $*L_{tbb} > *L_{tbb}$ can be expected only when $*X_b < 0$ (adaptation) in the comparison ear is less than zero) i.e. a_2 (unstable adapted neural units) units are produced in the comparison ear. This means that 'negative adaptation' (or action of the efferent system innervating the outer hair cells) must be present in the comparison ear". Vyasamurthy (l982).

The relation of adaptation to the intensity of the adapting stimulus is in conflict. Simple adaptation (Loudness adaptation measured at the intensity of the adapting stimulus) is linear with the intensity of the adapting stimulus upto 60 dB. Beyond 60 dB there is 1eveling off of adaptation. The levelling off of simple adaptation above 60 dB may be attributed to the adaptation of comparison ear (Jerger). According to Palva and Karja (Weiler 1972), levelling off of simple adaptation may be due to spread of stimulation to internal hair cells.

Vyasamurthy (1982), objectively answered the controversies prevailing with regard to the levelling off of simple adaptation above 60 dB.

In the study by Vyasamurthy (1982), the influence of adaptation of non-test ear was not made use of for measuring loudness adaptation unlike SDLB technique.

Simple adaptation due to spread of stimulation to inner hair cells, could no more be entertained in the light of the results of his study.

These explanations may thus not be tenable. Levelling off of adaptation was explained on the basis of the 'eclipse phenomenon'.

From available data X_b (at 60 dB) in SDLB = X_b(at 80 dB) in SDLB = 20 dB (Weiler et al 1982) $X_b > 0$ can be expected in the adapting ear during post-adapted balances in SDLD method.

In the comparison ear, two conditions will prevail during the post adapted balance (1) the conditions due to 'binaural interaction' and (2) the conditions due to adaptation of the comparison ear through cross over of the adapting stimulus.

The combined effects of the two conditions in the comparison ear would be loudness of the post-adapted test tone in the comparison ear will be louder than the pre adapted test tone of the comparison ear at 80 dB SPL.

At high intensity levels i.e. 80 dB SPL, there exists 2 types of efferent systems. The efferent system which functions at low levels ($\leq$60 dB) is concerned with 'negative adaptation' and the efferent system which functions at high intensity levels is concerned with 'inhibition' i.e. arresting or inhibiting the sensory inputs from firing impulses. The efferent system responsible for negative adaptation is the efferent system innervating the outer hair cells and responds to low intensity sounds. The efferent system responsible for 'inhibition' is the efferent system innervating the inner hair cells and responds to high intensity sounds only.

Thus, according to the eclipse phenomenon, the increase in adaptation in the adapting ear, resulting from the increase in the intensity of the adapting stimulus will be eclipsed by binaural interaction in SDLB method, because at 80 dB two efferent systems operate together and the net result will be that the loudness of the post adapted test/in the comparison ear will be almost equal to the preadapted test tone in the comparison ear. Thus, there is no loudness gain in the comparison ear as seen in SDLB method at 60 dB i.e. at 80 dB the efferent system innervating the IHC leading to inhibition is also present. In SDLB method, there is only loudness gain in the comparison ear at 60 dB and there is loudness gain and loudness loss in the comparison ear at 80 dB.

In support of this phenomenon of loudness gain, many studies can be mentioned.

Cody and Johstone (1982) found a reduction in ipsilateral desensitization in the presence of contralateral stimulation.

Kemp (1978) saw that an acoustic click presented through a speaker? produced a brief wave of pressure in the ear. Wilson (1980) did a replication of Kemp (1978) study and found the same results.

Moutain (1980) also observed similar sound pressure and showed that the amplitude of the reflected distortion tone could be affected by stimulation of the crossed olivocochlear bundle (COCB).

Gerken (1984) reports that the evoked response amplitude is greater in the presence of a continuous tone or an amplitude enhancement of wave-V.

Siegel and Kim (1971) found that stimulation of the efferent nerve fibres to OHC causes a change in the non linear behavior of acoustically and electrophysiologically recorded responses.

Flock (1982)- through stimulation of efferent nerve fibres, the OHC may exhibit active components injecting energy into the auditory system.

Starr and Wernick (1968) found that with the cochlea destroyed, stimulation of the COCB increased the spontaneous activity of 42% and reduced the activity of 16% of cells recorded in the cochtear nucleus.

Need for the study:

The present study has been designed to observe 'Binaural Interaction' i.e. the efferent action of monaural stimulation on the periphery of the contralateral auditory system. (Vyasamurthy 1982)using ABR.

When a continuous stimulus is presented in one ear for 7 min.or more, loudness gain can be expected in the contralateral ear.

The expected loudness gain in the contralateral ear, can be observed in the form of an increase in absolute amplitude and a reduction in absolute latency of waves I of ABR,

Furthermore, hypersensitivity as reported by Gerken (1984) can also be studied by observing changes in the VIth peak of ABR in the presence of continuous ipsilateral stimulation.

This increase in hypersensitivity in the neuron of medial geniculate body, cen also be expected during 'binaural interaction'.

To verify this aspect, changes in absolute amplitude and latencies in VI peak in the presence of continuous contralateral stimulation was also studied.

Brief plan of study:-

Two studies were carried out. In the first study, BSER were recorded. The stimuli used was logon stimuli of 80 dB HL. Then a continuous tone of 60 dB HL was presented in the contralateral ear. The ton e was presented for 7 min. After 7 min. without switching off the tone, in its presence BSERs were again recorded. This time too, logon stimuli of 80 dB HL were used. The frequency of the logon stimuli and continuous tone was always the same.

The second study, was similar to the first study except that the continuous tone was presented in the ipsi-lateral ear.

The following null hypothesis have been proposed.

1. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute latency of wave-I of ABR.

2. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave—I of ABR.

3. Continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute latency of wave-I of ABR.

4. Continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave-I of ABR.

5. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute latency of wave-VI of ABR.

6. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave-VI of ABR.

7. Continuous ton e of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute latency of wave-VI of ABR.

8. Continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave-VI of ABR.

9. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the ipsilateral ear has no significant effect on the absolute latency of wave-VI of ABR.

10. Continuous tone of the same frequency as the logon stimuli i.e. 2 KKz in the ipsilateral ear has no significant effect on the absolute amplitude of wave-VI of ABR.

11. Continuous tone of the same frequency as the logon stimuli i. e. 2 KHz in the ipsilateral ear has no significant effect on the absolute latency of Wave-I. of ABR.

12. Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the ipsilateral ear has no significant effect on the absolute amplitude of wave-I. of ABR.

Terms Used:-

l. Binaural interaction - the efferent action of monaural stimulation on the periphery of the contralateral auditory system. (Vyasamurthy 1982).

2. Loudness gain - increase in the loudness of the post adapted test tone due to the action of the efferent system innervating the outer hair cells.

3. Hypersensitivity - i.e. measurable aspects of the central auditory system has changed, so that the system is more excitable than in its resting state (Gerken 1984).

4. Absolute latency - refers to the time relationship between stimulus onset and associated response.

5. Absolute amplitude - refers to the height of a given wave component i.e. measured from the peak of the wave to the following trough.

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REVIEW OF LITERATURE

REVIEW OF LITERATURE

The auditory electroencephalic responses (AER) can be divided arbitrarily into four classes of responses. They are divided on the basis of latency, different anatomical sources and different properties. Based on latency they are classified into:

1. Early response which is compromised of a series of very fast waves of latency 4 to 8 msec. These responses arise from the brainstem (Jewett and Williston 1971; Sohmer 1972).

2. Middle response which is compromised of a series of 'fast waves' of 5 to 100 Hz (Goldstein 1969) and of latency 8 to 50 msec. These responses arise from the primary cortical proj action areas.

3. Late response which is comprised of slow waves 2 to 10 Hz and of latency 50 to 300 msec. They arise from the primary cortical and secondary association areas (Appleby 1964; Scott, 1965).

4. The very late response of latency 300 msec to several seconds. They are described as the expectancy wave which is the last peak in the late response and the contingent negative variation which is a long latency negative potential. This response arises from the frontal cortex (Walter et al 1964).

