

ORIGINAL ARTICLE

EFFECT OF CHRONIC ALCOHOL INTAKE ON AUDITORY SYSTEM WITH CHANGES IN AUDITORY BRAINSTEM EVOKED RESPONSES

Harinder J.Singh¹, Sharanjit Kaur², Amandeep Kaur³, Lily Walia⁴, Anand Sharma⁵

Author's Affiliations: ¹Associate Professor, ⁴Professor & Head; ⁵Professor, Dept. of Physiology; ²Associate Professor, Dept. of Pharmacology, MMMC&H, Solan; ³Medical Officer, NHM, Ludhiana

Correspondence: Dr Harinder J.Singh E-mail: drharinder14@gmail.com

ABSTRACT

Objective: Alcohol affects the auditory threshold, processing of tones and frequency change at different levels of auditory processing system.¹ ABR is the sensitive tool for identifying the various changes in auditory processing unit.

Material and Methods: A total of 52 subjects were divided in group 1 with 26 non-alcoholics males and Group 2 (in lower case) with 26 alcoholic males. Chronic alcoholics who were taking alcohol for more than 8 years (300ml/day) without the history of any neurological and audiological problem and none of them were taking any medication that was oto-toxic were included in our study. Brainstem auditory evoked potential was performed on these subjects and results were analyzed statistically.

Results: There was a significant increase in latency of wave V in alcoholics (5.678 ± 0.2271 ms) as compare to non-alcoholics (5.874 ± 0.2969 ms). ($p = 0.0102$) The mean value of inter peak latency for I-V in group -1 (non-alcoholics) was 3.88 ± 0.26 ms and in group 2 (alcoholics) was 4.19 ± 0.42 ms which showed a statistically significant increase in group 2. ($p = 0.0020$). There was also a statistically significant increase in inter peak latency for III-V in group 2 (2.495 ± 0.389 ms) as compared to group 1 (2.228 ± 0.35 ms). ($p = 0.0119$) but there was no significant result noted for changes in other waveform pattern and inter peak latency I-III.

Key words: Alcoholism, Brainstem auditory evoked potential, latency waves, inter peak latency.

INTRODUCTION

Chronic alcohol intake is one of the most popular abused substance to affect the hearing mechanism. It is known to cause increase in the hearing threshold by altering the central auditory processing particularly at level of summation of auditory signals. Initially this temporary threshold shift in hearing mechanism may become permanent if alcohol taken over a period of time.^{1,2} The mechanism of alcohol toxicity is because of increase in fluidity of neuronal cell membrane and change in neurotransmitters.³ Chronic intake of alcohol affects auditory brainstem responses and causes delay in neurotransmission time, which reflects damage to central auditory pathways in the form hearing loss.⁴

Brainstem auditory evoked potentials (BAEPs) are a common non-invasive objective method to evaluate the integrity of central auditory pathways. It is one of the best measure used for the identification of cochlear and retrocochlear disorders and for threshold testing. Calibrated clicks are delivered to one ear, and

electrical events are recorded in the form of seven waves (I-VII) that appear at certain latent period of time. Any delay or absence of the peaks can locate the brainstem lesions. So this is an sensitive objective tool to measure the function and abnormalities through the entire auditory pathway from cochlea to cortex.^{5,6} There was a study conducted in patients with sensorineural deafness due to alcoholism and the results showed prolongation of wave I in 57% of cases and prolongation of inter peak latency I-V in 43% of cases.⁶ Certain studies showed significantly delay in appearance of wave V and inter peak latencies III-V and I-V.⁷ The current study was designed to evaluate the abnormalities in Auditory brainstem response (ABR) waves in chronic alcoholics to detect early changes in auditory processing unit.

METHODOLOGY

This study was conducted in Department of Physiology, M.M. Medical College and Hospital, Kumarhat-

ti, Solan. Himachal Pradesh. The study protocol was duly approved by institutional ethics committee. Our study was conducted to observe the effects of alcohol on brainstem auditory evoked potentials. 26 non-alcoholics males as controls (Group 1) and 26 chronic alcoholic male subjects (Group 2) as cases were included in our study. All the subjects were of the age group of 25 to 55 years with a mean age of 43.00 ± 10.13 in group I and 46.370 ± 8.367 in group 2 ($p > 0.05$). The subjects were randomly selected from the general population of Solan, Himachal Pradesh and surrounding areas of Solan district.

