ISSN: 0975-1556

Available online on www.ijpcr.com

International Journal of Pharmaceutical and Clinical Research 2022; 14(1); 728-733

Original Research Article

To Record the Visual Evoked Potential in Smokers and Non-Smokers and Comparison Between Two Groups for Any Changes in P100

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Received: 10-11-2021 / Revised: 27-12-2021 / Accepted: 20-01-2022
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Conflict of interest: Nil

Abstract

Objectives: To record the visual evoked potential in smokers and non-smokers aged 20-40years and to compare between two groups for any changes in P100.

Materials and Methods: Age matched 120 male smokers and 120 male non smokers in the age group of 20-40 years were studied for visual evoked potential. Smoking in terms of pack years was noted. Data was statistically analyzed.

Results: Visual evoked potential was affected in smokers with prolongation of latency and decrease in amplitude of P100 in both the eyes than non smokers, with is statistically highly significant. There is a significant positive correlation between smoking history expressed in pack-years and latency of P100 in smokers group.

Conclusion: Smoking causes degeneration in optic nerve shown by increased latency of P100 which increases as the number of pack-years increase.

Keywords: Visual Evoked Potential (VEP); Visual reaction time (VRT); Smokers.

Introduction

Tobacco smoke contains Nicotine and carbon monoxide. They harm peripheral nerves as well as central areas of brain. [1]Nicotine causes demyelination. [2, 3] Again, long-term smokers who have already developed COPD suffer from hypoxia which is clearly linked to neuropathy. [4]

Additionally, individual differences in personality [5, 6]together with situational factors such as induced stress [7]are important intervening variables determing the effects of cigarette smoking on electrocortical activity.

Chronic cigarette smoking appears to be associated with deficiencies in executive functions, cognitive flexibility, general intellectual abilities, learning and/or memory processing speed, and working memory. [8]

The effect of smoking on visual pathway can objectively be very well observed through visual evoked potential (VEP). This is a quick neurophysiologic, low-cost, non-invasive test which assesses the functional integrity of visual system. Therefore, this study aims to record the visual evoked potential in smokers and non-smokers aged 20-40years and to compare between two groups for any changes in P100.

Materials and Methods:

The smoking history of each subject of the test group was expressed in terms of pack-years. Pack-years of smoking is defined as the number of packs (one pack is equal to 20 cigarettes) smoked per day multiplied by the duration of smoking (in years). Subjects were instructed to restrain from smoking one hour before the tests.

Electrophysiological studies for the evaluation of VEP was carried out using RMS EMG EP MARK II supplied by Recorders And Medicare Systems (Pvt) Limited. Procedure for VEP recording as recommended by the International

Federation of Clinical Neurophysiology (IFCN) Committee was followed with stimulus configuration consisting of the transient pattern reversal method in which a black and white checker board was generated (full field) and displayed on VEP monitor (color 14") by an electronic pattern generator inbuilt in Evoked Potential Recorder (RMS EMG EP

MARK II).

After screening into study or control group according to inclusion and exclusion criteria subjects were instructed to come to Research Laboratory with their hair washed without applying oil. Study was conducted in Research laboratory, Department of Physiology, Patna Medical College & Hospital, Patna, Bihar.

Statistical analysis:

Student's unpaired 'T' test has been used to find out the significance of homogeneity of study characteristics between two groups of subjects. Simultaneous comparison of all 3 groups of cases is carried out by one-way ANOVA test which was followed by Tukey's post hoc test for pairwise comparison. Spearman's coefficient of correlation test was applied to find the correlation among 3 groups of cases with respect to pack years. Differences were considered significant at $p < 0.05$ level. The data has been analyzed by using SPSS 18 (Trial Version) USA, Chicago.

Results:

This is a Case-control study with 120 male smokers and matched controls of 120 male nonsmokers is taken to study the effect of smoking on VEP.

The study groups are age matched as the p value is 0.06. (Table 1)

Table 2: This table depict that there is highly significant difference in latency of both groups.

Table 3: This tables depict that there is highly significant difference in amplitude of both groups.

Table 4: shows Comparison of latency between smokers with relation to pack years in right eye and left eye, shows that latency of left eye is affected in smokers. On comparison of different groups latency of subjects in group II (2-5 pack years) are most affected and value is statistically significant.

Table 5 shows Comparison of amplitude between smokers with relation to pack years in right eye and left eye, shows that amplitude of right eye is affected in smokers. On comparison of different groups amplitude of subjects in group II (2-5 pack years) are most affected and value is statistically significant.

Table 6 shows Spearman's correlation of pack years with latency and amplitude of

VEP of smokers. There is positive correlation for latency of both eyes with a significant P value.

