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A Study of Brain Bioelectrical Activity in E-Cigarette Users

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Abstract

Case reports describing seizures among electronic cigarette (EC) users have recently appeared in the literature. However, objective studies evaluating brain functional activity in EC users remain limited. The aim of this study is to evaluate the functional state of the brain in electronic cigarette users by analyzing electroencephalographic bioelectrical activity compared with healthy volunteers.

Methods and Findings: The main group included 10 individuals without neurological complaints who had regularly used EC for more than one year. The control group consisted of 20 healthy volunteers who had never used EC. EEG recordings were obtained, background activity and functional tests. Spectral analysis of background EEG was performed in the alpha, beta-1 and beta-2 frequency bands. Statistical analysis was conducted using nonparametric methods. EEG changes were observed as bursts of beta-range activity in frontal, temporal and parieto-occipital regions. Epileptiform patterns were detected in 60% of cases. Spectral analysis of background activity demonstrated significantly higher spectral power in the beta-1 and beta-2 frequency ranges in EC users compared with controls ($p < 0.01$).

Conclusions: EC users exhibited electrophysiological signs of increased cortical excitability. These findings may reflect a subclinical neurotoxic effect of EC aerosol components and suggest the presence of a latent period preceding potential neurological manifestations.

Keywords

Electronic Cigarettes; EEG; Brain Bioelectrical Activity; Neurotoxicity; Cortical Excitability.

Introduction

In recent years, reports have begun to appear in the literature regarding seizures in e-cigarette users and their potential neurotoxicity [1]. Seizures have been reported among both new e-cigarette users and experienced users. In some cases, seizures began immediately after the first puff, while in others, they occurred after several weeks of regular use. Some e-cigarette users have had to stop using vapes due to recurrent seizures. In many cases, there were no documented pre-existing medical conditions or comorbidities. It has been suggested that not only nicotine but also other components of e-cigarette aerosols, such as glycerin, propylene glycol, and flavorings, may have neurotoxic properties [1,5,7,10].

However, studies investigating the bioelectrical activity (BEA) of the brain in individuals who systematically use electronic cigarettes (EC) are extremely limited. Electroencephalography (EEG) is an objective method for assessing the functional state of the brain.

Aim of the study

To assess the functional state of the brain in e-cigarette users in comparison with healthy volunteers based on electroencephalographic analysis of bioelectrical activity.

Materials and Methods

The study involved 30 volunteers under 30 years of age, divided into two groups: the main group (n=10) consisted of individuals with a history of regular EC use of over 1 year, and the control group (n=20) consisted of individuals who had never used EC. Inclusion criteria for all participants included the absence of neurological complaints, chronic diseases, medication, and drug or alcohol use. All subjects underwent recording of their brain bioelectrical activity using electroencephalography (EEG) on a 16-channel Biola Neuro Scope NS416A device using standard methods. The baseline EEG recording (without presentation of afferent stimuli) lasted from 3 to 5 minutes. Next, tests were performed with eyes open, rhythmic photo stimulation, and hyperventilation (3 minutes each).

EEG spectral analysis was performed using a 16-lead fast Fourier transform algorithm in the computerized encephalograph software. Artifact correction was performed before mathematical analysis of the EEG. EEG segments lasting 3-5 minutes of background recording were analyzed. Absolute spectral power ($\mu\text{V}^2/\text{Hz}$) was calculated in the alpha (8-14 Hz), beta-1 (14-20 Hz), and beta-2 (20-35 Hz) ranges.

Statistical analysis was performed using nonparametric statistical methods (descriptive statistics: median, quartiles of spectral power of alpha and beta range rhythms; one-way analysis of variance) in the STATISTICA 10.0 program.

Results

Visual analysis of the EEG in the study group revealed a disturbance in the zonal distribution of the alpha rhythm in the background recording in all participants, with a tendency toward alpha rhythm hyper synchronization in the frontal-central areas. Dysfunction of the brainstem structures varied in severity and was most often manifested by generalized bursts of alpha waves, and less frequently by generalized bursts of theta, beta, or polymorphic sharp waves (Figure 1). During stress tests, pronounced irritative changes in the brainstem activity were observed, manifested as localized bursts of beta waves in the frontal, temporal, or parieto-occipital areas. In 60% of cases, epileptiform signs (sharp waves) were recorded in the frontal,

temporal, or parieto-occipital areas (Figure 1).

