Central recruitment in individual with auditory neuropathy

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Abstract Auditory neuropathy (AN) describes patients with dysfunction of the auditory nerve in the presence of preserved cochlear outer hair-cell receptor functions in presence of normal otoacoustic emissions and/or cochlear microphonics. In individuals with auditory neuropathy speech are disproportionate to their hearing sensitivity and reported to be dependent on cortical evoked potentials. In individuals with AN, who have normal cortical potentials have better speech identification scores when compared to those with abnormal cortical potentials reflect relation between the cortical potentials and the speech identification scores. One group comparison research design was used for present study. The purpose of the study was to compare shift in latency of LLR peaks at different sensation level in subjects with auditory neuropathy and age matched normal individuals. 6 subjects (11 ears) diagnosed as having auditory neuropathy and 6 subjects (12 ears) with normal hearing Sensitivity participated for the study. Pure tone audiometry, immittance, reflexometry and otoacoustic emissions were administered. ABR was recorded for all the subjects at a repetition rate of 11.1 at an intensity of 90 dB nHL. LLR was carried out at different intensity levels for/da/speech stimulus at an intensity of 90 dB nHL. Latency of N1 and P2 of LLR was calculated at different sensation levels for both the groups. Descriptive analysis was carried out to find out the mean and standard deviation for latency of N1 and P2 for both, AN and normal hearing group.

There was delay in latency of N1 and P2 for individuals with auditory neuropathy.

Keywords Auditory neuropathy · Late latency response · Central recruitment · Cortical potential

Introduction

Auditory neuropathy (AN) describes patients with dysfunction of the auditory nerve in the presence of preserved cochlear outer hair-cell receptor functions [10]. The integrity of cochlear function in this population is provided by the presence of evoked oto-acoustic emissions and/or cochlear microphonics (CM), and the abnormal neural transmission or dys-synchrony is indicated by the absence of auditory brainstem responses and middle ear muscle reflexes. In Auditory neuropathy subjects, in spite of abnormal or absent ABRs, the cortical potentials is present to speech and non speech signals [3–7, 10–13]. Speech perception abilities in these individuals are disproportionate to their hearing sensitivity and reported to be dependent on cortical evoked potentials [8, 10]. In individuals with auditory neuropathy, who have normal cortical potentials have better speech identification scores when compared to those with abnormal cortical potentials reflect relation between the cortical potentials and the speech identification scores [7]. Also the AN individuals with higher amplitude in cortical potentials reflect better speech identification scores and also show hearing aid to be more beneficial than those with lesser amplitude [14]. However, all the individuals with AN do not benefit with the hearing aids.

So far we cannot yet determine precisely the existing diagnostic methods in the clinical praxis, the exact region of the auditory affection. Such difficulty to determine the

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exact location of the change along with the diversity found in the audiological and neurological findings between the cases has raised objection on the relevant procedure for auditory habilitation and rehabilitation of the patients with such alteration.

In individuals with auditory neuropathy the acceptance of hearing aids is very less. One of the major reasons for non acceptance of the hearing aids is the poor speech identification scores. However, there is a need to explore whether the rejection of hearing aids is only due to poor speech identification scores or there is some other mechanism which is affecting the hearing aids acceptance.

The purpose of the present study was to compare shift in latency of Late Latency Response (LLR) peaks at different sensation level in subjects with auditory neuropathy and age matched normal individuals.

Methods

The study aimed to compare the latency shift between normals and AN subjects in LLR waveform at different sensation levels reference to pure tone threshold. To accomplish the goal two groups of subjects were taken. A total of 16 subjects were participated in the study. Participants were grouped into two groups. Group I: included individuals with normal hearing (control group), group II: included individuals diagnosed as auditory neuropathy. Normal subjects consist of 12 ears with normal hearing from 12 individuals. The age range was between 18 and 23 years with the mean age of 19.5 years.

For these Subjects air conduction thresholds was within 15dBHL from 250 Hz–8 kHz at octave frequencies and bone conduction thresholds within 15 dB from 250 Hz–4 kHz at octave frequencies. All the subjects had good speech identification scores ($> 90\%$), “A” type tympanogram with normal acoustic reflex thresholds, indicative of normal middle ear function. Presence of OAEs for non-linear broad band click stimulus, indicative of normal outer hair cells functioning. No abnormality was observed in click evoked ABR at 90 dBnHL at 11.1 rate of repetition with band pass filter between 100 to 3,000 Hz and there were Presence of LLR for/da/stimulus at an intensity of 90 dBnHL band pass filtered between 1 to 30 Hz and repetition rate of 1.1/s, along with No history of any observable medical or neurological impairment.

