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NEUROPHYSIOLOGICAL CHARACTERISTICS OF SLEEP BRUXISM IN CHILDREN

Master: Shadimetova *N.M.*
Master of child neurology and medical genetics Department, Tashkent Pediatric Medical Institute, Uzbekistan,
shadimetovanihola@gmail.com
Research supervisor: PH D. Saidkhodzhayeva S. N.
Department of neurology with pediatric neurology and medical genetics Tashkent Pediatric Medical Institute, Uzbekistan

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Abstract:
An international panel of experts has redefined bruxism as repetitive jaw muscle activity characterized by clenching or grinding of teeth and/or tensing or thrusting of the mandible that can occur while awake (i.e., wakeful bruxism) or during sleep (i.e., sleep bruxism). Determining the nature of the phenomenon, taking into account comorbid disorders, will provide an opportunity to reduce the drug burden on the patient, and, on the other hand, to increase the effectiveness of therapy. 80 patients with sleep bruxism were examined, patient history and comorbid disorders observed in sleep bruxism were studied. EEG in 3 (3.75%) patients with bruxism revealed epileptiform activity, no specific changes were detected on polysomnograms, at the same time, there was a significantly higher proportion of awakening after falling asleep than in the control group (P<0.01).

Keywords: Sleep Bruxism, Electroencephalography, Polysomnography.

INTRODUCTION.

Sleep bruxism is a sleep-related movement disorder characterized by involuntary and non-functional grinding or clenching of teeth. Sleep bruxism is more common in children than in adults [1], the prevalence among children varies widely - from 13% to 49% [2,3,4,5].

Recently, an international panel of experts redefined bruxism as repetitive jaw muscle activity characterized by clenching or grinding of teeth and/or tensing or thrusting of the mandible that can occur while awake (i.e., awake bruxism) or during sleep (i.e., bruxism). sleep) [8]. Wakeful bruxism is commonly thought of as a habit of clenching the jaw in response to stressful and anxious states [9], while sleep bruxism is a sleep-related rhythmic chewing activity usually associated with awakening (from sleep) [10,11].

Both awake bruxism and sleep bruxism are divided into primary, not associated with any other disease, or secondary, associated with neurological disorders or considered a side effect of drugs [12,13,14,15].

Bruxism is classified by neurologists as a manifestation of parasomnia along with somnambulism and sleep-talking, however, the opinion of bruxism as a manifestation of segmental muscular dystonia is not excluded, it is considered as a marker, which is to a certain extent characteristic of children with epilepsy [6]. Researchers point to a higher prevalence of sleep bruxism in the population with epilepsy [7].

AI Boldyrev united epilepsy, somnambulism, enuresis and bruxism under the concept of "epileptic circle", and gave a significant place to bruxism as a manifestation of epilepsy. The inconsistency of specialists, and sometimes contradictions in this matter, is the reason for the late start or improper treatment of children with bruxism.

The study of the phenomenon of bruxism in children will resolve the issue of the relationship between epilepsy and bruxism and determine the tactics of managing patients with this phenomenon.

Purpose of the study. To determine the neurophysiological changes characteristic of sleep bruxism in children.

Materials and research methods. The object of the study was 80 children with sleep bruxism at the age of 7 to 14 years, the average age of children was 8.95±0.01. There were 46 boys (57.5%) among them, 34 girls (42.5%). A routine EEG study was performed using the Neurosoft Rossiya apparatus, and polysomnography (PSG) was performed in disputable situations. The exclusion criteria were bruxism on the background of dental diseases, patients with active epilepsy.

The control group consisted of 28 patients of the same age without acute somatic and neurological diseases.

Research results. Parents of children complained of headaches in 12 (15%) cases, somnambulism in 24 (30%) cases, somnoloquia in 14 (17.5%) cases,

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nocturnal enuresis in 18 (22.5%) cases, in 12 (15%) had a combination of these types of sleep disorders.

The history of patients was analyzed, hereditary predisposition for epilepsy was determined in 7 (8.7%) patients with bruxism, in the control group, the burden of epileptic syndromes was not determined. Obstetric anamnesis was aggravated in 49 (61.25%) patients with bruxism and 4 (14.3%) patients in the control group (P<0.001).

According to the anamnestic data in relatives of the first kinship of the study groups, somnambulism was determined in 24 (30%) and 2 (7.1%) cases (P<0.001), somnoloquia in 19 (23.75%) and 5 (17.8%) cases (P<0.05), nocturnal enuresis in 29 (36.25%) and 1 (3.6%) cases (P<0.001) in the group of patients with bruxism and the control group, respectively.