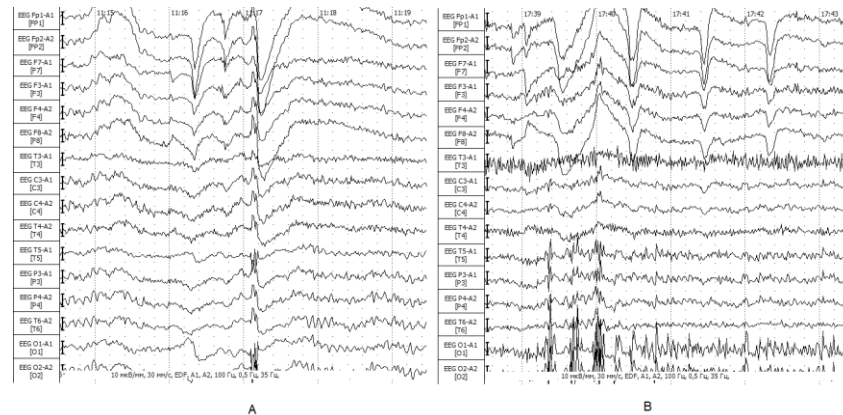


Figure 1: A - generalized burst of polymorphic sharp waves in the background recording of vaper A (eyes closed); B - burst of bilaterally synchronous beta waves in the parietal-occipital areas during the recording with open eyes of vaper B.

Irritative changes of bioelectrical activity were most frequently recorded in several regions simultaneously: frontotemporal and/or temporoparietal areas. In 80% of cases, they were detected in temporo-parieto-occipital regions, and in 60–70% of cases in frontotemporal regions. Local irritative changes of bioelectrical activity were practically not recorded in the background EEG in the main group, therefore, spectral analysis was used to assess the power of the beta rhythm in the background recording.

Spectral analysis of baseline EEG recordings in the main group revealed a median alpha rhythm spectral power of $55.49 \mu\text{V}^2/\text{Hz}$ [26.98; 109.54]; beta-1 rhythm $10.08 \mu\text{V}^2/\text{Hz}$ [7.45; 6.26]; and beta-2 rhythm $11.97 \mu\text{V}^2/\text{Hz}$ [9.37; 16.86].

In the control group, the median spectral power values were alpha rhythm $51.31 \mu\text{V}^2/\text{Hz}$ [30.37; 93.05]; beta-1 rhythm: $8.92 \mu\text{V}^2/\text{Hz}$ [5.64; 13.43]; beta-2 rhythm: $7.85 \mu\text{V}^2/\text{Hz}$ [5.03; 11.19].

A statistically significant increase in spectral power was found in the low-frequency beta-1 range and the high-frequency beta-2 range in the main group compared with the control group ($p < 0.01$). Significant differences were also found in the beta-1/beta-2 ratio ($p < 0.01$), due to increased power in the beta-2 range in the main group (Figure 2). No statistically significant intergroup differences were found for alpha rhythm spectral power.

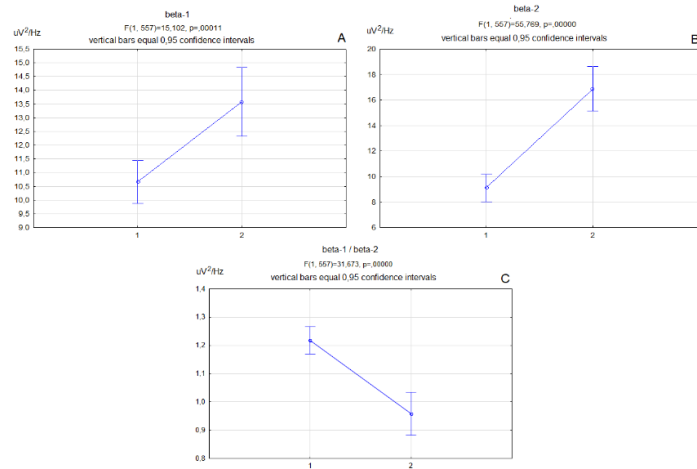


Figure 2: Results of one-way ANOVA of the spectral power of the background EEG recording rhythms in the control (1) and main (2) groups: A – in the beta-1 range; B – in the beta-2 range; C – the ratio of the beta-1/beta-2 ranges.

