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Neurophysiological Aspects of Pain Syndromes of the Maxillofacial Region

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Abstract: The article considers modern neurophysiological methods of studying pain syndromes of the maxillofacial region (MFR). It is shown that in recent years, clinical and neurophysiological features of various types of chronic facial pain, as well as the functional state of afferent systems and mechanisms of interaction of afferent and efferent pathways in chronic pain have been actively studied. The possibilities of neurophysiological study of the trigeminal system for establishing the pathogenesis of pain syndromes of the MFR are analyzed.

Keywords: pain, facial pain, evoked potentials.

The features of pain syndromes of the maxillofacial region are largely related to its complex structural and functional organization and the organization of the nervous system. Numerous sensitive somatic and autonomic receptors are located in a relatively small area. This area is innervated by the trigeminal, glossopharyngeal, facial and sublingual nerves, and also includes nodes of the autonomic nervous system. In addition, distant receptors (hearing, vision, smell), as well as respiratory and digestive organs (nasal cavity and sinuses, oral cavity, maxillary system, nasopharynx) are located in the CHL. The proximity of CSF to the central areas of higher mental functions determines the features of pain processes in this area, which are often accompanied by neurotic disorders that can transform into psychogenic facial pain [1, 2].

Pain in the maxillofacial region (CRO), which can be felt on the surface of the face or in the oral cavity (orofacial pain), can manifest as acute toothache, usually disappearing after dental treatment, chronic pain or recurrent facial pain (prosopalgia). The most severe course is observed with trigeminal facial pain, especially with trigeminal neuralgia (HTN).

Neuralgia of the glossopharyngeal, intermediate and upper laryngeal nerves is less common. Neuralgia of individual branches of the trigeminal nerve, such as the lingual, superior and inferior alveolar nerves, is even less common. All these cases are partial forms of HTN, which are manifested by characteristic pain attacks and the presence of trigger zones. Odontogenic neuralgia is distinguished separately, which develops as a result of traumatic damage to the lunular nerves during tooth extraction, injections during conduction anesthesia, or fractures of the mandible bones. Pain in the throat can vary in localization, duration, time of occurrence, the presence of vegetative symptoms and provoking factors. A clinical examination reveals a variety of pain syndromes presented in the working classification. Paraclinical studies (neuroimaging, ultrasound and other methods) help to clarify the picture of the disease.

The purpose of this review is to evaluate the possibilities of neurophysiological methods in the study of facial pain.

Trigeminal neuralgia (HTN) and methods of its diagnosis Typical neurogenic prosopalgia is HTN, which is divided into symptomatic and idiopathic forms. The peculiarity of typical HTN is the paroxysmal nature of pain attacks with sudden and lightning onset and end of pain. This type of seizures makes it possible to compare these neuralgias with epileptic paroxysms. With an exacerbation of HTN, bioelectric activity changes in a spike-wave type, which is a characteristic feature of epilepsy. This indicates that changes in sensory flow from the periphery activate the central mechanisms of HTN, forming an algogenic system of the paroxysmal type. This process confirms the effectiveness of antiepileptic drugs in the treatment of NTN.

Trigeminal evoked potentials: In 60% of patients with NTN, bilaterally synchronous waves are recorded in the Δ- and θ-frequency ranges, more pronounced in the central leads, as well as "fast wave — slow wave" complexes. This indicates a pathological impulse from the median nonspecific structures of the brain stem and a decrease in the threshold of convulsive readiness. In 40% of patients, three-dimensional localization of the sources of pathological activity reveals its generator in the central part of the brain stem, either on both sides or on the side opposite to the pain. This corresponds to anatomical localization in the reticular formation of the brainstem and the nonspecific nuclei of the thalamus, while various parts of these structures may be involved in the process.

The symmetry of changes and the severity of epileptoid transformations in the late components of evoked potentials also indicate the involvement of nonspecific median structures of the brain stem in the pathogenesis of typical HTN. Studies of trigeminal somatosensory evoked potentials (TSWP) have shown differences between patients with trigeminal neuropathy and HTN. In the case of trigeminal neuropathy, the main factor is structural nerve damage that affects only the trigeminal system, altering the early components of the trigeminal nerve, without affecting the transmission of sensory information in other afferent systems of the brain.

Disorders of the central afferentation mechanisms and the formation of a pathological pain system, including nonspecific median structures of the brain stem, cortex, and other afferent systems of the brain, play an important role in the pathogenesis of HTN.

In the study of trigeminal somatosensory evoked potentials In patients with NTN, it was found that when the mandibular branch of the trigeminal nerve is stimulated, there is no peak of P1 on both sides, and the appearance of peak N1 occurs earlier than in the control group. Irritation of the maxillary branch causes a slight increase in the latency of the positive peak on the pain side, while on the opposite side there is an increase in the latency periods of the first wave, especially in its negative phase. Each TSWP wave consists of positive and negative peaks, which reflect the electrical activity of various nervous structures. The peaks of P1 and N1 are associated with the activity of the fibers of the peripheral branches of the trigeminal nerve, and P2 and N2 are the result of the excitation of two consecutive generators: the cells of the Gasser node and the fibers of the trigeminal root. Peaks of P3 and N3 are formed at the level of the descending tract and trigeminal nuclei in the brainstem.

