1. INTRODUCTION

1.1 Definition of stuttering
Stuttering is a developmental disorder characterized by frequent and protracted sound prolongations, sound, syllable, word and phrase repetitions and silent blocks that interfere with the efficient production of speech (Bloodstein, 1995; Guitar, 2006). Wingate (1964) gave a standard definition of stuttering as disruption in the fluency of verbal expression, which is characterized by involuntary, audible or silent, repetition or prolongation in the utterance of short speech elements and words of one syllable. Bloodstein (1995) suggested that it is best to characterize stuttering by what is measurable. Chronic stuttering is distinguished when: (a) symptoms have lasted for more than 6 months, (b) more than 3% out of 100 syllables are stuttered, (c) disfluencies are involuntary, (d) sound, syllable and word repetitions with more than 2 iterations occur, (e) sound prolongations longer than 1 second occur, (f) broken words occur, (g) a fixed posture is observed, (h) an increase of pitch or volume occurs, (i) a word is spoken with excessive visible or audible tension, and (j) associated physical movements occur (Bloodstein, 1995; Nicolosi et al., 2004; Natke, 2000; Ochsenkühn & Thiel, 2005; Yairi, 1997).

Another way of measuring disfluency types for the assessment and diagnosis of stuttering is to count the number of Stutter-Like-Disfluencies (SLDs) and Other Disfluencies (ODs) per 100 syllables spoken. If a child exhibits more than 3 SLDs per 100 syllables, stuttering should be suspected in that child (Ambrose & Yairi, 1999). The terminology accounts for “the fact that judgments of overt speech behaviour as ‘stuttering’ are made in the ear of the listener” (Yairi et al., 2001). A SLD is defined as a disfluency that consists of part-word repetitions, prolongations, blocks, and to some extent single syllable word repetitions (Ambrose, 2006). In contrast, ODs consist of interjections (e.g., “hm”, “um”, “ah”), revision/abandoned utterances (e.g., “Helen went/ Helen took her bike to school”, “I thought/ Why don’t we go and see that match?”), and multi-syllable/phrase repetitions (e.g., “maybe, maybe”, “I would like I would like to go home”) (Ambrose & Yairi, 1999).
1.2 Salient features of stuttering:

1.2.1 Core behaviors: stuttering is associated with core behaviors which are basic behaviors of stuttering, which include repetitions, prolongations and blocks. These stuttering behaviors seem involuntary to the stutterers. Repetitions are typically first core behavior to develop. These are simply a sound, syllable or single syllable word repeated several times. The speaker is apparently “stuck” on a sound and continues repeating it until the following sound can be produced. Prolongation of sounds may develop somewhat later than repetitions. In prolongation the sound or airflow continues, but movement of one or more articulators is stopped. Blocks are typically the last core behavior to develop. They occur when the stutterer inappropriately stops the flow of air or the voice and often the movement of articulators as well.

1.2.2 Secondary behaviors: Persistent stuttering is associated with secondary characteristics that define the disorder beyond the speech characteristics described above. Stutterers learn these behaviors as a consequence of their effort to finish the core behaviors quickly or to their effort to avoid them. Specifically, people who persist in stuttering into the school age and adult years, often present with facial grimaces, eye blinking, jaw and neck tension, reduced eye contact, hand tapping or other extraneous body movements (Conture & Kelly, 1991; Riva-Posse et al., 2008). These secondary characteristics can be broadly classified into two categories. First category is “escape behavior”, which occurs when the speaker is stuttering and attempts to get out of stuttering and finish the word. Examples are eye blink, head nods or interjection of extra sounds. These often are followed by the termination of stuttering and are thus rewarded. Second category is “avoidance behavior” which occurs when a speaker anticipates stuttering and tries to keep from stuttering by changing the word, using pause, or using an eye blink. These are rewarded because they sometimes prevent a stutterer from stuttering. Secondary behaviors interfere with the ability of stutterers to communicate clearly and efficiently with their conversation partners. People who stutter may also use circumlocution as a strategy for avoiding speaking words that contain sounds that they
have developed a fear of over their lifetime (Bloodstein, 1995; Plexico, Manning & Levitt, 2009).

1.2.3 Feelings and attitude: As a result of both the primary and secondary characteristics of this disorder social isolation may begin in the early school years. It is common for children who stutter to avoid participating in classroom activities that require speaking in public, such as verbally responding to teachers’ questions or completing oral presentation portions of projects, thus leaving some listeners to misjudge the children as incompetent (Betz, Blood, & Blood, 2008; Dorsey & Guenther, 2000; Franck, Jackson, Pimentel, & Greenwood, 2003; Guntupalli, Everhart, Kalinowski, Nanjundeswaran, & Saltuklaroglu, 2007). The potential for academic problems often influences a child’s self identify and future academic and career goals.

Treatment for individuals at advanced stages of stuttering is often ineffective for reasons not yet understood (De Nil & Kroll, 1995; De Nil, Kroll, & Houle, 1998; Yaruss, 2001). Genetic and environmental factors are implicated in the disorder, and their influences on neural development likely play a role in determining stuttering onset, recovery and treatment response.

1.3 Development of Stuttering

Stuttering onset is most likely to begin during the preschool years. Sixty-five percent of children who stutter begin to do so prior to age 2.5 and 85% prior to age 3.5 (Yairi & Ambrose, 1999). In India, prevalence of stuttering is approximately 22 persons per one lakh population (NSSO, 2002). The overall prevalence of the disorder in childhood is 5% but decreases to 1% in adulthood (Bloodstein, 1995). The drop in prevalence is reflective of the number of children who recover prior to puberty. A large number of children may actually recover close to the onset of the disorder. In fact, 74% of preschool children who stutter recover (Yairi & Ambrose, 1999). For those children who do recover, the usual length of recovery following stuttering onset ranges 2 to 3 years (Yairi & Ambrose, 1999). As the typical age of onset ranges between 2 to 5 years old (Bloodstein and Ratner, 2008) it is reasonable to deduce that most children who recover do so between
ages 4 and 8 years. However, for those who do not recover, the psychosocial and socioeconomic costs are large (Craig, Blumgart, & Tran, 2009; Klein & Hood, 2004; Messenger, Onslow, Packman, & Menzies, 2004; Vanryckeghem, Brutten, & Hernandez, 2005).

