Electrocochleography in Individuals with Auditory Dys-Synchrony

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Abstract

Auditory neuropathy is characterized by the absence or severe impairment of auditory brainstem responses with the preservation of otoacoustic emissions and/or cochlear microphonics. In the present study extratympanic electrocochleography was recorded in individuals with auditory dys-synchrony and those with normal hearing. Cochlear microphonics was present in all the individuals in both groups. Amplitude of the cochlear microphonics was significantly higher in individuals with auditory neuropathy/dys-synchrony subjects when compared to normal hearing individuals Latency of the cochlear microphonics was also significantly prolonged in individuals with auditory neuropathy/dys-synchrony. Further, for the normal hearing individuals the amplitude of the cochlear microphonics was enhanced when noise was presented in the contralateral ear whereas in individuals with auditory neuropathy/ dys-synchrony there was no difference in the amplitude across the two conditions. Presentation of contralateral noise did not affect the latencies in both the groups. Summating potential was absent in 40% of normal hearing individuals and action potential was present in all the individuals with normal hearing, whereas both the potentials were absent in all the subjects with auditory neuropathy/dys-synchrony. There was a significant reduction in the amplitude of summating potentials and action potentials in individuals with normal hearing when it was recorded in the presence of noise.

Introduction

Auditory neuropathy/dys-synchrony is a hearing disorder affecting auditory nerve function in the presence of preserved cochlear outer hair cell activity (Starr, Picton, Sininger, Hood & Berlin, 1996). The hearing loss is characterized by disproportionate effects on auditory temporal processes relative to pure tone thresholds with speech perception severely impaired (Starr et al. 1991; Zeng, Oba, Garde, Sininger & Starr, 1999). The site of lesion may be at the level of inner hair cells, auditory nerve fibers or the synapse (Starr et al., 1996, Berlin et al., 1998). Therefore auditory neuropathy/dys-synchrony appears to consist of a number of varieties depending on the site of lesion (Starr, Sininger & Pratt, 2000). Auditory neuropathy like conditions have been associated with disorders such as several neonatal illnesses (Deltnre et al.,1997; Berlin et al., 1998), neonatal hyperbilurubinemia (Deltenre et al., 1997; Rance et al., 1999), hypoxia (Harrison, 1998) and mitrochondrial diseases (Deltenre et al., 1997).

Physiological tests generally used in diagnosing auditory dys-synchrony are auditory brainstem responses and otoacoustic emissions. By clinical definition a subject with auditory neuropathy/dys-synchrony will have abnormal or absent auditory brainstem response with

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presence of otoacoustic emissions. Because normal auditory brainstem response can be recorded only when multiple neurons fire synchronously at the onset, even minor variation in the timing of neural discharge after each stimulus can make the auditory brainstem responses unrecognizable (Kraus et al., 2000).

Another evoked potential which can be used to check the integrity of outer hair cells and auditory nerve is electrocochleography. Electrocochleography is a measurement of stimulus related electrical potentials which include the cochlear microphonics, summating potentials and the action potentials of the auditory nerve (Ruth, 1994). These three potentials can be recorded independently or in various combinations (Ferraro, 2000). It has been reported that the cochlear microphonics recorded from subjects with auditory neuropathy/dys-synchrony is either of normal amplitude (Santarelli & Arslan, 2002) or they have higher amplitude and it persists for several milliseconds after a click stimulus (Starr, Sinninger, Winter, Derbery, Oba & Michalewski, 1998; Starr, Sinninger, Nguyen, Michalewski, Oba & Abdala, 2001; Starr, Sininger & Pratt, 2000; Santarelli & Arslan, 2002; Berlin, 1999). Very few studies have investigated summating potentials in subjects with auditory neuropathy/dys-synchrony and the results are equivocal. A few investigators have reported that in subjects with auditory neuropathy/dys-synchrony the amplitude of summating potentials is within normal limits (Santarelli & Arslan, 2002; Sheykholeslami, Kaga & Kaga, 2001) whereas others have reported there is an absence of summating potentials (Starr, 2001). There are also reports that the amplitude of summating potentials is abnormal i.e. a large positive summating potentials is noted in some of the subjects with auditory neuropathy/dys-synchrony (O'Leary, Mitchell, Gibson & Sanli, 2001). The compound action potentials in auditory neuropathy/dys-synchrony subjects is generally absent (Liang, Liu & Liu, 1999; Wang et al., 2002; Santarelli & Arslan, 2002) or is of very small amplitude (Wang et al., 2002) and is present at only high sensation levels (Liang et al., 1999).