Inclusion criteria: Alcoholics who have been consuming more than 300 ml of alcohol daily for more than 8 years and abstinence of alcohol for 10 days prior to this study were included in our study. These patients were recruited from college staff only giving this above history. All alcoholics were without the history of any neurological and audiological problem and none of them were taking any medication that was oto-toxic. The subjects were free from symptoms of Wernick's encephalopathy like nytagmus and ataxia.

Exclusion criteria: All the subjects having history of clinical auditory abnormality, subjective symptoms of hearing loss, diabetes mellitus or hypertension were excluded from our study.

All the subjects were interviewed on a proforma which include clinical history for each subject. The subjects were explained about the study and informed consent was taken before their participation. The test performed on these subjects was short latency auditory evoked potential using Neuro- Perfect 2-channel EMG NCV EP PC based machine in Physiology Department.

BAEP Recording Procedure: The subjects were seated in front of machine. High quality EEG electrodes were used. Spots were marked on scalp of subjects and these spots were rubbed with acetone to remove oil. The electrodes were dipped in conductive jelly and pressed on each spot with adhesive tape. The ground electrode was placed on midline point on forehead. The active electrodes were placed on left mastoid and on right mastoid region. Reference electrode was placed on vertex of skull. The impedance of electrodes was kept below 5 ohm. Filter setting was kept at 10 Hz as low filter and at 3000Hz as high filter. 2000 clicks were given at the rate of 11.1 per second with intensity of 60 decibels above normal hearing threshold. A series of 5 waves were recorded during first 10 milliseconds of both right and left ears and its latencies and inter peak latencies were noted. Then we take the average of these 2000 sweeps using computer techniques. The mean wave latency I, II, III, IV, V and inter peak latencies I-III, I-V and III-V of auditory brainstem response (ABR) were measured.^{8,9} The data was analysed statistically between group 1 and group 2.

RESULTS

Our study compared the latencies and interpeak latencies between alcoholics and non-alcoholics. The mean latency V in group 1 (non-alcoholics) was 5.678 ± 0.2271 ms and in group 2 (alcoholics) was 5.874 ± 0.2969 ms. There was a significant increase in latency of wave V in alcoholics as compare to non-alcoholics (Table 1) ($p = 0.0102$).

Table 1: Comparative evaluation of latencies of ABR waves in group 1 (non-alcoholics) and group 2 (alcoholics)

ABR latency waves (in milliseconds)	I (ms) Mean \pm S.D.	II(ms) Mean \pm S.D.	III(ms) Mean \pm S.D.	IV(ms) Mean \pm S.D.	V(ms) Mean \pm S.D.
Group 1	1.795 ± 0.2246	2.730 ± 0.2032	3.450 ± 0.2240	4.716 ± 0.2446	5.678 ± 0.2271
Group 2	1.688 ± 0.2725	2.698 ± 0.2084	3.380 ± 0.2431	4.740 ± 0.2561	5.874 ± 0.2969
p-value	0.1263 *	0.5739 *	0.2854*	0.7328*	0.0102 **

* Non significant, ** significant, ABR= Auditory brainstem response, SD = Standard deviation

Table 2: Comparative evaluation of inter-peak latencies of ABR waves in group 1 (non-alcoholics) and group 2 (alcoholics)

ABR INTERPEAK LATENCY (in milliseconds)	I-III(ms) Mean \pm S.D.	I-V(ms) Mean \pm S.D.	III-V(ms) Mean \pm S.D.
Group 1	1.654 ± 0.3597	3.883 ± 0.2579	2.228 ± 0.3485
Group 2	1.692 ± 0.3514	4.199 ± 0.4225	2.495 ± 0.3892
p-value	0.7011*	0.0020***	0.0119**

* Non significant, ** significant, *** highly significant, ABR= Auditory brainstem response, SD = Standard deviation.

