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ORIGINAL ARTICLE / ARAŞTIRMA MAKALESİ

Assessment of Brainstem Reflexes with Transcranial Magnetic Stimulation in Bruxism: The Role of Central Mechanisms in Pathophysiology

Bruksizmde Beyin Sapı Reflekslerinin Transraniyal Manyetik Stimülasyon ile Değerlendirilmesi: Merkezi Mekanizmaların Patofizyolojideki Rolü

Bilgin Ali ŞENTÜRK[1], Gaye YILDIRIM[2], Ayşegül Özer ÇELIK[3], İbrahim ÖZTURA[4]

ABSTRACT

Objectives: We aim to prove the central etiology hypothesis for bruxism, we plan to examine the structural components that contribute to the occurance of RMMA/Bruxism such as cortical, subcortical structures and as a key roleplaying component, the brainstem structures by investigating the MEP, CSP, MIR (SP1 and SP2) and the blink reflex (R1 and R2) and central conduction time.

Materials and Methods: In this study, a total of 77 subjects investigated to find any difference between the two groups. The MIR and blink reflexes studied by transcranial magnetic stimulation (TMS) and electric stimulation. The cortical silent period (CSP) evoked in contralateral masseter muscle by TMS. Central motor conducting times were evaluated.

Results: The absence of SP2 (component of MIR), R2 (component of blink reflex) latency and left APB (abductor pollicis brevis) muscle F wave latency are found to be significantly different between two groups.

Conclusions: As a result of our study, the loss of the SP2 component and the prolongation of the latency of the R2 component, and the localization of reflex circuits in the brainstem, although their pathways are separate, bruxer suggested that the pathophysiology may have a central origin in most of the bruxers.

Keywords: Bruxism, Transcranial Magnetic Stimulation, Masseter Inhibitory Reflex

Bilgin Ali ŞENTÜRK (✉)
Dt. Basic Neurosciences Ph.D., Health Sciences Institute, Dokuz Eylul University, Izmir, Turkey. bilginalisenturk@yahoo.com

Gaye YILDIRIM
Specialist Neurologist, Ordu, Turkey.

Ayşegül Özer ÇELIK
Specialist Dr. Urla Goverment Hospital, Department of Neurology, Urla, Izmir.

İbrahim ÖZTURA
Prof. Dr. Medical School, Neurology Department, Department, Dokuz Eylül University, Izmir, Turkey

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ÖZ

Amaç: Bu çalışmada bruksizmin merkezi mekanizmalarla ilgili olabileceği hipotezini kanıtlamak için MEP, CSP, MIR (SP1 and SP2), göz kırpma refleksi (R1 and R2) ve merkezi iletim zamanını inceleyerek RMMA/Bruksizm oluşumuna katkıda bulunan kortikal, subkortikal yapılar ve anahtar rol oynayan beyin sapı yapıları gibi yapısal bileşenlerin incelenmesi amaçlanmaktadır.

Gereç ve Yöntemler: Bu çalışmada, iki grup arasında herhangi bir fark olup olmadığını bulmak için toplam 77 kişi ile çalışılmıştır. MIR ve göz kırpma refleksleri, transkraniyal manyetik stimülasyon (TMS) ve elektrik stimülasyonu ile incelenmiştir. Kortikal sessiz periyot (CSP) kontralateral masseter kasta TMS ile uyarılmıştır. Merkezi motor iletim süreleri değerlendirilmiştir.

Bulgular: SP2 (MIR komponenti)'nin olmayışı, R2 (blink refleks komponenti)'nin latansı ve sol APB (Abdüktör pollisis brevis) kastan ölçülen F dalgası latansı incelendiğinde iki grup arasında istatistiksel olarak anlamlı şekilde fark bulunmuştur.

Sonuç: Çalışmamız sonucunda SP2 bileşeninin kaybolması ve R2 bileşeninin gecikme süresinin uzaması ve beyin sapındaki refleks devrelerinin lokalizasyonu, yolları ayrı olmasına rağmen, patofizyoloji bruksistlerin çoğunda merkezi bir kökene sahip olabileceğini desteklemektedir.

Anahtar Kelimeler: Bruksizm, Transkraniyal Manyetik Stimülasyon, Masseter Inhibitor Reflex

INTRODUCTION

Bruxism may be classified as noctrunal or diurnal bruxism according to circadian manifestations, as primary (idiopathic) and secondary (iatrogenic) according to the presence of underlying neurological or psychiatric disease and drug use (Guaita & Högl, 2016).

