THE EFFECT OF CONTRALATERAL NOISE ON THE MIDDLE EVOKED RESPONSE

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TO MY PARENTS

CERTIFICATE

This is to certify that the Dissertation entitled "THE EFFECT OF CONTRALATERAL NOISE ON THE MIDDLE EVOKED RESPONSE" is the bonafide work submitted in part fulfilment for M.Sc, in Speech and Hearing of the student with Register No. 8412

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This is to certify that this Dissertation entitled "THE EFFECT OF CONTRALATERAL NOISE ON THE MIDDLE EVOKED RESPONSE" has been prepared under my guidance and supervision.

ACKNOWLEDGEMENT

I express my sincere gratitude to Dr. M.N. Vyasamurthy for his invaluable guidance.

My thanks are due to Dr. M.Nithya Seelan, Director, All India Institute of Speech & Hearing, Mysore.

I thank Dr.(Miss) S. Nikam, Prof, and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore, for providing necessary instruments to carryout my study.

My thanks to Mr. J.Dayalan Samuel for his help.

I would like to thank Yasmeen, Gayatri and Sinha for their support and help.

My deepest gratitude to all the subjects without whom the study would have been impossible.

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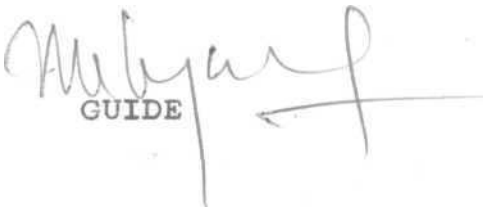

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LIST OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

INTRODUCTION

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"From our Vantage point in 1982, we can see that the late components of the auditory evoked potentials receive very little attention these days as a routine threshold diagnostic procedure, while the auditory brainstem response appears to be part of the routine audiometric test battery in many clinics today. If I may be so bold, I would like to suggest that there even seems to be a resurgence of interest in primary cortical, or as I prefer to call them, Middle latency or Middle component responses. There are not the number of corresponding studies that .I feel there aught to be within this middle latency time domain. I think there are a number of very intersting experimental questions that have yet to be answered about the development of this response and about what this response can tell us about the development of auditory behavior". Mendel (1983)

EEA has been used for about 15 years as a non voluntary test for assessing hearing in those persons who cannot, or will not, respond to sound behaviorally in a consistent manner. Of the time domains constituting the auditory evoked potentials, there has been increasing interest in the middle components (8-50 ms). Hood, (1975).

The maximal response for middle latency components is obtained at the vertex and is symmetrical around the point.

(Peters and Mendel, 1974; Piton Hillyard and Krauz et al 1974). Middle latency components are usually recorded from the vertex (C_3) referenced to a mastoid or earlobe, with a narrow band-pass filter of 25 to 200 Hz. As intensity is increased, latency slightly decreases, where as amplitude substantially increases. (Madell and Goldstein, 1972; Piton et al 1977? Thornton, Mendel and Anderson 1977).

While click stimuli tend to evoke somewhat larger latencies and greater amplitude changes compared to tone bursts (Zerlin and Naunton, 1974; Zerkin,Naunton and Mowry, 1973), tonal stimuli have been found to provide reasonably sensitive frequency specific responses (Mousheglan, Rubert and Stillman, 1973; Kupperman and Mendel 1974; Metarland, Vivien and Goldstein, 1977; Thornton et al 1977).

Stimulus onset, or rise time, also effects amplitude, whereas stimuli duration or spectrum has little influence (Lane, Kupperman and Goldstein 1971; Skinner and Antenoro 1971).

Stimulus rates of one to ten stimuli per second demonstrate little effect on amplitude although higher rates do produce some overall amplitude decline (Goldstein, Rodman and Karlovich, 1972;? McFarland, Vivien end Wolf et al 1975). A rapid adaptation of middle latency responses may occur, since substantial decrease

in overall component amplitudes are found with the first 500 stimulus presentations, and greater numbers of stimuli, produce no significant amplitude reduction (Goldstein et al, 1972; McFarland et al 1975; Vivion, Goldstein and Wolf et al 1977).

Some reports indicate difficulty in obtaining reasonable waveform in neonates (Engel, 1971; Davis, Hirsh and Shelnutt et al 1974; Skinner and Glattke 1977). Other studies have, however been relatively successful, and note little difference between adult and infant morphology for middle components as a function of intensity, or rate of stimulus presentation. (McRandle, Smith and Goldstein 1974; Goldstein and McRandle, 1976, Mendel, Adkinson and Harker 1977) The major differences between these populations are that neonates demonstrate slightly shorter latencies and smaller amplitudes than do adults.

The effect of endogenous factors on middle latency components are minimal. They remain essentially unchanged with attention to the stimulus train, ignoring the stimulus as in reading a book, or sitting with eyes open or closed (Picton and Hillyard 1974; Mendel and Goldstein 1969a). The various stages of natural sleep (Mendel and Goldstein 1971; Mendel 1974; Mendel and Kupperman 1974) or sleep deprivation (Mendel and Goldstein 1969b) have little effect on the middle latency responses.

Similarly, light sedation does not diminish the overall response (Kupperman and Mendel 1974; Mendel and Hosick 1975; Mendel, Hosick and Windman et al 1975). However, when complete anaesthesia is attained, middle latency responses are eliminated (Goff, Allison and Lyons et al 1977) even when recordings are taken from the surface of the cortex (Celesia and Puletti, 1971).

The major problem with middle responses seems to be the large intersubject variability obtained for the AER measures of threshold relative to behaviorindices. Thus, despite the frequency specific sensitivity of the middle latency components and their attractiveness as an audiometric tool (Davis, 1976b, Picton et al 1977), more refinement of the procedures is apparently necessary before they can be employed as an objective measurement technique for hearing evaluation. However, recent work by Galambos and his associates (Galambos, Makeig and Talmachaff 1981) have defined a series of middle latency components obtained with 40 stimulus presentation per second. This '40 Hz evoked potential' appears to reflect the number and basilar membrane location of the auditory nerve fibres, a given tone exites. Such an auditarially sensitive measure is a promising new approach for the clinical application of middle latency componentss.

Presentation of a contralateral masking stimulus of moderate intensity does not appear to affect component amplitudes (Gutnik and Goldstein 1978). Similarly, binaural stimulus presentation also shows little effect on the wave-form at low-to-moderate levels of intensity (Peters and Mendel 1974), but does produce an overall reduction in component amplitudes when intensities are greater than 70 dBHL (Dobie and Norton 1980). In contrast to this result, Kadobayashi et al (1984) observed that the early components of the middle latency responses for binaural stimulation had larger amplitudes than those for monaural stimulation.

Statement of the problem:

The present study is aimed at studying if there is any effect on the amplitude and latency of the middle response when noise is presented to the contralateral ear. The study is also aimed at studying if there is any difference in the amplitude and latency of the V peak when measured using patients response intervals of 10 ms and 20 ms. And lastly, the study is aimed at finding if any binaural interaction takes place in the middle evoked response.

Questions:

1. Is there any effect of contralateral noise on the latency and amplitude of the middle response at sensitivity = 0.2?

2. Is there any effect of contralateral noise on the latency and amplitude of the middle response at sensitivity = 1?

3. Is there any effect of contralateral noise on the latency and amplitude of the VI peak at sensitivity = 0.2?

4. Is there any effect of contralateral noise on the latency and amplitude of the VII peak at sensitivity = 0.2?

5. Is there any effect of contralateral noise on the latency and amplitude of the VI peak at sensitivity = 1?

6. Is there any effect of contralateral noise on the latency and amplitude of the VII peak at sensitivity = 1?

7. Is there any difference in the amplitude and latency of the V peak when measured using patient response interval of 10 ms and 20 ms?

8. Is there any difference between the combined amplitude of middle response (amplitude of response when right ear was stimulated + amplitude of response when left ear was stimulated) and the binaural amplitude response (amplitude of middle response obtained when both the ears were stimulated)?

Implications of the study:

1. It provides information regarding clinical usefulness of middle evoked response audiometry.

2. It provides information regarding the amplitude and latency of middle evoked response in normals using 0.2 uV and 1 uV sensitivity.

3. It provides information regarding the amplitude and latency of the VI and VII peak in normals using 0.2 uV and 1 uV sensitivity.

4. It provides information about the amplitude of middle evoked response for binaural stimulation.

5. It provide information about the amplitude and latency of the V peak in normals while using a patient response interval of 10 ms and 20 ms.

Terms used:

Middle Latency Response: Are those responses having latency between that at the early responses, the endocochlear and brainstem potentials on the one hand and the late cortical potentials on the other.

Binaural Interaction (BI): Is derived by subtracting the sum of the left and right monaural responses from the binaural response.

Latency: Refers to the time relationship between stimulus onset and associated response.

Amplitude: Refers to the height of a given wave component i.e. measured from the peak of the wave to the following trough.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Along with the development of computers and especially the averaging technique it has been made possible to record human auditory evoked potentials (EP's) appearing as very weak electrical signals at the surface of the skull. Today, we can use the auditory evoked potentials as part of our clinical procedures for the diagnosis of audiological and otoneurological disorders. These methods are referred to as electric response audiometry(ERA) (Davis, 1976).

ERA is often referred to as an 'Objective Test'. Audiologists commonly classify the various audiometric tests available into subjective, behavioral and objective categories. Objective tests require no active co-operation from the subject who can only influence the results by interfering with the test procedures. The objectivity usually only relates to the subject as often the results may require considerable 'subjective' interpretation.

Judgements of responses for clinical purposes are generally made from the visual display of the AER. Among the factors which make one response more identifiable than another are amplitude of the peaks and the amount of biologi noise in the tracing. Clinically, it is desirable to obtain readily identifiable responses from small numbers of stimuli over a short period of time.

There are many different auditory evoked potentials that can be recorded from the human scalp in response to various acoustic stimuli (Picton,Woods and Baribeau-Braun et al 1977).

In the 10-50 msec latency range there are various scalp muscle reflexes that can occur in response to loud acoustic stimuli (Bickford, 1972). The inion response in the neck muscles appears to depend upon vestibular rather than cochlear connections (Tounsend and Cody, 1971). The postauricular muscle reflex, on the other hand, is initiated by cochlear stimulation. It is a bilateral reflex recorded from a localized region of the mastoid process at the level of the external auditory meatus (Yoshi and Okudaina, 1969; Dauck, Gibson and Humphries, 1973; Strelete, Katzand Hohenberger et al, 1977). The amplitude of the response varies with repetition rate, attention, head position, and muscle tension. It usually has two major components - a mastoid - negative wave peaking at 12.15 msec., followed by a positive wave at 18-25 msec. The threshold for eliciting this reflex is usually between 60 and 70 dBnHL. Other scalp muscle reflexes can also be elicited by local auditory stimuli, particularly in the temporalis or frontalis muscles (Picton, Hillyard and Krausz et al, 1974). All of these muscle reflexes can distort the middle latency brain responses. It is therefore best to record the middle responses during sleep, when reflex muscle activity is minimized.

Stretz et al (1977) found the middle components to be free of myogenic contamination during sleep and dependant on the electrode position.

