CORTICAL EVOKED POTENTIALS IN APHASICS

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DEDICATED TO,

THE FIELD OF AUDIOLOGY

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" THE WIND BENEATH MY WINGS "

CERTIFICATE

This is to certify that this dissertation entitled **"CORTICAL EVOKED POTENTIALS IN APHASICS"** is a bonafide work in part of fulfillment for the degree of Master of Science (Speech and Hearing) of the student **(Register No.MSHM0102)**

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CERTIFICATE

This is to certify that this dissertation entitled "CORTICAL EVOKED POTENTIALS IN APHASICS" has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any diploma or degree.

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DECLARATION

This dissertation entitled "CORTICAL EVOKED POTENTIALS IN APHASICS" is the result of my own study under the guidance of **Dr. C.S. Vanaja,** Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and not been submitted in any other University for the award of any degree or diploma.

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INTRODUCTION

Aphasia is an acquired communication disorder caused by brain damage, characterized by an impairment of language modalities, speaking, listening, reading and writing, it is not result of sensory deficit, a general intellectual deficit or psychiatric disorder (Chapey and Hallowell, 2001). It has been reported that at least 60-70% of all aphasic subjects, fluent as well as nonfluent, display some degree of impairment in auditory comprehension of language (Boiler, Kim, and Mack, 1977). It is very important, therefore, to assess aphasic subject's auditory abilities. There are different ways in which one can assess the auditory abilities of an aphasic person. Conventional pure-tone audiometry and speech audiometry will yield information regarding hearing sensitivity and speech understanding. However, pure tone audiometry is mainly a test of awareness but speech recognition involves cortical function also and therefore speech recognition is subject to the effects of aphasia. Apart from this certain other behavioral tests have also been frequently employed to assess central auditory processing abilities of such subjects.

Divenyl and Robinson (1989) assessed the non-linguistic auditory capabilities through psychophysical tests in eight left-cerebral vascular accident (CVA) aphasics, four right -CVA non-aphasic subjects and eight normal male subjects. The tests included frequency discrimination, frequency sweep discrimination, and gap detection, gap discrimination, and assessment of magnitude of the frequency uncertainty effect in the detectability of tones in noise. The results of these tests were compared with the measures of auditory comprehension obtained from Boston diagnostic aphasia examination and token test. It was observed that the non-linguistic auditory performance of the three groups differed significantly from each other. For the left-CVA subjects, frequency discrimination, frequency sweep discrimination, the frequency uncertainty effect in the detectability of tones in noise were the best predictors of verbal auditory comprehension. The right -CVA subjects displayed marked deficits with regard to all pitch-related tests. The findings stressed the importance of considering the presence of nonlinguistic auditory dysfunctions when evaluating linguistic auditory capabilities in aphasia.

Musiek, Baran and Pinheiro (1994) reported a single case study of an aphasic subject following trauma. The auditory performance was assessed using pure-tone audiometry, dichotic digit, and dichotic consonant-vowel (CV) tests. The results revealed that while pure tone thresholds were within normal limits, results of dichotic CVs and dichotic digit tests demonstrated depressed scores.

The aforementioned studies or procedures used by these studies are limited in that they are dependent upon some type of response, which the subject may or may not be capable of performing. Although behavioral data have provided useful information about non-linguistic auditory capabilities of aphasic subjects. Objective techniques using cortical evoked potentials such as late latency response (LLR), P300 and mismatch negativity (MMN) might more directly assess neurological functioning without relying solely on behavioral output measures and tell us how well the person is processing auditory information. A negativity, instead of the positive P300 observed in controls, was reported in aphasic subjects from leads corresponding to the affected hemisphere (Onofrj, Fulgente, Malatesta, Locatelli and Comi, 1995). MMN was found to be present in 89% of normal subjects and 79% of aphasic subjects to tone stimuli while for speech stimuli MMN was present in 100% of normal subjects and 54% of aphasic subjects (Wertz et al., 1998). It has been reported that the presence of MMN was related to good auditory comprehension and absence of the MMN was associated with poor auditory comprehension. Poor comprehension and absence of MMN were related to lesions in the temporal lobe. Good comprehension and presence of a MMN response were related to lesions that spared the temporal lobe (Auther, Wertz, Miller and Kirshner, 2000).

Selinger, Prescott and Shucard, (1989) reported that electrophysiological measures indicated that the aphasic subject responded differently from the normal group particularly when the task required processing of verbal information. The pattern of electrical asymmetry in the aphasic group seems to be an indicator of severity as measured by traditional aphasia examination.

Thus, these studies indicate that there is a need for using a test battery approach comprising of behavioral (non-audiological and audiological) and electrophysiological measures to assess the auditory abilities of aphasic subjects.

AIM OF THE STUDY

The present study was designed to study cortical evoked potentials (LLR, P300, and MMN) in aphasics.

NEED OF THE STUDY

The following points justify the need for the study:

- There have been reports in literature regarding studies carried out on the aphasics using objective tools to assess the auditory comprehension abilities. This study had been taken to delineate the pattern of linguistic deficits and performance of adult aphasics on objective audiological tools. The widely used linguistic tools provide information about the linguistic assets of the aphasics subjects and diagnostic tools helps in localizing the lesions. They are the tools, which are subjective in nature; the objective audiological tools may enhance our knowledge about functional organization of the brain. The combined use of linguistic and objective audiological techniques may provide an in-depth understanding of auditory comprehension of aphasics.

- There is general agreement that the sense of hearing is fundamental to the development and promotion of normal speech and language. It would follow, therefore that hearing assessment of an individual with aphasia is an essential component of overall communication management and should be conducted prior to the initiation of clinical treatment.

- Conventional pure-tone audiometry and speech audiometry will yield information regarding hearing sensitivity and speech understanding. However, speech recognition involves cortical function also and is therefore subject to the effects of aphasia. Thus using audio logical tests, attempt can be made to differentiate peripheral and central hearing loss, which can be of immense help in rehabilitation of these subjects.

- There is ample evidence indicating that cortical lesions often result in diminished non-verbal auditory perception as well. A generic auditory dysfunction may parallel or even underlie to some degree, linguistic auditory comprehension dysfunctions as well as fluency of language (Divenyl and Robinson, 1989). Thus having knowledge about auditory processing abilities of a person will help in planing out the rehabilitation of an aphasic subject.

REVIEW OF LITERATURE

There is ample evidence indicating that cortical lesions often result in diminished non-verbal auditory perception as well. A generic auditory dysfunction may parallel or even underlie to some degree, linguistic auditory comprehension dysfunction's as well as fluency of language (Divenyl and Robinson, 1989). It is important to assess such subject's auditory abilities as having knowledge about auditory processing abilities of a person, will help in planning out the rehabilitation of an aphasic subject accordingly. Both behavioral and objective audiological tools can assess auditory abilities of such subjects. Various audiological behavioral and electrophysiological tests can be used to study the central auditory processing abilities of aphasics.

Behavioral Tests

Shanks and Ryan (1976) administered a dichotic consonant-vowel (CV)-syllable listening task to a group of eleven non-brain-injured adults and to a group of eleven adult aphasics. The results of this revealed that the group of non-brain-injured adults showed a slight right ear advantage for dichotically presented CV-syllables. In comparison with the control group, the aphasic group showed a bilateral deficit in response to the dichotic CV-syllables, superimposed on a non-significant right ear advantage. The aphasic group demonstrated a great deal of intersubject variability on the dichotic task with six aphasics showing a right ear preference for the stimuli. The non-brain-injured subjects performed more homogeneously on the task. The two subgroups of aphasics, group with right ear advantage and group with left ear advantage, performed significantly different on the dichotic listening task. These results were analyzed in terms of a functional model of auditory processing. In view of this model, the bilateral deficit in dichotic performance of the aphasic group was accounted for by the presence of a lesion within the dominant left hemisphere, where the speech signals from both ears converge for final processing. The right ear advantage shown by one aphasic subgroup was explained by a lesion interfering with the corpus colossal pathways from the left hemisphere; the left ear advantage observed within the other subgroup was explained by a lesion in the area of the auditory processor of the left hemisphere.

Niccum, Rubens and Seines (1983) carried out a study to identify relations among dichotic listening performance, specific language functions and Computerized topography (CT) scan evidence of lesion location in order to determine whether the nature of these relations would support either "dominance effect" or "lesion effect" interpretations of the dichotic scores. Twenty-five aphasic subjects completed digit dichotic listening test, the Boston Diagnostic Aphasia Examination (BDAE), an experimental sentence comprehension test, and a digit immediate memory span test. Right ear scores and the ear advantage accurately reflected the integrity of the central auditory structures for all but one of the subjects, and thus was interpreted as "lesion effects." These same right ear scores also were related more strongly to performance on phrase repetition subtests of the BDAE and to scores obtained on sentence comprehension test than to the other language measures analyzed. Both "dominance" and "lesion" effect interpretations were discussed, but the "lesion effect" interpretation more adequately characterized the data. That is, the integrity of the posterior, superior temporal area seemed to be essential for perception of right ear stimuli on the digit dichotic test and for accurate performance on specific language tests.

Moore and Papanicolaou (1988) investigated the role of the right hemisphere in recovery from aphasia following left-hemisphere stroke using a dichotic-listening procedure. Thirty-one stroke subjects were divided into three groups: (a) subjects who were recovering from aphasia (aphasic group, n = 11), (b) subjects who had experienced mild strokes with only transient dysarthria (dysarthric group, n = 10), and (c) subjects who had sustained right-hemisphere stroke with no language disturbance (nonaphasic group, n = 10). In addition, a group of normal, healthy volunteers served as a control group (n = 11). Results show that, like the control subjects, the dysarthrics and nonaphasics showed a strong right-ear advantage (REA) for dichotically presented CV syllables. This is usually thought to be an indication of left-hemisphere dominance. By contrast, the aphasic group showed left-ear advantage (LEA) suggesting a shift in cerebral dominance for language. The possibility that the results were due to sensory degradation of the auditory messages (lesion effect) was also explored. This idea was rejected in favour of an explanation based on increased right-hemisphere mediation of language following left-hemisphere aphasiogenic lesions.

Hugdahl, Wester and Asbjornsen (1990) reported dichotic listening performance to CV syllables in an aphasic right-handed male subject. The subject was tested three times with dichotic listening; one week, five weeks and 6 months after the hemorrhage. During the first test, he could not speak, but understood well. During the second test he uttered one-syllable words, and could answer "yes" and "no" to questions. During the third test, he could speak whole sentences, although slow and "stutter-like." Dichotic listening performance showed an almost perfect match with speech recovery. During the first test he showed a left ear advantage which changed to no ear advantage during the second test, and to a right ear advantage during the third test. The overall performance was markedly reduced during the first test, but improved during the following test.

Thus, a review of literature indicates that dichotic listening may be a valid complement to traditional language assessment procedures after unilateral brain lesions. However, these behavioral tests are limited in that they are dependent upon some type of response which subject may or may not be capable of performing. Evoked potentials might more directly assess neurological functioning without relying solely on behavioral output measures.

Electrophysiological Tests/Evoked potentials Tests

Evoked potentials refers to a series of electrical changes occurring in the peripheral and central nervous system, usually related to the sensory pathways (McPherson and Starr, 1993, cited in McPherson, 1996). Depending on the sensory system that is being stimulated, the evoked potentials may be referred to as auditory evoked potential (AEP), visual evoked potential (VEP) or somatosensory evoked potential (SSEP). Each of these sensory evoked potentials may be further categorized according to specific use. For example, the auditory evoked potentials can be further categorized as brainstem auditory evoked potential, the middle latency auditory evoked potential and cortical evoked potential (McPherson, 1996).

The cortical evoked potential are low voltage (microvolt), discrete electrical potentials occurring in the electroencephalogram (EEG) to a time locked sensory stimulus (McPherson, 1996). These cortical evoked potentials can be broadly categorized into LLR, P300, Mismatch Negativity (MMN), N400.

LLR

The long latency auditory evoked potentials are characterized by components comprising the time domain of 50to 500 msec (McPherson and Starr, 1993, cited in McPherson, 1996) and are labeled according to their polarity and latency at the vertex (Picton, Wood and Proulx, 1978, cited in McPherson, 1996). Major components in the long latency auditory evoked potentials include a positive component at about 60-80 msec having an amplitude of about 7 microvolts, a negative component at about 100-120msec with an amplitude of about 10 microvolts, a positive component at 160-180 msec with an amplitude of 6 microvolts, and a negative component at 200-250msec having an amplitude of 6 microvolts (McPherson and Starr, 1993, cited in McPherson, 1996).

Neurophysiological Basis of LLR

Responses at about 60 msec (P60 -N100) are probably late thalamic projections into auditory cortex and part of the specific sensory system (McPherson, 1996). The majority of research suggests that the P1 60 is generated in the primary auditory cortex with in the sylvian fissure contralateral to the side of stimulation (Makela and Hari, 1990, cited in McPherson, 1996). Scherg, Vajsar and Picton (1989, cited in McPherson, 1996), suggested that the N200 has its origins in the supratemporal gyrus. McCarthy et al., 1989(cited in Tonnquist-Uhlen, 1996) reported that frontal lobe could also be generator for N2.

Clinical applications of LLR

LLR has been increasingly used in studying the specific auditory perceptual skills, measure of cerebral dysfunction in head trauma subjects, evaluation of language and speech motor disorders.

Selinger et al. (1989) examined the relationship between an auditory-event related potential probe technique measure of differential hemispheric processing and traditional tests of aphasia in aphasic subjects. Ten aphasic and ten normal subjects were taken for the study. They were given a verbal and a music task Electrophysiological measures indicated that the aphasic subjects responded differently from the normal group, particularly when the task required processing of verbal information. During the verbal task, the aphasic group showed higher amplitude for right hemispheric responses as compared to left i.e eight out of ten aphasic subjects showed higher amplitude for right than left hemisphere responses for P2 during the verbal task, seven out of ten exhibited same patterns of asymmetry for N2. The normal group showed little hemispheric task related asymmetries. It was concluded that the pattern of electrical asymmetry in the aphasic group seems to be an indicator of severity as measured by traditional aphasia examination.

