AUDITORY EVOKED POTENTIALS IN STUTTERERS

Register No. M9902

Independent Project submitted as part fulfillment for the First year MSc. (Speech and Hearing), submitted to the University of Mysore, Mysore.

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

MAY 2000

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

CERTIFICATE

This is to certify that this Independent Project entitled **"AUDITORY EVOKED POTENTIALS IN STUTTERERS'** is the bonafide work in part fulfillment for the degree of Master of Science (Speech and Hearing)of the student with *Register No.* M9902

DIRECTOR
All India Institute of Speech & Hearing
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Mysore
May, 2000

CERTIFICATE

This is to certify that this independent project entitled "AUDITORY EVOKED POTENTIALS IN STUTTERERS" has been prepared under my guidance and supervision.

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May, 2000

DECLARATION

This Independent Project entitled: **"AUDITORY EVOKED POTENTIALS IN STUTTERERS"** is the result of my own study under the guidance of **Ms. Vanaja,** C.S., Lecturer in Audiology, AIISH, Mysore, and has not been submitted earlier at any University for **any** other diploma or degree.

Mysore **Register No. M9902**

May, 2000

Dedicated

to

Amma and Appaji

Acknoeledgments

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I wish to than myfamily, my relatives, all my juniors and seniors for their support and well wishes.

A special message for all fellow "Crusaders* keep going guys, nice and strong. Bad days will have to end soon. Good luck!

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INTRODUCTION

Stuttering is defined in the International Classification of Disorders as " disorders in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at the time is unable to say it because of an involuntary, repetitive prolongation / cessation of a sound " (World Health Organisation, 1977). This definition complies with the standard definition of Wingate which regards repetitions and prolongation of a sound or a syllable (audible or silent) as crucial elements in stuttering (Wingate, 1964).

Several theories have been put forth to understand the cause and the nature of stuttering. However none of them have been able to do so adequately. Bloodstein (1981) divided these theories into three groups : stuttering as a neurotic response, stuttering as a learned behaviour, and stuttering as a physiological deficit.

Stuttering as a Neurotic Response:

Psychoanalytic explanations for stuttering were prevalent 40 years ago (FenicheL.1945). Stuttering was variously viewed as satisfying oral or anal erotic needs and/or as an expression of repressed hostility. Thus, the moment of stuttering represented the unconscious need to suppress speech, Implicit in these and later formulations (Glauber, 1958 ; Travis, 1957) is that stuttering is but one symptom of a neurotic conflict which would also be evident both in other neurotic symptoms and in disturbed interpersonal relationships, particularly those with parents (Andrews et al., 1983).

Stuttering as Learned Behaviour:

Several attempts have been made to arrive at concepts regarding stuttering within the framework of learning theory (Bratten and Shoemaker, 1967; Shames and Sherrick,1963; Sheehan, 1953). These formulations view stuttering either as an instrumental avoidance response or as an approach avoidance conflict. Some theories attribute stuttering to the interaction of at least two distinct behavioral phenomena (Bloodstein,1993).

Stuttering as a **Physiological Deficit:**

These models refer to the period of stuttering as a momentary failure in performing complicated coordinations involved in fluent speech (West, 1958 ; Adams, 1974,1978 ; Perkins et al., 1976, 1979 ; Orton and Travis, 1978). These "breakdown theories" originate from the proposition that stutterers have a reduced physiological capacity to coordinate speech (Andrews etal., 1983).

The impetus for studying auditory function in stutterers has arisen from two major theories about the etiology and possible site(s) of lesion for stuttering and stuttering behaviours. On the one hand some researchers have used measures of auditory function to investigate cerebral dominance of language. This pertains to the theory proposed by Orton (1928) and Travis (1931) that stutterers do not develop complete dominance for left hemisphere for language, and/or for control of the motor activity of the speech mechanism. Differences between stutterers and non-stutterers would suggest a possible site of lesion in the cortical area (Stager, 1990).

On the other hand, some researchers have used measures of auditory function to investigate possible abnormality along the auditory pathway. This pertains to the hypothesis that stuttering is related to problems with auditory feedback during speech production. Since a majority of stutterers have normal hearing sensitivity, any problems with auditory feedback are more related to deficits in the central, rather than peripheral auditory system (Stager, 1990).

The defect may have two possible forms, a central auditory processing problem which is demonstrated in certain tests involving an auditory comprehension task, or it can be restricted to the speech-auditory feedback, and then would be found only during actual speech production (Postma and Kolk,1992).

A number of investigations have been undertaken to study the auditory processing in stutterers. However, the results are not conclusive. Also, linguistic behavioral tests using speech material are language specific and therefore difficult to use in a multilingual county like India (Radhika, 1998). Hence, objective tests like evoked potentials are more valid and useful, as they are direct reflectors of changes in the nervous system as the stimuli is processed.

The studies investigating auditory processing dysfunction in stutterers using Auditory evoked potentials have yielded conflicting results. Reports of anomalies in auditory brainstem evoked responses in stutterers include increased interpeak latency differences especially between waves I and V (Blood and Blood, 1984 ; Stager, 1990). However some investigations have

shown no significant differences between stutterers and non-stutterers in terms of interpeak latency intervals (Newman, 1985 ; Blood and Blood, 1990 ; Decker etal., 1982).

The results on Auditory Middle latency responses have been quite equivocal. Hood (1987) and Vikram (1997) found latency of wave Pb to be significantly longer for adult stutterers than for the controls whereas Dietrich et al .(1995) have reported a shorter Pb latency in stutterers compared to the non-stutterers. Finitzo et al. (1990), found reduced amplitudes for waves PI, Nl and P2 for mild stutterers when compared with normals.

There is a need for further investigations to contribute in the direction of substantiating the findings obtained through evoked potentials in the past. Also there is a dearth of studies investigating the auditory pathway at both the brainstem and cortical level in a single subject. Hence, doing so would help in deterrmning whether there is any disturbance in the auditory processing of stutterers, and identifying the site of dysfunction.

Aim of the Study:

The present investigation aimed to study the following auditory evoked potentials in stutterers.

- Auditory Brainstem Evoked Potentials
- Auditory Middle Latency Evoked Potentials
- Late Latency Exogenous Potentials
- Late Latency Endogenous Potentials

REVIEW OF LITERATURE

All the studies that have used measures of auditory function in stutterers are directed to investigate the cerebral dominance for language and/or probe into the anomalies in the auditory pathway. Hence, the following review of research will be dealt with reference to the above mentioned ideas.

Stuttering and Cerebral Dominance:

Initially Orton (1938) and Travis (1931) proposed that many children go through a stage of disfluency because language has not yet lateralised to the appropriate hemisphere. As the child grows older, the language lateralizan'on process becomes more complete and the disfluency disappears. However, a subgroup retain their abnormal bilateral_____representation and continue to stutter.

Consequent to the Orton-Travis thesis many investigators addressed the prevalence of right and left handedness amongst stutterers, contending that if stuttering were a disorder due to abnormal cerebral laterality, such an abnormality should be reflected in a different matrix of handedness between stutterers and non-stutterers. Due to varying definitions of handedness and varying methods of ascertaining the presence / absence of stuttering in populations, investigators derived conflicting data and arrived at disparate results (Bryngelson, 1935 ; Milisen and Johnson, 1936 ; McAllister, 1937 ; Daniels, 1940 ; Spadino, 1941 ; Meyer, 1945 ; Despert, 1946).

