PAMR: AN OBJECTIVE TOOL TO MEASURE HEARING SENSITIVITY IN INDIVIDUALS WITH NORMAL HEARING AND HEARING IMPAIRMENT

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I dedicate this work to

my beloved

Appa and Amma

CERTIFICATE

This is to certify that this dissertation entitled ***"PAMR*: An Objective Tool to Measure Hearing Sensitivity in Individuals with Normal Hearing and Hearing Impairment**" is the bonafide work submitted in part fulfillment for the degree of Master of Science (Audiology) of the student, Register No. 07AUD005. This has been carried out under the guidance of a faculty of this institute and has not been submitted earlier to any other University for the award of any Diploma or Degree.

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This is to certify that the dissertation entitled ***"PAMR*: An Objective Tool to Measure Hearing Sensitivity in Individuals with Normal Hearing and Hearing Impairment"** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any Diploma or Degree.

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DECLARATION

I declare that this dissertation entitled ***"PAMR*: An Objective Tool to Measure Hearing Sensitivity in Individuals with Normal Hearing and Hearing Impairment"** is the result of my own study and has not been submitted in any other university for the award of any diploma or degree.

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1. 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).

The PAMR was initially used for the threshold estimation in 1960s and 70s. But there were many reports on the variability in recording the PAMR responses (Cody & Bickford, 1969; Picton, Hillyard, Krausz & Galambos, 1974; Bochenek & Bochenek, 1976). The PAMR responses are affected by number of factors such as subject state when they are more relaxed (Gibson, 1975; Humphries, Gibson & Douek, 1976) and asleep (Davis, 1976; Streletz, Katz, Hohenberger & Cracco, 1977), muscle tone, head and eye position (Patuzzi & Thomson, 2000), and recording filter pass band (O'Beirne & Patuzzi, 1999). Because of the large variability in recording PAMR within and between the subjects it was later not used for the threshold estimation.

The much of the variability in recording PAMR is due to the uncontrolled eye movement (Patuzzi & O'Beirne, 1999b). The PAMR can be enhanced by turning the eyes towards the stimulation ear since there is a direct connection between the muscle tension and PAMR. The muscle tension is enhanced by the eye rotation. Hence, they reported that by controlling the eye movement the PAMR can be used to estimate the hearing threshold reliably.

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. They also correlated the PAMR threshold with the behavioral audiometric threshold in individuals with sensorineural hearing loss and found a good correlation. Since PAMR is reliably recorded in adults it can also be recorded in infants in lesser time. Hence, the authors also suggest that the PAMR can be used as a screening tool with complement to ABR.

There were not many studies on PAMR in threshold estimation by controlling the eye movements. The studies done on PAMR on normal hearing individuals were less and with limited number of subjects. There is also very less information on estimating the hearing threshold in individuals with sensorineural hearing loss. There is no data available on PAMR on individuals with auditory neuropathy.

Need for the study

- PAMR can be well recorded in almost 80 % of the normal population near the threshold (Purdy et. al., 2005). Hence, an extensive study on hearing loss population might testify the importance of PAMR as a clinical tool.

- 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.

- The selection of hearing aid by prescriptive method is based on the behavioral threshold. Hence, obtaining behavioral threshold is important, which may be done using PAMR.

- 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. Responses were considered to be inconsistent if the thresholds varied more than 10 dB within a test session (Kumar & Jayaram, 2006). PAMR, if it is found to be reliable can be used to estimate the threshold since ABR will be absent in these subjects and cannot be used for threshold estimation.

- The prevalence and incidence of AN/AD in children are highly variant in different studies. Davis and Hirsh (1979), suggested that one in every 200 hearing-impaired children will have audiologic findings consistent with a contemporary diagnosis of AN. On the other hand, Berlin, Hood, Goforth-Barter and Bordelon (1999) estimated that AN/AD is present in at least 4% of children having permanent hearing loss. Rance et al. (1999) found that the likely

prevalence of AN/AD in a group of 5199 infants with neonatal and familial risk factors for hearing loss was 0.23%. Of the 109 children with permanent moderate or greater hearing loss within that group, the AN /AD prevalence rate was 11 %. As the ABR is absent in subjects 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.

- 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).

Thus, the current study was taken up.

Aim of the Study was to:

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

2. Review of literature

A sound evoked myogenic potential could be recorded from electrodes placed over the post auricular muscle located behind the pinna (Bickford, Jacobson & Galbraith, 1963; Jacobson, Cody, Lambert & Bickford, 1964). Kiang, Crist, French and Edwards (1963) first identified the PAMR, which was referred earlier as the 'sonomotor response' (Davis & Lowell, 1965). The post auricular muscle response (PAMR) in humans is likely to be a vestigial version of the Preyer reflex that causes the ears of some animals to move in response to sound (Gibson, 1978).

Douek, Gibson and Humphries (1973) and Humphries, Gibson and Douek (1976) obtained a response by stimulating the cochlea with clicks filtered to various frequencies. The averaging responses obtained from behind the ear, appeared between 12-20 ms. They termed this response as Crossed Acoustic Response (CAR). The authors also suggested that that the origin of this response could be a complex rather than a simple source. It is likely that at near-threshold levels of stimulus a neurogenic element is prominent. When the stimulus gets louder, the response is swamped by a powerful myogenic element originating in the post-auricular muscle fibers, supplied by the facial nerve.

The existence of such a reflex is no surprise when they consider that the muscles involved phylogenetically mobilize the pinna to locate the source of a sound (Douek, Gibson and Humphries, 1973). A similar reflex in the guinea-pig was described by Preyer (1881) and hence it was known as Preyer reflex. But its latency

was only 6 ms and, therefore, must not be confused with the crossed acoustic response which has a latency of 12-20 ms.

The latency difference might be due to the number of synapses in the pathway. The equal latency in normal persons suggests that the impulses pass through an equal number of synapses. It is the crossed nature of this acoustic response that has suggested the name Crossed Acoustic Response (Douek, Gibson & Humphries, 1973). However, the usage of the term 'Post Auricular Muscle Response' by Yoshie and Okudaira (1969) continued to denote this myogenic response.

Proposed pathway for PAMR:

The pathway of post-auricular muscle reflex activity was described by Douek, Gibson and Humphries (1973). A sound stimulus is converted in the cochlea to afferent nervous information which passes via the auditory fibers of the Vlllth cranial nerve to the brain stem. Here it is relayed bilaterally to the efferent motor neurones of the facial nerve nucleus. The efferent activity stimulates the post-auricular muscle fibers, which, lying superficially behind the ear, produce an electrical response that is easy to detect.

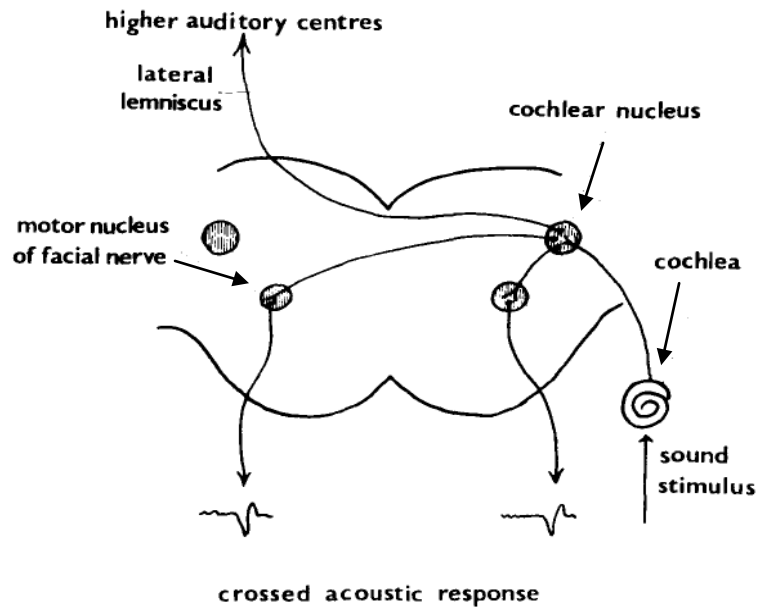

Figure 1: The pathway of PAMR described by Douek, Gibson and Humphries (1973).

Gibson (1975) has suggested a brainstem pathway consisting of the ventral cochlear nucleus, superior olivary nucleus, the nucleus of the lateral lemniscus and then either the reticular formation or the inferior colliculus. From here, the neural activity travels along the facial nerve to the PAM, producing the PAMR. It is at some point within the brainstem that the response is `split' and relayed bilaterally to the motor nuclei of the facial nerve on both sides of the head to produce the bilateral response. It is clear from these studies that the cochlea is the receptor organ driving the PAMR.

Bickford, Jacobson and Cody (1964) have found that its amplitude could be enhanced or abolished by contraction or relaxation of the post auricular muscle and that a local anesthetic blocks the post-auricular branch of the facial nerve abolished the response unilaterally. Yoshie and Okudaira (1969) and Gibson (1975) reported that PAMR can be obtained from subjects with abnormal vestibular function but normal hearing, but is absent in deaf subjects with normal vestibular function. Hence, this is the suggestion of cochlear origin.

O’Beirne and Patuzzi (1999) reported that the PAMR is often much larger than the more commonly recorded auditory brainstem response (ABR) and its amplitude alters with the muscle tone of the Post Auricular Muscle. The PAMR is often so large that it can be seen clearly in the raw (unaveraged) trace. It is also clear that the PAMR is of muscular, rather than neural, origin. The higher signal to noise ratio of the PAMR relative to the ABR means that less amplification is required to observe it and much less averaging is needed to produce a stable averaged waveform. Only 20 averages are necessary to produce a stable PAMR trace, when electrode placement is optimized.

Variability of PAMR:

Variability in recording PAMR was observed within and between subjects (Patuzzi & Thomson, 2000). This variability appears to result from factors such as

a) muscle tone,

b) head and eye position,

c) subject state, and

d) recording filter pass band.

PAMR is reduced when subjects are more relaxed (Gibson, 1975; Humphries, Gibson & Douek, 1976). Similarly PAMR is also reduced when subject is asleep (Davis, 1976; Streletz et at., 1977).

Cody and Bickford (1969) found the response to be absent in at least one ear of 32% of their subjects and absent bilaterally in 7% of their subjects. Picton et al. (1974) described PAMR as `highly variable from subject to subject and even within subjects'. Because of this variability, they considered that the PAMR would have least clinical application.

Bochenek and Bochenek (1976) stated that the disadvantage of the PAMR is the inconsistency or variability of its appearance. Such an individual variability is a very serious problem for applying the response as an index of objective audiometry.

O'Beirne and Patuzzi (1999) stated that the small amplitude of the PAMR in at least some subjects is not due to a small muscle mass for the Post Auricular Muscle. Rather PAMR occurs sporadically (eg. 18 out of 100 presentations), in such subjects, but with a near normal amplitude and latency. The sporadic appearance of the PAMR in these subjects contrasts with the more typical subjects, who produced PAMR responses reliably for nearly every presentation of a moderately intense stimulus (50 dB SL or higher). This could be the reason for poor amplitude in those individuals.

Enhancement of PAMR:

Research has shown that there are various ways through which the amplitude of post auricular muscle response can be enhanced. They are

a) neck traction (Cody & Bickford, 1969),
b) head lowering (Yoshie & Okudaira, 1969; Dus & Wilson, 1975),
c) teeth gritting (Dus & Wilson, 1975; Gibson, 1978),
d) propping the head forward (Yoshie & Okudaira, 1969; Thornton, 1975b),
e) pushing the head against a force (Clifford-Jones, Clarke & Mayles, 1979), or
f) lateral eye movement (Patuzzi & O'Beirne, 1999b; Patuzzi & Thomson, 2000).

Much of the variability in presence or absence of PAMR or PAMR amplitude may be due to uncontrolled eye movements in the test population (Patuzzi and

O'Beirne, 1999b). It has been known for many years that lateral movement of the eyes is associated with movement of the ears, at least in some subjects, and the phenomenon is known in the neurological literature as `Wilson's occuloauricular phenomenon' (Wilson, 1908; cited in Patuzzi & O'Beirne, 1999b).

