A COMPARISON BETWEEN MLD & III PEAK OF BSERA

Register No: 8611

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An Independent project submitted as part fulfilment for
Frist year M.Sc. (Speech and Hearing)
to the University of Mysore

All India Institute of Speech & Hearing
MYSORE-570 006.

MAY-1987

MY BELOVED PARENTS

CERTIFICATE

This is to certify that the Independent Project entitled " A COMPARISON BETWEEN MLD AND III PEAK OF B.S.E.R.A " is the bonafide work on part fulfilment for the Degree of Master of science (Speech and Hearing) of the student with Register No.8611

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CERTIFICATE

This is to certify that the Independent Project entitled "A comparison between MLD and III peak of B.S.E.R.A" has been prepared under my supervision and guidance.

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DECLARATION

I hereby declare that this Independent Project entitled "A COMPARISON BETWEEN MLD and III PEAK OF B.S.E.R.A" is the result of my own study under the guidance of Dr.M.N.Vyasamurthy, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore.

Date; May 1987 Register No.8611

ACKNOWLEDGEMENT

I acknowledge my indebtedness to my teacher and guide Dr.M.N.Vyasamurthy for his invaluable help and guidance rendered to me in every aspect of this project.

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

I extend my sincere thanks to Dr.(Miss) S.Nikam, Prof. and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore for permitting me to carry out this project.

I am thankful to sreedevi, Asha, Chandrashekhar, Ravi Nigam, and Maya for their invaluable assistance to me.

I am also indebted to all those who served as my subjects for this study.

My sincere thanks are also due to Ms Rajalakshmi R Gopal who typed this project report for me in short time.

There are many others also who helped me in one way or the other and I am grateful to all of them.

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INTRODUCTION

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Masking level difference (MLD) is an important psycho-acoustic phenomenon.

MLD may be defined as a psychoacoustic phenomenon in which binaural auditory sensitivity for either tones or speech may be improved in the presence of masking noise by the introduction of an interaural phase difference on either the binaural signal or on the masking noise (Lynn, et al 1981). In simpler terms MLD can be discribed as the difference between binaural masked thresholds obtained under homophasic and antiphasic conditions. In homophasic (NoSo) condition the noise and signal are in phase in the 2 ears. In antiphasic condition (Nos_{π} or N_{π} So) the phase of either the signal or noise is reversed (by 180°) at the 2 ears. In the homophasic condition, noise has its maximum masking effect on threshold sensitivity and is the moat difficult condition to detect the presence of a signal. In the antiphasic condition i.e. either when the signal or noise is reversed (180) at the 2 ears, the noise has less masking effect on threshold sensitivity. Hence a release of masking occurs and threshold sensitivity for the signal is improved. The MLD represents the improvement in threshold sensitivity under antiphasic listening condition relative to the homophasic condition and is usually expressed in dB.

The MLD values in normal hearing subjects range from 3 dB at high frequencies to around 15 dB at low frequencies, especially at 500 Hz (Green and Yost, 1975; Jeffress, 1972). Results of Lynn et al in their study of binaural MLDs in neurological disorders suggest that abnormally small MLDs occur in patients with CHS lesions below the cerebral hemisphere.

It has been known since the work of Lichlida and Hirsch that masked thresholds improve under antiphasic listening conditions and that some form of cross corelational analysis information received binaurally from the 2 ears in the CNS is responsible for the release of the masking phenomenon. The region of superior olivary complex in caudal pons would seam to the particularly suited to carry out this function since this is the 1st anatomical site where integration of information from the 2 ears occurs. Therefore MLD is considered as a SOC phenomenon.

ABR is a measure of the synchronous neural activity of the 8th nerve and auditory brain stem. It is the early auditory evoked potentials i.e. latency between 4—8 m.sec.

Since 1970, the BSER technique has emerged as a vital adjunct to the clinical equipment of the audiologist, otologist, neurologist, neurosurgeon and paediatrician who jointly determine hearing sensitivity, lesion site and CNS integrity.

pathology and maturation. BSER applications in audiologic otologic disorders and site of lesion t sting have shown that the responses are well suited for the detection of hearing abnormalities (shares and Albright, 1980). They became popular because of reproducibility, case of administration, low inter and intra subject variability and accuracy in estimating hearing sensitivity (Clemis and McGee, 1979y Sohmer and Feinmesser, 1970, 1973, 1974).

Still another recent application has been the use of BSER in neurological disease (Starr and sohmer and Celesin, 1978). BSER has been of great assistance in diagnosing various brain stem lesions, the determination of CNS integrity and the assessment of patients with various CNS abnormalities. It is also used in the examination of High risk neurologically impaired children (Jacobson, 1985).

An important advantage of BSERA is that it is not affected by sedatives where as late response is affected by it and also BSERA response is more stable.

Principle used in the brain stem audiometry is that when an individual is quite and relaxed, his brain wave activity shows a definite pattern. In the presence of external auditory stimulus there will be a change in the brain wave activity. This response of change in brain activity is obtained by fixing electrodes - one on the vertex and one each on the right and left ear mastoid.

Based on several studies it has been found that the different peaks in the BSERA waveform originate at different relay stations between the auditory nerve and cerebral cortex. Also it is found from several studies that the III peak of the BSERA originate from the superior olivary complex.

Need for the present study:

From the above explanations it is evident that there are studies suggesting that both MLD and the III peak of BSERA waveform have their origins in the superior olivary complex. Therefore the present study was designed to find out whether any significant relationship existed between the magnitude of MLD and the amplitude of the III peak of BSERA.

Limitations of the present study:

1. The MLD values are obtained using pure tone stimulus of 500 Hz frequency only.
2. The sample size was limited to 20 normal hearing adults.
3. The age range was limited.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Brief review of literature about (1) Masking level difference (MLD) and (2) BSERA.

1) Masking Level Differences A binaural masking level differences may be defined as the improvement in masked threshold sensitivity for a signal that occurs on transition from a homophasic listening condition to an antiphasic one. Homophaaic listening occurs when each of the 2 stimuli, signal and masker, is either interaurally in phase or interaurally out of phase with itself (Noso). Antiphasic listening occurs when either of the stimuli, signal or masker is interaurally out of phase with itself while its companion is in phase (Glsen, Noffsinger and Carhart, 1976).

