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A Search for Early Predictors of Mental and Speech Disorders: Neurophysiological Aspects

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Abstract—Data from neurophysiological examinations were summarized for children with mental development disorders of perinatal genesis. Hypothetical sources of slow activity were found in the frontotemporal cortex bilaterally. An increase in the power of slow components was shown to correlate with the severity of mental developmental disorders. New data were obtained to indicate that local and distributed ("global") sources of EEG rhythms have a potential as neuromarkers of psychoverbal retardation in early ontogeny. A high level accuracy was observed for a classification of the children on the basis of these potential neuromarkers.

Keywords: neuromarkers, mental developmental disorders, transcranial direct current stimulation (tDCS), independent component analysis (ICA)

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INTRODUCTION

The possibility to study the mechanisms of mental activity depends to a great extent on the potential of natural psychological experiments, the diagnostic and treatment tools available in modern medicine, and the mathematical methods of data processing. Current neuropsychology originates from neurosurgical clinical research in adults and, rare, children [1–5]. In pediatric neuropsychology, a corrective–educational approach is finding application in the cases where higher mental functions and speech are underdeveloped in children because of immaturity or dysfunction of the brain systems responsible for their support [6, 7]. A while ago, abroad range of electrical influences on the brain provided a potent impetus for studies of treatments, rehabilitation of lost mental functions, and stimulation of compensatory reserves [8–11].

We have used left-hemispheric transcranial direct current stimulation (tDCS) in children with psychoverbal retardation [12–15] and observed a significant increase in α activity in the age-related focus (parieto-occipital cortical areas) and a significant decrease in slow activity in the frontotemporal areas of the left hemisphere in cases where beneficial changes occurred in the development. After repeated tDCS courses, the average frequency of α activity in the age-related parieto-occipital focus increased to 9–10 Hz in the eyes-closed state, its spectral power grew higher, and an accent became detectable in areas of the unstimulated (right) hemisphere. A decrease in the β-band spectral power was additionally observed in the right posterior temporal cortex in the eyes-open and eyes-closed states. Thus, both local positive changes in EEG and systemic effects on neurophysiological parameters were detected in children with positive dynamics of their development after tDCS.

The current system of screening newborns for health problems on the outpatient basis is such that a child is no longer required to visit a specialist regularly when pathological neurological signs and symptoms improve or disappear by the end of the first year of life. However, the child may again attract attention of specialists at approximately three years old with what is formulated as "delayed consequences of perinatal damage to the CNS". Several problems become evident by the age three, including a weak response when spoken to, lack of speech production, feral behavior, poor copying behavior, undeveloped self-care skills and indexical gestures, etc. The period of 2–3 years of age with latent risk factors for delayed consequences is often a blank spot for both neurologists and neurophysiologists.

The objective of this work was to identify the specifics of bioelectrical activity of the brain that may serve as neurophysiological predictors of retardation in HNF and speech development in children at early ontogenetic steps, using independent component analysis (ICA) of EEG components.

METHODS

The study included children with lower speech development rates; motor alalia; general speech underdevelopment; emotional-volitional immaturity; defects in the development of attention, thinking, and communicative functions; etc. EEG examinations were performed in two steps, depending on the child's age. The study did not include children with epilepsy or cerebral palsy and patients on long-term treatment with psychoactive drugs.

At the first step, we selected older children (4–9 years old) with mental retardation. Their choice was based on our previous finding that independent EEG components are significantly different in groups of children with more severe defects in psychoverbal development, in particular, those with autism spectrum disorder (ASD) [14]. A control group (group 1) included 70 children with a history of perinatal encephalopathy, but without developmental retardation (44 boys; mean age 6.4 years; standard deviation (SD) = 1.4 years). Group 2 included 42 children with ASD (36 boys; mean age 5.9 years; SD = 1.7 years). A total of 112 children were examined at this step (mean age 6.4 years; SD = 1.6 years). A psychological and logopedic testing was performed, and the ASD group was divided into two subgroups differing in the extent of retardation (severe and mild forms). The groups of children were age-matched and did not differ in age by the Mann–Whitney criterion at each step of the study.

