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T. P. Kalashnikova, G. V. Anisimov, N. A. Savel'eva
Perm, Russia

EPILEPTIFORM ACTIVITY AND SPEECH DYSONTOGENESIS IN PRESCHOOL CHILDREN

Abstract. The article demonstrates the correlation between lateralization of epileptiform activity and the nature of speech disorders in preschool children based on the comparison of clinical types of speech dysontogenesis with localization of benign epileptiform childhood patterns (BECP) identified by electroencephalography. In 55% of cases, children with articulatory developmental dyspraxia (ADD) have BECP with localization in the left parietal-central-temporal area. In patients with motor dyspraxia of development (MDD), BECPs were identified mostly in the right occipital-temporal area in 71% of cases. The formation of occipital-temporal interzonal connections of the right hemisphere forms the basis for speech acquisition. These connections determine the development of the non-verbal auditory gnosis which is manifested by correlation of the visual image of an object with the sound characteristic of it.

The right hemisphere dysfunction has a more global character and distorts the process of lateralization in the speech ontogenesis. In the long run, this fact determines the severity of speech disorders in the form of motor developmental dysphasia. The articulatory developmental dyspraxia is based on the kinesthetic and dynamic articulatory dyspraxia associated with the dysfunction of the parietal and prefrontal areas of the brain. As a result, the patients do not have the opportunity to reproduce the standard articulatory positions, and the mechanism of switching from one articulatory position to another in the process of word reproduction is affected.

Thus, some children with speech impairments develop a peculiar dysontogenetic syndrome which is clinically manifested as MDD or ADD, and its neurophysiological pattern is BECP.

Keywords: preschool children; speech dysontogenesis; children with speech disorders; speech disorders; motor dysphasia; articulation dyspraxia; electroencephalography, electroencephalogram; epileptiform activity.

About the author: Kalashnikova Tat'yana Pavlovna, Doctor of Medicine, Professor.
Place of employment: The First Medical Pedagogical Centre “Lingva Bona” (Perm, Russia).
About the author: Anisimov Grigoriy Vladimirovich, Candidate of Medicine.
Place of employment: The First Medical Pedagogical Centre “Lingva Bona” (Perm, Russia).
About the author: Savel'eva Natal'ya Aleksandrovna, Candidate of Medicine.
Place of employment: The First Medical Pedagogical Centre “Lingva Bona” (Perm, Russia).

Introduction. Child speech function is vulnerable, and its impairment has a number of significant consequences [10; 9; 5; 3]. In spite of the information available about speech function, there are still certain questions pertaining to etiopathogenesis of speech disorders, their correction and prevention. The electroencephalogram (EEG) of children with speech underdevelopment registers epileptiform activity, in particular, benign epileptiform childhood patterns (BECP) [14; 16; 15; 4]. Benign epileptiform childhood patterns in EEG were first described by EEG technician Yvette Gastaut in 1952 under the name "functional adhesions". BECP is a five point electric dipole including a sharp wave and a slow wave. By their morphology the complexes resemble ORS spikes on the electroencephalogram (see: Fig.).

Benign epileptiform childhood patterns in EEG. The question about correlation of BECP and speech disorders is urgent enough. At present, BECP is considered to be a specific electroencephalographic marker of functional immaturity of the brain. The pattern is not specific for epilepsy.

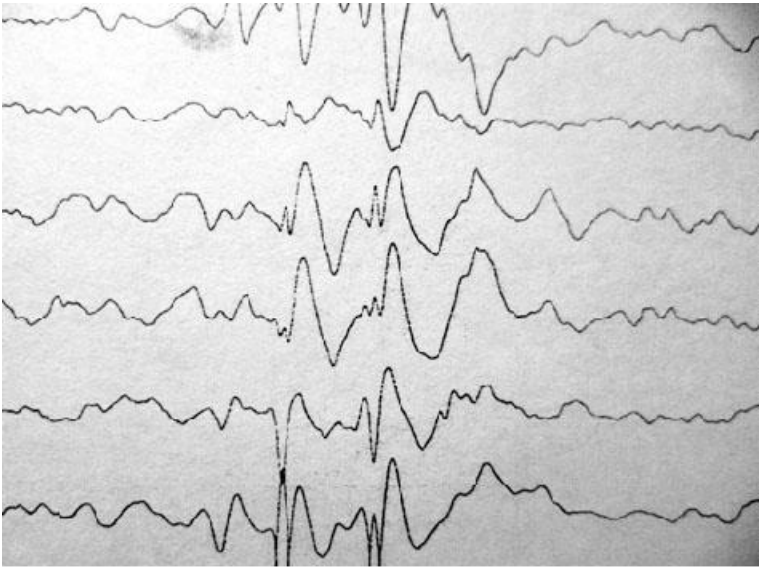

Fig. Patient A. Age 5. Diagnosis – motor developmental dysphasia

K. Yu. Mukhin and his co-authors single out in their work a number of

conditions associated with BECP [8]. Apart from idiopathic focal epileptic forms here belong tics, enuresis, attention deficit hyperactivity disorder (ADHD) and stammering. The frequency of diagnosing BECP in children with ADHD is 25%, headaches caused by strain – 23%, speech disorders – 18%, enuresis – 5%, and tic – 3%. In addition, 18% of children with ADHD in EEG turned out to be clinically healthy. H. Doose and W. K. Baier argued in their work published in 1989 [12] that BECPs are regulated by the autosomal dominant gene with age-dependent penetrability and variable expressiveness [13]. The emergence of epileptiform activity is observed during the critical period of the child's development – from 3 to 5 years of age [11; 1].

