Relationship between Auditory Long Latency Response and Speech Identification Scores in Individuals with Auditory Neuropathy

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Abstract

The present study aimed at investigating the relationship between speech identification scores and ALLR parameters in quiet and at 0 dB SNR in individuals with auditory neuropathy. In the process, 6 individuals with auditory dys-synchrony and 15 individuals with normal hearing in the age range of 12-40 years participated in the study. Speech identification scores were assessed by bi-syllabic words with and without noise. Cortical auditory evoked potentials were recorded for click and speech stimuli /ba/, /ga/ and /da/. Results revealed that there was no significant correlation between speech identification scores and parameters of ALLR on both normal hearing individuals and individuals with AD. However, the presence of ALLR does correlate with speech identification scores in both the groups in both the conditions. Speech evoked ALLRs had larger amplitude than click evoked in both the groups and conditions (with and without noise). Among the speech stimulus, /da/ elicited more number of ALLR responses in individuals with AD in both the conditions. To conclude, speech evoked ALLR can be recommended for clinical use for both normal hearing individuals and for clinical population (individuals with auditory dys-synchrony). /da/ stimulus could be used to elicit ALLR in individuals with AD.

Introduction

Auditory neuropathy (AN), more recently referred to as auditory dys-synchrony (Berlin, Hood & Rose 2001), is one of the hearing disorders in which cochlear outer hair cell function is spared but neural transmission in afferent pathway is disrupted. The integrity of cochlear function in these individuals is indicated by the presence of evoked otoacoustic emissions and/or cochlear microphonics (CM). The abnormal neural transmission or dys-synchrony in the auditory nerve fibers is indicated by the absence of auditory brainstem responses and acoustic reflexes (Rance et al., 2002).

Audiological and electrophysiological test findings in auditory neuropathy are suggestive of a retro-cochlear pathology, but the exact site of pathology and pathophysiological mechanism leading to auditory neuropathy is not known. Two physiological explanations proposed for the neurophysiological manifestations observed include, dys-synchronized spikes and/or reduced spike of the auditory nerves (Rance et al., 2002).

Some possible sites of lesion that could produce the audiometric and electrophysiological profile of AN include: inner hair cells, synaptic junction between inner hair cell and type I

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afferent nerve fibers, spiral ganglion cells, demyelinization of type I auditory nerve fibers and reduce number of type I auditory nerve fibers. Therefore, AN consists of many varieties, depending on the site of lesion (Starr, Picton, Sininger, Hood & Berlin, 1996).

Hearing sensitivity in individuals with auditory neuropathy may range from normal hearing to profound hearing impairment (Rance, Beer & Cone-Wesson, 1999; Starr, Sininger & Pratt, 2000). A majority of the individuals with auditory neuropathy have low frequency hearing loss with wide range of speech identification scores. These individuals typically have speech identification scores that are out of proportion to their degree of hearing impairment and do not benefit from conventional amplification. Poor speech perception abilities in these patients are attributed to abnormal temporal coding and asynchrony (Zeng, Oba, Sininger & Starr, 1999; Kraus et al., 2000; Rance, McKay & Grayden, 2004; Zeng, Kong, Michalewski & Starr, 2004).

Need for the study

In auditory neuropathy/dys-synchrony, auditory brainstem responses are severely disrupted. Hence, it might be expected that more central evoked responses such as the middle latency response and cortical auditory evoked potential (CAEP) would be similarly affected. However, CAEPs may be recordable in some cases of auditory neuropathy/dys-synchrony because these potentials are less dependent on synchronous firing of the auditory nerve than auditory brainstem responses. Many individuals with AD had normal CAEP latencies and amplitudes (Starr et al., 1996; Rance et al., 2002). Hence, the current study has been designed to record ALLR in individuals with normal hearing and also with AD.

Infants with auditory neuropathy and possible hearing impairment are being identified at very young ages through the implementation of hearing screening programs. The diagnosis is commonly based on evidence of normal cochlear function but abnormal brainstem function. This lack of normal brainstem function is highly problematic when prescribing amplification in young infants because of lack of thresholds. Cortical auditory evoked potentials may, however, still be evident and reliably recorded to speech stimuli presented at conversational levels. In these clinical populations, it can also be used to evaluate the benefits with rehabilitative measures. Thus, click and speech is used as stimulus to record ALLR.