According to the updated concensus, the definition of bruxism is explained with repetative masticatory muscle activity characterized by clenching or grinding of the teeth and by bracing or thrusting of the mandibula involuntarily. In healthy indiviuals bruxism shouldn't be considered as a

disorder. The absence of tooth contact supports that it is a central movement. In the concensus a grading system as has been proposed for possible, probable and definitive bruxism (Lobbezoo et al., 2018).

It can also be defined as the involuntary, unconscious, and excessive grinding of teeth (Lal & Weber, 2022).

High-level activities of the chewing muscles have negative oral consequences such as chewing muscle pain, temporomandibular joint pain, periodontal problems, tooth wear, prosthodontic problems (Raphael et al., 2016).

Its etiology is multifactorial, both peripheral (morphological) and central (pathophysiological and psychological) factors play a role. While it was thought to be caused by defects in the occlusal and orofacial region structures in the past, the findings from the recent studies gradually raise the importance of central factors in the pathogenesis (Winocur et al., 2007; Nélio et al., 2015; Manfredini et al., 2017).

Understanding "chewing automatism" defined as rhythmic masticatory muscle activity (RMMA) without tooth-grinding is important to understanding the pathogenesis of bruxism. This activity is observed in 60% of healthy people (Lavigne et al.,1996), it is 3 times more common in bruxers (Lavigne et al., 2003) and is usually associated with sleep arousals (unconscious and transient changes in brain electroencephalography (EEG) activity, lasting 10-15 seconds, with or without changes in cardiac rate and muscle tone).

Brainstem structures play a particularly key role in the genesis and control of rhythmic jaw movements and RMMA. Numerous nuclei in the brain stem (nucleus reticularis pontis oralis, pontis caudalis and parvocellularis) and neurotransmitters (serotonin, dopamine, GABA, and noradrenaline) regulate the tone of the muscles in the formation of the chewing movement (Lavigne et al., 2003). Studies have demonstrated that dopamine reduction in the mesocortical and nigrostriatal pathways causes disinhibition in the frontal cortex, resulting in increased RMMA and bruxism.

As we aim to prove the central etiology hypothesis for bruxism, we plan to examine the structural components that contribute to the occurance of RMMA/bruxism such as cortical, subcortical structures and as a key roleplaying component, the brainstem structures by investigating the MEP, CSP, MIR (SP1 and SP2) and the blink reflex (R1 and R2) and central conduction time.

MATERIALS AND METHODS

Our study was conducted in the Neurophysiology Laboratory of Dokuz Eylül University and Ethics Committee approval was obtained from Dokuz Eylül University. Forty bruxers (25 females and 15 males) and 37 normal subjects (17 females and 20 males) aged between 16-73 years were included in the study. Participants were selected from volunteers who were admitted to the Neurology Department Sleep Disorders Center of Dokuz Eylül University with various complaints and met bruxism diagnosis criteria after the examination.

All participants provided an informed written consent form.

Inclusion criteria were determined based on the moderate and severe chronic bruxism criteria set by the American Sleep Disorders Association (Table 1) (Koyano et al., 2008).

Cortical motor evoked potentials (c-MEP), CSP, MIR (Silent periods 1 and 2 (SP1 and SP2)), and central conduction time (CMCT) were measured using the TMS method by recording from both masseter muscles. The blink reflex (R1 and R2) was studied by the electrical stimulation method.

Our study was approved by Dokuz Eylul University Non-Interventional Research Ethics Committee with protocol number 3155-GOA and decision number 2017/06-34.

Table 1. Inclusion and exclusion criterias of the study

Inclusion Criteria	Exclusion Criteria
• The patient's awareness about bruxism •Awarness of grinding sounds or clenching at sleep •If one or more of the followings presents additionally • Tooth ware • Jaw muscle fatigue, pain • Masseter muscle hypertrophy	• Known neurological disease • History of epilepsy • Craniofacial pain • Temporomandibular joint disorder • Chronic headache • Missing more than 2 molar teeth, except wisdom tooth • Use of removable dentures • Pregnancy • Cardiac Pacemaker • Cochlear implant • Metal-containing implant • Menstrual period for female patients

Neurophysiological Examination:

Transcranial Magnetic Stimulation (TMS) patient selection questionnaire was applied to all subjects; eligible subjects were included in the study. Before the examination, a demographic data form consisting of age, height, weight,

body mass index (BMI), and neck circumference was completed.

TMS is a method of generating an electrical field in the brain by means of electromagnetics and may be used for neuromodulation and neurostimulation when applied sufficiently to depolarize neurons (Rossi et al., 2009; Herrero et al., 2018).

