PAMR: An Objective Tool to Measure Hearing Sensitivity in Individuals with Normal Hearing and Hearing Impairment

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Abstract

The present study was aimed to find the percentage of occurrence of post auricular muscle response (PAMR) in individuals with normal hearing and to estimate the hearing threshold in hearing impairment. The individuals with hearing impairment were divided into two groups. One group with individuals having sensorineural hearing loss and the other group with individuals having auditory neuropathy. PAMR was used to estimate the hearing threshold by using the protocol given by Purdy et al. (2005). The results showed that, for individuals with normal hearing the presence of PAMR at 80 dBnHL was 100% and above 90 % at 20 dBnHL for both males and females. No gender effect and ear effect was found for latency measures in individuals with normal hearing. In individuals with sensorineural hearing loss, the PAMR thresholds were significantly correlated with the puretone averages (PTA1 & PTA2). No ear effect was seen in individuals with sensorineural hearing loss. Hence, the PAMR can be used to estimate the hearing threshold in individuals for whom ABR cannot be done due to increased muscle tension and also for difficult to test population. The results also showed that the PAMR was not an effective tool to measure the hearing sensitivity in individuals with auditory neuropathy as most of the individuals in this group did not have a recordable PAMR.

Introduction

The post-auricular muscle response (PAMR) is a large sound-evoked muscle action potential that can be measured on the skin surface over the muscle behind the pinna. Bickford, Jacobson and Galbraith (1963) and Jacobson, Cody, Lambert and Bickford (1964) showed that a sound evoked myogenic potential could be recorded from electrodes placed over the post auricular muscle located behind the pinna. The PAMR can be evoked bilaterally from monaural sound stimuli such as clicks or tonebursts (Yoshie & Okudaira, 1969). The unique advantage of the PAMR was the sound-evoked PAMR is a large bipolar muscle action potential recorded at the skin surface just behind the ear. The PAMR can be much larger than the ABR, with amplitude that changes with the muscle tone in the post auricular muscle (Gibson, 1975).

There were many reports on the variability in recording the PAMR responses (Cody & Bickford, 1969; Picton, Hillyard, Krausz & Galambos, 1974; Bochenek & Bochenek, 1976). Until recently, because of the large variability in recording PAMR within and between the subjects it was not used for the threshold estimation. Patuzzi and O'Beirne (1999b) observed that the variability in recording PAMR was due to the uncontrolled eye movement and PAMR can be enhanced by turning the eyes towards the stimulation ear since there is a direct connection between the muscle tension and PAMR.

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Purdy, Agung, Hartley, Patuzzi and O'Beirne (2005) found the percentage of occurrence of PAMR in individuals with normal hearing is above 80% at the softest intensity levels when the eyes are turned towards stimulated ear. And also, good correlation between the PAMR threshold and the behavioral audiometric threshold were found in individuals with sensorineural hearing loss. Hence, the authors also suggest that the PAMR can be used as a screening tool with complement to ABR.

Need for the study

Though PAMR is acoustically elicited, it has not been extensively studied about its consistency and its clinical utility. If click evoked PAMR found to give consistent result, it can be used as quick tool to predict behavioral threshold. PAMR can be well recorded in almost 80 % of the normal population near the threshold (Purdy et. al., 2005). Hence, extensive studies on hearing loss population might testify the importance of PAMR as a clinical tool. If found reliable, it can also be used for other group of subjects such as difficult to test population since it has greater amplitude than ABR and also, can be recorded even when they are active (Purdy et al., 2005).

As the ABR is absent in individuals with AN/AD, it is difficult to estimate the threshold in children where behavioral threshold cannot be established. The PAMR may help us to estimate the threshold in these children if it is found to be an effective tool in adults. And also the classification of degree of individuals with auditory neuropathy may not be possible in most of the cases because responses were inconsistent and had peaked audiograms. Responses from 40% of the patients are judged as inconsistent (Kumar & Jayaram, 2006). PAMR, if found reliable, can be used to estimate the threshold since ABR will be absent in these subjects and cannot be used for threshold estimation. Thus, the current study was taken up.

