Auditory Steady State (Response in Subjects with Auditory Dys-synchrony

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A dissertation submitted in part fulfillment of the final year
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Dedicated

To

Papu

For all your love, inspiration and

keeping me on one firm condition.

Certificate

This is to certify that this dissertation entitled *"Auditory Steady State (Response in Subjects -with Auditory Dys-synchrony"* is a bonafide work done in part fulfillment for the degree of Master Science (Speech and Hearing) of the student (Register No. 02SH0016)

Director

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May, 2004

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Certificate

This is to certify that this dissertation entitled *"**Auditory Steady State (Response in Subjects with Auditory Dys-synchrony**"* has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any other Diploma or Degree.

Guide

Mysore
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DECLARATION

I hereby declare that this dissertation entitled ***"Auditory Steady State (Response in Subjects with Auditory Dys-synchrony"*** is the result of my own study under the guidance of *Dr. C. S. Vanaja, Lecture in Department of Audiology, All India Institute of Speech and Hearing, Mysore,* and has not been submitted earlier in any other University for the award of any other Diploma or Degree.

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INTRODUCTION

Auditory neuropathy or auditory dys-synchrony (AD) is a term used to describe subjects who display auditory characteristics consistent with absent or abnormal auditory brainstem response in the presence of otoacoustic emission or cochlear microphonics (Starr, Pictone, Sininger, Hood & Berlin, 1996). Other common features, of subjects with auditory dys-synchrony are absent acoustic reflexes to tonal stimuli in the presence of normal middle ear function, abnormal masking level difference (MLD) and poorer than expected word recognition scores (Tlumak, 2002).

Proposed etiologies of auditory dys-synchrony have been diverse and include neonatal hyperbilirubinemia (Stein et al.,1996), severe illness during the neonatal period (Deltenere, Mansbach, Bozet, Clerex & Hecox, 1997), a part of a generalized metabolic toxic or inflammatory neuropathy (Starr et al., 1996). Some patients also may have an accompanying generalized neuropathy affecting other cranial and/or peripheral nerves (Starr et al., 1996). The other etiologies, which can lead to occurrence of auditory neuropathies, include genetic factors as in hereditary sensory motor neuropathy (Raglan, Prasher, Trinder and Rudge, 1987), hereditary sensory and autonomic neuropathy (Wright and Dyck, 1995), and the neuropathy accompanying Friedrich's ataxia (Cassandro, Mosca, Sequino, De-Falco and Campanella, 1986). The demyelinating neuropathy of the Guillian-Barre syndromes may at times involve the auditory neuropathy (Rooper and Chiappa, 1986).

Difficulty in the understanding of speech is a consistent finding in subjects with auditory dys-synchrony (Davis & Hirish, 1979; Kraus, Ozdamar, Stein & Reed, 1984 and Starr et al., 1996). Because there is some evidence that temporal cues play a role in the encoding of human speech (Zeng, Oba, Garde, Sininger, & Starr, 1999), it is possible that the poor speech comprehension are associated with the reduced temporal synchrony of eighth nerve firing (Zeng et al., 1999). Synchronized aggregate neural responses have been shown to reflect perceptually important acoustic features in speech, (Mc.Gee et al., 1996; Steinschneider et al.,1994). Synchrony related to stimulus timing is especially important in the auditory system, (Eggermant, Phillips, Sinex Cited in Kraus et al., 2000), where the spectral and temporal complexity of a signal such as speech elicits responses from a broad neural population resulting in patterns of synchronized activity. So loss of synchronous firing of auditory neurons may lead to poor identification or speech recognition.

Electrophysiological test generally used in diagnosing auditory dys-synchrony is ABR. By clinical definition subject with auditory dys-synchrony will have abnormal or absent ABR with presence of OAE. Because, normal ABR is recorded only when multiple neurons fire synchronously, at stimulus onset even minor variation in the timing of neural discharges after each stimulus can make the ABR unrecognizable (Kraus et.al.2000). Hence it does not provide opportunity to examine the role of synchrony in perception in subjects with auditory dys-synchrony.

Speech representation is not only dependent on peripheral synchrony. It has been reported that the auditory cortex can adjust to the faulty signal representations present at earlier stages along the auditory pathway (Kraus et.al. 2000). In fact, the grossly intact cortical potentials recorded in these cases suggest that the patient with auditory dys-synchrony can use varied and limited inputs to perceive complex signals (Kraus et.al.2000). It has been reported that auditory middle latency response (AMLR) and auditory late latency response (ALLR) are less dependent on synchronous firing when compared to ABR and can be expected to be present if the problem is less severe (Hood., 1999). A review of literature shows that AMLR & ALLR are present in some subjects with AD whereas it is absent or abnormal in a few subjects with auditory dys-synchrony (Starr et al., 1991). Investigations have also indicated that speech identification is poorer in subjects with abnormal ALLR when compared to subjects with normal ALLR (Kraus et al., 2000). It has been reported that subjects with normal ALLR benefit more from hearing aid when compared to those with abnormal ALLR (Vanaja & Manjula, 2002).