The Orton-Travis thesis lay fairly dormant until Jones (1966) noted that all four stutterers who were to be operated upon for a cerebral disease, became aphasic following Amytal injection in either their right or left carotid artery (Wada test ; V/ada and Rasmussen, 1960). This suggested that both hemispheres were contributing significantly to language production. A repeat Wada test elicited aphasia only after injection to the non-operated side. The patients no longer had bilateral speech representation and they no longer stuttered. However subsequent investigations by Andrews, Quinn and Sorby (1972) and Luessenhop, Boggs, Laborwit and Walle (1973), using the Wada technique and controlling for some of the confounding variables in the Jones' study failed to replicate the earlier observation of bilateral motor speech control in stutterers.

Investigations using the Wada technique are difficult to interpret for several reasons: handedness was different; neurological integrity was heterogeneous; onset of stuttering was not always controlled; age of subjects varied; a number of potentially important observations were not always adequately explained (Moore, 1984).

Other Methods used in Studying Hemispheric Processing in Stutterers

Tachistoscopic Visual Procedures:

Most researchers using Tachistoscopic Visual Procedures have incorporated meaningful linguistic stimuli in a linguistic decision task (Hand and Haynes, 1983 ; Moore, 1976 ; Plakosh, 1978). Moore (1976)

reported a left field (right hemisphere) preference in most of the stutterers participating in the study.

Electromyographic Studies:

Travis (1931) hypothesized that stuttering results from the asynchronous arrival of nerve impulses in the bilaterally paired jaw muscles. In 1934, Travis presented EMG data recorded from the left and right masseter muscles of twenty four adult stutterers and non-stutterers. He reported that action potentials from normal subjects were " practically identical", while those from stuttering subjects were " strikingly different". Other EMG investigations (Morley, 1935 ; Steer, 1937 ; Strother, 1935) seemed to support the findings of Travis (1931) and it was believed that competition between the cerebral hemispheres during motor speech behavior resulted in out-of-phase arrival of action potentials that disrupted speech.

Williams (1955) failed to find significant differences in amplitude and timing of action potentials between the two sides of the jaw in stutterers and non-stutterers. Differences found between the stuttering and non-stuttering groups were attributed to the excessive muscular tension and different patterns of jaw movements accompanying stuttering. Hence the electromyographic differences seen were viewed as an outcome of stuttering than its cause.

Alpha Recordings:

Increased suppression of alpha brain wave frequency (8 to 13 Hz) has been demonstrated over the hemisphere, primarily processing specific kind of information under a specific task condition. An advantage of this procedure being that hemispheric processing using a variety of stimuli including more natural units of language (phrases, sentences and connected discourse) can be studied over time.

Douglass (1943) and Knott and Tjossen (1943) found that stutterers as a group had less percept time alpha in their right occipital area compared to their left occipital areas during silence, while the non-stutterers evidenced otherwise.

Moore and Haynes (1980) found that comprehension of connected verbal discourse was unaffected in male stutterers who also demonstrated reduced right hemispheric alpha, a finding which could also reflect the right hemisphere's superiority in processing semantic aspects of language. He suggested that" stuttering may emerge when both hemispheric processing of incoming information and motor programming of segmental linguistic units is in the right hemisphere (a non-segmental processor). These processing differences may be related to an inability, under certain circumstances, to handle the segmentation as it relates to motor programming in some stutterers."

McFarland and Moore (1982), Moore, Craven and Faber (1982), Moore and Haynes (1980a), Moore and Lang (1977), Moore and Lorendo

(1980) found that alpha was suppressed over the right posterior temporo-parietal areas in stutterers. Moore and Haynes (1980b) found that stutterers recalled fewer words across word lists than non stutterers. These findings may reflect the stutterers right hemisphere verbal short term memory to have a shorter span (Zaidel, 1979).

Boberg et al. (1983) gathered hemispheric alpha asymmetry data from anterior and posterior brain sites before and after treatment. Prior to treatment stutterers showed less alpha over the right posterior frontal region for verbal tasks, while after treatment there was less alpha over the left posterior frontal region. These findings suggest that alpha ratios over frontal motor areas may implicate motor programming aspects of stuttering and that increased fluency accompanying treatment, shifts alpha suppression from the right to the left hemisphere. Similar results were reported by McFarland and Moore (1982) who recorded alpha hemispheric asymmetries before and after treatment. The results showed right hemispheric alpha suppression during baseline (relatively high frequency of stuttering) with a gradual and consistent suppression of left hemisphere as fluency increased. This indicates that following treatment, which increases fluency, stutterers apparently show a shift to more segmental left hemispheric processing strategies.

On the other hand Pinsky and McAdam (1980) performed alpha recording on five adults stutterers and five fluent speakers over hemispheres during performance of cognitive tasks, contingent negative variation with either an articulatory or bilaterally symmetrical response and readiness potential with same responses. All subjects showed consistent patterns of

cerebral laterality indicative of localisation of speech functions in the left hemisphere.

Cortical Blood Flow:

Wood, Stumps and Sheldon and Proctor (1980) subjected two stutterers to cerebral blood flow measurements while reading aloud. During disfluent moments, both stutterers showed higher cortical blood flow in Broca's area on the right compared to the left hemisphere. However during fluent speech , a greater flow was observed in the left hemisphere as compared to the right. These results provide support to the relationship proposed between stuttered speech and hemispheric processing reported by Boberg et al. (1983) and McFarland and Moore (1982).

CT and PET Scans:

CT and PET Scans have also been used to study Cerebral Dominance. Wu et al. (1997) investigated the role of the dopamine system using 6FDOPA for PET on three patients with moderate to severe developmental stuttering in comparison with six normal controls. Stuttering subjects showed significantly higher 6FDOPA uptake than normal controls in medial prefrontal cortex, deep orbital cortex, insular cortex, extended amygdala, auditory cortex and caudate tail. Elevated 6FDOPA uptake in ventral limbic cortical and sub-cortical regions is compatible with the hypothesis that stuttering is associated with an overactive presynaptic dopamine system in the brain regions that modulate verbalization.

Fox et al.(1996) using PET scans showed stuttering induced widespread over activation of the motor system in both cerebrum and cerebellum with right cerebral dominance. Stuttered reading lacked left lateralized activations of the auditory system which are thought to support the self monitoring of speech and selectively deactivating multiple neural systems used for speaking.

Strub and Black (1987) tested two siblings for stuttering for speech - language, neurological and neuropsychological functions, dichotic listening auditory evoked responses. EEG and CT scan asymmetry. The data showed abnormal cerebral dominance on variables investigated. CT scans showed atypical asymmetry especially in occipital regions. The above findings suggest hemispheric processing differences between stutterers and non-stutterers.

Auditory Evoked Potentials:

Auditory Evoked Potentials have been used for determining the hemispheric dominance as well, to investigate the central auditory processing in stutterers. Some of the studies in which hemispheric processing is investigated through measures of auditory evoked potentials are discussed here. Averaged evoked responses (AER) are a neuro-electrical measure of the cortical activity. It is a non invasive technique where changes in cortical electrical activity are averaged over trials.

Ponsford, Brown, Marsh and Travis (1975) used AER to investigate hemispheric differences between stutterers and non-stutterers. The stimuli used were meaningful words embedded in phrases. Stutterers showed greater inter-subject variance as opposed to normals whose responses were most different in the left hemisphere.

Zimmerman and Knott (1974) recorded the Contingent Negative Variation; Two control conditions with non-verbal stimuli (tones) requiring a non-verbal response were compared with two experimental conditions in which meaningful linguistic stimuli (words) were used. In one experimental condition the subjects indicated whether or not they thought they would stutter on the word presented by pushing one of the keys marked "yes" and "no". In the second condition subjects were instructed to speak each word upon signal. Results revealed differences between stuttering and non-stuttering groups for frontal electrodes placed over Broca's area on the left and its contralateral homologue on the right. They stated " when processing verbal stimuli, stutterers appear to show more variable interhemispheric relationships than the non-stutterers."

