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# Coffee consumption influence upon the clinico-neurophysiological manifestations of primary sleep bruxism

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**Abstract**

**Background:** Caffeine is one of the most widely-used psychoactive substances, having multiple stimulating effects. Caffeine is being considered as eliciting a dose-dependent effect on sleep bruxism.

**Material and methods:** There were investigated 100 patients with primary sleep bruxism. Patients were clinically examined, there were given several questionnaires, and they underwent a series of investigations: sleep bruxism recording; surface electromyography, occlusal reflex determination, jc.SSR (*jaw clenching sympathetic skin response*) recording and masseter muscle ultrasonography.

**Results:** In patients with primary sleep bruxism, there were observed various coffee intake patterns/24 h: "abstinent" group – 32%; "1-3 cups" group – 38.1%; "4-6 cups" group – 19%; ">6 cups" group – 11%. People who consume >6 cups/24 h, have increased the number of nocturnal clenches. Pathological occlusal reflex indices were observed in the "1-3 cups" group (76.3%), "4-6 cups" group (89.5%), ">6 cups" (100% cases), "abstinent" group (50%). Disorders associated with the temporomandibular joint and the bruxism-associated pain reach pathological values in individuals who consume 4-6 or more cups of coffee. Excessive caffeine consumption leads to the increase of the amplitude of the sympathetic autonomous potential (jc.SSR, A2, mV) without significant changes in the central autonomous regulation time (jc.SSR, T, s).

**Conclusions:** There were observed various coffee intake patterns in patients with primary sleep bruxism. Excessive coffee consumption is associated with the stress level. The masseter muscle thickness and dental wear show no statistically significant elevation trends, under the influence of various caffeine doses.

**Key words:** sleep bruxism, caffeine, clinical-neurophysiological indices.

## Introduction

Caffeine is widely-used on a daily basis, being a psychoactive substance with multiple stimulating effects – it reduces the total duration of sleep, changes the pattern and the quality of night sleep, increases the wakefulness level and the information processing speed, modifies the activity of the cortico-cerebral neurons and influences the psycho-emotional status [22, 24].

It has been assumed that caffeine has a dose-dependent effect on sleep bruxism [24]. Caffeine consumption and the incidence of bruxism do not differ essentially based on the gender of the individuals, thus coffee consumption in males reaches the following levels: 3-8 cups – 3.8%; more than 8 cups – 7.5%; respectively in women: 0-3 cups – 4.6%; 3-8 cups – 3.9%; more than 9 cups – 5.9% [24]. Svenson et al. [29] have highlighted the dangerous threshold for the onset/maintenance of sleep bruxism – caffeine consumption of more than 6 cups per day. Increased caffeine consumption has been shown to increase the severity of sleep bruxism [17], and the consumption of 6 and more cups of coffee per day increases the risk of sleep bruxism by 1.4 times [21]. However, in other investigations, these data have not been confirmed [1]. Caffeine stimulates the masticatory muscles [8], it increases the subjective sensations pertaining to muscle tension, along with an increase in anxiety tendencies – representing typical signs of sleep bruxism [22]. Bastien et al. [3] have conducted a study on the bioelectric activity of the masseter muscles in patients with sleep bruxism under the action of caffeine and did not detect any significant differences in comparison to the placebo group.

A slightly less studied aspect is the role of the autonomous nervous system in the pathogenesis of primary sleep bruxism. Under the influence of coffee, the sympathetic muscular activity increases by 54.1%, but the studied problem becomes even more complex in light of investigations, according to which the administration of caffeine-free coffee also causes the essential increase in sympathetic muscle activity [6].

Currently, there are no known factors that contribute to the worsening or maintenance of bruxism-associated disorders under the influence of caffeine, and there are not known the clinico-neurophysiological indices that may be informative for the monitoring of patients with primary sleep bruxism with various patterns of coffee consumption (intake of various caffeine doses).