Studies have revealed that dys-synchronization does not affect only afferent functioning but also has an effect on the functioning of the efferent system. One of the audiological tests which have been used widely to assess the integrity of efferent system is contralateral suppression of otoacoustic emissions. Subjects with auditory neuropathy/dys-synchrony demonstrate significantly reduced or no suppression of otoacoustic emissions (Hood, Berlin, Bordelon & Rose, 2003). Other investigators have reported similar findings (Abdala, Starr & Sininger, 2000; Hood & Berlin, 2001). However the effect of contralateral suppression on cochlear microphonics has not been studied.

The present study was conducted with the aim of studying summating potentials in individuals with auditory neuropathy/dys-synchrony using the extratympanic method as there are equivocal findings on summating potentials in subjects with auditory neuropathy/dys-synchrony. Study also aimed at investigating the effect of contralateral suppression on cochlear microphonics, summating potentials and action potentials in subjects with auditory neuropathy/ dys-synchrony as the effect of efferent system stimulation on these potentials has not been studied in these individuals.

Method

Nine participants with auditory neuropathy (18 ears), ranging in age from 10 to 22 years and twenty four participants (31 ears) with normal hearing ranging in age between 10 to 26 years

of age were included in the study. Pure tone thresholds were obtained at octave intervals between 250 Hz to 8000 Hz for air conduction and between 250 Hz to 4000 Hz for bone conduction. Immittance evaluation was carried out with a probe tone frequency of 226 Hz. Ipsilateral and contralateral acoustic reflex thresholds were measured for 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz. Both ABR and extratymapanic electrocochleography was recorded using Interacoustics EP15 system with insert earphone ER-3A and otoacoustic emission was recorded using ILO 292 instrument.

The auditory brainstem responses were recorded using rarefaction click stimuli with a repetition rate of 11.1/sec at 80 dB nHL intensity. For recording ECochG participants were made to relax on a reclining chair. Silver chloride (AGCL) electrode with conducting gel and a TIPTRODE was used for recording ECochG. The noninverting electrode was placed in the ear canal, ground electrode was placed on the nasion and the inverting electrode was placed on the opposite ear mastoid. It was ensured that impedance for each electrode was less than 5 KΩ. Broad band clicks with a repetition rate of 11.1/sec were presented at 80 dBnHL. ECochG was recorded separately for stimuli of rarefaction, condensation and alternating polarity.

Transient evoked otoacoustic emissions evoked by clicks presented at 65 dBSPL for the linear clicks were recorded. The probe with a tip was positioned in the external ear canal and was adjusted to give flat stimulus spectrum across the frequency range. The response was acquired using the linear averaging method. The two averaged TEOAE waveforms of each memory buffer composed of 260 accepted click trains were automatically cross-correlated and used to determine the reproducibility of the measured TEOAEs by the software. Responses were accepted when the reproducibility was 80% or greater. Broadband noise was fed through GSI-16 audiometer for recording contralateral suppression of otoacoustic emissions and electrocochleography. The intensity of broadband noise was 50 dBSPL for the control group and for experimental group contralateral noise was presented at 30 dBSL with reference to threshold obtained for broadband noise.

