Original Article   **Open Access**

Effect of Different Treatment Modalities on Masseter Inhibitory Reflex in Patients with Sleep Bruxism: A Case-Control Study

Nabaa Saeed Mousa [1] , Farqad Bader Hamdan [2*] , Balsam Saadi Abdulhameed [3]

[1] Neurophysiology Unit, Al-Yarmouk Teaching Hospital, Baghdad, Iraq
[2] Department of Physiology, College of Medicine, Al-Nahrain University, Baghdad, Iraq
[3] Department of Oral and Maxillofacial Surgery, Al-Imamain Al-Kadhemain Medical City, Baghdad, Iraq

* **Corresponding Author:** Farqad B. Hamdan
Address: Department of Physiology, College of Medicine, Al-Nahrain University, Baghdad, Iraq
E-mail: farqadbhamdan@colmed-alnahrain.edu.iq - farqadbhamdan@yahoo.com

Abstract

Background: Sleep bruxism (SB) is a parafunctional oral habit that is frequently related to sleep arousals. The masseter inhibitory reflex (MIR) causes reflex inhibition of voluntary contractions of the elevator muscles induced by intense peri- or intraoral mechanical or electrical stimulations. This study aimed to investigate the changes in MIR of patients with SB, and the effect of different treatment modalities on MIR.

Methods: In this case-control study, 100 individuals were randomly assigned to four groups (n=25) of conservative treatment (G1), occlusal splint treatment (G2), low-level laser therapy (LLLT) treatment (G3); and control (G4). The MIR was tested in all participants before and one month after treatment.

Results: All MIR components of the patients had longer right and left SP1 and right and left SP2 latencies (P=0.017, P=0.043, P<0.001, and P=0.04, respectively), and shorter right and left SP1 and right and left SP2 durations (P=0.021, P=0.021, P<0.001, and P<0.001, respectively) as compared to the control group. The right SP1 and SP2 latencies were prolonged in G3 versus G1 and G2 (P=0.026 and P<0.001, respectively); whereas, the left SP2 latency was prolonged in G2 compared with G1. The right and left SP2 duration was not significantly different among the three treated groups. The left SP1 duration was not significantly different among the three treated groups. Gender had no effect on MIR parameters.

Conclusion: SB patients had an abnormal MIR response. LLLT was the most effective compared to other treatment modalities.

Keywords: Bruxism, Masseter Muscle, Low-Level Laser Therapy, Sleep

Background

Sleep bruxism (SB) is an oromandibular disorder defined as a stereotyped movement disorder that occurs during sleep and is characterized by tooth grinding and/or clenching (1). SB shows no gender difference, in contrast to awake bruxism, and is more common in females (2,3). This condition is becoming more common in the younger population (4).

Bruxism is often diagnosed clinically (5) based on patient's history (e.g., complaints of grinding noises) and presence of typical signs and symptoms. Early detection of bruxism is advantageous due to its potential complications and its negative impact on the quality of life (3).

The masseter inhibitory reflex (MIR) has been studied in the recent years due to increased awareness about bruxism among doctors and a growing need for quick and effective diagnostic tools for bruxism to monitor jaw muscle activation. MIR is a brainstem response that causes reflex inhibition of voluntary contractions of the jaw-closing muscles evoked by intense peri- or intraoral mechanical or electrical stimulation (6).

MIR is mostly seen as a protective reflex, although it also aids in synchronization of mandibular movements during mastication and articulation (7). MIR is the most commonly used neurophysiological test to study the function of the third trigeminal division and the mandibular nerves (8). It was discovered that the use of MIR could be beneficial for evaluation of SB (6).

Currently, no definite treatment exists for SB. The available treatment approaches reflect varying degrees of efficacy in dealing with the potentially detrimental effects of SB (9). Behavioral treatments for managing SB include avoiding the risk factors and triggers, patient education, relaxation techniques, sleep hygiene, hypnotherapy, biofeedback, and cognitive-behavioral therapy (10,11).

Several medications have been linked to both a decrease and an increase in SB activity, lending credence to the possibility of central mechanisms involved in the origin of SB (12). The orofacial motor activity is thought to be mediated by the dopaminergic, serotoninergic, and adrenergic systems. However, there is still lack of evidence for both the efficacy and safety of drugs for SB patients, and pharmacological treatments should be considered only in seriously afflicted symptomatic patients as short-term therapy (12).

Physical therapy is also used to treat this condition, with the most common methods being transcutaneous neuromuscular stimulation, microcurrent electrical neuromuscular stimulation, cryotherapy, ultrasound, infrared therapy, kinesiotherapy, massage therapy, acupuncture, low-level laser therapy (LLLT), and the use of an occlusal splint (13-15).

Occlusal appliances have been widely employed in clinical practice; however, their effect seems to be transitory and highly variable among patients. Irrespective of the appliance design, the majority of trials demonstrated a decrease in SB activity in the first 2 weeks of treatment (16,17). Furthermore, around 20% of patients had an increase in EMG activity during sleep when using an occlusal appliance, particularly the soft mouth guard kind (18).

LLLT, on the other hand, is a non-invasive infrared light treatment that is used directly at the lesion site. Its principal effect is based on the light absorption process. This soft laser has a wavelength range of 630 to 1300 nm (19). Despite its unknown exact mechanism of action, LLLT activates tissues and has analgesic and anti-inflammatory effects in direct irradiation (19-21).

This study aimed to investigate the changes in MIR of patients with SB, the effect of different treatment modalities on MIR, and the link between MIR and other confounding factors such as the patients' age and gender, and duration of bruxism.

Methods

Study design:

This case-control study was conducted at the neurophysiology department of Al-Imamain Al-Kadhemain Medical City in Baghdad from October 2020 to November 2021. This study was approved by the Iraqi Board of Medical Specialization (Decision No. 291; Date 21/1/2021). All participants were informed about the technique and purpose of the study, and all the patients and controls signed informed consent forms for participation in the study.