Middle Latency Components:

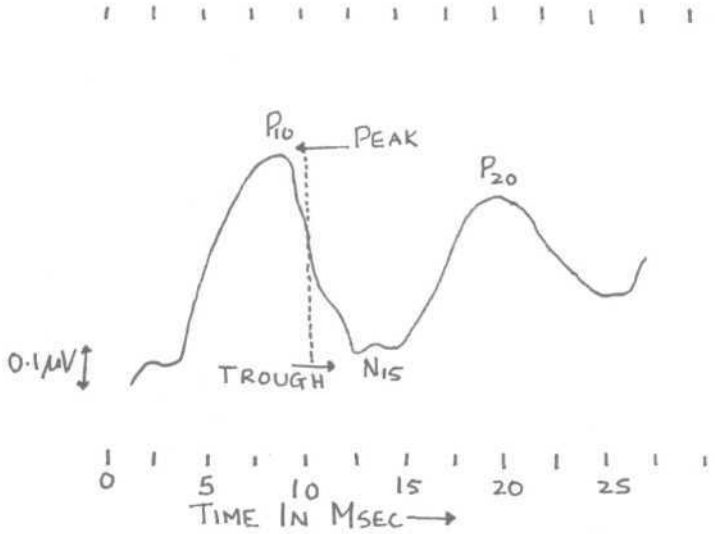
The responses are so called because their latency lies between that of the early responses, the endocochlear and brainstem potentials on the one hand and the late cortical potentials on the other; the latencies of the various peaks of the middle responses range from 10 to 50 msec and have amplitudes ranging from 0.5 to 3.0 uV. They are generally denoted by the labels No, Po, Pa and Nb, although Pb and Nc are occasionally seen with somewhat longer time bases eg. (10-80 msec). When the middle latency components were initially reported (Geisler, Frishkoff and Rosenblith, 1958) they were called the 'early' AER responses,but have more recently been termed the middle - latency responses. (Picton, Hillyard and Krausz et al, 1974; Davis, 1976b), due to the defination and increased interest in the auditory brainstem response which occurs before the middle latency group (Jewett and Willistone, 1971; Skinner and Glattke, 1977; Starr, Sohmer and Celesia, 1978).

The peak latencies of the middle evoked response usually occur in the following limits depending on the bandpass of the

system used (Goldstein and Rodman, 1967). No-8-10 msec; Po-10-13 msec; Na-16-30 msec, Pa-30-45 msec; Nb 40-60 msec and Pb 55-80 msec. The latency of the responses is quite consistent within the same subject and Pa is, perhaps, the most-stable of the individual peaks. The peak to peak amplitudes vary from about 0.7 to 0.3 uV with the largest peak being Nb. Goldstein and Rodman (1967) found that the combination of Na and Pa provided the best means of identifying the responses. The No and Pb peaks are not always identifiable.

Suzuki, Yasuhito and Horiuchi (1981) observed auditory evoked responses to tone pips within the first 25 msec. Following signal onset. These were labelled P10, N15 and P20. The number refers to the latency of the response and the proceeding letter indicates whether the wave is positive (p) or negative (N).

In a pilot study conducted by Beattie, Meretti and Warren, (1984), it was revealed that evoked responses within the 25 msec, period following the presentation of tone pips were characterized by the V pattern (P10-N15-P20).



Typical evoked response waveform to a 40 dBnHL tone pip. The frequency was 2 KHz and the rise-full time was 2 ms. (Beattie, Moretti and Warren, 1984).

Effects of Stimulus Frequency and Intensity on the Middle Components of the Averaged Auditory Electroencephalic Response:

Goldstein and Rodman (1967) studied the early components of averaged evoked responses to rapidly repeated auditory stimuli. They observed that the latency of the Na, Pa and Nb peaks remained relatively consistent at suprathreshold and threshold levels for most subjects. Although some latency increase was seen with a decrease in sensation level, no clear differences were noted in the total group in the response configuration, or latency as a function of sensation level. For the majority of subjects for sensation levels of 60, 30 and 10 dBSL, a vertex negative peak was seen at 31.25 to 35.00 msec, and another vertex negative peak at 46.25 to 50.00 msec. Another positive peak occured at about 10 to 12 msec, for many subjects at 60 dBSL.

Thornton, Mendel and Anderson (1977), observed that in their study, there was a trend for greater amplitudes with low frequency stimuli. (Na-Pa) did not show consistent increase in amplitude as a function of sensation level for any of the tone burst stimuli. However, later measures (Pa-Nb) did show a consistent amplitude growth with increaing tone burst. Good agreement across subjects in composite AAER peak latencies was found. Responses to 4 KHz stimuli were found to be smaller and showed a more gradual growth in amplitude, so detection at low stimulus levels was more difficult.

Kupperman and Mendel (1974); Mendel et al(1975); and McFarland, Vivion and Goldstein (1977) have showed that the middle components can be elicited with tonal stimuli of low as well as high frequencies, in adults and similar results were reported by Mendel, Adkinson and Harker (1977) in older infants.

Madell and Goldstein (1972) observed that amplitudes of the AER for a given sensation level varied considerably between subjects. Latency varied slightly between subjects, but the response configuration were essentially the same. Most of the subjects showed quite consistent response patterns

and only small amplitude differences between trials. Some subjects maintained consistent response configurations between trials, but with considerably different amplitudes. Although most subjects demonstrated a fairly consistent relation between amplitude and sensation level, a few evidenced little relation between the two. It was also seen that latency decreased as sensation level increased upto about 40 dBSL. The mean latencies at 50 dBSL were Po, 11.3 ms; Na, 20.8 ms; Pa 32.4 ms; and Nb 45.5 ms. These values were similar to these reported by(Goldstein and Rodman 1967; Mendel and Goldstein 1969a and b). For all peak-to-peak measures, they found that amplitude increased as sensation level increased.

Zerlin et al (1973), tested 4 awake subjects with one third octave clicks centered at 500 Hz, 1 KHz, 2 KHz and 4 KHz. A latency decrease of 5 ms between 500 and 4 KHz was reported in their study.

A failure to find a systemic increase of amplitude for the Na-Pa peaks with increasing level for tone pips was observed in a study by Kupperman and Mendel (1974). This result was found to be puzzling, however, the results for the Pa-Nb peaks showed the anticipated trend. Although the absolute amplitudes differed, the trend for the Pa-Nb peaks corraberated with the earlier reports using click stimuli-Madell and Goldstein (1972) and Mendel(1973).

Mendelson and Salamy (1981), observed that the latencies for Po were slightly shorter than those for Pb and these were reported to be considerably longer than these reported by other investigators. This may be attributable to the extremely brief duration of the click stimuli? the relatively wide response filter band pass that was employed, or a combination of these factors.

Amplitude of evoked responses to tones of high intensity was measured by Picton, Goodman and Bryce (1970). They observed a definite decline in the amplitude intensity curve above 70 dB ISO. Similar results were also obtained in other laboratories (Davis and Zerlin, 1966? Butler et al, 1969). This decline, was found not to be related to cross hearing, but did occur at an Intensity very close to the stapedius reflex threshold. The stapedius reflex occurs at too long a latency, however to be a causative factor.

Origin of the Middle Response:

Okitsu et al (1977) who studied the middle latency response to click stimuli, suspect that the origin of the Po peak may be different from that of Na and Pa Peak. In a later study by Okitsu et al (1980), they studied the middle components of the auditory evoked response in young children while awake and asleep.

They summarized that the middle components may be divided into four groups comprising Po, Na, Pa and peaks later than Nb; and that each group has different origins of pathways of the response.

Controversy about the source of generators for the middle components still exist. Geisler et al (1958), did one of the earliest studies using averaging computers. They concluded that the response was cortical in origin because (a) it was repeatable from the same subject; (b) it was recordable from a wide area of the scalp; (c) monaural stimulation evoked a bilateral response; (d) the symmetrical placement of electrodes recorded virtually the same response and (e) these latencies were comparable to onset latencies for the somato sensory and visual system. In a later, more extensive study of these evoked response components, Geisler (1960) concluded that their characteristics are similar to animal cortical responses; their activity can be detected at the cortex, but they are not necessarily cortical in origin.

Harker et al (1977) found the middle latency responses to be of neurogenic origin. This was supported by Goff (1978) and Mendel (1979). However, Bickford et al (1964) and Borsanyi (1964) suggested the response to be or myogenic origin.

Picton et al (1974) found a myogenic response to acoustic stimuli with a latency similar to the neurogenic response.. Pa. But, the myogenic response was found to be highly variable, and Picton concluded that the middle latency components largely represent potentials from cortex and thalamus. Beagley, also appoints the site of generation of the middle responses, almost certainly to be situated in the auditory radiations in tne thalamic region, and in the primary auditory cortex in the temporal lobe.

Based upon animal experiments the assumption has been made that the middle components one of neurogenic origin, generated in the primary auditory cortex (Walloch 1975; Brugge and Imig (1978).

Vaughan and Ritter (1970) found early human scalp components with a maximum amplitude, superior to the estimated level of the sylvian fissure and inverting in polarity below this level.

In preoperative temporal lobe recording, Ruhm et al (1976) found evidence indicating that the temporal lobe is the generating site of the middle component.

Celesia (1976) found auditory evoked potentials in man with a latency of about 15 msec and later, when recording directly from the Heschl's gyri. When recording from the perisylvian area.

responses with a latency of about 30 msec were found. However, Coff et al (1977) found the early part (15 msec) of these components to be suppressed by barbiturate anaesthesia and concluded that it could not be the primary cortex response and that subsequent neurogenic auditory evoked responses were too late to be generated in the primary cortical area.

The earlier middle - latency components (No, Po, Na) might arise from the medial geniculate and polysensory nuclei of the thalamas, while the later portions of the wave forms are found over wide areas of association cortex (Geisler et al 1958? Picton Hillyard and Krausz et al 1974; Davis, 1976b).

However, recent clinical evidence with bilateral auditory cortical damage suggests that these responses do not arise from the primary auditory cortex (Parving, Salomon and Elberling et al, 1980).

There is general agreement that the early components of the auditory evoked response are derived from generators with in the auditory nerve and brainstem, while the middle evoked potential's components, reflect activation of the thalamus and cerebral cortex (Picton and Smith, 1978). However, this is no consensus as to the precise origin of waves P10, N15 or P20 (Borg 1981; Rowe 1981). These authors agree that interpretation

of the evoked response waves in specific anatomical terms is questionable because of the auditory pathway. This complexity is well stated by Rowe (1981) - "Controversy continues over whether the activity arises in nuclear structure or tracts or ipsilaterally or contralaterally to the stimulated ear or bilaterally. The complex spatial arrangement of the auditory system structures, the combined sequential and simultaneous activation of generators, and the overlapping of transient and sustained activity from multiple sites probably preclude any specific correlation between a given brainstem site and a particular response peak".

Uncertainity in associating specific waves with anatomic structures also is due to the use of scalp electrodes which are situated some distance from the neural generators. The electrical activity from the auditory system is transmitted to the scalp via a volume conductor (brain, tissue, extracellular fluid) this has in homogeneous electric properties (Borg, 1981).

Borg (1981) suggested that the generators of the evoked potentials are located in the relay stations of the auditory pathway rather than in the tracts. He speculated that depolarization of nerve cell bodies is the main source, but that presynaptic and postsynaptic events may be involved as well.

Davis and Hirsh (1979) suggested that N15 may represent the electrical output of dendrites and cell bodies in the gray matter of one or more of the brainstem nuclei of the auditory system.

The above review indicates that the complexity of the auditory system precludes assigning specific generator sites to waves P10, N15 and P20.

Comparison Between Early and Middle Latency Evoked Response:-

For the past several years, auditory brainstem response (ABR) has been shown to be a valuable tool for assessing electrophysiological thresholds to sound stimuli. With its sudden popularity, it has overshadowed an older procedure, middle latency evoked response (MLER). MLER is said to be a valid and reliable auditory electrophysiologic procedure. (Ruhm et al 1977; Mendel and Goldstein, 1961).

Recently reported experience would seem to indicate the MLER to be advantageous over ABR in regarding to obtaining frequency specific auditory information (Thornton et al 1977; McCandless 1978; Picton et al 1977). However, in emphasizing clicks, a stimulus best suited for ABR, it is difficult to surmise whether ABR or MLER is better for measuring electro-

physiologic responses close to threshold. However, other studies report that threshold for clicks determined by EEA using the middle components of the AER agree closely with voluntary behavioral thresholds for clicks determined on the same subjects. (Geisler, Frishkoff, and Rosenblith 1958, Lowell, Williams, Ballinger and Alving 1961? Goldstein and Rodman 1967? Madell and Goldstein 1972).

Musick and Geurkink (1981) compared ABR and MLER sensitivity near threshold. They observed that considerably more subjects yielded middle latency waves Pa and Pb along with brainstem wave V than brain stem waves I and III and middle latency wave Pc. This was most evident at a low SL(5 dB) and this difference decreased as intensity increased.