Latency and amplitude of cochlear microphonics, summating potential and action potential (N1) was measured. Latency and amplitude of cochlear microphonics was estimated from the waveform for rarefaction and condensation stimuli whereas latency and amplitude of the summating potential and action potential was estimated from waveform obtained for alternating polarity stimuli. The amplitude of cochlear microphonics was estimated from the average of two waveforms. Peak to peak amplitude values of summating potential and N1 action potential was measured from the alternating waveform.

Results and Discussion

The latency and amplitude of the cochlear potentials (Cochlear microphonics, summating potentials), action potential and amplitude of OAE were recorded in quiet and in the presence of noise. Figure 1 shows the representative sample of ECochG in an individual with normal hearing

Mean and standard deviation (SD) was calculated separately for each group. Wilcoxin signed ranks test was administered to check if there is a statistically significant difference between the measures obtained in quiet and in the presence of noise. Mann-Whitney "U" test

was carried out to check if there is a significant difference between the measures obtained for the two groups. SPSS software version 10 was used to carry out the statistical analysis.

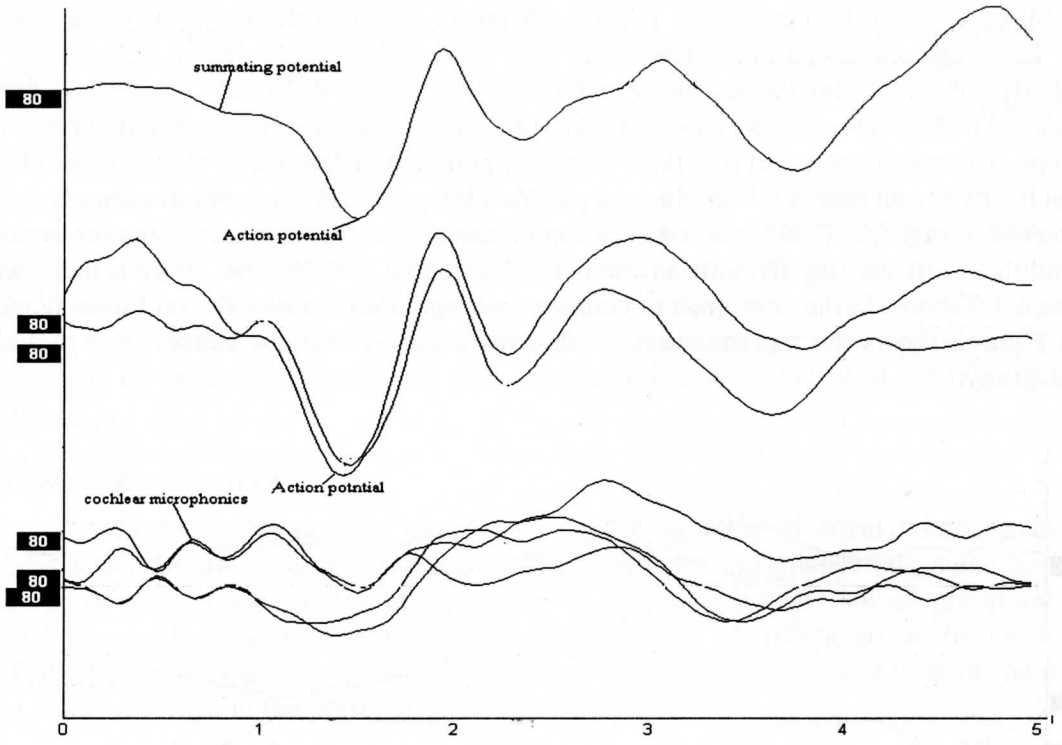

Figure 1: Representative sample of ECochG in an individual with normal hearing

1. Cochlear microphonics

Cochlear microphonics could be recorded in all the subjects in both the conditions i.e. in quiet and in the presence of noise. The mean and standard deviation values of latency and amplitude of cochlear microphonics are given in Table 1 for both the groups. It is clear from the table that the mean amplitude and latency for the cochlear microphonics in individuals with auditory dys-synchrony is more than that of the normal hearing individuals.