At the second step, we analyzed the data obtained for younger children (147 children in total, 1–4 years old). Like at the first step, group 1 (control) included children without developmental retardation (26 children, 12 boys, mean age 3.5 years, SD = 0.56 years). Another group (group 2S) included 20 children with more severe retardation, including ASD (11 boys, mean age 3.2 years, SD = 0.95 years). A stepwise discriminant analysis with inclusion was carried out to evaluate the specificity of a neuromarker in estimating the extent of retardation. A group with moderate developmental defects (group 2Mo) was isolated for the purpose and included 30 children (23 boys, mean age 3.4 years, SD = 0.73 years). One more additional group (group 2Mi) included children with mild impairments (71 children, 48 boys, mean age 3.3 years, SD = 0.81 years). Spectral characteristics of EEG components isolated previously were evaluated relative to the control group [12]. Slow-wave (2–7 Hz) and α (7–11 Hz) ranges were examined.

EEG was recorded at the resting state for 2–4 min. Silver/silver chloride electrodes were positioned according to the international 10–20 system. EEGs from 19 channels were recorded using a computerized EEG system (Mitsar, Russia). The resistance of the EEG electrodes did not exceed 5 kOhm. The recording was monopolar with respect to the left and right ear silver/silver chloride electrodes (a combined reference/common average reference was used). The cut-off frequencies of high- and low-frequency filters were 0.5 and 45 Hz, respectively. Eye movement artifacts were corrected by filtering the principal components corresponding to eye movements with a focus at the frontal sites Fp_1 and Fp_2 [16]. Principal component analysis was used because artifact-free EEG recordings are short in children [17]. In addition, the analysis did not include the EEG fragments that contained high-amplitude fast-activity artifacts with frequencies of 20–35 Hz and amplitudes higher than 40 μV or slow oscillations with frequencies of 0–1 Hz and amplitudes higher than 50 μV; potential changes greater than 120 μV were also excluded.

Spectral components were isolated by blind source separation via independent component analysis (ICA). Spectral component isolation was carried out using the Infomax procedure described in EEGLab [18]. Putative sources of independent EEG components were localized by sLORETA low-resolution tomography [19]. The method analyzes the topography of a component to identify its potential sources and Brodmann areas where the sources presumably occur. Based on the localization data, all components were associated with EEG channels according to the international 10–20 system.

At the first step, the power spectra were compared between the main EEG bands: θ (4–8 Hz), α (8–13 Hz), and β (13–20 Hz). Spectra were calculated using a standard epoch of 4 s, an epoch overlap of 50%, and the Hanning time window. The average power was taken as a logarithm to normalize the distribution in order to eliminate the age-related changes. Differences in spectral power in the above bands were additionally evaluated from the Cohen effect with a threshold; the results were considered significant at $d > 0.6$.

The EEGs recorded in the resting state with eyes open was used for component separation by ICA. Before further processing, the entire array of EEG recordings was filtered at the 0.5–35-Hz frequency band.. High-order filters (894) were used. Spectra were calculated using a standard epoch of 2 s, epoch overlap of 50%, and the Hanning time window. The average power was taken as a logarithm to normalize the distribution. Differences were evaluated by MANOVA.

RESULTS

The group of older children with ASD and greater developmental retardation showed significant changes in the EEG θ band in several areas as compared with the control group (Table 1) Components were localized in both right and left hemispheres, including frontotemporal areas (components *ICF*4, *ICF*8, and *ICF*3), parietal and posterior temporal areas (components *ICP*3, *ICPz*, and *ICT*6), and occipital cortical areas (components *ICO*1 and *ICO*2). Independent

Table 1. Comparative analysis of the powers of EEG spectral components in the children with ASD (mild and severe forms) and control children in the waking state with eyes open