## Material and methods

In the study, there were enrolled 100 people with primary sleep bruxism. All patients were investigated according to the modern diagnostic protocol regarding bruxism, by applying clinical and paraclinical dental exam procedures (anamnesis, dental examination with the identification of dental wear, palpation of the masticatory muscles, computed tomography, etc.). The assessment of the presence and the severity of the clinical disorders were achieved by applying specialized questionnaires [25, 31, 36]: the bruxism questionnaire; the Fonseca questionnaire; the multifactorial questionnaire of bruxism-associated pain; the emotional stress questionnaire; the sleep questionnaire; the questionnaire regarding professions. For the quantitative assessment of emotional stress, we have applied the visual analogue scale (VAS). The division of

the stresogenic professions, was performed according to the criteria proposed by Nishimura [20].

We have assessed the degree of dental abrasion, according to the following grades: 0 – no abrasion; 1 – dental abrasion within the enamel boundaries; 2 - dental abrasion with a <1/3 crown destruction; 3 – dental abrasion with a >1/3 crown destruction [25].

In order to record the *total number of clenches* (TNC) and the *total clench time* (TCT), we have applied the SleepGuard SG5 portable device (Holistic Technologies Inc., USA). The investigation of the occlusal reflex (masseter inhibitory reflex), has been conducted according to the method developed by Tzvetanov et al. [32], with the application of the Neuro-MVP-micro diagnostic complex (Neurosoft). After recording the electromyogram, the analysis was performed based on the presence/absence of the *muscle silence period* SP2 with the latency of 40-60 ms and the minimum duration of 20 ms. Normal SP2 is highlighted as Type I, partially inhibited SP2 is classified as type 2 (EMG amplitude is 20 to 80% of the initial amplitude) and SP2 – Type 3 is considered to be the one without essential changes in the EMG amplitude.

The evoked autonomous potentials during jaw clenches (*jaw clenching sympathetic skin response* – jc.SSR) were recorded using the multifunctional computerized complex Neuro-MVP-micro (Neurosoft). We have analyzed the amplitude ($A_2$, mV) and the recovery period back to the initial levels (T, s) of the autonomous-sympathetic potential, evoked at the standardized maximum jaw engagement using cotton rolls as an interface in the region of premolars/molars (autonomous evoked potentials under standardized conditions).

The thickness of the masseter muscles has been determined by using the Envisor C device (Philips) and a 7.5 MHz linear transducer [10]. We have determined the thickness gradient for the masseter muscle (GCM), under conditions of muscle relaxation-maximum jaw clenching, based on the formula [11]: GCM = (Ga-Gr/Ga)*100%, where: Gr – the thickness of the masseter muscle (mm) during relaxation; Ga – the thickness of the masseter muscle during maximum jaw engagement. In healthy people, GGM is equal to 25%. The echostructure of the masseter muscle was determined by the analysis of the local and diffuse echogenetic features: normal echogenicity in the projection of the masseter muscle – 0 points; local pathological echogenicity – 1 point; diffuse pathological echogenicity – 2 points. The echogenicity index in healthy individuals is 0.23±0.07 points.

The amount of caffeine consumed was evaluated according to the literature recommendations [24], based on the number of coffee cups consumed in 24 hours (the estimated caffeine content in a cup depends on the type of drink: soluble coffee – 61-70 mg; espresso coffee – 97-125 mg; cocoa – 10-17 mg; tea – 15-75 mg; hot chocolate – 30 mg).

*Inclusion criteria used in the research*: a positive clinical diagnosis of primary sleep bruxism, confirmed by the objective recording of sleep bruxism episodes; partial edentulism (no more than 1-3 teeth); age of patients – 18-50 years; presence of the patient consent for participating in the research; cooperating patients.

