AGE RELATED CHANGES IN AUDITORY LATE LATENCY RESPONSES

REG NO. 9683

AN INDEPENDENT PROJECT SUBMITTED AS PART FULFILMENT OF

FIRST YEAR M.Sc., (SPEECH AND HEARING) TO THE
UNIVERSITY OF MYSORE,

ALL INDIA INSTITUTE OF SPEECH AND HEARING

MYSORE - 578 886

MAY 1997

DEDICATED TO

RESEARCH

DECLARATION

I hereby declare that this Independent Project entitled **"AGE RELATED CHANGES IN AUDIOTORY LATE LATENCY RESPONSES"** is the result of my own study under the guidance of Mrs.Vanaja Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore, has not been submitted earlier to any University for any other Diploma or Degree.

Mysore
May 1997

Reg. No. **M9603**

CERTIFICATE

This is to certify that the Independent Project entitled "AGE RELATED CHANGES IN AUDIOTORY LATE LATENCY RESPONSES" is a bonafide work in part fulfillment for the First Year M.Sc, in Speech and Hearing of the student with Reg. No. M9603

Mysore
May 1997

Director
All India Institute of
Speech and Hearing
Mysore - 6

CERTIFICATE

This is to certify that the Independent Project entitled "AGE RELATED CHANGES IN AUDIOTORY LATE LATENCY RESPONSES" has been prepared under my supervision and guidance.

Mysore
May 1997

Guide 10/6/97

Mrs. Vanaja C.S
Lecturer in Audiology
AIISH, Mysore

ACKNOWLEDGMENT

First and foremost, I extend my heartfelt gratitude to my teacher and guide Mrs Vanaja. Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore for her in valuable guidance and help.

I also thank -

* Dr. S. Nikam, Director, AIISH, Mysore for allowing me to takeup this project.
* A big thanks to all the subjects who particpated in the project.
* To my family - thanks for being with me.
* To the Gals of my class Haripriya, Kanaka, Rohima, Poonam, Sonia, Suzan, Roopa, Elza, Preethi, Veena, Sindhu without your help and emotional support I couldn't have finished this project.
* To Prabha and Rajeev P. Thanks for helping me feed the extensive data.
* To Prakash - thoughts of you kept me motivated ! .
* To Maruthi Computers Shylaja, Sudhakar, Shivu and Venu without your quick and efficient service my IP would have been just a dream.

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INTRODUCTION

The history of science is marked by a slow but steady progress from unknown about the ear and has been built from bricks, laboriously fashioned over the years, even centuries. Each new finding provides an immediate solution for some problem but for others it suggests still another question. Though the tower of physiological measures available to examine the audio vestibular system is well based, it would never be considered complete. Today's edifice is the result of yesterday's efforts and is the structure on which to build tomorrows achievement.

The capacity for hearing in an individual can be tested subjectively or objectively. The test results gives us an indication as to how essential audition is as a link to the outside world. But assessment of audiological function through volumetric responses in case of children and adults with neurological, emotional and social impairment is not always possible. In such cases, the study of evoked potentials recorded from the scalp have appeared successful. The decade of the 80 's is called the era of evoked response audiometry. This is because a hoard of studies concerning evoked potentials have come up in the past 10 years.

Auditory evoked potentials have also been used to enhance the objectivity in the assessment of central processing disorders. A review of literature suggests that the auditory brainstem responses is very sensitive to eighth

never and lower brain stem lesion. However its usefulness in the assessment of the upper brain stem and cortical lesions is limited. (Watanabe, Hara, Miyazaki, Yamada, 1981). Consequently reviewed interest has shown that middle latency and late latency responses as possible ways of objectively delineating problems of the central auditory system.

While many investigators suggests that MLR has potentially valuable clinical application, it3 usefulness is hampered by the still unresolved issue surrounding origin, maturational effect as well as the influence of technical parameters in the test outcome (Musiek, Verkest and Gollegly, 1988). Partially as a consequence of these limitations, renewed interest has also focussed on potentials occurring after MLR.

Auditory late latency responses are recorded in a time period from about 50 to 250 ms after the acoustic stimulation at a relatively slow rate (one stimulus every 1 or 2 sec). Amplitude of ALR is larger usually within 3 to 10 u volts range and occasionally larger. The main components of ALR are P1 (50-80 ms), N1 (100-150 ms), P2 (150-200ms) and M2 (180-250 ms) (Hall, 1992). The labels for these peaks refer to the expected voltage polarity of the response as recorded from the vertex.

The ALR was actually the first auditorily stimulated electrical response to be recorded from central nerve system. In 1939, Pauline Davis and his colleagues, described an 'on-

response' to sound in EEG and used the term 'k-complex' to describe it (Davis, Davis, Loomis, Harvey and Hobert, 1939).

Precise anatomic generators of ALR aren't known and presumably were from the auditory cortex suggested by Knight (1988) studying the patients with lesions of superior temporal gyrus and inferior parietal lobe. The audiologists have suggested that the superior temporal gurus and lateral temporal gyrus are important to the generation of long latency auditory evoked potential at least to 200 ms (Scherg, 1989; Hari, 1990).

ALR is optimally evoked by a tone burst stimuli of relatively long duration (greater than 5 sec) with stimulus rates of 2/sec or less at an intensity level of 50-60 db (Davis and Zerlin 1966, Onishi and Davis, 1968, Antinoro, Skinner and Jones, 1967, Beagley and Knight, 1967). In contrast to shorter latency AERs subject attention to stimulus has pronounced effects as the response of ALR and also affected by drugs (sedatives) (Cody, Klass and Bickford 1967). Despite the clinical limitation of inter-intra subject variability susceptibility to state of arousal and drugs, the clinical implications of ALR is extensive. In general ALR have 2 clinical application.

1) Neurodiagnosis (adults and children)

2) Estimation of hearing sensitivity (mostly in children).

In adults, AERs are more applied for neuro diagnosis than for estimation of auditory sensitivity. The clinical application of ALR on peripheral auditory assessment is minimal since its affected by state of arousal and drugs taken. The ALR implications in CNS diseases include alcoholism, apnea, autism, CVA, coma, congenital neonatal hypotonia, Down's syndrome, freidreich ataxia, Gilles de le Tourette syndrome, head injury, Huntingtons's chorea, learning disability, mental retardation, Parkinson's disease, tumor (Hall, 1992).

However to use the late latency responses as a clinical tool, the alterations due to normal aging must be well established. This is especially important for higher level auditory functioning. Its been reported in literature that ALR have been recorded for a range of age group from neonates premature infants to geriatric population. It usually seen that ALR latency decreases and amplitude increases as a function of age during childhood upto 10 years of age, although the most prominent alterations occur within first year of life and to a lesser intent within 2 to 5 years of age (Barnet and Goodwin, 1965).