Participants:

Seventy-five patients with primary SB of either sex were randomly assigned to three groups of 25 individuals each: conservative treatment group (G1), occlusal splint treatment group (G2), and LLLT treatment group (G3). Another 25 age- and sex-matched healthy volunteers served as the control group. Randomization was conducted utilizing Microsoft Excel (version 2013), with a total of 75 patients assigned randomly to the three groups. This randomization was executed in block form, with groups comprising of 25 patients each. A set of 75 opaque envelopes, labeled with sequential numbers ranging from 1 to 75, were prepared. Each envelope contained information about the designated group, following a predetermined random order. These envelopes remained securely sealed until the commencement of treatment. A maxillofacial surgeon referred all of the patients. Their age ranged from 5 to 60 years, and all had a normal mentality. The study excluded participants with secondary bruxism related to mental health conditions (e.g., anxiety and depression, confirmed by a psychiatrist), neurological disorders (such as Huntington's disease and Parkinson's disease), sleep apnea, and individuals who were using certain antidepressants, antipsychotics, and selective serotonin reuptake inhibitors.

It should be noted that seven patients were dropped out during the third visit: three from G1, two from G2, and two from G3.

Clinical examination:

The participants were examined for symptoms such as tooth grinding accompanied by a distinctive sound that may even awaken the bruxer's bed partner, TMJ pain, masticatory and cervical muscle pain, headache (especially in the temporal zone when the patient wakes up in the morning), hypersensitive teeth, excessive tooth mobility, poor sleep quality, and tiredness. The clinical diagnosis was done using the ICSD third edition (sleep-related bruxism).

Out of the total number, 12% of those with SB had diabetes mellitus distributed in the three treatment groups, 6.7% had hypertension in G1 and G2, and only one (1%) patient in the G1 group had thalassemia.

Treatment modalities:

Group 1 consisted of 25 participants who were instructed to change their lifestyle by changing their sleep hygiene through a set of instructions aimed at correcting personal habits and environmental factors that disrupt sleep quality. These included abstaining from coffee, tea, stimulants, chocolate, and caffeine-containing medications, avoiding smoking and drinking alcohol (if any) at least 6 hours before going to bed, eating light meals before going to bed, and limiting physical or mental activity before bedtime and maintaining healthy sleeping circumstances (quiet and dark room).

Second, during the trial period, the patients were given the drug phenyltoxamine Mag salicylate "Myogesic" tablet, which is a mixture of 35 mg orphenadrine citrate and 450 mg acetaminophen three times a day (22).

Group 2 included 25 patients with SB who were instructed to wear an occlusal splint during the study period (6 weeks) which is a custom-fabricated hard acrylic appliance that fits over the occlusal and incisal surfaces of the maxillary or mandibular teeth (17). Throughout the trial period, the patients were instructed to use it at night every day. The patients were informed about its significance, as the device is only useful when the patient wears it.

Group 3 included 25 patients who underwent LLLT with Quicklase 12w Dual Plus 6" 810+980 nm diode laser (GaAlAs Laser Diode Class IV; Quickwhite Ultimate Lasers, UK). This machine produces a semi-conductive laser with a wavelength: of 810 and 980 ± 10 nm, an output power of 0.3 W, and energy density of 4 J/cm2 (19-21).

LLLT was performed in non-contact mode using a special probe for bio-stimulation with 1.5 cm distance from the skin. A handheld single laser probe was used to deliver the laser beam. It was applied perpendicular to the skin behind, in front of, and above the joint area, as well as into the external acoustic meatus. Each tender spot received 120 seconds of LLLT. LLLT was administered over 2 weeks, through 3 sessions per week. The MIR results were evaluated 2 weeks after the termination of treatment (4 weeks post-treatment initiation), and subsequently, another assessment was conducted 2 weeks later (6 weeks post-treatment initiation) to monitor the response.

MIR:

The MIR was examined using Keypoint EMG equipment from Medtronic (Denmark). After instructing the patients to clench at maximum strength with auditory feedback, two surface recording electrodes (the active electrode over the lower third of the muscle belly and the reference electrode about 2 cm below the angle of the mandible) were placed unilaterally on the masseter muscle to record the EMG signals. The ground electrode was placed on the patient's forehead (23).

With the patient clenched, electric shocks were delivered by placing a stimulator over the studied side's mental nerve. The current merely required to elicit a perceptible sensation under the cathode determines the stimulus strength. The detection threshold ranged between 6 and 12 mA.

This process was repeated until five identical responses at a fixed stimulus strength of transiently inhibited electromyographic activity were registered. This transient post-excitatory inhibition is called the silent period (SP). The average of five replies was used for statistical purposes, and the latency (measured in milliseconds) which is the time measure from the beginning of post-excitatory inhibition and duration (measured in milliseconds) which is the time during which the muscle electrical activity is still inhibited by MIR were investigated. The procedure described above was repeated on the contralateral master muscle.

The filter setting was turned to 50Hz-20 kHz, with a sweep speed of 20 ms/division, a sensitivity of 0.5 V/division, and an impulse duration of 0.5 ms. Throughout the trial, the ambient temperature was 25˚C and the skin temperature was kept between 32-34˚C. The MIR was assessed in each of the three study groups before treatment (baseline data), 2 weeks later, and 1 month later.

Statistical analysis:

According to the findings presented by Huynh et al, (24) with a targeted power of 90%, and maintaining a significance level of α=0.05, the minimum required sample size for all groups was calculated to be 100 patients, with 25 individuals allocated to each group.

