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Smoking induced alterations in auditory pathways: Evidence from evoked potentials

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ABSTRACT

Objectives: Auditory Brainstem Responses (ABR) are used for assessment of integrity of auditory pathway. Given the widespread prevalence of smoking, interpretation of ABR data must be done in light of smoker/nonsmoker status.

Materials and Methods: The present study was done on 30 normal, healthy non-smoker males and 30 healthy, smoker males in the age group of 18-40 years. Approval of Institutional Ethics Committee and written informed consent was taken from all volunteers. ABR was recorded using Biopac MP 150 system. The recordings were done in a state of abstinence of 12 hours (chronic smoking state) and 10 minutes after smoking (acute smoking state). In the non-smoker group, only one set of recordings were done. The peak latencies and amplitudes of the ABR waves were analyzed.

Results: Analysis of data revealed a significant prolongation of the latencies of wave I and II and Inter-peak latency I-III in chronic smoking state as compared to non-smoking state. The amplitudes of waves I, II and V were also significantly reduced in chronic smoking state. A comparison between the non-smokers and acute smoking state revealed significantly prolonged latency of waves I and II in the acute smoking state accompanied by a significant decrease in the amplitudes of all waves of ABR. There were no significant differences in latencies and amplitudes of the chronic and acute smoking state.

Conclusion: Smoking led to an increase in latency and decrease in amplitude, thereby indicating that it adversely affected the auditory pathway. Thus, interpretation of data of ABR should consider smoking as a confounding variable.

Keywords: Smoking, Auditory brainstem response, Hearing

INTRODUCTION

Cigarette smoking is a major health concern in today's era. According to the National Family Health Survey-4, nearly 44.5% men and 6.8% women, consume tobacco in one form or the other.[1] Cigarette smoking is the primary delivery system of nicotine. Each cigarette contains about 9–13 mg of nicotine that is rapidly absorbed and transported by blood to receptors in the central nervous system.[2] The nicotinic acetylcholine receptors have a widespread distribution in the auditory pathways. Cholinergic neurons project from the trapezoid body to cochlear nucleus.[3] In addition to these, there are cholinergic olivocochlear pathways from superior olivary complex to hair cells in cochlea.[4] Thus, it is very likely that nicotine absorbed from smoking may exert an influence on auditory pathways.

Auditory brainstem responses (ABRs) are non-invasive tests to study the integrity of the auditory pathways. They are categorised as waves I to V, reflecting activation of peripheral, pontomedullary and brainstem portions of the auditory pathway.[5] ABRs serve as a useful tool in diagnosis of hearing impairment. They have been shown to be effected by commonly consumed substances such as caffeine.[6] Studies have demonstrated that smoking and nicotine consumption effect the ABR. Increased pack years of smoking have been documented to be positively associated with high frequency hearing loss.[7] Bhargava *et al*. reported that smoking led to a decrease in amplitude of waves III and IV of ABR in rats.[8] Knott *et al*. evaluated waves I, III and V following real or sham smoking and reported no significant changes in latencies or amplitudes of waves I and III after smoking.[9] However, there was an increase in amplitude of wave V following smoking. Kumar and Tandon evaluated ABR in chronic smokers and observed a prolongation of latencies of waves I and III.[10] In their study, Harkrider *et al*. observed a decrease in amplitude and increase in latency of wave I with nicotine. The latencies and amplitudes of waves III and V showed no significant change.[11]

Most of the studies have evaluated the effects of nicotine using alternative delivery methods like patches or have observed chronic effects. There is little evidence to substantiate the effects of smoking (a more realistic method of nicotine consumption in a country like ours!) on ABR in chronic and acute smoking states in the same subset of individuals. This study evaluated the effect of smoking on auditory pathways in chronic smoking state and acute smoking state and compared it to controls.

MATERIALS AND METHODS

The study group comprised 30 normal, healthy non-smoker males and 30 healthy, normal smoker males in the age group of 18–40 years. Smokers with at least 5 pack years of smoking (1 pack-year was taken as 1 pack of cigarettes having 20 cigarettes being smoked daily for 1 year) along with no history of head injury, epilepsy, hearing impairment, migraine, sleeping problems, drug abuse, diabetes and hypertension were recruited for the study.

