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KEY PATHOPHYSIOLOGIC FACTORS DETERMINING RECOVERY IN FACIAL NERVE LESIONS: CURRENT SCIENTIFIC UNDERSTANDING

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Abstract: Anatomical features of the facial nerve largely determine its vulnerability to various pathogenic effects. A complex tortuous passage through the bony canal of the temporal bone, the presence of narrow areas (especially in the area of the labyrinthine segment, where the diameter of the canal exceeds the diameter of the nerve by only 0.1-0.2 mm), relatively poor blood supply to the intracanal part — all these factors create prerequisites for nerve compression with minimal edema or inflammatory reaction. On the other hand, the presence of about 10,000 axons in the nerve and its multifunctionality (motor, parasympathetic, sensory and gustatory fibers) determine the variety of clinical manifestations in its lesion.

Key words: facial neuropathy, Bell's palsy, pathophysiology, axonal regeneration, neuroplasticity, neuroinflammation, neurotrophic factors, electroneuromyography, fMRI, synkinesias, prognostic biomarkers, cortical reorganization, BDNF, cytokines, personalized therapy.

Introduction. The etiology of facial neuropathy is heterogeneous and includes idiopathic (Bell's palsy) and secondary forms. The idiopathic form accounts for 60-75% of all cases, while secondary forms can be caused by various factors: traumatic injuries, infectious agents (herpes simplex virus, Varicella-Zoster virus, borrelia), tumor processes (auditory nerve neurinoma, cholesteatoma), metabolic disorders (diabetes mellitus) and autoimmune diseases (Guillain syndrome-Barre, sarcoidosis) [1].

The pathogenesis of facial neuropathy is a complex cascade of interrelated processes. Regardless of the primary etiological factor, the key mechanism of neuropathy development is nerve compression in the bone canal due to edema and inflammatory reaction. Compression leads to impaired microcirculation, which exacerbates edema and contributes to the development of ischemia of the nerve trunk. Ischemia, in turn, activates the processes of lipid peroxidation and oxidative stress, which leads to damage to the myelin sheath and, with prolonged exposure, to axonal degeneration. Depending on the degree and duration of ischemia, the damage may be limited to segmental demyelination (favorable prognosis) or progress to Wallerian degeneration (unfavorable prognosis with a high risk of incomplete recovery and complications)[3].

Facial neuropathy is one of the most common mononeuropathies characterized by damage to the VII pair of cranial nerves, followed by the development of paresis or paralysis of facial muscles. Epidemiological data indicate the frequency of this pathology in the range of 20-35 cases per 100,000 population per year, with no significant gender differences and the possibility of manifestation at any age. The social significance of the problem is due not only to the high prevalence of the disease, but also to its significant impact on the quality of life of patients, their psycho-emotional state and social adaptation[4].

The idiopathic form of facial neuropathy (Bell's palsy) accounts for about 60-75% of all cases, while secondary forms can be caused by traumatic injuries, infectious agents (herpes simplex virus, Varicella-Zoster virus, borrelia), tumor processes, metabolic disorders and autoimmune diseases. Regardless of the etiological factor, the initial link in pathogenesis is nerve compression in the bone

canal due to edema and ischemia, which triggers a cascade of pathophysiological reactions leading to demyelination and, in severe cases, axonal degeneration[5].

The clinical picture of facial neuropathy is characterized by acute or subacute development of peripheral paresis of facial muscles with typical symptoms: facial asymmetry, inability to wrinkle the forehead, close the eye and show teeth on the affected side. Depending on the level of the lesion, concomitant symptoms may occur: impaired lacrimation, taste sensitivity, hyperacusis, and impaired salivation[6].

Despite the fact that the majority of patients (approximately 70-85%) experience spontaneous restoration of facial nerve function within 3-6 months, in a significant proportion of patients the recovery process is incomplete or accompanied by the development of complications such as contractures, pathological synkinesia and hemifacial spasm. Prognostically unfavorable factors are considered to be old age, complete paralysis of the facial muscles, a marked decrease in the amplitude of the M-response during electroneuromyography (more than 90% of the norm), the absence of clinical signs of recovery within 3 weeks of the onset of the disease, as well as the presence of pain behind the ear at the onset of the disease[7]. The pathophysiological mechanisms that determine the nature and degree of restoration of facial nerve function remain the subject of active research. Modern concepts consider the healing process as a complex interaction between local factors in the area of nerve damage (inflammation, ischemia, oxidative stress, demyelination, axonal degeneration and regeneration) and central mechanisms of neuroplasticity, covering various levels of the nervous system from the facial nerve nucleus to the primary motor cortex and associative areas of the cerebral cortex[8].