Discussion

Under normal conditions, the beta rhythm reflects a state of active wakefulness. The EEG beta-2 range (>20 Hz) is typically associated with high cortical activation and intense cognitive activity. However, in our study, electronic cigarette users demonstrated increased beta-range spectral power even during passive wakefulness. Moreover, the increase in beta rhythm spectral power was mainly due to the high-frequency beta-2 range. The high-frequency component is typically associated with irritative phenomena in brain structures. According to the literature, pathological increases in beta-range spectral power may indicate neurotoxic effects leading to disruption of the balance between excitation and inhibition. Additionally, increased beta spectral power may occur under conditions of stress, anxiety, or emotional arousal [2].

Based on available literature, both specific and nonspecific mechanisms may contribute to disruption of the excitation–inhibition balance in neuronal cells and neural networks in electronic cigarette users.

Specific mechanisms involve neurotransmitter imbalance and/or altered receptor sensitivity. In nicotine-containing electronic cigarettes, the leading mechanism is thought to be the direct effect of nicotine on nicotinic acetylcholine receptors (nAChRs) [3]. These receptors, located in the neocortex, amygdala, hippocampus, and thalamus, activate NMDA receptors and increase neuronal excitability. Nicotine acts on nAChRs expressed in dopaminergic neurons of the ventral tegmental area, as well as on local GABAergic interneurons and afferent terminals, thereby altering the excitability of dopaminergic neurons through both direct effects and changes in local GABAergic and glutamatergic transmission [4,5]. Such neuroadaptation in neurotransmitter systems leads to changes in neuronal excitability, synaptic plasticity, and neurotransmitter release, thereby modulating susceptibility to seizures [6].

Nonspecific mechanisms of increased neuronal excitability may include membrane damage and dysfunction of ion channels caused by pyrolysis products of e-liquids and other toxic agents, including heavy metal nanoparticles. These factors enhance oxidative stress and spontaneous lipid peroxidation [7].

Propylene glycol (PG) and vegetable glycerin (VG), two other key components of electronic cigarettes, may act

synergistically in promoting seizure development. Inhalation of toxic doses of propylene glycol may cause hyperosmolar metabolic acidosis, while the osmotic properties of glycerin may lead to dehydration, also increasing seizure risk [1].

Inhalation of vapors containing flavoring agents and other chemicals present in e-liquids may exert synergistic neurotoxic effects, increase neuronal excitability and disrupt synaptic transmission. Studies have shown that flavored electronic cigarettes produce aerosols containing reactive oxygen species, which contribute to oxidative stress-induced cellular damage [8]. Emerging evidence indicates that oxidative stress may influence mechanisms underlying seizures, epilepsy, and epileptogenesis [9].

Numerous metals have been identified in e-liquids and their aerosols [10,11]. The concentrations of these metals—except cadmium—may exceed those found in traditional cigarettes. These metals can originate from the atomization chamber or other components of the device and may enter the aerosol. Inhalation of such metals can disrupt normal levels of chromium and nickel in the body, leading to toxicity affecting multiple organs. Copper is associated with mitochondrial oxidative stress and DNA fragmentation [10,11].

Normally, the blood–brain barrier (BBB) restricts the transport of potentially toxic substances from the bloodstream into the brain. Increased BBB permeability associated with electronic cigarette use may result from low-grade systemic inflammation, including neuroinflammation [12]. Small cerebral vessels interact with neurons through glial cells—particularly astrocytes—and are functionally closely interconnected [13]. Increased oxidative stress, decreased expression of the tight junction protein zonula occludens-1, and increased expression of vascular adhesion molecules have been demonstrated in an in vitro BBB model using mouse brain microvascular endothelial cells [14]. Activation of microglia in the hippocampus has also been observed in offspring of mice exposed to electronic cigarette aerosols in vivo [15].

Conclusions

Disturbances in the balance between excitation and inhibition in brain neurons are reflected in EEG recordings as irritative changes or epileptiform activity. However, the presence of epileptiform discharges on EEG does not necessarily correspond to clinical manifestations such as partial or generalized seizures. In our cohort, all examined individuals exhibited irritative changes in brain bioelectrical activity of varying severity, ranging from low-amplitude local beta bursts to generalized bursts of polymorphic sharp waves. Nevertheless, electronic cigarette users in the main group reported no neurological complaints. These findings suggest that before the neurotoxic effects of electronic cigarettes manifest clinically as seizures, there may exist a latent subclinical period of neurotoxicity characterized by electrophysiological correlates on EEG in the form of irritative or epileptiform alterations in brain bioelectrical activity.

Availability of data and materials

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Competing interests

The authors declare no conflicts of interest

Ethics statement

All participants gave written informed consent before any study procedures. The protocol received prior approval from the Local Ethics Committee of St. Petersburg Medical and Social Institute (19.12.2025).

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