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The Efficacy of Gabapentin versus Stabilization Splint in Management of Sleep Bruxism

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Abstract

Purpose: This study aimed to determine if the use of gabapentin is more efficacious than a stabilization splint with regard to the intensity of masseter muscle contractions and/or sleep quality for patients experiencing sleep bruxism (SB).
Materials and Methods: Twenty patients with SB participated in this clinical study. They were randomly divided into two treatment groups: stabilization splint group (n = 10) and gabapentin group (n = 10). The first polysomnographic examination was performed before the beginning of the experiment for all the participants. At the end of a 2-month period of stabilization splint therapy or gabapentin usage, a second polysomnographic recording was made.
Results: Statistically significant reductions in the number of SB episodes per hour and per night, bruxism time index, total duration of SB episodes per night and number of SB episodes in stages NR I and NR II ($p < 0.05$) were observed in both groups after treatment. Both treatments significantly reduced the mean intensity of masseter muscle contractions during SB episodes. Moreover, the participants treated with gabapentin showed a significant improvement in total sleep time, slow wave sleep (stage III), and sleep efficiency ($p < 0.05$).
Conclusions: Gabapentin could be an effective treatment modality in SBs, especially in those with poor sleep quality.

Bruxism is defined as a diurnal or nocturnal parafunctional activity that includes involuntary rhythmic or spasmodic clenching, gnashing, or grinding of teeth.[1] According to the international classification of sleep disorders,[2] sleep bruxism (SB) has been defined as "a stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep."

Some consequences of SB are tooth attrition, temporomandibular joint and muscle pain, temporal headaches, and

marital problems as a result of grinding sounds.[1,3,4] Diagnostic procedures include clinical evaluation, ambulatory monitoring sleep laboratory investigations, and other methods.[5,6] The clinical approach comprises the patient's history, orofacial examination, and tooth wear classification.[3,7]

There is no definitive treatment for bruxism. Management of SB comprises psychological, orodental, and pharmacological strategies.[3] Psychological approaches include explaining causes or exacerbating factors of SB, sleep hygiene instructions, hypnotherapy, biofeedback, and relaxation strategies.[8–10]

Orodental therapies consist of stabilization bite splints.[3] The hard occlusal splint, covering a full dental arch, is particularly useful for patients who are severe or frequent grinders.[3,11] Patient compliance with these oral devices is reduced over time, and splints may therefore be considered as bumpers that prevent tissue damage or influence oral habits.[12,13]

Pharmacologic management of SB is controversial, as different treatment strategies have resulted in suppression or exacerbation of this condition.[3,7] A wide range of sedative and muscle relaxant, serotonin related, dopaminergic, and cardioactive agents have been suggested.[3,10]

Based on the current data, central primary efferents are the major drivers of bruxism.[14,15] Therefore, centrally acting agents, which also affect the sleep structure, might be effective on SB. Considering this mechanism, the effects on SB of antiepileptic drugs such as tiagabine and gabapentin have been investigated.[16,17] However, in the absence of definitive evidence, the appropriate treatment for SB is still a matter of debate.

The objective of the present study was to compare the treatment efficacy of occlusal stabilization splint and gabapentin on SB, using polysomnographically determined outcome measures for the quantification of SB.

Materials and methods

Design and setting

This single-blind, randomized clinical trial was carried out at the Mashhad Dental School and Ebne Sina Hospital, Iran. This study was approved by the Ethics Committee of Mashhad University of Medical Sciences, and written informed consent was obtained from all participants.

Participants

At the beginning of the study, 24 patients (13 women and 11 men; mean age, 28.3 ± 7.1 years; range, 18 to 50 years) with the complaint of SB were recruited from the clinic of occlusion and prosthetic department of Mashhad Dental School. Patients who fulfilled the inclusion criteria of SB according to the international classification of sleep disorders[18] participated in this trial:

(1) The patient had a complaint of tooth grinding or clenching;
(2) One or more of the following occurred:
    (a) abnormal wear of teeth in a natural dentition,
    (b) a history of tooth grinding sounds for at least 3 nights per week,
    (c) jaw muscle discomfort;
(3) Polysomnography demonstrated both of the following:
    (a) jaw muscle activity during sleep; and
    (b) absence of associated epileptic activity.