The aim of the given research was to compare the clinical variants of speech dysontogenesis with localization of BECPs in EEG in preschool children.

Materials and methods. We have analyzed clinico-anamnestic and electroencephalographic data of 39 children aged 3-5. All children were divided into two clinical groups in accordance with the character of speech dysontogenesis consistent with ICD-10. The first observation group included patients with articulatory developmental dyspraxia – ADD (18 children). The second group united children with motor developmental dysphasia – MDD (21 children). All children under observation had BECPs in EEG.

Articulatory developmental dyspraxia is interpreted as selective trivial but stable mispronunciation with no total polimorphemic violation of pronunciation, and with changes in tone and muscle contraction capability; it may be accompanied by mild disorders of innervation of articulation organs [6].

In accordance with ICD-10, *specific speech articulation disorder* (F-80.0) may be considered to be synonymous to the term *articulatory developmental dyspraxia*. This disorder is based on impairment of differentiation of fine motor kinesthetic positions of the tongue, palate and lips – i.e. kinesthetic dyspraxia [7].

Motor developmental dyspraxia is defined as a specific developmental speech disorder meaning that the child's ability to use spoken language is on the level which is lower than the one corresponding to his age with various degrees of oral comprehension development. Criteria for motor developmental dyspraxia diagnostics may include early emergence, consistency with absence of the period of typical development and tendency to progressive improvement; late formation of phrase speech (after 3 years of age); reduction of active vocabulary; violation of word syllabic structure (shortening or transposition of syllables) and the grammatical structure of speech; domination of non-verbal means of

communication [5; 3; 7].

All the children were subject to complex uniform observation including clinical and neuropsychological observation with assessment of higher cortical functions, and EEG in the state of quiet wake with visual and quantitative assessment.

EEG was registered via the method of monopolar stimulation in accordance with the international system "10 – 20" on a 16 channel electroencephalograph "Neuron-Specter 4/ ВП". Ear electrodes were used as the referent ones. The following tests were performed: opening and closing eyes, photostimulation and hyperventilation. Clinical assessment of EEG included analysis of background rhythm, zonal differences, activation response, hyperventilation and mastering rhythm of a flashing light, diagnosis of pathological types of activity.

The presence of disorder of psychological development and sensory dysfunction (poor hearing or visual acuity) served as criteria for exclusion. The results obtained were subject to statistical procession.

Analysis of localization of BECP in the children of groups 1 and 2 revealed different localization with reference to brain sides.

BECP with localization in the left parietal and central temporal region was registered in children with ADD in 55% of cases. 71% of patients with MDD showed BECP with accent on the right temporo-oscipital region.

Taking into account the principles of speech development and hemispheric asymmetry, the results obtained have patho-genetic significance. Development of the speech functional system begins with formation of temporo-oscipital connections of the right side of the brain which lie at the basis of non-verbal hearing gnosis manifested by the connection of the visual image of an object with the sound associated with it [2]. Non-verbal hearing gnosis is the basic neuropsychological syndrome for development of other stages of speech formation – verbal hearing gnosis, phonemic analysis, etc. The right hemisphere dysfunction which is more closely connected with subcortical brain structures has a global character and distorts the process of lateralization in speech development and causes difficulty of transformation of images into word symbols. In the long run, it determines the severity of speech disorder in the form of motor developmental dysphasia.

Kinesthetic and dynamic articulatory dyspraxia connected with dysfunction of parietal and prefrontal brain regions and with malformation of interzonal connection of fronto-temporal and parietal-temporal regions of the left hemisphere lies at the basis of articulatory developmental dyspraxia. As a

result, patients are not able to reproduce model articulatory positions necessary for correct pronunciation of the sound. What is more, the mechanism of switching articulatory positions in the process of reproduction of a word is also damaged. BECP in the right side in children with ADD is accompanied by the above described neuropsychological syndromes.

Thus, a certain number of children with speech underdevelopment demonstrate a specific dysontogenetic syndrome which is clinically manifested as MDD or ADD, and its neurophysiological pattern is BECP. And BECP lateralization is coordinated with the character of speech disorder. For practical purposes it is important to stress that the presence of BECP is not a contraindication to rehabilitation work and massage, including the logopedic one.

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