Another evoked potential testing that has gained popularity in the recent years is Auditory Steady State Responses (ASSR). ASSR are periodic scalp potentials that arise in response to regularly varying stimuli such as sinusoidal amplitude /or frequency modulated tones (Kuwada, Batra & Moher, 1986). The ASSR may not require the degree of neural synchrony needed for identification of transient waveform (Rance, Dowel, Rickards, Beer & Clark, 1998). ASSR may be elicited with slow modulation as well as high modulation rate (eg., 40 Hz & 80 Hz). Stimuli modulated at 40 Hz rate generates a response with derived latency of approximately 30 msec and is thought to result from the

superimposition of transient middle latency response (Galambous, Makeing & Talmas Voff cited in Rance et al. 1999) where as potentials elicited by tonal stimuli modulated at rates around 80 to 90 Hz, appears to have linear generators to the late components of the ABR (Cohen, Rickards, & Clark, 1991)

Rance et al., (1999) studied ASSR for high modulation rate in subjects with auditory dys-synchrony and results showed a weak co-relation between behavioral threshold and ASSR threshold, ASSR could be recorded in all the subjects but only at high sensation levels. The range of observed SSEP/behavioral difference values for these cases was higher than expected. This inconsistency suggests that the abnormality affecting ABR is likely to have influenced the ASSR result also.

NEED AND AIMS OF THE STUDY

Extraction of ABR from the EEG signal achieved by averaging requires precise synchrony of neural firing for response definition. Even minor variation in timing of neural discharges after each stimulus can make the ABR unrecognizable. And pathophysiological changes in neural conduction properties associated with demyelization are likely to have profound effect on ABR, which are reliant on the relatively precise synchronous response of a population of auditory nerve fibers to a transient acoustic stimulus (Starr et al., 1996). The ASSR, on the other hand may not require the degree of neural synchrony needed for identification of transient waveform. Hence they may be more resistant to the neural dys-synchrony that appears to arise in case of auditory dys-synchrony (Rance et al., 1999).

Clinical experience with subjects with auditory dys-synchrony shows that speech identification scores are poor in clients with auditory dys-synchrony but there is variability in the speech identification score observed in different clients. A review of literature also indicates that in subjects with auditory dys-synchrony, ASSR is present at high SL for tone modulated at a higher rate but there is no information on ASSR for tones modulated at lower rate. Based on the results of studies on AMLR and ALLR in subjects with auditory dys-synchrony (Starr et al., 1991), it can be hypothesized that subjects with normal ASSR at low SL will have better speech identification score than those with abnormal ASSR. The second hypothesis is that the speech identification will be poorer in subjects with abnormal ASSR for both 40 Hz and 80 Hz modulations than those with abnormal ASSR for 80 Hz modulations and normal ASSR for 40 Hz modulations.

Hence the first goal of the investigation was to study ASSR for 40 Hz and 80 Hz modulation frequencies in subjects with auditory dys-synchrony. The second goal of the study was to investigate the relationship between the results of ASSR and speech identification scores.

REVIEW OF LITERATURE

Auditory dys-synchrony is a hearing disorder in which peripheral hearing appears normal, but the eighth nerve fiber and brainstem functioning are abnormal (Kraus, Ozdamar, Stein & Reed, 1984; and Starr et al. 1991). Subjects with this disorder have normal otoacoustic emissions (OAE) and cochlear microphonic (CM) potentials, but exhibit an absent or severely abnormal auditory brainstem response (ABR) (Starr et al., 1996). A normal ABR is recorded only when multiple neurons fire synchronously at stimulus onset. Hence failure to record these potentials would be associated with an auditory perceptual deficit disproportionately affecting those perceptions that are dependent on temporal features of the acoustic signal (Starr et.al., 1991)

Neural synchrony is a fundamental neurobiologic process underlying, sensory, motor, and cognitive events. Synchrony related to stimulus timing is especially important in the auditory system (Phillips cited in Kraus et al., 2000), where the spectral and temporal complexity of a signal such as speech elicits responses from a broad neural population resulting in patterns of synchronized activity. Eggermont (1997) has stressed the importance of neural synchrony across populations of neurons in the signaling of differences between steady state and dynamic stimuli. In addition, synchronized aggregate neural responses have been shown to reflect perceptually important acoustic features in speech (McGee, Kraus, King, & Nicol, 1996; Steinschneider et al., 1994).

A review of literature indicates that a number of investigations have been carried out to study the results of various behavioral and electrophysiological tests in subjects

with auditory dys-synchrony. A brief of review of these investigations is presented in this section.

Behavioral tests

The hearing thresholds in subjects with auditory dys-synchrony can range from normal hearing to profound loss (Starr et. al., 1996). It is not uncommon for hearing thresholds to fluctuate dramatically from day to day or even during test. The configuration of audiogram varies. Sininger & Oba (2001) reported that 43% of subjects show a flat audiometric shape, and 28% have a reverse sloping loss with higher thresholds for low-frequency stimuli than for higher frequencies. Speech audiometry will reveal poor speech perception relative to the degree of hearing loss (Tlumak, 2002). Subjects with auditory dys-synchrony have dysfunction of speech perception that is out of proportion with their pure-tone loss (Rance et. al., 2002).