In a recent study of this effect, an increase in muscle tone in the Post Auricular Muscles was found in 96% of normal subjects (Urban, Marczynski & Hopf, 1993). Its absence has been used in the differential diagnosis of various neurological disorders (Schmidt & Thoden, 1978).

Jacobson et al. (1964) stated that the amplitude of the PAMR can be greatly modified by changing head position and lateral movement of the eyes. However, less known about the effect of eye movement on PAMR for more than 20 years.

Patuzzi and O'Beirne (1999b), proposed three schemes in which they tried to explain the mechanism behind the enhancement of PAMR with eye rotation. In the first scheme the enhancement of the PAMR with eye rotation occurs at the motor nucleus of the facial nerve. They presume it is because Exitatory Post Synaptic Potentials (EPSPs) from abduscens neurones depolarize the motor neurones, bringing them closer to firing threshold. Also the authors expected that eye rotation would increase the amplitude of the sound-evoked PAMR and the background EMG almost concomitantly, since such a depolarization would make it more likely that both the sound-evoked and the non-sound-evoked EPSPs within these neurons would reach threshold more easily.

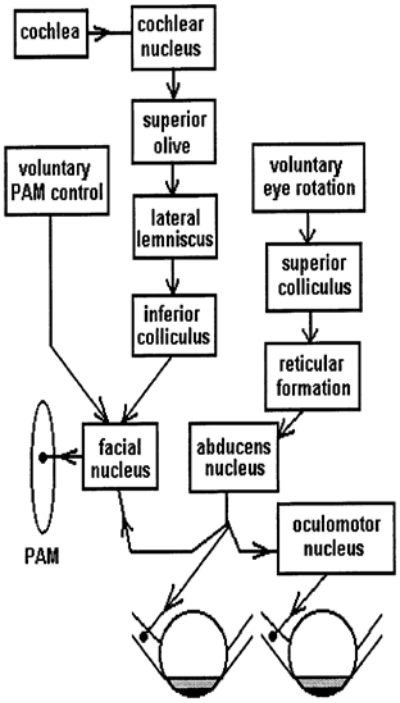

Figure 2: Pictorial representation of Scheme 1 (Patuzzi and O'Beirne, 1999b).

In the second scheme (Figure 3), eye rotation potentiates the PAMR in the auditory brainstem, in which case, eye rotation might increase the PAMR without altering the level of the tonic or voluntary EMG. Moreover, with this scheme, voluntary contraction of the PAM on its own may not potentiate the PAMR. This is because the sound-evoked neural drive is unable to pass through the brainstem without some other potentiating influence (e.g. eye rotation). That is, for scheme 2, an increase in tonic EMG would not be sufficient to enable or potentiate the PAMR. Neither it may be necessary if the neural drive from the auditory brainstem produces a sufficiently large EPSP within the cells of the facial nucleus which can evoke firing in the motor neurones on its own.

Both these two proposals (Figure 2 and 3) assume that the neural drive producing the non-sound evoked muscle activity does not itself pass through the auditory brainstem. It is certainly considered the scheme in Figure 3 to be improbable, since the EMG and PAMR are always seen to be co-activated, and both voluntary contraction of the PAM and eye rotation produce a similar potentiation of the PAMR, even though eye rotation produces less of an effect. (Patuzzi & O' Berine, 1999b)

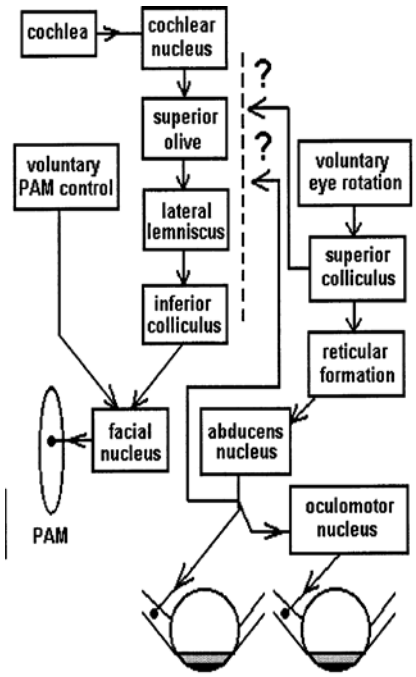

Fig 3: Pictorial representation of Scheme 2 (Patuzzi and O'Beirne, 1999b).

In the third scheme (Figure 4), however, all neural drive activating the post auricular muscle (from voluntary contraction, transient sounds or eye rotation) is assumed to pass through the auditory brainstem. Since the pinna is fundamentally part

of the auditory system this possibility cannot be ruled out. In animals with highly mobile pinna, changes in pinna position are associated with altered auditory mapping within the central nervous system (Middlebrooks & Knudsen, 1987).

As a result, it is reasonable that all neural control of pinna movement should be relayed via the auditory brainstem, providing it with an `efference copy' of the movements of the pinna, allowing re-mapping of the auditory fields as required to maintain a fixed body image as the pinnae rotate. Such feedforward `recalibration' of the maps within the central nervous system occurs routinely in the visual system, where visual maps along the central visual pathways are constantly re-calibrated as the eyes move relative to the head (Weyand & Malpeli, 1993).

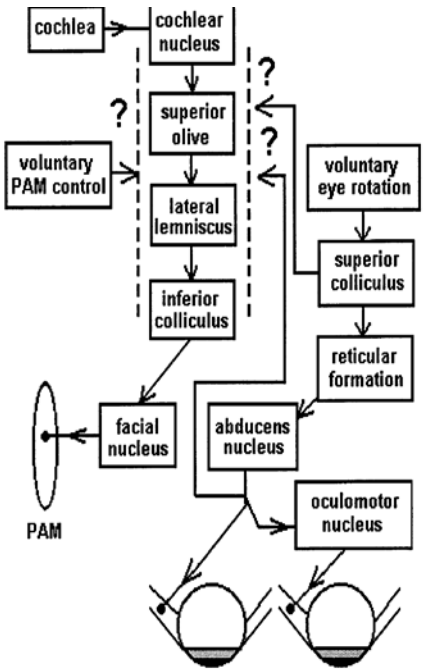

Figure 4: Pictorial representation of Scheme 3 (Patuzzi and O'Beirne, 1999b).

In any case, if all neural control for pinna movements (voluntary, co-activation with the eye, or sound evoked) were relayed via the auditory brainstem, then concomitant changes in PAMR and EMG amplitude would provide no evidence as to the site at which the PAMR response was potentiated along the reflex pathway (Patuzzi & O' Berine, 1999b).

To summarize the findings of Patuzzi and O' Berine (1999b)

- Eye rotation has a powerful influence on the magnitude of the PAMR and that there is a strong relationship between the PAMR and background EMG activity in most subjects, under a variety of conditions, during and after eye rotation. Any method used to increase the EMG also increased the PAMR (if it was present) and any action that increases the PAMR (other than altering the acoustic stimulus) also increased the EMG. First, anything that increases the muscle tone of the PAM appears to increase the PAMR As a result, the effects of eye movement would have been (and are) only apparent in the most relaxed of subjects, because once the PAMR is potentiated by other means, the eye movements are useless.
- As for the underlying neural circuitry, the increases in EMG and PAMR amplitude it was observed with eye rotation are certainly consistent with a potentiation of the reflex at the motor nuclei of the facial nerve, presumably due to depolarization of the membrane potential of these neurones. That is, the EPSPs from the auditory neurones probably add to the EPSPs from the eye-rotation neurones to reach action potential threshold with eye rotation.

Motor units contributing to PAMR:

De Grandis and Santoni (1980), recorded from individual motor units in the Post auricular muscle using sub-dermal needle electrodes and claimed that at least three motor units were responsible for each subject. The important point made in their study was that the PAMR on the skin surface arises because a brief acoustic stimulus has the ability to synchronize the ongoing spontaneous electrical activity of the motor units of the Post Auricular Muscle.

O'Beirne and Pattuzi (1999) reported that the largest PAMR recorded from a subject was about 250 µv pp (peak to peak). In the same subject, the largest single spontaneous spikes had a peak to amplitude of about 80 µv pp. The simplest argument would suggest that at least three (250 µv pp/80 µv pp) motor units gave rise to the PAMR. However, many of the spikes in the spontaneous PAM activity had an amplitude smaller than 80 µv pp (whether due to unit size or distance from the electrode is not clear), indicating that the number of contributing motor units was probably more than three. Certainly, there was no clear quantization in the PAMR amplitude from such a small number of motor units. They also attempted to identify and count the individual motor units and concluded that the number of motor units contributing to the PAMR was clearly more than three. Based on the relatively large size of many of the spikes observed in the spontaneous activity, they suggested that not more than 10 motor units could be contributing.

Recording of PAMR:

The distribution of the PAMR around the post-auricular area has been studied by various researchers (Yoshie & Okudaira, 1969; Picton et al., 1974; Streletz et al., 1977; Buffin, Connell & Stamp, 1977). However, few studies have considered the distribution of the potential across the pinna (Streletz et al., 1977). This is important for clinical application of the PAMR.

O'Beirne and Patuzzi (1999) studied the distribution of the response over the post-auricular area and the dorsal surface of the pinna from two subjects in detail using an array of 12 small electrodes. Averaged responses (n = 900) from these 12 active electrode locations were recorded using a forehead reference electrode and a recording bandwidth of 10 - 200 Hz (with a 50 Hz notch filter). The click stimulus was given at 45 dB SL. The PAMR was found to have the largest amplitude when recorded directly over the body of the PAM (electrode location 'PAM 5' on Figure 5). The response was smaller when recorded further away from the main body of the muscle, consistent with resistive attenuation of the electrical potentials by the skin and tissue. An inverted version of the PAMR waveform was found on the dorsal surface of the pinna.

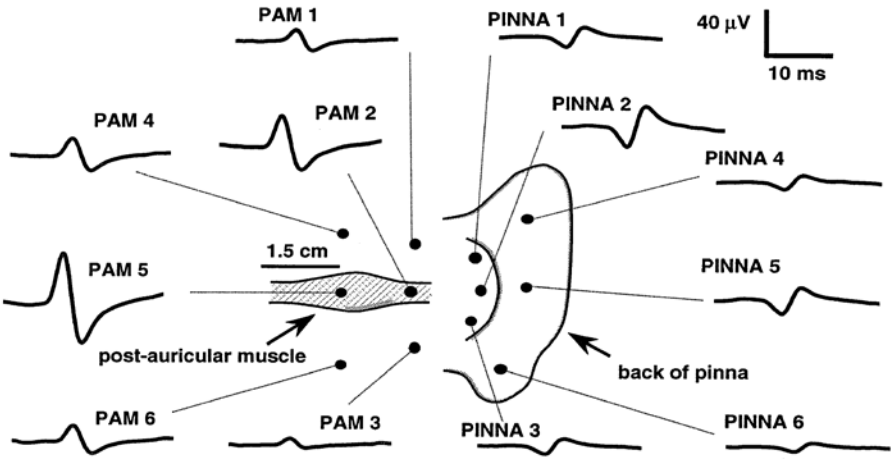

Figure 5: PAMR obtained from 12 active electrode placements (O'Beirne & Patuzzi, 1999)

The largest of these pinna waveforms had amplitude of 48 WV pp, recorded from electrode location 'PINNA 2' (Figure 5). From these results, it was clear that the largest response could be obtained by recording the PAMR with an active electrode directly over the Post Auricular Muscle and with the reference electrode at position PINNA 5 (Figure 5) on the back of the pinna. Figure 6 shows the averaged response recorded from electrode location `PAM 5', superimposed on the inverted response recorded from location `PINNA 2'. This allows a direct comparison of the differences in latency and amplitude between the two waveforms.

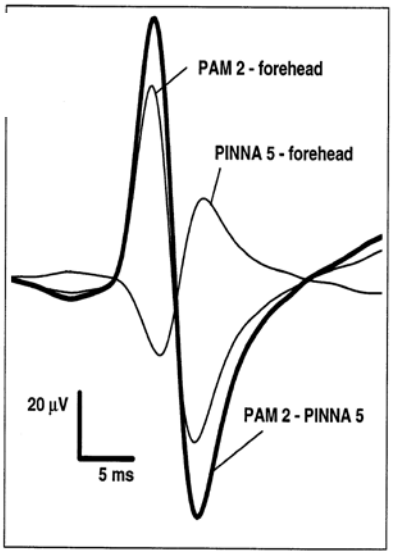

Figure 6: The largest signal (heavy curve) shows PAMR by measuring differentially between the skin directly over the PAM (position PAM 5) and the rear of the pinna (position PINNA 2). Waveforms recorded at these sites relative to the forehead are shown for comparison (light curves).