Table 1: Mean and SD of latency and amplitude of CM in control and experimental group

Conditions	Control Group		Experimental group	
	Mean	SD	Mean	SD
Latency (msec) without Noise	0.33	0.1606	0.45	0.193
Latency (msec) with noise	0.33	0.1432	0.52	0.2315
Amplitude (μv) without noise	0.08	0.03	0.23	0.07
Amplitude (μv) with noise	0.20	0.1036	0.21	0.06

However there was an overlap in the range obtained for the two groups. In the control group the latency ranged from 0.10 to 0.67 msec whereas it ranged from 0.20 to 0.90 msec for the experimental group. The amplitude ranged from 0.14 μv to 0.36 μv for the experimental group and it ranged from .03 μv to .15 μv for the normal group. There was an increase in the

mean amplitude value of the cochlear microphonics for the control group when noise was presented to the contralateral ear whereas for the experimental group there was no difference in the amplitude of the cochlear microphonics across two conditions.

Mann-Whitney U test showed that the difference between the two groups for the mean latency value and the mean amplitude value was statistically significant at .05 and .01 level respectively. (Z= 2.083 for latency and Z=5.691 for amplitude). Wilcoxin signed ranks test was administered to find out if there is a significant difference in latency and amplitude between two conditions. The results revealed that there was no significant difference in the latencies obtained between the two conditions for both the groups. (Z=0.90; p>.05) for the control group and for the experimental group (Z= 0.962; p>.05). The amplitude of the cochlear microphonics across the two conditions differed significantly in the control group (Z = 4.868; p<. 01) but there was no significant difference in the amplitude of cochlear microphonics across two conditions (Z=0.604; p>.05). Figure2 shows the representative waveforms obtained form an individual with auditory dys-synchrony.

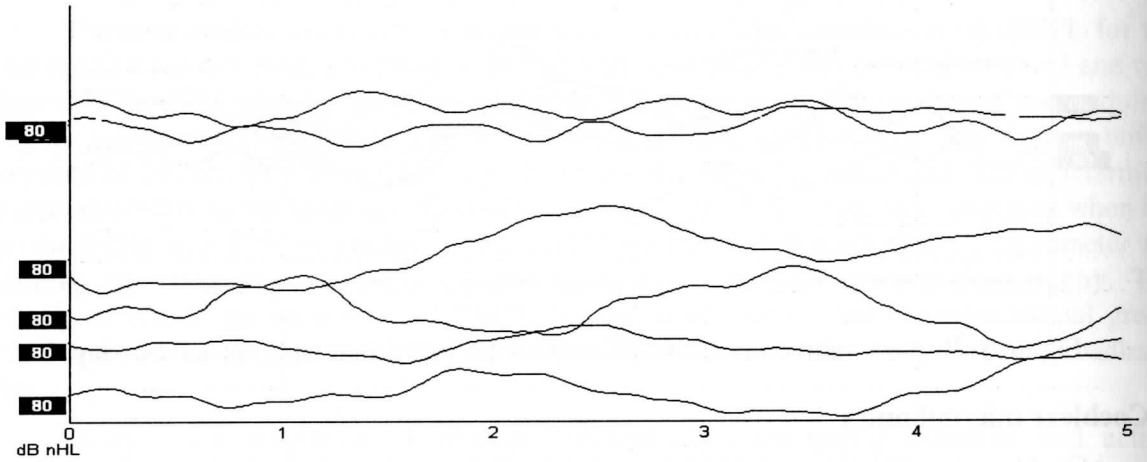

Figure 2: Presence of Cochlear Microphonics and absence of summating potential and action potential in an individual with auditory dys-synchrony

The mean latency of cochlear microphonics in normal hearing individuals is comparable with that reported in literature. In the present study the amplitude of the cochlear microphonics in the control group is lesser than that reported in literature. Starr et al., (2001) reported mean amplitude of 0.38 μv for the cochlear microphonics. The difference in amplitude obtained in the two studies is probably due to methodological differences. Starr et al. measured the amplitude of the cochlear microphonics from the subtracted averages to condensation and rarefaction waveforms and measured the amplitude at the peak where it had maximum amplitude. It has been found that the amplitude of cochlear microphonics in the subtracted waveform is twice that of the cochlear microphonics found in the separate averages to the condensation and rarefaction stimuli (Starr et al., 2001). In the present study due to technical limitations the subtracted waveform could not be obtained.