There is dearth for literature on LLR in aphasics. Investigations carried out indicate that LLR can be used to assess the specific auditory perceptual skills and as a measure of cerebral dysfunction in head trauma subjects .

P300

P300 is a component of human auditory evoked potential that is not affected by the physical parameters of the eliciting stimuli. This component rather appears to reflect the cognitive processing of stimulus information on the part of the subject. Literature has evidenced the importance of the task relevance to P300 elicitation. So an "oddball paradigm" is generally used to elicit this response, in which, a deviant or rarely occurring stimulus is presented in a series of frequent or standard stimuli. In this procedure, comparisons are made between the averaged responses of standard and deviant stimuli. Investigators have reported the use of active discrimination (Kilney, 1991, Saravanan, 1997) or signal detection paradigms (Paul and Sutton, 1972) or passive paradigms, in which no active behavioral response (Polich, 1989, Kiruthika , 2001) is required to elicit P300.

Neurophysiological Basis of P300

The P300 originates from non-specific, unknown neural generators and is felt to be an electrophysio logical manifestation of strategies used by the central nervous system (CNS) in selective attention activities, including frontal cortex (Courchesene, 1978), auditory cortex of superior temporal lobe (Kilney and Robertson, 1985),hippocampus and associated brain sites (Okada, Kaufman and Williamson, 1983), Comparison of recording obtained from implanted electrodes and surface electrodes revealed that P300 was generated from sequential generators for the N_2-P_3-N_4 subcomponents, with N_2 and N_4 from the non-specific sensory system and P_3 from the specific auditory system. Consequently, it is proposed that there is a sequentially non-specific - specific-nonspecific (N_2-P_3-N_4) activation of overlying generators for the P300. Although the precise generators are still not fully resolved, there is evidence of a subthalmic and medial geniculate origin with other activity noted in the gyrus orbitalis, rostral thalamus and anterior commissure. In a very simplistic manner, one could say that P300 includes responses from frontal cortex, centroparietal cortex and hippocampus (McPherson, 1996)

Clinical applications of P300

P300 has been used by audiologists and other professionals in varied areas such as identifying defective cognitive function, evaluation of specific auditory perceptual skills, measure of cerebral dysfunction in head trauma subjects, evaluation of language and speech motor disorders and in detecting dichotic deficits.

Onofrj et al. (1995) recorded P300 in six subjects with lesions of frontal, parietal, and temporal lobe, in four subjects affected by primary progressive aphasia (PPA) and in

56 age matched controls with the linked earlobe reference (LER) and with a computer calculated average reference (AR), excluding the two linked earlobe derivations. The results indicated that latencies, amplitudes and scalp distribution of the earlier components (P_1, N_1, P_2, and N_2) were within normal limits for both linked earlobe reference and average reference recordings. P3 scalp distribution in subjects was normal when linked earlobe reference was used, with the exception of two subjects affected by bilateral medial temporal lobe lesions. When P3 was recorded using average reference, the scalp distribution was statistically different from normal distributions in all subjects. Negativity, instead of the positive P3 observed in controls, was recorded in subjects. It was suggested that this finding might have clinical applications.

Yingling, Hosobuchi and Harrington (1990) applied a passive P300 response paradigm in prediction of recovery from coma. They concluded that P300 can be used to assess cognitive function of unconscious subjects and ultimately contribute to prognosis of communicative and cognitive outcome.

Thus, a review of literature indicates that P300can be used to assess the cognitive function, specific auditory perceptual skills and in planing out rehabilitation of an aphasic subject.

N400

A negative potential that has been associated with language and occurs about 400msec following the completion of a sentence in which an ambiguous construct occurs characterizes N400 potential. It has an amplitude of about 6 μV and a width of about 80-100msec. A similar response seen at 500 msec, is a positive going wave and occurs when last word in the sentence is a different word (not just contextually but literally) than the preceding word. This later response has an amplitude of about 20 μV and a width of about 80-100 msec (McPherson, 1996).

Neurophysiological Basis of N400

The generator sources of N400 are not known but the response is highly related to decision making processes and higher level processing task (Fischler, Bloom, Childers, Roucer and Perry, 1983). The N400 has been used to study language and linguistic features of speech. Wood, Knight and Neveille, (1984) suggested that such potentials are a series of multiple endogenous sources.

Clinical applications of N400

N400 has been increasingly used in studying the language processing in normals and disordered population such as specific language impairment, learning disabled and aphasic population.

Keurs, Brown and Hagoort (2002) recorded cortical evoked potentials from the scalp while Broca's subjects and non-aphasic control subjects read open- and closed-class words that appeared one at a time on a computer screen. Separate waveforms were computed for open- and closed-class words. The non-aphasic control subjects showed a modulation of an early left anterior negativity in the 210-325ms as a function of vocabulary class and a late left anterior negative shift to closed-class words in the 400-700ms epoch. An N400 effect was present in both control subjects and Broca's subjects. Early electrophysiological differences were considered to reflect the first availability of word-category information from the mental lexicon. The late differences can be related to post-lexical processing. In contrast to the control subjects, the Broca's subjects showed no early vocabulary class effect and no late anterior shift to closed-class words. The results support the view that an incomplete and/or delayed availability of word-class information might be an important factor in Broca's agrammatic comprehension.

Cobianch and Giaquinto (2000) studied the utility of cortical evoked potentials in studying language processing in subjects suffering from aphasia. Three kinds of potentials were obtained over time, P300, P340 and N400 in response to spoken words in

Lexical-semantic processing impairments in aphasic subjects with left hemisphere lesions and non-aphasic subjects with right hemisphere lesions were investigated by Hagoort, Brown and Swaab (1996) by recording N400 while subjects listened to auditorily presented word pairs. The word pairs consisted of unrelated words, or words that were related in meaning. The related words were either associatively related, e.g. "bread-butter", or were members of the same semantic category without being associatively related, e.g. "church-villa". The latter relationships are assumed to be more distant than the former ones. In elderly control subjects, the N400 amplitude to associatively and semantically related word targets was reduced relative to the N400 elicited by unrelated targets. Compared with this normal N400 effect, a varying pattern was seen in different subject groups. Aphasic subjects with only minor comprehension deficits (high comprehenders) showed N400 effects of a similar size as the control subjects. In aphasic subjects with more severe comprehension deficits (low comprehenders) a clear reduction in the N400 effects was obtained, both for the associative and the semantic word pairs. The subjects with right hemisphere lesions showed a normal N400 effect for the associatively related targets, but a trend towards a reduced N400 effect for the semantically related word pairs. A dissociation between the N400 results in the word pair paradigm and P300 results in a classical tone oddball task indicated that the N400 effects were not an aspecific consequence of brain lesion, but were related to the nature of the language comprehension impairment. It was concluded that comprehension deficits in the aphasic subjects are due to impairment in integrating individual word meanings into an overall meaning representation. Right hemisphere subjects are more specifically impaired in the processing of semantically more distant relationships, suggesting the involvement of the right hemisphere in semantically coarse coding.

Thus, a review of literature indicates that N400 is increasingly used in studying the language processing and comprehension deficits of aphasic subjects.

Mismatch Negativity (MMN)

Originally discovered in 1975 by Naatanen and colleagues, the MMN is an electrical brain response, a negative component, elicited by infrequent changes (i.e.

two young subjects suffering from a recent aphasia in whom expressive deficits were particularly marked. Recordings were made on three occasions for each subject at the same time of the day. The second recording session took place after six months and the final one after one year. Eighteen right-handed subjects in normal health (10 women, 8 men) were enrolled as controls. Results indicated that P300 was useful to monitor attention and discrimination and it improved over time. Both of the subjects lacked a defined P340 potential, but longitudinal recordings could show a very slow reappearance over time although with a frequently inverted polarity. Apparently, the subjects with expressive aphasia did not improve over a one-year interval. In contrast to P340, N400 was relatively preserved. This potential is linked to semantic judgement. The bilateral representation indicated that the right hemisphere was involved in some kind of language processing. It was concluded that apparently, long latency auditory evoked potentials enable us to study receptive processes in subjects with expressive aphasia. Moreover, they appear to be particularly suitable for monitoring the recovery of neural mechanisms responsible for language.

Kitade, Enai, Sei and Morita (1999) investigated N400 component in aphasic subjects with some impairments of language processing. Meaningful and meaningless words in Kana (Japanese characters) were used as stimuli under a visual oddball paradigm. Increases in N400 latency and amplitude in the aphasic group were significant in comparison with the control group. In the aphasic group, N400 latency correlated significantly with the performance intelligence quotient employed besides language quotients. Moreover, the N400 effects were seen more clearly in the left hemisphere than in the right hemisphere for both groups. It was proposed that the abnormal variations in amplitude or latency of N400 in the aphasic group reflect language processing functions (controlled processing and automatic processing) that are different between slight and severe types of aphasia. Moreover, N400 effects are sensitive to intellectual abilities besides language ability. It was concluded that N400 effects in the left hemisphere, for the aphasic group, are a reflection of active language processing as the substitution function.

deviant stimuli) in a sequence of a repetitive (i.e. standard) auditory stimulus or features of the auditory stimulus, usually peaking 100 to 300 msec following stimulus onset, it may be seen as an enlarged N1, a second negative peak, or an attenuation of the P2 wave (Picton, 1990). It is a potential, evoked in a passive condition, by a deviant stimuli presented within a series of standard stimuli and hence reflects a process specific to stimulus change . As MMN reflects auditory processing of very fine stimulus differences, it may provide a useful means for assessing neurophysiologic mechanisms involved in the auditory perception (Kraus, McGee, Carrel and Sharma, 1995), both in normals and disordered population.

Neurophysiological Basis of MMN

MMN generators are localized on the basis of scalp-distribution, magnetoencephalographic (MEG), intracranial and brain-lesion data. Various studies on MMN indicates that a major MMN source is located in the auditory cortex (Alho, 1995). However, the exact location of this MMN generator appears to depend on which feature of sound is changed (e.g., frequency, intensity, or duration), as well as on the complexity of sound (e.g., a simple tone vs complex sound).Moreover, recordings of frequency-change MMN from the cat auditory cortex have shown that in addition to primary auditory area, AI, an MMN also is observed in recordings from the area All (Csepe, Karmes and Molnar, 1987). However, Kraus, McGee, Littman, Nicol and King, (1994) observed no MMN in responses of the primary auditory cortex of the guinea pig, where as MMN to changes in tone frequency and phonetic stimuli occurred in responses apparently generated in the non-primary auditory cortex. MMN recordings also have provided evidence that simple and complex sounds are processed or represented by different neuronal populations in auditory cortex. For example, the MMN to change in a phoneme was abolished in aphasic subjects with temporal-parietal lesions, while a prominent MMN was elicited in the same subjects by frequency change in a simple tone (Aaltonnen, Tuomainen, Laine and Niemi, 1993). There is some evidence for contribution of frontal-lobe activity to the MMN, which might be related to the involuntary switching of attention to a change in the acoustic environment. In addition, intracranial MMN recordings in animals suggest that at least in some species, MMN subcomponents may be generated in the thalamus and hippocampus (Alho, 1995).

Clinical applications of MMN

Despite its rather recent discovery, the MMN already holds a number of promising applications. These applications might be divided into four main categories. The MMN might be an indictor of a) functional state of cortex b) sensory and perceptual abilities c) pathology of automatic processing d) neural plasticity. Studies of MMN in subjects with cochlear implants (Kraus et al, 1995, Ponton and Don, 1995), central auditory processing disorders (Alho, Sainio, Sajaniemi, Reinikainen, and Naatanen, 1990), objective diagnosis of aphasic subjects (Aaltonen et al., 1993), language disorders (Korpilahti and Lang, 1994) and learning disorders (Kraus and McGee, 1994) provide an insight into the processes underlying these disorders.

Auther et al. (2000) evaluated MMN response to speech stimuli in aphasic adults to determine the relationships among aphasic subjects auditory comprehension, site of lesion and the presence or absence of MMN. It was observed that the MMN response was significantly related to auditory comprehension. MMN was present in 89% of the aphasic's subjects with good auditory comprehension and in 25% of the aphasic subjects with poor auditory comprehension. The presence of MMN was related to good auditory comprehension and absence of the MMN was associated with poor auditory comprehension. Poor comprehension and absence of MMN were related to lesions in the temporal lobe. Good comprehension and presence of a MMN response were related to lesions that spared the temporal lobe

Iilvonen et al. (2001) studied the ability of left-hemisphere stroke subjects (n = 8; aged 42-62 yrs) with Wernicke's type of aphasia as well as healthy control Ss (n = 8) to process sounds preattentively and attentively by recording MMN and behavioral responses. The results showed that for the right-ear stimulation, the mismatch negativity (MMN) was significantly smaller in the subjects than controls over both hemispheres. For the left-ear stimuli, the MMN was significantly smaller in the subject group than in the control group over the left hemisphere, whereas no group differences were obtained over the right hemisphere. In addition, the Nl amplitude was reduced bilaterally for the right-ear stimulation (with the reduction being larger over the left hemisphere), whereas no significant effects on the Nl amplitude were found for the left-ear stimulation. Behaviorally, the subjects detected significantly fewer deviant tones than did the controls irrespective of the stimulated ear. It was concluded that the long-latency auditory evoked potentials could be used to probe auditory processing deficits that are difficult to define with behavioral measures. Especially by recording MMN to monaural stimuli, the discrimination accuracy can be separately determined for the left and right temporal lobe.