AN subjects: this group consists of 11 ears of 6 subjects who diagnosed as having auditory neuropathy. The age range was between 20 and 25 with mean age of 22.5 years. In this group Subjects had minimal to mild sensorineural hearing loss and diagnosed as having auditory neuropathy. All the subjects had poor speech identification scores ($< 50\%$). All of them had “A” type tympanogram with

absence of acoustic reflex, indicative of no middle ear function.

Presence of Oto Acoustic Emissions (OAEs) for non-linear click stimulus, indicative of normal outer hair cells functioning. Abnormality observed in click evoked ABR at 90 dBnHL at 11.1 rate of repetition with band pass filter between 100 to 3,000 Hz. Presence of LLR for/da/stimulus at an intensity of 90 dBnHL band pass filtered between 1 to 30 Hz and repetition rate of 1.1/s.

No history of any observable medical impairment has been observed.

All the audiological tests were carried out in the acoustically treated room with adequate illumination. A calibrated double channel clinical audiometer OB922 with TDH-39 head phones housed in MX-41/AR cushions with audio cups was used to estimate air conduction threshold and establish the speech identification scores. B-71 bone vibrator was used for bone conduction testing, calibrated immittance meter, GSI-TYMPSTAR used to assess the middle ear status. ILO-292 Echo port plus used for measuring otoacoustic emission, to confirm outer hair cells functioning. Auditory brain stem response (for click stimulus) and long latency responses (using/da/stimulus) was recorded using Biologic evoked potential system. The stimuli were delivered using ER-3A insert receiver.

Pure tone threshold for air conduction were obtained at octave frequencies between 250 and 8,000 Hz and from 250 to 4,000 Hz for bone conduction. Modified Hughson–Westlake procedure [1] was used to obtain pure tone threshold. Speech identification score was obtained at 40 dBSL with reference to speech recognition threshold in each ear independently. Phonetically balanced list was used for the same. Tympanometry was carried out using 226 Hz probe tone frequency. Acoustic reflexes were checked at 500, 1,000, 2,000 and 4,000 Hz pure tones, for both ipsi and contra laterally to rule out middle ear pathology. Transient Evoked Oto Acoustic Emissions (TEOAEs) were recorded using nonlinear broad band click. A total of 256 sweeps were presented to elicit TEOAEs. The eliciting stimulus was presented at around 75 dB peSPL. Prior to the TEOAEs recording appropriate probe fit was obtained. TEOAEs were considered to be present when the amplitude was greater than +6 dB SPL with a reproducibility of 80 %. TEOAEs were recorded to rule out the outer hair cells functioning based on presence or absence of it.

Auditory Evoked Potentials (AEP) recording: for AEP recording i.e., for both ABR and LLR recording. Subjects were asked to sit comfortably and relax on a reclining chair facing away from the instrument. They were also instructed to avoid extraneous movements of head neck and limbs during testing, as might interfere with the EEG recording. Subjects were shown videos during LLR recording to minimize the attention related factor for the same.

Table 1 Biographical information for normal hearing participants

No. of cases	Age range (in years)	Right ear mean PTA (in dB)	Left ear mean PTA (in dB)	Right ear mean SRT (in dB)	Left ear mean SRT (in dB)	Right ear mean SIS (in %)	Left ear mean SIS (in %)	Type of Tympanogram (right/left)
12	18–23 (mean:19.9)	9.86	10.6	12.5	14.58	100	100	A/A

No number, *PTA* pure tone audiometry, *SRT* speech recognition threshold, *SIS* speech identification score

Table 2 Biographical information for AN participants

No. of cases	Age range (in years)	Right ear mean PTA (in dB)	Left ear mean PTA (in dB)	Right ear mean SRT (in dB)	Left ear mean SRT (in dB)	Right ear mean SIS (in %)	Left ear mean SIS (in %)	Type of tympanogram (right/left)
12	20–25 (mean:22.5)	32.47	27.47	25	38.3	45.3	37.3	A/A

No number, *PTA* pure tone audiometry, *SRT* speech recognition threshold, *SIS* speech identification score