*Exclusion criteria used in the research:* age not in the established range; other clinical forms of bruxism (central nervous system disorders, epilepsy, parkinsonism, etc.); the presence of anomalies and inflammatory signs in the stomatognathic system; the presence of signs of organic damage with the decompensation of the masticatory muscles activity (a bioelectric activity of less than 30 mcV as recorded on EMG, during a state of relaxation); various acute and chronic diseases during the exacerbation period; parasitosis; alcoholism, drug addiction, toxicomania, mental illness; treatments with psychotropic, anticonvulsant or miorelaxant drugs; lack of the patient consent for participating in the research; non-cooperating patients.

The obtained results were processed with the statistical software package *Statistics for Windows*, v. 11.0 (StatSoft, Inc., USA).

## Results

Of all the investigated patients (n = 100), in 32% of cases, caffeine is consumed sporadically ("abstinent" group), and in 68% of cases, there was observed a constant caffeine consumption trend: 38% – 1-3 cups of coffee; in 19% of cases – 4-6 cups of coffee and in 11% of cases – more than 6 cups. Caffeine consumption in patients with sleep bruxism is higher in young people, and with ageing, caffeine consumption decreases considerably – the correlation between age and consumption is negative and statistically significant ($R_{xy}$ = -0.534, p <0.001).

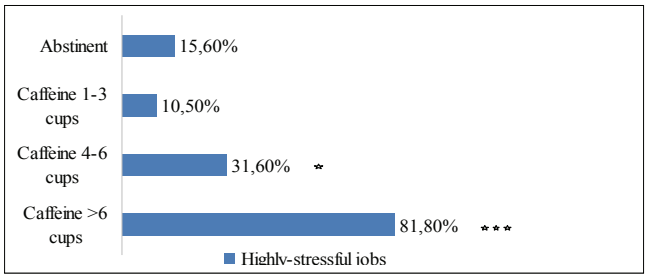


**Fig. 1. Frequency of highly-stressful jobs vs. caffeine consumption per 24 hours in patients with primary sleep bruxism.**

**Note:** statistical differences vs. "abstinent" group (* – p <0.05; *** – p <0.001).

Frequency of highly stressful jobs *vs.* caffeine consumption in patients with primary sleep bruxism: "abstinent" group – 15.6%, "1-3 cups" group – 10.5%, "4-6 cups" group – 31.6%, ">6 cups" group – 81.8 % (fig. 1).

The indices of emotional stress *vs.* caffeine consumption in patients with primary sleep bruxism are essentially different in relation to the caffeine dose. VAS-stress in the "abstinent" group is 4.8 ± 0.31 pt, in patients who consume 1-3 cups of coffee – 5.2 ± 0.33 pt, in patients who consume 4-6 cups of coffee – 6.1 ± 0.32pt, in patients who consume more

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**Table 1**

**Age of patients with primary sleep bruxism, clinical manifestations of bruxism, and the ultrasonographic indices of the masseter muscles *vs.* caffeine consumption per 24 hours**

| Indices | Caffeine consumption | | | |
|---|---|---|---|---|
| | Abstinent (n = 32) | 1-3 cups (n = 38) | 4-6 cups (n = 19) | >6 cups (n = 11) |
| Age, years | 37.1 ± 1.42 | 34.4 ± 0.98 | 32.6 ± 0.92* | 28.5 ± 1.12*** |
| Bruxism questionnaire, pt. | 3.9 ± 0.25 | 4.8 ± 0.28* | 4.9 ± 0.33* | 5.9 ± 0.31*** |
| Pain questionnaire, pt. | 4.11 ± 0.51 | 4.40 ± 0.43 | 4.43 ± 0.42 | 5.90 ± 0.33* |
| Fonseca questionnaire, pt. | 41.32 ± 3.66 | 48.39 ± 4.01 | 56.43 ± 3.96** | 61.94 ± 4.15*** |
| USG, GGM index, % | 27.8 ± 0.48 | 28.0 ± 0.49 | 28.7 ± 0.51 | 29.0 ± 0.52 |
| USG, echogenicity, un. | 1.28 ± 0.10 | 1.30 ± 0.09 | 1.39 ± 0.10 | 1.43 ± 0.11 |
| Dental wear, un | 1.71 ± 0.24 | 1.79 ± 0.23 | 2.12 ± 0.18 | 2.13 ± 0.15 |

**Note:** statistical differences vs. "abstinent" group (* – p <0.05; ** – p <0.01; *** – p <0.001).

than 6 cups – 6.7 ± 0.23 pt. There were observed statistical significant differences in comparison with the "abstinent" group in the group of patients that consume 4-6 cups of coffee per 24 hours (p <0.01) and in the group with a consumption of more than 6 cups/24 h (p <0.001).