The amplitude of cochlear microphonics was higher in individuals with auditory dys-synchrony subjects than normal hearing individuals in the present study. In addition a long

ringing cochlear microphonics up to 1.4 msec was also observed in two of the individuals with auditory dys-synchrony. The cochlear microphonics prominence in auditory dys-synchrony subjects (Starr et al, 1998, 2001) as well as its persistence for several milliseconds after a click stimulus has already been reported by several authors (Berlin, 1999; Starr et al, 2001), who considered this finding an indication of an abnormal cochlear function (Starr et al., 2001). Santarelli and Arslan (2002) suggested that in subjects with auditory dys-synchrony the enhancement of both cochlear microphonics amplitude and duration of cochlear microphonics may result from the pathology in the afferent and efferent loop.

No effect was observed for amplitude of the cochlear microphonics in individuals with auditory dys-synchrony when contralateral noise was presented but the amplitude was enhanced in normal hearing individuals when contralateral noise was presented. The absence of enhancement of amplitude in individuals with auditory dys-synchrony/neuropathy may be due to deficit in the afferent system. The evidence comes from the previous study on contralateral suppression of otoacoustic emissions and acoustic reflexes (Hood et al., 2003; Berlin et al., 2005).

2. Summating potentials

The summating potentials could be recorded in 60% of normal hearing individuals whereas it was absent in all the subjects with auditory dys-synchrony. The mean and standard deviation of latency and amplitude of summating potentials for the control group are given in Table 2. It can be observed from the table that there is no difference in the latency of the summating potentials across the two conditions whereas there is a reduction in the amplitude when it was recorded in the presence of contralateral noise.

Table 2: Mean and standard deviation of the summating potentials in the control group

Conditions	Mean	SD
Latency (msec) without noise	0.70	0.20
Latency (msec) with noise	0.70	0.19
Amplitude (μv) without noise	0.21	0.11
Amplitude (μv) with noise	0.12	0.07

Wilcoxon signed ranks test revealed that there is no significant difference in the latency of the summating potentials whereas there is a significant difference found for the amplitude of the summating potentials between two conditions ($Z= 1.357$; $p>.05$ for the latency, and $Z=3.927$; $p<. 01$ for the amplitude).

The range of amplitude of summating potentials varied between 0.10 μv to 0.48 μv in normal subjects. Previous investigators have reported amplitude range between 0.04 μv to 1.30 μv (Ferraro & Durrant, 2004; Ferraro, 2003; Chatrian, Wrich, Edwards, Turella, Kaufman & Snyder, 1985). The latencies of the summating potentials in normals varied between 0.44 msec to 1.17 msec with a mean latency of 0.7 msec. Similar results have been reported by previous investigators (Chatrian et al., 1985). But the upper limit was higher in the present study than reported by earlier investigators. The smaller amplitude and longer latency observed in the present study may be attributed to the placement of electrodes. Extratympanic placement was used in the present study whereas majority of earlier investigators have used transtympanic recording (Santarelli & Arslan, 2002). It has been well established in literature that the amplitude

of potentials is higher in transtympanic method compared to extratympanic method (Starr et al., 2001; Santarelli & Arslan, 2002).

