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# Study of Local EEG Specificities in Children with Mental Development Disorders Using Independent Component Analysis

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**Abstract**—Baseline EEGs in the frequency range of 3–13 Hz in children with mental disorders of perinatal origin during wakefulness with the eyes open were analyzed using independent component analysis. In cases of severe mental retardation, a significant increase in the power density spectra of the θ band was revealed in the left-sided frontotemporal and right-sided temporal cortices, which allows us to consider these regions to be putative sources of slow activity and markers for a lesion or immaturity in the fronto-thalamic system, as well as for the temporal areas responsible for the auditory analysis and synthesis of speech signals and the integration of audio-visual information.

*Keywords*: EEG, independent component analysis (ICA), perinatal pathology, mental development disorders

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Changes in children's mental development due to a perinatal pathology in the central nervous system (CNS) are among the neurophysiological models allowing us to understand those cerebral mechanisms that are responsible for disorders (dysfunctions, damages, and immaturities) in higher mental processes. However, the specific features in the behavior of retarded children, such as reduced or absent responses to instructions during investigations, disinhibition, and inadequate emotional reactions, create considerable difficulties for the researchers' attempts to obtain a sufficiently prolonged artifact-free EEG recording during waking, particularly, with the eyes closed. Due to this fact, a search for EEG markers for developmental disorders in natural conditions of wakefulness, i.e., with the eyes open, is important.

In recent years, new possibilities for the understanding of functional significance of the brain bioelectrical activity have been offered by independent component analysis (ICA), which belongs to a class of methods for separating signals from sources which cannot be directly recorded using available detectors [1, 2]. The method is based on the assumption about statistical independence of temporal series corresponding to signals from the "generators" of components. The objective of its application to the EEG analysis is in separating independent signals generated by different brain "sources."

In this study, we used the ICA method to estimate the differences in the EEG spectral power in the frequency range of 3–13 Hz in children with different degrees of retardation in mental development during wakefulness with their eyes open.

## METHODS

We investigated 108 children at ages of 3–8 years with complications caused by perinatal damages to the CNS. The groups were matched with respect to the representation of preschool and school children to reduce the effect of age-related factors (the mean values across the groups did not reliably differ by Student's *t* test). According to the results of the psychologo-logopedic test for the development of higher mental functions and speech [3], the children were conventionally divided into four groups.

The first group was control, it was composed of 17 children studying at preschool and school educational institutions, according to their age-group programs of education (first grade), at a mean age of 5 ± 2 years.

Children with mental development disorders were divided into the following three subgroups.

Group 2Mi included 39 children with a mild degree of retardation in development (rate delays in speech development, general speech underdevelopment, mild dysarthria and other disorders in expressive and impressive speech unaccompanied by delays in the development of other mental processes), at a mean age of 5.5 ± 2 years.

Group 2Mo included 35 children with a moderate degree of retardation in development who studied at specialized kindergarten and schools (classes) of corrections specialization. These 35 children had delays in mental development, nonsevere forms of autistic spectrum disorders with combined disorders in the development of higher mental processes (visuospatial perception, attention, memory, thinking, impressive and expressive speech, communicative functions, etc.); the mean age was $5.2 \pm 3$ years.

Group 2Se included 17 children with a severe degree of developmental retardation with underdeveloped age-specific self-help skills (eating, dressing, toilet use, etc.) and expressed learning difficulties (a weak reaction to an address in voice, imitation and utilization behavior, a low cognitive interest, etc.); the mean age was $4.7 \pm 2$ years.

EEGs were recorded in a wakeful state with the eyes open (for about 2 min) using a Mizar electroencephalograph and the WinEEG software. We used 19 silver chlorine bridge electrodes localized on the child's scalp according to the International 10–20 System. Reference electrodes were placed on the earlobes or the forehead. The resistance of electrodes did not exceed 5 kΩ. The sampling rate of EEG was 250 Hz. The parameters of high frequency filter and low frequency filter were, respectively, 0.53 Hz and 50 Hz. The common average reference (*Av*) monopolar scheme was used in the analysis.

