

Prescription of Hearing Aids using Auditory Steady State Responses (ASSR)

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Abstract

Hearing aid prescription involves setting the gain at different frequencies and other parameters including compression ratio and compression knee-point. Verification of hearing aid can be done using subjective techniques such as functional gain and objective techniques such insertion gain or electrophysiological tests. In the present study, intensity-amplitude functions were obtained from measures of loudness growth using Auditory Steady State Responses (ASSR). Using this, the gain and compression ratio of the hearing aid were estimated. The relationship between amplitude and intensity of the ASSR was compared in a group of adults having normal hearing with that adults having moderate and moderately-severe sensorineural hearing loss. This was done to propose a method to derive information on hearing aid characteristics from the amplitude- intensity function of the ASSR. This procedure enabled determination of some basic properties of hearing aids, such as average gain, compression ratio. The study also aimed at comparing the gain and compression ratio estimated by ASSR with that predicted by NAL-NL1 and FIG6 prescriptive procedures. From the results of the study it can be inferred that, the gain prescribed by ASSR-PF can also be useful in prescribing hearing aid gain as it was comparable with other prescriptive formulae. Thus, the ASSR serves as an objective tool in verifying the hearing aid prescription process for difficult-to-test population such as infants, young children in whom reliable behavioural responses cannot be obtained.

Key words: gain, compression, intensity-amplitude function, prescriptive procedures.

Introduction

Hearing aid fitting follows three main steps. They are assessing hearing loss, prescribing an aid to compensate for this hearing loss and verifying that this aid provides adequate benefit (Scollie & Seewald, 2001). Each step has its own contribution in hearing aid fitting. Hearing assessment evaluates the hearing threshold, speech identification, maximum comfort levels (MCL) and loudness discomfort level (LDL) at different frequencies. Prescription sets the gain and other parameters including compression ratio and compression knee-point of a selected aid so that the average spectrum of speech sounds is amplified to levels within the range between the unaided thresholds and the loudness discomfort levels of an individual (Cornelisse, Gagné, & Seewald, 1991; Stelmachowicz, Mace, Kopun, & Carney, 1993; Byrne & Dillon, 1986; Cornelisse, Seewald, & Jamieson, 1995). Verification provides some measurement of how well the sounds are heard when the aid is used at its prescribed settings (Stelmachowicz, Kopun, Mace, Lewis, & Nittrouer, 1995).

Fitting hearing aids in adults and older children with hearing loss can be guided by subjective responses to amplified sounds (Picton, et al., 1998). One of the popular subjective measures for selection of a hearing aid is the 'functional gain'. The 'functional gain' a patient

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receives can be determined by obtaining the difference between the unaided and aided thresholds for a particular stimulus (Dillon, 2001). In the case of difficult-to-test population with hearing loss who are unable to provide behavioral responses, objective methods - such as real ear measures and electrophysiological measures - must be relied upon to guide the hearing aid fitting and verification process.

Over the years, data have begun to accumulate which suggest that the ASSR threshold estimates are reasonably accurate in predicting the behavioral thresholds. A number of investigators have reported that ASSR thresholds correlate well with behavioural thresholds. (Cone-Wesson, Dowell, Tomlin, Rance, & Ming, 2002). The amplitude of the ASSR can be used in the estimation of loudness growth function. This information can be used in setting the hearing aid parameters. The validity of using ASSR in hearing aid selection has been evaluated (Vanaja & Manjula, 2004; Damarla & Manjula, 2007) and it has been found that ASSR can be used in setting the gain of the hearing aid.

Apart from setting the gain of the hearing aid, the ASSR can also be used for setting the compression ratio of the hearing aid. The Auditory Steady State Response - Prescription Formula (ASSR-PF) enables determination of some of the basic properties of hearing aids, such as, gain across frequencies and compression characteristics based on the dynamic range of hearing (Zenker, Fernáandez, & Barajas, 2005). In this ASSR-PF procedure, the amplitude-intensity function of the ASSR can be used to derive the information on hearing aid characteristics such as gain and compression ratio. The setting of the gain and compression ratio is done by comparison of the amplitude-intensity function of the ASSR for the clients with hearing impairment with that of those with normal hearing.

Recent studies have proposed that assessment of auditory evoked potentials, and specifically ASSRs, could serve as useful tools in the fitting and verification of the hearing aids (Cone-Wesson, Parker, Swiderski, & Ricakrds, 2002; Picton et al., 1998; Zenker, Fernandez, & Barajas, 2006).