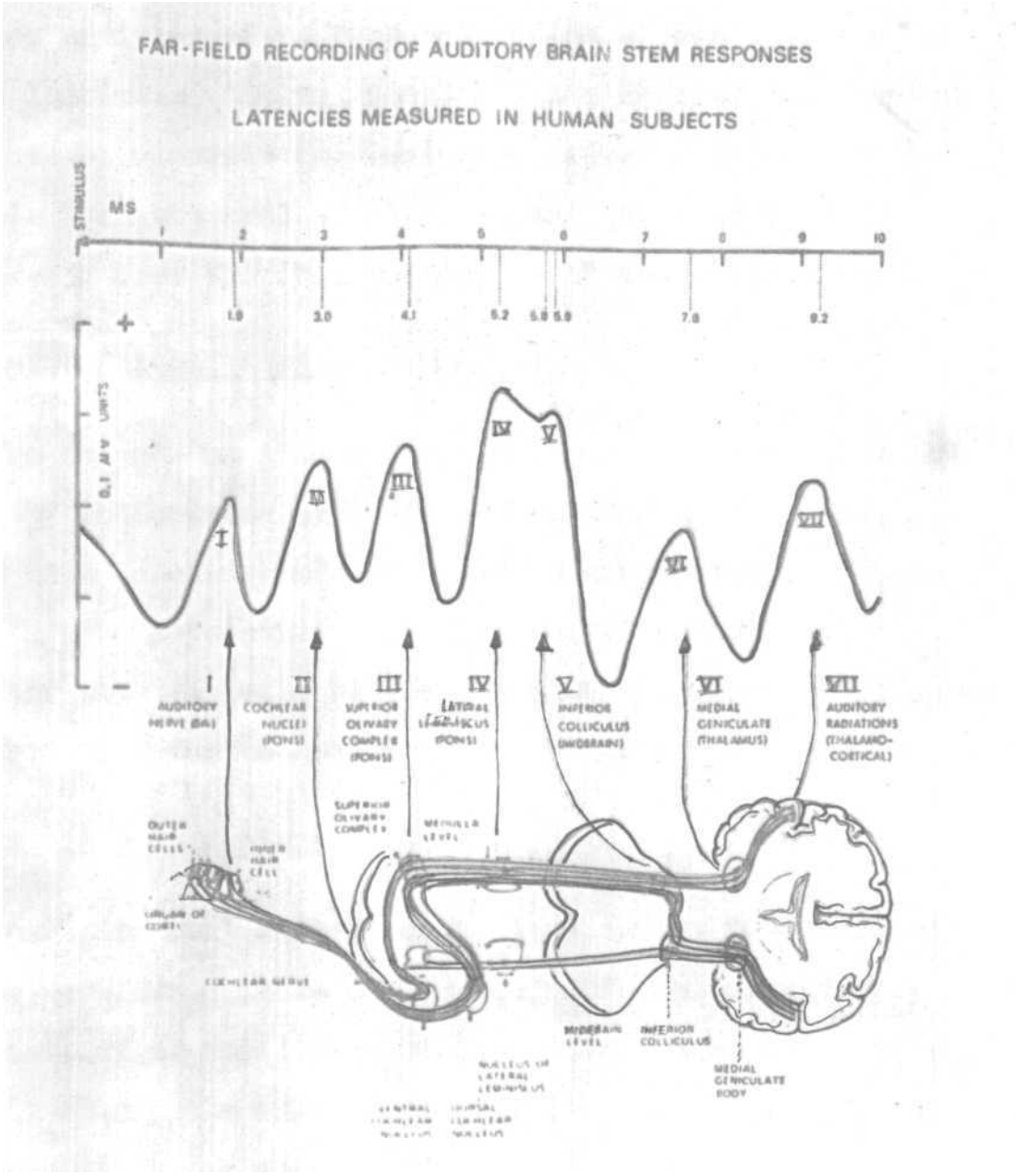


Fig 4 - Anatomic correiation of components of short latency auditory evoked response (Ref. Keith, R.W., Central auditory dyafunction, New York, Gruno & Stratton, Inc. p.11, 1977)

Auditory Brain Stem evoked Responses were measured and utilised in this study, because they are little affected by sleep, wakefulness, tension and awareness of stimuli. The auditory system can be repetitively and rapidly stimulated and its physiological and anatomical pathway have sufficient reliability. Thus, ABR has high reliability.

In the first 10 msec after a click stimulus, a series of small waves can be recorded. The terminology used to describe these waves is conflicting Jewett named them I to VII in Roman numerals. Sohmer numbered then 1, 2, 3, 4a and 4b.

Based on data from several species it is seen that the acoustic nerve transmission of action potential from the cochlea to the brain stem occupies a time course which is compatible with Wave-I latency and ao there is agreement that the first positive peak is produced by the acoustic nerve activity (Cat-Achar and Starh 1980; rat-Henry 1979; human-Sohmer et al 1974; Hashimoto et al 1981).

Wave-II most likely arises from the cochlear nuclei (Jewett 1970).

Wave-III is thought to arise from the superior olivary complex which is known to be the 1st stage of bilateral innervation.

Wave-IV is generated in the ventral nucleus of the lateral lemniscus and is dependant on crossed and uncrossed projections to this area.

Wave-V is from the inferior colliculus from crossed projections.

Wave-VI is from the medial geniculate body which is a complex neural structure that receives ascending multi-sensory input and descending corticofugal input.

Wave-VII originates from the primary auditory cortex.

The waves are generally agreed to have these provence (Skinner 1977; Jewett 1970; Beagley and Sheldrake 1978).

BSER reflect graded post synaptic potential (PSP) rather than all or none action potential discharged at the cell some or transmitted along the axonal projections. This hypothesis is based on the following conclusions.

When stimulus rise time was prolonged, only latency increased and amplitude was unaffected (Galambos et al 1978), i.e. latencies reflect the rate at which threshold for synaptic activation is attained.

When excitability of muscarinic and nicotinic reception were altered, only the amplitude varied (Bhargava et al 1978).

When the intensity of stimuli was decreased or stimulus rate was decreased, there was a decrease in amplitude and increase in latency. (Pratt and Sohmer 1976; Buchwald and Huang 1978; Hyde et al 1976). The reverse was obtained when the intensity of stimuli was increased. This suggests that the synaptic threshold is reached faster leading to decrease in latency, and post synaptic activation is attained in a larger population of neurons leading to an. increase in amplitude.

Effects of stimulus Parameters as given by Moore (1983). There are only 3 primary stimulus parameters frequency, intensity and time.

Effect of frequency:- There is a change in Wave-I for low intensity stimuli across different frequencies, but for high intensity stimuli, there is no change in wave-I across different frequencies.

Effect of Intensity:- It is maximum for latency of wave-I when there is a change of intensity from 30 to 70 dB SPL. The amplitude of wave IV, V complex is least affected by intensity changes.

Effect of time parameters: As the duration of the stimulus is increased, the latency of all the components of the wave increases and the amplitude decreases.

With longer rise decay time, the latency of the waves increases.

As the rate of stimulation increases, the latency increases and the amplitude decreases.

Loudness adaptation is a decrease in the loudness of a steady stimuli over time. According to physiologists, it is a decrease in the response of s single neural unit to a steady stimulus, or the decrease in response magnitude to a constant stimulus level. (Young and Sachs 1973).

There are 3 methods, by which adaptation can be measured (Scharf, 1983).

1. Measurements without recourse to Interaural Loudness or Monaural method.
2. Measurements of Adaptation Based on Interaural Loudness matches or Binaural method.
3. Measurements of Adaptation Based on Lateralisation Judgements.

Experiments based on monaural procedures are - the procedure of absolute judgement by Lawrence et al (1949). Here a 1 KHz tone was either increased, or decreased or left unchanged over a 30 sec listening period. Observers had to **say** if the loudness had increased or decreased or remained unchanged. 9 observers, said 82% of the time that the loudness decreased of an unchanging tone which was presented at 15 dB SL. 66% of the time they said that the loudness increased

of the unchanging tone when it was presented at 70 dBSL. At 15 dB, when the tone was increased, they said it was unchanged. At 70 dB, when the tone was decreased they said it was unchanged. From these findings, one **can** conclude that the loudness of a soft tone decreases over time, but the loudness of a moderate tone increases over time.

In another study of absolute judgement by, Fishkenet al (l977), Scharf and Horton(1978), subjects were to assign numbers to represent the loudness of a continuous tone before and after 2 min and 3 min presentation. They observed a reduction in loudness for a 1 KHz tone at low sensation levels and none at high sensation levels.

Cross modality matching, is a task where the subject has to adjust the intensity of a non-auditory stimuli at specified time intervals, to make it equal in subj ective magnitude to the loudness of a continuous sound (Scharf 1983) Gummlich (1971) had 30 subjects, press a button the length of time necessary to match the loudness of a one third octave band of noise. Results suggested an increase in loudness equivalent to 20 dB over the 1st minute, followed by a decrease in loudness equivalent to 12 dB. At the end of 60 min. the continuous noise caused an increase in loudness.

Another study by Gruber and Braune (1974) using a vibratory stimulus on the forearm to match the magnitude of loudness of an auditory stimuli, found no adaptation to a 75 dB narrow band noise presented for 31 mins.

In the tracking procedure, the subject has to keep the loudness of a continuous sound constant. A study was done by Mirabelle et al (1967). 72 subjects showed 2 to 5 dB adaptation to a 3500 Hz tone, 1 KHz tone and wide band noise presented for 10 min when the intensity of the stimuli were 70 dB SPL. When the stimuli were 90 dB SPL, on the contrary they showed an increase in loudness. Wiley, (1972) observed no adaptation to tones between 10 dB and 60 dB at either 500 Hz or 4 KHz which were presented for 30 sec.

Contrary to all these procedure where no adaptation or minimum adaptation was observed, Weiler and Gross (1973) showed that adaptation could be measured using monaural heterophonic procedures. Weiler (1973) observed 17.75 dB (Mean) adaptation to a 60 dB SPL tone. There was a related increase in adaptation, with adapting stimuli i.e. there was no levelling off of adaptation.