The artifacts of winks and horizontal eye movements were corrected using ICA [4]. The EEG fragments containing high-amplitude artifacts of quick oscillations at frequencies of 25–35 Hz and amplitude exceeding 40 μV were also excluded from the analysis; potentials were visually analyzed, and spikes of potential exceeding 150 μV were deleted. After the described filtration, the $F_8$ and $F_7$ leads were excluded, using ICA, from subsequent analysis because of the considerable repression of activity in these channels (caused by artifacts due to electrode movements resulting from retarded children's specific behavior, such as motor anxiety and crying).

Fragments containing more than eight epochs of artifact-free recording (over 30 s) were used for quantitative analysis of the baseline EEG. Individual EEGs in all groups were pooled when separated into independent components. The analysis resulted in 17 topographies of independent EEG components, which were further used for sorting out individual EEG components. This allowed us to calculate the spectrum of EEG components individually for each case of localization of the sorted-out activity, thus reducing their mutual effects.

To determine the localization of hypothetical sources of independent EEG components, we used the method of standardized low resolution tomography (sLORETA) [5]. This method makes it possible to determine, on the basis of the component's topography, its putative sources and Brodmann's areas where it is supposedly localized. According to the localization data, all the components were associated with EEG leads of the International 10–20 System.

To normalize the distributions, the mean spectral power values for the rhythms were logarithmized. The logarithms of the spectral power for independent components were compared in the low-frequency (3–7 Hz) and α activity (8–13 Hz) ranges. To determine the statistically significant differences between groups of children with different level of developmental retardation, we used the two-factor ANOVA. To qualitatively evaluate the intergroup differences in the EEG spectral power, we calculated Cohen's effect size.

## RESULTS

Significant differences between all investigated groups of children have been distinguished in the slow-wave range at the fronto-temporal leads $Fp_1$, $F_3$, $T_3$, $T_4$ and $T_6$ using ANOVA: $F(15, 276.5) = 1.98$, $\varepsilon = 0.75$, $p < 0.016$. No changes of this kind have been detected in the α range with the eyes open: $F(15, 276.5) = 1.28$; $\varepsilon = 0.83$, $p < 0.21$.

Qualitative analysis of the spectral power for independent components in slow frequencies (using Cohen's effect) has revealed a gradual growth of differences in correspondence with the degree of severity in mental development disorders (Table 1). In particular, the analysis has revealed no differences at any EEG lead between the first group of children without development disorders and the 2Mi subgroup. At the same time, the comparison of the children of subgroup 2Se (with the severest disorders) with all the remaining groups has revealed differences practically at every lead (Table 1, Figs. 1, 2, 3).

We also marked spectral peaks in the EEG components where significant spectral power differences in slow rhythms have been revealed (Fig. 3): at $Fp_1$ at the frequency of 5.6 Hz (Fig. 1) and at $F_3$ and $T_3$ at the frequency of 5 Hz (Figs. 2, 3).

Analysis of the cumulative spectral power for independent components (across all frequency ranges) demonstrated the trend towards interregional differences (central leads $Cz$, $C_3$ and $C_4$ were excluded from the analysis) (Table 2). In particular, in the first group of children (without development delays), regional differences in this parameter between the anterior-central and posterior regions were practically unexpressed. Group 2 (with mental development disorders) demonstrated the emergence of some accent on slow components in the temporo-parietal and occipital regions (Brodmann's areas 21–22, associative areas 19, 37, 39, 40, 42, etc.). The degree of expression decreases in children with more severe forms of developmental delays.