1.3.1 Genetic Transmission of Developmental Stuttering

Developmental stuttering is a neurological disorder known to be genetic in origin (Riaz et al., 2005; Shugart et al., 2004; Suresh et al., 2006; Wittke-Thompson et al., 2007). However, the mechanisms underlying genetic transmission and expression are not understood (Ambrose, Cox, & Yairi, 1997; Felsenfeld, 2002; Starkweather, 2002). It seems likely that the presentation of stuttering is the result of a complex interaction between genetic susceptibility to the disorder and the environment (Ambrose et al., 1997; De Nil, 1999; Kidd, 1984; Starkweather, 2002). Early evidence for a genetic influence came from the fact that boys who stutter outnumber girls who stutter 2:1 in preschool years, but that this increased to 5:1 in later school years and adulthood (Ambrose, Yairi, & Cox, 1993; Ambrose et al., 1997). Thus, boys are more susceptible to develop stuttering and to persist. The perceived protective mechanism of the female genotype from stuttering itself, and from persisting to stutter, is very interesting but has not been satisfactorily explored. Geshwind and Galaburda (1985) speculate that the known higher amounts of testosterone in boys may tend to make their speech coordination break down more easily under environmental pressures. Kidd, Kidd, and Records (1978) attribute the difference to a sex linked inherited predisposition to stutter. None of these explanations has won acceptance. All we know that more boys than girls begin to stutter. There is also evidence of familial inheritance of the disorder (Ambrose et al., 1993; Ambrose et al., 1997; Drayna, Kilshaw, & Kelly, 1999; Kidd, 1984). Other evidence comes from concordance studies comparing fraternal and identical twins who stutter. Identical twins have a greater concordance for stuttering relative to fraternal ones (Andrews, Morris-Yates, Howie, & Martin, 1991; Felsenfeld et al., 2000; Howie, 1981). In other words, an individual whose identical twin stutters is much more likely to stutter also, compared to an individual whose fraternal twin stutters. More recent studies have made efforts to isolate the chromosomes responsible for the disorder.
offering direct evidence of genetic involvement (Riaz et al., 2005; Shugart et al., 2004). However, this has proved to be challenging as stuttering does not follow a simple pattern of transmission (Felsenfeld, 2002; Riaz et al., 2005; Suresh et al., 2006; Wittke-Thompson et al., 2007). One genomewide linkage study suggested that stuttering may have a locus on chromosome 12q (Riaz et al., 2005).

There is some evidence from adoption studies to suggest that the role of the environment cannot be ignored. A higher number of people who stutter and who were also adopted were found to have a history of stuttering in their adoptive families than would otherwise be expected by chance (Bloodstein, 1995). Additionally, a biological family history of stuttering was found to be only slightly more predictive of stuttering occurrence than an adoptive family history of stuttering in a group of adopted people who stutter (Felsenfeld & Plomin, 1997).

In summary, the etiology of childhood stuttering remains unknown, but because of its onset early in speech development and its genetic component it has been posited that predetermined neuroanatomical or neurophysiological distinctions increase an individual’s susceptibility to the disorder (De Nil, 1999; De Nil, 2004; Kidd, 1984).

1.4 Theories of stuttering:

There is great diversity in proposed theories which attempt to explain the phenomenon of stuttering. Researchers have made various speculations about stuttering, usually based on models of other disorders or extrapolated from current understanding of normal speech processes. Bloodstein (1995) proposed three broad categories of the theories of stuttering; (1) theories that attempt to define the etiology of stuttering, (2) theories associated with the moment of stuttering and, (3) theories which attempt to reformulate existing theories in either of the earlier mentioned areas.
1.4. 1 Theories of Stuttering Etiology

These theories describe the conditions under which stuttering first develops (Bloodstein, 1995). Five main theories under this category are (1) Theory of Cerebral Dominance (2) Diagnosogenic Theory (3) Genetic Disorder Theory (4) Demands and Capacities Theory and (5) Covert Repair Hypothesis. The details of these theories are given below.

1.4.1.1 Theory of Cerebral Dominance – Travis (1931) proposed this theory. According to this theory, stuttering may be considered as an inability to co-ordinate the messages sent from both cerebral hemispheres for the movement of speech musculature. It also proposes that one hemisphere is dominant in controlling for the synchronization of messages. Hence, in the absence of one hemisphere dominance, the two hemispheres would function independently, which may cause poorly coordinated timing of speech movements and stuttering may be manifested. As this theory makes a link between cerebral dominance, handedness and stuttering, it is also sometimes known as the “handedness theory”. The concept of handedness is based on an early belief that many children who stutter were left-handed, ambidextrous or had been shifted to right-handedness early in life (Travis, 1931). Based on observation of aphasic patients, it was believed that right-handed people had dominant left hemispheres and vice versa for left-handed people. It was further suggested that ambidextrous children had no cerebral dominance. According to Travis (1931), society’s pressure for children to use the right hand in many activities, ultimately attempted to change left-handed children’s cerebral dominance, which might lead to problems like stuttering.

1.4.1.2 Diagnosogenic Theory – This theory of stuttering was proposed by Wendell Johnson in 1942 (Bloodstein, 1995). This theory explains stuttering as a result of attempting to avoid stuttering (Bloodstein, 1984). Johnson (1959) suggested that the attempt to avoid stuttering was caused by parent’s misdiagnosing normal disfluencies as stuttering. The parents attempted to correct these disfluencies, or showed adverse reactions to disfluencies and these attempt created feelings of anxiety in the child leading to the child believing that he/she was really ‘disfluent’ and therefore became so. Johnson further rejected the concept of primary stuttering. Johnson proposed that the disfluency
noted in very young ‘stuttering’ children was no different to normal childhood disfluency (Bloodstein, 1984). This statement highlights the importance of differential diagnosis in very young children, which continues to receive research attention today (Ambrose & Yairi, 1995; Ambrose & Yairi, 1999; Meyers, 1986; Myers & Wall, 1981; Yaruss & LaSalle, 1998).