Other psychophysical tests, which have been used to asses subjects with auditory dys-synchrony are gap detection test, modulation transfer function tests, temporal integration, and masking level difference. As reported by Starr et.al., (1991), subjects with auditory dys-synchrony showed normal or near normal temporal integration function. In contrast detecting short silent intervals or gaps in acoustic signals (in gap detection test) was uniformly impaired in the subjects with dys-synchrony, their gap detection thresholds were 2 to 25 times greater than the normal threshold (Zeng et, al. 1999**).** Shivaprakash & Manjula (2003) studied temporal resolution using gap detection

test in two subjects with auditory dys-synchrony. Their results showed that there is variation in the temporal resolution of subject with auditory dys-synchrony.

Zeng et al., (1999) also studied modulation transfer function in subjects with auditory dys-synchrony. Results showed that subjects with auditory dys-synchrony had impaired sensitivity to both slow and fast temporal fluctuation whereas sensitivity to slow temporal fluctuation was good in normal subjects and sensitivity decreased as fluctuation rate was increased. Rance, McKay, and Grayden (2004) Studied frequency resolution, temporal resolution and frequency discrimination in subjects with auditory dys-synchrony. Results showed that frequency resolution was normal in most of the subjects. but the temporal resolution was abnormal in many of the subjects. Frequency discrimination ability varied significantly; with some subjects (typically those with good speech perception) showing normal limens and other subjects showing severely depressed ability to detect frequency differences.

Starr et al., (1991) reported a marked impairment of those auditory perceptions that are dependent on temporal cues. These include lateralization, masking level difference, monaural detection of a silent gap in a sound and monaural threshold elevation for short duration tones. In contrast, auditory function reflecting intensity or frequency discriminations (difference limens) were only minimally impaired.

Thus, a review of literature on auditory dys-synchrony and psychophysical tests indicates that there is impairment in temporal resolution which is essential for speech

perception or speech identification ability. The review also shows that there is heterogeneity in subjects with auditory dys-synchrony and the results of tests depend on degree of dys-synchronous firing.

Auditory evoked potential studies

Kraus et al., (1984) described four subjects with audiometric findings ranging from normal hearing to moderate hearing loss with absent ABR. They showed ABR abnormalities, which was out of proportion to the pure tone hearing loss. It was also found that MLR was normal in one out of five subjects. In the remaining four subjects MLR was reported to be absent. Hence a neuropathology of brainstem was suspected in those subjects.

Starr et al., (1996) administered electrophysiological tests on ten subjects with hearing impairment. The study showed that it was possible to detect other types of auditory evoked potentials in five subjects eventhough their brainstem potentials were absent. Middle latency response were detected in one out of five subjects with absent brainstem potentials and long latency components (N100, P200) were recorded in three out of four subjects with absent ABR. Cognitive potential (P300) evoked in an auditory discrimination target detection task, were also present in the two of the subjects tested.

Normal LLR and MMN to speech stimuli have been reported in a subject who had normal peripheral hearing with absent ABR (auditory dys-synchrony), by Kraus et al (2000). Rance, Con-Wersson, Wunderlich and Dowell (2002) studied cortical event

related potentials in eighteen children with auditory dys-synchrony. Results showed that approximately 50% of children with auditory dys-synchrony showed event related potential of normal latency, amplitude and morphology. Sharad (2001) recorded auditory evoked potentials (ABR, MLR, LLR and MMN for intensity devience) in seven subjects with auditory dys-synchrony. Analysis of results showed that in a majority of the subjects MLR was absent but LLR and MMN were present. Only in one subject, all the potentials were absent.

Thus, absent or abnormal MLRs and LLRs in a few subjects suggest deficits in auditory processing or communicative problems that cannot be explained by peripheral hearing loss alone. On the other hand, the fact that MLRs and LLRs were observed in some of the subjects indicated that neural signals are indeed reaching auditory pathway central to brainstem.

The Auditory - Steady State Response (ASSR) is an auditory evoked potential, elicited with modulated tones that can be used to predict hearing sensitivity in patients of all ages (Stach, 2002). ABR extraction from the EEG signal, achieved by averaging, requires precise synchrony of neural firing for response and even minor variation (<0.5 ms) in the timing of neural discharges after each stimulus can make the ABR unrecognizable (Rance et al., 1998). The ASSR on the other hand, may not require the degree of neural synchrony needed for identification of transient waveforms.