Since in individuals with AD, the audiometric configuration varies widely, different speech sounds composed of different spectral energy composition would be preferable to obtain ALLR in individuals with AD. This might suggest the processing of different speech signal having different frequency energy concentration. There is a dearth of information in which they correlate whether speech evoked or click evoked ALLR parameters represents the speech perception ability in these individuals. Thus, the present study was under taken to record ALLR using three different speech stimulus having different spectral energy.

To date, only few studies have investigated the ALLRs using speech stimuli in auditory neuropathy individuals to predict speech identification abilities. However, these studies had a small number of subjects and reported conflicting results. Cortical auditory evoked potentials elicited using speech stimuli were not compared with the speech perception abilities to find which one correlates the best, whether the click or the speech evoked cortical potentials. No study has been done to correlate ALLR in noise and speech identification scores in noise. So research is required to optimize whether click evoked or speech evoked cortical potentials correlates better with speech identification scores in noise in individuals with AD and normal hearing individuals.

Aims of the study

Thus the current study was taken up with the aim to:

- know whether the ALLR vary for different speech sounds in quiet and with ipsilateral noise in normal hearing individuals and individuals with AN/AD.
- investigate the relationship between the click evoked ALLR and speech identification scores in quiet and in noise in individuals with normal hearing and with AN/AD.
- investigate the relationship between the speech evoked ALLR and speech identification scores in quiet and in noise in individuals with normal hearing and with AN/AD.
- know whether the non-speech stimulus or speech stimulus is better to elicit ALLR in individuals with AN/AD.
- know which speech sounds is more suitable to elicit ALLR in individuals with auditory dys-synchrony.

Method

Subjects

The subjects in the present study were in the age range of 12-40 years and were divided into two groups.

- Individuals with normal hearing (control group)
- Individuals with auditory neuropathy (clinical group)

Control group

A total of 15 ears from 15 subjects with normal hearing in the age range of 15 to 38 years were evaluated. The criteria considered for the selection of subject were as follows:

Subject selection criteria:

- Pure tone threshold were within 15 dB HL at octave frequencies between 250 to 8000 Hz for air conduction and between 250 to 4000 Hz for bone conduction.
- All the subjects had 'A' type tympanogram with normal acoustic reflex thresholds.

- Speech identification scores were greater than 90%.
- Speech identification scores in the presence of noise at 0 dB SNR were assessed and all of them had scores above 60%.
- Good ABR waveform morphology was present for all the individuals at 80 dB nHL for both 11.1 and 90.1/sec repetition rate.
- TEOAE's were present in all the subjects for both the ears.
- No history of any otological or neurological problems was reported.

Clinical Group

In the clinical group, 25 ears from 16 subjects with auditory neuropathy in the age range of 13 to 40 years were evaluated. The following criteria were considered for the selection of the subject:

- All the subjects had pure tone audiometry thresholds ranging from normal to moderate sensorineural hearing loss.
- Subjects had speech identification scores ranging from 0-100%.
- Speech identification scores in noise at 0 dB SNR were poor.
- All the ears tested had "A" type tympanograms with absent acoustic reflexes.
- TEOAE's or cochlear microphonics was present in all the ears tested.
- ABR was absent at 80 dB nHL for all the subjects even at 11.1/sec repetition rate.
- No history of any other observable otological or neurological problems was reported.

Stimulus generation

Syllables /ba/ /ga/ and /da/ were spoken by a male speaker and digitally recorded into a computer with the PRAAT software version 4.2.01 with a sampling frequency of 44,000 Hz and 16 bit resolution. These stimuli were edited in such a way that the voice onset time, burst portion and a little portion of the vowel was retained to make the syllable duration approximately 150 ms. The stimuli durations were 147 ms for /ba/, 146 ms for /ga/ and 150 ms for /da/.