SPSS version 25.0 (SPSS Inc., Chicago, IL, USA) was used for all statistical analyses. The data were subjected to the Shapiro-Wilk normality test. Continuous data were presented as mean ± standard deviation and analyzed using either the student t-test (between two groups) or ANOVA (among more than two groups) followed by the least significant difference as the post hoc for pairwise comparisons. Numbers and percentages were used to express the categorical variables. The Pearson's correlation test was performed to investigate the potential relationship between the neurophysiological parameters and age and bruxism duration. A statistically significant difference was defined as a P value less than 0.05.

Results

Demographic information of the study population:

The Shapiro-Wilk normality test revealed that the data had a normal distribution. Age, gender, and presence of comorbidities were not different among the three treatment groups and the control group (P>0.05). The right side was more affected in the G3 group; whereas, the left side was more affected in the G1 group (P=0.025); whereas, no difference was found between G1 and G2. In addition, the G3 group had a longer duration of bruxism in comparison to G1 and G2 groups; additionally, G2 was longer than G1 (P=0.009), as indicated in Table 1.

MIR:

Figure 1 shows the MIR of a control group and a SB patient after 2 weeks and 1 month of LLLT.

Table 2 compares the MIR components between patients with SB and controls. The left and right SP1 and right and left SP2 latencies of the bruxers were significantly longer compared to the control values (P=0.017, P<0.001, P=0.04, and P=0.043, respectively). The duration of the right SP1 and SP2, and left SP1 and SP2 was significantly shorter in bruxers than controls (P=0.021, P<0.001, P=0,.021, and P<0.001, respectively). In 17 patients, the SP2 was not recorded on either side (5 in the G3, 8 in the G2, and 4 in the G1 group).

Table 1. Demographic information and baseline clinical data of the study population.

Variable	Treatment group			Control (n=25)	P-value
	G1 (n=25)	G2 (n=25)	G3 (n=25)		
Mean age (years)	34.96±13.36ab	38.37±12.7a	31.0±8.98b	37.56±11.1a	0.126
Gender [N(%)]					
Male	8(32%)	8(33.33%)	9(37.5%)	12(58%)	0.644
Female	17(68%)	16(66.67%)	15(62.5%)	13(52%)	
Comorbidities [N(%)]					
None	19(76%)	19(79.17%)	20(83.33%)	22(88%)	
Diabetes mellitus	3(12%)	2(8.33%)	4(16.67%)	3(12%)	0.407
Hypertension	2(8%)	3(12.5%)	0(0%)	0(0%)	
Thalassemia	1(4%)	0(0%)	0(0%)	0(0%)	
Affected side [N(%)]					
Right	5(20%)	4(16.67%)	10(41.67%)		
Left	10(40%)	6(25%)	1(4.17%)	-	0.025
Bilateral	10(40%)	14(58.33%)	13(54.17%)		
Headache [N(%)]					
Yes	8(32%)	6(25%)	11(45.83%)		0.546
No	17(68%)	18(75%)	13(54.17%)	-	
Mean duration (months)	14.36±10.87a	21.83±14.78c	27.38±17.13b	-	0.009

G1 = conservative treatment group; G2 = splinting group; G3 = LLLT group;
A significant difference between groups is indicated by a different superscripted letter (P<0.05).
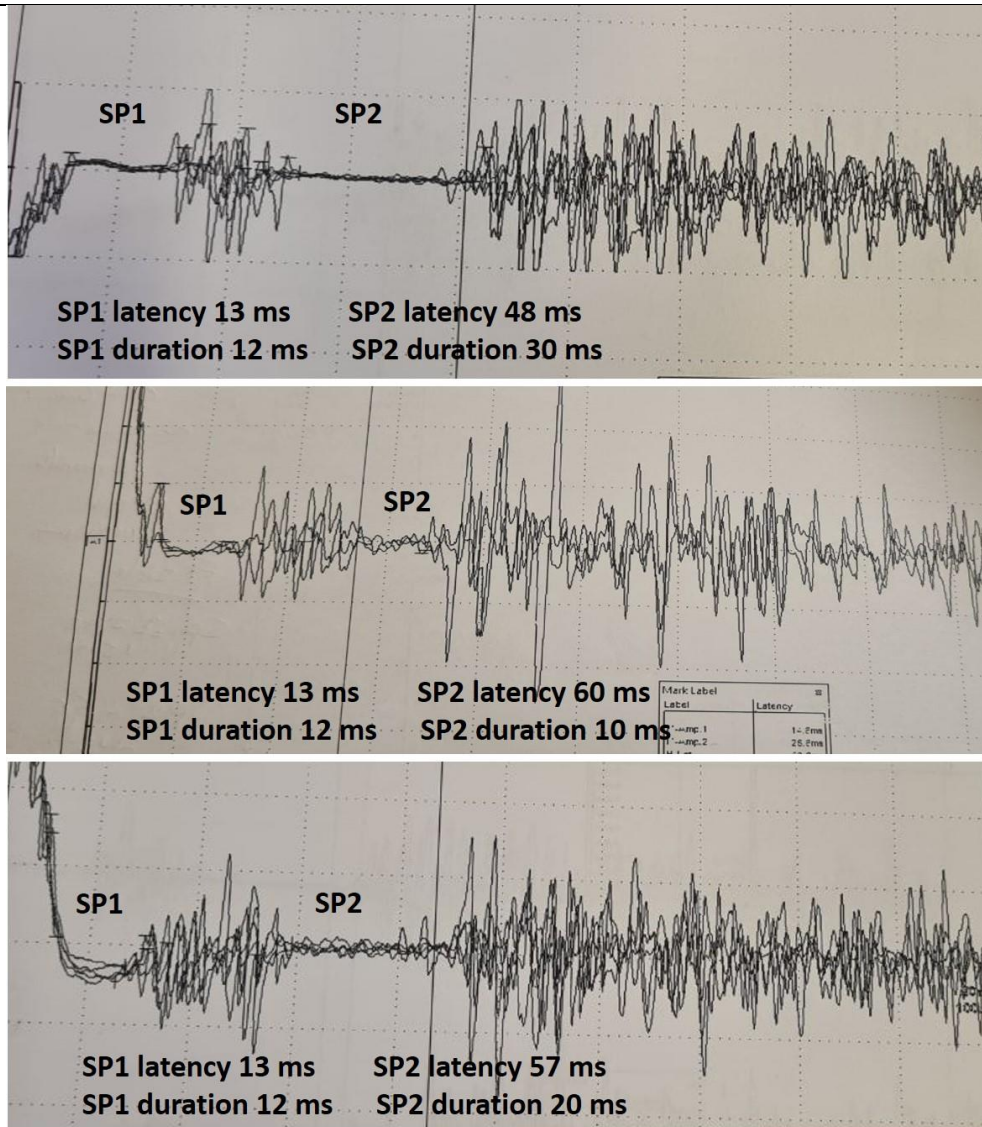A 95% confidence interval of the mean was estimated in the analysis.