Molt and Brading (1994) used a sixteen channel topographic brain mapping procedure to examine hemispheric patterns for dichotically presented consonant-vowel stimuli and noted the P300 and N200 components. No ear advantage differences were observed between stutterers and non-stutterers. Stuttering subjects demonstrated significantly less cross hemispheric amplitude differences for the P300 component. Similar results were observed for the N200 component, thus indicating differences in hemispheric activity patterns.

Ferrard et al. (1991) performed simultaneous measurements of P300 brain potentials (using tones of 500Hz. and 2000Hz as frequent and in frequent stimuli respectively), and laryngeal positioning prior to vocal fold closure and vocal fold vibration. No significant differences were found in the temporal patterning of three activities between the ten stutterers and ten non-stutterers, who participated in the study.

Pinsky and McAdam presented data of the Contingent Negative Variation recording using a non-linguistic stimuli (lOOOHz tone) under two response conditions. One condition required subjects to press a button with each thumb simultaneously when a tone stopped. For the second condition subjects uttered a fluent word at the termination of the tone. The results provided insufficient evidence to support hemispheric asymmetries between stutterers and non-stutterers. In the various above mentioned studies, differences may well be due to the differences in behavioral tasks and the nature of stimuli used.

Dichotic Listening:

Dichotic listening paradigms have been used in the largest number of investigations exploring hemispheric processing strategies in stutterers. These paradigms, according to some investigators, provide a relatively simple test of the Orton-Travis thesis. The stutterers lack of suitable hemispheric dominance should therefore be revealed through an appropriate dichotic test (Rosenfield and Jerger, 1985).

One of the early investigations using dichotic listening was by Curry and Gregory (1969). They tested twenty adult stutterers and twenty non-stutterers as controls, all of who were reportedly right handed. The dichotic tests included in their study were the dichotic word test, dichotic environmental sound test and the dichotic pitch discrimination test. The dichotic word test involved recognition of pairs of highly familiar consonant-vowel-consonant words presented in groups of six pairs with 0.5 seconds separating each pair. After presentation of each group of six pairs, subjects were to recall the twelve words in any order. Seventy five percent of non stutterers demonstrated right ear advantage i.e. their right ear scores were higher than their left ear. This was true for only forty five percent of the stutterers. The mean of absolute difference between two ears in non-stutterers was twice as greater as that seen in stutterers.

Sussman and MacNeilage (1975) employed a dichotic test paradigm and pursuit auditory tracking. Their experiment involved matching the frequency of a variable tone in one ear to the frequency of an externally varied tone in the other ear. The former tone was altered by a transducer attached to the tongue / jaw. The subject varied the frequency of this tone by appropriately moving the tongue / jaw. Results revealed no differences in the dichotic listening paradigm between stutterers and non-stutterers. On the tracking paradigm however, normals had a right ear advantage whereas stutterers did not.

Tsunoda and Moriyama (1972) conducted the Tsunoda's cerebral Dominance test and standard audiometry on fifty-seven adult Japanese stutterers. Seventy nine percent of normal controls showed a preference for

vowel sounds in the left ear, but this pattern existed only for about thirty nine percent of the stutterers. This suggested the existence of a sub-group among stutterers in whom stuttering may be due to abnormal cortical function resulting from niinimal brain damage. No information regarding subjects handedness and age was provided.

Blood and Blood (1989) compared eighteen male eighteen female stutterers between the ages of eighteen to thirty six years, with a matched control group. All subjects were right handed and were to respond to a six item dichotic word test using a gestural double response paradigm. Results revealed significant differences between the stutterers and the controls in the magnitude of ear preference in both male and female stutterers. Blood (1985) investigated seventy six stutterers and seventy six non-stutterers in the age range of seven to fifteen years, using dichotically presented synthetic syllables. Results indicated that although the direction of ear preferences was same for stutterers and non-stutterers, the magnitude of ear preferences for the two groups was significantly different Fifty five percent of the stutterers showed a right ear preference. These subjects formed the largest group followed by the ambilateral group and left ear preference group. According to them reporting mean data for stutterers in dichotic listening paradigms is inappropriate without a sub-group and individual data analysis.

Strong and Frick (1983) administered dichotic CV listening task to ninety rigjht handed boys, in the ages of five, seven and nine years, half of the subjects being stutterers and the other half being non-stutterers. Two and a half times as many stutterers as non-stutterers were found to display either a left ear or a no ear advantage.

Quinn (1972) investigated hemispheric processing using the dichotic listening paradigm in sixty eight right handed stutterers and age/sex matched controls. No significant differences between the two groups were observed. Dorman and Porter (1975), evaluated sixteen right handed adult stutterers with the controls on a task if writing down the responses to synthetically generated CV dichotic stimuli. There was no marked difference between stutterers and non-stutterers. Also Slorach and Noehr (1973) examined fifteen stutterers in the age range of six to nine years. They presented dichotic digit pairs and tested not only free recall of digits but also reports from the stutterers as to what digit they heard in which particular ear. Stutterers scores were akin to those of the controls. Gruber and Powell (1974) tested twenty eight right handed fluent and disfluent children using dichotic digit pairs. Free recall reports of both the groups failed to reveal significant differences between them. Here, one should note that since four percent of children stutter and only one percent of adults stutter, the mechanism /type of stuttering may be different from that among adults.

Manning and Reinsche (1978) tested the auditory assembly abilities of thirty stuttering and thirty non-stuttering children from first to fourth grade matched for age, grade level, sex and mis-articulations. They were presented with meaningful consonant-vowel-consonant syllables with four silent interphonemic intervals (100, 200, 300 and 400msecs.). There was no significant difference in the overall performance between the two groups.

Pinsky and McAdam (1980) tested five adult stutterers and five fluent speakers, all of who except one (stated to be " weakly right handed ") were

right handed. Both groups yielded similar scores on the dichotic listening procedure.

Sapna (1999) tested twenty young adult male stutterers in the age group of seventeen to thirty years on the dichotic CV paradigm at various lag times of 0, 30 and 90msecs. Of the twenty stutterers, five were mild, nine moderate and six severe grade stutterers. Significant right ear preference was not demonstrated by the stutterers at 0 and 30msec lag times. Also scores were seen to diminish with increase in the severity of stuttering.

Thus, studies using dichotic listening paradigms have yielded conflicting results regarding cerebral dominance in stuttering, one of the reasons being an array of contaminating variables influencing the results. Some of these are : Handedness, order of reporting sounds as per instructions and failing to confirm the ear advantage by employing a test retest experiment (Rosenfield and Jerger, 1985).

The non-auditory tests conducted to determine the cerebral dominance in stutterers have indicated insufficient cerebral dominance for language. However the audiologjcal investigations have yielded equivocal results.

Stuttering And Auditory Feedback:

The notion that stuttering might be due to a defect in the auditory feedback mechanism has been discussed by several authors (Fairbanks, 1954; Mysak, 1960; Butler and Stanley, 1966; Timmons and Boudreau,

1972). The central nervous system dysfunction can affect the auditory feedback and fluency relationships in one of the two ways (Toscher and Rupp, 1978).

1. A neurological dysfunction may block or distort the feedback signal or it may cause an inability to rectify correctly the observed disfluency.

2. The feedback might be distorted before or during its transmission through the neurological system by non-neural physiological factors.

Phase Disparity Between AC **and BC Tones:**

In 1957, Stromsta exploited the fact that two pure tones, 180 degree out of phase but equal in frequency and amplitude, will cancel each other out. Stutterers and normal speakers listened to an AC tone introduced to the ear and to a bone conducted tone of the same frequency simultaneously introduced at the teeth. Subjects were asked to vary the phase and amplitude of the AC tone until a critical adjustment was achieved at which no sound was audible to them. There was a significant difference between stutterers and non-stutterers in the relative phase angle of the AC and BC sounds at 2000Hz.