Data collection

Speech audiometry

Speech identification scores were assessed with and without noise using speech material developed by Vandana (1998). The stimuli were presented through supra-aural headphones (TDH-39) using calibrated diagnostic audiometer (GSI-61). Speech perception in noise (SPIN) scores was assessed at 0 dB SNR by using SPIN CD developed by Vargesh (2004). SIS and SPIN scores were established at 40 dB above the SRT (speech recognition threshold) level.

Auditory Long Latency Responses (ALLRs) recording

Subjects were instructed to sit comfortably on a reclining chair and relax during the testing and to stay awake during the testing. They were also instructed to ignore the stimulus and restrict the movement of head, neck and eye during testing. Preparation of the subjects and electrode montage used to record ALLR was the same as used for ABR recording. The parameters used to record ALLR are given in Table 2.

Table 2: Parameters used to record ALLR

Stimulus parameters		**Acquisition parameters**	
Transducer	Insert ear phones ER-3A	Amplification	50,000
Type of stimulus	Clicks and speech stimuli /ba/, /ga/, and /da/.	Analysis window	-100 to 500 ms
Duration of the stimulus	Click- 100µsec /ba/- 147 ms, /ga/- 146 ms and /da/- 150 ms	Filters	1– 30 Hz
Intensity	80 dB SPL	Notch filter	Off
Presentation ear	Monaural	Artifact rejection	100 µV
Stimulus polarity	Alternating	Electrode montage:	
No of sweeps	300	Non-inverting	Vertex (Cz)
Repetition rate	1.1/s	Inverting	Test ear mastoid (A1/A2)
Ipsilateral masking	Without noise With noise at 80 dB SPL (0 dB SNR)	Ground	Non test ear mastoid (A2/A1)

The recording was done twice at each presentation level to check for the reliability. The waveforms elicited in this manner were shown to three experienced audiologists and they were asked to identify N1, P2 waves. They were not told about the condition and the stimulus for which the responses were obtained. The latencies and amplitudes identified in this way were compared across the judges and the waveforms in which the latencies and amplitude marked by at least two judges were similar were taken for analysis.

Results

Cortical auditory evoked potentials were present in all the normal hearing individuals. In some individuals with AD, AEP's were absent for all the stimuli or certain stimulus.

N1:

The mean value obtained for click evoked N1 latency in quiet condition was shorter for normal hearing individuals than the clinical group as evident from the Table 3. No ALLR could be recorded using click at 0 dB SNR in the clinical group. Though there was difference between N1 latency obtained for different speech stimuli in both the conditions between the groups, no specific pattern could be observed. N1 latency shift observed in the presence of noise in the clinical group was more than that in normals for /ba/ and /da/ stimulus. For /ga/ stimulus the N1 obtained in the presence of noise was less. The N1 latency obtained in both the groups for different stimuli is given in Table 3.

Table 3: Mean, SD for N1 latency and Z-values with significance level obtained for click and different speech stimulus in two conditions between both the groups.

Parameter	Control group		Clinical group		
N1 latency (msec)	Mean (N= 15)	Standard deviation	Mean	Standard deviation	/Z/
Click without noise	118.00	9.01	134.71 (N= 7)	19.81	1.872
Click with noise	121.46	6.86	-	-	-
/ba/ without noise	164.33	9.86	156.50 (N= 14)	15.38	**1.966***
/ba/ with noise	171.13	14.12	208.66 (N= 3)	62.93	1.245
/ga/ without noise	162.93	10.88	165.41 (N= 12)	13.30	0.782
/ga/ with noise	170.46	14.42	165.50 (N= 2)	7.77	-
/da/ without noise	158.73	9.67	164.38 (N= 18)	18.92	0.272
/da/ with noise	172.26	13.11	193.62 (N = 8)	44.00	1.164

*p< 0.05

For comparison of N1 latency obtained between the groups for each stimulus, Mann Whitney U test was carried out. A statistically significant difference was obtained for N1 latency elicited in quiet only for /ba/ stimulus and not for the other three stimuli (Table 3).

To check for the correlation between speech identification scores and N1 latency, Spearman's rank correlation was carried out. Both the groups did not show any significant correlation between speech identification scores and N1 latency evoked by all the stimuli in both with and without noise conditions. Both the groups showed a significant reduction in speech identification score in the presence of noise. However, SIS was poor in clinical group than in control group in both the conditions.