Need for the study

Fitting the hearing aid includes setting the gain and compression characteristics of the hearing aid depending on the hearing threshold and loudness growth of an individual. For this, ASSR can be used as an objective tool. It has been shown that the FG obtained through ASSR and that obtained through sound field audiometer were highly correlated (Vanaja & Manjula, 2004). Further, the FG obtained through ASSR and the IG were also well correlated (Damarla & Manjula, 2007). There are very few studies that have evaluated the usefulness of ASSR in setting the gain as well as compression parameters of the hearing aid (Zenker, Fernandez, & Barajas, 2005). Thus, the present study aims at evaluating the usefulness of the ASSR in setting the gain as well as the compression parameters of the hearing aid.

Objectives

The aims of the present study were

1. To estimate the gain of a hearing aid by the measurement of hearing threshold using ASSR.
2. To estimate the compression ratio of the hearing aid by the measurement of dynamic range, i.e., the difference between the uncomfortable level and the threshold, using ASSR.
3. To compare the gain obtained by ASSR and that estimated by NAL-NL1 and FIG6.
4. To compare the compression ratio obtained by ASSR and that estimated by NAL-NL1 and FIG6.

Method

The following method was adopted to investigate the aims of the study.

Participants

Eighty participants were included in the three groups. Their age ranged from 15 to 55 years, with a mean age of 31.2 years and standard deviation of 3.1 years. The participants were divided into three groups:

- Group I comprised of individuals (N=40) with normal hearing.
- Group II comprised of individuals (N=20) with moderate degree of flat sensorineural (SN) hearing loss in both the ears.
- Group III comprised of individuals (N=20) with moderately severe degree of flat sensorineural (SN) hearing loss in both the ears.

Instruments used

- A calibrated double channel diagnostic audiometer for pure tone audiometry and speech audiometry.
- A calibrated diagnostic immittance meter to confirm the normal middle ear function through tympanometry and acoustic reflex measurement.
- GSI Audera (version 2.6) to record the ASSR through insert earphones.

Procedure

The testing was carried out in a sound treated environment. Pure tone audiometric thresholds were obtained using modified Hughson - Westlake procedure (Carhart & Jerger, 1959). Speech audiometry was performed to establish the speech reception threshold, speech identification scores and uncomfortable level for speech. Immittance evaluation was carried out to ensure normal middle ear functioning. These measurements were carried out on each participant to ensure that the participants met the selection criteria.

The data were collected in two phases.

Phase I: Calculating the hearing aid parameters using NAL-NL1 and FIG6.

Phase II: Calculating the hearing aid parameters using ASSR-PF.

Phase I: Calculating the Hearing Aid Parameters using NAL-NL1 and FIG6

The gain for moderate level sounds (65 dB SPL) was calculated at 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz. For each participant, the gain for moderate level sounds at these four frequencies was computed manually, for both NAL-NL1 and FIG6 using the respective prescriptive formula. The compression ratio was calculated by feeding the audiogram information into the NOAH (3.0) software and simulating a double channel hearing aid with appropriate gain. The default values for the compression ratio at 500 Hz and 2000 Hz as prescribed by NAL-NL1 and FIG6 were noted.

Phase II: Calculating the hearing aid parameters using ASSR-PF

The participant was made to sit comfortably on a reclining chair. He/she was instructed to relax, close the eyes and sleep, if possible while recording the ASSR using the calibrated GSI Audera equipment. The site of electrode placement was prepared with skin preparing paste. Disc type silver coated electrodes were placed with conduction gel. The non-inverting electrode (+) was placed on high forehead (Fz), ground electrode was placed on non-test ear mastoid and the inverting electrode (-) was placed on the test ear mastoid. It was ensured that the impedance of each electrode was less than 5 k Ohms and that the inter-electrode impedance difference was less than 2 k Ohms. The ASSRs were recorded using the insert earphones. ASSR measurements were performed using high modulation frequency of 74, 81, 88, 95 Hz for 500, 1000, 2000 and 4000 Hz respectively, with an amplitude modulation rate of 100% and frequency modulation of 10%.

To find out the dynamic range through ASSR, the testing was initiated at the behavioural threshold level and the intensity was increased in 10 dB steps till the intensity level of UCL-5dB was reached. This was done separately for each of the four test frequencies, i.e., 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz. The amplitude level of the ASSR at each measurement was noted down for the participant.

For participants in Group I, the intensity - amplitude curve was obtained at the four different frequencies, i.e., 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. For participants in Group II and III, the gain at the four frequencies and the compression ratio at 500 and 2000 Hz were determined using the ASSR-PF formula. This procedure namely the Auditory Steady State Response-Prescription Formulae (ASSR-PF) enables determination of some basic parameters of hearing aids, such as dynamic range, frequency response, gain, compression factor, Input-Output function and Maximum Power Output (Zenker, Fernandez, & Barajas, 2005). In the present study, the gain at four frequencies and compression ratio at two frequencies using ASSR-PF were computed for each participant in Group II and Group III.

