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NEUROVEGETATIVE MECHANISMS OF THE DEVELOPMENT OF NONEPILEPTIC SEIZURES (SYNCOPES) IN ADOLESCENCE AND THEIR DIFFERENTIAL DIAGNOSIS

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Abstract: Non-epileptic paroxysms, particularly syncope, represent a major diagnostic challenge in pediatric and adolescent neurology. During adolescence, intensive neuroendocrine remodeling combined with autonomic nervous system instability often manifests as transient loss of consciousness. Misdiagnosis frequently leads to inappropriate antiepileptic drug prescription.

This clinical study aimed to investigate the neurovegetative mechanisms underlying non-epileptic syncopal episodes in adolescents and to identify distinct autonomic and electrophysiological criteria for precise differential diagnosis.

A prospective, controlled cohort study evaluated adolescents presenting with recurrent transient loss of consciousness. Patients with confirmed organic heart diseases or primary epilepsy were excluded. Evaluation methods focused on multi-system autonomic assessments, including the head-up tilt table test, prolonged video - EEG monitoring to capture paroxysmal events, and spectral analysis of heart rate variability to assess sympathetic-parasympathetic balance.

Keywords: adolescents, non-epileptic paroxysms, syncope, neurovegetative regulation, tilt table test, heart rate variability, differential diagnosis.

INTRODUCTION The period of adolescence is characterized by rapid somatic growth of the body, hormonal restructuring and active integration of neuroendocrine systems. During this transitional period, a temporary imbalance in the control mechanisms between the central and peripheral nervous systems creates the basis for the development of various paroxysmal states. Among adolescents, sudden short-term fainting spells - syncopes (states resulting from hypoxia and a temporary decrease in cerebral perfusion) constitute the most common group of non-epileptic seizures in neurological practice. The relevance of this problem is determined by the fact that fainting episodes cause social maladaptation and cause a high level of anxiety in the adolescent and his parents.

For a long time, syncope in adolescence was considered a simple functional condition. However, modern neurocardiological and electrophysiological studies show that syncope is based on a complex pathophysiological chain - an imbalance of neurotransmitters, a decrease in the sensitivity of baroreceptors and excessive reactivity of the autonomic nervous system. The international literature has repeatedly noted that the clinical similarity of syncope developing against the background of autonomic dysfunction with paroxysmal epileptic seizures (the presence of myoclonic contractions, elements of tonic tension) leads to diagnostic errors. Unjustified prescription of antiepileptic drugs (hyperdiagnosis) is a serious toxic load for the adolescent body.

Although cardiogenic and neurogenic types of syncope have been studied separately in existing scientific works, the synchronous chain of vegetative homeostasis disorders and changes in central bioelectric activity, which are characteristic of adolescence, has not been fully elucidated. In clinical practice, there is a lack of clear algorithmic approaches to which neurophysiological and

autonomic biomarkers are leading in the differential diagnosis of non-epileptic seizures. In many local hospitals, cases are limited to only standard short-term video monitoring (EEG) examinations, which is insufficient to reveal the true nature of the seizure.

The aim of this study is to identify the neurovegetative mechanisms of the development of non-epileptic seizures (syncope) in adolescence and to develop clinical-electrophysiological and autonomic diagnostic criteria that allow them to be distinguished from epileptic paroxysms.

Research objectives:

a) comprehensive assessment of the initial state of the autonomic nervous system (tone, reactivity) in adolescents with a history of fainting episodes;

b) analysis of synchronous changes in brain bioelectrical activity and heart rhythm parameters during attacks using video-EEG and cardiomonitoring;

c) determination of the contribution of neurogenic and vascular factors in the development of syncope using orthostatic tests (Tilt-test);

d) formulation of clinical recommendations for differential diagnosis of syncope in adolescents and reduction of hyperdiagnosis.

The scientific novelty of the study is that it was proven for the first time, through the integration of electrophysiological and cardiohemodynamic tests, that the formation of non-epileptic seizures in adolescents is not a central neuronal disorder, but a temporary desynchronization of the autonomic baroreflex circuit that controls peripheral vascular tone. It was argued that the flattening effect on the EEG during the seizure is ischemic, not epileptic in nature.

The results obtained allow for the widespread introduction of pathogenetic correction methods aimed at correcting patient referral and restoring vegetative homeostasis in pediatric neurology and pediatric practice, avoiding expensive and unnecessary drug therapy. The developed diagnostic algorithm increases the quality of differential diagnostics in clinics.

METHODS Study design and patient selection unification. The study was organized on a clinical basis and prospectively studied the condition of a group of adolescents with syncope paroxysms. In order to exclude subjective approaches in the formation of the clinical cohort, examinations were carried out on the basis of fully standardized protocols. Letters of consent were obtained from the parents of the patients in accordance with the rules of bioethics.