The most noticeable changes in TSWP in NTN relate to a decrease in the component composition of the first wave during stimulation of the mandibular branch. In addition, there is an increase in the amplitude of the P2 peak, but less pronounced on the pain side, with an increase in the latency of the third wave and its component peaks.

In the study of TSVP on the affected side, there is also an elongation of the latent periods of the early components and a decrease in their amplitude, and in some cases, their complete disappearance. This may indicate the presence of structural abnormalities in the trigeminal nerve system and confirms the peripheral compression origin of the disease. Thus, the method of registration of TSVP can serve as an effective tool for diagnosing NTN and determining the level of nerve damage. Dynamic observation during periods of exacerbation and remission showed that the increase in amplitude and distortion of the typical configuration are caused by additional signals and are functional in nature during exacerbation of the disease. Thus, the registration of

TSWP confirms the involvement of both peripheral and central mechanisms in the development of NTN.

The study of nociceptive flexor reflexes (NFR), such as corneal, blinking, and trigeminal-cervical reflexes, is used to assess pain syndromes in CHL, as they allow quantifying the pain threshold. The relationship between the pain threshold and the occurrence of these reflexes plays an important role in the analysis of pain processes. The NFR study provides an opportunity to assess the state of the anti- and nociceptive systems, as well as the role of mediators involved in pain control, and clinical syndromes associated with chronic pain or impaired pain perception.

The implementation of NFR is associated with the activation of A-δ and C-fibers. For example, these receptors are dominant in the cornea, so the corneal reflex is considered nociceptive. The blinking reflex also involves the activation of nociceptive fibers, which manifests itself in late responses (peaks R2 and R3). When the first branch of the trigeminal nerve is stimulated, three responses are recorded in the M.sternocleidomastoideus: C1, C2 and C3, and C3 occurs during nociceptive stimulation.

The study of the trigeminal-cervical reflex showed that with migraine, response latencies are shortened, with cervicogenic pain, the amplitudes decrease, and with chronic tension headache and excessive headache, no changes are observed. The analysis of the parameters of reflex responses of the NFR helps to assess both the mechanisms of pain formation at various levels of the nervous system and the integrative mechanisms of pain control.

Acute pain is characterized by a decrease in NFR thresholds, which reflects the activation and dominance of the nociceptive system. In chronic pain, there are two possible variants of NFR changes: 1) lowering the thresholds, which indicates the insufficiency of the analgesic system in the absence of a nociceptive stimulus; 2) increasing the thresholds, which indicates increased activity of the antinociceptive system, but not effective enough to eliminate pain. In the case of paroxysmal pain, a decrease in thresholds is observed only before or during a paroxysm.

The blinking reflex (MR)— an analog of the corneal reflex, includes afferents of the first branch of the trigeminal nerve, efferents of the facial nerve and their nuclei in the brainstem. MR is recorded from the circular eye muscle when the first branch of the trigeminal nerve is stimulated. The reflex arc includes the fibers of the first branch and the sensory nucleus of the trigeminal nerve, the nucleus of the facial nerve, as well as the posterior longitudinal bundle, which together with the reticular formation of the brainstem performs a coordinating and regulating function. The monosynaptic part of the reflex includes the first branch of the trigeminal nerve and its sensory nucleus, and the polysynaptic part is carried out through the insertion neurons of the posterior longitudinal bundle, which allows impulses to be transmitted to the motor neurons of the opposite side. Under normal conditions, the latency of the contralateral late response may exceed the latency of the ipsilateral component by 1-5 ms. This method allows us to assess the functional state of the first branch of the trigeminal nerve, the facial nerve and the brainstem [11, 12].

There are two main theories explaining temporomandibular joint (TMJ) dysfunction: 1) jaw occlusion disorder and 2) psychophysiological. It is believed that an imbalance in occlusion of the jaws is the cause of functional disorders of the TMJ, and that its elimination improves the patient's condition. However, the presence of patients with normal occlusion suffering from prosopalgia indicates that pain can be caused not only by a violation of occlusion. Pain caused by a muscle spasm occurs earlier than pain caused by changes in the joint itself. Microtrauma of articular elements may be associated with changes in occlusion, inflammation, and degenerative processes. Both theories recognize that the pain of TMJ dysfunction includes a myogenic component.

