PHYSIOLOGICAL ASPECTS OF PAIN SENSITIVITY

Pazilova Sevara Bakievna
Senior teacher of the Department of Physiology, Tashkent Pediatric Medical Institute
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Abstract*. The article describes the pathophysiological mechanisms of the formation and development of somatogenic and neurogenic pain syndromes. The pathophysiological and neurochemical aspects of hyperalgesia are considered. The results of immunohistochemical studies of the participation of excitatory amino acids, neuropeptides, nitric oxide, and Ca2+ ions in the mechanisms of sensitization are presented. The structural aspects of neurogenic pain syndromes are the formation in the central nervous system of aggregates of interacting sensitized neurons with impaired inhibitory mechanisms and increased excitability. Scientists associate the features of the pathogenesis of somatogenic and neurogenic pain syndromes with the principles of therapy for pathological pain.*
Keywords*: transduction, perception, somatogenic pain, neurogenic pain.*

Pain is an unpleasant feeling with negative emotional experiences that almost everyone has experienced in their lives. Physiological pain provides a signaling function, warns the body of danger and protects it from possible excessive damage. The perception, conduct and analysis of pain signals in the body are provided by special neuronal structures of the nociceptive system, which are part of the somatosensory analyzer. Therefore, pain can be considered as one of the sensory modalities necessary for normal life and warns us about harmful effects.

However, there is another type of pain that is pathogenic to the body. This pain makes people unable to work, reduces their activity, causes psychoemotional disorders, leads to regional and systemic microcirculation disorders, is the cause of secondary immune depressions and disorders of the visceral systems. Such pain is called pathological. In a biological sense, it poses a danger to the body, causing a whole range of maladaptive reactions.

Pain is always subjective, and its final assessment is determined by the location and nature of the injury, the nature of the damaging factor, the psychological state of a person and his individual life experience. Pain is defined as "an unpleasant sensation and emotional ordeal associated with existing or potential tissue damage, or experienced at the time of this damage." Between the site of injury and the moment of pain perception lies a whole series of complex electrochemical phenomena, united by the term "nociception". Nociception involves four physiological processes:

Transduction is a process in which the damaging effect is transformed into electrical activity at the ends of sensitive nerves. Transmission is the conduction of nerve impulses through the system of sensitive nerves. Modulation is a process in which the nociceptive transmission is modified under the influence of neural influences. Perception is the final process in which transduction, transmission and modulation, interacting with individual physiological characteristics, create a final subjective emotional sensation perceived as pain. Traditionally, two main theories of pain perception are considered. According to the first one, put forward by M. Frey, there are pain receptors in the skin, from which specific afferent pathways to the brain begin. It was shown that when human skin was irritated with the help of metal electrodes, the touch of which was not even felt, "points" were detected, the threshold stimulation of which was perceived

as a sharp unbearable pain. The second theory proposed by Goldscheider suggests that any sensory stimulus reaching a certain intensity can cause pain. In other words, there are no specific pain structures, and pain is the result of the summation of thermal, mechanical and other sensory impulses. Initially called intensity theory, it later became better known as "pattern" or "summation" theory. However, recent studies devoted to the study of the anatomy and physiology of pain have largely "reconciled" these two oppositional theories. Currently, it has been established that there are two types of peripheral distal sensory neurons that most actively respond to nociceptive stimuli. Based on the study of the response characteristics of these subtle afferents, three types of them were identified: mechanosensitive, thermosensitive and polymodal nociceptors. There are five main components in the overall structure of pain:

Perceptual component, allows you to determine the location of the damage.

The emotional-affective component reflects the psycho-emotional reaction to damage.

The vegetative component is associated with a reflex change in the tone of the sympatho-adrenal system.

The motor component is aimed at eliminating the effects of damaging stimuli.

The cognitive component is involved in the formation of a subjective attitude to the pain experienced at the moment based on accumulated experience.

Acute pain is a new, recent pain that is inextricably linked to the damage that caused it, and, as a rule, is a symptom of some kind of disease. Such pain disappears when the damage is repaired. Chronic pain often acquires the status of an independent disease, lasts for a long period of time, and the cause of this pain may not be determined in some cases.