**Table 1.** Intergroup differences in the spectral power of independent components in the slow-frequency range as estimated using Cohen's effect

| EEG leads | Cohen's effect size for the spectral power logarithm | | | | | |
|---|---|---|---|---|---|---|
| | Compared groups | | | | | |
| | 1–2Mi | 1–2Mo | 2Mi–2Mo | 2Mo–2Se | 2Mi–2Se | 1–2Se |
| $Fp_1$** | 0.09 | 0.85* | 0.62* | 0.43 | 1.11* | 1.44* |
| $Fp_2$ | 0.25 | 0.77* | 0.53* | 0.39 | 1.00* | 1.24* |
| $F_3$** | –0.12 | 0.43 | 0.47 | 0.82* | 1.31* | 1.41* |
| $Fz$ | –0.30 | 0.41 | 0.41 | 0.26 | 0.69* | 0.87* |
| $F_4$ | 0.02 | 0.41 | 0.32 | 0.65* | 1.11* | 1.34* |
| $T_3$** | –0.12 | 0.51* | 0.58* | 0.78* | 1.48* | 1.49* |
| $C_3$ | 0.21 | 0.33 | 0.12 | 0.45 | 0.71* | 0.88* |
| $Cz$ | –0.07 | 0.06 | 0.13 | 0.40 | 0.64* | 0.56* |
| $C_4$ | 0.28 | 0.83* | 0.66* | 0.37 | 1.34* | 1.38* |
| $T_4$** | 0.27 | 0.86* | 0.47 | 0.61* | 1.25* | 1.84* |
| $T_5$ | 0.17 | 0.43 | 0.29 | 0.44 | 0.72* | 0.82* |
| $P_3$ | –0.22 | 0.28 | 0.37 | 0.73* | 1.19* | 1.40* |
| $Pz$ | –0.09 | 0.21 | 0.25 | 0.71* | 1.14* | 1.30* |
| $P_4$ | –0.35 | 0.19 | 0.79* | 0.43 | 1.46* | 0.65* |
| $T_6$** | 0.31 | 0.87* | 0.45 | 0.55* | 1.13* | 1.72* |
| $O_1$ | 0.26 | 0.54* | 0.30 | 0.62* | 1.06* | 1.23* |
| $O_2$ | 0.31 | 0.55* | 0.19 | 0.38 | 0.54* | 1.00* |

* The effect size at a level of $d > 0.5$; ** significant intergroup differences.

**Table 2.** Regional specificities of the spectral power of independent components at rest with the eyes open (% of the common EEG spectrum)

| Cortical regions (EEG leads) | Cumulative spectral power values | | | |
|---|---|---|---|---|
| | groups of children | | | |
| | 1 | 2Mi | 2Mo | 2Se |
| Frontotemporal ($Fp_1$, $Fp_2$, $F_3$, $F_4$, $Fz$, $T_3$, $T_4$) | 41.6 | 31.2 | 30.2 | 35 |
| Parieto-temporal, occipital ($T_5$, $T_6$, $P_3$, $P_4$, $Pz$, $O_1$, $O_2$) | 45.7 | 58.9 | 48.7 | 46.6 |

## DISCUSSION

The important role of the fronto-thalamic system in complex forms of mental activity and the regulation of the level of wakefulness was shown by many studies [6–11]. The fronto-thalamic slow waves are currently considered as signs of this system's non-optimality (its immaturity or dysfunction) [12, 13].

Despite the fact that the presence of slow activity in the EEG spectra is typical of children, its proportion should decrease with a child's growth, giving place to the α rhythm as the leading one with an accent in the parieto-occipital cortices in the normal state [14, 15]. The slow activity is also preserved in children with mental development disorders at later stages of ontogenesis, prevailing over rapid forms of activity. Edelman considered the underdeveloped rhythmic activity in the α range to be a correlate of a low level in the CNS plasticity and the immaturity of the α-rhythm generators [16].