Based on the self-assessment data, pathological sleep in caffeine-abstinent patients was found only in 12.5% of cases (4 patients out of 32); a minimal degree of caffeine consumption (1-3 cups) leads to pathological sleep in 5 patients out of the 38 investigated (13.2%); a moderate coffee consumption (4-6 cups) is associated with the presence of pathological sleep in 63.2% of cases (12 patients out of the 19 investigated) and an excessive caffeine consumption is followed by sleep quality disturbance in 81.8% (9 out of the 11 patients investigated). In comparison to the caffeine-abstinent patients, statistically significant differences were observed for cases with a caffeine consumption of 4-6 cups and more of coffee (p <0.001). The duration of night sleep tends to decrease, but the differences between the investigated groups are insignificant: "abstinent" group – 7.6 ± 0.58 hours; "1-3 cups" group – 7.5 ± 0.53 hours; "4-6 cups" group – 6.8 ± 0.51 hours; ">6 cups" groups – 6.2 ± 0.45 hours.

Clinical manifestations of sleep bruxism, as assessed on the data collected from the clinical questionnaires, have shown certain peculiarities. An integral assessment (bruxism questionnaire) revealed a worsening of symptoms regardless of the caffeine dose, with a more pronounced increase in individuals that consume 6 cups of coffee per 24 hours (Table 1). The values of bruxism-associated pain reach statistical significance (p <0.05) in individuals that consume more than 6 cups of coffee per day. Temporomandibular joint-associated disorders (Fonseca questionnaire) reach statistically significant pathological values in individuals that consume 4-6 cups of coffee and are further aggravated by increasing the caffeine dose.

Another situation can be observed after the analysis of the GGM index and the echogenicity of the masseter muscles during the consumption of various doses of caffeine – pathological trends can be observed without reaching any statistically significant values (Table 1). Likewise, in the case of dental wear – there were not found any statistically significant values in all the groups that were investigated, based on the dose of consumed caffeine. The correlation coefficient between dental wear and caffeine consumption is 0.238 (p> 0.05).

The manifestations of the episodes of sleep bruxism (TNC, TCT) were also more pronounced in younger people who consumed more caffeine (fig. 2).

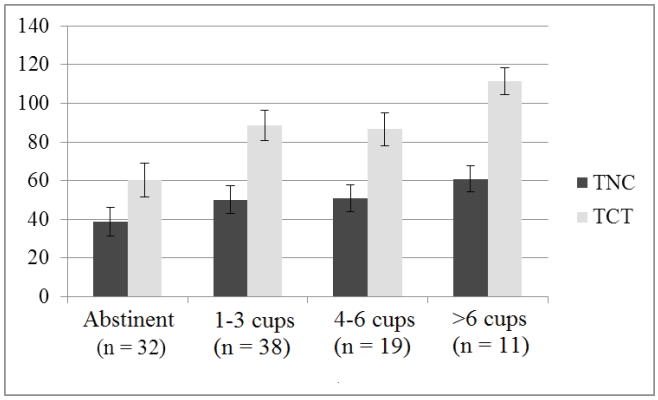


**Fig. 2. Quantitative indices of bruxism *vs.* consumption of caffeine per 24 hours in patients with primary sleep bruxism.**

**Note:** vertically – the total number of clenches (TNC, un.) and their total duration (TCT, s); statistical differences vs. "abstinent" group (* – p <0.05; *** – p <0.001).

