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Original Research Article

Effect of Chronic Smoking on the Auditory Pathway by BERA

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Abstract

Background: As per National Family Health Survey-4, about 44.5% men and 6.8% women, have evidence of consume tobacco consumption. Smoking affects the auditory pathway.

Materials and Methods: This study was performed on N=30 normal, healthy non-smoker males and N= 30 healthy, smoker males in the age group of 18-30 years. Informed written consent was taken. After abstinence of 12 hours for chronic smokers the recordings were done. BERA waves and amplitudes were recorded in both the groups.

Results: The latencies of waves I, II and III and inter-peak latency I-III in chronic smokers were delayed and significant as compared to non-smokers. The amplitudes of waves I, II and III were decreased in chronic smokers.

Conclusion: Smoking increases the latency and decreases the amplitudes, hence smoking alters the auditory pathway. Hence, while interpretating BERA, smoking as a confounding factor must be considered.

Keywords: Auditory Pathway, Brainstem Evoked Response Audiometry (BERA), Smoking.

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Introduction

Cigarette contains nicotine that is absorbed and transported by blood to the receptors. [1] The nicotinic acetylcholine receptors are present in the auditory pathways. Thus, nicotine absorbed from the smoking affects the auditory pathways. Increased pack years of smoking have reported high frequency hearing loss.[2] Bhargava et al. reported a decrease in amplitude of waves III, wave IV in rats. [3] Knott et al. reported no changes in latencies or amplitudes of waves after smoking. [4] Kumar and Tandon reported prolonged latencies of waves I and III in chronic smokers. [5] Harkrider et al. reported decrease in amplitude and prolonged wave I latency with nicotine.

The latencies and amplitudes of waves III and V were normal.[6] BERA is a non-invasive test. BERA elicits potentials from the brain stem. The BERA waves are recorded by electrodes placed over the scalp and mastoid.[7] BERA assesses cranial nerve VIII till auditory cortex.[8] The BERA waves I-VII, recorded within a 10-millisecond time.[9] Few studies are there about the impact of smoking on ears by BERA. Hence, aim is to study the effect of smoking on the auditory system by BERA.

Aim: To evaluate the impact of smoking on the auditory pathway by brainstem evoked response audiometry

Material and Methods: Written informed consent was taken from the every participants. A case control study was conducted in the Neurophysiology Lab of the Dept of Physiology, Patna

Inclusion Criteria: 60 healthy volunteers were selected between 18–30 years of age. Group I had N= 30, normal, healthy non-smoker control male subjects, Group II had N=30 male healthy, normal

smokers. Smokers with at least 5 pack years of smoking were enrolled. In one pack-year, 1 pack of cigarettes containing 20 cigarettes being smoked daily for one year [10] Both smokers and controls were abstain from caffeine and nicotine substances for at least 12 hours before the procedure. Hypothesis was BERA wave's latency and interpeak intervals would be delayed and amplitudes would be decrease in chronic smoker's subjects. Sample size was calculated by AI - therapy statistics BETA. Large size effect was (0.8). Alpha was 0.05.[9] 80% power with one tailed hypothesis analysis was done.[11]

Exclusion Criteria: Diabetes mellitus, family history of hearing disorder, anti-psychotic drugs, furosemide, neuropathy, epilepsy, head injury, cardiac arrhythmia, hypertension, drug noise induced hearing loss were excluded from the study.

Rennie's and Webbers tests were performed for hearing. Weight in kilograms and height in centimetres were measured. BMI was calculated as weight divided by the height in square meters (kg/m2).[12]

Active electrode was placed at the mastoid. Reference was placed at the vertex of the skull. Ground was placed at the forehead. The right ear was tested first and opposite ear masking with 40 dB white noise was done. The scalp or skin site was cleaned with alcohol and gel was applied. At least 2 readings were taken for the re-producibility of BERA waves and interpeak latency. A brief click stimuli was given by the headphone on right ear with 80 dB intensity and 2 KHz frequency.[11] Filter was set between 100 Hz to 3000 Hz.[8] 2000 responses were averaged. Skin to electrode impedance was kept below 5 kohm. BERA was recorded by 10–20 International system.[10]

Statistical Analysis: Data analysis by GRAPHPAD QUICKCALCS student unpaired t-test. BERA waves were compared between Group 1 and Group 2. The statistical significance was at p value < 0.05.