1.4.1.3 Genetic Disorder Theory – This theory suggests that stuttering has a genetic basis and it is based on the observation that stuttering runs in families. Generally person’s inherited susceptibility along with environmental factors eventually leads to the development of stuttering (Felsenfeld et al., 2000; Kidd, 1984; Yairi & Ambrose, 1996). Further, it is also proposed that genetics may also play a role in the persistence or recovery of stuttering (Ambrose, Cox, & Yairi, 1997).

1.4.1.4 Demands and Capacities Theory (DCT) – This theory is based on the premise that stuttering is caused when a person’s capacity for speech is inadequate to meet the demands placed on the person (Adams, 1990). This theory was proposed by Starkweather (1987) and although he did not explain the theory in more detail it has received numerous interpretations over the years (Adams, 1990). People could have a reduced capacity in cognitive, linguistic, motoric and/or emotional areas. There may be different sources of demands, including environmental, communication partners and/or the stutterer’s own demands (Adams, 1990). Few stuttering therapy approaches are based on DCT. For example, parents are asked to slow down their rate of speech when talking with their child with stuttering so that the demands being placed on the child to reply with a similar rate of speech will get reduced (Costello & Ingham, 1984).

1.4.1.5 Covert Repair Hypothesis (CRH) – This theory proposes that stuttering occurs due to disruption in the process of transforming thoughts into speech. Further it states that instances of disfluency are self repairs which reflect a person’s impaired ability to phonologically encode, and their attempts to adapt for this (Postma & Kolk, 1993). Postma and Kolk suggested that persons with stuttering are slow in their ability to activate intended sounds. When they attempt to activate sounds at a faster rate than their
phonological encoding system is capable of doing, it leads to increase in the chance of an error occurring in the sounds selected. When the speaker detects these errors they may attempt to correct it midspeech which results in the perception of a stutter (Postma & Kolk, 1993).

1.4.2 Theories of the Moment of Stuttering

1.4.2.1 The Breakdown Hypothesis – Bloodstein (1981) defined a stuttering moment as the “momentary failure of the complicated co-ordinations involved in speech”. These difficulties are aggravated by environmental pressures which serve as triggers to the event of stuttering. Environmental pressures include emotional/psychoemotional stress and speech anxiety. Different studies have proposed the stuttering as a “momentary breakdown” which has been linked to motor deficits (Adams, 1974; Perkins, Rudas, Johnson & Bell, 1976), cerebral planning deficits (Travis, 1931), and language processing deficits (Moore & Haynes, 1980).

1.4.2.2 The Repressed Need Hypothesis – This theory suggests that stuttering is a symptom of unconscious anxious/obsessive thoughts. It is based on a belief that stuttering is the manifestation of an unconscious attempt to repress speech (Fenichel, 1945). Several explanations for this avoidance of speech has been proposed like fear of expressing inappropriate words/thoughts or reflection on aggressive thoughts. This theory largely relies on clinical observation and case studies with less scientific or objective data available. Psychotherapies for stuttering, which are based on this theory, have been largely unsuccessful (Bloodstein, 1995).

1.4.2.3 The Anticipatory Struggle Hypothesis – This theory is based on the premise that people stutter because of their belief that speech is difficult. A similar effect is seen in many real life activities. For example, it is likely that when an individual concentrates on fear they are in fact increasing the chances of that fear occurring (Bloodstein, 1995). There are different interpretations of this theory like stuttering being an attempt to exert voluntary control over individual speech movements rather than allowing the automatic
process of speech production (West, Ansberry, & Carr, 1957). According to Sheehan (1953) stuttering is the result of conflict between the desire to speak and the desire to remain silent. Van Riper (1971) suggested that stuttering is triggered by anticipation of word difficulty, followed by three physical reactions (tension in speech organs, focus on first sound rather than whole words, and unnatural positioning of speech organs) which lead to stuttering.

1.4.3 Reformulated Theories

1.4.3.1 Learning Interpretations- These theories attempt to explain how stuttering is learned and maintained by examining surrounding factors/variables. This model includes theories such as stuttering as an operant behaviour, where disfluency increases when children are attempting to gain attention and if they are rewarded for this it could reinforce the stuttering behaviour (Shames & Sherrick, 1963). Further Shames and Sherrick (1963) proposed that punishment of non-fluent responses may lead to the maladaptation from simple repetition behaviours to characteristic stuttering responses. Stuttering has been proposed as a conditioned behaviour, based on the observation that feelings of stress are capable of causing disfluent behaviour in normally fluent individuals. It was proposed that if children are made to repeatedly experience these stressful feelings in a given situation, then the emotional response and subsequent stuttering is aroused with even neutral environmental cues (Brutten & Shoemaker, 1967).

1.5 Auditory Processing and Developmental Stuttering

Stuttering may be related with problems with auditory processing. This includes problems with retaining auditory images, making figure ground distinction, or selecting meaningful from non meaningful auditory signals. Stuttering is less prevalent in deaf and hard-of-hearing individuals (David, 2006) and stuttering may be improved when auditory feedback is altered, such as masking, delayed auditory feedback (DAF), or frequency altered feedback (Gordon, 2002). There is some evidence that the functional organization
of the auditory cortex may be different in stutterers. In addition, in persons with stuttering reduced activation in the left auditory cortex has been observed (Guitar et al., 2006).

The auditory defect may have two possible forms, a central auditory processing problem 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).

1.5.1 Auditory Feedback in Normal and Stuttered Speech Production

The integration of auditory feedback from self generated speech sounds into upcoming motor commands is important for the stability and control of speech production. For example, children with profound hearing impairment experience greater difficulty acquiring and maintaining speech than their normal hearing peers (Campisi, Low, Papsin, Mount, & Harrison, 2006; Kishon-Rabin, Taitelbaum-Swead, Ezrati-Vinacour, & Hildesheimer, 2005; Moeller, Hoover, Putman, Arbataitis, Bohnenkamp, Peterson, Lewis et al., 2007; Moeller, Hoover, Putman, Arbataitis, Bohnenkamp, Peterson, Wood et al., 2007). Also, adults with acquired hearing loss show a gradual degradation of their previously proficient articulatory ability that is partially restored after cochlear implantation (Kishon-Rabin, Taitelbaum, Tobin, & Hildesheimer, 1999). The importance of auditory feedback for speech motor control in normal speakers has been demonstrated via perturbation studies. Various studies have shown the compensatory impact perturbing the volume (Bauer, Mittal, Larson, & Hain, 2006), pitch (Burnett, Senner, & Larson, 1997), phonetic accuracy (Houde & Jordan, 1998) and timing (Jones & Striemer, 2007) of auditory feedback has on the kinematic and acoustic outcomes of speech production in normal speakers. Computational neural network models of speech production have also been used to demonstrate the importance of auditory feedback for articulatory control (Guenther, Husain, Cohen, & Shinn-Cunningham, 1999; Perkell et al., 2000).

