**Doctoral School in Medical Sciences**

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# THE CLINICAL-NEUROPHYSIOLOGICAL PATTERN IN PATIENTS WITH BRUXISM AND SELF-HELP OPTIONS

**323.01 STOMATOLOGY**

**Abstract of Doctoral Thesis in Medical Sciences**

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## 1. CONCEPTUAL FEATURES OF THE RESEARCH

**The actuality of the topic**. Bruxism is a widely spread condition; in general about 85-90% of the population clench their teeth during certain periods of their life, in 5% of these people clenching transforms into a clinical disorder [3]. The average prevalence of bruxism is about 20% of the population, affecting approximately 14-18% of children, 13% of adolescents, 5-6% of adults and about 3% of people over 60 [9, 10, 11].

There are various theories and hypotheses regarding the etiology and pathogenesis of bruxism: occlusal, muscular, psycho-behavioral, humoral, genetic and others [1, 9, 11, 19]. The high incidence of sleep bruxism is explained by the influence of psycho-emotional factors with a predominant role of emotional stress [7, 9, 20]. It has been shown that emotional stress and anxiety are important factors for the appearance/maintenance of bruxism. Some authors consider occlusal disorders to be the primary etiological factor, however clinically it has been proven that bruxism can be found in patients with normal occlusal indices; moreover, there are patients with significant occlusal disorders without any signs of bruxism [13, 20].

In recent years there were discovered neurogenic interactions between masticatory muscles and different brain centers; it is supposed that there may exist a "bruxism generator" at brainstem level [9, 10], this fact increasing the significance of neurophysiological investigations of this disorder. In many cases, sleep bruxism is latent (subclinical) without being identifiable by means of dental methods [20]. In subclinical variants of sleep bruxism, disease outbreak is already at stages when various complications (dental abrasion, root fractures, etc.) occur. Based on this, it becomes imperative to develop methods for diagnosing bruxism at the initial stages by applying pathogenetically based neurophysiological methods.

Clinical manifestations and management of bruxism depend on the action of multiple etiological and pathogenic factors, among which the daily cofactors of bruxism (such as alcohol, caffeine, nicotine, etc.) play an important role [2, 4, 11, 14]. The role of these cofactors from a diagnostic, clinical and therapeutic aspect is partially deciphered. A slightly less studied aspect is the clinical manifestation of bruxism at different periods of life.

In recent years, there has been achieved great success in the treatment of sleep bruxism and its complications [9, 10, 15]. Currently, there persists the issue of creating individualized programs for the complex treatment of primary sleep bruxism with the inclusion of dental treatment and daily self-help methods (relaxation techniques, meditation, massage etc.) [1, 5, 6, 8, 20].

**The hypothesis of the study**. Clinical manifestations of primary sleep bruxism depend on the patients'age, the presence/intensity of intrinsic factors (tooth damage, temporomandibular joint dysfunction etc.) and extrinsic factors (psycho-emotional disorders, professional activity, daily cofactors of bruxism – alcohol, caffeine, nicotine etc.), the study of which will allow to elucidate some new pathogenetic aspects of primary bruxism, to optimize the diagnosis with the identification of the predominant clinical-neurophysiological patterns, will allow to create individualized treatment programs and self-help recommendations in a day-to-day setting, which can contribute to the enhancement of the complex treatment.

**The purpose of the research**. Optimization of the diagnosis and treatment of primary sleep bruxism based on identifying of the clinico-neurophysiological particularities of bruxism manifestation in relation to the patients' age and the influence of the daily cofactors, creating pathogenetically-based self-help methods.

**The objectives of the research.**

1. The analysis of the clinical particularities, ultrasonographic indices of masticatory muscles and

of the autonomous regulation processes in stomatognathic system in patients with primary sleep bruxism based on their age and the influence of the daily cofactors of bruxism.
2. The study of the peculiarities of manifestation of primary sleep bruxism based on the prevalence of nasal or oral diurnal respiration.
3. The elaboration of pathogenetically-based daily self-help methods in patients with primary sleep bruxism by applying respiratory-individualized splints and by stimulation of the reflexogenic zones of the stomatognathic system.
4. The increase the treatment efficacy of the primary sleep bruxism based on the combination of different types of splints and self-help methods.

**Methodology of the scientific research**. The methodology of the scientific research was based on the concept of diagnosis and treatment of sleep bruxism following European and American dental guidelines [3, 9]. The research was based on the concepts regarding sleep bruxism that include the role of the patients' age, the influence of psycho-emotional factors and of the bruxism cofactors (alcohol, caffeine, nicotine, etc.) [2, 7, 9, 10, 14]. The researches were based on the multi-systemic and multifactorial concept of dental disorders correction in patients with bruxism [9]. The investigation and interpretation of the results was accomplished based on the concept that refers to the complex interrelation of bruxism and emotional stress [17].

**Scientific novelty and originality.**

• Scientific originality relies on the analysis of clinico-neurophysiological changes in patients with primary sleep bruxism based on their age, with the identification of clinical particularities, occlusal reflexes, autonomous regulation processes in stomatognathic system and structural-ultrasonographic indices of the masticatory muscles.
• Novel is the identification of peculiarities regarding the activity of the stomatognathic system in patients with primary sleep bruxism under the influence of psychoactive substances (alcohol, caffeine, nicotine) and with a various level of patients' physical activity.
• Novel are the obtained results that reflect the thermo-vasomotor-metabolic changes of the masticatory muscles in the patients with primary sleep bruxism based on the patients' age, the action of the psychoactive substances (alcohol, nicotine, caffeine) and the level patients' physical activity.
• For the first time there has been demonstrated the influence of the psychoactive substances (alcohol, caffeine, nicotine) and of physical activity upon the processes of autonomous regulation in the stomatognathic system in patients with primary sleep bruxism.
• Original is the systematization of the main components of the disorders in patients with sleep bruxism (psychogenic, reflective-occlusal, myogenous, arthrogenic, dento-periodontal, respiratory, autonomous, algic), which form the predominant clinical-neurophysiological patterns.
• For the first time there have been identified the particularities of the occlusal reflex associated with the efficacy of applying myorelaxing splints in patients with primary sleep bruxism.
• Novel is the obtained data regarding the scientific argumentation of the possibilities of correcting bruxism-associated disorders by means of applying the splint-respiratory treatment and by stimulation of the auricular reflexogenic zones of the stomatognathic system.

**The main scientific results submitted for the defense.**

1. In patients with primary sleep bruxism, there are present various variants of the clinical neuro-neurophysiological patterns, the structure and the expressivity of which depend on the age and the action of daily cofactors of bruxism.
2. Based on the complex diagnosis (clinical, psychological, neurophysiological, electrophysiological, radiological) there are evidenced clinico-neurophysiological particularities

of the primary sleep bruxism, which make the differentiated treatment possible.

3. Clinical manifestations of primary sleep bruxism may be associated with diurnal oral breathing syndrome, requiring an individualized treatment.

4. The indication and application of myorelaxing splints based on clinical-neurophysiological indices substantially increases their effectiveness.

5. Splint-respiratory treatment and the reflex-bruxism-regulating self-help method in patients with primary sleep bruxism diminishes the frequency and intensity of nocturnal episodes of bruxism, improves the psycho-emotional condition and the autonomous regulation processes in the stomatognathic system.

**The implementation of the scientific results**. The scientific results were implemented in the research process and clinical activity at the Municipal Stomatological Center (Chisinau), Republican Stomatological Policlinic, "Megalux Dent" Dental Clinic.