The ASSR-PF gave information about some critical parameters for fitting hearing aids. First, the hearing dynamic range established from the ASSR hearing threshold and loudness discomfort level; second, the hearing aid characteristics supposed to amplify the entire range of speech into the dynamic range of a particular hearing loss; third, the difference between the hearing loss and the lower limit of the speech dynamic range provided the amount of the gain required by the hearing aid; fourth, the compression factor determined by the degree of hearing loss relative to the long-term average speech spectrum (LTASS) based on the amplitude growth function of the electrophysiological Auditory Steady State Response of the participants.

The dynamic range, gain and compression ratio were obtained from the amplitude projection procedure (APP) as depicted in the Figure 1. The amplitude level function for the group of participants with normal hearing (Group I) was represented by the solid line curve and the amplitude level functions for the group of participants with moderate and moderately severe hearing impairment (Group II & III) were represented by dashed and dotted curves respectively.

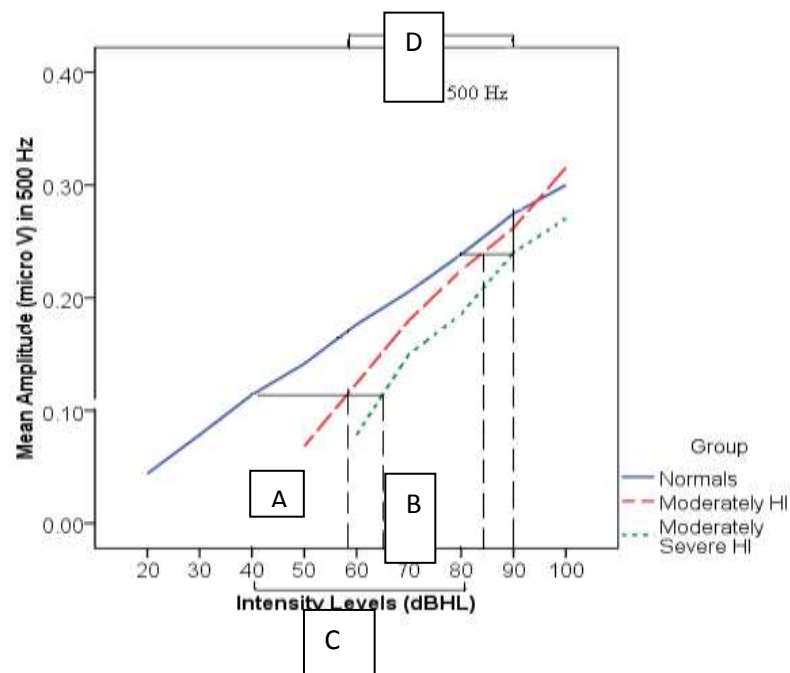


Fig. 1: The amplitude projection procedure (APP) for calculation of gain and compression ratio at 500 Hz.

The dynamic range of speech (40 to 80 dB) was projected upward from the abscissa to the normal amplitude intensity function for each of frequency. Then, the gain requirement is estimated as the difference between the point at which the dotted line (A or B) intersected the X-axis and the lower limit of the input dynamic range (i.e., 40 dB). The compression ratio is given by the ratio of output dynamic range of the participant to the input dynamic range.

From Figure 1, for the group with moderate hearing loss, the gain was calculated as the difference between the hearing loss (59 dB, A) and the lower limit of the LTASS (40 dB), or $59 - 40 = 19$ dB. The compression ratio was calculated by the ratio of the normal speech dynamic range ($80 - 40 = 40$ dB, C) to the ratio of the dynamic range of the participant ($85 - 59 = 24$, D). Thus, the compression ratio was $40/24 = 1.6$.

The gain at all the four frequencies obtained by NAL-NL1 prescriptive rule was compared with the gain at all the four frequencies obtained through Auditory Steady State Response-Prescriptive Formula (ASSR-PF). The compression ratio (CR) prescribed by NAL-NL1 was compared with the values obtained by ASSR-PF for all the participants at 500 Hz and 2000 Hz. The same procedure was repeated for FIG6 also. This was done in order to compare ASSR based hearing aid prescription with that of NAL-NL1 and FIG6 prescription in terms of gain and compression ratio.

Results and Discussion

The data collected were statistically analyzed, using statistical package for social sciences (SPSS). These results are being discussed below.

The target gain prescribed by ASSR-PF, NAL-NL1 and FIG6 were within 6 dB of each other for the moderate hearing loss group (Group II). The results of the present study, for moderate hearing loss (Group II), indicated that there was a significant difference between the gain prescribed by ASSR-PF and NAL-NL1 at 500 Hz. At 1000 Hz and at 2000 Hz, there was a significant difference between ASSR-PF and FIG6. At 4000 Hz, there was no significant difference in the amount of gain prescribed between any of the three prescriptive formulae.