Perturbing the timing of auditory feedback in people who are fluent is known to induce a variety of articulation disturbances. Specifically, delayed auditory feedback varied between 200 ms and 400 ms during reading aloud results in a reduced number of correct words, increased total reading time, monosyllabic sound substitutions, omissions,
insertions and additions including repetitions (Fairbanks, 1955; Fairbanks & Guttman, 1958; B. S. Lee, 1950; B. S. Lee, 1951; Stuart, Kalinowski, Rastatter, & Lynch, 2002; Yates, 1963). Conversely, delayed auditory feedback has been shown to positively influence speech fluency in people who stutter (Adamczyk, 1959; Kalinowski, Stuart, Sark, & Armson, 1996; Ryan & Van Kirk, 1974; Soderberg, 1968; Stuart, Kalinowski, Armson, Stenstrom, & Jones, 1996; Stuart, Kalinowski, & Rastatter, 1997). The degree of fluency enhancement varies depending on a number of variables (e.g. delay duration, feedback intensity), the context and the individual (Armson, Kiefte, Mason, & DeCroos, 2006; Wingate, 1970). As a result of the variable responses reported in the literature, the clinical effectiveness of altered auditory feedback as a treatment tool remains controversial (Antipova, Purdy, Blakeley, & Williams, 2008; Lincoln, Packman, & Onslow, 2006; O'Donnell, Armson, & Kiefte, 2008; Pollard, Ellis, Finan, & Ramig, 2009; Stuart, Kalinowski, Rastatter, Saltuklaroglu, & Dayalu, 2004; Stuart, Kalinowski, Saltuklaroglu, & Guntupalli, 2006; Wingate, 1970).

The basis for the variable response of adults who stutter to delayed auditory feedback is not known. Various theories have been put forward to describe how delayed auditory feedback induces fluent speech in some individuals who stutter. It has been proposed that delayed auditory feedback results in speech improvement by forcing the person who stutters to assume a new pattern of speech movement (Goldiamond, 1965). The new pattern is claimed to be established and maintained via operant learning principles with the delayed auditory feedback functioning as aversive negative reinforcement. As pointed out by Wingate (1970), the conceptualization of this process is unclear and incomplete. However, there is some evidence to support the claim that a new speech pattern is learned (Ryan & Van Kirk, 1974). It has also been proposed that the delayed auditory feedback is corrective in nature thereby improving fluency. However, the contrary that delayed auditory feedback is distorted feedback seems to be obvious (Wingate, 1970). Some authors have posited that the key to delayed auditory feedback’s effectiveness is the reduction of meaningful feedback (Wingate, 1970) denying the person who stutters the ability to rely on this potentially inefficient control system. This assertion is somewhat supported by the observation that masking of auditory feedback also induces fluent
speech in some individuals who stutter (Sutton & Chase, 1961; Wingate, 1970). Lastly, it has been proposed that delayed auditory feedback is effective because of the tendency of individuals to slow their speech rate, prolong vowel duration and increase vocal intensity and fundamental frequency (Wingate, 1970). However, changes in speech characteristics such as a slower rate cannot be the only reason that delayed auditory feedback is effective, as it has been demonstrated to have similar fluency enhancing effects, even at fast rates of speech (Kalinowski et al., 1996; Stuart et al., 2002). The effects of altered auditory feedback on speech fluency in people who stutter demonstrate the importance of auditory processing in the disorder. Advancing our understanding of the role auditory processing plays in the speech production of people who stutter may begin to elucidate the mechanisms behind fluency inducing altered auditory feedback.

1.5.2 Auditory processing in normal and stuttered speech production:

Behavioural studies of auditory processing in adults and children who stutter have yielded evidence of central auditory processing differences in these populations relative to fluent age-matched peers. Rousey, Goetzinger and Dirks (1959) reported that 20 stuttering children showed below normal performance in sound localization. Lack of sound localization skills may be indicative of temporal lobe disorders (Jerger, Wekers, Sharbrough, & Jerger, 1969). Various studies have employed batteries of audiometric tests to behaviourally evaluate central auditory processing in adults children who stutter. Rousey, Goetzinger and Dirks (1959) reported that 20 stuttering children showed below normal performance on sound localization. Hall and Jerger (1978) reported that adults who stutter performed poorly relative to fluent adults on a subset of such tests. They concluded that the results suggested the presence of a subtle central auditory processing deficit in adults who stutter. Anderson, Hood and Sellers (1988) conducted a similar study and found that adolescents who stuttered performed poorly on only one subtest as compared to a group of age-matched control participants. They similarly concluded that if a deficit exists it is subtle.
Evidence of a subtle central auditory processing deficit has also been demonstrated in children who stutter. For example, children who stutter have been found to have higher thresholds on backward masking tasks than children who do not stutter (Howell, Rosen, Hannigan, & Rustin, 2000). Howell et al. also found a positive correlation between backward masking thresholds and stuttering severity in children who stutter. In a follow-up study Howell and Williams (2004) investigated children who stutter on a battery of audiometric tests including backward masking tasks. Based on the profile of performance on the audiometric battery of tests, Howell et al. (2004) reached the conclusion that children who stutter had a different developmental pattern of central auditory processing abilities relative to their fluently speaking age-matched peers but they did not specify the nature of that difference.