Cortical Motor Evoked Potential (c-MEP) and Cortical Silent Period (CSP):

C-MEP recording was performed from the contralateral masseter muscle by performing 6-8 single stimulations. Optimal MEP responses for the masseter muscle were obtained by performing a stimulation at a magnitude of 40% of the maximum device output (Pauletti et al., 1993).

The CSP refers to an interruption in electromyographic activity when cortical magnetic stimulation is applied while the target muscle is in voluntary contraction (Rossini & Rossi, 2007), and the duration of the activity increases as the magnetic stimulation intensity is increased (Cantello et al., 1992). The silent period elicited by transcranial stimulation is generated by intracortical inhibition systems (Cohen et al., 1992).

Masseter inhibitory reflex (MIR):

MIR is a reflex inhibition that occurs in the muscles that close the jaw as a result of mechanical, electrical, or magnetic stimulation of the mouth or face area and can only be demonstrated by electrophysiological methods. It is a protective reflex related to mandibular movements during mastication and articulation (Huang et al., 2014). It is generated by stimulation of the mental nerve, and consists of two silent periods that occur as an interruption in the electromyography (EMG) recording performed while the ipsilateral and contralateral masticatory muscles are in voluntary contraction: silent period 1(SP1) and silent period 2(SP2). The afferent stimuli of SP1 are connected to an inhibitory interneuron which is located in the H region surrounding the ipsilateral trigeminal motor nucleus and directly reaches the motor nucleus, and they cross at the motor nucleus level. SP2 is a polysynaptic reflex with a latency of 45-50 seconds (Cruccu et al., 1991). Interneurons responsible for SP2 are located in the bulbar reticular formation near the trigeminal nucleus caudalis (Huang et al., 2014). It is thought that SP2 is probably conveyed to the spinal trigeminal tract by A-beta fibers, and occurs by interacting polysynaptically with ipsilateral and contralateral interneurons and inhibiting masseteric motor neurons (Cruccu et al., 1984).

For MIR recording, patients were asked to perform voluntary contraction at 30% of their maximum contraction, and this level was determined by audiovisual feedback. The responses were superimposed while establishing the duration, the time from the onset of the MEP response to the point at which basal activity reappears was marked and recorded.

Central Motor Conduction Time:

It was calculated using the F wave latency method. Ag-Ag surface cup electrodes were placed on the left abductor pollicis brevis (APB) muscle using the belly-tendon method. The cortical stimulation point was designated by using similar studies in the literature as references to obtain cortical evoked potential. The latency of the motor response (M response) obtained by performing supramaximal electrical stimulation of the median nerve at the wrist level was recorded. Among the F waves obtained after 10 consecutive supramaximal stimulation, the one with the shortest latency was chosen.

Peripheral conduction time was calculated by the formula of ((F latency + M latency) – 1) x0.5 (Hallet, 2007).

Central Conduction time was calculated according to the formula of the (Udupa & Chen, 2013); left APB MEP latency – peripheral conduction time.

Blink Reflex:

Medelec EMG device was used. The active electrode of Ag-Ag surface cup electrodes was placed lateral to the lateral epicanthus and the reference electrode was placed 2 cm lateral to the active electrode. Stimulation intensity was increased at 10-second intervals until stable responses were recorded. Latencies of ipsilateral R1 and contralateral R2 responses were recorded.

Statistical Analysis:

The samples were normally distributed according to Kolmogorov Smirnov test and parametric statistics were used. In this study we investigated whether central factors

affect bruxism or not as has been done in previous studies (Galloway et al., 2013; Huang et al., 2014). We didn't assess the amount of correlation between central factors with bruxism.

Data were assessed by SPSS 18 program. In addition to descriptive statistics, chi-square for categorical variables from hypothesis tests, t-test for continuous variables were used. A $p<0.05$ value was considered statistically significant.

RESULTS

Demographic Data:

There was no significant difference between the groups in terms of age (p=0.334), in terms of BMI (p=0.617), in terms of neck circumference (p=0.631) (Table 2).

Table 2. Demographic data

	Normal(n=37)	Bruxer(n=40)	P<0.05
Age	41.49±12,355	44.45±14.22	0.334
Body Mass Index	25.85±4.3752	25.33±4.212	0.617
Neck Circumference (cm)	36.32±4.3	35.85±4.165	0.631

Neurophysiological Parameters:

1) Motor-evoked potentials in masseter muscle (c-MEP): c-MEP obtained from the left masseter muscle with stimulation from the right hemisphere was found to be mildly longer in the bruxer group compared to the control group (6.21±1.15/6.14±0.912 ms). There was no statistically significant difference between the groups. (p right=0.757, p left=0.336) (Table 3).