The target gain prescribed by ASSR-PF, NAL-NL1 and FIG6 were within 14.3 dB of each other for the moderately severe hearing loss group (Group III). The results of the present study, in moderately severe hearing loss (Group III), indicated that there was a significant difference between the gain prescribed by ASSR-PF and NAL-NL1 at 500 Hz. At 1000 Hz and 2000 Hz, the results indicated that there was no significant difference between NAL-NL1, FIG6 and ASSR-PF. At 4000 Hz, there was a significant difference between ASSR-PF and FIG6.

In Group II, the results indicated that there was a significant difference between the compression ratio values at 500 Hz and 2000 Hz. In Group III, Bonferroni multiple comparison tests indicated that there was no significant difference between the compression ratio values obtained by ASSR-PF and NAL-NL1 at 500 Hz. In Group III, the results indicated that there was a significant difference between the compression ratios at 2000 Hz prescribed by ASSR-PF, NAL-NL1 and FIG6. The gain and compression ratio for Groups I, II and III are discussed below.

I. Moderate hearing loss (Group I)

A. Gain

The target gain prescribed by ASSR-PF, NAL-NL1 and FIG6 were within 10.9 dB of each other. Zenker, Fernandez, and Barajas (2005) in their study, reported that there was a significant difference between the gain prescribed by ASSR-PF and NAL-RP, POGO, and Berger formulae. The results of the present study indicate that there was a significant difference between the gain prescribed by ASSR-PF and NAL-NL1 at 500 Hz only. ASSR – PF provided more gain than NAL-NL1. Picton (2003) has reported that this can be because the difference between the physiological threshold and behavioural threshold is higher at low frequencies. Here, the ASSR over estimates the threshold at 500 Hz and this will lead to increase in the amount of gain at that frequency. To overcome this, a correction factor can be incorporated in the present ASSR-PF to obtain the better estimation of gain at 500 Hz.

Dillon (2001) reported that the gain prescribed by NAL-NL1 is relatively lower at 500 Hz when compared to the other prescriptive formulae such as DSL i/o, FIG6 and IHAF. As the NAL-NL1 formula tends to maximize the speech intelligibility, the low frequency parts of the speech which are more intense and less important than the high-frequency parts, i.e., relatively little low-frequency gain is required to maximize contribution to the Speech Intelligibility Index (SII) at the low frequencies. As the other procedures tend to normalize the loudness, they do not reduce the gain because they attempt to place speech at each frequency at the level needed to give normal loudness for that frequency.

The gain obtained at 1000 Hz, 2000 Hz and 4000 Hz was not significantly different between ASSR-PF and NAL-NL1, although the gain prescribed by NAL-NL1 was higher than that of ASSR-PF. As the ASSR-PF formula is based on the dynamic range of the LTASS. It gives more emphasis to the speech frequencies. The underlying rationale of NAL-NL1 prescription procedure is to maximize the speech intelligibility, subject to the overall loudness of speech at any level being more than that perceived by a person with normal hearing.

The gain obtained by ASSR-PF and FIG6 was not significantly different at 500 Hz and 4000 Hz although ASSR-PF prescribed higher gain. This may be attributed to the fact that FIG6 procedure prescribes a flat frequency response, for all input levels, for a flat audiogram. In the present study also, the participants had a flat configuration of audiogram.

The gain obtained by ASSR-PF and FIG6 was significantly different at 1000 Hz and 2000 Hz. At these frequencies, ASSP-PF prescribed significantly higher gain than FIG6. This may be because the FIG6 procedure specifies the gain to normalize loudness, whereas, the ASSR-PF prescribes the gain based on the long-term average speech spectrum, (LTASS).

B. Compression ratio

The compression ratio obtained by ASSR-PF was significantly lower than NAL-NL1 and FIG6 at 500 Hz and 2000 Hz. This may be attributed to the fact that ASSR-PF prescription is based on intensity-amplitude function wherein at higher intensities the amplitude of ASSR in individuals with hearing impairment equals that of individuals with normal hearing leading to reduction in the dynamic range and thus the compression ratio.

II. Moderately severe hearing loss (Group III)

A. Gain

As in the group with moderate hearing loss, the results in this group also indicated that there was a significant difference between the gain of ASSR-PF and NAL-NL1 at 500 Hz. ASSR-PF provided significantly higher gain than NAL-NL1. Picton (2003) reported that this can be because of the difference between the physiological threshold and behavioural threshold is higher. Thus, the ASSR over estimates the threshold at 500 Hz. This will lead to increase in the amount of gain at that frequency prescribed by ASSR-PF than that by NAL-NL1. To overcome this, a correction factor can be incorporated in the present ASSR-PF to get a lower better estimation of gain at 500 Hz, as the low frequency components of speech are louder.

The gain obtained at 1000 Hz, 2000 Hz and 4000 Hz was not significantly different between ASSR-PF and NAL-NL1. Although ASSR-PF and NAL-NL1 formulae are based on the dynamic range of the LTASS, the NAL-NL1 prescribed gain was not significantly higher than that of ASSR-PF.