Aim of the study was to:

- Estimate the percentage of normal hearing individual having PAMR responses.
- Find the PAMR responses in individuals with sensorineural hearing loss and individuals with auditory neuropathy.
- Establish the relationship between behavioral thresholds with the click evoked PAMR threshold in individuals with hearing impairment.
- Compare the PAMR parameters in individuals with normal hearing sensitivity and individuals with hearing impairment.

Method

The subject group was divided into three. Group I consisted of 30 individuals (60 ears) with normal hearing with the age range of 18 to 54 years (Mean - 22.4 years), group II consisted of 14 individuals (25 ears) with sensorineural hearing loss with the age range of 23

to 77 years (Mean - 47.2 years) and group III consisted of 10 individuals (20 ears) with bilateral auditory neuropathy with the age range of 18 to 40 years (Mean - 25.2 years).

Subject selection criteria

Group I

All subjects had hearing sensitivity within 15 dBnHL in both ears at frequencies 250 to 8 kHz with 'A' type tympanogram with normal of acoustic reflexes. TEOAEs were present and no abnormality in click evoked ABR in all of these subjects.

Group II

All subjects had hearing loss and the severity ranged from mild to profound degree with speech identification scores proportional to severity of hearing loss and air-bone gap not exceeding 10 dBHL. All had 'A' type tympanogram with present, elevated or absent acoustic reflexes and absent transient otoacoustic emissions. Latencies of click evoked ABR waves were appropriate to the degree of their hearing loss with good wave morphology at higher repetition rate in all of them.

Group III

All subjects had hearing sensitivity ranging from normal hearing to profound hearing loss and Speech identification scores were disproportionate to severity of hearing loss in all of them. All had 'A' type tympanogram with absent acoustic reflexes but presence of transient otoacoustic emissions. Absent ABR or poor ABR wave morphology with prolonged latencies were observed in all these subjects and were disproportionate to their degree of hearing loss. All of these subjects were diagnosed as primary auditory neuropathy by an experienced neurologist.

All the subjects participated in the present study did not have any symptoms or history of middle ear dysfunction and the middle ear pathology was ruled out by an otologist.

Instrumentation

A calibrated two channel diagnostic audiometer (OB 922- version 2.0) with TDH-39 head phone and B-71 bone vibrator were used to obtain pure tone thresholds and speech identification scores. A calibrated immittance meter (GSI- tympstar) was used to assess the middle ear function. ILO V6 OAE instrument was used to measure the TEOAEs. An evoked potential system [Intelligent Hearing System (USB Jr.)] was used to record the ABR and post auricular muscle response.

Procedure

The purtone thresholds for both AC and BC were tracked using modified Hughson and Westlake method (Carhart & Jerger, 1959). Speech identification scores (SIS) were calculated in percentage at 40 dB SL from SRT by using the speech material developed by Vandana (1998). Tympanometry was carried out using 226 probetone and acoustic reflexes

were found for frequencies 500, 1 k, 2 k and 4 kHz. TEOAEs were measured using the default setting in ILO V6 TEOAEs with 260 sweeps and non linear click trains at 85 dBpeSPL.

ABR was recorded in all the subjects participated in the study at two repetition rates (11.1/sec & 90.1/sec). PAMR was recorded in all the subjects by seating them in a comfortable chair. The inter electrode and intra electrode impedance were maintained at 2 kohm and 5 kohm respectively. They were instructed to turn the eyes towards the stimulated ear during the stimulus presentation. The PAMR was recorded by using protocol given by Purdy et al. (2005).