More recently, central auditory functioning was evaluated behaviourally and with electroencephalography in adults who stutter (Hampton & Weber-Fox, 2008). Behaviourally, adults who stutter performed less accurately and demonstrated longer reaction times in response to the prompt tone in a standard oddball paradigm. However, a small subgroup of adults who stutter was found to be driving the results. The same subgroup of poor performing adults who stutter also demonstrated abnormal evoked auditory waveforms. Hampton and Weber-Fox (2008) concluded that this subgroup demonstrated deficient non-linguistic auditory processing.

Objective tests like AEPs are valid and useful measures to study auditory processing in persons with stuttering as they reflect changes in auditory system as stimuli is processed.

1.6 Auditory Evoked Potentials (AEPs) and auditory processing:

Evoked potentials (EPs) are bioelectric potentials recorded using electrodes placed on the body. In AEPs, the potentials are elicited by giving external auditory stimuli through ear phones. Many different AEPs can be recorded from human scalp and each may be related to particular aspect of auditory processing. Monitoring of spontaneous bioelectric activity from the central nervous system was first described by Berger (1929). After ten years Davis gave effects of auditory stimulation on human brain wave (Davis, 1939). The
process of extracting stimulus related bio-electric events from the ongoing EEG activity set the stage for future clinical development in various aspects of what was called as electric response audiometry (ERA) by Davis (1976).

**Classification of AEPs**

AEPs may be classified in five ways, two of which are used in table 1.1.

One classification scheme, which depends upon the temporal relation between the recorded response and the evoking stimulus, distinguishes among transient, sustained and steady state responses. Transient responses are the potentials evoked by a change in the stimulus such as an onset or offset, whereas sustained potentials are evoked by the continuation of the stimulus (Picton & Fitzgerald, 1983). Transient responses are evoked by an auditory stimulus that is presented at a rate sufficiently slow that the response to one stimulus has finished before the next stimulus occurs; steady-state responses are evoked when stimuli are presented at a rate sufficiently high to cause an overlapping of the responses to successive stimuli (Regan, 1982).

Human AEPs can also be divided into first, fast, middle, slow, and late (Davis, 1976). Because the same potential may change its latency with changes in the stimulus, there is some necessary overlap between these divisions. A sustained potential is usually classified on the basis of its onset latency. A steady state potential is usually categorized by its frequency (the reciprocal of time) or by its "apparent latency", a measurement derived from the phase of the response recorded at different stimulus rates (Regan, 1982).

Three other ways of categorizing the AEPs are not used in the Table. Evoked potentials may be considered in relation to the perception of the stimuli and divided into exogenous and endogenous potentials (Sutton, Braren, & Zubin, 1965). Exogenous potentials are determined by the physical nature of the stimuli, whereas endogenous potentials are determined by the conceptual meaning of the stimulus. In general, the later the evoked potential, the more likely it is endogenous in nature. A fourth classification distinguishes evoked potentials on the basis of their generators. According to this classification scheme, evoked potentials can be considered as cochlear, brainstem, cortical, or
myogenic. Unfortunately, the sources for many of the scalp-recorded evoked potentials are not known. Finally, evoked potentials can also be classified on the basis of the type of evoking stimulus. One may thus consider the evoked potentials to clicks, frequency modulated tones, or speech.

Table 1.1 Classification of AEPs

<table>
<thead>
<tr>
<th>Time</th>
<th>Transient</th>
<th>Sustained</th>
<th>Steady State</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First</strong> (0-5 ms)</td>
<td>Cochlear Nerve Action potential (N1, N2)</td>
<td>Summating potential</td>
<td>Cochlear Microphonic</td>
</tr>
<tr>
<td><strong>Fast</strong>  (2-20 ms)</td>
<td>Auditory Brain stem response (I-VII)</td>
<td>Cranial nerve VIII and brain stem</td>
<td>Frequency Following Response (FFR)</td>
</tr>
<tr>
<td><strong>Middle</strong> (10-100 ms)</td>
<td>Middle Latency Response (No, Po, Na, Pa, Nb, Pb)</td>
<td>Brainstem, mid brain, and cortex</td>
<td>40 Hz potential</td>
</tr>
<tr>
<td><strong>Slow</strong>  (50-300 ms)</td>
<td>Vertex Potential (P1, N1, P2, N2)</td>
<td>Cortical sustained response</td>
<td>Steady State potentials to amplitude modulated tones</td>
</tr>
<tr>
<td><strong>Late</strong>  (250-1000 ms)</td>
<td>Late Positive waves (P3a, P3b), Slow Negative Wave (SNW)</td>
<td>Contingent Negative variations</td>
<td></td>
</tr>
</tbody>
</table>

Different anatomical sites from where the AEPs are recorded have been shown in fig 1.1.
Details about various important AEPs are as follows:

1.6.1 Auditory Brainstem Responses (ABR) - These are potential differences generated in the VIII cranial nerve and the auditory brainstem system when a person’s ear is stimulated with click sounds. The ABR response is commonly found between one and fifteen milliseconds from the time of stimulation. The waveform associated with an ABR consists of up to 7 deflections, or peaks, labeled I-VII (as shown in fig 1.3) all occurring prior to 10 milliseconds. The earliest peaks, I and II, are attributed to activity in the auditory nerve. The sources of peaks III-VI are disputable but in general are thought to reflect activity between the cochlear nucleus, superior olivary complex, inferior lateral lemniscuses and colliculus (Parkkonen, Fujiki, & Mäkelä, 2009). Different neural generators of ABR peaks are shown in fig 1.2.
1.6.1.1 Test Procedures for ABR recording

Test Environment:
ABR test should be administered in a quiet test environment. A sound treated room with is desirable when recording responses to low intensity stimuli but may not be necessary when testing is performed using only high intensity stimuli. Insert ear phones are helpful in attenuating external sounds and are highly desirable for testing patients of all ages.

Patient Considerations:
Recording of early-evoked potential are best obtained when the patient is quiet and relaxed in order to avoid muscle artifacts. Patients are usually placed in a reclining position with a good support to the neck and are instructed to close their eyes, relax and sleep during the recording process.
1.6.1.2 Characteristics of ABR

1.6.1.2.1 Absolute Latency:
The time interval between the stimulus onset and the peak of a wave form referred to as the latency of the response. This latency is more precisely, the absolute latency of a peak because it is related to the stimulus rather than to other peaks in the response. Fig 1.4 shows absolute latencies of different peaks of ABR wave in a normal hearing person. Normative values for absolute latency (Linda Hood, 1998) are Wave I (1.6 ms ± 0.2 ms), Wave III (3.7 ms ± 0.2 ms) and Wave V (5.6 ms ± 0.2 ms).