ASD group compared with the control group	Statistical difference	Component (IC)/area: EEG θ band (4–8 Hz)							
		ICF3	*ICF4*	*ICF8*	*ICP3*	*ICPz*	*ICT6*	*ICO1*	*ICO2*
Severe forms	$F[1,93]=$	**20.31**	10.36	**14.38**	7.89	9.96	**17.71**	9.37	8.18
	$p <$	**0.00002**	0.002	**0.0003**	0.006	0.002	**0.0001**	0.003	0.005
	Cohen Size-effect, *d*	1.09	0.79	0.80	0.69	0.79	1.08	0.74	0.72
Mild forms	$F[1,87]=$	12.29							
	$p <$	0.0007							
	Cohen Size-effect, *d*	0.92							
ASD group compared with the control group	Statistical difference	Component (IC)/area: EEG β band (13–30 Hz)							
		ICF3	*ICF4*	*ICF8*	*ICP3*	*ICPz*	*ICT6*	*ICO1*	*ICO2*
Severe forms	$F[1,93]=$	**16.9**		7.4		8.7	12.4		7.3
	$p <$	**0.0001**		0.007		0.004	0.0007		0.008
	Cohen Size-effect, *d*	0.95		0.63		1.35	0.95		0.70
Mild forms	$F[1,87]=$	11.0							10.1
	$p <$	0.002							0.002
	Cohen Size-effect, *d*	0.84							0.88

The results significant after the Bonferroni correction are in bold ($p < 0.0004$). The Cohen Size-effect was considered significant at >0.6. Empty cells indicate lack of significant differences.

sources of the θ band were detected only for the component *ICF*3 in milder ASD. After a Bonferroni correction, significant differences were observed for only two components, *ICF*4 and *ICT*6, in the right hemisphere and the component *ICF*3 in the right hemisphere.

The α band (eyes open) did not display significant changes by Student's *t*-test. In the β band, significant differences from the control were more often detected in cases with more severe developmental retardation in the group of older children. However, the total number of local sources was lower than in the slow band. In particular, the components *ICF*8, *ICT*6, and *ICO*2 were left on the right (but the differences were observed only without the Bonferroni correction) and the components *ICF*3 (was left after the correction) and *ICPz* on the left. A local β-band source (component *ICO*2) was additionally isolated in the children with mild ASD, in contrast to the θ band. The source was significant by the Cohen effect size, but nonsignificant by Student's *t*-test.

It should be noted that the component *ICF*3 in the older retarded children showed substantial differences from the control group in both of the EEG bands under study and that the differences were observed in cases differing in ASD severity (being more significant in severe cases) (Table 1). The component was broadly distributed through many cortical areas and was consequently designated global.

At the second step, significant differences in the spectral power of slow activity in a range of 2–7 Hz (that is, with expansion to lower frequencies) was revealed in the younger children in the waking state with eyes open by spectral component analysis. Slow activity in the younger children with developmental retardation was significantly higher than in the control group, as was observed in the older retarded children. Several local sources were also the same in the younger children. This was the case with the component *ICF*3 on the left (with a broad distribution and a center presumably in the inferior frontal gyrus, Brodmann area 44 according to sLORETA; Fig. 1a) and the component *ICT*6 (in the posterior temporal and occipital areas, with a center located presumably in Brodmann area 37 according to sLORETA; Fig. 1c). An additional local source, the component *ICT4* (in the temporal area, with a center located presumably in Brodmann area 21 according to sLORETA; Fig. 1b), was detected in the younger children. As is seen from Fig. 1, differences were greater in the right hemisphere.

A stepwise discriminant analysis of the data from all children showed that a correct classification is possible at a rate of 85% for the group of severely retarded children (group 2S) compared with the control group. MANOVA confirmed the difference for seven components: *ICF*3, *ICC*3, *ICP*3, *ICC*z, *ICT*4, and *ICT*6 of the θ band and *ICT*6 of the α band ($F[7,38] = 3.70$, $p < 0.004$; Wilks' $\lambda = 0.59$). In the moderately retarded children (group 2Mo), a correct classification rate of