P2:

It can be inferred from the Table 4 that the mean P2 latencies elicited by different stimuli in different conditions were longer in clinical group compared to that of the controls. Though there was difference in P2 latency evoked for different speech stimuli in both with and without

noise conditions between the clinical and control groups, no specific trend was observed. P2 latency shift in the presence of noise in clinical group was more than that was observed in normals for /ba/ and /da/ stimulus.

Table 4: Mean, SD and Z-values with significance level for P2 latency for click and different speech stimulus for control and clinical group in both the conditions.

Parameter	Control group		Clinical group		
P2 latency (msec)	Mean (N= 15)	Standard deviation	Mean	Standard deviation	Z - value
Click without noise	181.71	7.84	184.85 (N= 7)	14.36	1.097
Click with noise	186.78	8.65	-	-	-
/ba/ without noise	215.14	12.66	219.42 (N= 14)	21.41	0.000
/ba/ with noise	225.00	14.81	248.00 (N= 3)	31.04	1.365
/ga/ without noise	217.78	4.47	228.16 (N= 12)	14.79	**1.957***
/ga/ with noise	226.64	9.21	234.00 (N= 2)	9.89	-
/da/ without noise	211.35	11.41	227.70 (N= 17)	17.02	**2.554***
/da/ with noise	225.14	12.40	253.00 (N= 8)	26.81	**2.133***

*p< 0.05

The Mann Whitney U test was carried out for the comparison of P2 latency evoked by each of the 4 stimuli and between the groups. There was a significant difference obtained for P2 latency evoked by /da/ and /ga/ in quiet and for /da/ evoked P2 latency at 0 dB SNR. None of the other stimulus condition was significantly different.

Spearman's rank correlation was carried out to check for relationship between SIS and P2 latency. There was no significant correlation between speech identification scores and P2 latency for both the groups. SIS obtained at 0 dB SNR showed a significant reduction in comparison to SIS obtained without noise in both the clinical and control group. However, the clinical group showed poor SIS than the control group.

N1-P2:

The mean amplitudes obtained from the clinical group were comparatively lesser than the control group in both the conditions. However, the amplitudes elicited in the presence of noise were lesser than amplitudes elicited without noise in both the groups. In both the groups, the amplitudes elicited by speech stimuli were greater than the amplitude evoked by the click stimulus without noise. The mean amplitude can be seen in table 5.

Table 5: Mean, SD along with Z-values with significance level for N1-P2 amplitude elicited by click and speech stimulus in two conditions for both the groups

Parameter	Control group		Clinical group		
N1-P2	Mean (N= 15)	Standard deviation	Mean	Standard deviation	/Z/
Click without noise	3.04	0.66	1.76 (N= 7)	0.67	**3.034***
Click with noise	1.97	0.44	-	-	-
/ba/ without noise	3.50	1.42	2.71 (N= 14)	0.76	1.811
/ba/ with noise	2.32	0.84	1.92 (N= 3)	1.26	0.772
/ga/ without noise	3.58	0.97	2.71 (N=12)	1.24	**2.172***
/ga/ with noise	2.24	0.81	1.45 (N= 2)	0.94	-
/da/ without noise	3.56	1.08	2.55 (N= 17)	1.21	**2.834***
/da/ with noise	2.41	1.18	2.12 (N= 8)	0.99	0.097

*p< 0.05

Comparison of N1-P2 amplitude between control and clinical group for each stimulus was done using Mann Whitney U test. A significant difference was noted between the groups for N1-P2 amplitude evoked by click, /ga/ and /da/ without noise, which can be seen in the Table 5.

On carrying out Spearman's rank correlation test, it was found that there was no correlation between SIS and N1-P2 amplitude evoked for any of the stimulus. SIS obtained at 0 dB SNR was significantly reduced in both the groups in comparison to SIS obtained without noise. SIS obtained in the clinical group were poor than SIS obtained in control group, in both the conditions.