1.6.1.2.2 Interpeak Latency
The time between peaks in the ABR is referred to as interwave latency intervals, interpeak latencies, and interwave latencies. The interpeak latencies used in clinical interpretation of ABR waveforms are those for wave I – III, wave III – V, and wave I – V. Normative values for interpeak latency (Linda Hood, 1998) are Wave I – III (2.0 ms ± 0.4 ms), Wave III – V (2.0 ms ± 0.4 ms) and Wave I – V (4.0 ms ± 0.4 ms).

Fig 1.3: Normal ABR waveform showing its absolute latencies of different peaks
1.6.1.2.3 Interaural Latency Differences

Interaural latency differences compare the absolute latencies of wave V obtained from stimulation of the right versus left ears at equal intensity levels. When the peripheral hearing sensitivity is similar in both ears the latency of wave V should differ by no more than 0.2 ms to 0.4 ms between two ears (Lindahood, 1998).

1.6.1.2.4 Latency Intensity Function

As the intensity of the stimulus decreases the latencies of the peaks of the ABR increases and response amplitude of the peak decreases. These latencies increases occur slowly for intensities from 90 to 60 dBnHL and then increase more rapidly at lower levels. LI function for Wave V in normal hearing person is 0.3 ms per 10 dB (Lindahood, 1998). In cochlear hearing loss there is steeper LI function when compared to conductive or normal hearing. In auditory nerve or brainstem disorders, the latency of wave V is generally prolonged at all intensities.

1.6.1.2.5 Rate changes

Increasing the rate at which stimulus are presented results in latency and amplitude changes in the ABR. High stimulus rates can be employed to evaluate neural synchrony and recovery and use of higher rates may sensitize testing to subtle neural disorders.
When the stimulus rate is increased from about 10 stimuli per second to 100 stimuli per second. Wave V latency increases by approximately 0.5 ms in normal individuals (Linda Hood, 1998).

1.6.1.2.6 Amplitude
As the stimulus intensity decreases response amplitude decreases. The lower amplitude earlier peaks (e.g. Wave I and III) may become obscured in the background noise first with remaining visible at the lowest intensities. The wave V/I amplitude ratio is obtained by dividing the peak to peak amplitude of wave V by the peak to peak amplitude of wave I. Normative value for wave V/I ratio = ≥ 1.0 (Linda Hood, 1998).

1.6.1.3 Clinical utility of ABR
ABR can be used for hearing screening and to estimate hearing thresholds of difficult to test populations. The ABR is currently the most popular AEP for screening and threshold estimation. The middle and long latency responses have the disadvantage of being at least somewhat dependent on attention and arousal.

ABR can be used for site-of-lesion testing. The ABR is useful for differentiating conductive, sensorineural and retrocohlear disorders. It can assess the integrity of auditory system in central auditory processing disorder upto the level of brainstem.

1.6.2 Auditory Middle Latency Responses (AMLR) - The AMLR responses are series of negative and positive waves which are found between twelve and fifty milliseconds from the time of stimulation, and are identified as Na, Pa, Nb, Pb. This period reflects the response of the auditory cortical and subcortical locations involving the midbrain, reticular formation and thalamocortical pathways. AMLR is exogenous i.e., Responses that are directly dependent on stimulus characteristics (e.g. polarity) & are independent of whether the patient is attending, processing or discriminating the stimulus. The AMLR is used clinically in the electro physiologic determination of hearing thresholds in the lower frequency range, the assessment of cochlear implant function, the assessment of auditory pathway function, and the localization of auditory pathway lesions.
1.6.2.1 Components of AMLR

AMLR consists of biphasic waveform with a negative wave occurring at about 20 ms (Na), a positive wave occurring at about 30 msec (Pa), a second negative wave occurring at about 40 msec (Nb) and second positive wave occurring at about 50 msec (Pb). The Pb component of the AMLR is often identified as the P1 component of the LLR. The wave amplitudes range from 0.5 to 3.0 µV. AMLR wave usually consists of 3 positive and 3 negative peaks, which are labeled as No, Po, Na, Pa, Nb, Pb (Goldstein & Rodman, 1967). A normal AMLR waveform is displayed in fig1.5.

Fig 1.5: Normal AMLR waveform showing its components

1.6.2.2 Neural generators of AMLR

Na – The Na component receives contributions from subcortical regions of the auditory system, specifically the medial geniculate body of the thalamus (Fischer, Bognar, Turjman, & Lapras, 1995; and perhaps portions of the inferior colliculus (Hashimoto, 1982). However, evidence from intracranial electrophysiologic recordings and magnetic responses in human suggests that generation of the Na component also involves the primary auditory cortex within the temporal lobe – Medial tip of Heschl’s gyrus (Liegeois-Chauvel, Musolino, Badier, Marquis & Chauvel, 1994).
Pa – In the 1980s, studies of AMLR utilizing scalp electrodes in patients with cortical lesions confirmed the major role of primary auditory cortex in generation of the Pa component (Kraus, Ozdamar, Hier, & Stein, 1982). Based on investigations in patients with temporal lesions, however, subcortical (e.g. thalamic) structures also appear to contribute to the Pa component (Simpson & Naeser, 1987). Therefore, the Pa component actually is the product of activity within both subcortical and cortical regions of the auditory system (Jacobson, 1990). Human pial surface recording demonstrate positive peak of Pa latency over temporal and parietal lobes (lee et al., 1984). Human neuromagnetic recording show a positive peak at approximately 30ms (pelizzone et al., 1987). A positive peak of approx. 30 ms was reported from within the brain (Goff et al., 1977). Recent studies have shown that Pa is produced by tangentially oriented dipole sources in auditory cortex.

Nb – Nb component of AMLR arises from Thalamo-cortical tracts and lateral Supratemporal gyrus (STG).