91% was achieved with all θ-band components (*ICFp*2, *ICC*3, *ICT*4, *ICT*5, *ICP*3, *ICPz*, and *ICT*6) and α-band components (*ICF*3, *ICF*4, *ICT*3, *ICC*3, *IC*C4, *ICP*3, and *ICT*6); MANOVA $F[14,41] = 5.00$, $p < 0.00002$; Wilks' $\lambda = 0.37$. In the mildly retarded children (group 2Mi), a correct classification rate of 85% was achieved with the θ-band component *ICCz* and the α-band components *ICF*4, *ICC*4, *ICPz*, and *ICP*4; MANOVA $F[5,46] = 5.07$, $p < 0.0009$; Wilks' $\lambda = 0.65$. Thus, the correct classification rate could be no less than 85% in the above groups according to the discriminant analysis.

DISCUSSION

For 20 years, we have examined children with consequences of perinatal damage to the CNS at various stages of diagnosis and therapy. Although diverse signs and symptoms are characteristic of deviations in ontogeny, there are still common neurophysiological regularities.

Modern methods of EEG analysis often detect a predominance of slow-rhythm sources, while significant changes in fast activity are far less common [12–15, 20, 21]. Because a function underdeveloped for the age (immature) is the problem, it is possible that the problem has the appearance of a slowing down of brain rhythms at the level of neurophysiological mechanisms, while structural changes are rare detected in the brain by MRI. We have observed a broader distribution of slow-activity sources in the most severe cases of developmental retardation; the sources were mapped to the frontotemporal and parieto-occipital areas [12]. This is possibly one of the neurophysiological mechanisms that underlies total underdevelopment of psychic functions in mental retardation, when "acceleration" of cortical rhythms and mental processes during ontogeny is insufficient for the patients to catch up with their normally developing age-mates.

The following should be noted about the specifics of interhemispheric relationships. Spectral peaks corresponding to "deceleration" at frequencies of 5–6 Hz have previously been isolated in the frontotemporal areas of the left hemisphere in severely retarded children (the components *ICFp*1, *ICF*3, and *ICT*3) [12]. In this work, a global (broadly distributed) component with a maximum close to the motor centers of speech (the component *ICF*3 close to Broca's area on the left) was isolated in comparisons of different age groups. The component was observed in both β and θ bands in the older children and was detectable in patients differing in the severity of retardation. The component can be used as a neuromarker of various retardation forms at various ontogenetic stages. Pathological activity of the mirror neuron system in the EEG from the frontal and central cortical areas has been described in the literature for autistic children and is especially distinct in Broca's area [22].

A certain predominance of right-hemispheric sources of fast activities (13–30 Hz) was observed in our studies in the EEGs of older children with severe ASD. An increase in β band (20–30 Hz) in the frontocentral areas with an accent in the right hemisphere has been described for autistic patients of a broad age range (3–26 years old) [21]. Right-hemispheric accents of changes in brain activity have been described for adult patients (ERP analyses in schizophrenia) in other studies [23, 24].

A right-hemispheric accent was observed only for slow frequencies of no more than 5 Hz in the youngest group of retarded children with autistic signs. Slow-activity sources of the right hemisphere were the same in the younger and older children only in the temporal area (the components *ICT4* and *ICT6*). The presence of slow-activity sources in the right temporal area at an early age is possibly a predictor of developmental retardation and risk of autism. Cases where normally developing children withdraw into themselves and display speech regression after an illness, vaccination, etc., are well known in clinics [21]. Distorted processes whereby external events are reflected in a child's brain (mirror neuron system) lead to a chain of deviations in the development of copying behavior, adequate social responses in adaptation, and correct recognition of other people, that is, to social brain developmental problems [25].

If we compare the distribution of sources within one hemisphere, severely retarded children tend to display a predominance of sources in the anterior (frontotemporal) areas of the left hemisphere and more posterior (parietotemporal and occipital) areas of the right hemisphere. Phylogenetically younger structures, which include the left frontotemporal area, are thought to be the most vulnerable in disorders of the early ontogeny [6]. The fronto-thalamic system has been shown to play an important role in complex mental activities and the regulation waking levels in children [26].

