

Pathology in children with congenital cleft lip and palate.

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Abstract: In children with congenital cleft lip and palate (CCLP), under the influence of endogenous (hereditary) or exogenous (causing disruption of embryogenesis) factors, changes occur in various organs and systems of the body. The commonality of CGN with multiple organ pathologies in the body is explained by the presence of connective tissue dysplasia (CTD). The formation of the multifactorial nature of CGN is realized due to the genetic susceptibility of the organism to the action of externally damaging environmental factors in relation to the fetus. In 75% of children, CRHN is an isolated congenital defect, but it can be combined with other congenital defects.

The data presented in the article may be useful for the development of preventive and therapeutic measures, in the prognosis and management of children with this pathology from an individual professional and family perspective.

Key words: children, etiology, cleft lip and palate, factors, defects, deformation of the maxillofacial area

Introduction. Congenital cleft lip and palate (CWPN) is one of the most common human malformations. Among congenital malformations, the share of VARHN is 20–30%. It is diagnosed in an average of 1 in 1000 newborns. The number of children with BPH is constantly increasing. Over the past 100 years, the incidence of this defect has increased 3 times. Growth is associated with an increase in teratogenic and genetic factors WGN can be detected in many diseases, monogenic, chromosomal, multifactorial forms. In most cases, the nature of VRHN is multifactorial, associated with the simultaneous impact of genetic and environmental influences. According to clinical and genealogical studies, in 85% of children VRHN is a multifactorial malformation, in 15% it is hereditary (monogenic or chromosomal in nature). The hereditary origin of VRHN is mainly due to gene mutations. The proportion of chromosomal mutations is only 7–8% [1; 3; 14]. The formation of the multifactorial nature of VRHN is realized by the genetic susceptibility of the organism to the action of externally damaging environmental factors in relation to the fetus. In 75% of children, VRGN is an isolated birth defect, but it can be combined with other birth defects. Changes in the anatomical and topographic structures of the palate and nasopharynx are accompanied by hypertrophy of the tongue root, disorders of the Eustachian tube and middle ear, disorders of sucking, swallowing, breathing, hearing, and speech. In many children, pathology of the immune, bronchopulmonary, nervous and gastrointestinal systems, as well as the gastrointestinal tract is detected [11; 12; 13]. Cleft lip and/or palate is a severe malformation of the maxillofacial region, manifested by a violation of the continuity of the upper lip, alveolar process and palate, and accompanied by significant functional disorders. Difficulties in restoring impaired vital functions of nutrition, breathing and speech, anatomical restoration of the upper lip, nose and upper jaw in the conditions of a growing organism are the reason for the disability of children with cleft lip and palate for many years [1; 4].

In this malformation of the face, morphological features are revealed in the antenatal period, and after birth, disturbances of the following vital functions are observed:

- 1) malnutrition: normal sucking during newborns and infancy is impossible in most cases due to the lack of separation of the nasal and oral cavities, and subsequently chewing disorders are associated with anomalies of the dentition and occlusion disorders;
- 2) speech: the lack of integrity of the velopharyngeal sphincter makes it difficult to form correct speech (speech is slurred, quiet with a nasal tinge), and may be the cause of delayed speech development
- 3) hearing: cleft and incorrect attachment points of the palatal musculature cause dysfunction of the Eustachian tube;
- 4) nasal breathing: deformation of the terminal and osteochondral part of the nose leads to a change in air flow, disrupts adequate breathing and contributes to the development of various concomitant pathologies of the ENT organs. A cosmetic defect in the form of a violation of the anatomy of the upper lip, deformation of

the nose at birth causes a negative reaction from parents, family, and subsequently people around you. The harmonious development of a child with a cleft is difficult due to his cosmetic defect and functional disorders [1; 14].

Objective: The aim of the study was to identify etiological risk factors and the mechanism of RHN development, as well as to analyze the frequency of occurrence of this pathology.

Material and methods of research. According to the literature and numerous research data, 70–85% of children with VRHN have signs of perinatal encephalopathy, which occurs in most cases with syndromes of intracranial hypertension and increased neuroreflex excitability. Cytological examination of the cerebral cortex in children with perinatal encephalopathy revealed significant changes in the spatial organization of the pre-central cortex. It can be assumed that the areas of the brain Those responsible for motor functions lag behind in their development due to the disruption of the embryogenesis process. The relationship between the anatomical structures of the brain and the facial structures is often the cause of neuropsychiatric disorders in children with GLN. According to the data, such children have a decrease in the level of social adaptation, impaired emotional development, the formation of negative self-esteem, and communication difficulties. In this regard, in order to improve the quality of life of children with developmental disabilities, it is necessary to create a special individual educational program aimed at the timely correction of neuropsychiatric disorders. [1; 9; 11].

Results and discussion. The results of the study showed that children with VRHN are 5 times more likely to have manifestations of DST from the cardiovascular system in the form of overgrowth of connective tissue, the development of microcirculatory disorders, dystrophic changes in cardiomyocytes. In the tissues of the upper lip on the side of the cleft there are signs of disorganization of connective tissue (dystrophic changes in muscle fibers, their partial replacement by connective tissue structures with the development of focal sclerosis). The main essence of the pathogenesis of DST in children with VRHN is energy dysfunction associated with metabolic disorders and insufficient energy production in the mitochondria. In this regard, in the course of many somatic diseases, in organs and tissues with high energy needs, significant disorders of energy metabolism occur with signs of oxidative stress and hypoxia. [11; 14].

Conclusion. The urgency of this problem increases due to the proven need for surgical treatment of children at an earlier age, starting from the neonatal period [5, 3, 12, 13]. Primary cheilorhinoplasty in the neonatal period makes it possible to normalize the myodynamic balance of the muscles of the oral region at an earlier time and thereby eliminate the effect of the deforming factor on the middle zone of the face, which leads to a more correct formation of the facial skeleton. Recovery anatomical integrity of the palate prevents the development of hearing loss. Early surgical intervention contributes to the formation of correct breathing, sucking, swallowing and the development of speech, the formation of a normal biocenosis of the nasopharynx and "local" secretory immunity, the prevention of the occurrence of frequent ARVI and chronic diseases of the nasopharynx, the improvement of the physical parameters of the child, etc. Analyzing the data of literature sources and our own data, we can conclude that the pathogenetic essence of VRHN is DST that arises in the process of embryogenesis. The development of DST is associated with the influence of external environmental and genetic factors. Fetal VRHN is formed under the influence of external environmental factors in the presence of a genetic predisposition. [1, 3, 5, 7].

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