Using a similar method, Stromsta (1972) noted an unusual phase disparity between stutterers left and right ears. The stutterers adjusted the amplitude and phase of the two AC tones heard in either ear, until they cancelled an identical BC tone. At the point at which cancellation was achieved, the air conducted tones of the two ears had a phase disparity at

several frequencies that was twice as wide for the stutterers as for the non-stutterers. Stromsta (1957) concluded that stutterers as a group tended to differ from normals in transmission of feedback signals.

Acoustic Reflex Studies:

The acoustic reflex due to its concurrent presence / initiation during the vocalization process, was investigated by some authors. Webster and Lubker (1968), suggested that temporal abnormalities of the acoustic reflex unique to stutterers changes the synergy/ synchrony of air and bone conducted components of the speech signal in a way as to initiate and maintain stuttering behaviour.

Shearer and Simmons (1965) investigated stapedius muscle activity in stutterers and non-stutterers during ongoing speech. In stutterers, the parallelism between stapedius muscle activity and vocalization was less consistent. The muscle activity seemed to be delayed with respect to vocalization. In general, however differences between the two groups was not striking.

Hall and Jerger (1978) compared the acoustic reflex to external sound in stutterers and controls. Reflex threshold was equivalent in the two groups, but reflex amplitude was smaller in the stuttering group. Hannley and Dorman (1982), however failed to note any differences between stutterers and non-stutterers.

These findings do not clarify the relationship between acoustic reflex and stuttering.

Tests of Central Auditory Dysfunction:

The auditory feedback defects in stutterers might only be a part of a more comprehensive disorder of function in their central auditory perceptual mechanism (Rosenfield and Jerger, 1985). A number of investigations have attempted to explore this question using clinical audiometric measures and techniques developed specifically to assess the central auditory dysfunction. Both behavioral and electrophysiological techniques have been used for this purpose.

Behavioral Tests:

In 1959 Rousey et al. investigated sound localization abilities in twenty normal, seven hemiplegic, twenty stuttering and twenty emotionally disturbed children to reveal a relatively poorer sound localizing ability. Gregory (1964) further pursuing audiometric studies contended that there was no significant difference between adult stutterers and non-stutterers in tests of sound localization, binaural loudness balance and understanding of speech by frequency filtering. This was supported by Kamyama (1964) and Asp (1968).

Jerger and Hall (1978) assessed central auditory function in ten stutterers and ten non-stutterers. Performance of the two groups was

compared for seven audiometric procedures including acoustic reflex between stutterers and non-stutterers.

The above findings do not clarify the relationship between acoustic reflex and stuttering.

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The findings with regard to the tests of central auditory processing in stutterers are conflicting. However, it is suggested that a test battery approach be followed in assessing the central auditory functioning in stutterers as it permits comparison of the performance on several measures of auditory function (Hall and Jerger, 1978).

Electrophysiological Tests:

Auditory evoked potentials provide an objective measure of the central auditory processing. Blood and Blood (1984), performed brainstem evoked response testing on eight adult stutterers (four severe and four

moderate) and eight non-stutterers. Stutterers demonstrated prolonged central conduction time as measured by the interpeak latency differences between waves I to V. Five of the stutterers manifested abnormalities unilaterally, while three of the subjects showed abnormal responses bilaterally. No relationship was found between brainstem evoked responses and the severity of stuttering.

Stager (1990) measured interpeak latency differences between waves I and V, amplitude ratio between waves V and I and latency shifts in wave V between low and high stimulus repetition rates in ten male stutterers and twelve male non-stutterers (with normal hearing sensitivity). As a group, stutterers did not differ significantly from non-stutterers on any of the measures. Individually half the stutterers demonstrated latencies greater than two standard deviation from non-stutterers means on at least one measure.

Newman et al. (1985) obtained brainstem evoked responses of both the ears of active stutterers, recovered stutterers and non-stutterers, both male and female adults, at click rates of 11.1 and 71.1 per second. No significant differences were obtained between stutterers and non-stutterers. However female subjects (stutterers and non-stutterers) showed faster neural conduction times than males.

Smith, Blood and Blood (1990), recorded the brainstem evoked responses when the subjects engaged in overt speech, whispering, silent articulation and covert verbal rehearsal tasks. Results revealed that stutterers demonstrated significantly larger wave V to wave I amplitude ratio than non-stutterers. However, no significant differences were found between

stuttering and non-stuttering subjects for absolute / interpeak latencies of the waves during the verbal rehearsal tasks.

Decker et al. (1982), compared the latencies of waves I, in and V, interpeak latency differences between waves I and V, the amplitude of wave V and the comparison between the right and left monoaural stimulation waveforms with the binaural stimulation waveforms. No abnormality in the responses of stutterers was observed.

According to Stager (1990), lack of significant differences between stutterers and non-stutterers can be due to choice of those parameters which are associated with assessment of auditory sensitivity and not necessarily intactness of the brainstem. This means to say that measures of auditory sensitivity which include latency of wave V, slope of the latency intensity function and intensity required to observe the first definitive response, do not always assess the brainstem pathway. The measures for the latter would be amplitude ratio of wave V to I, interpeak latency differences between waves I and V, and latency shift in wave I when the stimulus presentation rate is increased.

Pool, Freeman and Finitzo (1987) identified cortical dysfunction over the medial frontal and left temporal cortex in three stutterers using multichannel long latency evoked potential recording. Finitzo et al. (1990), tested twenty adult stutterers and examined PI, Nl and P2 components of the auditory evoked responses. They found no significant differences in terms of latency, however amplitude was reduced in mild to moderate stutterers as compared to normals but not reduced for the severe

stutterers. Hence reductions in bihemispheric amplitude was noted although reductions were persistently greater over the left hemisphere. This suggested a left temporal cortex dysfunction in stutterers.

Dietrich et al.(1995), recorded middle latency from ten male stutterers and ten controls using a variety of filter passbands in response to clicks presented binaurally at various rates. The latency of Pb wave was found to be significantly shorter in groups of subjects who stuttered. Hood (1987) and Vikram (1997) reported increased Pb latencies in the stuttering group. Vikram (1997) attributed differences in findings of Diedrich et al. (1995) and Hood (1987) to the differences in stimulus parameters used in the two studies. The conflicting results to the results obtained by Dietrich et al. in his study even on using similar stimulus parameters was accounted by the differences in subject selection, i.e. the various subcategories / subgroups in the stuttering population would manifest a variety of results in the auditory middle latency responses.

Hence, there is a lot of controversy as to the performance of stutterers on tests assessing tests of central auditory processing. It may be hypothesised however, that a central auditory processing problem may better manifest on using speech stimuli to record the evoked potentials than using non-speech stimuli.

METHODOLOGY

The present investigation aimed to study the following auditory evoked potentials in stutterers:

- Auditory brainstem evoked potentials (ABR)
- Auditory middle latency evoked potentials (AMLR)
- Late latency exogenous potentials (LLR)
- Late latency endogenous potentials (P300 and Mismatch Negativity)

Subjects:

Sixteen stutterers, ten males and six females between the ages of six to thirty years participated in the study. The range of stuttering severity was from mild to severe as determined by the Stuttering Severity Index (SSI).