Furthermore the amplitude of summating potentials was suppressed after the presentation of contralateral stimuli. The suppression of amplitude of the summating potentials may be due to activation of the efferent system. Fex (1959) and Gans (1977) reported that efferent stimulation reduces the amplitude of the summating potentials. Additional measures of recording summating potentials may be necessary if we are to define whether this cochlear event is normal in subjects with auditory dys-synchrony. The measure is important because the generators for summating potentials include both types of hair cells with inner hair cells considered the principle generators (Durrant, Wang, Ding & Salvi, 1998; Zheng, Ding, McFadden & Henderson, 1997).

3. Action potentials

Action potentials could be recorded from all subjects in the control group but it was absent in all individuals with auditory dys-synchrony. The mean and standard deviation of latency and amplitude of action potentials for control group are given in Table 3. It can be seen from the table that there is no difference in the latency of the action potentials between the two conditions and a reduction in the amplitude of action potential was observed when recorded in presence of contralateral noise.

Table 3: Mean and SD value of action potentials in the control group

Conditions	Mean	SD
Latency (msec) without noise	1.46	0.15
Latency (msec) with noise	1.46	0.15
Amplitude (μv) without noise	0.51	0.22
Amplitude (μv) with noise	0.32	0.20

Wilcoxon sign rank test revealed that there is no significant difference in the latency of action potentials between the two conditions whereas there is a significant difference in the amplitude of action potentials across two conditions ($Z=0.174$; $p>.05$ for latency and $Z= 4.863$; $p<. 01$ for amplitude). The amplitude value of action potentials in the present study ranged from 0.28 μv to 1.20 μv. Previous researchers have reported amplitude of action potentials which varied between 0.6 μv to 5 μv (Ferraro, 2003; Ferraro & Durrant, 2004; Chatrian et al., 1985). The variations of amplitude across different studies are due to methodological differences such as the intensity used for recording ECochG and different electrodes used for recording ECochG (Ferraro, 2003; Ferraro & Durrant, 2004; Chatrian et al., 1985).

Further, there was no change in the latency of action potentials but there was reduction in amplitude of action potentials when the noise was presented to the contralateral ear. Folsom and Owsley (1987) also reported a reduction in the amplitude of action potentials but no change in the latency of action potentials after the presentation of contralateral noise. The reduction in amplitude of action potentials is attributed to the activation of the efferent system. It has been reported that activation of efferent system suppresses the amplitude of action potentials (Folsom & Owsley, 1987; Libermann, 1989).

The action potentials were absent in all the subjects with auditory neuropathy/dys-synchrony. The absence of action potential is expected in subjects with auditory dys-synchrony as there is a dysfunction of the auditory nerve. However a few investigators have reported

presence of wave I in ABR or presence of N1 in ECochg in some of the subjects with auditory dys-synchrony (Santarelli & Arslan, 2002; Starr et al., 1996). Santarelli and Arslan (2002) using transtympanic ECochG reported presence of N1 action potential component with broad morphology in few of the subjects with auditory dys-synchrony. Closer the electrode placement to the generator sites higher the chances of recording the action potentials as it enhances the signal to noise ratio. Therefore chances of recording wave I during ECochG is higher compared to far field recording.

Thus the present study reveals presence of cochlear microphonics in all individuals but the amplitude was higher and latency longer in individuals with auditory dys-synchrony. Summating potentials and action potentials were absent in all the individuals with auditory dys-synchrony whereas summating potential was present in 60% of normal hearing individuals and action potential was present in all normal hearing individuals. Further on presentation of contralateral noise there was an enhancement in amplitude of cochlear microphonics and suppression in the amplitude of OAE's, action potential and summating potential in normals whereas in individuals with auditory dys-synchrony there was no change in the amplitude of OAE's and cochlear microphonics when it was recorded in the presence of noise.

Conclusions

Thus it can be concluded from the present study that the summating potentials and action potentials cannot be measured using extratympanic recording in subjects with auditory dys-synchrony. Amplitude of cochlear microphonics is higher in individuals with auditory dys-synchrony when compared to that of normal hearing individuals and contralateral noise does not have any effect on the cochlear microphonics in individuals with auditory dys-synchrony.

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