The high incidence of abnormally small MLDs in populations with normal sensitivity to pure tones and speech but with evidence of subcortical central lesions, such as patients with multiple sclerosis, suggests that the MLD tasks can be of diagnostic value in detecting retrocochlear lesions. But in person with hearing loss or significant interaural differences in threshold sensitivity or both, the MLD tests are not always reliable in differentiating cochlear from retrocochlear (Olsen et al 1976).

The MLD in normal listeners ranges approximately 8 to 12 dB varying some what with the type of signal being presented, response task and whether the signal or the noise is out of phase interaurally. Patients with hearing loss from peripheral lesions of the middle ear, inner ear or auditory nerve have MLDs that are often smaller than normal. This reduction in MLD size has been attributed to such factors as:

1. differences in threshold sensitivity between ears for the signal and the noise.
2. distortion in signal transduction in the inner ear, and
3. alteration in the transmission of information from the peripheral ear to the central auditory system.

Lynn et al (1981) report that cortical lesions have been shown to have no significant effect in MLDs for either pure tones or speech, but patients with multiple sclerosis or other brain stem abnormalities have MLDs that are significantly smaller than normal. These findings would suggest that MLDs are mediated in the CNS some where below the auditory cortex, where the potential exists for some form of auditory processing that correlates binaural information received from the 2 ears. From theae results they conclude that small MLDs occur in patients with CNS lesions below the cerebral hemisphere i.e. only in eases with pontomedullary involvement of the brain stem. Normal MLD scores would not rule out the possibility of central auditory involvement but could be interpreted as an indication

of an involvement of the auditory system at the pontonedullary level, so MLD helps in localization of CNS lesions when used aa a part of a battery of other tests.

schoemy and Carhart (1971) reports that clinical disorders, cochlear involvement is known to affect the MLD size. They demonstrated that the MLDs for 500 Hz for person with unilateral Meniere's disease were smaller than in typical for persons with normal hearing.

It is generally assumed that retrocochlear function, particularly correlation process with in CHS are responsible for the MLD phenomenon (Jeffress, 1972). If so, one would expect that pathologic conditions disrrupting process in the CMS would interfere with normal release from masking.

Olsen, Noffsinger and Carhart (1976) report from their study on MLD in clinical population that the mean results at 500 Hz for patients with cortical lesions and noise induced hearing loss closely approximated the average performance of the normal hearing group for S_{π} No and SoN_{π}. patients with other pathologies had smaller mean MLDs, ranking progressively in decreasing magnitude as follows - Preabycusis, conductive hearing impairment, multiple sclerosis, Meniere's disease and 8th nerve tumors.

Above authors also report that speech MLDs were similar to the ordering observed for 500 Hz MLD. i.e. to Largest mean

MLD was obtained in the group with cortical lesion and the smallest in the groups with 8th nerve tumor.

MLDs for pure tones are greatest for low frequencies i.e. around 15 dB (Hirsh, 1948). For speech they are thought to be equivalent to the average of the MLDs for the frequency range critical to speech understanding (Levitt and Rabinn,1967; Carhart et al 1966).

Several theories and models have been put forward to explain MLD (eg. Durlach, 1972; Jeffress, 1972; Hafter et al 1969). However, Green and Yost (1975) point out that none of the existant models fits well into the neurophysiological mechanisms.

Harrison and Howe, 1974y Gibson, 1978, have reported that the olivary nuclei referent the most caudal brain stem structure receiving auditory afferent information from both ipsilateral and contralateral ears. Further, Monshegion et al (1964) have shown that single unit discharge patterns in the medial superior olive (MSO) are differently affected by ipsi-lateral and contralateral stimulations.

Hannley et al (1993) have compared auditory brain stem responses (A3R), the binaural masking level difference (MLD)

for a 500 Hz pure tone and acoustic reflex in 20 patients with confirmed multiple sclerosis. Their interest was to determine whether abnormalities in the 3 measures were related or whether they varied independently, because MLD, wave III of ABR, and the acoustic reflex have in common demonstrated mediation by lower brain stem structures in the region of superior olivary complex (SOC) (Borg, 1973; Jewett, 1970; Lev and sohraer, 1972; Bucheald and Huang, 1975). The results indicated that the size of the MLD varied with the integrity of wave III of ABR. When the ABR was abnormal there was a lack of wave III in one or both ears and the crossed acoustic reflex was abnormal and there was no release from masking.

Thus Hannley, et al(1903) have concluded that both the wave III of ABR and the MLD have a common neuromachanism in the region of SOC.

Vyasamurthy et al (1985) measured the ABR tracings in 3 conditions (i) homophasic (NoSo); (ii) antiphasic (NoSπ) and (iii) antiphasic (Nπ So) in normal hearing subjects. They observed significant increase in the latency of III and V peak of ABR in antiphasic condition in comparison to homophasic condition. But no significant difference in amplitude of the waveforms and inter peak (V-III) latencies between homophasic and antiphasic condition was reported.

They further suggest that the prolongation of III and V waves latencies during antiphasic condition suggests that some changes in neural mechanisms in SOC may be taking place. Since the interpeak (V-1) latency also shows prolongation effect during antiphasic condition, the prolongation effect is not likely to the changes in the peripheral level. Obviously SOC is involved in the MLD phenomenon.

Hence it has been concluded that MLD is a SOC phenomenon.

BSEA:

The clinical application of the auditory brain stem response (ABR) has provided a unique diagnostic dimension that has transcended inter disciplinary boundaries. In the audiology community, no other test procedure has caused so much interest, generated such attention and been so widely accepted. The reason for the rapid acceptance of ABR is its ability to objectively detect, localize and monitor auditory and neurO—logical deficits in difficult to test populations (Jacobson, 1985.

In this, no overt response is necessary from the patient, only minimal cooperation is required. The adult patient must remain immobile and the baby showEd sleep.