The study was approved by Institutional Ethics Committee and written informed consent was taken from the subjects before the study. The smokers and controls were asked to abstain from caffeine and nicotine containing substances for at least 12 h before recording of ABR. The ABR was then recorded in state of abstinence for smokers and for controls. Only one set of recordings was done for non-smoker group. The smokers were allowed to smoke their own brand of cigarette to ensure that the blood nicotine levels were not affected by change of brand and ABR was again recorded after 10 min of smoking.

Recording of ABR

The ABRs were recorded with disk electrodes from standard scalp locations of the 10–20 International system using Biopac MP150 digital data acquisition system. The electrode at Cz was active and FPz was ground. The scalp or skin site was prepared by cleaning with alcohol and subsequently applying skin preparation gel and EEG paste. A1 and A2 were used as reference sites. The contact impedance between skin and electrode was kept at <5 KΩ.

A click stimulus of duration 0.1 ms at an intensity of 90 dB pe SPL and a bandpass of 100–3000 Hz was used to record ABR. A total of 1000 responses were averaged. The latencies of waves I–V, interpeak intervals I–III, III–V and I–V and amplitudes of waves were recorded.

Estimation of cotinine levels

Five millilitres blood was collected using aseptic precautions from venipuncture and serum was obtained. The serum was used to estimate levels of cotinine as per manufacturer's specification (Cotinine ELISA by Calbiotech Inc.). The blood sample for smokers was taken after 12 h abstinence (chronic smoking state) and 10 min after smoking (acute smoking state) and for non-smokers only once.

Data analysis

The data obtained were analysed by ANOVA using SPSS 21.

RESULTS

The present study evaluated the effect of smoking on ABR in chronic and acute smoking states and compared it to healthy controls. The smokers and controls were age matched with the mean age of the smokers being 28.7 ± 2.7 years and that of non-smokers, 26.9 ± 1.8 years. The cotinine levels in non-smokers were 0.28 ± 0.12 (ng/ml) which was significantly less than in smokers both in chronic smoking state (77.63 ± 3.39 ng/ml) and in acute smoking state (188.47 ± 30.29 ng/ml). The difference in cotinine levels in smokers in both states was also statistically significant.

The state of chronic smoking was after 12 h' abstinence and acute smoking was 10 min after smoking. The results are summarised in [Tables 1-3].

The results revealed significant prolongation of latencies of waves I and II in chronic smoking state. The latencies of other waves of ABR were also increased, but the changes were not significant. The prolongation of latencies was accompanied by significant increase in interpeak latency I–III in chronic smoking state. The amplitudes of all waves of ABR were reduced in comparison to non-smokers, the changes in waves I, II and V were significant.

Table 1: Peak latency (Mean±SD in ms) of auditory brainstem responses waves.

Wave	Non-smoker	Chronic smoker	Acute smoker
I	1.36±0.22	1.58±0.18***	1.57±0.16***
II	2.38±0.18	2.60±0.17 **	2.66±0.16**
III	3.64±0.13	3.66±0.16	3.72±0.17
IV	4.71±0.24	4.76±0.23	4.81±0.20
V	5.41±0.23	5.59±0.20	5.56±0.25

***P<0.001, **P<0.01. Significance is for comparison between non-smokers and smokers-both chronic and acute separately

Table 2: Amplitude (Mean ± SD in μv) of auditory brainstem responses waves.

Waves	Non-smoker	Chronic smoker	Acute smoker
I	0.95±0.22	0.29±0.15***	0.33±0.16*
II	0.42±0.23	0.29±0.13*	0.22±0.15*
III	0.46± 0.22	0.29±0.17	0.25±0.16*
IV	0.18 ± 0.10	0.15±0.09	0.12±0.08*
V	0.89 ± 0.26	0.55±0.23*	0.53±0.25*

***P<0.001, *P<0.05. Significance is for comparison between non-smokers and smokers-both chronic and acute separately

Table 3: Interpeak latency (Mean±SD in ms) of auditory brainstem responses waves.

Wave	Non-smoker	Chronic smoker	Acute smoker
I-III	2.12±0.16	2.32±0.13*	2.18±0.11
III-V	1.74±0.23	1.90±0.22	1.79±0.19
I- V	4.01±0.22	4.07±0.25	3.97±0.23

*P<0.05. Significance is for comparison between non-smokers and chronic smokers

The changes in latencies and amplitudes in chronic and acute smoking states were non-significant.

