Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2017/447

Original Article

Brain Stem Evoked Response Audiometry Responses in Tinnitus Patients - A Study on Auditory Evaluation in a Tertiary Teaching Hospital of Hyderabad

Urooj Ahmed Choudhary[1], Imtiaz Ahmed Khan[2]

[1]Assistant Professor, Department of Ear, Nose and Throat, Deccan College of Medical Sciences, Hyderabad, India, [2]Associate Professor of Ear, Nose and Throat, Deccan College of Medical Sciences, Hyderabad, India

Abstract

Background: The cause of tinnitus is theorized as due to abnormal spontaneous nerve activity somewhere along the auditory pathway. Majority of the causes of such neuronal activities are unknown. Auditory evaluation is done in all patients with tinnitus to rule out organic brain diseases which may also cause tinnitus. Brain stem evoked response audiometry (BERA) is one such test in the armamentarium of audiological evaluation laboratory.

Aim: The aim of the study is to study the results of BERA performed in all patients undergoing audiological evaluation for tinnitus.

Materials and Methods: Patients were divided into two groups; 78 patients with tinnitus and 34 normal individuals as control group. Total audiological evaluation was done in individuals of both groups. Absolute latencies of Wave I, III, and V, inter peak latencies (IPLs) as well as the interaural latency difference of Wave V (ILD-V) was recorded.

Observations and Results: There was no statistical significant difference between both groups. Few tinnitus patients showed abnormal prolonged absolute latencies, IPLs, and increased ILD-V. There was some asymmetry in results between different study subgroups.

Conclusions: BERA results can variable in tinnitus patients. Few patients have normal results while others showed prolonged absolute or IPLs or increased ILD-V difference.

Key words: Tinnitus, BERA, Audiometry and wave I , III and V

INTRODUCTION

Tinnitus is one of the main aural symptoms with which patients attend the Outpatient Department (OPD) of ear, nose, and throat (ENT) department. After excluding the common causes of tinnitus patients are subjected to a battery of audiological evaluation tests mainly to rule out organic brain lesions. Once the organic brain lesions are ruled out as the cause of tinnitus further establishment of pathogenesis and actual site of neuronal activity remains a dilemma to the practicing ENT surgeons as they are duty bound to explain to the patients the real problem for the tinnitus. Primarily tinnitus is a symptom associated with primary ear disease but may also occur in people with normal hearing.

Access this article online

IJSS
www.ijss-sn.com

Month of Submission : 07-2017
Month of Peer Review : 08-2017
Month of Acceptance : 09-2017
Month of Publishing : 09-2017

Although the initial involvement of auditory end organs is the inducing factor in producing tinnitus, sustained plastic changes, and abnormal neuronal activity within the subcortical and cortical structures of the auditory and non-auditory pathways remain dominant in the causation of tinnitus.[1] Until recently, (1980) tinnitus was believed to be generated in cochlea alone.[2,3] However, later the involvement of not only cochlea but also auditory pathways and cerebral cortex were found to be the source

Corresponding Author: Dr. Imtiaz Ahmed Khan, Department of Ear, Nose and Throat, Deccan College of Medical Sciences, Hyderabad, India. Phone: +91-8978638159. E-mail: iak.knl99@gmail.com

of neuronal activity for tinnitus; with actual abnormality underlying tinnitus evading the human studies.[4] Another theory put forward for the chronicity of tinnitus is lack of habituation in tinnitus patients who have frequent negative associations which reinforce its perception.[5] Brain stem evoked response audiometry (BERA) has been used by many authors in an attempt to understand the pathophysiology of tinnitus.

Such potentials recorded are used to understand the synchronous discharge of fibers in the auditory pathway and identify the presence of abnormal neuronal activity; long latency auditory evoked potentials (P1, N1, P2, and P3) studies revealed abnormal response in tinnitus patients.[2] Recently, an auditory evoked magnetic field study taking a different approach reported significant differences in cortical frequency organization and positron emission tomographic study described abnormally asymmetric activity in the auditory cortices of tinnitus individuals.[6] BERA can be used in evaluating tinnitus patients for a number of reasons, including its objectivity in evaluating the cochlea, and the brainstem auditory pathways. It remains the test of choice when patients present with symptoms that suggests a cochlear or retrocochlear organic lesion.[7] In addition, some BERA findings are thought to be indices of central tinnitus. These indices included: Abnormal morphology of auditory brainstem response (ABR) waveform, fluctuation of Wave III and V and prolonged transmission time.[8] Thus, ABR may contribute to clarify tinnitus origin and this is very important for managing following up such patients.[9]

MATERIALS AND METHODS

Aim of the Study

The aim of the study is to study and evaluate the results of BERA performed in all patients undergoing audiological evaluation for tinnitus.

Study Period

The study period is November 2016 - July 2017.