Wertz, et al. (1998) evaluated MMN in nine normals and twenty-four aphasic adults to tone and speech stimuli to determine aphasic person auditory discrimination and studied the relationship of MMN measures with the severity of aphasia. It was found that while MMN was present in 89% of normal subjects and 79% of aphasic subjects to tone stimuli, for speech stimuli MMN was present in 100% of normal subjects and 54% of aphasic subjects. The duration of MMN was significantly related to severity of aphasia as rated by the Western Aphasia Battery, Porch Index of Communicative Ability, and the token Test. It was concluded that not all-aphasic people show an early, preconscious orientation response to tone and speech stimuli

Aaltonen et al, (1993) recorded MMN from four subjects (two aphasic subjects with anterior lesion and two with posterior lesion) in order to study if discrimination of synthetic vowels is impaired by left posterior brain damage. The stimulus material included both speech (vowels) and non-speech (sinusoidal tones) sounds. The results showed that while MMN to synthetic vowels disappeared after left tempro-parietal lesion, all the subjects, including those with posterior lesions had MMN for sine waves, suggesting that vowels and pure tones are atleast partly processed in different cortical areas.

Csepe, Osman-Sagi, Molnar and Gosy (2001) compared MMN recorded in four aphasic subjects with that of age, gender and education matched controls. The MMN changes elicited by tone, vowel, voicing stop consonant and place-of articulation contrasts were recorded over both hemispheres. The results of MMN amplitude, latency and distribution differences between aphasics and controls were analyzed in detail. An extensive neuropsychological investigation was performed in order to highlight the assumed dissociation and possible interactions between the impaired acoustic/phonetic perception and deficient comprehension in aphasic subjects. The principal finding was that MMN elicited by pitch deviations is not sensitive enough to distinguish between subjects and age-matched controls. The MMN elicited by consonant contrasts was found to be the most vulnerable in aphasic subjects investigated. The MMN elicited by voicing ([ba:] vs. [pa:]) and place-of-articulation ([ba:] vs. [ga:]) could be characterized by total lack, distorted or very limited distribution and correlated with the subjects' performance shown in the behavioral phoneme discrimination task. The magnitude of the deficient phoneme (vowel and consonant contrasts) processing shown by MMN anomalies was proportionally related to the non-word discrimination and did not interact with the word discrimination performance. It was concluded that the impact of deficient speech sound processing on higher level processes may depend on the type of aphasia, while the presence of perceptual deficits in processing acoustic/phonetic contrasts seems to be independent of the type of aphasia.

MMN for duration discrimination and Speech perception

MMN is a powerful tool for understanding neural mechanisms underlying speech perception for the following reasons: (1) It is the neurophysiologic reflection of just perceptible acoustic differences. (2) It reflects the representation of dynamic properties of speech signal-the inherently changing sound structure of speech. (3) It reflects dynamic neural properties of brain. (4) With respect to more long-term dynamic processes it is modifiable with learning and experience over time. (5) Importantly MMN reflects the preconscious stimulus processing-it can be distinguished from attention / motivation / cognitive factors. (Kraus and Cheour, 2000)

Adequate processing of temporal information such as duration has been found to play an important in auditory perception. However a review of literature indicates that a majority of studies using MMN have been carried out using frequency, intensity deviance or speech stimuli and very few studies investigated MMN for duration deviance. Joutsiniemi et al. (1998) studied amplitude and latency of duration MMN in 40 healthy subjects in the range of nine to eighty -four years. Four thousand stimuli were presented at 80dB SPL binaurally through headphones. The standard stimuli were 75 msec, 700Hz sinusoidal tones with an interstimulus interval of 300 msec. Two types of deviant stimuli were presented in the same block randomly (50 msec and 25msec, 700 Hz pips) both with 6% probability. The results indicated that 25 msec deviant tone evoked a clear response in 39 subjects while 50 msec deviant tone evoked an observable MMN in only 32 subjects. Also the MMN peak amplitude for 25 msec deviants was significantly larger than for 50 msec deviants. There was no significant difference in peak latency.

Srividya (2002) investigated the minimum duration deviance needed to elicit MMN in normal hearing adults. The latency, amplitude and duration of MMN were compared for 10, 5 and 3msec duration deviance in normal hearing adults and children as well as in normal hearing children and in children with learning disorder. Sixteen adults, sixteen children and six children with learning disorder served as subjects. Auditory "odd ball" paradigm was used to record MMN for duration for both the groups. The results indicated that in normal hearing adults, the minimum duration deviance needed to elicit MMN is 2 msec. For all the deviancies, peak latency of MMN is significantly greater in children when compared to that of adults while children with learning disorder had abnormal MMN for duration, indicating some difficulty in processing subtle changes in duration.

The aforementioned studies indicate that MMN can be used to probe auditory processing deficits that are difficult to define with behavioral measures and that MMN could be a powerful tool for understanding neural mechanisms underlying auditory perception.

Thus, a review of literature indicates that cortical evoked potentials such as LLR, P300, MMN enable us to study auditory processing abilities of aphasic subjects which are difficult to define with behavioral measures and provide an in-depth understanding of auditory comprehension of aphasics. Moreover, they also appear to be useful means for assessing neurophysiologic mechanisms involved in auditory perception and thus can be particularly suitable for monitoring the recovery of neural mechanisms responsible for language. Hence the study was planned in this direction.

V

METHOD

I. Subjects

The study consisted of two groups of subjects, experimental group and control group.

Experimental Group: The experimental group consists of five aphasics. The patients were diagnosed on the basis of neurological finding obtained from the neurologists as well on basis of results of WAB administered at the department of speech-language pathology.

The following criteria was taken into consideration while selecting the clients:

- All subjects should be diagnosed as aphasics by a speech language pathologists and /or neurologists.
  Post onset period of greater than 60 days.
- Age range of fifteen to seventy years.

Control group: The control group included five non-aphasics matched for hearing acuity, age, gender, language and handedness with the subjects in experimental group.

II. Tools for the study

The following tools were used in the study.

1. A calibrated audiometer.
2. A calibrated immittance meter
3. Biologic Navigator evoked potential system with EP 317 software(Bio-logic Inc) to generate stimuli and record P300 and MMN

III*. *Test procedure

Pure Tone thresholds were obtained from all the subjects using modified Hughson-Westlake procedure (Carhart and Jerger, 1959) to assess the type and degree of loss. Immittance screening to rule out middle ear pathology followed this. If the subjects could not follow the instructions or showed inconsistent responses, auditory brainstem response was carried out to estimate the threshold.

P300, LLR and MMN recording: Subjects were seated comfortably and were instructed not to pay any attention to the stimuli.

a) Setting up the instrument -The parameters, which were used for recording, are shown in Table1 and Table2.

Tablel- Protocol used for P300 recording

Type of stimulus	Tone- Burst
Polarity	Rarefaction
Intensity and Frequency	Infrequent stimuli:2000Hz at 80dBnHL Frequent Stimuli:500Hz at 80dBnHL
No. of stimuli	500 stimuli were presented of which 80% was frequent stimuli and 20% was randomized infrequent stimuli
Transducer	Headphone
Presentation	Monaural
Mode	Air Conduction
Rate	0.9/sec
Analysis time	500 msec
Gain	50,000
Filters	l-30Hz

Table2- Protocol used for MMN recording

Type	Tone Bursts
Polarity	Alternating
Frequency	1000Hz
Intensity	60dBnHL
Rate	3.1/sec
Number	500 stimuli were presented of which 80% was frequent stimuli and 20% was randomized infrequent stimuli.
Duration Standard stimuli Deviant stimuli	 50 msec 55 msec
Transducer	Headphone
Presentation	Monaural
Mode	Air Conduction
Analysis time	500 msec
Gain	50,000
Filter setting	0.1-30 Hz

b) Preparing the subjects

(i) Electrode placement - Electrode sites for two channel recording were selected. For P300, the non-inverting electrodes were connected to upper forehead (Fz) and parietal (Pz) with common at non-test ear mastoid. The inverting electrode was connected to mastoid of test ear. For MMN, the non-inverting electrodes were connected to vertex

(Cz) and parietal (Pz) with common at lower forehead (Fpz). The inverting electrode was connected to tip of the nose.

The electrode sites were prepared before placing the electrodes by cleaning with an abrasive skin preparing paste. Each electrode was dabbed with a paste or a gel and taped to the site securely with a plaster. It was ensured that the absolute impedance of each electrode site was < 5000 ohm and inter-electrode impedance was <2000 ohm.

(ii) *Instructions to the subject*- After the electrode placement, the following instructions were given to them:

They were instructed to relax themselves and at the same time to remain alert, through out the test.

They were asked to keep their eyes open and to fixate their vision to one spot to minimize alpha interference.

P300 was recorded in passive condition, by instructing the subjects to relax and not to pay attention to what they were listening.

MMN was recorded by instructing the patient to relax and not to pay attention to what they were listening.

(iii) *Data Collection*- After instructing the subjects, the earphones were placed on the ears, being careful not to dislodge any electrodes, The electrode leads and electrode box were as far away from the earphones as possible to minimize artifacts, Roth P300 and MMN were then recorded from the subject using the protocol described earlier.

c) Identification of P300 waveform

LLR peaks were identified from the response for the frequent stimuli. The P300 was identified in the response for the infrequent stimuli by visual inspection. The third positive peak from the baseline, following the N200 was considered as P300. The latency of LLR and P300 was recorded.

d) **Analysis by MMN**

The MMN response was obtained by subtracting the response for the frequent stimulus from the response for the infrequent stimulus, at both *CZ* and Pz sites, For the identification of MMN true response through visvial detection the following criteria was used: -

- MMN is the first negative trough in the subtracted waveform with absolute amplitude less than-0.3μv in the latency range of N_1 P_2 or N_2P_2 complex of LLR.
- LLR should be present in the unsubtracted frequent and/or infrequent waveform.
- A positive peak should follow the negative trough.

RESULTS AND DISCUSSION

The information collected from case history, medical history, results of radiological investigations, WAB, pure-tone audiometry, immittance evaluation, auditory brainstem response (wherever applicable), cortical evoked potentials, P300 and MMN of five aphasic subjects is described and discussed.

As it was a small sample and a heterogeneous group, no statistical analysis was carried out. Pure-tone audiogram was described based on the classification given by Clark (1981, cited in Harrell, 2000). Classification given by Liden, Harford and Hallen, (1974) was used to classify the tympanograms obtained for 226Hz-probe tone. LLR and P300 recorded in passive condition was compared with the normative data given by Kiruthika (2001) and data obtained from control subjects. MMN recorded from control subjects was used to classify MMN as normal or abnormal. The mean values given by the corresponding investigators ± 2SD was considered as normal for all the potentials.

Subject 1

a. **Case History:** A 49 year old male reported with a history of attack of paralysis on right side due to high blood pressure 8 months back. Immediately after the attack, the client lost the ability to speak normally and had slurred speech. Word finding difficulty, repetition of utterances were reported. The client was hospitalized for 18days. Speech was reported to be improving in clarity.

b. **Medical History:** Right side Hemiplegia with hypertension with aphasia

c. **Radiological investigations:** MRI reports revealed acute cerebral haemotoma in left basal ganglion region with minimal midline shift. CT scan results showed left internal capsule having slight mass with CVA and left perisylvalian haemotoma.

d. **WAB:** WAB was done at the Department of speech pathology. The subject was diagnosed as a fluent aphasic with word retrieval problem i.e anomic aphasia.

e. **Audiometric and Immittance evaluation results:** Pure-tone audiometry couldnot be done as subject failed to understand the instructions. Immittance evaluation showed "A" type tympanogram with reflexes present indicating normal middle ear function.

f. **ABR results:** The absolute latency for V^{th} peak of ABR could be recorded till 30dBnHL and was found to be within normal limits for clicks presented at 30.1 /sec repetition rate.

g. **LLR results:** Figure 1-1 and 1-2 shows LLR waveform for the right ear and left ear. LLR peaks could not be identified due to poor waveform morphology in right ear as well as left ear for Fz . For Pz recording latency of P1 and N1 peaks was found to be within normal limits while latency of P2 and N2 was comparatively delayed in both right ear and left ear.

h. **P300 results:** P300 was absent in recordings for both the ears for both Fz and Pz recording. P300 waveforms for both ears are shown in Figure 1-1 and 1-2.

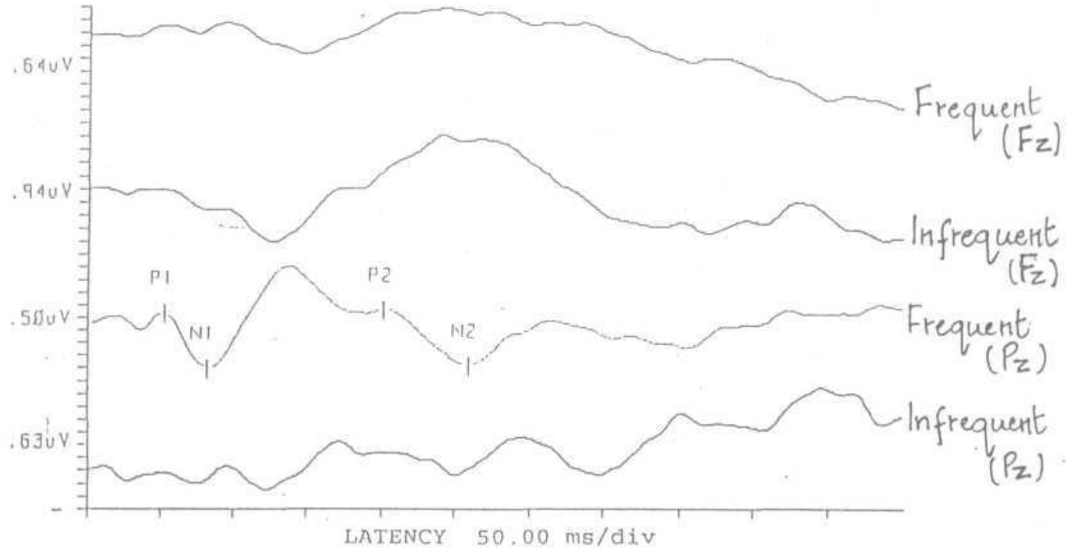


Figure: 1-1 LLR and P300 waveforms of subject 1 for the right ear stimulation.