Fig. 1. Masseter inhibitory reflex recorded from a control subject (upper), SB patient after 2 weeks of treatment (middle), and 1 month after LLLT (lower).

MIR Component	Patients (n=75) Mean± SD	Controls (n=25) Mean± SD	P-value
Table 2. Comparison of the MIR components between patients with SB and controls.			
Right SP1			
Latency (ms)	12.16±1.31	11.51±1.11	0.017*
Duration (ms)	12.42±1.27	13.08±1.0	0.021*
Right SP2			
Latency (ms)	58.22±8.27	52.00±4.13	<0.001*
Duration (ms)	19.60±8.14	30.6±3.79	<0.001*
Left SP1			
Latency (ms)	11.92±1.04	11.42±1.03	0.04*
Duration (ms)	12.27±1.10	12.88±1.17	0.021*
Left SP2			
Latency (ms)	55.87±8.12	52.36±4.18	0.043*
Duration (ms)	21.26±8.26	29.72±3.66	<0.001*

SP = silent period; ms = millisecond; SD: standard deviation

A significant difference between groups is indicated by a * sign ($P<0.05$).

A 95% confidence interval of the mean was estimated in the analysis.

Regarding the MIR data before treatment, the right SP1 latency was not different between G1 and G2 while the right SP1 latency values in G1 and G2 were different from those in G3 and control group (P=0.026). The right SP1 duration was not different among the three treatment groups and the control group (P=0.136). Moreover, the right SP2 latency was different between G1 and G3, and between G1 and G2 but not between G2 and G3 and the latter two and the control group (P<0.001). Regarding the right SP2 duration, no difference was demonstrated among the three treatment groups but the difference with the control group was significant (P<0.001).

Concerning the left SP1 latency and duration, no difference was found among the three treatment groups or between the treatment and control groups (P=0.083 and P=0.0116, respectively). Considering the left SP2 latency, G1 was different from G2 and both G2 and G3 from the control group (P=0.007). SP2 duration was not different among the treatment groups but the treatment groups were different from the control group (P<0.001), as indicated in Table 3.

Table 4 shows that the right-sided SP2 latency was significantly reduced (P=0.001) and SP2 duration was significantly prolonged (P=0.003) over time (onset-6 weeks) in G3 patients as compared to G1 and G2 groups that showed no change in the MIR parameter between baseline, 2 weeks and 1 month.

Before starting the treatments, the right-sided SP2 duration was inversely linked to age (r=-0.247; P=0.048) and positively linked to bruxism duration (r=0.330; P=0.007). Likewise, the left-sided SP2 latency, had a positive relationship with bruxism duration (r=0.320; P=0.011), as shown in Figure 2.

Table 3. Baseline data of the MIR in the treatment groups versus the control group.

Components	Treatment groups			Controls (n=25) Mean± SD	P-value
	G1 (n=25) Mean± SD	G2 (n=25) Mean± SD	G3 (n=25) Mean± SD		
Right SP1					
Latency (ms)	11.2±0.82a	11.5±1.22a	11.83±1.2ab	12.16±1.31b	0.026
Duration (ms)	12.52±1.23abc	12.33±1.0b	12.42±1.56abc	13.1±1.0c	0.136
Right SP2					
Latency (ms)	53.17±8.75a	58.95±7.65c	62.82±4.97bc	51.96±4.13a	<0.001
Duration (ms)	21.22±8.33a	19.2±8.73a	18.27±7.44a	30.56±3.79b	<0.001
Left SP1					
Latency (ms)	11.16±0.75a	11.58±1.1ad	11.54±1.18ac	11.92±1.04bcd	0.083
Duration (ms)	12.36±0.81abc	12.33±0.92abc	12.12±1.48b	12.88±1.17c	0.116
Left SP2					
Latency (ms)	52.64±7.34ac	58.8±7.6b	56.55±8.53ab	52.36±4.18c	0.007
Duration (ms)	21.86±8.65a	18.75±8.8a	22.1±7.42a	29.72±3.66b	<0.001

G1 = conservative treatment group; G2 = splinting group; G3 = LLLT group; SP = silent period; ms = millisecond; SD: standard deviation
A significant difference between groups is indicated by a different superscripted letter (P<0.05).
A 95% confidence interval of the mean was estimated in the analysis.

