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A Comparitive Study of Brainstem Auditory Evoked Response In Chronic Alcoholics And Non Alcoholics

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Abstract: *Alcohol is the most commonly used abuse drug in the world. There are at least 208 million people with alcoholism around the world. Long term Alcohol consumption mainly affects all body systems mainly nervous system. In nervous system it affects auditory pathways due to its toxic effects on auditory nerves resulting in various auditory disturbances.*
Aims & objectives*: To study and compare Brainstem evoked auditory response in both Right (Rt) and Left(Lt) ear of chronic alcoholics and non alcoholic controls of Latencies (ms) Wave I ,Wave III Wave V and Interpeak latency (ms) I-III , I-V.*
Materials & Methods*: The study was carried out in the Department of Physiology. It was a case control study which included 120 male subjects in the age group of 25-50 years selected randomly from population. It includes 60 chronic alcoholic men consuming alcohol more than 21 units/week. Control group includes 60 healthy men of same age, BMI and not consuming alcohol.*
Results: *The mean values of latency in wave I in non alcoholic controls and alcoholics was statistically insignificant and there was statistically very highly significant increase in mean latencies of waves III and V in chronic alcoholics as compared to non alcoholics. Mean Interpeak latencies duration of wave I-III and wave I-V in chronic alcoholics was statistically very highly significantly increased as compared to non alcoholic controls.*
Conclusion: *Long term alcohol consumption affects auditory pathways and delayed auditory transmission time.*
Keywords*: Alcoholics, Brainstem Auditory Evoked Response(BERA), Interpeak latency(ms), Latency(ms),*

I. Introduction

Alcohol (ethanol) is a substance that impacts the social, psychological, health, and economical spheres of our existence. Alcoholism is characterized by significant physiological, psychological and social dysfunctions associated with persistent and excessive use of alcohol. The evoked potential (EP) techniques provides unique and sensitive indices of brain function, yielding data on the level of sensory, perceptual, and cognitive processing. An EP is obtained by recording the time-locked brain electrical activity following the delivery of a discrete stimulus in any sensory modality. Signal averaging techniques make it possible to extract the time-locked neuro electric signal from the background random 'noise.' These time-locked signals exactly represent activity at neural generators from the peripheral organ to higher integrative centers of the brain [(1)]. Brainstem auditory evoked responses (BAEPs) are the potential recorded from the vertex and ear in response to brief auditory stimulus to assess the conduction through auditory pathway up to mid brain; it gives idea about the severity of hearing deficit and functions of middle portion of brainstem. More recently evoked related potentials have been used to assess the functional integrity of the brains of alcoholic patients. Deviations from standard BAEPs latencies and amplitudes generally reflect various diseases affecting the auditory nerve and central auditory pathways [(2)]. BAEPS are commonly used for non invasive, objective clinical diagnosis of disease of inner ear, cerebello pontine angle and central auditory pathway. Several studies have demonstrated that a single dose of alcohol causes significant increase in auditory brainstem Transmission Time in rats, cats, and man.However, functional brainstem deficits have not been reported in alcoholic patients.[(3)] So the present study was carried out to evaluate the long term effects of alcohol on the auditory pathway with brainstem auditory evoked response, in chronic alcoholics and non alcoholic controls. In view of possibility of advocating these neurophysiological tests to detect subclinical central and peripheral neuropathy at an early stage of disease in chronic alcoholics.

II. Aims and Objectives

To study and compare Brainstem evoked auditory response in both Right (Rt) and Left (Lt) ear of chronic alcoholics and non alcoholic controls Latencies (ms) Wave I,
Wave III, Wave V, Interpeak latency (ms) I-III, I-V.

III. Materials and Methods

The present study was carried out in Department of Physiology. Before commencement of the study, it was approved by the institutional ethical committee. It was a case control study carried for 3 years.