Thus there is an essential need to collect normative data across age group to adequately assess the age related changes in late latency responses.

Therefore the current study was undertaken:

1) To compare the latency, amplitude and morphology in ALR waveform at difference intensity levels.

2) To study the ALR waveform in adults and children.

3) To compare the waveforms obtained for any significance differences between the 2 groups.

REVIEW OF LITERATURE

The central nervous system generates spontaneous random neuro electric activity in the absence of sensory stimulation. These neural activity can be recorded using needle/scalp electrode. From the on going EEG activity it is possible to extract those and record those neural activity related to specific type of sensory stimulation. The recording of sensory evoked potential (SEP) is based on the assumption that there is an exact temporal relationship that exists between the sensory stimulation presented and the neural response pattern evoked.

The monitoring of spontaneous EEG activity generated from CNS and recorded from the human scalp was first described by Berger (1929). This pioneering effort was followed by the works of (Davis, Davis, Loomis, Harvey and Hobart, 1939) who first reported alterations in the human EEG pattern brought about by the introduction of sensory stimulation. This extraction of stimulus related, neuro electric events from on going EEG activity set the stage for future clinical development in various aspects of sensory evoked potential measurements. An SEP can be evoked by auditory, visual or somato sensory stimuli. An auditory evoked potential (AEP) is an activity within the auditory system that is produced or stimulated by acoustic stimuli. The major auditory evoked responses are electro cocleography (Ecochg), auditory brainstem response (ABR),

auditory middle latency response (AMLR), auditory late latency response (ALR), auditory P300 response, depending on site of origin of the wave form, and time taken for these wave forms to appear after stimulation.

The auditory late latency waveform is recorded in a time period from about 50-250ms after acoustic stimulation at a relatively slow rate (one stimulus per 1 sec or 2 sec). The amplitude of. the ALR is. large, usually 3 to 10 uv range and occasionally larger. The main components and their characteristic latency values are P1 (50-80 ms); N1 (100-150 ms); P2 (150-200 ms); N2 (180-250 ms) (Hall, 1992).

The ALR was the first auditory electrical response to be recorded from the central nervous system. The observation by Davis, Davis, Loomis, Harvey, Hobart (1939), showed that with the introduction of repeatable auditory stimuli, 3mall but consistent changes in EEG activity were recorded and the potentials were between 50 to 200 ms consistently. The response description was extended by Gastaut (1953) and Baucaud, Btoch and Paillard (1953) who suggested the term 'v-potentials' to emphasize the vertex-maximal scalp distribution. Using techniques like photographic super imposition (Abe 1954); on line-summing Devices (Davis, 1964, Goldstein, 1961) and signal averaging HAVOC (Davis, Mart, Yoshie, Zerlin, 1966) a proliferation of studies on ALR as an accurate, objective method of evaluating auditory acuity took

place, since high quality ALR could be recorded and it continues unabated till today.

ORIGIN OF ALR WAVEFORM:

The neuro anatomic origin of ALR has for many years been the object of study and debate. The lack of good animal model has been a source of difficulty and its doubtful if an animal analog exists in non-primates (Hardin and Castellucci, 1970). It was showed by Davis (1939) that ALR could be recorded from electrodes at numerous scalp location with maximum amplitude from midline electrodes over frontal region. He suspected diffuse, non specific generators in thalamo cortical regions. The association cortex of the frontal lobe was postulated as site of generation by Pilton, Hillyard, Krausz and Galambos (1974). The prime candidates for ALR generation are the post synaptic potentials of radically oriented pyramidal cells and their apical dandrites in a study conducted by Geutzfeldt and Kuhnt (1967). Results of a series of investigation of scalp topography and neuromagnetic correlation of ALRs in humans (Papanicolaou, Baumann, Rogers, Saydjari, Amparo and Eisenberg, 1990) as well as monkeys (Arezzo et al 1975) placed generators in the region of the Sylvian fissure and superior temporal plane in the temporal lobe. Evidence has been accumulated that, several concurrent sources contribute to scalp potentials in the latency region of the ALR (Wolpaw and Penry 1975).

Advances in two areas (1) dipole source analysis (Scherg and Voncramon 1985; Scherg 1990), (2) cortical evoked magnetic field (AEMF) (Hari 1990) have created a new insight into the location of ALR generators. In the dipole source analysis, the location, strength and orientation of a small number of electrical equivalent dipoles that would cause observable scalp potential distribution AEMF are associated primarily with lateral components of current flow that is, with flow tangential to the surface of the skull. Relative to AEP, they have the advantage that the induced field are spatially more restricted to scalp regions over lying the generator sites.

Laterality with respect to ALRs have always been a fundamental concern whether contra lateral advantage that is present for speech stimuli processing is also present for ALRs. Studies in this area has yielded conflicting results, including no amplitude differences between hemispheres for verbal stimuli and shorter latency values for ALRs recorded from hemisphere contralateral to the stimulus (Butler, Keidel and Spreng 1969).

What ever the source of location the exact electro physiology of neural elements that give rise to ALR is not unequivocally established.

CLINICAL UTILITY OF ALR:

The ALR potentials is of special interest for audiology because they can be used as a valued physiological indicator that the auditory nerve impulse have activated some parts of the brain (Barnet and Lodge 1966; Rapin and Graziam 1967). They found this method particularly useful for young or uncooperative children including infants. ALR applicability lies in its use as a potential diagnostic tool for neurological examination of premature infants (as in the study of Rottevel et al 1985). Barnet and Lodge (1966) reported that ALR can be elicited in the absence of ABR. Since ALR and MLR are generated at level of mesencephelon they of neuro audiological interest. Rapin and Schimmel(1977) reviewing extensive experience, concluded that ALRs are suitable to provide information about perceptual aspects of audition rather than to detect threshold, probably due to unfavourable S/N ratio Barnet et al (1978) reported persisting abnormality in ALR in infants with marasmus after treatment. Watanabe (1981) found a close correlation between a favourable out come and normal ALRs in new borns with intracranial haemorrhage.