Depending on the pathogenesis, pain syndromes are divided into:

Somatogenic pain syndromes

Neurogenic pain syndromes

Psychogenic pain syndromes

Pain syndromes resulting from the activation of nociceptive receptors in trauma, inflammation, ischemia, and tissue stretching are classified as somatogenic pain syndromes. Clinically, among them are: post-traumatic and postoperative pain syndromes, pain with joint inflammation, myofascial pain syndromes, pain in cancer patients, pain with damage to internal organs and many others. The development of neurogenic pain syndromes is associated with damage to the structures of the peripheral or central nervous systems involved in conducting nociceptive signals. Examples of such pain syndromes are neuralgia (trigeminal, intercostal, and others), phantom pain syndrome, thalamic pain, and causalgia. The leading role in the mechanism of development of psychogenic pain syndromes is assigned to psychological factors that initiate pain in the absence of any serious somatic disorders. Often, pain of a psychological nature occurs due to overstrain of any muscles, which is provoked by emotional conflicts or psychosocial problems. Psychogenic pain can be an integral part of a hysterical reaction or occur as a delusional hallucination in schizophrenia and disappear with adequate treatment of the underlying disease. Psychogenic pain also includes pain associated with depression, which does not precede it and does not have any other cause. Thus, peripheral damage triggers a cascade of pathophysiological and regulatory processes affecting the entire nociceptive system from tissue receptors to cortical neurons. At the same time, if we briefly characterize the pathogenesis of somatogenic pain syndromes, we can note the following most important links:

Irritation of nociceptors in case of tissue damage

Isolation of algogens and sensitization of nociceptors in the area of damage

Increased nociceptive afferent flow from the periphery

Sensitization of nociceptive neurons at various levels of the central nervous system

In this regard, the pathogenetically justified use of drugs aimed at somatogenic pain syndromes is considered;

Limiting the entry of nociceptive impulses into the central nervous system is achieved with the help of various kinds of blockades with local anesthetics, which can not only prevent the sensitization of nociceptive neurons, but also contribute to the normalization of microcirculation in the damaged area, improving the restoration of damaged tissues. The use of nonsteroidal and/or steroid anti-inflammatory drugs provides suppression of algogen synthesis, reduction of inflammatory reactions and thereby reduces the sensitization of nociceptors. To activate the structures of the antinociceptive system that control nociceptive impulses in the central nervous system, a whole range (depending on clinical indications) of medicinal (narcotic and non-narcotic analgesics, benzodiazepines, alpha-2-adreno receptor agonists and others) and non-medicinal (percutaneous electroneurostimulation, reflexotherapy, physiotherapy) agents can be used reducing pain sensitivity and negative emotional experience. Indispensable pain syndromes caused by damage to peripheral nerves or structures of the central nervous system are one of the clinical paradoxes. Indeed, a violation of the integrity of the nerve should lead to a decrease in sensory sensations in the area innervated by it. However, patients with complete limb denervation, for example, with brachial plexus injury, often experience excruciating pain in a paralyzed arm.

It is believed that neurogenic pain syndromes occur when structures associated with carrying out nociceptive signals are damaged. Clinical observations are an important proof of this position. Thus, in patients after damage to peripheral nerves in the area of constant soreness, in addition to paresthesia and dysesthesia, there is an increase in the thresholds for injection and nociceptive electrical stimulus. In patients with syringomyelia, a pronounced pain syndrome occurs when the pathological process spreads to the dorsal horns of the spinal cord, while temperature and pain sensitivity decrease occurs when the pathological process spreads to the dorsal horns of the spinal cord, while temperature and pain sensitivity decrease. In patients with multiple sclerosis, who also suffer from attacks of pain paroxysms, sclerotic plaques were found in the afferents of the spinothalamic tract. Isolated damage to the ventrolateral quadrants of the spinal cord, along with the occurrence of spontaneous pain and dysesthesia, causes a decrease in pain and temperature sensitivity.

In the clinical examination of patients with thalamic pain that occurs after cerebrovascular disorders, there is also a decrease in temperature and pain sensitivity. At the same time, the lesions detected by computed tomography correspond to the sites of passage of somatic sensitivity afferents in the brain stem, midbrain and thalamus. Spontaneous pain occurs in people with damage to the somatosensory cortex, which is the final cortical point of the ascending nociceptive system. All this indicates that neurogenic pain syndrome can occur regardless of the location of damage to the pain pathways.

The following medicines are currently considered a priority: anticonvulsants and drugs that enhance inhibitory reactions in the central nervous system — benodiazepines, GABA receptor agonists, calcium channel blockers, excitatory amino acid antagonists, peripheral and central Na channel blockers.

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