TNC Index *vs.* caffeine consumption in patients with primary sleep bruxism: "abstinent" group – 38.7 ± 7.29 un., "1-3 cups" group – 50.1 ± 7.14 un., "4-6 cups" group – 50.8 ± 6.88 un., ">6 cups" group – 60.9 ± 6.63 un. TCT Index *vs.* caffeine consumption in patients with primary sleep bruxism: "abstinent" group – 60.37 ± 8.63 s, "1-3 cups" group – 88.67 ± 7.91 s, "4-6 cups" group – 86.61 ± 8.55 s, ">6 cups" group – 111.45 ± 6.89 s.

Caffeine influences the *occlusal reflex* index, depending on the amount consumed. In people who do not consume caffeine (abstinent), SP2 has a normal inhibition in 16 patients (50%), the other 16 patients (50%) have pathological inhibition. Regular consumption of 1-3 cups of coffee per day leads to pathological disruptions of the SP2 activity in 29 patients (76.3%, p <0.05 as compared to the "abstinent"

group), consumption of 4-6 cups causes pathological disorders of the SP2 inhibition processes in 16 patients (89.5%, p <0.01) and excessive coffee consumption (more than 6 cups) has resulted in the presence of pathological variants of SP2 in all the patients from that group (100%, p <0.001).

**Table 2**

**Indices of autonomous-sympathetic manifestations in the stomatognathic system *vs.* caffeine consumption per 24 hours in patients with primary sleep bruxism**

| jc.SSR Indices | Caffeine consumption | | | |
|---|---|---|---|---|
| | Abstinent (n=32) | 1-3 cups (n=38) | 4-6 cups (n = 19) | >6 cups (n = 11) |
| jc.SSR, $A_2$, mV | 2,15 ± 0,26 | 2,73 ± 0,22 | 2,82 ± 0,34 | 3,5 ± 0,38** |
| jc.SSR, T, s | 9,8 ± 0,31 | 10,0 ± 0,36 | 9,9 ± 0,35 | 10,1 ± 0,34 |

**Note:** statistical differences vs. "abstinent" group (** – p <0.01).

In our investigations for the first time, it was shown that excessive caffeine consumption (more than 6 cups per day) leads to disturbances in the sympathetic autonomous activity in the stomathognathic system (jc.SSR, $A_2$, Table 2) without increasing the central regulation time (jc.SSR, T, s) in the processes of autonomous regulation.

## Discussion

Considering that in the pathogenesis of sleep bruxism, the dysfunction of the cerebral structures takes the main place, it can be assumed that different psychoactive substances (alcohol, caffeine, etc.) can influence the central nervous system, eliciting an important effect on the pathogenetic mechanisms of sleep bruxism. Under day-to-day conditions, people use different psychoactive substances to achieve effects of relaxation, pleasure, or in order to relieve excessive stress or pain [13]. In this regard, we may conclude that sleep bruxism can be viewed as a disease associated with the states of psycho-emotional tension, with the high levels of anxiety and emotional stress, that may require the usage of psychoactive remedies [2, 24, 34, 35]. The analysis of literature data [3, 6, 8, 17, 21, 22, 24] and our own results, highlight a close link between the effects of caffeine and the manifestations of sleep bruxism.

We have established that the higher caffeine consumption in younger people correlates with more severe manifestations of sleep bruxism. Based on these data, it is difficult to establish the main cause – the young age or the excessive caffeine consumption. A recent study, based on questioning of a group of 113 students in Poland, did not reveal any statistically significant associations between coffee consumption and the incidence of bruxism [4].

The stressor psychosocial factors are in a close association with age, these play an important role, both in the pathogenesis of sleep bruxism and as well they influence the consumption degree for psychoactive substances [24]. Under conditions of chronic stress, muscular dysfunctions in the stomatognathic system occur in 34-46.2% of cases [28]. In our study, the presence of stresogenic professions is associated in 81.8% of cases with excessive caffeine consumption, and the manifestations of emotional stress (VAS-stress) are substantially higher at the consumption of 4 or more cups of coffee. The obtained results correlate with the ones from the scientific literature, according to which the effects of caffeine upon the psycho-emotional state are dose-dependent [26, 27].