Table 4. MIR at the treatment onset and two weeks and one month after different treatment modalities

Treatment modality		Right SP1		Right SP2		Left SP1		Left SP2	
		Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)
G1	Onset	11.2±0.82	12.52±1.23	53.17±8.75	21.22±8.33	11.16±0.75	12.36±0.81	52.64±7.34	21.86±8.65
	2 weeks	11.16±0.75	12.44±1.1	52.16±7.38	21.48±7.67	11.12±0.78	12.48±0.71	52.0±6.66	22.17±6.96
	1 month	11.18±0.73	12.45±0.96	51.18±5.82	24.9±4.76	11.14±0.71	12.45±0.51	52.34±5.78	23.14±5.96
p-value		0.983	0.962	0.669	0.164	0.982	0.816	0.946	0.835
G2	Onset	11.5±1.22	12.33±1.0	58.95±7.65	19.2±8.73	11.58±1.1	12.33±0.92	58.8±7.6	18.75±8.88
	2 weeks	11.54±1.1	12.46±1.1	56.83±7.75	18.67±9.6	11.58±1.1	12.45±0.88	57.14±8.05	20.5±8.91
	1 month	11.59±1.0	12.4±0.96	55.73±6.54	21.5±1.37	11.55±1.37	12.64±1.05	6.32±7.62	21.41±8.53
p-value		0.963	0.914	0.361	0.529	0.992	0.558	0.578	0.828
G3	Onset	11.83±1.2	12.42±1.56	62.82±9.0a	18.87±7.4a	11.54±1.18	12.12±1.48	56.55±8.53	22.1±7.42
	2 weeks	11.79±0.98	12.5±1.35	57.17±4.8b	19.25±6.7a	11.25±1.15	12.37±1.56	55.96±7.58	21.04±7.63
	1 month	11.32±0.89	12.4±1.26	55.1±3.88b	24.6±4.57b	11.18±1.0	12.64±1.65	55.09±5.48	24.95±5.0
P-value		0.186	0.970	<0.001	0.003	0.508	0.543	0.817	0.147

G1 = conservative treatment group; G2 = splinting group; G3 = LLLT group; SP = silent period; ms = millisecond
A significant difference between groups is indicated by a different superscripted letter (P<0.05).
A 95% confidence interval of the mean was estimated in the analysis.

As indicated in Table 5, the SP2 latency and duration on the right side were strongly linked to the affected side (P<0.001), and the left-sided SP2 latency and duration were both significantly associated with the affected side (P=0.002 and P=0.001, respectively).

Variables	Right SP1		Right SP2		Left SP1		Left SP2	
	Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)	Latency (ms)	Duration (ms)
Gender								
Male	11.4±1.04	12.6±1.38	59.04±6.85	19.17±8.0	11.64±1.15	12.32±1.22	57.5±6.97	20.1±8.81
Female	11.56±1.15	12.33±1.21	57.76±9.0	19.83±8.3	11.31±0.95	12.25±1.04	54.97±8.63	21.9±8.81
p-value	0.555	0.398	0.554	0.758	0.198	0.798	0.244	0.414
Affected side								
Right	11.68±0.82	12.53±1.43	60.89±7.17	20.39±6.35	11.42±0.84	12.05±1.35	50.74±7.2	27.63±7.0
Left	12.52±1.43	12.06±0.97	51.0±7.19	27.23±7.28	11.53±1.18	12.29±0.92	56.67±7.46	22.4±7.93
Bilateral	11.43±1.37	12.54±1.3	60.7±7.14	14.8±5.91	11.38±1.06	12.38±1.04	58.93±7.54	16.32±5.84
p-value	0.720	0.403	<0.001*	<0.001*	0.884	0.579	0.002*	<0.001*
Headache								
No	11.59±1.07	12.5±1.24	57.23±6.93	20.2±9.17	11.43±0.93	12.3±1.03	55.0±6.75	21.47±8.64
Yes	11.37±1.18	12.3±1.32	59.69±9.92	18.69±6.35	11.41±1.18	12.22±1.22	57.25±9.91	20.91±7.78
p-value	0.424	0.511	0.243	0.467	0.913	0.760	0.291	0.798

Table 5. Association of MIR parameters and demographic data

G1 = conservative treatment group; G2 = splinting group; G3 = LLLT group; SP = silent period; ms = millisecond A significant difference between groups is indicated by a * sign (p < 0.05).

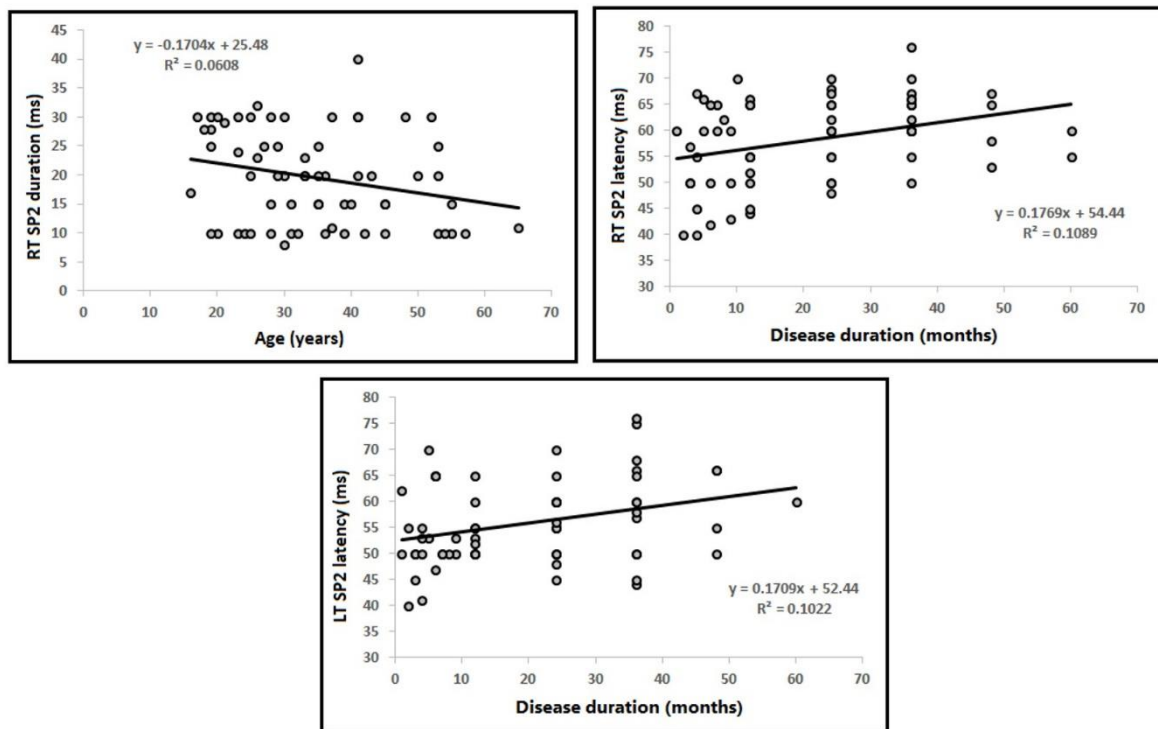A 95% confidence interval of the mean was estimated in the analysis.

