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Factors Affecting Sleep in External Appearance

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Abstract: The article discusses the pathophysiological and behavioral mechanisms of chronic insomnia development in headache. Particular attention is paid to the effect of sleep disorders on primary headaches - migraine and cluster headache. Data on the use of melatonin in the complex treatment of chronic headaches are presented.

Keywords: sleep, sleep disorders, headache, migraine, cluster headache, melatonin.

The connection between sleep and headache is well known: lack of sleep can trigger another attack, and good sleep can relieve pain. Back in 1853, M. Romberg suggested that “an attack (of a migraine) usually ends in a deep and refreshing sleep” [2]. And in 1873, E. Living expressed the opinion that "the easiest way to stop a migraine attack is to fall asleep" [3].

In the International Classification of Headache Third Revision (ICHD-3, 2018), sleep is mentioned 38 times [4], and headache is indicated in the International Classification of Sleep Disorders among the symptoms of sleep disorders [5]. Sleep-related headache is one of the common complaints of headaches (migraine or other) by the American Academy of Sleep Medicine.

Neuroanatomical structures that provide pain perception and sleep

Studying the pathophysiology and function of the hypothalamus simultaneously in headache and sleep disorders may explain the relationship between pain perception involving the trigeminal nerve and sleep induction. The trigemino-cervical complex, which plays an important role in the formation of headache, is connected to a number of stem structures. Nociceptive information enters the nuclei of the thalamus, the activation of which is of great importance in the development of migraine and cluster headache. The trigeminal nerve sends afferent impulses to intra- and extracranial structures and blood vessels, the spinal trigemino-cervical complex (Fig. 1A) [6]. In the modulation of sensory trigeminal activation, such vasoactive substances as calcitonin-gene-related peptide and substance P are important. Secondary neurons of the trigeminal system send ascending impulses to thalamocortical neurons. In addition, there are direct and indirect connections with the periaqueductal gray matter and the hypothalamus. The third thalamocortical neuron, in turn, has a synaptic connection with the cortex, including motor, somatosensory and visual. Monoaminergic neurons, in particular noradrenergic, serotonergic, dopaminergic, and periaqueductal gray matter, affect the thalamus (orexin) and the basal forebrain. Thus, during the development of a headache, excitation is transmitted (Fig. 1B). The trigeminovascular complex receives direct and indirect modulating impulses: direct ones come from the somatosensory cortex and the island, and indirect ones come from the hypothalamus. This complex provides pro- and antinociceptive interactions. The suprachiasmatic nucleus of the hypothalamus receives direct impulses from the cells of the retinal ganglion and indirect signals from the environment (light - dark). The posterior thalamic neurons receive light signals from the retina, these neurons project onto the sensory and visual cortex, which

is a neuronal substrate for the development of photophobia during a migraine attack and increased sensitivity to light. The monoaminergic nucleus inhibits the ventrolateral preoptic nucleus, which leads to stimulation of the hypothalamic (orexinergic) system and the nucleus of the bridge. When going to sleep, the orexinergic system activates the ventrolateral preoptic nucleus. Direct inhibition of the system and a decrease in orexinergic support for the state of sleep determine the rapid transitions from sleep to wakefulness and vice versa. The orexinergic system, including neuropeptides (orexin A and B), is present in the posterior, lateral, and paraventricular hypothalamus [7]. Orexin receptors are located in the prefrontal cortex, thalamus, and subcortical areas and are involved in the modulation of nociceptive neurotransmission, thermoregulation, neuroendocrine and autonomic functions [8]. These zones are also involved in the modulation of nociceptive activation of the trigeminovascular complex [9].

Before a migraine attack, patients often yawn, feel hungry and feel sleepy, which may be caused by changes in the hypothalamus and orexin neurons and is confirmed by neuroimaging studies using functional magnetic resonance imaging before and during attacks migraine [10]. Thus, a connection is established between the systems that regulate the processes of sleep and wakefulness, and the systems involved in the formation of headaches.

A biobehavioral model of the mechanisms of connection between chronic insomnia and chronic headache was proposed, which involves three basic points:

1) attempts to overcome headaches can accelerate and intensify sleep disturbances;
2) violation of the physiology of sleep increases the tendency to headache;
3) over time, these cycles interact and serve to transform or transition from episodic headache to chronic [11].

Among the important prerequisites, there are traits of an anxious personality, a tendency to activate the sympathetic nervous system, which, together with psychological stress, can be a trigger for sleep disorders. Efforts to overcome sleep disorders (compensatory daytime sleep, taking sleeping pills before bed and caffeine in the daytime) lead to the consolidation of chronic insomnia. This model explains the interaction between biological and psychological factors in the development of sleep disorders [11].

Comorbidity of sleep disorders and various types of headache

According to ICGB-3 (2018), there are:

- ✓ primary headaches that are not the result of any disease;
- ✓ secondary or symptomatic headaches, in which pain is a symptom of the underlying disease;
- ✓ cranial neuralgia and other facial pains;
- ✓ pain due to psychiatric disorders.

The group of primary headaches includes migraine, tension headache, cluster headache and other rarer variants. Primary headaches are more common in the population - they account for 85-90% of all headaches. It is primary headaches, such as migraine and cluster headache, that are more often accompanied by sleep disturbances.

The connection between headache and sleep disorders is multifaceted. On the one hand, headaches can be the result of sleep disorders, a typical example is hypnic headache. Patients with migraine and trigeminal autonomic cephalalgia, which includes cluster headache, report attacks of headache during sleep. On the other hand, insomnia can cause the development of headache. Population studies have revealed comorbidity of migraine and restless legs syndrome, migraine and narcolepsy.

Another aspect is that some medications used to treat headaches can cause sleep disturbances. Beta-blockers, which are the first line of preventive therapy for frequent migraine attacks, cause heavy dreams and frequent nocturnal awakenings. They reduce the secretion of endogenous melatonin, which leads to suppression of the sleep signal and can cause sleep disturbances and fragmentation.

So, according to polysomnography, an increase in awakenings was shown against the background of taking propranolol compared with placebo. Tricyclic antidepressants (amitriptyline), used in the treatment of frequent migraine attacks, not only reduce Another aspect - some drugs used to treat headaches can cause sleep disturbances. Beta-blockers, which are the first line of preventive therapy for frequent migraine attacks, cause heavy dreams and frequent nocturnal awakenings. They reduce the secretion of endogenous melatonin, which leads to suppression of the sleep signal and can cause sleep disturbances and fragmentation. So, according to polysomnography, an increase in awakenings while taking propranolol was shown compared with placebo. Tricyclic antidepressants (amitriptyline), used in the treatment of frequent migraine attacks, not only reduce.

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