Experiments based on binaural procedures. Here loudness adaptation is measured by matching the loudness of a comparison sound in the unadapted ear, to that of the adapting or test

sound in the adapted ear. The comparison sound is presented before and after the presentation of the adapting sound. The comparison sound may be presented while the test sound is on or after it is switched off. i.e.concomittant or residual or delayed or simultaneous.

Delayed dichotic loudness balance procedure, involves presentation of the comparison tone only once, just after termination of the adapting sound.

Earlier studies show that adaptation was present using this method, later studies show an absence of loudness balance.

Simultaneous dichotic loudness balances are of 2 kinds i.e. either comparison tone is presented just once before termination of the adapting sound, or the comparison tone is presented repeatedly throughout the adaptation period. Hood (1950) found a roughly linear relation over the range of intensity values he investigated. Jerger (1957), too demonstrated a linear relation, at least for adapting stimuli from 10 dB to 60 dBSPL, but above this level he found that the function flattens out or there is levelling off of adaptation. There was more adaptation in SDLB than MH technique even though both measure concomittant adaptation.

Measurement of adaptation based on lateralisation. Here one ear is exposed to a steady sound and an equally intense

sound is introduced to the unadapted ear. The sound is lateralised to the ear where the loudness is greater. A quantitative measure of loudness is obtained by varying the comparison sound so that there is median plane localisation. Modern studies generally show more adaptation for lateralisation at 2 KHz and above than at low frequencies, with the amount of adaptation increasing with level of the steady sound.

Adaptation using this method, like SDLB technique, occurs when a steady tone to one ear is accompanied by an intermittent tone to the other ear.

No adaptation was measured if interaural interaction was eliminated using monaural techniques or if the frequency of the comparison tone was different from the test frequencies (Fraser et al 1970; Morgan and Dirks 1973; Bray et al 1973).

There are so many controversies in this area of auditory adaptation. This was because there was no straight forward technique for the measurement of adaptation.

A new objective technique viz. objective residual monaural loudness adaptation (ORMLA) was developed (Vyasamurthy 1977) to study the phenomenon of loudness adaptation. Adaptation

was measured using this technique. Based on the results, a revised model for loudness coding during auditory adaptation was developed, which solved the controversies regarding auditory adaptation.. "The essence of the revised model are:

1. The adapted neural units are of 3 types (i) stable adapted neural units (a) (2) positive unstable adapted neural units (a_1) and 3) negative unstable adapted neural units (a_2).

2. Simple category and complex category (terms used by Frishkopf and Goldstein) may be the sources for (a) units i.e. (a) units originate from the place of maximum stimulation of the basilar membrane, (a) units for low frequencies originate from 'complex category' and (a) units for $\geq$ lKHz originate from simple category.

3. The source of (a_1) units is the neural units innervating the inner hair cells (NIIH) and they are produced as a result of the efferent action.

4. The source of (a_2) units is the neural units innervating the outer hair cells (NIOH) and they are produced as a result of the afferent action.

5. (a_2) units perform a reverse function of (a_1) units and also that of (a) units. In other words (a_2)

units (responsible for negative adaptation) will be contributing to the loudness during the adaptation process.

6. (a_1) and (a_2) units regain their original preadapted state whenever a test tone at an intensity higher than the adapting intensity is presented during the post adapted state.

7. For high stimulus intensities, loudness is determined by the total number of activated neural units from the following (i) simple category(or complex category) (ii) NIIH (3) NIOH.

8. For low or moderate intensity stimulus, loudness is determined by the total number of activated neural units from simple category (or complex category) and NIOH.

9. The revised model can be used to explain the possible Mechanisms of Perstimulatory Adaptation (Concomittant Binaural).

Adaptation measured using SDLB method is known as 'perstimulatory adaptation' (or concomitant binaural).

Ward (1973) has concluded that 'perstimulatory adaptation' reflects a change in the 'potency' of stimuli in regard to localization following monaural stimulation, it

is demonstrable only by binaural presentation of test signals and therefore involves complex judgemental process relatively high up the auditory chain (through the possible efferent action of monaural stimulation on the periphery of the contralateral auditory system

cannot be ruled out... For the moment, perstimulatory adaptation remains a provoking enigma.

Using the deductions from the results of the study and using data for MH technique and SDLD technique, it may be possible to understand the mechanism involved in 'Perstimulatory adaptation'.

The mechanisms of perstimulatory adaptation can be understood if the answer to the following questions are known.

1. Why do we observe $X_n < X_b > 0$, when the adapting intensity is $\leq$ 60 dB SPL in SDLB method?
2. Why do we observe levelling off of adaptation above 60 dB SPL, in SDLB method?

Here X_b is adapation at baseline X_n is adaptation measured at an intensity higher than the adapting stimulus. Since the deductions from the results of the present study are from a monaural technique (ORMLA) there deductions cannot be applied straightaway to the available data of SDLB method. Therefore, it is necessary to consider the adapting ear and the comparison

ear separately. After knowing the conditions which prevail (during the post-adapted conditions) in the two ears, the combined effects can be reasonably predicted.

Regarding the 1st question - why do we observe $X_n < X_b > 0$ in SDLB technique at 60 dB SPL adapting intensity, the answer may be-Available data for MH technique at an adapting intensity of 60 dB SPL is X_b=11.75 dB (mean) (Weiler et al 1977). From the deduction of the present study, $a > a_2$ can be expected in the adapting ear during the post adapted condition of SDLB method because $X_b > 0$ is $a + a_2 > 0$.

The available data for SDLB at 60 dB SPL adapting intensity is 20 dB (Mean) (Weiler 1972). Thus, it follows that SDLB method yields $X_b > 0$ i.e. the comparison of the data available for MH technique and the data of SDLB, shows that X_b(SDLB)>X_b(MH) (i.e. 20 > 11.75). The fact that SDLB method yields more adaptation than the MH technique at identical intensity and at identical adapting period, is an indication for the influence of the comparison ear in the results. Weiler et al (1979) showed no significant correlation between the amounts of adaptation obtained for MH and SDLB techniques on the same subjects. Glass and Weiler (1979) observed that MH technique and SDLB technique yielded :

$X_h - X_b > 0$ and $X_n < X_b > 0$ respectively for nearly the same stimulus parameters (Weiler et al 1972). Thus, the above

findings show that SDLB and MH techniques yield different results although both measure concomittant adaptation. The difference in the results obtained in the 2 methods must be due to the influence of the comparison ear.

The influence of comparison ear (CE) can be expected through 'binaural interaction[1]. The result X_b(SDLB) >X_b(MH) can be expected provided $*L_{tba} > *L_{tbb}$ (Loudness of the post adapted test tone in the CE. > loudness of the preadapted test tone in the comparison ear. But $*L_{tba} > *L_{tbb}$ can be expected only when $*X_b < 0$ i.e. a_2 results in in the C.E. This means that 'negative adaptation' (or action of the efferent system innervating the outer hair cells) must be present in the C.E. In this context, the author likes to recall wards (1973) suggestion that "... there may be a possibility for the efferent action of monaural stimulation on the periphery of the contralateral auditory system in 'perstimulatory adaptation' ".

The above discussion clearly reveals that X_b (SDLB) > X_b (MH) is due to 'binaural interaction' as correctly guessed by Ward (1973). Thus, the presence of 'binaural interaction' which was not well understood, perhaps, now appears, to be clear. Thus ' binaural interaction' can be defined as the efferent action of monaural stimulation on the periphery of the contralateral auditory system.

Regarding the question - why do we observe levelling off of adaptation above 60 dB SPL in SDLB technique? Available data: X_b(at 60 dB) in SDLB = X_b(at 80 dB) in SDLB = 20 dB (Weiler et al 1972).

Due to 'binaural interaction[1] in the comparison ear, $*L_{tbb}$(at 80) < $*L_{tba}$(at 80). The situation in the adapting ear and the comparison ear, together is

X_b(80) in SDLB = L_{tbb}(at 80) - L_{tba}(at 80) + $*L_{tba}$(at 80)

" $*^{L}$tba (at 80)- At adaptinng intensity of 80 d B SPL in SDLD Adaptation at adapting intensity of 60 dB SPL in SDLB.

X_b(60) in SDLB = L_{tbb}(at 60) - L_{tba} (at 60)+

$*L_{tba}$(at 60) - $*L_{tbb}$(at 60).

The available data show:

X_b (at 80) in SDLB = X_b (at 60) in SDLB

\ L_{tbb}(at 80) - L_{tba}(at 80) $^{+}$ $*L_{tba}$(at 80) -

$*L_{tba}$(at 80) = L_{tbb}(at 60) - L_{tba}Ut 60) +

$*L_{tba}$ (at 60) - $*L_{tbb}$ (at 60).

Rearranging we get:

Loudness in the adapting ear due to the increase in the intensity of the adapting stimulus (i.e. increase in intensity from 60 to 80) = Loudness increase in the C.E. due to the efferent action for 60 dB stimulus + Loudness loss in the C.E. due to the efferent actior for 80 dB stimulus.