In analysing the percentage of possible responses for each of the brainstem and middle latency waves for all SL's combined, wave Pa had the highest occurance followed by wave V, wave Pb, Wave III, wave Pc and wave I. When combining all middle latency and brainstem waves being analysed for all SL's, MLER waves yielded a significantly higher percentage of responses than ABR waves. ABR latencies decreased with increases in intensity and were consistent and predictable. Latencies for the middle waves were more variable and less predictable.

especially wave Pc. However, (Freeman 1965; Mendel and Goldstein, 1969a) have reported middle components, elicitable in a replicable fashion under distraction? over extended time percods (Freeman 1965; Mendel and Goldstein 1969b); during sleep (Mendel and Goldstein 1969b; 1971a and b; Mendel, 1974; Mendel and Kupperman 1974), and with the subject pharmacologically sedated (McRandle and Goldstein, 1973; Mendel, Hosick, Windman, Davis, Hirsh and Dinger 1975; Mendel and Hosick 1975). They can be elicited from neonates (McRandle, Smith and Goldstein, 1974; Goldstein and McRandle 1976), and from young infants (Mendel, Adkinson, and Harker 1975). Because of their rapid recovery (Ruhm, Walker and Flanigin 1967; Goldstein, Rodman and Karlovich 1972; McFarland, Vivion, Wolf and Goldstein 1975; Vivion 1975; Vivion, Goldstein, Wolf and McFarland 1977), middle components can be elicited by rapidly repeated stimuli allowing clinical derivation of an AER.in a resonably brief period.

McFarland, Vivion and Goldstein (1977) did not observe consistent or reproducible middle component peaks between 8 and 25 ms, in all subjects for all stimulus conditions. The peaks Po and Na, did not emerge clearly enough to warrant extensive description or analysis. Either the filter conditions employed, or an unfavourable signal-to-noise ratio, or a combination of both probably account for these peaks not being clearly observable in the AER's.

Thornton et al (1977) were not able to identify two of the earliest middle component peaks. No and Po, in their composite AER's even though they employed slightly different filter conditions.

The mean latencies for the middle responses Pa and Pb reported by Museik and Geurkink (1981) were found to be in-fairly good agreement with other published data (Davis, 1976; Goldstein and Rodman 1967; and, Madell and Goldstein 1972). However, there is less agreement in terms of the latency of wave Pc. The greater amplitude for the Middle Latencies are said to be a chief factor in being able to read these waves more easily than the brainstem waves. This difference is especially noted at the extremely low SLs. The larger amplitudes for the middle latency waves is probably attributable to a greater number of nerve fibres being involved at a higher cortical level.

Museik and Geurkink (1981), feel that for measurements near auditory threshold, MLER may be as good or a better procedure than ABR. But, this viewpoint must be tempered with some reservations. It must be remembered that the subjects tested were adults having normal hearing. Jerger and Mauldin (1978) reported an estimating high frequency (1 KHz to 4 KHz) thresholds for a hearing impaired population for which a 15 dB standard

error was found using ABR. McFarland et al (1977) using MLER also tested a hearing impaired population. No standard error for thresholds was reported. However, these two studies were considerably different in the methodologies employed and direct comparisons are impossible.

MLER is said to be a sensitive measure near threshold. This may be of most value in pediatric testing, but high repetition rates are more commonly employed with ABR than MLER. However, this advantage may be partially off set by the fact that MLER often requires fewer trials to obtain a readable waveform.

In summation, it is important for those involved in measuring auditory electrophysiological responses at low SLS not to limit their procedures to ABR testing. Rather, the potentials of other electrophysiological measures such as MLER should be kept in mind. It is only by the use of these various measures clinically and experimentally that the best procedure will be realized (Musick and Geurkink 1981).

Effects of Noise:-

Masking is said to occur when one sound makes another sound difficult or impossible to hear, or when the threshold of the signal (the masker) has been elevated by a second signal or noise (the masker) (Moore, 1983).

The masking phenomena studied in the psychoacoustic lab can also be studied in the lab of physiology. Although, physiological studies of masking are not numerous (Gerken 1971).

Blegrad (1972) examinad the effect of contralateral masking in a patient in whom; in all probability, one cochlea had been deprived of its efferent innervation. Stimulation of the better ear with white noise exerted a definite influence on the tracings from the operated ear. The finding suggests that the contra-lateral masking effects, is due to a central mechanism, rather than to an action upon the opposite cochlea.

Rosenhammer and Hohnkuist (1983) compared monaurally evoked ABRs to clicks at 70 dBnHL in the presence of contralateral masking by white noise at 60, 70, 80 and 90 dBnHL with the corresponding ABR's without contralateral masking. They observed that the latency of wave-I did not change significantly with contralateral noise at any one of the four levels. The latency of wave-III was significantly prolonged only at the noise level of 90 dBHL. The latency of wave-V was significantly increased at the noise levels of 80 and 90 dBHL. The average latency pro-longation were on the order of 0.05 msec. The findings suggest the latency increments to be attributable to central masking than to acoustic cross over or stapedies reflex elicitation. Contra-lateral white noise at levels below 80 dBHL did not seem to effect the ABR to clicks above 65 dBnHL.

In contrast to the above finding, Rajalakshmi (1983), observed that there was no effect of contralateral noise on brainstem evoked response elicited using 2 KHz and 4 KHz logon stimulus. She concluded that there may not be any central masking effect operating when the noise is presented to the contralateral ear, while testing the test ear during BSERA. If the central masking phenomenon had operated, the amplitude and latency of brainstem evoked response would have changed during contralateral noise condition.

Reid and Thornton (1983), in their study, observed that contralateral masking had no statistically significant effect upon the BSER. A possible explanation for this finding may be found in the hypothesis of Gersuni and his associates (Gersuni 1971). They proposed that a different mechanism and pathway within the auditory system exists for short duration sounds and for longer duration sounas. If the wide band click stimulus produces neural activity within the onset responding 'short duration part of the auditory pathway and the continuous wide-band masking noise causes neurons in the long duration path of the pathway to fire, then, perhaps for this reason, contralateral masking will not have an effect on the BSER.

Evans (1974) suggested that the auditory system divides into two subsystems that can be differentiated anatomically and functionally at the brainstem level at least, and they may be related to the processing of localization, and pattern information respectively.

Freigang, VonSpecht and Oeken (1974) applied white noise to the contralateral ear of normally hearing persons at 0, 30 and 40 dB levels several times in an alternating sequence, while constant stimulation of the ipsilateral ear with a 1 KHz tone at 70 dB was applied simultaneously. They noticed that there was a small increase in amplitude during stimulation of the opposite ear with white noise at 30 dB and a reduction in amplitude at 40 dB. The latencies were found to increase in both cases. These changes were explained by the authors in terms of a central mechanism.

Ananthanarayan and Gerken (1983) in their study, observed two contrasting effects on components of the ABR. One was partial masking of wave-III, and the other was amplitude enhancement of wave V. It is possible that wave V generators receive input via a pathway not reflected in wave III. The increased latency of wave V could seem to indicate though that the wave V generator(s) are also driven by a sound afrected by partial masking, hence the enhancement of wave V would be a central effect. They state

that another possible passive basis is that the enhancement of wave V may be due to the selective masking of wave VI. The fact, however, that wave VI did not always exhibit masking makes this explanation unlikely.

Three experiments were carried out by Reid, Birchall and Moffat (1984). In the first experiment, in six unilaterally deaf people a wide band click at 90 dBSL, relative to the threshold in the normal ear, was presented to the deaf ear and the amount of masking needed to wash out the sound crossing over to the good ear was measured. In the second experiment clicks at 70 dB, 80 dB and 90 dBSL were presented to five unilaterally deaf subjects in the deaf ear and the brainstem responses evoked from the normal hearing ears were recorded. In the third experiment, ABRs were recorded from nine normally hearing subjects. Clicks at 70 dB, 80 dB and 90 dB were presented to one ear, both with and without 50 dBSL of contralateral masking. Results of the first experiment showed that, in all subjects, the stimulus was masked out when 50 dBSL masking was used. In the second experiment, it was seen that wave V was present in all the subjects when a stimulus of 90 dBSL was presented to the deaf ear, this response was abolished by masking the normally hearing ear at 50 dBSL. In the third experiment, it was found that for waves I to V there were no significant latency or amplitude differences between the results obtained with

and without masking at each stimulus level, although, there was a 19% increase in the amplitude of wave V with masking at a 90 dBSL click level. However, for wave VI the amplitude was significantly reduced with masking when the stimulus was set at 90 dBSL, but there was no significant effect at the lower stimulus level. Without masking, the amplitude of wave-VI increased noticeably with an increases in stimulus intensity. With 50 dBSL of masking such an increase was not evident. It was also seen that, with no masking, the amplitude of wave-VI at a 90 dBSL stimulus level differed significantly from the amplitude at a 70 dBSL and at an 80 dBSL stimulus level. With masking at 50 dBSL the difference between stimulus levels was not significant. It was also seen that as the stimulus intensity increased the latency of wave VI decreased. This latency change was more noticeable with no contralateral masking. An explanation given for this phenomenon was that the presence of wave V from the contralateral ear is affecting wave V and VI from the test ear.

< Central masking is said to occur when the level of a sound presented to one ear is affected by the presentation of a masker to the contralateral ear. Prasker and Cohen (1984) studied the selective effects of contralateral masking on brainstem potentials. They observed that the mean reduction in amplitude of wave V from monaural clicks to clicks with pulsed noise at the contralateral ear is 41% whilst the mean increase with binaural clicks is 52%.

The latency of wave V did not seem to be affected significantly by the different stimulus conditions. The computer sum of the responses recorded with the click and noise pulses presented independently resulted in no significant effect on wave V amplitude. As the computer sum represents to a first approximation ihe binaural summation of activity at the two ears, the efferent suppression, or central masking, may be responsible for the difference between the computer sum and the actual recording with the click and pulse noise presented simultaneously.The fact that only wave V was affected by the pulsed white noise at the contralateral ear suggests that the area of mediation of the central masking effect is caudal to the site of generation of wave V. The enhancement of wave V amplitude on binaural click presentation may be attributed to binaural summation of activity from the two ears. There is evidence that certain neural elements are only activated by dichotic stimulation thus leading to a greater amplitude when recorded from remote electrodes, however, as the increase in amplitude of wave V with binaural stimulation is not twice. the monaural response amplitude, the difference may be due to the summation of 'suppressed' activity from the two ears. On binaural stimulation either side influences the afferent impulses at the other via the efferent pathway leading to reduced activity at the two ears. The summation of activity at the two sides resulting in an amplitude greater than twice the monaural response but less than twice the monaural sum.

In this respect it is of note that Galambos (1956) and others (Fex, 1959; Desmedt and Manaco 1961; Sohmer 1965) have shown that direct stimulation of Rasmussen's efferent oliva cochlea, bundles reduces the afferent click evolved activity in the auditory nerve.

The effect of ipsilateral noise on the middle components of the AER was investigated by Smith and Goldstein (1973). Peak to peak amplitudes were measured in response to clicks in the presence of white noise win the same ear. Peak to peak amplitude varied directly with signal to noise ratio. Amplitudes measured from negative signal-to-noise and silent control condition did not differ from each other. Smith and Goldstein assumed that wave forms from the inaudible (masked) signals took their shapes from the ongoing activity of the brain.

Gutnick and Goldstein (1978) studied the effect of contralateral noise on the middle components of the averaged electroencephalic response. Three levels of signal (20, 40 dBSL and silent control) and four levels of masker (20, 40 and 80 dBSL and silent control) were used. The results of this study showed that the amplitude for conditions with signal at silent control was significantly different from the amplitude for conditions with the signal at 20 and 40 dBSL. The continuously on masker, did not produce aggregate time looked neural activity.

necessary for a replicable AER. Rather,the AER took its shape from the filtered and summed back ground electroencephatic activity and other electrophysiologic noise. The masker in the contralateral ear did not degrade the middle component AER evoked by the signal in the test ear when the masker was at 20 or 40 dBSL. The masker at 20 or 40 dBSL produced +0.7 and -0.7 dB of threshold shift, both of which were non significant. The significant masker level effect, was attributable to pair wise comparison involving the 80 dBSL masker. It produced a mean signal threshold shift of -16.6 dB which may be attributable either to activation of the acoustic reflex or to transcranial masking. The 80 dBSL masker appeared to reduce the AER amplitude only when it significantly reduced the loudness of the signal.

