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FEATURES OF CEREBRAL HEMODYNAMICS IN CHILDREN WITH CONGENITAL HEART DEFECTS

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Abstract. *Congenital heart defects (CHDs) are a leading cause of congenital pathology in children and are a major cause of infant and early childhood morbidity and mortality. In recent decades, advances in cardiac surgery and intensive care have significantly increased the survival rate of children with CHDs, leading to an increase in the number of patients with chronic disease and varying degrees of severity. Current research shows that the severity of CHDs is determined not only by the anatomical defect but also by the degree of hemodynamic impairment, the severity of hypoxemia, and the impact of the defect on systemic and cerebral perfusion. Cyanotic forms of CHD, accompanied by chronic hypoxia, are particularly unfavorable, negatively impacting the development of organs and systems, including the central nervous system.*

Key words: *congenital heart defects, young children, cognitive development, psychomotor development*

ОСОБЕННОСТИ ЦЕРЕБРАЛЬНОЙ ГЕМОДИНАМИКИ У ДЕТЕЙ С ВРОЖДЕННЫМИ ПОРОКАМИ СЕРДЦА

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Аннотация. *Врожденные пороки сердца (ВПС) занимают ведущее место в структуре врожденной патологии у детей и являются одной из основных причин младенческой и ранней детской заболеваемости, и смертности. В последние десятилетия благодаря развитию кардиохирургии и интенсивной терапии значительно повысилась выживаемость детей с ВПС, что привело к увеличению числа пациентов с хроническим течением заболевания и различной степенью тяжести порока. Современные исследования показывают, что тяжесть ВПС определяется не только анатомическим дефектом, но и степенью гемодинамических нарушений, выраженностью гипоксемии и влиянием порока на системную и церебральную перфузию.*

Особенно неблагоприятными являются цианотические формы ВПС, сопровождающиеся хронической гипоксией, которая оказывает негативное влияние на развитие органов и систем, в том числе центральной нервной системы.

Ключевые слова: *врожденные пороки сердца, дети раннего возраста, когнитивное развитие, психомоторное развитие*

ТУҒМА ЮРАК НУҚСОНЛАРИ БЎЛГАН БОЛАЛАРДА МИЯ ГЕМОДИНАМИКАСИНИНГ ХУСУСИЯТЛАРИ

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Изоҳ. *Туғма юрак нуқсонлари (ТЮН) болаларда туғма патологиянинг асосий сабаби бўлиб, гўдаклар ва эрта болалик даврида касалланиш ва ўлимнинг асосий сабабидир. Сўнгги ўн йилликларда юрак жарроҳлиги ва интенсив терапия соҳасидаги ютуқлар ТЮН билан касалланган болаларнинг омон қолиш даражасини сезиларли даражада оширди, бу эса сурункали касалликка чалинган ва турли даражадаги оғирликдаги беморлар сонининг кўпайишига олиб келди. Ҳозирги тадқиқотлар шуни кўрсатадики, ТЮНнинг оғирлиги нафақат анатомик нуқсон, балки гемодинамик бузилиш даражаси, гипоксемиянинг оғирлиги ва нуқсоннинг тизимли ва мия перфузиясига таъсири билан ҳам белгиланади. Сурункали гипоксия билан бирга келадиган ТЮНнинг цианотик шакллари айниқса ноқулай бўлиб, марказий асаб тизимини ўз ичига олган органлар ва тизимларнинг ривожланишига салбий таъсир кўрсатади.*

Калит сўзлар: *туғма юрак нуқсонлари, ёш болалар, конитив ривожланиш, психомотор ривожланиш*

Introduction. Cerebral hemodynamics in children with congenital heart defects (CHD) is characterized by complex disturbances обусловленные both the anatomical features of the defect and systemic changes in circulation. In recent years, most researchers have considered impaired cerebral blood flow as one of the key mechanisms underlying neurological and neurocognitive disorders in this group of patients. According to Goncharova E.A. (2019) and Smirnov V.A. (2020), children with CHD show changes in cerebral perfusion already at early stages of life, manifested by a decrease in linear blood flow velocity and its redistribution among different brain regions. Similar results are presented in the studies by Licht D.J. (2018), who demonstrated that newborns with critical heart defects exhibit reduced cerebral blood flow even before surgical treatment. At the same time, Mebius M.J. (2020) emphasizes that cerebral hemodynamic disturbances are universal in nature and are observed in both cyanotic and acyanotic forms of CHD; however, their severity depends on the type of defect and the degree of hemodynamic impairment [3,4,15].