A special variant of myofascial syndrome is observed in patients with trigeminal neuralgia (HTN+). Palpation of the masticatory muscles on the pain side of these patients shows muscle density and tension, as well as pronounced soreness. In all patients, painful seals and dots are

found in at least one of the masticatory muscles, but palpation of these muscles does not cause paroxysms of HTN. There are no seals on the opposite side, and they are also absent in the muscles that lower the lower jaw (maxillohyoid, chin-hyoid, and biconvex). Both hypesthesia and hyperesthesia are found in the sensations of sensitivity, as well as the absence of changes in sensitivity. All patients with HTN+ experience limited mouth opening due to severe pain. After a paroxysm of HTN, a spasm of the masticatory muscles occurs, which is considered to be the result of a sensorimotor reflex. This spasm leads to local ischemia in the muscles, the release of inflammatory mediators that increase the sensitivity of nociceptors, activate "dormant" receptors, which makes them more excitable under any kind of irritation. As a result, the spasmodic muscle becomes an additional source of nociceptive impulses.

EMG in patients with NTN in the stage of prolonged exacerbation shows a decrease in the average amplitude of bioelectric muscle activity on the pain side, while on the opposite side muscle activity does not change, which distinguishes this from myofascial syndrome. This can serve as a differential diagnostic sign between nociceptive and neuropathic pain. Signs of increased reflex excitability of the trigeminal system, such as hyperesthesia, paresthesia, dysesthesia, allodynia, and others, are often observed in sensitive disorders and accompany the clinical picture of irritation.

In HTN caused by the tunnel compression mechanism, bilateral changes in trigeminal evoked potentials (TVP) are revealed, such as an increase in the latency of early components and a decrease in their amplitude, which indicates structural disorders in the trigeminal nerve system. In chronic pain syndrome, signs of dysfunction of the brain stem structures are often found, for example, an increase in the latency of the R2 peak in the blinking reflex and the latency of the amplitude of stem evoked potentials (ASVP). Pain syndrome in the facial area is often accompanied by impaired function of the masticatory muscles, which is also detected during EMG stimulation of these muscles.

In chronic facial pain, EMG can show a decrease in the amplitude of the M-response on the pain side, as well as its deformation and stretching. Latency indicators may decrease or remain within the normal range. [1, 7, 8].

One of the new methods for studying pain syndromes in the maxillofacial region is the study of laser evoked potentials (LVP). It has been shown that when the skin of the hands of healthy subjects is stimulated with short pulses of an infrared laser, a potential arises in the area of the vertex, the amplitude of which correlates with the intensity of pain. Similar cortical responses were also recorded when stimulating tooth pulp, but using a laser allows the stimulus to be standardized and applied to different areas of the body. The mechanism of action of infrared laser stimulation is a rapid increase in skin temperature, which causes activation of intraepidermal nociceptors. This stimulation selectively activates A-delta and C-fibers, which are the main afferent nociceptive fibers. Late HDL reflects the activity of A-delta fibers, and late HDL reflects the activity of myelin-free fibers.

M. masseter also uses the method of extraceptive suppression of voluntary muscle activity, which is used to study the mechanisms of headache and facial pain. This method involves perioral electrical stimulation, which causes two consecutive periods of inhibition in the tonic EMG activity of the masticatory muscles: early (ES1) with a latency of 10-15 ms and late (ES2) with a latency of 25-55 ms. The degree of exteroceptive suppression in the masticatory muscles increases with homotopic nociceptive activity in the trigeminal afferents, which helps to quantify the intensity of headache and facial pain. It is believed that the early period of inhibition (ES1) is associated with the oligosynaptic activation of interneurons of the trigeminal complex, which have an inhibitory effect on the motor neurons of the masticatory muscles, and the late period (ES2) is associated with a polysynaptic reflex arc involving neurons of the medullary part of the spinal trigeminal nucleus. There is also evidence that ES2 can be detected with heterotopic pain stimulation.

To measure the intensity of pain, the registration of CVD (spontaneous and evoked potentials) is widely used. The early components of the CVD (N65—P120) reflect the intensity of the physical stimulus, and the amplitude of the late components (N140—P300) correlates with the subjective perception of pain. A decrease in the amplitude of the late components of CVD is associated with the administration of analgesics, which confirms that these components reflect the subjective perception of pain. However, the amplitude of the late CVD components is subject to significant variability, depending on psychological factors such as attention, memory, and emotional state, which makes it difficult to accurately interpret these data to control pain. Therefore, the nociceptive reflex of withdrawal remains the most reliable method for assessing pain.

In recent years, methods of functional mapping of brain neuronal activity in acute and chronic pain, such as positron emission tomography and functional MRI, have been actively introduced. These methods make it possible to register the local hemodynamic reaction and metabolism in the brain, which correlate with the electrical activity of neurons. The use of functional mapping makes it possible to visualize changes in neuron activity in response to painful stimuli, which helps to investigate the neurophysiological and neuropsychological mechanisms of pain. The study of the mechanisms of central regulation of human sensitivity in normal and pathological conditions is an important task of modern neurophysiology, clinical neurology and neurostomatology, and neurophysiological and neuroimaging methods remain the main tools in this field.

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