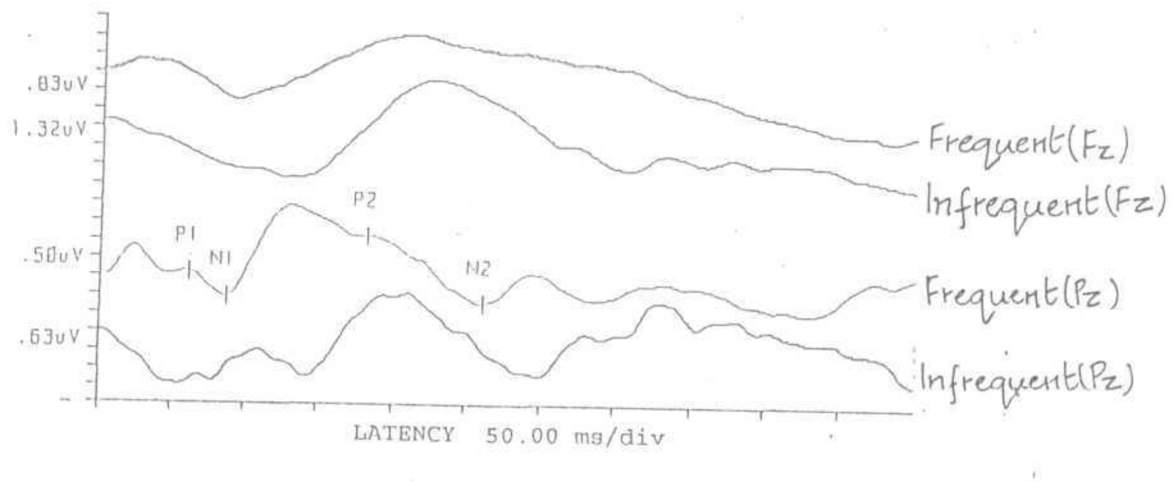


Figure: 1-2 LLR and P300 waveforms of subject 1 Tor the left ear stimulation.

i. MMN results: In right ear, the latency, amplitude and duration of MMN in both Cz and Pz recording was within normal limits while in left ear MMN was found to be absent for both Cz and Pz recording. The MMN waveforms for both ears are shown in Figure 1-3 and 1-4.

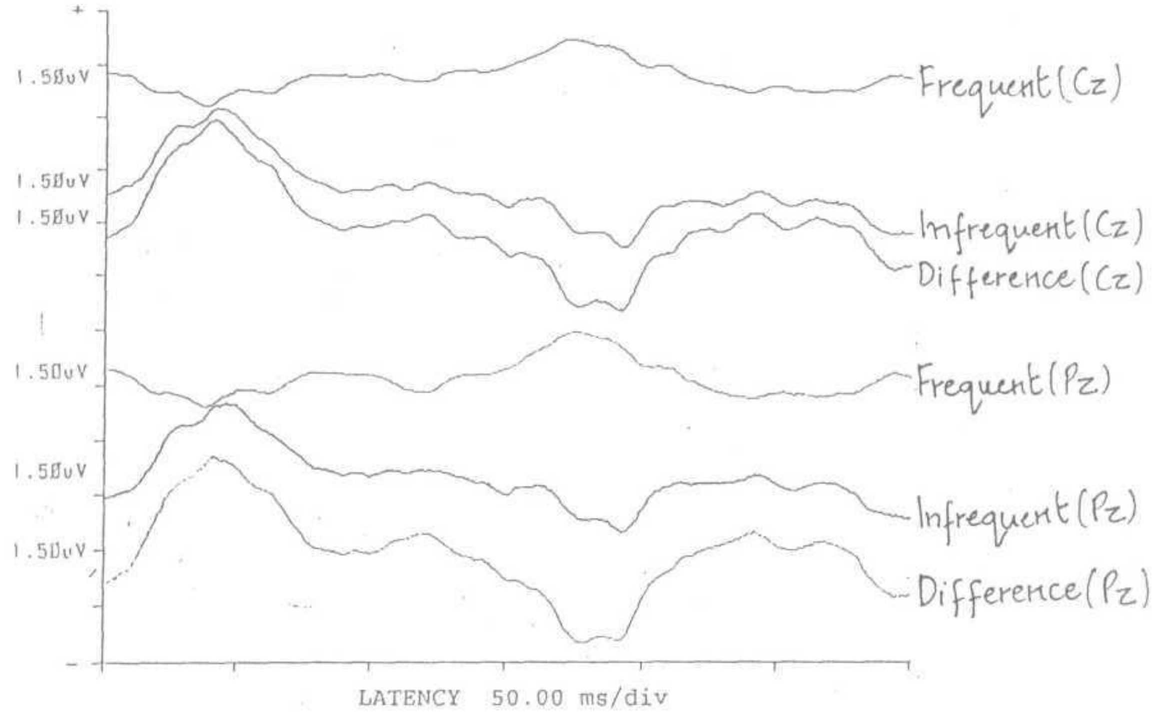


Figure: 1-3 MMN waveforms of subject 1 for the right car stimulation

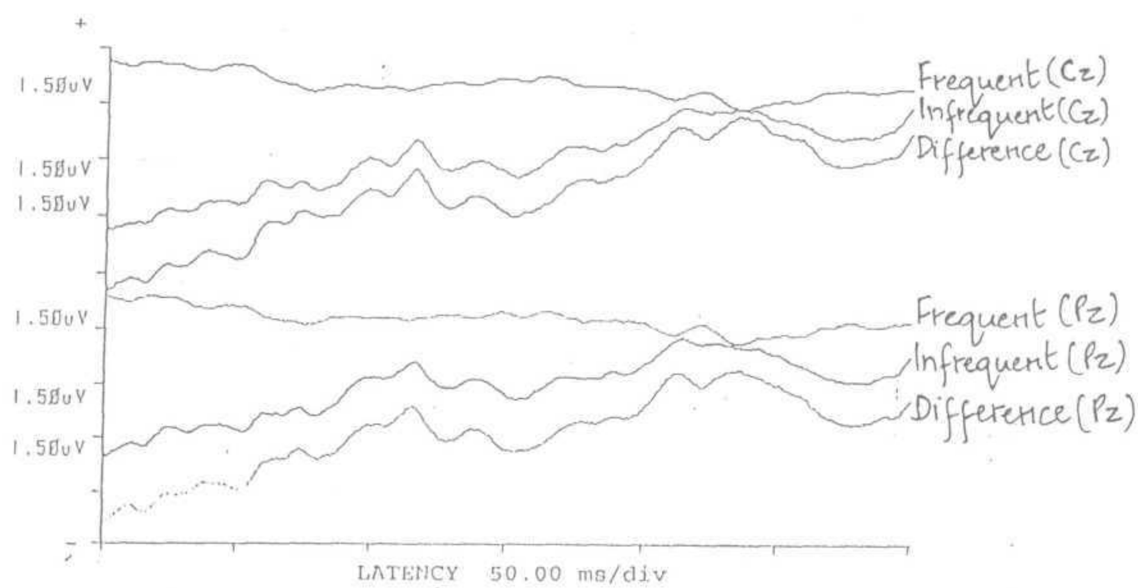


Figure: 1-4 MMN waveforms of subject I for the left ear stimulation

Comments:

The findings of various tests done for subject 1 are summarized in Table 3

Table 3: Results of various tests done for subject 1

Age/Sex	49yr/M			
Duration	8 months			
WAB Results	Anomic aphasia			
Radiological Findings				
CT Scan	Left internal capsule with slight mass with CVA with left perisylvalian haemotoma.			
MRI	Acute cerebral haemotoma in left basal ganglion region.			
LLR	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
PI	Absent	Normal	Absent	Normal
Nl	Absent	Normal	Absent	Normal
P2	Absent	Prolonged	Absent	Prolonged
N2	Absent	Prolonged	Absent	Prolonged
P300	Absent in Both Ears			
MMN	Right Ear		Left Ear	
	Cz	Pz	Cz	Pz
Latency	Normal	Normal	Absent	Absent
Amplitude	Normal	Normal	Absent	Absent
Duration	Normal	Normal	Absent	Absent

Results of WAB indicated anomic aphasic, a symptom seen in almost all cases of generalized lesion (Hegde, 1994). The absence of LLR peaks for Fz recording in both ears could be explained as: Evoked potentials have multiple generators and what is recorded in a far-field response is a resultant of all these generators. The presence of LLR will depend on the proximity of site to the generator also on dipole orientation of the potentials at that particular generator. Therefore what is recorded from Fz may not be same for Pz recording. So absence of LLR peaks could be attributed to the fact that the generators which are strongly contributing to Fz recording may be more affected. Henkin, Rabin, Gadoth and Pratt (2002) reported that P2 and N2 also reflects semantic processing. Though auditory comprehension is good in anomics but impairment to varying extent can be seen (Hegde, 1994). Therefore the prolongation of P2 and N2 in both ears for Pz recording could be due to deficits in auditory comprehension. Kiruthika (2001) observed absence of P300 even in a few normals (8 out of 30 subjects). The subject had a lesion in sub-thalamic region which is also generator site of P300 (McPherson, 1996). It is therefore difficult to say whether the absence of P300 is due to lesion or it is a normal variation. Iilvonen et al. (2001) reported that by recording MMN to monaural stimuli, the discrimination accuracy could be separately determined for the left and right temporal lobe. Hence absence of MMN in left ear indicates that probably there is a lesion in right temporal lobe also, which is affecting duration discrimination.

Subject 2

a. Case History: A 39 year old male reported with a complaint of irrelevant speech consequent to head trauma due to a road accident 3 months back. He was unconscious for 4 days and was kept in intensive care unit (ICU). With gradual recovery, he had symptoms of fluent aphasia, irrelevant speech, word retrieval deficits and preservations.

b. Medical History: Cranial fossa floor fracture with post-traumatic amnesia.

c. **Radiological investigations:** Reports were not available.

d. WAB results: WAB showed that subject had fluent aphasia with word retrieval problem i.e anomic aphasia.

e. **Audiometric and Immittance evaluation results:** Pure-tone audiometry results showed that hearing was within normal limits in both the ears. Immittance evaluation showed "A" type tympanogram with reflexes present indicating normal middle ear function.

f. **LLR results:** LLR was normal in both the ears. The LLR waveform of subject is shown in Figure 2-1 and 2-2 for the right ear and left ear respectively.

g. **P300 results:** P300 could not be identified due to noisy waveform in the right ear for both Fz and Pz recording and for Fz recording in the left ear while it was found to be present at normal latency for Pz recording in left ear. P300 waveforms for both ears are shown in Figure 2-land 2-2.

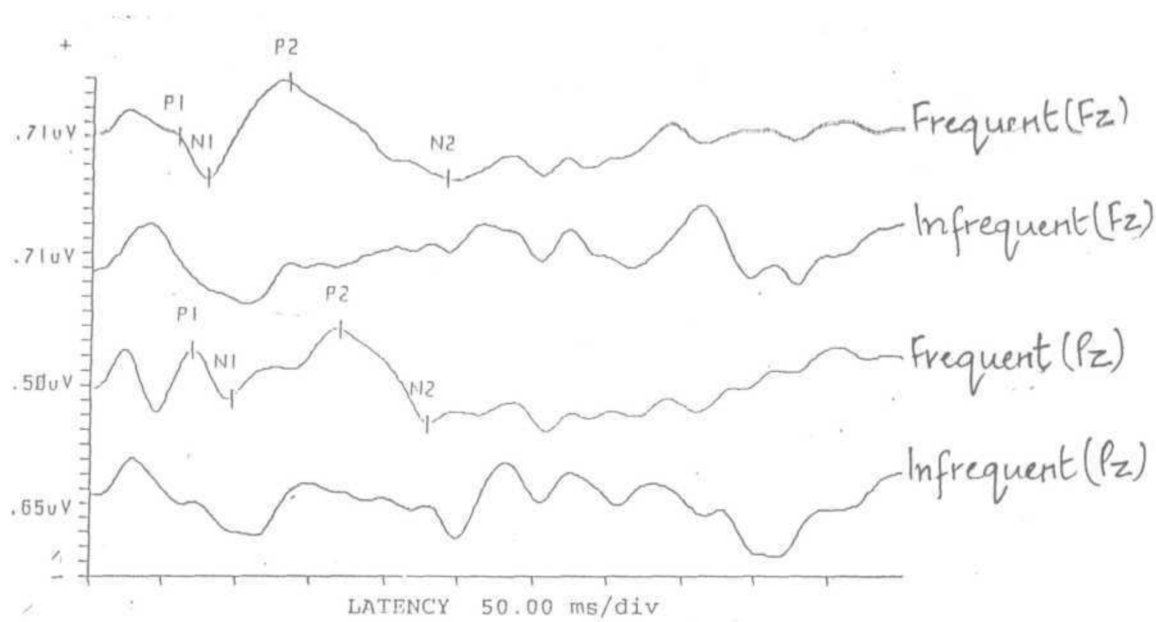


Figure: 2-1 LLR stiul P300 waveforms of subject 2 for (he right e;ir stimulation.