Binaural Interaction:-

Binaural Interaction (BI) potential is derived by subtracting the sum of the left and right monaural responses from the binaural response (Parker and Salt 1982).

According to Dobie and Berlin (1979), binaural interaction is defined as any deviation from the predictions of a non interactive linear model. This model assumes that there are two sets of BSER 'generators' one for each ear, and that binaurally evoked BSER's would be predicted exactly by summation of the separate monaurally evoked BSER traes. This summated response (P) is

subtracted from the binaurally evoked response (B) to yield a difference trace (D), which may be regarded as representing derived, binaural interaction.

Dobie and Berlin (1979) observed the presence of BI in the brain stem evoked response of the guinea pig; linear summation of the monaural BSER did not predict the binaural BSER. This nonlinearity affected both, the peak- to-peak amplitude of wave IV and the latency of peak V. since the effects of binaural interaction on the BSER wave form are complex, the magnitude of BI ia best estimated by the magnitude of DN_1P_2 complex in the (D) trace, rather than by comparison of wave IV amplitude from peak measurements. The magnitude of BI was large (92.8% of wave IV amplitude on the average), and varied very little in a test-retest situation. Defenite BI was run at stimulus intensity levels of 87 and 67 dBpeSPL, with interaural intensity difference of + 20 dB and with interaural time difference of upto $\pm$ 1000 JUS. They also noticed that the latency of this interaction (3.3 to 4.0 ms) was most consistent with an effect at the level of 3rd order neurons, many of which are likely to arise from the superior olivary complex. Binaurally innervated neurons may receive excitatory input from both ears (E-E) or excitatory input frorp6ne ear and inhibitory input from the other (E-I), either type can explain the effect observed, and units of both types are present in the superior olivary complex (Moushigian, Ruport, Gidda, 1975).

Hall and Goldstein (1968) in their study on unanesthetized cats observed that by far, the most commonly encountered binaural interaction was one in which the response to binaural stimulation was judged to be greater in size than the response to stimulation of either ear alone at the same frequency and sound pressure level. This interaction is referred to as summation. In a less frequently encountered form of binaural interaction, stimulation of one ear made the response to stimulation of the other ear weaker. They termed this form of interaction 'inhibition'.

Denker and Houze (1982) found evidence of binaural interaction in ABR at equal sensation levels. The fact that difference trace deflections are present even when subjective loudness level is balanced across conditions suggests a qualitative difference in the neural response to binaural summation.

Parker and Sclt (1982) in their study on factors affecting the binaural interaction of the auditory brainstem response observed that binaural interaction potentials exhibited marked phase change between temporal and mastoid locations. It was seen that differential electrode pairs using locations where Binaural Interaction potentials were antiphasic, gave large Binaural Interaction potentials. Symmetrical displacement of the binaural image to the left and right by introducing (a) interaural intensity or (b) interaural time delay differences did not necessarily produce symmetric difference in the BI potential.

Galbraith, Aine, Squires and Buchwald (1983) studied BI in auditory brain stem responses of mentally retarded in-dividuals. The retarded individuals did not differ from control subjects when the amplitude of binaural auditory brainstem responses were compared to the computer summation of such responses evoked by left and right ear stimulation? however, there was evidence for a general binaural interac-tion effect.

Yamachi, Yamamoto, Nakamura and Iwanaga (1981) studied binaural interaction in auditory brainstem responses. The results of the study suggest that the neural organization the binaural interaction takes place at the level where waves V and VI are generated. And trapezoid body transection leads to a loss of this effect in waves V and VI.

Gardi and Berlin (1981) did a study for a search for the neural structures responsible for the generation of the BI component. They concluded as follows: the BIC must be generated by neurons more rostrally situated along the auditory pathway than those comprising the central nuclei of the inferior colliculus. They also discovered that, in general, higher frequency tone pips were more likely to generate a BIC than lower frequency tone pips. Thus the BIC waveform can be accounted

for by the presence of high frequency neurons firing in response to equal intensity signals presented binaurally to each ear. Based on this reasoning, the only primarily high frequency sensitive region inthe proposed generator area is the lateral superior olive.

Peters and Mendel (1974) in a study of middle latency auditory evoked potentials, found that monaural and binaural clicks of equal loudness yielded equal response amplitudes and latency. Though this was not a test of BI, the findings are in contrast to those observed by Dobie and Norton (1980) who found BI in middle latency responses. They observed that binaural responses were much larger than monaural responses, even when elicited by stimuli 20-30 dB less intense. They feel that this difference in the two studies could be due to the difference in the neural mechanisms underlying the generation of the middle component auditory evoked potential and the generation of the auditory brainstem response. Denker and Houie (1982) have contradicted this statement by saying that the two mechanisms are identical.

Kadobayashi et al (1984), compared middle latency responses for monaural and binaural stimuli. The early components of the middle latency responses for binaural stimulation had larger amplitudes than those for monaural stimulation. This has not been reported by other authors, although comparison of brainstem

auditory evoked potentials for monaural and binaural stimuli has been made (Ainslie and Boston (1980); Dobie and Norton (1980), in which, all peak-to-peak amplitudes of the early components under binaural stimulation were found to be larger than those obtained under monaural stimulation. This suggests that impulses from the right and left ears elicited the response in the brainstem.

Thus, from the review of literature on middle evoked response, one can see that no pertinent literature is available regarding the effect of contralateral noise and binaural interaction on the middle evoked response, hence this study has been taken up.

METHODOLOGY

METHODOLOGY

Subjects:

Thirteen female and nine male subjects in the age range of 18 to 23 years were selected from the student population of All India Institute of Speech and Hearing.

The subjects selected for the study, had no history of any ear discharge, earache, tinnitus, giddiness, headache, brain damage or exposure to loud sounds.

All the subjects had hearing sensitivity within 20 dBHL (ANSI, 1969) in the frequencies 250 Hz, 500 Hz, 1KHz, 2KHz and 4 KHz.

Instruments used:

Beltone 200-C audiometer with TDH-39 earphones and circum-aural cushion Mx-41/AR were used for obtaining pure tone thresholds at 2 KHz.

Electric Response Audiometer Model TA-1000 was used. It consisted of a SLZ-9793 desk top console which contains all of the; operating controls, indications and readouts for the system, SLZ 9794 preamplifier which is an isolated preamplifier with frequency response and gain specifically designed for ERA, a set of standard silver chloride electrodes, TDH-39 earphones and

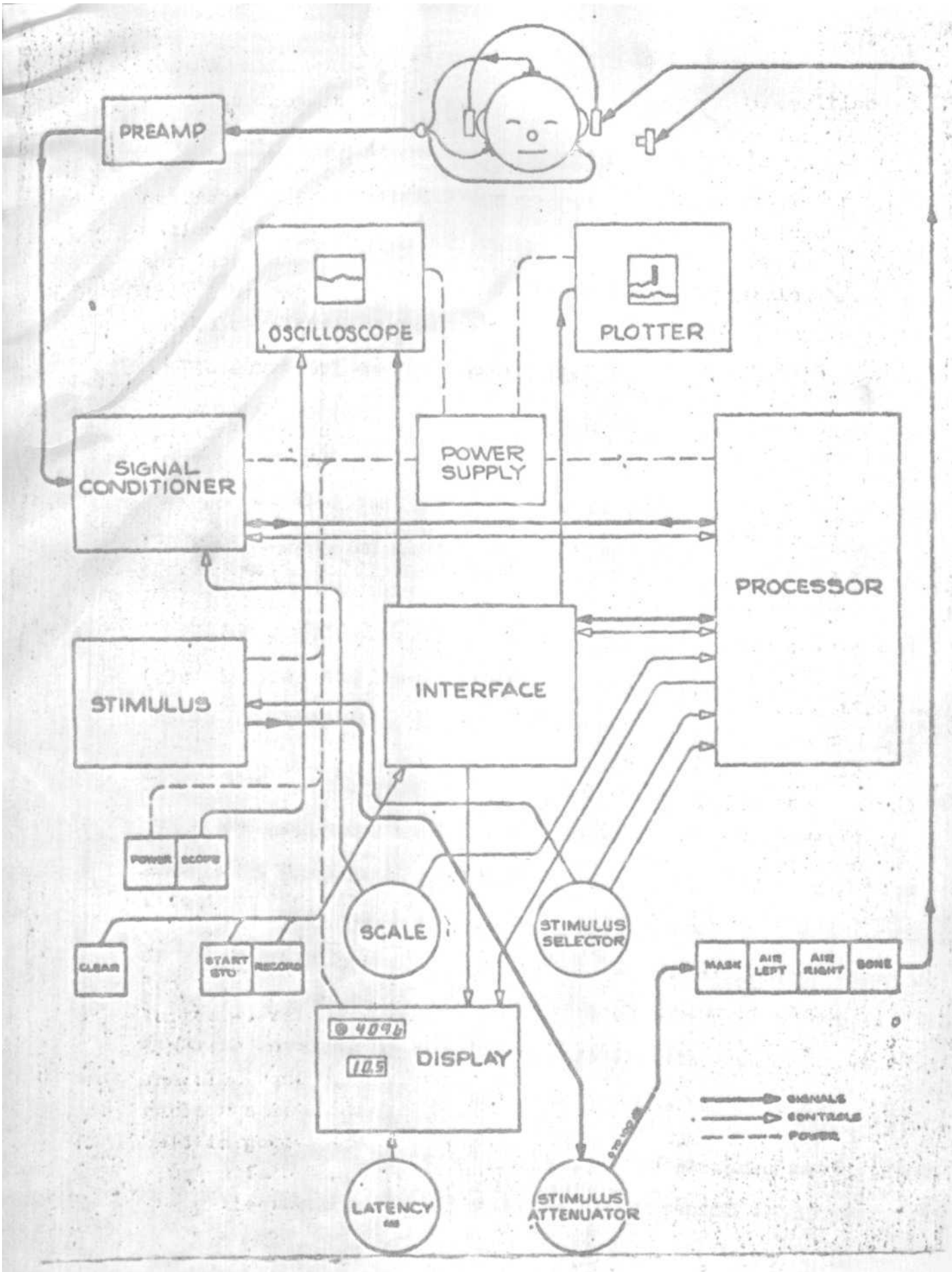


Fig. 8 - Flow chart of ERA: TA-1000 used in the present study.

circumaural cushion MX-41/AR.Calibrated paper was used to record the responses and electrolyte gel, adhesive tape and spirit was used for purpose of fixing the electrodes.

TA-1000 operates with four knobs and nine push button switches.

The knobs are:-

1. The stimulation 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 Attenuator Knob, which permits selection of acoustic logon stimuli from 0 dBHL to 100 dBHL.

3. The Scale Function Knob, which permits selection of system sensitivity and number of average response samples, that is for 1024 samples 0.5, 1 and 5 uV per division sensitivity are available. For 2048 samples, 0.2, 0.5, 1 and 2 uV per division sensitivities are available. And for 4096 samples 0.1, 0+2, 0.5 and 1 uV per division sensitivities are available.

4. The Latency Control Knob, provides a curson mark on the oscilloscope display of the Middle Response Wave for precise

determination of latency. Readout of latency in msec to 0.2 ms 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 selected number of samples has accumulated or when any average memory channel is full. The average can be stopped to evaluate intermediate results and restarted without disturbing the average action.
5. Record: Records the wave onto the calibrated paper.
6. Mask: Provides broad band noise to the contralateral ear only when either air left or air right stimulus is presented.
7. Air Left: Provides stimulus to the left ear phone.
8. Air Right: Provides stimulus to the right ear phone.
9. Bone:Provides stimulus to the 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 EEC position,after a clear, oscilloscope will display the patients ongoing EEC activity.