Table 1: Parameters used to record PAMR

Stimulus parameters		Acquisition parameters	
Stimulus type	Clicks	Transducer	Insert (ER -3A)
Stimulus duration	100 microsec	Mode	Monaural stimulation
Stimulus rate	17.1/sec	Electrode type	Disc electrode
Polarity	Alternating	Electrode montage	- ve : post auricular muscle(on the test ear mastoid) + ve: behind the pinna of the test ear. Ground: forehead
Intensity	80 dB, 50 dB and 20dB nHL for normal hearing subjects. Variable for subjects with SN hearing loss and auditory neuropathy	Analysis window	40 ms
		Filter settings	10 Hz – 300 Hz
		Notch filter	On
		No. of sweeps	250
		No of channels	Single channel
		Gain	10,000

For individuals with normal hearing three intensity levels were taken for finding the percentage of occurrence of PAMR (80, 50 and 20 dBnHL). For individuals with hearing impairment the threshold were estimated using PAMR by decreasing the intensity levels from 80 dB steps till PAMR was not observed and increasing in 10 dB steps till PAMR was observed. If not observed at 90 dBnHL the PAMR was recorded at 99 dBnHL. The minimum intensity at which the responses were observed was considered as the PAMR threshold.

The pi, ni and pii were marked in the obtained waveform based on the agreement between three experienced audiologists. The absolute latency and absolute amplitude were measured for each of these peaks. The data obtained were analyzed using SPSS (Version 16) software. Descriptive statistics was done to all the parameters of PAMR for each intensity level.

Results

Individuals with normal hearing:

The major peaks observed in individuals with normal hearing are pi, ni and pii across three intensity levels. The PAMR response could be recorded from almost 100 % of the normal hearing population at 80 dBnHL and approximately 90 % at 20 dBnHL either from right or left ear (Figure 1). However, the pii peak was not commonly observed in individuals with normal hearing.

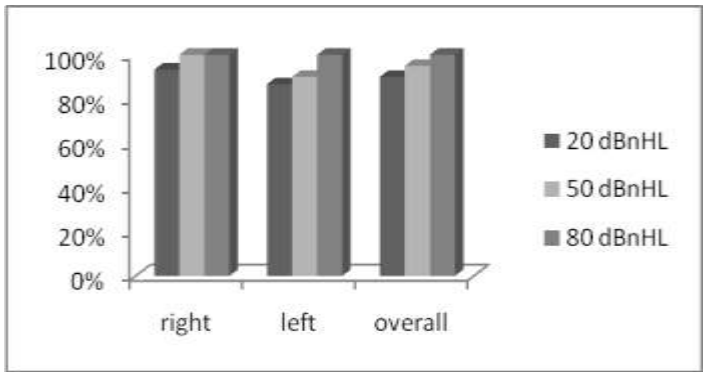

Figure 1: The percentage of PAMR occurrence in right and left ear and also for the both ears together (overall) obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

The effect of intensity, ear and gender on pi and ni latencies of PAMR was determined by Mixed ANOVA results. There was significant effect on pi latency [$F (2, 48) = 103.74$, $p < 0.001$)] and ni latency [$F (2, 48) = 35.942$, $p < 0.001$)] when the intensity is decreased from 80 dBnHL to 20 dBnHL. The Bonferroni post hoc analysis showed there were significant difference between 80 and 50 dBnHL, 50 and 20 dBnHL and also 80 and 20 dBnHL at $p < 0.001$ for both pi and ni latencies.

The Mixed ANOVA also revealed no significant difference in pi and ni latency between the males and females and also between right and left ear. The data of pi and ni latencies of males and females were combined and shown in the Figure 2.

There was a large amount of variation seen in the amplitude of ni which can be seen in Figure 3. Mixed ANOVA was used to determine intensity, ear and gender difference on pi and ni amplitude. The results revealed that, there was significant effect on pi amplitude [$F (2, 48) = 35.015$, $p < 0.001$)] and ni amplitude [$F (2, 48) = 28.03$, $p < 0.001$)] when the intensity is decreased from 80 dBnHL to 20 dBnHL. The Bonferroni post hoc analysis showed there were significant difference between 80 and 50 dBnHL, 50 and 20 dBnHL and also 80 and 20 dBnHL at $p < 0.001$ for both pi and ni amplitude.