Therefore, the increase in the adaptation, in the adapting ear, resulting from the increase in the intensity of the adapting stimulus (i.e. raising the intensity from 60 to 80 dB) will be 'eclipsed' by 'binaural interaction' in SDLB method.

The effects of 'binaural interaction' for 60 dB and 80 dB adapting stimulus are different. When the adapting intensity is 60 dB SPL (or less), the efferent system innervating the outer hair cells will be responsible for the increase in loudness of the post adapted test tone in the C.E. When the adapting intensity is 80 dB SPL or more, the efferent system innervating the inner hair cells will be responsible for the decrease in loudness of the post-adapted test tone in the C.E.

In sum. it can be stated that the levelling off of adaptation observed in 6DLB method, is mainly due to the 'binaural interaction'." (Vyasamurthy 1982).

In support of the phenomenon of loudness gain, many studies can be mentioned:

Johnstone and CodyCl982)did a study on anasthesized guinea pigs. They measured the amount of temporary threshold shift (TTS) to an ipsilateral stimulus (10 KHz, 107 dBSPL for 1 min).

Then TTS was determined using the same stimuli, but in the presence of contralateral stimuli (10K, 80 dB SPL for 1 min). This procedure was repeated with contralateral stimulus (7 KHz, 80 dB SPL for 1 min) after injecting strichnine. The results showed that in the presence of contralateral stimuli (10 KHz, 80 d B SPL for 1 min) TTS induced in the ipsilateral ear was significantly reduced. This significant reduction was not observed when the contralateral stimulus was set at a different frequency of the ipsilateral stimulus, or when strichnine was injected,

Cody and Johnstone propose that the reduction in ipsi-desensitisation in the presence of contralateral stimulation is the result of acoustically evoked activity of cochlear efferent action. Since there was no reduction in T.T.S. after administration of strichnine, it suggests that the reduction seen is real and that it is possibly the result of acoustically activated efferent neuron activity. The absence of this crossed efferent effect for contralateral stimuli set at different frequency of the ipsilateral stimuli also rules out the possibility of generalized effects likes changes in cochlea blood flow.

Kemp (1978) using a signal averaging technique, measured the response of the closed external acoustic meatus to acoustic impulse, near to the threshold of audibility. A brief wave of

pressure was produced in the ear. This response was present in all normal ears tested, but was absent in ears with cochlear deafness. He attributed this emission to have its origin probably in the cochlea, responding mechanically to auditory stimulation and dependent upon the normal function of the cochlea transduction process. He suggests that probably the population of outer hair cells generate this mechanical energy.

This study was replicated by Mountain and Wilson (1980) who observed the same results.

Flock (1982) through stimulation of the efferent nerve fibres, found that the O.H.c. exhibit an active component, injecting energy into the auditory system.

Gerken and Ananthanarayan (1982) did an experiment, where a tone on tone forward masking stimulus configuration was used. The stimuli were suprathreshold, so that they could measure the post stimulatory effects of the masker in terms of changes in the probe evoked ABR. According to the definition, masking refers to the elimination of a component of the ABR, and partial masking to an amplitude reduction and/or latency increase of a component of ABR, but the results showed an amplitude increment of wave-V or an enhancement of Wave-V in the forward masking condition. This they attribute to a central effect.

They conclude that the exact process underlying wave-V enhancement are not clear, but the phenomenon is robust.

Several stimulation and behavioral experiments demonstrate a phenomenon referred to as stimulation hypersensitivity (Gerken 1979; Gerken et al 1982). It is described as sustained alterations from the resting state of the auditory system. This means that some measurable aspect of the central auditory system is more excitable or sensitive, than in its resting state. It is due to various conditions normal and pathological. The underlying nature of hypersensitivity.is not yet known, but its occurrence indicates an interaction between the peripheral and central portion of the auditory system.

The sensitivity of auditory neurons to electrical stimulation can be increa decreased by brief sounds. Only when the sustained sound is of intensity 60 dB SPL, does the evoked response amplitude increase. Facilitation of this sort by a sustained sound was termed enhancement. This enhancement was produced in the medial geniculate body, but was not obtained from all portions of the medial geniculate body. The enhancement was not present beyond the termination of the sustained stimuli.

Spoendlin (1975) reports that the efferents to the outer hair cells synapse with the hair cells and that the enormous nerve supply to the outer hair cells would tally with a concept of a more monitoring role of the outer hair cell system.

Pickles (1982) reports that the centrifugal fibres to the cochlear nucleus are both excitatory and inhibitory.

Fex et al(l982) report that they have found Aspartate amino transferare (AA tase) like immunoreactivity only in the medial system of efferents. They conclude that the efferent terminals to the outer hair cells may participate in the recycling of the released neurotransmitter using AA tase.

Comis and Whitefield (1968) report that acetylcholine, the neurotransmitter of the efferent system innervating the outer hair cells, (ESIOHC) is an excitatory neurotransmitter.

Hoffmann et al (1983) report the presence of enkephalin like peptides (putative neuroactive substances) in the efferent terminals of outer hair cells.

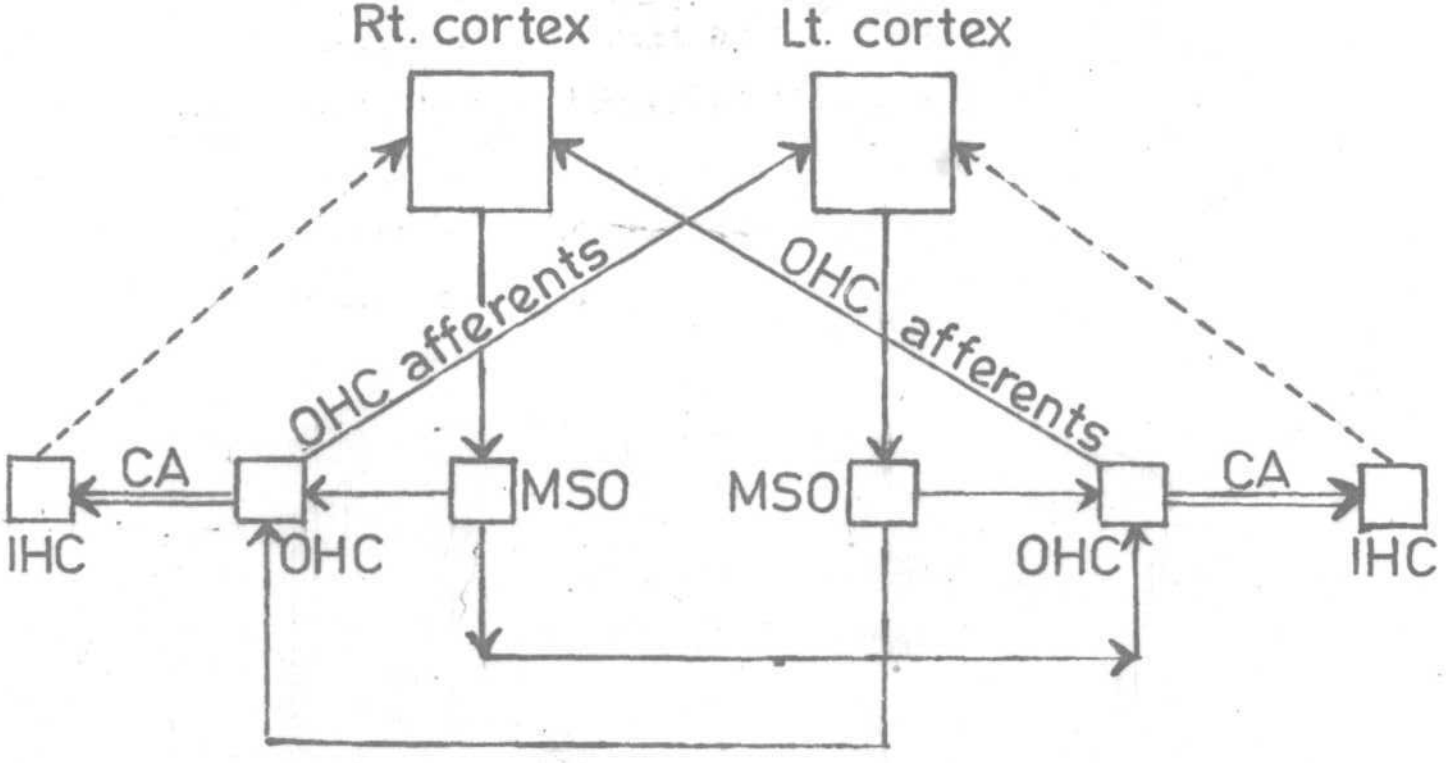
From these studies, one can assume that the action of the ESIOHCS is to increase the loudness of the post adapted test tone.

Kemp(l978,79); Zwislocki(l980) Zureck(l981); Zwislocky and Kletsky(l982); Neely and Kim(l983); Davis (l983) have suggested that there is an active mechanism in the cochlea. This mechanism could be responsible for the greater sensitivity and sharp tuning expressed by the tips of the neural tuning curves.