Katz and Miledi (1965) carried out a series of experiments on the propagation of electrical activity in single muscle fibres. They found that the extracellular potentials were negative-going above the body of a muscle, but were positive-going above a myotendinous junction (the point at the end of the muscle where it attaches to its tendon).

O'Beirne and Patuzzi (1999) and Streletz et al. (1977) observed that PAMR waveform is inverted on the pinna in relative to over the post auricular muscle. This (inversion) may be explained by the occurrence of a compound muscle action potential arising in the post auricular muscle which spreads towards its tendinous insertion on the ear structures (Katz & Miledi, 1965).

Feneis (1994) reported that tendinous aponeurotic end of the post auricular muscle inserts into a cartilaginous ridge (the `ponticulus') on the pinna. Hence, it is likely, that the inverted PAMR waveform on the pinna is the result of electrotonic spread from the tendinous insertion of the post auricular muscle into the pinna (Streletz et al., 1977).

O'Beirne and Patuzzi (1999) stated that in the clinical context, the mechanism causing the inversion of the PAMR on the pinna is not important, but the increased response amplitude possible when recording differentially from post auricular muscle to pinna is very useful. They also listed out other advantages of recording the PAMR with the active electrode over the Post Auricular Muscle and the reference electrode on the dorsal surface of the pinna include

(i) an improved signal to noise ratio,

(ii) reduction of the very low-frequency background electrical noise from the frontalis muscle of the forehead and the jaw and neck,

(iii) a reduction of external electrical interference from nearby equipment,

(iv) more convenient placement of the electrode pairs and

(v) the elimination of blink artifacts.

Spectral analysis of the PAMR:

The distortion of the PAMR due to system bandwidth limits has been discussed by Thornton (1975a), who studied the effect on the PAMR waveform of reducing the low-pass limit of his recording system from 4 kHz to 500, 200 and 100 Hz. Thornton suggested that the previously described PAMR waveforms, consisting only of a negative-going and positive-going peak (e.g. Yoshie & Okudaira, 1969; Douek, Gibson

& Humphries, 1973) were actually distorted versions of the PAMR, caused by recording the response using a system with insufficient bandwidth.

Thornton believed that the PAMR actually consisted of five peaks labelled P1, N1, P2, N2 and P3 and that it was by excessive low-pass filtering that the response was reduced to two peaks. (O'Beirne & Pattuzi, 1999) stated that though Thornton (1975a) succeeded in showing that lowpass filtering the five-peaked signal could produce a two-peaked waveform, the five-peaked waveform was, itself, produced by waveform distortion due to highpass filtering in his own recording system.

O'Beirne and Pattuzi (1999) stated that the true PAMR waveform is a simple bipolar waveform, and the frequency spectrum of the averaged waveform lies mostly between 25 and 200 Hz, broadly centered around 90 Hz. It is not centered at 600 Hz, as described by Thornton (1975a).

The average voltage spectrum of the electrical activity recorded from the surface of the skin above the post auricular muscle (and not the spectrum of the average waveform) can be separated into components that are due to the firing of muscle action potentials from the post auricular muscle and electrical activity from other sources, such as neural activity, distant muscle activity and external electrical interference. The voltage spectrum of the electrical activity attributable to the PAM itself extends from 10 Hz to approximately 550 Hz, with a broad spectral peak centred between 70 and 110 Hz. Since most of the PAMR signal energy lies between 25 and 200 Hz, a system bandwidth from 10 to 300 Hz distorts the signal little (O'Beirne & Pattuzi, 1999).

The effect of maturation on PAMR:

Buffin, Connell and Stamp (1977) recorded the PAMR from 241 subjects and reported that the latency of the PAMR was significantly extended in infants. This longer latency has been attributed to a number of variables, including incomplete myelination and reduced synaptic efficiency in the central nervous system (Eggermont, 1985; Goldstein, Krumholz, Felix, Shannon & Carr, 1979). Both decrease the conduction velocities of the responses along their neural pathways leading to increase in latency.

O'Beirne and Pattuzi (1999) studied PAMR in two children, and found similar results. They also demonstrated that PAMR testing is equally easy in adults and children but the PAMR waveforms are later and last longer in very young babies.

Threshold estimation using PAMR:

Yoshie and Okudaira (1969) found that the click-evoked PAMR thresholds ranged from 0 to 20 dB in normal-hearing adult subjects. Thornton (1975b) estimated threshold through PAMR in individuals with normal hearing and hearing impairment (mostly of cochlear origin). He found that the mean difference between the click-evoked PAMR threshold and the 2 kHz audiometric threshold was 9 dB. But they reported wide variability in the amplitude of PAMR in these subjects. This might be due to the uncontrolled eye movement during the recording.

(O'Beirne & Pattuzi, 1999) optimized the stimulus condition by controlling the eye movement found that thresholds of PAMR were near the audiometric thresholds. They also found that PAMR can also be recorded reliably using the tonebursts. For 500

Hz they obtained PAMR threshold at 40 dB SL, 2 kHz at 20 dB SL and 8 kHz at 40 dB SL respectively.

Purdy et al. (2005) studied the percentage of occurrence of PAMR in normal hearing individuals at various intensity levels and estimated hearing sensitivity in hearing impaired. They used four conditions to record PAMR. They are monaural eyes turned and eyes front condition and binaural eyes turned and eyes front condition. The results were as follows:

- There were a higher proportion of subjects with recordable PAMR for binaural and eyes-turned conditions, and at higher intensity levels.
- All subjects had a recordable PAMR for the binaural eyes-turned condition at stimulus levels. of 80 and 65 dB nHL.
- Across all intensity levels for eyes-turned conditions, almost 80% of subjects had a PAMR.
- For the eyesfront conditions, over 80% of subjects had a PAMR at intensity levels of 80, 65, and 50 dB nHL. At 20 - 35 dBnHL only 45 - 65% of subjects had PAMR.
- Latencies were 1 - 2 ms longer for the softest (20 dB nHL) intensity tested. Latencies were slightly longer with binaural stimulation and with eyes facing front, but these trends were not statistically significant.
- There were significant intensity effects on PAMR latencies and amplitudes, with PAMR getting later and smaller with decreasing intensity.

- Amplitudes reduced by about 67% with the reduction in click intensity from 80 to 20 dB nHL. The amplitude intensity function is steepest and PAMR is clearly larger for the eyes-turned, binaural condition.
- The first two PAMR peaks were significantly larger for binaural compared to monaural listening condition.
- When equivalent monaural and binaural conditions are compared there is a 44% amplitude enhancement with binaural stimulation (range 15 - 70%).
- All PAMR peaks were larger for eyes-turned conditions than for eyes-front conditions
- Three of the twenty subjects with average hearing thresholds (1, 2, and 4 kHz). of 47 -57 dB HL did not have a PAMR with eyes turned at 80 dB nHL, the highest intensity tested.
- For the seventeen subjects with a PAMR, the mean difference between the pure-tone average (PTA) threshold and the PAMR threshold was 5.39 dB (SD 11.90) when subjects had their eyes turned. With eyes to the front, the discrepancy between PAMR thresholds and the PTA was greater (mean 19.38 dB, SD 17.27).
- PAMR thresholds were significantly higher with eyes front compared to eyes turned.
- For the subjects with PAMR present, there were significant correlations between PAMR eye-turn thresholds and the 1, 2, and 4 kHz pure-tone average and 2 kHz and 0.5 kHz audiometric thresholds.

- For the eyes-front condition, there was no correlation between PAMR threshold and pure-tone average, or 2 kHz or 0.5 kHz audiometric thresholds

It can be concluded that there is less information available regarding the use of PAMR to estimate threshold though it has larger amplitude and requires lesser time to record compared to conventional ABR. There is also dearth of information regarding PAMR in clinical population. Hence, the present study is taken up to know the occurrence of PAMR in stimulus optimized condition in individuals with normal hearing and also to see the utility of PAMR as a tool to measure the hearing sensitivity in clinical population.

3. Method

The study was carried out to find the percentage of occurrence of PAMR in individuals with normal hearing. Attempt was also made to see the correlation between the PAMR threshold and behavioral threshold in individuals with hearing impairment.

Subjects

To accomplish the aim three groups of subjects were taken for the study. The groups were as follows:

- Group I: Consisted of 60 ears from 30 subjects (15 males & 15 females) with normal hearing sensitivity. The mean age was 22.4 years with the age range of 18 to 54 years.
- Group II: Consisted of 25 ears from 14 subjects with Sensorineural hearing loss. The mean age was 47.2 years with the age range of 23 to 77 years.
- Group III: Consisted of 20 ears from 10 subjects with bilateral auditory neuropathy. The mean age was 25.2 years and the age range was between 18 to 40 years.

The subjects were taken for the study on the basis of the following criteria:

Group I: *Individuals with normal hearing*

- Bilateral air conduction and bone conduction thresholds were within 15 dB HL in the octave frequencies from 250 Hz to 8000 Hz and 250 Hz to 4000 Hz respectively.

- Speech identification scores (SIS) were greater than or equal to 90%.
- All of them had ‘A’ type tympanogram with presence of acoustic reflexes indicating normal middle ear function.
- Ttransient otoacoustic emissions (TEOAE) were present in all the subjects.
- No abnormality in click evoked auditory brainstem response was observed in them.
- None of them had any history of otological symptoms (ear ache, ear discharge, and tinnitus or hearing loss).
- No history of neurological symptoms or any other general body weakness was reported by them.

Group II: *Individuals with Sensorineural hearing loss*

- Severity of hearing loss ranged from Mild to Profound having either a flat or sloping configuration of air conduction thresholds (PTA was 26.6 dB HL to 105 dB HL).
- The air-bone gap did not exceed 10 dB HL.
- Speech identification scores were proportionate to the severity of the hearing loss.
- All of them had ‘A’ type tympanogram with present, elevated or absent acoustic reflexes.

- No transient otoacoustic emissions could be recorded from any of them, indicating cochlear damage.
- Latencies of click evoked ABR waves were appropriate to the degree of their hearing loss with good wave morphology at higher repetition rate indicating absence of retrocochlear pathology.
- None of them reported to have acute or chronic ear infections (ear pain or ear discharge).
- Middle ear pathology was ruled out by an otologist.
- None of them had any neurological problems or any other general body weakness.

Group III: *Individuals with auditory neuropathy/ dysynchrony*

- Air conduction thresholds ranged from normal hearing to severe degree of sensorineural hearing loss (PTA was 15 dB HL to 85 dB HL).
- The air- bone gap was within 10 dB HL.
- All of them had poor speech identification scores and were disproportionate to their severity of hearing loss.
- All the subjects had 'A' type tympanogram with absent ipsilateral and contralateral reflexes.
- All of them had presence of transient otoacoustic emission(TEOAE).

- Absent ABR or poor ABR wave morphology with prolonged latencies was recorded in all the subjects which were disproportionate to their degree of hearing loss.
- None of them had history of acute or chronic ear infections (ear pain or ear discharge).
- Middle ear pathology was ruled out by an experienced otologist.
- All the clients underwent neurological evaluation and they were ruled out of having other neurological problem and general weakness.
- All the clients were diagnosed as having primary auditory neuropathy by the neurologist.

Instrumentation

The following instruments were used for the study:

a) 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.

b) A calibrated immittance meter (GSI- tympstar) was used to assess the middle ear function.

c) ILO V6 OAE instrument was used to measure the TEOAEs.

d) An evoked potential system [Intelligent Hearing System (USB Jr.)] was used to record the ABR and post auricular muscle response.

Test environment:

All the audiological tests and recording of post auricular muscle response were carried out in a sound treated room. The noise level in the room was as per ANSI (1991; S3.1).