Study design: Selections of subjects - 120 male subjects in the age group of 25-50 years were selected randomly from general population. The subjects were divided into two groups, Study group: It includes 60 chronic alcoholic men consuming alcohol more than 21 units/week (1unit=10grams), 1 unit =30ml of alcohol for greater than 5 yrs of duration without abstinence and not having clinical overt neuropathy.[4] Control group: It includes 60 healthy men of same age, socio economic status, BMI and not consuming alcohol. Detailed History of the subject, type, quantity, frequency and duration of alcohol intake was recorded, Alcohol dependence screening was done by using alcohol dependence data Questionnaire (SADD) was used to detect alcohol dependence (Annexure B) Alcohol consumption in units were quantified. General and systemic examination was carried out. ENT examination including, otoscopic examination, tuning fork hearing test was done, to rule ear diseases.

***Exclusion criteria*:** Subjects with: HIV, Tuberculosis, Diabetes, Thyroid disorder, Hypertension, Smokers, Acoustic handicap, Multiple sclerosis , Head , Ear injury, systemic illness -Uremia, stroke, Hepatic encephalopathy, long term medication known to cause neuropathy, i.e anti epileptic, Antipschycotic, Antidepressant drugs,Radiotherapy and chemotherapy.

Parameters selected:

1) **Height in cm:** Standing height of the each participant was measured in cms.[9]
2) **Weight in Kg:** weight was noted in Kg by using KRUPS weighing machine in light weight garments without foot wears.
3) **Body mass index –** wt in Kg/ Ht in m^2
4) **Brainstem evoked auditory response (BERA):** absolute latency in (ms) of waves I, III, V and interpeak latencies in (ms) of I-III and III-V of both Right(Rt) and Left(Lt) ears was measured with **RMS BERA (PUNE) -32 supersec Recorders and medicare system private limited.**

IV. Results

The study population included 120 participants, 60 were alcoholics and 60 were non alcoholic controls healthy male subjects of same age, socio economic status, not consuming alcohol selected randomly from general population. Age, Height, Weight and BMI was statistically non significant in chronic alcoholics as compared to non alcoholic controls.

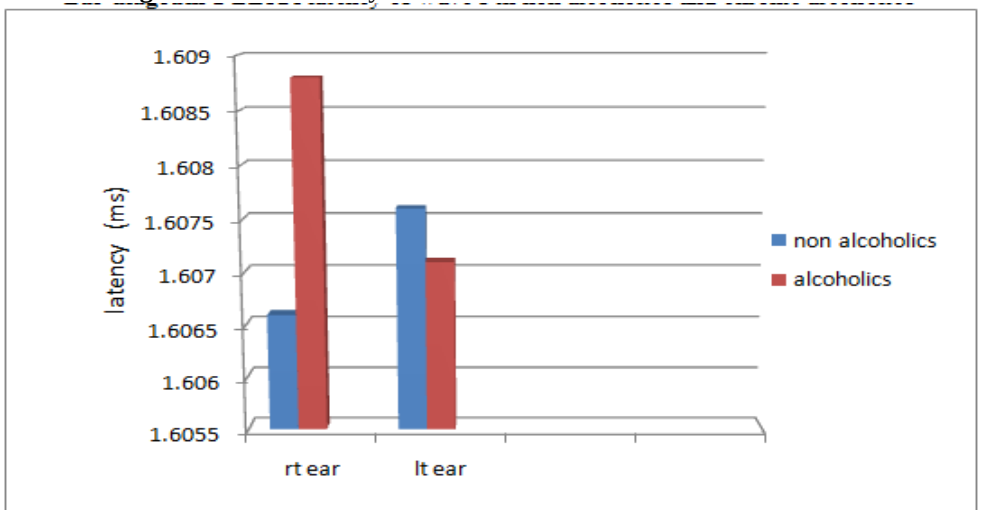
Table 1: Comparison of Brainstem evoked auditory response in non alcoholic controls and chronic alcoholics