Other criteria for subject selection were as given below:

- No history or present complaint of hearing loss or any other otological problem
- No concomitant speech-language disorder
- No history of a neurological disorder or gross neurological symptoms
- No intellectual deficits

Instrumentation:

A calibrated Madsen OB-822 with TDH-39 earphones lodged in MX-41/AR ear cushions was used for pure tone audiometry. A calibrated Grasen-Stadler -33 Middle Ear Analyser, Version 3.1 was used to perform immittance audiometry. The electrophysiological unit, Biologic Auditory Evoked Potentials system with the following accessories was used to record the auditory evoked potentials.

- Silver Chloride disc electrodes for recording the potentials.
- TDH-39 earphones with MX-41/AR ear cushions, to present the stimulus.

Testing Procedure:

Pure tone audiometry was conducted to ensure normal hearing sensitivity (thresholds below 25dBHL) at octave frequencies from 250Hz to 8000Hz.

Immittance evaluation was performed on the subjects to check for normal middle ear functioning indicated by a static compliance between 0.5-1.75ml, a peak compliant pressure between -lOOdaPa to +60daPa, and presence of both ipsilateral and contralateral reflexes at100dBSPL for the frequencies 500Hz,1000Hz and 2000Hz.

The auditory evoked potentials were recorded in the following order.

l.ABR

2. MLR

3. Mismatch Negativity (MMN)

4.LLR and P300

1. **ABR:**

The ABR was recorded for each ear at three repetition rates. The electrode sites chosen and their connections to the electrode box were made as shown in figure M. 1.

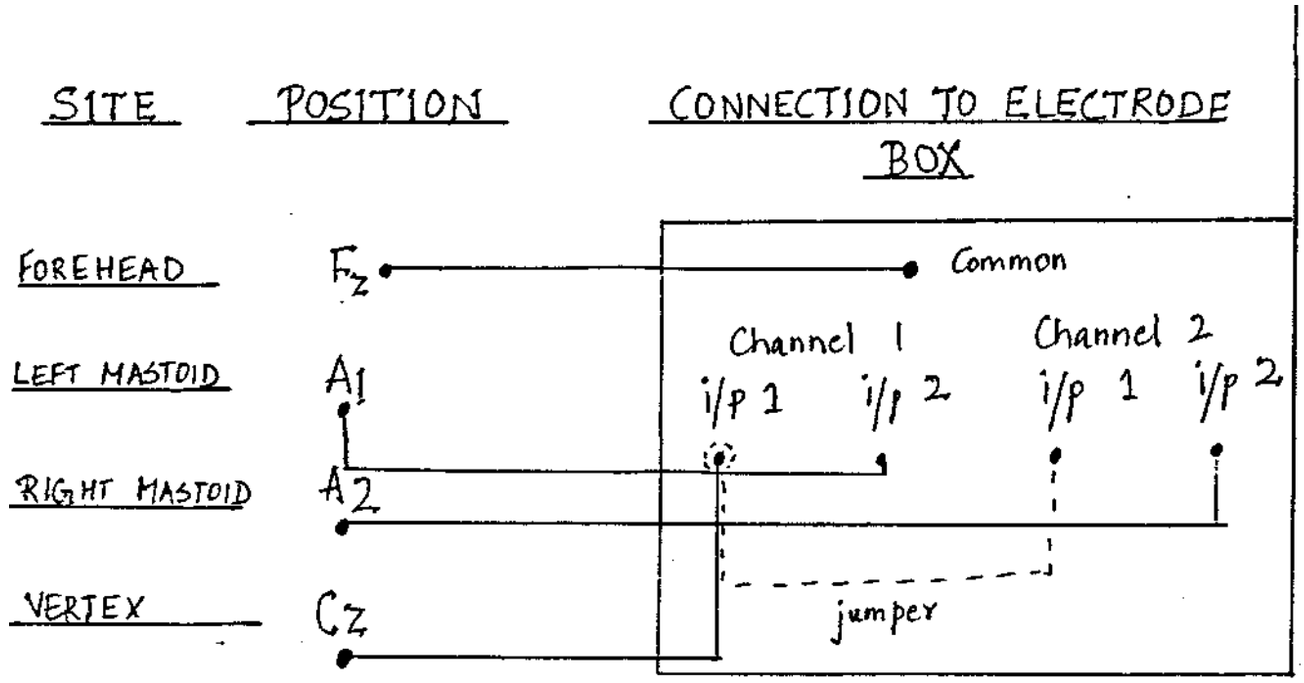


Figure M .1. : Electode sites and their connection to the electrode box.

The electrode impedance was kept less than 5k-ohms and the interelectrode impedance was kept less than 2k-ohms.

Instructions:

The subjects were asked to sit comfortably on the chair and relax. They were instructed to avoid extraneous movements of the head, neck and jaw during the course of recording the potentials.

Stimulus Parameters

Stimulus : clicks
Polarity : rarefraction
Rate : ll.lc/s, 60.1c/s, 90.1c/s
Filter Setting : 100Hz-3kHz
Montage : Cz/Al :: Cz/A2
Transducer ; : Headphones
Maximum Stimuli : 2000
Intensity : 70dBnHL

2. **MLR:**

The middle latency responses were recorded from both the ears. The electrode impedance was kept less than 5k-ohms and the interelectrode impedance was kept less than 3k-ohms.

For recording these potentials, the electrode placement, instructions and stimulus parameters were same as that used to record the ABR, but for the parameters mentioned below.

Repetition Rate : 9.1/s
Filter-Setting : 5-250Hz

3. MMN:

The electrode sites chosen with their connections to the electrode box were made as shown in figure M.2. The electrode and the interelectrode impedance were kept less than 5k-ohms and 2k-ohms respectively.

Instructions:

The subjects were asked to sit comfortably on the chair, relax and shut their eyes. They were instructed to avoid any extraneous movements of the head, neck and jaw during the course of recording the potentials.

Stimulus Parameters

Stimulus : alternate tone bursts
Polarity : rarefaction
Rate : 1.1/s
Filter Setting : 0.1-3.0kHz
Montage : Cz/Al :: Pz/A2
Transducer : Headphones
No. of Stimuli : 100 artifact free (odd) stimuli
Intensity of the
frequent stimuli : 65dBnHL
infrequent stimuli : 62dBnHL
Probability ratio : 5
Frequency of both the stimuli : 1000Hz.

4. LLR and P300:

The electrode placement used to record these two potentials was kept the same as for recording the MMN i.e. as shown in figure M.2.

Instructions:

The following instructions were given to the subjects:

"You will be presented with two stimuli / tones, one of which will be more frequent than the other. Count the number of rarely occurring stimuli."

Stimulus Parameters:

The parameters set to record these potentials were same as those used for recording the MMN, but for the parameters mentioned below:

No. of Stimuli	:	60 artifact free (odd) stimuli
Frequency of the		
frequent stimuli	:	lOOOHz
infrequent stimuli	:	2000Hz
Intensity of both the stimuli	:	70dBnHL

Analysis:

The following measures were studied from the ipsilateral and contralateral waveforms of ABR.