The exclusion criteria consisted of:

(1) loss of more than two teeth and wearing a partial denture,
(2) presenting gross malocclusion,
(3) taking any medication with a known influence on sleep structure or SB (e.g., selective serotonin reuptake inhibitors, psychotropic, antidepressant, antianxiety, anticonvulsive, and analgesic drugs)
(4) being diagnosed with psychological, neurotic, or renal disorders.

Interventions

Participants were assigned to one of two treatment groups in order of admission to the study. Trial design was parallel with the allocation ratio of 1:1. In group A, a stabilization splint, a hard acrylic splint covering the maxillary dental arch, was used for treatment of bruxism. Splint fabrication and adjustment was performed according to the procedure explained by Okeson.[19] For each patient, an alginate impression was made from the maxillary arch and poured immediately with die stone. Later a 2-mm thick clear resin sheet was adapted to the cast, using a pressure adaptor (Biostar, Great Lakes Orthodontics Products, Tonawanda, NY). The outline of the appliance was then cut off the cast with a separating disk. The cut was made at the level of the interdental papilla on the buccal and labial surfaces of the teeth. The labial border terminated between the incisal and middle thirds of the anterior teeth. A large acrylic bur was used to smooth any rough edges. The appliance was then placed over the patient's maxillary teeth and evaluated for proper fitness.

To locate the condyles in their most musculoskeletally stable position (Centric Relation: CR), the bilateral manual manipulation technique was used.[19] When the CR position was carefully located, the appliance was removed from the mouth, and sufficient acrylic resin was added to the anterior and posterior regions of the occlusal surface. The appliance with the setting acrylic was returned to the mouth, and the patient was guided to close the mandible in CR. After the appliance was removed, the occlusal surface was visualized to ensure all mandibular teeth had made indentations in the acrylic.

When the appliance was adequately smoothed, it was returned to the mouth, and the CR contacts (marked by red articulating paper) were carefully checked so they occurred on flat surfaces with equal occlusal forces. Afterwards, the eccentric guidances were adjusted to produce smooth, continuous pathways. During the protrusive and laterotrusive movements, guidance by the mandibular canines was the goal.

Once the stabilization appliance was properly adjusted and polished, the patient was instructed to wear the appliance 8 to 10 hours at night and return in 2 to 7 days for evaluation. At that time, the occlusal marks on the appliance were reexamined. All patients in this group (mean age, 31.7 ± 9.2) were asked to wear the splint at night for 2 months. During this period they were followed up every week for any discomfort caused by the appliance.

In group B (mean age, 26.1 ± 5.2), patients were treated with gabapentin ([Aminomethyl] cyclohexaneacetic acid, Razak Company, Tehran, Iran). For patients' adaptation to the medication, they were given 1 capsule (100 mg) orally at bedtime for the first 3 nights. Then the dosage applied was 200 mg/night for the next 3 nights. Thereafter the dosage was increased up to total of 300 mg/night and continued for 2 months.

The first polysomnographic examination was performed before the beginning of the experiment for all participants. At the end of a 2-month period of stabilization splint therapy (in group A) or gabapentin usage (in group B), a second polysomnographic recording was made.

Polysomnographic variables

Polysomnographic all-night recordings were obtained between 10:30 pm and 6:00 am for the following clinical parameters: total sleep time (TST), non-REM sleep latency (NRSL), REM sleep latency (RSL), stages I, II, III of non-REM sleep (NR I, II, III), sleep efficiency (SE), number of SB episodes per night (N Epi./n), number of SB episodes per hour (N Epi./h), total duration of SB episodes per night (T Dur./n), bruxism time index (BTI), number of SB episodes in stage NR I (Epi./NI), number of SB episodes in stage NR II (Epi./NII), number of SB episodes in stage NR III (Epi./NIII), position of sleep (supine, right, left), maximum voluntary contraction of masseter muscle recorded before sleep (MVC), and the mean intensity of masseter muscle contractions (EMG) during SB episodes. Body mass index (BMI) was also calculated for each patient.