Table 1: Age distribution of subjects studied

Age (Years)	Cases	Controls	Total No.	Total %
20-24	10	14	24	10.0
25-29	37	40	77	32.0
30-34	42	36	78	32.5
35-40	31	30	61	25.4
Total	120	120	240	100
Mean+/-SD	31.8+/- 5.7	31.3+/-5.2		

*Student's unpaired t test, Samples are age matched with P= 0.06

Table 2: Comparison of latency between smokers and nonsmokers in both right eye and left eye

Pack years	<2 (n=56)	2-5 (n=35)	>5 (n=9)	P* Value	Significant pairs**
Visual evoked potential	Mean + SD	Mean + SD	Mean + SD		
Latency msec, Right eye	121.81 + 5.71	128.81 + 5.29	111.21 + 4.91	0.051	
Latency msec, Left eye	108.21 + 4.01	108.82 + 5.80	111.81 + 4.04	0.001S	I&II

* Student's unpaired t test, HS – Highly significant

Table 3: Comparison of amplitude between smokers and nonsmokers in both right eye and left eye:

Visual Evoked Potential	Cases	Controls	Mean Difference	p-value
	Mean + SD	Mean + SD		
Amplitude (uV) Right eye	4.81 + 1.91	7.20 + 2.17	2.81	<0.001 HS
Amplitude (uV) Left eye	3.01 + 1.89	6.91 + 2.83	3.10	<0.001 HS

* Student's unpaired t test, HS – Highly significant

Table 4: Comparison of latency between smokers with relation to pack years in right eye and left eye:

Pack years	<2 (n=56) I	2-5 (n=35) II	>5 (n=9) III	P Value	Significant pairs
Visual evoked potential	Mean + SD	Mean + SD	Mean + SD		
Amplitude (uV) , Right eye	6.21 + 2.81	4.01 + 1.71	5.71 + 2.01	0.03	II & III
Amplitude (uV), Left eye	6.29 + 2.01	4.44 + 1.01	5.13 + 1.15	0.30	

Table 5: Comparison of amplitude between smokers with relation to pack years in right eye and left eye:

Visual Evoked Potential	Cases	Controls	Mean Difference	p-value
	Mean + SD	Mean + SD		
Latency (msec) Right eye	128.29 + 5.12	112.31 + 4.81	4.61	<0.001 HS
Latency (msec) Left eye	131.21 + 5.48	119.20 + 4.01	4.18	<0.001 HS

Student's unpaired t test, HS – Highly significant

Table 6: Spearman's correlation of pack years with latency and amplitude of VEP of smokers:

Visual Evoked Potential		P value
Latency msec, Right eye	0.31	0.01 S
Amplitude (uV), Left eye	-0.080	0.40 NS
Latency msec, Right eye	0.36	0.002 S
Amplitude (uV), Left eye	-0.111	0.30 NS

S- Significant

Discussion:

Cigarette smoking affects almost every system in the human body and it is accepted as a risk factor for various cancers, heart diseases, strokes, emphysema and many eye diseases as shown by various studies.

Smokers generally have a higher probability of respiratory symptoms which are often accompanied by pulmonary function abnormalities with a greater annual rate of decline in FEV1 and maximal expiratory flow volume as compared to non-smokers. Many studies done [9] on healthy tobacco smokers had found that spirometric parameters were significantly lower in all smokers (even mild smokers) than in non-smokers. Our study also showed similar results. This decline in PFT is even evident in teenagers who had smoked only for a few years. This indicates that smoking cause narrowing in the diameter of airways.[10,11]

Study by Rose FC, on smokers with optic neuritis found that there was high incidence of color vision defects in smokers when compared with nonsmokers. Vascular effects of smoking may be due to a direct effect of nicotine which could act either by depressing retinal ganglion cell function, block transmission in demyelinating nerve fibers, blocking synaptic transmission at lateral geniculate

body or depressing receptor cells in striate cortex.[12]

Change in photic driving balance, a monitor of 'lability-stability' of the CNS [12], depended on (mouth) nicotine which in turn was (positively) related to neuroticism personality score. Higher nicotine delivery predicted increases in PD response at the lower frequency (16 Hz) but decreases at the higher frequency (26 Hz).[13]

An increase in VEP latency clinically means degeneration in the quality of sight. Study by Rose FC, on smokers with optic neuritis found that there was high incidence of color vision defects in smokers when compared with nonsmokers. Vascular effects of smoking may be due to a direct effect of nicotine which could act either by depressing retinal ganglion cell function, block transmission in demyelinating nerve fibers, blocking synaptic transmission at lateral geniculate body or depressing receptor cells in striate cortex.[13]

The delayed response to visual stimuli in smokers might be due to various patho-physiological changes probably like atherosclerosis of arteries and arterioles supplying cerebral hemisphere. This may be the result of tobacco smoking which leads to abnormal increase in total blood triglycerides, enhanced blood coagulability due to increased fribrinogen. There is reduction in small airways function with low levels of PaO2 and PaCO2 which might lead to decreased cerebral blood flow. Smokers develop elevated carboxyhaemoglobin levels which might impair function of central nervous system by affecting oxygen transport and its utilization leading to cognitive dysfunction and perceptual motor delay in smokers.[14]

