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ORIGINAL ARTICLE

Occlusal splint for sleep bruxism: an electromyographic associated to Helkimo Index evaluation

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Abstract This study aims to evaluate long-term effects of using an occlusal splint in patients with sleep bruxism (SB), using surface electromyography (EMG) of masseter and temporalis muscles, as well as the Helkimo Index. The subjects were 15 individuals aged from 19 to 29 years, bearers of SB, with presence of signs and symptoms of temporomandibular disorders (TMD), which never have used occlusal splints. The subjects answered the Helkimo's Index and underwent EMG before and after 60 days of occlusal splints use. There was no indication of a significant decrease in mean EMG levels over the therapy in the muscles. A significant decrease in TMD signs and symptoms were observed in SB patients after 60 days of occlusal splints therapy.

Keywords Sleep bruxism · Electromyography · Occlusal splints · Masseter · Temporalis

Introduction

According to the International Classification of Sleep Disorders (ICSD), sleep bruxism (SB) is defined as a

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"disturb characterized by tooth grinding or clenching movements throughout the sleep" [1, 2]. It is related that the intensity of maximum teeth clenching during the SB is high, and it could exceed the conscious level of this movement [3]. The drawn out resistance and repetition of teeth clenching, as well as the parafunctional hyperactivity, could induce traumatic lesions in the masticatory system.

SB etiology involved local, systemic, psychological, and hereditary factors [4, 5]. Concerning about local factors, occlusal interferences were considered as a main cause factor of bruxism in the past. However, recently, the pathophysiological factors have been considered as the main cause of bruxism. More specifically, disturbances in the central dopaminergic system have been described in relation to bruxism. In recent studies, bruxism seems to be mainly regulated centrally, not peripherically [5, 6]. The relationships between inadequate occlusion and bruxism are still not very consistent [7].

The severe SB can cause signs and symptoms of temporomandibular disorders (TMD), as well as adjacent structures of the masticatory system. Excessive teeth wearing, pain in the temporomandibular joints (TMJ) and masticatory muscles, and/or headaches are common findings. SB can also cause sensibility at touch examination and masticatory muscle hypertrophy, especially on the masseter, which is a superficial and powerful muscle [8].

Sleep is the physiologic state of nonconscious or unconscious. The sleep cycle is divided in four stages called nonrapid eye movement (NREM) followed by a rapid eye movement (REM) period.

During a normal sleep cycle, the subject goes through the light stages 1 and 2 to the deeper stages 3 and 4 in NREM phase and than into the REM one. The SB can be founded in all sleep levels, but it occurs more frequently in stages 1 and 2 [9].

The sleep polissonographic studies indicated that SB occurs generally during the fragmental periods of sleep, with the increase in electroencephalographic and electromyographic (EMG) activities, as well as the increase in cardiorespiratory frequency [9–12].

Medications for symptoms control, muscle exercises, physiotherapy, occlusal splints, and occlusal rehabilitation are some of the described treatments for bruxism [8].

Occlusal splints are frequently used in SB, to protect teeth from damages resulting from the contraction force of mandibular muscles or to reduce the orofacial pain by relaxing masticatory muscles [13, 14]. These devices are also called "splints for muscle relaxing" or "stabilizing splints" [15]. However, the efficacy of the occlusal splints to reduce masticatory muscle activity remains questionable [14, 16], and there are not enough scientific evidence regarding the effects of splints on SB to date, mainly because of the difficulty in recording SB activity in multiple nights and for a longer observation period [17].

Considering the aforementioned data, this study aims to evaluate the long-term effects of an occlusal splint, through the surface EMG values of the masseter and temporal muscles and the Helkimo Index, before and after 60 days of splint use in SB bearers.

Materials and methods

Subjects

The sample was composed of 15 subjects (14 female and 1 male) ranging from 19 to 29 years old (average of 22.13+ 2.72 years). All subjects were undergraduate students in the city of São José dos Campos, São Paulo, Brazil.

The inclusion criteria adopted demanded that patients were all undergraduate students, bearers of SB, with or without presence of signs and symptoms of temporomandibular disorder and that they have never been treated with occlusal splints.

Clinical diagnosis of SB was made under world standards of diagnosis, based upon patient history and orofacial examination [22]. Teeth's wearing was evaluated with a dental mirror and adequate light. Upper and lower casts were made to analyze teeth wear degree [18–20]. Diagnose of muscular hypertrophy was also taken considering patient age and dental facial morphology [21].