Jerger and Jerger (1985) conducted study on patients with arteriosclerotic, cerebrovascular disease, multiple sclerosis and developmental dysphasia used click at 80 db NHL to eilicit LLR and found that in developmental dysphasia P2 couldn't be reliably discerned and in multiple sclerosis

in the LLR potentials were absent. No conclusive evidence regarding arteriosclerosis was obtained. ALR has been used as a useful diagnostic tool in difficult to test children. Study conducted by Small (1969) and Small (1971) found lower amplitude in LLR in Autistic children than normals. Autistic showed greater latency variation and the peak latencies were shorter. Longer latencies were reported in children with low IQ by Chalke (1965). Barnet & Lodge (1967) concluded after studying downs syndrome children that, they had greater amplitude than their normal peer group. Another study by Yellin, Lodwig and Jerison (1980) on Downs syndrome, used binaural tone pips of several inter stimulus intervals greaterthan is and compared between young adults with Downs syndrome (Trisomy 21) and normal young adults. Results indicated that (1) AER amplitude and latency for both experimental group increased with lengthening of ISI. (2) ALR peak latencies of DS were longer than peak latencies of normals for all ISI employed (3) The amplitude of DS group tended to be larger than that of normal adult group.

ALR applicability in cases with hearing loss has also been researched. Jerger & Jerger (1985) studied amplitude of ALR to intensity and frequency changes and compared it to behavioural performance in one normal and one SN hearing loss subject. The results indicate that behavioural function at 3 frequency 500, 1k and 4k with Cochler disturbances show steeper function than normal subject. The comparison of the ALR function and behavioural function showed that this

steepness is reflected in ALRs also. At all test frequency the function relating to increment size to AER amplitudes are steeper for subjects with cochlear loss than with normal hearing. Another study conducted by Bochenek and Bochenek (1972) studied the vertex response in normal hearing subjects and those with SN and CD hearing loss. Used 64, 1024 Hz, pure tones presented every 2 sec in 3 groups of ears. 77 with normal hearing (Group A) 28 with CD loss (Group B) and 36 with SN loss (Group C). The % of evoked responses obtained at subjective threshold was not the same. It was greatest in Group C and least in Group A. But the differences wasn't statistically significant. The latency of the N1 peak at 40 DB above the threshold was shortest in Group C (mean value at 86.9 ms) longer in group B (Mean value = 90.2 ms) and longest in Group A (100.2 ms). The amplitude (N1 -P2) of the response became larger when the duration of ISI increased. In some ears with SN loss, rapid dimunition of latency of the evoked response as the stimulus intensity was increase can be considered analogues to the loudness recruitment (Cody 1968, Knight, Beagley 1969). They concluded that ALR can be used as an objective test to detect presence or absence of peripheral hearing loss. Applicability of ALR is in assessing central auditory processing disorders have also been studied. A study conducted by Jirsa and Clontz (1990) on 24 children diagnosed as possible cases were selected from the clinical group. The age ranged from 9.2 years to 11.6 years. The results showed significant differences in LLR

potentials between children with confirmed CAD with their normal peers. A significant latency increase for N1, P2, and P3 component in CAD. The inter peak latency interval P2 - P3 was significantly longer in CAD. In terms of amplitude difference only P3 differed between the 2 groups.

FACTORS AFFECTING ALR:

Factors affecting ALR have been reviewed under 3 headings:

1) Stimulus characteristics
2) Acquisition characteristics
3) Subject characteristic

1) STIMULUS CHARACTERISTICS:

Acoustic stimuli are necessary for generation of all ALRs stimulus properties such as frequency, duration, intensity, rate and type exert profound, often interrelated ALR measurement.

a) Stimulus type:

Different studies on ALR have used different types of stimuli. Davis et al (1966) used /tone pips. Rapin et al (1966) used Clicks. Mc Candles Best found (pure tones) better than clicks. Davis, Bowers and Hirsh (1968) found better than used tonal stimuli that traditionally used to elicit ALR. (Optimal ALR stimuli have rise time (RT), fall time (FT) and plateau time of greater than 10 ms (Onishi and Davis

1968). The RT and FT of over 20 ms in even more effective in eliciting ALR (Skinner and Jones 1968

A study conducted by Spoor, Timmer, and 0' denthal (1969) to find the relationship between N1 peak latency and N1 - P2 peak to peak voltage of the evoked auditors response elicited by amplitude and frequency modulated tone bursts. Found that N1 - P2 voltage of the response grown as the intensity of the stimulus is increased, and the latency simultaneously reduced.' Lenhardt (1971) studies effect of frequency modulated tone on N1 peak latency and N1 - P2 amplitude using two kinds of stimuli i.e., low frequency, ramp with initial frequency of 500 Hz and high frequency ramp with initial frequency of 2000 hz. It was presented as 40/60 dB SL to 2 normal hearing adults. Results indicated N1 -P2 amplitude reduced as the ramp duration increased from 25 to 2000 ms. The amplitude became progressively smaller as frequency region increased from 500 Hz to 2000 hz also decreased when intensity of the stimuli was reduced from 60 dBSL to 40 DBSL. The N1 pak latency increased as ramp duration increased. The view that transition between 2 frequency activate additional units was maintained. A study on effect of phase in version of the stimuli on amplitude at 200 hz and 2000 hz conducted by Butler and Kluskens (1971). Results indicated that larger amplitude response for Sπ was significantly larger than for So when the tonal stimuli was 200 hz. Mo stastistically significant difference between Sπ and So at 2000 hz was seen. Lenhardt (1973) studied the

influence of verbal association on ALR on 18 adult subjects. Affective loading of pleasant and unpleasant and neutral were grafted on to pure tones (0.5, 1.0, 2.0, 4.0 khz) presented at 80 db PL for 500 ms. Results indicated pleasant qualities produced a greater. N1 - P2 amplitude change than unpleasant. The percentage of amplitude range for pleasant was more than unpleasant of higher frequency with 0.5 khz a reference. To study effect of word meaning on ALR, Sharrard (1973) presented 64 word messages played forward and then reversed to 8 femal subjects. Amplitude and latency was measured at N90 and P170. It was found that reversal of word message revealed a reduction of amplitude of N1 and P2 • Latency was not significantly affected.

b) Stimulus duration:

Extensive studies on the effect of duration on ALR has been studies. Davis and Zerlin (1966), Onishi and Davis (1968) conducted studies with stimulus of 1000 Hz tone burst with linear onset offset ramps. Varying RT, FT and PT produced complex effect on ALR latencies and amplitude. No change in latency (N1 or P1) and amplitude (N1 - P1) as the RT/FT duration was varied between 0-30 ms. With a relatively brief RT/FT of 3 ms and reduction of PT from 30 ms to 0 ms produced a corresponding reduction in ALR amplitude. Also found that steeper slopes for RT/FT resulted in shorter ALR latencies. A study conducted by Onishi and Davis (1968) reported that ALR latencies decreased with increased duration

especially at low stimulus levelsa and with small RT/PT. Kodera, Hink, Yamada and Ichisuzuki (1979) studied effect of linear rise times (5, 10, 20 ms) with 1000 hz tone burst at 60 DBSPL on 8 normal hearing adults between 24-32 years. Results indicated that longer rise times were associated with longer latencies and smaller amplitudes.

c) Stimulus intensity:

One of the first observations made about ALRs was that amplitude increased as stimulus intensity increased with amplitude calculated from trough of N1 to peak of P2 since its the most stable measurement (Antinoro, Skinner and Jones 1969; Beagley and Knight 1967). The amplitude increase occurs steeply with in the first 20-30 db, above the threshold and then the amplitude increase is gradual with increasing intensity levels and in some people reaching a plateau above approximately 75 db (Beagley and Knight 1967; Davi3 and Zerlin 1966; Onishi and Davis 1968).