It can be concluded from the results that there was no correlation between speech identification scores and parameters of ALLR in the clinical group. But in the control group, even though there was a significant correlation found in between SIS and parameters elicited by stimuli in few conditions, definite trend were not observed. Most importantly it could be observed from the data that /da/ stimulus could elicit ALLR from most of the individuals with AD in both the conditions. Click could elicit ALLR from a few individuals with AD in without noise, but failed to record ALLR in the presence of noise.

Discussion

The ALLR data obtained from the individuals with normal hearing was statistically analyzed. The results obtained from the statistical analyses are discussed below.

Latency

It has been noticed in the current study that the latencies of N1 and P2 evoked by speech stimuli were longer than those elicited by click in normal hearing individuals. This difference in latencies between click and speech stimulus was statistically significant in normal hearing individuals but not in the clinical group. This difference between the groups could be due to the pathological condition. The prolonged latencies obtained for speech evoked ALLR than click could be because a single mechanism in the auditory cortex might be involved in general temporal processing for speech and non-speech stimuli, but may underlie further processing of verbal stimuli (Liegeois-Chauvel, Graaf, & Laguitton 1999). Another reason could be due to the rise time of the stimulus i.e., click has steeper rise time than speech stimulus which can lead to shorter ALLR latencies (Onishi & Davis 1968).

Most of the individuals with AD had ALLR for speech stimuli than for click. This could be because the click is a short duration signal with steeper rise time and hence it requires high synchronous firing. However, synchrony is affected in individuals with AD, leading to abnormal ALLR. One more reason could be due to impaired detection of short duration signals in individuals with AD (Zeng et al., 2005). As click is a short duration stimulus, ALLR responses might have been severely affected than for speech evoked ALLR.

ALLR recorded for the speech stimulus in the increasing order from the individuals with AD was /ga/, /ba/ and /da/. The presence of ALLR for the speech stimulus dominated by different frequency spectral energy can be explained in terms of spectral and temporal theories. Since in individuals with AD, phase locking is affected leading to dys-synchrony in low frequency auditory nerve fibers (Rance, McKay & Grayden 2004; Zeng et al., 2005) ALLR elicited for /ba/ and /ga/ stimuli were more affected. However, the high frequencies which are represented by the place of excitation on the basilar membrane are unaffected (Starr, Picton & Kim, 2001). As the energy concentration was greater in high frequency for /da/, it might have resulted in the presence of ALLR in most of the individuals with AD.

The mean latency values in the presence of noise were increased when compared to ALLR evoked without noise for all the stimuli in both the groups. The shift in the latencies between conditions was statistically significant in control group but not in clinical group. This difference between the groups could be due to the pathological condition. Since N1 and P2 are obligatory potentials; the presence of noise at 0 dB SNR would have decreased the audibility of the stimulus. Hence, it led to prolongation of latencies in the presence of noise (Martin & Stapells, 2005). In addition to that, in individuals with AD, 0 dB SNR can cause disruption in the synchrony of auditory nerve fibers (Kraus et al., 2000). In most of the individuals with AD, both dys-synchronization and reduced number of fibers often coexists. This produces an average discharge pattern similar to background activity and exaggerates the masking affects seen in

these individuals (Zeng et al., 2005). This over masking affect could have lead to absence of ALLR in the presence of noise along with dys-synchrony in most of the individuals with AD.

The mean latencies of ALLR elicited for all the stimuli in both the conditions were greater for the clinical group than in control group; even though it was not statistically significant. It was also observed that some individuals with auditory neuropathy had normal latencies, whereas some had greater latencies. Large variation in latency was seen in individuals with auditory neuropathy.

The variability in latency across the individuals may be due to degree of dys-synchrony and underlining patho-physiology. In individuals with AN, one of the possible site of lesion is demyelination of auditory nerve fibers. Demyelination results in an increase in membrane capacitance and a decrease in membrane resistance. Thus, it leads to a delay excitation, reduction in the velocity of action potential propagation and an increase in conduction vulnerability (McDonald & Sears, 1970; Rasminsky & Sears, 1972). The repetitive activation of demyelinated fibers results in a progressive increase in conduction time of action potential and may lead to intermittent or total in their propagation (Rasminsky & Sears 1972). Therefore, the latencies of the evoked potentials would lead to prolongation. Another possible site of lesion in these individuals is axonal neuropathy. This axonal neuropathy reduces the number of neural elements but doesn't directly affect the conduction speed. The refractory periods of these fibers also tend to be normal and are capable of firing at higher rates. Therefore the classic signs of axonal neuropathy are reduction in whole nerve action potential rather than an increase in latency or broadening of potentials (Kuwabara, Nakajima & Hattori 1999). This might have lead to the latency variations observed in the clinical group.