**The approval of the scientific results**. The results were presented at national and international scientific forums: XVIII Congress of The Association of Stomatologists from the Republic of Moldova (Chisinau, 2016); Scientific Conference – Days of the State University of Medicine and Pharmacy "Nicolae Testemitanu" (Chisinau, 2016); National symposium with international participation NANO-2016 (Chisinau, 2016); Congress of physiologists from CIS countries (Soci, 2016); V Congress of Biochemists from Russia (Soci-Dagomys, 2016); VIII International Congress of the Romanian Dental Association for Education (Iasi, 2016); XX International Congress of UNAS (Bucharest, 2016). The endorsement of the thesis title took place during the meeting of the Scientific Council of USMF "Nicolae Testemitanu" – minutes no. 4/6.2 from July 4$^{th}$ 2016. The positive opinion of the Research Ethics Committee for the study was obtained: minutes no. 37 of 04.05.2016. The results were approved at the meeting of the Department of Therapeutic Dentistry of USMF "Nicolae Testemitanu" from 19.12.2018, Scientific Seminar on Dentistry Profile from 20.03.2019. The research was conducted at the Department of Therapeutic Dentistry of USMF "Nicolae Testemitanu", Dental Clinic "Fala Dental "and at the University Clinic “Neuronova”.

**Key words**: primary sleep bruxism, clinical neurophysiological pattern, self-help, co-factors of bruxism.

## 2. MATERIAL AND RESEARCH METHODS

### 2.1. General characteristics of the researched group (sample volume, study design, inclusion/exclusion criteria)

In the study there were enrolled patients aged 18-50: the group aged under 35 included 70 patients, between 35-50 – 30 patients; women in total – 68, under 35 years – 50 women, between 35-50 –18; men in total – 32, under 35 years – 20, between 35-50 – 12. We have analyzed the action of the co-factors of bruxism (alcohol, caffeine, nicotine, hypodynamic activity) and the effectiveness of complex treatment with the use of splints and self-help methods.

*Criteria for inclusion in the research*: Positive clinical diagnosis of primary sleep bruxism, confirmed by objective recording of the nocturnal episodes; total dentition or partial edentulism up to 1-3 teeth; age of patients between 18-50 years; presence of patient’s consent for participation in research; cooperating patients.

*Criteria for exclusion from the research*: age does not fit the established limits; other clinical forms of bruxism (central nervous system disorders, epilepsy, parkinsonism, etc.); the presence of anomalies and inflammatory signs in the stomatognathic system; presence of signs of organic damage (bioelectric activity recorded via electromyography (EMG) in the state of relaxation is

less than 30 mcV); various acute and chronic diseases during periods of aggravation, parasitosis; alcoholism, drug addiction, toxicomania, mental illnesses; treatment with psychotropic medicines, anticonvulsants, miorelaxants; lack of patients' consent for participation in the research; non-cooperating patients.

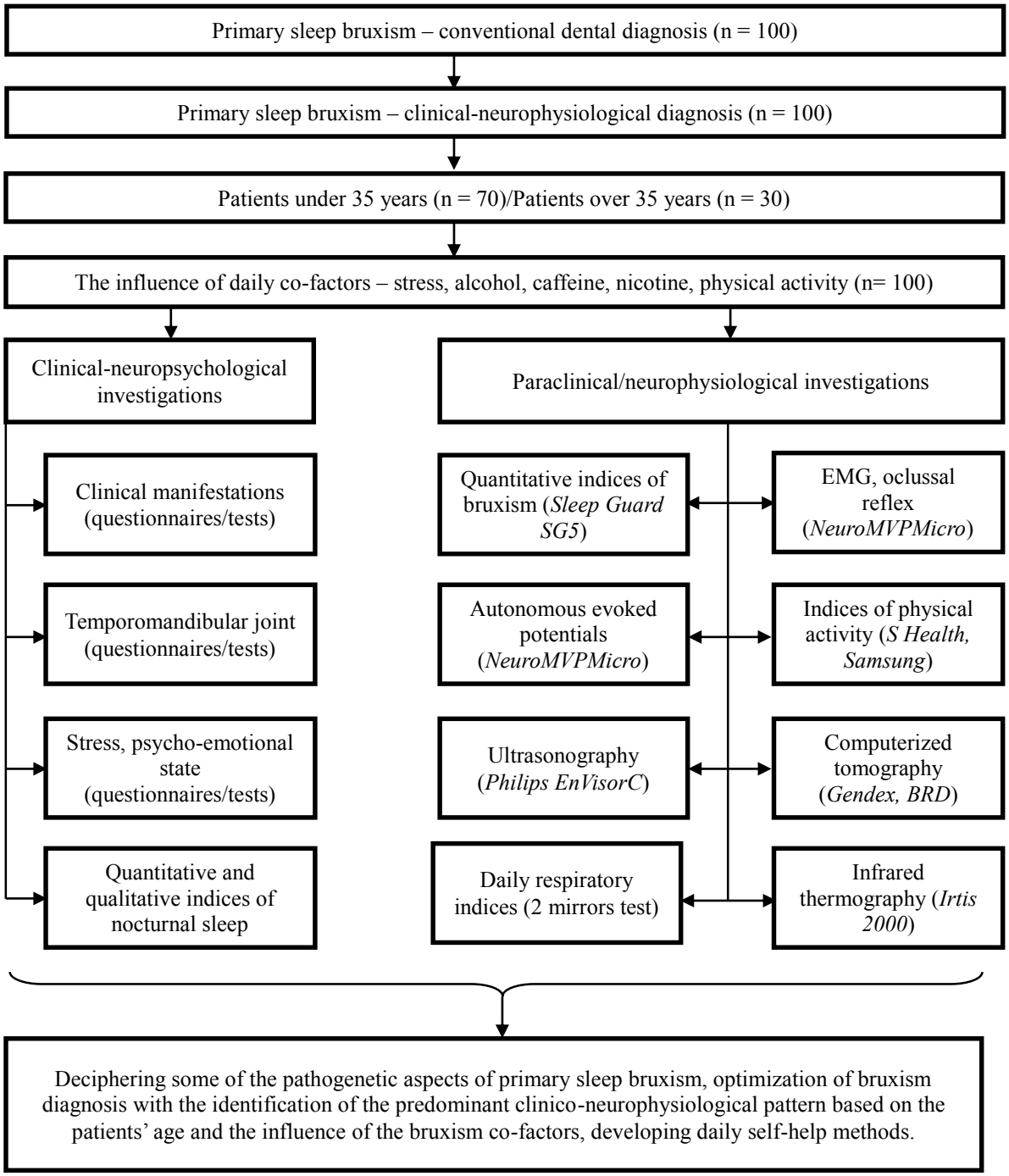


Figure 21. **The study design regarding the patients with primary sleep bruxism based on the patients' age and the action of co-factors of bruxism.**

### 2.2. Clinical and psychometric methods

We have applied specialized questionnaires and tests [9, 10, 18]: bruxism questionnaire, Fonseca questionnaire, multifactorial questionnaire of bruxism-associated pain, sleep quality questionnaire, palpation of the masticatory muscles, evaluation of dental abrasion, evaluation of the emotional stress level using the visual analogue scale (VAS). In order to analyze the type of

breathing (nasal/oral/mixed) and the hypoxic resistance we have applied the "2 mirrors test" and the Shtange test.

**2.3. Electrophysiological and neurophysiological methods**

The surface electromyography recordings of the masticatory muscles, the determination of the masseter inhibitory reflex (MIR), and the recording of the jaw clenching sympathetic skin responses (jc.SSR) were performed by means of the diagnostic complex Neuro-MVP Micro (Neurosoft). In order to determine the Total Number of Clenches (TNC) and Total Clench Time (TCT) we have applied the portable Sleep Guard SG5 (Hollistic Inc., USA).