The analysis of the data revealed a significant increase in latencies of waves I and II in acute smoking state when compared to non-smoking state. The latencies of other waves were prolonged but it was not significant. There were non-significant changes in interpeak latencies in comparison to non-smokers. Acute smoking led to a significant decrease in amplitudes of all waves of ABR in acute smoking in comparison to non-smokers.

DISCUSSION

The present study evaluated the effect of smoking on ABR in chronic (12 h abstinence) and acute (10 min after smoking) states in comparison to healthy non-smokers. The results revealed significant increase in latencies of waves I and II and interpeak latency I–III in chronic smoking state in comparison to non-smoking. This was accompanied by a significant decrease in amplitudes of waves I, II and V. Acute smoking also led to a significant prolongation of latencies of waves I and II along with significant decrease in amplitudes of all waves when compared to healthy non-smokers. There were no significant differences in the latencies, interpeak latencies and amplitudes of waves in chronic and acute smoking states.

The different waves of ABR are known to have specific generator sites with wave I arising from VIII nerve, II from cochlear nuclei, III from superior olivary nucleus and IV and V from lateral leminiscus and inferior colliculus.[5]

Our findings in chronic smoking state are similar to those of Kumar and Tandon who reported prolongation of latencies of waves I and III in chronic smokers.[10] Knott reported no change in latency or amplitudes of waves I and III in chronic and acute smoking state.[9] This was similar to our study wherein there was no significant difference in latencies of these waves in chronic and acute smoking state. There was a significant increase in amplitude of wave V after smoking, a finding unlike ours. The author attributed the change in amplitude to the effect of nicotine on brainstem.[9]

Harkrider *et al.* investigated the effects of nicotine patch on ABR in non-smokers and found a significant prolongation in latency and decrease in amplitude of wave I.[11] However, the main difference between their study and ours was that they recruited non-smokers and used nicotine patch to evaluate the effects on ABR.

The present study was different from previous ones as it evaluated both the acute and chronic effects and compared to controls rather than study the effects on the two states in isolation.

However, one drawback of this study was that cigarette smoking was used as vehicle for nicotine delivery and not nicotine patch, which is the mode of delivery of pure nicotine. Cigarette smoking is a more realistic method of nicotine administration in India and hence it was used for this study.

Studies in smokers and animals chronically exposed to nicotine have shown that nicotine led to upregulation of nicotinic receptors.[12,13] Smoking and nicotine have been shown to bring about an increase in alertness and information processing in humans.[14,15] The findings of the present study reveal a decrease in neuronal function, a finding that cannot be explained by nicotine induced upregulation of receptors.

The possible reason for the changes in latency and amplitudes of the peripheral components of ABR in this study could be the nicotine induced reduction in cochlear blood flow

leading to sensory loss and cochlear damage.[16] The blood flow reduction causes changes in endocochlear potentials, cochlear microphonics and eighth nerve potentials. It is also possible that the central effects of nicotine were due to the alteration in efferent neural discharge through olivocochlear bundle leading to modulation of the hair cell response.[17]

Another possible mechanism of alterations in ABR could be the effect of other substances like carbon monoxide in cigarette smoke. Smokers' blood has been demonstrated to have higher levels of carbon monoxide.[18] The levels of carboxyhaemoglobin range from 0.5% to 1.5% in normal individuals, whereas the levels in smokers are 5–10%. This increase in carbon monoxide is associated with hypoxemia as carbon monoxide replaces oxygen in haemoglobin.[19] Since auditory pathway has high metabolic rate, it is susceptible to hypoxia and the carbon monoxide induced hypoxia could have brought the alterations in ABR.[20,21]

The present study demonstrates that smoking alters the conduction in auditory pathway. Since cigarette smoke contains various substances, it cannot be definitely concluded as to what substances cause this change. However, the role of nicotine and carbon monoxide seem to be the major factor leading to this effect. Also in diagnostic reporting of ABR, smoking can be a confounding variable and hence it is important to study the effects in greater detail in future work.

CONCLUSION

Smoking effects auditory pathway and brings about changes in latency and amplitude. Since smoking is very common, these changes must be taken into consideration while doing clinical reporting of Auditory Brainstem Responses.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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