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CLINICAL AND NEUROLOGICAL CHARACTERISTICS OF CHILDREN WITH DEVELOPMENTAL SPEECH DELAY DEPENDING ON THE PRESENCE OF RESIDUAL BRAIN DAMAGE

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Abstract. *Developmental speech delay (DSD) is a frequent neurodevelopmental condition in early childhood and is often accompanied by a wide range of neurological, behavioral, and cognitive disturbances. Increasing attention is being paid to the role of perinatal and early postnatal factors, particularly residual brain damage (RBD), in shaping the clinical phenotype of speech dysontogenesis.*

Keywords: *Developmental speech delay; residual brain damage; perinatal risk factors; neurodevelopment; neurological status; language impairment; children.*

КЛИНИКО-НЕВРОЛОГИЧЕСКИЕ ОСОБЕННОСТИ ДЕТЕЙ С ЗАДЕРЖКОЙ РЕЧЕВОГО РАЗВИТИЯ В ЗАВИСИМОСТИ ОТ НАЛИЧИЯ РЕЗИДУАЛЬНОГО ПОРАЖЕНИЯ ГОЛОВНОГО МОЗГА

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Аннотация. *Задержка речевого развития (ЗРР) является одной из наиболее распространённых форм нейроразвития в раннем детском возрасте и часто сопровождается неврологическими, когнитивными и поведенческими нарушениями. В последние годы особое внимание уделяется влиянию перинатальных факторов риска и резидуального поражения головного мозга (РПГМ) на формирование клинических вариантов речевого дизонтогенеза.*

Ключевые слова: *задержка речевого развития, резидуальное поражение головного мозга, перинатальные факторы риска, нейроразвитие, неврологический статус, речевые нарушения, дети.*

НУТҚ РИВОЖЛАНИШИ КЕЧИКИШИ БЎЛГАН БОЛАЛАРДА РЕЗИДУАЛ БОШ МИЯ ЗАРАРЛАНИШИ МАВЖУДЛИГИГА ҚАРАБ КЛИНИК-НЕВРОЛОГИК ХУСУСИЯТЛАР

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Аннотация. *Нутқ ривожланишининг кечикиши (НРК) эрта болалик даврида учрайдиган нейроривожланиш бузилишларининг энг кенг тарқалган шаклларидан бири ҳисобланади ва кўпинча неврологик, когнитив ҳамда хулқ-атвор бузилишлари билан кечади. Сўнгги йилларда перинатал хавф омиллари ҳамда бош миянинг резидуал зарарланишининг нутқ дизонтогенезининг клиник вариантлари шаклланишига таъсирига алоҳида эътибор қаратилмоқда.*

Калит сўзлар: *нутқ ривожланишининг кечикиши, бош миянинг резидуал зарарланиши, перинатал хавф омиллари, нейроривожланиш, неврологик статус, нутқ бузилишлари, болалар.*

Introduction.

Developmental speech delay (DSD) remains one of the most common neurodevelopmental disorders encountered in pediatric neurological practice. According to epidemiological data, speech and language disorders affect up to 12–15% of preschool-aged children and often represent the earliest manifestation of broader neurodevelopmental dysfunction [1,2]. Impaired speech development significantly limits communication abilities, disrupts social interaction, and negatively influences cognitive development and academic performance in later childhood [3].

The formation of speech is a complex multilevel process that depends on the integrity and functional maturity of cortical and subcortical brain structures, sensorimotor integration, executive regulation, and emotional-behavioral control [4,5]. Disruption of these mechanisms during critical periods of brain development may lead to persistent speech dysontogenesis. In this context, perinatal and early postnatal factors have attracted increasing attention as potential determinants of unfavorable neurodevelopmental outcomes [6].

Residual brain damage, resulting from antenatal hypoxia, prematurity, birth trauma, or early neonatal complications, is considered an important pathogenetic factor contributing to diffuse neurological dysfunction and delayed speech acquisition [7,8]. Nevertheless, clinical practice demonstrates substantial heterogeneity among children with DSD, including patients with relatively isolated speech impairment and those with combined neurological, motor, and behavioral abnormalities. Comparative studies addressing the influence of residual brain damage on the clinical-neurological structure of developmental speech delay remain limited.