Auditory steady state evoked potentials can be recorded with different rates of stimulation (Richards and Clark, 1984). Rates of a few stimuli per second elicit steady-

state versions of the late auditory-evoked potentials (Maiste and Picton, 1989) but their responses are small and vary with the state of the subject. The 40-Hz response is the steady state version of the middle latency evoked potentials (Dimitrijevic, John, Roon, & Picton, 2001). Auditory steady-state responses to stimulus rate of 80-100 Hz may represent the steady - state versions of the transient brainstem ABRs (Aoyagi, Kiren, Furuse, Fuse, 1994). These 80-Hz auditory steady-state responses are less affected by sleep than the 40 Hz response (Cohen, Richards, and Clark (1991) and can be recorded reliably in sleeping children (Aoyagi et al., 1994).

Rance et al., (1999) studied ASSR for high modulation rate in subjects with auditory dys-synchrony and results showed a weak co-relation between behavioural threshold and ASSR threshold. They could record the potentials in all the subjects but only at high sensation levels. The range of observed SSEP/behavioural difference values for these subjects was higher than expected. In addition to providing estimates of hearing thresholds, steady-state responses might be able to evaluate how well the auditory system processes differences in frequency and intensity at supra threshold intensities. This has been studied in normal-hearing subjects by correlating the "independent amplitude frequency modulation" responses at different intensities to speech discrimination test performed at the same levels. (Dimitrijevic et al., 2001).

Thus limited research available on ASSR indicate that ASSR to different modulation rates may supplement information provided by ABR while testing subjects with auditory dys-synchrony.

Relationship between the Speech Identification Score and other Test results

Speech Identification Scores and Psychophysical Tests

Speech understanding in auditory dys-synchrony subjects appears to be unrelated to the behavioral audiogram, suggesting that the distortion of suprathreshold cues rather than access to the speech spectrum is the limiting factor. Speech perception ability in fact appears to be related to the degree to which temporal precision is disrupted in the auditory brain stem response and central auditory pathway. Rosen (cited in Kraus et al., 2000) reported that the suprasegmental components of speech typically are expressed over hundreds or thousands of milliseconds, and fine structure of speech, characteristic of many consonants, occur in the tenth-of-millisecond range

Starr et al., (1991) found that marked impairment of monaural timing is consistent with poor speech and word comprehension that was disproportionate to the extent of pure tone hearing loss. It was observed that the subject with auditory dys-synchrony had difficulty distinguishing words differing in their vowels but could distinguish words bound on their high frequency consonants. In contrast to the loss or marked impairment of auditory percepts dependent on temporal cues, which were out of proportion to the changes in threshold, those percepts utilizing frequency or intensity features were less affected. They hypothesized that a disorder of the peripheral part of the auditory system at the eighth cranial nerve principally affecting the temporal precision of the neural coding and or its transmission centrally, could account for the loss of auditory percepts dependent on temporal cues and those evoked potentials sensitive to acoustic transients.

Kraus et al., (2000) reported a subject with auditory dys-synchrony whose speech perception in quiet was excellent for sentences and words, where contextual and multiple acoustic cues are available, but were markedly impaired in noise. Results of this study indicated that synchrony is more critical for understanding speech in the presence of noise. They also observed that subjects with auditory dys-synchrony had perceptual difficulty in response to sound containing critical acoustic information at stimulus onset, rather than to stimuli requiring discrimination of durational cues within a syllable. The subject had good discrimination for speech sounds along |ba-wa| continuum but poor discrimination for |da-ga| contrast, because difference between |da| and |ga| occurs at stimulus onset while the difference between |ba| and |wa| occurs with in the syllable. It is due to the deficits in the representation of transient stimulus cues related to the onset of voiceless syllables, where as longer duration harmonic aspects of voicing appeared to be preserved.

A few investigations have compared the results of psychophysical tests with speech perception to understand the characteristics of perceptual abilities of subjects with auditory dys-synchrony. Shivaprakash & Manjula (2003) studied temporal resolution (using gap detection) and speech identification score in two subjects with auditory dys-synchrony. Results showed that the subject showed poorer temporal resolution ability performed poorer on speech identification task when compared to the subjects with better temporal resolution ability. They hypothesized that speech identification scores varies with the degree of auditory dys-synchrony in neurons.

Zeng et al., (1999) stated that in their subjects temporal integration ability could not explain the poor speech recognition in auditory dys-synchrony subjects. However the impaired ability to follow temporal fluctuations is likely the underlying cause for the poor speech recognition. It has been assumed that the main effect of the dys-synchronous activity is a smeared temporal representation of the acoustic stimulus. If the listening task is merely detection of either presence of or absence of a sound, then this smeared representation would not present a difficult perceptual problem. However, if the task was discrimination of two different waveforms, one with gap and one without gap (i.e, speech), then the smearing in the internal representations would result in a much more difficult perceptual task. Thus, it can be hypothesized that subjects with auditory dys-synchrony have severe discrimination of fine temporal aspects, which occur within speech sound.

Rance, McKay, and Grayden (2004) reported that the degree of temporal disruption was strongly correlated with speech perception ability. They hypothesized that the inability of some subjects to perceive amplitude modulation fluctuation at modulation rate as low as 10Hz suggests a degree of temporal processing abnormality far greater than that reported for subjects with sensory neural hearing loss and is sufficient to disrupt perception of amplitude envelope cues in normal speech. It was also observed that subjects with good speech perception had normal DLF where as subjects with severely depressed DLF showed poor speech perception.