A history of febrile seizures was identified in 7 (8.75%) patients with bruxism; no febrile seizures were noted in the control group children (P<0.05). Also, in the examined patients, various hyperkinesis in the form of frequent blinking, biting of the lips and tongue, tics of various localization, "sniffing" of the nose, twitching of the neck or shoulder are much more often detected in 23 (28.75%) patients with bruxism compared to the control group - 2 (7.1%), although the statistical data were unreliable (P<0.05).

Also, in patients with the phenomenon of bruxism, increased anxiety, hyperactivity, impulsivity, stereotyped movements, and onychophagia are determined.

It should be noted that 15 (18.75%) patients were seen by a neurologist with a diagnosis of ADHD, 18 (22.5%) patients with a diagnosis of minimal cerebral dysfunction, 12 (15%) patients with a diagnosis of parasomnia, 23 (28.75%) patient diagnosed with tic hyperkinesis. In the literature, often comorbid disorders of bruxism are ADHD, migraine, tension headaches, other motor phenomena during sleep [1,2,3], often the phenomenon of bruxism is accompanied by increased anxiety [16], and finally, epilepsy. According to the authors, children with ADHD in combination with sleep bruxism have a high prevalence of oppositional defiant disorder, but associations with anxiety disorders are unreliable [17].

The results obtained as a result of the EEG showed that 17 (21.25%) patients had an inherent hypersynchronization of the alpha rhythm with a high alpha index. In 73 (91.25%) patients, paroxysmal activity was recorded in the form of bilaterally synchronous theta-wave periods, with an amplitude of up to 160 μV during a test with hyperventilation, which was interpreted as diffuse slow-wave activity.

Sleep spindles in patients with bruxism tended to generalize, had a higher amplitude than in patients in the control group - up to 50 μV. Functional immaturity of the brain was determined in 4 (5%) patients. generalized epileptiform activity, in 1 (1.25%) patient, epileptiform changes were focal in nature (table 1). Although these children were clinically seizures were not observed.

Table 1.
Electroencephalogram results in children with bruxism.

EEG changes	Abc	%	Focus localization
Generalized epileptiform changes	2	2,5%	-
Diffuse slow-wave activity, functional immaturity of the brain	73	91,25%	-
Epileptiform focal changes with secondary generalization (the focus is located in the left hemisphere)	1	1,25%	Temporoccipital
Functional immaturity of the brain	4	5%	-

A polysomnographic study was performed in 2 children with generalized epileptiform activity and 11 children without it.

As shown in Table 2, sleep latency, total sleep time, and sleep efficiency did not differ between children with bruxism and controls.

No differences were found in the distribution of sleep stages (N1 sleep stage, N2 sleep stage and R sleep stage) or arousal/event index. However, children in the bruxism group had a significantly higher proportion of awakenings after falling asleep than in the

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control group (P<0.01). The K-complex tended to generalize in 3 (4.1%) patients without epileptiform activity on the EEG.

The teeth grinding phenomenon on EMG did not coincide with any particular phase of sleep, but tended to occur during REM sleep, after an episode of bruxism there were signs of awakening on the EEG, and motor restlessness. While in patients with epileptiform activity, the phenomenon of teeth grinding was determined diffusely in all stages of sleep.

Table 2. Parameters of polysomnography in children with bruxism and the control group.

PSG parameters	Control	Patients with bruxism	P
Total sleep time (min)	497,5±59,0	501,2±65,5	> 0.05
Sleep latency (min)	44,2 ±28,7	38,5 ±25,1	> 0.05
Sleep efficiency, %	87,5 ±6,3	85,2 ±5,7	> 0.05
Sleep stage N1 (min)	7,4± 6,1	8,6± 4,1	> 0.05
Sleep stage N2 (min)	251,7±36,0	249,8±19,8	> 0.05
Sleep stage N3 (min)	29,3 ±4,9	23,1 ±2,8	> 0.05
Stage R sleep (min)	104,3±26,4	106,3±25,4	< 0.05
Waking up after falling asleep (min)	26,4 ±23,1	53,4 ±28,1	> 0,01
Excitation/event index	4,2± 2,1	9,8± 1,1	> 0.05

Thus, with the phenomenon of bruxism, no specific changes are detected on the electroencephalogram, but the presence of comorbid conditions testifies in favor of the fact that bruxism can serve as a signal for further examination of the child to rule out epilepsy.

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