Under normal conditions, cerebral perfusion is maintained by autoregulatory mechanisms that ensure stable cerebral blood flow

despite fluctuations in systemic arterial pressure. However, as noted by Fedorova L.A. (2018, 2019) and Soul J.S. (2017), these mechanisms are impaired in children with CHD, leading to increased brain sensitivity to changes in systemic hemodynamics. Khalil A. (2016, 2017) demonstrated in his studies that alterations in cerebral blood flow develop already during the prenatal period and are accompanied by redistribution of blood flow with a decreased proportion directed to the brain. These findings are supported by the work of Peyvandi S. (2022), which indicates that impaired cerebral perfusion is one of the key factors in the development of neurodevelopmental disorders in children with CHD. Overall, the results of these studies indicate the systemic nature of cerebral circulation disturbances.

Chronic hypoxia is one of the leading factors contributing to impaired cerebral hemodynamics. Alekseeva T.M. (2021), Kuznetsov A.V. (2017), and Sidorov P.I. (2019) note that reduced blood oxygenation leads to compensatory vasodilation of cerebral vessels; however, these mechanisms do not ensure adequate oxygen delivery to brain tissues. In international studies, Goff D.A. (2019) showed that chronic hypoxia leads to structural brain changes, including white matter injury. Rollins C.K. (2022) and Newburger J.W. (2022) emphasize that hypoxia disrupts neuronal migration and myelination processes. Limperopoulos C. (2019, 2023) demonstrated that children with CHD exhibit slowed brain growth and reduced brain volume associated with impaired oxygen supply (215, 220). Similar findings are reported by Kelly C.J. (2022) and Watson C.G. (2023), who point to delayed maturation of the cerebral cortex. Thus, the impact of chronic hypoxia on brain development is supported by a wide range of studies [1,5,7].

Disorders of cerebral hemodynamics are closely associated with changes in neural tissue metabolism. Alekseeva T.M. (2021) and Emelyanova N.V. (2018) note that under hypoxic conditions there is a shift to anaerobic metabolism with lactate accumulation and the development of acidosis (1, 18). Sun L. (2018, 2019) showed that fetuses with CHD have reduced cerebral oxygen consumption, which is accompanied by decreased brain volume (352, 353). McQuillen P.S. (2017) and Miller S.P. (2018) emphasize that metabolic disturbances play a key role in white matter injury. Panigrahy A. (2019, 2021) demonstrated a relationship between metabolic changes and structural brain abnormalities detected by MRI. In summary, metabolic disturbances represent an important link in the pathogenesis [12,13].

Systemic hypoxemia and heart failure significantly exacerbate cerebral hemodynamic disturbances. Petrova N.N. (2019) and the clinical guidelines of the Ministry of Health of the Russian Federation (2021) indicate that reduced cardiac output leads to decreased cerebral perfusion and the development of ischemia. Mebius M.J. (2020) and McElhinney D.B. (2017) note that the combination of hypoxemia and hypoperfusion is the most unfavorable factor leading to central nervous system injury [2,14]. Licht D.J. (2018) and Peyvandi S. (2022) emphasize that these disturbances are particularly pronounced in children with critical heart defects. During the perioperative period, these changes may be further aggravated. Andropoulos D.B. (2017, 2018) and Wypij D. (2021) showed that fluctuations in systemic hemodynamics during surgical interventions additionally impair cerebral perfusion [6,10,11]. These findings are supported by multiple studies.

Analysis of the literature indicates that disturbances of cerebral hemodynamics in children with congenital heart defects are formed under the influence of a complex of interrelated factors, including chronic hypoxia, impaired autoregulation of cerebral blood flow, metabolic disorders, and systemic hemodynamic changes. Comparison of different studies shows that pathological alterations in cerebral perfusion begin already during the prenatal period and persist postnatally, becoming more pronounced under the influence

of surgical treatment. Despite the substantial body of accumulated data, questions remain regarding individual variability of cerebral hemodynamics and adaptive mechanisms, which substantiates the need for further research and the development of methods for early diagnosis of cerebral circulation disorders in children with CHD.

Thus, disturbances of cerebral hemodynamics in children with congenital heart defects represent a complex, multilevel syndrome that includes motor, cognitive, and behavioral disorders. A comparison of international studies indicates a high prevalence of these disturbances, their multifactorial nature, and their significant impact on patients' quality of life. Current perspectives emphasize the importance of early detection of neurodevelopmental disorders and the need for a comprehensive, multidisciplinary approach to diagnosis and rehabilitation, thereby underscoring the relevance of further research in this field.

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