Siegal and Kim(1982) state that the active mechanism is controlled by the central nervous system, through the activity of the efferent synapse on the outer hair cells.

In discussing the function of the efferent auditory system, Crane (1983) comments "OHC afferents are part of the servo central 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 OHC and that they, rather than the inner hair cell afferents are the source of efferent excitation".

Fex et al (1982) suggest that the acoustic stimulation of crossed olivocochlear bundle may be expected to result in the increase of the sensitivity of OHC afferents through the recycling of the released neurotransmitter.



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

(Vyasamurthy, 1985)

Vyasamurthy (1985) guesses that acetylcholine could be the neurotransmitter released by the efferent system innervating the outer hair cells as Comis and Whitefield (1968) have reported, that acetylcholine is an excitatory neurotransmitter and that acetylcholine could be the neurotransmitter released by the OHC afferent.

By putting all the above pieces of information together, a neural model of the efferent mechanism for loudness gain during auditory adaptation was proposed (Vyasamurthy, 1985).

The model (fig.2) suggests that the efferent system passing through the medial superior olive is responsible for the loudness gain (recycling of the released neuro-transmitter).

METHODOLOGY

METHODOLOGY

Subjects:

15 subjects were tested in this study. The subjects were students of All India Institute of Speech and Hearing.

The subjects had to satisfy the following criteria.

1. No history of any ear discharge, earache, tinnitus, guiddiness, headache, brain damage or exposure to loud sounds.
2. No family history of hearing loss.
3. Age range from 18 to 25 years.
4. Hearing sensitivity within 20 dB HL (ANSI 1969) in the frequencies 500 Hz, 1 KHz, 2 KHz, 4 KHz.

Instruments used:-

Maico-MA-27 portable audiometer with TDH-39 earphone and circumaural cushion MX-41/AR was used, to provide a continuous pure tone to the contralateral ear through the right ear phone.

The audiometer was calibrated for both intensity and frequency for pure tones as per the specifications given by ANSI 1969.

GSI-10 Bekesy audiometer with the B.C.transducer was used to provide continuous pure tone to the ipsilateral ear.

To measure auditory brain stem evoked responses, an Electric Response Audiometer model TA-1000 was used. It consisted of a SLZ 9793 desk top console which contains all of the operating controls, indication and readouts for the system. SLZ 9794 preamplifier which is an isolated EEG preamplifier with frequency response and gain specifically designed for ERA. Also a set of standard silver chloride electrodes, TDH-39 earphone and circumaural cushion MX-41/AR, calibrated paper to record the responses, electrolyte get, adhesive tape and spirit.

TA-1000 operates with 4 knobs and 9 push button switches. The knobs are:-

1. The stimulus function knob which permits selection of frequencies 2 KHz, 4 KHz or 6 KHz at a repetitive rate of 5 or 20 stimuli per second, and patient's response intervals of 10 ms or 20 ms immediately following the acoustic logon stimulus.
2. Stimulus attenuation knob which permits selection of acoustic logon stimuli from 0 dB HL to 100 dB HL.

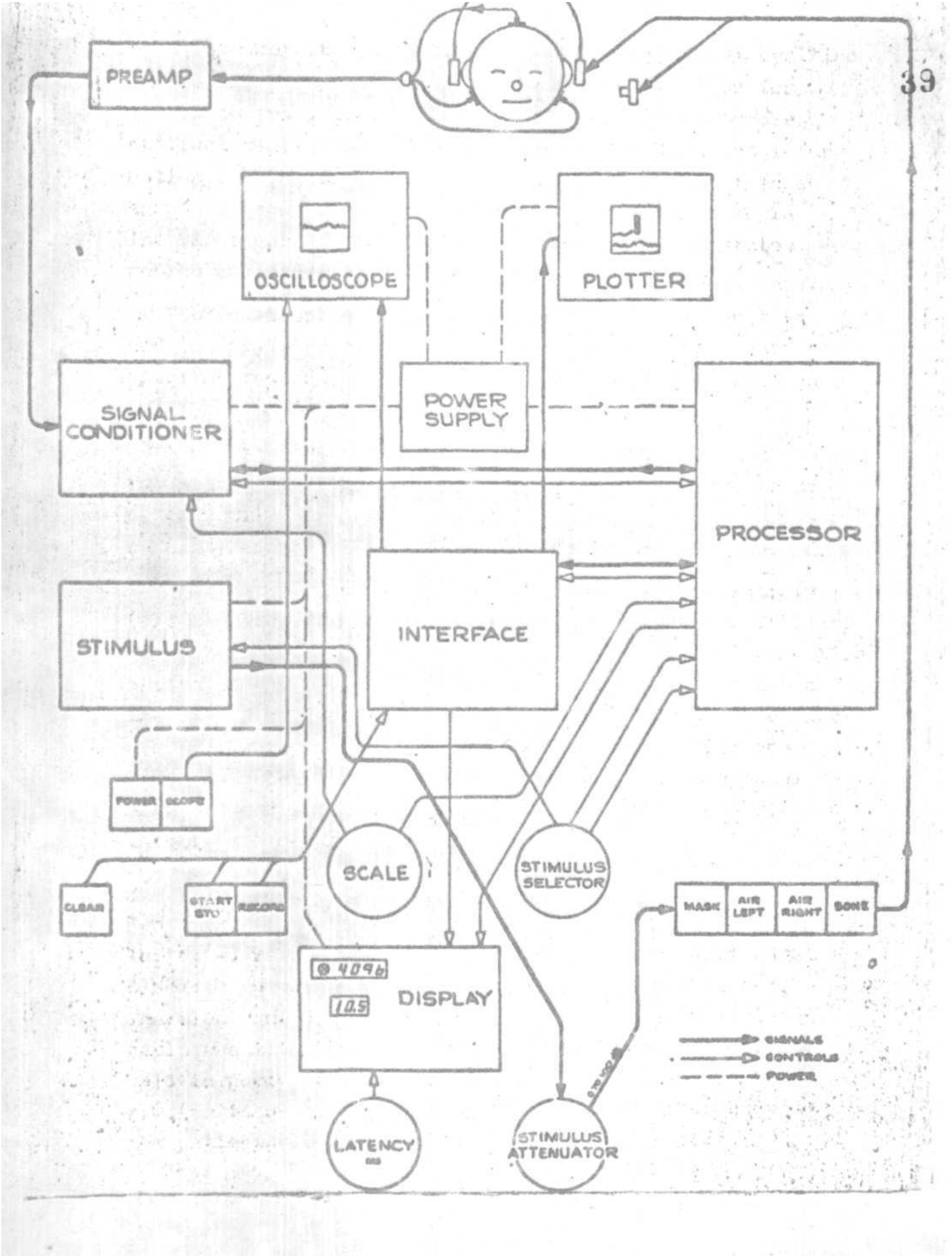


Fig. 8 - Flow chart of ERA: TA-1000 used in the present stud

3. The scale function knob which permits selection of system sensitivity and number of average response samples i.e. for 1024 samples 0.5, 1 and 5 uV per div. sensitivities are available. For 2048 samples, 0.2, 0.5, 1 and 2 uV per div sensitivities are available. For 4096 samples 0.1 uV, 0.2 uV, 0,5 uV and 1 uV per div sensitivities are available.

4. The latency control knob provides a curson mark on the oscilloscope display of the BSER wave for a precise determination of latency. Readout of latency in msec to 0.1 msec is displayed in digital form directly above this control.

The push button switches are:

1. Power
2. Scope - gives an oscilloscope display of the wave.
3. Clear - clears the microprocessor.
4. Start/stop - indicates the microprocessor average function. The average function is automatically terminated when the related number of samples has accumulated or when any averager memory channel is full. The average can be stopped to evaluate intermediate results and restarted without disturbing the averager action.
5. Record - records the wave on to the calibrated paper.
6. Mask - provides broad band noise masking to the contra-lateral ear only when either air left or air right stimulus is presented.

7. Air left-stimulus to left ear phone.
8. Air right - stimulus to right ear phone.
9. Bone - stimulus to bone vibrator.

TWF/Run/EEG switch - This switch should be in run position for normal operation. When in the TWF position, after a clear, the oscilloscope will display a characteristic test waveform to confirm oscilloscope operation. In the EEG position, after a clear, oscilloscope will display the ongoing patient EEG activity.

A stimulus generator was used to generate logon signals. The logon stimulus is characterised by 3 peaks in a 50% - ve 100% + ve, 50% - ve sequence followed by a 50% + ve, 100% - ve 50% + ve sequence reversing on each successive stimulus.

Test environment:

The study was carried out in an acoustically sound treated dimly lit room at All India Institute of Speech and Hearing.

Procedure:

The subj ect was explained the nature of the test. They were made to lie down comf6rtably on a bed with a pillow to reduce neck muscle tension and thereby artefacts. The subject was told to either relax with eyes closed or sleep.

The preamplifier was located very near to the subject and the subject's electrode cable was pinned to the pillow.