The use of the ICA method in this study has allowed us to reveal, in retarded children, hypothetical slow-activity sources in not only frontal, but also temporal cortical regions responsible for the auditory analysis and the synthesis of speech signals, as well as

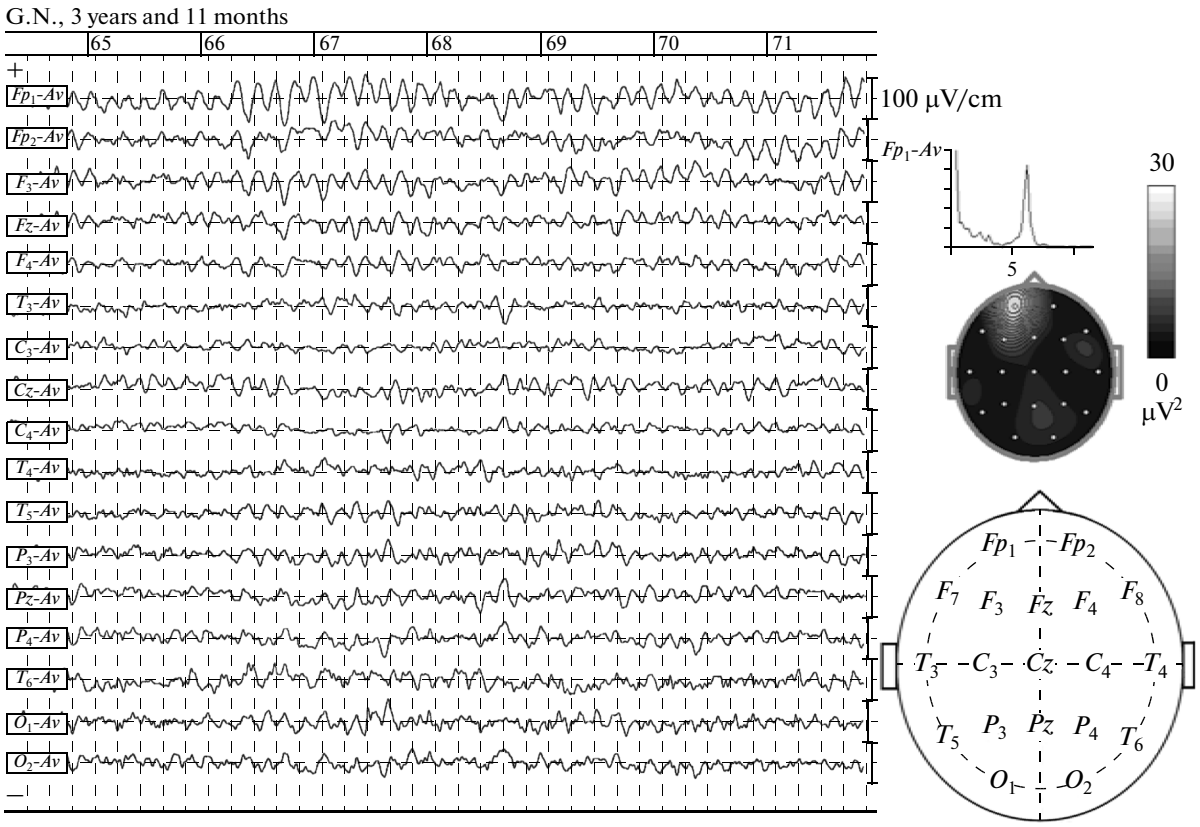


**Fig. 1.** A fragment of a child's native EEG recording from group 2Se at rest with the eyes open (left). The placement of electrodes according to the International 10–20 System, the recording method is monopolar relative to the averaged electrode. Frequency band: a low-frequency filter with a time constant of 0.3 s, a high-frequency filter for 50 Hz. Numbers on the top are 1-s recording marks; calibration signal over all channels is 100 μV/cm. On the right, the EEG spectrum at the $Fp_1$ lead (ordinate, $\mu V^2$; abscissa, Hz) with a peak at the frequency of 5.6 Hz. These data are represented below as a topogram for the spectral power.

the audio-visual information integration [6, 17]. It has been shown that an increase in the power of slow components corresponding to the *degree of severity* of developmental delays, including the presence of spectral peaks at the frequencies of 5–6 Hz in the indicated regions of the left hemisphere.