**2.4. Imaging methods**

We have used computerized tomography (Gendex, Germany), ultrasonography (USG) (Envisor C, Philips) of the stomatognathic system [12] and infrared thermography (IRTIS 2000, Russia) [22].

**2.5. The treatment of primary sleep bruxism and the self-help methods**

Patients received dental treatment based on indications (abrasions, fissures, etc.). We have applied the Reflex-Bruxism-Regulating (RBR) method, which is based on the stimulation of the auricular reflexogenic zones of the stomatognathic system. Myorelaxing splints were used by 50 patients with sleep bruxism (SB) as monotherapy and by 50 patients in combination with the reflex-bruxism-regulating method. Splint-respiratory treatment was applied to 13 SB patients with predominant oral respiration and to 30 patients with predominant nasal breathing. The respiratory training was used in order to diminish the psycho-autonomous disorders [21] and to adapt the stomatognathic system to the applied splints.

**2.6. Mathematical and statistical processing of the research material**

The results obtained were processed with the help of the software package for statistical analysis *Statistics for Windows*, *Release 11.0 StatSoft, Inc.* (US). We have determined the arithmetic mean and the standard error (SE). We have used the Student test to compare the averages of a specific feature between two groups. In order to determine the degree and the type of correlation between the studied values we have used the Pearson and Spearmen correlation coefficients. We have calculated the efficacy coefficient in order to assess the effectiveness of the applied treatment, by using:

$$\beta = 1 - \frac{(F-N)}{(I-N)} \times 100\% \quad (1)$$

where: I – the value of the parameters before the treatment; F – final parameters after the treatment; N – parameters in healthy people.

## 3. THE CLINICAL-NEUROPHYSIOLOGICAL PATTERN OF PRIMARY SLEEP BRUXISM BASED ON THE AGE OF THE PATIENTS AND THE INFLUENCE OF DAILY COFACTORS

**3.1. Manifestations of bruxism based on the patients' age**

Based on the fact that increased stress reactivity contributes to the occurrence of several disorders of SB, people with SB have a much higher level of stress activity, and in most cases they refer to A type personalities [7, 11, 17]. We have studied the level of emotional stress and the professions of the patients vs. the patients' age. In healthy people, highly stressful professions constituted up to 30% of the cases; the expression of VAS-stress – 3.80 ± 0.35 pt. In patients with SB of various age (18-50 years, n = 100), highly stressful professions amounted to 24% of the cases; VAS-Stress – 5.71 ± 0.31 pt. In patients with SB, aged under 35, highly stressful professions made up to 21.4% of the cases; VAS-stress – 5.99 ± 0.28 pt. In patients with SB, aged over 35,

highly stressful professions amounted to 30% of the cases; VAS-stress – 5.43 ± 0.35 pt. Sleep quality in patients with SB, aged under 35 (statistical significance in comparison to the data regarding older patients): normal sleep – 20 patients (28.6%), $p>0.05$; borderline disorders – 38 patients (54.3%), $p<0.01$; pathological sleep – 12 patients (17.1%), $p<0.001$. The quality of sleep in patients with SB, aged over 35, based on self-assessment, is the following (statistical significance in comparison to the data regarding younger patients): normal sleep – 4 patients (13.3%), $p>0.05$; borderline disorders – 8 patients (26.7%), $p<0.01$; pathological sleep – 18 patients (60.0%), $p<0.001$. In patients, aged under 30, clinical manifestations of SB are more pronounced in comparison with older patients: bruxism questionnaire – $p<0.05$; pain questionnaire – $p>0.05$; Fonseca questionnaire – $p<0.05$, gradient of masseter muscle thickness (GMT) – $p<0.001$.

The pain pattern in patients aged under 35 can be characterized as psycho-emotional-myogenous circadian pain and in older patients as psycho-emotional-myogenous-arthrogenic circadian pain (figure 31).

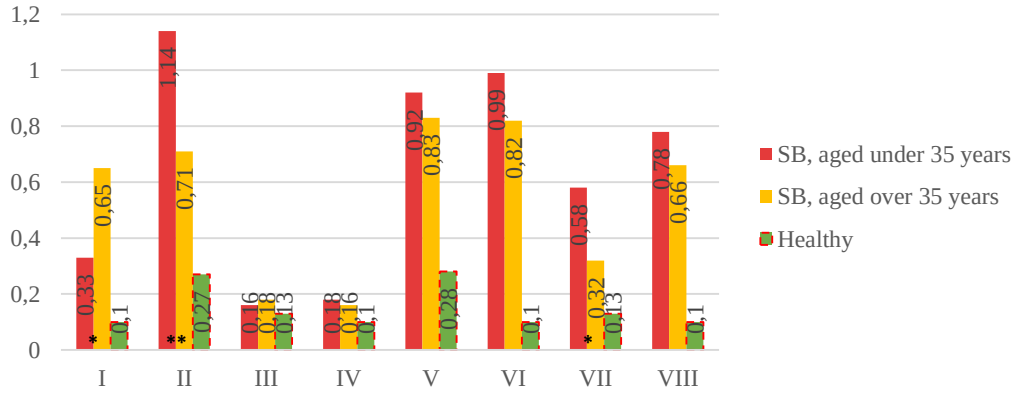


Figure 31. **The intensity of bruxism-associated pain in patients with sleep bruxism in correlation with age.**

***Note:*** *vertically – symptoms intensity (pt.); horizontally – bruxism-associated pain scales (factors): I – arthrogenic; II – myogenous; III – loco-regional referral; IV – autonomous; V- psycho-emotional; VI – circadian; VII – pain intensity; VIII – pain duration. Statistically significant differences: SB, patients under 35 years vs. patients over 35 years – (* – $p < 0,05$; ** – $p < 0,01$).*

TNC indices in healthy individuals were 3.4 ± 0.95 units (un.); in patients with SB (n = 100) – 50.4 ± 5.96 un; in patients with SB, aged under 35 – 65.4 ± 5.26 un.; in patients with SB, aged over 35 – 35.4 ± 6.44 un.; TCT indices in healthy individuals were 3.6 ± 0.68 seconds; in patients with SB (n = 100) – 86.5 ± 7.93 s; in patients with SB, aged under 35 – 110.5 ± 8.22 s; in patients with SB, aged over 35 – 61.5 ± 6.91 s. The analysis of the occlusal reflex indices (MIR) revealed that the SP2 (occlusal reflex phase 1) variants – SP2 – normal inhibition, and SP2 – partial inhibition are informative for the differentiation of occlusal disorders among healthy individuals and patients with SB in general, regardless of age ($p<0.001$), however, for the differentiation of occlusal disorders between patients with SB under and over 35 years old, the SP2 component – no inhibition is more informative ($p<0.05$, Figure 32).

Normally, in healthy individuals in 100% of cases in the postural position of the mandible (state of relaxation), based on the data acquired via infrared thermography, the activity of the thermo-vasomotor-metabolic processes in the anterior temporal muscle (AT) predominates the one from the masseter muscle (MM), AT>MM).

In patients with SB, aged under 35 in 11.4% of cases, there can be observed a predomination

of the thermo-vasomotor-metabolic activity of the masseter muscles (AT<MM), indicating the discoordination of these processes, in comparison to the healthy individuals, and in individuals aged over 35, in already 23.3% of cases the thermo-vasomotor-metabolic processes predominate in the masseter muscle (AT<MM).