The gain obtained by ASSR-PF and FIG6 was significantly different at 4000 Hz. As FIG6 is based on the rationale that high-frequency components contribute more to speech intelligibility, it provided significantly higher gain than ASSR-PF.

The FIG6 procedure specifies the gain to normalize loudness, and it is based on average loudness data that relates equal-loudness and threshold curves. Whereas, the ASSR-PF prescribes the gain based on the long-term average speech spectrum.

B. Compression ratio

The compression ratio prescribed by ASSR-PF is significantly lower than that by FIG6 and NAL-NL1 at 500 Hz. Byrne, Dillon, Ching, Katsch, and Keidser (2001) have reported that with the increase in degree of hearing loss, the FIG6 prescribes higher compression ratio than the other prescriptive procedures. However, Dillon (2001) reported that with the increase in degree of hearing loss, the compression ratio should be lesser to make the input-output function more linear.

The compression ratio prescribed by ASSR-PF is significantly lower than that by FIG6 and NAL-NL1 at 2000 Hz. This may be because; the NAL-NL1 tends to use less compression than the other procedures such as DSL-i/o, FIG6 and IHAF which differ considerably (Byrne, et al., 2001).

Byrne, et al., (2001) reported that for the present, such prescriptions must be based mainly on logic as there is very limited evidence on which compression thresholds (CTs) and ratios (CRs) are best. It is observed that FIG6 procedure prescribes higher compression ratio than other procedures. The FIG6 procedure prescribes more compression at high frequencies.

However, a high degree of compression could result in unacceptable sound quality. There is little information on which to judge the amount of compression needed to maximize comfort or the amount of compression that can be used before sound quality is perceived as being degraded (Moore, et al., 1998). More information can be obtained if done on subjects to see if the prescribed compression ratios are right or to check the quality of speech with different compression ratios.

Conclusions

Several studies have reported that the auditory steady state responses could be used to estimate the frequency specific auditory sensitivity. These studies have reported that there is a good correlation between behavioural thresholds and the thresholds estimated from ASSR. Electrophysiological tests like ASSR can assist in hearing aid prescription since they can measure frequency specific auditory thresholds. Thus, the present study aimed at investigating the gain and compression ratio obtained through ASSR based prescriptive formula (ASSR-PF) proposed by Zenker, Fernandez, and Barajas, (2005). The study also aimed at comparing it with the gain and compression ratio obtained through NAL-NL1 and FIG6 prescriptive procedures.

1. In Group II with moderate hearing loss, the following observations were noted for the gain prescribed by ASSR-PF, NAL-NL1 and FIG6.
 - There was a significant difference in gain between ASSR-PF and NAL-NL1 at 500 Hz ($p < 0.001$), the mean gain provided by ASSR-PF was 4 dB higher than NAL-NL1.
 - There was no significant difference in gain between ASSR-PF and NAL-NL1 at 1000 Hz, 2000 Hz and 4000 Hz ($p > 0.05$).
 - There was a significant difference in gain between ASSR-PF and FIG6 at 1000 Hz and 2000 Hz ($p < 0.001$). The mean gain provided by ASSR-PF was 4.1 dB, and 3.8 dB higher than FIG6 at 1000 Hz and 2000 Hz respectively.
 - There was no significant difference in gain between ASSR-PF and FIG6 at 500 Hz and 4000 Hz ($p > 0.05$).

2. In Group II with moderate hearing loss, the following findings were observed for the compression ratio prescribed by ASSR-PF, NAL-NL1 and FIG6.
 - There was, a significant difference in the prescription of compression ratio by ASSR-PF, NAL-NL1 and FIG6 ($p < 0.001$) at 500 Hz and 2000 Hz. The mean compression ratio prescribed by FIG6 was 0.6 and 0.1 higher than ASSR-PF and NAL-NL1 respectively.
3. In Group III with moderately severe hearing loss, the following findings for the gain prescribed by ASSR-PF, NAL-NL1 and FIG6 were observed.
 - There was no significant difference in gain between ASSR-PF and NAL-NL1 at 1000 Hz, 2000 Hz and 4000 Hz ($p > 0.05$).
 - There was a significant difference in gain between ASSR-PF and NAL-NL1 at 500 Hz ($p < 0.001$), the mean gain prescribed by ASSR-PF was 3.9 dB higher than NAL-NL1.
 - There was a significant difference in gain between ASSR-PF and FIG6 at 4000 Hz ($p < 0.001$), the mean gain prescribed by FIG6 was 4.5 dB higher than ASSR-PF.
 - There was no significant difference in gain between ASSR-PF and FIG6 at 500 Hz, 1000 Hz and 2000 Hz ($p > 0.05$).
4. In Group III with moderately severe hearing loss, the following findings were noted for the compression ratio prescribed by ASSR-PF, NAL-NL1 and FIG6.
 - There was no significant difference in the prescription of compression ratio by ASSR-PF and NAL-NL1 at 500 Hz, ($p < 0.001$), however, there was significant difference in the prescription of compression ratio by ASSR-PF and FIG6 at 500 Hz ($p > 0.05$), and compression ratio prescribed by FIG6 was 1.1 dB higher than ASSR-PF.
 - There was a significant difference in the prescription of compression ratio by ASSR-PF, NAL-NL1 and FIG6 at 2000 Hz ($p < 0.001$), FIG6 prescribed 1.1 dB and 0.8 higher than ASSR-PF and NAL-NL1 respectively.