2) Cortical silent period (CSP): There was no statistically significant difference between the groups for CSP durations obtained with right and left stimulation (p right stimulation=0.757, p left stimulation=0.991) (Table 3).

3) Central Motor Conduction Time: There is no significant difference between the groups in terms of central motor conduction time (NK:7.75 ± 2.993 and B:7.75 ± 2.12 ms) (p=0.998) (Table 3).

4) Masseter Inhibitory Reflex (SP1 and SP2): While SP1 was detected both in the normal control (NC) and bruxer (B) groups, the absence of SP2 was prominent in the bruxer group and was statistically significant (**p=0.00**). (Table 4), (Fig. 1) (Fig. 2)

Table 3) Latency of motor evoked potentials (c-MEP), durations of cortical silent period (CSP) obtained from both masseter muscles with stimulation from the right and left hemispheres time

	Normal		Bruxer		P<0.05 (R/L)
	R hemisphere stimulation	**L hemisphere stimulation**	**R hemisphere stimulation**	**L hemisphere stimulation**	
c-MEP latency	6.14±0.912	6.098±0.81	6.21±1.15	6.3±0.954	0.757/0.336
CSP (ms)	50.95±25.746	50,42±26.41	54.021±24.92	50.5±24.22	0.757/0.991
CMCT(ms)	7.75 ± 2.993		7,75± 2.993		0.998

CSP: Cortical silent period **L**: Left **R**: Right **CMCT**: Central motor conduction

Table 4. MIR SP1 and SP2 components

	Normal	Bruxer	P<0.05
Duration of SP1 (ms)	24.17±5.64	29.07±22.17	0.196
Duration of SP2 (ms)	36.22±7.8	35.1±9.02	0.678
Presence of SP1 %	100	100	
Presence of SP2 %	**84**	**35**	**0.00**

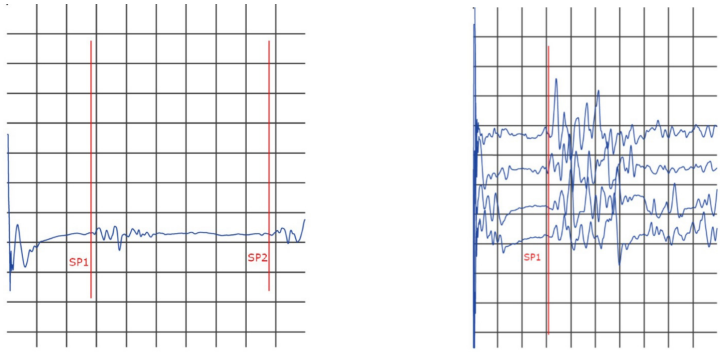

Figure 1: Masseter Inhibitory Reflex

5) APB muscle cortical MEP, M response, and F latency: No significant difference was found between the normal control and the bruxwe group in terms of cortical MEP latency (21.26 ± 2.75/20.57. D2.04 ms) and M response latency (2.94 ± 0.37/ 3.01 ± 0.46 ms) obtained by recording from the APB muscle (MEP latency p=0.216 and M latency p=0.472).

F wave latency was found to be significantly shorter (23.6 ± 2.51 /25.07 42.34 ms) in the bruxer group, and this difference was statistically significant (**p=0.01**). As the mean age of the groups is similar, this difference may be explained by the variation in the limb length of the subjects as well as the involvement of the lower brainstem upper spinal region when interpreted in conjunction with the impact on the R2 response of the blink reflex. (Malick et al., 2000).

6) Blink reflex: While R1 latency was similar in both groups (N: 11.28 ± 1.313 and B: 11.27 ± 1.314 ms) (p = 0.982), R2 latency was significantly prolonged in the bruxer group (35.08±4.445 /32.755±4.613 ms) and this difference was statistically significant (**p=0.027**) (table 5).

Table 5. Blink reflex (R1 and R2) and F wave latency recorded from the left median nerve

	Normal	Bruxer	P<0.05
R1 latency (ms)	11.28 ± 1.313	11.27± 1.314	0.982
R2 latency (ms)	**32.755±4.613**	**35.08±4.445**	**0.027**
F wave latency (ms)	25.07±2.34	**23.6±2.51**	**0.01**

DISCUSSION

Demographic Data:

Studies have reported that bruxism is more common in females and similarly, the number of females in the bruxer group selected was also found to be higher in our study (Melis &Abou-Atme, 2003). However, no statistically significant difference was observed.