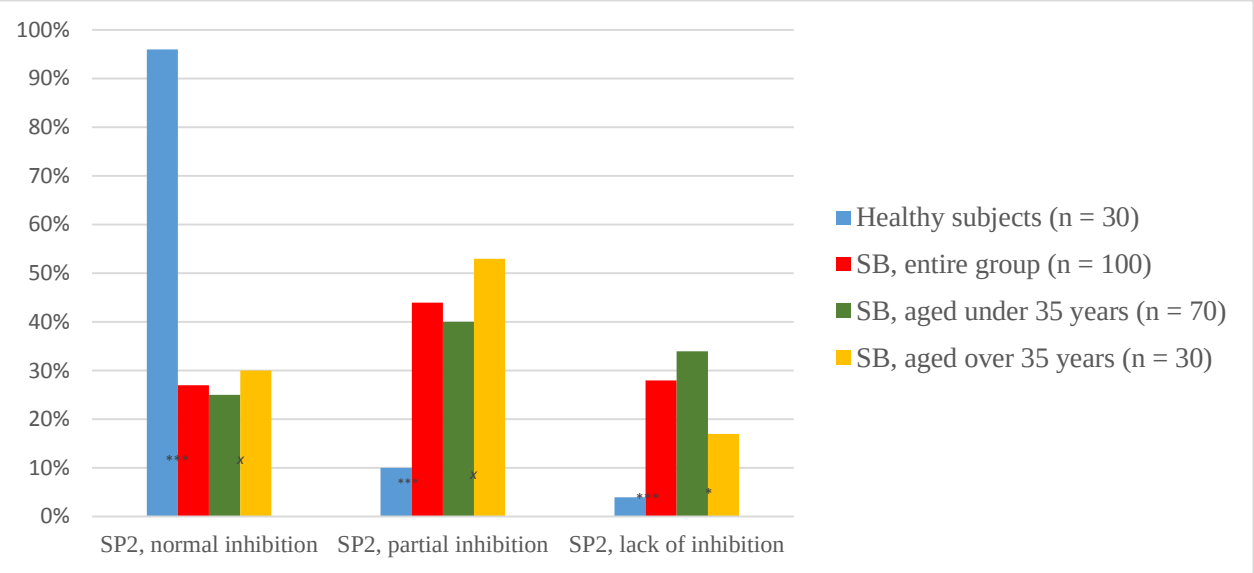


Figure 32. **Frequency of EMG indices of the occlusal reflex (MIR) in healthy individuals and patients with sleep bruxism.**

***Note:*** *vertically – the frequency of SP2 variants; statistically significant differences ($^{*}$ – $p < 0,05$; $^{***}$ – $p < 0,001$; $^{x}$ – $p > 0,05$).*

The obtained results show that the sympathetic autonomous response in the standardized jaw-clenching conditions is informative in the detection of sympathetic autonomous disorders in the masticatory muscles: the amplitude of the autonomous-sympathetic response at a standardized jaw clenching (jc.SSR-S, A2) in patients aged under 35 – 2.52 ± 0.17 mV, in individuals aged over 35 years – 3.11 ± 0.21 mV ($p<0.05$); the restoration period for the evoked autonomous-sympathetic potential (jc.SSR-S, T) in patients under 35 – 9.3 ± 0.35 s, in individuals aged over 35 years – 10.5 ± 0.43 s ($p<0.05$ ). These indices can be used with the aim of differentiating the patients with SB from the healthy individuals and for determining the severity of the autonomous disorders based on the age of patients with SB.

### 3.2. Daily cofactors of the primary sleep bruxism

Stressogenic professions in alcohol-abstinent patients constitute 26.3%, in patients who consume 1-3 un. of alcohol the presence of stressful professions is 22.0%, and at the consumption of more than 3 un. of alcohol – 27.3%. The expression of VAS-stress in abstainers – 5.2 ± 0.34 pt; alcohol consumption of 1-3 un. – 5.6 ± 0.39 points (pt); alcohol consumption of more than 3 un. – 6.3 ± 0.36 pt. The duration of nighttime sleep in patients with SB based on the alcohol consumption is of 7.8 ± 0.56 hours in abstainers; 7.2 ± 0.48 hours at a consumption of 1-3 un. of alcohol and of 6.4 ± 0.43 hours at a consumption of more than 3 un. Consumption of 1-3 un. of alcohol leads to the occurrence of pathological sleep in 9 out of 59 patients (15.3%), but excessive alcohol consumption (more than 3 un. in 24 hours) is associated in 81.8% of cases with the presence of pathological sleep (18 patients out of 22 investigated, $p<0.001$ vs. abstainers). Clinical expression of SB is much higher in people with excessive alcohol consumption (Table 31).

TNC vs. alcohol consumption in patients with primary sleep bruxism: abstainers – 38.4 ± 3.75 un., alcohol consumption 1-3 un. – 47.7 ± 7.02 un., alcohol consumption of more than 3 un. – 65.1 ± 3.96 un. TCT vs. alcohol consumption in patients with SB: abstainers – 58.75 ± 7.38 s, alcohol consumption 1-3 un. (n = 59) – 84.9 ± 8.15 s, alcohol consumption of more than 3 un. – 120.43 ± 8.46 s. Out of 19 abstinent patients, normal inhibition of SP2 was found in 9 individuals (47.4%), pathological inhibition – in 10 patients (52.6%). In patients with an alcohol consumption of 1-3 un. of alcohol, an SP2 with normal inhibition was observed in 7 individuals (11.9%, $p<0.01$ in comparison to abstainers) and in 52 individuals (88.1%, $p<0.01$), there was a various degree of pathological SP2 values (partial inhibition/no inhibition). The most significant changes for SP2 were observed at the consumption of more than 3 un. of alcohol: normal inhibition is present in only 3 patients (13.6%, $p<0.05$) and pathological MIR was observed in 19 patients (86.4% $p<0.05$).

Table 31. **Age of patients, clinical manifestations of bruxism and ultrasonographic indices of the masseter muscle vs. alcohol consumption per 24 hours.**

| Indices | Alcohol consumption | | |
|---|---|---|---|
| | **Abstainers (n=19)** | **1-3 un. (n=59)** | **>3 un. (n=22)** |
| Age, years | 38.7 ± 1.22 | 32.6 ± 1.16** | 28.3 ± 0.82*** |
| Bruxism questionnaire, pt. | 3.1 ± 0.18 | 3.6 ± 0.25 | 7.8 ± 0.22*** |
| Pain questionnaire, pt. | 5.20 ± 0.42 | 5.08 ± 0.39 | 3.85 ± 0.31** |
| Fonseca questionnaire , pt. | 42.34 ± 3.58 | 53.49 ± 3.91 | 60.23 ± 4.28** |
| USG, GMT index, % | 26.7 ± 0.59 | 28.4 ± 0.48 | 30.3 ± 0.51*** |
| USG, echogenicity, un. | 1.10 ± 0.11 | 1.38 ± 0.10 | 1.49 ± 0.11* |
| Dental wear, un. | 1.40 ± 0.21 | 1.90 ± 0.18 | 2.50 ± 0.16*** |

***Note**: statistical differences vs. abstainers (* – $p < 0,05$; ** – $p < 0,01$; *** – $p < 0,001$); there are indicated the arithmetic mean and standard error (M ± SE).*

In patients with higher alcohol consumption (>3 un.), there is also a statistically significant increase in the sympathetic activity in the stomatognathic system. jc.SSR-S, A2 mV: alcohol >3 un. vs. abstainers – 2.2 ± 0.19 ($p<0.001$); jcSSR-S, T, s: alcohol >3 un. vs. abstainers – 10.4 ± 0.34 ($p<0.05$). According to the infrared thermography data, simultaneously with the increase of the amount of the consumed alcohol, there is an increase in the thermo-vasomotor-metabolic disorders in AT/MM muscles, with the most serious disorders being observed in patients that consume excessive amounts of alcohol (more than 3 un./24 hours) in comparison to healthy individuals ($p<0.001$).