Pb – Pb component of the AMLR arises from auditory cortex. (Posterior region of the Planum Temporale). Thalamo-cortical tracts, lateral Supratemporal gyrus (STG) and antero-lateral Heschl's gyrus are possible generators of Pb wave.

1.6.2.3 Clinical utility of AMLR:

The AMLR is used clinically in the electrophysiologic determination of hearing thresholds in the lower frequency range. It is used as a measure of establishing threshold because of its frequency sensitivity.

AMLR can also be used as a means of neurological diagnosis. It gives us information about the integrity of auditory pathways when considered along with other AEPs.

1.6.3 Late Latency Responses (LLR) - The LLR responses are auditory evoked potentials which are found between 50 and 300 milliseconds from the time of stimulation, after the ABR and MLR. LLRs can be acquired with the use of pure tone stimulation.
(tone-bursts). The LLR response is of very low frequency (under 30Hz) and has a common voltage range between three and ten microvolts.

Fig 1.6: Normal LLR waveform showing its components

1.6.3.1 Components of LLR: The major components are waves N1, P1, N2, P2 (as shown in fig 1.6).

- **P1** - The P1 response is primarily an exogenous potential occurring at about 60ms (between 55 and 80 ms). The P1 response appears to be strongly related to stimulus parameters.

- **N1** - The N1 is primarily an exogenous potential, occurring at about 100ms (between 90 & 110ms). According to David.L.Woods (1995) the N1 wave reaches maximum amplitude at fronto central sites where it shows an early peak at 95 – 100 ms and a 2nd peak at 120 - 130 ms. It returns to baseline at 180-130 ms.

- **P2** - The P2 occurs at about 160ms (between 145 & 180ms) and is primarily an exogenous potential.

- **N2** - The N2 is the first of the primarily endogenous potentials occurring at about 200ms (between 180 & 250ms). The range of values were given by Mc pherson (1996). The N2 is highly related to attention as is the entire N1-P2-N2 response, being related to the acoustic features of audition. The N2 wave is not invariable and may or may not be present in normal subjects. The P1 differs greatly from the N1, P2, N2 components of the
LLR and in some instances the P1 of the LLR is also considered to be the Pb in the AMLR.

The simplest measurement condition consists of an infrequent stimulus presented randomly within a series of frequent and predictable stimuli.

1.6.3.2 Neural Generators of LLR:

P1- P1 has its origin in the temporal lobe (Sherg and Von Cramon, 1985).

N1- N1 is associated with activity of the nonspecific polysensory system within the contralateral supratemporal auditory cortex (Knight et al, 1988).

P2- P2 is associated with the nonspecific polysensory system demonstrating activity in the auditory cortex (Vaughan and Ritter, 1970).

N2- N2 is part of the nonspecific polysensory system in the supratemporal auditory cortex (Velasco et al, 1989).

Davis (1939) showed that the LLR could be recorded from electrodes locations on the scalp; with maximum amplitude from midline electrodes over frontal regions. Diffuse non-specific generators in thalamocortical regions were suspected.


Vaughan &Ritter (1970) demonstrated in 6 normal subjects a clear polarity inversion at the Sylvian fissure level for a component in the 200ms range. Superior to sylvian fissure this component was of positive voltage and below it was negative voltage. They suggested that the source of ALR was a dipole within the primary auditory projection cortex on the supratemporal plane.

LLRs appear to reflect the response of the auditory cortex with contribution made by limbic system.
1.6.3.3 Clinical applications of LLR: Despite the clinical limitation of inter-intra subject variability susceptibility to state of arousal and drugs, the clinical implications of LLR is extensive. In general LLR have two clinical applications:

1. Neurodiagnosis (adults and children)
2. Estimation of hearing sensitivity (mostly in children)

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

1.6.4 P300

P300 wave is a parietocentral positivity that occurs when a subject detect an informative task relevant stimulus (Picton, 1992). The P300 is a component within an extended auditory late response time frame recorded using an oddball paradigm (standard and target signal). One stimulus, a frequent and predictable stimulus (the standard signal), generates an auditory late response. The other stimulus which is infrequent (rare), unpredictable (presented randomly), and different (deviant) in some way from the first signal-the oddball or target signal-produces a positive wave in the latency region. Target signal produces a positive peak in the latency of 300ms, which is also called P3. A missing, rare or a deviant signal can elicit P300 response. It is often described as cognitive evoked response as it depends on the detection of the difference between frequent vs. rare signals. Fig 1.8 shows P300 waveform with its component.
Fig 1.7: Normal P300 waveform showing its components

Morphology of endogenous response waveforms is dependent on details of test paradigm and subtle variations in the subject’s attention to the stimuli. Anticipation of the stimulus, processing time affects amplitude and latency of P300.

1.6.4.1 Components of P300 - Components of a normal P300 wave include P300a and P300b. P300a is passive response which has shorter latency. It is evoked independent of attention to the target stimuli. P300b refers to conventional P300 response that appears 300 ms after presentation of the rare stimulus in oddball paradigm.

1.6.4.2 Neural generators of P300 - Diverse regions of the brain contribute to the generation of P300 including sub cortical structures – hippocampus, other structures within the limbic system and the thalamus, auditory regions in cortex, frontal lobe.

1.6.4.3 Clinical applications of P300 - Since we are uncertain about the cerebral origin of P300 wave and since we do not know the exact role it plays in cognitive processing, the clinical usefulness of this wave is limited. However, it is useful in neurodiagnosis of central auditory processing disorders which may be related with disorders like Schizophrenia, autism, dementia, Multiple Sclerosis etc.

ABR, AMLR, and LLR recordings were included in our study; however P300 could not be included due to instrumental constraints.
1.7 NEED FOR THE STUDY

Behavioural studies suggest that at least a subgroup of adults and children who stutter present with subtle differences in performance on behavioural tests of central auditory function that vary across development relative to age-matched fluently speaking controls. Due to the inconsistent results across studies, it appears that indirect audiometric tests of central auditory processing lack the sensitivity required to fully characterizing the differences that may exist in this domain between people who stutter and people who speak fluently. Also, linguistic behavioral tests using speech material are language specific and therefore difficult to use in a multilingual country like India. Hence, further investigation of auditory processing in adults and children who stutter with direct measures of cortical auditory structure and function using electrophysiological tests may prove more informative.

