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AUDITORY BRAINSTEM RESPONSES IN EARS WITH UNIFORM SENSORI-NEURAL HEARING LOSS EXHIBITING NO ABNORMAL TONE DECAY.

By
(Reg. No.9)
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An Independent Project submitted to the University of Mysore in Partial fulfilment of the requirements for the Degree of Master of Science in Speech & Hearing.

May, 1983

CERTIFICATE

This is to certify that the Independent Project entitled "Auditory Brainstem Responses in ears with uniform Sensori-neural Hearing Loss exhibiting no Abnormal Tone Decay" is the bonafide work in part fulfillment for the Degree of Master of science in Speech & Hearing, of the student with Reg. No.

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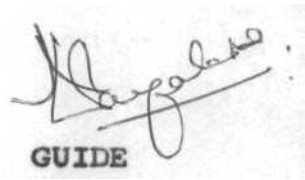
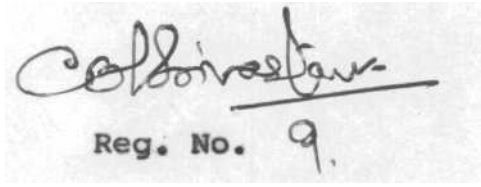
CERTIFICATE

This is to certify that this Independent Project has been prepared under my supervision and guidance.



DECLARATION

The Independent Project entitled " Auditory Brain-stem Responses in Ears with uniform Sensori-neural Hearing Loss exhibiting no Abnormal Tone Decay" is the result of my own study undertaken under the guidance of Mr. Jesudas Dayalan Samuel, Lecturer in Audiology, Ail India Institute of Speech and Hearing, Mysore - 570006 and has not been submitted ear-lier at any University for any other Diploma or Degree.



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CHAPTER - I

INTRODUCTION

The auditory brain-stem responses (ABR) represent probably the most exciting advance in Electric Response Audiometry to date.

The ABR is a sequence of variation in the potential between two electrodes placed on the surface of the skull recorded within 10 msec after the transient acoustic stimulation. (These responses are obtained from surface electrodes by a completely safe and non-traumatic technique which may be performed by any sensible person without the necessity for medical training (Gibson, 1978). The responses are used for objective assessment of hearing acuity and its clinical application has recently become a target of extensive research (Yamada et. al, 1976; Kodera et. al, 1977). This is undoubtedly a satisfactory tool for threshold determination at high frequencies (2KHz and above) (Davis, 1976). Also, this is an effective method of evaluating the auditory pathway from the peripheral endogram through the brain-stem. Hence, ABR is found to be a valuable adjunct to the neuro-otologic evaluation.

Depth and scalp electrode recordings in animals with experimental lesions and clinical studies in potentials with brain stem lesions have shown that the first three component of the response of waves are generated by eigth nerve (Wave I), the cochlear nuclei (Wave II) and from the region of superior olivary complex (Wave III) in the caudal pons. Waves IV and V represent activity

from the region of the nuclei of the lateral lemniscus and the inferior colliculus in the caudal midbrain respectively. Wave VI is thought to originate from the region of medical geniculate body of the thalamus and Wave VII from the region of the auditory radiations.

To differentiate between Cochlear and retrocochlear lesions in sensori-neural hearing loss is one of the most important tasks of ABR.

The importance of utilizing ABR in the evaluation of retro-cochlear lesions have been emphasized by Selters and 8rackman(1977). The underlying assumption is that a tumor has the potential to exert pressure on the auditory nerve, thereby showing a desynchroni-zing neural activity sufficiently to prolong brain stem component latencies and distort waveform markedly. They found that the inter-aural latency difference is normally less than 0.2 m sec but was 0.4 m sec. or greater for 35 acoustic tumor cases and for 7 or 10 Other temporal bone tumor cases.

Previous to this experiment, two experimental studies on cats had suggested the idea that an increase in latency could be used to detect tumors that compress the auditory nerve. Chinn and Millers (1975) attempted to stimulate tumors by inflating rubber catheters against cat auditory nerves. They observed that the cortical electric responses to clicks increased in latency possibly as a direct affect of the pressure on the auditory nerve. Wang and Dallos (1972) observed that auditory nerve latencies were normal in cats with hair cell lesions that were produced by various doses

of Kanamycin sulfate. From these two studies came the expectation that cochlear lesions would result in normal auditory nerve latencies, while tumor pressure lesions Mould increase neural latencies.

ABR in retrocochlear lesion has been studied by various authorities. In cases of acoustic neuromas and meningioma the ABR were abnormal and delay in appearance of $Jewett_5$ - FF_7 complex were found (Terkildsen, 1977). BERA had an excellent success rate 98% in determining presence of carebro-pontine angle lesions (Glossaock, 1979). 98% of patients Mith surgically confirmed acoustic neuromas had positive BERA findings (House and Brackman, 1979). Rosenhall (1981) studied 30 patients of cerebro-pontine angle tumor with BSER. In 8 cases no BSER could be evoked. In 13 cases Mith a Wave V present this wave was significantly delayed in all cases (II_5) and the I - V interval was prolonged in all these cases in which these parameters could be estimated. In 9 cases only the earlier components of BSER (I & III) could be distinguished although Mave II was often missing. In all the cases studied by Rosenhall BSER was pathological indicating a cerebro-pontine angle or lesion.

But some investigators have also found the false positive results of ABR in nontumor cases. The following are the examples -

Clemis and Mitchell (1977) obtained 36% false positive in a material comprising 96 nontumor patients (that however included 11 cases with conductive hearing loss). Clemis and Mc.Gee (1979) obtained false positive rate of approximately 30% of 115 nontumor patients.

Brackmann and Selters (1979) reported false positive rate of 14% in 266 patients with obvious cochlear and hearing loss.

ILD of wave V with asymetric cochlear loss with varying interaural differences concerning pure tone audiogram were studied by Rosenhammer et al 1980. They found false positive rate of 9% of ILDs at 90 dB HL.

Glosscock et al (1979) reported a false +ve rate of 4% in 221 patients with meinare's disease.

Thomson et al (1978) reported a false positive rate of approximately 10% of Meinere's patients.

The explanation of these false positive results are not well understood. Therefore, it is thought that the degree of hearing loss may be acting as variable. To control this factor the present study is attempted to find out whether the latency depends on energy reaching to the cochlea or it depends on the sensation level.

Rosenhamer (1981) studied 110 consecutive cochlear ears with click thresholds not in excess of 60 dB HL. There were 11 ears with rising, 22 ears with flat and 77 ears with sloping audiograms. Click intensities administered were 80 dB HL and 60 dB HL. Wave V latency increments relative to normal values were related to hearing thresholds at 4 KHz. Wave V was identifiable in all the ears at

80 dB ML and/or 60dB SL whereas waves I and III often failed to appear at 80 dB HL and occasionally at 60 dB SL. At 80 dB HL click level, the wave V latency increment was related to the 4KHz hearing loss (statistically significant at level 0.05 in the high frequency loss group).

In this study bilateral sensorineural hearing loss exhibited no decays showing flat type of audiometric configuration will be studied because there is paucity in the literature on this topic. It is the aim of the paper to find out whether the latencies (I - VI) vary from the normal hearing subjects.

Cochlear lesions too alter the ABR and the alternations caused by peripheral lesions are quantitatively of the same kind as those caused by central disorders. Nevertheless the changes are generally smaller (Provided the cochlear loss is not very severe and they may be systematically correlated with the actual hearing loss which is not the case with central lesions (Rosenhamer, 1980).

Wave V has been the most valuable brain stem responses due to its large amplitude and its persistence with reduction of intensity.

The waves I, III and V are usually prominent and therefore the amplitudes of these waves have been generally studied by some investigators.

In the present study, the amplitude of Wave I, III and V will be studied and will be compared with the normal values to observe whether the amplitudes of these waves differ from the normal values or not.

Rosenhamer (1980) felt that the ABR changes in cochlear hearing loss are generally moderate and may be related to the degree of hearing loss.

In this study the attempt will be made to know whether the ABR is related to the degree of hearing loss or not.

NEED FOR THE STUDY

Inter aural latency difference has diagnostic value in cases of acoustic neuroma. To find the inter aural difference stimulus is presented at high intensity HL, irrespective of the degree of hearing loss (except profound hearing loss cases). It is possible that the degree of hearing loss may act as variable. To control this factor, it is necessary to find out whether the latency depends on the energy reaching to the cochlea or it depends on the sensation level.

Also, there is paucity in the literature about effect of degree of hearing loss on the amplitudes of ABR.

Hence, there is a need to find the effect of degree of hearing loss on latency and amplitude of ABR.

NULL HYPOTHESIS

Following are the null hypotheses of the present study.

1. There is no significant difference between the absolute latency obtained from sensori-neural hearing loss subjects and normal hearing subjects.

(a) There is no significant difference between the mean absolute latency value of Wave I obtained from the sensorineural hearing loss subjects and normal hearing subjects.

(bj There is no significant difference between the mean absolute latency value of Mave II obtained from the sensori-neural hearing loss subjects and normal hearing loss subjects.

(c) There is no significant difference between the mean absolute latency of Mave III obtained from the sensori-neural hearing loss subjects and the normal hearing subjects.

(d) There is no significant difference between the mean absolute latency value of Mave IB obtained from the sensori-neural hearing loss subjects and the normal hearing subjects.

(e) There is no significant difference between the mean absolute latency value of wave V obtained from the sensori-neural hearing loss subjects and the normal hearing subjects.

(f) There is no significant difference between the mean absolute value of Wave VI obtained from sensori-neural loss subjects and the normal hearing subjects.

(2) There is no significant difference between sensori-neural hearing loss subjects and normal hearing subjects in amplitude of ABR.

(a) There is no significant difference between the mean absolute amplitude of Wave I obtained from the sensori-neural hearing loss subjects and the normal hearing subjects.

(b) There is no significant difference between the mean absolute amplitude of wave III obtained from the sensori-neural hearing loss subjects and normal hearing subjects.

(c) There is no significant difference between the mean absolute amplitude value of wave V obtained from sensori-neural hearing loss hearing subjects and the normal hearing subjects.

A BRIEF PLAN OF THE STUDY

Some subjects of varying degrees of bilateral sensori-neural hearing loss will be selected for the purpose of the study. The criteria for selecting the subjects will be their age range should b between 18 years to 36 years, their audiometric configuration must be flat. Otoscopically the middle ear should be normal.

Pure tone audiomatry will be dona to check test retest validity. The retrocochlear lesion will be ruled out by administering the test Carhart's tone decay. If any of the subjects will show even mild abnormal tone decay, the subject will be rejected from the study.

Brain-stem electrical response audiometry (BERA) will be done to all the selected subjects. 100 dB HL and 80 dB HL logan stimuli at 2KHz Mill be monaurally administered. Absolute latencies of each wave (I through VI) will be determined. Amplitudes of wave I, III and V will be computed in microvolts.

The responses will be compared with the responses obtained from normal hearing subjects (Uma Devi, 1983). The data will be suitably analyzed and discussed.

CHAPTER - II

REVIEW Of LITERATURE

Auditory brain-stem response (ABR) is a sequence of variation in the potential between two electrodes placed on surface of the skull recorded within 10 ms after the transient acoustic stimulation. The ASR is useful for objective assessment of hearing acuity and its clinical application has recently become a target of extensive research (Yamade et al, 1976; Kodera et al, 1977). This is undoubtedly a satisfactory tool for threshold determination at high frequencies (2 KHz and above) (Davis, 1976). Also, this is an effective method of evaluating the auditory pathway from the peripheral endorgan through the brain stem. Hence, ABR is found to be a valuable adjunct to the neuro-otologic evaluation. Review of literature is discussed under following headings

HISTORICAL ASPECTS

The presence of electrical potential in the brain was first noted by Caton (1875) Mho Managed to record electrical changes in the exposed brain of rabits and monkeys.

After around fifty years of this investigation Hans Berger (1929) a neurologist from Jena recorded the first human electroencephalogram (EEG) froa the elecrtodes placed on the scalp. There was initial reluctance by physiologists to accept Barger's findings as they doubted the reliability of his technique. He has used a simple galvanometer which was not able to measure accurately

voltages as minute as those obtained from the surface of the scalp and an optical recording system which was itself prone to error.

Berger (1930) described a change in the rythm of the electroencephalogram when he either dropped a steel ball into a dish or exploded a fire cracker to produce a sudden loud noise. He was fascinated more, however by the first electrical rhythm that he had described which is now known as alpha-rythm. Ha noted that at rest the electroencephalogram was characterised by large slightly irregular waves occuring at the rate 8-12 per second. If the subject opened his eyas or began some mental activity, than the rythm was inhibited and smaller, physiologists faster waves replaced it. It worried physiologists at the time that the largest brain activity should be recorded whilest the brain was resting. It was felt therefore by many that Berger's recording were artifacts. It was only after the work of Adrian and Mathews (1934) that their scepticism was refuted. Adrian and Mathews (1934) used a valve amplifier and an accurate pen recording apparatus which left no doubt as to authenticity of Berger's work.

Because of technical limitations, it was not until 1960s that evoked response audiometry became a practical clinical study. In 1967 an important contribution was made to the physiological

measurement of auditory responses. Using the click stimuli, two Israeli physicians Sohmer and Feinmesser observed an Evoked polyphasic response, recorded from the vertex of a human subject. This evoked response consisted of five positive-direction waves occuring within the initial 12.5 m sec post stimulus.

In 1970, Jewett noted these same wave forms in the first 10 m secs, post stimulus measured in human subjects.

In 1971, 3ewett and Williston described a method of eliciting brain stem evoked responses (BER) by means of far field (remote electrodes) technique. Nevertheless, early papers (Terkildsen et. al 1973, 1974, 1975, Thorton 1975) continued to use the terms "Surface recorded" or "far field electro-cochleography" to refer to the brain stem response. This engineering term "far field electrococh1eography"was used to describe the situation where electrodes on the surface of the scalp recorded on the distant neural generators.

Though an early report of the ABR in three human subjects were presented by Jewett et al (1970) but the classic paper was published by Jewett and Williston (1971). This later paper provided a more detailed description of ABR properties in human subjects and outlined the influence of various stimulus and procedure related factors on response parameters.

Jewett and Williston (1971) demonstrated that the normal human ABR consisted of five to seven vertex positive waves occuring in first nine milliseconds following a click stimuli (Fig. 1). This wave series was impressively consistent across within subjects. Wave V was the most prominent component of the response and the most robust in its resistance to the effects of increased stimulus repetition rate. Wave VI was a fairly consistent part of the response but wave VII occured inconsistently across subjects.

Jewett and Williston (1971) also compared vertex and ear canal electrode recordings and found that waves 1 and II corresponce for the two configurations, thereby confirming the earlier observations of Sohmer and Feinmesser (1967). The early waves of the response (Wave I through IV) were found to be particularly sensitive to increases in stimulus repetition rate i.e. the resolution of these waves was markedly reduced at higher repetition rates. It was also observed that waves I through IV were present in recordings taken from the mastoid process. When the vertex electrode was moved seven centimeters anteriorily or laterally, the response was unaffected and the authors concluded that this findings confirmed their definition of far field potentials, i.e. potentials arise from distant generators.

Finally Jewett and Williston (1971) demonstrated that tone pip stimuli as well as clicks could be used to elicit the ABR. Lower frequency tone pips, however resulted in a

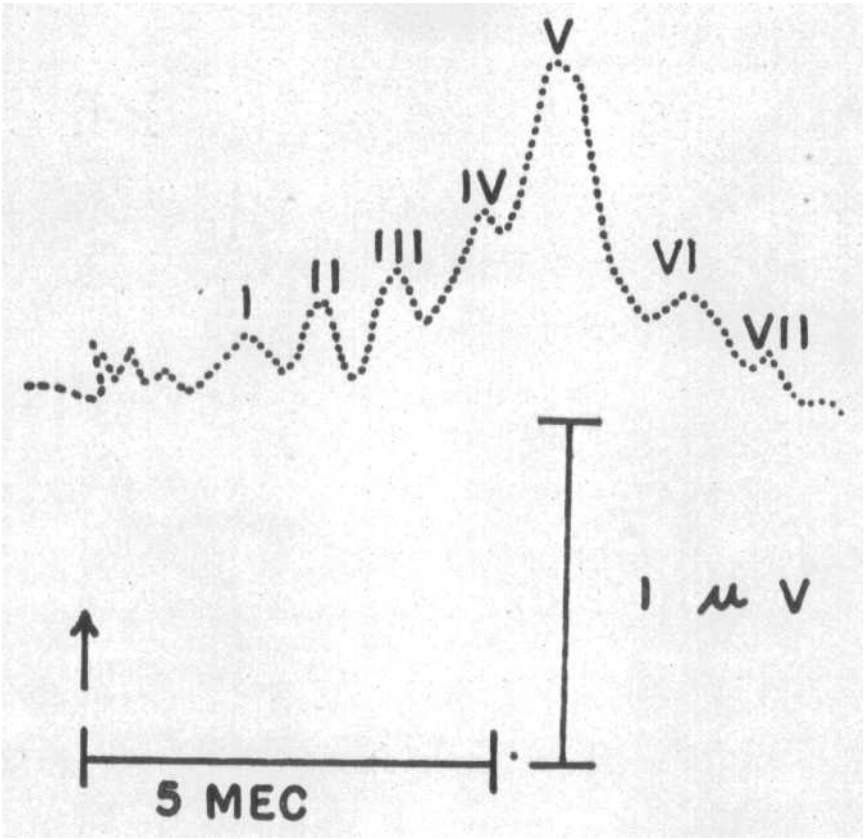

Figure 1 .An example of the auditory brain stem response elicited by high intensity clicks in a normal adult subject. Thia waveform appeared in a report by Jetwett and Williaton (1971); it was the response of subject B in their figure 1.

less distinct waveform than higher frequency tone pips.