Table 3 Depicting the parameters used to record ABR and LLR

Stimulus parameters		
Stimuli	Click	/da/
Duration	100 ms	40 ms
Rate	11.1/s,90.1/s	1.1/s
Polarity	Rarefaction	Alternative
Sweeps	1,500	200
Intensity	90dBnHL	Variable
Transducer	ER-3A insert receiver	ER-3A insert receiver
Acquisition parameters		
Filter	100–3,000 Hz	1–30 Hz
Notch filter	On	Off
Artifact rejection	31 %	31 %
Gain	1,00,000	50,000
Time window	10 ms	500 ms
Replications	Two times	Twice
Electrode montage	Inverting-left mastoid (M1), right mastoid (M2), ground–M2/M1	Inverting–M1/M2, ground–M2/M1 non inverting–high forehead (FpZ)

LLR testing was initiated at 80 dBnHL for both the groups. Intensity was then gradually reduced if the observable LLR was noticed. Intensity was reduced by 10 dBnHL for both the group still no response was obtained. LLR was recorded twice at each presentation level to check for replicability. The responses were stored for further analysis. Later wave forms were recalled and analyzed for N1-P2 Complex the intensity at which N1-P1 occurred was noted.

Wave form analysis

The latency of P1, N1, P2 and N2 component of LLR were noted. The data obtained for the subjects were analyzed as follows:

Morphology of the LLR wave form across individuals with normal hearing and auditory neuropathy were discussed. The mean, standard deviation and range were computed for different components for both the groups obtained at different intensities. The **latency changes** in LLR component with respect to change in the intensity were analyzed for individuals in both the groups. Finally the differences in latency shift at equal SL levels for both the group were compared for N1 and P2 component.

Results

Descriptive analysis was carried out to find out the mean and standard deviation for latency of N1 and P2 for both the AN group and the normal hearing group. The details are given in table below.

On analyzing overall there was a delay in latency of N1 and P2 for individuals with auditory neuropathy. On subtracting the values at different sensation levels the latency of both the N1 and P2 is prolonged compared to the normal hearing individuals. Particularly at the lower sensation levels the latency is prolonged in individuals with auditory neuropathy compared to the normal hearing individuals.

Table 4 Mean and standard deviation for latency of N1 and P2 peaks of late latency response (LLR)

	Normals				Auditory neuropathy			
	N1		P2		N1		P2	
	Mean	S.D	Mean	S.D	Mean	S.D	Mean	S.D
30 dBSL	152.05	15.46	179.14	12.82	185.23	14.02	227.82	38.63
40 dBSL	137.37	13.45	166.45	16.11	154.82	5.46	196.37	22.53
50 dBSL	112.66	20.12	152.69	15.67	134.56	18.05	175.30	30.07
60 dBSL	97.13	14.52	140.99	12.61	116.13	9.64	158.44	11.99
70 dBSL	90.39	8.40	131.10	16.00	98.46	11.59	100.69	12.22

SL sensation level

Discussion

The latency of N1 and P2 is prolonged at all the sensation levels in individuals with auditory neuropathy compared to the normal hearing individuals. The higher shift in latency in individuals with auditory neuropathy could be due to the dys-synchronous firing of the nerve fibers. As there is a dys-synchronous firing of the nerve fibers in individuals with auditory neuropathy at different intensity levels the amount of action potentials being conducted will be less compared to the normal hearing individuals Tables 1, 2, 3 and 4. The prolongation could also be due to probably a central recruitment happening in individuals with auditory neuropathy. An abnormal growth of N100 has been reported in sensorineural hearing loss [2]. It was speculated that there is an abnormal growth of loudness in these subjects. However it is well learnt that the measures of intensity processes are normal in individuals with AN [15]. The mechanisms underlying altered cortical excitability in AN may reside within the cortex. An animal model of AN showing increased excitability of auditory cortex did not have a corresponding excitability change of inferior colliculus [9] The abnormal excitability of auditory cortex in AN may be likened to the central excitability changes encountered in disorders of other sensory systems following de-afferentation. This central recruitment might be one of the reason for non-acceptance of the hearing aids in individuals with AN, as it might be creating an abnormal loudness.

In conclusion, the significant Latency shift information in individuals with auditory neuropathy may serve as an objective measure to explain the underlining neural mechanism. This will probably help to define the varieties of AN types based on physiological processing information and also to choose a rehabilitation strategy accordingly e.g, Selection of type of compression circuit, while choosing hearing aid for AN individuals. However, the findings of the present study should be interpreted cautiously as it has been done on a small data. Further, the fitting of hearing

aids in the AN individuals can add more information in this regard.

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