

The Use of Methods of Functional Analysis of the Dental System Makes it Possible to Individualize and Systematize the Sequence of Screening and Monitoring of Patients With Bruxism

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Relevance. According to a number of authors, bruxism is a genetically determined disease. So children whose parents noted the presence of episodes of Brooks behavior were also susceptible to this disease. 20-50% of bruxists have at least one direct relative who gritted his teeth in childhood, while 87% suffered from this disease in adulthood. In a cohort study involving twins in Finland, the highest matching coefficient was found among monozygotic twins, as opposed to dizygotic ones. Gaidarova (2003) established that bruxism is a genetically determined disease with an autosomal dominant type of inheritance.

Nevertheless, genetic markers of bruxism have not yet been found. Further research is needed at the population level in order to establish a possible genetic component in the development of bruxism. Most likely, the mechanism of development of the disease is due to genetic polymorphism, and not the presence of a gene responsible for the occurrence of pathological muscle activity. It is worth noting that tools for assessing parafunction at the population level are most often based on questionnaires in which the patient is asked to indicate the presence or absence of episodes of bruxism in the patient and his relatives.

The physiology of sleep has been analyzed by many scientists in order to find possible biomarkers for the occurrence of nocturnal bruxism. During sleep, two main phases periodically alternate in humans: slow and fast sleep, and at the beginning of sleep the duration of the slow phase prevails, and before waking up, the duration of REM sleep increases. In a healthy person, sleep begins with the first stage of slow sleep (Non-REM sleep), which lasts 5-10 minutes. Then comes the 2nd stage, which lasts about 20 minutes. Another 30-45 minutes falls on the period of 3-4 stages. After that, the sleeper again successively returns to the 3rd and 2nd stages of slow sleep. After that, the first episode of REM sleep occurs, which has a short duration of about 5 minutes. This whole sequence is called a cycle. The first cycle has a duration of 90-100 minutes. Then the cycles are repeated, while the proportion of slow sleep decreases and the proportion of REM sleep gradually increases, the last episode of which in some cases can reach 1 hour. On average, with a full healthy sleep, there are five complete cycles. The object of the researchers' attention was activation reactions (awakening reactions, RP) to the EEG during sleep [1.3.5.7.9.11.13.15.17.19].

Wake-up reactions are short awakenings lasting less than 3 seconds, which are characterized by high frequencies on the EEG (including alpha frequencies, theta frequencies and frequencies above 16 Hz). RP is repeated 6 to 14 times during an hour of sleep, as a reaction to external (environmental) and internal (physiological or pathological) stimuli. Episodes of sleep bruxism, in turn, are associated with rhythmic activity of the chewing muscles. This is a specific activity of the chewing muscles, which is characterized by rhythmic, pseudo-chewing movements of the lower

jaw, occurring once or twice per hour of sleep, at a frequency of approximately 1 Hz. RMMA is observed in 60% of healthy people and in 80% of patients with nocturnal bruxism. RMMA episodes occur predominantly during stages 1 and 2 of sleep, only 10% of episodes occur during REM sleep. From 70% to 88% of RMMA episodes correlate with activation reactions to EEG and are an integral part of them.

Although the number of RP does not usually differ in healthy people and patients with parafunctions, there is experimental evidence that bruxists have a higher sensitivity to activation reactions. In a study, Kato (2003) demonstrated that microactivation reactions caused by auditory or vibrotactile stimuli were followed by episodes of RMMA in 11% of cases, and episodes of compression and friction of teeth occurred in 71% of cases. The pathophysiology of bruxism is associated with the activation of the autonomic nervous system, its sympathetic division. Oromotor activity in bruxism is only the final element of the stage process. Lavinge (2008) described a chain of events that begins with the activation of the sympathetic nervous system and a decrease in the activity of the parasympathetic nervous system (the interval between 8 and 4 minutes before the occurrence of bruxism), followed by activation of the cortex with the presence of a waves on the EEG (4 seconds before the occurrence of bruxism), an increase in respiratory rate and heart rate (1 seconds before bruxism), an increase in the tone of the supra-lingual muscles (0.8 seconds before bruxism), and at the end - an episode of bruxism. This confirms the etiopathogenetic hypothesis of the central origin of bruxism, where the activity of the masticatory muscles and, accordingly, the episode of compression or friction of teeth are a peripheral reflection of this central activation [2.4.6.8.10.12.14.16.18.20.22.24].

Thus, these studies refute the occlusive theory of Bruxism. About 80% of episodes of bruxism occur during the transition from phase 3 to phase 2 and from phase 2 to phase 1 of non-REM sleep (during the transition from deep sleep to surface sleep) and are associated with activation reactions to EEG (RP): short-term 3-15 second periods in which cortical activation occurs, associated with an increase in the activity of the sympathetic nervous system). RP are physiological episodes that recur during sleep, cyclically grouped into a so-called cyclic alternating pattern. Sleep bruxism is more often observed during REM sleep in patients with mental or neurological disorders and in patients receiving treatment with drugs that act on the central nervous system. To date, polysomnographic studies have confirmed that there is a link between activation reactions to EEG (autonomous activation of the cortex preceding activation of the musculature that closes the lower jaw) and episodes of sleep bruxism [21.23.24.25].

Conclusion. Thus, activation of the autonomic and central nervous system is the main factor responsible for the initiation of bruxism. At the same time, most patients with sleep bruxism do not report a deterioration in sleep quality. As a rule, the organization of sleep in patients with bruxism is not impaired in terms of duration and effectiveness, as well as the stage of the process. When analyzing the microstructure of sleep in bruxists, a drop in K-complexes was revealed, which normally occur up to five times per minute and represent the cortex's response to exogenous (for example, sound) or internal (for example, changes in blood pressure) factor.

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