The concept of inclusion and exclusion criteria. The study group included adolescents aged 12 to 17 years who had experienced at least several episodes of brief loss of consciousness in the past few months. The diagnosis was made based on the criteria for cardiogenic and neurogenic paroxysms. Patients with structural brain diseases (tumors, complications of severe trauma), organic heart defects (valve pathology, pronounced cardiomyopathies), and primary status epilepticus were excluded from the group to ensure the safety of the research object and the impartiality of the results.

Neurovegetative and Electrophysiological Testing Protocol. *Heart rate variability analysis:* To assess the balance of the sympathetic and parasympathetic divisions of the autonomic nervous system in patients, the cardiographic frequency spectrum was analyzed at rest. The spectral

power indicators, which indicate the influence of sympathetic tone and the vagus nerve, were compared.

Passive Orthostatic Test: To test vascular regulation, patients were placed in a vertical position at a certain angle on a special rotating table. During the test, arterial blood pressure and heart rate were dynamically measured every minute. The goal was to assess the vascular response to orthostatic loading as a neurogenic reflex.

Long-term Video-EEG monitoring: Multi-channel bioelectrical activity was recorded to visualize the state of the central nervous system during clinical attacks. Functional loads (hyperventilation and photostimulation) were applied during the observation.

RESULTS

As a result of clinical and instrumental observations, it was found that the majority of paroxysmal fainting in adolescents is not epileptic in nature, but depends on neurotransmitters and vascular dysfunction. In most cases, it turned out that attacks were provoked by standing for a long time, staying in a hot or humid room, emotional stress, or sudden changes in body position (orthostasis). In the pre-seizure period (prodromal phase), a complex of obvious autonomic symptoms, such as darkening of the forehead, ringing in the ears, and profuse sweating, was observed in adolescents, which is an atypical criterion for primary epileptic paroxysms.

During the tilt-test, two main variants of the vascular reactions of adolescents were observed:

1. Vasodepressor variant: A sharp decrease in arterial blood pressure and loss of vascular resistance.

2. Cardioinhibitory variant: A sudden slowdown in heart rate (bradycardia) or a short-term state of asystole.

Heart rate variability analyses showed an overactive parasympathetic nervous system (vagus effect) in adolescents. It was confirmed that insufficient activation of the sympathetic system in response to orthostatic loading leads to reflex hypotension and cerebral hypoxia.

No sharp waves, spike-wave complexes, or foci of epileptiform activity were detected in the video-EEG recorded during the seizure. At the moment of loss of consciousness, an initial increase in general slow-wave activity (delta and theta rhythms) was observed in the cerebral cortex, followed by a short-term decrease in bioelectric activity (flat EEG). These changes reflect a hypoxic state resulting from a temporary decrease in cerebral perfusion, and upon recovery of consciousness, the EEG pattern completely returned to normal.

Due to the complex diagnostics carried out, this diagnosis was completely refuted in several adolescents who were previously under observation with suspicion of epilepsy. It was proved that the mild myoclonus (tremors) observed during seizures were not epileptic in nature, but were a transient response of cortical neurons to deep brain hypoxia. During the examinations, no complications detrimental to the health of adolescents were noted.

DISCUSSION

The results of our study further strengthen the international scientific view on the neurovegetative nature of non-epileptic seizures, especially syncope, in adolescence. Rapid growth of body parts in adolescence outpaces the rate of innervation of the vascular system, which leads to instability of general and cerebral hemodynamics.

The most common diagnostic error in clinical practice is the misdiagnosis of hypoxic myoclonus (muscle contractions) during syncope as an epileptic seizure. Our neurophysiological data (recording the moment of paroxysm during video-EEG monitoring) showed that in non-epileptic syncope, EEG changes are only in the form of diffuse slowing. This is a universal response of brain tissue to ischemia, which is fundamentally different from the synchronous neuronal discharges in epilepsy.

Heart rate variability and the Tilt-test results show that the pathophysiological basis of syncope in adolescents is a temporary deficiency of baroreflex regulation. In a healthy organism, when the body position changes to vertical, the sympathetic system should reflexively activate and maintain vascular tone. In adolescents, however, there is a disruption in this chain or, as a result of hyperreactivity of the vagus nerve, the vessels dilate and blood accumulates in the lower extremities (venous pooling). As a result, the volume of blood returning to the heart decreases, and the brain experiences short-term oxygen starvation.

This study highlights the need for clinical neurophysiology to integrate autonomic testing beyond standard EEG in the assessment of syncope. This approach is the only way to reduce overdiagnosis and protect adolescents from inappropriate drug therapy.

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