Historical perspectives of bSeRA:

Auditory brain stem evoked responses started with the discovery of bioelectric potentials in animals, 1st described

by Galvani and Circa (1791). Caton (1875) was the first to publish evoked potential recordings obtained from the exposed brain of rabbits and monkeys. Following this Berger (1929), 1st recorded brain electric potentials from the human scalp, which was later called on electroencephalogram (EBG). This was followed by the work of Loomis et al (1938) who 1st reported alterations in human EEG patterns brought about by the introduction of sensory stimulation. Davis et al (1939) initially described the results of a series of auditory evoked cortical potentials obtained from alert and sleeping humans. Their observation showed small, but consistent changes in raw EEG tracings with the introduction of repeatable auditory stimuli.

It was sohmer and Feinmesser (1967) who 1st offered the amount of evoked potentials generated from the brain stem while attempting for an alternative procedure to surgical methods of recording the cochlear potentials (Action potential and Cochlear microphonics). Later Jewett et al (1970,71) definitively identified and described the origin of the far field scalp recorded ABR. Jewett and willistin (1971) showed that acoustically generated 'early potentials' could be detected from a wide area of the skull. They concluded that BSER is a 'far field technique' and the position of the active electrodes are net so crucial.

Animal studies:

In most of the studies cat was used. Jewett (1970), Lev and Sohmer (1972), Buchwald and Huang (1975), Starr and Achor (1978) and Allen and Starr (1978) in different animals found that wave I and V reflected activity from unilateral generators; waves II and II originated in bilateral generators; and wave IV appeared to have its origin in either a midline or bilateral generators. The above studies concluded that the composite activity of as many as six brain stem generators were reflected in ABR.

Human studies:

Lev and sohmer (1972) speculated the similarity between the cat and human ABR generators.

Subsequent to this, sohmer et al (1974), Starr and Achor (1978); Starr and Hamiton(1978), stockard and Rossiter (1977) examined alterations of the ABR in patients. Martin and Coats (1973), Martin and Moore (1977), Picton et al (1974) made togo-graphical analysis of scalp distribution of human ABRs and found that wave I was restricted to the ipsilateral mastoid (with respect to stimulated ear). Picton et al (1974) concluded that waves I and IV represented activity of the auditory nerve and brain stem auditory nuclei.

Goff et al (1977) after investigating ABR in normal young adults in pre and post anesthetic conditions indicated and a subcortical leminiscal origin for the ABR wave components.

Differences between results obtained in man and those obtained in experimental animals:

The data from small animals cannot be used to identify the neural generators of the ABR in man because the auditory nerve in man is much longer (Lang, 1981) than it is in the eat since man has a larger head.

Another difference between man and experimental animals such as cat is the smaller size of the auditory nuclei in man relative to head size i.e. the volume of the cochlear nucleus in man is not much different from that in thecat, but because the human head size is larger, these structures are smaller relative to the head size. This fact together with the much longer distance from the recording electrode to the neural generators in man is responsible for the much smaller amplitude of the potentials recorded from human subjects compared to those recorded from snail animals. (Holler and Janetta, 1985).

Terminology - Jewett and Williston (1971) used the term 'auditory'Evoked Far Fields' to differentiate the response from near field techniques. Hecox and Calambos (1974) used

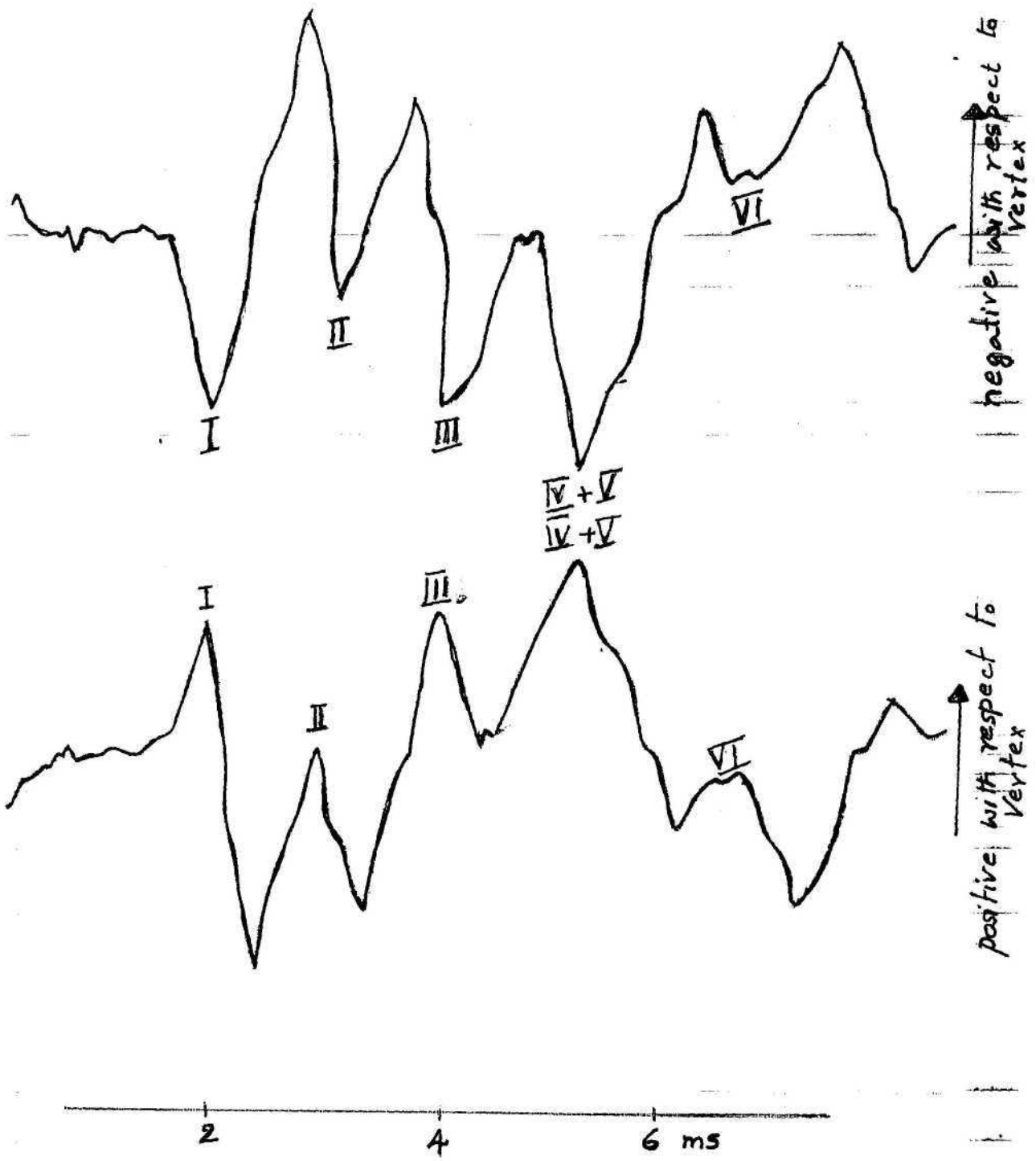
the term 'Brain Stem Auditory Evoked Responses' which seems the most appropriate discription. Gibson (1978) calls it as 'Acoustic brain stem electrical responses'