The electrodes and the skin surface were cleaned with spirit. Electrode gel was smeared on the electrodes. Each electrode with the electric gel was applied to the cleaned skin and fixed on with the help of adhesive tape.

The 3 electrodes were place as follows:-
Red or signal electrode was placed on high forehead,white or reference electrode on mastoid of test ear i.e. right ear.

Black or ground electrode on mastoid of nontest ear i.e. left ear.

The right earphone was removed from the earphone band of TA-1000 and in place the right earphone from MA-27 portable audiometer was attached.

The earphones were placed after the red light on the preamplifier went off. If the red light came on again after placing the earphones, then the detached right earphone of TA-1000 and left earphone of MA-22 were manipulated such that the red light went off. The power push button switch was switched on. The TWF/Run/EBS switch was set to 'run'. The stimulus function knob was turned to select a stimulus of 2 KHz at a repetitive rate of 20 stimuli per second and patient's response interval of 10 ms.

The stimulus attenuator knob was turned to select a stimulus of 80 dB. The scale function knob was turned to provide 2048 stimuli at 0.2 uV per div sensitivity.

By means of push button stimulus was given to the left ear through air conduction.

First 'clear' push button was pressed to clear the microprocessor, and then the 'start' push button. After 2048 samples were accumulated, the average function automatically stopped. The stimuli to the left ear was switched off. The response was recorded on to the calibrated paper by pressing push button 'record'.

Latencies were marked on the wave by reading them out from the digital display. The digital display was obtained by pressing the push button 'scope'.

In the contralateral ear i.e. the right ear a 2 KHz continuous tone was presented for 7 mins. The tone was presented at 60 dB HL. It was generated by the portable audiometer MA-27.

After presentation of the continuous tone for 7 mins. BSER recordings were again done using the same setting as before. Thus BSER was done in the presence of the continuous tone in the contralateral ear. At the end of 7 min, the

continuous tone was not terminated.

Once the related number of samples were accumulated i.e. 2048 and the average function automatically stopped the continuous tone was withdrawn in the contralateral ear.

The stimulus to the left ear was discontinued. The response was recorded on the calibration paper.

Two graphs were obtained:
One was BSER to 80 dB HL stimuli (2 KHz logon). The other was BSER to 80 dB HL stimuli in the presence of continuous tone in the contralateral tone.

The same procedure was carried out at 4 KHz. Two minutes rest was allowed in between the two test frequencies viz. 2 KHz and 4 KHz.

The above procedure was repeated, but with the presentation of continuous tone in the ipsilateral ear.

Ipsilateral tone was presented through the BC vibrator because continuous tone and clicks had to be presented simultaneously which was not possible using ear phones.

The B.C. vibrator was placed on the forehead and not on the mastoid because mastoid placement interferred with the recording of responses and so the red light on the preamplifier was continuously 'on'.

When the BC vibrator was placed on the forehead, the red light on the preamplifier went off.

A minimum distance had to be maintained between the electrode and the BC vibrator, or else the electrode would pick up the vibrations of the BC vibrator rather than the responses.

The continuous tone was presented at 70 dB HL, for one subject and for the other subjects the continuous tone was presented at 55 dB HL. The reason for selecting a lower level was that at higher intensity levels the vibration of the BC vibrator was interfering with the results.

This test was only carried out at 2 KHz and not at 4 KHz, became the vibrations at 4 KHz interferred with the recording of impulses and thus affected the responses.

The following were determined:

1. Latency - the latency was read in 0.1 msec increments from the displayed digital value, or by counting the number of verticle lines on the calibrated paper from the point the curve starts to the peak of the wave being measured. Each vertical striation =0.1 msec.

2. Absolute amplitude = $\frac{T \times S}{M} \times \frac{N}{n}$

Where, M = masker amplitude

T = the amplitude of the desired trace feature

S = the sensitivity

n is the number of samples actually counted.

N is the number of samples present on the scale.

The efferent action due to stimulation of the COCB was determined by the increase in absolute amplitude and the decrease in latency in waves I and VI. In this study the peaks of the waves were numbered using Roman symbols I to VII as given by Jewett.

The procedure can be summarized as:

1. BSER to 2 KHz logon stimuli of 80 dB HL.
2. 2 KHz continuous tone in the contralateral ear of 60 dB HL for 7 mins.
3. BSER to 2 KHz logon stimuli of 80 dB HL in the presence of 2 KHz continuous tone in the contralateral ear of 60 dB HL.

The same procedures at 4 KHz were repeated in the presence of continuous tone in ipsilateral ear.

1. BSER to 2 KHz logon stimuli of 80 dB HL.
2. 2 KHz continuous tone in the ipsilateral ear of 55 dB HL for 7 mins.

3. BSER to 2 KHz logon stimuli of 80 dB HL in the presence of 2 KHz (55 dB HL) continuous tone in the ipsilateral ear for 3 subjects (The continuous tone was at 70 dB HL in one subject).

The data collected were statistically treated and analyzed using Wilcoxon matched pairs signed ranks test.

RESULTS AND DISCUSSION

RESULTS AND DISCUSSION

Two experiments were carried out. In one, continuous, tone was presented in the contralateral ear. In the other, continuous tone was presented in the ipsilateral ear.

In the first experiment, BSERs were determined under 2 conditions viz. condition A (BSER to logon stimuli of 80 dB HL before the presentation of the continuous tone) and condition B (BSER to logon stimuli of 80 dB HL in the presence of continuous tone of 60 dB HL in the contralateral ear after presentation of the continuous tone in the contralateral ear for 7 min).

In all the tests, the frequency of the continuous tone was the same as the frequency of the logon stimuli.

Table-1 shows the absolute latency values of Wave-I for BSERs obtained under the two conditions A and B. The frequency of the continuous tone and the logon stimuli was 2 KHz.

From the table-1 it can be seen that either there is a reduction in latency in condition B or there is no difference in latencies in condition A and B.

The difference between the two conditions was evaluated with the Wilcoxon T. The difference was statistically significant ($T=0$, $N = 10$, $P < 0.01$).

Thus the null hypothesis - "Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contra-lateral ear has no significant effect on the latency of Wave-1 of BSER" is rejected.

Table-2 shows the absolute amplitude values(μ-v) of Wave-1 for BSERs obtained under the two conditions A and B for 2 KHz tone and logon stimuli.

From the table-2 it can be seen that either there is an increase in amplitude in condition B or there is no difference in amplitude in conditions, A and B.

The difference between the two conditions was evaluated with the Wilcoxon T. The difference was statistically signi-ficant ($T = 0$, $N = 12$ $P < 0.01$)

Thus the null hypothesis-"continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute amplitude of P Wave-1 for BSER* was rejected.

Table-3 shows the absolute latency values (msec) of Wave-VI for BSER obtained under the two conditions A and B for 2 KHz tone and logon stimuli.

From the table-3 it can be seen that in most of the subjects there is a reduction in latency in condition B.

The difference between the two conditions A and B was evaluated with the Wilcoxon T. The difference was statistically significant (T = O N = 10 P < 0.01).

Thus the null hypothesis - continuous tone of the same frequency i.e. 4 KHz in the contralateral ear has no significant effect on the absolute latency of Wave-1 for BSER is rejected.

Table-4 shows the absolute amplitude values (AAvolts) of wave-1 for BSER obtained under the two conditions A and B measured at 4 KHz.

There is an increase in absolute amplitude in most of the subject for wave-1 after the presentation of the continuous tone.

The difference between the two condition A and B was evaluated with /the Wilcoxon T. The difference was statistically significant (T = 0 N =12 P < 0.01).

Thus the null hypothesis - continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave-1 for BSER is rejected.

Thus from the above tables it can be said that 2 KHz and 4 KHz continuous tone in the contralateral ear produces a facilitatory effect in the test ear, which is observed by a significant increase in absolute amplitude and decreases

in absolute latency of wave-I.

Wave-VI was also analysed to see if the continuous tone has a similar effect on wave-VI as wave-I.

Table-5 shows the absolute latency values (msecs) of wave-VI for BSERs obtained under the two conditions A and B measured at 2 KHz.

The difference between the 2 condition A and B was evaluated with the Wilcoxon T. The difference was statistically significant (T= -3, N=10, P < 0.01).

Thus the null hypothesis - continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the contralateral ear has no significant effect on the absolute latency of wave-VI for BSERS is rejected.

Table-6 shows the absolute amplitude values (m volts) of wave-VI for BSERs obtained under the two conditions A and B measured at 2 KHz.

The difference between the 2 conditions A and B was evaluated with the Wilcoxon T. The difference was statistically significant (T= -3, N=10 P < 0.01).

Thus the null hypothesis - continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the

contralateral ear has no significant effect on the absolute amplitude of wave-VI for BSERs'is rejected.

Table-7 shows the absolute latency values (msecs) of wave-VI for BSERs obtained under the two conditions A and B measured at 4 KHz.

The difference between the 2 conditions A and B was evaluated with the Wilcoxon T. The difference was statistically significant ($T = 4$, $N = 11$, $P < 0.01$).