A stimulus generator was used to generate logon signals. The logon stimulus is characterized by three peaks in a 50% negative, 100% positive, 50% negative sequence followed by a 50% positive, 100% negative, 50% positive sequence revarsing 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. The ambient noise level present in the test room were below the proposed maximum allowable noise levels.

Procedure:

All the subjects were screened at 20 dBHL in the frequencies 250 Hz, 500 Hz, 1 KHz, 2 KHz and 4 KHz to find the presence or absence of a hearing loss in both the ears.

The study consisted of three different experiments.

Experiment-1:

Eight female and 7 male subjects were explained the nature of the test. They were made to lie down comfortably on a bed

with a pillow to reduce neck muscle tension and there by artefacts. The subjects were told to either relax with eyes closed or to sleep.

The preamplifier was located very near to the subjects 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 electrode gel was fixed to the cleaned skin with the help of adhesive tape.

The 3 electrodes were placed as follows:

Red or signal electrode was placed on the high forehead.
White or reference electrode on the mastoid of the test ear.
BlacK or ground electrode on the mastoid of the nontest ear.

The earphones were placed and the power button was switched on. The TWF/Run/EEG 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 20 ms.

The stimulus attenuator knob was turned to select a stimulus of 60 dB.

The scale function knob was turned to provide 2048 stimuli at 0.2 uV per division sensitivity.

By means of the push button, the logon stimulus was presented to the right ear in four females and four males, and to the left ear in four females and three males.

The 'clear' push button was then pressed to clear the microprocessor, and the start push button was switched on. After the samples were accumulated, the average function automatically stopped. The response was recorded on to the calibrated paper and the number of samples accumulated was noted down since it was observed that in some cases, the samples did not accumulate upto 2048, even after a number of tries.

Next, the response was recorded with the presence of masking noise in the contralateral ear.

The same procedure was repeated to obtain recordings at 80 dB, 100 dB, and at 4 KHz - 60 dB, 30 dB and 100 dB in the absence and presence of contralateral noise.

The following were determined from the recordings;

1. Latency: The latency of peak Po was determined by counting the number of vertical lines on the calibrated paper from the point the curve starts to the peak of the wave. Each vertical straitian is equal to 0.2 ms.

2. Amplitude: The magnitude of the patients response in micro volts was determined using the formula:

$$= \frac{N}{n} \times \frac{TxS}{M} \quad .$$

where,

N = number of samples present on the scale.

n = number of samples actually counted.

T = The amplitude of the desired trace feature.

S = The sensitivity.

M = Masker amplitude.

The same procedure was then carried out on nine females and four males using 2048 samples, 1 uV per division since it was observed that with reduction in sensitivity,the peak Po appeared more clear.

Experiment-II:

To find the difference in amplitude and latency of peak V of the early response when recorded using patient response interval of 10 ms and 20 ms.

Six female and two males who had acted as subjects in experiment-I were explained the nature of the test. They were made to lie down comfortably on a bed with a pillow. Earphones were placed on the subject after fixing the electrodes.

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 interval at 10 ms.

The stimulus attenuator knob was turned to select a stimulus of 80 dB.

The scale function knob was turned to provide 2048 samples at 0.2 uV per division sensitivity.

With this setting, the early response was obtained and latency and amplitude of the V peak was determined.

The same procedure was repeated to obtain an early response at 4 KHz, 80 dB.

The amplitude and latency of peak V using patient response interval of 20 ms was measured directly from the response recording obtained in experiment-I.

The data obtained was then analysed statistically using Wilcoxon matched pairs signed, rank test.

Experiment-III:

To determine Binaural Interaction.

Thresholds for the subjects were determined for a 2 KHz pure tone. Only those subjects having equal thresholds in both ears were chosen for the experiment.

Seven female and three male subjects were first explained

the nature of the test. They were made to lie down comfortably on a bed with a pillow. The electrodes were fixed and the earphones were placed on the subject.

A 70 dBSL logon stimulus was presented to the right ear and the middle response was recorded while keeping the stimulus function knob at frequency 2 KHz, repetitive rate of 20 stimuli per second and patients response interval at 20 ms. The scale function knob was set at 2048 samples, 0.2 uV p r division sensitivity.

The same procedure was repeated to obtain a middle response in the left ear and middle response when both the ears were presented with logon stimuli simultaneously.

The amplitude of peak Po was calculated using the formula

$$= \frac{N \times TxS}{n \; M}$$

Binaural Interaction was calculated using the formula.

Binaural Interaction = Binaural Response - predicted Response.

Predicted response = Response of right ear + response of left ear.

RESULTS AND DISCUSSIONS ,

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RESULTS AND DISCUSSION

Results:

The results were analysed statistically using Wilcoxon signed ranks test. Table la, shows the latency and amplitude of peak Po, measured for a stimulus of 2 KHz and table lb shows the Latency and amplitude of peak P0, measured for a stimulus of 4 KHz with sensitivity at 0.2 uV, in the presence and absence of contralateral noise. The results show that at 2 KHz 60 dBHL five out of fifteen subjects show clear peaks in the absence of contralateral noise and only four out of fifteen subjects show clear peaks in the presence of contra-lateral noise. At 2 KHz 80 dBHL, only one subject shows a clear peak in the absence of contralateral noise, where as four out of fifteen subjects show clear peaks in the presence of contra-lateral noise. At 2 KHz 100 dBHL six subjects show clear peaks in the presence and absence of contralateral noise. At 4 KHz 60 dBHL, three of the subjects show clear peaks in tne absence of contralateral noise, while in the presence of contralateral noise, four subjects show clear peaks. At 4 KHz 80 dBHL, three subjects show clear peaks in the absence and presence of contra-lateral noise. At 4 KHz 100 dBHL, out of fifteen subjects, four subjects show a clear response in the absence of contralateral noise where as five subjects show clear responses in the presence of contralateral noise.

Table-la: Showing the latency and amplitude of peak Po, for 2 KHz at sen&itivity 0.2 uV in the presence and absence of contralateral noise.

Sl.No. of subjects	2 KHz											
	60		60+N		80		80 + N		100		100 + N	
	Latency ms.	Amplitude uV.	Latency ms.	Amplitude uV.	Latency ms.	Amplitude uV.	Latency ms	Amplitude uV.	Latency ms	Amplitude uV.	Latency ms	Amplitude uV.
1.	12.8	0.35	12.6	0.3	-	-	10.4	0.7	12.4	0.25	-	-
2.	-	-	-	-	-	-	-	-	14.6	1.0	-	-
3.	-	-	-	-	-	-	-	-	11.6	0.2	11.2	0.4
4.	11.6	2.04	12.2	0.45	12	0.7	11	2.4	10.8	0.45	10.6	0.6
5.	13.8	1	11.8	0.35	-	-	9.8	1.0	11.8	0.25	9.8	1.16
6.	-	-	-	-	-	-	-	-	-	-	-	-
7.	-	-	-	-	-	-	-	-	-	-	-	-
8.	-	-	-	-	-	-	-	-	-	-	-	-
9.	-	-	-	-	-	-	-	-	-	-	10.0	2.75
10.	13.4	0.5	-	-	-	-	-	-	11.0	1.2	10.4	0.7
11.	-	-	-	-	-	-	13.8	0.35	13.6	0.25	13.40	0.3
12.	-	-	-	-	-	-	-	-	-	-	-	-
13.	-	-	-	-	-	-	-	-	-	-	-	-
14.	13.0	0.55	13.6	0.35	-	-	-	-	-	-	11.2	0.7
15.	-	-	-	-	-	-	-	-	-	-	-	-

Table-lb: Showing the latency and amplitude of peak Po, for 4 KHz at sensitivity 0.2 uV in the presence and absence of contralateral noise.

Sl.No. of subjects	4 KHz											
	60		60+N		80		80+N		100		100+N	
	Latency ms	Amplitude μV.	Latency ms	Amplitude μV.	Latency ms	Amplitude μV.	Latency ms	Amplitude μV.	Latency ms	Amplitude μV.	Latency ms	Amplitude μV.
1	12.8	0.4	13.6	0.2	12.2	0.4	10.2	0.4	12.2	0.25	10.2	0.25
2	-	-	-	-	-	-	-	-	-	-	-	-
3	-	-	-	-	-	-	-	-	11.0	1.71	10.2	2.8
4	13	0.5	13.4	0.3	-	-	-	-	-	-	-	-
5	12	0.2	10.6	1.72	10.6	1.44	10.4	4	10.8	4.5	10.2	5
6	-	-	-	-	-	-	-	-	-	-	-	-
7	-	-	-	-	-	-	-	-	-	-	-	-
8	-	-	-	-	-	-	-	-	-	-	-	-
9	-	-	-	-	-	-	10.4	1.5	-	-	-	-
10	-	-	-	-	-	-	-	-	-	-	-	-
11	-	-	13.5	0.15	14.2	0.4	-	-	-	-	13.4	0.5
12	-	-	-	-	-	-	-	-	12-	0.85	11.8	0.38
13	-	-	-	-	-	-	-	-	-	-	-	-
14	-	-	-	-	-	-	-	-	-	-	-	-
15	-	-	-	-	-	-	-	-	-	-	-	-

Table-2a shows the latency and amplitude of peak Po, measured for a stimulus of 2 KHz and Table-2b shows the latency and amplitude of peak Po measured for a stimulus of 4 KHz with sensitivity at 1 uV, in the presence and absence of contralateral noise. The results show that at 2 KHz 80 dBHL, only one of the subjects showed a clear peak in the absence of contralateral noise, where as in the presence of contralateral noise, two out of the thirteen subjects showed clear peaks. At 2 KHz 80 dBHL seven subjects showed clear peaks in the absence of contralateral noise and six subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 80 dBHL+ Four subjects showed clear peaks in the absence of contralateral noise and two subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 100 dBHL, six out of the thirteen subjects showed clear peaks in the presence and absence of contealateral noise.

Table-3a shows the amplitude and latency of the VI peak measured at 2 KHz and Table-3b shows the amplitude and latency of the VI peak measured at 4 KHz with sensitivity at 0.2 uV, in the presence ana absence of contralateral noise. The results show that at 2 KHz 60 dBHL six subjects showed clear peaks, whereas in the presence of contralateral noise, eight subjects showed clear peaks. At 2 KHz 80 dBHL, ten out of the fifteen subjects showed clear peaks, whereas in the presence of contra-

Table-2a: Showing the latency and amplitude of peak Po for 2 KHz at sensitivity 1 uV in the presence and absence of contralateral noise.

Sl.No. of Subjects.	2 KHz							
	80		80+N		100		100+N	
	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV
1	-	-	-	-	12.2	1.0	-	-
2	-	-	-	-	10.2	0.94	10.2	1.0
3	-	-	-	-	12.2	0.5	10.8	1.25
4	11.0	0.56	11.0	1.25	-	-	10.8	0.56
5	-	-	-	-	-	-	-	-
6	-	-	-	-	12.2	2.5	11.2	0.75
7	-	-	-	-	-	-	-	-
8	-	-	-	-	-	-	-	-
9	-	-	-	-	-	-	-	-
10	-	-	12.0	0.5	11.6	0.7	11.6	0.5
11	-	-	-	-	11.2	2.9	10.6	2.0
12	-	-	-	-	-	-	-	-
13	-	-	-	-	12.0	1.37	-	-

Table-2b: Showing the latency and amplitude of Peak Po, for 4 KHz at sensitivity 1 uV, in the presence and absence of contralateral noise.