Data are accumulating on the significant role of neuroimmune interactions in the pathogenesis of facial neuropathy and subsequent recovery. Changes in the profile of pro- and anti-inflammatory cytokines, activation of microglia and macrophages, migration of T-lymphocytes to the affected area – all these processes can both contribute to further nerve damage and create a favorable environment for regeneration, depending on the specific immunological phenotype of the reaction[9]. In recent years, special attention has been paid to the study of the molecular mechanisms of axonal regeneration and remyelination, including the role of neurotrophic factors (nerve growth factor NGF, brain-derived neurotrophic factor BDNF, neurotrophin-3), signaling molecules (Wnt, Notch, Sonic hedgehog), transcription factors (c-Jun, STAT3, ATF3) and extracellular components the matrix. Understanding these mechanisms opens up prospects for the development of targeted therapeutic strategies aimed at stimulating regenerative processes and preventing the formation of pathological reinnervation patterns[7]. No less important are the processes of central neuroplasticity, which compensate for functional deficits. Modern methods of functional neuroimaging (fMRI, PET) make it possible to visualize the reorganization of cortical representations of facial muscles after peripheral damage to the facial nerve. Various patterns of cortical reorganization associated with favorable and unfavorable restoration of function have been identified, which indicates the possibility of using neuromodulation methods to optimize the processes of central plasticity[1].

The clinical heterogeneity of the outcomes of facial nerve neuropathy (from complete recovery to persistent deficiency with complications) indicates the existence of individual characteristics of pathophysiological processes that determine the course of the disease. Genetic factors, age-related changes, concomitant diseases, and previous damage to the nervous system can modify both local processes in the area of nerve damage and central mechanisms of neuroplasticity, which necessitates a personalized approach to prognosis and therapy [2]. Despite significant progress in understanding individual pathophysiological aspects of facial neuropathy, the overall picture of the mechanisms determining the nature and degree of function recovery remains incomplete. The lack of a comprehensive understanding of the relationship between local processes in the area of nerve damage and the central mechanisms of neuroplasticity makes it difficult to develop effective therapeutic strategies and sound prognostic criteria. The clinical picture of facial neuropathy is characterized by acute or subacute development of peripheral paresis of the facial muscles of the corresponding half of the face. Typical symptoms are facial asymmetry at rest and during movement, the inability to furrow

the forehead, close the eye completely (Bell's symptom), and difficulty baring teeth on the affected side. Depending on the level of damage, concomitant symptoms may occur: hyperacusis (when a nerve is affected proximal to the branch of the stapes muscle), impaired lacrimation (when the large stony nerve is involved), impaired taste sensitivity in the anterior two-thirds of the tongue (when a nerve is affected proximal to the drum string), impaired salivation (when secretory fibers are involved in the submandibular and sublingual salivary glands)[3].

The diagnosis of facial nerve neuropathy is based on the characteristic clinical picture, electroneuromyography (ENMG) data and, if necessary, neuroimaging methods to exclude the secondary nature of the lesion. ENMG allows you to determine the degree of nerve damage (neuropraxia, axonotmesis, neurotmesis), assess the dynamics of recovery and predict the outcome of the disease. Of particular value is the determination of the amplitude of the M-response in the acute period (a decrease of more than 90% from the norm is associated with an unfavorable prognosis) and the identification of signs of denervation activity (fibrillation potentials and positive acute waves) with needle EMG. Neuroimaging techniques (MRI, CT) are used in cases of atypical course of the disease, recurrent symptoms, and suspected bulky formation[4].

The course and prognosis of facial neuropathy vary widely. The majority of patients (approximately 70-85%) experience spontaneous restoration of function within 3-6 months. However, in some patients, the recovery process is incomplete or accompanied by the development of complications. The most common complications include: contracture of facial muscles (persistent tension and shortening of muscle fibers), pathological synkinesia (involuntary contraction of some facial muscles with voluntary contraction of others), crocodile tears (lacrimation during eating due to aberrant reinnervation) and hemifacial spasm. Prognostically unfavorable factors are considered to be old age, complete paralysis of the facial muscles, a marked decrease in the amplitude of the M-response in ENMG, the absence of clinical signs of recovery within 3 weeks of the onset of the disease, as well as the presence of pain behind the ear at the onset of the disease.

