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ASSESSMENT OF THE MORPHOFUNCTIONAL CONDITION OF THE ORAL CAVITY IN FACIAL NERVE PALSY

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Abstract. This comprehensive scientific study is devoted to investigating the destructive and functional changes occurring in the organs and tissues of the oral cavity as a result of various degrees of damage to the facial nerve (VII cranial nerve). The article analyzes in detail the biomechanical consequences of mimic muscle atony, neurogenic impairment of the secretory activity of salivary glands, as well as the transformation of oral microflora and local immunity[1,2,3].

Based on international neurostomatological protocols from 2024–2026, the diagnostic effectiveness of electromyography, laser Doppler flowmetry, and sialography is demonstrated with statistical evidence.

Keywords: N. facialis, Bell’s palsy, m. orbicularis oris, xerostomia, hyposalivation, neurogenic atrophy, electromyographic amplitude, microcirculation index, periodontal status, dysarthria.

INTRODUCTION

Facial nerve paralysis is not only a neurological disorder, but also a serious stomatological problem. According to the 2025 report of the World Health Organization (WHO), 92% of patients with peripheral facial paralysis experience varying degrees of impairment in oral functions, including mastication, speech, and salivation[4,5,6].

Approximately 40% of these patients develop chronic dental diseases, such as dental caries, periodontitis, and oral candidiasis, within the first six months following the onset of paralysis.

Early and accurate evaluation of the morphofunctional condition of the oral cavity is therefore critically important not only for restoring nerve function but also for preventing irreversible destructive changes in oral tissues.

The morphological structure of the facial nerve determines its central role in the functioning of the oral cavity[7,8]. The chorda tympani branch of the nerve provides parasympathetic innervation to the submandibular and sublingual salivary glands. When paralysis occurs, disruption of these nerve fibers leads to impaired conduction, resulting in the cessation of secretory granule formation in the acinar cells of salivary glands.

Morphologically, this process may lead to serous–mucous transformation of glandular tissue, followed by fibrotic degeneration. According to statistical data, in severe paralysis the viscosity of saliva increases by approximately 3.5 times, which completely disrupts the self-cleansing mechanism of the oral cavity[9,10].

One of the main indicators of the morphofunctional condition of the oral cavity is the state of the mimic muscles, particularly the orbicularis oris muscle (m. orbicularis oris). Atony of this muscle prevents hermetic closure of the lips, which leads to impairment of labial and labiodental articulation.

Electromyographic (EMG) studies show that within the first 48 hours of paralysis, the amplitude of bioelectrical potentials in muscles decreases from the normal 500–700 μV to 30–40 μV. If electrical silence persists on EMG after 21 days, it indicates the development of neurogenic muscle atrophy. In such cases, muscle tissue is gradually replaced by connective tissue, leading to the formation of muscle contractures[11,12].

Table 1. Morphofunctional condition of oral cavity organs in facial nerve paralysis (2025 statistics).

Organ/System	Morphological change	Functional consequence	Assessment method

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Salivary glands	Acinar atrophy	Hyposalivation (dryness)	Sialometry, pH-metry
Buccinator muscle	Neurogenic hypotrophy	Mastication efficiency −60%	Masticatiography
Tongue (anterior 2/3)	Degradation of taste receptors	Ageusia (loss of taste)	Gustometry
Mucous membrane	Epithelial desquamation	Stomatitis, ulcers	Cytological smear
Blood vessels	Capillary spasm	Hypoxia, trophic disorders	Laser Doppler flowmetry

In assessing morphofunctional status, the biomechanics of the masticatory apparatus also plays a significant role. Due to dysfunction of the buccinator muscle (m. buccinator) on the paralyzed side, the food bolus accumulates in the vestibule of the oral cavity between the cheek and teeth[13,14]. This leads to retention and decomposition of food debris, creating favorable conditions for the proliferation of cariogenic microflora such as Streptococcus mutans.

Microbiological studies conducted in 2026 demonstrated that the dental caries index (DMFT) on the paralyzed side is 2.4 times higher than on the healthy side.

The biochemical composition of saliva also serves as an important indicator of the severity of nerve damage. In facial nerve paralysis, the balance of calcium and phosphorus ions in saliva becomes disrupted, reducing the remineralization capacity of tooth enamel[15,16].

Additionally, decreased levels of enzymes such as kallikrein and lysozyme reduce the antibacterial resistance of the oral mucosa. According to statistics, 55% of patients with Bell's palsy develop catarrhal gingivitis within the first month after the onset of the disease.

Studies of oral mucosal microcirculation using Laser Doppler Flowmetry (LDF) revealed that the perfusion index decreases from the normal 18–20 p.u. to 10–12 p.u. on the affected side[17]. This indicates the presence of chronic tissue hypoxia and slowed regenerative processes. Morphologically, this condition is characterized by perivascular edema and plasmorrhagia of small blood vessels.

Table 2. Effectiveness and predictive value of instrumental diagnostics

Diagnostic method	Sensitivity (%)	Specificity (%)	Clinical significance
Surface EMG	94	88	Detection of muscle recovery
Sialography	82	75	Evaluation of gland duct patency
Gustometry	76	91	Detection of taste neuropathy
3D facial scanner	98	95	Measurement of facial asymmetry

During the rehabilitation process, the assessment of morphofunctional status should be dynamic and continuous. According to 2026 neurostomatological standards, facial movements and the position of the oral commissure should be evaluated every two weeks using the House–Brackmann scale[18].

If morphofunctional indicators do not recover within three months, this may indicate the need for surgical decompression of the facial nerve or nerve–muscle plastic surgery.

In particular, morphological imbalance between the right and left sides of the maxillofacial system may lead to compensatory hypertrophy of the masticatory muscles (m. masseter). Since patients tend to chew on the healthy side, this may eventually result in temporomandibular joint (TMJ) dysfunction and pain syndrome.

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Statistical analyses show that 28% of patients with long-term facial nerve paralysis develop pathological displacement of the joint condyle and articular disc.

CONCLUSION

Facial nerve paralysis is not merely a disorder of facial expression, but rather a complex morphofunctional crisis of the oral cavity. Decreased salivary gland function, muscle atrophy, and impaired mucosal microcirculation mutually aggravate each other.

Modern instrumental diagnostic methods such as electromyography, laser Doppler flowmetry, and 3D facial scanning allow precise evaluation of the depth of pathology and facilitate the development of individualized treatment plans.

In modern medical practice in 2026, the neurostomatological approach plays a key role in achieving complete social and functional rehabilitation of patients.

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