The VRT was shorter in smokers than in non- smokers in our study and also in a study done by Ichaporia et al 1991.[15] This could be due to stimulant action of nicotine which enhances the effect of visual attention. In general small doses of nicotine have stimulating and arousal action on CNS (whereas large dose suppress it) especially in cortical neurons, limbic system and reticular activating system.[16] Thus, smoking enhances response to preparation and execution.[17,18]

Though there are different opinions regarding effects of smoking on VEP, studies suggest that immediately after smoking reaction time becomes faster than baseline[18]and there is increased amplitude, decreased latency of P100[19] produced due to the stimulant effect of nicotine on CNS.[20]

Conclusion:

The data was statistically analyzed which revealed that smokers had Increased latency& Decreased amplitude of P100 waves of VEP in both eyes. There exists a significant positive correlation between smoking history expressed in pack-years and latency of P100 in smokers group.

References:

1. Hughes JR. Motivating and Helping Smokers to Stop Smoking. J Gen Intern Med 2003; 18: 1053–1057.
2. Faden A, Mendoza E and Flynn F. Subclinical neuropathy associated with chronic obstructive pulmonary disease: possible pathophysiologic role of smoking. Arch Neurol 1981; 38: 639–642.
3. Northrop-Clewes CA and Thurnham DI. Monitoring micronutrients in cigarette smokers. Clin Chim Acta 2007; 377: 14–38.
4. Pfeiffer G, Kunze K, Brüch M, Kutzner M, Ladurner G, Malin JP, et al. Polyneuropathy associated with chronic hypoxaemia: prevalence in patients with chronic obstructive pulmonary disease. J Neurol 1990; 237: 230–233. 1990; 237: 230–233.
5. Ashton, C. H., J. E. Millman, R. Telford and J. W. Thompson. The effects of caffeine, nitrazepam and cigarette smoking on the contingent negative variation in man. Electroencephalogr Clin Neurophysiol 37: 5%71, 1974.
6. Cinciripini, P. M. The effects of smoking on electrocortical arousal in coronary prone (Type A) and non-coronary prone (Type B) subjects. Psychopharmacology (Berlin) 90: 522-527, 1986.
7. Golding, J. F. and G. L. Mangan. Arousing and de-arousing effects of cigarette smoking under conditions of stress and mild sensory isolation. Psychophysiology 19: 449--456, 1982.
8. Durazzo TC, Meyerhoff DJ, Nixon SJ. Chronic cigarette smoking: Implications for neurocognition and brain neurobiology. Int J Environ Res Public Health. 2010;7:3760–3791.
9. Mehta V, Desai N and Patel S. When Pulmonary Function Test is Available, Should we Wait for the COPD Symptoms to Develop? J Clin Diagn Res 2016; 10: OE08–OE12.
10. Kenfuni, M. M., Gallouo, M., alafifi, mahmoud, Tsikambu, A. C. D., Alafifi,

R., Moataz, A., Dakir, M., Debbagh, A.,& Aboutaieb, R. (2022). Pyonephrose : Risk factors, clinical, para-clinical and anatomopathological profile about 19 cases. Journal of Medical Research and Health Sciences, 5(2), 1770–1773. https://doi .org/10.52845/JMRHS/2022-5-2-1

11. Urrutia I, Capelastegui A, Quintana JM, Nerea M, Xavier B, Jordi S, et al. Smoking habit, respiratory symptoms and lung function in young adults. Eur J Public Health 2005; 15: 160–165.
12. Fletcher C and Peto R. The natural history of chronic airflow obstruction. Br Med J 1977; 1: 1645–1648.
13. Mangan, G. L. The Biology of Human Conduct: East-West Models of Temperament and Personality. Oxford: Pergamon Press, 1982.
14. Smoking and optic neuritis. Postgraduate Medical J. 1975;51:382–385.
15. P, Deshpande KP, Phatak VK, MS. The study of auditory and visual reaction times in chronic smokers. Int J Med Health Sci. 2013;2(1):18–22.
16. Ichaporia RB, Kulkarni SP, Malthi A and Parulkar VG. Study of reaction time in smokers. J Postgrad Med 1991; 37: 209–210.
17. Conrin J. The EEG effects of tobacco smoking--a review. Clin Electroencephalogr 1980; 11: 180–187.
18. Pritchard W, Sokhadze E and Houlihan M. Effects of nicotine and smoking on event-related potentials: a review. Nicotine Tob Res 2004; 6: 961–984.
19. Woodson PP, Baettig K, Etkin MW, Kallman WM, Harry GJ, Kallman MJ, et al. Effects of nicotine on the visual evoked response. Pharmacol Biochem Behav 1982; 17: 915–920.
20. Afshan A, Bhutkar MV, Reddy R, Patil RB. Effect of chronic smoking on intraocular pressure and audio-visual reaction time. Int J Biol Med Res. 2012;3(2):1760–1763.