Fig. 2. Scatter plot and regression line between MIR right SP2 duration and age (upper left), right SP2 latency and bruxism duration (upper right), and left SP2 latency and bruxism duration (bottom).

Discussion

In the present study, all MIR components of the patients had a longer latency and shorter duration than the control group; also, 17 patients showed absent SP2 component. These findings were consistent with those published elsewhere (6,24,25). In contrast, Luco (26) discovered that in patients with SB, the SP2 was significantly shortened or nonexistent; whereas, the SP1 was somewhat accelerated and shortened. When assessing the MIR components, we examined both the latency and duration, considering both sides as patients may experience bruxism on either or both sides. Consequently, the data could reveal prolonged latency or shortened duration on either side. The significant distinction arises when comparing the data of treated groups to control values. Therefore, minor discrepancies between G1 and G2 at baseline regarding latency or duration would not substantiate differences in parameters post-treatment.

These findings can be explained by first realizing that the MIR is a period of motor activity inhibition of the jaw-closing muscles. Under typical settings, two silent intervals are recorded on each side of unilateral electrical stimulation. The earliest inhibition period is mediated by one inhibitory interneuron whose generator is located in the midpons, whereas the later longest inhibition period includes fibers descending in the spinal trigeminal tract to the level of the pontomedullary junction and inhibitory interneuron projecting onto bilateral motor neurons of the masseter muscle (7,27,28). It has been proposed that a decrease in duration and degree of suppression may represent reduced inhibitory interneuron activity in the brainstem (24,26).

The second theory is that bruxism is a centrally mediated condition characterized by impaired basal ganglia and limbic system control (6,24,26). Polysomnography and electromyographic findings revealed abnormal contractions of the masseter and temporalis muscles, indicating increased excitability in the trigeminal motor system, which could be attributed to increased excitability of the trigeminal motor nucleus itself or abnormal activation of the motor nucleus via cortical or subcortical pathways.

Gastaldo et al, (6) and Huang et al. (25) discovered that motor-evoked potentials recorded on the masseter muscle were normal, but the SP2 recovery cycle was abnormal in their studies. They concluded that the motor pathway of the jaw-closing muscles is normal in SB, but the excitability of the brainstem interneurons in the reflex circuit is altered.

The unusual SP1 outcome in the current study could be related to the fact that this first inhibitory period appears to be insensitive to peripheral conditioning and suprasegmental modulation, and its latency varies little, implying that it is likely mediated by a small number of afferents.

According to a recent study, the first mandibular inhibitory phase is substantially delayed (even more than 20 ms) in individuals with demyelinating polyneuropathies and severe diabetic polyneuropathy (25). SB is thought to be caused by central nervous system mechanisms (29), rather than peripheral ones; hence, it does not affect the SP1 characteristics.

Seventeen of the total number of patients lacked SP2. Others obtained similar results utilizing magnetic stimulation (24,26). It has been noted that a missing SP response is similar to a jaw jerk and that it may be absent bilaterally in elderly patients or patients with malocclusion (30).

Only the SP2 was altered in the LLLT group as compared to the other treatment groups in the present study. The latency was greatly reduced after 2 weeks and also after 1 month of treatment; however, the length was significantly increased after 1 month of treatment (Tables 3-6).

It is worth noting that the SP2 was re-measured in the 17 individuals who had no SP2 after 6 weeks of therapy. Most noteworthy, all five patients treated with LLLT had a latency of 60 to 65 ms and a duration of 10 to 25 ms; whereas, three patients out of four in the G1 and four patients out of eight in the G2 had absent SP2. These data highlight the well-known effect of LLLT on modifying the MIR and thus the SB.

To the best of our knowledge, this is the first study to demonstrate the effect of LLLT on MIR components in SB patients. Previous research demonstrated a substantial difference in signs and symptoms (rather than MIR) of the parafunction following the use of this method, at least when compared to the occlusal splint (24,31,32).

As previously indicated, no comparable research is present; however, several publications discussed the effect of splint treatment on bruxism. The position of the bite is crucial in oral appliance therapy for SB. If the bite on the device is in the molar region, the MIR remains suppressed (producing excessive bite force); however, if it is in the cuspid or incisal region, the MIR is reactivated and the bite force is reduced to normal levels (8,33-35).

These principles were adopted early in the prototype stage of the Luco Hybrid OSA Appliance,

yielding great results. In the majority of the cases, the forward bite effectively reduced SB symptoms within 2 weeks. This is attributable to the reactivation of the MIR, which results in control of the bite force with the cuspid bite, which confirms prior research (36) and is confirmed by EMG home sleep study recording.

Age had a negative correlation with SP2 duration. Age-related changes in several aspects of neuromuscular control begin around the age of 50-60 years, increase in the 70s, and become more pronounced in the 80s (37,38). Furthermore, the jaw-jerk reflex was absent in five of nine participants over the age of 70 years (39). A bilateral absence of the jaw-jerk reflex, even in contracting muscles, has been thought to be a common finding in adults over the age of 70 years (38,40).