Neurophysiological Parameters:

About Neurophysiological parameters, there was no statistically significant difference between the groups in terms of MEP latency, CSP durations obtained with right and left stimulation, MIR, APB muscle cortical MEP, M response latency, but the absence of SP2 was prominent in the bruxer group and statistically significant (p=0.00). F wave latency was found to be significantly shorter in the bruxer group and the difference is statistically significant (p=0.01). Lastly R2 latency was significantly prolonged in the bruxer group and the difference is statistically significant (p=0.027).

In this study there was no significant difference between two groups in terms of age, BMI and neck circumference.

Neurophysiological Assessment:

In our study, no difference between the groups in terms of MEP latency and central motor conduction time was observed, however in the literature, MEP latency, amplitude, and central motor conduction time were also found to be similar to normal controls (Gastaldo at al., 2006; Huang et al., 2014). This similarity between the groups can be considered as that corticobulbar tract functions are also normal in bruxers and there is no change in cortical motor system excitability (Gastaldo et al., 2006).

In a study of Parkinson's patients, while MEP latencies and amplitudes remained the same, the duration of the CSP was prolonged after Levodopa treatment. The dopamine reduction in the basal ganglia and the nigrostriatal pathway has been proposed to lead to abnormal silent periods may be as a result of reduced facilitation of the motor cortex leading to excitation of the cortical inhibitory interneurons (Priori et al., 1994; Inghilleri et al., 1996). In our study, CSP, CCT, and MEP latencies for both cortical hemisphers were found to be similar. This conclusion indicated no significant excitability differences between cortexes in both hemispheres among two groups.

In a previous study aiming to understand the abnormal networks related to the excitability of masticatory pathways in patients with sleep bruxism the MIR after electrical stimulation and auditory startle reaction (ASR) were examined in sleep bruxers and control groups. The duration of SP1 and SP2 components of MIR was found to be shorter in sleep bruxers than controls means a lower suppression of SP's in sleep bruxer group. The ASR responses including masseter muscle found to be similar in both groups meaning the integrity of brainstem pathways mediating ASR are intact in sleep bruxers unlike dopamine related pathophysiology like dystonia, restless leg syndrome in which exaggerated ASR responses were observed as an indicator of disinhibition of reticulospinal pathways (İnan et al., 2017).

Plamen Tzvetanov 2009, found S2 in MIR reduced in intensity and duration or absent in 66.7 % of the individuals with episodic TTH (Tansion Type Headache). The changes

observed in TTH were claimed to be due to hyperexcitability of the raticular nuclei, that has inhibitor effect on the medullar inhibitor interneurons (Tzvetanov et al., 2009).

It has been claimed that SP1 and SP2 disappeared in mid-pontin lesions, only one of the SP1 and SP2 components were affected in isolated lesions of different regions, and therefore these pathways are independent from each other (Ongerboer et al.,1990). Stimulation of the bulbar reticular formation located in the pontomedullary junction area caused an inhibitory effect on bilateral trigeminal motor nuclei. (Takatori et al. 1981).

In our study, the most remarkable finding is that while SP1 is obtained in both healthy subjects and bruxers (Figure 1, SP2 is significantly absent in bruxers (Figure 2). The absence of SP2 in bruxers was found to be insignificant in one of two previous studies (p=0.053) (Gastaldo et al. 2006) and the absence of SP2 in bruxers was found to be significant in the other study (p=0.041) (Huang et al., 2014).

The inhibitory effect conveyed to the trigeminal motor nucleus is considered to originate from the ventral nucleus pontis caudalis and has a role in the SP2 mechanism (Tanaka et al., 1999; Lund & Kolta, 2005; Lund & Kolta, 2006; Kolta et al., 2010). While SP1 is not affected, the loss of only SP2 in bruxers may be the result of a pathology involving the brain stem region especially lower ponds and medulla similar as R2 component.

Other remarkable findings in our study are also associated with the blink reflex. The blink reflex is the response of contraction of the orbicularis oculi muscle to the stimulation of the supraorbital nerve. It has two components of R1 and R2. R1 has a short latency (13 ms) and the response is ipsilateral. The R2 component has a long latency (40 ms) and the response is bilateral. R1 is an oligosynaptic response and its circuit is located in the pons like SP1. The afferent fibers of R2 descend from the pons to the most caudal part of the spinal trigeminal nucleus in the spinal trigeminal area in the lateral medulla. R2 ascends bilaterally and polysynaptically, medial to the spinal trigeminal nucleus and in the lateral medullary reticular area, and reaches the facial nerve (Scott et al., 2003).