Statistical analysis

We performed an independent-samples *t*-test to evaluate whether participants in the two groups had significant differences in demographic variables. Independent-samples *t*-test was used to compare sleep- and bruxism-related variables in each step between groups A (stabilization splint) and B (gabapentin). We used paired samples *t*-test to compare sleep and bruxism variables of each group before and after the treatment. $P < 0.05$ was considered statistically significant.

Results

Of 24 participants admitted to this study, four were excluded. The reasons for these were that SB in two patients was not confirmed based on the first polysomnographic recordings, and two patients did not report for the second polysomnographic examination. Therefore, 20 patients including 11 women and 9 men took part in the study. In addition to SB, masticatory muscle disorders were observed in 85% of the patients, and symptoms of derangement of the condylar disk complex were reported in 15% of the participants.

Independent-samples *t*-test revealed no significant differences in demographic variables of sex ($p = 0.66$), age ($p = 0.16$), and BMI ($p = 0.45$) between the two groups (Table 1). Data in Table 2 show that at baseline, bruxism and sleep-related variables were not significantly different between the two groups, except for NR III, which was significantly greater in the gabapentin group ($p = 0.048$).

Table 1 Demographic data of patients (n = number of subjects)

Demographic data	Splint group	Gabapentin group	P values[1]
Mean age (SD)	31.7 (9.2)	26.1 (5.2)	0.16
Male *n*	5	4	
Female *n*	5	6	0.66
Mean BMI (SD)	21.9 (3.1)	20.7 (2.5)	0.45
Total *n*	10	10	

[1]Independent-samples *t*-test.

Table 2 Comparison of sleep and bruxism variables during the first (baseline) polysomnographic test between the two groups

Variables	Splint group Mean (SD)	Gabapentin group Mean (SD)	*P* values[1]
Sleep			
TST[2] [min.]	(43.2) 387	382 (40.9)	0.794
NR SL[3] [min.]	21.5 (8.4)	24.2 (14.1)	0.598
RSL[4] [min.]	112.7 (16.6)	97.9 (22.4)	0.111
NR I[5] [%]	10.8 (2.0)	11.3 (1.8)	0.578
NR II[6] [%]	51.9 (2.5)	54.1 (2.2)	0.296
NR III[7] [%]	15.7 (2.5)	18.1 (2.6)	**0.048***
SE[8] [%]	87.4 (4.6)	86.3 (4.8)	0.608
Bruxism			
N Epi./n[9]	24.7 (12.7)	21.1 (7.8)	0.454
N Epi./h[10]	3.9 (2.2)	3.4 (1.5)	0.600
T Dur./n[11] [sec]	237 (112.7)	203 (70)	0.432
BTI[12] [%]	1.12 (0.8)	0.98 (0.4)	0.381
Epi./NI[13]	12.7 (5.9)	9.4 (4.4)	0.174
Epi./NII[14]	10.7 (5.5)	10.5 (4.2)	1.000
Epi./NIII[15]	1.3 (1.8)	1.2 (0.9)	0.879
Supine SP[16] [%]	47 (10.3)	55 (12.2)	0.161
Right SP[17] [%]	30 (6.4)	29 (9.9)	0.793
Left SP[18] [%]	23 (11.9)	16 (6.1)	0.233
MVC[19] [μv]	545 (125.3)	465 (72.3)	0.080
EMG[20] [μv]	183 (39)	189 (40.3)	0.777

*$P < 0.05$.

[1] Independent-samples *t*-test, [2]Total sleep time, [3]Non REM sleep latency, [4]REM sleep latency, [5]Non REM sleep stage I, [6]Non REM sleep stage II, [7]Non REM sleep stage III, [8]Sleep efficiency, [9]Number of SB episodes per night, [10]Number of SB episodes per hour, [11]Total duration of SB episodes per night, [12]Bruxism time index, [13]Number of SB episodes in stage NR I, [14]Number of SB episodes in stage NR II, [15]Number of SB episodes in stage NR III,[16]Supine sleep position,[17]Right sleep position,[18]Left sleep position,[19]Maximum voluntary contraction of masseter muscle recorded before sleep, [20]Mean intensity of masseter muscle contractions during SB episodes.