Procedure:

Puretone audiometry:

The behavioral puretone thresholds were obtained at the octave frequencies from 250 Hz to 8 KHz for air conduction and 250 Hz to 4 kHz for bone conduction. The thresholds were tracked using modified Hughson and Westlake method (Carhart & Jerger, 1959).

Speech identification scores:

Speech identification scores (SIS) were calculated in percentage at 40 dB SL from SRT. Speech material developed by Vandana (1998) was used to obtain SIS.

Immittance:

Tympanogram was established by sweeping the pressure from positive to negative, using 226 Hz probetone. Acoustic reflexes were measured at 500 Hz, 1 kHz, 2 kHz and 4 kHz tones. The change of admittance of tympanic membrane by 0.03ml after the onset of the reflex eliciting signal was considered as presence of acoustic reflex.

Transient otoacostic emissions (TEOAEs):

TEOAEs were measured using the default setting in the instrument with 260 sweeps and non linear click trains at 85 dBpeSPL. The TEOAE was considered to be present if the overall amplitude over the noise floor was greater than or equal to 6 dB with the reproducibility of greater than or equal to 50% (Glattke, Pafitis, Cummiskey & Herrer, 1995). The absence of TEOAEs with the presence of hearing loss was considered as an indication of cochlear pathology. The presence of TEOAE in the presence of hearing loss with ABR being absent was considered as having auditory neuropathy/auditory dysynchrony.

ABR recording:

Prior to ABR recording the electrode sites were cleaned using abrasive gel (Nuprep). The silver chloride disc type electrodes were placed on the scalp at electrode placement sites with adequate amount of conductive paste. The inter electrode impedance was maintained less than 2 kohm and intra electrode impedance was within 5 kohm. The electrodes were taped using surgical plaster to prevent any dislodging of electrodes.

The subjects were instructed to sit comfortably, close their eyes and relax on a reclining chair. They were instructed to avoid head and limb movement during testing to avoid artifacts. The ABR was recorded using the following protocol:

Table 1.

Parameters used to record ABR

Stimulus parameter		**Acquisition parameters**	
stimulus	clicks	Mode of stimulation	Monaural
polarity	Alternating	Electrode montage	Fz: +ve A1/A2 : - ve A2/A1: Ground
Number of sweeps	1500	Filter setting	100 – 3000 Hz
Stimulus rate	11.1/sec &90.1/sec	Transducer	Insert ear phone (ER-3A)
Intensity	90 dB nHL	Analysis window	10 ms
		Replicability	Twice
		Gain	1,00,000
		Notch filter	On

The wave I, III and V latency and morphology of ABR was visually inspected to check for interpeak latency and ABR morphology. This was done to rule out absence or presence of retrocochlear pathology along with TEOAE and puretone audiometric results. Based on the audiological test results the subjects were selected and put them

under the respective group as per the criteria mentioned above. Later all of them underwent PAMR testing.

PAMR recording:

The electrode sites (behind the pinna, mastoid and forehead) were cleaned using abrasive gel. The silver chloride disc type electrodes were placed on the electrode placement sites with adequate amount of conduction paste. The inter electrode impedance was maintained less than 2 kohm and intra electrode impedance was within 5 kohm. The electrodes were taped using surgical plaster to prevent any dislodging of electrodes.

The subjects were instructed to sit comfortably on a chair and to turn the eyes towards the ear in which the stimulus was presented. They were also instructed to turn their eyes only during the stimulus presentation. The PAMR was recorded using the protocol recommended by Purdy et al. (2005) as given below:

Table 2.

Parameters used to record PAMR

Stimulus parameters		**Acquisition parameters**	
Stimulus type	Clicks	Transducer	Insert ear phone (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 used to elicit the post auricular muscle response. The PAMR was recorded twice at 80 and 50 dBnHL and at the 20 dBnHL it was recorded thrice for replicability.

For individuals with sensorineural hearing loss and auditory neuropathy:

The PAMR was recorded at 90 dBnHL initially and reduced in 20 dB steps till response wase not observed. The intensity was then increased in 10dB steps till the response was observed. If the response was not present at 90 dBnHL, then the PAMR was recorded at 99 dBnHL. If the PAMR was not observed at 99 dBnHL the testing was terminated. The PAMR was recorded twice at higher intensity and thrice near threshold for replicability. If any adaptation was noticed in PAMR due to repeated recordings, a rest period of 2 minutes was given to the subjects before the next recording. The minimum intensity at which the responses were observed was considered as the PAMR threshold. The waveforms were stored in the computer and retrieved later for analysis.

Waveform analysis:

Recorded waveforms were shown to three qualified audiologist to mark Pi, Ni, Pii PAMR waves separately. If there was an agreement in identifying the peaks among the audiologists then the waveforms were taken for further analysis. The absolute latency and absolute amplitude for each of these peaks were measured.

Statistical analysis:

- Descriptive statistics (mean and standard deviation) for the amplitude and latency of pi, ni, and pii were computed for all the intensity levels in individuals with normal hearing and hearing impairment.
- Mixed ANOVA were used to compare the latency and amplitude within these intensity levels and across gender and ear for normal hearing group.
- Paired t-test was used to see the ear effect for amplitude of the PAMR peaks.
- PAMR threshold was compared with PTA1 and PTA2 of the individuals with sensorineural hearing loss. Correlation analysis was done to see whether PAMR threshold correlate best with PTA1 or PTA2. PTA1 was calculated by finding out the average of the puretone air conduction thresholds of 500 Hz, 1 kHz and 2 kHz. PTA2 was established by averaging puretone air conduction thresholds of 1 kHz, 2 kHz and 4 kHz.
- Wilcoxon signed rank test was used to compare latency and amplitude of pi and ni across intensities in individuals with hearing loss.
- No statistical analysis was done for the data obtained in individuals with auditory neuropathy since the number of individuals for whom the PAMR was observed was less.

4. Results

The aim of the present study was to determine the percentage of the occurrence of PAMR at three intensity levels (20 dBnHL, 50 dBnHL and 80 dBnHL) using click as a stimulus in individuals with normal hearing. The study was also aimed to compare PAMR threshold with behavioral threshold to know whether it can be used to estimate auditory threshold with individuals with hearing loss. The latency, amplitude and threshold values from 30 individuals with normal hearing sensitivity (60 ears), 14 individuals with hearing impaired (25 ears) and 10 individuals with auditory neuropathy (20 ears) were analyzed using statistical package for social sciences (SPSS) software version 16.

The variables present in this study were:

- Independent variable (intensity levels, ear and gender).
- Dependent variable (latency, amplitude and threshold).

The following statistical analyses were done within and across the subject groups:

- Descriptive statistics for all the parameters of PAMR.
- Mixed ANOVA to see the significant main effects and interaction between stimulus intensity levels, ears and gender as an independent variable for control group.
- Bonferroni post hoc analysis was done to see the pair wise (intensity levels) difference when mixed ANOVA showed a significant difference.

- Paired t-test was done to see the significant difference between the ear for the control group.
- Karl pearsons correlation was done to see the correlation between PAMR thresholds and puretone audiometric thresholds (PTA1 and PTA2). Correlation analysis was not done in control group due to lesser variation in PTA1 and PTA2. The correlation analysis was also not done in individuals with auditory neuropathy since the PAMR could not be obtained from most of the individual.
- Wilcoxon signed rank test was done to see the difference between the intensity level and ear effect in the clinical group.
- Mann Whitney test to compare the PAMR parameters across individuals with sensorineural hearing loss and individuals with auditory neuropathy due to small sample size.

The details of the results obtained from the different statistical analysis are discussed below.

Individuals with normal hearing sensitivity:

The major peaks observed in individuals with normal hearing are pi, ni and pii across three intensity levels. Figure 7 shows the PAMR waves obtained in a normal hearing individual at three intensity levels.

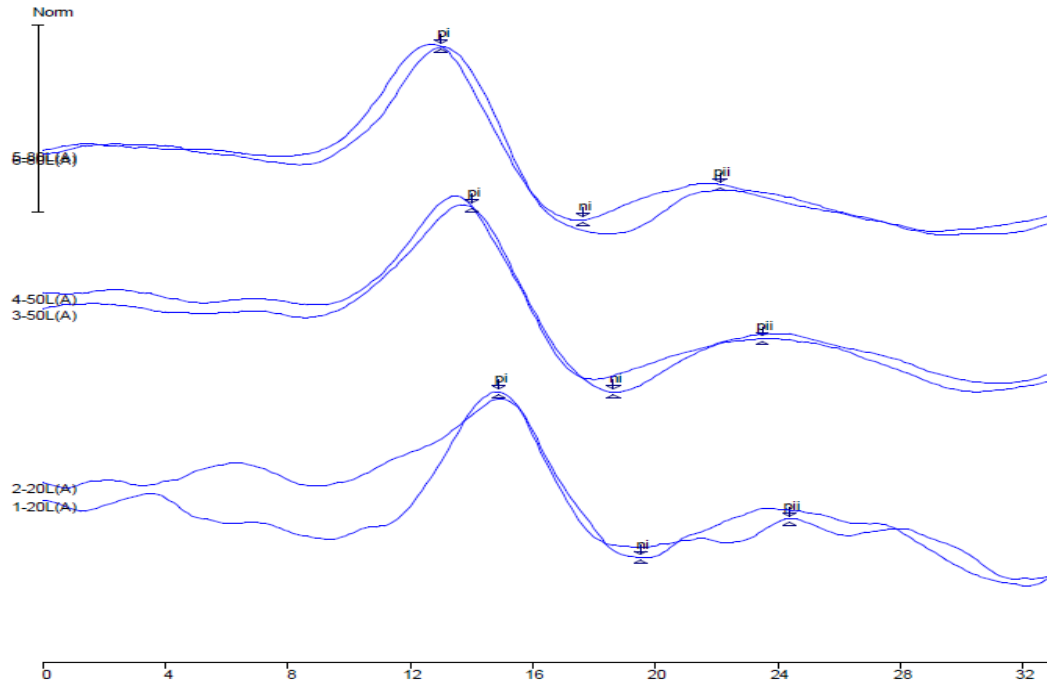

Figure 7: The click evoked PAMR obtained at 80, 50 and 20 dBnHL in a normal hearing individual.

The percentage of occurrence of PAMR was shown in Figure 8. The overall occurrence of pi and ni peaks of PAMR in individuals with normal hearing was about 90% at 20 dBnHL. It can be seen from the Figure 4.2 that the PAMR obtained from left ear and combined response from both ears had a similar pattern of the percentage of occurrence. A reduction in percentage of occurrence in PAMR was observed with the decrease in intensity from 80 dBnHL to 50 dBnHL and to 20 dBnHL. However, PAMR recorded from right ear showed same percentage of occurrence at both 80 dBnHL and 50 dBnHL. Most importantly, 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. However, the pii peak was not commonly observed in individuals with normal hearing.

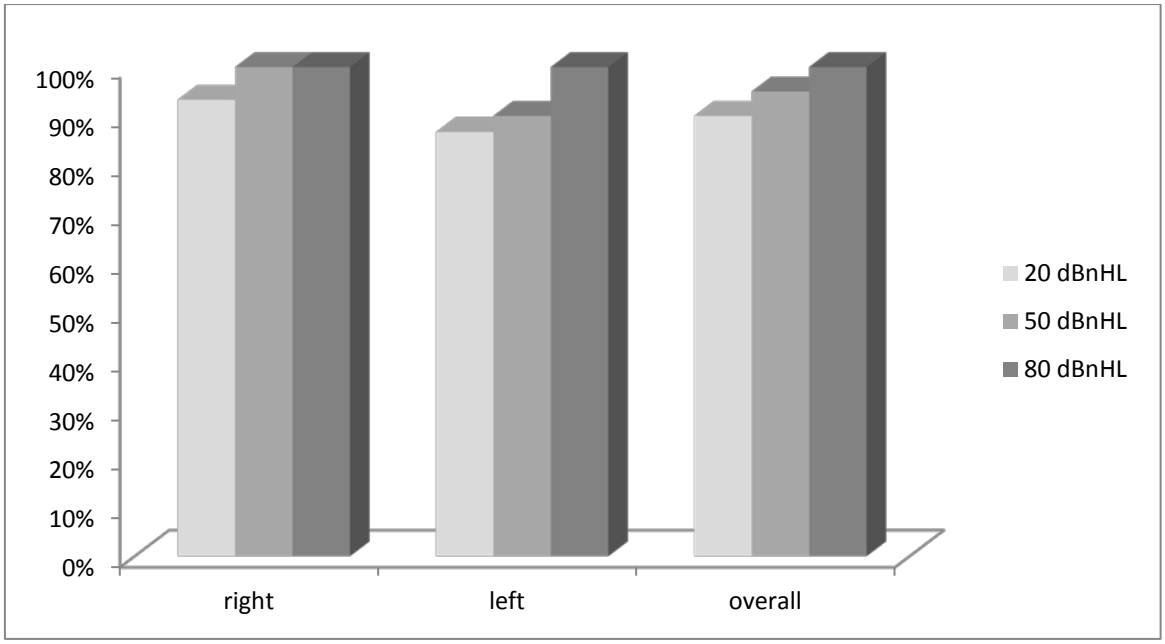

Figure 8: The percentage of PAMR occurrence in right and left ear and also for the both ears together (overall) obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

Latency measures:

The mean, S.D and range for latency of pi and ni were calculated for the three intensity levels in both ears and genders. From the Table 3 it can be noted that the latency increases when the intensity is decreased from 80 to 20 dBnHL for both pi and ni peaks.