Amplitude

The amplitude of ALLR elicited for all the speech stimuli was greater than click evoked ALLR in both the groups. However, it was not statistically significant. This amplitude of N1-P2 being greater for speech stimulus than click stimulus might be due to the duration of stimulus leading to temporal integration. The longer duration stimulus activated the neurons other than simply onset detectors in generation of ALLR waves (Alain, Woods & Covarrubias 1997) and minimal duration required for the temporal integration to take place is $\geq$ 30 msec (Forss, Makela, McEvoy & Hari, 1993).

The amplitude of N1-P2 complex also reduced at 0 dB SNR for all the stimuli when compared to without noise in both the groups. This difference in N1-P2 amplitude elicited in both the conditions was statistically significant in control but not in clinical group. Since ALLR is an exogenous potential, the presence of noise reduces the audibility of the stimulus leading to reduction in amplitude of N1-P2 (Martin & Stapells, 2005). Besides this, in individuals with

AD, the reduction in the amplitude of ALLR could be due to disruption of synchrony being more in the presence of noise (Kraus et al., 2000). The reduction in the amplitude was greater for /ba/ and /ga/ when compared to /da/ as phase locking ability is affected in individuals with AD (Zeng, Oba & Garde 2001).

The amplitude of ALLR elicited was greater for control group than clinical group in both with and without noise conditions. However, it was not significant. In clinical group, some individuals had normal N1-P2, whereas some had abnormal amplitude. The reduction in amplitude in the clinical group can be due to the site of the lesion and severity of the pathology (Kumar & Vanaja 2008).

Relationship between speech identification scores and ALLR

None of the groups showed significant correlation between SIS and parameters of ALLR in both the conditions. The lack of correlation between speech identification scores and ALLR could be due to the wide variability in ALLR parameters recorded from both the groups especially in individuals with AD. Another reason could be, ALLR is affected by large number of factors like sleep or drowsiness, background EEG etc.

However, the presence of ALLR did correlate with speech identification scores. Individuals who had greater than 60% of speech identification scores showed ALLR for all the stimuli. The reason for correlation between the presence of ALLR and speech identification scores is that the presence of cortical auditory evoked potential reflects some amount of preserved synchrony in central auditory system which contributes to better speech understanding despite the distortion that occurs at 8th nerve and auditory brainstem in these individuals (Kraus et al., 2000 & Rance et al., 2002).

Conclusions

It can be concluded from the above results that the speech elicits better ALLR than click. Hence, speech evoked ALLR can be recommended in clinical use for both normal hearing individuals and in clinical population (individuals with auditory dys-synchrony). /da/ stimulus could elicit ALLR from more number of individuals with AD in both the conditions. Hence, it could be a useful stimulus to elicit ALLR in individuals with AD. There was no significant relationship between speech identification scores obtained and parameters of ALLR recorded in both the conditions for both the groups. But there was a good relation between the presence of ALLR for different stimuli and speech identification scores obtained in both the conditions in individuals with AD. It can also be concluded that optimal auditory nerve and auditory brainstem synchrony do not appear to be essential for understanding speech in quiet listening conditions. However, synchrony is critical for understanding speech in the presence of noise.

Clinical implication of the present study

The study can have the following implications:

- It can be used as an electrophysiological tool to evaluate the processing of speech sounds in normal population as well as in the impaired population.
- The present study also suggests the usage of speech stimulus for eliciting ALLR in individuals with auditory neuropathy.
- It also suggests the usage of /da/ stimulus to elicit ALLR response in individuals with AD.
- ALLR can be used to assess the hearing ability in individuals with auditory neuropathy from whom behavioral thresholds cannot be obtained.
- Using different stimuli dominated by different spectral energy helps us in estimating the severity of pathology across speech spectrum.

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