In a study examining blink and MIR together in individuals with dystonia and blepharospasm, no significant abnormality was found in R1 and R2 (Ongerboer De Visser & Kuypers, 1978; Pauletti et al., 1993). However, a significant shortening was observed in the recovery times of SP2 and R2. This condition has been regarded as a decrease in the control of the basal ganglia over the brainstem reflex networks (Aramideh & Ongerboer de Visser, 2002).

On the other hand neuroplastic changes occurs as a result of periferal changes in the oral-facial and mastication systems also indicates the affect and relation of central mechanism involving masticatory activity and bruxism.

A study hypothesised that bidirectional or multidisectional motor movements may differentially alter excitability of the tongue and first dorsal interosseous (FDI) muscle groups, found significant neuroplastic changes in the tongue motor cortex area (MI) as a result of bidirectional and multidirectional training of the tongue. Multidirectional training results in a great number of motor map sites that show a significant increase in excitability (Boudreau et al., 2013).

Previous TMS studies in healty subjects support the idea that repeated standardized jaw movements can result in neuroplastic changes centrally. These changes include chorticomotor control of jaw closing muscles (Lida et al., 2014).

A significant increase in the amplitude of the masseter muscle MEP's (p=0,036) after four weeks of altering the occlusal vertical dimension (OVD) and a significant increase in numerical rating scale scores of masticatory ability after four weeks may indicate an adaption of the masticatory systems to the altered oral conditions (Deng et al., 2018).

Such results also indicates the importance of periferal affects on masticatory system and also strenght the affect of periferal factors on bruxism.

In summary, since it is considered that the R2 response is polysynaptic response and is predominantly originated from synaptic connections in the medullary region, the absence of SP2 and the prolonged R2 latency which were statistically significant in bruxers as a result of our study can be considered to be an indicator of medullar involvement (Scott et al., 2003). Although the R2 circuit is also polysynaptic like SP2, it descends to more caudal than SP2 and is conveyed via different pathways. Although it is conveyed via different pathways, the fact that both responses were affected together in our study strengthens our hypothesis of the central origin of bruxism. Also significantly shorter latency of F. Waves of the bruxer group than the control group may be a sign of the lower brainstem where the late blink reflex component R2 traces were found and the upper cervical area interaction (Malick et al., 2000). However, more comprehensive studies with larger numbers of patients are warranted.

Limitations of The Study:

The main limitation of this study is that the cases were selected by dental examination and patient reports. Polysomnographic examination was not performed.

The second limitation is this study is an open study not blind. Also bruxer group wasn't classified as sleep, awake and both.

Another limitation is that SP1 and SP2 were assessed while bruxers were awake. As voluntary contraction is required when evaluating MIR, it is impossible to evaluate it during sleep. The effect of wakefulness and awareness on reticular formation structures and brainstem reflex circuits is unknown.

While assessing the MIR, it is necessary to have a voluntary contraction, but as the masticatory force increases, SP2 decreases, and as the stimulation intensity increases, it increases (Gastaldo et al., 2006). In order to minimize this condition, the bite level was monitored audiovisually on the monitor, and each individual was ensured to have the same intensity of stimulation. Since conditions such as sense of discomfort, pain, irritability, and anxiety can affect inhibitory trigeminal reflex circuits, efforts have been made to make people as calm and neutral as possible (Gastaldo et al., 2006). Although both cases were tried to be minimized, they may have influenced reflex responses.

Our examinations were done in a separate room in which the TMS device and EMG device were found in the neurology department.

This study cannot be generalized due to the number of participants.

CONCLUSION

The pathophysiology of bruxism has not been elucidated yet. Both central and peripheral causes are considered to be factors. As a result of our study, the loss of the SP2 component and the prolongation of the latency of the R2 component, and the localization of reflex circuits in the brainstem, although their pathways are separate, bruxer suggested that the pathophysiology may have a central origin in most of the bruxers.

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CONFLICTS OF INTEREST STATEMENT

There is no conflicts of interest in this study.