A detailed analysis of anamnestic data, perinatal risk factors, neurological status, and speech characteristics may contribute to a better understanding of pathogenetic mechanisms underlying different clinical variants of DSD and provide a basis for improving early diagnostic and therapeutic approaches.

Purpose of the research.

To conduct a comprehensive clinical and neurological assessment of children with developmental speech delay and to identify differences in anamnestic, neurological, and speech characteristics depending on the presence of residual brain damage.

Materials and methods.

Study Population. The study included 305 children aged 3 to 7 years diagnosed with developmental speech delay (ICD-10: F80) who were examined on an outpatient basis. All children were assessed by a pediatric neurologist and a speech-language specialist. Participants were divided into three groups: Group I (DSD + RBD) – 120 children with developmental speech delay and clinically confirmed residual brain damage. Group II (DSD without RBD) – 185 children with developmental speech delay without signs of residual brain damage. Control group – 40 neurologically healthy children with age-appropriate speech development. The groups were comparable in terms of age and sex distribution (Figure 1).

Anamnestic and Perinatal Assessment. Anamnestic data were collected through structured parental interviews focusing on antenatal, intranatal, and early postnatal periods. Perinatal risk factors were evaluated using standardized scoring systems widely applied in pediatric neurology, allowing stratification of risk into antenatal, intranatal, and early neonatal domains [9,10].

Neurological Examination. A comprehensive neurological examination was performed in all participants, including assessment of cranial nerve function, muscle tone, tendon reflexes, coordination, gait, postural stability, and the presence of hyperkinetic or extrapyramidal signs. Neurological findings were interpreted in accordance with established pediatric neurological guidelines [11].

Speech and Language Assessment. Speech development was evaluated using structured logopedic examination tools assessing phonation, articulation, receptive and expressive speech, vocabulary size, phrase construction, and lexical-grammatical organization [12,13]. Parental questionnaires were additionally used to assess speech-related complaints, behavioral characteristics, and learning difficulties.

Statistical Analysis. Quantitative data were expressed as mean values with standard deviations (M ± SD) and 95% confidence intervals. Group comparisons were performed using Welch's t-test or one-way ANOVA. Categorical variables were analyzed using Fisher's exact test or Pearson's chi-square test. Statistical significance was set at $p<0.05$.

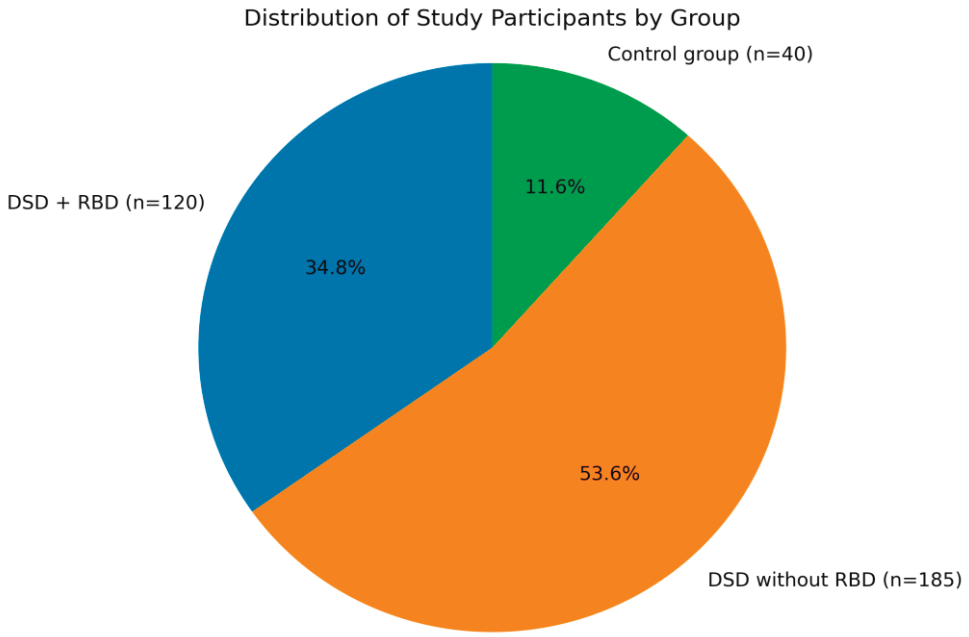


Figure 1. **Demonstrates the proportional distribution of participants across the study groups. The largest proportion of the sample was represented by children with developmental speech delay without residual brain damage, followed by children with developmental speech delay and residual brain damage, while the control group constituted the smallest part of the cohort. The predominance of clinical groups reflects the study's focus on comparative analysis of developmental speech delay phenotypes. The comparable age and sex distribution across groups supports the validity of intergroup comparisons and minimizes potential demographic confounding.**

Results.