Gender did not affect the MIR parameters in the current study. Many studies' findings contradict this conclusion. Women's reflexes were uniformly faster, except for the latency of the second phase of depression, and they had a long silent period (41). Furthermore, various limb stretch reflexes in women were consistently slower than in men (42). Furthermore, women under the age of 70 years had a lower occurrence of the reaction than men, but this difference vanished beyond that (43).

The following limitations apply to this study: first, the small sample size. Second, the superiority of LLLT in improving MIR is called into question. Even though MIR showed abnormalities during the day, due to the paroxysmal nature of the parafunction, a mild improvement in MIR parameters at night may occur while the patient is wearing the occlusal splint. Nonetheless, since voluntary contractions of the jaw-closing muscles are required, MIR could not be evaluated while the person was sleeping. Third, patients receiving conservative and occlusal splint treatment must comply with the treatment in wearing the splint or taking the medications; whereas LLLT does not require patient cooperation. Fourth, because of the short duration of treatment (6 weeks), there is no certainty that the improvement with LLLT is permanent or transitory.

Conclusion

Patients with SB had an abnormal MIR response. SP2 parameters improved considerably 1 month later in the LLLT group, indicating that this modality was superior to others. SP2 duration was negatively correlated with age; however, gender did not influence the MIR components.

References

1. De Laat A, Macaluso GM. Sleep bruxism as a motor disorder. Mov Disord. 2002;17 Suppl 2:S67-9. PMID: 11836759 doi: 10.1002/mds.10064
2. Macedo CR, Silva AB, Machado MA, et al. Occlusal splints for treating sleep bruxism (tooth grinding). Cochrane Database Syst Rev. 2007;2007(4):CD005514. doi: 10.1002/14651858.CD005514.pub2. PMID: 17943862
3. Shetty S, Pitti V, Babu CLS, et al. Bruxism: a literature review. J Indian Prosthodont Soc. 2010;10(3):141-8 PMID: 21886404 doi: 10.1007/s13191-011-0041-5
4. Sari S, Sonmez H. The relationship between occlusal factors and bruxism in permanent and mixed dentition in Turkish children. J Clin Pediatr Dent. 2001;25(3):191-4. PMID: 12049076 doi: 10.17796/jcpd.25.3.84m695q650622568
5. Kalantzis A, Scully C. Oxford Handbook of Dental Patient Care, The Essential Guide to Hospital Dentistry. 2nd ed. New York: Oxford University Press. 2005;332.
6. Gastaldo E, Quatrale R, Graziani A, et al. The excitability of the trigeminal motor system in sleep bruxism: a transcranial magnetic stimulation and brainstem reflex study. Orofac Pain. 2006;20(2):145-55. PMID: 16708832
7. Schoenen J, Agrégé MD. Exteroceptive suppression of temporalis muscle activity in patients with chronic headache and in normal volunteers: methodology, clinical and pathophysiological relevance. Headache. 1993;33(1):3-17. doi: 10.1111/j.1526-4610.1993.hed3301003.x
8. Cruccu G, Deuschl G. The clinical use of brainstem reflexes and hand-muscle reflexes. Clin Neurophysiol. 2000;111(3):371-87. PMID: 10699396 doi: 10.1016/s1388-2457(99)00291-6
9. Huynh N, Manzini C, Rompré PH, et al. Weighing the potential effectiveness of various treatments for sleep bruxism. J Can Dent Assoc. 2007;73(8):727-30. PMID: 17949541
10. Lobbezoo F, van der Zaag J, van Selms MK, et al. Principles for the management of bruxism. J Oral Rehabil. 2008;35(7):509-23. PMID: 18557917 doi: 10.1111/j.1365-2842.2008.01853.x
11. Jadidi F, Castrillon E, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. J Oral

Rehabil. 2008;35(3):171-83. PMID: 18254794 doi: 10.1111/j.1365-2842.2007.01781.x

12. Winocur E, Gavish A, Voikovitch M, et al. Drugs and bruxism: a critical review. J Orofac Pain. 2003; 17(2):99-111. PMID: 12836498

13. Tsukiyama Y, Baba K, Clark GT. An evidence-based assessment of occlusal adjustment as a treatment for temporomandibular disorders. J Prosthet Dent. 2001;86(1):57-66. PMID: 11458263 doi: 10.1067/mpr.2001.115399

14. Kato T, Montplaisir JY, Guitard F, et al. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res. 2003;82(4):284-8. PMID: 12651932 doi: 10.1177/154405910308200408

15. Venezian GC, da Silva MA, Mazzetto RG, et al. Low-level laser effects on pain to palpation and electromyographic activity in TMD patients: a double-blind, randomized, placebo-controlled study. Cranio. 2010;28(2):84-91. PMID: 20491229 doi: 10.1179/crn.2010.012

16. Ommerborn MA, Schneider C, Giraki M, et al. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. Eur J Oral Sci. 2007; 115(1):7-14. PMID: 17305711 doi: 10.1111/j.1600-0722.2007.00417.x

17. Nascimento LL, Amorim CF, Giannasi LC, et al. Occlusal splint for sleep bruxism: an electromyographic associated to Helkimo Index evaluation. Sleep Breath. 2008; 12(3):275-80. PMID: 17987334 doi: 10.1007/s11325-007-0152-8

18. Okeson JP. The effects of hard and soft occlusal splints on nocturnal bruxism. J Am Dent Assoc. 1987; 114(6):788-91. PMID: 3475357 doi: 10.14219/jada.archive.1987.0165

19. Melis M, Di Giosia M, Zawawi KH. Low level laser therapy for the treatment of temporomandibular disorders: a systematic review of the literature. J Craniomand Prac. 2012;30(4):304-12. PMID: 23156972 doi: 10.1179/crn.2012.045

20. Zakrzewska JM. Multi-dimensionality of chronic pain of the oral cavity and face. J Headache Pain. 2013;14(1):37. PMID: 23617409 doi: 10.1186/1129-2377-14-37