Parameters	Ear	Non alcoholics (n=60)	Chronic alcoholics (n=60)	P value
		Mean ±SD	Mean ±SD	
Latency wave I (ms)	Rt	1.606±0.0165	1.6088±0.013	P>0.05
	Lt	1.607±0.022	1.6071±0.012	P>0.05
Latency wave III (ms)	Rt	3.670±0.055	4.138±0.090	P<0.001
	Lt	3.677±0.260	4.145±0.089	P<0.001
Latency wave V (ms)	Rt	5.698±0.065	6.155±0.1048	P<0.001
	Lt	5.666±0.072	6.166±0.100	P<0.001
Interpeak latency wave I-III (ms)	Rt	2.128±0.0155	2.387±0.038	P<0.001
	Lt	2.134±0.026	2.391±0.037	P<0.001
Interpeak latency wave I -V (ms)	Rt	4.041±0.0494	4.370±0.057	P<0.001
	Lt	4.085±0.075	4.3701±0.054	P<0.001

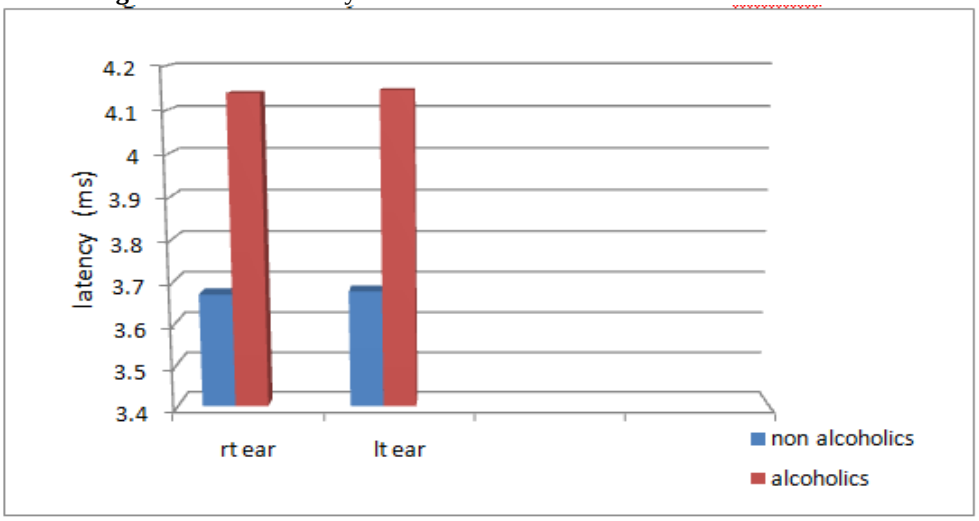
p<0.001 –very highly significant, p>0.05 non significant.

The mean values of latency in wave I in non alcoholic controls and alcoholics was statistically insignificant (p>0.05) andthere was statistically very highly significant increase (p<0.001) in mean latencies of waves III and V in chronic alcoholics as compared to non alcoholics .Mean Interpeak latencies duration of wave I-III and wave I-V in chronic alcoholics was statistically very highly significantly increased (p<0.001) as compared to non alcoholic controls.

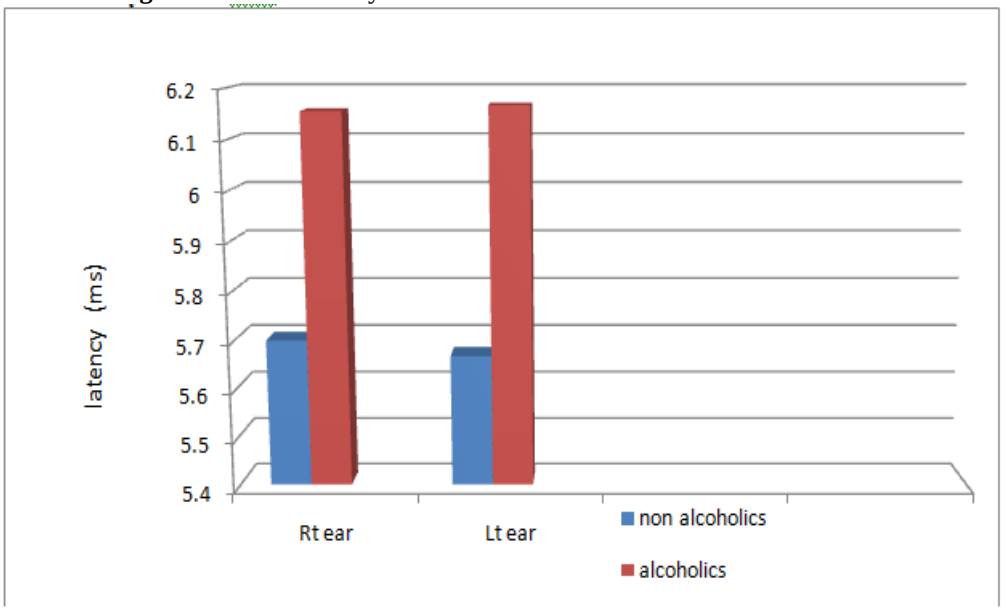
Bar diagram 1 BERA latency of wave I in non alcoholics and chronic alcoholics