Anamnestic and Perinatal Characteristics. Children with developmental speech delay demonstrated a significantly higher burden of unfavorable perinatal factors compared to the control group, which is consistent with previously reported associations between early neurobiological vulnerability and delayed speech development [3,6]. The most pronounced risk profile was observed in children with residual brain damage. In this group, complicated pregnancy, maternal chronic diseases, premature birth, neonatal hypoxia, and early neurological dysfunction were significantly more frequent ($p<0.001$), reflecting the impact of early brain injury on subsequent neurodevelopmental outcomes [6–8]. Antenatal and early neonatal risk scores were significantly higher in the DSD + RBD group, whereas intranatal risk factors were relatively more prominent in children with DSD without residual brain damage. Despite these differences, the overall perinatal risk level in both clinical groups corresponded to a high-risk category, indicating a substantial contribution of early developmental adversity to the formation of speech delay phenotypes [4,7].

Neurological Findings. Neurological examination revealed diffuse neurological signs in both groups of children with developmental speech delay, supporting the concept of DSD as a condition frequently accompanied by mild but persistent neurological dysfunction [5,9]. However, children with residual brain damage demonstrated significantly higher frequencies of muscle tone abnormalities, altered tendon reflexes, coordination disorders, postural instability, tic-like hyperkinesias, and motor stereotypies ($p<0.05$), which are characteristic markers of early diffuse brain involvement [6,8]. Although the qualitative spectrum of neurological symptoms was similar between the two clinical groups, their severity and persistence were notably greater in children with residual brain damage. This finding indicates a more pronounced involvement of central nervous system regulatory mechanisms and reduced neurodevelopmental compensation capacity in this subgroup [4,7].

Speech Characteristics. All children with developmental speech delay exhibited significant impairment across core speech domains compared to controls ($p < 0.001$), confirming the central role of speech dysfunction in the clinical structure of DSD [1,2]. The most affected parameters included phonetic articulation, active vocabulary, phrase length, and lexical-grammatical organization, which are considered key indicators of expressive speech maturation [12,14].Children with residual brain damage demonstrated significantly lower speech scores than children without residual brain damage in all assessed domains, suggesting a close association between structural-functional brain immaturity and

the severity of speech impairment [3,6]. Parental reports revealed a high prevalence of babbling words, word distortions, limited verbal initiative, reliance on gestures, and difficulties in social communication, particularly in the DSD + RBD group. These features reflect impaired speech programming and reduced integration of speech with cognitive and social functions [4,5,13].

Discussion.

The results of the present study confirm that developmental speech delay is a heterogeneous neurodevelopmental condition strongly influenced by perinatal and early postnatal factors. Residual brain damage significantly aggravates both neurological and speech manifestations of DSD, leading to a more complex clinical phenotype characterized by combined motor, regulatory, and speech deficits [6–8].

The predominance of antenatal and early neonatal risk factors in children with residual brain damage supports the concept of early disruption of cortical-subcortical networks responsible for speech programming, motor coordination, and executive control [4,5]. In contrast, children with DSD without residual brain damage appear to represent a functionally milder variant of speech dysontogenesis, although still associated with significant speech impairment.

The close association between speech disorders and diffuse neurological signs underscores the integrative nature of neurodevelopment and highlights the importance of comprehensive neurological assessment in children with speech delay. These findings emphasize the need for early multidisciplinary intervention aimed at both speech rehabilitation and neurological support.

Conclusion.

Developmental speech delay is characterized by complex clinical and neurological heterogeneity. The presence of residual brain damage is associated with a higher burden of perinatal risk factors, more pronounced neurological dysfunction, and deeper speech impairment.Early identification of high-risk children and comprehensive clinical-neurological evaluation are essential for prognostic stratification and optimization of individualized rehabilitation programs. A multidisciplinary approach may significantly improve long-term neurodevelopmental outcomes in children with developmental speech delay.

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