After 2 months of treatment, sleep variables of SE and NR III in participants treated with gabapentin were significantly greater than patients who received a stabilization splint (Table 3). NRSL in the splint group was significantly greater when compared with the gabapentin group ($p = 0.031$), therefore, patients who received gabapentin could go to sleep more quickly. Likewise, NR II was significantly higher in the splint group ($p = 0.035$). Regarding bruxism variables, the percentage of bruxism episodes in the supine sleep position was greater in the gabapentin group ($p = 0.016$); however, other parameters

Table 3 Comparison of sleep and bruxism variables during the second (after treatment) polysomnographic test between the two groups

Variables[1]	Splint group Mean (SD)	After treatment Gabapentin group Mean (SD)	*P* values[2]
Sleep			
TST [min.]	380.3 (51)	411.7 (45)	0.150
NRSL [min.]	20.4 (7.9)	10.9 (9.9)	**0.031***
RSL [min.]	117.9 (28.4)	117.8 (20.1)	0.995
NR I [%]	12.4 (4.5)	10.3 (3.9)	0.272
NR II [%]	55.6 (3.1)	52.7 (2.6)	**0.035***
NR III [%]	15.9 (3.9)	20.9 (4.0)	**0.012***
SE [%]	86.7 (4.3)	93.1 (3.9)	**0.003***
Bruxism			
N Epi./n	15 (8.2)	11.3 (4.8)	0.236
N Epi./h	2.4 (1.4)	1.7 (0.8)	0.156
T Dur./n [sec]	123.2 (62.1)	93.6 (38.1)	0.215
BTI [%]	0.57 (0.31)	0.35 (0.14)	0.060
Epi./NI	7 (4.0)	5.2 (3.5)	0.301
Epi./NII	7.5 (4.4)	5.7 (1.9)	0.255
Epi./NIII	0.6 (0.7)	0.6 (0.5)	1.000
Supine SP [%]	49.3 (7.2)	57.8 (7)	**0.016***
Right SP [%]	28.7 (7.7)	24 (5.7)	0.141
Left SP [%]	22 (6.5)	18.2 (8.6)	0.278
MVC [μv]	536.5 (106.3)	457.4 (64.6)	0.060
EMG [μv]	152.6 (34)	139.7 (33.2)	0.402

[1]Please refer to abbreviations explained in Table 2.
[2]Independent-samples *t*-test.
*$P < 0.05$.

Table 4 Comparison of sleep and bruxism variables between the first (baseline) and second (after treatment) polysomnographic tests in the splint group

Variables[1]	Baseline mean (SD)	Splint group After treatment mean (SD)	*P* values[2]
Sleep			
TST [min.]	(43.2) 387	380.3 (51)	0.160
NRSL [min.]	21.5 (8.4)	20.4 (7.9)	0.526
RSL [min.]	112.7 (16.6)	117.9 (28.4)	0.632
NR I [%]	10.8 (2.0)	12.4 (4.5)	0.368
NR II [%]	51.9 (2.5)	55.6 (3.1)	**0.010***
NR III [%]	15.7 (2.5)	15.9 (3.9)	0.841
SE [%]	87.4 (4.6)	86.7 (4.3)	0.546
Bruxism			
N Epi./n	24.7 (12.7)	15 (8.2)	**0.001***
N Epi./h	3.9 (2.2)	2.4 (1.4)	**0.002***
T Dur./n [sec]	237 (112.7)	123.2 (62.1)	**0.000***
BTI [%]	1.12 (0.8)	0.57 (0.31)	**0.010***
Epi./NI	12.7 (5.9)	7 (4.0)	**0.001***
Epi./NII	10.7 (5.5)	7.5 (4.4)	**0.008***
Epi./NIII	1.3 (1.8)	0.6 (0.7)	0.226
Supine SP [%]	47 (10.3)	49.3 (7.2)	0.759
Right SP [%]	30 (6.4)	28.7 (7.7)	0.696
Left SP [%]	23 (11.9)	22 (6.5)	0.980
MVC [μv]	545 (125.3)	536.5 (106.3)	0.246
EMG [μv]	183 (39)	152.6 (34)	**0.005***

[1]Please refer to abbreviations explained in Table 2.
[2]Paired *t*-test.
*$P < 0.05$.

did not show significant differences between the two groups (Table 3).