The results also revealed a difference between the ears in ni amplitude when the intensity is decreased. Hence, paired t-test was administered and the results showed that there was significant difference between two ears at 50 dBnHL ($p < 0.05$) and at 20 dBnHL ($p < 0.01$).

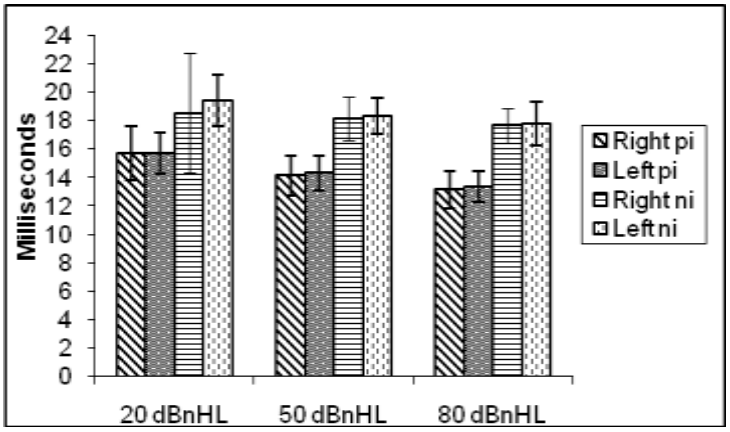

Figure 2: The Mean, SD of overall (Males & females combined) pi and ni latency obtained at 80, 50 and 20 dBnHL from right and left ear in individuals with normal hearing.

The Mixed ANOVA showed no difference between the genders and hence the data of pi and ni was combined and shown in the Figure 3. The results also showed that there was no interaction between the intensity, ear and gender for both pi and ni latencies and amplitudes.

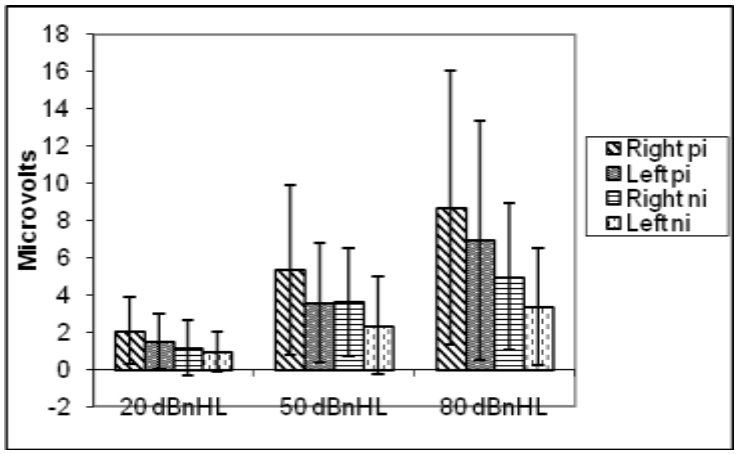

Figure 3: Mean and S.D of Overall (Males & Females combined) pi and ni amplitude for right and left ear obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

The percentage occurrence was around 40% for right ear and 15% for the left ear at 80 dBnHL and it even reduced in both ears at 20 dBnHL. Wilcoxon signed rank test results indicated that there was a significant difference in latency when the intensity was decreased from 80 to 50 dBnHL in the left ear ($p < 0.05$). It also indicated that there was a significant difference in amplitude when the intensity is decreased from 80 to 20 dBnHL for right ear ($p < 0.01$) and left ear ($p < 0.05$). However, no significant difference was found in other intensities for both the ears and also between the ears for pii latency and amplitude.