The true importance of Jewett and Williston's(1971) report is sometimes overlooked and the paper is remembered for a comparatively insignificant aspect - their suggestion that component waves be labelled with Roman numerals I through VII. This labelling convention remains in common use today.

Most workers obtain these responses by placing the active electrode on the mastoid skin or on the earlobe and positioning the reference electrode on the surface of the wcalp at the vertex or the midline of the forehead immediately beneath the hairline. There is a series of five or more deflections that may be recorded from these sites (Jewett, Romano and Williston, 1970; Sohmer and Feinmesser 1971; Thorton, 1975). Lev and Sohmer (1972) compared the experimental results obtained in cats with human recordings and postulated the relation of each wave to important parts of the auditory tract.

The auditory brainstem responses have been used as a method of threshold audiometry (Sohmer and Feinmesser, 1973; Terkildsen, Osterhammel and Huis in't veld, 1973). The most valuable application of these responses will lie, perhaps, in the neuro-otologic field. Sohmer, feinmesser and Szabo (1974) have found that, in cases of known brain-stem lesions, it is possible to determine the point at which the auditory tract is

damaged by noting the numbers of waves which remain intact. For instance, in the case of a patient with a petrous bone meningiotna, only the first two deflections were visible. Starr and Archor (1975) as well as Selters and Brackman (1979) have applied this technique into the evaluation of the auditory pathway in such a manner as to identify patients with cerebello-pontine angle lesions, multiple sclerosis and a variety of neurological disorders.

Glasscock et al (1979) also found that brain stem evoked response audiometry was useful in determining the presence of cerebellopontine angle lesions and was helpful in establishing whether the hearing loss is of cochlear or retrocochlear type. They concluded that BSER is a valid method of dealing with suspected malingerers and those who cannot respond to standard audiomatric techniques. SERA has been helpful in determining the threshold of hearing.

ANATOMICAL AND NEUROPHYSIOLOGICAL ASPECTS OF ABR

An understanding of the central auditory pathway is needed to appreciate ABR. Most of the studies mentioned have involved animals such as cat and some caution must be exercised in trans-lating this work into the realm of human physiology. Kiang(1968) warns that there is considerable variation in appearance of the various nuclei among different species as the pathway proceeds centrally. The pathway becomes more complex, the higher the evolutionary status of the animal.

Central Auditory pathways:

The central nervous system (CNS) includes both ascending and descending pathways: the term ascending is used to denote those tracts that convey information toward the cortex from the periphery to the lower centers, of CNS. Descending alludes to those that convey information to these lower centers, or the periphery, from the cortex or higher centers. Afferent fibers innervated by movement of the hair cells in the organ of corti form the first order, or primary, neurons of the ascending auditory pathway* These neurons originate in the cochlea. Fig. 2 traces route of an auditory stimulus as it traverses in reaching the cortex as described by Wever (1949), Durrant and Lorrinic (4977) and Sanders (1977).

The auditory pathways use a succession of four neurons enroute to the cerebrum. The first order neurons situated in the modiolous from the spiral ganglion of the cochlea. Nerve fibers pass from the internal auditory meatus into the cranial cavity at the level of the upper medulla end pons. As soon as they enter the CNS, the auditory fibers branch off to the first of a series of neuron centers, the cochlear nucleus, there the fibers divide into two branches, one travelling to the dorsal portion (dorsal cochlear nucleus), the order to the ventral portion (Ventral cochlear nucleus). The cochlear nuclei form a

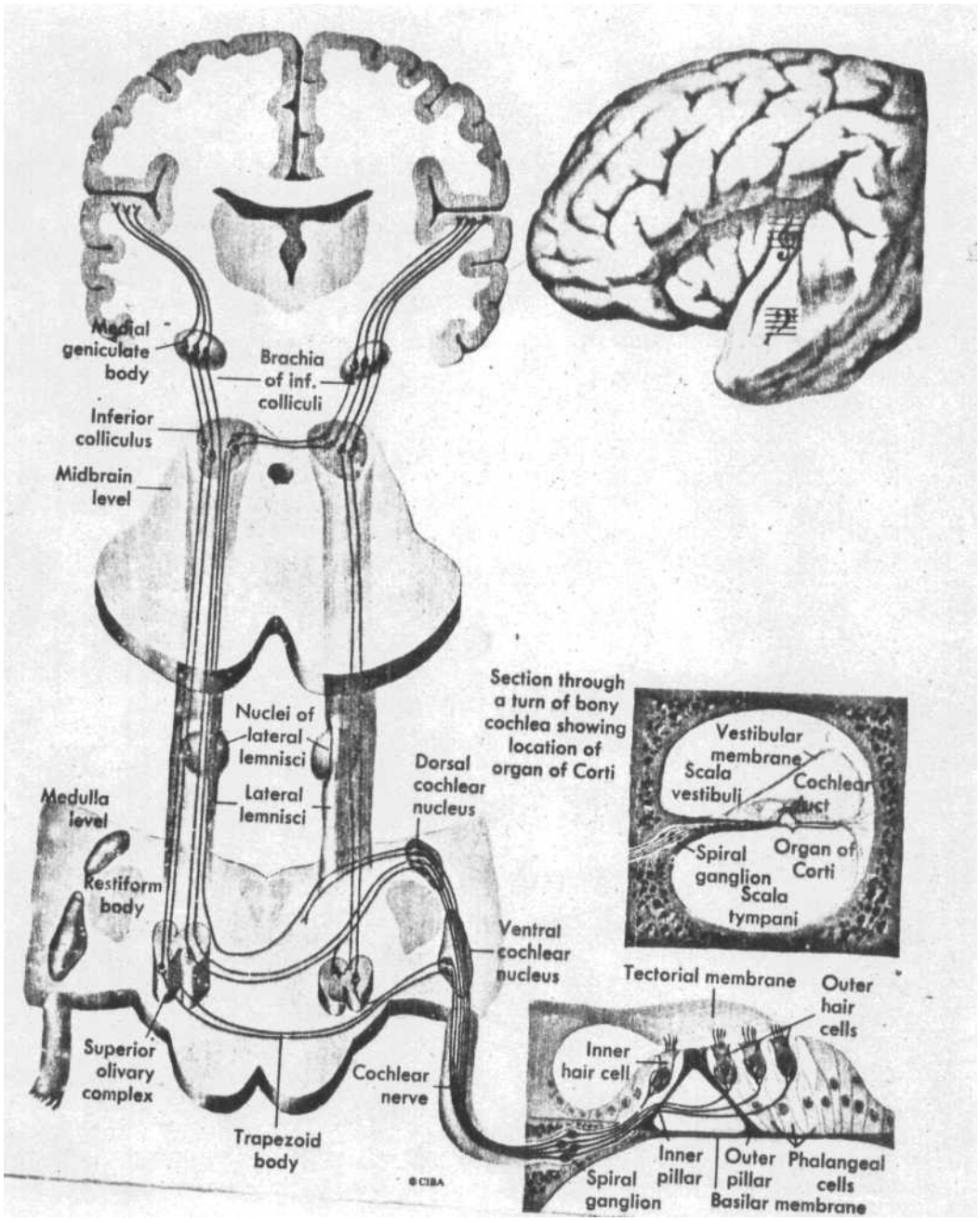

Fig. 2: Central pathways of hearing

kind of substitution linking the fibers from the spiral ganglion to the second order neurons, with fibers from these continuing upward to higher neural centers. Many second order neurons cross over with the trapezoid body from both nuclei and synapse with the contra-lateral cells of the superior olivary nucleus of the pong. Other second order neurons ascend ipsilaterally (on the same side), eventually crossing over and ending in the inferior colliculus, a lower auditory center in the midbrain. All second order nerve tracts reach the lateral lemniscus, from which the third order neurons ascend to nuclei in the upper brain stem. Some fibers go only to the inferior colliculus, but the bulk send a branch to this nucleus and have their main ending in the medial genicu-late body, from where the final radiation arises to the auditory cortex.

While the bulk of fibers cross over at the cochlear nucleus to the side of opposite ear, both ears send a portion of the fibers upward on the ipsilateral side. Thus both fibers from each cochlear nucleus travel both to the cerebral cortex of the same hemisphere and to that of the opposite hemisphere. Hence, since each side of the brain receives information from both ears, removal of one temporal lobe does not cause deafness in either ear. The provision, which amounts to two sets of data for each cortex is thought to be an important factor in some of the more subtle auditory skills.

Auditory evoked responses are generally thought to represent far field reflections of electric events occuring in the neural

pathways from cochlear endorgan as they proceed to the cortex. The responses are grouped according to the period of latency in milliseconds (m sec) from the onset of the auditory stimulus to the recording of the electrical activity. They are referred to as slow(cortical) responses (50 to 600 m sec), middle (12 to 50 m sec) and fast (0 to 10 m sec) responses. It is the fast group which includes the brain stem pathways and is considered to be of great clinical significance.

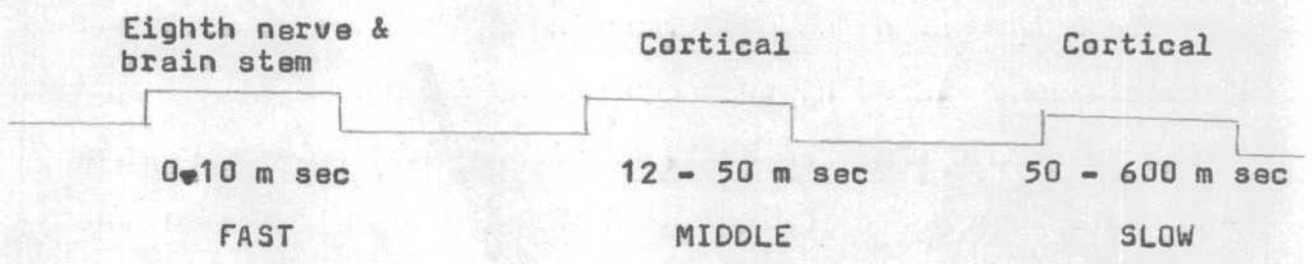

Fig. 3 - Far field responses are grouped by latencies and generator site.

More specific localizations of brain stem disorders involving the auditory pathways may be seen in the recordings of the auditory evoked brain stem potentials (AEP) (Fig. 4). Depth and scalp electrode recordings in animals with experimental lesions and clinical studies in potentials with brain stem lesions have shown that the first three component of the response or "waves" are generated by the 8th nerve (Wave I), the cochlear nuclei (Wave II) located at the lateral portion of the

FAR FIELD RECORDING OF AUDITORY BRAIN STEM RESPONSES

LATENCIES MEASURED)N HUMAN SUBJECTS

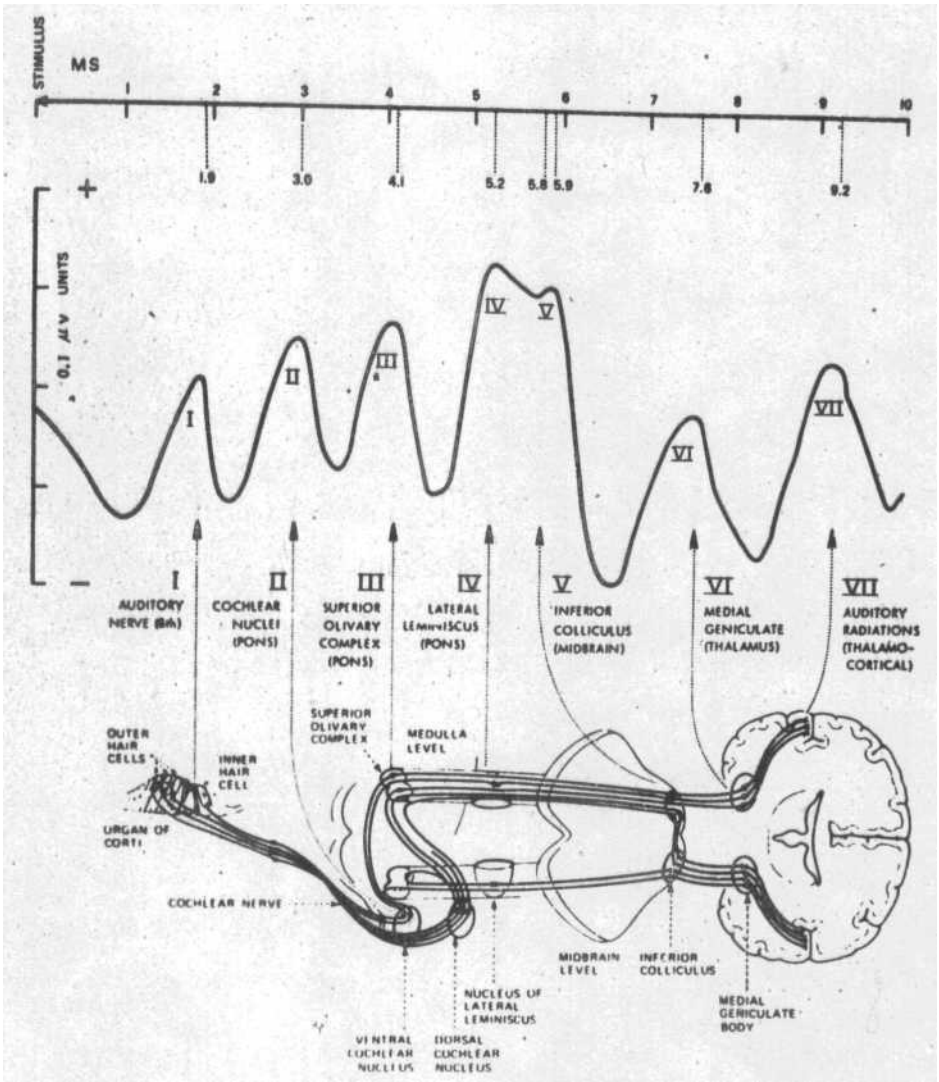

Fig 4 - Anatomic correlation of components of short latency auditory evoked response (Ref. Keith, R.W., Central auditory dysfunction. New York, Grune & Stratton, Inc. p.11, 1977)

junction of pons and medulla and from the region of superior olivary complex (Wave III) in the caudal pons. Waves IV and V represent activity from the region of the nuclei of the lateral lemniscus (Rostral pons) and the inferior colliculus in the caudal midbrain respectively. Wave VI is thought to originate from the region of the medial geniculate body of the thalamus and wave VII from the region of the auditory radiations.

NORMAL RESPONSE PATTERNS

The use of the ABR for clinical purposes obviously involves the recognition of abnormal results. Such recognition depends on a knowledge of normal ABR characteristics. Normal values for ABR parameters are morphology, latency and amplitude. Particular emphasis Mill be placed on the description of parameter variation due to nonpathologic factors.

Response Morphology

In the present context morphology refers to visual appearance or waveform. It is a more subjective parameter than either latency or amplitude, because morphology cannot be specified in measurable units such as milliseconds or microvolts.

The visual appearance of the ABR in different papers may vary. Although most investigators display positive waves at the vertex as upward deflections, some display the same waves as downward deflections. Attention to this seemingly minor point can avoid confusion when comparing published waveforms.

Several investigators have observed that Waves IV and V often are fused together into what has been called in"IV.V" complex. Variations in the waveform of the IV - V complex, based on the relative height and separation of the two waves have received attention in recent literature.

Chiappa et al (1979) described six variant forms in normal young adults. Furthermore, they found that 58% of their 52 normal adult subjects had the same IV-V complex waveforms in both ears.

In normal adult subjects wave V is most frequently observed component of the ABR in response to high intensity clicks, whereas wave II and IV are seen with the least frequency (Rowe,1978). Fria (1980) opines that wave III is a prominent feature of the normal human ABR.

Rowe (1978) observed morphological differences between ears in approximately 20% of the 25 normal adult subjects evaluated. Wave I through V were clearly defined in the right ear responses of these subjects, but waves II and IV were poorly defines.

Response Latency

The time relationship between any response and the stimulus eliciting that response is commonly called latency. For ABR this parameter is designed as absolute wave latency

or interwave latency. Fig. 5 Absolute latency conforms to the traditional definition; the time relationship between stimulus onset and associated response. Interwave latency, refers to the time difference between two component waves, eg. the I - V interwave latency both absolute and interwave latency values are typically specified in milliseconds(m sec).