REFERENCES

1. Aramideh M, Ongerboer de Visser BW. Brainstem reflexes: Electrodiagnostic techniques, physiology, normative data, and clinical applications. Muscle Nerve. 2002; 26(1):14–30.
2. Boudreau, S.A., Lontis, E.R., Caltenco, H. et al. Features of cortical neuroplasticity associated with multidirectional novel motor skill training: a TMS mapping study. Exp. Brain. Res. 2013; 225(4):513–526.
3. Brocard F, Verdier D, Arsenault I, J. P. Lund, Kolta A. Emergence of ıntrinsic bursting in trigeminal sensory neurons parallels the acquisition of mastication in weanling rats. J. Neurophysiol. 2006; 96(5): 2410–2424.
4. Cantello R, Gianelli M, Civardi C, Mutani R. Magnetic brain stimulation: The silent period after the motor evoked potential. Neurology. 1992;42(10):1951-9.
5. Cohen L. G, Bandinelli S, Topka H. R, Fuhr P, Roth B. J, Hallett M, Topographic maps of human motor cortex in normal and pathological conditions: mirror movements, amputations and spinal cord injuries. Electroencephalogr. Clin. Neurophysiol. Suppl., 1991;43:36-50.
6. Cruccu G, Agostino R, Fornarelli M, Inghilleri M, Manfredi M. Recovery cycle of the masseter inhibitory reflex in man. Neurosci. Lett. 1984;49(1-2):63–68.
7. Cruccu G, Pauletti G, Agostino R, Berardelli A, Manfredi M. Masseter inhibitory reflex in movement disorders. Huntington's chorea, Parkinson's disease, dystonia, and unilateral masticatory spasm", Electroencephalogr. Clin. Neurophysiol. Evoked Potentials. 1991;81(1):24-30.
8. Deng H, Gao S, Lu S, Kumar A, Zhang Z, Svensson P. Alteration of occlusal vertical dimension induces signs of neuroplastic changes in corticomotor control of masseter muscles: Preliminary findings. J. Oral Rehabil. 2018;45(9):710-719.
9. Galloway G. M, Dias B. R, Brown J. L, Henry C. M, Brooks D. A, Uggie E. W. Transcranial magnetic stimulation-may be useful as a preoperative screen of motor tract function. J. Clin. Neurophysiol. 2013;30(4):386-389.
10. Gastaldo E, Quatralo R, Graziani A, Eleopra R, Tugnoli V, Tola M.R. Granieri E. The excitability of the trigeminal motor system in sleep bruxism: A transcranial magnetic stimulation and brainstem reflex study. J. Orofac. Pain 2006;20(2):1-11.
11. Guaita M, Högl B. Current treatments of bruxism. Curr. Treat. Options. Neurol. 2016;18(10):2-15.
12. Hallett M. Transcranial Magnetic Stimulation: A Primer. Neuron 2007; 55(2):187-196.
13. Herrero Babiloni A, De Beaumont L, Lavigne GJ. Transcranial magnetic stimulation: Potential use in obstructive sleep apnea and sleep bruxism. Sleep Med. Clin. 2018;13(4):571-582.
14. Huang H, Song Y. H, Wang J. J, Guo Q, Liu W. C. Excitability of the central masticatory pathways in patients with sleep bruxism. Neurosci. Lett. 2014; 558:82-86.
15. 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.
16. Inghilleri M, Berardelli A, Marchetti P, Manfredi M. Effects of diazepam, baclofen and thiopental on the silent period

evoked by transcranial magnetic stimulation in humans. Exp Brain Res. 1996;109(3):467-72.