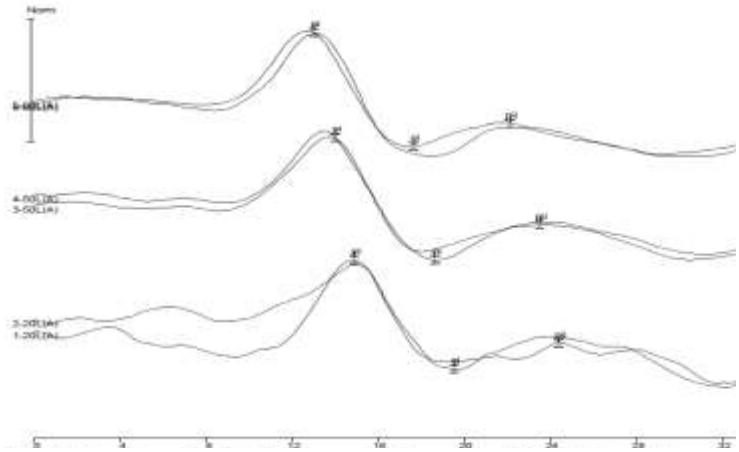

Figure 4: The click evoked PAMR obtained at 80, 50 and 20 dBnHL in a normal hearing individual.

Individuals with sensorineural hearing:

The PAMR was present in 19 ears out of 25 ears of sensorineural hearing loss tested. The PAMR was recorded in mild, moderate, moderately severe, severe hearing loss and profound sensorineural hearing loss. All the individuals who had mild, moderate and moderately severe sensorineural hearing loss had PAMR peaks. However, all the four ears with profound hearing loss did not have any recordable PAMR. Two out of five ears with severe hearing loss also did not have any PAMR.

Karl Pearson correlation coefficient revealed that there was a significant correlation between PTA 1(average of 500, 1 K and 2 kHz AC thresholds) and PTA 2 (average of 1 K, 2 K and 4 kHz AC thresholds) and PAMR threshold for both right ear and left ear. The results were shown in the Table 2.

Table 2: Karl Pearsons rank correlation coeffiecient and Mean difference of PTA1 & PTA2 with PAMR thresholds.

Thresholds	R - PAMR		Thresholds	L - PAMR	
	r-value	Mean Diff. (dB)		r-value	Mean Diff. (dB)
R - PTA1	0.844**	4.48	R- PTA1	0.911**	6.14
R - PTA 2	0.816*	5.53	L- PTA2	0.828**	7.95

[** $p < 0.001$ and * $p < 0.05$]

Note: R-PAMR: Right PAMR thresholds; L-PAMR: Left PAMR thresholds.
R-PTA1: Right PTA (500 Hz, 1 kHz& 2 kHz); L-PTA1: Left PTA (500 Hz, 1 kHz & 2 kHz).
R-PTA2: Right PTA (1 k, 2 kHz & 4 kHz); L-PTA 2: Left PTA (1 kHz, 2 kHz & 4 kHz).

The data obtained for left ear at 50 dBnHL was one and hence, the data obtained at 60 dBnHL was taken for the analysis instead of 50 dBnHL. So, between the ears comparison at 50 dBnHL could not be done. Wilcoxon signed Rank test results showed that there was a significant difference in the pi and ni latency in both ears when the intensity is decreased

from 90 to 70 dBnHL ($P < 0.05$). However, there was no statistically significant difference in latency for other intensities in both ears.

Wilcoxon signed rank test also revealed that there was a significant difference was obtained for ni amplitude between 90 and 70 dBnHL for both ears ($p < 0.05$). Whereas, only for the right ear, there was a significant difference in pi amplitude at 90 and 70 dBnHL ($p < 0.05$). No other conditions such as between the intensity levels within the ear or between the ears at the same intensity level could show a significant difference.