Beagly and Sheldrake (1978) observed an interesting coincidence. The absolute latency of ABR component waves in response to high intensity clicks is approximated by Roman numeral designing the wave; eg. Wave I latency falls between 1.0 and 2.0 m sec., wave II between 2.0 and 3.0 m sec. and so on. Table 1 shown the mean absolute latency values for normal young adults reported by various studies. Separate values are shown for waves I through VI in response to high intensity clicks. The No. of subjects, click intensities and filter settings used in each study are also indicated. The standard deviation of normal latency values reported by Lev and Sohmer (1972) and Amadeo and Shagass (1973) was greater for values beyond III; but in these early papers the inherently inconsistent IV - V complex was labelled as one wave, and this might account later reports (Starr and Achod, 1975; Rosenhamer et al 1978; Rowe 1978; Stockard et al 1978; Chiappa et al (1979) observed approximately the same standare deviation for all A8R component waves; this value was typically 0.3 m sec or less.

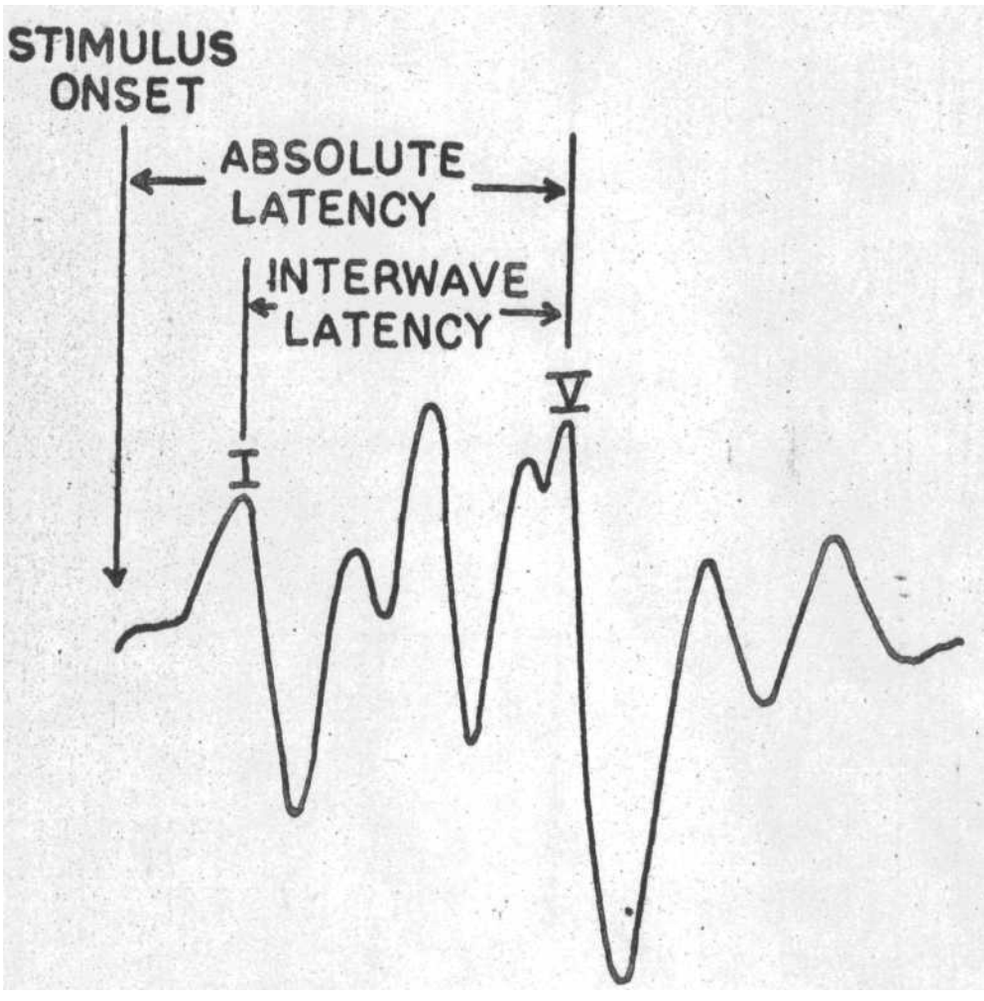

Figure 3 : The distinction between abaolute end interwave latency for component waves of the ABR. By definition, absolute latency is the time(in milliseconds) from stimulus onset to the occurrence of a given wave peak; in this figure, the absolute latency of wave V is represented. Interwave latency is the time difference (in mllliaeconda)between the absolute latencies of two ABR waves; In this figure the 1 to V (1-V) interwava latency ia depicted.

The variation between studies for a given ABR wave latency might reflect differences in the number of subjects evaluated and, or the click intensity and filter settings employed.

Despite differences between studies the data in Table 1 demonstrate a notable trend. The waves occur at approximately 1.0 m sec intervals from roughly 1.7 to 5.7 m sec. in response to high intensity clicks.

Selters and Brackman (1977) reported that the wave V latency difference between ears of the same normal adult subject was less than 0.2 m sec. Rowe (1978) reported that normal inner-ear latency differences were within 0.4 m sec for waves I through V in 95% of the 25 subjects evaluated.

Normal interwave latency values have been reported that several combinations of ABR component waves(of Stockard & Rossiter, 1977). There is an increasing tendency, however to focus on the I-III, III-V and I - V interwave latencies. The I - III value estimates transmission time through the ponto-medullary function and lower pons and the III - V values estimates transmission time from caudal pons to caudal midbrain levels. The I - V latency estimates the time needed for impulses to travel the entire system and is sometimes called 'Central' or "brainstem" transmission time. As will become

Investigation			Click Intensity.	Filter	Absolute latency					
					I	II	III	IV	V	VI
Jewett & Williston	(1971)	11	60-75dB	10-10,000	1.7	-	-		4.6-5.1	-
Lev & Sohmer	(1972)	10	65 dB	250-5,000	1.5	2.5	3.5	5.0	6.7	-
Amadeo & Shagass	(1973)	4	60 dB	10-80,000	1.6	2.8	3.7		5.6	-
Picton et al.	(1974)	20	60 dB	10- 3,000	1.5	2.6	3.5	4.3	5.8	7.4
Starr & Achor	(1975)	6	65 dB	100-3,000	1.6	2.8	3.8	4.8	5.5	7.1
Rosenhammer et al	(1978)	20	60 dB	180-4,500	1.7	2.9	3.9	5.2	5.9	7.6 ?
Rowe	(1978)	25	60 dB	100-3,000	1.9	2.9	3.8	5.1	5.8	7.4 ^
Stockard et al	(1978)	50	60 dB	100-3,000	1.8	2.9	3.9	5.2	5.8	- ^
Chiappa et al	(1979)	50	60 dB	100-3,000	1.7	2.8	3.9	5.1	5.7	7.3

TABLE A - A comparison of mean absolute latency for each ABR wave across Laboratories studying normal subjects. The No.of subjects, click intensity & Bandpass filter settings used in each study are also shown.

Investigation	N-	I - III	Interwave Latency	
			III - V	I - V
Chiappa et al	1979 50	2.1(.15)	1.9(.16)	4.0 (.23)
Gilroy & Lynn	1978 15	2.05(.15)	-	3.83(.13)
Rowe	1978 25	1.97(.16)	1.97(.20)	3.94(.22)
Stockard & Rossiter	1977 125	2.1 (.2)	1.9 (.2)	4.0 (.2)

TABLE B - The mean and standard deviation in parenthesis of interwave latency values from several investigators.

ABSOLUTE LATENCY IN ms

TABLE - C

	2 KHZ Logon Stimuli						4 KHZ Logon Stimuli				
	I	II	III	IV	V	VI	I	II	III	IV	V
100 dB HL	.89	1.8	2.9	4.2	4+7	6+2	.95	2.0	3.0	4.3	4.8
80 dB HL	1.2	2.2	3.2	4.4	5.0	6.5	1.1	2.2	3.2	4.4	5.0

ABSOLUTE AMPLITUDE IN uv

	2 KHz Logon Stimuli			4 KHz Logon Stimuli		
	I	III	V	I	III	V
100 dB HL	.36	.27	.58			
80 dB HL	.26	.29	.51			

Table: Showing the Normative data of Uma Devi (1983).

Logon stimuli of 100 dB HL and 80 dB HL at 2 KHz and 4 KHz were used to elicit the auditory brain-stem responses.(N-10).

evident later, these estimates can prove valuable for clinical purposes. Several studies have reported normal values for these interwave latencies and Table 2 presents a comparison of published findings for young adult subjects. As shown the I - IV intarwave latency approximates 4.0 m sec time and slightly more than half of this time can be attributed to the I - III interwave latency.

Uma Devi (1983) tested 20 ears of 10 normal hearing subjects. Logan stimuli of 100 dB HL and SO dB HL at 2 KHz and 4 KHz were used to elicit the ABR responses. The mean absolute latency for each wave are shown in Table C. The mean absolute amplitude is also given in the Table C

Response Amplitude

In the context of ABR parameters, response amplitude refers to the height of a given wave component and it is usually measured in microvolts (mV) from the peak of the wave to the following through (assuming that vertex positive waves are displayed as upward deflections). This measurement is sometimes called absolute amplitude. The absolute amplitude of ABR component waves can also be expressed in relation to one another, and these measurements are commonly called relative amplitude (Fig. 6)

The variation of normal values for ABR wave component amplitudes have been observed to be substantial by number of

investigators (Amadeo and Shagass 1975; Chiappa et al 1979; Starr and Archor 1975). Stockard et al (1978) reported the mean amplitude in response to high intensity clicks to be 0.15 and 0.38 V for Waves I and V respectively.

In recognition of the inherent variability of absolute amplitude measurements, Starr and Achor (1975) suggested measuring the relative amplitude of waves V and I. In 50 normal subjects they found that the ratio of V:1 amplitude always exceeded 1.0 in response to click intensities below 65 dB. Similar ratios for 60 dB click-evoked ABRs were reported by Chiappa et al (1979). Stockard et al (1978) found the mean V:1 ratio of 2.53 in 100 normal ears.

Fig. 6- The distinction of absolute and relative wave amplitudes for the ABR. Most often absolute wave amplitude is the height of the wave (in microvolts) from its peak to the following trough, as shown in the figure 6 for waves I and V (A & B respectively) but relative amplitude is the ratio of the absolute amplitudes for two ABR waves. For eg. in this figure the relative amplitude of wave V to wave I would be B divided by A. Absolute amplitude measures show wide variation between and within the subjects (Amadeo & Shagass, 1973), Starr & Achor (1975) but relative measures are most consistent and are better indices for comparing amplitude phenomenon between subjects and within the same subject on different occasions (Starr & Archor 1975; Stockard et al (1978).

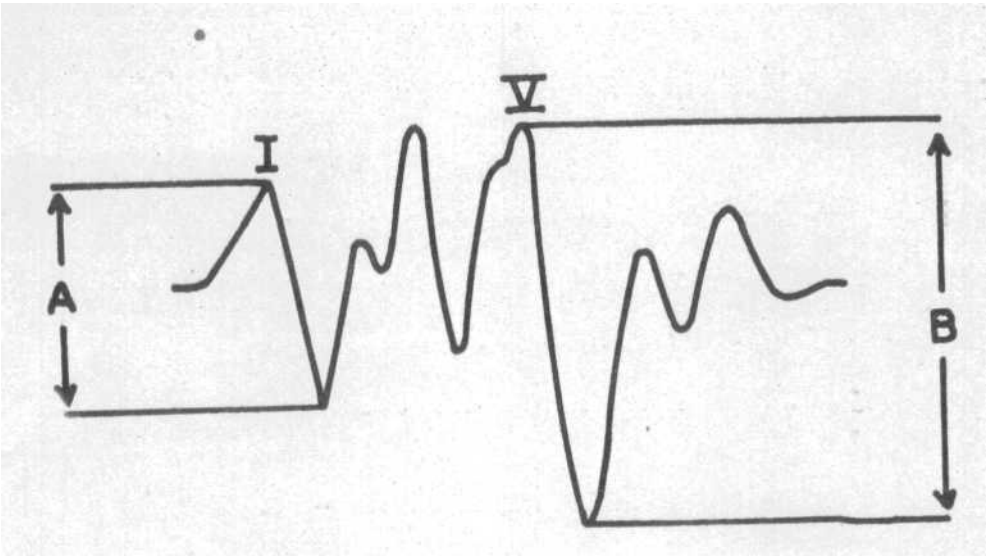

Figure 6: The distinction of absolute and relative wave amplitudes for the ABR. Most often, abaolute wave amplitude is the height (in microvolts) of the wave from its peak to the following trough, as shown above for waves I and V (A end B, respectively); but relative amplitude ia the ratio of the abaolute amplitudes far two ABR waves.

Some investigators measure absolute amplitude from the peak of the wave to the baseline and others measure from the peak to the proceeding through.

FACTORS AFFECTING THE ABR

Based on the clinical experience as well as inspection of SSER waveform appearing in the literature Elberg (1979) opines that the frequency composition of BSER recordings is influenced by a variety of different conditions- age of the patient, normal vs. abnormal hearing ability, site of lesion and degree of pathology, site of electrode placement, type of acoustic stimulus (click, tone burst etc), stimulus intensity, stimulus repetition rate, monaural vs. binaural stimulation etc. Some of these factors are described as follows:

1. Repetition Rate:

For repetition rates upto around 10/sec. all available results indicate no significant influence from repetition rate, provided that the silent interval is much longer than the actual stimulus duration (duty cycle 5%).

All higher repetition rates than 10/S or shorter intervals than 100 ms a gradual increase in latencies and reduction in amplitude has been noted.

Response amplitude for different waves have been reported to react differently to increasing repetition rate with wave V

showing the least reduction (Terkildsen et al 1975; Pratt & Sohmer 1 1975).

In clinical practice, repetition rates in the range 10-20/s are the most commonly used; 20/s giving rise to small changes in responses but saving examination time.

2. Stimulus level:

Three different Mays of expressing stimulus level can be found to dominate. Two of them audiological and the third acoustics

Expressing the level in dS HL means a scale where the reference is the average hearing threshold for the particular stimulus as measured on a group of young, normal hearing listeners. In the other author audiological scale, dB SL (sensation level) the reference level is the hearing threshold for the particular sound as measured on the particular subject being stimulated. In the third scale, the acoustic one i.e. commonly used stimulus level is expressed in dB pc SPL (peak equivalent sound pressure level). This means the sound level reference 20/ per Pa of a continuous pure tone with a peak sound pressure equal to the peak sound pressure of the stimulus.

THE INFLUENCE OF RETROCOCHLEAR LESION OF ABR

The importance of utilizing auditory brainstem evoked responses as an adjunct to the neuro-otologic evaluation of

patients mith suspected retrocochlear lesions has been emphasized by Selters and Brackmann (1979). The underlying assumption is that a tumor has the potential to exert pressure on the auditory nerve, thereby showing or desynchronizing neural activity sufficiently to prolong brain stem component latencies and distort waveform markedly. Selters and Brackmann found that ILD is normally less than 0.2 m sec, but was 0.4 ms or greater for 35 acoustic tumor cases and for 7 or 10 other temporal bone tumors.

When certain circumstances exist, the interpretation of these ABRs is not always straight forward. Cochlear hearing loss of varying degrees and slopes can yield prolonged wavelet latencies not unlike that observed in some patients with retrocochlear involvement (Coats ' Martin, 1977; Rose & Harner, 1978; Selters & 8rackmann, 1979, Komada et al 1979). Profound hearing loss increase the possibility of obtaining distorted responses. Further confusion may arise when one is confronted mith unilateral high frequency sensori-neural hearing loss. In many of these cases, because of reduced peripheral integrity, the interaural wave V latency difference may be large and may mislead the interpretation.

Using 4 KHz tone burst with rise decay time of 0.3 m sec and a plateau of 1 m sec Terkildsen et al (1977) found abnormal brain stem response in tmo patients of acoustic neuromas, even though conventional tests revealed cochlear type of hearing loss. In the third case of meningioma the authors obtained a similar

type of response and in this case the conventional tests clearly pointed to the presence of retrocochlear disease. Characteristic finding were a broadening of the whole nerve action potential and a delay in the appearance of $Jewett_5$-FFP_7 complex

Thomson (1978) applied ABR examination to 27 patients with surgically verified acoustic neuromas. The main indicator of retrocochlear disease was interaural latency difference (ILD) of Jewett-5 wave, the IT_5. Though in the literature it is described that J_5 latency tends to increase with age, in this investigation the authors did not find statistical significance. The authors tried to find out the correlation between tumor size and IT- but they were unable to find any correlation. The authors recommend that this technique can be used without any correlation factors when the stimulus is a 2 KHz filtered tone pip.

Glasscock et al (1979) performed BERA (Brain Stem Evoked Response Audiometry) to over 500 patients (639) ears) and concluded that BERA has an excellent success rate 98% in determining the presence of cerebellopontine angle lesions. They have opined that this method is helpful in establishing whether a hearing loss is of cochlear or retrocochlear type.

House and Brackmann (1979) have reported that 98% of patients with surgically confirmed acoustic neuromas had positive BERA findings.