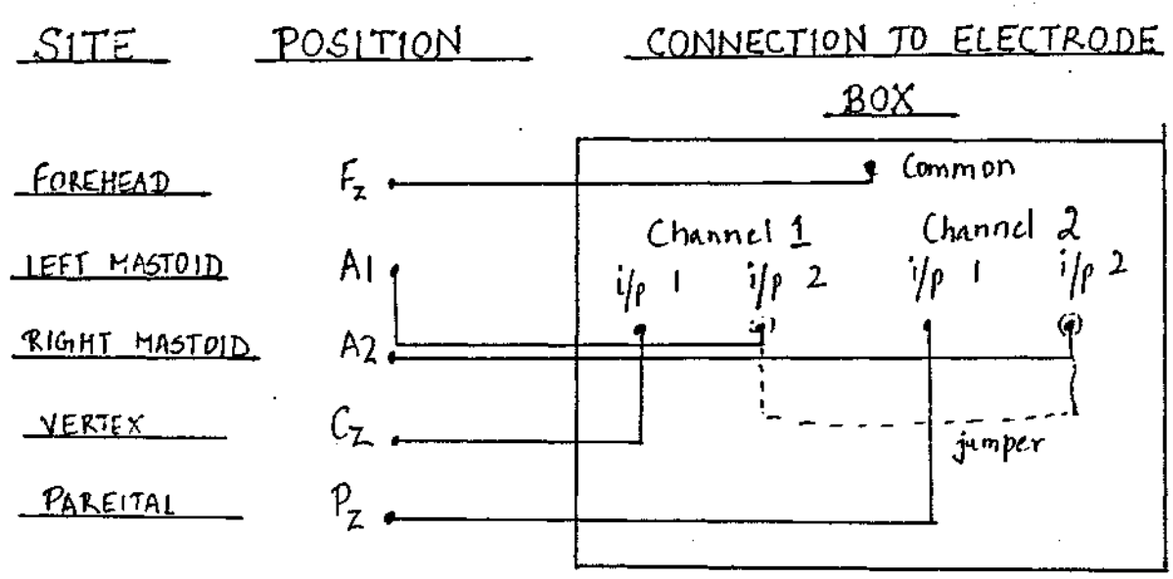


Figure M.2. : Electrode sites and their connectios to the electrode box.

1. Absolute latencies for wave I, III and V
2. Interpeak latency differences between I-III, III-V and I-V
3. Amplitude ratio between wave V and wave I.

Latency values of Na, Pa and Nb and NaPa amplitude were studied from the waveforms of AMLR.

Absolute latencies of P1, .NI, P2 and N2 and the NI-P2 amplitude were studied from the Cz wave obtained for the frequent stimuli while recording the P300.

P300 was measured from the recording for the infrequent stimuli from both the Cz and Pz sites.

MMN was studied by subtracting the waveform for frequent stimuli from the waveform of the infrequent stimuli. Both the Cz and Pz recordings were considered.

Latency was recorded at the peak of the waves. If there was no sharp peak the latencies were recorded by placing the cursor at the centre of the plateau.

Amplitude was measured by placing two cursors, one at the peak and the other at the immediate trough.

The duration of the MMN potentials was recorded from the onset of the potentials to its offset.

RESULTS AND DISCUSSION

The present investigation aimed to study the following auditory evoked potentials in sixteen stutterers between the ages of six to thirty years.

- Auditory bramstem evoked potentials (ABR)
- Auditory middle latency evoked potentials (AMLR)
- Auditory late latency exogenous potentials (ALLR)
- Auditory late latency endogenous potentials (P300 and Mismatch Negativity)

In order to determine whether any of the stutterers demonstrated abnormality in any one/more measures, the data obtained from each subject was compared with the normative data (Saravanan, 1997; Paul, 1997; Shankar, 1997; Saoji, 1998; Krithika, 1999), established using the same instrumentation and test protocols used in this study. Any latency and amplitude measure which was beyond the range of normative values was identified as deviant.

Out of the sixteen stutterers who participated in the study, nine stutterers showed deviancy in at least one measure of Auditory evoked potentials.

The results of the performance of stutterers on the various potentials were as follows:

ABR:

	11.1/s (msec)		60.1/s (msec)		90.1/s (msec)	
	Right	**Left**	**Right**	**Left**	**Right**	**Left**
Peak I						
Mean	1.62	1.71	1.82	1.85	1.92	1.95
SD	0,21	0.23	0.22	0.25	0.31	0.32
Peak III						
Mean	3.62	3.60	3.80	3.88	4.01	4.03
SD	0.29	0.24	0.31	0.33	0.28	0.25
PeakV						
Mean	5.51	5.63	5.78	5.78	5.97	6.07
SD	0.29	0.27	0.25	0.31	0.32	0.35
IPL I-III						
Mean	2.11	2.43	2.38	2.46	2.55	2.61
SD	0.22	0.34	0.31	0.29	0.45	0.39
IPL III-V						
Mean	2.22	2.18	2.54	2.32	2.19	2.62
SD	0.28	0.35	0.26	0.31	0.27	0.23
IPL I-V						
Mean	4.18	4.02	3.96	4.16	4.32	4.48
SD	0.38	0.41	0.28	0.31	0.35	0.42
Amplitude Ratio						
Mean	4.2	3.1	3.8	4.6	**5.2**	3.1
SD	0.34	0.48	0.26	0.51	0.38	0.22

Table R.l. Mean and Standard Deviation of the absolute latencies, interpeak latency differences and wave V/I amplitude ratio in stutterers

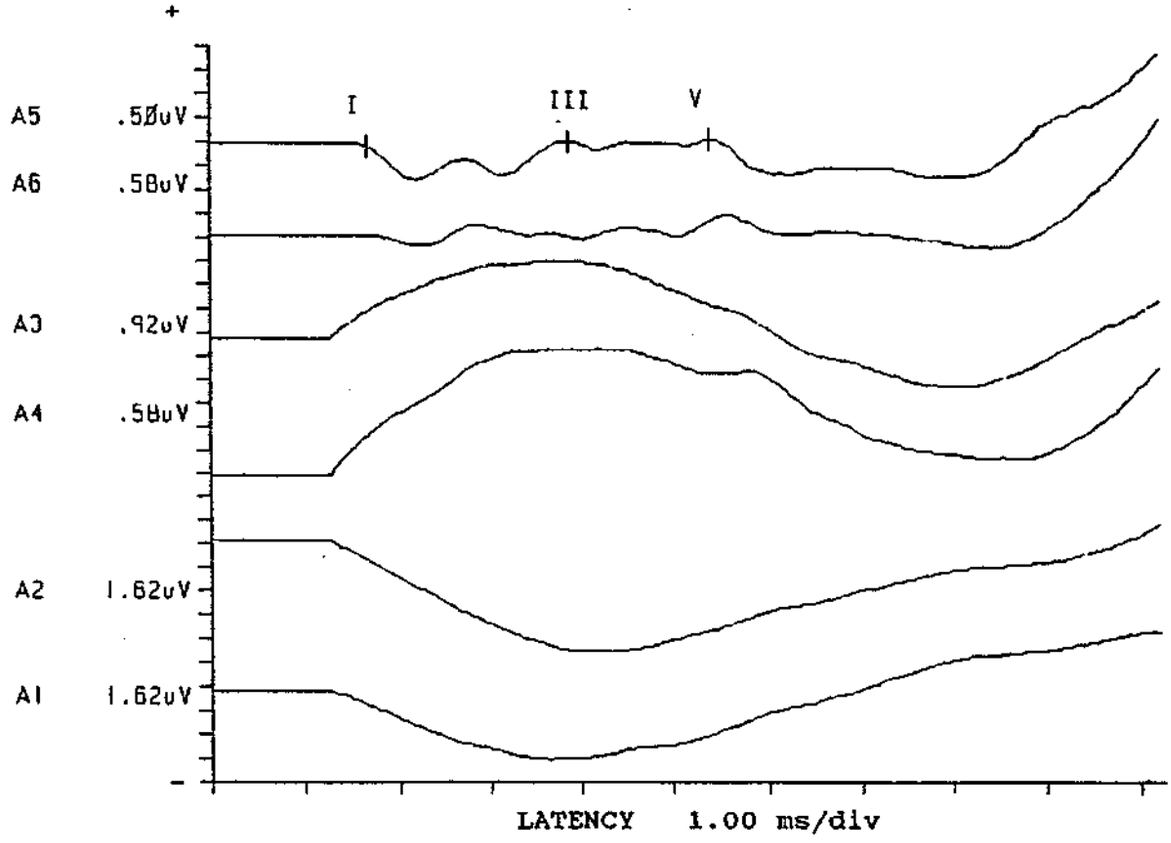
Table R.I. shows the mean and standard deviations for the absolute latencies, interpeak latency differences and wave V/I amplitude ratio at all the repetition rates, which are comparable to the normative values (Saoji, 1998). Also, inspection of the data of individual subjects revealed no deviancy in these measures. These results are different from that reported by Stager (1990), who had observed anomalies in terms of latencies in some of their stuttering subjects.