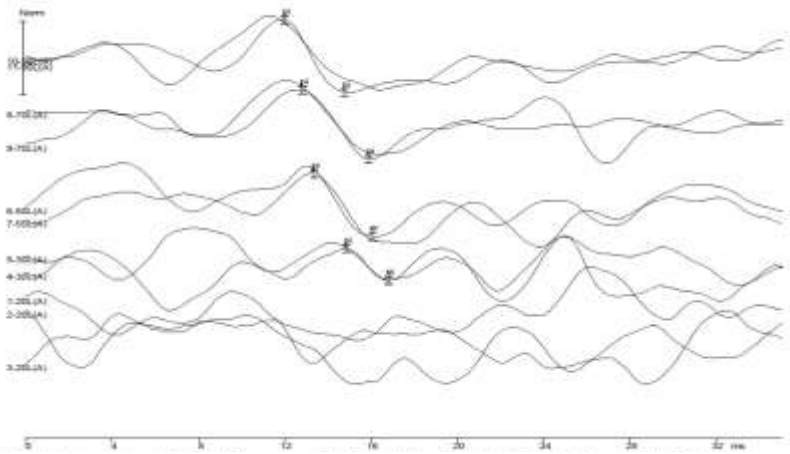

Figure 5: The click evoked PAMR recorded in a mild sensory neural hearing loss individual.

Individuals with auditory neuropathy

PAMR is recorded in 20 ears with auditory neuropathy. Out of 20 ears, only 3 ears had PAMR peaks. One subject who had normal hearing sensitivity in puretone air conduction threshold (both PTA1 and PTA2) in both ears had PAMR responses bilaterally. In right ear, the PAMR threshold was 30 dBnHL and left ear it was 50 dBnHL. Another subject who had mild hearing loss with the PTA 1 of 36.6 dBHL and PTA 2 of 28.3 dBHL also had PAMR response at 90 dBnHL. There was no trend seen in the latency and amplitude of pi and ni with respect to the intensity levels. The amplitudes obtained were much lesser. However, statistical analysis could not be done due to less number of data.

Group comparisons

The comparison was made at 80 dBnHL and 50 dBnHL between Group I and Group II. At 50 dBnHL only right ear comparison was made since, the number of data in left ear at 50 dBnHL in group with sensorineural hearing loss was too less. The individuals with auditory neuropathy were not compared with the control group since the number of data available was less and hence statistical analysis could not be done. Mann Whitney U test revealed that there was no significant difference between the two groups in latency and amplitude for both ears.

Discussion

The overall PAMR could be observed in 90% of the individuals with normal hearing at softest intensity levels. The results obtained in this study were consistent with the results obtained by Purdy et al. (2005). The possible reason could be the Excitatory Post Synaptic Potentials (EPSPs) from the auditory neurones probably add to the EPSPs from the eye-rotation neurones to reach action potential threshold with eye rotation (Patuzzi & O'Beirne, 1999 a, b).

The latency of pi and ni is significantly prolonged when the intensity was decreased. The results were consistent with the findings of Yoshie and Okudaira (1969); O'Beirne & Patuzzi (1999) & Purdy et al. (2005). The possible reason could be due to the larger excitatory post-synaptic potentials (EPSPs) in one or more of the neurones in the neural pathway reaching a firing threshold sooner with the higher intensity stimuli than with lower intensity stimuli, thereby initiating action potentials earlier (O'Beirne & Patuzzi, 1999).

The amplitude of pi and ni increased significantly when the stimulus intensity is increased. The findings were similar to the findings by O'Beirne & Patuzzi (1999) and Purdy et al. (2005). There was also large variations seen in the amplitude of pi and ni was seen in the current study. The possible reason could be due to the small average amplitude of the PAMR over many presentations was because of sporadic appearance of the PAMR, rather than by a small PAMR amplitude in every trace (O'Beirne & Patuzzi, 1999). Hence, for the clinical use of PAMR the amplitude measure may not be considered because of its larger variability.

There was a significant difference in ni amplitude across the ears. There was also mean difference noticed in pi amplitude between the ears which was not statistically significant. O'Beirne and Patuzzi (1999) reported that there was an increase in electromyography in the left post auricular muscle with eye rotation to the left and the EMG was largest in the right PAM with eye rotation to the right in two of the subjects tested. However, these authors do not mention about the amplitude difference between the two ears.