Jerger et al (1980) reported 4 cases of intracranial tumors. All the cases had normal pure tone audiogram in both ears, but other tests like speech, impedance and ABR varied widely. The authors concluded that consideration of the overall pattern of results on all three measures in combination with audiometric sensitivity level can lead to relatively precise site localization of brain-stem auditory disorders.

Using acoustic clicks stimuli Rosenhall (1981) studied 30 patients of cerebro-pontine angle with BSER. In 8 cases no BSER could be evoked. In 13 cases With a suave V present, this wave was significantly delayed in all cases. The inter aural time differende (IT_5) and the I-V interval was prolonged in all these cases in which these parameters could be estimated. In 9 cases only the earlier components of BSER (I and III) could be distinguished although suave II was often missing. In all the cases studied by Rosenhall BSER Mas pathological indicating a cerebro-pontine angle or lesion.

INELUENCE OF COCHLEAR LESIONS UPON ABR

It is known that ABR is easily distorted by retrocochlear lesions affecting the auditory nerve and/or central auditory pathMays. However, this does not mean that cochlear lesions do not alter ABR and the alterations caused by peripheral lesions are quantitatively of the same kind as those caused by central disorders. Nevertheless the changes are generally

smaller (provided the cochlear loss is not very severe) and they may be systematically correlated with actual hearing loss which is not the case with central lesions (Rosenhamer, 1981).

The fact that ABR changes in cochlear hearing loss are generally moderate and may be related to the degree of hearing loss (within certain limits) makes it possible to correct A8R parameters for peripheral lesions (Rosenhamer, 1981).

Rosenhamer et al (1980) compared ABRs to (80 d8 HL) unfilterad and filtered clicks with centre frequencies in 23 recruiting ears steeply sloping audiograms with sharp cut off at 1, 1.5 and 2 KHz, found that the ABRs to filtered clicks were less distinct (and showed longer latencies) than those to unfiltered clicks.

Clemeis and Mitshell (1977) and Clemis & Mc Gee (1979) preferred 1, 2 and 4 KHz tone pips in differentiating between cochlear and retrocochlear lesions, but obtained a false positive rate of approximately 30%.

To obtain frequency specific audiograms, frequency specific stimuli were used by Trenque and Gezeand (1978) (Frequency ramp burts and Zollner & Patterson (1980)(damped wave trains).

However, most investigators also imply BRA as a means of neuro-otologic diagnosis with the aid of selectively masking noise stimulate with unfiltered squares waves or full cycle or half cycle sinusoids.

Picton et al used short tone pips in notched noise.

It may well be postulated that such very time precise stimuli will produce better synchronization of nerve impulse volleys in the auditory nerve and central auditory pathways and thus more distinct ABR.

Coats (1978) using BRA for differentiating cochlear and retrocochlear lesions, added responses to condensation and rarefaction clicks, seemingly without any harmful effects upon the diagnostic power of this technique.

Rosenhamar et al (1980) studied 14 ears with high frequency loss, they were not able to detect any statistically significant latency differences between ABR to rarefaction clicks and alternating clicks (80 dB HL) nor between ABRs to condensation and alternating clicks.

As in normally hearing subjects, wave latencies decrease whan stimulus intensity increases. However as several authors have shown latency intensity curves (L-1 curves)in cochlear hearing loss ears are generally steeper than in normally hearing ears, especially when recruitment is established by ABLB or other tests (Coats, 1979; Galambos and Hecox 1978; Skinner and Glattke 1977; Kamada et al 1979, whereas the wave V latency at low stimulation levels (above hearing threshold may be considerably longer than in normal ears at equal click hearing levels this latency

will approach normal values at higher stimulation levels (Kavanagh and Beadsley 1979). Shifting the intensity axis graded in dB HL) to the left by a distance that ia equal to the patient's subjective click threshold Mill result in a L-1 curve with the intensity axis graded in dB SL. Such a displacement may show graphically that the wave U latency of the cochlear ear may even be below that of normal ear at sufficiently high SLs.

There is a little information in the literature on ABR as a function of other stimulation characteristics in cochlear hearing loss.

Moller and Blegrad (1976) found that ABR amplitudes were approximately 40% greater on binaural stimulation than in monaural stimulation in patients with symmetrical hearing loss.

Trenque and Gezeaud (1978) found that the wave V latency increase on stimulation with frequency ramp bunts (FR8) vs. pure tone bursts (PTB)was greater in patients with cochlear hearing loss than in normal subjects when comparing 2-4 KHz FRB with 2 KHZ PTB, whereas the opposite was found when comparing 1-2 KHz FRB with 1 KHz PTB.

The relations between ABR parameters and stimulation and recording characteristics found in normally hearing subjects hold in cochlear hearing loss (eg. the findings that wave latencies increase with decrease of tone bursts or tone pip frequency(Kodera et al 1977) and that latencies shorten with increase of the preamplifier cut off frequency.

Some authors (eg. Coats, 1978) make use of complete L-1 curve for differentiating cochlear and retrocochlear lesions. Although the L-1 curve in cochlaar hearing loss is steeper than the normal curve, the fact remains that the wave latencies are mostly longer at all stimulus hearing levels.

Rosenhamer et al (1980) measured wave V latencies and I-V, III-V IPL at 80 dB HL (unfiltered clicks of alternating polarity) in 11 cochlear ears with rising 22 ears with flat and 77 ears with sloping audiograms. They found that in the three groups a clear increase of wave V latency related to the hearing loss at high frequencies (the pure tone threshold at 4 KHz was used as independent variable) but there was no significant increase and 4 KHz hearing contrary to this.

Jerger & Mauldin (1978) found that the V latency increment in patient with cochlear hearing loss was even better correlated with the slope of audiogram between 1 & 4 KHz (the latency increased by 0.2 ms for each 30 dB decrease of the threshold difference at 4 and 1 KHz) than the pure tone average at 1,2 and 4 KHz. In the investigation they applied clicks 70-90 dB HL and the material was comprised of 185 cochlear ears.

Coats & Martin (1977) observed that patients with cochlear high frequency loss showed increasing N_1 and V latencies and decreasing N_1-V intervals with increasing hearing loss at 4 and 2 KHz (these results were obtained both with condensation and rarefaction at approximately 80 dB HL.

In a subsequent article coats (1978) presented L-1 curves with a longer latencies and steeper slope in groups of cochlear patients with different degrees of hearing loss with different at 4 and 8 KHz; both latencies and slope increased with high frequency loss.

Galambos and Hecox (1978) confirmed that L-1 curves in recruiting (cochlear hearing loss) ears take a steeper course than normal but made the reservation that L-1 curves in patients with severe cochlear loss may slow the normal slope.

Moller and Blegrad (1976) established that the latency increase of wave V was less pronounced in patients with gradually sloping high frequency loss, the longest latencies were seen in patients with steeply sloping loss. In each group of the patients the latency increment increased with the pure tone average from 0.5 to 4 KHz (this investigation comprised of 48 patients with symmetrical loss, binaural high level stimulation was used.

Rossi et al (1979) showed in 80 patients using clicks in 90dB SPL that not only did the IV-V latency increase but also did the IV-V amplitude decrease with hearing loss at 3 and 4 KHz. On the other hand they did not find any relations between the characteristics of the IV-V complex and the configuration of the pure tone audiogram.

Thomson et al (1978) who stimulated with 2 KHz tone bursts maintained that the V latency is not significantly prolonged provided the pure tone threshold at 2 KHz does not exceed 60-70d8 HL.

Yamada et al (1979) studied L-I curves in 3 patients with low frequency loss, 3 patients flat loss, 3 patients with steeply sloping high frequency loss and 3 patients with gradually sloping high frequency loss and found that upward dislocation and increased steepness, seen in all four groups to be particularly evident in the patients with gradually sloping audiograms.

Kavanagh and Beardsley (1979) also found that the ABR may may be sensitive to cochlear hearing loss but met with normal V latencies at high click levels in 24 cochlear patients with high fraquency loss in order of 50-60 dB HL.

From studies of normally hearing subjects it is known that increased age and male sex may bring slight prolongations of absolute latencies and possibly of IPLS and consequently the ABR values of a sensorineural patient always be compared with corresponding normative values for the and and sex of the patient. In fact, Jerger and Hall (1980 found the wave V latency to be 0.2ms shorter in female than in mala patients with corresponding degrees of cochlear hearing loss, they also observed a slight influence of age upon the V latency in accordance with that seen in normal subjects.

The principal ABR (if recordable) latency parameters observed in neuro-otologic diagnoses are - (1) the absolute V latency (2) the I¾V interval (provided wave I is observed (3) the inter aural latency difference with regard to wave V+

Selters and Brackmann (1977) reported that if the ILDs are more than 0.2 ms, there is a probability of acoustic tumor.

They themselves have obtained 12% false positive results in 54 nontumor patients with sensori-neural hearing loss and at a later stage (Srackmann & Selters, 1979). They reported a false positive rate of 14% in 266 patients with obvious cochlear hearing loss.

ILD of Wave V with asymmetric cochlear hearing loss with varying inter aural differences concerning pure tone audio-gram were studied by Rosenhamer et al 1980. They found positive rate of 9% of ILDs at 90 dB HL. On the other hand they found that the ILDs were never positive i.e. the latency was never longer on stimulation of the poor ear at the click sensation, level corresponding to 90dB HL in the poor ear.

Clemis and Mitchell (1977) obtained 36% false positive in a material comprising 96 nontumor patients (that however in-cluded 11 cases with conductive hearing loss).

Clemis and Me Gee (1979) got 30% false positives in 115 nontumor patients.

Glosscock et al (1979) reported a false positive rate of 4% in 221 patients with meniere's disease.

Using clicks as auditory stimuli Bauch et al (1981) recorded brain stem evoked responses from six selected configurations, histories and diagnoses. Although none of the patients were diagnosed medically as having retrocochlear disease, ABR latencies and waveform morphology were considered abnormal in some cases. By contrast other patients with similar high frequency configurations yielded normal ABRs. Hence the author describes the potential difficulty encountered in the interpretation of ABRs whan hearing loss exists.

Rosenhamer (1981) performed a study in order to investigate robustness of BSER in cochlear hearing loss by recording BSER in 100 consecutive cochlear ears with click thresholds not in excess of 60 d8 HL. There were 11 ears . . . . . , 22 ears with flat and 77 ears with sloping audiograms. Click intensity presented were 80 dB HL and 60 dB HL and the BSER were observed for replicability and latencies of Waves I, III and V as wall as I-V and III-V interpeak intervals. Wave values were related to hearing thresholds at 4KHz. Wave V was identifiable in all the ears at 80 dB HL and/or 60 dB SL whereas wave I and III

often failed to appear at 80 dB HL and occasionally at 60 dB SL. At 80 dB HL click level, the wave V latency increment was related to the 4 KHz hearing loss (statistically significant at level 0.05 in the high frequency loss group, increasing by approximately 0.1ms for each 10 dB starting at 30 dB HL. Increasing click intensity to 60 dB SL tended to bring waves I and III into appearance and to offset the V latency increase (even inverting it in highly recruiting flat loss ears). Interpeak intervals were not significantly affected by cochlear hearing loss.

HEARING ASSESSMENT BY BRA IN COCHLEAR HEARING LOSS

Jerger and Mauldin (1978) on testing 185 (mainly adults) patients with cochlear hearing loss (275 ears) found that the pure tone average at 1, 2, and4 KHz might be roughly calculated from ABR threshold to unfiltered clicks by multiplying his thresholds by 0.6 a factor i.e. however dependent upon the contour of the audiogram particularly between 1 and 4 KHz.

Kodera et al (1977) obtained frequency specific thresholds in a material of 13 patients with cochlear hearing loss. They used 0.5, 1 and 2 KHz tone pips (rise and fall time 5 ms, no platea of alternating polarity and followed wave V down to the response threshold. However, they noted that the response threshold) might be 20 or 25 dB higher than the corresponding pure tone threshold.

Clemis and Mitchell (1977) also used diamond-shaped pips (rise and fall time 1 ms, no plateau) of alternating polarity; the pip frequencies were 2, 4, and 8 KHz, and the average discrepancy between response and puretone thresholds was approximately 10 dB in 22 patients with sensorineural hearing loss, obviously of cochlear origin. These authors refer the response threshold to the normal ABR threshold which in turn found to be 12 dB (on the average) in excess of the normal subjective pip threshold.

Zollner and Paderson (1980) employed 1, 4 and 8kHz damped wave trains DMT, exponentially decaying sinusoids with a 10% decrement between successive amplitudes) in a material comprising 15 patients with cochlear hearing loss they found a closer agreement between response thresholds (referred to normal subjective DMT thresholds) and corresponding pure tone thresholds in patients with flat loss in patients with marked high frequency loss in whom the response thresholds were even found to be lower. than the pure tone thresholds.

Don et al (1979) used unfiltered clicks and masking high pass noise (with the spectrum level kept constant after determining the power of the unfiltered noise just sufficient to mask the ABR to a 70 dB HL clicks) with a very sharp low frequency cut off (96 dB/octave) in order to obtain desired responses

down to threshold from each of the five basilar membrane segments with centre frequencies of 0.5, 1, 2, 4 and 8 KHz. They found that in normally hearing subjects the ABR (wave V) could be traced down to a click intensity of 10 dB HL (in the two extreme regions, however only down to 30 dB HL) and they used these values for evaluating the hearing threshold in each of the five basilar membrane segments in one patient with a 4 KHz dip, one patient with a low frequency loss and one patient with a flat audiogram. In all three patients, they were able to demonstrate a remarkable agreement between the pure tone audiogram and the BRA.

CHAPTER - III

METHODOLOGY

Eight subjects (10 ears) having flat type of varying degree of sensori-neural hearing loss were tested by ABR. Retrocochlaar pathology was ruled out by Carhart's tone decay test. In ABR, all the ears were tested by using 100 dB HL and 80 dB HL logon stimuli at 2 KHz. Latencies of each wave (1 through VI) were determined. Amplitudes of wave I, III and V were computed in microvolts. The responses Mere compared with the responses derived from normal ears using the same instrument in the same environment (Uma Devi, 1983). The methodology is described under the following headings.

1. SUBJECTS:

The study consisted of 6 males and 2 females ranging in age from 12 years to 36 years. The subjects were selected based on the following criteria.

a) Sensorineural hearing loss in both the ears.
b) Negative otoscopic findings.
c) No history of external or middle ear pathology.
d) Negative tone decay test in both ears (Carhart's tone decay test.
e) Flat type of audiogram in atleast one of the ears.

(If both the ears showed flat type of sensori-neural hearing loss, both the ears Mere tested by ABR. Same May, if only one ear exhibited flat type of sensori-neural loss, only that particular ear was tested).

Responses of ten ears of eight subjects are included in this study.

INSTRUMENTATION

(A)
Audiometer: An advanced diagnostic Audiometer Maico MA 22 with TDH-39 earphones placed in MX-41/AR cushions were used for pure tonetesting. This audiometer allows tasting at 11 frequencies from 125 Hz through 8,000 Hz, also has hearing level ranging from - 10 to 110 dB HL for pure tone.

Calibration: Calibration of the audiometer was maintained using Bruel & Kjaer calibration unit. It consists of artificial ear (type 4152), sound level meter (2203) and octave filter set (1603). Calibration was done in a sound treated room.

Periodic checking was made to keep the unit in calibration throughout the period of study.

(B)
ERA Instrument: The teledyne Avionics Avionics TA-1000 Electric response audiometer is a clinical diagnostic system incorporating the essential precision, versatality and reliability in a simple compact and convenient instrument (fig. 7).

The TA-1000 system consists of (a) SLZ 9793 desk-top console, (b)the SLZ 9794 preamplifier and (c) an access of group.

(a) The SLZ 9793 console contains all the operating controls, indicators and read outs for the system. It provides the patient auditory stimulus, and accepts patient electrical responses from the preamplifier. The signal conditioning and digital averaging extract the patient's BSER or ECochG responses from the background noise oscilloscopic display and ink-on-paper recording provide an on-going moni- . tor as well as a permanent record of responses.

(b) The SLZ 9794 preamplifier is a totally EEG preampli-\0 fier with frequency response and gain specifically designed for electric response audiometry, patient electrical response i3 sensed by a set of three electrodes, and after amplification, is conducted to the console by an inter connecting cable.

(c) The accessory group used includes the following components- A binaural air-conduction headset with cardset. P interconnecting cables, chart, paper and pens. Sets of electrodes, electrolyte, gel and plaster.

Functions of the controls:

TA-1000 is operated with only four knobs and nine push-butt switches. All knobs are clearly marked to indicate their functions.

Push-button switches are of two types: alternate acting, i.e. push-on, push OFF, and momentary acting i.e. push to initiate. All push buttons indicate.

The following is the description of the four knobs:

1. The STIMULUS function switch permits the selection of 2 KHz, 4 KHz, or 6 KHz acoustic logan stimulus equivalent frequencies at repetition rates of 5 or 20 stimuli per second and patient response intervals of 10 ms or 20 ms immediately following the logan stimulus.

2. The STIMULUS attenuator establishes the presentation level, permits selection of acoustic logan stimulus from 0 to 100 dB HL.