4 KHz

Sl.No. of subjects.	80		80+N		100		100+N	
	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms.	Amplitude μV	Latency ms.	Amplitude μV
1	-	-	-	-	-	-	-	-
2	-	-	-	-	-	-	10.5	1.0
3	-	-	-	-	12.4	0.37	10.6	3.37
4	12.2	0.44	-	-	12	0.62	11.8	2.0
5	11.6	0.5	-	-	11	0.5	-	-
6	11.4	1.25	11.2	0.87	10.8	0.37	10.6	0.81
7	-	-	-	-	-	-	-	-
8	-	-	-	-	-	-	-	-
9	-	-	-	-	-	-	10.7	0.5
10	12.4	0.75	12.2	0.81	12.4	0.62	12.0	1.12
11	-	-	-	-	12.4	0.5	-	-
12	-	-	-	-	-	-	-	-
13	-	-	-	-	-	-	-	-

Table-3a: Showing the latency and amplitude of the VI peak for 2 KHz with sensitivity at 0.2 uV in the presence and absence of contralateral noise.

Sl.No. of subjects	2 KHz											
	60		60+N		80		80+N		100		100+N	
	Latency ms.	Amplitude μV	Latency ms.	Amplitude μV	Latency ms.	Amplitude μV	Latency ms.	Amplitude μV	Latency ms.	Amplitude μV	Latency ms	Amplitude μV
1.	7.0	0.2	7.1	0.15	6.0	0.25	6.6	0.05	-	-	6.2	0.05
2.	7.0	0.10	7.0	0.43	6.5	0.25	6.2	0.3	5.8	0.55	5.7	0.45
3.	6.4	0.55	6.4	0.4	6.2	0.15	6.2	0.3	-	-	-	-
4.	-	-	-	-	-	-	6.4	0.35	-	-	-	-
5.	-	-	-	-	6.4	0.2	6.8	0.05	-	-	5.6	0.33
6.	6.8	0.25	6.4	0.25	6.2	0.10	5.8	0.4	6.0	0.56	-	-
7.	-	-	-	-	-	-	6.6	0.15	6.8	0.45	6.6	0.15
8.	-	-	-	-	6.2	0.4	6.0	0.3	6.0	0.21	5.8	0.35
9.	-	-	7.2	0.3	-	-	-	-	6.6	0.3	-	-
10.	-	-	-	-	6.8	0.2	-	-	-	-	-	-
11.	-	-	-	-	-	-	-	-	-	-	-	-
12.	-	-	7.0	0.18	7.0	0.18	7.0	0.1	6.6	0.3	6.6	0.25
13.	7.2	0.2	6.8	0.3	6.8	0.3	6.6	0.35	6.0	0.5	-	-
14.	-	-	-	-	-	-	-	-	-	-	-	-
15.	-	-	7.2	0.13	6.8	0.15	6.6	0.25	6.0	0.4	6.0	0.5

Table-3b: Showing the latency and amplitude of the VI Peak for 4 KHz with sensitivity at 0.2 uV in the presence and absence of contralateral noise.

Sl.No. of Subjects	4 KHz 60 Latency ms	60 Amplitude µV	60+N Latency ms	60+N Amplitude µV	80 Latency ms	80 Amplitude µV	80+N Latency ms	80+N Amplitude µV	100 Latency ms	100 Amplitude µV	100+N Latency ms	100+N Amplitude µV
1	-	-	-	-	-	-	-	-	6.4	0.4	6.8	0.25
2	6.2	0.3	6.2	0.2	6.2	0.35	-	-	5.8	0.7	6.0	0.55
3	-	-	-	-	6.2	0.10	6.0	0.2	6.2	0.63	5.8	0.6
4	6.6	0.25	6.6	0.3	6.6	0.4	6.6	0.3	6.0	0.55	-	-
5	6.8	0.2	7.0	0.18	-	-	-	-	-	-	-	-
6	6.6	0.2	-	-	6.4	0.25	6.2	0.45	6.2	0.6	6.2	0.4
7	-	-	6.8	0.4	6.2	0.4	-	-	6.6	0.4	6.0	0.4
8	6.4	0.2	6.4	0.2	6.2	0.2	6.2	0.35	6.2	0.47	6.0	0.48
9	-	-	-	-	7.0	0.18	7.0	0.18	6.6	0.2	-	-
10	6.6	0.4	6.6	0.4	-	-	-	-	-	-	-	-
11	-	-	-	-	-	-	-	-	6.2	0.42	6.6	0.52
12	-	-	7.4	0.13	7.0	0.25	7.2	0.15	6.8	0.6	6.8	0.4
13	-	-	-	-	-	-	6.2	0.7	6.4	0.7	6.4	0.55
14	-	-	-	-	-	-	-	-	-	-	-	-
15	7.2	0.2	-	-	6.8	0.25	-	-	6.4	0.46	6.0	0.5

lateral noise, eleven subjects showed clear peaks. At 2 KHz 100 dBHL eight subjects showed clear peaks, and only seven subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 60 dBHL seven subjects showed clear peaks in the absence and presence of contralateral noise. At 4 KHz 80 dBHL, nine subjects showed clear peaks, seven subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 100 dBHL twelve subjects showed clear peaks in the absence of contralateral noise and 10 subjects showed clear peaks in the presence of contralateral noise.

Table-4a shows the amplitude and latency of the VII peak measured at 2 KHz and Table-4b shows the amplitude and latency of the VII peak measured at 4 KHz with sensitivity/in the presence and absence of contralateral noise . The results show that at 2 KHz 60 dBHL, in the absence of contralateral noise, two out of the 15 subjects showed clear peaks, where as in the presence of contralateral noise, none of the subjects showed clear peaks. At 2 KHz 80 dBHL five subjects showed clear peaks and in the presence of contralateral noise, four subjects showed clear peaks. At 2 KHz 100 dBHL five subjects showed clear peaks in the absence of contralateral noise and 6 subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 60 dBHL, only one subject showed clear peaks, where is in the presence of contralateral noise, five subjects showed clear peaks. At

at 0.2 uV

Table-4a: Showing the Latency and Amplitude of the VII Peak for 2 KHz with sensitivity at 0.2 uV in the presence and absence of contralateral noise.

Sl.No. of subjects.	2 KHz											
	60		60+N		80		80+N		100		100+N	
	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV
1	-	-	-	-	-	-	-	-	-	-	8.2	0.05
2	-	-	-	-	-	-	8.6	0.2	8.0	0.05	8.0	0.15
3	-	-	-	-	7.4	0.15	-	-	-	-	-	-
4	-	-	-	-	-	-	-	-	-	-	-	-
5	-	-	-	-	-	-	-	-	-	-	-	-
6	-	-	-	-	-	-	-	-	8.0	0.2	-	-
7	-	-	-	-	-	-	-	-	8.4	0.25	7.8	0.4
8	-	-	-	-	8.0	0.05	8.0	0.05	7.8	0.35	7.0	0.10
9	-	-	-	-	-	-	-	-	-	-	-	-
10	-	-	-	-	-	-	-	-	8.1	0.11	8.0	0.25
11	-	-	-	-	8.6	0.1	-	-	-	-	8.6	0.25
12	8.6	0.15	-	-	-	-	-	-	-	-	-	-
13	9.2	0.05	-	-	8.4	0.3	8.4	0.05	-	-	-	-
14	-	-	-	-	-	-	-	-	-	-	-	-
15	-	-	-	-	8.8	0.05	8.8	0.05	-	-	-	-

Table-4b: Showing the Latency and Amplitude of the VII Peak for 4 KHz with sensitivity at 0.2 uV in the presence and absence of contralateral noise.

	4 KHz											
	60		60+N		80		80+N		100		100+N	
Sl.No. of Subjects	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV
1	-	-	-	-	-	-	-	-	8.6	0.05	-	-
2	7.9	0.05	8.0	0.2	-	-	-	-	8.2	0.05	-	-
3	-	-	-	-	7.8	0.05	-	-	7.8	0.09	-	-
4	-	-	-	-	-	-	-	-	7.4	0.25	-	-
5	-	-	-	-	-	-	-	-	-	-	-	-
6	-	-	8.2	0.05	8.2	0.05	8.2	0.05	8.0	0.2	-	-
7	-	-	9.4	1.5	-	-	-	-	8.6	0.2	8.4	0.2
8	-	-	8.4	0.05	7.9	0.15	7.8	0.05	-	-	-	-
9	-	-	-	-	-	-	-	-	-	-	-	-
10	-	-	-	-	-	-	-	-	-	-	-	-
11	-	-	9.4	0.2	-	-	-	-	-	-	-	-
12	-	-	-	-	-	-	-	-	-	-	-	-
13	-	-	-	-	-	-	-	-	8.3	0.15	8.6	0.15
14	-	-	-	-	-	-	-	-	-	-	-	-
15	-	-	-	-	-	-	-	-	-	-	-	-

4 KHz 80 dBHL three subjects showed clear peaks and in the presence of contralateral noise, two subjects showed clear peaks. At 4 KHz 100 dBHL, seven subjects showed clear peaks, but in the presence of contralateral noise only two subjects showed clear peaks.

Table-5a shows the amplitude and latency of the VI peak measured at 2 KHz and Table-5b shows the amplitude and latency of the VI peak measured at 4 KHz, with sensitivity at 1 uV in the presence and absence of contralateral noise. The results show that at 2 KHz 80 dBHL, six subjects had clear peaks, where as in the presence of contralateral noise, only one of the subjects showed clear peaks. At 2 KHz 100 dBHL, six subjects show clear peaks in the absence/contralateral noise and five subjects show clear peaks in the presence of contralateral noise. At 4 KHz 80 dBHL five subjects show clear peaks, but in the presence of contralateral noise, only two subjects show clear peaks. At 4 KHz 100 dBHL, ten subjects show clear peaks and in the presence of contralateral noise, eight subjects showed clear peaks.

Table-6a shows the amplitude and latency of the VII peak measured at 2 KHz and Table-6b shows the amplitude and latency of the VII peak measured at 4 KHz with sensitivity at 1 uV in the presence and absence of contralateral noise. The results show that at 2 KHz 80 dBHL two out of the thirteen subjects had

Table-5a : Showing the latency and amplitude of the VI Peak for 2 KHz with sensitivity at 1 uV in the presence and absence of contralateral noise.

Sl.No. of subjects.	2 KHz							
	80		80+N		100		100+N	
	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV
1	-	-	-	-	-	-	-	-
2	-	-	-	-	6.0	0.25	5.9	0.19
3	6.8	0.25	-	-	-	-	-	-
4	6.2	0.13	6.2	0.13	5.4	0.3	5.3	0.25
5	6.2	0.37	-	-	5.9	0.5	5.8	0.37
6	-	-	-	-	-	-	-	-
7	5.8	0.19	-	-	5.8	0.19	-	-
8	-	-	-	-	6.0	0.56	6.0	0.13
9	-	-	-	-	6.2	0.13	6.0	0.37
10	-	-	-	-	-	-	-	-
11	6.6	0.19	-	-	-	-	-	-
12	-	-	-	-	-	-	-	-
13	6.5	0.19	-	-	-	-	-	-

Table-5b: Showing the latency and amplitude of the VI Peak for 4 KHz with sensitivity at 1 uV in the presence and absence of contralateral noise.

Sl.No. of subjects	4 KHz							
	80		80+N		100		100+N	
	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV	Latency ms	Amplitude μV
1	-	-	-	-	-	-	-	-
2	-	-	-	-	6.0	0.56	5.9	0.5
3	-	-	-	-	6.5	0.5	6.5	0.5
4	6.4	0.25	-	-	6.1	0.5	6.1	0.06
5	6.2	0.25	-	-	6.0	0.75	5.9	0.3
6	6.4	0.13	6.1	0.25	5.9	0.25	5.8	0.19
7	5.9	0.25	-	-	5.9	0.37	-	-
8	-	-	-	-	6.2	0.69	6.2	0.75
9	-	-	-	-	6.4	0.5	6.4	0.44
10	6.4	0.37	6.4	0.25	-	-	-	-
11	-	-	-	-	6.8	0.5	6.6	0.37
12	-	-	-	-	6.5	0.31	-	-
13	-	-	-	-	-	-	-	-

Table-6a: Showing the latency and amplitude of the VII Peak for 2 KHz (with sensitivity at 1 uV) in the presence and absence of contralateral noise.