For example, higher values of the slow-activity power spectra have been revealed in children with rough developmental delays (group 2Se) as compared with groups 1 and 2Mi (children without delays in development and with mild speech disorders) across all 17 leads under analysis (Table 1). At the same time, when comparing the groups less contrasting in the level of development (2Mi and 2Mo, 2Mo and 2Se), we found differences only for half of the analyzed EEG leads.

In other words, we have found a wider distribution of independent slow-activity sources in the group of children with severe disorders of development, which may be basic for the total underdevelopment of mental functions characteristic not only of developmental delays (in a certain sense, a temporary phenomenon), but also of mental retardation, as a rougher disorder, without the required "acceleration" in the cortical rhythmic and mental processes in the course of ontogenesis.

It is also possible that such a specific synchronization of slow-activity sources in many investigated cerebral cortices is associated with more rigid and less plastic connections formed in the cases of perinatal CNS lesions, as well as with disturbed neuroembryogenesis, due to chronic fetal hypoxia [18–20]. There are assumptions that the increased synchronization of neuronal ensembles in the dominant hemisphere may interfere with the development of autonomy in individual regulatory centers at the cortical level [21].

We possibly deal with some mechanism that brings disturbances to a certain specified succession of a child's psychophysiological development [22] in ontogenesis with the leading role in this succession belonging to the fronto-thalamic system. It is known that a child's brain with a severe damage may not compensate for the affected deficient fragment in the process of development or even may lose the potential for subsequent maturation and become still more depleted with becoming older [23].

The following specific aspects have been revealed in comparing the basic (α and θ) ranges of EEGs in