To summarize, it can be stated that the extreme speech perception difficulties experienced by some of the subjects with auditory dys-synchrony may reflect their

inability to cope with the dynamic nature of speech signals (Rance et al., 2004). To accurately discriminate phoneme in running speech, or even in individual words, a listener must be able to perceive the characteristic spectral shapes of phonemes, and in addition, be able to rapidly update this perception to follow the flow of speech sounds. Further more, a listener must also be able to follow within- phoneme changes in spectral pattern that give cues to co-articulation. Inability to perceive frequency changes, amplitude changes, and temporal changes due to dys-synchrony may lead to a smearing of their spectral shape perception, and a reduced ability to use amplitude envelope cues in speech (Rance et al., 2004).

Speech Identification Scores and electrophysiological tests

To understand the relationship between evoked potentials and speech perception in subjects with auditory dys-synchrony, it is necessary to consider some underlying mechanism for both. Late latency response appears to be more strongly related to auditory perception than are the compound action potential (CAP) and ABR, which are highly dependent on neural synchrony (Hood, 1999). Obligatory cortical related potentials are dependent on neural synchrony, but to a lesser extent than the ABR. The cortical related potentials are thought to reflect postsynaptic potentials in the dendritic zone of neurons within the auditory cortex (Speckmann and Walden cited in Ranee et al., 2002), where as the ABR appears to reflect the volume conducted action potentials of the axons. It is therefore possible to obtain cortical related potentials when the ABR peaks are absent, although such a circumstance indicates abnormal functioning of the auditory system (Rance, Cone-Wesson, Wunderlich, and Dowell, 2004).

ABR peaks tend to cancel when discharges are separated by fractions of a millisecond. In contrast, for cortical potentials, the waves are so slow that contributions separated by several milliseconds contribute to these later waves. While the ABR reflects highly synchronous discharges with microsecond precision, the synchrony required for cortical potentials is on the order of several milliseconds. These differences demand that the rate of stimulation be slower for cortical than for brainstem responses, limiting the precision with which timing information can be represented. It has also been reported that MLR, $P_1/N_1/P_2$, MMN, and P_{300} cortical responses were robust and present in quiet, however cortical potentials deviated from normal. They hypothesized that, the neural representation of these signals depends on the precise neural synchrony known to characterize signal neuron activity in auditory cortex in response to the onset of acoustic signals (Phillips cited in Krous et al., 2000).

Rance et. al., (2002) studied speech perception and cortical event related potentials in children with auditory dys-synchrony. They studied unaided and aided speech perception assessments (PBK words), and cortical event related potentials. They observed that the response presence (at normal latencies) was consistent with reasonable speech perception ability and response absence was consistent with negligible speech perception ability.

Vanaja and Manjula (2002) studied the usefulness of cortical evoked potential in predicting benefit derived from amplification in subjects with auditory dys-synchrony. Results showed that subjects with absent cortical evoked potential benefit lesser from

hearing aid and subjects with better morphology of cortical evoked potential benefit more from hearing aid.

As discussed earlier, one of the first steps in the perception of speech is to discriminate changes in the frequency and amplitude of a sound. The ability of the brain to detect changes in frequency and amplitudes can be assessed by recording ASSR to modulated in frequency and amplitude of suprathreshold tones (Dimitrijevic, John, van Roon, and Picton, 2004). Recording both 40 Hz and 80 Hz modulation response results in better relationships with word recognition score than with just one range of modulation frequencies.

The general idea behind using amplitude frequency modulation to predict word recognition score is that speech contains acoustic information that varies rapidly in intensity and frequency. The threshold at which amplitude modulation and frequency modulation became detectable can be used to assess the ability of the auditory system to recognize changes in intensity.

The electrophysiologic thresholds for detecting modulation using steady-state responses are close to those obtained behaviorally (John & Domitrijevic cited in Dimitrijevic et al., 2004). It has been suggested that the responses to rapid AM and FM might provide useful measurements of frequency and intensity discrimination (Dimitrijevic et al., 2001). Evaluating the steady state response at faster rates may help detect individuals who have problems processing rapid formant-transitions in speech

(Tallal et al.,1996). The ability of the auditory system to follow rapid changes in the frequency or amplitude of the sound is assessed psychophysically by means of temporal modulation transfer function (Viemeister, 1979). Because the temporal modulation of speech sounds may serve as cues to facilitate the recognition of consonants (Shannan, Zeng, Kamath, Wygonski & Ekelid, 1995), in the same way the ability of the steady state responses to follow rapid modulation frequencies may also relate to the ability of the auditory system to understand speech.

The presence or absence of a response may be more important for discrimination of speech than the absolute amplitude of the responses. Absolute amplitude may vary with stimulus intensity and with volume conduction in the head and these effects may distort the relationships with speech discrimination (Dimitrijevic et al., 2001).