From the results of the study it can be inferred that, the gain prescribed by ASSR-PF can also be useful in prescribing hearing aid gain as it was comparable to NAL-NL1, except at 500 Hz. At 500 Hz a correction factor is required for ASSR-PF to be more efficient for hearing aid prescription. Thus, ASSR serves as an objective tool in verifying the hearing aid prescription process for difficult-to-test population such as infants, young children in whom reliable behavioural responses cannot be obtained.

Clinical implications

Use of ASSR, an objective measure, for prescribing gain and compression ratio for individuals with hearing loss will be highly useful. This is especially true for prescribing hearing aid for the difficult-to-test populations.

References

- Byrne, D., & Dillon, H. (1986). The National Acoustic Laboratories' (NAL) new procedure for selecting the gain and frequency response of a hearing aid. *Ear and Hearing, 7*, 257-265.
- Byrne, D., Dillon, H., & Ching, T. (2001). The NAL-NL1 procedure for fitting non-linear hearing aids: characteristics and comparisons with other procedures. *Journal of the American Academy of Audiology, 12*, 37-51.
- Carhart, R. & Jerger, J. (1959). Preferred method for clinical determination of pure-tone thresholds. *Journal of Speech and Hearing Disorders, 24*, 330-345.
- Cone-Wesson, B., Parker, J., Swiderski, N., & Rickards, F. (2002). The auditory steady state response: full term and premature infants. *Journal of the American Academy of Audiology, 13*, 260-269.
- Cornelisse, L. E., Seewald, R. C., & Jamieson, D. G. (1995). The input/output formula: A theoretical approach to the fitting of personal amplification devices. *Journal of the Acoustical Society of America, 97*, 1854-1864.
- Cornelisse, L.E., Gagné, J.P., & Seewald, R.C. (1991). Ear level recording of the long term average spectrum of speech. *Ear and Hearing, 12*, 47-54.
- Damarla, V.K.S., & Manjula, P. (2007). Application of ASSR in the Hearing Aid Selection Process. *The Australian and New Zealand Journal of Audiology, 29* (2), 89-97.
- Dillon, H. (2001), Hearing aids (pp.106-107). Boomerang press, Sydney: Thieme.
- Picton, T. W., Durieux-Smith, A., Champagne, S. C., Whittingham, J., Moran, L. M., Giguère, C., & Beauregard, Y. (1998). Objective evaluation of aided thresholds using auditory steady-state responses. *Journal of the American Academy of Audiology, 9*, 315-331.
- Picton, T.W., John, M.S., Dimitrijevic, A., & Purcell, D. (2003). Human auditory steady state responses. *International Journal of Audiology, 42* (4), 177-219.
- Scollie, S., & Seewald, R. (2001). Hearing aid fitting and verification procedures for children. In J. Katz (ed.), *Handbook of Clinical Audiology* (pp. 687-706). Philadelphia, PA: Lippincott, Williams and Wilkins.
- Stelmachowicz, P. G., Kopun, J., Mace, A., Lewis, D. E., & Nittrouer, S. (1995). The perception of amplified speech by listeners with hearing loss: Acoustic correlates. *Journal of the Acoustical Society of America, 98*, 1388-1399.
- Stelmachowicz, P. G., Mace, A., Kopun, J., & Carney, E. (1993). Long-term and short-term characteristics of speech: Implications for hearing aid selection for young children. *Journal of Speech and Hearing Research, 36*, 609-620.
- Vanaja, C.S., & Manjula, P. (2004). ASSR: An objective tool for hearing aid fitting. Paper presented in 36th National Conference of the Indian Speech & Hearing Association, Mysore.

- Zenker, F., FernándeZ, R., & Barajas, J.J. (2005). The ASSR-PF the auditory steady state response prescription formulae *XIX International Evoked Response Audiometry Study Group Biennial Symposium*, La Havana, 12-16.
- Zenker, F., FernándeZ, R., & Barajas, J.J. (2006). Fitting hearing aids in early childhood based on auditory evoked potentials in steady states. *Acta Otorrinolaringol Esp*, 57 (9), 388-393.