Damage to the parieto-occipital areas and the temporo-parieto-occipital (TPO) subregion leads to semantic aphasia and an impaired understanding of complex speech constructs in adults, while the meaning of individual words is understood correctly [3]. Indicators of speech understanding, execution of verbal instructions, and attention were the lowest in children with ASD, who had defects in many mental functions [14]. An intense thickening of the cortex in the TPO area has been observed in younger children (below three years old) together with similar processes in visual cortical fields and the prefrontal oculomotor area, which are associated with the formation of visual perception and manipulative activities [27].

Local EEG activity sources were additionally isolated in the inferior parietal and occipital areas of the two hemispheres (*ICP*3, *ICP*4, *ICPz*, *ICO*1, and *ICP*2). The posterior association area (area 37) is

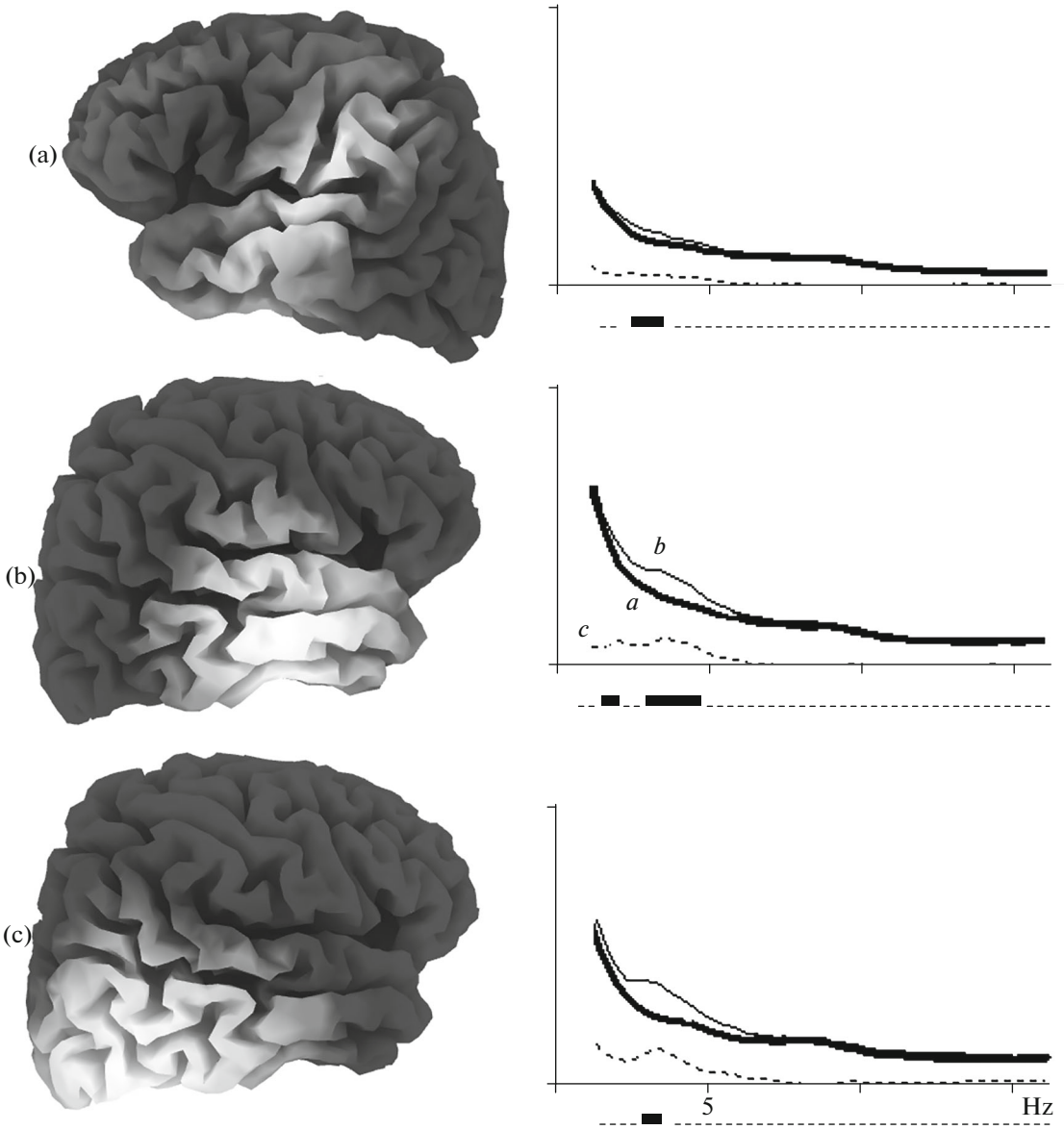

Fig. 1. Significant between-group differences in the spectral power of EEG components in younger children the state of calm waking with eyes open. X axis: EEG frequency (Hz); Y axis: spectral power. Lines: *1*, group 1; *2*, group 2S; and *3*, between-group difference (regions of values significant at $p < 0.05$ are shown with bars at the bottom of each plot). Hypothetical localizations of the sources of the EEG components according to sLORETA are shown with brain images on the left of each plot. (a) Component *ICF*3 has a broad distribution and a putative center in the inferior frontal gyrus, Brodmann area 44; (b) component *ICT*4 is in the temporal area and has a putative center in Brodmann area 21; (c) component *ICT*6 is in the posterior temporal–occipital area and has a putative center in Brodmann area 37.

known to play a substantial role in perceiving and recognizing complex visual images and to act, together with the inferior parietal areas, as a higher integrating component in analysis of visual information [22]. The right occipital area is associated with mechanisms of visual recognition of real and symbolic objects [24] and the processes involved in identifying the emotional state of another person. A psychic blindness area has even been placed into the occipital area in early maps of the brain.

It is rather difficult to determine whether the mechanisms of the above phenomena are associated with dysfunction, imbalanced interhemispheric relationships, or deficits and insufficient development of one of the hemispheres. A special role of the left hemisphere in the mechanisms of many speech-associated mental processes has been highlighted in many studies, which have additionally emphasized that interhemispheric interactions are of importance for the optimal brain function [2–6, 10, 29–31]. The role of the right hemisphere at a younger age has been accen-