Concerning sleep-related variables in the splint group, paired *t*-test did not show significant differences between the baseline and after-treatment polysomnographic tests, except for NR II ($p = 0.010$) (Table 4). In the gabapentin group, there was a significant increase in variables of TST, NR III, and SE, but a significant decrease in NRSL after 2 months of treatment (Table 5).

A statistically significant reduction in most bruxism variables, such as N Epi./n, N Epi./h, T Dur/N, BTI, Epi./NI, and Epi./NII was observed in both groups after treatment (Tables 4 and 5). Moreover, both groups showed a significantly reduced mean intensity of masseter muscle contractions (EMG) during SB episodes (Tables 4 and 5).

Discussion

In this clinical experiment, only patients who reported frequent and longstanding SB, verified by a roommate or family member, were selected. Their SB was confirmed by polysomnography.

In a search of the MEDLINE database, we found literature reports suggesting the effects of anticonvulsant agents on the treatment of nocturnal bruxism. A drug named tiagabine (Gabitril, Cephalon, Frazer, PA), an antiepileptic agent used for management of refractory partial seizures, has been evaluated for its effects on bruxism and temporomandibular pain.[16] Tiagabine with doses of 4 to 8 mg at bedtime could efficiently suppress nocturnal bruxism in 4 of 5 patients.[16]

Brown and Hong[17] reported a case of antidepressant-induced bruxism successfully treated with gabapentin. Gabapentin has been approved by the US Food and Drug Administration as an adjunctive therapy for control of seizures. Although gabapentin is an analog of gamma-aminobutyric acid (GABA), it is neither a GABA agonist nor antagonist, and its mechanism of action is not well-understood.[20] However, like some other anticonvulsants, it appears to block voltage-dependent sodium channels and potentiate GABA responses in the brain.[21] This agent has no known significant drug interactions and generally benign side effects (including fatigue, dizziness, somnolence, and ataxia),[20] especially at the low dosage used in the present investigation.

Previous studies have demonstrated the effects of gabapentin and other antiepileptic drugs on sleep structure.[22–26] In an add-on study of patients with epilepsy,[23] gabapentin increased REM sleep, decreased awakenings, and decreased stage I sleep, but also showed a significant improvement in slow-wave (deep) sleep.

Most hypnotics and modern antidepressants either reduce stages III to IV or leave them unchanged. Because decreased stage III to IV (deep) sleep is seen in nocturnal bruxism[27] and gabapentin increases these stages, this may be the mechanism by which gabapentin reduces nocturnal bruxism.

Table 5 Comparison of sleep and bruxism variables between the first (baseline) and second (after treatment) polysomnographic tests in the gabapentin group

Variables[1]	Baseline mean (SD)	Gabapentin group After treatment mean (SD)	*P* values[2]
Sleep			
TST [min.]	382(40.9)	411.7 (45)	**0.000***
NRSL [min.]	24.2(14.1)	10.9 (9.9)	**0.001***
RSL [min.]	97.9(22.4)	117.8 (20.1)	0.077
NR I [%]	11.3(1.8)	10.3 (3.9)	0.357
NR II [%]	54.1(2.2)	52.7 (2.6)	0.743
NR III [%]	18.1(2.6)	20.9 (4.0)	**0.002***
SE [%]	86.3(4.8)	93.1 (3.9)	**0.000***
Bruxism			
N Epi./n	21.1 (7.8)	11.3 (4.8)	**0.000***
N Epi./h	3.4 (1.5)	1.7 (0.8)	**0.000***
T Dur./n [sec]	203 (70)	93.6 (38.1)	**0.000***
BTI [%]	0.98 (0.4)	0.35 (0.14)	**0.001***
Epi./NI	9.4 (4.4)	5.2 (3.5)	**0.011***
Epi./NII	10.5 (4.2)	5.7 (1.9)	**0.001***
Epi./NIII	1.2 (0.9)	0.6 (0.5)	0.140
Supine SP [%]	55 (12.2)	57.8 (7)	0.557
Right SP [%]	29 (9.9)	24 (5.7)	0.157
Left SP [%]	16 (6.1)	18.2 (8.6)	0.657
MVC [μv]	465 (72.3)	457.4 (64.6)	0.660
EMG [μv]	189 (40.3)	139.7 (33.2)	**0.000***

[1]Please refer to abbreviations explained in Table 2.
[2]Paired *t*-test.
*$P < 0.05$.