The inter peak latency I-III, I-V and III-V in group 1 (non-alcoholics) was 1.654 ± 0.3597 ms, 3.883 ± 0.2579 ms and 2.228 ± 0.3485 ms and in group 2 (alcoholics) was 1.692 ± 0.3514 ms, 4.199 ± 0.4225 ms and 2.495 ± 0.3892 ms respectively. The inter peak latency I-V showed statistically significant increase in group 2 as compared to group 1 (Table 2) ($p = 0.0020$). Also the inter peak latency III-V showed significant increase in group 2 as compared to group 1 (Table 2) ($p = 0.0119$).

Our results revealed that ABR latency V; inter peak latency I-V and III-V has statistical significant prolongation in group 2 when compared to group 1 (Table 2) ($p < 0.05$) showing that chronic alcoholism affects the central auditory pathway with prolongation of transmission of nerve impulse in chronic alcoholics. Data was analysed using student's unpaired 't' test comparing the means and standard deviation between group 1 and group 2. The differences between the means was considered significant when $p < 0.05$.

DISCUSSION

Chronic alcohol intake was related to hearing loss due to neuronal degeneration as suggested by few researchers like Sandra Beatriz et al, Nordahl et al, Golabek et al.^{10, 11, 12} but some contradicted this relation like studies done by Propelka et al and Itoh et al who found no association between alcohol consumption and hearing loss.^{13, 14} Previous studies also showed that chronic alcoholism was a known cause of hearing loss of the sensorineural type at high frequencies (4000 – 8000 Hz).¹⁵

Auditory threshold (AT) measurement method is a standard behavioural procedure for measuring auditory sensitivity. Verma et al. in their study had shown to affect the auditory threshold in alcohol-dependent patients for higher frequencies.¹ The studies done in past show the effects of alcohol on brain auditory evoked potentials with variable results. Our result mostly correlates with other studies which show delay in latencies of ABR waves II, III, IV and V in alcoholics.¹⁷ In a study conducted in 2002, the authors have shown that 57% patients with hearing loss due to chronic alcoholism have latency prolongation of wave I and 43% patients with hearing loss due to chronic alcoholism shows latency prolongation of wave V, showing that auditory pathways were involved in sensorineural hearing loss.⁶ Our observations also showed the increase in latency V and increase in inter-peak latency I-V and III-V in alcoholic group.

The wave V originates from inferior colliculus.¹³ The statistical comparison in our study showed the increasing trend of wave latency V in alcoholics as compared to non-alcoholics. The possible reason for

this increase in latency V is because of demyelination of auditory pathways.¹⁶ Our study was also in agreement with a previous study showing the prolongation of III-V wave. The prolongation of III-V wave suggests alcoholic cerebellar degeneration.¹⁷ There were also prolonged I-V and I-III inter peak intervals in patients of Wernicks–korsakoff syndrome group in another study.¹⁸ Our study also shows the increase in inter peak latency I-V showing the disturbances in neurological functions in chronic alcoholics.

The results observed in alcoholics could be explained due to degeneration in auditory pathway. It can also be due to peripheral hearing loss due to auditory nerve atrophy which can lead to increase in latencies and increase in inter peak latencies.¹⁹ Another reason for these changes is change in membrane transmission, neuronal loss, death of axons and demyelination of nerves which produces delay in absolute latencies in alcoholics.¹⁶ The chronic alcohol consumption leads to depletion of vitamin B12 stores in liver and greater vitamin B12 intake would be required to preserve cochlear functioning.²⁰ In our study as a limitation of this study, we could not demonstrate the causal association between vitamin B12 and hearing loss.

CONCLUSION

Chronic alcohol consumption can lead to brainstem damage, resulting in hearing degradation depending upon the quantity of alcohol ingested and time duration for this intake. Deviations from normal wave pattern or delay in peak latencies and inter peak latencies can detect various pathologies in auditory processing unit.