tuated in other studies [1, 32–36]. This mosaic picture indicates that the involvement of many brain areas corresponds to the complexity of processes underlying the formation of higher mental functions and speech in the ontogeny, even when the scenario is abnormal.

Although there are well-known limitations to the use of theoretical findings of neurophysiologists in medicine, it should be noted that the neuromarkers isolated in our work allow a classification of children with various mental developmental problems with a high level accuracy (85–91%). As early as 1992, EEG studies in patients with early childhood autism showed that a special "EEG syndrome" has a high potential (more than 80%) in certain nosological forms [20].

CONCLUSIONS

A higher number of slow-activity sources was detected in severely retarded children by independent component analysis of the EEG. In the right hemisphere, slow-activity sources common for older and younger children were detected in the temporal area (components *ICT*4 and *ICT*6). The presence of a slow-activity source in the right temporal area at an early age is possible to interpret as a predictor of risk for psychoverbal retardation. A global component (*ICF*3) with a broad distribution in the vicinity of the motor centers of speech of the left hemisphere was observed in both of the age groups. The component was isolated in both β and θ bands in the older children and was detectable in patients differing in the severity of retardation. Based on the findings, the component may play a role as a neuromarker of psychoverbal disorders in abnormal ontogeny. Discriminant analysis showed that correct classifications based on the neuromarkers are possible with a high level of accuracy (85–91%).

COMPLIANCE WITH ETHICAL STANDARDS

Conflict of interests. The authors declare that they have no real or potential conflict of interest.

Statement of compliance with standards of research involving humans as subjects. All procedures were in accordance with the ethical standards of the 1964 Helsinki Declaration and its later amendments and were approved by the local Ethics Committee at the N.P. Bechtereva Institute of the Human Brain (St. Petersburg). The parents of all individual participants involved in the study voluntarily gave their written informed consent for participation after being informed about potential risk and benefits and the nature of the study.