This study was previously approved by the ethical committee from UNIVAP—University of Vale do Paraíba.

Method

The selected subjects were submitted to an EMG analysis of masseter and temporal muscles in rest and isometric positions. Patients then received a rigid flat and plane occlusal splint, constructed with acrylic resin, which they used during the sleep time and were clinically evaluated each 2 weeks during 60 days of treatment. Occlusal splints

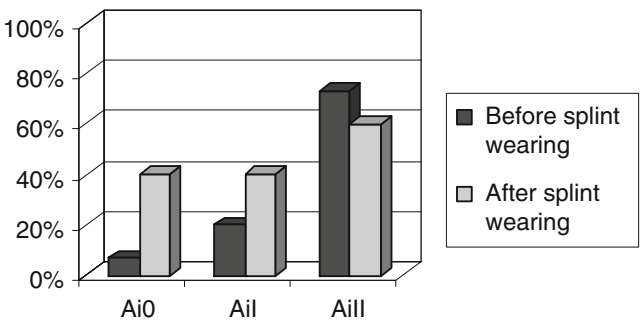

Fig. 1 The Helkimo Anamnestic Dysfunction Index in patients with TMD before and after occlusal splint therapy. *Ai0* Absence of anamnestic signs and symptoms of dysfunction, *AiI* anamnestic signs and symptoms of light dysfunction, *AiII* anamnestic signs and symptoms of severe dysfunction

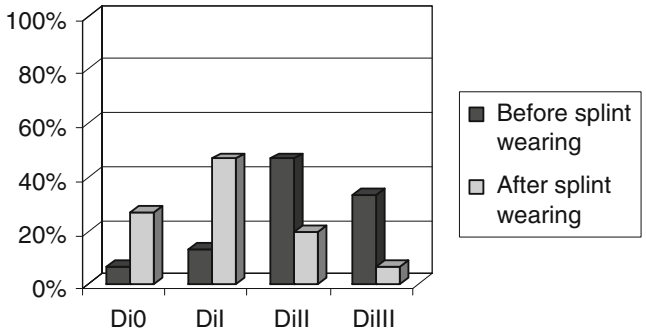

Fig. 2 The Helkimo clinic dysfunction index in patients with TMD before and after occlusal splint therapy. *Di0* Absence of clinical symptoms of dysfunction, *DiI* light clinical symptoms of dysfunction, *DiII* moderate clinical symptoms of dysfunction, *DiIII* severe clinical symptoms of dysfunction

were checked, and in case of instability, they were adjusted to achieve again functional stability. Simultaneous occlusal contacts of all mandibular supporting cusps were verified by a 12-μm-thick articulation paper. After this period, subjects were re-evaluated by the Helkimo Index [22] and masseter and temporal muscles EMG.

The patients gave the necessary information to obtain the index for anamnestic dysfunction of masticatory system, validated by Helkimo. According to the answer of each individual, the sign and symptoms of dysfunction are scored as A_i0—absence of sign and symptoms of dysfunction, A_iI—light sign and symptoms of dysfunction such as TMJ noises, masticatory muscles fatigue, and sensation of TMJ rigidness, A_i2—intense sign and symptoms of dysfunction such as limited jaw opening and clocking episodes, TMJ open lock, pain during mandible movement, and pain during TMJ palpation or in masticatory muscles. The index for clinic dysfunction of masticatory system, also validated by Helkimo, was obtained in clinic examination, and it demonstrates the degree of TMJ dysfunction based in the presence of 05 common symptoms, such as muscular pain, TMJ pain, pain during mandible movements, limited TMJ function, and mobility index. This method was described in detail previously [22].

The quantitative interpretations of EMG signs considered three fundamental characteristics: duration, amplitude, and frequency [23]. Duration corresponds to the activation period of the muscle; the amplitude expresses the level of activity sign, which gives data about the intensity of muscle activity. The root mean square (RMS) value allows evaluating the sign amplitude [24]. Finally, the frequency means the tax of muscle cell excitation.

The EMG study used a system of EMG signs registration (EMG System do Brasil, Brazil) of eight channels of analogical input with amplification of a thousand times, filter with frequency range of 20 and 500 Hz, and digital analogical converter of 12 bits of resolution. The frequency sampling was of 2,000 Hz by channel. The EMG registration was realized using bipolar surface electrodes of 10 mm of diameter, after realizing muscle function proof of masseter and temporal [25]. The cleansing of skin was done with ethylic alcohol 70% to reduce the impedance.