Frequency of highly stressful professions vs. caffeine consumption in patients with SB: abstainers (n = 32) – 15.6%, consumption of 1-3 cups (n = 38) – 10.5%, consumption of 4-6 cups (n = 19) – 6%, consumption of more than 6 cups (n = 11) – 81.8%. VAS-stress in abstainers was 4.8 ± 0.31 pt., in patients who consumed 1-3 cups – 5.2 ± 0.33 pt.; in patients who consumed 4-6 cups – 6.1 ± 0 , 32-pt., in patients who consume more than 6 cups – 6.7 ± 0.23 pt. Statistical differences in comparison to abstainers were observed in the group of patients who consume 4-6 cups in 24 hours ($p<0.01$) and in the group with a consumption of more than 6 cups ($p<0.001$). According to self-assessment, pathological sleep in caffeine-abstinent patients was found in 12.5% of cases; minimal caffeine consumption (1-3 cups) leads to pathological sleep in 13.2% of cases; moderate consumption (4-6 cups of coffee) is associated with the presence of pathological sleep

in 63.2% of cases, and excessive caffeine consumption is manifested by the disturbance of sleep quality in 81.8% of cases. The duration of night sleep tends to decrease, however the differences between the studied groups are insignificant: abstainers – 7.6 ± 0.58 hours; 1-3 cups of coffee – 7.5 ± 0.53 hours; 4-6 cups – 6.8 ± 0.51 hours; more than 6 cups – 6.2 ± 0.45 hours. The clinical manifestations of SB in correlation with the consumed caffeine doses have certain peculiarities with the intensification of disorders at caffeine overuse (Table 32).

TNC indices vs. caffeine consumption in patients with SB: abstainers (n = 32) – 38.7 ± 7.29 un., consumption of 1-3 cups (n = 38) – 50.1 ± 7.14 un., consumption of 4-6 cups (n = 19) – 50.8 ± 6.88 un., consumption of more than 6 cups (n = 11) – 60.9 ± 6.63 un. TNC indices vs. consumption of caffeine in patients with primary sleep bruxism: abstainers – 60.37 ± 8.63 s, consumption of 1-3 cups – 88.67 ± 7.91 s, consumption of 4-6 cups – 86.61 ± 8.55 s, consumption of more than 6 cups – 111.45 ± 6.89 s. In individuals who do not consume caffeine there can be observed a SP2 with normal inhibition in 16 patients (50%) and with a pathological inhibition – 16 patients (50 %). Consumption of 1 to 3 cups of coffee per day leads to pathological disorders of the SP2 activity in 29 patients (76.3%, $p<0.05$ in comparison with abstainers), consumption of 4-6 cups causes pathological disorders of SP2 inhibition in 16 patients (89.5%, $p<0.01$) and excessive consumption (more than 6 cups) results in the appearance of pathological variants of SP2 in all patients (100%, $p<0.001$) in this group.

Table 32. **Patients' age, clinical manifestations of bruxism, and ultrasonography indices of masseter muscle vs. caffeine consumption per 24 hours in patients**

| Indices | Caffeine consumption | | | |
|---|---|---|---|---|
| | **Abstainers (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, GMT 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. abstainers (*- $p<0.05$; ***- $p<0.001$); there are indicated the arithmetic mean and standard error (M ± SE).*

In our investigations for the first time, it was observed that excessive caffeine consumption (more than 6 cups per day) leads to disturbances of the sympathetic autonomous activity in the stomatognathic system: jc.SSR-H, A2, mV: 4.6 ± 0.38 vs. abstainers 3.1 ± 0.41 ($p<0.05$); jc.SSR-S, A2 mV: 3.5 ± 0.38 vs. abstainers 2.15 ± 0.26 ($p<0.01$). Unlike other psychoactive factors (nicotine, alcohol) under the influence of caffeine, there is no evidence of increased conduction time (jc.SSR, T, s) in autonomous regulation processes, the fact that confirms the stimulating effects of caffeine. The analysis of thermo-vasomotor-metabolic indices of AT/MM muscles according to the infrared thermography data has revealed pathological disorders in individuals that consume more than 6 cups in 24 hours.

Frequency of stressogenic professions vs. nicotine consumption is the following: 10.0% in

abstainers, 12.0% in smokers of 1-5 cigarettes and 40.0% in smokers of more than 5 cigarettes. Indices of emotional stress expression vs. nicotine consumption in patients with SB is as follows: abstinent patients – VAS-stress 5.1 ± 0.29 points; smokers of 1-5 cigarettes – 6.1 ± 0.33 pt.; smokers of more than 5 cigarettes – 5.9 ± 0.21 pt. Statistical differences ($p<0.05$) compared to abstainers was evidenced in smokers of 1-5 and more than 5 cigarettes in 24 hours. According to the self-assessment of sleep in 23.3% of cases, there is a pathological sleep in non-smoking patients; smoking of 1-5 cigarettes per day leads to the occurrence of pathological sleep in 36.0% of cases, and excessive smoking (more than 5 cigarettes) is associated with pathological sleep in 31.1% of cases. Sleep duration is as follows: abstainers- 7.2 ± 0.56 hours; smokers of 1-5 cigarettes – 7.6 ± 0.48 hours; smokers of more than 5 cigarettes in 24 hours – 6.5 ± 0.53 hours. Differences between groups are statistically insignificant ($p>0.05$).

There has been observed an important clinical particularity in smokers – the consumption of more than 5 cigarettes/24 hours leads to the increase in subjectively assessed disorders (bruxism questionnaire) and the predominance of temporomandibular joint dysfunction (Fonseca questionnaire) (Table 33). In abstinent individuals, TNC values were equal to 41.4 ± 6.45 un., and TCT – 63.34 ± 6.83 s. In patients with SB who smoke 1-5 cigarettes (n = 25) the TNC had values of 43, 8 ± 6.0 un., and TCT – 77.96 ± 8.15 s; in patients with SB who smoke more than 5 cigarettes (n = 45) the TNC has values of 63.0 ± 5.37 un. and TCT – 117.18 ± 7.76 s. In non-smokers, there were observed normal values of SP2 component of occlusal reflex in 10 patients (33.3%) and pathologic SP2 was found in 20 patients (66.7%); in smokers of 1-5 cigarettes pathological SP2 has been observed in 18 patients (72%) and in smokers of more than 5 cigarettes pathological SP2 is present in 35 patients (77.8%).

Table 33. **Patients' age, clinical manifestations of bruxism, and ultrasonography indices of masseter muscle vs. nicotine intake per 24 hours in patients.**

| Indices | Nicotine intake | | |
|---|---|---|---|
| | **Abstainers (n = 30)** | **1-5 cigarettes (n = 25)** | **>5 cigarettes (n = 45)** |
| Age, years | 37.2 ± 1.24 | 27.0 ± 1.21*** | 35.4 ± 1.35 |
| Bruxism questionnaire, pt. | 3.7 ± 0.24 | 4.4 ± 0.31 | 6.5 ± 0.22*** |
| Pain questionnaire, pt. | 4.28 ± 0.44 | 4.6 ± 0.41 | 5.25 ± 0.38 |
| Fonseca questionnaire, pt. | 49.08 ± 3.85 | 45.62 ± 4.01 | 61.36 ± 4.42* |
| USG, GGM indices, % | 27.5 ± 0.57 | 28.4 ± 0.54 | 29.3 ± 0.48* |
| USG, echogenicity, un. | 1.12 ± 0.11 | 1.46 ± 0.12* | 1.47 ± 0.10* |
| Dental wear, un. | 1.62 ± 0.25 | 1.84 ± 0.21 | 2.35 ± 0.15** |

***Note:*** *statistical differences vs. abstainers (* – $p<0.05$; ** – $p<0.01$; *** – $p<0.001$); there are indicated the arithmetic mean and standard error (M ± SE).*

In patients who smoked more than 5 cigarettes per day there is observed the statistically significant increase of the period jc.SSR-S, T, s: 11.0 ± 0.32 vs. abstainers – 9.5 ± 0.31 ($p<0.01$). With the increase of the number of cigarettes, there is observed an increase of the thermo-vasomotor-metabolic disorders, with the most serious disorders in smokers of more than 5 cigarettes ($p<0.001$).