There is some confusion regarding the labelling of the various peaks of BSERA. The 4th and 5th are often merged. Some workers do not recognize the 4th and 5th waves as being separate. Sohmer et al label the 4th, 5th and 6th waves as N4a, N4b and N5 respectively. Whereas Jewett and others designate them as NIV, NV, and NVI respectively. (Fig.I).

Now it is standard to display the waveform with the negative peaks displayed as a down going peak. But still, there is no definite rule about the ABR waveform display. The only vital requirement is that all the responses should be clearly labelled in Roman or Arabic numerals and the polarity with respect to either the inastoid or electrode must be specified (Gibson, 1973).

Classification:- Auditory evoked potentials comprise a series of neuro electric responses qenerated at all levels of the auditory mechanisms. Using scalp electrodes, as many as 15 AEPs have been identified with in the first 500 ms. post stimulus onset (Picton, Hillyard and Galambos, 1974; Picton, woods and Healey, 1977).

Brain stem response reflects origins of neural activity. Davis (1976) described responses in order of their latency epoch



4KHz stimulus 20/sec 90dB HL.

A typical young adult BER waveform showing the labelling of the peaks according to Jewett's classification. The upper trace shows the response with vertex negative (mastoid positive) waves as an upwards deflection. The lower trace is in the opposite polarity.

Fig. 1

and oay be expressed as follows - 'First '(C:M, SP and acoustic nerve 0-2 ms).
'Fast' (acoustic nerve and auditory brain stem response: 2-lOms)
'Middle' (Thalamus and auditory cortex : 8-50 ms)
'Slow' (primary and secondary areas of the cerebral cortex: 50-300 m.3secs) and
'Late' (primary and association areas of cerebral cortex!300+ms).

BSER generation: The ABR latency epoch consists of 5 to 7 wave peaks measured with la the 1st 10 ms. In the newborn and infant population, the response usually consists of only 3 wave peaks (I, III and V) whose latency and amplitude differ from adult values (Jacobson and Johnson (1992).

Based on studies from several species, it is found that:-

1. Ist wave in the BSER sequence is produced by acoustic nerve activity (Cat-Jewett (1970); Hoshimoto, Ishiyami and Yoshimoto(1981).
2. The cochlear nucleus contribute to the II BSER wave (Buchwald and Huang, 1975).
3. Dendritic post-synaptic potentials of the medial superior olivary complex is responsible for wave III.
4. Wave IV generation is postulated as 'post symptic potential' activity with in the lateral leminiscus cell population (Buchwald, 1983).
5. Inferior colliculus is responsible for V wave generation (Buchwald, 1983).

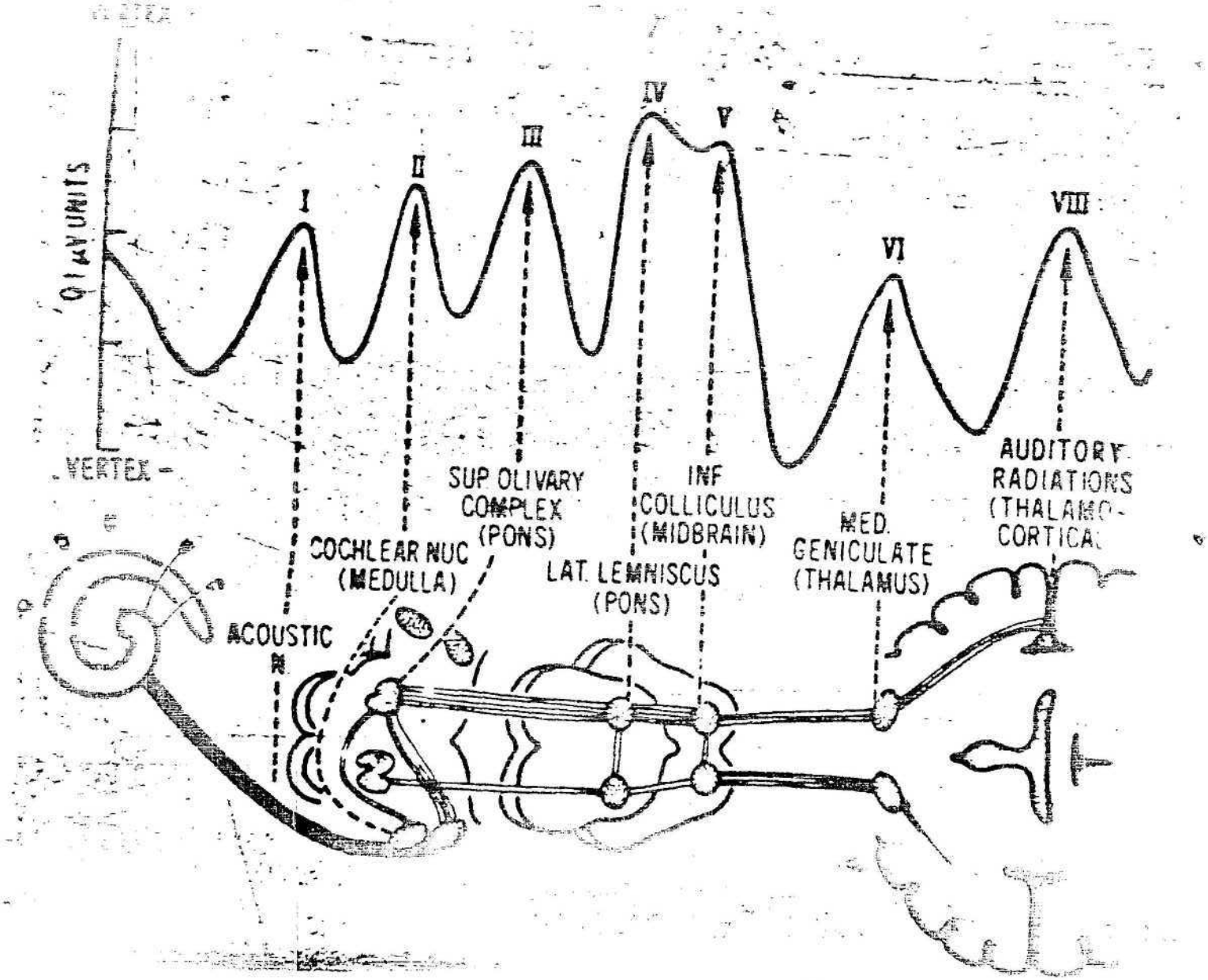
6. Wave VI arises from medial gesticulate body and it is irregularly present (Chiappa, Gladstone and Young, 1979)
7. Wave VII arises from auditory radiations (thalamocortical) and is also irregularly present.