The studies have reported that considerable variability characterises the amplitude intensity relationship, but the changes in amplitude is more regular for tones versus clicks. The amplitude increase as a function of intensity were steeper for lower frequency stimuli (500 Hz) than for HF stimuli (8000 hz) (Antinoro; Skinner; and Jones 1969). Picton et al (1977) reported that there is a non-linear increase in response amplitude ie., N1 - P1 amplitude increased rapidly just above the response threshold but then

grows more gradually for higher intensity levels and may decline at very high intensity levels. Also females show greater overall amplitude increase and a steeper slope in int-amplitude function. Larger amplitude in patients with clinical evidence of loudness recruitment than those with tone decay was reported by Shimizu (1968).

ALR latency changes with intensity have also been studied. It was found that, latency increases as stimulus intensity decreases. Also this relationship isn't entirely linear since the latency changes is greater for intensity below 45-50 DBSPL (Rapin et al 1966). He noted that there is very little change for the N1 or P2 component as click stimulus intensity increases except at intensity levels very close to the auditory threshold.

d) Inter-stimulus interval (ISI) and rate:

ALR is highly dependent on ISI (Davis et al 1966, Hari et al 1982). The duration of the stimuli used in eliciting ALR is about 50-60 ms or even longer. Consequently total accumulated duration constitutes a considerable portion of the analysis time. Also for ALR the recovery time is longer (Hall 1992). It was found by Davis et al (1966), Hari et al (1982), Rothman, Davis and Hay (1970) that, though latency doesn't change markedly, the amplitude increases as ISI is lengthened, and concomitantly the, stimulus rate is decreased. They reported that, the greater amplitude increase occurs for ISI lengthened up to 8 sec and occurs for

higher intensity levels. For ISI values greater than 4 sec yielded larger amplitude values than for lesser than 4 sec (Hari et al 1982).

e) Monaural vs Binaural stimulation:

Studies conducted by Butler, Keidel and Spreng (1961) showed that monaural acoustic stimulation produces an N1 component that is consistently shorter in latency when recorded from the hemisphere contralateral to the stimuli in comparison to the Ipsilateral recording. Pantev, Ho ke, Lutkenhoner, Lehnertz and Spittka (1986) reported that there is no binaural summation for the ALR but there is summation for its magnetic analog. The ALR amplitude is greater for binaural than monoaural stimulation (Butler et al 1969, Davis and Zerlin, 1966, Davis et al 1968).

AQUISITION CHARACTERISTICS:

a) Electrodle placement:

The studies done to determine the neural source of the ALR led to studies on electrode placement (Goff, Allison, and Vaughan 1978, Kooi, Tipson, Marshall 1971, Wood, Wolpaw 1982). The response was largest when recorded at vertex in studies conducted by Davis (1939). It was supported by Abe (1954), Cody et al (1964a) Teas (1965) that the vertex (a site within 2-3 cm lateral or anterior) was optimal. Vaughan and Ritter (1970) recorded ALR from different coronal electrode array and concluded that, there is both diminishing

response amplitude at greater distance from midline and polarity reversal in the sylvaian fissure region. Its seen that ALR can be reliably recorded with a non-inverting electrode located any where over the frontal position of the scalp of the head, especially the midline, but its usually has maximum amplitude with the vertex site (Cody, Bickford 1965; Cody and Klas 1968, Davis et al 1966). A study comparing ALR for cephalic and non-cephalic sites were done by Wood and Wolpaw (1982). It was found that no significant voltage gradients at neck or below were seen, whereas there were large voltage gradients for various electrodes location on the head. They recommended a non-cephalic reference site such as the balanced sterno vertebral point since its both inactive and minimally affected by EKG artifact.

b) Analysis time:

Studies conducted by Hall (1992) concluded that since the ALR are long latency responses, the analysis time should extend for atleast for 250 msec post stimulus. It was studied that the responses mainly were low frequency energy and it therefore a minimum time period between data points of 1 ms or even more provides adequate temporal resolution and accuracy for amplitude calculations.

c) Filters:

Filters selectively remove part of some thing from the whole. In the ALR measurements, filter reject electrical

activity at certain frequencies and pass energy at other frequencies. Studies conducted by Sayers, Beagley and Marshall (1974), Yamamoto, Sakabe and Kaiho (1979) found frequency composition of ALR in region under 30 Hz. Therefore recommended filter setting of 1 or 3 Hz to 30 or 100 Hz. And its typically employed in ALR recordings.

III) SUBJECT CHARACTERISTICS:

Non-pathologic subject characteristics are those factors that may influence the outcome of AER recording in any subject with in normal peripheral and central auditory system. They are age, gender, attention, state of arousal, drugs, sleep etc. The influence of each of these factors varies markedly among the AER.

a) Attention and state of arousal :

Psychological variables are apparently important, when delivering to the subject a lengthy monotonous set of stimuli. Vaughan and Kitter (1970) noted marked effects on response morphology can occur simply by changing from periodic to irregular stimulation sequences. Keating and Ruhnn (1971) found that ALR variability was reduced with the subject reading, in comparison with counting the stimuli or simply sitting quietly. An increase in SVP amplitude with increased stimulus oriented attention was noticed in a study by Davis '64, Picton and Hillyard 1974. They found that the threshold changes were most marked near the threshold and may

differ between peaks. The influence of sleep was recognised by researchers (William, Tepas, Horlock 1962), but complexities of the sleep effect cames to be appreciated later. In sleep, latency increased and intensity at which ALR is first observed in subjects with the normal hearing increased by 20-40 db (Cody et al, 1967). Amplitude became variable in sleep (Rapin, Schimmel and Cohen 1972, Weitzman and Kremen 1965). The amplitude of the N2 component is markedly increased during sleep (Ornitz, Ritro, Carr, Panman and Walter 1967, Picton and Hillyard 1974). A study conducted by Davis 1964, Hillyard, Hink, Schwent and Picton (1973) concluded that N1 and P2 components are larger when the subject is paying close attention to the stimulus or listening for a change in some aspect of the stimulus. The N1 amplitude increased by 50%.

b) Effect of drugs:

The influence of drugs on AER is well known. Sedation with chloral hydrate (a tranquilizer) increases the variability of ALR waveform was concluded by Skinner and Antinoro (1969). Measurement of ALR under sedation is not recommended. Lader (1977) concluded that use of diazepam results in amplitude reduction of N1, P2 and N2 with little effect on latency. Pfefferbaum et al (1979) said that the use of Droperidol, proceduses a latency prolongation of about 10 ms in P1 and N1 component with amplitude reduction. The use of phenothiazine in the treatment if schiozophrenia

produces dose dependent decrease in amplitude (Roth and Cannon 1972). Hergel and Herman (1990) found that lithium increase P1 latency and increases P1 - N1 amplitude. The effect of alcohol an ALR has also been studied. Gross, Begleith, Tobin and Kissin 91966) found that amplitude of ALR is said to be affected to a great extent by alcohol intoxication. The amplitude of the N1 - P2 complex decreased by acute alcohol consumption. In general primary sensory region are more resistant to the effects of alcohol and the association areas are more suspectible.