3. The SCALE function switch permits selection of system sensitivity and number of averaged response samples. For 1024 samples, 0.5 uv, 1 uv, 2 uv and 5 uv, 1 uv and 2 uv/ division sensitivities are available. For 4096 samples 0.1 uv, 0.5 uv and 1 uv/division sensitivities are available.

4. The LATENCY control position a cursor mark on the oscilloscope display for precise determination of time delay from stimulus peak to any point on the averaged patient response. Readout of latency in milliseconds, to 0.1 ms resolution is displayed in digital form directly above this control.

The nine push button switches are as follows:

1. POWER switch energizes the system and indicate the system status.

2. SCOPE switch controls the oscilloscope display.

3. CLEAR push-button clears the micro-processor averager memory, resets the simple display counter and corrects the micro processor operating mode to correspond to the current control status.

4. START/STOP push-button initiates the micro-processor averager function. As the number of samples accumulates, the averager can be stopped to evaluate intermediate results and restarted without disturbing the averager action. The averager function is automatically terminated when the selected number of samples has accumulated, or when any averager memory channel is full; automatic termination requires a clear, to permit restart.

5. RECORD push button indicates the platter read out if the averager is not active.

6. MASK push-button applies broad band noise masking to the contralateral ear only when either AIR LEFT or RIGHT stimulus is active. Masking level is determined by HL setting of STIMULUS attenuator.

7. AIR LEFT applies the stimulus to the left earphone.

8. AIR RIGHT applies the stimulus to the desired earphone.

9. BONE push button applies the stimulus to bone vibrator transducer.

Paper advancer thumb wheel when rotated downward advances the plotter chart paper.

The limit indicator, in the samples window, all light briefly indicate the presence of excess input to the system. At high sensitivities i.e. 0.1 uv, 0.2 uv and 0.5 uv/division, this indicator will be relatively active, depending on the individual patient. Patient responses, occuring when the limit is on, are rejected from the averaged responses and are neither accumulated nor counted.

The TWF/RUN/EEG should be in RUN for normal operation. When in the TWF position, after a clear the oscilloscopic will display a characteristic test waveform to confirm oscilloscope operation. In the EEG position after a CLEAR, the escilloscope will display the ongoing patient EEG activity, the raw signal from which the averaged response is desired. Fig. 8 shows the flow chart of the system.

The telex 1470 earphone, used with the TA-1000 is heavily damped.

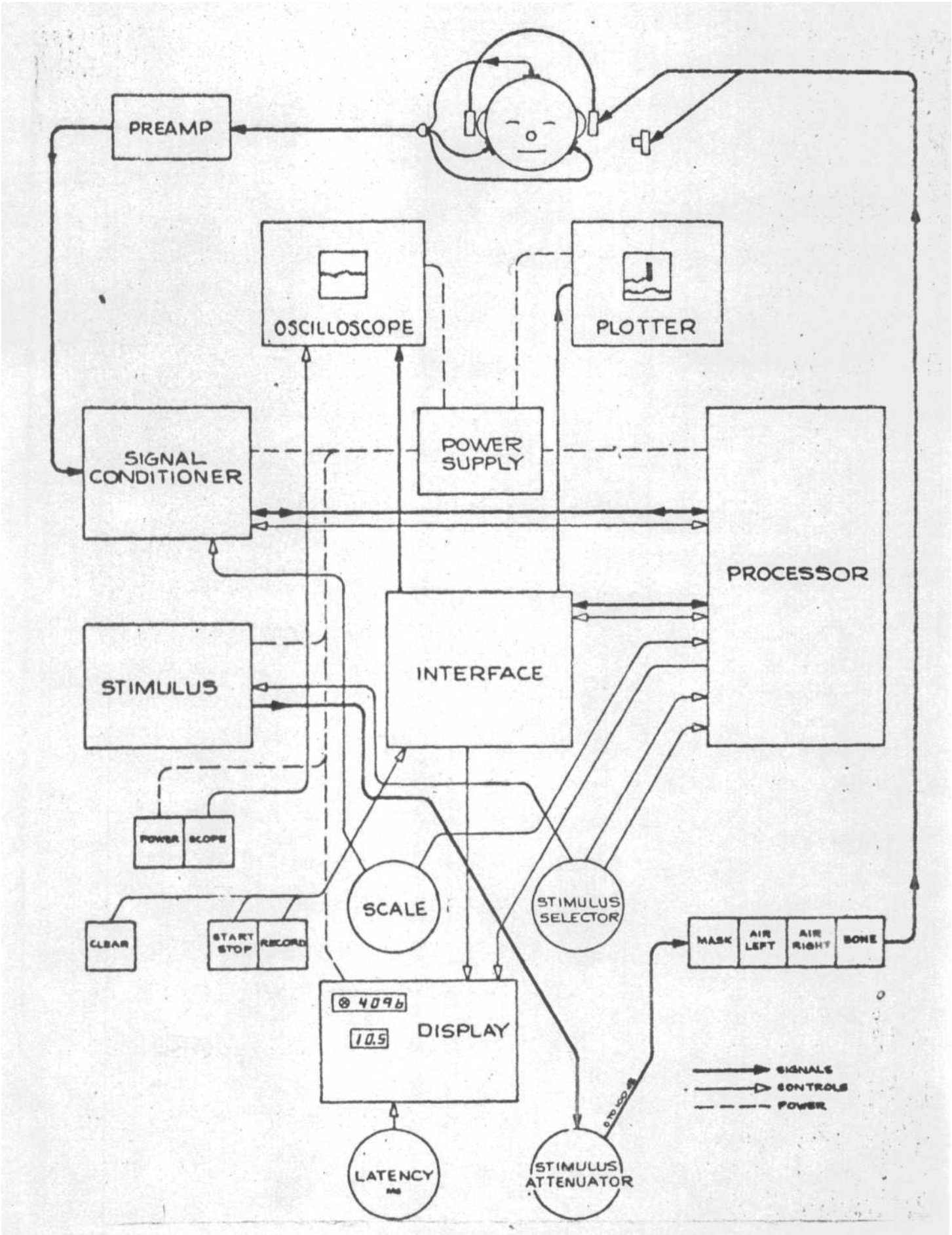

Fig. 8 - Flow chart of ERA: TA-1000 used in the present study.

The tone logon of TA-1000 is a very brief tone pip with very specific rise and decay characteristics. The logon of Gabor is defined as the pure sine wave modulated by a Gaussian distribution function. Gabor in 1947 recognized that rise and decay characteristics of a modulation envelope and the sinusoidal tone being modulated bore a unique time precision (duration) vs. frequency precision (band width) relationship. for a given envelope the value obtained by bandwidth in Hertz was a constant. It is now possible, with the help of hind sight, to visualize Gabor and his associates laboriously computing this figure of macit, TX BW for a series of specific modulation envelopes. On the modulation envelope they tested, the Gaussian distribution curve gave the lowest value and hence the strong endorsement of this curve.

The manufacturers of TA-1000 tested linear ramp, half-sine squared, dual exponential and many other possible functions including the Gaussian distribution curve. They found that their selected functions provide better that the Gabor logon and then by only a slight margin. In recognition of Gabor's pioneering efforts and in view of near identity of the TA-1000s stimulus envelope to a original description the Teledyne Avionics have retained the logon designation.

The TA-1000 stimulus logan is characterized by 3 peaks; in & 50% negative, 100% positive, 50% negative sequence,

followed by a 50% positive sequence reversing on each succe-ssive stimulus.

Stimuli are provided with 2000Hz, 4000Hz and 6000Hz center frequencies; each with a spectrum band width approxi-mately equal to the centre frequency.

The logon stimulus has been determined to be approxi-mately -25 dB effective than a pure tone of the same fre-quency, in terms of hearing threshold SPL.

Latency determination, particularly of wave V is the end result of BSER audiometry. System timing from stimulus to readout, is the key of precise latency determination. In the TA-1000, precise system timing is be design, with each portion of the system controlled and integrated with respect to all components. The system timing diagram is given (fig. 9), illustrates the events between the system trigger pulse and the analysis window.

In the present study logon stimulus of 2 KHz is used. Hence, the electrical spectrum and acoustical spectrum plots are shown in the fig. 10... as specified by Teledyne Avionics.

MEASUREMENT OF PURE TONE THRESHOLDS

Puretone audiometry was done in a sound-treated two roomed situation.

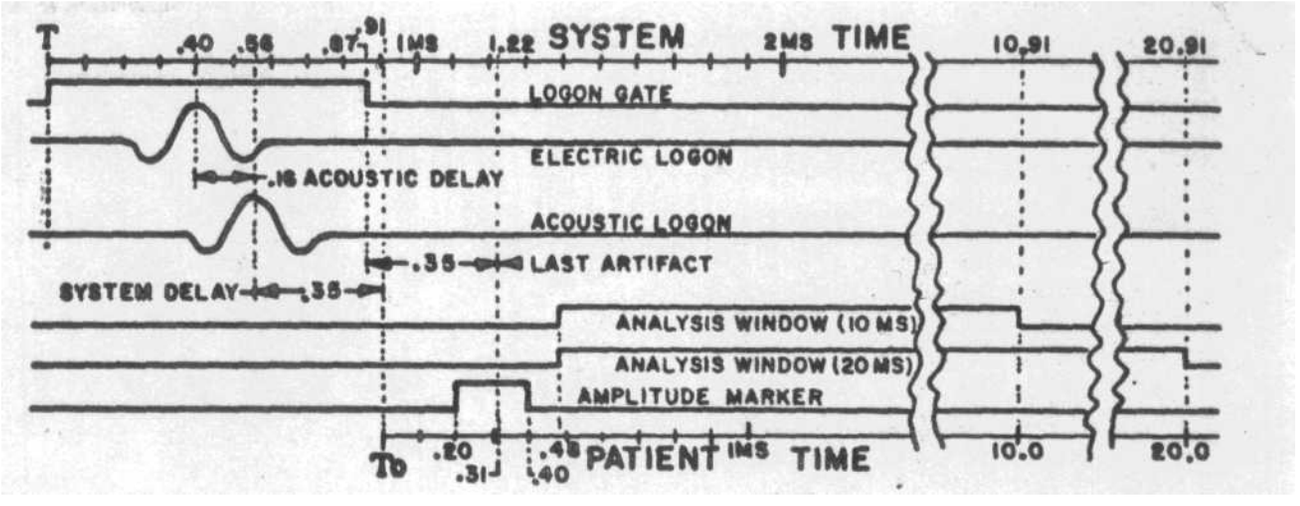

Fig. 9 - System timing Diagram of ERA. Figure illustrates the events between the system trigger pulse and the analysis window.

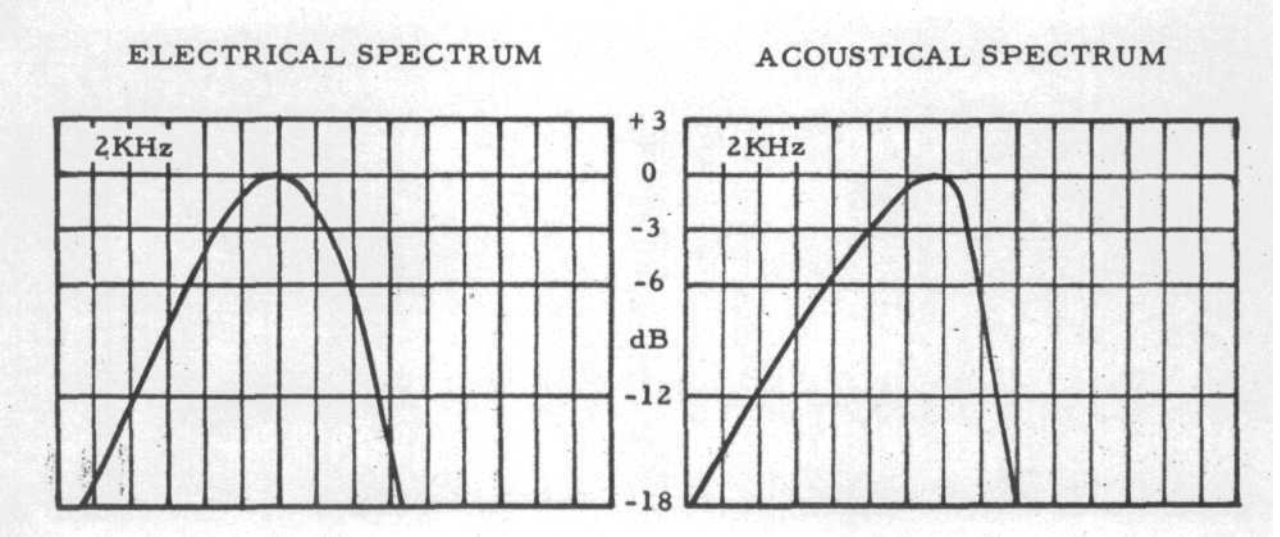

Fig. 10 - Electric and acoustic plots of Logon stimuli (2 KHZ)

Threshold for pure tone were established for the subjects. Hughson -Westlake (1944) procedure was used to establish thresholds as described by Carhart and Jerger (1959).

First a tone at 40 dB was presented to the right ear. If the subject responded presentation level Mas decreased in 10 dB steps until the stimulus became inaudible. Once, the level of inaudibility reached, the level of the tone Mas increased in 5dB steps. And, if the subject did not respond at initial 40 dB presentation level, the intensity level Mas increased in 10 dB steps until the subject responded for the tone. After this, the intensity was decreased for 5 dB and checked the threshold. The level where the subject perceived the stimulus at 50% of the time, is recorded as his/her threshold.

The following instructions were given to the subjects for pure tone audiometry.

"Me are going to test your hearing. I am going to place an earphone over each ear but we shall test only one ear at a time. The object of the test is to find the point where you can just barely detect the presence of the tone. We shall start each time with the tone off. Then, I shall gradually introduce the tone until you can just hear it. As soon as you first hear the tone, signal me. Then, I will make the tone louder. So you can hear it wall. I shall then make the tone softer until you signal me that you can no longer hear it. Then I will make it louder or

softer and turn it on and off while you tell me whether or not you can hear the tone each time, until I am satisfied that we have the point where you can just detect the presence of the tone. Then me will shift it to a different tone and start the process all over. You can signal that you hear the tone by raising your finger. Keep raising the finger as long as you hear the tone. When you no longer hear the tone, do not raise your finger. Do you hear better with one ear than with the other? If so, we mill test the better ear first; If not, we will begin with the right ear. Are you ready? Raise the finger when you hear the tone and keep raising until you cease to hear the tone (Newby, 1958).

CARHART'S TONE DECAY TEST

After establishing the threshold, Carhart's (1957) tone decay test was administered at 250 Hz, 500 Hz, 1K and 2K.

For this, the sustained tone was presented to the subject at his threshold level. He was instructed to respond by raising his finger, as long as he hears the tone. The tone was presented for full one minute. In case, the subject stopped responding before one minute criterion is met, the subject was not taken up for study.

Therefore, all the subjects included in the study did not exhibit abnormal tone decay.

AUDITORY BRAIN-STEM RESPONSE AUDIOMETRY

The ASR experiment was carried out in the sound treated

room of Audiology unit of All India Institute of Speech and Hearing, Manasagangothri, Mysore - 570 006.

i. Bower source: The main A.C. current was canalized to I.T.L. model SUS-200L stabilizer with input 170-270 volts and output of 230 volts. This was stepped down by Kardio S.No.101 to 110 volts which is the requirement of the instrument to function properly.

ii. The Experimental sound treated room had the following characteristics:

(A) Humidity was neither too high or low to the point where either the subject or experimenter were uncomfortable.

(B) The room was away from noisy environment.

(C) A dim light of zero power voltage was put on while experiment was carried out. Curtains were put in the window to control the direct light.

Prior to every test the stabilizer output was checked to ensure a consistent voltage of 200 volts. The chart papers in the plotter was also checked for its proper position. The tabulator pen holder was uncaped.

The subject was asked to lie on the foam bed provided to him/her. The pillow was also provided to avoid muscle tension. The subject was made to relax for sometime. He/She

informed that "We are going to test your hearing. This is not exactly treatment of your problem. Relax properly. After some time the electrodes will be placed behind your ear. Ear-phones will also be placed on your ears. All that you have to do is relax and if possible sleep."

Electrodes were checked with a gentle tug on both ends. They were cleaned with cotton soaked in rectified spirit. (Electrodes were of solid sterling silver).

Cotton soaked in rectified spirit was briskly rubbed on both the wastoids and vertex of the subject and where the ele-ctrodes were supposed to be placed. This was then wiped with dry cotton.

Sufficient quantity of Beckman electrode electrolyte (electrolyte gel) was placed on the electrodes to fill the recess in the electrode to the "slightly rounded" condition and to get applied to the skin. Electrode was placed on the previously cleaned area, pressing gently. The excess of paste which oozed out from the electrode holes and sides was cleaned with dry cotton. Then Johnson adhesive of 2 X 2 cms approxi-mately was used to hold the electrode into firm contact all around.

Electrode placement was as follows:

Red (+) - Signal placed on vertex.