The V/I amplitude ratio for two subjects was reduced (Table R.S.I.), the wave V amplitude being less than half of the wave I amplitude. Though ABR amplitude criteria has less of a diagnostic value, reduction in V/I amplitude ratio should be regarded as indicative of retrocochlear pathology.

Subject	Measure	Value
1	V/I amplitude ratio (Left ear)	0.21
3	V/I amplitude ratio (Left ear)	0.28

Table R.S.1. Stutterers with deviant V/I amplitude ratio

Only one subject (subject 4.) did not demonstrate ABR peaks at higher repetition rates (60.1 and 90.1 c/s). The recorded waveforms of the subject are presented in figure R.F.I. This may be indicative of a breakdown of neural synchrony when the central auditory processor at the brainstem level is stressed.



LATENCIES (ms)

	I	II	III	IV	V	VI	I'	V'
A5	1.68				3.88			5.40

Wave forms : Repition rates

A5,A6 : 11.1/s

A3,A4 : 60.1/s

A2,A1 90.1/s

Figure : R.F.1 : Absent ABR peaks at higher repetion rates in subjects 4.

Thus, except in three subjects, none of the other subjects showed anomalies at the brainstem level. The slow neural conduction time? at this level was linked with disordered feedback by Stager (1990).

MLR:

Table R.2. shows the mean and standard deviation values for latencies of Na, Pa and Nb peaks along with the NaPa amplitude. These measures fall within the normative data reported by Paul (1997).

	Na (msec)		Pa (msec)		Nb (msec)		NaPa	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Right Ear	23.48	3.37	35.58	4.12	50.26	4.33	0.86	0.50
Left Ear	24.46	3.89	40.48	4.25	52.67	4.84	0.78	0.53

Table R.2. Mean and Standard deviation values for latencies of Na, Pa and Nb along with NaPa amplitude

These results are in line with the results obtained by Vikram (1997) who reported normal mean Na, Pa and Nb peak latencies in the stuttering group. Examination of individual data, however, revealed that four subjects displayed prolonged Pa and Nb latencies (Table R.S.2.). The MLR waveform for one of the subjects is presented in figure R.F.2.

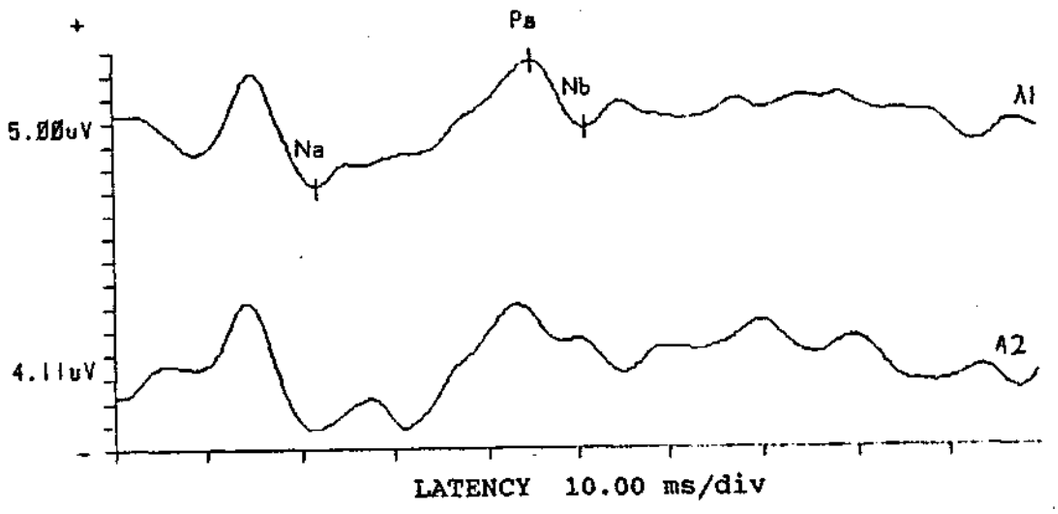
Subject	**Measure**	**Value** (msec)
2	Absolute latencies Pa	44.85
	Nb	50.89
4	Pa	46.22
	Nb	54.83
6	Pa	55.12
	Nb	53.65
7	Pa	48.83
	Nb	52.06
	Amplitude NaPa	0.12µV

Table R.S.2. Stutterers with deviant MLR latencies and amplitude

This highlights the importance of inspecting individual data in stutterers and that relying only on group statistics in a population with high intersubject variability, may lead to erroneous conclusions. The NaPa amplitude was also reduced for one of the subjects with delayed Pa and Nb. latencies. In two subjects, MLR waveform morphology was poor in the left ear. Among them, one subject had normal MLR peak latencies and amplitude values.

The abnormalities in MLR suggest an anomaly in the Thalamo-Cortical projections and the reticular formation which are proposed to activate the AMLR from the primary and secondary auditory cortex (Shi Di and Barth, 1992). These thalamo-cortical projections form a part of the cortico-striato-pallido-cortical loop. This loop performs the function of internal feedback to result in the final execution of language in the form of

internal feedback to result in the final execution of language in the form of speech. Stuttering could occur in the subjects as a result of dysfunction in this loop (Mazziotta, Phelps and Wapenski, 1985).



LATENCIES (ms)

Na	Pa	Nb	Pb
21.64	44.85	50.89	

A1 : ipsilateral wave form
A2 : Contralateral Wave form

FIGURE : R.F.2 : Abnormal MLR is a stutterer

LLR:

Exogenous Potentials:

Table R.3. displays the mean and standard deviation data for the latencies of peaks PI, Nl, P2, N2 and the N1P2 amplitude which are similar to the non-stutterers as per the normative data established by Shankar (1997). A delay in the N2 peak latency (270.78msec) in one stutterer may be indicative of slow neural conduction at the level of N2 generating site.

PI (msec)		Nl (msec)		P2 (msec)		N2 (msec)		N1P2 (uV)	
Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
74.42	6.04	121.38	9.08	168.62	11.02	237.76	11.54	1.63	1.45

Table R.3. Mean and Standard deviation data for LLR peak latencies and N1P2 amplitude in stutterers

P300 and Mismatch negativity (MMN):

As a group, stutterers were comparable to normals (Table R.4. and R.5.) as indicated by the normative data established for P300 (Saravanan, 1997) and MMN (Krithika, 1999).

	Latency (msec)	**N2P3 Amplitude (uV)**
Mean	303.65	11.45
SD	12.45	6.02

Table R.4. Mean and Standard deviation values for P300 latency and N2P3 amplitude in stutterers.

	Duration (msec)	Latency (msec)	Amplitude (μV)
Mean	58.86	208.78	2.84
SD	19.94	25.28	1.20

Table R.5. Mean and Standard deviation data for MMN in stutterers

However, on an individual basis two subjects had reduced N2P3 amplitudes (2.4, 2.2). Among them, one had delayed Pa and Nb latencies and the other subject not showing any visible MMN. The MMN waveform for the subject is presented in figure R F.3.

The reduced N2P3 amplitude indicates either a less or inefficient processing of the incoming stimuli (Barret, 1993). According to McPherson (1996), a decreased P300 amplitude is suggestive of decreased perceptual sensitivity. A stimulus processing deficit at the frontal, temporal and temporo-pareital association cortex (proposed generating sites of P300) can be suspected.