(Fig.2)

The most prominant and consistent components are waves III and V which appear 2 and 4 m.sec. later thanwave I. Waves III-IV-V are some what variable in shape between individuals. Wave III is sometimes double peaked and waves IV and V may overlap to a variable extent (Chiappa, Gladstone and Young (1979). Wave V is the moat consistent and prominent component and it is much less affected by increasing stimu-lus presentation rates (Dan, Allen and Starr, 1977).

According to Buchwald (1903) there is a general agreement among investigations of both human and animal BSER upon the following points.

l.The BSER are a series of volume conducted neural potentials recordable from the scalp which originate from the primary auditory pathway of the brain stem.
2.The BSER show (positive) peaks and (negative) troughs when the scalp electrode registers positively against a second non-caphalic or cephalic reference electude
3.The peaks and troughs occur with latencies less than 10 m.sec. following an intense auditory stimulus.
4.The interval between positive peaks are approximately 1m.sec.



The presumed correspondence between BER component waves (I through VII, upper position of the figure) and anatomical structure in the primary ascending auditory pathway (lower position of the figure).

Fig - 2

5. Peak latencies for any given subject are unchanging over successive trial or recording sessions.
6. BSER latencies and amplitudes are little affected by changes in arousal level or by sleep.

Developmental studies also emphasize the concept of different generator systems for the different BSER waves as their maturation proceeds. During the 3rd week of development in the Kitten, a marked enhancement of wave III amplitude is produced by fast click rates (ghipley et al 1980).

Wave V appear significantly later than the I-IV waves (Jewett and Romano, 1972). The inter peak latencies (I to V) are prolonged early in development, but as maturation takes place, they shorten and become similar to adults. (Shipley, Buchwald and Norman, 1980; Jewett et al 1972).

Studies relating III peak afid SOC:

According to Anchor and Starr (1980) extensive lesions of the SOC had no effect on the BSERs prior to wave III. Also recordings of surface BSERs have shown maximum amplitude in the SOC which coincides with that of wave III (cat, Jewett, 1970; Lev, and Sohmer, 1972). Additional support is by lesion studies. Lesion of inferior colliculus and lateral leminisucus, but spared the SOC, produced no change in wave III (Cat-Lev and Sohmer, 1972, Achor and Starr, 1980).

Following an extensive unilateral SOC lesion, which destroyed most of the M.S.O, wave III was eliminated to ipsilateral stimulation while contralateral stimulation produced a small residual positivity (Achor, and starr,1980).

Physical characteristics of ABR: It is critical to the measurement of ABR that normative data be collected with in the individual laboratory or clinic. The criteria for ABR interpretation are based in general on the following:—

1. Latency (1 ms) of individual wave forms.
2. Latency differences between primary peak components (inter-peak latency).
3. Peak amplitude in microvolts.
4. 1-V amplitude ratio
5. Waveform morphology.

Among there diagnostic decisions are often based on the alterations in latency and amplitude (Schwartz and Berry, 1985).

Factors influencing the normal ABR:

Heterogeaity in stimulus recording, analysis of parameters etc. have led to small but significant differences which can cloud ABR interpretation.

1) Filter characteristics:- Unless the electrical interference of the background activity is overcome, the morphology of ABR will be lost, stockard and Sharbrough (1978) reported that

increasing the low frequency setting resulted in a progressive decreases in wave latency and also waveform amplitude increases with low frequency cut off.

Laukli and Mair (1981) observed that raising the low frequency out off from 2 to 100Hz resulted in a loss of the slow component of the response and a decrease in peak latencies

2. Time Domain averaging: Number of samples: Since the ABR is at most only 1% of the amplitude of the ongoing EEG activity (stockard and shaibrough, 1978) the desired response remains concealed with in this background activity.

There are several methods for eliminating unwanted electrical activity and improving the SN ratio from the desired ABR. Among there are (1) band pass filtering (2) artifact rejection(3) electrode placement and (4) common mode rejection. But the powerful tool is time domein averaging (Thoroston, 1982).

3) Repetition Rate: Jowett and Williston (1971) reported that wave morphology altered as repetition rate was increased from 2.5 to 50/sec. The latency of wave V increases as repetitition rate is changed from 21.1/sec to 81.1/sec. For clinical practices it appears that the latency of wave is not seriously affected until stimulus rate exceeds approximately 30/sec. (Hyde et al 1976).