Result: The smokers and controls were age matched. The mean age of the smokers were 23.7 ± 2.6 years and that of non-smokers were 23.9 ± 1.7 years. Mean values of waves I, II, III, IV, V and inter-peak latencies I-III, III-V, I-V latency of Group 1 at 80 dB , 2 KHz were 1.34 ± 0.20, 2.36 ± 0.16, 3.62 ± 0.10, 4.69 ± 0.21, 5.39 ± 0.21, 2.10 ± 0.14, 1.71 ± 0.21, 3.99 ± 0.21 and Group 2 were 1.46 ± 0.16, 2.45 ± 0.15, 3.69 ± 0.13, 4.74 ± 0.20, 5.45 ± 0.18, 2.17 ± 0.11, 1.80 ± 0.21, 4.06 ± 0.24 (ms) respectively. Mean values of amplitudes of waves I, II, III, IV, V of Group 1 at 80 dB , 2 KHz were 0.93 ± 0.01, 0.40 ± 0.22, 0.43 ± 0.21, 0.15 ± 0.09, 0.86 ± 0.25 and Group 2 were 0.88 ± 0.12, 0.29 ± 0.12, 0.32 ± 0.16, 0.12 ± 0.10, 0.78 ± 0.01(μv) respectively.

Table 1: Peak latency of BERA waves

Waves	Control Mean ± SD (ms)	Chronic Smoker Mean ± SD (ms)	P value
I	1.34 ± 0.20	1.46 ± 0.16	0.01*
II	2.36 ± 0.16	2.45 ± 0.15	0.02*
III	3.62 ± 0.10	3.69 ± 0.13	0.02*
IV	4.69 ± 0.21	4.74 ± 0.20	0.34
V	5.39 ± 0.21	5.45± 0.18	0.23

***Significant P < 0.05, P < 0.001**

Table 2: Amplitude of BERA waves

Waves	Control Mean ± SD (ms)	Chronic Smoker Mean ± SD (ms)	P value
I	0.93 ± 0.01	0.88 ± 0.12	0.02*
II	0.40 ± 0.22	0.29 ± 0.12	0.01*
III	0.43 ± 0.21	0.32 ± 0.16	0.02*
IV	0.15 ± 0.09	0.12 ± 0.10	0.22
V	0.86 ± 0.25	0.78 ± 0.01	0.08

***Significant P < 0.05**

Table 3: Interpeak latency of BERA waves

Waves	Control Mean ± SD (ms)	Chronic Smoker Mean ± SD (ms)	P value
I-III	2.10 ± 0.14	2.17 ± 0.11	0.035*
III-V	1.71 ± 0.21	1.80 ± 0.21	0.10
I-V	3.99 ± 0.21	4.06 ± 0.24	0.23

***Significant P < 0.05**

Mean values of latencies of waves I, II, III in Group 2 chronic smokers were delayed and significant. The mean values of interpeak latency of I–III in Group 2 chronic smokers was delayed and significant. The amplitudes of waves I, II, III were decreased and significant in group 2 chronic smokers.

Discussion

The above study compared the effect of smoking by BERA in non-smokers healthy subjects and chronic smokers. The significant increase in latencies of waves I, II and III and interpeak latency I–III as well as significant decrease in amplitudes of waves I, II and V in chronic smokers as compared to control group. The BERA waves generated are wave I from VIII nerve, II from cochlear nuclei, III from superior olivary nucleus and IV and V from lateral leminiscus and inferior colliculus.[13] Kumar and Tandon supported above findings.[5] Knott study disagreed with above findings.[4] Nicotine alters the auditory pathway by altering amplitudes and latencies of BERA waves.[4] Harkrider et al. reported a significant prolonged latency and decrease amplitude of wave I in nicotine patch used non-smokers.[6] Studies have shown that nicotine led to up regulation of nicotinic receptors in smokers.[14,15] This present study revealed a decrease in neuronal function could be the nicotine induced reduction in cochlear blood flow.

Conclusion

Smoking alters the auditory pathway and affects the latency and amplitude. Hence, while reporting the BERA waves in patients, nicotine as a confounding factor must be considered.

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