17. Kolta A, Morquette P, Lavoie R, Arsenault I, Verdier D. Modulation of rhythmogenic properties of trigeminal neurons contributing to the masticatory CPG, Progress in Brain Research. 2010; 187(1):137-148.
18. Koyano K, Tsukiyama Y, Ichiki R, Kuwata T. Assessment of bruxism in the clinic. J Oral Rehabil. 2008;35(7):495-508.
19. Lal SJ, Weber, DDS KK. Bruxism Management. 2022 Oct 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan.
20. Lavigne G, Kato T, Kolta A, Sessle B. J. Neurobiological mechanisms involved in sleep bruxism. Critical Reviews in Oral Biology and Medicine. 2003;14(1):30-46.
21. Lavigne G. J, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit. Rev. Oral Biol. Med. 2003;14(1):30-46.
22. Lavigne G. J, Rompré P. H, Montplaisir J. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. J. Dent. Res. 1996;75(1):546-552.
23. Lida T, Komiyama O, Obara R, Baad-Hansen L, Kawara M, Svensson P. Repeated clenching causes plasticity in corticomotor control of jaw muscles. Eur. J. Oral. Sci. 2014;122(1): 42-8.
24. Lobbezoo F, Ahlberg J, Glaros AG, Kato T, Koyano K, Lavigne GJ, et al. Bruxism defined and graded: an international consensus. J. Oral Rehabil. 2013;40(1):2-4.
25. Lobbezoo F, Ahlberg J, Raphael KG, Wetselaar P, Glaros AG, Kato T, et al. International consensus on the assessment of bruxism: Report of a work in progress. J. Oral Rehabil. 2018;45(11):837-844.
26. Lund, J. P.; Kolta, A., Adaption of the central masticatory pattern to the biomechanical properties of food, International Congress Series, 2005;1284: 11-20.
27. Lund, J.P., Kolta, A. Generation of the central masticatory pattern and its modification by sensory feedback. Dysphagia. 2006; 21:167-174.
28. Malick A, Strassman R. M, Burstein R. Trigeminohypothalamic and reticulohypothalamic tract neurons in the upper cervical spinal cord and caudal medulla of the rat, J. Neurophysiol. 2000;84(4):2078-112.
29. Manfredini D, Serra-Negra J, Carboncini F, Lobbezoo F. Current concepts of bruxism. Int. J. Prosthodont. 2017;30(5):437–438.
30. Melis M, Abou-Atme Y. S. Prevalence of bruxism awareness in a sardinian population. Cranio 2003;21(2):1-8.
31. Nélio V, Ângelo T, Ribeiro O A, Baptista A. Bruxism – Literature review. International Journal of Dentistry and Oral Health. 2015;1(5):1-5.
32. Ongerboer De Visser B. W., Cruccu G, Manfredi M., Koelman M.Th.J.H. Effects of brainstem lesions on the masseter inhibitory reflex: Functional mechanisms of reflex pathways. Brain. 1990;113(3):781-792.
33. Ongerboer De Visser B. W., Kuypers H. G. J. M. Late blink reflex changes in lateral medullary lesions: An electrophysiological and neuro-anatomical study of wallenberg's syndrome. Brain. 1978;101(2):285-294.
34. Pauletti G, Berardelli A, Cruccu G, Agostino R, Manfredi M. Blink reflex and the masseter inhibitory reflex in patients with dystonia. Movement Disorders 1993;8(4):495-500.
35. Priori A, Berardelli A, Inghilleri M, Accornero N, Manfredi M. Motor cortical inhibition and the dopaminergic system: Pharmacological changes in the silent period after transcranial brain stimulation in normal subjects, patients with parkinson's disease and drug-induced parkinsonism. Brain. 1994;117(2):317-23.
36. Raphael KG, Santiago V, Lobbezoo F. Is bruxism a disorder or a behaviour? Rethinking the International consensus on defining and grading of bruxism. J. Oral Rehabil. 2016;43(10):791-8.
37. Rossi S, Hallet M, Rossini P.M. Safety, ethical considerations, and application guidelines for the use of transcranial magnetic stimulation in clinical practice and research. Clinical Neurophysiology 2009;120(12):2008-2039.
38. Rossini P. M, Rossi S. Transcranial magnetic stimulation: Diagnostic, therapeutic, and research potential. Neurology, 2007;68(7):484-8.
39. Scott G, Westberg K. G, Vrentzos N, Kolta A, Lund J. P. Effect of lidocaine and NMDA injections into the medial pontobulbar reticular formation on mastication evoked by cortical stimulation in anaesthetized rabbits. Eur. J. Neurosci. 2003;17(10):2156-2162.
40. Takatori M, Nozaki S, Nakamura Y. Control of trigeminal motoneurons exerted from bulbar reticular formation in the cat. Experimental Neurology. 1981; 72(1): 122-140.
41. Tanaka S, Kogo M, Chandler S. H, Matsuya T. Localization of oral-motor rhythmogenic circuits in the isolated rat brainstem preparation, Brain Res. 1999; 6;821(1):190-199.
42. Tsuboi A, Kolta A, Chen C. C., Lund J. P. Neurons of the trigeminal main sensory nucleus participate in the generation of rhythmic motor patterns. Eur. J. Neurosci. 2003;17(2):229-239.
43. Tzvetanov P, Rousseff RT, Radionova Z. Abnormalities of masseter inhibitory reflex in patients with episodic tension-type headache. J Zhejiang Univ Sci B. 2009;10(1):52-6.
44. Udupa K, Chen R. Central motor conduction time. Handb Clin Neurol. 2013; 116: 375-86. Winocur E, Hermesh H, Littner D, Shiloh R, Peleg L, Eli I. Signs of bruxism and temporomandibular disorders among psychiatric patients. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 2007; 103(1):60-63.

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