Highly stressogenic professions occur in 25.0% of cases in patients with normal motor activity (5.18 ± 0.25 km/24 hours) and in 23.5% of cases in patients with reduced motor activity

(2.50 ± 0.11 km/24 hours). The emotional stress (VAS – stress) indices are characterized by the following values: normal motor activity – 5.2 ± 0.31 pt.; hypodynamic activity 6.2 ± 0.32 pt. ($p<0.05$). In patients with normal physical activity the presence of pathological sleep is observed in 6.3% of cases and in patients with hypodynamic activity, pathological sleep is present in 41.2% of cases ($p<0.001$). Sleep duration tends to decrease in patients with hypodynamic activity: normal activity – 7.7 ± 0.51 hours; hypodynamic activity – 6.4 ± 0.48 hours. Patients with hypodynamic activity have pronounced clinical manifestations of SB at a subjective level (bruxism questionnaire etc.) in association with pathological changes in the masticatory muscles (pathological ecogenity, thickness of the muscles) (Table 34).

The TNC in individuals with normal motor activity is equal to 34.8 ± 7.88 un., and in those with reduced motor activity 66.3 ± 6.94 un. TCT in individuals with normal motor activity is 55.33 ± 7.79 s and in people with reduced motor activity 122.65 ± 8.32 s. Normal physical activity is associated in 20 patients (62.5%) with MIR indices within the norm limits, and in 12 patients (37.5%) the SP2 inhibition processes are deregulated. Reduced physical activity is associated with MIR indices within the normal range in 7 patients (10.3%) and with varying degree of deviation in 61 patients (89.7%, $p<0.001$ in comparison with abstainers). Autonomous indices are very informative in patients with SB and hypodynamic activity: the amplitude of the autonomous-sympathetic response to a normal jaw clenching (jc.SSR-H, A2, mV) 4.4 ± 0.31 vs. normal motor activity 3.2 ± 0.36 ($p<0.05$), jSSSR-S, A2, mV 3.2 ± 0.25 vs. normal motor activity 2.4 ± 0.24 ($p<0.05$).

Table 34. **Patients' age, clinical manifestations of bruxism and ultrasonography indices of masseter muscle vs. motor activity per 24 hours in patients.**

| Indices | Motor activity | |
|---|---|---|
| | **Normal (n = 32)** | **Reduced (hypodynamic activity) (n = 68)** |
| Age, years | 29.6 ± 1.12 | 37.0 ± 1.34*** |
| Bruxism questionnaire, pt. | 3.6 ± 0.29 | 6.1 ± 0.33*** |
| Pain questionnaire, pt. | 3.95 ± 0.36 | 5.47 ± 0.45* |
| Fonseca questionnaire, pt. | 43.85 ± 3.49 | 60.19 ± 4.52* |
| USG, GGM indices, % | 27.65 ± 0.59 | 29.15 ± 0.43* |
| USG, echogenicity, un. | 1.20 ± 0.11 | 1.50 ± 0.09* |
| Dental wear, un. | 1.75 ± 0.16 | 2.12 ± 0.22 |

***Note:*** *statistical differences: hypodynamic activity vs. normal motor activity (* – $p<0.05$;*** – $p<0.001$); there are indicated the arithmetic mean and standard error (M ± SE).*

The thermo-vasomotor-metabolic ratio of AT/MM muscles according to infrared thermography data in patients with SB and hypodynamic activity is essentially deregulated ($p<0.001$) in comparison with indices in healthy individuals.

## 4. DAILY SELF-HELP TECHNIQUES IN THE COMPLEX TREATMENT OF PRIMARY SLEEP BRUXISM

### 4.1. The effectiveness of the reflex-bruxism-regulating method in correlation with the level of emotional stress

In patients with SB and signs of emotional stress (VAS-stress >5 pt.) under the influence of

the RBR method there were observed improvements of VAS-stress ($p<0.001$), sleep quality ($p<0.01$) and autonomous regulating processes ($p<0.05$). Correction efficacy (β) for TNC – 56.0%, TCT – 65.0%. In patients with SB *with no signs of emotional stress* (VAS-stress <5 pt.) the application of the RBR method caused positive tendencies ($p>0.05$) for VAS-stress, night sleep quality and autonomous regulation processes. The correction efficacy (β) for TNC – 63.0% and TCT – 69.0%.

In *healthy individuals with signs of emotional stress* (VAS-stress >5 pt.) the application of the RBR method caused positive changes in the psycho-emotional, autonomous state and TNC, TCT indices, but in *healthy individuals without signs of emotional stress* (VAS-stress <5 pt.), the application of the RBR method led to a harmonization of psycho-emotional, autonomous and motor functions (TNC, TCT).

Based on the obtained results, it can be assumed that under the influence of the RBR method the transformation of the stress-pathogenic bruxism into the stress-sanogenic one occurs.

**4.2. The effectiveness of the reflex-bruxism-regulating method in combination with myorelaxing splints**

The analysis of the results in patients with SB who used splints and did not apply RBR method revealed that in the cases of normal inhibition of SP2, the indices of masticatory muscle hyperactivity (TNC, TCT) show a tendency towards normalization, however the values do not reach the statistically significant threshold even after 6 months ($p>0.05$) (Table 41). The analysis of autonomous indices (jc.SSR-H, A2 and jc.SSR-H, T) revealed a slightly pronounced change in all the periods of investigation. It should be noted that the level of emotional stress tended to diminish, however the values did not reach the threshold of statistical significance. Another picture can be observed in patients with SB who used splints with SP2 variants *partial inhibition or no inhibition* – there is a statistically significant decrease ($p<0.05$) of TNC and TCT values. At the same time, there is observed a statistically significant decrease of the indices of the sympathetic autonomous regulation in the stomatognathic system (jc.SSR-H, A2), the stress level diminishes statistically respectively ($p<0.05$) (Table 41).

Table 41. **The comparative analysis of the efficacy of myorelaxing splints in patients with primary sleep bruxism in correlation with occlusive reflex indices (MIR).**

| MIR | Indices | Monitoring period | | | | β, % |
|---|---|---|---|---|---|---|
| | | Pretreatment | Treatment | | | |
| | | | 1st month | 3 months | 6 months | |
| Normal inhibition (n = 13) | TNC, un. | 49.6 ± 5.92 | 46.4 ± 5.81 | 39.7 ± 5.38 | 34.6 ± 6.35 | 32.0 |
| | TCT, s. | 85.2 ± 7.84 | 79.6 ± 7.23 | 69.9 ± 6.91 | 64.5 ± 6.94 | 25.0 |
| | jc.SSR-H, $A_2$, mV | 3.64 ± 0.21 | 3.61 ± 0.32 | 3.91 ± 0.19$^{\#}$ | 3.71 ± 0.23 | 5.0 |
| | jc.SSR-H, T, s. | 10.30 ± 0.38 | 10.10 ± 0.34 | 9.92 ± 0.37 | 9.71 ± 0.33 | 18.0 |
| | VAS-stress, pt. | 5.7 ± 0.38 | 5.5 ± 0.39 | 5.2 ± 0.51 | 5.1 ± 0.46 | 32.0 |
| Partial inhibition/no inhibition (n = 37) | TNC, un. | 47.5 ± 5.78 | 42.3 ± 5.52 | 35.6 ± 5.49 | 30.9 ± 5.83$^{*}$ | 38.0 |
| | TCT, s. | 82.3 ± 7.90 | 74.6 ± 7.46 | 63.4 ± 7.92$^{\#}$ | 60.2 ± 6.9$^{*\#\#}$ | 28.0 |
| | jc.SSR-H, $A_2$, mV | 4.05 ± 0.28 | 3.78 ± 0.31 | 3.38 ± 0.23$^{\#\#}$ | 3.32 ± 0.22$^{*\#}$ | 37.0 |
| | jc.SSR-H, T, s. | 10.11 ± 0.33 | 9.82 ± 0.31 | 9.71 ± 0.38 | 9.55 ± 0.35 | 81.0 |
| | VAS-stress, pt. | 5.4 ± 0.33 | 4.8 ± 0.39 | 4.3 ± 0.31$^{*}$ | 4.4 ± 0.37$^{*}$ | 63.0 |