Caffeine consumption has a pronounced effect not only on the psycho-emotional state but also on the sleep; caffeine consumption 16 hours before sleep, induces a much more superficial sleep [14]. We have determined that caffeine prolongs sleep latency, reduces the total sleep time and sleep efficiency, and the sleep quality is subjectively assessed as being a lower one. Caffeine consumption is associated with the *difficulty of staying asleep* (DSA) [15]. Our results have highlighted the interrelation between the dose of consumed caffeine and the quantitative/qualitative sleep disorders.

In the scientific literature, there are published researches which show that increased caffeine consumption, along with its negative influence upon the psycho-emotional status, as well on the duration and quality of sleep, increases the severity of sleep bruxism [17]. Our results prove that caffeine consumption, regardless of dose, increases the total duration of jaw clenches (TCT), but the consumption of more than 6 cups per day is the landmark dose that increases this index the most (TCT – 111.45 ± 6.89 s, p < 0.001). In comparison with the strong effect of caffeine on the TCT index, the changes of the total number of clenches (TNC) are less pronounced. In this context, it is important to mention the results obtained by Bastien et al. [3], which have shown that the bioelectric activity of the masseter muscle in patients with sleep bruxism under the influence of caffeine consumption did not change significantly when compared to placebo. Perhaps that caffeine mostly maintains and prolongs the pathological activity of masticatory muscles rather than it initiates new episodes of bruxism – this hypothesis is confirmed by the greater change in total clench time (TCT) in comparison with total number of clenches (TNC). In the scientific literature, there is a focus on the caffeine's ability of stimulating the masticatory muscles [8]. In recent years, there have been published several researches, analyzing the structural features of the masticatory muscles by applying ultrasonography. A normal ultrasonographic map of the masseter muscle is expressed by the presence of hyperecogenic strips parallel to the long axis of the muscle [10]. The obtained results show a trend towards increasing of the echogenicity of the masseter muscle that follows the increase in the amount of consumed caffeine, pathological changes occur in the muscles, fascia and connective tissues, expressed as local and/or diffuse changes in the echogenicity. The polymorphism of these changes that occur under the influence of excessive caffeine consumption, emphasizes the broad spectrum action of this psychoactive substance on the whole body and in particular, on the stomatognathic system.

The *feedback* connection between the occlusal contact

and the occlusal muscular force regulates the *occlusal reflex*, which is known in the scientific literature as the trigemino-trigeminal reflex, anti-nociceptive inhibitory reflex of the brain stem [15]. The first inhibitory phase of the occlusal reflex is determined by the inhibitory disinaptic neuronal network (inhibitory interleaving neurons, located in the trigeminal motor nucleus), and the second phase is determined by the inhibitory polysinaptic network (neurons located in the lateral reticulate formation at the level of ponto-medullary junction). These neural networks are strongly influenced by the activity of the limbic, cortical, cerebellar and hypothalamic structures [23, 33]. These modulating effects explain the variability of the occlusal reflex that was observed in our studies, based on the dose of consumed caffeine and the psycho-emotional state of the patients with primary sleep bruxism.

According to the scientific literature data, patients with bruxism did not reveal statistically significant correlations between EMG muscle activity during wakefulness/sleep and the degree of dental abrasion [9, 29]. As a result of the study, there was observed no valid correlation between dental wear and age of patients, between dental wear and occlusal factors, as well as in regard to the degree of temporomandibular joint dysfunction. These data prove that dental wear is associated to a large extent with extra-stomatognathic factors. According to the data from the literature, the presence of dental abrasion is not the main criterion for the diagnosis of sleep bruxism, because it may be found in other diseases as well (acid reflux, long-term consumption of acidic juices, etc.) [12, 19].