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

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Abstract

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

Introduction

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

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

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

Need for the study

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

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

Aim of the study was to:

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

Method

The subject group was divided into three. Group I consisted of 30 individuals (60 ears) with normal hearing with the age range of 18 to 54 years (Mean - 22.4 years), group II consisted of 14 individuals (28 ears) with sensorineural hearing loss with the age range of 23

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

Subject selection criteria

Group I

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

Group II

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

Group III

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

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

Instrumentation

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

Procedure

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

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

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

Table 1: Parameters used to record PAMR

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

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

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

Results

Individuals with normal hearing:

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

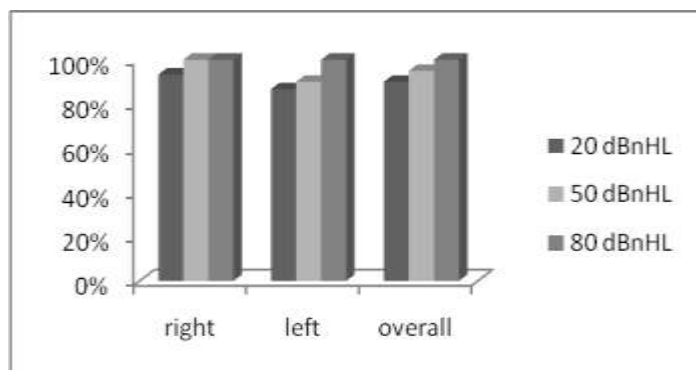


Figure 1: The percentage of PAMR occurrence in right and left ear and also for the both ears together (overall) obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

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

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

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

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

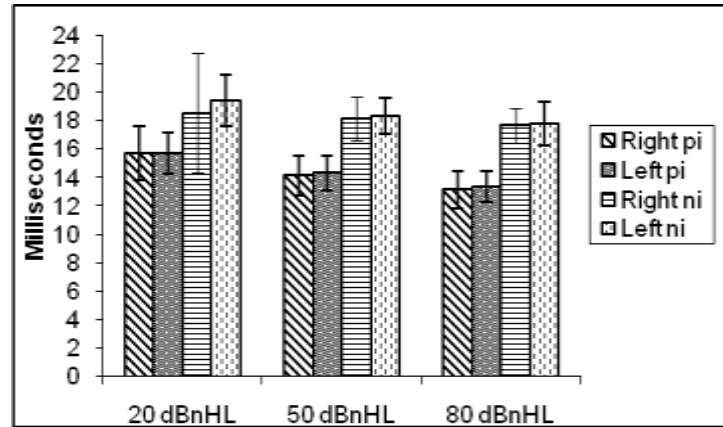


Figure 2: The Mean, SD of overall (Males & females combined) pi and ni latency obtained at 80, 50 and 20 dBnHL from right and left ear in individuals with normal hearing.

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

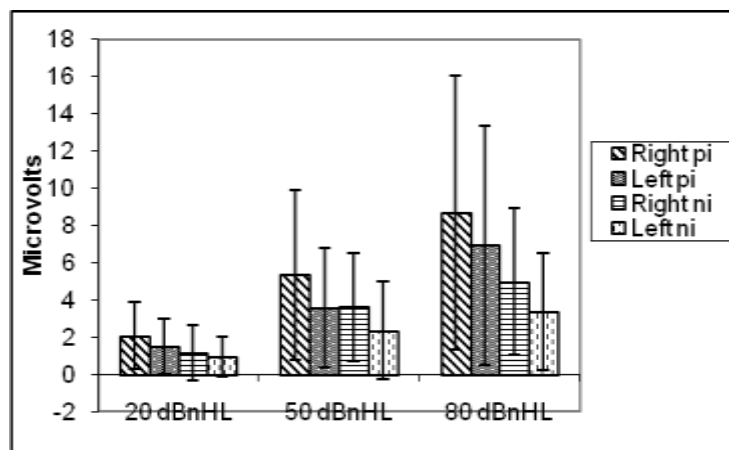


Figure 3: Mean and S.D of Overall (Males & Females combined) pi and ni amplitude for right and left ear obtained at 80, 50 and 20 dBnHL in individuals with normal hearing.

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

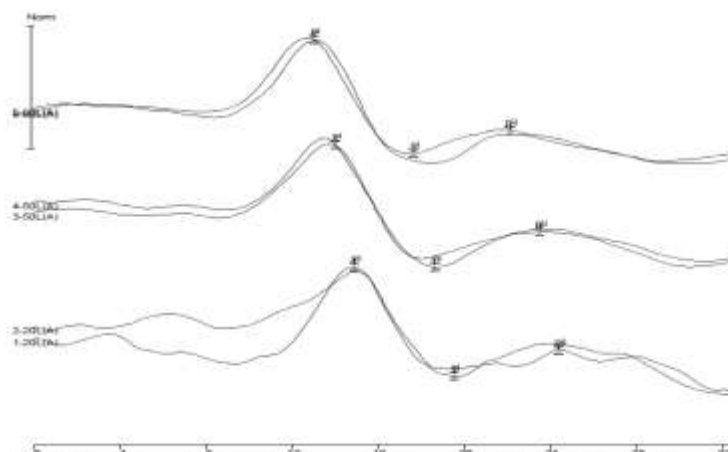


Figure 4: The click evoked PAMR obtained at 80, 50 and 20 dBnHL in a normal hearing individual.