***Note:*** *statistical differences: vs. period of pre-treatment ($^{*}$ – $p<0.05$); vs. the use of splints in combination with RBR method ($^{\#}$ – $p<0.05$, $^{\#\#}$ – $p<0.01$); there are indicated the arithmetic mean and standard error (M ± SE).*

Under the influence of myorelaxing splints in 6 months there occur positive tendencies in the thermo-vasomotor-metabolic activity of AT/MM muscles assessed by means of infrared thermography method. Another pattern is manifested in patients with SB who used myorelaxing splints altogether with RBR method (Table 42). An essential fact is that the therapeutic effect is much more pronounced in patients with *partial inhibition/no inhibition* of SP2 compared to patients with SP2 inhibition within the normal limits. These peculiarities are manifested at the motor, psycho-emotional and autonomous level. The results denote that the *SP2 – partial inhibition /no inhibition* is an important criterion that determines the effectiveness of application of splints.

Table 42. **The efficacy of myorelaxing splints in combination with the reflex-bruxism-regulating method in patients with primary sleep bruxism in correlation with occlusal reflex indices (MIR).**

| MIR | Indices | Monitoring period | | | | β,% |
|---|---|---|---|---|---|---|
| | | Pre-treatment | Treatment | | | |
| | | | 1st month | 3 months | 6 months | |
| Normal inhibition (n = 14) | TNC, un. | 51.2 ± 6.02 | 46.8 ± 5.86 | 35.8 ± 6.22 | 32.6 ± 7.13 | 39.0 |
| | TCT, s. | 87.8 ± 8.13 | 77.4 ± 7.64 | 58.8 ± 7.92$^{*}$ | 56.9 ± 9.56$^{*}$ | 37.0 |
| | jc.SSR-H,$A_2$, mV | 3.91 ± 0.28 | 3.51 ± 0.25 | 3.10 ± 0.29$^{*\#}$ | 2.90 ± 0.39$^{*}$ | 56.0 |
| | jc.SSR-H, T, s. | 10.61 ± 0.41 | 10.12 ± 0.34 | 9.71 ± 0.31 | 9.64 ± 0.32 | 20.0 |
| | VAS-stress, pt. | 5.5 ± 0.35 | 5.2 ± 0.38 | 4.1 ± 0.33$^{**}$ | 4.0 ± 0.41$^{**}$ | 88.0 |
| Partial inhibition/no inhibition (n = 36) | TNC, un. | 53.3 ± 6.39 | 44.8 ± 6.24 | 26.3 ± 5.63$^{**}$ | 23.4± 5.75$^{***}$ | 60.0 |
| | TCT, s. | 90.7 ± 7.96 | 68.9 ± 7.53$^{*}$ | 38.1±6.48$^{***\#}$ | 31.8±6.93$^{***\#\#}$ | 68.0 |
| | jc.SSR-H,$A_2$, mV | 3.51 ± 0.24 | 2.97 ± 0.28 | 2.43±0.2$^{***\#\#}$ | 2.38 ±0.41$^{***\#}$ | 80.0 |
| | jc.SSR-H, T, s. | 10.58 ± 0.31 | 10.16 ± 0.36 | 9.62 ± 0.30$^{*}$ | 9.41 ± 0.42$^{*}$ | 50.0 |
| | VAS-stress, pt. | 5.8 ± 0.37 | 5.2 ± 0.41 | 4.0 ± 0.39$^{***}$ | 4.1 ± 0.28$^{***}$ | 85.0 |

***Note:*** *treatment vs. pre-treatment ($^{*}$ – p<0.05; $^{**}$ – p<0.01; $^{***}$ – p<0.001); vs. the use of splints without RBR method ($^{\#}$ – p<0.05; $^{\#\#}$ – p<0.01); there are indicated arithmetic mean and standard error (M ± SE).*

The analysis of vascular-metabolic activity of AT/MM muscles under the influence of splint reflex-bruxism-regulating treatment revealed an improvement of these indices by 7.2% in patients with normal SP2 and 8.3% in patients with pathologic SP2.

The obtained results give the possibility to recommend splints especially in patients with *SP2 – partial inhibition/no inhibition*. This criterion is an objective one; it reflects the pathogenetic mechanisms of SB with differentiation of patients in accordance with occlusal reflex manifestations.

### 4.3. The effectiveness of Splint-Respiratory Treatment in patients with primary sleep bruxism in accordance with the type of daytime breathing

Splint-Respiratory Treatment (SRT) in patients with oral treatment (n=13) improved respiration in 76.9% of cases, out of which 46.2% of cases constituted the pattern of nasal respiration. In patients with SB and nasal respiration (n=30) the application of SRT did not modify the predominant type of respiration. In patients with SB and nasal respiration, the efficiency of correction (β) under the influence of SRT was 29.0% for TNC and 36.0% for TCT; in patients with SB and oral respiration syndrome (ORS) the efficiency of SRT is 40.0% for TNC and 52.0% for

TCT. SRT in patients with nasal respiration improves thermo-vasomotor-metabolic rapport of AT/MM muscles in 6.7% of cases, but in patients with ORS in 38.5% of cases. The analysis of modifications of VAS-stress indices, sleep quality and autonomous adjusting (jc.SSR-H) in the stomatognathic system in patients with nasal respiration and ORS under the influence of SRT evidenced positive tendencies of these processes. We distinguished the particularities of the manifestation of oral symptoms (there is indicated the frequency of manifestation and statistical differences respectively-patients with nasal respiration vs. oral respiration): xerostomia 10.0/53.8% ($p<0.01$), marginal gingivitis 3.3%/30.8% ($p<0.05$), glossoptosis 0/38.5% ($p<0.01$), tongue ulceration 0/30.8% ($p<0.05$).