Caffeine has been shown to influence the activity of cortico-cerebral neurons and to strongly alter the psycho-emotional state, it increases anxiety and amplifies the subjective sensation of muscle tension [22]. There is evidence that the effects of caffeine are associated with the calcium release in the sarcoplasmic reticulum and the inhibition of its reabsorption, this being associated with the alteration of the neuromuscular function and with the increase in the muscle contracting force [30]. In recent years, the caffeine-emotions-bruxism interaction is studied based on the involvement of the adenosine neuromodulator, release of serotonin, acetylcholine, dopamine, etc. [16]. The obtained results indicate that the activity of the stomatognathic system is closely related to the processes of autonomous regulation, as highlighted by the jc.SSR method. The hypothalamus is the main generator of jc.SSR, and the modulation of the autonomous reaction is accomplished by several brain structures – the reticulate formation, the limbic system, etc. [37]. Under the influence of caffeine, we have established an increase of $A_2$, which is a sign of increased activity of ergotropic centers (sympathicotonia), but the central regulation time has not increased, which confirms the stimulating effects of caffeine.

The preventive results obtained in our studies, indicate that the analysis of caffeine effects on patients with primary sleep bruxism requires a multisystemic approach (Figure 3).

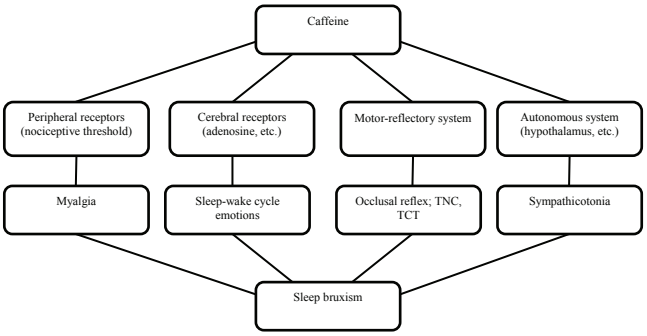


**Fig. 3. Interaction scheme of caffeine effects and the manifestations of primary sleep bruxism.**

Finally, we may conclude that the disorder expression degree under the action of caffeine depends not only on the amount of caffeine consumed, but also on the frequency of consumption, because the discontinuation of consumption leads to the occurrence of abstinence, which lasts up to 5 days – the period necessary for the readaptation of the adenosine sensitivity of the cerebral receptors [24]. During the abstinence periods, psychomotor and autonomous disorders worsen, significantly diminishing the quality and the duration of sleep. Patients with sleep bruxism, who consume excessively caffeine, are advised to gradually reduce the dose.

## Conclusions

1. In patients with primary nocturnal bruxism, there were observed different patterns of caffeine consumption per 24 hours: "abstinent" group – 32.0%; "1-3 cups" group – 38.0%; "4-6 cups" group – 19.0%; ">6 cups" group – 11.0%. Caffeine consumption in patients with primary sleep bruxism decreases with ageing.

2. The presence of highly-stressful jobs, an increased level of emotional stress and hypersympathicotonia are associated with excessive caffeine consumption. Minimal caffeine consumption (1-3 cups) leads to pathological sleep in 13.8% of cases, and excessive consumption (>6 cups) is associated with pathological sleep in 81.8% of cases.

3. Caffeine intake, regardless of dose, increases the overall duration of night clenches, in comparison with a less pronounced effect on the total number of jaw clenches. A pathological occlusal reflex has been observed at the consumption of 1-3 cups of coffee in 76.3% of cases, in 89.5% of cases in the "4-6 cups" group, and in 100% of individuals from the ">6 cups" group; in the abstinent individuals – the occlusal reflex was pathological in 50% of cases.

4. The temporomandibular joint-associated disorders and the myogenic-algic manifestations, reach a significant value at the consumption of 4-6 or more cups of coffee, the thickness of the masseter muscle and dental wear exhibit no statistically significant tendencies of increase under the influence of different doses of caffeine.

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