Original Research Article

Effect of smoking on auditory P300 event related potential

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Abstract

Background: P300 represents the long latency auditory event related potentials that serve as an objective measure of information and cognitive processing. Smoking remains a serious global threat, yet a modifiable risk factor that may influence cognitive functioning. While the adverse effects of smoking are well appreciated, effect of smoking on the cognitive marker P300 has received less attention. **Objectives:** To assess the effect of smoking on auditory P300 event related potential. **Materials and Methods:** 60 male individuals in the age group of 40-60 years were recruited for the present study. 30 were smokers and 30 were non-smokers. The latency and amplitude of P300 event related potential was recorded at Cz and Pz and compared between smokers and non-smokers. $p< 0.05$ was considered statistically significant. **Results:** There was a significant increase in latency of P300 at both Cz and Pz in smokers compared to the non-smokers. There was a significant decrease in the amplitude of P300 at both Cz and Pz in smokers compared to the non-smokers. **Conclusion:** These results suggest that smoking causes significant alteration in auditory P300, suggesting impaired cognitive processing. Hence intervention may be suggested at earlier stages to smokers to maintain good cognition.

Keywords: cognition, non-Smokers, P300, smokers

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Introduction

The tobacco consumption rate in India is around 28.6%, representing a major health burden to the country.[1] Cigarette smoking, being a predominant form of tobacco consumption acts as a preventable risk factor for morbidity and mortality.[2] Cigarette smoking harms nearly every organ in the body. Cognitive decline is a debilitating health problem in the elderly people.[3] With the increase in the life expectancy of the elderly population in India, [4] it becomes imperative to identify the risk factors that affect cognitive functioning in the elderly age group. In an Indian study it is identified that low cognitive scores are associated with 7% reduction in the quality of life.[5]

Cigarette smoking is a modifiable risk factor that may influence cognitive functioning. While the general health consequences of cigarette smoking are well addressed, [6,7]only few research articles addressed the effect of smoking on neuro cognition and these articles revealed conflicting results.[8,9] Hence the degree to which smoking uniquely contributes to cognitive functioning needs to be established. Auditory P300 represents the long latency auditory event related potentials that serve as an objective measure of information and cognitive processing.[10] Existing data from previous Indian studies are derived from responses

to a questionnaire.[11,12] Unlike these neurocognitive tests auditory P300 event related potentials are least influenced by personality traits and educational status.[13]

Hence the present study aimed to study the effect of smoking on auditory P300 event related potentials by comparing the latency and amplitude of auditory P300 event related potential between the smokers and non-smokers.

Materials and Methods

This cross sectional study was carried out at the Research Laboratory, Department of Physiology of our Institution, after obtaining permission from the Institutional Ethics Committee. Around 60 subjects were selected for the study based on the inclusion and the exclusion criteria. The sample size was estimated using software open epi version 3.01 taking into consideration mean (S.D) of the cognitive parameters based on a previous study with 80% power and 95% confidence interval.[14]

Inclusion criteria: Age group – 40 – 60 years, Males and Current cigarette smokers.

Exclusion Criteria: Females, Bidi smokers, Known alcoholics, Diabetics, Hypertensives, Ex-smokers, individuals with hearing deficits and under treatment on sedatives, anti-psychotics

Methodology

Informed consent was obtained from all the study participants. The demographic details, smoking history was obtained from them. Basic anthropometric measurements were measured and physical examination was carried out. For the smokers pack years were calculated by multiplying the number of packs smoked/day with number of years of smoking. Then the study participants were taken to the research laboratory of department of Physiology for the measurement of auditory P300 event related potentials. They were classified into two groups as Smokers and Non-Smokers.

Recording of P300

Subjects were informed about the nature of procedure. The subject were made to relax on a couch in the research laboratory with closed eyes. Rare tone (2 KHz) and frequent tone (1 KHz) of 85dB were applied on both ears together in 20% and 80 % in frequency in random through headphones. Total 300 stimuli were applied at rate of 1 stimulus/sec. The recording sites on scalp were cleaned with spirit. One active electrode was attached on vertex (Cz), one as ground electrode to forehead (Fz) and two reference electrodes were fixed to right and left mastoid designated as A1 and A2 respectively. All the electrodes were plugged to a junction box keeping skin to electrode impedance below 5 K ohms. Subjects were asked to avoid sleep and identify the rare stimulus, counting in loud voice. The signals were picked up by electrodes and filtered, amplified, averaged, displayed on the screen of EMG EP MK II equipment (Electromyography, Evoked potential machine, MK II model, Recorders and Medicare System Private Ltd. Chandigarh, India) and recorded. Two reproducible recordings were taken for a subject and averaged together to obtain the final measurement. Latency and amplitude of P300 wave for rare stimuli were measured.