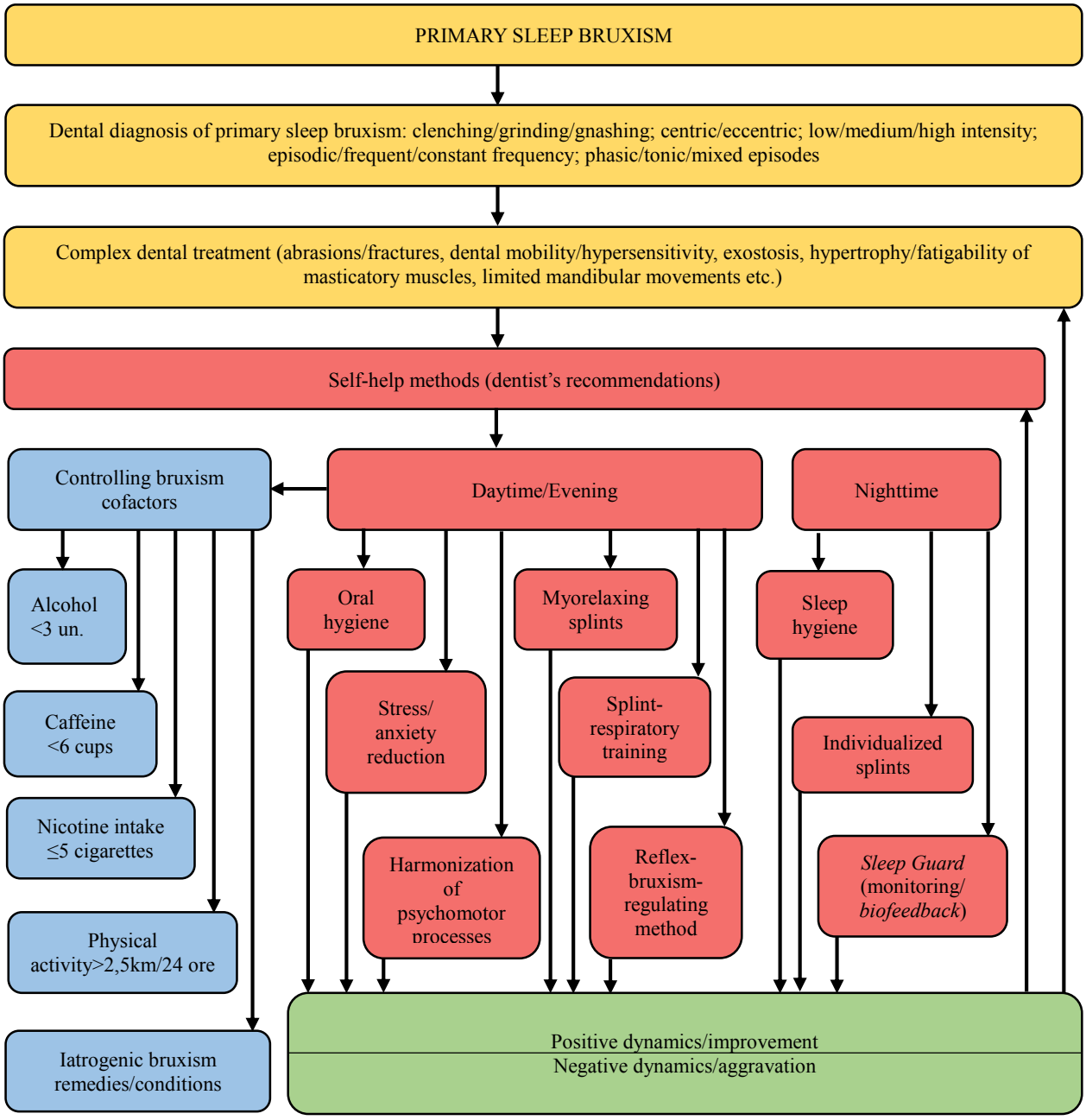


Figure 42. **Algorithm of combining the dental treatment and self-help methods in patients with primary sleep bruxism**.

As a result of the accomplished investigations, we have developed an algorithm combining the dental treatment and the self-help methods in patients with SB, which is presented in Figure 42.

## GENERAL CONCLUSIONS

1. In the process of diagnosis, treatment and monitoring of primary sleep bruxism, the particularities of predominant clinical-neurophysiological patterns are assessed (psychogenic, neurogenic-reflectory, myogenous, arthrogenic, dental-periodontal, autonomous-sympathetic, algic) manifestations of which depend on excessive/ moderate caffeine consume in 57.0%/11.0%, nicotine in 25.0%/45.0%, alcohol in 59.0%/22.0% and the presence of hypodynamic activity in 68.0% of cases.
2. The expression of primary sleep bruxism manifestations is dependent on the age of the patients: manifestations in younger individuals (18-35 years) are characterized by more severe disorders of the nocturnal episode of bruxism ($p<0,05$), pathological activity of masticatory muscles according to infrared thermography, myogenic-spastic and more pronounced reflectory-occlusal disorders ($p<0,001$); manifestations in elderly patients (35-50 years) are characterized by predominance of local and diffuse myogenic structural disturbances ($p<0,01$), more pronounced arthrogenic pain ($p<0,05$), autonomous-sympathetic dysfunction in the stomatognathic system ($p<0,05$), more severe quantitative ($p<0,05$) and qualitative ($p<0,001$) disorders of nighttime sleep.
3. In patients with primary sleep bruxism excessive consumption of psychoactive substances and hypodynamic activity have a negative influence on sleep bruxism manifestations with the increase of severity of disorders in the following order: nocturnal episode and associated clinical disorders – nicotine<hypodynamic activity<caffeine<alcohol; pathological occlusal reflex – nicotine<alcohol<hypodynamic activity<caffeine; the pathological activity of masticatory muscles according to infrared thermography – hypodynamic activity<nicotine<caffeine<alcohol; thickness and pathological ecogenity of masseter muscle – caffeine<nicotine = hypodynamic activity<alcohol; autonomous-sympathetic disorders in the stomatognathic system – hypodynamic activity<nicotine = caffeine<alcohol; night sleep disorder – nicotine<hypodynamic activity<alcohol = caffeine; degree of dental wear – hypodynamic activity = caffeine<nicotine<alcohol.
4. In patients with primary sleep bruxism, the values of the autonomous-sympathetic response at habitual and standardized jaw clenching, the indices of infrared thermography of the masticatory muscles reflect the degree of impairment of the autonomous regulation processes in the stomatognathic system and the severity of the bruxism in correlation with the patients' age and the action of the cofactors of bruxism (alcohol, nicotine, caffeine, hypodynamic activity).
5. The elaborated reflex-bruxism-regulating method diminishes the emotional stress and autonomous-sympathetic disorders in the stomatognathic system ($p<0,05$), the severity of the nocturnal episodes of bruxism ($p<0,001$), improves the sleep ($p<0,001$), it is simple in accomplishment, does not provoke side effects or complications. The combination of splints with the reflex-bruxism-regulating method greatly enhances the effectiveness of the treatment. The efficacy of nocturnal splints in patients with primary sleep bruxism depends on the variant of expression of the occlusal reflex: there is lesser efficacy in the normal inhibition variant; and there is greater efficacy in partial inhibition/no inhibition ($p<0,05$).
6. In patients with primary sleep bruxism in 13% of cases, there was present normal diurnal oral breathing syndrome that is accompanied with more frequent and more serious manifestations of nocturnal episodes, more pronounced pathological activity of masticatory muscles according to infrared thermography, sleep quality diminishing, more frequent presence of xerostomia, marginal gingivitis, glossoptosis and ulceration of the tongue. In patients with primary sleep bruxism and oral breathing syndrome it is effective to apply splint-respiratory therapy.
7. The use of myorelaxing splints, splint-respiratory treatment and the reflex-bruxism-regulating method contribute to an increased control over the manifestations of primary sleep bruxism with the transformation of the stress-pathogenic bruxism into stress-sanogenic bruxism.

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## LIST OF ABBREVIATIONS

AT – anterior temporal muscle;
EMG – electromyography;
GGM – the gradient of the masseter muscle thickness;
j.SSR-H, A2 –amplitude of the autonomous-sympathetic response to normal jaw clenching;
jc.SSR, T – period of restoration of evoked autonomous-sympathetic potential;
jc.SSR-S, A2 –amplitude of the autonomous-sympathetic response to standardized jaw clenching;
MIR – occlusal reflex;
MM – the masseter muscle;
OBS – oral breathing syndrome;
pt. – points;
RBR – reflex-bruxism-regulating method;
SB – sleep bruxism;
SRT – splint-respiratory treatment;
TCT – total clench time;
TNC – total number of clenches;
un. – units;
USG – ultrasonography;
VAS – Visual Analogue Scale.