Clinic-eeg correlation somatogenous of conditioned febrile seizures in children

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Abstract: The article describes the outcomes of febrile seizures in children. The main provoking factors for the development of febrile seizures are acute respiratory viral infection, pneumonia and acute intestinal infection, and concomitant diseases (rickets of the II degree, allergic diseases, helminth invasion, I degree of malnutrition, diffuse goiter of the II degree, chronic diseases of the upper respiratory tract and urinary tract systems) create favorable conditions for the development of afebrile seizures. With febrile seizures, mainly generalized tonic-clonic seizures were observed, which can serve as a criterion for the development of afebrile seizures.

Keywords: febrile convulsions, children, seizure, epilepsy.

Relevance: Febrile seizures (FS), being the most common paroxysmal conditions among children from the age of 6 month, up to 5-7 years, belong to the group of diseases that do not require a mandatory diagnosis of epilepsy [11,12]. According to the draft classification of 2001, the term febrile seizures was replaced by febrile seizures, since in the clinical picture of this condition not only convulsive, but also convulsive paroxysms can be observed. Thus, FS is defined as an episode of epileptic seizures that occur in preschool children with hyperthermia that is not associated with neuroinfection, and refers to benign, age-dependent, genetically determined conditions in which the brain is susceptible to epileptic seizures that occur in response to high temperature.

In 10-30% of cases, AF is noted in the debut of many epileptic syndromes and epilepsies [1,7,12], so they have a history of 10-45% of patients with idiopathic focal epilepsy, and in 7% of children with Roland epilepsy, relatives are identified having a history of AF [1,7,10,12]. C.P. Panayiotopoulos (2005) notes that a maximum AF frequency of up to 30% of cases is observed in the debut of benign occipital epilepsy [9]. In recent years, AF has been described that occurs in the framework of idiopathic focal epilepsy of infancy [7,11,12]. The frequency of AF in patients with benign myoclonic epilepsy of infancy reaches 27%, is less common in the history of patients with the following forms of idiopathic generalized epilepsy: Doose syndrome - 11%, juvenile abscess epilepsy - 12%, Tassinari syndrome - 15%, epilepsy with isolated generalized convulsive 15%, juvenile myoclonic epilepsy - in isolated cases [7,11,12]. It has been noted that an early predictor of the transformation of AF into idiopathic focal epilepsy may be the appearance on the EEG more often when recording benign childhood epileptiform patterns in sleep, with these forms of epilepsy there are exclusively typical AFs that are often associated with sleep [7,12]. At present, genetic, social, exo, and endogenous factors of febrile seizures have been studied [6–9].

The hypothesis that a genetically determined predisposition to convulsive states is a consequence of a generalized defect in the metabolism of catecholamines in the central nervous system is widely recognized [3]. In the early eighties of the XX century, the American neurophysiologist J. Zabara hypothesized that stimulation of the vagus nerve can prevent the development of epileptic seizures [5]. Later, in the study of patients with epilepsy in the interictal period, the following autonomic...
disorders were revealed: an increase in parasympathetic activity in lesions of the right hemisphere and, accordingly, in sympathetic activity in the left hemisphere focus [6,12]. In addition, as a result of studies of children with febrile seizures, it was found that increased excretion of amines in children with febrile seizures is a consequence of ergotropic (sympathetic-adrenal) hypertonicity, which is based on genetically caused or resulting as a result of perinatal pathology dysfunction of higher suprasegmental vegetative centers, and above all, the structures of the limbic-reticular complex [6]. As for the identification of changes in EEG in children with AF, they are non-specific. Perhaps a study of the state of the ANS in children with AF and comparing them with the EEG results will help to identify the relationship of these disorders, showing the unidirectional nature of current processes in AF and epilepsy.

The purpose of the study: The aim of our work is a comparative description of the clinical manifestations and results of EEG studies of somatogenically caused febrile seizures.

**Materials and methods:** 20 patients with febrile seizures were examined, of which 10 children had simple febrile seizures, and 10 children had complex AF, from 6 months to 3 years of age. The examination plan for each child consisted of the collection and analysis of clinical and medical history of the disease and life, and the study of neurological status. The EEG study was carried out in conditions of relative rest, background recording was recorded in a sitting position, for 5 minutes. Registration and assessment of the bioelectric activity of the brain was carried out in a state of wakefulness and physiological daytime sleep. EEG recording was carried out on a 16-channel computer electroencephalograph under the name Neuron-Spectrum-2. To register the EEG, a 10-20 electrode arrangement was used, including 21 cup electrodes with a ground electrode in the center of the anteroposterior region. The alphanumeric and numeric designations of the electrodes were in accordance with the international 10-20 layout. Allocation of electric potentials was carried out in a monopolar way with an averaged common. The advantage of this system is the less laborious process of applying electrodes with sufficient info. The average duration of EEG registration in each patient was standard and the possibility of conversion to any bipolar was 40-60 minutes.

**Results of the study:** in 16 children (80%), a burdened obstetric and pediatric history was revealed, namely, the pregnancy proceeded against the background of iron deficiency anemia (20%; 4 patients) suffered from SARS during pregnancy (25%; 5 patients), threats of abortion (5%; 1 patient), hypoxic-ischemic encephalopathy (20%; 4 patients) and other pathologies (10%; 2 patients). The typical age period of the onset and development of febrile seizures in children was 10-28 months. The temperature increase at the time of febrile seizures ranged from 37.5 to 41 °C. The causes of fever that caused febrile seizures were ARVI (60%; 12 people), lacunar angina (25%; 5 people), pneumonia (15%; 3 people). An analysis of the hereditary family background in 2 children revealed the presence of relatives suffering from febrile seizures.

**References**


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