Table 3.

Mean, S.D and Range for pi and ni latency obtained for Right and Left ear in males and females with Normal Hearing Sensitivity

Parameter	Gender	Int	Right			Left		
			Mean	S.D	Range	Mean	S.D	Range
Pi (ms)	Males	80	13.08 (N=15)	1.45	12.13- 18.25	13.32 (N=15)	1.28	12.25- 17.17
		50	13.87 (N=15)	1.38	13.20 -18.75	14.13 (N=14)	1.13	12.68 -16.28
		20	15.25 (N=14)	1.86	13.50 -19.13	15.46 (N=13)	1.20	14.18 - 19.08
	Females	80	13.76 (N=15)	1.63	10.95- 16.65	13.74 (N=15)	1.71	11.25 - 15.32
		50	14.85 (N=15)	1.67	11.93- 16.50	14.55 (N=14)	1.25	12.38 - 16.25
		20	16.30 (N=14)	1.74	12.97 -18.90	15.95 (N=13)	1.70	12.97 -17.76
ni (ms)	Males	80	17.70 (N=15)	1.14	15.50- 22.35	17.74 (N=15)	1.79	15.90 - 22.88
		50	18.29 (N=15)	1.13	15.90 - 22.75	18.18 (N=14)	1.17	16.30 - 20.27
		20	19.03 (N=14)	1.91	15.90 - 23.16	19.30 (N=13)	1.87	16.30 -22.13
	Females	80	18.10 (N=15)	1.76	15.53 - 20.02	18.06 (N=15)	1.79	15.97 - 20.28
		50	18.42 (N=15)	2.13	16.15 - 20.23	18.43 (N=14)	1.39	16.05 - 23.40
		20	19.70 (N=14)	2.07	15.68 - 22.65	19.46 (N=13)	1.81	15.60 - 22.13

Mixed ANOVA [(3) intensity X (2) ears X (2) gender] was done for pi and ni latency separately to see the interaction among the variables. The results showed a significant main effect in the latency of pi [F (2, 48) = 103.74, $p < 0.001$)] and ni [F (2, 48) = 35.942, $p < 0.001$)] respectively, when the intensity is decreased from 80 to 20 dBnHL. The results of Mixed ANOVA showed no significant main effect between the ears for pi [*F* (1, 24) = 0.205, $p > 0.05$] and ni [F (1, 24) = 1.01, $p > 0.05$] latencies. The mixed ANOVA results also revealed no interaction between the intensity levels and gender for pi [*F* (2, 48) = 0.964, $p > 0.05$] and ni [F (2, 48) = 0.942, $p > 0.05$)], between ear and intensity for pi [F (2, 48) = 0.232, $p > 0.05$] and ni [F (2, 48) = 0.076, $p > 0.05$] and between ear and gender for pi [*F* (1, 24) = 0.331, $p > 0.05$] and ni [F (1, 24) = 0.046, $p > 0.05$] latencies respectively. There was no interaction seen between ears, genders and intensity levels for pi [*F* (2, 48) = 0.577, $p > 0.01$] and ni [F (2, 48) = 1.557, $p > 0.01$] too.

Since Mixed ANOVA showed significant interaction between the intensity levels, Bonferroni post hoc analysis was carried out to see the pair wise difference for both pi and ni latencies. The results are shown in the Table 4. From the table it can be concluded that there is a significant difference between all the three intensity levels for both pi and ni wave latency.

Table 4.

Bonferroni test results of pi and ni latency across three intensity levels obtained in individuals with normal hearing.

Intensity	50 dBnHL		80 dBnHL	
	pi	ni	pi	ni
20 dBnHL	Significant**	Significant**	Significant**	Significant**
50 dBnHL			Significant**	Significant**

[** $p < 0.001$]

Since the mixed ANOVA did not show any gender effect, data obtained from males and females were combined and mean and S.D were obtained for both pi and ni latencies. The results were shown in Figure 9. It can be observed that there is no difference between right ear and left ear in the overall latency (males and females combined) for pi latency.

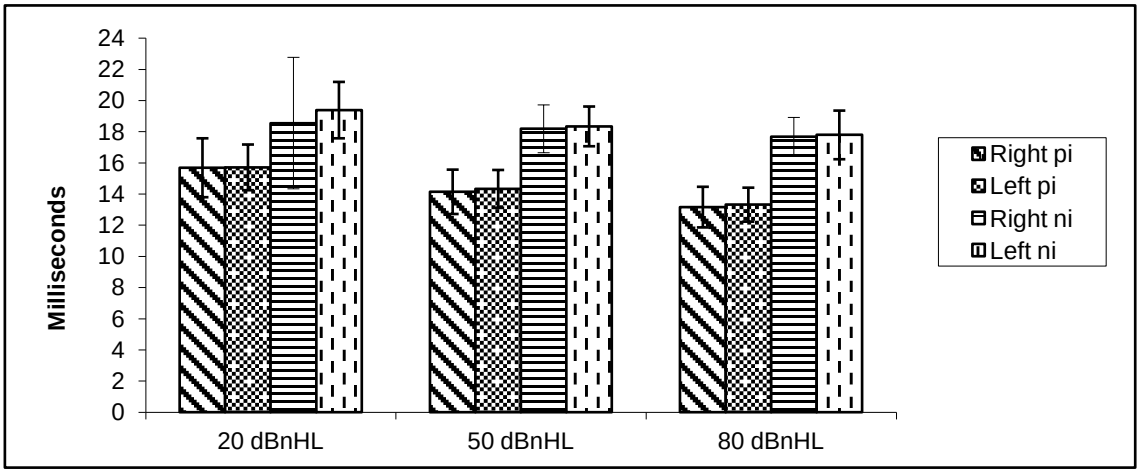

Figure 9: The Mean, SD of overall (Males & females combined) pi and ni latency obtained at 80, 50 and 20 dBnHL from right and left ear in individuals with normal hearing.

Amplitude measures:

The Mean, S.D and Range of pi and ni amplitude for males and females were calculated and shown in the Table 5. From the Table it can be observed that the mean amplitude decreased as the intensity was reduced from 80 to 20 dBnHL for both pi and ni amplitude. It can also be noted that there was a difference in amplitude between the two ears for both pi and ni amplitudes.

Mixed ANOVA results shows that there is a significant main effect in the amplitude of pi [$F(2, 48) = 35.015$, $p < 0.001$)] and ni [$F(2, 48) = 28.03$, $p < 0.001$)] when the intensity was decreased from 80 to 20 dBnHL. The mixed ANOVA results also revealed that there was no ear effect in the pi amplitude [$F(1, 24) = 3.616$, $p > 0.05$]. However, there was a statistical significant ear effect for ni amplitude [$F(1, 24) = 7.307$, $p < 0.05$]. The mixed ANOVA also showed no interaction between the intensity levels and gender for pi [$F(2, 48) = 1.206$, $p > 0.05$] and ni [$F(2, 48) = 0.537$, $p > 0.05$] amplitudes and for ear and intensity for pi [$F(2, 48) = 1.418$, $p > 0.05$] and ni[$F(2,48) = 2.504$, $p > 0.05$]. There was also no statistically significant interaction seen between ear and gender for pi [$F(1, 24) = 0.046$, $p > 0.05$] and ni [$F(2, 48) = 1.407$, $p > 0.05$] amplitudes and also ear, gender and intensity levels for pi [$F(1, 48) = 0.591$, $p > 0.05$] and ni [$F(1, 47) = 0.817$, $p > 0.05$] amplitudes.

Table 5.

Mean, S.D and Range for pi and ni amplitude obtained for Right and Left ear in males and females with Normal Hearing Sensitivity.

Parameter	Gender	dB nHL	Right			Left		
			Mean	S.D	Range	Mean	S.D	Range
Pi (µv)	Males	80	9.22 (N=15)	7.87	2.08 -2.41	6.85 (N=15)	6.09	1.43 - 21.14
		50	5.17 (N=15)	4.50	0.89 -14.95	4.17 (N=13)	3.30	0.92 -12.83
		20	1.94 (N=14)	1.59	0.32 - 6.51	1.41 (N=13)	1.23	0.33 -4.21
	Females	80	6.61 (N= 15)	6.25	1.36 -23.78	5.55 (N= 15)	6.58	1.43 - 23.73
		50	4.44 (N= 15)	4.55	0.49 - 18.09	2.8171 (N= 14)	3.06	0.69 - 10.97
		20	1.95 (N=14)	2.02	0.41 - 8.15	1.62 (N= 13)	1.73	0.40 - 6.25
ni (µv)	Males	80	4.73 (N=15)	4.20	12.27 - 0.45	3.16 (N=15)	3.90	11.26 - 0.76
		50	2.90 (N=15)	2.55	9.54 - 0.23	2.89 (N=13)	2.89	10.87 - 0.25
		20	0.75 (N=14)	0.91	2.54 - 0.45	0.97 (N=13)	1.15	2.84 - 1.39
	Females	80	4.36 (N=15)	3.53	13.54 - 0.77	2.71 (N=15)	2.25	7.71 - 0.23
		50	3.59 (N=15)	3.23	8.86 - 0.29	1.70 (N=14)	2.25	9.26 - 0.23
		20	1.49 (N=14)	1.73	6.01 - 0.58	0.91 (N=13)	1.00	2.80 - 0.15

Bonferroni post hoc analysis was carried out to see the difference across the intensity levels for pi and ni amplitude as the ANOVA revealed significant interaction between the intensity levels. The result obtained is given in the Table 6.

Table 6.

Bonferroni test results of pi and ni amplitude across three intensity levels obtained in individuals with normal hearing.

Intensity	50 dBnHL		80 dBnHL	
	pi	ni	pi	ni
20 dBnHL	Significant**	Significant**	Significant**	Significant**
50 dBnHL			Significant**	Significant**

[**p < 0.001 level]

Paired t-test was carried out to see the ear difference for ni amplitude since mixed ANOVA results showed a significant ear effect. From the table 7 it can be observed that there was a significant difference between the two ears at 50 and 80 dBnHL. But there was no ear difference seen at 20 dBnHL. The paired t- test was not carried out for pi amplitude since mixed ANOVA did not show any ear effect.

Table 7.

t-values along with significance level for ni amplitude between the ears at three intensity levels obtained in individuals with normal hearing.

Intensity	t	df	Significance
20 dBnHL	1.049	25	0.304
50 dBnHL	2.061	26	0.049*
80 dBnHL	2.817	29	0.009**

[** $p < 0.01$, *$p < 0.05$ level]

Since the mixed ANOVA did not show any gender effect, data obtained from males and females were combined and the mean and S.D for pi and ni amplitude was obtained. The results were shown in Figure 10. It can also be observed that there is a difference in amplitude between right ear and left ear in the overall amplitude (males and females combined) for pi. However, the Mixed ANOVA results showed there was no significant difference between the ears. It can also be seen that there was a ear difference for ni amplitude which was statistically significant.