Treatment of facial nerve neuropathy is aimed at reducing edema and inflammation of the nerve in the acute period, protecting the cornea from drying out, stimulating regeneration processes and preventing complications. The basis of drug therapy consists of corticosteroids prescribed in the first 72 hours after the onset of the disease (prednisone at a dose of 1 mg / kg / day, followed by a dose reduction). If a herpetic etiology is suspected, antiviral drugs (acyclovir, valacyclovir) are additionally prescribed. Physiotherapy methods (electrical stimulation, laser therapy, magnetic therapy) and physical therapy are used to stimulate nerve regeneration and prevent atrophy of facial muscles. In cases of severe damage with an unfavorable prognosis and with secondary forms of neuropathy, surgical treatment (nerve decompression, neuroplasty) may be considered.

Despite significant advances in the study of facial neuropathy, many aspects of pathogenesis, outcome prediction, and treatment optimization remain poorly understood. Current research areas include the development of new methods for early diagnosis and prediction of outcomes, the search for effective neuroprotective and neuro-regenerative strategies, the study of the mechanisms of central neuroplasticity and their role in functional recovery, as well as the development of personalized treatment approaches tailored to the individual characteristics of patients.

This study is aimed at a comprehensive study of facial neuropathy with a focus on clinical and electrophysiological correlations, analysis of prognostic factors and evaluation of the effectiveness of various therapeutic approaches. Special attention is paid to identifying early predictors of an unfavorable outcome and developing personalized therapy algorithms aimed at optimizing recovery processes and preventing complications. Transcranial magnetic stimulation (TMS) has attracted increasing attention in recent years as a method of noninvasive neuromodulation with a wide range of potential clinical applications. TMS technology is based on the principle of electromagnetic induction, which makes it possible to generate local electric fields in the nervous tissue through short-term magnetic pulses without significantly reducing their intensity when passing through the bone structures of the skull. Initially used as a diagnostic tool for assessing the functional state of the corticospinal

pathways, TMS is now being actively researched as a therapeutic method for various neurological and psychiatric disorders. In facial nerve neuropathy, TMS can affect several key links in the pathophysiological process. First, the stimulation promotes the activation of neuroplastic processes in the motor cortex, which controls the facial muscles, which can compensate for the functional deficit. Secondly, TMS can modulate cortical excitability and enhance downward influences on the facial nerve nucleus in the brainstem. Thirdly, neuromodulation stimulates the production of neurotrophic factors that play an important role in axon regeneration and remyelination. Finally, TMS has anti-inflammatory potential, which may be especially important in the early stages of the disease.

One of the key aspects of the application of TMS is the determination of optimal stimulation parameters, among which frequency is a particularly important characteristic. Modern protocols include low-frequency stimulation (< 1 Hz), which usually causes a decrease in cortical excitability, and high-frequency stimulation (≥5 Hz), which predominantly enhances cortical activity. The choice of frequency mode may be crucial for therapeutic effectiveness in facial neuropathy, taking into account the features of pathogenesis and the stages of recovery processes.

Despite the encouraging preliminary results of the use of TMS in facial neuropathy, comparative studies analyzing the effectiveness of various frequency protocols are extremely insufficient. There are no well-founded clinical recommendations for choosing optimal stimulation parameters, taking into account the age of the disease, the degree of nerve damage and the individual characteristics of patients[1,5].

The present study is aimed at conducting a detailed comparative analysis of the therapeutic efficacy of low-frequency (1 Hz) and high-frequency (10 Hz) transcranial magnetic stimulation in the context of restoring facial nerve function in patients with neuropathy. Particular attention is paid to the multifactorial assessment of treatment outcomes, including clinical scales, electroneurographic parameters and neuroimaging data, as well as the identification of potential predictors of a positive response to various TMS protocols.

The conducted study is a comprehensive comparative analysis of the effectiveness of transcranial magnetic stimulation of various frequencies in modulating the recovery process in facial neuropathy. The results obtained indicate a differentiated effect of low-frequency (1 Hz) and high-frequency (10 Hz) TMS protocols on the dynamics of facial muscle function restoration.

Clinical evaluation data using the House-Brackmann scale and the facial neuromotor function assessment system showed that high-frequency TMS (10 Hz) provides a statistically significant advantage in recovery rate and final functional outcome in patients with acute facial neuropathy (duration of disease up to 14 days) compared with low-frequency TMS and the control group. At the same time, the greatest effect was observed with stimulation of the contralateral motor cortex, corresponding to the projection of facial muscles.

Conclusions: Thus, the purpose of this study is a comprehensive study of the main pathophysiological mechanisms affecting the recovery process in facial neuropathy, with a focus on the interaction of local and central factors that determine the nature and degree of functional recovery. Special attention is paid to identifying potential biomarkers and predictors of recovery that can be used for personalized prediction of disease outcomes and optimization of therapeutic approaches.

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