. c) Gender:

Onishi and Davis (1968) reported that ALR amplitude in general tended to be larger and the amplitude versus intensity function steeper for females than males. Another study conducted with infants, children, and adults to record ALR with in a background of complex verbal and non-verbal auditory stimuli in verbal and non-verbal condition revealed that females have higher amplitude response from left hemisphere than male subjects. Whereas the male showed higher amplitude response form right hemisphere than females. (Museik, Verkest and Gollegly, 1988).

d) Age:

Research has demonstrated that ALR can be recorded from both premature, full term, new born and older children (Hall 1992). A 3tudy conducted by Rotteveel, Colon, Noter mans,

Steeling and Visco (1985) on 25 mature healthy new born of 1-1-5 days with a follow up at 3 months of age concluded that ALR show an early complex within the latency reach of 100 ms and a slow 'w' shaped late complex distinct at 3 weeks. At 1-5 days the children showed a primary complex at first 150 ms after stimulation ie., "NaPbNcP1N1". At 3 months of age a separation of PbPi with Pb at 36 ms and P1 at 80 ms occurred. The 'w' shaped slow secondary complex P2N2P3N3P4 showed latency decrease between 1-5 days and 3 months.

Gibbs and Gibbs (1950) noted that a spontaneous 'cortical rythm' resembling the adult 'alpha rythm' in denotes in the waking stage at about 5-6 hz instead of at the adult 10 Hz. The infants like adults show 2 stages of sleep namely 'quite stage' with high voltage slow wave EEG activity and an 'active stage' frequency muscular movement and low voltage fast EEG activity (Barnet and Goodwin 1965). They suggested that the response to 50-100 stimuli separated by intervals of atleast one and preferably 2-3 sec must be summed and high voltage is more releable than low voltage in active stage (Barnet and Goodwin 1965).

Another study conducted by Rapin and Graziam (1967) reported that N1 wave at about 100 ms was small or flat topped or entirely absent in normal sleeping infants and the P2 - N2 complex dominates the response (as in older children). Either P2/N2 may be double peaked. Larger waves slower than N2 and more variable in form and latency often

appeared in high voltage sleep. The latency that can be measured most relinsly was found at P2 at 200 ms approx. as in adults. Onishi and Davis (1968) studied 3 normal infants between 4 to 12 months of age a sleep with out sedatin. They found that the latencies at 4 months are slightly larger but the difference in waveform amplitude and latency related stage of sleep, intensity of stimulus are much more important that the differences related to age. The V potential gradually emerges from the first vague responses at 23-29 weeks. N1 at 180-270 ms in followed by a slow P2 at 600-900 ms. At 35-37 weeks P2 at 300 ms is the most prominent wave. Next P1 and N2 appear and the normal neonate pattern is established, but by 45 weeks N1 is decreased and P2 (at about 320 ms) is the major component. They felt that the maturation of V potential pattern is almost complete few weeks after full term birth and in very nearly complete at 4 months. After 4 months, there is a slight further shortening of latency and particularly reduction is variability in wave form latency (Davis, Hirsh, Shelnut and Bower, 1967). A high voltage slow activity stage of sleep the voltage of v potentials increased considerably particularly the P2 -N2 complex. N2 often develops a secondary 'hump'. The threshold was within 10 DB of an adult listener and near threshold the latency of P2 was some what prolonged as in older children (Onishi and Davis 1968).

Another study conducted on preterm to 3 months postterm by Rotterveel, Colan, Stegeman and Visco (1987) revealed that

ALR in young infants and new born consists of fast and slow component respectively the primary and secondary complex. In primary complex Na, Pb, N_c, P1 and N1 are recognizable as low voltage peaks and troughs at 3 months post term date. In the secondary complex, the high voltage components are P2, N2, P3, N3 and P4. At term date Pb and P1 are fused and P2is bifid (P_2 and P^1 Rotteveel et al 1985).

The primary and secondary complex undergo changes in complexity and are recognizable from 25 weeks onwards. The transition period according to them starts at about 36 weeks and is very prominent at the term date. The premature wave form is characterized by N2P, P2P/ (p - premature 3tage) N2P, P3P4P, P3p, P4P, ie., this negativity (N2P) is preceeded by a low voltage shorter positivity and negativity and is followed by slow positivity. Initially, before 30 wks (CA) P2P is not more than a small hump in the descending negativity N2P which follows PbP1. At term date P2, the hall mark in the ALR, is often bifid. The transitional wave form emerges about 36 weeks CA with a bifid P2 - N2 and P3 - N3 - P4 complex. At 3 months post term date, P2 and N2 are the hall marks in the complex. Rotteveel et al (1987) concluded that, N2 shows a latency decrease from 28 ms at 28-29 weeks CA to 16 ms at 3 months. PbP1 decreases from about 100 ms to about 80 ms at term date. At 3 months post term P3 can be recognised at about 35 ms, Nc at about 50 ms, P1 at about 80 ms. N1 decreases in latency from about 140 ms in preterm period to about 100 ms at 3 months. P2P initially a low voltage small

(*CA - CHRONOLOGICAL AGE).

notch at pre term, grows into a broad high voltage component P2 at 3 months. N2P shows a latency of 200-250 ms. They felt that at term date latency is difficult to define because of the presence of P2 and P2' which often are used P3, N3 and P4 occur in the 300 to 600 ms time domain.

The amplitude values of the component of the primary complex didn't show important changes at different CA levels for N_a (-0.2 to -0.9 uv) PbP1 became more clear as the amplitude slightly increased (-0.1 uv to 0.6 uv). The amplitude of N1 fluctuates across the CA levels from -1.9 uv (at 32-33 weeks) to 0.2 uv (50-52 weeks). The amplitude of P3P4P complex increased between 25 to 30 weeks CA. Around 30 weeks P3P becomes separated from P4PP shows a gradual amplitude decrease to 3 months post term date.