According to the results of the present investigation, the amounts of TST, non-REM sleep stage III (deep sleep), and SE recorded were significantly increased in the gabapentin group. This is consistent with the positive effects of gabapentin on sleep quality and amount of deep sleep, reported previously.[24–26] In addition, significant reduction in NRSL in this group suggests that compared with the baseline, patients who received gabapentin spent less time in bed before falling asleep.

On the other hand, sleep-related variables were not improved in patients treated with the stabilization splint. Likewise in an investigation carried out in Belgium,[27] splint therapy did not improve sleep quality.

A significant reduction in bruxism variables, including the number of SB episodes per night, number of SB episodes per hour, total duration of SB episodes per night, bruxism time index and number of SB episodes in stages NR I and NR II was observed in both groups after treatment. The differences between the two groups concerning these variables were not statistically significant, suggesting the equivalent efficiency of these treatment strategies in controlling bruxism.

In both treatment groups of the present study, a significant reduction in the mean intensity of masseter muscle contractions during SB episodes was observed. This was not unexpected with the stabilization splint, since a number of investigators have reported a reduction in jaw muscle activity with the use of occlusal splints.[1,3,19] Nevertheless, some other studies have shown no effects.[28,29] In a qualitative systematic review, authors concluded that the bite splint may be justified when a reduction of jaw closing muscle activity (e.g., tooth grinding) is desired, or as an emergency device in patients with acute temporomandibular pain and, possibly, restricted jaw opening.[30]

In addition to its antiepileptic function, gabapentin is widely used for the treatment of neuropathic pain and is a novelty in the pharmacological management of spasticity.[20,31] In an open-label clinical and neurophysiological study, Serrao et al suggested that gabapentin would be helpful in the treatment of stable, longstanding muscular cramps associated with different diseases.[32]

It has been demonstrated that blocking presynaptic glutamate release with gabapentin effectively reduces the manifestation of muscular spasticity in patients with spinal cord injury.[33–35] This inhibition of glutamate release might be responsible for the reduction of masseter muscle contractions with gabapentin observed in the present study.

According to the findings of this experiment, both treatment modalities might be effective in the management of bruxism events. Moreover, gabapentin could improve the variables related to sleep quality. Therefore, the treatment of choice would depend on the specific clinical situation and the patient's acceptance.

Because of the small sample size of the present study, the results must be interpreted with caution. For future research and to specify an optimal duration for drug therapy, it is suggested to design clinical trials in larger populations, over longer periods, and with different dosages of gabapentin recommended in the pharmacologic literature. In addition, concomitant use of gabapentin and stabilization splint might be more helpful in the management of SB and needs to be addressed in further investigations.

Conclusions

Given this study's limitations, gabapentin was shown to be a potential effective treatment modality in sleep bruxers, especially in those with poor sleep quality.

References

1. Dawson PE: Functional Occlusion from TMJ to Smile Design (ed 3). St. Louis, Elsevier, 2007, p. 334
2. AASM: International Classification of Sleep Disorders: Diagnostic and Coding Manual (ed 2). Westchester, IL, American Academy of Sleep Medicine, 2005
3. Lavigne GJ, Manzini C, Kato T: Sleep bruxism. In Kryger MH, Roth T, Dement WC (eds): Principles and Practice of Sleep Medicine. St. Louis, Elsevier, 2005, pp. 946-959
4. Lavigne GJ, Khoury S, Abe S, et al: Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil 2008;35:476-494
5. Lavigne GJ, Rompre PH, Montplaisir JY: Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996;75:546-552
6. Lavigne GJ, Rompre PH, Poirier G, et al: Rhythmic masticatory muscle activity during sleep in humans. J Dent Res 2001;80:443-448