REFERENCES

1. Verma RK, Panda NK, Basu D, Raghunathan M. Audi-vestibular dysfunction in alcohol dependence. Are we worried? *Am J Otolaryngol* 2006 Jul-Aug;27(4):225-8.
2. Kähkönen S, Marttinen Rossi E, Yamashita H. Alcohol impairs auditory processing of frequency changes and novel sounds: a combined MEG and EEG study. *Psychopharmacology (Berl)* 2005 Feb;177(4):366-72.
3. Melgaard B. The neurotoxicity of ethanol. *Acta Neurologica Scandinavica* 1983;67(3):131-142.
4. Smith, E.S. & Riechelmann, H. Cumulative life-long alcohol consumption alters auditory brainstem potentials.. *Alcoholism: Clinical & Experimental Research* 2004;28(3):508-515.
5. Weber BA. Patient specific normative values of Auditory brainstem response audiometry. *AJA* 1992 Nov; 24-26.
6. Zhelyazkova Z, Benchev R. Auditory evoked Brainstem response (ABR) of Patients with hearing loss, suffering from chronic alcoholism. *Balkan Journal of Otolaryngology and Neuro-Otology* 2002; 2(1):26-29.

7. Begleiter H, Porjesz B and Chou CL. Auditory brainstem potentials in chronic alcoholics. *Science* 1981;211:1064-6.
8. Rosenhamer H, Lindstrom B, Lundborg T. On the use of click-evoked electric brain stem responses in audiological diagnosis. III. Latencies in cochlear hearing loss. *Scand Audiol* 1981;10: 3-1 1.
9. Jewett D, Romano M, Williston J. Human auditory evoked potentials: possible brain stem components detected on the scalp. *Science* 1970;167:1517-8.
10. Ribeiro SBA, Jacob LCB, Alvarenga KDF, Marques JM, Campelo RM, Tschoeke SN. Auditory assessment of alcoholics in abstinence. *Rev. Bras. Otorrinolaryngol.* 2007;73:1590/S003 – 72992007000400004
11. Legatt AD, Arezzo IC, Vaughan HG Jr. The anatomic and physiologic bases of brainstem auditory evoked potentials. *Neurol Clin* 1988;6:681-704.
12. Golabek W, Niedzielska G. Audiological investigation of chronic alcoholics. *Clinl Otolaryngol* 1984;9:257 -61.
13. Popelka MM, Cruickshanks KJ, Wiley TL, Tweed TS, Klein BE, Klein R, Nondahl DM. Moderate alcohol consumption and hearing loss: a protective effect. *J Am Geriatr Soc* 2000;48:1273 -78
14. Itoh A, Nakashima T, Arai H, Wakai K, Tamakoshi A, Kawamura T et al. Smoking and drinking habits as risk factors for hearing loss in the elderly; epidemiological study of subjects undergoing routine health checks in Aichi, Japan. *Public Health* 2001;115(3):192 – 6.
15. Pearson P, Dawe LA, Timnay B. Frequency selective effects of alcohol on auditory detection and frequencydiscrimination thresholds. *Alcohol and alcoholism.*1999;34(5):741-749.
16. F Diaz, F Cadaveirs and C Grau. Short and middle latency auditory evoked potentials in abstinent chronic alcoholics: preliminary findings. *Electroencephalography and clinical Neurophysiology* 1990;77:145-50.
17. Chu N, Squires K, Starr A. Auditory brainstem response in chronic alcoholics. *Electroencephalogr Clin Neurophysiol* 1982;54:418-25.
18. Chan YW, Mcleod JG, Tuck RR, and Feary PA. Brainstem auditory evoked response in chronic alcoholics. *Journal of Neurology, neurosurgery and Psychiatry* 1985; 48:1107-12.
19. Matas CG, Filha VA, Okada MM and Resque JR, Auditory evoked potentials in individuals over 50 years. *Pro Fono R Atual Cient* 2006 Sep-Dec;18(3).
20. Halsted CH, Villanueva JA, Devlin AM, Chandler CJ. Metabolic interactions of alcohol and folate. *J Nutr* 2002; 32:2367S–2372S.