Institute of Study

Owaisi Hospital and Research centre attached to Deccan College of Medical Sciences, Hyderabad.

112 individuals were included in the present study. Among them, 78 were patients with tinnitus, attending the ENT OPD of Deccan Medical College a tertiary teaching institute of Hyderabad. Another 34 individuals with normal hearing were included as a control group. An institutional ethical committee clearance was obtained before the commencement of the study. All the individuals were given a formal consent letter approved by the ethical committee for filling up and recorded.

Inclusion Criteria

1. 34 individuals with normal hearing were included as a control group.
2. Patients with tinnitus as the main complaint were included.
3. Patients aged between 30 and 70 years were included.
4. Patients with loss of hearing without middle ear pathology were included.

Exclusion Criteria

1. Patients with chronic suppurative otitis media were excluded.
2. Patients with diabetes mellitus, hypertension, cervical disorders, and other neurological disorders were excluded.
3. Patients with thyroid functional disorders were excluded.
4. Patients with anemia and nutritional disorders were excluded.
5. 5. Pregnant women were excluded.
6. Patients with allergic disorders were excluded.
7. Patients with malignancies or on immuno suppressants were excluded.
8. Patients with psychological disorders were excluded.
9. Patients with noise exposure, acoustic trauma, or previous ototoxic medication.
10. Patients with retrocochlear organic lesions requiring further computerized tomography scan evaluation were excluded.

The age and sex of the patients recorded. All patients were submitted to Otologic examination, basic audiologic evaluation (pure tone audiometry, speech audiometry, and impedance audiometry). The subjective nature of the tinnitus matched by the patients was noted in the categories of pure tone, narrow band, or uncertain and tabulated. Frequencies of 3 and 6 kHz were also tested to avoid inclusion of individuals with audiograms that displayed minor dips. Tinnitus matching for intensity and frequency were also done. BERA was done using Smart-EPs of intelligent hearing system. This was done through two-channel recording using four disposable electrodes applied according to the Smart-EP manual specification as the following sites: High frontal Fz (positive electrode), low frontal Fpz (ground electrode). The last two electrodes were placed on the left and right mastoids as negative or reference electrodes depending on the recording side. All electrodes were connected to the pre-amplifier of the Smart-EP equipment. ABR was recorded ipsilaterally in response to click stimuli presented at 90 dBnHL and traced down to threshold in 10 dB steps using alternating polarity

and 19.3 s_1 repetition rate. Stimuli were delivered through ER3A-insertphone. The absolute latencies of Wave I, III, and V, inter peak latencies (IPLs) I0-III, III-V, and I-V as well as the interaural latency difference of Wave V (ILD-V) was calculated. Standard statistical methods were used to analyze the data in the study.

OBSERVATIONS AND RESULTS

The present study included two groups. In Group A, control group 34 individuals without hearing abnormality were included. There were 24 males and 10 females in this group. The patients were aged from 30 to 63 years with a mean age of 53.60 ± 1.2. In Group B, 78 patients with tinnitus were included consisted of 56 males and 22 females. The patients were aged from 30 to 69 years with a mean age of 56.42 ± 1.6. There was a sex age match statistically which was significant with *P* value at 0.498 (*P* value significant at < 0.05) (Table 1).

The duration of tinnitus, laterality, and bilateral involvement was recorded. The subjective nature of the tinnitus matched by the patients was noted in the categories of pure tone, narrow band, or uncertain and tabulated in the patients of Group B. As there were patients with hearing loss included in Group B, subgroups of patients with or without hearing loss developed which was also recorded and tabulated (Table 2).

Absolute and IPLs of Wave I, III, V, and ILD-V were calculated and compared at 90dBHL between both Group A and B as well as subgroups of B (with and without hearing loss). They were considered prolonged if they increased by more than 2 standard deviation from absolute and IPLs in control. BERA values in terms of absolute and IPLs in normal hearing tinnitus patients were not significantly different from control group in general. On the other hand, ILD-V was abnormally prolonged in 40% (24/46) of normal hearing tinnitus patients of Group B and was significantly prolonged when compared with control Group A. All the values were statistically significant as the *P* value was below 0.05 (Table 3).