4) stimulus intensity: As intensity is reduced below 60dBHL, the earlier waves tend to disppear while wave V decreas a in

amplitude and increases in latency studies by D.E.Rose (1984) and Worthington and Peters (1980) shows that visual detection of wave V was possible ia 75% of the cases at intensities between 10 and 20 dB SL and III Peak in 50-60% of the subjects at approximately 30 dB SL.

5. Effects of contralateral masking: The Presentation of broad band noise to the non test ear seems to have only minimal effect on the ABR (Chiappa et al 1979; Humes and Ochs, 1982, Reca and Thornton 1983). Hence the introduction of bread band noise is an effective method for limiting participation of the non test ear.

6. Transducer types:— If the earphones are not matched, then only one earphone should be used to record the ASR in both ears. Schwartz and Berry (1985) reported that the amplitude of wave I for the prezoalectric earphone is more than twice that obtained with the standard audiometric transucer.

7. Electrode location: It is found that changes in electrode position can result in alterations in waveform latency and amplitude (Schwartz and Berry, 1985).

Effect on 'subject' characteristics on the Normal ABR:

1. Age: Infants have 3 vertex positive waves (I, III and V) having different latencies and amplitude compared to the adult norms (Lox, Hack, and Metz, 1981, Jacobson et al 1981).

By 3 months they are similar to adult pattern (salamy, 1976). Beagley and Sheldrake (1978) found no increase in latency between 11 to 79 years who exhibited normal hearing.

2. Gender: Females generally present with earlier response latencies than males of the same age because of their smaller head circumference (Beagley and Sheldrake, 1978; Goldman et al 1991). They also reported that females displayed a waveform amplitude of a magnitude of 0.080 - 0.130 uV greater than those of their male counterparts.
. Robier and Reynand (1984) report that the gender difference was observed only for wave V latency and that I-V is larger in man than in women, where as I—III is identical.
3. Phamacologic agents: stockard et al (1980) reported a general preservation of all waveform parameters in normal subjects undergoing general anesthesia. Gibson (1978) also reports that BSER is not affected by sedatives general anesthetic agents and relaxants.
4. Audiogram: Peak latency and phase differences may be enhanced in persons With sensory hearing deficits(Stockard and stockard, 1983).

Test-Rest-Reliability! It is essential to mention a word about Test-Rest-Reliability. ABR has excellent test-retest-

reliability. Rosenhamer et al (1978) and Thornton (1975) tested 6 subjects each on different occasions and found statistically significant test-retest-reliability. Tha latency of V peak is remarkably constant even from subject to subject and occurs between 4.9 - 5.5 ms. at 80dB HL in normal hearing adults (Gibson, 1970).

Inspite of the good test retst reliability it is seen that the review of literature shows considerable variability in the results with reference to the various parameters affecting ABR.

METHODOLOGY

METHODOLOGY

Subjects: 20 normal hearing (10 males and 10 females) subjects within the age of 18 to 24 years (mean age 21.5 years) were selected for this study. The subjects were selected on the following criteria:

1. They should have hearing sensitivity with in normal limits i.e. within 20 dB HL (ANSI 1969) at 500Hz.
2. They should not have had any history of chronic ear discharge, tinnitus, giddiness, earache or any other otological complaints.
3. They should be able to relax and feel comfortable with electrodes on, within 10-15 minutes after their placement.
4. They should not have had any history of epilepsy or other neurological complaints.

Equipment:

The equipment used was, electric response audiometry, model TA-1000 with Telex 1470 A earphone, mounted in MX41/AR Supra aural cushions and Grason-staddler Audiometer (GSI-10) with TBH-50 p earphones mounted in MX 41—/AR superaural cushions. The equipments wore calibrated periodically using Bruel and Kjaer instruments,

Test Environment:

The study was carried out in an acoustically sound treated

room at the All India Institute of speech and Hearing. The ambient noise levels present in the test room were below the proposed maximum allowable noise levels.

Procedure:

There were 2 stages in the experiment carried out in this study:

1) Determining MLD (2) B.S.E.R.A (Amplitude of III Peak)

1) Determining MLD: Before obtaining the MLD values, the subjects were screened at 20dB HL (ANSI 1969) for 500Hz pure tone in right ear.

Instructions: The subjects were instructed as follows "You will be hearing a pulsed tone in the presence of noise in both ears. Indicate when you hear the pulsed tone.

For obtaining masking level difference (MLD) values each subject was presented binaurally with a narrow band noise of 60 dB SPL centred around 500Hz and 500Hz pulsed tone which had an on/off time of 200m.secs under following conditions,

i) homophasic (NoSo) i.e. when both the noise and signal are in phase at the 2 ears.

ii) antiphasic (NoS_{π}) i.e. when the phase of the signal is reversed (180) at the 2 ears,

iii) antiphasic (N_{π} so) i.e. when the phase of the noise is reversed (180*) at the 2 ears.

2) B.S.E.R.A:

Instructions: The subjects were instructed to lie in a relaxed position on the examination table. They were allowed to sleep, (without sedation).

Electrode placement: Before the electrodes were placed, the skin on the mastoid, forehead and the electrodes were cleaned with cotton soaked in rectified spirit. Electrode gel was placed in the electrodes to fill the recess in the electrodes to the slightly rounded condition and to get applied to the skin.

The red or the signal electrode was placed on the forehead.

The white or the reference electrode was placed on the mastoid of the test ear. i.e. right ear.

The black or the ground electrode was placed on the mastoid of the nontest ear.i.e,. the left ear mastoid.

Johnson adhesive paste was used to hold the electrodes in position.

Each electrode was plugged into the correspondingly coloured receptable on the patient electrode cable from the preamplifier.

The head set was adjusted so that the placement of the head phones were comfortable to the subject.

The instrument was set as follows:-

1) Stimulus frequency on 2KHz, 20 pulses per second and 10 ms sample time.

2. The scale switch on 2048 saopies and 0.2 uV/Division.
3. Stimulus intensity at 80 dB HL

B.S.E.R. waveform was taken for each subject at 2000Hz at the intensity level 80dB HL in right ear.