White (-) - Reference placed on low mastoid area on the stimulated ear side.

Black - Guard placed on the low mastoid area of the non-stimulated side.

The electrode end of the preamplifier patient electrode cable was attached to the bed surface near the hear and held in position with adhesive plaster. Each electrode was plugged into the patient electrode cable observing the color code. If reversal of the (-) is desired, white and black plugs at the patient electrode cable were reversed rather than removing and replacing the electrodes (for convenience). Preamplifier was positioned in a convenient locatidn and plugged with the 3 pin patient electrode cable plug into the corresponding preamplifier (they have blue colour code).

Preamplifier and the ERA were interconnected by means of the cable and receptacles which are colour coded (yellow).

Headphones were placed on the ears of the subject in such a way that is was comfortable for the subject.

Power and scope buttons were pressed.

The pre-amplifier high input light was checked. If the red light flashes, it is an indication that the input is greater than 50 microvolts. In such cases, various factors such as wrong placement of the electrodes, pasting of the Johnson plast, tension of the subjects etc. were checked. When the red light of preamplifier was autenemies audomatically off, the condition was assumed to be right. It was an indication that there was no high input. Hence, the experiment could be started.

ERA was set as follows:

- TWF/RUN/EEG was kept on RUN position.
- STIMULUS frequency was set on at 20 pulses per second and 10 ms sample time.
- The SCALE switch was kept on 2048 samples and 0.2 uv/DIV.
- Stimulus intensity was first kept on 100 dB HL.
- CLEAR was pressed and then AIR RIGHT or AIR LEFT button was pressed as desired by the investigator.

The samples was rejected when:

(1) an automatic stop occured before 2048 samples.

(2) when rapid averaging of amplitude was observed, a four division marker was observed in the left side which as test progresses and trace reaches full oscilloscope amplitude,

a two division maker and finally one division was observed If one division was observed before 500 samples or not observed even when 2048 samples were achieved.

When adequate samples and divisions were observed, there was automatic stop after 2048 samples. The internal lamp of the START/STOP was automatically off. Than, RECORD button was pressed. Recording was done by the system on the plotter by tabular pen. To determine absolute latencies the cursor was positioned on each wave and latencies could be red directly in 0.1 ms increments from the displayed digital value.

Whan this part of the experiment was over, the stimulus intensity was over, the stimulus intensity was set at 80 d8 HL. Other conditions remained the same. Latencies were again measured in the same way as it was done for stimulus intensity at 100 dB HL.

Amplitude of ABR was determined for I, III and Vth wave. To determine the amplitudes in microvolts (Mv), the marker amplitude M was noted down either in 1, 2 or 4 divisions. And amplitude of wave I, III and V were noted down. Max. value 4 divisions. SCALE switch amplitude S was .2 uv/div. for eg. a trace feature is 2.5 divisions high and the marker

is 2 division high and the scale switch is set to .2 uv/div.

T=2.5

M=2

S = .2

$$\text{Amplitude} = \frac{TS}{M}$$

$$= \frac{2.5 \times .2}{2}$$

$$= .25$$

All the subjects were tested in the same manner.

CHAPTER - IV
RESULTS

Eight subjects were selected for this study. Out of these eight subjects, six were males and two were females. Their age ranged from 18 years to 36 years with a mean age of 23 years. All these subjects had bilateral sensori-neural hearing loss with varying degrees. They did not exhibit abnormal tone decay in either of the ears. All the ears showing fiat type of audiogram were tested by means of ABR. Hence, a total number of 10 ears were tested. Each ear was tested by using 100 dB HL and 80 dB HL logon stimuli at 2 KHz. Latencies of each wave (I through VI) were determined. Amplitude of wave I, III and V in microvolts (uv) were computed. Later the data was compared with the data obtained from normal hearing subjects by Uma Devi (1983). As far as the morphology of waves are concerned, all the waves were clearly distinct.

Results are discussed in the following steps:

Table 1 shows the absolute latencies of each wave (I through VI for 100dB HL and 80 dB HL logon stimuli.

It can be seen from the table that the latency (eace wave) increases as the stimulus parameter is changed from 100 dB HL to 80 dB HL.

No general tendency of the increase of the absolute latencies depending on hearing level could be observed.

SL No	Name	Age/sex	PTA (dB)	INTENSITY											
				100 dB HL						80 dB HL					
				I	II	III	IV	V	VI	I	II	III	IV	V	VI
1		16Y M		1.1								3.2			
	HM		38(R)		1.9	3.0	4.3	4.9	6.5	1.2	2.2		4.4	5.2	6.5
2	A	18Y M	45(R)	0.9	1.9	2.7	4.0	4.4	5.7	1.1	2.0	2.9	$.0	4.6	6.1
3	HM	26Y M	46(L)	0.9	2.1	3.1	4.2	5.0	6.3	1.2	2.3	3.3	4.6	3.0	6.5
4	GM	18Y M	51(R)	1.0	1.8	3.0	4.2	4.8	6.3	1.3	2.2	3.3	4.4	3.0	6.8
5	K	18Y F	56(R)	0.9	1.9	2.9	3.9	4.7	5.6	1.4	2.3	3.3	4.3	5.0	6.2
6	RG	36Y M	56(L)	0.9	1.9	3.0	4.2	4.7	6.4	1.4	2.3	3.3	4.4	3.0	6.3
7	GM	18Y M	58(L)	0.9	2.1	2.7	3.8	4.8	5.8	.....C N T					
8	R	20Y M	65(R)	0.8	2.2	3.3	4.2	4.9	6.3	1.2	2.8	3.6	4.8	5.4	6.5
9	P	24Y F	68(R)	0.7	2.2	3.3	4.2	4.8	6.3	1.0	2.4	3.5	4.7	5.4	6.6
10	M	23Y M	76(L)	1.2	2.2	3.0	4.2	4.9	5.9	.....C N T					

TABLE:1- Showing the absolute latencies of wave I through VI of each subject by ABR at 100 and 80 dB Logon (2KHz) stmuli.

Key: HL= Hearing level, CNT = could not test, R- right.ear L- Left ear

Table 2 shows the absolute amplitude of Wave I, III and V for each subject at two intensity levels - 100 dB HL and 80 dB HL.

No general tendency of increase or decrease of amplitudes could be observed as the stimulus parameter was changed from 100 dB HL to 80 dB HL.

A general tendency of change in amplitude depending on the hearing level could not be observed.

Latency of each wave (I through VI) and amplitudes of I, III and V are discussed in the following fashion:-

Table 3 shows the absolute latencies of Wave I for each subject for 100 dB HL and 80 dB HL logon stimuli.

Mean of the latencies of Sensori-neural hearing loss subjects at 100 dB HL was .92 ms. The mean of the latencies of normal hearing subjects was .89. The mean differences were not statistically significant at .05 level (Table 12). Also, mean deviation from the mean of normal hearing subjects in percentage was 30%.

Similarly, the mean latency values at 80 dB HL was 1.225 ms and the mean latency t value of normal hearing subjects was 1.235 ms. The difference between these two means were not statistically significant (Table 12).

SI. No.	Name	Age/Sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				I	III	V	I	III	V
1	HM	26Y M	38(R)	.26	.32	.78	.24	.24	.46
2	A	18Y M	45(R)	.12	.40	.96	.16	.18	.88
3	HM	26Y M	46(L)	.24	.26	.66	.20	.20	.50
4	GH	18Y M	51(R)	.10	.16	.80	.22	.18	.57
5	K	18Y M	56(R)	.48	.14	.74	.26	.10	.34
6	RG	36Y M	56(L)	.08	.22	.92	.08	.10	.52
7	GH	18Y M	58(L)	.32	.06	.80	..... C N T		
8	R	20Y M	65(R)	.24	.10	.48	.05	.11	.20
9	P	24Y F	68(R)	.34	.14	.36	.12	.12	.20
10		25Y M	76(L)	.06	.32	.36	..... C N T		

TABLE 2 - Showing the absolute amplitude measured in terms of micro volts (uv) of Wave I III & V of each subject tested by ABR at 100 & 80 dB HL logon (2 KHz) stimuli

KEY: Hearing level, CNT - Could Not Test, R - Right ear , L - Left ear.

I N T E N S I T Y									
SI. No.	Name	Age/ Sex	PTA (dB)	100 dB HL			80 dB HL		
				Subjects' Latency (ms)	Normal Latency Value(ms)	Diffe-rence	Subjects Latency (ms)	Normal Latency Value(ms)	Diffe-rence
1	HM	26Y M	38 (R)	1.1	.89	.21	1.2	1.2	0
2	A	18Y M	S5 (R)	0.9	.89	.01	1.2	1.2	-0.1
3	HM	26Y M	46 (L)	0.9	.89	.01	1.2	1.2	0
4	GH	18Y M	51 (R)	1.0	.89	.11	1.3	1.2	0.1
5	K	18Y F	66 (R)	0.8	.89	-0.09	1.4	1.2	0.2
6	RG	48Y M	56 (L)	0.9	.89	.01	1.4	1.2	0.2
7	GH	18Y M	58 (L)	0.9	.89	.01	CNT	1.2	-
8	R	20Y M	65 (R)	0.8	.89	-0.09	1.2	1.2	0
9	P	24Y f	68 (R)	0.7	.89	-0.19	1.0	1.2	-0.2
10	N	25Y M	76 (L)	1.2	.89	.31	CNT	1.2	-
	Mean age=23 Y			M=.92 SD =.1475	M-.89 SD=.0788	MD% =30	M=1.225 SD= .1388	M=1.235 SD= .1308	MD% = 2.5

TABLE.3 : Showing the absolute latencies of Wave I for each subject for 100 & 80 dS Logon(2KHz) stimuli. Differences from the mean of the Wave I from normal hearing subjects are also shown.

KEY; M = mean, SD= Standard deviation; MD% Mean/from the mean of the normal subjects in percentage.
PTA: Pure tone average. CNT - could not test. HL- Hearing level.

"Source: Uma Devi, Independent project submitted to University of Mysore, 1983.

The mean deviation from the mean of the normal hearing subjects was 2.5%.

The latency difference in normal subjects for a 20dB increase in intensity, is (1.2-.89) ms = 0.31 ms. In sensori-neural hearing loss subjects, the latency difference for an increase of nearly 40 dB loss is (.31 - .21) ms = 0.10 ms. Comparison of the latency increase in normal and sensori-neural hearing loss subjects shows that the latency increase in S-N loss subjects is not in proportion to that of normal subjects. Hence it can be concluded that the I wave latency values in SN loss subjects depend on the energy reaching to the cochlea, but not on SL. In other words the latency of wave I does not depend on the degree of sensori-neural hearing loss.

Table 4 shows the absolute latencies of wave II of each subject for 100 and 80 dB logon stimuli.

At 100 dB HL the mean latency value is 2.02 ms. When compared to normal latency value (1.84 ms), the difference statistically significant at .05 level (Table 12). But, mean deviation from the mean of the normal hearing subjects was found to be 18.0%.

Sl. No.	Naae	Age/ Sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				Subjects' Absolute Latency(ms)	Normal Absolute Latency(ms)	Diffe-rence (ms)	Subjects' Absolute Latency(ms)	Normal* Absolute Latency (ms)	Diffe-rence (ms)
1	HM	26Y M	38(R)	1.9	1.84	.06	2.2	2.24	-0.04
2	A	18Y M	45(R)	1.9	1.84	.06	2.0	2.24	-0.24
3	HM	26Y M	46(L)	2.1	1.84	.26	2.2	2.24	-0.04
4	GH	18Y M	51(K)	1.8	1.84	-0.04	2.3	2.24	0.06
5	K	18Y F	56(R)	1.9	1.84	.06	2.2	2.24	-0.04
6	RE	36Y M	56(L)	1.9	1.84	.06	2.3	2.24	0.06
7	GH	18Y M	58(L)	2.1	1.84	.26	CNT	2.24	
8	R	20Y M	65(R)	2.2	1.84	.36	2.8	2.24	0.56
9	P	24Y r	68(R)	2.2	1.84	.36	2.4	2.24	0.16
10	N	25Y M	76(L)	2.2	1.84	.36	CNT	2.24	
	Mean age=23Y			M = 2.02 SD=1549	M = 1.84 SD=1759	MD%=18.0	M = 2.3 SD=2329	M = 2.24 SO -.1875	MD% 6.0

TABLE -4:Showing the absolute latencies of wave II of each subject for 100 and 80 dB Logon (2KHz) stimuli. Difference from normative data is also computed.

KEY: M= Means SD = Standard Deviation; MD% = Mean Deviation from the mean of normal hearing group in percentage. CNT - could not test, HL- Hearing level

*Source: Uma Devi, Independent Project submitted to University of Mysore, 1983

At 80 dB HL the mean latency value is 2.3 ms and the mean of the latency of normal hearing subjects was 2.24. The difference between these two means were not statisticallt significant. Mean deviation from the mean of the normal hearing group in percentage was found to be 6.0.

For 20 dB of increase brings the change about 2.24-1.84)ms = .40 in normal hearing subjects. Whereas, in sensori-neural hearing loss subjects for about 40 dB loss the difference in latency will be .36-.06) = .20. This is not in proportion to the normal hearing subjects.

Therefore, it can be concluded that the latencies of wave II does not depend on SL, it depends on energy reaching to the cochlea.

Table 5 shows the absolute latencies of wave III of each subjects for 100 dB HL and 80 dB HL logon stimuli.

At 100 dB HL the mean latency in SN loss subjects was 3.0 ms. In normal hearing subjects it was found to be 2.98ms. The differences between these two means were not statistically significant. The mean deviation from the normal hearing subjects were calculated to be 13.0%.

Sl No.	Name	Age/ Sax	PTA (dB)	INTENSITY					
				100 dB HL			80 dS HL		
				Subjects' Absolute Latency(ms)	Normal Absolul:e Latency)(ms)	Diffe-rence (ms)	Subjects' Absolute Latency(ms)	Normal Absolute Latenc%(ms)	Diffe-rence (ms)
1	HM	26Y M	38(R)	3.0	2.98	.02	3.2	3.2	0
2	A	18Y M	45(R)	2.7	2.98	-0.28	2.9	3.2	- 0.3
3	HW	26Y M	46(L)	3.1	2.98	0.12	3.3	3.2	0.4
4	GH	18Y M	51(R)	3.0	2.98	0.02	3.3	3.2	0.1
5	K	18Y f	58(L)	2.7	2.98	0.08	CNT	3.2	0.1
6	RG	36Y M	56(L)	3.0	2.98	0.02	3.3	3.2	0.1
7	GH	18Y M	58(L)	2.7	2.98	0.82	CNT	3.2	-
8	R	20Y M	65(H)	3.3	2.98	0.32	3.6	3.2	0.4
9	P	24Y f	68(R)	3.3	2.98	0.32	3.5	3.2	0.3
10	N	25Y M	76(L)	3.0	2.98	0.02	CNT	3.2	-
	Mean age=23Y			M =3.0 SD =2054	M=2.98 SD =1460	MD%=13.0 M-2.3 SD=2070		M= 3.2 SD =1556	MD%= 10.0

TA8LE-5: Showing the absolute latencies of wave III for each subject for 100 and 80 dB Logon (2KHz) Stimuli. Differences from the mean of the wave III obtained from normal subjects are also shown.

KEY: M = Mean; S.D.= Standard Deviation; MD% = Mean Deviation from the mean of normal hearing groups in percentage. CNT= could not test, HL = Hearing level

Source: Uma Devi, Independent project submitted to University of Mysore, 1983

Similarly at 80 d8 HL the mean latency of the sensorineural hearing loss subjects and normal hearing subjects were 3.3 and 3.2 ms respectively. The mean differences were not statistically significant. Mean deviation from the mean of normal hearing subjects were 10.0%.

The latency difference in normal hearing subjects for a 20dB increase in the intensity is (3.2-2.98)ms = .22 ms In sensorineural subjects latency difference for an increase of nearly 40 dB loss is (0.02v0.02)ms = 0. The comparison of latency increase in normal hnd sensorineural hearing loss subjects shows that the latency increase in sensorineural hearing loss subjects is not in proportion to that of normal subjects.

Hence, it can be concluded that the latency of wave III does not depend on SL, it depends on the energy reaching to the cochlea.

The Table 6 shows the absolute latencies of wave IV.

At 100 dB HL the mean latency value was 4.12 whereas the mean latency value for normal hearing subjects was 4.22. The differences are not statistically significant (Table 12). The mean deviation from the mean of normal hearing group in percentage was found to be 10.0.

At 80 dB HL the mean latency value was 4.45 and the mean latency value of normal hearing subjects was 4.42. The differences were not statistically significant.

The latency difference in the normal hearing subjects for a 20 dB increase in the intensity is (4.42-4.22)ms=.20ms. Whereas in sensorineural hearing loss subjects the latency difference for a increase of nearly 40 dB is (-0.02-0.08)ms = -.10 ms. The comparison of latency increase in normal and sensori-neural hearing loss subjects shows that the latency increase in sensori-neural hearing loss subjects is not in proportion to that of normal subjects.