Table 1: Showing the age, sex incidence, and mean and SD values of the study (*n*=112)

Observation	Group A-34	Group B-78
Male	24	56
Female	10	22
Mean age	53.60	56.42
SD	1.2	1.6
P value	0.498	0.498

SD: Standard deviation

Table 2: Showing the laterality, nature of tinnitus, and duration of tinnitus (*n*=78)

Observation	Male-56	Female-22
Unilateral	26	08
Bilateral	30	14
Duration of tinnitus (years)		
<1	18	06
1-3	21	17
>3	15	09
Patients with hearing loss	22	10
Patients without hearing loss	34	12
Pure tone (%)	36.40	38.24
Narrow band (%)	32.80	36.35
Uncertain (%)	30.80	25.41

DISCUSSION

Tinnitus is a frequent and often causes devastating effect on the social life of the patients. It is a symptom of auditory system disorders and a variety of other non-auditory system pathological conditions. The sensation of tinnitus may be associated with auditory perception defects at various levels of the auditory processing. The only clinical available measure of tinnitus is the psychoacoustical description of pitch and loudness which is based on subjective match between tinnitus and external sounds. Electrophysiological evidences were tried to explain the pathophysiology of tinnitus as many workers thought tinnitus due to impaired brain process [9]. In the present study, BERA was used to evaluate the auditory pathway at the brainstem level. In genereal, there was no significant difference between normal hearing tinnitus patients and normal hearing individuals. This result was similar to the study of Kamal *et al.*[10] and Barnea *et al.*[11] and McKee and Stephans.[12] However, BERA absolute and IPLs were prolonged in some tinnitus patients, and this agreed with Kehrle *et al.*[1] and Rosenhall and Axelsson[13] who reported the presence of BERA values abnormality in patients complaining of tinnitus. They showed two types of abnormalities; (1) prolongation of Wave I accompanied by a prolongation of Waves III and V. These findings were consistent with a lesion in the peripheral auditory system, (2) lengthening of the IPLs reflecting the increased neural conduction time in the brainstem. These two patterns occurred most often in tinnitus patients with normal hearing or slight hearing loss.[14] In the present study, different patterns of BERA values were found in normal hearing tinnitus patients suggesting central auditory pathway affection. The first type was the abnormal prolongation of Wave V absolute latency which occurred in 46.65% of cases suggesting lower brainstem affection. The second type was the increased ILD-V which was found in 53.35% of patients. This is consistent with Kehrle *et al.*[1] who reported prolonged ILD-V in three of their tinnitus patients. Another type of abnormal value was

Table 3: Showing the mean and SD values of Absolute and IPLs of I, III, and V Waves and ILD-V (*n*=112)

Observation	Control Group-34	Group B (normal hearing)-32	Group B (with hearing loss)-46	*P*
Wave I	1.76±0.04	1.7±0.14	1.9±0.12	0.179
Wave II	3.3±0.06	3.6±0.18	3.7±0.17	0.237
Wave III	5.40±0.13	5.8±0.12	5.60±0.18	0.312
IPL-I-III	2.09±0.11	2.2±0.16	2.0±0.13	0.456
IPL-III-V	2.0±0.19	2.3±0.11	2.4±0.10	0.490
IPL-I-V	4.30±0.16	4.90±0.21	4.50±0.11	0.471
ILD-V	0.12±0.003	0.21±0.004	0.22±0.14	0.043

IPLs: Inter peak latencies, SD: Standard deviation, ILD-V: Interaural latency difference of Wave V

of BERA was prolonged Wave III in 34.42% and Wave I in 27.30%. The patterns of IPLs abnormalities included prolonged I-V in 28.80%, III-V in 10.60% and finally 09.48% patients. This could be due to permanent changes in auditory system by electrical activity (tinnitus) which might have changed the central transmission of electrical impulses which in turn might be modifying external stimuli transmission. This might give us an idea about the disturbances caused by tinnitus. BERA abnormalities could be also due to abnormal brainstem activity either at the level of the inferior colliculus (IC), cochlear nuclei, or medial superior olivary complex. For example, IC activity could be normal but it receives abnormal input activity from lower centers (e.g., dorsal cochlear nucleus, medial superior olivary complex) or higher centers (e.g., medial geniculate body).[15] Alternatively, input to the IC may be entirely normal; however, the IC itself has an intrinsic abnormality like membrane alterations that raise the resting potential of IC neurons.[16] A combination of extrinsic and intrinsic abnormalities is also possible. These entire abnormalities can results in tonotopic organization of auditory maps.[17] Tinnitus is caused by abnormal spontaneous hyperactivity in the auditory pathways 23 and that the absence of abnormal BERA parameters in tinnitus patients might be due to the masking effect of the stimulus that masks the abnormal activity in the central pathways.[18-20] Shulman and Goldstein[21] hypothesized the tinnitus dys-synchrony-synchrony theory which considers tinnitus to be an abnormal, conscious, auditory percept occurring as a result of an initial dys-synchrony in pre- or post-synaptic neuronal transmission within the peripheral or central nervous system (cortical or subcortical). This dys-synchronized activity interferes with brain homeostasis and acts as an aberrant auditory stimulus expressed through the auditory system as tinnitus.