The latency values of the III peak of the B.S.E.R. wave-form was noted down from the graph.

Amplitude of the III peak of B.S.E.R. was also determined in microvolts (uV). For this the marker amplitude was noted down. The scale switch amplitude 's' was .2 uV/Division.

For example, a trace feature is 2.4 division high and the marker is the division high and the scale switches is set to 0.2 uV/<3ivision. i.e. T=2.4;

M=1

S=0.2

$$\therefore \text{ amplitude} = \frac{TS}{M} = \frac{2.4 \times 0.2}{1} = \frac{0.48}{} \text{ uV}$$

All the 20 subjects were tested in the same manner.

RESULTS AND DISCUSSION

RESULTS AND DISCUSSION

The data of the present study was subjected to relevant statistical analysis and the results are displayed in the Tables 1 to 4.

Table-1 shows the MLD values obtained for 500Hz pure tone in NoS_{π} and N_{π} so conditions (i.e. in the former condition signal is out of phase by 180° in both the ears and in the latter condition noise is out of phase in both the ears by 180°) in males.

Table-2 shows the MLD values obtained for 500Hz pure tones in NoS_{π}- and N_{π} So conditions in females.

Table-3 shows the amplitude and latency of III peak of B.S.E.R.A. when right ear was tested using 2000Hz logon stimulus at 80d8 for 2048 samples in males.

Table-4 shows the amplitude and latency of III peak of B.S.E.R.A. when right ear was tested using 2000Hz logon stimulus at 80dB for 2048 samples in females.

Results of the analysis: Analysis was done using product moment correlation:

	values
1. Correlation of :MLD (noS_{π})coodition and amplitude of III peak in males.	-0.2147
2. Correlation of MLD (NoS_{π}) condition and amplitude of III peak in females.	-0.3081

Table-1: MLD values for 500Hz pure tone - males

Subjects	MLD in (Noso - NoS$_{\pi}$) condition in dB	MM) io (Noso - N$_{\pi}$SO) condition in dB
1	10	7.5
2	10	10
3	10	10
4	12.5	12.5
5	15	15
6	12.5	10
7	12.5	10
8	12.5	10
9	7.5	5
10	12.5	10
Mean	11.5	Mean 10
Standard deviation	2	Standard deviation 2.5

Table-2: MLD values for 500Hz puce tone in females

Subject	MLD ia (NoSo - NoS_{π}) condition in dB	MLD in (NOSO - N_{π} So) condition in dB
1.	10	10
2.	12.5	12.5
3,	15	12.5
4.	15	12.3
5.	12.5	10
6.	15	12.5
7.	10	10
8.	10	7.5
9.	10	7.5
10.	12.5	10
Mean	12.25	Mean 10.5
Standard deviation	2.1	Standard deviation 1.87

Table-3: Amplitude of III peak when right ear was tested using 2000Hz logon stimulus at 80dB for 2048 samples - males.

Subjects	Intensity of stimulus.	amplitude in uV	Latency
1.	80 dB	0.74	3.2
2.	80 dB	0.56	3.4
3.	80 dB	0.52	3.3
4.	80 dB	0.30	3.3
5.	80 dB	0.40	3.2
6.	80 dB	0.20	3.2
7.	80 dB	0.28	3.3
3.	80 dB	0.30	3.5
9.	80 dB	0.26	3.4
10.	80 dB	0.36	3.4
		Mean Value of ampli-tude. 0.39	
		Standard devia-tion 0.16	

Table-4: Amplitude and Latency of III peak of SSER when tight ear was tested using 2000Hz logon stimulus at 80<dB for 2048 samples - females.

Subjects	Intensity of the stimulus	Amplitude (uV)	Latency (m.sec.)
1.	80 dB	0.28	3.2
2.	80 dB	0.52	3.0
3.	80 dB	0.24	3.2
4.	80 dB	0.54	3.2
5.	80 dB	0.54	3.2
6.	80 dB	0.50	3.2
7.	80 dB	0.36	3.1
8.	80 dB	0.60	3.0
9.	80 dB	0.56	3.2
10.	80 dB	0.42	3.4
	Mean value of amplitude.	0.46	
	Standard deviations	0.11	

3. Correlation of MLD (N_{π}so) condition and amplitude of III peak in males.	-0.1839
4. Correlation of MLD (N_{π} So) condition and amplitude of III peak in females.	-0.5769

From the results it is evident that there is no correlation between the MLD values in both the conditions i.e. NoS_{π} and N_{π} SO and the change in amplitude of III peak of BSERA.

Discussion

some of the earlier studies relating the magnitude of MLD and the amplitude of III peak of BSER have arrived at some correlation between the 2 measures. But the present study has shown a negative correlation between there 2 values. However this result can be justified if the intracranial recording of ABR by Moller and Jannetta (1982, 1983) are taken into consideration. They have reported that When a recording electrode on the 8th nerve is moved from a location near the porus acousticus to a location that is close to the brain stem, the amplitude of the potential decreases and the shape of the potential changes. The potentials recorded from the 8th nerve near the porus acousticus have shorter latencies than do those recorded from the nerve at a location near the brain stem. In addition, when responses are recorded near the brain stem a slow negative potential is seen to follow the sharp negative peak and a 2nd negative peak is seen about 1ms after the 1st negative

peak. This 2nd negative peek is most likely generated by second order auditory neurons located in the cochlear nucleus while the slow potential is probably generated by dendrites in the coehiear nucleus.