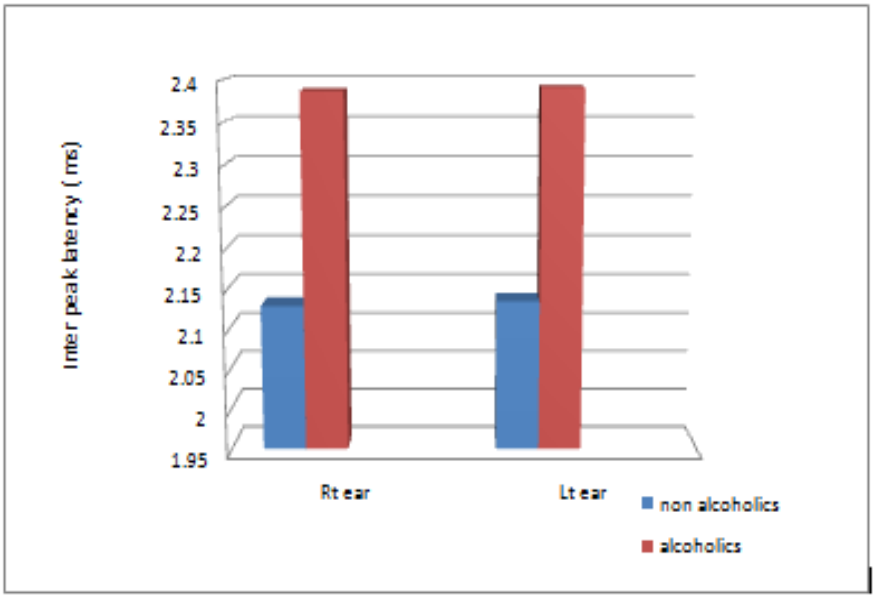
Bar diagram 2 BERA latency of wave III in non alcoholics and chronoic alcoholics



Bar diagram 3 BERA latency of wave V in non alcoholics and chronic alcoholics



Bar diagram 4 BERA Wave I-III Interpeak latencies in non alcoholics and chronic alcoholics



V. Discussion

In this modern world, alcohol abuse is a most common social and economical problem, it is emerging as the third leading cause of death in the world. Long term alcohol abuse affects various body organs cardiovascular system, gastrointestinal tract, respiratory system, central nervous system, peripheral nervous system etc. Electrophysiological tests are considered as unique and sensitive indices of brain function investigation so far. Hence the study was carried out to study and compare the electrophysiological tests Brainstem evoked auditory response, in chronic alcoholics and non alcoholic controls. It was a case control study. 120 participants were consisting of two groups as 60 chronic alcoholics and 60 non alcoholic controls. The chronic alcoholics who were fulfilling the selection criteria, selected randomly from the general population. Controls were selected from non teaching staff of institute. The two groups were matched for age & height, Wt and BMI. Brainstem evoked auditory response**:** Comparison of waves I , III, V, latencies and I-III, I-V interpeak latencies of both Rt and Lt ears in non alcoholic controls and chronic alcoholics.

