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### **PATHOGENETIC FEATURES OF BRONCH-OBSTRUCTIVE SYNDROME IN CHILDREN**

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**Abstract:** Bronchial obstructive syndrome (BOS) in children has been attracting the attention of both researchers and medical practitioners for many years, due to the widespread and heterogeneous nature of its development, and therefore, difficulties in differential diagnosis. Of particular difficulty is the differentiation of various manifestations of airway obstruction in young children due to the high incidence of biofeedback as the main syndrome, secondary syndrome, and even iatrogenic.

**Keywords:** bronchial obstructive syndrome, infectious pathology, inflammation, hypersecretion;

Bronchial asthma (BA) and various acute bronchial obstructive pathologies - obstructive bronchitis (OB), bronchiolitis, acute stenosing laryngotracheitis (OSLT - false croup), whooping cough - diseases in which violation of the airway, especially the bronchi, is the main manifestation. The identification and treatment of biofeedback is a key point in the diagnostic and therapeutic algorithms of these diseases [1,2]. The term "bronchial obstruction" refers to a pathological condition resulting from impaired patency of the bronchi with subsequent increase in resistance to air flow during ventilation and characterized by episodes of shortness of breath as a result of bronchoconstriction, inflammatory infiltration, hypersecretion and dyskrinia of the submucous glands, edema and hyperplasia of the mucous membrane of the respiratory tract, in particular [1, 3]. From a biological point of view, bronchial obstruction has a protective and adaptive nature, preventing the penetration of various foreign agents, including infectious pathogens and allergens, into the alveoli, thus preventing the development of pneumonia, and therefore is a universal mechanism for the protection of

the respiratory tract through inflammation, hypersecretion of mucus, mucociliary transport, cough and even bronchospasm [3,4].

**Pathogenesis.** During the formation of biofeedback, inflammation of the airways develops with the participation of epithelial and endothelial cells, granulocytes, macrophages, monocytes, and there may be T-cell activation in response to an infectious antigen or allergen [2]. As a result of inflammation, a violation of the geometry of the small bronchi is formed due to thickening of the wall, closure of the lumen by mucus and cellular detritus, increased release of pro-inflammatory cytokines, increased bronchial hypersensitivity, impaired neuroregulatory mechanisms in connection with parasympathetic hyperreactivity [5,7]. Violation of mucociliary clearance (the process of cleansing the respiratory tract) occurs both with excessive sputum formation and with insufficient sputum, which leads to stagnation of sputum, as a result of which the bronchial drainage function suffers and the ventilation function of the lungs is impaired, the effectiveness of the protective mechanisms, mucociliary transport and cough, are reduced. Colonization of the bronchi by pathogenic microflora is growing. Violation of sputum transport leads to the maintenance and progression of inflammation and bronchial obstruction [6,10].

Clinically, the degree of impaired bronchial patency with various pathologies depends on the ratio of individual components of bronchial obstruction, the presence of genetically determined bronchial hyperreactivity, the characteristics of causative factors and inflammation. The most important pathophysiological components of acute BOS in children are edema of the bronchial mucosa, mucus hypersecretion and bronchospasm [8,9].

With the protracted nature of BOS, hyperplasia of the mucous membrane develops, and with chronic inflammation typical of bronchial asthma and bronchopulmonary dysplasia (BPD), the phenomena of fibrosis and sclerosis gradually form, which indicates structural restructuring of the bronchi (remodulation) [7, 8]. Most often and clinically bright, BFB is manifested in children of the first years of life, which is due to morphofunctional features of the respiratory system: narrow airways, insufficient elasticity of the lungs, soft cartilage of the bronchial tree, insufficient stiffness of the chest, tendency to develop edema, hypersecretion of viscous mucus, weak development of smooth muscles of the bronchi [3, 6]. A special place in the formation of increased bronchoreactivity and the development of biofeedback is occupied by neuroreflective mechanisms, the basis of which is the functioning of the autonomic nervous system. Autonomic effects in young children are differently represented in different organs, tend to generalize, are very labile, quickly transfer from one system to another [7]. In healthy infants,

the tone of the parasympathetic section of the autonomic nervous system (vagotonia) predominates, which causes narrowing of the bronchi, vasodilation, sweating, peristalsis and an increase in the tone of the digestive organs, which is manifested by pastiness, the development of edema and hyperproduction of dense secretions, gastroesophageal disease, and gastroesophageal disease development of biofeedback. Under conditions of vagotonia, the normal balance between bronchoconstriction and bronchodilation with the involvement of various receptors is disturbed. This dysfunction can lead to the development of reflex bronchospasm and bronchial hyperreactivity, leading to the formation of non-allergic AD mechanisms.  $\beta$ 2-adrenergic receptors are abundantly present in the airways and are present on smooth muscle cells, epithelial cells, submucosal gland cells, numerous inflammatory cells, in alveoli and presynaptic nerves [3,5,6]. The function of the  $\beta$ 2-adrenergic receptor depends on the connection with the Gs protein, stimulated by adenylate cyclase, which increases the level of cyclic adenosine monophosphate in the cell, the high content of which, in addition to relaxing the smooth muscles of the bronchi, inhibits the release of mediators of an immediate hypersensitivity reaction from inflammatory cells (primarily from mast cells), which is important for children with atopy. After this, the sensitivity of  $\beta$ -adrenergic receptors to further stimulation decreases. Another group of receptors is muscarinic (cholinergic). Of their 5 types, M1, M2, and M3 receptors are present in the lungs. The tone of the muscles of the bronchi is determined mainly by parasympathetic (cholinergic) innervation (vagotonia) and is significantly enhanced in bronchial asthma. The neurotransmitter acetylcholine, released in the nerve endings through cholinergic receptors, leads to contraction of smooth muscles and enhances the secretion of submucosal glands. Triggers for exacerbation of the process (histamine, cold air, physical activity) initiate obstruction by a direct effect on the receptors and activation of the cholinergic pathway, increasing bronchial hyperreactivity. Smooth muscle cells are capable of producing interleukins, growth factor, and pro-inflammatory cytokines that can initiate, provoke, or support inflammation. In response to sensitization,  $\gamma$ -interferon is released in smooth muscle cells, which is able to interact with the M2 receptor, inhibiting its function, which subsequently leads to an increase in the release of acetylcholine [3,4,5,6,7].

Thus, the anatomical and physiological characteristics of the respiratory system and the vegetative orientation of young children determine the significant frequency of biofeedback and the characteristic features of its clinical manifestations. So, the leading one in the development of bronchial obstruction in OB is pronounced edema of the bronchial mucosa and

hypersecretion of viscous mucus, in contrast to bronchial asthma, in which bronchospasm is the basis of bronchial obstruction [7].

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**FROM THE HISTORY OF THE FORMATION OF THE INTERNATIONAL  
CULTURAL COOPERATION OF THE PEOPLES OF CENTRAL ASIA (ON THE  
EXAMPLE OF UZBEKISTAN WITH KAZAKHSTAN)**

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