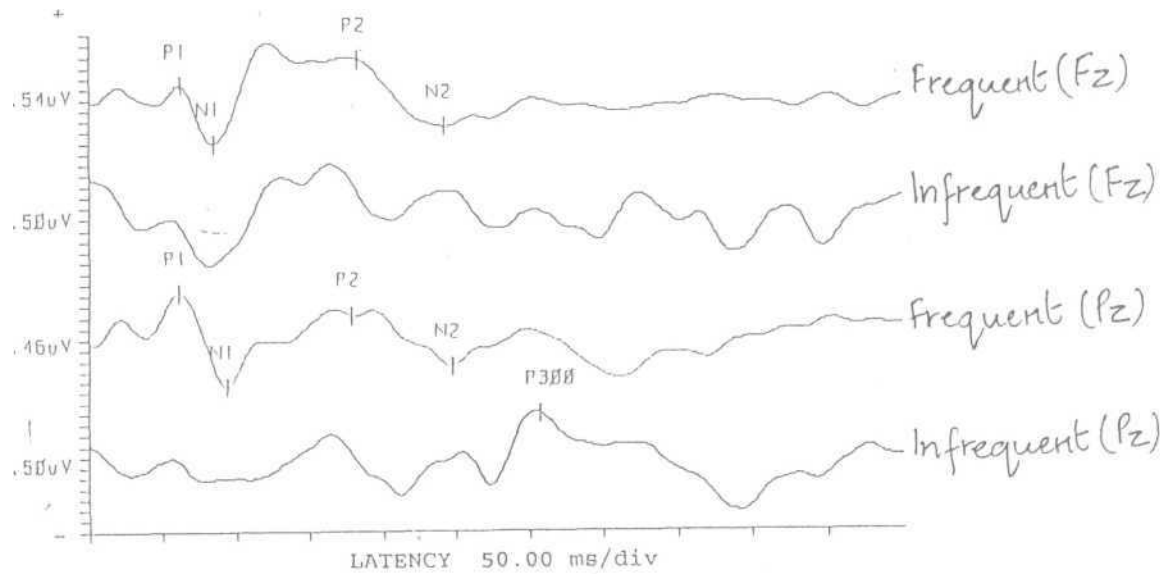


Figure: 2-2 LLR and P300 waveforms of subject 2 for the left ear stimulation

h. MMN results: MMN couldnot be recorded as the subject had lot of involuntary movements.

Comments:

The findings of various tests done for subject 2 are summarized in Table 4

Table 4: Results of various tests done for subject 2

Age/Sex	39yr/M			
Duration	3 months			
WAB Results	Anomic aphasia			
Radiological Findings				
CT Scan	Details not available.			
MRI	Details not available.			
LLR	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
PI	Normal	Normal	Normal	Normal
Nl	Normal	Normal	Normal	Normal
P2	Normal	Normal	Normal	Normal
N2	Normal	Normal	Normal	Normal
P300	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
	Absent	Absent	Absent	Normal
MMN	Couldnot be done due to lot of involuntary movements.			

Presence of LLR waveforms indicates that generators of LLR are spared. Absence of P300 in right ear can be explained as due to damage to subthalmic or left frontal cortex, the generators site of P300 (McPherson, 1996). These findings correlate well with results obtained on behavioral tests (WAB). Absence of P300 for Fz recording in left ear could be attributed to deviance in scalp distribution of the response

Subject 3

a. **Case History:** A 59 year old male reported with a history of attack of paralysis 7 months back following which he lost his speech. He was under coma for 8 days. His comprehension had recovered gradually. Poor intelligibility and articulation errors were reported. He had a similar attack at the age of 28 years also but had recovered completely from it.

b. **Medical History:** Right hemiplegia with associated cardiac problems.

c. **Radiological investigations:** MRI reports revealed early stage sub-acute haemotoma in left parietal lobe with perilesional edema, age related cerebral atrophy, chronic infarct with encephalomalacic changes in right fronto-temporo-parietal lobes. CT scan results showed intracerebral haemorrage.

d. **WAB results:** WAB showed that subject had conduction aphasia.

e. **Audiometric and Immittance evaluation results:** Pure-tone audiometry couldnot be done as subject failed to understand the instructions. Immittance evaluation showed "A" type tympanogram with reflexes present at normal hearing level in right ear and elevated in left ear indicating normal middle ear function.

f. **ABR results:** The absolute latency for V^{th} peak of ABR could be recorded till 30dBnHL and was found to be within normal limits for clicks presented at 30.1 /sec repetition rate for right ear. However for left ear Vth peak was found to be absent at presentation level lower than 50 dBnHL indicating a mild hearing loss in left ear.

g. **LLR results:** As shown in Figure 3-1 and 3-2, latency of all the peaks was normal except for N2 recorded from Pz for right ear stimulation. However latency was found to be comparatively prolonged for all the LLR peaks in left ear for both Fz and Pz recording.

h. P300 results: P300 was found to be absent in right ear for both Fz and Pz recording. However latency of P3 peak was found to be within normal limits for both Fz and Pz recording in the left ear.P300 waveforms for both ears are shown in Figure 3-1 and 3-2.

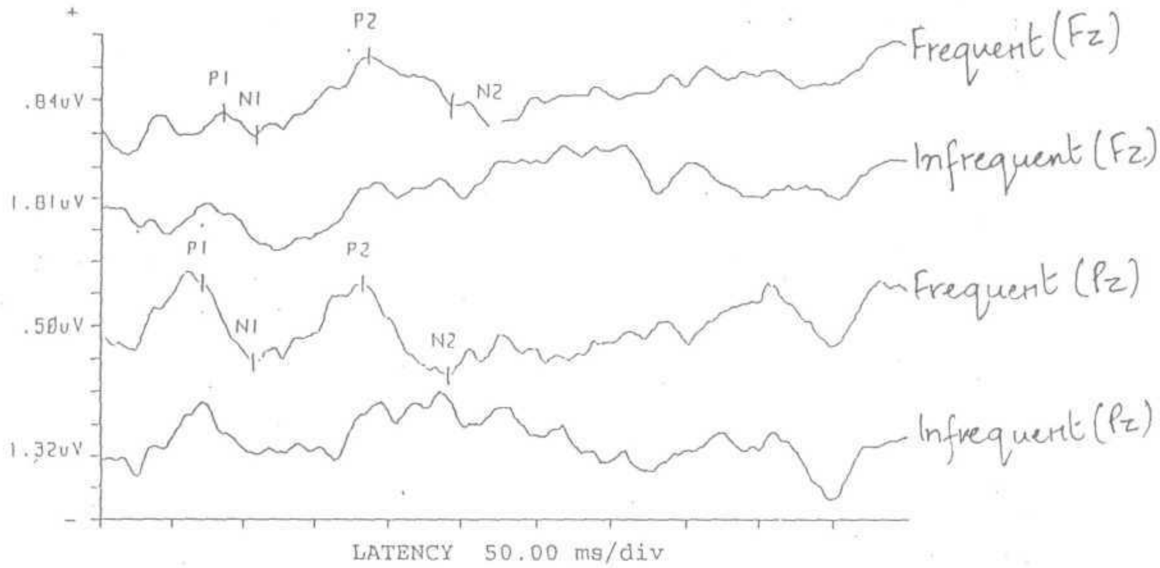


Figure: 3-1 LLR and P300 waveforms of subject 3 for the right ear stimulation.

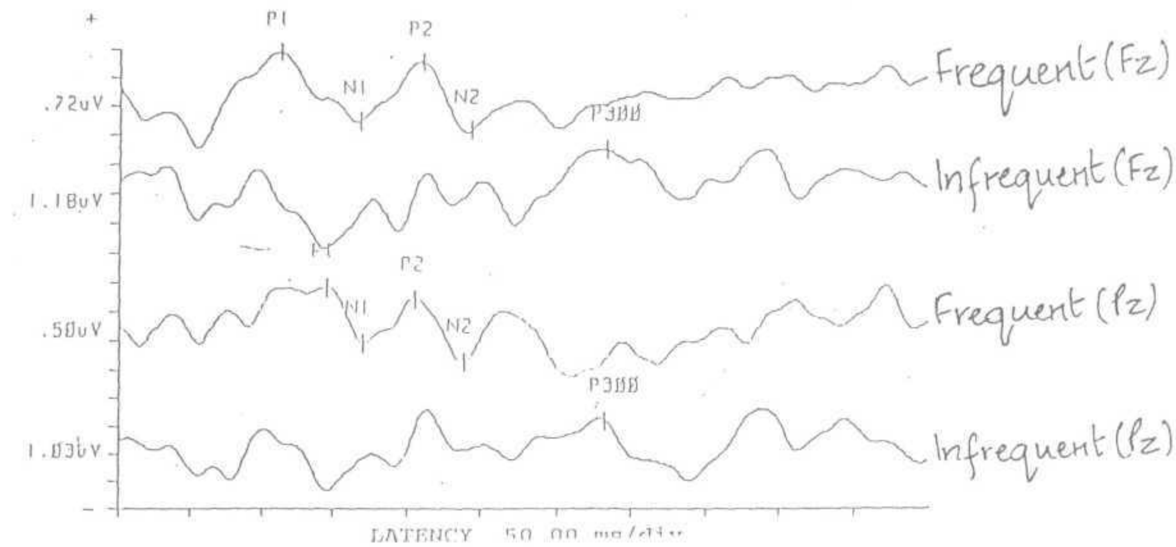


Figure: 3-2 LLR and P300 waveforms of subject 3 for the left ear stimulation.

i. **MMN results:** In right ear, the latency, amplitude and duration of MMN in both Cz and Pz recording was within normal limits while in left car MMN was absent for both Cz and Pz recording. The MMN waveforms for both ears are shown in Figure 3-3 and 3-4

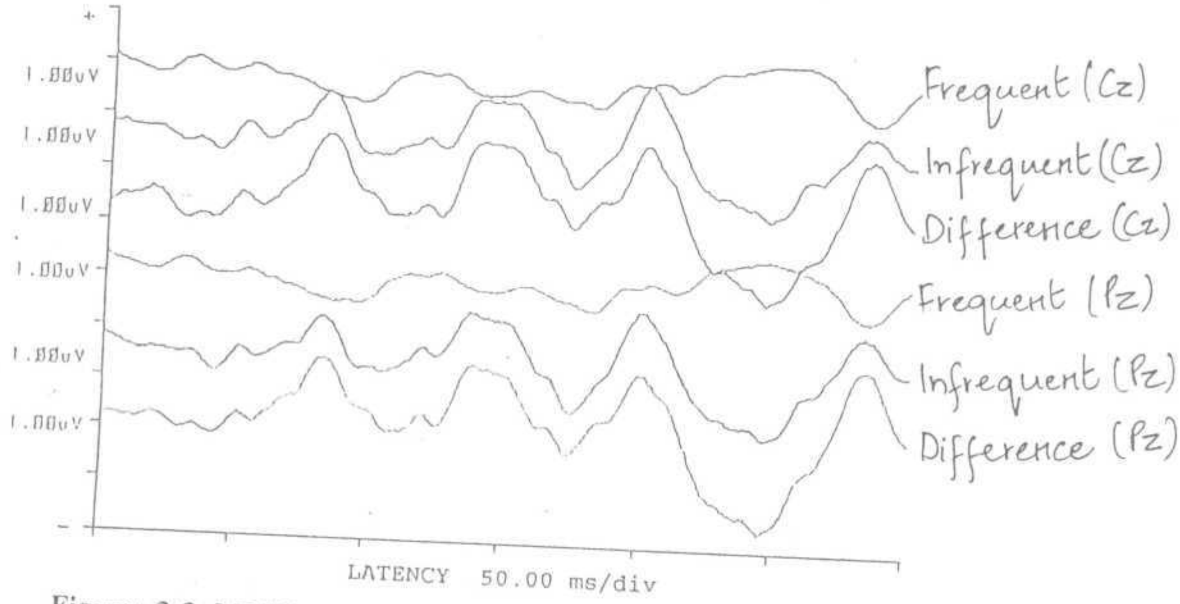


Figure: 3-3 MMN waveforms of subject 3 for the right ear stimulation

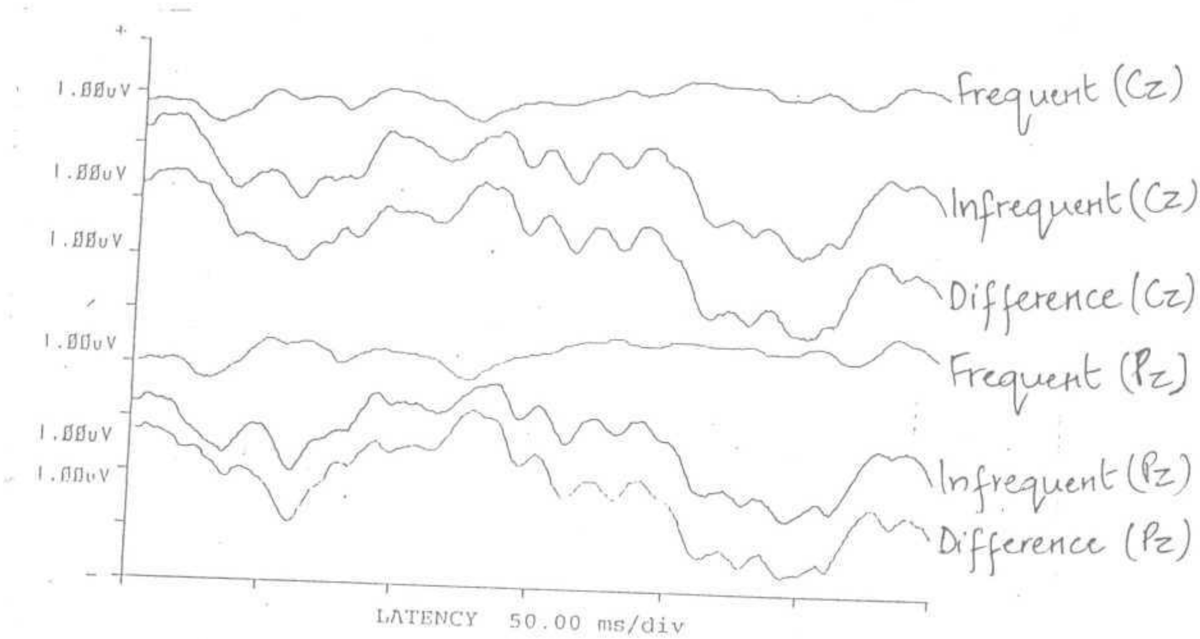


Figure: 3-4 MMN waveforms of subject 3 for the left ear stimulation

Comments:

The findings of various tests done for subject 3 are summarized in Table 5

Table 5: Results of various tests done for subject 3

Age/Sex	59yr/M			
Duration,	7 months			
WAB Results	Conduction aphasia			
Radiological Findings				
CTScan	Intracerebral haemorrage.			
MRI	Early stage sub-acute haemotoma with perilesional edema.			
LLR	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
PI	Normal	Normal	Prolonged	Prolonged
Nl	Normal	Normal	Prolonged	Prolonged
P2	Normal	Normal	Prolonged	Prolonged
N2	Normal	Normal	Prolonged	Prolonged
P300	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
	Absent	Absent	Normal	Normal
MMN	Right Ear		Left Ear	
	Cz	Pz	Cz	Pz
Latency	Normal	Normal	Absent	Absent
Amplitude	Normal	Normal	Absent	Absent
Duration	Normal	Normal	Absent	Absent

This subject had conduction aphasia, a condition caused due to damage to arcualate fasciculus that connects Brocas area with Wernicks area or due to damage to left temporal lobe (Wernickes, 1874, cited in Hegde, 1994). The prolongation of LLR peaks may be because of hearing loss in left ear. However MMN is not that much affected by mild hearing loss (Sivaprasad, 2000). Similar to subject 1, in this subject also absence of MMN was seen in left ear, which could be because of damage to right temporal lobe affecting duration discrimination. When the lesion is anterior to arcualate fasciculus , it mainly effects frontal cortex (Love and Webb, 1986) and absence of P300 in right ear could be attributed to damage to this area. The presence of MMN in right ear indicates that certain areas of superior temporal gyrus might be spared (Aaltonen et al., 1993)

Subject 4

a. Case History: A 63 year old male reported with a complaint of irrelevant speech consequent to right sided paralytic attack 2 years back. He had loss of speech for 15 days following the attack. He started speaking in monosyllables after 15 days. His comprehension was fair and could speak in 3-4 word utterances, though intelligibly was still poor.

b. Medical History: The subject had right sided paralytic stroke, diabetes and hypertension.