Mean latencies of wave I of Rt ear of controls was 1.6066±0.0165(ms) and of chronic alcoholics was 1.6488±0.0130(ms) and in Lt ear of controls was 1.6075±0.0222(ms) and in chronic alcoholics was 1.647±0.0126(ms). The mean latency of wave I was statistically insignificant ($p>0.05$) in chronic alcoholics than non alcoholic controls.

Mean latencies of wave III of Rt ear of controls was 3.6701±0.0552(ms) and of chronic alcoholics was 4.1383±0.0903(ms) and in Lt ear of controls was 3.6770±0.2606(ms) and in chronic alcoholics was 4.145 ±0.0891(ms). The mean latency of wave III was statistically very highly significantly increased ($P<0.001$) in chronic alcoholics than non alcoholic controls.

Mean latencies of wave V of Rt ear of controls was 5.6983± 0.0650(ms) and of chronic alcoholics was 6.155±0.1048 and in Lt ear of controls was 5.6666±0.0.0728(ms) and in chronic alcoholics was 6.1666±0.1002(ms). The mean latency of wave V was statistically very highly significantly increased ($P<0.001$) in chronic alcoholics as compared to non alcoholic controls. H. Begleiter et al (1981); found that alcohol consumption prolongs BAEP latencies of wave II, wave III. Auditory brainstem potentials were recorded from abstinent chronic alcoholics and control subjects and found that latencies of peaks II, III, IV, and V were significantly delayed in the alcoholic patients as compared to control subjects. Brainstem transmission time was longer in alcoholics than in controls. Their study provided systematic evidence that chronic alcohol abuse results in brainstem deficits and it may be because of possible demyelination of auditory tracts.[(5)]

Chan et al (1985) also found significant influence of alcohol consumption on BAEP latencies, whereas the impact of neurological disturbances like Wernicke-Korsakoff syndrome was stronger. [(6)]

Diaz et al. (1990) who studied Auditory brainstem evoked potential, Each ear was tested separately,found that the latencies of peaks II, III, IV, and V were significantly delayed in alcoholic subjects compared with control subjects. Cadaveira et al. (1991) found additionally that the parameters most sensitive to long-term alcohol consumption were (in descending order) peak V latency, and the I–V and III–V intervals. B.Porjesz(1982) et al also found that the latencies of peaks II, III, IV**,** and V were significantly delayed in the alcoholic patients compared to control subjects and found Brainstem transmission time was longer in alcoholics than non alcoholic controls.

Mean duration of wave I-III interpeak latency of Rt ear in controls was 2.128±0.01555 (ms) and in chronic alcoholics was 2.38711±0.0382 (ms) and for Lt ear of controls was 2.134±0.0263 (ms) and in chronic alcoholics was 2.391±0.0373 (ms) The mean latency of wave I-III was statistically very highly significantly increased (P<0.001) in chronic alcoholics than non alcoholic controls.Mean duration of wave I-V interpeak latencies in Rt ear of controls was 4.04±0.0494 (ms) and in chronic alcoholics was 4.3701±0.0575 (ms) and for Lt ear in controls was 4.085±0.0755 (ms) and in chronic alcoholics was 4.3735±0.0549 (ms) .The mean latency of wave I-V was statistically very highly significantly increased (P<0.001) in chronic alcoholics than non alcoholic controls.RR TUCK (1983) et all found that, mean value of I-V interval was prolonged in chronic alcoholics. There was also prolongation of I-V and I-III intervals in the Wernicke-Korsakoff syndrome group than in the group without the syndrome.The presence of prolonged I-III interval in an alcoholic raised the possibility of Wernicke's encephalopathy.[6]