A longitudinal study conducted by Barnet et al (1975) on normal children from 10 days to 3 years indicated latency changes such that P2 decreases from 230 to 150 ms; N2 from 535 to 320 ms; P3 from 785 to 635 ms. While the adult values are just under 100, 200 and 300 for N1, P2 and P3. They concluded ALR latency decrease and amplitude increases as a function of age during childhood upto 10 years of age, although the most pronounced alterations occur within first year of life and to a lesser extent within 2 to 5 years range (Barnet et al 1975, Barnet and Goodwin 1965). Another study conducted by Callaway and Halliday (1973) reported that the ALR peak to peak amplitude increases by approximately 50%

from 6 years to 15 years. They also reported that a decrease in latency for both N1oo and P175 was noted with more marked decrease for *PITS.* The Nioo latency is approximately constant from 10 years to 70 years of age where as, P175 latency increase by about 25% over that age range as reported by (Goodwin, Squiries, Henderson and Starr 1978b). In adults, the ALR amplitude decrease with age at the rate of 1 uv every 5 years (Goodwin et al 1978). Callaway 1975, Goodin et al (1978) reported age related decrease in ALR latency upto 15 years with an increase in latency for persons older than 15 years. It is also reported that P2 latencies is shorter for older subjects (average age 63 years is compared to younger subjects (average age 22 years) (Spink, Johansen and Pirsig, 1979).

In retrospect, one realises the importance of various factors affecting the ALR latency, amplitude, and waveform like type, frequency, and intensity of stimulus, filter setting, inter stimulus intervals, rate, age, gender, drugs, state of arousal etc. Therefore it is essential to obtain normative data specific to particular age groups with the parameters provided in the available software and those that will be used for clinical population.

Thus the following project aims to study the age related changes in late latency responses.

METHODOLOGY

The present project was undertaken to study the following objectives:

1) To study the latency amplitude and morphology of the LLR waveform at different intensities.

2) To obtain normative data of the LLR waveform for children and adults.

3) To compare for any significant difference in waveform between children and adult.

SUBJECT:

In total 60 subjects were taken for this study. They were divided into two groups:

Group-I - Consisted of 30 subjects (males and females) between the age range of 18 to 22 years. They were graduates and undergraduates who volunteered for the study.

Group-II - Consisted 30 subjects (males and females) between the age range of 7 to 10 years such that 10 children were selected in each range of 7-8; 8-9; 9-10 years.

The mean age range of Group I was 18.5 years and Group II was 8.5 years.

It was confirmed that their hearing was within normal limits. Other criteria for subject selection were -

* All the subjects volunteered for the experiment.
* No history of acute or any chronic ear infection, headache, tinnitus, vertigo or any other otological problems.
* No history of any medical and neurological impairment like hypertension, essential tumors, disarthria, etc.
* The subjects were alert and aware and relaxed when the electrodes were placed for the duration of the study.

EQUIPMENT:

An electrophysiological test Unit-Biologic System Corps, Navigator equipped with the LLR software version 5.44 for Model 317 evoked potentials was used.

TDH-39 earphones were used to deliver the tone burst stimuli.

TEST ENVIRONMENT:

The tests were conducted in a sound treated room.

A) PROCEDURE:

The first step in the procedure was selecting the subject. The criteria mentioned under the subject selection criteria were considered. Bilogic Corp.System - Navigator was used to collect the LLR waveforms at different intensity.

The subject was made to sit on a chair and was asked to relax.

B) INSTRUCTIONS TO THE SUBJECT:

* The subject were instructed to stay alert but relaxed throughout the recording.

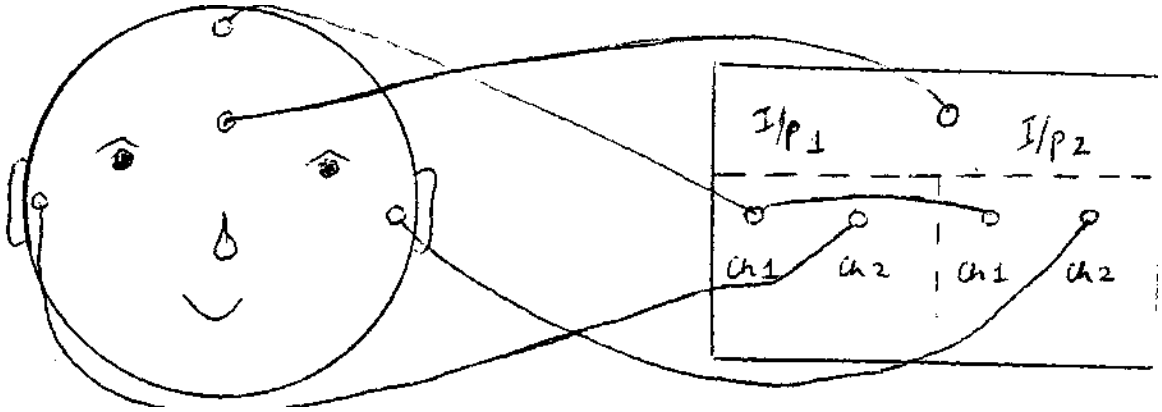
* The subjects were asked to keep their eyes open and concentrate on a spot to relax all neck and jaw muscles.

* The subject was told that he will hear a tone burst and he will have to keep alert during the presentation of the stimuli and throughout the test.

C) ELECTRODE PLACEMENT:

The area of placement of electrode was cleaned with cotton dipped in rectified spirit. The rubbing was done till the surface appeared red indicating high vascularity. Electrodes were cleaned and the required amount of gel was put on the electrodes and using a piece of plaster were placed in positions. There were four electrode which were used for LLR testing. One was placed at vertex (CZ), second on the forehead (FF2), and the 3rd and 4th on the mastoid region behind the auricle. The electrode at the vertex served as positive, one on the forehead served as common

electrode and ones on the mastoid served as negative electrodes.



The above diagram illustrates the placement of the electrodes and their connection to the electrode box. After the electrode was placed, the impedance was checked. If the impedance was within the specified limits, the earphones were placed without dislodging the electrodes (Blue-left ear; red-right ear).

D) STIMULUS PARAMETERS:

Stimulus	Alterating tone bursts
Frequency	1000 Hz
Rise/fall time	10.0
Plateu	30.0
Rate	l.1(/s)
Max. Stimuli	300
Gain	5000
Band pass filter	1.00-3.00

* *The* test procedure and the storing procedure were adopted from the software for LLR (as given in the manual).

* The test was done at 70 dB nHL, 50 dB nHL, 30 dB nHL. The response was stored for further analysis.