Statistical Analysis

The baseline characteristics and the cognitive parameters were compared between the smokers and the non-smokers using unpaired t test. The correlation between pack years and the cognitive parameters were assessed by Pearson's correlation test. p value < 0.05 was considered statistically significant. SPSS version 20 was used for statistical analysis.

Results

Of the total 60 study participants, 30 were smokers and 30 were non-smokers. Table 1 represents the baseline characteristics of the study participants.

Table 1: Baseline characteristics of the study participants

Parameters	Values
N	60
Age	50.16 ± 5.56
Latency (C_z) (ms)	290.89 ± 23.42
Amplitude (C_z) (µv)	5.62 ± 1.66
Latency (P_z) (ms)	288.03 ± 24.42
Amplitude (P_z) (µv)	5.46 ± 1.74
Smokers	
Pack years	22.26 ± 16.51
Duration	10.05 ± 4.72

Values expressed as Mean ± S.D, n – sample size, ms – milliseconds, µv – microvolts

Table 2 represents the comparison of the P300 Latency and amplitude at C_z and P_z between the smokers and the non-smokers. The latency of P300 for the rare stimuli was significantly prolonged in smokers compared to non-smokers, at both C_z and P_z ($p < 0.001$). The amplitude of P300 for the rare stimuli at both C_z and P_z was significantly lower in smokers compared to the non-smokers ($p < 0.001$).

Table 2: Comparison of P300 latency and amplitude (at C_z and P_z) between smokers and non-smokers

Parameters		Smokers	Non-Smokers
P300 Latency (Rare stimuli)	C_z (ms)	301.95 ± 20.86*	279.82 ± 20.67
	Pz (ms)	302.29 ± 21.84*	273.78 ± 17.78
P300 Amplitude (Rare stimuli)	P_z (µv)	4.60 ± 0.79*	6.65 ± 1.68
	C_z (µv)	4.69 ± 0.55 *	6.22 ± 2.15

* $p < 0.001$

Table 3 represents the correlation between the pack-years and cognitive parameters. There was significant positive correlation between the pack years and the P 300 latencies ($p < 0.01$). However no significant correlation was found between the amplitude of P300 and the pack years.

Table 3: Correlation between the Cognitive parameters and the pack years among smokers

Parameters	P300 Latency C_z (ms)		P300 Latency P_z (ms)	
Pack years	**R**	**p**	**r**	**p**
	0.44	0.01*	0.49	0.001*

Discussion

The present study compared the auditory P300 event related potential between the smokers and the non-smokers. The results showed a significant increase in the latency and decrease in the amplitude of P300 waves recorded at C_z and P_z in the smokers compared to the non-smokers. Similar results were observed by other studies.[15,16] Hedges and Bennett in their systemic review suggested a strong dose-dependent influence of smoking on cognition.[15] Anokhin and co-workers reported that chronic smoking may induce reversible changes in the brain and the effect on P300 may serve as a risk indicator for nicotine dependence.[16] Global brain atrophy and structural and biochemical abnormalities in anterior frontal regions, subcortical nuclei and commissural white matter are some of the neurological changes observed due to chronic effects of nicotine.[15,16] Prefrontal cognitive dysfunction was observed in chronic cigarette smokers by Guney F and his colleagues.[17] Sudharkody et al observed that prevalence of cognitive impairment significantly increased with pack year of smoking.[12]

The results of the present study goes in contrast with the findings of Houlihan ME, as they observed reduced latency of P300 in smokers.[18] However Houlihan ME recorded P300 in smokers immediately after smoking. Hence the reduction in P300 latency may be due to acute effect of nicotine on cognition. Increased cortical arousal and enhanced regional cerebral blood flow are some of the reasons quoted as acute effects of nicotine. They also show enhanced attention and improved consolidation.[9] Similar results were observed by Dumatar C, where short term memory, alertness, visual-retention and motor-

coordination improved immediately after smoking.[11]

However in the present study, as the smokers were asked to abstain from smoking overnight, the effect of smoking abstinence on cognition may not be excluded. Several studies report that even 14 hours of smoking abstinence may delay the neural processing. Acute abstinence from cigarette smoking may alter dopaminergic transmission in the striatum and the prefrontal cortex of nicotine-dependent human subjects.[19,20]

In the present study mean pack years of the smokers was strongly evident of chronic smoking, hence chronic effects of nicotine may be attributed as a major cause for prolongation in P300 latency.

However this study has its own limitations. It is a cross sectional study with small sample size. Hence a longitudinal study with neuroimaging details may serve as a better indicator of effect of smoking on cognition.

Conclusion

Chronic cigarette smoking may significantly influence cognitive function as evident by the long latency of the event related potential P300. Smoking being a modifiable risk factor, quitting smoking at an earlier stage may help to maintain adequate cognition at later stages. Auditory P300 may be suggested as a routine screening test for cognitive assessment in smokers.

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Conflict of interest: Nil

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