Y W CHAN (1985) et all studied BERA in Chronic alcoholics with Wernicke-Korsakoff syndrome and BERA recordings were performed in an isolated room using a Medelec modified averaging system and found Abnormalities in all the interpeak latencies, the I-V and I-III intervals. There was prolonged I-V interval, with a prolonged I-III interval .The presence of prolonged I-III interval in an alcoholic raised the possibility of Wernicke's encephalopathy. Elisebeth Stephanie Smith (2004) et al studied the alcohol consumption of the head and neck tumor patients corresponded to high-risk, dangerous, and risky alcohol consumption behavior, whereas that of the plastic surgery patients corresponded to risky and low-risk consumption behaviour They found prolongation of latencies I, III, V, and prolongation of interpeak latencies I-III, I-V inter peak latencies. The latency I–V of the high-risk, dangerous, and risky drinkers was found to be significantly delayed compared with low-risk drinkers and a logarithmic relationship between BAEP latencies and cumulative lifelong alcohol consumption was obtained.[7]

H.Begleiter et all(1981), studied BERA Waves provide systematic neurophysiological evidence of increased neural transmission time in the brainstem of alcoholic patients who show no clinical signs of corticobulbar or corticospinal tract deficits,data indicate that in the most peripheral part of the auditory pathway there is significant increase in latency of III-V. This significant slowing in neural transmission time reflects a decrease in conduction velocity not elicited by deficits at the peripheral organ. But suggesting pathological changes in the medulla and the pontine formation. Various morphological abnormalities of the auditory brain stem potential have been described in patients with neurological disorders, and electrophysiological deficits have been found to be related to specific neuroanatomical lesions.[5]

Long history of alcohol abuse were suspected of central pontine myelinosis. The pathological changes usually involve the central part of the base of the mid- to upper pons and are characterized histologically by loss of myelin sheaths and oligodendroglia, whereas nerve cells, axis cylinders, and blood vessels remain relatively intact. Demyelination of the auditory tracts and nuclei at the level of the caudal and mid-pons adjacent to the basis pontis has been shown to result in a significant increase in Brainstem transmission time. This demyelination cannot readily be identified by clinical diagnosis and in most cases its presence is only detectable by neurophysiological tests. [5]

Alcohol itself may be ototoxic to the outer hair cells. In general the outer hair cells(OHC), in the basal turn are most vulnerable to ototoxic injuries. Morizono and Sikora et al reported ototoxicity after local application of alcohol to the inner ear of guinea pigs. In their experiment, a reduction in cochlear microphonics was observed, indicating that the OHCs were damaged. A disturbance in the end cochlear environment by alcohol and its metabolites may also result in abnormal outer cell motility. Alcohol may influence neurotransmission in the inner ear. It has been suggested that alcohol suppresses the central nervous system by inhibiting excitatory transmission via N-methyl-Daspartate receptors and enhancing inhibitory transmission via y-aminobutyric acid subtype A ($GABA_A$**)** receptors. A number of studies have confirmed the presence of cholinergic and GABAnergic efferent's on OHCs in animals and so alcohol may suppress the OHCs via these efferent pathways. Middle ear impedance may be affected by alcohol. DPOAE (Distortion Product otto acoustic emissions) arc generated in the cochlea by non-linear interaction of two primary tones introduced externally through the middle ear. They also have to pass through the middle ear before being detected by a microphone in the ear canal.

Therefore, the middle ear transfer function may play an important role in determining the frequency characteristics of DPOAEs. Study demonstrated that consumption of moderate amounts of alcohol induces reductions in DPOAE amplitudes at high frequencies in humans. These amplitude changes are completely reversible. This suggests that alcohol not only affects hearing via the central nervous system, but also influences the function of OHCs. However, the effects of chronic alcohol consumption on the central and peripheral auditory systems are still unclear and require further investigation. [8]

VI. Conclusion

The present study was undertaken with the aim of studying the Brainstem evoked auditory response, in chronic alcoholics and comparing the results with the corresponding non alcoholic controls .Long history of alcohol abuse causes central pontine myelinosis. The pathological changes involve the central part of the base of the mid- to upper pons and are characterized histologically by loss of myelin sheaths and oligodendroglia, whereas nerve cells, axis cylinders, and blood vessels remain relatively intact. Demyelination of the auditory tracts and nuclei at the level of the caudal and mid-pons adjacent to the basis pontis has been shown to result in a significant increase in Brainstem transmission time.

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