The auditory evoked potentials (AEP) are important in the assessment of the auditory pathways, especially in individuals with communication disorders. The AEP are objective tests and, therefore, do not require verbal responses from the patient (Jerger J, Musiek FE., 2000). The results through AEPs by various authors are contradictory. The present study was undertaken to contribute in the direction of substantiating the findings obtained through AEPs in the past.

The therapeutic process progress can be monitored through changes in latency and amplitude of such potentials given the plasticity of Central Auditory Nervous System (Hayes EA, Warrier CM, Nicol TG., 2003). Neuronal plasticity resulting from training and perceptual learning has been shown to involve alterations in neural connections and activity at multi levels of the auditory pathway. For example unilateral sound deprivation in adolescent ferrets has been shown to lead to sub cortical changes (Moore, 1993). Evoked potentials, reflecting the precise timings of synchronous events in the neural encoding of stimuli, provide a sensitive index of neurophysiologic alterations related to training. However, there are less evidence to suggest that the correlations between the changes in evoked potentials with that of stuttering. Hence there was a need to find out the relation between the two. Pre and post therapy AEP evaluations would help us to
understand the changes seen in AEP measures which may be attributed to effectiveness of speech therapy.

1.8 AIM OF THE STUDY

The aim of the present study was to find out the following:

a. Whether there is presence of central auditory processing disorder in person with stuttering which may be indicated by deviant findings in wave latency and amplitude of ABR, AMLR or LLR measures. And

b. Whether there are any changes in AEP measures after speech therapy which may indicate whether the functional neuroplasticity has been taken place or not.

1.8.1 OBJECTIVES:

1. To find out whether there is improvement in stuttering severity of persons with stuttering after the speech therapy is administered.
2. To characterize the auditory evoked potentials of ABR, MLR, and LLR in persons with stuttering.
3. To compare the ABR, MLR and LLR measures in persons with stuttering with that of persons with no stuttering.
4. To compare pre and post measures of ABR, MLR and LLR in persons with stuttering as well as in persons with no stuttering.
5. To compare pre and post measures of ABR, MLR and LLR in persons with stuttering based on sides of ear.
6. To compare pre and post measures of ABR, MLR and LLR measures in persons with stuttering based on age.
7. To compare pre and post measures of ABR, MLR and LLR in male and female in persons with stuttering based on gender.
1.9 HYPOTHESES:

Following Null hypothesis were made for the present study:

1. There is no improvement in stuttering severity in persons with stuttering when pre therapy and post assessments are compared.

2. There is no significant difference in any parameters of ABR in persons with stuttering when compared with the mean values of ABR measures in persons with no stuttering.

3. There is no significant difference in pre and post measures of ABR in persons with stuttering.

4. There is no significant difference in pre and post measures of ABR in persons with no stuttering.

5. There is no significant difference in pre and post measures of ABR in persons with stuttering based on side of ears.

6. There is no significant difference in pre and post measures of ABR in persons with no stuttering based on side of ears.

7. There is no significant difference in pre and post measures of ABR in persons with stuttering and persons with no stuttering based on side of ears.

8. There is no significant difference in pre and post measures of ABR in persons with stuttering based on age variable.

9. There is no significant difference in pre and post measures of ABR in persons with no stuttering based on age variable.
10. There is no significant difference in pre and post measures of ABR in persons with stuttering based on gender variable.

11. There is no significant difference in pre and post measures of ABR in persons with no stuttering based on gender variable.

12. There is no significant difference in pre and post measures of ABR in persons with stuttering and persons with no stuttering based on gender variable.

13. There is no significant difference in pre and post measures of ABR between persons with stuttering and persons with no stuttering based on gender variable.

14. There is no significant difference in any parameters of AMLR in persons with stuttering when compared with the mean values of AMLR measures in persons with no stuttering.

15. There is no significant difference in pre and post measures of AMLR in persons with stuttering.

16. There is no significant difference in pre and post measures of AMLR in persons with no stuttering.

17. There is no significant difference in pre and post measures of AMLR in persons with stuttering between sides of ears.

18. There is no significant difference in pre and post measures of AMLR in persons with no stuttering between sides of ears.

19. There is no significant difference in pre and post measures of AMLR in persons with stuttering and persons with no stuttering between sides of ears.

20. There is no significant difference in pre and post measures of AMLR in persons with stuttering based on age variable.
21. There is no significant difference in pre and post measures of AMLR in persons with no stuttering based on age variable.

22. There is no significant difference in pre and post measures of AMLR in persons with stuttering based on gender variable.

23. There is no significant difference in pre and post measures of AMLR in persons with no stuttering based on gender variable.

24. There is no significant difference in pre and post measures of AMLR in persons with stuttering and persons with no stuttering based on gender variable.

25. There is no significant difference in pre and post measures of AMLR between persons with stuttering and persons with no stuttering based on gender variable.

26. There is no significant difference in any parameters of LLR in persons with stuttering when compared with the mean values of LLR measures in persons with no stuttering.

27. There is no significant difference in pre and post measures of LLR in persons with stuttering.

28. There is no significant difference in pre and post measures of LLR in persons with no stuttering.

29. There is no significant difference in pre and post measures of LLR in persons with stuttering based on side of ears.

30. There is no significant difference in pre and post measures of LLR in persons with no stuttering based on side of ears.

31. There is no significant difference in pre and post measures of LLR in persons with stuttering and persons with no stuttering based on side of ears.
32. There is no significant difference in pre and post measures of LLR in persons with stuttering based on age variable.

33. There is no significant difference in pre and post measures of LLR in persons with no stuttering based on age variable.

34. There is no significant difference in pre and post measures of LLR in persons with stuttering based on gender variable.

35. There is no significant difference in pre and post measures of LLR in persons with no stuttering based on gender variable.

36. There is no significant difference in pre and post measures of LLR in persons with stuttering and persons with no stuttering based on gender variable.

37. There is no significant difference in pre and post measures of LLR between persons with stuttering and persons with no stuttering based on gender variable.