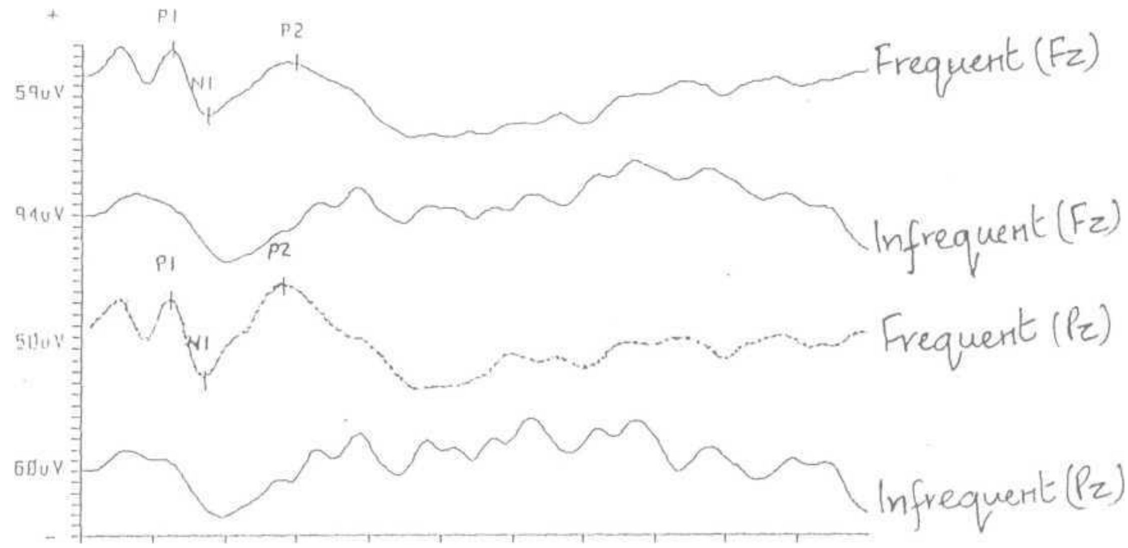
c. Radiological investigations: CT scan results showed that subject had infarct in left parietal lobe

d. WAB results: WAB showed that subject had Broca's aphasia with fair comprehension for complex forms and commands.

e. Audiometric and Immittance evaluation results: Pure-tone audiometry results showed that hearing was within normal limits in both the ears. Immittance evaluation

f. LLR results: Latency of LLR peaks was within normal limits for both Fz and Pz recording in left ear and right ear except N2 which was found to be absent for both Fz and Pz recording in right ear. The LLR waveforms for both ears are shown in Figure 4-land 4-2.

g. P300 results: P300 was absent in right ear for both Fz and Pz recording. However it was found to be within normal limits for both Fz and Pz recording in the left ear. P300 waveforms for both ears are shown in Figure 4-1 and 4-2.



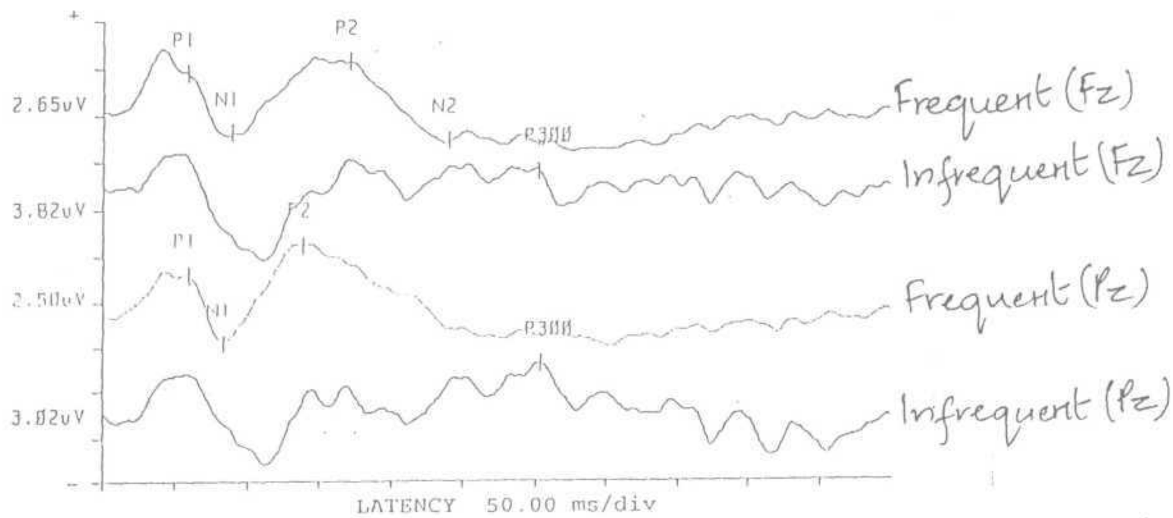


Figure: 4-2 LLR and P300 waveforms of subject 4 for the left ear stimulation.

h. MMN results: For both right and left ear, the latency, amplitude and duration of MMN in both Cz and Pz recording were within normal limits. The MMN waveforms for both ears are shown in Figure 4-3 and 4-4.

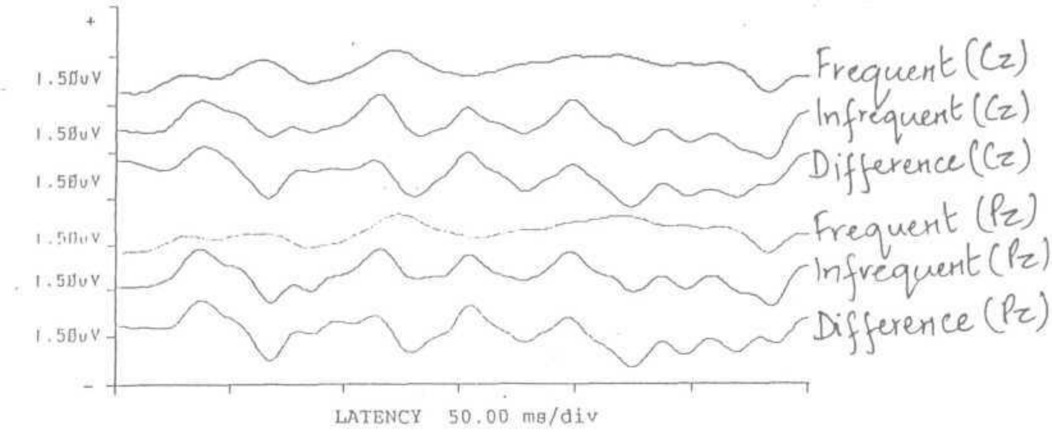


Figure: 4-3 MMN waveforms of subject 4 for the right ear stimulation .

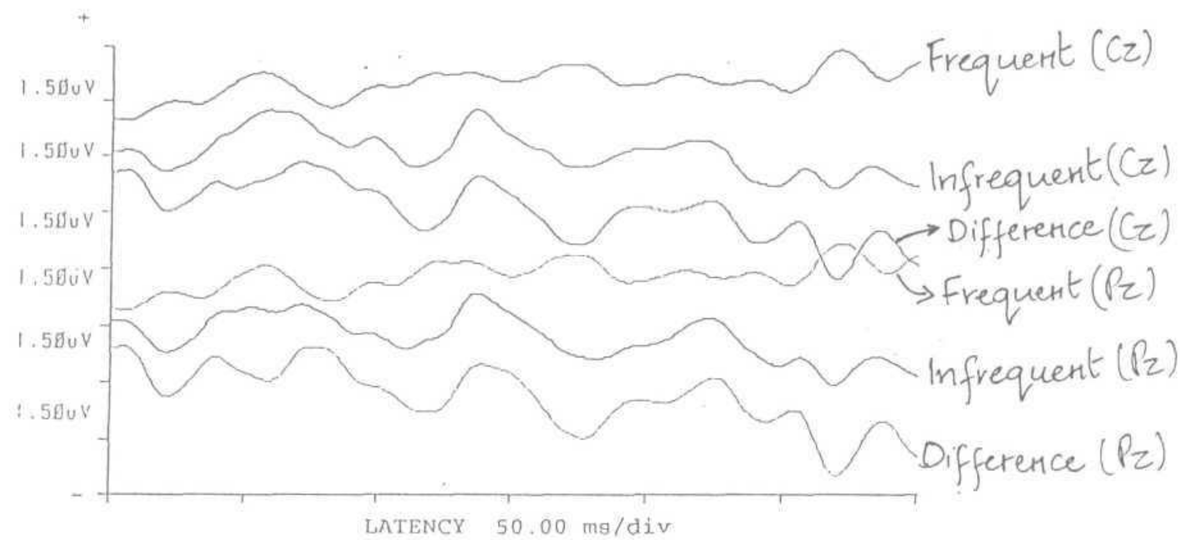


Figure: 4-4 MMN waveforms of subject 4 for the left ear stimulation.

Comments:

The findings of various tests done for subject 4 are summarized in Table 6

Table 6: Results of various tests done for subject 4

Age/Sex	63yr/M			
Duration	2 years			
WAB Results	Broca's aphasia			
Radiological Findings				
CTScan	Recent Infarct Left parietal Lobe(left MCA territory)			
MRI	Not Probed.			
LLR	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
P1	Normal	Normal	Normal	Normal
N1	Normal	Normal	Normal	Normal
P2	Normal	Normal	Normal	Normal
N2	Absent	Absent	Normal	Normal
P300	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
	Absent	Absent	Normal	Normal
MMN	Right Ear		Left Ear	
	Cz	Pz	Cz	Pz
Latency	Normal	Normal	Normal	Normal
Amplitude	Normal	Normal	Normal	Normal
Duration	Normal	Normal	Normal	Normal

Results of WAB indicated Brocas aphasia, a condition in which the lesion is in frontal lobe (Brocas, 1861,cited in Hegde, 1994). McPherson (1996) reported that frontal lobe is one of the generator of P300 and McCarthy et al., 1989(cited in Tonnquist-Uhlen, 1996) reported that frontal lobe could also be generator for N2. The absence of N2 and P300 in the right ear indicates that probably there could be damage to left frontal lobe. The presence of MMN in both ears indicate that primary auditory cortex is intact or has resolved due to spontaneous recovery since auditory processing of very fine stimulus differences is present.

Subject 5

a. **Case History:** A 15 year old male reported with a complaint of unclear speech consequent to surgery for convulsions 6 months back. The subject had stopped speaking 8 days after surgery. Gradually expressive language had recovered as reported but speech was still unclear. He was taking anti-convulsion drugs since 3 years.

b. **Medical History:** Focal seizures, right temporal abscess.

c. **Radiological investigations:** MRI results showed Cortical dysphasia (?),Gliosis in right temporal region(?) and he underwent surgery for the same .

d. **WAB results:** WAB showed that subject had fluent aphasia with word retrieval problem i.e anomic aphasia.

e. **Audiometric and Immittance evaluation results:** Pure-tone audiometry results showed that hearing was within normal limits in both the ears. Immittance evaluation showed "A" type tympanogram with reflexes present indicating normal middle ear function.

f. LLR results: As shown in Figure 5-1 and 5-2, latency of all the peaks was normal except for Nl, P2, N2 recorded from Fz in left ear.

g. P300 results: P300 was found to be absent in both the ears for both Fz and Pz recording. P300 waveforms for both ears are shown in Figure 5-1 and 5-2.