* Later waveforms were recalled and analyzed.

RESULTS AND DISCUSSION

LLR waveform were elicited at 70 dB nHL, 50 dB nHL and 30 dB nHL for adults and children. The following table A, B, C, D & E summaries the changes in peak latencies and amplitude at different intensities.

TABLE A (P1 PEAK LATENCY)•

Int.	Mean A	SDA	MINI.A	MAX.A	MEANc	SDc	MINI.c	MAX.c
70	61.60	10.8	50.4	89.07	76.64	10.52	50.04	87.9
50	63.87	11.37	50.98	85.56	76.46	12.94	52.15	95.92
30	65.65	11.15	43.36	96.10	62.58	330.03	55.08	97.86

TABLE B (N1 PEAK LATENCY):

Int.	Mean A	SDA	MINI.A	MAX.A	MEANc	SDc	MINI.c	MAX.c
70	97.13	16.14	71.47	141.81	113.20	22.14	69.46	176.97
50	101.58	13.95	80.28	141.81	120.71	77.94	65.63	187.01
30	113.30	15.67	89.66	154.21	93.52	21.68	76.18	127.25

TABLE C (P_2 PEAK LATENCY):

Int.	Meanx	SDA	MINI.A	MAX.A	MEANc	SDc	MINI.c	MAX.c
70	172.54	16.23	189.28	266.63	142.86	29.48	91.92	176.97
50	169.37	17.89	107.38	212.13	140.02	38.80	77.49	185.76
30	170.62	16.03	149.43	196.12	129.58	23.29	100.79	181.66

TABLE D (N2 PEAK LATENCY):

Int.	Mean	SDA	MINI.A	MAX.A	MEANc	SDc	MINI.c	MAX.c
70	225.25	19.29	189.28	266.63	225.51	22.74	159.98	286.55
50	238.21	24.51	200.41	291.83	222.53	23.32	157.05	254.32
30	242.97	30.83	168.18	284.80	213.89	24.62	169.67	267.72

TABLE E (Ni P_2 AMPLITUDE):

Int.	MeanA	SDA	MINI.A	MAX.A	MEANc	SDc	MINI.c	MAX.c
70	5.68	2.65	1.37	12.01	1.89	1.88	0.08	6.89
50	4.28	1.54	1.82	7.86	1.49	1.6	0.09	5.67
30	2.48	1.39	0.53	0.21	1.22	0.84	0.16	3.24

Table A, B, C, D & E indicate P1, N1, P_2, N_2 & N1 P_2 values at 70 dB nHL, 50 dB nHL and 30 dB nHL for adults and children respectively.

Tables A, B, C, and D indicated that for adults, the peak latency values P1 N1 P2 & N2 lengthened as the intensity decreased. This finding was in correlation with studies cited in literature (Rapin et al. 1966) However this range was not consistent in children.

Table E indicated that for adults children there was a definite consistent decease in amplitude with increase in

intensity. This was in correlation with the studies quoted in literature (Antinoro Skinner and Jones, 1969).

The mean peak latencies and amplitude at 70 dB nHL was also compared between the 7 years, 8 years, 9 years and adult groups. The following table la, lb, 2a, 2b, 3a, 3b, 4a, 4b and 5a, 5b, summarizes the results.

Table la (Peak latency P1)

	7 Years	8 Years	9 Years	Adults
Mean	77.61	72.78	68.34	61.60
SD	8.12	9.54	11.89	10.80
Min	65.63	56.26	50.04	50.04
Max	85.56	87.90	87.31	89.07

Table lb (t **Test scores)**

Group compared	t score	Probability
7 - 8	1.38	0.64
8 - 9	1.55	0.52
7 - 9	2.14	0.28
Adults	0.0001	-4.3

Table la compares the mean peak latency of P1 both for children of 7 years, 8 years, 9 years and adults elicited at 70 dBnHL (ipilateral). As seen from the table, the mean peak latency for P1 peak appeared at 77.61 ms for 7 yrs,

72.78 ms for 8 yrs, 68.34 ms for 9 yrs and 61.60 ms for adults. It indicated a definite and consistent decrease in P1 latency as the age increased. The P1 peak could be identified for all subjects at 70 dBnHL.

Table lb indicated the t scores obtained when the respective groups were compared. As seen the difference between peak latencies of adults and children was statistically significant. This was not found between the age group of children and could be attributed to the number of children used in each age group (i.e. N=10)

Table 2a (mean latency Nl)

	7 Years	8 Years	9 Years	Adults
Mean	123.99	102.19	114.37	97.13
SD	20.99	25.18	16.48	16.14
Min	88.63	64.46	81.45	71.47
Max	153.53	150.60	134.78	141.81

Table 2b (t Test scores)

Group compared	Probability	t score
7 - 8	1.43	0.60
8 - 9	12,.33	0.21
7 - 9	1..62	0.48
Adults	3.34	0.0017

Table 2a compares the mean latency for Nl for both Adults, children of 7 years, 8 years, 9 years. The mean latency for Nl falls at 123.99 ms for 7 yrs, 102.19 ms for 8 yrs, 114.37 ms for 9 yrs and 97.13 ms for adults. Again the mean latency for Nl indicated a decrease in latency with increase in age except at 8 years which indicated a latency value lesser than 9 yrs old. Nl could be identified for all subject at 70 dBnHL.

Table 2b indicated t scores for groups compared. There was no significance difference between the mean latency for Nl for 7 - 8 yrs, 8-9 yrsi, 7-9 years but there was a significant difference between that of adults and children. Again this could be attributed to the number of children used in age Group (N=10).

Table 3a (Peak latency P2 and N1P2 amplitude)

	7 Years	8 Years	9 Years	Adults
Mean	138.82	132.92	156.83	172.54
SD	19.57	30.34	15.91	16.23
Min	103.14	91.42	135.95	189.28
Max	157.05	175.80	176.97	266.63
N1P2 Amplitude (Mean)	0-97	2.19	2.42	5.68

Table 3b (t scores)

Group compared	t Scores for latency	Probability of peak latency	t score for Amplitude	Probability of amplitude
7 - 8	2.40	0.18	4.86	0.03
8 - 9	3.63	0.04	1.47	0.57
9 - 10	1.51	0.53	3.29	0.09
Adults-Children	6.03	0.00	1.51	0.00

Table 3a, compares the mean peak latency of P2 between adult and children of 7 years, 8 years and 9 years of age at 70 dBnHL.

The mean peak latencies for P2 indicated a decrease in latency between 7 to 8 years and then a consistent increase from 8 yeas to adulthood.