To summarize, a review of literature indicates that there is a relationship between cortical evoked potential and speech identification scores. In subjects with auditory dys-synchrony the measurement of cortical related potentials allows a gauge of the severity of the synchrony disorder. When cortical related potentials are present and short latency potentials are absent, it signifies that some synchrony, at least at the cortical level is preserved. This residual synchrony encodes some temporal information needed for speech perception. ASSR may also provide additional information that can explain the speech perception abilities of an individual.

METHOD

Subjects

The subjects were divided into two groups, experimental group and control group. Experimental group included seven subjects with auditory dys-synchrony, in the age range of 15 to 40 years, and who satisfied the following criteria.

a) Poor speech identification scores (disproportionate to pure tone average) given by Owens (1971) criteria.

b) "A" type tympanogram with absent ipsilateral and contralateral reflex.

c) Absent or severely abnormal ABR.

d) Presence of OAE.

e) Native speaker of Kannnada.

f) No history of speech & language problems.

Control group constituted often subjects, with normal hearing, in the age range of 15 to 40 years. All the ears in the control group had normal pure tone thresholds less than 15dBHL in the octave frequencies ranging from 250 to 8000 Hz (ANSI, 1989). Immittance evaluation ruled out middle ear pathology. There was no history of any otologic or neurological disorders.

Instrumentation

The following instruments were used for the study.

1) A calibrated two channel diagnostic audiometer was used for pure tone and speech audiometry.

2) A calibrated middle ear analyzer for examining the status of the middle ear.

3) A calibrated evoked potential system to record ABR

4) GSI Audera (Version 1.0.2.2) for recording ASSR.
5) Evoked otoacoustic emissions were measured using a calibrated otoacoustic emission analyser.

Material

Speech identification material developed by Vandana (1998) was used for checking speech identification score.

Test procedure

- Pure tone threshold, Immitance evaluation, measurement of otoacoustic emission and auditory brain stem responses were carried out to ensure that the participants meet the criteria of subject selection.
- Speech recognition threshold were obtained using Kannada paired word list (Rajshekher, 1976) and speech identification score was obtained at 40 dB SL (re: SRT), using standard speech identification list in Kannnada given by Vandana (1998). It ensured that the presentation level was at least below the subject's UCL. The test material was spoken by a female native Kannnada speaker and it was recorded into a computer through a mic in a sound treated room. The computerized material was, scaled using the Audio lab software so that all words were of similar intensity. Before each list, a 1 kHz calibration tone was recorded to adjust the VU meter of the audiometer to zero. A batch file was created with an inter-stimulus interval of four seconds. The material was then transferred to a digital tape recorder from which it was again transferred to a CD using a CD writer. During speech identification test, the subjects were asked to repeat sounds that are heard. The percentages of correct scores were calculated.

- For recording ASSR, subjects were seated comfortably in an armchair and were asked to relax the jaw and neck muscles. Stimuli were presented through TDH-39 earphones placed in MX-41/AR ear cushions. Electrode site were cleaned, before placing electrode at vertex (Cz), forehead (FPz) and two mastoids (Ml and M2). The electrode placed on the vertex formed the non-inverting electrodes, the electrode on the two mastoids were the inverting electrodes, while the common or ground electrode was placed on the forehead. Recording was done using the protocol given in Table 1. It was ensured that impedance at each electrode site was less than 5kohms and the interelectrode impedance difference was less than 3kohms.The test stimuli were 1000 Hz, 2000 Hz and 4000 Hz tones, amplitude and frequency modulated at rate of 80 Hz and 40 Hz. The modulations of both slow and fast rate were used to check the response contribution from both brainstem as well as higher level. An amplitude modulation depth of 100% and a frequency modulation width of 10% were combined to have maximum response amplitude. It was ensured that the subjects were awake while recording ASSR for 40Hz modulation rate. To obtain ASSR threshold, the level of the stimulus was increased from low level in 10 dB steps until a response could be detected.

TABLE 1: Protocol for ASSR

Types of stimuli	Amplitude & frequency modulated tone
Frequency	500Hz, 1kHz, 2kHz
Transducer	Supra aural earphone
Modulation frequency	80Hz & 40Hz
Amplitude modulated percentage	100%
Frequency modulated percentage	10%
Noise level criteria	- 134.7
Intensity	Was varied to get response

RESULTS

The results of 40 Hz and 80 Hz ASSR of the experimental group and a comparison of these results with those of control group are presented in this section. Non-parametric analysis was carried out to study the correlation between the results of ASSR and speech identification score. A brief clinical history and audiometric profile of the experimental group also included in this section.

Clinical history

Among seven subjects, six of them reported with the complaint of reduced hearing sensitivity and difficulty in understanding speech mainly in noise, one subject reported with complaint of only reducing hearing sensitivity. It was reported to be progressive in all of them. Negative family history was reported in all the subjects. In all seven subjects no other associated clinical symptoms were reported except one subject who had mastoidectomy in right ear.