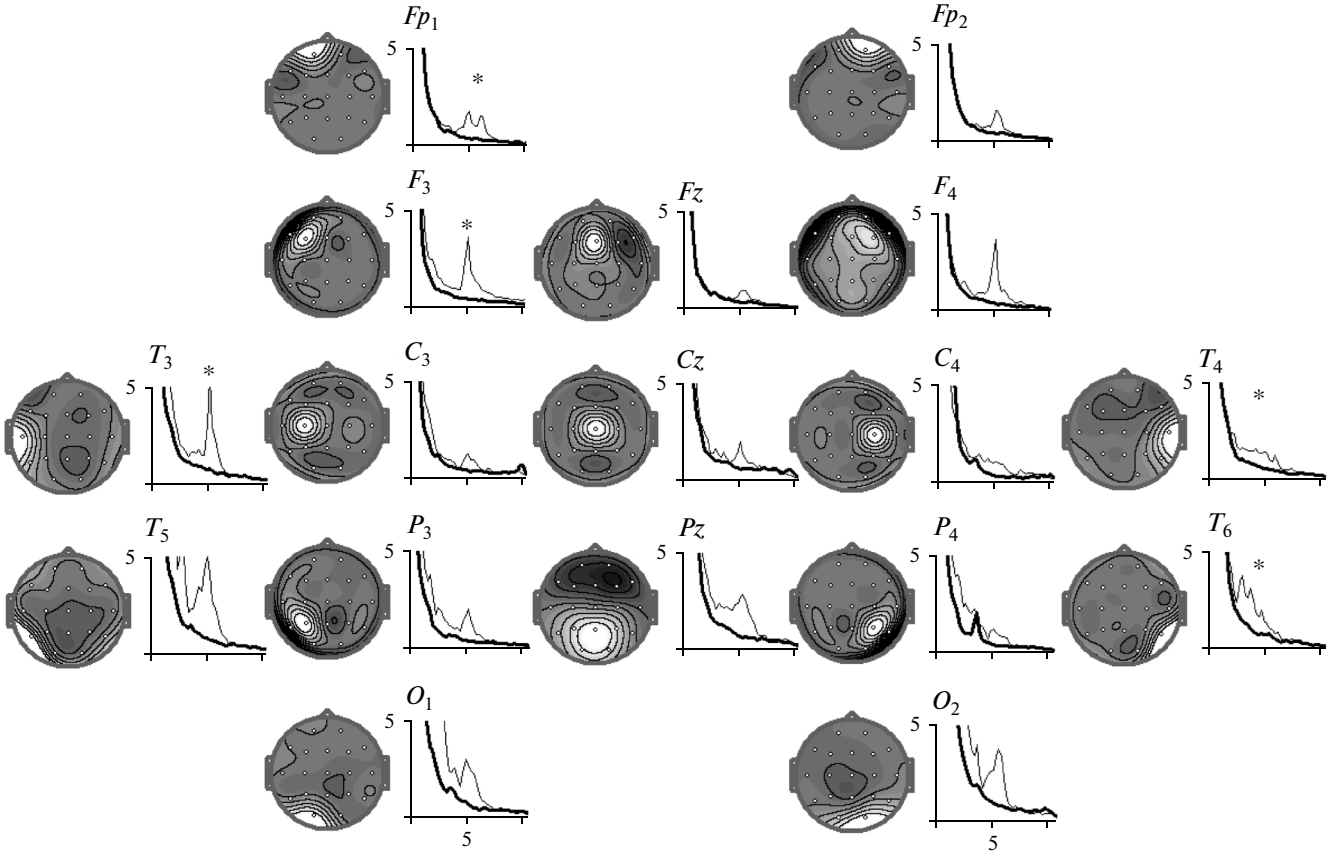


**Fig. 2.** Comparative analysis of the power spectra for independent EEG components in the state of calm wakefulness with the eyes open in children with mental development disorders in groups 2Mi (the bold line) and 2Se (the fine line). The abscissa shows the analyzed EEG frequencies (Hz); the ordinate shows the power spectra values in relative units. * A statistically significant growth in the spectral power of slow frequencies in group 2Se compared with group 2Mi.

children. We have revealed some putative generators of slow frequencies in wakefulness with the eyes open, while no significant differences have been found between independent components in the α-rhythm range with the eyes open. This allows us to use the detected slow-activity generators in the fronto-temporal cortices as markers for developmental disorder risks, particularly, when it is impossible to evaluate the expression of the main (α) rhythm in a waking state due to difficulties with recording a prolonged artifact-free EEG in a retarded child with the eyes closed. The insufficient informativeness of α range in children younger or older than the subjects of our study has also been noted by other authors describing speech disorders and the attention deficit hyperactivity disorder (ADHD) against a residual organic background [21, 24].

The manifestations of interhemispheric asymmetry seemed contradictory in our studies. For example, the frontal cortex as sources of slow rhythms prevailed in the left hemisphere, the sign of asymmetry across posterior temporal areas was inverted into the right hemisphere, and these sources were symmetrical across the anterior temporal areas (the interhemispheric differ-

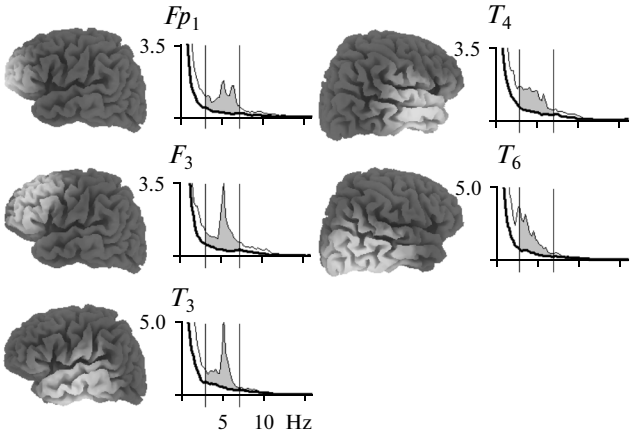


**Fig. 3.** Significant differences in spectral power for independent EEG components in the state of quiet wakefulness with the eyes open in groups 1 (the thick line) and 2Se (the thin line). The abscissa shows the analyzed EEG frequencies (Hz); the ordinate shows the power spectra values (relative units). The supposed localization of the sources for independent EEG components is marked to the left of each graph on the brain model, according to sLORETA.

ences are nonsignificant) (Fig. 3). It is only in the left hemisphere areas that significant spectral differences between slow frequencies were combined with spectral peaks at frequencies of 5–6 Hz.