The mean N1P2 amplitude falling at 0.97ms for 7 years, 2.19ms for 8 years, 2.42ms for 9 years and 5.68ms for adult & children indicated a very consistent increase in amplitude of the waveform N1P2 with an increase in age.

Table (3b) indicates t- scores comparing the respective groups for any significant difference. The table indicated no significant difference between the different age groups of children except between 8-9 years that indicated a significant difference. Also the amplitude between 7-8 **years** indicated a statistical significant difference. There **was** a

definite significant difference between adults and children for P2 peak latency and N1P2 amplitude.

Table 4a (latency of N2)

	7 Years	8 Years	9 Years	Adults
Mean	223.64	222.72	230.13	225.25
SD	25.37	26.94	15.73	19.29
Min	204.14	159.98	212.72	189.28
Max	286.55	253.74	264.29	266.63

Table 4b (t - test scores)

Group compared	t score	Probability
7 - 8	1.12	0.85
8 - 9	2.93	0.09
7 - 9	2.59	0.15
Adults - Children	1.43	0.02

Table 4a compares the mean latency of N2 between 7 years, 8 years, 9 years and adults. The variation in mean latency for N2 was not consistent with age. With mean latency appearing at 223.64ms for 7 year, 22.72 ms for 8 years, 230.13 years for 9 years and 225.57 ms for adults.

The results obtained also indicated an increase in P^2 latency values from 138.82 ms in 7 years to 132.57 ms in adults which is consistent with the study reported by Goodin, Squires, Henderson and Starr (1978) that indicated an 25% increase in P2 latency with age.

Mean peak latencies obtained for adults at 61.60 ms for P1, 97.13 ms for N1, 172.54ms for P2 and 225.57ms for N2 falls well within the latency range suggested by Hall (1992) of P1 (50-80ms), N1(100-150ms), P2(150-200ms) and N2 (180-250ms). N2 waveform could be identified for all subjects at 70 dBnHL.

Table 4b indicates the t-scores obtained while comparing the respective groups for statistically significant difference. As indicated by the table, there was a statistically significant difference between adults and children which was not there between the different age groups in the children.

Table 5

Comparing Adult Males vs Females

	Mean		**SD**		**Mini**		**Max**		
	(M)	**(F)**	**(M)**	**(F)**	**(M)**	**(F)**	**(M)**	**(F)**	**Proba-bility**
P1	57.97	63.61	6.3	11.84	50.40	50.40	69.15	89.08	0.02
N1	92.37	101.01	11.69	18.10	75.59	71.49	111.30	141.81	0.11
P2	172.33	173.38	17.28	15.59	158.22	150.60	217.99	204.51	0.70
N2	244.06	253.74	23.09	14.01	189.28	219.16	266.63	261.36	0.07
N1P2	**6.11**	5.30	**2.40**	2.80	2.90	1.37	11.60	12.01	0.57

Table 5 compares the mean, standard deviation, range and t-scores for P1, N1, P_2, N2 and N1P2 between adult males, females.

The peak latencies at P1, N1, P2 and N2 were consistently longer for females than for males but the difference was not statistically significant. But the amplitude of the female population was lower than the male population, but not statistically significant.

Table 6

Comparing Children Males vs Females

	Mean		**SD**		**Mini**		**Max**		
	(M)	**(F)**	**(M)**	(F)	**(M)**	**(F)**	**(M)**	(F)	**Proba-bility**
P1	71.59	73.79	11.68	9.44	50.40	56.26	87.90	85.56	0.46
N1	112.14	107.33	34.93	25.50	11.34	76.18	150.60	153.53	0.06
P2	147.12	142.04	22.50	25.72	91.42	74.93	175.80	176.97	0.60
N_2	225.50	228.73	25.39	19.83	159.98	199.24	286.55	264.29	0.35
NlP_2	2.28	1.83	2.10	2.21	0.08	0.10	6.89	6.61	0.85

Table 6 compares the mean, SD, Range and t-test scores for P1, N1, P2, N1 and N1P2 between children males and females. Unlike the adult population, females did not show a consistent lengthening of latencies at the peak P1, N1, P2, N2. But the amplitude of the females is lesser than males in adults. But the differences at peak latency and amplitude was not significantly different.

The results obtained in this study was consistent in many respects with the few studies reported in literature. In the study conducted by Callaway & Halliday (1973). They reported of a latency decrease with age in a group of children between 6 years to 15 years. They also reported of an increase in amplitude with increase in age. This trend had been found in the present study also for all the peaks except P2.

Although LLR may be recorded in infants, complete maturation of N1 and P2 does not occur until adolescence (Davis & Onishi,1969, Goodin, Squires,Henderson & Starr 1978)

The present study also supported the above in that LLR latency values decreased while the amplitude increased with age and the LLR may not reach adult values, until the teen years. These findings are consistent with those regarding myelination which indicated that, where as some brainstem structures, may complete myelination process during the first year of life, myelination of high level structures continues throughout adolescence and early adulthood (Yakovlev and Lecours, 1967).

SUMMARY AND CONCLUSION

Auditory late latency responses are recorded in a time period from about 50 to 250 ms after the acoustic stimulation at relatively slow rate (one stimulus every 1 or 2 sec). Amplitued of ALR is larger usually within 3 to 10 mv range and ocassinally larger. The main components of ALR are P1(50-80ms) N1(100-150ms),P2(150ms-200ms) and N2(180-250 ms) (Hall, 1992).

30 adults with normal hearing between the age range of 17 years and 23 years and 30 children with normal hearing, 10 in each age group of 7-8 years, 8-9 years and 9-10 years. The aims of the study were -

1) To study the latency amplitude and mophoiogy of the LLR waveform at different intensities.

2) To obtain normative data of LLR waveform for children and adults.

3) To compare for any significant difference in waveform between children and adult.

The LLR waveform were elicited for 70 dB nHL, 50 dB nHL and 30 dB nHL using an electrophyiology unit (Biologic system Corps. Navigator). It was observed that N1, P1, N2, P2 and N1P2 could be identified for all subjects at 70 dB nHL consistently. The data were subjected to the following statistical analysis - Mean, standard diviation, Range and T-

test. The results indicated that, there was significant difference between adults and children for all the peak latency (N1, P1, N2, P2) and for amplitude (N1P2).

There was no significant difference between males and females for adults and children, though the females consistently showed longer latencies than males in adults. There was a significant difference only for N1 peak latency between 7-8 years age group, P2 peak latency between 8-9 years age group and P2 peak latency between 7-9 years age group.

In conclusion there were difinite changes in mean peak latencis and amplitude with the aging process in late latency response.

Limitation of the study:

1) Numbers of subjects used was small

2) It was difficult to keep the children alert since the test procedure was long.

3) Multiple electrods placement that could have yielded better results was not used.

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