Audiometric profile

The degree of hearing loss ranged from minimal to moderately severe sensory hearing loss. The hearing loss was symmetrical in all the subjects except one, who had profound mixed hearing loss in one ear and mild sensory neural hearing loss in the other ear. She had undergone mastoidectomy in the ear with profound hearing loss. The configuration of the audiogram was irregular. However, a majority of the subject had more loss at frequencies below 2 kHz and better threshold at 4 kHz.

Auditory steady state response

40 Hz ASSR

ASSR could not be recorded for one ear with profound hearing loss. Among the remaining thirteen ears, ASSR was present in all the ears except one, when the carrier frequencies used were 1000 Hz and 2000 Hz. However for 4000 Hz carrier tone, the responses were present in nine ears, absent in three ears and noisy in one ear. The difference between ASSR threshold and behavioral threshold ranged from 45 to 95dB with a mean of 65.9, 74.09 and 68.75 dB at 1000 Hz, 2000 Hz and 4000 Hz respectively. This difference is much higher than that observed for the control group. As shown in the Table 2, the mean difference between ASSR threshold and behavioral threshold in the control group was 37.5, 37.7 and 41.38 at 1000 Hz, 2000 Hz and 4000 Hz respectively.

Table - 2: Summarizes the mean difference between ASSR threshold and behavioral threshold in normal and experimental group.

	Normal group		**Experimental group**	
	Mean	**SD**	**Mean**	**SD**
1000 Hz	37.5	8.78	65.90	13.38
2000 Hz	37.7	11.53	74.90	15.94
4000 Hz	41.38	12.69	68.75	11.25

80 Hz ASSR

ASSR for tones modulated at 80 Hz were absent in all the subjects except one. The differences between pure tone thresholds and ASSR thresholds in that subject were at 50, 85, 85 in the right ear and 80, 75, 75 in the left ear at 1000 Hz, 2000 Hz and 4000 Hz respectively. This is again much higher than that reported for normal subjects by Thenmozhi (2004). The differences between ASSR threshold and normal threshold in normal subjects were 36.37, 32.62 and 34.12 dBSL with a SD of 11.7, 13 and 13.7 at 1000 Hz & 2000 Hz and 4000 Hz respectively.

Speech identification score:

The mean speech identification score ranged between 8 to 84% with a SD of 23.71. Only one subject had speech identification score of more than 50% (84% in right ear and 68% in left ear). In the remaining subjects speech identification score ranged from 8% to 48%.

Correlation between ASSR and Speech identification score:

Pearson product moment correlation was calculated with the difference between 40 Hz ASSR threshold and behavioral threshold as independent variable and speech identification score as dependent variable. Separate analysis was carried out for 40 Hz responses obtained at 1 kHz, 2 kHz and 4 kHz. The results revealed a very low correlation between the two variables (r =0.12 for 1 kHz & SIS, r =0.07 for 2 kHz & SIS and r =0.16 4 kHz & SIS). However the correlation values obtained were not statistically significant.

DISCUSSION

The results of the present study are discussed in context of the other reports in literature in this chapter.

In the present study, auditory steady state responses with 40 Hz modulation rate were present in a majority of the subjects with auditory dys-synchrony. But ASSR for 80 Hz modulation rate were either absent or noisy in all the subjects except one. Rance et.al., (2002) reported that ASSR for tones modulated at 80 Hz were present only at high intensity levels. There is a dearth for literature on ASSR for 40 Hz modulated tones.

The absence of 80 Hz steady state response in a majority of the subjects with auditory dys-synchrony can be explained by the hypothesis that, the steady state potentials, when elicited by tonal stimuli modulated at rates around 80 to 90 Hz, appears to have similar generators to the late components of the ABR. It has been reported that auditory dys-synchrony could be associated with a loss of myelin and could be localized to the Type 1 afferent auditory nerve fibers (Starr et al., 1996). Partial or complete loss of myelin can have profound effects on the generation, and velocity of propagation of action potential with in the auditory nerve fibers (Rance et all., 1999). The pathophysiologic changes in neural conduction properties associated with demyelination are likely to have profound effects on ABRs, which are reliant on the relatively precise synchronous response of a population of auditory nerve fiber. The results of this investigation indicate that the abnormality affecting the ABR probably influence the results of ASSR to 80 Hz also. The other possible explanation is that these subjects were not able to perceive the amplitude modulations presented at the rate of 80Hz.