Local lesions in the posterior temporal areas of the right hemisphere, as some authors believe, obstruct the development of the "left-hemispheric" hearing and speech functions and memory in the course of ontogenesis, which, in turn, determine the development of reading and writing skills [25]. If EEG deviations are localized in the anterior regions of the left hemisphere, a predominance of disorders is described in the motor components of writing; if phonological and morphological disorders are present, the accent of EEG changes is bilaterally marked in the temporal and posterior associative areas of the right hemisphere.

Regarding the interpretation of the data, it is appropriate to recall Jackson's statement that to locate the damage which destroys speech and to locate speech are different things (cited from [26]). It seems difficult to obtain more accurate functional specific features of EEG and structural changes due to heterochronicity of the development of cortical areas, as well as due to different probabilities of lesions or underdevelopment in any structures of the brain in the pre- and postnatal period. Moreover, the question of damage of which hemisphere more negatively affects subsequent child's development is a subject of active disputes so far [26–29].

According to today's neuroimaging data, disorders in the development of the language system in early childhood are often unassociated at all with lesions in a particular brain structure [30]. Researchers consider the intactness of a certain area of the brain not so much as a crucially important factor for the speech function as the preservation of potential connectivity between multiple speech-controlling areas, while the participation of even one of them in this connectivity is only considered to be a necessary link.

The issues about what center of speech (motor or sensor, Broca's or Wernicke's area) is the leading one have been discussed for a long time. Long ago, Pribram stated that if a lesion was localized posterior to Wernicke's area of the brain, the corresponding speech defect was more severe in terms of neurosurgical practice [31]. It is near this cortical projection (EEG leads $T_3$ and $T_4$) that we distinguished independent "sources" of slow activity in both hemispheres. According to the positron-emission tomography data, if the connections between Wernicke's area and other associative areas (if the area itself is preserved) are broken in adults, not only speech perception becomes complicated, but also the capacity to translate thoughts into words [32]. Wernicke's area is regarded in this context not so much as a rigidly concentrated center where the neurophysiological processes providing the speech function are focused but rather as a "neuronal gate" mainly performing the switching on/off function, where impulses from various sensory and associative fields converge and where "neural pattern of images" are translated into "form words."

Our studies have shown that selecting action-specific targets in children with mental retardation near the cortical projections of the above-mentioned cortical areas (in transcranial micropolarization, TCMP) increases the quality and efficacy of correcting the disturbed functions [33–35]. This may be connected with the fact that an action-specific target is initially a hypothetic source of low frequencies in a retarded child's EEG. The child's inborn native language ability, in fact, fails in mental retardation cases caused by a perinatal CNS pathology [30, 36]. The presence of speech is the main indicator of a child's mental development wellbeing, which is closely connected with the experience transfer function in the process of individual development.

## CONCLUSIONS

On the basis of the obtained data and taking into consideration a significant predominance of slow rhythms in the EEGs of mentally retarded children, we can assume that their hypothetic sources can be detected in the fronto-temporal cortices. It has been shown that the intensity of slow components increased corresponding to the degree of mental retardation severity, including the presence spectral peaks at frequencies of 5–6 Hz in the left hemisphere.

The use of the hypothetic generators of slow frequencies as the targets of action in performing a TCMP significantly increases the correctional effect of this action on speech functions and can serve as an indirect confirmation in favor of association of the described phenomena with one of the brain mechanisms underlying the formation of speech disorders of perinatal origin.

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