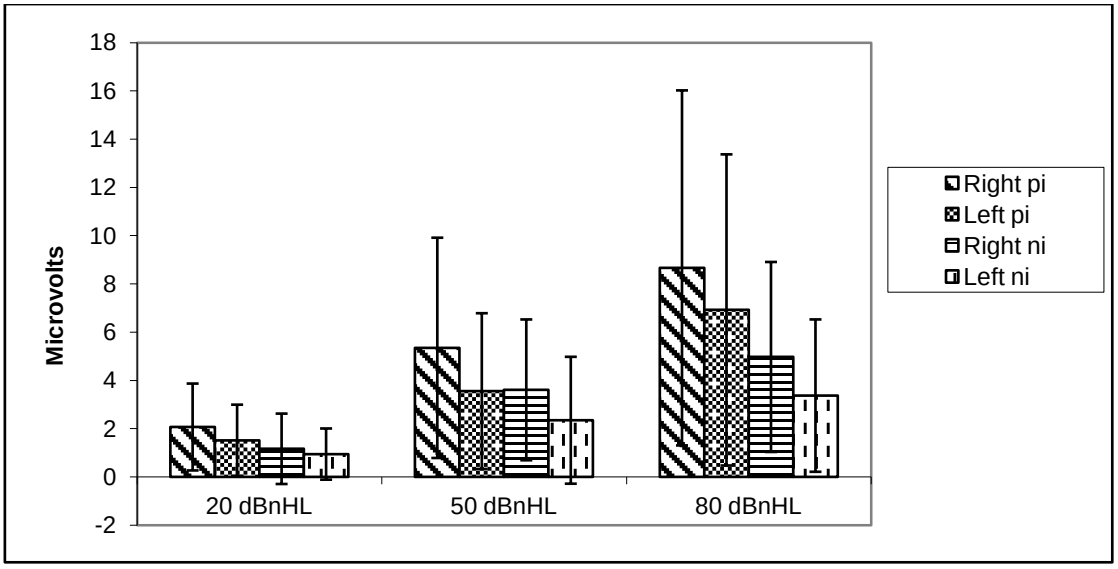

Figure 10: Mean and S.D of Overall (Males & Females combined) pi and ni amplitude for right and left ear obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

pii peak:

The number of individuals for whom the pii peak could be observed was less. The percentage of occurrence of pii was shown in the Figure 11. From the figure it can be noted that the percentage occurrence was around 40% for right ear and 15% for the left ear at 80 dBnHL. It can also be noted that the percentage of occurrence of pii peak even reduced in both ears at 20 dBnHL.

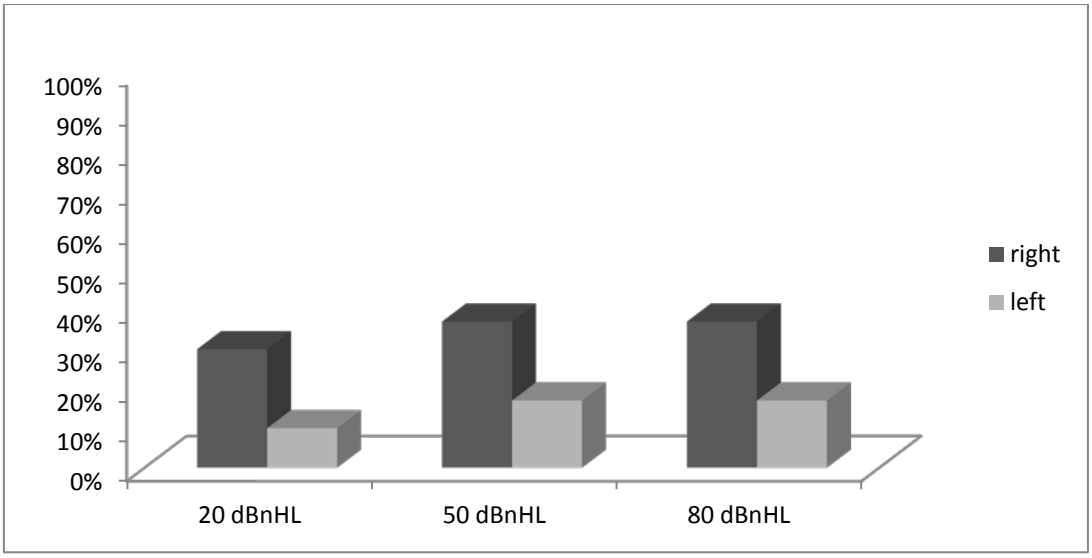

Figure 11: The percentage of occurrence of pii in right and left obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

pii latency:

The Mean, SD and range were calculated for pii latency for right ear and left ear and shown in the Table 8. There is no particular trend seen in pii latency when intensity level is decreased from 80 to 20 dBnHL as compared to pi and ni peak latencies.

Since the number of individual for whom pii peak was observed was less, Wilcoxon signed rank test was carried out to see the intensity effect as well as the ear effect for the latency. The results were given in Table 9. The results indicate that there was a significant difference in latency when the intensity was decreased from 80 to 50 dBnHL in the left ear. However, no significant difference was found in other intensities for both the ears. There was also no significant difference obtained between the ears.

Table 8.

Mean, S.D and Range for pii latency obtained from right and left ear in males and females with normal hearing

Latency	Gender	Int	Right			Left		
			Mean	S.D	Range	Mean	S.D	Range
Pii (ms)	Males	80	22.72 (N= 7)	2.06	20.40 - 26.70	24.50 (N=2)	1.23	23.63 -25.38
		50	23.38 (N= 7)	2.34	20.60 -27.68	24.99 (N=2)	1.15	24.18 -25.81
		20	23.11 (N=5)	3.76	18.50 -25.15	-	-	-
	Females	80	23.19 (N=5)	1.51	22.20 -25.80	22.15 (N=3)	1.16	21.45 -23.50
		50	23.46 (N=5)	0.51	22.88 -24.10	23.03 (N=3)	1.30	21.98 -24.50
		20	25.05 (N=4)	0.20	24.80 -25.30	23.37 (N=3)	0.57	22.75 -23.88

Table 9.

Z-values along with the significant level for pii latency between the intensity levels and ears obtained in individual with normal hearing.

Latency	Z-values	Significance level
Between Intensity for Right Ear		
pii 50 Rt – pii 20 Rt	1.599	0.110
pii 80 Rt – pii 20 Rt	1.364	0.173
pii 80 Rt – pii 50 Rt	1.883	0.060
Between Intensity for Left Ear		
pii 50 Lt – pii 20 Lt	1.069	0.285
pii 80 Lt – pii 20 Lt	1.604	0.109
pii 80 Lt – pii 50 Lt	2.023	0.043*
Between Ears at Same Intensity Levels		
pii 20 Lt – pii 20 Rt	1.342	0.180
pii 50 Lt – pii 50 Rt	1.461	0.144
pii 80 Lt – pii 80 Rt	1.461	0.144

[*$p < 0.05$ level].

pii amplitude:

The mean and SD were calculated for pii amplitude for right ear and left ear and was shown in the Table 10. From the table it can be noted that the amplitude of pii is

much smaller than pi and ni peaks. However, the amplitude reduced with the reduction in the intensity from 80 to 20 dBnHL.

Table 10.

Mean, S.D and Range for pii amplitude obtained from right and left ear in males and females with normal hearing,

Amp	Gender	Int	Right			Left		
			Mean	S.D	Range	Mean	S.D	Range
pii (µv)	Males	80	0.91 (N= 7)	0.43	0.10 - 1.55	1.28 (N=2)	1.59	0.15 - 2.41
		50	0.56 (N=7)	0.42	0.13 - 0.94	0.40 (N=2)	0.59	0.02 - 0.82
		20	0.51 (N=5)	0.79	0.00 - 1.92	-	-	-
	Females	80	0.44 (N=5)	2.01	2.66 - 2.97	0.93 (N=3)	0.86	0.15 -1.87
		50	0.36 (N=5)	2.05	3.82 - 1.40	0.29 (N=3)	1.43	1.92 - 0.78
		20	0.07 (N=4)	1.47	2.11 - 1.37	0.10 (N=3)	0.29	0.08 - 0.45

To see the intensity effect and ear effect in pii amplitude Wilcoxon signed rank test was carried out. The results were shown in Table 11. From the table it can be noted

that there is a significant difference in amplitude between 80 to 50 dBnHL in the left ear and right ear. However, no significant difference was found in other intensities for both the ears. There was also no significant difference between the two ears.

Table 11.

Z-value and significance level for pii amplitude between the intensity levels and for ears obtained in individuals with normal hearing.

Amplitude (µv)	Z-Value	Significance
Between the Intensity for Left Ear		
pii 80 Lf – pii 20 Lt	1.604	0.109
pii 80 Lf – pii 50 Lt	2.023	0.043*
pii 50 Lf - pii 20 Lt	0.000	1.000
Between the Intensity for Right Ear		
pii 80 Rt – pii 20 Rt	1.244	0.214
pii 80 Rt – pii 50 Rt	3.059	0.002**
pii 50 Rt – pii 20 Rt	0.178	0.859
Between Ears at Same Intensity Levels		
pii 20 Lt – pii 20 Rt	0.447	0.655
pii 50 Lt – pii 50 Rt	1.095	0.273
pii 80 Lt – pii 80 Rt	1.095	0.273

[** $p < 0.01$, * $p < 0.05$ level].

Individuals with sensorineural hearing loss:

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. Figure 12 shows PAMR waveform obtained in a individual with mild sensorineural hearing loss.

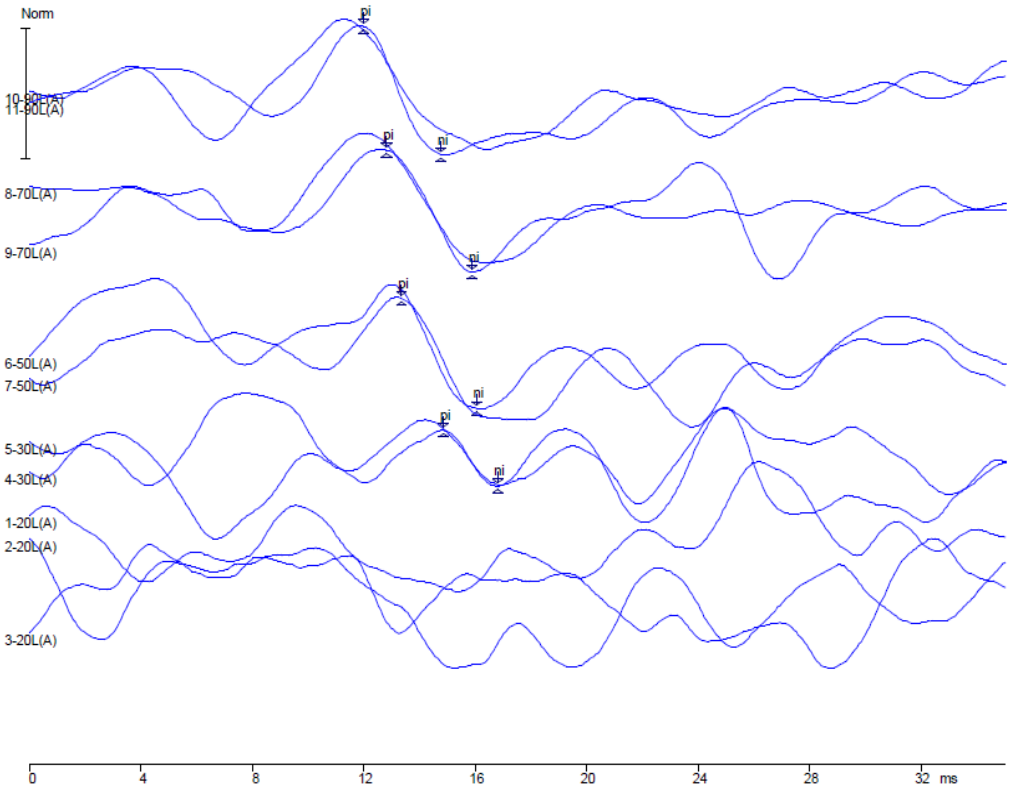

Figure 12: The click evoked PAMR recorded in a mild sensory neural hearing loss individual.

