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The Effect of Changes in the Cardiovascular System on the Organs of the Oral Cavity in Children With Disabilities

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Annotation: The problem of cardiovascular diseases is one of the most important problems in pediatric practice due to the variety of clinical forms, the complexity of diagnosis and treatment, as well as the predisposition to the formation of a heart defect (wool). Currently, heart defects are one of the main causes of childhood disability and death. According to modern concepts, heart failure in children is a progressive clinical and pathophysiological syndrome that occurs as a result of cardiac or extracardial causes, leads to characteristic symptoms (edema, respiratory failure, impaired physical development, inability to perform physical exercises) and is accompanied by hemodynamic changes, enters the Saras of structural restructuring of the heart, neurogumoral and molecular diseases.

Rossano J. (2012) states in his work that heart failure (58% to 70%) in most cases is diagnosed in children of the first year of life with congenital heart defects. Cardiomyopathies are the most common cause of heart failure in children whose hearts are normally formed. Currently, the prevalence of heart failure in children is increasing, which is due to advances in the surgical treatment of complex congenital heart defects and an increase in the life expectancy of children with cardiomyopathy against the background of modern therapy methods. In the process of studying heart failure, the pathophysiological concepts that underlie its appearance and development have also changed. In the 1950s, the" weak myocardial" theory prevailed, directing the say-actions of doctors to increase the contractile function of the heart with the main drug of the time - cardiac glycoside. Later, this theory was replaced by cardiorenal conception of the development of a heart defect, which explained the appearance of edema syndrome by the fact that the heart cannot provide adequate renal blood flow. This model of cardiac defect pathogenesis justified the treatment of patients with diuretic drugs [1.2]. In the 1970s and 1980s, heart failure was largely considered as a hemodynamic disorder, and the "cardiosirculation" model was formulated. In this model, a decrease in the systolic function of the heart and a change in the tone of peripheral arteries and veins played an important role with a further increase in previous and subsequent loads. This theory served as the basis for the practical introduction of peripheral vasodilators and non-glycosidic inotropic drugs [2.4.6.8].

The modern theory of the development of heart failure - neurogumoral - determines the main role in the activation of local or tissue neurogormones as a result of stretching of myocardial fibers or their damage. These mainly cover the sympathetic-adrenal system (SAT) and its effectors - angiotensin II and aldosterone, the renin-angiotensin-aldosterone system (Raat) and its effectors - angiotensin II and aldosterone, as well as the natriuretic peptide system (NPT) acting against it. The sympathetic-adrenal system is activated in the early stages of cardiac activity, due to autonomic

balance, a deficiency is caused. Raat is then activated due to renal hypoperfusion due to low cardiac output and increased renin due to sympathetic stimulation of the kidneys. In the short term, these mechanisms adapt: the heart rate increases, blood pressure and cardiac output increase, thereby maintaining the blood supply to the organs. Over time, these reactions become incompatible, with long-term neurogormone hyperactivation leading to myocyte hypertrophy, apoptosis, fibroblast proliferation, interstitial collagen accumulation, myocardial remodeling, and decreased ability of cardiac ventricles to contract. Currently, the neurogumoral model is widely recognized and confirmed by the results of numerous studies, which are enzyme inhibitors that convert the heart to angiotensin (aafi), showing the survival of patients with heart failure as a result of neurogumoral unloading with angiotensin receptors. Angiotensin, 0-adrenoblocagorami, is included in the spironolactone-blocking receptor class. However, a number of studies have shown flaws in this model. In particular, in the work of Mann D and others (2002), despite complete treatment with 0-adrenoblocagorami, spironolactone-blocking receptors, and angiotensin-converting enzyme inhibitors, the heart defect in most patients continues to develop, albeit at a slower rate [1.3.5].

Later, in addition to neurogormones, it was found that immune mechanisms also play a role in the development of heart defects, the involvement of which determines the "incomplete authority" of the neurogumoral theory. Levine B. A study by (1990) for the first time demonstrated that increased tumor necrosis factor-A (O'no-A) levels in patients were associated with cardiac defect severity. Based on numerous works on this topic, a new theory of the development of a common heart defect was proposed, based on the idea of activation of immunity and systemic inflammation. According to this concept, the non-specific activation of macrophages and monocytes performed in severe disorders of microcirculation determines the evolution of anti-inflammatory cytokine (TNF-a, interleukin-1, interleukin-6, etc.) dysfunction. The action of cytokines has a negative inotropic effect, the development of cardiac remodeling in the form of destruction of the collagen matrix, the emergence of ventricular expansion, cardiomyocyte hypertrophy, increased apoptosis and impaired endothelial-dependent relaxation of arteries [2.4.6.8.10].

Currently, the development of heart failure is explained not only by damage to the myocardium, but also by damage to the endothelium of the vascular wall. In 1998, Murad F., Furchgott R. and Ignarro l was awarded the Nobel Prize in medicine for studying the role of endothelium in the pathogenesis of various cardiovascular diseases and for discovering the endothelial relaxation factor-nitric oxide(no). Recent studies have reliably shown the important and independent role of endothelium in the development of cardiovascular pathology [1.3.5.7.9]. Endothelial vasodilators and antiagregant agents (NO, prostacyclin, bradykinin, prostaglandin F2a, endothelial hyperpolarization factor), vasoconstrictors, and proaggregants (endothelin-1, angiotensin II, serotonin, prostaglandin F2a, leukocyclines, leukotans, leukotrimins, leukotrieni S4, D4, thromboxane A2) are the factors that develop releases. Some researchers associate the manifestation of endothelial dysfunction with an imbalance between vasodilating, angioprotective, antiproliferative factors on the one hand and the production of vasoconstrictor, prothrombotic, proliferative factors on the other. Other theories are related to the lack or presence of NO production in the arterial wall [11.13.15.17.19].

The main role in the mechanism of development of endothelial dysfunction is played by oxidative stress, strong vasoconstrictors (endoperoxides, endothelins, angiotensin II), as well as the production of cytokines and TNF-a, which suppress the production of NO. With prolonged exposure to harmful factors (hemodynamic overload, hypoxia, intoxication, inflammation), endothelial function is impaired and impaired, vasoconstriction, increased vascular wall elements, and thrombus formation are responses to general stimuli. Determining the importance of hereditary factors in the

pathogenesis of heart defects and the development of gene technologies opens up great opportunities for predicting a heart defect and developing new approaches to its treatment. About 30% of patients with hereditary diseases have hereditary syndromes caused by chromosomes (Downa, Ternera, Di-Djordji syndrome) or gene mutations (Nunan, Alajilya, holta-orama syndrome). For the remaining 70% of patients with hereditary diseases, genes (dominant or recessive hereditary) that affect embryonic development and regulation of cardiovascular systems have been identified and studied in recent years. Mutations that alter protein function in one of these genes, under certain conditions, can lead to the appearance of various hereditary diseases. In addition, these genes play a role in regulating cardiac activity throughout life [10.12.14.16.18.20].

Conclusion. Genetic factors determine the response to drug therapy, which is important for the clinical course and treatment of heart defects. At the same time, the main mechanisms responsible for changes in the myocardium are related to polymorphism of the angiotensin-converting enzyme (AAF) gene, pathology of the dystrophin gene, and mutation of the actin gene. Polymorphisms in genes encoding proteins involved in drug metabolism, such as CYP450 liver enzymes, alter the response to a number of pharmacological agents used to treat heart defects. The modern concept of pathogenesis of heart failure combines all of the above concepts based on complementarity, since all disorders in a patient with heart failure cannot be explained using a single theory of pathogenesis and even more so solved.

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