Hence, It can be concluded that the latency of wave IV does not depend on SL, it depends on energy reaching to the cochlea.

Table 7 shows the absolute latencies of the wave V of each subject for 100 and 80 dB logon stimuli.

At 100 dB HL the mean latency value of the sensorineural hearing loss group was 4.79 ms. The normal mean value was 4.77. When compared these two means, they were not statistically significant. The Mean deviation from the mean of normal hearing group in percentage was computed to be 2.0.

Sl. No.	name	Age/ sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				Subjects' Absolute Latency(ms)	Normal Absolute Latency(ms)	Diffe-rence (ms)	Subjects' Absolute Latency(ms)	Normal . Absolute Latency(ms)	Diffe-rence (ms)
1	HM	26Y M	38(R)	4.3	4.22	.08	4.4	4.42	-0.02
2	A	18Y M	45(R)	4.0	4.22	-0.22	4.0	4.42	-0.42
3	HM	26Y M	46(L)	4.2	4.22	-0.22	4.6	4.42	-0.18
4	GH	18Y M	51(R)	4.2	4.22	-0.02	4.4	4.42	-0.02
5	K	18Y F	56(R)	3.9	4.22	-0.32	4.3	4.42	-0.12
6	RG	36Y M	56(L)	4.2	4.22	-0.02	4.4	4.42	-0.02
7	GH	18Y M	58(L)	3.8	4.22	-0.42	CNT	4.42	-
8	R	20Y M	65(R)	4.2	4.22	-0.02	4.8	4.42	0.38
9	P	24Y F	68(R)	4.2	4.22	-0.02	4.7	4.42	0.28
10	N	25Y M	76(L)	4.2	4.22	-0.02	CNT	4.42	
	Mean age=23Y			M =4.12 SD =.1619	M=4.22 SD =1712	MD%= 10.0	M=4.45 SD=2507	M= 4.42 SD=.1852	MD% = 3.0

TABLE-6: Showing the absolute latencies of wave IV of each subject for 100 and 80 dB Logon(2KHz) stimuli. Differences from the mean of the wave IV obtained from normal hearing subjects are also shown.

KEY: M= Mean, S.D = Standard Deviation; MD% = Mean Deviation from the mean of normal hearing group in percentage. CNT= could not test, HL = Hearing level, R= right, L= Left

Source: Uma Devi, Independent project submitted to University of Mysore, 1983

Sl. No.	Name	Age/ Sex	PTA (dB)	INTENSITY					
				Subjects' Latency value(ms)	Normal* Latency Value(ms)	Diffe-rence (ms)	Subjects Latency Value(ms)	Normal* Latency value(ms)	Diffe-rence (ms)
1	HM	26Y M	38(R)	4.9	4.77	0.13	5.2	5.03	0.17
2	A	18Y M	45(R)	4.4	4.77	-0.37	4.6	5.03	-0.43
3	HM	26Y M	46(L)	5.0	4.77	0.23	5.1	5.03	0.07
4	GH	18Y M	51(R)	4.8	4.77	0.03	5.0	5.03	-0.03
5	K	18Y F	56(R)	4.7	4.77	-0.07	5.0	5.03	-0.03
6	RG	36Y M	56(L)	4.7	4.77	-0.07	5.0	5.03	-0.03
7	GH	18Y M	58(L)	4.8	4.77	0.03	CNT	5.03	
8	R	20Y M	65(R)	4.9	4.77	0.13	5.4	5.03	0.37
9	P	24Y F	68(R)	4.8	4.77	0.03	5.4	5.03	0.37
10	N	25Y M	76(L)	4.9	4.77	0.13	CNT	5.03	-
				M=4.79	M=4.77	MD%=2.0	M=5.08	M=5.03	MD% 5.75
Mean age =23Y				SD=.1663	SD=.1976		SD=.2587	SD =.1750	

TABLE-7 Showing the absolute latencies of Wave V of each subject for 100 and 80 dB Logon (2KHz) stimuli. Differences from the mean of the Wave V obtained from normal hearing subjects are also shown.

KEY! M = Mean; SD= Standard Deviation; MD= Mean Deviation from the mean of norman hearing group in percentage. CNT = could not test, HL= Hearing level, R= right, L= Left

S0URCE Uma Devi, Independent project submitted to University of Mysore, 1983

At 80 dB HL the mean values were 5.08 for sensori-neural Loss subjects and 5.03 for normal hearing subjects. These differences were not statistically significant. The mean deviation from the mean of normal hearing group was found to be 5.75%.

The latency difference in the normal hearing subjects for a 20 dB increase in the intensity is (5.03-4.77)ms = .26ms. whereas in sensori-neural hearing loss subjects, latency difference for a increase of nearly 40 dB is (0.13-0.13)ms = 0. The comparison of latency increase in normal and sensori-neural hearing loss subjects shows that the latency increase in cochlear hearing loss subjects is not in proportion to that of normal hearing subjects.

Hence, it can be concluded that the latency of wave V in sensori-neural hearing loss subjects depend upon the energy reaching to the cochlea, not on SL.

Table 8 shows the absolute latencies of wave VI of each subject, tested at 100 and 80 dB logon stimuli.

At 100 dB HL the mean latency value in our subjects was 6.11 ms. In normal hearing subjects the mean value was 6.26. The differences between these two means were not statistically significant. The mean deviation from the mean of normal hearing subjects was computed to be 18.75%.

Sl. No.	Name	Age/ Sex	PTA (dB)	INTENSITY					
				100dB HL			80 dB HL		
				Subjects' Absolute Latency(ms)	Normal* Absolute Latency(ms)	Diffe-rence (ms)	Subjects' Absolute Latency (ms)	Normal* Absolute Latency(ms)	Diffe-rence (ms)
1	HM	26Y M	38(R)	6.5	6.26	0.24	6.5	6.58	-0.08
2	A	18Y M	45(R)	5.7	6.26	-0.56	6.1	6.58	-0.48
3	HM	26Y M	46(L)	6.3	6.26	0.04	6.5	6.58	-0.08
4	GH	18Y M	51(R)	6.3	6.26	0.04	6.8	6.58	0. 22
5	K	18Y F	56(R)	5.6	6.26	-0.66	6.2	6.58	-0.38
6	RG	36Y M	56(L)	6.4	6.26	0.14	6.5	6.58	-0.08
7	GH	18Y M	58(L)	5.8	6.26	-0.46	CNT	6.58	
8	R	20Y M	65(R)	6.3	6.26	0.04	6.5	6.58	-0.08
9	P	24Y F	68(R)	6.3	6.26	0.04	6.6	6.58	0.02
10	N	25Y M	76(L)	5.9	6.26	-0.36	CNT	6.58	
	Mean	age-23Y		M= 6.11 SD= .3247	M= 6.26 SD= .2345	MD%=18.75	M-6.46 SD=.2199	M= 6.58 SD=.2912	MD%= 11.75

TABLE-8 Showing the absolute latencies of wave VI of each subject for 100 and 80 dB Logon (2KHz) stimuli. Differences from the Mean of the wave VI obtained from normal hearing subjects are also shown.

KEY: M= Means S.D.= Standard Deviation; MD%= Mean Deviation from the mean of normal hearing subjects in percentage. CNT= could not test, HL= Hearing Level, R=Right, L= Left

SOURCE: Uma Devi, Independent project submitted to University of Mysore, 1983.

At 80 dB HL the mean latency value in sensori-neural hearing loss subjects and normal hearing subjects were 6.46 and 6+58 respectively. The differences were not statistically significant. The mean deviation from the mean of normal hearing subjects is calculated to be 11.75%.

The latency difference is the normal hearing subjects for a 20 dB increase in the intensity is (6.58-6.26)ms= .32 ms. In sensorineural hearing loss subjects latency difference for a increase of nearly 40 dB is (-0.36-0.24)ms = -60ms. The comparison of latency increase in normal and sensorineural hearing loss subjects shows that the latency increase in cochlear hearing loss group is not in proportion to that of normal hearing subjects.

Therefore it can be concluded that the absolute latency of wave VI does not depend on SL, it is dependent on energy reaching to the cochlea.

Table 9 shows the absolute amplitude of wave I for each subject, tested at 100 and 80 dB logon stimuli.

At 100 dB HL the mean value of hearing loss group was .224 uv and normal hearing group was .362 uv. The mean difference was found to be significant at .05 level but it was not significant at .01 level. The amplitude of sensori-neural

Sl. No.	Name	Age/ sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				Subjects' Absolute Amplitude (uv)	Normal* Absolute Amplitude (vu)	Diff- rence (vu)	Subjects' Absolute Amplitude (vu)	Normal* Absolute Amplitude (vu)	Diffe- rence (Vu)
1	HM	26Y M	38(R)	.26	.36	-0.10	0.24	.26	-0.02
2	A	18Y M	45(R)	.12	.36	-0.24	0.16	.26	-0.10
3	HM	26Y M	46(L)	.24	.36	-0.12	0.20	.26	-0.06
4	GH	12Y M	51 (R)	.10	.36	-0.26	0.22	.26	-0.04
5	K	18Y M	56(R)	.48	.36	0.12	0.26	.26	0
6	RG	48Y M	56(L)	.08	.36	-0.28	0.08	.26	-0.18
7	GH	18Y M	58(L)	.32	.36	-0.04	CNT	.26	-
8	R	20Y M	65(R)	.24	.36	-0.12	0.05	.26	-0.21
9	P	24Y F	68(R)	.34	.36	-0.02	.12	.26	-0.14
10	N	25Y M	76(L)	.06	.36	-0.30	CNT	.26	-
		Mean age:23Y		M = .224	M= .362	MD%=13.6	M =.166	M=.265	MD%=-9.37
				SD = .1349	SD= .1598	.	Sd=.0768	SD = .1203	

TABLE 9.Showing the Absolute Amplitude of Wave I for each subject for 100 and80 dB Logon(2 KHz) stimuli. Differences from the mean of the wave 1 obtained from the amplitude of Wave I

KEY: normal hearing subjects are also shown.

M= Mean; S.D. = Standard Deviation; MD% = Mean Deviation from the mean of normal hearing group in percentage. CNT= could not test, HL= hearing level, R=Right, L=Left

"SOURCE: Uma Devi, Independent Project submitted to University of Mysore, 1983

hearing loss subjects was lesser than normal hearing subjects. However, the total mean deviation a from the mean of normal hearing subjects was found to be 13.6%.

At 80 dB HL the mean of sensori-neural hearing loss subjects was .166 uv. When compared with the normal hearing subjects whose mean was .265 uv, the mean difference were statistically significant at +05 level but not at .01 level. The amplitude in sensori-neural hearing loss subjects was lesser than normal hearing subjects. However, the total mean deviation from the mean of the normal hearing group was 9.37%.

Therefore, it can be concluded that the amplitude of wave I is lesser in cases of cochlear hearing loss as compared to normal hearing subjects.

Table 10 shows the absolute amplitude of wave III for each subject tested at 100 dB HL and 80 dB HL logon stimuli.

At 100 dB HL the mean amplitude values of sensori-neural hearing loss group was found to be .212 uv and the mean of normal hearing subjects was .2795 uv. Statistically these differences were not significant. However, as evident from the table, the amplitudes of sensorivneural hearing loss subjects were lesser reduced except two cases Sl. No. 1 and 10. It can be concluded

Sl. No.	Name	Age/ Sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				Subjects' Absolute Amplitude (vu)	Normal * Absolute Amplitude (vu)	Difference (vu)	Subjects' Absolute Amplitude (vu)	Normal * Absolute Amplitude (Vu)	Difference (vu)
1	HM	26Y M	38(R)	.32	.27	.05	.24	.29	-0.05
2	A	18Y M	45(R).40		.27	0.13	.18	.29	-0.11
3	HM	26Y M	46(L)	.26	.27	-0.01	.20	.29	-0.09
4	GH	12Y M	51 (R)	.16	.27	-0.11	.18	.29	-0.11
5	K	18Y F	56(R)	.14	.27	-0.13	.10	.29	-0.19
6	RG	48Y M	56(L)	.22	.27	-0.05	.10	.29	-0.19
7	GH	18Y M	58(L)	.06	.27	-0.21	CNT	.29	-
8	R	20Y M	65(R)	.10	.27	-0.17	.11	.29	-0.18
9	P	24Y F	68(R)	.14	.27	-0.13	.12	.29	-0.17
10	N	25Y M	76(L)	.32	.27	0.05	CNT	.29	
				M= .212	M=.2795	MD%=-5.8	M =.153	M= .2965	MD% =13.62
		Means= 23Y age SD = .1104 aye		SD =.1695			SD =.0531	SD =.1339	

TABLE 10-Showing the Absolute Amplitude of Wave III of each subject for 100 and 80 dB Logon (2kHz) stimuli. Differences from the mean of the amplitude of wave III obtained from normal hearing subjects are also shown.

KEY: M = Mean; SD = Standard Deviation; MD = Mean Deviation from the mean of normal hearing group in percentage.

*SOURCE Uma Devi, Independent Project submitted to University of Mysore, 1983

that there is a tendency of reduced amplitude in sensori-neural hearing loss subjects. The total mean deviation from the mean of normal hearing group is 5.8%.

At 80 dB HL the mean amplitude of the sensori-neural hearing loss subjects and normal hearing subjects were .153 uv and .2965 uv respectively. These mean differences were found to be statistically significant at .05 level. The total mean deviation in percentage was 13.62. As evident from the table that all the subjects who could be tested had reduced amplitude.

Therefore, it can be concluded that there is a tendency of wave III amplitude to be reduced in cases of sensori-neural hearing loss subjects with compare to normal hearing subjects. But they may or may not be statistically significant.

Table 11 shows the absolute amplitude of wave V of each subject for 100 dB HL and 80 dB HL logon stimuli.

At 100 dB HI the mean amplitude was .686 uv whereas the normal mean values was .587. The mean differences were not statistically significant. The total mean deviation of normal hearing subjects in percentage was computed to be 10.6. Out of 10 subjects only three showed reduced amplitude. Hence, a general tendency of reduced amplitude could not be observed at 100 dB HL.

SL. No.		Age/ sex	PTA (dB)	INTENSITY					
				100 dB HL			80 dB HL		
				Subjects' Absolute Amplitude (uv)	Normal * Absolute Amplitude (uv)	Difference (uv)	Subjects' Absolute Amplitude (uv)	Normal* Absolute Amplitude (uv)	Difference (uv)
1	HM	26Y M	38(R)	.78	.58	0.20	.46	.51	-0.05
2	A	18Y M	45(R)	.96	.58	0.38	.88	.51	0.37
3	HM	26Y M	46(L)	.66	.58	0.08	.50	.51	-0.01
4	GH	12Y M	51(R)	.80	.58	0.22	.57	.51	0.06
5	K	18Y F	56(R)	.74	.58	0.16	.34	.51	-0.17
6	RG	48Y M	56(L)	.92	.58	0.34	.52	.51	0.01
7	GH	18Y M	58(L)	.80	.58	0.22	CNT	.51	
8	R	20Y M	65(R)	.48	.58	-0.10	.20	.51	-0.31
9	P	24Y F	68(R)	.36	.58	-0.22	.20	.51	-0.31
10	N	25Y M	76(L)	.36	.58	-0.22	CNT	.51	-
Mean age=23Y				M = .686 SD = .2168	M= .587 SD= .1925	MD%=10.6	M= .458 SD = .2214	M= .512 SO= .1535	MD%=5.12

TABLE 11 Showing the Absolute Amplitude of wave V of each subject for 100 and 80 dB Logon (2kHz) stimuli. Differences from the mean of the Amplitude wave V obtained from the normal hearing subjects are also shown.

KEY: M= Mean; SD = Standard Deviation; MD% = Mean Deviation of normal hearing group in percentage. CNT= could not test, HL=Hearing level, R=Right, L=Left

*SOURCE: Uma Devi, Independent Project submitted to University of Mysore, 1983

At 80 dB ML the mean amplitude value of sensori-neural loss subjects was .458 uv. The normal amplitude value was .512 uv. The mean differences were not statistically significant. But it can be said that the sensori-neural hearing loss subjects had lesser absolute amplitude value than the normal hearing subjects, In the table it is evident that out of 8 subjects tested at 80dB HL, five subjects showed lesser amplitude than the normal amplitude Value.

Table 12 shows the positive values (critical ratio) for testing the significance of the mean between the two groups, i.e. sensorineural hearing loss subjects and normal hearing subjects. As described earlier, in this study ABR of cochlear hearing loss subjects are compared with the normal hearing subjects. The normal hearing subjects were tested by Uma Devi (1983). Her data is presented in the Appendix - 8.