Individuals with sensorineural hearing:

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

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

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

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

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

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

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

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

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

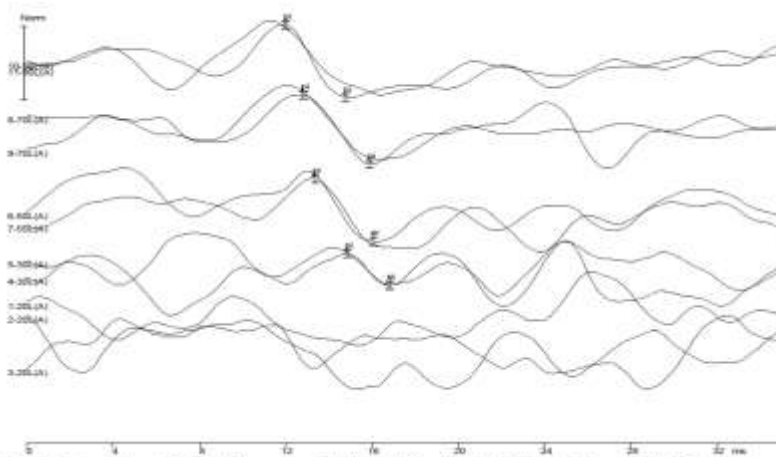


Figure 5: The click evoked PAMR recorded in a mild sensory neural hearing loss individual.

Individuals with auditory neuropathy

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

Group comparisons

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

Discussion

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Conclusion

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

References

- Bickford, R. G., Jacobson, J. L., & Galbraith, R. F. (1963). A new audio motor system in man. *Electroencephalography and Clinical Neurophysiology*, 15, 921-925.
- Bochenek, W., & Bochenek, Z. (1976). Postauricular (12 msec latency) responses to acoustic stimuli in patients with peripheral, facial nerve palsy. *Acta Oto-Laryngologica*, 81, 264-269.
- Cody, D. T., & Bickford, R. G. (1969). Averaged evoked myogenic responses in normal man. *Laryngoscope*, 79, 400 - 416.
- Carhart, R., & Jerger, J. F. (1959). Preferred method for clinical determination of puretone threshold. *Journal of Speech Hearing Disorders*, 24, 330-345.
- Gibson, W. P. R. (1975). *The Crossed Acoustic Response - A Post-Aural Myogenic Response*. Doctoral Thesis, University of London, London.
- Jacobson, J. L., Cody, D. T., Lambert, E. H., & Bickford, R. G. (1964). Physiological properties of the post-auricular responses (sonomotor) in man. *The Physiologist*, 7, 167-167.
- Kumar, A. U., & Jayaram, M. M. (2006). The prevalence and audiological characteristics in individuals with auditory with auditory neuropathy/auditory dysynchrony. *International journal of audiology*, 45(6), 360 -366.
- O'Beirne, G. A., & Patuzzi, R. B. (1999). Basic properties of the sound evoked post-auricular muscle response (PAMR). *Hearing Research*, 138, 115-132.
- Patuzzi, R. B., & O'Beirne, G. A. (1999a). A correlation method for detecting the sound-evoked post-auricular muscle response (PAMR). *Hearing Research*, 138, 147-162.

- Patuzzi, R. B. & O'Beirne, G. A. (1999b). Effects of eye rotation on the sound-evoked post-auricular muscle response (PAMR). *Hearing Research*, 138, 133-146.
- Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials. 1: Evaluation of components. *Electroencephalography and Clinical Neurophysiology*, 36, 179-190.
- Purdy, S. C., Agung, K. B., Hartley, D., Patuzzi, R. B., & O' Beirne, G. A. (2005). The post-auricular muscle response: An objective electrophysiological method for evaluating hearing sensitivity. *International Journal of Audiology*, 44(11), 625-630.
- Thornton, A. R. D. (1975b). The use of post-auricular muscle responses. *Journal of Laryngology and Otology*, 89, 997-1010.
- Vandana (1998). *Speech Identification Test in Kannada*. Unpublished Master's dissertation submitted in University of Mysore, Mysore.
- Yoshie, N., & Okudaira, T. (1969). Myogenic evoked potential responses to clicks in man. *Acta Oto-Laryngologica (Supplement)*, 252, 89-103.