21. Herpich CM, Amaral AP, Leal-Junior EC, et al. Analysis of laser therapy and assessment methods in the rehabilitation of temporomandibular disorder: a systematic review of the literature. J Phys Ther Sci. 2015;27(1):295-301. PMID: 25642095 doi: 10.1589/jpts.27.295

22. Fernández-Sánchez MT, Díaz-Trelles R, Groppetti A, et al. Nefopam, an analogue of orphenadrine, protects against both NMDA receptor-dependent and independent veratridine-induced neurotoxicity. Amino Acids. 2002;23(1-3):31-6. PMID: 12373515 doi: 10.1007/s00726-001-0106-6

23. İnan R, Şenel GB, Yavlal F, Karadeniz D, Gündüz A, Kızıltan ME. Sleep bruxism is related to decreased inhibitory control of trigeminal motoneurons, but not with reticulobulbar system. Neurol Sci. 2017;38(1):75-81. PMID: 27629540 doi: 10.1007/s10072-016-2711-x

24. Huynh NT, Rompré PH, Montplaisir JY, Manzini C, Okura K, Lavigne GJ. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. Int J Prosthodont. 2006; 19(5):435-41. PMID: 17323720

25. Huang H, Song YH, Wang JJ, et al. Excitability of the central masticatory pathways in patients with sleep bruxism. Neurosci Lett. 2014;558:82-6. PMID: 24269982 doi: 10.1016/j.neulet.2013.11.014

26. Luco K. How sleep bruxism and tension headaches affect the masseter inhibitory reflex. J Sleep Disor: Treat Care. 2017;6:1-4. doi: 10.4172/2325-9639.1000198

27. Cruccu G, Iannetti GD, Marx JJ, et al. Brainstem reflex circuits revisited. Brain. 2005; 128(Pt 2):386-94. PMID: 15601661 doi: 10.1093/brain/awh366

28. Aramideh M, Ongerboer De Vısser BW. Brainstem reflexes: electrodiagnostic techniques, physiology, normative data, and clinical applications. Muscle Nerve. 2002;26(1):14-30. PMID: 12115945 doi: 10.1002/mus.10120

29. Wassell R, Naru A, Steele J, et al. Applied occlusion. London: Quintessence. 2008;26-30.

30. Cruccu G, Ongerboer de Visser BW. The jaw reflexes. The International Federation of Clinical Neurophysiology. Electroencephalogr Clin Neurophysiol Suppl. 1999;52:243-7. PMID: 10590991

31. Salgueiro MDCC, Bortoletto CC, Horliana ACR, et al. Evaluation of muscle activity, bite force and salivary cortisol in children with bruxism before and after low level laser applied to acupoints: study protocol for a randomised controlled trial. BMC Complement Altern Med. 2017;17(1):391. PMID: 28789647 doi: 10.1186/s12906-017-1905-y

32. Kobayashi FY, Castelo PM, Gonçalves MLL, et al. Evaluation of the effectiveness of infrared light-emitting diode photobiomodulation in children with sleep bruxism: Study protocol for randomized clinical trial. Medicine (Baltimore).

2019;98(38):e17193. PMID: 31567965 doi: 10.1097/MD.0000000000017193

33. Nakashima K, Takahashi K. Exteroceptive suppression of the masseter, temporalis and trapezius muscles produced by mental nerve stimulation in patients with chronic headaches. Cephalalgia. 1991;11(1):23-8. PMID: 2036666 doi: 10.1046/j.1468-2982.1991.1101023.x

34. Schoenen J, Bottin D, Sulon J, et al. Exteroceptive silent period of temporalis muscle in menstrual headaches. Cephalalgia. 1993;11(2):87-91. PMID: 1860134 doi: 10.1046/j.1468-2982.1991.1102087.x

35. Wallasch TM, Göbel H. Exteroceptive suppression of temporalis muscle activity: findings in headache. Cephalalgia. 1993;13:11-4. PMID: 8448781 doi: 10.1046/j.1468-2982.1993.1301011.x

36. Cruccu G, Agostino R, Inghilleri M, et al. The masseter inhibitory reflex is evoked by innocuous stimuli and mediated by A beta afferent fibres. Exp Brain Res. 1989;77:447-50. PMID: 2792292 doi: 10.1007/BF00275005

37. Pyykko I, Jantti P, Aaalto H. Postural control in elderly subjects. Age Ageing. 1990;19(3):215-21. PMID: 2363386 doi: 10.1093/ageing/19.3.215

38. Drummond J, Newton J, Scott J. Orofacial ageing. In: Barnes IE, Walls A, eds. Gerodontology. Oxford: Wright. 1994:17-28.

39. Ongerboer de Visser BW, Goor C. Electromyographic and reflex study in idiopathic and symptomatic trigeminal neuralgias: latency of the jaw and blink reflexes. J Neurol Neurosurg Psychiat 1974;37:1225-30. PMID: 4457616 doi: 10.1136/jnnp.37.11.1225

40. Kimura J, Daude J, Burke D, et al. Human reflexes and late responses. Report of an IFCN committee. Electromyogr Clin Neurophysiol. 1994;90:393-403. PMID: 7515783 doi: 10.1016/0013-4694(94)90131-7

41. Kossioni AE, Karkazis HC. Jaw reflexes in healthy old people. Age and Ageing.1998;27:689-95. PMID: 10408662 doi: 10.1093/ageing/27.6.689

42. Carel RS, Korczyn AD, Hochberg Y. Age and sex dependency of the Achilles tendon reflex. Am J Med Sci. 1979;278:57-63. PMID: 484592 doi: 10.1097/00000441-197907000-00007

43. Milne J S, Williamson J. The ankle jerk in older people. Geront Clin 1972;14:86-8. PMID: 4665887 doi: 10.1159/000245378