MMN has been demonstrated to provide information about the central processing of fine acoustic differences in speech (Kraus, McGee, Carrell and Sharma, 1995). Absence / anomalies in MMN may therefore indicate the inability of the central processing to detect fine acoustic differences in speech. This would in turn lead to a disordered auditory feedback and cause stuttering which reflects the attempt of a stutterer to overcome the suspected, but in reality a nonexistant error (Bloodstein, 1981; Maraist and Hutton,1957). Further investigations using fine acoustic

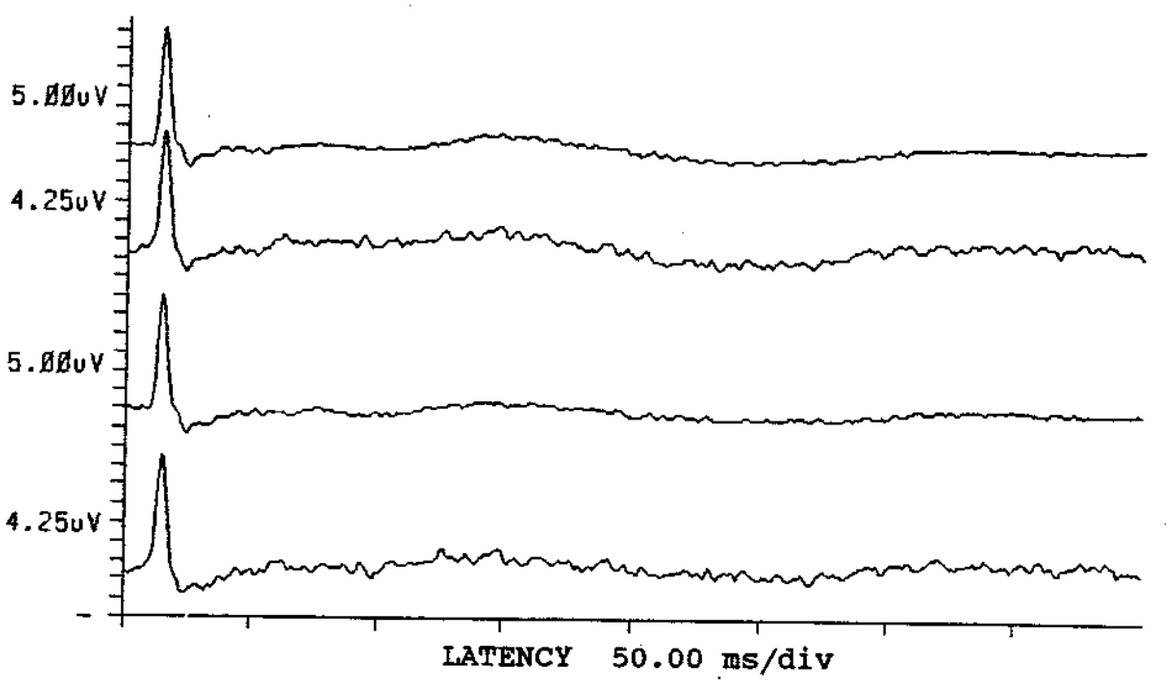


Figure : R.F.3 : No visible MMN in a stutter

differences in speech stimuli in eliciting MMN are needed which will help in substantiating the results obtained through this study.

Thus, in this study nine stutterers showed anomalies in one or more measures of ABR, MLR, LLR, P300 and MMN. Although it can not be ascertained that these aberrations are related to stuttering in these subjects, yet the results do give an indication of a central auditory processing problem in stutterers. Also the variety of anomalies exhibited by some stutterers and not by all the stutterers supports the proposition that the stutterers form a heterogeneous group (Stager, 1990; Clutter and Freeman, 1984). Table R.P. showing the anomalies seen in Auditory evoked potentials in stutterers shows that the findings are scattered. Hence even among the group of stutterers demonstrating a central auditory processing disorder, the site of dysfunction can be anywhere along the auditory pathway. However the results of this study weigh more towards the cortical area being the site of dysfunction.

	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10	S11	S12	S13	S14	S15	S16
ABR	+	-	+	+	-	-	-	-	-							
MLR	-	+	-	+	-	+	+	+	-							
LLR	-	-	-	-	+	-	-	-	-							
P300	-	+	-	-	-	-	-	-	+							
MMN	-	-	-	-	-	-	-	-	+							

Table R.P. The performance of the subjects on each of the potentials

 Normal

\+ Deviant

\- No deviance

SUMMARY AND CONCLUSIONS

A number of investigations have been undertaken in the past to study auditory function in stutterers to determine the cerebral dominance for language and/or look for possible anomalies along the auditory pathway. Objective tests such as evoked potentials are more valid and useful in evaluating auditory processing as they are direct reflectors of changes in the nervous system as the stimuli is processed. The results obtained through auditory evoked potentials by various authors are contradictory. The present study was undertaken to contribute in the direction of substantiating the findings obtained through auditory evoked potentials in the past. Also studying the auditory pathway at both the brainstem and cortical level would help in evaluating the possible site of dysfunction, if any auditory processing deficits exist.

The present investigation aimed to study the following evoked potentials in stutterers:

- Auditory brainstem evoked potentials (ABR)
- Auditory middle latency evoked potentials (AMLR)
- Late latency exogenous potentials (ALLR)
- Late latency endogenous potentials (P300 and Mismatch negativity)

Sixteen stutterers, ten males and six females between the ages of six to thirty years participated in the study. The range of stuttering was from mild to severe as determined by the stuttering severity index (SSI). A calibrated electrophysiological unit, Biologic Auditory Evoked Potentials

system with silver chloride disc electrodes (to record the potentials) and TDH-39 lodged in MX-41/AR ear cushions (to present the stimulus) was used.

The results obtained did not indicate deviant amplitude and latency measures on any of the evoked potentials recorded for stutterers as a group. However, inspection of individual data revealed that nine out of sixteen stutterers who participated in the study showed deviancy in at least one measure of the auditory evoked potentials.

With respect to the ABR, two subjects showed reduced wave V/I amplitude ratio. One subject did not demonstrate ABR peaks at higher repetition rates.

Recordings of the MLR revealed prolonged Pa and Nb latencies for four subjects. In two subjects MLR waveform morphology was poor. In terms of LLR, one stutterer showed a delay in N2 peak latency.

For the endogenous potentials, two subjects had reduced N2P3 amplitudes and one subject showed no visible MMN.

To conclude, though it can not be ascertained that the deviancy shown by the nine stutterers is related to stuttering, yet results do indicate a central auditory processing problem in stutterers. Hence anomalies on the auditory evoked potentials may be used as a means to distinguish the organic from the psychogenic etiology of stuttering.

Also, it will not be wrong in concluding that stutterers among themselves form a heterogeneous population (Stager, 1990; Clutter and Freeman, 1984) and the site of central auditory processing dysfunction could be anywhere along the auditory pathway.

Further Suggestions:

* The variation in performance of stutterers grouped on the basis of severity, on the evoked potentials can be studied.

* The auditory evoked potentials can be recorded for normal-non fluent and stuttering subjects to determine if these potentials can be used as one of the means to differentially diagnose stuttering from normal non-fluency.

* The auditory evoked potentials (especially the endogenous potentials) can be measured in stutterers before and after therapy to evaluate changes in the central auditory processing, if any.

* P300 and Mismatch negativity performed using speech stimuli may lead to better manifestation of the central auditory processing deficit which may arise due to inability of the central auditory mechanism to detect fine acoustic differences in speech.

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