Sl.No. of subjects	2 KHz							
	80		80+N		100		100+N	
	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms.	Amplitude µV.	Latency ms	Amplitude µV
1	-	-	-	-	-	-	-	-
2	-	-	-	-	8.2	0.125	7.2	0.125
3	-	-	-	-	-	-	-	-
4	-	-	-	-	8.2	0.125	-	-
5	8.0	0.125	-	-	-	-	6.6	0.125
6	-	-	-	-	7.8	0.25	7.5	0.125
7	-	-	-	-	-	-	-	-
8	-	-	-	-	8.5	0.25	8.0	0.06
9	-	-	-	-	-	-	-	-
10	-	-	-	-	-	-	-	-
11	-	-	-	-	-	-	-	-
12	-	-	-	-	-	-	-	-
13	8.4	0.06	-	-	-	-	-	-

Table-6b: Showing the latency and amplitude of the VII Peak for 4 KHz(with sensitivity at 1 UV)in the presence and absence of contralateral noise.

Sl.No. of subjects	4 KHz							
	80		80+N		100		100+N	
	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV	Latency ms	Amplitude µV
1	-	-	-	-	-	-	-	-
2	-	-	-	-	-	-	-	-
3	-	-	-	-	-	-	-	-
4	-	-	-	-	8.0	0.125	8.0	0.125
5	-	-	8.3	0.125	-	-	-	-
6	-	-	8.0	0.06	7.6	0.125	7.6	0.06
7	-	-	-	-	-	-	-	-
8	-	-	-	-	8.1	0.125	-	-
9	-	-	-	-	-	-	-	-
10	-	-	-	-	-	-	7.0	0.19
11	-	-	-	-	8.8	0.19	-	-
12	-	-	-	-	-	-	-	-
13	-	-	-	-	8.0	0.19	-	-

clear peaks, but none of the subjects had clear peaks in the absence or contralateral noise. At 2 KHz 100 dBHL, four subjects showed clear peaks in the presence and absence of contralateral noise. At 4 KHz 80 dBHL, none of the subjects showed clear peaks but two subjects showed clear peaks in the presence of contralateral noise. At 4 KHz 100 dBHL five ot the subjects showed clear peaks and only three subjects showed clear peaks in the presence of contralateral noise.

Table-7 shows the amplitude and latency of the V peak when measured using patient response interval of 10 ms and 20 ms, at 2 KHz 80 dBHL and 4 KHz 80 dBHL. The results show that all the subjects have clear peaks at both 10 ms and 20 ms patient response interval.

Table-8 shows mean and standard deviation for the amplitude and latency of the V peak when measured using patient response interval of 10 ms and 20 ms. The mean values of the V peak latency using 10 ms patient response interval at 2 KHz and 4 KHz 80 dBHL were found to be 1 ms more than the V peak latency using 20 ms patient response interval. The standard deviation of the V peak latency measured using 10 ms patient response interval at 2 KHz and 4 KHz 80 dBHL is significantly higher than the standard deviation of the V peak latency measured using 20 ms patient response interval at 2 KHz and 4 KHz 80 dBHL.

Table-7: showing the amplitude and latency of the V Peak for patient response interval, of 10 ms and 20 ms, for 2 KHz (80 dBHL) and 4 KHz (80 dBHL' stimuli.

SI.No. Of subjects.	20 KHz 80dB 20ms Latency msec	Amplitude Mv	4 KHz 80dB 20 ms. Latency msec	Amplitude Mv	2 KHz 80dB 10ms Latency msec	Amplitude mv	8 KHz 80 dB 10 ms. Latency	Amplitude
1	4.8	0.6	4.4	0.8	5.1	0.5	5.1	0.3
2	4.6	0.3	4.6	0.4	4.7	0.65	4.4	0.6
3	4.4	0.4	4.7	0.4	4.6	0.95	4.8	0.85
4	5.0	0.6	5.0	0.54	5.2	0.75	5.3	0.5
5	4.6	0.7	4.6	0.7	4.7	0.75	4.8	0.4
6	4.6	0.7	4.6	0.7	4.5	0.5	4.6	0.4
7	5.0	0.4	5.0	0.55	5.2	0.85	5.3	0.4
8	5.1	0.425	5.2	0.45	5.4	0.7	5.4	0.7

Table-8: Showing the mean and standard deviation for the amplitude and latency of V peak when measured using patient response interval of 10 ms and 20 ms.

	2 KHz 80 dBHL 20 ms latency msec	2 KHz 80 dBHL 20 ms ampli-tude µ v	4 KHz 80 dBHL 20 ms latency msec	4 KHz 80 dBHL 20 ms ampli-tude µ v	2 KHz 80 dBHL 10 ms latency msec	2 KHz 80 dBHL 10 ms ampli-tude µ .V	4 KHz 80 dBHL 10 ms latency msec	4 KHz 80 dBHL 10 ms Ampli-tude µ v.
Mean	4.8	0.5	4.8	0.6	4.9	0.7	4.9	0.6
Standard Deviation	0.24	0.14	0.26	0.14	0.32	0.14	0.35	0.14

Table-9 gives the Wilcoxon T values of significance for the V peak latency and amplitude at 2 KHz 80 dBHL and 4 KHz 80 dBHL, using patient response interval of 10 msec, and 20 msec. The results show that the T values for the V peak amplitude at 2 KHz 80 dBHL and 4 KHz 80 dBHL for 10 msec, and 20 msec, patient response interval is more than the T value given in the table for Wilcoxon T test of significance at the 0.05 level of significance. The T value for the latency of the V peak at 4 KHz 80 dBHL for 10 msec, and 20 msec, patient response interval is more than the T value given in the table for the Wilcoxon T test of significance at the 0.05 level of significance. The T value for the latency of the V peak at 2 KHz 80 dBHL for 10 msec, and 20 msec, patient response interval is less than the T value given in the table for the Wilcoxon test of significance.

Table-10 shows the values of the right ear amplitude, right ear latency, left ear amplitude, left ear I=tency, combined amplitude Binaural amplitude and Binaural latency values of peak Po (Middle response). Table-10 also shows the T values for the amplitude of middle response measured binaurally (amplitude of middle response obtained when both the ears were stimulated) and for the combined amplitude of the middle response (amplitude of response when right ear was stimulated + amplitude

Table-9: Showing the Wilcoxon T values of significance for the V peak amplitude and latency at 2 KHz 80 dBHL and 4 KHz 80 dBHL for patient response interval of 10 ms and 20 ms.

	2 KHz 80 dBHL	4 KHz 80 dBHL
Latency	Significant T = 2 N = 8	Not significant T = 3 N = 7
Amplitude	Not significant T = 5.5 N = 8	Not significant T = 13.5 N = 8

Table value at 0.05 level of significance for N = 8 = 4

Table value at 0.05 level of significance for N = 7 = 2

Table-10: Showing Right ear amplitude, Right ear latency, left ear amplitude, left ear latency, combined amplitude, Binaural amplitude and binaural latency values of peak Po (middle response). And the Wilcoxon T values of significance for combined amplitude and binaural amplitude of peak Po (middle response)

Sl.No. of subjects	Right Latency msec.	Right Amplitude uV	Left Latency uV	Left amplitude uV	Right Left Predicted amplidute uV	Binaural Latency msec.	Binaural amplitude uV	Binaural Amplitude uV-Predicted Amplitude uV
1	10.3	0.25	11.0	0.325	0.575	12	0.4	-0.175
2	10.4	0.1	10.6	2.02	2.13	10.6	3.05	+0.93
3	12.0	0.4	11.8	1.1	1.5	11.4	2.875	+1.375
4	12.4	0.075	12.4	0.05	0.125	13.0	0.01	-0.025
5	13.8	0.25	14.0	0.2	0.45	13.8	0.65	+ 0.2
6	12	0.025	12.0	0.05	0.075	11.8	0.1	+0.025
7	11.8	0.9	13.8	0.3	1.2	13.0	2.02	+0.82
8	11.8	1.05	11.4	0.85	1.9	11.8	2.26	+0.36
9	10.5	0.05	12.1	0.3	0.35	12.8	1.1	+0.75
10	10.2	1.58	9.0	0.83	2.41	9.6	0.99	-1.42

(T = 11.5 N=10) Shows no significant difference
Table value at 0.05 level of significance for N=10=8).

of response when left ear was stimulated). The results show that the T value for the combined amplitude of middle response and the values of the binaural amplitude response is greater than the value given in the table for Wilcoxon T test of significance at the 0.05 level of significance.

According to the results obtained from the study, the peaks VI, VII and Po did not emerge clearly enough in all the subjects for all stimulus condition to warrant extensive description or analysis.

Thus, the questions - "Is there any effect of contralateral noise on the latency and amplitude of the middle response at sensitivity = 0.2?".

"Is there any effect of contralateral noise on the latency and amplitude of the middle response at sensitivity = 1 uV?."

"Is there any effect of contralateral noise on the latency and amplitude of the VI peak at sensitivity = 0.2 uV?".

"Is there any effect of contralateral noise on the latency and amplitude of the VII peak at sensitivity = 0.2 uV?".

"Is there any effect of contralateral noise on the latency and amplitude of the VI peak at sensitivity = 1 uV?".

And "Is there any effect of contralateral noise on the latency and amplitude of the VII peak at sensitivity = 1 uV" could not be completely answered.

The question, "Is there any difference in the latency and amplitude of the V peak when measured using patient response interval of 10 msec, and 20 msec?" has been answered. There is no significant difference in the amplitude of the V peak when measured using patient response interval of 10 msec and 20 msec at 4 KHz 80 dBHL and 2 KHz 80 dBHL. There is no significant difference in the amplitude of the V peak measured when using patient response interval of 10 msec and 20 msec at 2 KHz 80 dBHL. But a significant difference was found in the latency of the V peak, measured when using patient response interval of 10 msec and 20 msec at 2 KHz 80 dBHL.

Finally, the question, "Is there any difference between the combined amplitude of middle response (amplitude of response when right ear was stimulated + amplitude of response when left ear was stimulated) and the binaural amplitude response (amplitude of middle response obtained when both the ears were stimulated)?" has been answered. No significant difference has been found between the combined amplitude of the middle response and the binaural amplitude response.

Thus, the present study shows that, no consistent VI VII and Po peaks between 0 and 20 msec in all the normal subjects tested in the presence and absence of contralateral noise and using sensitivity of 0.2 and 1, is observed. The present study also shows that no significant difference is observed in the amplitude and latency of the V peak measured at 4 KHz 80 dBHL, using patient response interval of 10 msec and 20 msec. No significant difference is observed in the amplitude of the V peak measured at 2 KHz 80 dBHL, but significant difference is observed in the latency of the V peak at 2 KHz 80 dBHL when measured using patient response interval of 10 msec and 20 msec.

Further, the study also shows that there is no significant difference between the combined amplitude of the middle response and the binaural amplitude response.

Discussion:

Results of the present study shows that some subjects have identifiable Po Peaks when measured at 2 KHz and 4 KHz with sensitivity at 0.2 uV it is seen that all the subjects having an identifiable Po peak show a reduction in latency in the presence of contralateral noise for a stimulus of 2 KHz and 4 KHz at 100 dBHL and 80 dBHL.

At 2 KHz 60 dBHL, two subjects having identifiable Po peaks show an increase in latency in the presence of contra-lateral noise and at 4 KHz 60 dBHL? only one of the subjects having an identifiable Po Peaks has an increase in latency in the presence of contralateral noise. The other subjects show-ing clear peaks at 2 KHz and 4 KHz 60 dBHL, show a decrease in latency in the presence of contralateral noise.

Thus, at high intensities, above 60 dBHL, there is a decrease in the latency of peak Po in the presence of contra-lateral noise, whereas at 60 dBHL, the change in latency of the peak Po in the presence of contralateral noise is variable.

When the middle response is measured using sensitivity = 1 uV, it is seen that in all the cases having identifiable peaks Po, there is a decrease in latency in the presence of centralateral noise. In only two instances, there is no change in the latency of peak Po in the presence of contralateral noise. In none of the subjects having identifiable peaks is

these an increase in latency in the presence of contralateral noise.