Karl pearsons correlation coeffiecient was done to see whether there is a correlation between the behavioral auditory threshold with the PAMR threshold and the results were shown in the Table 12. From the Table it can be concluded that there is a good correlation between the PTA1 and PTA2 and the threshold obtained by the PAMR in both the ears. The difference between PTA1 and PAMR threshold and PTA2 and PAMR threshold were also computed and shown in the Table 12.

Table 12.

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 * $p < 0.05$]

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 (500 Hz, 1 kHz & 4 kHz).

Latency measures:

Table 13.

Mean, SD and Range of pi and ni latency obtained at 90, 70 and 50 dBnHL from right and left ear in Individuals with Sensorineural Hearing loss.

Latency (ms)	Intensity levels in dBnHL	Right			Left		
		Mean	S.D	Range	Mean	S.D	Range
pi	90 dB	13.51 (N=9)	1.66	11.93 - 17.02	13.48 (N=8)	1.69	11.63 - 16.05
	70 dB	13.92 (N=8)	1.44	12.00 - 16.17	13.69 (N=6)	1.45	12.53 - 16.45
	50 dB	15.33 (N=3)	2.56	12.82 - 17.95	13.20 (N=1)	-	-
ni	90 dB	17.13 (N=9)	1.60	15.86 - 19.95	17.21 (N=8)	2.35	14.22 - 20.15
	70 dB	17.59 (N=8)	1.66	16.20 - 20.25	17.45 (N=6)	2.21	15.53 - 21.55
	50 dB	18.96 (N=3)	2.26	17.10 - 21.48	5.93 (N=1)	6.02	-

The mean, S.D and range of pi and ni peaks for the individuals with sensorineural hearing loss at three intensity levels for both ears were calculated separately and shown in the Table 13. Data obtained from male and female individuals were not compared due to uneven number of data and also normal hearing group did not show any significant difference. However, from the table it can be noted that there is an increase in latency with decrease in intensity.

Wilcoxon signed rank test was carried out to see the intensity effect and ear effect for the pi and ni latencies in individuals with sensorineural hearing loss. Since the data obtained for left ear at 50 dBnHL is only one, the data obtained at 60 dBnHL was taken for the analysis instead of 50 dBnHL. Hence between the ears comparison at 50 dBnHL could not be done. The results were shown in the Table 14. From the table it can be observed that there is a significant difference in the latency in both ears when the intensity is decreased from 90 to 70 dBnHL. However, there was no statistically significant difference in latency for other intensities in both ears. From the table it can also be observed that there is no statistical significance between right and left ear latency for 90 and 70 dBnHL.

Table 14.

Z-value along with significant level for pi and ni latency difference between the intensity levels and ears in individuals with sensorineural hearing loss.

latency	Z-Value	Significance
Between the Intensity for pi latency in Right Ear		
pi 70 Rt – pi 50 Rt	1.604	0.109
pi 90 Rt – pi 50 Rt	1.604	0.109
pi 90 Rt – pi 70 Rt	2.521	0.012*
Between the Intensity for pi latency in Left Ear		
pi 70 Lt – pi 60 Lt	1.604	0.109
pi 90 Lt – pi 60 Lt	1.342	0.180
pi 90 Lt – pi 70 Lt	2.023	0.043*
Between the Intensity for ni latency in Right Ear		
ni 70 Rt – ni 50 Rt	1.604	0.109
ni 90 Rt – ni 50 Rt	1.604	0.109
ni 90 Rt - ni 70 Rt	2.521	0.012*
Between the Intensity for ni latency in Left Ear		
ni 70 Lt – ni 60 Lt	1.604	0.109
ni 90 Lt – ni 60 Lt	1.342	0.180
ni 90 Lt – ni 70 Lt	2.023	0.043*
Between Ears at Same Intensity Levels for pi latency		
pi 70 Lt – pi 70 Rt	0.135	0.893
pi 90 Lt – pi 90 Rt	0.734	0.463
Between Ears at Same Intensity Levels for ni latency		
ni 70 Lt – ni 70 Rt	0.135	0.893
ni 90 Lt – ni 90 Rt	0.000	1.000

[* $p < 0.05$]

Amplitude measures:

The mean, S.D and range of pi and ni amplitude for the individuals with sensorineural hearing loss at three intensity levels were calculated and shown in the Table 15. From the table it can be noted there is a decrease in amplitude with decrease in intensity.

Table 15.

The Mean, SD and Range of pi and ni amplitude at 90, 70 and 50 dBnHL for Individuals With Sensorineural Hearing Loss.

Amp (µv)	Int. levels in dBnHL	Right			left		
		Mean	S.D	Range	Mean	S.D	Range
pi	90 dB	4.01 (N=9)	2.81	1.55 - 8.75	3.74 (N=8)	3.83	0.69 -10.71
	70 dB	3.43 (N= 8)	2.75	0.98 - 8.68	2.92 (N=6)	2.64	0.46 - 6.29
	50 dB	4.05 (N=3)	2.45	1.34 - 6.10	5.84 (N=1)	-	-
ni	90 dB	3.27 (N=9)	2.61	8.76 - 0.72	2.46 (N=8)	2.71	6.97 - 0.62
	70 dB	2.27 (N= 8)	2.37	7.30 - 0.11	1.67 (N=6)	2.09	5.28 - 0.09
	50 dB	2.69 (N=3)	1.67	4.25 - 0.93	3.90 (N=1)	-	-

Wilcoxon signed rank test was carried out to see the amplitude difference in individuals with sensorineural hearing loss. Since the data for left ear at 50 dBnHL is only one, the data obtained at 60 dBnHL was taken for the analysis for the left ear alone. Hence between the ears comparison at 50 dBnHL could not be done. The results were shown in the Table 16. From the table it can be concluded that there is a significant difference was obtained for ni latency between 90 and 70 dBnHL for both ears. Whereas, significant difference in pi latency was observed between 90 and 70 dBnHL only for the right ear. 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.

Table 16.

Wilcoxon signed rank test results of pi and ni amplitude difference across the intensity levels in both ears and across the ears in individuals with sensorineural hearing loss.

Amplitude	Z-value	Significance level
Between the Intensity for pi amplitude in Right Ear		
pi 70 Rt – pi 50 Rt	1.604	0.109
pi 90 Rt – pi 50 Rt	1.604	0.109
pi 90 Rt – pi 70 Rt	2.521	0.012*
Between the Intensity for pi amplitude in Left Ear		
pi 70 Lt – pi 60 Lt	1.604	0.109
pi 90 Lt – pi 60 Lt	1.342	0.180
pi 90 Lt – pi 70 Lt	1.604	0.109
Between the Intensity for ni amplitude in Right Ear		
ni 70 Rt – ni 50 Rt	1.604	0.109
ni 90 Rt – ni 50 Rt	1.604	0.109
ni 90 Rt – ni 70 Rt	2.521	0.012*
Between the Intensity for ni amplitude in Left Ear		
ni 70 Lt – ni 60 Lt	1.069	0.285
ni 90 Lt – ni 60 Lt	1.342	0.180
ni 90 Lt – ni 70 Lt	2.023	0.043*
Between Ears at Same Intensity Levels for pi amplitude		
pi 90 Lt – pi 90 Rt	0.734	0.463
pi 70 Lt – pi 70 Rt	1.826	0.068
Between Ears at Same Intensity Levels for ni amplitude		
ni 70 Lt – ni 70 Rt	1.753	0.080
ni 90 Lt – ni 90 Rt	1.690	0.091

[* $p < 0.05$]

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 PTA1 of 36.6 dBHL and PTA2 of 28.3 dBHL also had PAMR response at 90 dBnHL.

Latency measures:

The Mean, S.D and Range for pi and ni latency for the three ears were calculated and shown in the Table 17. From the table it can be noticed that there is no trend seen in the latency pi and ni with respect to the intensity levels. However, statistical analysis could not be done due to less number of data.

Table 17.

Mean, S.D and Range for latency of pi and ni peaks obtained at 90, 70 50 and 30 dBNHL in individuals with auditory neuropathy.

Lat. (ms)		90 dBnHL	70 dBnHL	50 dBnHL	30 dBnHL
pi	Mean	12.20 (N= 3)	11.93 (N =2)	12.14 (N=2)	13.10 (N=1)
	S.D	0.59	0.98	0.38	-
	Range	11.68 – 12.85	11.23 – 12.63	11.87 -12.41	-
ni	Mean	14.67	14.57	14.59	14.45
	S.D	1.06	1.21	1.30	-
	Range	13.68 -15.79	13.71 – 15.43	13.67- 15.51	-

Amplitude measures:

The Mean, S.D and Range for pi and ni amplitude for the three ears were calculated and shown in the Table 18. From the table it can be noticed that there is no specific pattern seen in the amplitude of pi and ni with respect to the intensity levels. It can be noted that the amplitude is smaller in individuals with auditory neuropathy.

Table 18.

Mean, S.D and Range for amplitude of pi and ni peaks obtained at 90, 70 50 and 30 dBNHL in individuals with auditory neuropathy.

Amp. (µv)		90 dBnHL	70 dBnHL	50 dBnHL	30 dBnHL
pi	Mean	0.25 (N= 3)	0.15 (N=2)	0.26 (N=2)	0.05 (N=1)
	S.D	0.02	0.02	0.98	-
	Range	0.23 – 0.28	0.13 - 0.17	0.19 - 0.33	-
ni	Mean	0.78	0.53	0.43	0.080
	S.D	0.22	0.11	0.02	-
	Range	0.62 – 0.94	0.45 – 0.61	0.41 – 0.45	-

Group comparison:

The data obtained for individuals with normal hearing are at 80 dB, 50 dB and 20 dBnHL. The data was obtained at 90 dBnHL for individual with sensorineural hearing loss and reduced till there was a PAMR response and also the data was obtained at 80 dBnHL for individuals with sensorineural hearing loss. This data is being used for group comparison as normal hearing group data was not obtained at 90 dBnHL. Hence, the comparison was made at 80 dBnHL and 50 dBnHL between control and clinical group. 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 test was carried out to see the difference in latency of pi and ni peaks across the control and clinical group. The results of latency comparison were shown in the Table 19. It can be observed from the table that the latency of both groups did not vary significantly at 50 and 80 dBnHL.

Table 19.

Z-value and significance level for pi and ni latency across intensity levels and ears between control and clinical group:

Latency	Z-Value	Significance level
Between the group at 50 dBnHL		
pi lat 50 Rt	0.626	0.531
ni lat 50 Rt	0.438	0.661
Between the group at 80 dBnHL		
pi lat 80 Rt	0.050	0.960
pi lat 80 Lt	0.395	0.693
ni lat 80 Rt	1.600	0.110
ni Lat 80 Lt	0.663	0.508

Mann Whitney test was carried out to see the difference in amplitude of pi and ni peaks between the control and clinical group. The results of amplitude comparison were shown in the Table 20. It can be observed from the table that the amplitude of both groups did not vary significantly between individuals with normal hearing and individuals with sensorineural hearing loss at both 50 and 80 dBnHL.

Table 20.

Z-value and significance level for pi and ni amplitude across intensity levels and ears between control and clinical group.

Amplitude	Z-Value	Significance level
Between the group at 50 dBnHL		
pi amp 50 Rt	0.125	0.900
ni amp 50 Rt	0.000	1.000
Between the group at 80 dBnHL		
pi amp 80 Rt	1.500	0.134
pi amp 80 Lt	1.325	0.185
ni amp 80 Rt	0.633	0.527
ni amp 80 Lt	0.394	0.694

There was also similar trend seen with respect to the intensity between the two groups for both latency and amplitude. The latency was prolonged and amplitude was reduced when the intensity was reduced in both the groups which were statistically significant.

It can be concluded from the results that for individuals with normal hearing there is about 90% occurrence of PAMR even at 20 dBnHL at optimized recording condition. The latency was significantly prolonged when the intensity was decreased. Similarly the amplitude decreased with decrease in intensity. There was ear difference in ni amplitude alone. No ear difference was found for pi amplitude and pi, ni latency. There was no gender effect for any amplitude and latency measures. The pii peak occurrence in individuals with normal was very less and even lesser in left ear when compared to right ear. The latency was prolonged and amplitude was reduced when the intensity is decreased.