REFERENCES

1. Sumernitskaya, E.G., *Mozg cheloveka i psikhicheskie protsessy v ontogeneze* (Human Brain and Psychical Processes in Ontogenesis), Moscow: Mosk. Gos. Univ., 1985.
2. Tsvetkova, L.S., *Metodika neiropsikhologicheskoi diagnostiki detei* (Method of Neuropsychological Diagnostic of Children), Moscow: Pedagog. O-va Ross., 2000.
3. Luriya, A.P., *Vysshie korkovye funktsii cheloveka* (Human Higher Cortical Functions), St. Petersburg: Piter, 2008.
4. Dobrokhotova, T.A., *Neiropsikhiatriya (Neuropsychistry)*, Moscow: Binom, 2013, 2nd ed.
5. Khrakovskaya, M.G., *Afaziya, agnoziya, apraksiya. Metodiki vosstanovleniya* (Aphasia, Agnosia, and Apraxia: Rehabilitation Methods), St. Petersburg: Nestor–Istoriya, 2017.
6. Semenovich, A.V., *Neiropsikhologicheskaya korrektsiya v detskom vozraste* (Neuropsychological Correction in Childhood), Moscow: Genezis, 2017.
7. *Narusheniya pis'ma i chteniya u detei: izuchenie i korrektsiya (Disturbance of Writing and Reading in Children: Analysis and Correction)*, Velichenkova, O.A., Ed., Moscow: Logomag, 2018.
8. *Elektricheskaya stimulyatsiya mozga i nervov u cheloveka (Electrical Stimulation of Human Brain and Nerves)*, Bekhtereva, N.P., Ed., Leningrad: Nauka, 1990.
9. Pinchuk, D.Yu., *Transkranial'nye mikropolyarizatsii golovnogo mozga: klinika, fiziologiya* (Transcranial Micropolarizations of the Brain: Clinics and Physiology), St. Petersburg: Chelovek, 2007.
10. Penfield, W., *The Mystery of the Mind: A Critical Study of Consciousness and the Human Brain*, Princeton, NJ: Princeton Univ. Press, 2015.
11. Ilyukhina, V.A., *Nauchnye predvideniya v poznanii printsipov zhiznedeyatel'nosti mozga cheloveka, ikh razvitie i realizatsiya* (Scientific Predictions in Understanding of Human Brain Activity: Development and Implementation), St. Petersburg: Inform–Navigator, 2018.
12. Kozhushko, N.Yu., Evdokimov, S.A., Matveev, Yu.K., et al., Study of local EEG specificities in children with mental development disorders using independent component analysis, *Hum. Physiol.*, 2014, vol. 40, no. 5, p. 497.
13. Kozhushko, N.Yu., Evdokimov, S.A., and Matveev, Yu.K., Neurophysiological markers of abnormal development in children with mental disorders, *Hum. Physiol.*, 2018, vol. 44, no. 2, p. 202.
14. Kozhushko, N.Ju., Nagornova, Zh.V., Evdokimov, S.A., et al., Specificity of spontaneous EEG associated with different levels of cognitive and communicative dysfunctions in children, *Int. J. Psychophysiol.*, 2018, vol. 128, p. 22.
15. Kozhushko, N.Yu. and Evdokimov, S.A., Age-related changes in EEG formation during transcranial direct current stimulation, *Hum. Physiol.*, 2019, vol. 45, no. 4, p. 364.
16. Ille, N., Berg, P., and Scherg, M., Artifact correction of ongoing EEG using spatial filters based on artifact and brain signal topographies, *J. Clin. Neurophysiol.*, 2002, vol. 19, no. 2, p. 113.
17. Tereshchenko, E.P., Ponomarev, V.A., Kropotov, Yu.D., and Müller, A., Comparative efficiencies of different methods for removing blink artifacts in analyzing quantitative electroencephalogram and event-related potentials, *Hum. Physiol.*, 2009, vol. 35, no. 2, p. 241.