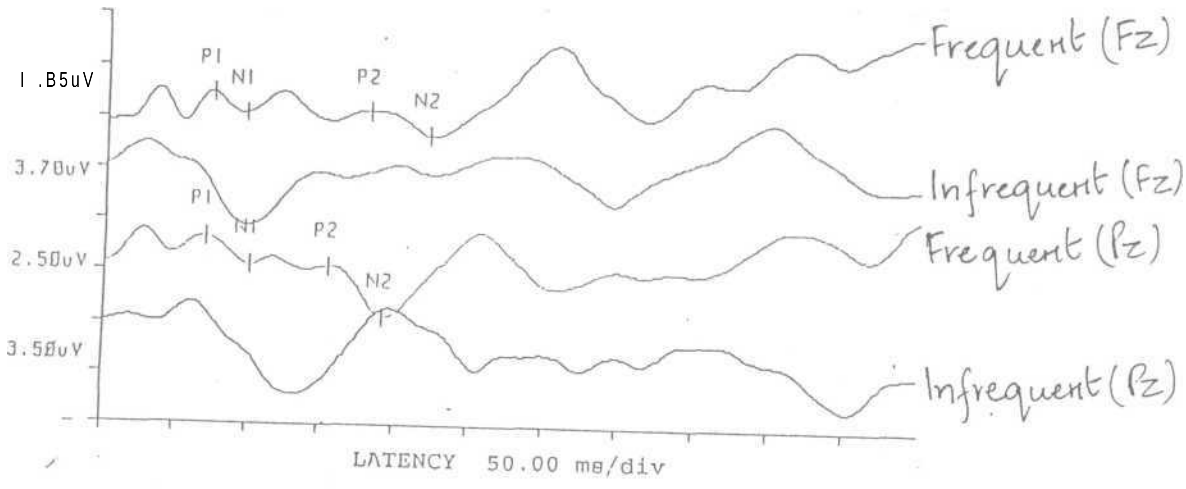


Figure: 5-1 LLR and P300 waveforms or subject 5 for the right ear stimulation.

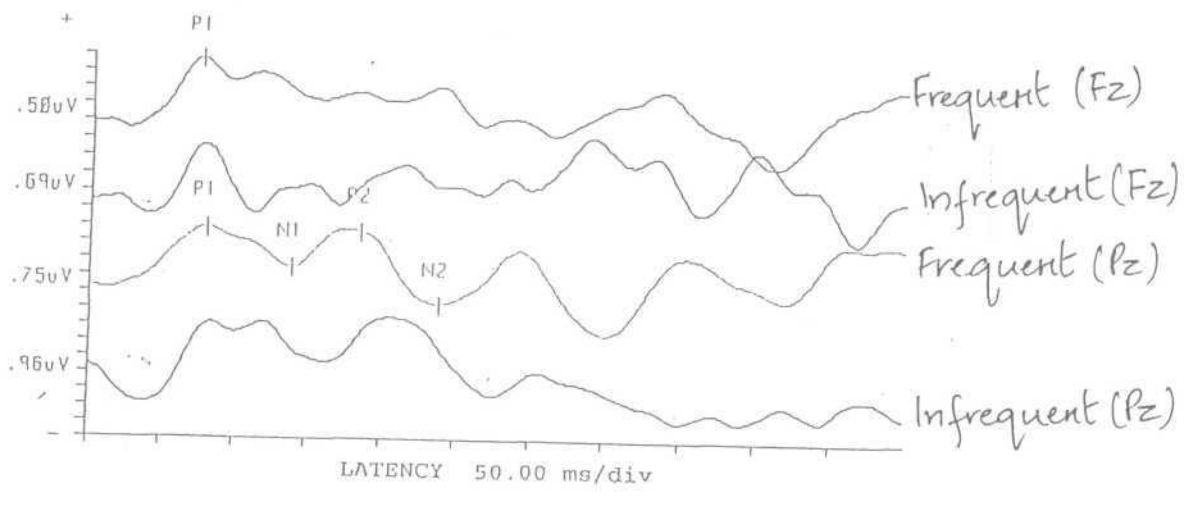


Figure: 5-2 LLR and P300 waveforms of subject 5 for the left ear stimulation.

h. MMN results: For left ear, the latency, amplitude and duration of MMN in both Cz and Pz recording were within normal limits while in right ear it was within normal limits for Pz recording and absent for Cz recording. The MMN waveforms for both ears are shown in Figure 5-3 and 5-4.

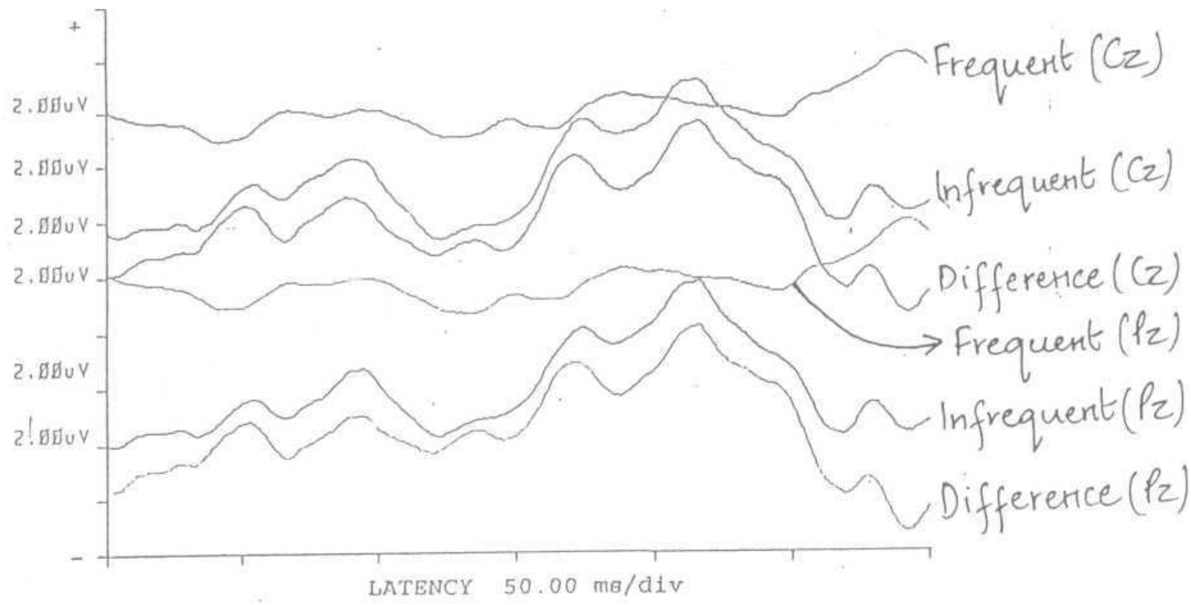


Figure: 5-3 MMN waveforms of subject 5 for the right ear stimulation.

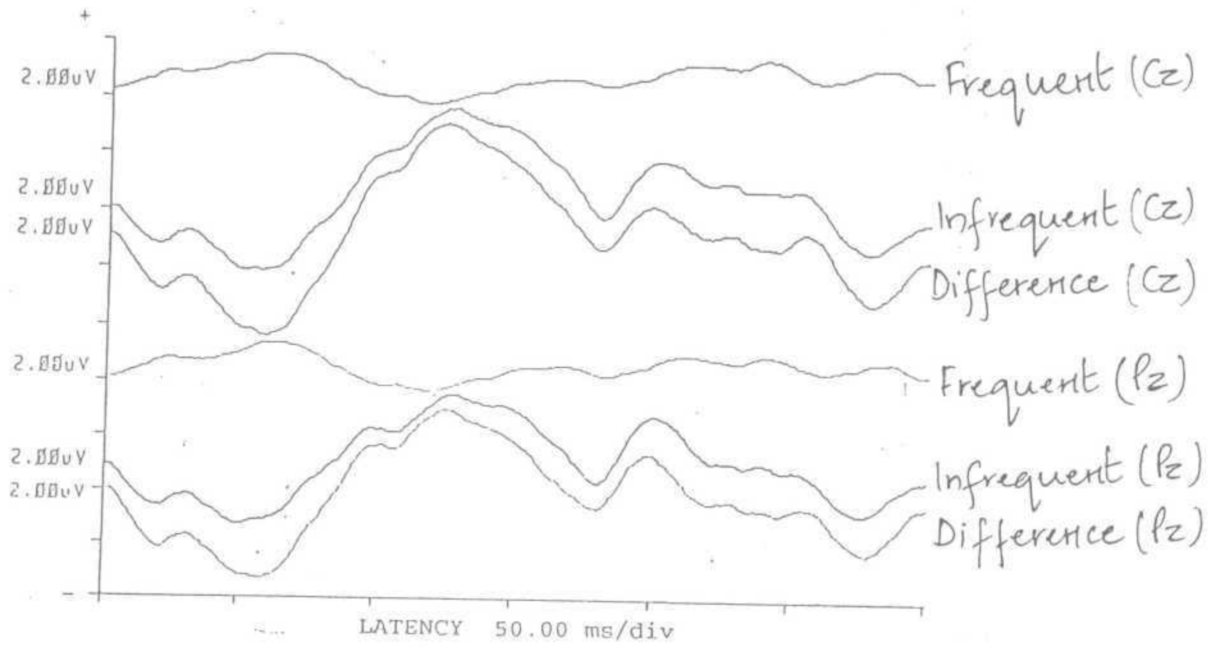


Figure: 5-4 MMN waveforms of subject 5 for the left ear stimulation.

Comments:

The findings of various tests done for subject 5 are summarized in Table 7

Table 7: Results of various tests done for subject 5

Age/Sex	15yr/M			
Duration	6 months			
WAB Results	Anomic aphasia			
Radiological Findings				
CTScan	Not Probed.			
MRI	Cortical dysphasia (?),Gliosis in right temporal region(?)			
LLR	Right Ear		Left Ear	
	Fz	Pz	Fz	Pz
P1	Normal	Normal	Normal	Normal
N1	Normal	Normal	Absent	Normal
P2	Normal	Normal	Absent	Normal
N2	Normal	Normal	Absent	Normal
P300	Absent in Both Ears			
MMN	Right Ear		Left Ear	
	Cz	Pz	Cz	Pz
Latency	Absent	Normal	Normal	Normal
Amplitude	Absent	Normal	Normal	Normal
Duration	Absent	Normal	Normal	Normal

The findings showing presence of Nl, P2, N2 in recording from one electrode site (Pz) and absent waveform from another electrode (Fz) in left ear indicates deviance in scalp distribution of the response i.e among multiple generators of these LLR peaks, generators which strongly contribute to Fz may be more affected. Kiruthika (2001) observed absence of P300 even in a few normals (8 out of 30 subjects). The subject had lesion in sub-thalamic region which is also generator site of P300 (McPherson, 1996).

Similar to subject 1, in this subject also it is difficult to say whether the absence of P300 is due to lesion or it is a normal variation. It is also difficult to explain absence of MMN for Cz recording in right ear as subject initial radiological investigations showed gliosis in right temporal region for which surgery was done. Details of post-operative radiological investigations were not available. So it is not known that whether there is any damage to left temporal lobe during the surgery leading to absence of MMN.

Concluding Remarks

Table: 8 Summary of results of various tests done for 5 subjects.

	Subject 1	**Subject 2**	**Subject 3**	**Subject 4**	**Subject 5**
Age/Sex	49yr/M	39yr/M	59yr/M	63yr/M	15yr/M
Duration	8 months	3 months	7 months	2 years	6 months
WAB Results	Anomic Aphasia	Anomic Aphasia	Conduction Aphasia	Brocas Aphasia	Anomic Aphasia
Radiological Findings	Left internal capsule with slight mass. Acute cerebral haemaotoama in left basal ganglion region	Details not available	Intracerebral haemorrage. Early stage sub-acute haemotoama.	Recent infarct left parietal lobe.	Gliosis in right temporal region.
LLR	Abnormal in both ears	Normal in both ears	Abnormal in left ear	Normal except For N2 in right ear	Abnormal for Fz recording in left ear.
P300	Absent in both ears	Abnormal in both ears	Absent in right ear	Absent in right ear	Absent in both ears
MMN	Absent in left ear	Couldnot be done.	Absent in left ear	Normal in both ears	Absent for Cz recording in right ear

As seen in Table 8, abnormality in LLR was seen in two of the three anomic subjects where as P300 was affected in all the three. MMN was also found to be affected in two anomics subjects while in third subject it couldnot be recorded. For Brocas aphasia only abnormality was seen in N2 and P300 recordings of right ear. In conduction aphasic, prolongation of LLR waveform was seen due to hearing loss in left ear where as MMN was found to be affected for left ear while abnormality in P300 was seen for right ear.

Selinger et al. (1989) reported that aphasic subjects responded differently from the normal group, particularly when the task required processing of verbal information in which higher amplitude was seen than non-verbal task .In this study, LLR was found to be present in a majority of the subjects which could be because of use of non-verbal stimuli. Further studies using verbal stimuli could give more information regarding this.

In a majority of the subjects P300 was found to be affected. P300 is related to cognition and occurs across sensory abilities (McPherson, 1996). Hillis (2001) reported that cognition is impaired in a majority of brain damaged patients. Thus the absence or abnormality of P300 may indicate that subjects cognitive abilities are impaired. It was also observed that in some subjects cortical evoked potentials could be recorded from one electrode site while it was absent when recorded from other electrode site for the same ear. Such results have also been reported by Onofrj et al. (1995) who found that there is a change in scalp distribution of P300 in aphasic subjects.

Auther et al. (2000) reported that MMN was absent in most of the aphasics with poor auditory comprehension using speech stimuli. Wertz et al. (1998) compared tone and speech stimuli and reported that while MMN was present in 79% of aphasic subjects to tone stimuli using frequency deviance, it was present in only 54% of aphasic subjects for speech stimuli. In the present study the duration deviance for tone stimuli was used and MMN was found to be affected in a majority of subjects. Probably duration deviance is more sensitive in detecting abnormality than frequency deviance. However this has to be confirmed using larger sample.

Iilvonen et al. (2001) reported that by recording MMN to monaural stimuli, the discrimination accuracy could be separately determined for the left and right temporal lobe. In this study, it was observed that in two aphasics, MMN was absent in one ear and present in another ear indicating that there is a damage to contralateral temporal lobe. Absence of MMN to duration differences indicates that auditory processing of very fine stimulus differences is affected.

To conclude, presence or absence of cortical evoked potentials in aphasics will depend on site of lesion. Recording of these potentials will throw light on auditory comprehension / cognitive abilities of an aphasic, which in turn will help in planning out the management.