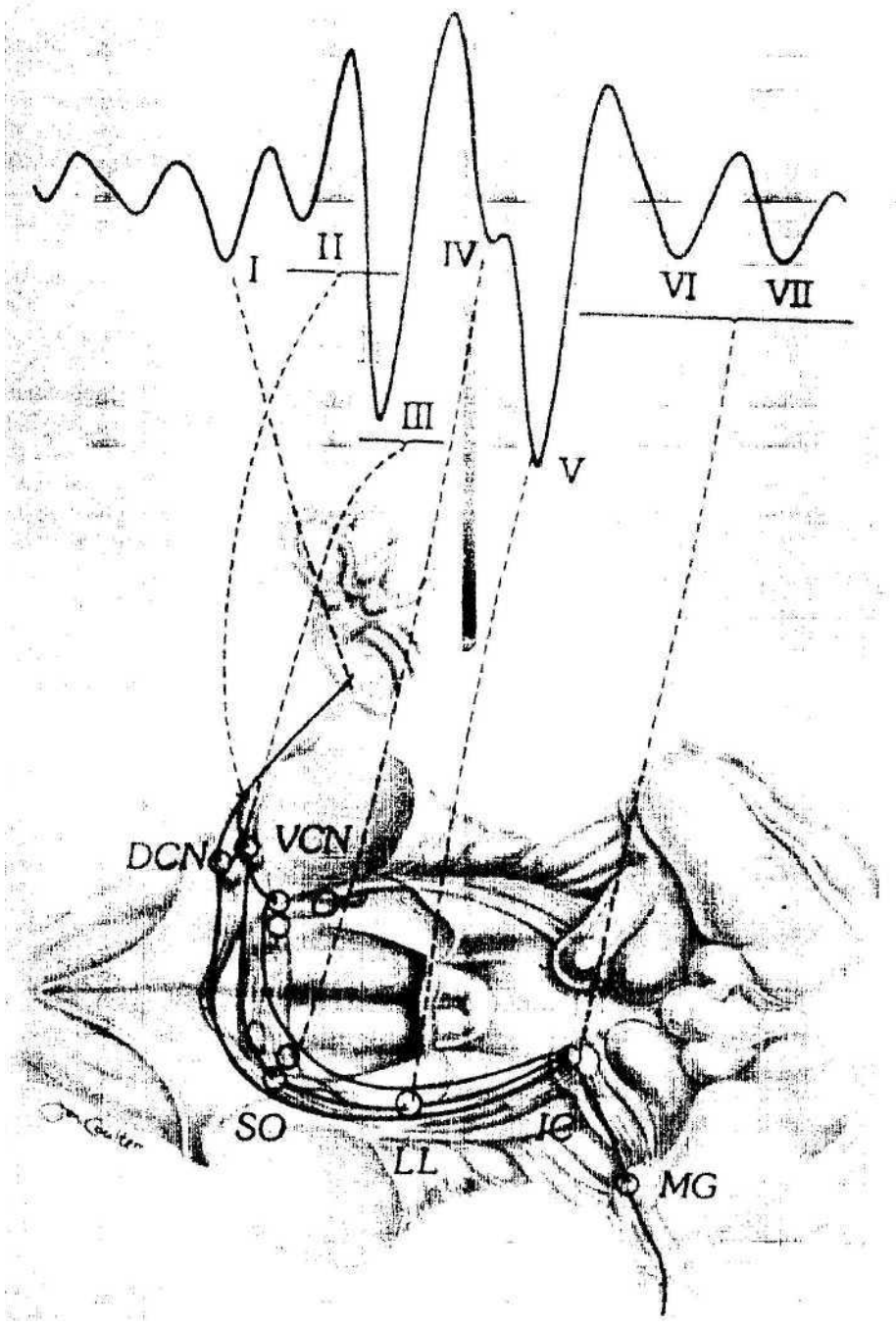
The cochlear nucleus in small animals dominates the brain stem and is located near the entrance of the 8th nerve, but in man is a comparatively small part of the brain stem and is pushed back words by the larger inferior cerebellar peduncle. It is therefore, difficult to gain direct access to the cochlear nucleus of man in a lateral approach. So Moller and Janetta (1982) concluded that 2nd peak is located in the brain stem and not in the nerve trunk along which the electrode is moved. When these intracranial recordings are compared to the ABRs recorded simultaneously from scalp electrodes, this peak is seen to appear with the same latency as does peak III of the ABR.

They also reported that in a patient who was operated upon for a tumor of the 4th ventricle it was possible to obtain direct access to the medial side of the cerebellar peduncle and thus the cochlear nucleus or its vicinity(Molier and Jannetta, 1933). Recordings from this location showed a potential with a large negative peak the latency of which was similar to this 2nd negative peak in the recording from the root entry zone of the 8th nerve. The initial positive deflection seen in the

recording is assumed to have originated in the proximal portion of the auditory nerve, where it enters the cochlear nucleus . It may therefore be assumed that thia second peak is generated by secondary auditory neurons located in the cochlear nucleus. This lends strong support to the hypothesis that peak III is generated mainly in the cochlear nucleus. (Fig-3)

Also the fact that peak III has a much larger amplitude than peak II support the hypothesis that peak III is generated by a relatively large nucleus. such as the cochiear nucleus. Therefore the present study shows that there is no one to one correlation between MLD values and BHER III peak amplitude.

FIGURE-3
Schematic illustration of the neural generators of the ABR in man.



SUMMARY AND CONCLUSIONS

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Few of the investigators have stated a relationship between MLD values sad the amplitude of III peak of BSERA in the past.

The present study was conducted to determine whether any significant correlation existed between the magnitude of MLD and the amplitude of III peak since there is a relationship between SOC and III peak of BSBRA and also between SOC and MLD.

20 normal hearing subjects (10 males and 10 females) with no history of otological problems or any other serious illness ware selected. MLD values for 500Hz pure tone in both NoS_{π} and N_{π} SO conditions were determined for all the subjects using the GSI-10 audiometer. Following this, the amplitude of III peak of BSERA was determined using model TA-1000 for 2000HZ logon stimulus at 80dB HL. The tests were carried out in sound treated rooms of All India Institute of Speech and Hearing.

The data collected was subjected to statistical analysis. Correlation between the 2 measures considered was determined using product movement correlation.

The results showed that no significant relationship existed between the magnitude of :MLD and the amplitude of III peak of BSERA. This may be attributed to the present hypothesis that both MLD and III peak do not have a common origin i.e. in SOC as it was previously assumed. Recently from the work of Moller and Jannetta (1983)it was been assumed that III peak arises from the cochlear nucleus instead of from SOC. This assumption leas a strong support to the result of the present study.

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