The ground wire was connected to the right wrist of the subject with electroconductor gel Lectron II (Pharmaceutical Innovations, USA) to increase the capacity of electroconduction and avoid external noise interferences [23].

The EMG evaluation occurred in a mandibular rest position for 10 s and after a maximum contraction (isometric) also for 10 s. An interval of 3 min between data collection was established to avoid muscle fatigue. After 60 days, the same protocol was applied.

The signals treatment was formed by complete wave rectification, linear cover by Butterworth of the fourth level, with 5-Hz frequency of cut, normalized in time base and amplitude, where the amplitude was normalized by mean [26]. The intensity variability of the EMG signal was calculated by the coefficient of variability.

Statistical analysis

The comparison between the EMG signs in different muscle situations was realized by the Kolmogorov–Smirnov test for verification of normal distribution of values found. A parametric test named *t*-paired test was used to verify the

Table 1 Descriptive data of electromyographic variations (rest/ isometry) from RMS values, in left and right sides of masseter and temporal muscles

Muscles	Number	Minimum	Maximum	Average	SD
Left temporalis before splint	13	0.02066	0.31408	0.066459	0.022994
Left temporalis after splint	13	0.01767	0.26929	0.057202	0.016874
Left masseter before splint	13	0.00966	0.09039	0.042797	0.006796
Left masseter after splint	13	0.00712	0.13266	0.054376	0.01434
Right temporalis before splint	13	0.01030	0.59303	0.142209	0.058218
Right temporalis after splint	13	0.00974	0.91433	0.181771	0.09349
Right masseter before splint	13	0.01412	0.14172	0.058602	0.011091
Right masseter after splint	13	0.00392	0.16378	0.045683	0.015369

Table 2 Values from the Kolmogorov–Smirnov test

	Number	Left temporalis	Left masseter	Right temporalis	Right masseter
Normal parameters	15	0.0027090± 0.11274273	0.0157403± 0.04198632	0.0813689± 0.39941567	−0.0074484± 0.04416618
Extreme variations	15				
Absolute		0.218	0.143	0.209	0.126
Positive		0.178	0.135	0.209	0.126
Negative		−0.218	−0.143	−0.183	−0.122
Kolmogorov–Smirnov *t*	15	0.844	0.555	0.808	0.490
Asymp. Sig. (two-tailed)	15	0.474	0.918	0.531	0.970

data about RMS (rest/isometry) for paired samples. The significance level adopted was 0.05. The score analysis of the Anamnestic and Clinical Helkimo Index was made using the χ^2 test.

Results

Considering the Helkimo's Anamnestic Dysfunction Index, before and after occlusal splint wear, it was observed that the perceptual indices A_i0 (absence of signs and symptoms of dysfunction) and A_iI (signs and symptoms of light dysfunction) increased from 6.7 and 20, respectively, to 40% for both indices. It was also noticed that a decreasing in the perceptual index of A_iII (signs and symptoms of severe dysfunction) occurred, as it could be observed on Fig. 1.

Considering the Helkimo's Clinical Dysfunction Index, it was verified that after occlusal splint wearing the indices of D_i0 (no symptoms of dysfunction) and D_iI (light dysfunction) increased from 6.7 and 13.3 to 26.7 and 46.7%, respectively. Moreover, a relative decreasing in DiII (moderate dysfunction) values from 46.7 to 20% and DiIII (severe dysfunction) from 33.3 to 6.7% was observed, which shows a reduction in subjects with moderate and severe dysfunctions (Fig. 2).

The descriptive data of EMG variations (rest/isometry) from RMS values in the masseter and temporal muscles, left and right sides, are shown on Table 1, and values from Kolmogorov–Smirnov tests are available on Table 2, which demonstrate normal distribution of EMG variables.

According to the values of the *t*-paired test (Table 3), with a confident interval of 95%, there was no statistically significant difference on the muscles studied before and after wearing occlusal splints for 60 days.

The difference between the averages of EMG values of both right and left masseter muscles (rest/isometry) was not significant.

A significant difference on the averages of RMS (rest/isometry) before and after the occlusal splint wearing both on right and left temporal muscles was not found.

Discussion

The results of our work revealed that after occlusal splint wearing for 60 days, the patients presented a decrease in signals and symptoms related to TMD according to the Helkimo Index, mainly in patients with severe symptoms of SB. However, EMG registers of masseter and temporal muscles have not shown significant differences.