Thus the null hypothesis - continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute latency of wave-VI for BSERs is rejected.

Table-8 shows the absolute amplitude values (μ volts) of wave-VI for BSERs obtained under the 2 conditions A and B measured at 4 KHz.

The difference between the 2 conditions A and B was evaluated with the Wilcoxon T. The difference was statistically significant ($T= -5$, $N=13$, $P < 0.01$).

Thus the null hypothesis - "Continuous tone of the same frequency as the logon stimuli i.e. 4 KHz in the contralateral ear has no significant effect on the absolute amplitude of wave-VI for BSERI' is rejected.

In wave-VI the continuous tone in the contralateral ear caused an increase in absolute amplitude and a decrease in absolute latency.

Thus a 2 KHz and 4 KHz continuous tone in the contra-lateral ear produced a facilitatory effect on the test ear.

This facilitatory effect is due to binaural interaction. It is the efferent action due to monaural stimulation on the periphery of the contralateral auditory system.

In the 2nd experiment, BSER were determined under 2 condition viz. condition I (BSER to logon stimuli of 80 dB HL) and condition II (BSER to logon stimuli of 80 dB HL in the presence of continuous tone of 55 dB HL in the ipsilateral ear, after presentation of the continuous tone in the ipsilateral ear for 7 mins).

The frequencies of the continuous tone and logon stimuli were kept constant. When the logon stimuli were of 2 KHz, even the continuous tone was of 2 KHz.

Table-9 gives the absolute latency values (msec) of wave-VI obtained under the 2 condition viz. condition I and condition II, measured at 2 KHz.

It is evident that continuous tone presented ipsilaterally influences BSER, causing a decrease in latency.

The difference between the 2 conditions was evaluated using Wilcoxon T. The difference was statistically significant ($T = 0$, $N = 3$, $P < 0.01$).

Thus the null hypothesis - "Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the ipsilateral ear has no significant effect on the latency of wave VI" is rejected.

Table-10 gives the absolute amplitude values (m volts) of wave-VI obtained under this 2 conditions I and II measured at 2 KHz.

The continuous tone presented ipsilaterally, caused an increase in amplitude of Wave-VI.

The difference between the 2 condition I and II was evaluated using Wilcoxon T. The difference was stistically significant ($T=0$, $N=4$, $P < 0.01$).

Thus the null hypothesis - "Continuous tone of the same frequency as the logon stimuli i.e. 2 KHz in the ipsilateral ear has no significant effect on the absolute amplitude of wave-VI" is rejected

Table-11 gives the absolute Latency values (msec) of wave-I obtained under the two conditions I and II measured at 2 KHz.

The continuous tone presented ipsilaterally, did not cause a change in absolute latency in 2 subjects. In 2 subjects, the continuous tone produced an increase in latency. There is no consistent change in wave I in the presence of the continuous tone presented ipsilaterally.

Since there is no consistent change in all the subjects tested, it is difficult to draw any conclusion. It needs further investigation using more number of subjects.

Table-12 gives the absolute amplitude values (m volts) of wave-I obtained under the two conditions I and III measured at 2 KHz.

From table-12, it can be seen that continuous tone presented ipsilaterally caused an increase in absolute amplitude in 2 subjects and a decrease in absolute amplitude in 2 subjects. There has been no consistent increase nor decrease in the absolute amplitude. Since there is no consistent change, it is difficult to draw any conclusion. It needs further investigation using more number of subjects.

Table-13 summarizes the experiments of the present study.

TABLE-l: Absolute latency values (msecs) of wave for BSERs under the 2 conditions A and B measured at 2 KHz.

Subject	Condition A(latency in msec)	Condition B(late: in msec).
1	1.5	1.l
2	1.1	1.1
3	1.3	1.1
4	1.1	1.0
5	1.3	1.2
6	1.3	1.2
7	1.3	1.2
8	1.3	1.2
9	1.4	1.4
10	1.1	0.9
11	1.2	1.2
12	1.0	0.9
13	1.5	1.5
14	1.3	1.2

TABLE-2: Absolute amplitude values (μv) of wave-1 for BSERs under the 2 conditions A and B measured at 2 KHz.

Subject	Condition A(amplitude in μ volts)	Condition B(amplitude in μ volts)
1	0.16	0.36
2	0.36	0.36
3	0.34	0.40
4	0.18	0.22
5	0.16	0.26
6	0.10	0.26
7	0.20	0.44
8	0.12	0.20
9	0.10	0.12
10	0.08	0.26
11	0.32	0.32
12	0.14	0.24
13	0.14	0.20
14	0 12	0.16

Table-3: Absolute latency values (msecs) of wave-1 for BSERs under the 2 conditions A and B measured at 4 KHz.

Subject	Condition A (latency in msecs).	Condition B (latency in msec).
1-	1.5	1.4
2	1.1	1.1
3	1.1	1.1
4	1.1	0.9
5	1.1	1.0
6	1.4	1.3
7	1.4	1.3
8	1.5	1.5
9	1.6	1.5
10	1.1	0.9
11	1.4	1.3
12	1.1	1.1
13	1.4	1.3
14	1.3	1.1

TABLE-4: Absolute amplitude values (μ volts) of Wave-I for BSER under the 2 condition A and B measured at 4 KHz.

Subjects	Condition A(Amplitude in μ volts)	Condition B(Amplitude in μ volts)
1	0.10	0.26
2	0.28	0.36
3	0.18	0.18
4	0.22	0.32
5	0.28	0.28
6	0.12	0.18
7	0.33	0.36
8	0.03	0.22
9	0.06	0.10
10	0.26	0.34
11	0.06	0.28
12	0.04	0.09
13	0.15	0.16
14	0.08	0.13

Table-5: Absolute latency values (msecs) of wave-VI for BSERs under the 2 conditions A and B measured at 2 KHz.

Subjects	Condition A (latency in msecs).	Condition B (latency in msecs).
1	6.8	6.7
2	6.2	6.1
3	6.7	6.4
4	6.2	6.1
5	6.1	5.8
6	6.9	6.3
7	6.4	6.2
8	7.2	7.1
9	6.9	6.8
10	6.3	6.4
11	7.1	7.1
12	6.8	6.8
13	6.7	6.7
14	7.0	6.8

Table-6: Absolute amplitude values (m Volts) of Wave-VI for BSERs under the 2 conditions A and B measured at 2 KHz.

Subject	Condition A (Amplitude in m Volts}	Condition B (Amplitude in m Volts)
1	0.18	0.22
2	0.04	0.l2
3	0.06	0.06
4	0.22	0.26
5	0.04	0.16
6	0.15	0.15
7	0.04	0.06
8	0.06	0.08
9	0.12	0.10
10	0.08	0.20
11	0.12	0.16
12	0.16	0.16
13	0.02	0.03
14	0.16	0.16

Table-7: Absolute latency values (msecs) of wave-VI for BSERs under the 2 conditions A. and B measured at 4 KHz.

Subject	Condition A(latency in msecs)	Condition B (latency in msecs).
1.	7.3	7.1
2	6.0	6.2
3	7.0	6.9
4	6.8	6.7
5	6.4	6.0
6	6.9	6.7
7	7.0	6.4
8	7.1	6.5
9	7.2	7.2
10	6.2	6.2
11	6.9	6.9
12	7.7	6.9
13	6.8	6.5
14	6.5	6.1

Table-8: Absolute amplitude values (m volts) of wave-VI for BSERs under the 2 conditions A and B measured at 4 KHz.

Subject	Condition A(amplitude in m volts)	Condition B (amplitude in m volts)
1	0.18	0.20
2	0.08	0.22
3	0.20	0.19
4	0.14	0.24
5	0.04	0.20
6	0.06	0.16
7	0.02	0.18
8	0.06	0.09
9	0.16	0.20
10	0.04	0.16
11	0.02	0.02
12	0.08	0.11
13	0.02	0.03
14	0.1	0.08

TABLE-9: Absolute latency values (msecs) of Wave-VI under the 2 conditions I and II measured at 2 KHz.

Subject	Condition-I(latency in msecs).	Condition-II (latency in msecs).
1(70 dBHL)	6.7	6.2
2(55 dBHL)	7.1	6.5
3(55 dBHL)	6.4	6.3
4(55 dBHL)	6.7	6.7

TABLE-10: Absolute amplitude values (m volts) of wave-VI obtained under the 2 conditions I and II measured at 2 KHz.

Subject	Condition-I (amplitude in m volts)	Condition-II (amplitude in m volts)
1(30 dBHL)	0.08	0.10
2(55 dBHL)	0.06	0.14
3(55 dBHL)	0.17	0.32
4(55 dBHL)	0.14	0.36

TABLE-11: Absolute latency values (msecs) of Wave-I under the two conditions I and II measured at 2 KHz.

Subject	Condition-I (latency in msecs)	Condition-II (latency in msecs)
1(70 dBHL)	1.1	1.1
2(55 dBHL)	1	1.2
3(55 dBHL)	1.1	1.1
4(55 dBHL)	1	1.3

TABLE-12: Absolute amplitude values (m volts) of Wave-I obtained under the two conditions I and II measured at 2 KHz.