The occurrence of pii peak in normal hearing individual was less and even lesser in left ear compared to the right ear. This is in contradiction to the findings of Purdy et al. (2005) where they found about 80% occurrence of pii peaks at 20 dBnHL. The possible reason for lesser percentage of occurrences of pii peak of PAMR in left ear could be due to the lesser amplitude of ni which was significant. Since there is a difference found in the pi and ni amplitude between the two ears with left ear having lesser amplitude the ongoing EMG level would have obscured the presence of pii peak more in left ear. This could be evident since the pii peaks were observed in individuals who had quite larger pi and ni amplitudes and not in the individuals who had lesser pi and ni amplitude.

There was no gender difference seen in individuals with normal hearing. As expected, the same origin would be responsible for the generation of PAMR for both the genders.

The possible reason for the observable PAMR peaks in individuals with severe sensorineural hearing loss could be that the PAMR is a large muscle potential and largely dependent on the EMG rather than the compound action potential of auditory pathway which is responsible for the other neurogenic responses. The stimulus used was greater than their hearing loss and could have been sufficient to produce the PAMR responses through the eye rotation.

The PAMR was not obtained in any of the ears with profound hearing loss. The possible reason could be that PAMR is a myogenic response which is mediated by the auditory pathway. The subjects tested had no responses in behavioral threshold in most of the frequencies. The residual hearing was above 100 dBHL. As the stimulus is not conveyed to the auditory pathway the PAMR did not occur. Hence, the results strongly suggest that the PAMR responses are mediated by the auditory system.

The threshold obtained using the PAMR is highly correlated with the PTA1 and PTA2 of individuals with sensorineural hearing loss. The results were consistent with the findings of Thorton (1975b) and Purdy et al. (2005) were they found significant correlation with 2 kHz and PTA 2 respectively. The possible reason could be that it is likely the high-frequency cochlear regions dominate the click-evoked PAMR, as is seen for click-evoked ABR (Purdy et al., 2005). This could account for the PTA 2 correlation. In the present study PTA1 also well correlated with PAMR thresholds. This could be due to the subject's pattern of hearing loss. Most of the individuals with hearing loss had flat pattern. There was also very high correlation between PTA1 and PTA2 in the present study. The latency increased and amplitude decreased with decrease in intensity similar to individuals with normal hearing. Possibly, same mechanism would have involved in both the groups.

Hence, PAMR can be used as an alternative tool to measure the hearing sensitivity in hearing impairment when ABR could not be done due to increased level of EMG. PAMR can also be used for threshold estimation for difficult to test population since the PAMR thresholds were better correlated with audiometric threshold.

The number of individual with auditory neuropathy for whom the PAMR was observed was meagre. The possible reason for absence of PAMR in individuals with auditory neuropathy could be due to the altered temporal processing and auditory dysynchrony of the auditory nerve. From this finding it is clear that PAMR is not an effective objective tool to measure the hearing sensitivity in individuals with auditory neuropathy.

The latency of pi and ni obtained in one individual did not show any trend with respect intensity levels. For decrease in latency with increase in the intensity levels greater degree of synchronous firing of auditory nerve is required. Since there was a dysynchrony in the firing of the auditory nerve the threshold for reaching the action potential for PAMR would have been similar across the intensity levels. However, it requires more number of data to confirm these findings.

There is no statistically significant difference in latency and amplitude of pi and ni between the individuals with normal hearing and individuals with sensorineural hearing loss.

The possible reason could be that the cochlear damage may not disrupt the neural processing to that extent where the trigger for PAMR is affected, unlike the auditory dysynchrony. Moreover, the synchrony of the auditory nerve could have been preserved in individuals in sensorineural hearing loss.

Conclusion

It could be concluded from the study that PAMR is an effective tool to measure the hearing sensitivity when recorded with eyes turn condition. It can be used to estimate the behavioral threshold precisely when the subjects are more tensed and may not relax and also when the ongoing EMG activity is very high. It can also be used to estimate the behavioral threshold in difficult to test population since it requires lesser time than other evoked potentials. PAMR is not an effective tool to estimate the behavioral threshold in auditory neuropathy.

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