Gutnick and Goldstein (1978), observed that with high intensity noise a€ 80 dBSL in the contralateral ear, there was a mean signal threshold shift of - 16.6 dB in the middle evoked response threshold, which they attribute to either activation of the acoustic reflex, or to transcranial masking.

Prasher and Cohen (1984) studied the selective effects of contralateral masking. They observed that only wave-V was affected by the pulsed white noise at the contralateral ear and thus, they suggest that the area of mediation of the central masking effect is caudal to the site of generation of wave-V.

Thus, based on the results of Prasher and Cohen (1984), in the present study increase in latency of the peak Po in the presence of contralateral noise, whose site of generation is distal to the site of generation of wave-V cannot be attributable to central masking effect.

The increase in latency cannot he attributed to activation of acoustic reflex or transcranial masking since, the increase in latency is observed at low intensity levels and not at high intensity levels as observed by Gutnick and Goldstein (1980).

Therefore, some other phenomenon must be operating. The present data is not sufficient to describe this phenomenon.

There is a reduction in the latency of peak Po observed at higher intensities at 2 KHz and 4 KHz. The present data, is not adequate enough to explain this.

The results of the present data show that in some subjects, at certain intensity and frequency levels, clear VI peaks are identifiable when measured using sensitivity = 0.2 uV. For subject 1, 5, and 11, there is an increase in latency in the presence of contralateral noise. Whereas for the other subjects, there is either a decrease or no change in latency in the presence of contralateral noise.

When measured for sensitivity 1 uV, those subjects having clear VI peak show either a decrease or no change in the latency in the presence of contralateral noise.

Reid, Birchall and Moffat (1984) carried out three experiments. The results they obtained, showed that for wave-VI the amplitude was significantly reduced with masking when the stimulus was set at 90 dBSL, but there was no significance effect at the lower stimulus levels. Without masking, the amplitude of wave-VI increased noticeably with an increase in stimulus intensity. It was also seen that with no masking, the amplitude of wave-VI at a 90 dBSL stimulus level differed

significantly from the amplitude at a 70 dBS and at 80 dBSL stimulus level.

In the present study, the VI peak does not show any consistent change in amplitude with change in intensity, and only a minimum amount of consistency is these in the change of latency as a function of change in intensity. Thus, the present data is not sufficient to explain the cause of decrease in latency of the VI peak in the presence of contralateral noise.

Results of the present study show that peak VII measured at 2 KHz and 4 KHz at different intensity levels, at sensitivity 1 uV and 0.2 uV is identifiable in only a few subjects and only at some intensity levels, there is no consistency seen in the results identified. Therefore, further research and more consistent data is required to come to some conclusion.

The present study answers the question "Is there any difference in the amplitude and latency of the V peak when measured using patient response interval of 10 msec and 20 msec?",

The results of the present study show that there is no significant change in latency and amplitude of the V peak when measured at 4 KHz 80 dBHL with patient response interval at 10 msec and 20 msec. When measured at 2 KHz 80 dBHL, there

is a significant change in the latency of peak V, but no significant change is observed in the amplitude of peak V when measured using patient response interval of 10 msec and 20 msec.

Moore (1983) has given the effects of stimulus parameters on ABR. He says, there are only three primary stimulus parameters, frequency, intensity and time. No studies have studied the effect of patient response interval. Thus, from the present study, one can assume that increase in patient response interval brings about a decrease in latency at 2 KHz 80 dBHL.

Further, the present study, has also been able to answer the question' Is there any difference between the combined amplitude of middle response (amplitude of response when right ear was stimulated + amplitude of response when left ear was stimulated) and the binaural amplitude response (amplitude of middle response obtained when both the ears were stimulated)?"

In the present study, no significant difference is present between the combined amplitude of middle response and the binaural amplitude response. Results of the study also, how that monaural and binaural responses for log on stimuli of equal sensation level does not yield equal response amplitudes. The amplitude yielded by administering logon stimulus to only one ear is less than the amplitude yielded by administering logon stimulus to both the ears simultaneously.

Dobie and Norton (1980) and Kadobayashi et al (1984) have also observed that the early components of the middle latency response for binaural stimulation had larger amplitude than those for monaural stimulation.

Dobie and Norton (1980) have reasoned that since this difference is present between the monaural and binaural response, the neural mechanism underlying the generation of the middle component auditory evoked potential is different from the neural mechanism underlying the generation of the auditory brain stem response. Denker and House (1982) have contradicted this statement by saying that the two mechanisms and identical.

Ainslie and Bosten (1980) have observed the amplitude of waves I, II and V for binaural stimulation are significantly larger than those for monaural stimulation. Therefore, according to Kadobayashi et al (1984) the peak to peak amplitudes of the early components of the middle response under binaural stimulation being larger than those obtained under monaural condition, can be attributed to impulses from the right and left ears eliciting the response in the brain stem.

In the present study also, since the amplitude of peak Po under binaural stimulation is larger than the amplitude of peak Po under monaural condition for peak Po, the impulses from the right and left ear are eliciting the responses in the brain stem.

SUMMARY AND CONCLUSIONS

SUMMARY AND CONCLUSIONS

The present study was aimed at investigating whether there is any effect of contralateral noise on the latency and amplitude of the VI, VII and Po peak. Simultaneously, the study was also aimed at seeing if there was any change in the amplitude and latency of the V peak when measured using patient response interval of lOmsc.and 20msec. And finally,the study attempted to find out if there was any difference between the combined amplitude of middle response and the binaural amplitude response.

The Electric Response Audiometer Model TA-1000 was used for the study. The study was divided into three experiments. In experiment-I, the latency and amplitude of the middle latency response was measured for eight normal females and seven normal males, in the presence and absence of contralateral noise. In experiment-2, six normal females and two normal males, who had acted as subjects in experiment-1, were taken as subjects and latency and amplitude of averaged evoked response was measured, while keeping the patient response interval at 10 msec. In experiment-3, seven normal females and three normal males having equal thresholds in both the ears, were taken for the study. Amplitude of the middle response from the left ear, right ear and when both the ears are stimulated binaurally, were recorded.

Conclusions:

1. Clear VI peaks were obtained when tested at sensitivity 0.2 uV in ___
   10/15 subjects at 2 KHz 80 dBHL;
   8/15 subjects at 2 KHz 100 dBHL;
   9/15 subjects at 4 KHz 80 dBHL; and
   12/15 subjects at 4 KHz 100 dBHL.

2. Clear VI peaks in the presence of contralateral noise were obtained when tested at sensitivity 0.2 uV in - -
   11/15 subjects at 2 KHz 80 dBHL;
   7/15 subjects at 2 KHz 100 dBHL;
   7/15 subjects at 4 KHz 80 dBHL; and
   10/15 subjects at 4 KHz 100 dBHL.

3. Clear VI peaks were obtained when tested at sensitivity 1 uV in ___
   6/13 subjects at 2 KHz 80 dBHL;
   6/13 subjects at 2 KHz 100 dBHL;
   5/13 subjects at 4 KHz 80 dBHL; and
   10/13 subjects at 4 KHz 100 dBHL.

4. Clear VI peaks in the presence of contralateral noise were obtained when tested at sensitivity 1 uV in - -
   1/13 subjects at 2 KHz 80 dBHL;
   5/13 subjects at 2 KHz 100 dBHL;
   2/13 subjects at 4 KHz 80 dBHL; and
   8/13 subjects at 4 KHz 100 dBHL.

5. In all the subjects, except 3 subjects, for the VI peak there was either a decrease or no change in latency when measured in the presence of contralateral noise at 2 KHz and 4 KHz, with sensitivity at 0.2 uV.

6. In all the subjects having clear VI peaks, there was a decrease, or in some no change in latency when measured in the presence of contralateral noise at 2 KHz and 4 KHz, keeping sensitivity at 0.2 uV.

7. Clear VII peaks were obtained when tested at sensitivity 0.2 uV in ___
   5/15 subjects at 2 KHz 80 dBHL;
   5/15 subjects at 2 KHz 100 dBHL;
   3/15 subjects at 4 KHz 80 dBHL; and
   7/15 subjects at 4 KHz 100 dBHL.

8. Clear VII peaks in the presence of contralateral noise were obtained when tested at sensitivity at 0.2 uV in - -
   4/15 subjects at 2 KHz 80 dBHL?
   6/15 subjects at 2 KHz 100 dBHL;
   2/15 subjects at 4 KHz 80 dBHL; and
   2/15 subjects at 4 KHz 100 dBHL.

9. Clear VII peaks were obtained when tested at sensitivity at 1 uV in ___
   2/13 subjects at 2 KHz 80 dBHL;
   4/13 subjects at 4 KHz 100 dBHL;
   None of the subjects at 4 KHz 80 dBHL; and
   5/13 subjects at 4 KHz 100 dBHL.

10. Clear VII peaks in the presence of contralateral noise were obtained when tested at sensitivity at luV in - -
    None of the subjects at 2 KHz 80 dBHL;
    4/13 subjects at 2 KHz 100 dBHL;
    2/13 subjects at 4 KHz 80 dBHL; and
    3/13 subjects at 4 KHz 100 dBHL.

11. Clear Po peaks were obtained when tested at sensitivity at 0.2 uV in ___
    at
    1/15 subjects/2 KHz 80 dBHL?
    7/15 subjects at 2 KHz 100 dBHL?
    3/15 subjects at 4 KHz 80 dBHL? and
    4/15 subjects at 4 KHz 100 dBHL.

12. Clear Po peaks in the presence of contralateral noise were obtained when tested at sensitivity at 0.2 uV in -
    4/15 subjects at 2 KHz 80 dBHL?
    7/15 subjects at 2 KHz 100 dBHL?
    3/15 subjects at 4 KHz 80 dBHL? and
    5/15 subjects at 4 KHz 100 dBHL.

13. Clear Po peaks were obtained when tested at sensitivity at 1 uV in ___
    1/13 subjects at 2 KHz 80 dBHL;
    7/13 subjects at 2 KHz 100 dBHL;
    4/13 subjects at 4 KHz 80 dBHL; and
    6/13 subjects at 4 KHz 100 dBHL.

14. Clear Po peaks in the presence of contralateral noise were obtained when tested at sensitivity at 1 uV in - -
    2/13 subjects at 2 KHz 80 dBHL;
    6/13 subjects at 2 KHz 100 dBHL;
    2/13 subjects at 4 KHz 80 dBHL; and
    6/13 subjects at 4 KHz 100 dBHL.

15. In all the subjects having clear Po peaks at sensitivity 0.2 uV, there was a decrease in latency at high intensity levels above 60 dBHL at 2 KHz and 4 KHz in the presence of contralateral noise. Whereas at 80 dBHL for 2 KHz and 4 KHz, the change in latency in peak Po in the presence of contralateral noise was variable.

16. In all the subjects having clear Po peaks at sensitivity = 1 uV, there was a decrease in latency when measured in the presence of noise at 2 KHz and 4 KHz.

17. Increase in sensation level does not bring about consistent increase in amplitude and decrease in latency in the middle evoked response.

18. There was no significant change in latency and amplitude of the V peak when measured at 4 KHz 80 dBHL with patient response intervals of 10 and 20 msec.

19. There was significant difference between the latency values of V peak (2 KHz 80 dBHL) obtained at 10 msec, and 20 msec; patient response intervals.

20. There was no significant difference between the amplitude values of peak V (2 KHz 80 dBHL) obtained at 10 msec and 20 msec patient response intervals.

21. The amplitude of Po peak was greater in binaural stimulation than in monaural stimulation.

22. Difference in amplitude for monaural and binaural response may be explained in terms of Kadobayashi et al (1984) finding that for peak Po, impulses from the right and left ear, elicit the response in the brain stem.

Limitations of the study:

1. A small population is tested.
2. The latency range is United to 20 ms.
3. The age range is limited.

Recommendations:

1. To carry out the study on a larger population, using stimulus rate of 5 stimuli per second.

2. To study the effect of contralateral noise on middle response by using patients response interval of 100 msec.

3. To study the effect of patient response interval on V peak using a larger population.

4. To study Binaural Interaction for peaks Pa and Pb of the middle latency response.

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