Subject	Condition-I (amplitude in m volts)	Condition-II (Amplitude in m volts)
1(70dBHL)	0.1	0.2
2 (55 dBHL)	0.21	0.3
3(55dBHL)	0.4	0.24
4(55dBHL)	0.4	0.2

TABLE-13: Summary of the data of all the experiments.

			Wave-I		Wave-VI	
			Condition-A	Condition-B	Condition-A	Condition-B
Experiment-I	2Khz	Latency Mean	1.26	1.15	6.66	6.52
		Latency S.D	(0.15)	(0.16)	(0.35)	(0.39)
			(t=O, N=10	P < 0.01*)	(t=3, N=10	*P < 0.01**)
		Amplitude Mean	0.18	0.27	0.10	0.14
		Amplitude S.D	(0.09)	(0.09)	(0.06)	(0.06)
			(t=0, N=12	P < 0.01*)	(t=3, N=10	P < 0.01*)
	4 KHz	Latency Mean	1.3	1.2	6.84	6.59
		Latency S.D	(0.18)	(0.2)	(0.45)	(0.38)
			(t=0, N=10	p<o.o1*)	**(t=4, N=11**	P<0.01*)
		Amplitude Mean	0.16	0.23	0.03	0.13
		Amplitude S.D.	(0.09)	(0.09)	(0.06)	(0.07)
			(t=0, N=12	P<O.01*)	(1=5, N=13	P.< 0.01*)

Contd..Table-13

In continuation of Table-13

			Wave-I		Wave-VI	
			Condition-I	Condition-II	Condition-I	Condition-II
Experi-ment-II	Latency	Mean	1.05	1.75	6.7	6.42
		S.D	(0.05)	(0.09)	(0.28)	(0.22)
			(t=0, N=3	P< 0.01*)	(t=0, N=3	P<0.01*)
	Amplitude	Mean	0.27	0.23	0.11	0.23
		S.D	(1.48)	(0.47)	(0.512)	(1.29)

* Significant Difference.

Discussion:

In the present study, monaural acoustic stimulation produced an increase in amplitude and a decrease inl latency in wave-1 positive peak in the contralateral ear. This change in the evoked response, may be due to Binaural interaction as defined by Vyasamurthy (1982) i.e. the effe-rent action of monaural stimulation on the periphery of the contralateral auditory system.

The change in evoked response can be due to loudness gain by the action of the efferent system innervating OHC as supported by(Spoendlin'l975; Cody and Johnstone 1982; Gerken 1984; Fex et al 1982; Comis and Whitfield 1968; Hoffmann et al 1983; Pickles 1982; Stopp et al 1983).

Wave-1 is produced by the auditory nerve activity. (Cat-Achor and Starr 1980; rat-Henry 1979; Human - sohmer et al 1970; Hashimoto et al 1981). Any change in sensitivity of OHC afferents will thus cause a change in wave-1.

The possible mechanism for this loudness gain may be based on the neural model of the efferent mechanism for loudness gain proposed by (Vyasamurthy 1985).

Acoustic stimulation in the contralateral ear leads to acoustically activated efferent neurons which may modify the

response of the OHC to acoustic trauma by the adapting tone i.e. the efferent action counteracts the effect produced by the adapting tone (Cody and Johnstone, 1982).

The process of counteracting the effect produced by the adapting tone could be said to be present because OHC afferents are part of the servo central system. So OHC afferents reflect a crude estimate of the acoustic level at the OHC and they are the source of efferent excitation (Crane 1983).

Efferent excitation or acoustic stimulation of COCB may be expected to result in the increase of the sensitivity of OHC afferents through the recycling of the released excitatory neurotransmitter (Fex et. al 1982). Vyasamurthy (1985) guesses that acetylcholine could be the neurotransmitter released by the efferent system innervating the outer hair cells as Comis and Whitefield (1968) report that acetylcholine is an excitatory neurotransmitter.

The efferent system passing through the medial superior olive could be responsible for the increase in amplitude and decrease in latency of wave-I.

Gerken (1984) determined evoked response to brief click stimuli from the medial geniculate nucleus in a concious

cat. He reports that an increase in stimulus intensity produced increased evoked response amplitude. An increase in evoked response amplitude was also produced in the presence of the continuous tone.

A similar increase in evoked response amplitude and a decrease in latency was observed in wave-VI peak in the present study. Wave-VI is produced by the medial Geniculate nucleus (MSN). The evoked response amplitude increased and latency decreased to logon stimuli from the MGN in the presence of continuous tone in the contralateral ear.

Gerken (1984) reports enhancement of evoked responses in a concious cat. In the present study similar enhancement was observed in man.

As speculated by Gerken (1984) the efferent action might be responsible for the enhancement.

A few subjects were tested with a continuous tone in the ipsilateral ear, to determine whether similar results (as those obtained using continuous tone in the contralateral ear) would result.

A continuous tone in the ipsilateral ear produced an increase in the amplitude and a decrease in latency of evoked

response only in wave-VI. i.e. enhancement was only present in the medial geniculate nucleus when the continuous tone was presented in the ipsilateral ear.

Thus the concept of the continuous tone as a masker fails at higher stimuli intensity as reported (60 dB SPL) by Gerken (1984).

The results of the Exp.II for wave-I of BSER are inconclusive and further studies (using large number of subjects) is required.

For all the subjects, 2 responses were determined for condition A i.e. responses to 80 dB HL without the presence of the continuous tone. The change in the evoked response in condition B was due to loudness gain or enhancement and not due to variability of the response.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The assumption that the action of the efferent system innervating the outer hair cells is to increase the loudness of the post adapted test tone is supported by many studies (Spoendlin 1975; Cody and Johnstone 1982; Gerken 1984; Fex et al 1982; Comis and Whitfield 1968; Haffamann et al 1983, Pickles 1982; Stoop et al 1983).

This loudness gain can be determined by using ABR audiometry. It is observed in the form of an increase in absolute amplitude and a reduction in absolute latency.

In the present study the efferent action of monaural stimulation on the periphery of the contralateral auditory system was determined using ABR audiometry.

Binaural interaction causes a loudness gain when the continuous tone is at a low intensity level (Vyasamurthy, 1982).

l4 normal hearing subjects within the age range from 18 years to 25 years were tested. BSER were determined using an electric response audiometer TA-1000. Logon stimuli of 80 dB HL were used. Then a continuous tone of 60 dB HL was presented in the contralateral ear. The tone was presented through the right earphone using a Maico MA-27 portable audiometer. The tone was presented for 7 mins.

Evenafter 7 mins, the tone was continued. During the 7 to 9 minutes period (i.e. in the presence of the continuous tone) BSERs were again recorded. Logon stimuli of 80 dB HL were used. The frequency of the logon stimuli was same as the frequency of the continuous tone. The test was carried out at 2 KHz and at 4 KHz. In all subjects the left ear was the test ear and continuous tone was presented in the right ear.

Loudness gain was observed in wave-I for most of the subjects i.e. there was loudness gain at the auditory nerve.

From the present study, one can conclude that binaural interaction takes place at the level of the auditory nerve. Monaural stimulation of low intensity causes a loudness gain in the contralateral ear.

Loudness gain was also observed in wave-VI i.e. at the level of the medial geniculate nucleus. The results of the present study agree with the results observed by Gerken 1984. He reports enhancement of response amplitude in the medial geniculate nucleus to continuous tone. He observed these findings in a concious cat.

In the present study enhancement of response amplitude in the medial geniculate nucleus to continuous tone was observed in humans.

Loudness gain was observed in terms of increase in the amplitudes of peaks I and VI for frequencies 2 KHz and 4 KHz.

Another experiment was carried out, where loudness gain was observed when a continuous tone was presented in the ipsilateral ear.

4 normals in the age range of 18 to 25 years were tested. BSERs were determined using an Electric Response Audiometer TA-1000.

Stimuli used was logon stimuli of 80 dB HL. Continuous tone was then presented via a BC transducer using GSI-10 Bekesy audiometer. The tone was of 55 dB HL and it was presented for 7 rains, ipsilaterally. Then without switching off the tone, in its presence, BSER were determined. Logon stimuli of 80 dB HL was used. The test was only carried out at 2 KHz.

Loudness gain was only observed in the 6th peak.

Continuous tone presented ipsilaterally causes loudness gain only in the medial geniculate nucleus.

Limitations of the study:

1. The size of the sample was small.
2. The age range of the subj acts was limited.

Recommendations:

1. To carry out the study on a larger population,
2. To carry out the study on subjects with a wider age range and determine if loudness gain varies with age.
3. To study if binaural interaction is present when the continuous tone is of a different frequency from that of the test stimulus.
4. To study if loudness gain is present when the continuous tone is of a high intensity ($\geq$ 80 dB HL).
5. To study if loudness gain is present in wave—I of BSER in the presence of continuous tone (contralateral ear) in cases with cochlear pathology.

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