Presence of steady state response to tone modulated at 40 Hz can be explained by one of the two hypothesis. ASSR when elicited by tonal stimuli modulated at rates around 40 Hz appears to have similar generator to the components of cortical evoked response. It has been reported in literature that cortical evoked potential may be present in subjects with auditory dys-synchrony (Kraus et. al., 2000; Vanaja & Manjula, 2002; Starr et. al., 1991). The presence of cortical evoked response and absence of ABR in subjects with auditory dys-synchrony has been attributed the difference in neural synchrony required for these two potentials. It has been reported that cortical evoked potentials require different neural synchrony compared to the synchrony required for relatively shorter latency responses (Kraus et.al., 2000). It is possible that in subjects with auditory dys-synchrony, ABR and higher modulation steady state responses which require higher synchronization is disrupted whereas slow neural synchrony (in order of several milliseconds or higher rates) required for cortical evoked potential and lower modulation steady state responses is intact. Thus according to the first hypothesis presence or absence of 40 Hz ASSR depends on the severity of dys-synchrony. The second hypothesis for presence of 40 Hz ASSR and absence of 80 HZ ASSR is that these subjects may fail to perceive amplitude and frequency modulation when presented at higher rate where as this perception is not affected when the tone is modulated at a lower rate. It has been reported that the ability of the brain to detect changes in frequency and amplitude, can be assessed by recording ASSR (Dimitigevic et al., 2004).

The presence of ASSR for tone modulated at 80 Hz in one of the subjects suggests that there is heteroginity in subjects with auditory dys-synchrony. It is possible that the severity of dys-synchrony is lesser in this subject and hence only ABR which

require highly synchronous firing was affected. However, if this was true, then ASSR to tone modulated at 40 Hz should have been present but results indicated that 40 Hz ASSR was present in one ear while it was absent in the other ear.

Speech identification score and ASSR

Statistical analysis showed week correlation between speech identification score and sensation level at which 40 Hz ASSR obtained. But correlation value was not significant. This could be because of small size. No statistical analysis was carried out to check correlation between 80 Hz ASSR and speech identification score as 80 Hz ASSR was absent in all the subject except one.

Noteworthy observation was that, the speech identification was relatively higher in this subject in whom 80 Hz ASSR could be recorded. And this subject didn't complain of difficulty in understanding speech. Presence of 80 Hz ASSR and absence of ABR suggest that, probably the severity of dys-synchrony is lesser in this subject compare to other subject in whom both 80 Hz ASSR and ABR was absent. This inturn would have led to better speech identification. Investigation with large number of subject may throw more light on this aspect.

To, conclude the results of the present study showed that 80 Hz ASSR was absent in a majority of subjects with auditory dys-synchrony but 40 Hz ASSR was present. There was no 40 Hz ASSR threshold in the present study.

SUMMARY AND CONCLUSIONS

Auditory dys-synchrony is a disorder in which cochlear amplification functions are normal but afferent neural conduction in the auditory pathway is disordered (Starr et al., 1996).

Difficulty in the understanding of speech is a consistent finding in subjects with auditory dys-synchrony (David & Hirish, 1979; Kraus et al., 1984 and Starr et al., 1996). Because there is some evidence that temporal cues play a role in the encoding of human speech (Saches, Vaight & Young, 1983), it is possible that the poor speech comprehension are associated with the reduced temporal synchrony of 8^{th} nerve firing (Zeng & Oba, 1999). Speech representation is not only depended on peripheral synchrony but also on cortical representation.

Electrophysiological test generally used in diagnosing auditory dys-synchrony is ABR. Extraction of ABR from the EEG signal achieved by averaging requires precise synchrony of neural firing for response definition. Pathophysiological changes with demyelization can change the timing of neural discharges due to stimulus and it can make the ABR unrecognizable (Starr et al., 1996). The ASSR, on the other hand may not require the degree of neural synchrony needed for identification of transient waveform. Hence they may be more resistant to the neural dys-synchrony that appears to arise in case of auditory dys-synchrony (Rance et al., 1999). A review of literature also indicates that in subjects with auditory dys-synchrony, ASSR is present at high SL for tone modulated at a higher rate but there is no information on ASSR for tones modulated at lower rate. Since the 40Hz ASSR has similar generator as cortical evoked response,

based on the results of studies on AMLR and ALLR in subjects with auditory dys-synchrony (Starr et al., 1991; Kraus et al., 2000), it was hypothesized that, subjects with ASSR present at low SL will have better speech identification score than those with ASSR absent or present at higher level. The second hypothesis was that the speech identification will be poorer in subjects with ASSR absent for both 40 Hz and 80 Hz modulations than those with ASSR absent for 80 Hz modulations and ASSR present for 40 Hz modulations.

To check the hypothesis ASSR and speech identification score were recorded from seven subjects with auditory dys-synchrony. ASSR was recorded using GSI Audera for tone modulated at 80 Hz as well as tone modulated at 40 Hz. ASSR threshold was established for tones with carrier frequencies of 1 kHz, 2 kHz as well as 4 kHz. Speech identification score were obtained using Speech identification material developed by Vandana(1998).

Analysis of the data revealed the following results:

1. 40 Hz ASSR was present in all the subjects except one subject in whom it was absent in one ear. In subjects with auditory dys-synchrony, the difference between ASSR threshold and behavior threshold was higher than that observed in the control group.
2. 80 Hz ASSR was absent in all the subjects except one, in whom it could be recorded at higher intensity.
3. Speech identification score was very poor less than 50% in all the subjects except one.
4. There was a very low correlation between speech identification score and SL at which 40 Hz ASSR was obtained but the value were not statistically significant.

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