CONCLUSIONS

BERA values in normal hearing tinnitus patients are different from patients with tinnitus and hearing loss. Few have normal response while others have prolonged absolute latencies, prolonged IPLs or increased ILD-V suggesting impaired neural firing synchronization and transmission in the auditory pathways in tinnitus patients. At the same time, the pathology causing tinnitus may not be identical in all patients with possible brainstem involvement in some cases.

REFERENCES

1. Kehrle HM, Granjeiro RC, Sampaio AL, Bezerra R, Almeida VF, Oliveira CA. Comparison of auditory brainstem response results in normal-hearing patients with and without tinnitus. Arch Otolaryngol Head Neck Surg 2008;134:647-51.
2. Lee CY, Jaw FS, Pan SL, Lin MY, Young YH. Auditory cortical evoked potentials in tinnitus patients with normal audiological presentation. J Formos Med Assoc 2007;106:979-85.
3. Smits M, Kovacs S, de Ridder D, Peeters RR, van Hecke P, Sunaert S. Lateralization of functional magnetic resonance imaging (fMRI) activation in the auditory pathway of patients with lateralized tinnitus. Neuroradiology 2007;49:669-79.
4. Nodar RH. Tinnitus reclassified: New oil in an old lamp. Otolaryngol Head Neck Surg 1996;114:582-5.
5. Melcher JR, Sigalovsky IS, Guinan JJ Jr, Levine RA. Lateralized tinnitus studied with functional magnetic resonance imaging: Abnormal inferior colliculus activation. J Neurophysiol 2000;83:1058-72.
6. Attias J, Urbach D, Gold S, Shemesh Z. Auditory event related potentials in chronic tinnitus patients with noise induced hearing loss. Hear Res 1993;71:106-13.
7. Shiomi Y, Nagamine T, Fujiki N, Hirano S, Naito Y, Shibasaki H, *et al*. Tinnitus remission by lidocaine demonstrated by auditory-evoked magnetoencephalogram. A preliminary report. Acta Otolaryngol 1997;117:31-4.
8. Dobbie RA. Physiological techniques used in the assessment of the auditory system. In: Keith R, editor. Auditory for the Physician. Baltimore MD: Williams & Wilkins; 1980. p. 14.
9. Shulman A, Seitz MR. Central tinnitus diagnosis and treatment: Observations of simultaneous binaural auditory brain responses with monaural stimulation in the tinnitus patient. Laryngoscope 1981;92:2025-35.
10. Kamal N, Tawfic S, Ahmed S. Evaluation of tinnitus. Egypt J Otolaryngol 1995;12:211-25.
11. Barnea G, Attias J, Gold S, Shahar A. Tinnitus with normal hearing sensitivity: Extended high frequency audiometry and auditory-nerve brainstem-evoked responses. Audiology 1990;29:36-45.
12. McKee GJ, Stephens SD. An investigation of normally hearing subject with tinnitus. Audiology 1992;31:313-7.
13. Rosenhall U, Axelsson A. Auditory brainstem response latencies in patients with tinnitus. Scand Audiol 1995;24:97-100.
14. Møller AR. Pathophysiology of tinnitus. Ann Otol Rhinol Laryngol 1984;93:39-44.
15. Jastreboff PJ. Phantom auditory perception (tinnitus): Mecha-tinnisms of generation and perception. Neurosci Res 1990;8:221-54.
16. Adams JC. Ascending projections to the inferior colliculus. J Comp Neurol 1979;183:519-38.

17. Kaltenbach JA, Godfrey DA, Neumann JB, McCaslin DL, Afman CE, Zhang J. Changes in spontaneous neural activity in the dorsal cochlear nucleus following exposure to intense sound: Relation to threshold shift. Hear Res 1998;124:78-84.
18. Arnold W, Bartenstein P, Oestreicher E, Romer W, Schwaiger M. Focal metabolic activation in the predominant left auditory cortex in patients suffering from tinnitus: A PET study with F deoxyglucose. Otorhinolaryngol 1996;58:195-9.
19. Shiomi Y, Tsuji J, Naito Y, Fujiki N, Yamamoto N. Characteristics of DPOAE audiogram in tinnitus patients. Hear Res 1997;108:83-8.
20. Mirz F, Pedersen B, Ishizu K, Johannsen P, Ovesen T, Stødkilde-Jørgensen H, *et al.* Positron emission tomography of cortical centers of tinnitus. Hear Res 1999;134:133-44.
21. Shulman A, Goldstein B. Tinnitus dyssynchrony-synchrony theory: A translational concept for diagnosis and treatment. Int Tinnitus J 2006;12:101-14.

How to cite this article: Choudhary UA, Khan IA. Brain Stem Evoked Response Audiometry Responses in Tinnitus Patients - A Study on Auditory Evaluation in a Tertiary Teaching Hospital of Hyderabad. Int J Sci Stud 2017;5(6):30-34.

Source of Support: Nil, **Conflict of Interest:** None declared.