18. Makeig, S., Bell, A.J., Jung, T.P., et al., Independent component analysis of electroencephalographic data, in *Advances in Neural Information Processing Systems*, Cambridge. MA: MIT Press, 1996, vol. 8, p. 145.
19. Pascual-Marqui, R.D., Standardized low-resolution brain electromagnetic tomography (sLORETA): technical details, *Methods Find. Exp. Clin. Pharmacol.*, 2002, vol. 24, suppl. D, p. 5.
20. Gorbachevskaya, N.L., Yakupova, L.P., Kozhushko, L.F., et al., Topographic EEG-mapping in children's psychiatry, *Fiziol. Chel.*, 1992, vol. 18, no. 6, p. 40.
21. Gorbachevskaya, N.L., Mamokhina, U.A., Vershinina, N.V., et al., Specific spectral characteristics of EEG in persons with autistic disorders, *Psikhiatriya*, 2018, vol. 78, no. 2, p. 48.
22. Yasuhara, A., Correlation between EEG abnormalities and symptoms of autism spectrum disorder (ASD), *Brain Dev.*, 2010, vol. 32, no. 10, p. 791.
23. Evdokimov, S.A., Pronina, M.V., Polyakova, G.Yu., et al., Analysis of independent components for event-related potentials from patients with an established diagnosis of schizophrenia, obsessive-compulsive and depressive disorders, *Zh. Vyssh. Nervn. Deyat. im. I.P. Pavlova*, 2014, vol. 64, no. 5, p. 500.
24. Kropotov, J.D., Pronina, M.V., Polyakov, J.I., and Ponomarev, V.A., Functional biomarkers in the diagnostics of mental disorders: cognitive event-related potentials, *Hum. Physiol.*, 2013, vol. 39, no. 1, p. 8.
25. Ramachandran, V.S., *The Tell-Tale Brain: A Neuroscientist's Quest For What Makes Us Human*, New York: W. W. Norton, 2010.
26. *Mozgovye mekhanizmy formirovaniya poznavatel'noi deyatel'nosti v predshkol'nom i mladshem shkol'nom vozraste* (Brain Mechanisms of the Cognitive Activity in Pre-School and Primary School Age), Machinskaya, R.I. and Farber, D.A., Eds., Voronezh: Modek, 2014.
27. Tsekhmistrenko, T.A., Vasilyeva, V.A., and Shumeiko, N.S., Structural rearrangements of the cerebral cortex in children and adolescents, *Hum. Physiol.*, 2017, vol. 43, no. 2, p. 123.
28. Glezer, V.D., *Zrenie i myshlenie* (Vision and Thinking), Leningrad: Nauka, 1985.
29. Bekhtereva, N.P., *Magiya mozga i labirinty zhizni* (Magic of the Brain and the Labyrinths of Life), Moscow: AST, 2007.
30. Pavlova, L.P., *Dominanty deyatel'nosti mozga cheloveka* (Dominants of Human Brain Acitivity), St. Petersburg: Inform–Navigator, 2017.
31. Njiokiktjien, C., *Symptoms and Syndromes in Neuropsychiatry*, Amsterdam: Suyi, 2008.
32. Nuss, R., Peterson, H., and Koch, D., Differential effects on congenital left and right brain injury on intelligence, *Brain Cognit.*, 1989, vol. 9, no. 2, p. 258.
33. Sergienko, E.A. and Dozortseva, A.V., Functional asymmetry of the cerebral hemispheres, in *Funktsional'naya mezhpolusharnaya asimmetriya* (Functional Interhemispheric Asymmetry), Moscow: Nauchnyi Mir, 2004, p. 218.
34. Tsvetkova, L.S. and Tsvetkov, A.V., Speech and the right brain hemisphere: aphasia vs anomie, *Teor. Prakt. Obshch. Razvit.*, 2014, no. 13, p. 70.
35. Gainotti, G., Lower- and higher-level models of right hemisphere language. A selective survey, *Funct. Neurol.*, 2016, vol. 31, no. 2, p. 67.
36. Skeide, M.A. and Friederici, A.D., The ontogeny of the cortical language network, *Nat. Rev. Neurosci.*, 2016, vol. 17, no. 5, p. 323.

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