TABLE 12 Showing the t values (Critical ratio) for testing the significance of the mean between the two groups i.e., Sensori-neural hearing loss subjects & normal hearing subjects.

t Values

	100 dS HL (df=28)	80 dB HL (df=26)
Latency Wave - 1	.74 (Not significant at at .05 level and .01 level)	-0.174 (Not significant at .05 level and .01 level)
Latency Wave - II	2.69 (significant at .05 level but not at.01 level)	.7042 (Not significant)
Latency Wave - III	.3094 (Not significant)	1.398 (Not significant)
Latency Wave - IV	1.519 (Not significant)	.3488 (Not significant)
Latency Wave - V	.2721 (Not significant)	.5896 (Not significant)
Latency Wave - VI	-1.4918 (Not significant)	- 1.08 (Not significant)

(contd. ii)

TABLE -12 (Contd)

	t Values	
	100 dB HL (df=28)	80 dB HL (df=26)
Amplitude Wave - I	- 2.351 Significant at .05 level but not at .01 level.	-2.136 Significant at .05 level but not at .01 level
Amplitude Wave - III	-1.13 (Not significant)	-2.90 (Significant at 0.05 level and also significant at .01 level)
Amplitude Wave - V	1.262 (Not significant)	-0.74 (Not significant)

KEY: df = Degree of Freedom

The sign (-) indicates that the mean value of Sensorineural hearing loss subjects group was lesser than the mean value of normal hearing subject group.

CHAPTER - V

DISCUSSION

It is well known that retrocochlear lesions alter auditory brainstem responses. But the question remains whether the sensorineural loss excluding retro-cochlear lesions affect on ABR. Rosenhamer (1980) has found that cochlear lesions also alter the ABR but the changes are generally smaller (provided that the cochlear loss is not very severe). Rosenhamer writes ABR changes in cochlear hearing loss are generally moderate and may be related to the degree of hearing loss. In this study the ears showing flat type of audiometric configuration, having sensorineural hearing loss exhibiting no abnormal tone decay are tested to see whether the degree of hearing loss is related to the ABR. The present study did not indicate that there may be some change in terms of latencies of ABR when compared to the normal hearing subjects. Hence, it is difficult to generalize the statement taht cochlear lesion may cause prolonged wave latencies. Also, this study points out that the degree of hearing loss are not related to that of ABR latencies. This is in accordance with the result of Moller and Blegrad (1976). They established that the latency increase of wave V was less pronounced in patients with flat loss than in patients with gradually sloping high frequency loss, the longest latencies were seen in patients with steepy sloping loss. In this group of patients, the

latency increment increased with pure tone average from 0.5 to 4 KHz (this investigation comprised a total of 48 patients with symmetrical loss, binaural high level stimulation was used). In the present study the ears were stimulated monaurally.

Kavanagh & Beardsley (1979) found that the ABR may be sensitive to cochlear hearing loss but met with normal V latencies at high click levels in 24 cochlear patients in high frequency loss in order of 50-60 dB HL. In the present study the variable 'High frequency loss' was controlled. The author selected only those subjects whose audiometric configuration was flat. In these subjects also the latencies were almost like normal hearing subjects.

Rosenhamer (1980) measured wave V latencies and I-V, III-V IPL at 80 dB HL (unfiltered clicks of alternating polarity in 11 cochlear ears with rising, 22 ears with fiat and 77 ears with sloping audiograms. They found in the three groups a clear increase of wave V related to the hearing loss at high frequencies (the pure tone threshold at 4 KHz was used as independent variable). The relation between the wave V latency increase and 4 KHz hearing loss was statistically significant (at level .05) only in a group with sloping audiograms. On the other hand at the

click level of 60 dB SL when they stimulated the cochlear ear at high level, no such suave V latency increments were observed in several ears with flat loss and in some ears with sloping audiograms. Our results also show that wave V latency increment was not observed in ears with flat loss (Though in this study logon stimuli at 2 KHz was used and we have tried to find the relation with pure tone average and latencies) whereas Rosen-hamer used click stimuli and they compared wave V latency with the threshold at 4 KHz).

Wave Q I, III and V are usually prominent hence, the amplitudes of these waves are usually studied. There is little information in the literature of amplitudes of wave I, III and V in cases with sensori-neural hearing loss, excluding retrococh-lear pathology. In the present study an attempt is made to study the amplitudes of ABR. It is found that the amplitudes of wave I and III were reduced. Because, the sample of this data is not large, the statement cannot be generalized. However, some studies to support or contradict the statement is recommended. But this study indicated that there was no significant difference in terms of amplitude of Vth wave when compared to normal hearing subjects.

CHAPTER - VI

SUMMARY & CONCLUSION

Eight subjects (ten ears) having flat type of varying degree of sensori-neural hearing loss were selected for the study. Out of these eight subjects six were males and two were females. Their age ranged from 18 years to 36 years (mean age = 23 years). Retrocochlear pathology was ruled out by using Carhart's tone decay test.

Brain stem electric response audiometry was done for all the selected subjects using the 100 dB HL and 80 d8 HL logen stimuli at 2 KHz. Absolute latencies of each wave (I through VI) were measured in terms of microvolts (uv).

The data was compared with the data derived from normal hearing subjects (Uma Devi, 1983). Following conclusions were drawn from the study:- .

(1) There was no stististically significant difference between the absolute latencies obtained from sensori-neural hearing loss subjects and normal hearing subjects except the latency of wave II at 100 dB HL.

(a) There was no statistically significant difference between the mean absolute latency values of wave I obtained from the sensori-neural hearing loss subjects and normal hearing subjects.

(b) At 100 dB HL there was a significant difference at .05 level only between the mean absolute latency of wave II obtained from sensori-neural hearing loss subjects and normal hearing subjects.

But there was no significant difference between the mean absolute latency of wave II obtained from the sensori-neural hearing loss subjects and normal hearing subjects.

(c) There was no statistically significant difference between the mean absolute latency of wave III obtained from the sensorineural hearing loss subjects and normal hearing subjects.

(d) There was no statistically significant difference between the mean absolute latency of wave IV obtained from the sensori-neural hearing loss subjects and normal hearing subjects.

(e) There was no statistically significant difference between the mean absolute latency of wave V obtained from sensori-neural hearing loss subjects and normally hearing Subjects.

(f) There was no statistically significant difference between the mean absolute latency of wave VI obtained from

sensori-neural hearing loss subjects and normally hearing subjects.

2. A general tendency of reduced amplitude of wave I, III and V of reduced amplitude S.N. hearing loss subjects when compared to normal hearing subjects were seen.

(a) There was statistically significant difference in mean absolute amplitude of wave I obtained from the sensori-neural hearing loss subjects and normal hearing subjects at .05 level.

(b) At 100 dB HL thare was no statistically significant difference between the mean absolute amplitude of wave III obtained from the sensori-naural hearing loss subjects and normal hearing subjects.

But at 80 dB HL there was statistically significant difference between the mean absolute amplitude of wave III obtained from the sensori-neural hearing loss subjects and normal hearing subjects at .05 level.

(c) There was no statistically significant difference between the mean absolute amplitude value of wave V obtained from the sensori-neural hearing loss subjects and normal hearing subjects.

From this study, it can be concluded that absolute latencies (I through VI) do not depend on the sensation level, but they depend on the energy reaching the cochlea.

Sensorineural hearing loss may affect the amplitude of wave I, III and V. The results show that the sensori-neural hearing loss reduces the amplitude of I and III waves.

A study can be undertaken to see whether the amplitudes are really reduced depending on the degree of hearing loss.

Limitations

1. The sample size is small
2. The probable role of sex on the obtained data was not studied.

Recommendations

The present study shows that the amplitude of I and III waves is significantly reduced in sensori-neural hearing loss subjects. It is worthwhile to test many sensorineural hearing loss (uniform hearing loss) subjects to confirm the effect of degree of hearing loss on the amplitude of audi-tory brain-stem responses.

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APPENDIX - A

Sl.No.	Name	Age/Sex	Probable aetiology of the hearing loss
1	HM	26 Y M	Hearing loss followed with tinnitus
2	A	18Y M	gradual hearing loss, idiopathic Noice induced
3	HM	26 Y M	Gradual loss, idiopathic
4	GH	18Y M	Noice induced, gradual
5	K	18 Y F	Hereditary
6	RG	36 Y M	Ototoxic
7	GH	18 Y M	Noise induced
8	R	20 Y M	Hereditary hearing loss
9	P	24 Y F	Gradual onset since 9 yrs. idiopathic
10	N	25Y M	Hereditary hearing loss

Table (i) - showing the probable aetiology ofthe hearing loss of subjects tested in this study.

APPENDIX - B

Sl. No.	Age/ sex	2 KHz Logan Stimuli			
		100dB_HL		80dB_HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	0.9	0.9	1.4	1.3
2	22Y M	1.0	l.P	1.3	1.3
3	17½YM	0.9	0.8	1.3	1.1
4	20Y F	0.8	0.8	1.1	1.1
5	19Y M	0.8	0.8	l.O	1.1
6	22½YM	l.O	0.9	1.3	1.3
7	22Y F	1.0	1.0	1.4	1.3
8	18½Y F	0.9	0.8	1.4	1.3
9	18Y M	0.9		1.4	1.1
10	20Y F	0.9	0.9 0.8	1.1	1.1
	Mean age=20	M=.89 S.D. = .7888		M= 1.235 SD= .1308	

Table (iii) - showing the absolute latencies of Wave I for 100dB HL and 80 dB HL Logan stimuli in normal subjects (N=10).

Source: Uma Devi, Independent project, 1983 submitted to the University of Mysore.

Key: M= Mean, SD= Standard Deviation.

APEENDIX - B

Sl. No.	Age/Sex	2 KHz Logan stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	2.0	2.0	2.4	2.4
2	22Y M	2.1	2.0	2.5	2.4
3	17½Y M	1.7	1.5	2.0	2.0
4	20Y F	1.9	1.8	2.1	2.0
5	19Y M	1.6	1.7	1.9	2.O
6	23½Y M	1.9	2.0	2.3	2.4
7	22Y F	2.0	2.0	2.5	2.3
8	18½Y F	1.9	1.8	2.2	2.2
9	18Y M	1.5	1.9	2.2	2.4
10	20Y F	1.8	1.7	2.4	2.2
	Mean age= 20	M = 1.84 SD = .1759		M = 2.24 SD = .1875	

Table (iii) - showing the absolute latencies of wave II for 100 dB HL and 80 dB HL Logan stimuli in normal hearing subjects.

Source: Uma Devi, Independent Project submitted to the University of Mysore, 1983.

Key: M = Mean, SD = Standard Deviation, Rt= Right Lt. = Left.

APPENDIX - B

Sl. No.	Age/ sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	3.2	3.0	3.3	3.2
2	22Y M	3.2	3.1	3.4	3.3
3	17½Y M	2.9	2.8	3.3	3.0
4	26Y F	2.7	2.7	2.9	2.9
5	19Y M	2.9	3.0	3.1	3.0
6	23½Y M	2.9	3.0	3.1	3.2
7	22Y F	3.1	3.0	3.3	3.3
8	18½Y F	2.9	2.9	3.2	3.2
9	18Y M	3.1	3.1	3.4	3.4
10	20Y F	3.1	3.1	3.3	3.2
	Mean age = 20	M = 2.985 = 3.0 SD = .1460		M = 3.2 SD = .1556	

Table (iv) - showing the absolute latencies of wave III in normal hearing subjects for 100dB HL and 80dB HL Logan stimuli (N=10).

Source: Uma Devi, Independent project submitted to the University of Mysore, 1983.

Key: M= Mean, SD= Standard Deflation, Rt.= Right, Lt.=left

APPENDIX - B

Sl. N.	Age/ Sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	4.6	4.3	4.6	4.3
2	22Y M	4.4	4.3	4.7	4.6
3	17½Y M	4.1	4.0	4.2	4.3
4	20Y F	4.0	4.0	4.2	4.1.
5	19Y M	4.3	4.3	4.5	4.4
6	23½Y M	4.0	4.0	4.3	4.4
7	22Y F	4.3	4.3	4.7	4.6
8	18½Y F	1.4	4.2	4.2	4.5
9	18Y M	4.1	4.2	4.7	4.3
10	20Y F	4.3	4.4	4.4	4.4
	Mean age=20Y	M=4.225 SD = .1712		M = 4.42 SD = .1852	

Table (v) - Showing the absolute latencies of Wave IV in normal hearing subjects for 100 dB HL and 80 dB HL Logan Stimuli (N = 10)

Source: Uma Devi, Independent project submitted to the University of Mysore, 1983.

Key: M=Mean, SD= Standard Deviation, Rt.=Right, Lt.=Left

APPENDIX - B

Sl. No.	Age/ sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	4.8	4.6	5.2	4.9
2	22Y M	5.0	5.0	5.3	5.2
3	17½Y M	4.8	4.7	5.1	5.0
4	20Y F	4.5	4.4	4.7	4.7
5	19Y M	4.7	4.5	5.0	4.9
6	23½Y H	4.8	4.7	4.9	5.0
7	22Y F	4.7	4.6	5.1	4.9
8	18½Y F	4.8	4.7	4.9	5.0
9	18Y M	5.0	5.0	5.2	5.3
10	20Y F	5.0	5.1	5.1	5.2
	Mean age=20Y	M . 4.77 SD = .1976		M = 5.03 SD = .1750	

Table (vi) . showing the absolute latencies of wave V in normal subjects for 100 dB HL and 80 dB HL Logan Stimuli (N = 10).

source: Uma Devi, Indendent project submitted to the University of Mysore, 1983.

Key: M = Mean, SD=Standard Deviation, RT= Right Lt.=Left

APPENDIX - B

NO.	Age/ sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	6.5	6.2	6.9	6.4
2	22Y M	6.6	6.4	7.0	6.7
3	17½Y M	6.8	6.2	7.1	6.7
4	20Y F	5.9	5.9	6.2	6.1
5	19Y M	6.3	6.3	6.4	6.5
6	23½Y M	6.1	6.1	6.1	6.4
7	22Y F	6.2	6.2	6.3	6.7
8	18½Y F	6.0	6.1	6.6	6.4
9	18Y M	6.2	6.3	6.6	6.8
10	20Y F	6.6	6.4	6.8	6.7
Mean age = 20Y		M = 6.265 SD = .2345		M = 6.58 SD = .2912	

Table (vii) - showing the absolute latencies of wave VI in normal subjects for 100 dB HL and 80 dB HL Logan stimuli (N = 10).

Source: Uma Devi, Independent project, submitted to the University of Mysore, 1983

Rey: M = Mean, SD = Standard Deviation, Rt.=Right,Lt.=Left

APPENDIX - B

Sl. No.	Age/ Sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	.32	.36	.12	.28
2	22Y M	.20	.33		.26
3	17½Y M	.26	.26	.20	.20
4	20Y F	.52	.38	.38	.38
5	19Y M	.26	.24	.26	.24
6	23½Y M	.70		.36	.60
7	22Y F	.70	.40	.18	.22
8'	18½Y F	.28	.40	.08	.26
9	18Y M	.10	.24	.08	*24
10	20Y F	.26	.48	.36	#36
	Mean age - 20Y	M = .2625 SD = .1598		M = .265 SD = .1203	

Table (viii)- showing the absolute Amplitudes of wave I measured in u volts in normal hearing subjects for 100 dB HL and 80 dB HL (N=10).

Source: Uma Devi, Independent project submitted to the University of Mysore, 1983.

Key: M = Mean, S.D.= standard Deviation, Rt.= Right Lt.= Left.

APPENDIX - B

Sl. No.	Age/ sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Lt.	Rt.	Lt.	Rt.
1	19Y M	.16	.28	.44	.30
2	22Y M	.09	.19	.20	.17
3	17½Y M	.04	.06	.10	.14
4	20Y F	.40	.46	.50	.42
5	19Y M	.44	.54	.28	.36
6	23½Y M	.26	.22	.30	.34
7	22Y F	.56	.50	.56	.46
8	18½Y F	.42	.34	.28	.36
9	16Y M	.10	.24	.08	.24
10	20Y F	.18	.19	.24	.12
	Mean age= 20Y	M = .2795 SD = .1695		M = .2965 SD = .1339	

Table (ix) - showing the absolute Amplitudes of wave III measured in u volts in normal hearing subjects for 100 dB HL and 80 dB HL (n=10)

Source: Uma Devi, Independent project submitted to the University of Mysore, 1983.

Key: M = Mean, SD= standard Deviation, Rt=Right, Lt.= Left

APPENDIX - B

Sl. No.	Age/ Sex	2 KHz Logan Stimuli			
		100 dB HL		80 dB HL	
		Rt.	Lt.	Rt.	Lt.
1	19Y M	.60	.70	.62	
2	22Y M	.20	.36	.40	.54.26
3	17½Y M	.68	.46	.47	.31
4	20Y F	.58	.56	.84	.70
5	19Y M	.94	.90	.72	.90
6	23½Y M	.60	.50	.48	.54
7	22Y F	.78	.54	.40	.62
8	18½Y F	.78	.56	.48	.58
9	18Y M	.34	.46	.42	.34
10	20Y F	.80	.38	.46	.36
	mean age = 20Y	M = .587 SD = .1925		M = .512 SD = .1535	

Table (x) - showing the absolute Amplitudes of wave III measured in u volts in normal hearing subjects for 100 dB HL and 80 dB HL (n = 10)

Source: Uma Devi, Independent project submitted to the University of Mysore, 1983.

Key: M = Mean, Sd= Standard Deviation, Rt.= Right, Lt.= Left.