Landulpho et al. [27] studied the effect of occlusal splint therapy on TMD by surface EMG of masseter and temporal muscles. The authors followed 22 patients with signs and symptoms of TMD during 90, 120, and 150 days of splints wearing. The polynomial regression analysis of the electric potential of RMS of temporal and masseter muscles, during

Table 3 Results for the *t*-paired test before and after the treatment with occlusal splint

There were no statistically significant results in EMG after occlusal splint therapy.

Muscles	*t* test results	*df*	Sig. (two-tailed)	Average variation	95% confidence interval of the variation	
					Lower	Higher
Left temporalis	−0.331	12	0.746	−0.0100078	−0.0758765	0.0558609
Left masseter	1.490	12	0.162	0.0181738	−0.0083957	0.0447433
Right temporalis	0.804	12	0.437	0.0899046	−0.1536021	0.3334112
Right masseter	−0.656	12	0.524	−0.0084353	−0.0364646	0.0195941

maximum biting (isometric), showed significant differences ($p<0.05$) between evaluation periods (90/120/150 days), reducing during the treatment.

The authors showed that the longer the therapy with splints, the more reduced was the EMG activity of masticatory muscles. Their study justifies our findings on EMG activity registers, as we used a 60-day period of occlusal splint wearing, which was a shorter period compared with the periods [27].

Holmgren et al. [28] observed, in a sample of 31 SB bearers using occlusal splints for 6 months, a great reduction in symptoms related to TMD, like pain on temporal and cervical regions, headaches, TMJ pain, and noises. Despite considerable relief, the SB behavior persisted. In the present study, when comparing data from the Anamnestic Index of Helkimo before and after occlusal splints wear, it was also observed that symptoms reduced from A_iII index to A_iI and A_i0.

Holmgren et al. [29] verified that the therapeutic mechanisms of occlusal splints are not yet completely elucidated, but it was suggested that it eliminates occlusal interferences and interrupt the feedback mechanism that supplies the SB.

In a recent study, Dubé et al. [14] compared the efficacy of the occlusal splint and a palatal device used as control in nine volunteer bearers of SB by nocturnal polissonography. Patients wore palatal devices for 2 weeks and were evaluated at the beginning and the end of treatment. A statistically significant difference in the reduction in bruxism episodes per hour (decrease of 41%, $p<0.05$) by using both mechanisms was observed. A great decrease in clenching episodes (50%, $p=0.06$) was also verified. These findings show that splint wear reduce the frequency of SB events and the activities related to nocturnal clenching, which prevent damages caused by SB behavior, as does a control device.

Harada et al. [17] also compared the efficacy of occlusal splint and a palatal device in 16 patients during 6 weeks with each device and used an interval of 2 month to a washout period. The authors found no statistically significant difference between the two types of device, but the findings showed that there was statistically significant reduction in the masseter EMG activity immediately after the insertion of splints. However, they were no significant changes in 2, 4, and 6 weeks after the insertion of either splint.

Baad-Hansen et al. [30] found a significant reduction in EMG activity in the masseter during sleep when patients used nociceptive trigeminal inhibitory (NTI) splint and, curiously, found no significant reduction in EMG activity in the masseter during sleep when patients used a standard occlusal splint, which converge to our findings using the same type of device. Conversely, the reduction in masseter EMG using NTI was not associated with a reduction in signs or symptoms of the patients.

Our data coincide with the literature, where it is possible to find an important reduction in signs and symptoms of SB, analyzed after treatment with occlusal splints for 60 days, evaluated by the Helkimo Index. However, the surface EMG analysis of masseter and temporal muscles did not show favorable results considering the same period, as no significant difference was found on electric potentials generated by these muscles. A recent study showed that cognitive–behavioral treatment itself may also induce improvement of SB symptoms [31]. This study did not have a sleep study, and we do not know the changes of SB during the treatment. More extensive investigations should be done to achieve a better understanding on occlusal splints mechanism on reducing TMD signs and symptoms and to determine new approaches of SB therapy.

Conclusion

Occlusal splint used for 60 days by SB bearers presented a significant reduction in the clinical signs and symptoms of TMD and seems to benefit patients with more severe symptoms of SB. However, the EMG evaluation of the surfaces of masseter and temporal muscles in bearers of SB under therapy with occlusal splint for 60 days did not present a significant difference before and after treatment.

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