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EXPLORE THE EFFECT OF SMOKING ON THE VEP RESPONSE AMONG SMOKERS: AN ANALYTICAL STUDY.

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Abstract

Aim: to explore the effect of smoking on the VEP response among smokers.

Materials and Methods: The present prospective case-control study was conducted in the Department of Physiology, Neta Ji Subhash Medical College and Hospital, Patna, Bihar. Age matched 100 male smokers and 100 male non smokers were recruited. Data was statistically analyzed.

Results: The male subjects selected with mean age of 45.76 years. 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.

Conclusion: concluded that Visual Evoke Potential was affected in smokers with prolongation of latency and decrease in amplitude of P100 in both the eyes when compared to non smokers.

Keywords: Smokers, VEP, P100, Latency

Introduction

World Health Organization estimates that worldwide 5 million deaths are caused prematurely by smoking every year.[1] In India, over 6, 00,000 people in the age group of 25-69 years die due to smoking every year.[2] Currently in India there are about 120 million people who either smoke cigarette or bidi.[3] Current percentage of male tobacco smokers are 24.3% in India to Global Adult Tobacco Survey 2010.[4]

It is by far well established that the smoking affects the blood flow. Many researchers quote that as smoking alters the normal circulation it has significant effects on cerebral perfusion as well and may lead to alteration in physiology regulated by the area of brain suffering hypoperfusion.[5]

Smoking increases the risk of macular degeneration, cataracts, and poor eyesight. Of the 40,000 active substances in tobacco smoke, most are hazardous to human health. These toxic chemicals affect ocular tissues through ischemic or oxidative mechanisms.[6] Many common ophthalmological disorders such as retinal vein occlusion, age-related macular degeneration, cataract, anterior ischemic optic neuropathy, thyroid ophthalmopathy, and primary open angle glaucoma have been found to be associated with smoking. Diminished retinal sensitivity and peripheral scotomas in the visual fields have been observed in healthy heavy smokers.[7,8]

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, noninvasive test which assesses the functional integrity of visual system. Through this, the study tried to observe and analyze the alterations in VEP in smokers. The aim of this study was to explore the effect of smoking on the VEP response among smokers.

Material & Methods

Study Design

The present prospective case-control study was conducted in the Department of Physiology, Neta Ji Subhash Medical College and Hospital, Patna, Bihar. The study protocol was reviewed by the Ethical Committee of the Hospital and granted ethical clearance. After explaining the purpose and details of the study, a written informed consent was obtained.

Inclusion criteria

Cases

- Patient who signed the “informed consent” form
- Male Patients ≥ 18 years of age
- History of smoking ≥15 cigarettes daily for at least 3 years

Controls

- Age matched
- Nonsmokers who did not smoke

Exclusion criteria

- Patients <18 years of age
- History of alcohol consumption
- Patients having preexisting ophthalmic complication
- Patients having a history of any neurological disorder

- Patients with history of diabetes mellitus and hypertension

Methodology

For all the participants the complete clinical history and physical examination followed by relevant clinical investigation were carried out and demographic data and smoking history was recorded.

VEP recordings were done in accordance with the standardized methodology of the International Federation of Clinical Neurophysiology committee recommendations and the International Society for Clinical Electrophysiology of Vision guidelines, and montages were kept as per the 10-20 International System of electroencephalogram (EEG) electrode placements.[9-11] The reference electrode (Fz) was placed 12 cm above the nasion, the ground electrode (Cz) at the vertex, and the active electrode (Oz) at approximately 2 cm above the inion.

The study parameters included P100 latency which is the time interval between the onset of a visual stimulus and the first maximum positive deflection or excursion of the VEP signal and P100 amplitude which is measured from the peak of N70 to trough of P100 wave.

Statistical Analysis

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2010) and then exported to data editor page of SPSS version 19 (SPSS Inc., Chicago, Illinois, USA). Descriptive statistics included computation of percentages.

Results

Table 1: demographic profile

Variables	Mean ± SD	
	Case	Control
Age	45.76±3.81	44.71±3.63
BMI	27.21±2.31	26.44±2.11
Duration of Smoking	5.79±1.81	-
Number of cigarettes per day	17.81±3.98	-

Table 2: Comparison of P100 wave latency in the right and left eyes among groups

P100 latency (ms)	Mean ± SD	
	Case	Control
Right Eye	115.76±7.11	94.71±3.13
Left Eye	115.21±7.21	95.04±3.01

Table 3: Comparison of P100 amplitude in the right and left eyes among groups

P100 amplitude (µvolts)	Mean ± SD	
	Case	Control
Right Eye	3.76±3.41	5.71±3.43
Left Eye	3.21±2.61	5.44±2.11

Discussion

This study was taken up to study the effects of cigarette smoking on vision through electrophysiological technique like VEP and by measuring visual reaction time. In this study the male subjects selected with mean age of 45.76 years. This study was taken up to study changes in VEP before clinical signs and symptoms related to vision appear in smokers.

Analysis of this study showed that VEP was affected in smokers with prolongation of latency and decrease in amplitude of P100 in both the eyes when compared to non smokers. 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 colour vision defects in smokers when compared with non smokers. 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]

Smoking is also associated with deficiencies in auditory-verbal learning or memory, general intellectual abilities, visual search speeds, processing speed and executive functions.[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 fibrinogen. There is reduction in small airways function with low levels of PaO_2 and $PaCO_2$ 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 perceptualmotor delay in smokers.[14]

Though there are different opinions regarding effects of smoking on VEP, studies suggest that immediately after smoking reaction time becomes faster than baseline[15] and there is increased amplitude, decreased latency of P100 produced due to the stimulant effect of nicotine on CNS.[16]

Conclusion

The present study concluded that Visual Evoke Potential was affected in smokers with prolongation of latency and decrease in amplitude of P100 in both the eyes when compared to non smokers. An increase in VEP latency clinically means degeneration in the quality of sight. To generalize the results large population based study is

required. The VEP abnormalities are nonspecific and are not characteristic of any specific etiology.

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