SUMMARY AND CONCLUSIONS

It is important to assess aphasic subject's auditory abilities as it has been reported that at least 60-70% of all aphasic subjects, fluent as well as nonfluent, display some degree of impairment in auditory comprehension of language (Boiler, Kim, and Mack, 1977). The auditory abilities of an aphasic person can be assessed using behavioral (audiological and non-audiological) as well as objective audiological tests. The behavioral tests, however, have limitations in the sense that they are dependent upon some type of response, which the subject may or may not be capable of performing. Although behavioral data have provided useful information about non-linguistic auditory capabilities of aphasic subjects, objective techniques using cortical evoked potentials such as late latency response (LLR), P300 and mismatch negativity (MMN) might more directly assess neurological functioning without relying solely on behavioral output measures and provides information about how well the person is processing auditory information which can be helpful in planning out the rehabilitation.

Literature regarding assessing auditory abilities of aphasic patient indicates that there is a need for using a test battery approach comprising of behavioral (non-audiological and audiological) and electrophysiological measures to assess the auditory abilities of aphasic subjects. Attempts were made to assess auditory comprehension of aphasics using behavioral tests. Recently electrophysiological tests have also been used. However there is dearth of literature for such studies.

Hence the present study was taken up to study cortical evoked potentials- LLR, P300, and MMN in aphasics. LLR and P300 was recorded at repetition rate of 0.9/sec by presenting the stimulus at 80dBnHL.MMN was recorded at repetition rate of 3.1/sec and stimulus was presented at 60dBnHL.These potentials were recorded using Bio-logic evoked potential system (navigator). Five aphasics in the age range of fifteen to seventy years were tested. Hearing thresholds and normal middle ear function were established based on pure-tone audiometry and immittance results. ABR was done for/patients who failed to understand the instructions. In LLR waveform absolute latency of PI, Nl, P2, N2 were measured from frequent waveform of P300 in Fz and Pz recording. P300 was recorded in passive condition by instructing the patient to relax and not to pay attention to stimuli. The absolute latency of P300 was measured from infrequent waveform in Fz and Pz recording The MMN was measured by subtracting the response for the frequent stimulus from the response for the infrequent stimulus, at both Cz and Pz recording sites. The MMN analysis included peak latency of MMN, amplitude and duration of MMN.

The analysis of results revealed that LLR was abnormal in two of the three anomic subjects where as P300 was affected in all the three. MMN was also found to be affected in two anomics subjects while in third subject it couldnot be recorded. For Brocas aphasia only abnormality was seen in N2 and P300 recordings of right ear. In conduction aphasic, LLR was found to be prolonged due to peripheral hearing loss in left ear. Abnormality in P300 was seen for right ear while MMN was found to be affected for left ear.

To conclude, presence or absence of these potentials throw light on auditory comprehension abilities of these patients which in turn can be helpful in planning out the rehabilitation. However, as it is known that aphasia is not a unitary disorder, individual variation does exists. Also since these potentials have multiple generators, during far-field recording its difficult to record potentials generated from a specific site. Hence, a lot of variation can be expected in cortical evoked potentials recorded in different aphasics making it difficult to explain site of lesion. Thus a detailed evaluation of auditory processing may provide a more complete understanding of the disorder of aphasia, as well as a more accurate profile of the aphasic individual.

Future Directions

1. Present study considered a very small group of aphasics which was a heterogeneous group. Future studies can select a large sample to form a homogenous group for the study.

2. Since aphasia is often believed to be a disorder showing considerable variability over time, cortical evoked potentials measured at regular intervals may prove to be a valuable tool for measurement of aphasic disturbances and in predicting the recovery.

REFERENCES

Aaltonen,Q., Tuomainen,J., Laine,M. & Niemi,P.(1993).Cortica! difficulties in tonal vs vowel processing as revealed by an ERP component called mismatch negativity(MMN). *Brain and language,* 44,139-151.

Alho, K. (1995). Cortical generators mismatch negativity & its magnetic counterpart (MMNm) elicited by sound changes. *Ear and Hearing,* 16,38-50

Alho, K., Sainio, K., Sajaniemi, N., Reinikainen, K.,& Naatanen, R.(1990).Event related brain potential of human new born to pitch change of an acoustic stimulus. *Electroencephalography and Clinical neurophysiology,* 77,151-155.

Auther, L.L., Wertz, R.T., Miller, T.A., & Kirshner, H.S.(2000).Relationship among the mismatch negativity (MMN) response, auditory comprehension and site of lesion in aphasic *adults Aphasiology,7,5/6,46*1 -470

Boiler, F., Kim, Y. & Mack, J.L. (1977). Auditory comprehension in aphasia. In H.Whitaker & H.A.Whitaker *(Ed.).Studies in neurolingusitics,(ppl-63).NewYork:* Academic Press.

Carhart, R. & Jerger, J.F. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of speech and hearing research,* 24,330-345.

Chapey, R. & Hallowell, B. (2001). Introduction to language strategies in adult aphasia. In Chapey, R. (Ed.), *Language Intervention strategies in aphasia and related Neurocommunication disorders* (4 th Edition),(pp3-15). New York: Lippincott Williams and Williams.

Cobianchi, A.,& Giaquinto, S.(2000).Can we exploit event-related potentials for retraining language after stroke?. *Disability Rehabilitation,* 15,22(9), 427-34. Abstract retrieved December 5,2002,from Pubmed database.

Courchesene, E.(1978). Neurophysiological correlates of cognitive development: Changes in long latency event related potential from childhood to adulthood. *Electroencephalography and Clinical neurophysiology,* 45,468-482.

Csepe, V., Osman-Sagi, J. Molnar, M.& Gosy, M. (2001). Impaired speech perception in aphasic patients: Event-related potential and neuropsychological assessment. *Neuropsychologia,* 39(11), 1194-1208. Abstract retrieved December 5,2002,from Pubmed database.

Csepe, V., Karmes, G. & Molnar, M. (1987). Evoked potential correlates of stimulus deviance during wakefulness and sleep in cat: animal model of mismatch negativity. *Electroencephalography and Clinical neurophysiology,* 66,571-578.

Divenyl, P.L. & Robinson, A.J. (1989). Nonlinguistic auditory capabilities in aphasia. *Brain and Language,* 37,290-326.

Fischler, I., Bloom, P.A., Childers, D.G., Roucer, S.F., & Perry, N.W.(1983).Brain potentials related to stages of sentence verification. *Psychophysiology,* 20,400-409.

Hagoort, P., Brown, C.M.,& Swaab, T.Y. (1996). Lexical-semantic event-related potential effects in patients with left hemisphere lesions and aphasia, and patients with right hemisphere lesions without aphasia. *Brain,* 119,627-649.

Harrell, R.W.(2000). Pure-tone evaluation. In J.Katz (Ed.), *Handbook of Clinical audiology* (5th ed.)(pp71-87). Philadelphia: Lippincott Williams and Williams.

Hegde, M.N.(1994). *A course book on Aphasia and other Neurogenic Language disorders.* San Diego: Singular Publishing Group, Inc.

Henkin, Y., Rabin, L.K., Gadoth, N. & Pratt, H. (2002). Auditory event related potentials during phonetic and semantic processing in children. *Audiology and Neuro-Otology,* 7,228-239

Hillis, A.E. (2001). Cognitive neuropsychological approaches to rehabilitation of language disorders: Introduction. In Chapey, R. (Ed.), *Language Intervention strategies in aphasia and related Neurocommunication disorders* (4th Edition),(pp386-398). New York: Lippincott Williams and Williams.

Hugdahl, K., Wester, K., & Asbjornsen, A. (1990). Dichotic listening in an aphasic male patient after a subcortical hemorrhage in the left fronto-parietal region. *International Journal Neuroscience,* 54(1-2), 139-146. Abstract retrieved December 5,2002,from Pubmed database.

Iilvonen, T.M., Kujala, T., Tervaniemi, M., Salonen, O., Naatanen, R. & Pekkonen, E. (2001). The processing of sound duration after left hemisphere stroke: Event-related potential and behavioral evidence. *Psychophysiology,* 38(4), 622-628. Abstract retrieved December 5,2002, from Pubmed database.

Joutsiniemi, S.L.,Uvonen, T., Sinkkonen, J., Huotilainen, M, Tewaniemi, M., Letitokosk, A., Rinne, T.& Naatanen, R.(1998). The mismatch negativity for duration decrement of auditory stimuli in healthy subjects. *Electroencephalography and Clinical neurophysiology,* 108,154-158.

Keurs, M.T., Brown, C.M., & Hagoort, P. (2002). Lexical processing of vocabulary class in patients with Broca's aphasia: an event-related brain potential study on agrammatic comprehension. *Neuropsychologia,* 40(9), 1547-61.

Kilney, P.(1991).Use of Electrophysio logic measures in the management of children with cochlear implants, brainstem, middle latency and cognitive(P300) responses. *American Journal of Otology,* 12,37-42.

Kilney, P. & Robertson, C.M.T. (1985). Neurological aspects of infant hearing assessment. *Journal of Otolarynology,* 14, (suppl.14), 34

Kiruthika, C. (2001). P300: A comparison between active listening paradigm & a passive listening paradigm. *Unpublished Independent project,* University of Mysore, Mysore.

Kitade, S., Enai, T., Sei, H., & Morita, Y. (1999). The N400 event-related potential in aphasia. *Journal of Medical Investigation,* 46(1-2), 87-95. Abstract retrieved December 5,2002,from Pubmed database.

Korpilahti, P. & Lang, H. (1994). Auditory event related potential components and Mismatch negativity in dysphagic children. *Electroencephalography and Clinical neurophysiology,* 91,256-264.

Kraus,N. & Cheour, M.(2000).Speech sound representation in the brain. *Audiology NeuroOtology,* 5,140-150.

Kraus, N. & Mcgee,T.(1994).MMN in assessment of central auditory function. *American Journal of A udiology,* 3,139-151.

Kraus, N., McGee, T., Carrell, T.P. & Sharma, A (1995). Neurophysiologic basis of speech discrimination. *Ear and hearing,* 16,19-30.

Kraus, N., McGee, T., Littman, T., Nicol, T. & King, T. (1994). Non- primary auditory thalamic representation of acoustic change. *Journal of Neurophysiology,* 72,1270-1277.

Liden, G., Harford, E., & Hallen, 0. (1974). Automatic tymapanometry in clinical practice. *A udiology, 13,126-139.*

Love, R.J. &Webb, W.G.(l986). *Neurologyfor the speech- language pathologist.* Boston: Butterworth Publishers.

McPherson, D.L. (1996). *Late potentials of the Auditory system.* San Diego: Singular Publishing Group, Inc.

Moore, B.D.,& Papanicolaou, A.C.(1988). Dichotic-listening evidence of right-hemisphere involvement in recovery from aphasia following stroke. *Journal of Clinical Experimental Neuropsychology,* 10(4), 380-386. Abstract retrieved December 5,2002,from Pubmed database.

Musiek, F.E., Baran, J.A & Pinheiro, M.L.(1994). Neuroaudiology: Case studies. San Diego: Singular Publishing Group, Inc.

Naatanen, R.(1975).Selective attention & evoked potentials in humans: A critical review. *Biological Psychology,* 2,237-307

Niccum, N., Rubens, A.B.,& Seines, O.A.(1983). Dichotic listening performance, language impairment, and lesion location in aphasic listeners. *Journal of Speech and Hearing Research,* 26(1), 42-49.

Okada, Y.C., Kaufman, L., & Williamson, S.J.(1983). The hippocampal formation as a source of the slow endogenous potentials. *Electroencephalography and Clinical neurophysiology,* 55,417-426.

Onofrj, M., Fulgente, T., Thomas, A., Locatelli, T., & Comi, G. (1995). P300 asymmetries in focal brain lesions are reference dependent. *Electroencephaloagraphy and Clinical Neurophysiology,* 94,432-439.

Paul, D.D. & Sutton, S.(1972).Evoked potential correlate of response criterion in auditory signal *detection.Science,* 177,312-320.

Picton, T.W,(1990). Auditory evoked potentials. In Daly.D.D., *Pedley, T.A.(Ed.),Current Practice of Clinical Electroencephalography* [2nd Edition],(pp625-678).NewYork:Raven press.

Polich, J.(1989). P300 from a passive auditory paradigm. *Electroencephalography and Clinical neurophysiology,* 74(4), 312-320.

Ponton, C.W.,& Don, M.(1995).The mismatch negativity in cochlear implant users .*Ear and hearing,* 16,130-143.

Saravanan, E.(1997).Age related changes in P300. *Unpublished Independent project*, University of Mysore, Mysore.

Selinger, M., Prescott, T.F. & Shucard, D.W.,(1989).Auditory event related potential probes and behavioral measures of aphasia. *Brain and language,* 36,377-390.

Shanks J., & Ryan, W. (1976). A comparison of aphasic and non-brain-injured adults on a dichotic CV-syllable listening tasks. *Cortex,* 12(2), 100-112. Abstract retrieved December 5,2002,from Pubmed database.

Sivaprasad, M.R. (2000). MMN in ears with Sensori-neural hearing loss. *Unpublished Masters Dissertation,* University of Mysore, Mysore

Srividya, B.A. (2002). MMN for duration deviance: study on normal children, adults & children with learning disorder. *Unpublished Masters Dissertation,* University of Mysore, Mysore.

Tonnquist-Uhlen, I. (1996).Topography of auditory evoked long -latency potentials in children with severe language impairment: The P2 and N2 components. *Ear and Hearing,* 17,314-326.

Wertz, R.T., Auther, L.L., Sims, P.B, Khalil, R.A., Kirshner, H.S., & Duncan, G.W. (1998). A comparison of the mismatch negativity (MMN) event related potential to tone and speech stimuli in normal and aphasic adults. *Aphasiology,* 12,7/8,499-507.

Woods, D.L., Knight, R.T. & Neveille, H.J.(1984). Bitemporal lesions dissociate auditory evoked potentials and perception. *Electroencephalography and Clinical neurophysiology,* 68,132-148.

Yingling, C.D., Hosobuchi,Y. & Harrington, M.(1990). P300 as a predictor of recovery from coma. *Lancet,* 336,873-878.