In profound sensorineural hearing loss there was no PAMR responses obtained. However, other degrees of hearing loss elicited the PAMR responses near their behavioral threshold. There was a good correlation between their puretone average thresholds and PAMR thresholds. The amplitude and latency showed a similar trend as that of individual with normal hearing with respect to the intensity level. There was no statistically significant difference in amplitude and latency between individuals with normal hearing and hearing impairment.

The number of individuals with auditory neuropathy who obtained PAMR was less. The amplitude was much smaller than the individuals with normal hearing and sensorineural hearing loss. However, the latency did not show any trend with respect to the intensity. Since the data was very less no statistical analysis could be carried out to see the intensity effects and group comparison.

Discussion

The results obtained from different statistical analysis for each group and across the groups for the parameters of PAMR responses were discussed below.

Individuals with normal hearing sensitivity:

The overall PAMR could be observed in 90% of the individuals with normal hearing at softest intensity levels. The results obtained in this study was consistent with the results obtained by Purdy et al. (2005) where they reported about 80 % of normal hearing individual elicited a PAMR responses at 20 dBnHL when the eyes were turned towards the test ear as compared to eyes front where they elicited PAMR responses only around 45 – 65%. Thus, the results of this study suggest that a good PAMR can be elicited when the stimulus conditions are optimized.

This might be due to the fact that the increase in Electromyography (EMG) and PAMR amplitude observed with eye rotation, presumably due to depolarization of the membrane potential of motor nucleus of facial nerve. That is, 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). They reported a decrease in latency of around 3 - 5 ms with high stimulus sound levels. The results were also consistent with the results obtained by O'Beirne &

Patuzzi (1999). They reported that the latency of the peaks of the PAMR decreased with an increasing stimulus level. Purdy et al. (2005) also reported that there were significant intensity effects on PAMR latencies with PAMR getting later for softer stimulus levels.

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). They also reported that there is a linear growth in amplitude of PAMR peaks when the intensity is increased. Similar findings were found by Purdy et al. (2005). They found that amplitude of PAMR reduced by about 67% with the reduction in click intensity from 80 to 20 dBnHL.

The large variation in the amplitude of pi and ni was seen in the current study. Similar findings were reported by Purdy et al. (2005). Though, they report that the amplitude reduced with decrease in intensity there was a larger amplitude difference seen between the individuals.

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. In other words, it was more likely that the neural circuitry was unable to drive the muscle reliably, rather than that there was only a small muscle (and PAMR) to be driven (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. There was no information available in the literature where they have compared between the two ears absolute amplitudes. (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 mean latency and the mean amplitude obtained for pii in their study was much shorter and larger than the results of the present study.

The amplitude of pii peaks was significantly reduced when the intensity is decreased in both the ears. The possible reason could be same as the reason for pi and ni amplitude difference when the intensity is decreased. The softer intensities showed no significant decrease in pii amplitude in both ears might be due to larger variation in the amplitude at those intensity levels.

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. There was no information available were they compared the latencies and amplitudes of PAMR across gender in individuals with normal hearing sensitivity. As expected, the same origin would be responsible for the generation of PAMR for both the genders.

Individuals with sensorineural hearing loss:

The PAMR was obtained in three out of five individuals with severe sensorineural hearing loss at the highest intensity levels. There was no information in literature where they have reported PAMR in severe sensorineural hearing loss. The possible reason 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). He found that click evoke PAMR is better correlated with the 2 kHz of the audiometric thresholds. The result of this study was also consistent with the results of Purdy et al. (2005). They found that for the subjects with sensorineural hearing loss for whom the PAMR was present, there were significant correlations between PAMR eye-turn thresholds and PTA 2 and 2 kHz and 0.5 kHz audiometric thresholds.

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 PTA2 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 hearing loss individual taken for the study had a hearing loss not varying more than 15 dBHL across the entire frequency range tested. Hence, there was a very high correlation between PTA1 and PTA2 in the present study.

The mean difference between the PAMR threshold and PTA1 and PTA2 were less than 8 dB for both ears. The results were consistent with the findings of Thorton (1975b). He reported that the mean difference between the click evoked PAMR threshold and audiometric 2 kHz threshold was 9 dB. The results were also consistent with the findings of Purdy et al. (2005). They reported that the mean difference between the PTA1 and PAMR threshold was 5.39 dB.

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 latency of pi and ni in individuals with sensorineural hearing loss were significantly prolonged with decrease in intensity levels. The possible reason could be the same which is responsible for the increase in the latency of pi and ni in individuals with normal hearing.

There was no statistically significant prolongation of pi at the lower intensities. This could be due to the fact that the number of subjects who elicited PAMR at lower intensities are lesser compared to the individual who elicited responses at higher intensities. Hence, the variability could have been more for lower intensities than higher intensities.

The amplitude of pi and ni in individuals with sensorineural hearing loss significantly reduced with decrease in intensity in both ears. This could be due to the same mechanism which decreases the amplitude in individuals with normal hearing also decreases the amplitude in individuals with sensorineural hearing loss. The relationship between the amplitude and latency were similar to the relationship seen in individuals with normal hearing sensitivity.

Individuals with auditory neuropathy:

The number of individual with auditory neuropathy for whom the PAMR was observed was meager. There is no information in the literature where they have recorded PAMR in individuals with auditory neuropathy.

The possible reason for absence of PAMR in individuals with auditory neuropathy could be due to the same reason of absence of stapedial reflex. The altered temporal processing and auditory dysynchrony of the auditory nerve could have resulted in absent PAMR responses. The dysynchrony in the auditory nerve would not have increased the EMG which would have resulted in absence of the PAMR responses. The dysynchrony in the auditory nerve could be due to axonal or dentritic loss, demyelination of the auditory nerve or a combination of the two (Starr, Terence, Picton & Kim, 2001). It is clear from the results of the present study that auditory nerve plays a key role in eliciting the PAMR responses. Once the conduction of the auditory stimuli is disrupted in the auditory nerve then PAMR responses would be absent.

The latency of pi and ni obtained in one individual did not show any trend with respect intensity levels. The possible reason could be the dysynchronous firing of the auditory nerve. 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.

The amplitude of pi and ni peaks are much smaller in the individuals with auditory dysynchrony. This could be due to the dysynchronous firing of auditory nerve in individuals with auditory neuropathy resulting in reduced amplitude.

From the findings it is clear that PAMR is not an effective objective tool to measure the hearing sensitivity in individuals with auditory neuropathy. However,

more number of data could have given more information on PAMR responses in these individuals.

Across the group comparison:

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. There was no information in literature were they have compared the latency and amplitude of PAMR in individuals with normal hearing and individuals with hearing impairment.

The possible reason could be that the cochlear damage does not affect the latency or amplitude of PAMR. The cochlear hearing loss 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. Hence high sound level stimuli would cause a greater degree of synchronous firing in the afferent neurones of the cochlea and would therefore evoke a response of greater amplitude in the cells of the nervous system (Hall, 1992) in both individuals with normal hearing and sensory neural hearing loss. So that the latency and amplitude trend with respect to the intensity was similar in the individuals in both the groups.

Hence, the prolongation in latency of pi and ni peaks only dependent on the intensity levels and not on ear or gender. Hence, for usage of PAMR as an objective tool the latency has a greater clinical significance than the amplitude which has wide range of variablity across the individuals.

5. Summary and Conclusion

The Post Auricular Muscle Response is a large sound evoked muscle action potential that can be measured in the skin surface over the muscle behind the ear. It was first identified and reported by Kiang et al. (1963). Douek, Gibson and Humphries (1973) postulated that the PAMR is mediated by the acoustic pathway. The PAMR was initially used for the threshold estimation. Due to the variability in recording the PAMR within and between the subjects the PAMR was later not used clinically for threshold estimation.

Patuzzi and O'Beirne (1999b) found that much of the variability in recording the PAMR might be due to the uncontrolled movement of the eyes. They explained that there is a direct link between the eye rotation and PAMR enhancement. They also showed that PAMR can be recorded reliably by controlling the eye movement till the behavioral threshold.

Purdy et al. (2005) found that PAMR was present in most of the individual with normal hearing up to the softest intensity level. They found that there is good correlation between the audiometric threshold and PAMR threshold in individuals with sensorineural hearing loss. However, these studies have been done on limited number of subjects with normal hearing and also there was limited information on PAMR responses in individuals with sensorineural hearing loss. There is also dearth of information PAMR on individuals with auditory neuropathy too.

Hence the present study was aimed 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 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.

Subjects taken were 60 ears with normal hearing, 25 ears with sensorineural hearing loss and 20 ears with auditory neuropathy. Click was used to elicit the PAMR responses. IHS smart evoked potential USB Jr. was used to record the responses. All the subjects were instructed to turn their eyes towards the ear tested during the recording to enhance the PAMR responses. The protocol used for recording PAMR as given by Purdy et al. (2005). The latency and amplitude of pi, ni and pii was obtained at three intensity levels (80, 50 and 20 dBnHL) for individuals with normal hearing and recording was initiated at 90 dBnHL and obtained till their behavioral threshold in individuals with sensorineural hearing loss and auditory neuropathy. For individuals with hearing impairment the PAMR was obtained till the threshold levels. The latency and amplitude of pi and ni peaks for all the intensity levels were obtained.

The following statistical analyses were done to see the significant differences in the PAMR parameters obtained from these individuals.

Descriptive statistics for all the parameters of PAMR, Mixed ANOVA for the intensity, ear and gender effect, Bonferroni post – hoc test for pairwise comparisons, paired t-test for comparing between the ears, Karl pearson correlation coefficient for

PAMR threshold and behavioral threshold in individuals with sensorineural hearing loss, Wilcoxon signed rank test for intensity and ear effect in individuals with sensorineural hearing loss and Mann Whitney test for comparing between the control and clinical group.

The results from these statistical analyses revealed that

- There is a high percentage of occurrences of PAMR peaks even at lower intensities in individuals with normal hearing. Hence, PAMR was maximally influenced by eye rotation. This could be because of EPSPs from auditory neurones probably adding to the EPSPs from the eye rotation neurones to reach action potential with eye rotation (Patuzzi & O'Beirne, 1999). As the percentage of occurrence is 100% in individuals with normal hearing at high intensity levels, it can be used as a tool to estimate the auditory threshold reliably.
- The latencies were prolonged and amplitude were decreased when the intensity was decreased in individuals with normal hearing and sensorineural hearing loss. Hence, there could be same neurones in the auditory system that could have been involved for both to increase the latency and decrease the amplitude when the intensity is decreased.
- Larger variability seen in the amplitude measures of PAMR. This might be due to sporadic appearance of PAMR in some of the individuals. There was also difference between the ears. Hence, the amplitude measure for PAMR may not be considered for clinical purpose.
- There was no gender difference seen between any of the parameters of PAMR in individuals with normal hearing. This is due to the fact that both the genders

have the same origin for the generation of PAMR. Hence, PAMR can be reliably recorded from both the genders.

- There was a high correlation between the PTA1 and PTA2 and PAMR thresholds in individuals with hearing impairment. This could be due to high frequency domination of clicks and also the pattern of hearing loss. Hence, PAMR can be used as a clinical too for estimating the threshold for individuals with increased muscle tension and difficult to test population.
- The PAMR elicited in ears with auditory neuropathy is less. The absence of PAMR in these individuals could be due to dysychronous firing in the auditory nerve. Hence, PAMR is not a reliable tool to estimate hearing threshold in the individuals with auditory neuropathy.

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 could 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.

Implications of the study:

- Data obtained from the group of individuals with normal hearing can be used as a normative for clinical purpose.

- It can be used to estimate the hearing threshold in individuals who have increased muscle tension and could not relax and also in difficult to test population.
- The study adds information to the literature.

Future research direction:

- The PAMR can be evoked by other stimuli such as tone burst and can estimate frequency specific behavioral threshold.
- The PAMR can be evoked in infants and children to see the difference in them.
- The PAMR can be evoked in individuals with facial nerve palsy who have conductive component for differential diagnosis.

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