7. Saletu A, Parapatics S, Anderer P, et al: Controlled clinical, polysomnographic and psychometric studies on differences between sleep bruxers and controls and acute effects of clonazepam as compared with placebo. Eur Arch Psychiatry Clin Neurosci 2010;260:163-174
8. Moss RA, Hammer D, Adams HE, et al: A more efficient biofeedback procedure for the treatment of nocturnal bruxism. J Oral Rehabil 1982;9:125-131
9. Hudzinski LG, Walters PJ: Use of a portable electromyogram integrator and biofeedback unit in the treatment of chronic nocturnal bruxism. J Prosthet Dent 1987;58:698-701
10. Pierce CJ, Gale EN: A comparison on different treatments for nocturnal bruxism. J Dent Res 1988;67:597-601
11. Okeson JP: Management of Temporomandibular Disorders and Occlusion (ed 5). St Louis, Mosby, 2003, p. 428
12. Dao TTT, Lavigne GJ. Oral splints: the crutches for temporomandibular disorders and bruxism? Crit Rev Oral Biol Med 1998;9:345-361
13. Dubé C, Rompré PH, Manzini C, et al: Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. J Dent Res 2004;83:398-403
14. Kato T, Thie NMR, Huynh N, et al: Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain 2003;17:191-213
15. Lobbezoo F, Naejie M: Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001;28:1085-1091
16. Kast RE: Tiagabine may reduce bruxism and associated temporomandibular joint pain. Anesth Prog 2005;52:102-104
17. Brown ES, Hong SC: Antidepressant-induced bruxism: successfully treated with gabapentin. J Am Dent Assoc 1999;130:1467-1469
18. American Sleep Disorders Association (ASDA) Diagnostic Classification Steering Committee: The International Classification of Sleep Disorders: Diagnostic and Coding Manual, Revised. Rochester, MN, ASDA, 1997, pp. 182-185
19. Okeson JP: Management of Temporomandibular Disorders and Occlusion (ed 6). St. Louis, Elsevier, 2008, pp. 130-163
20. Martindale SS: The Complete Drug Reference (ed 35). London, Pharmaceutical Press, 2007, pp. 437-438
21. Perucca E: The new generation of antiepileptic drugs: advantages and disadvantages. Br J Clin Pharmacol 1996;42:531-543
22. Bazil CW: Effects of antiepileptic drugs on sleep structure. Are all drugs equal? CNS Drugs 2003;17:719-728
23. Placidi F, Mattia D, Romigi A, et al: Gabapentin-induced modulation of interictal epileptiform activity related to different vigilance levels. Clin Neurophysiol 2000;111:1637-1642
24. Placidi F, Diomedi M, Scalise A, et al: Effect of anticonvulsants on nocturnal sleep in epilepsy. Neurology 2000;54(5 Suppl 1): S25-32
25. Legros B, Bazil CW: Effects of antiepileptic drugs on sleep structure: a pilot study. Sleep Med 2003;4:51-55
26. Foldvary-Schaefer N, Sanchez IDL, Karafa M, et al: Gabapentin increases slow wave sleep in normal adults. Epilepsia 2002;43:1493-1497
27. Nagels G, Okkerse W, Braem M, et al: Decreased amount of slow wave sleep in nocturnal bruxism is not improved by dental splint therapy. Acta Neurol Belg 2001;101:152-159
28. Okkerse W, Brebels A, De Deyn PP, et al: Influence of a bite-plane according to Jeanmonod, on bruxism activity during sleep. J Oral Rehabil 2002;29:980-985
29. Sjöholm T, Lehtinen I, Polo O: The effect of mouth guard on masseter muscle activity during sleep. J Sleep Res 2002;11(Suppl 1):209-210
30. Stapelmann H, Turp JC: The NTI-tss device for the therapy of bruxism, temporomandibular disorders, and headache—Where do we stand? A qualitative systematic review of the literature. BMC Oral Health 2008;29:8–22
31. Lapeyre E, Kuks JB, Meijler WJ: Spasticity: revisiting the role and the individual value of several pharmacological treatments. Neurorehabil 2010;27:193-200
32. Serrao M, Rossi P, Cardinali P, et al: Gabapentin treatment for muscle cramps: an open-label trial. Clin Neuropharmacol 2000;23:45-49
33. Kitzman PH, Uhl TL, Dwyer MK: Gabapentin suppresses spasticity in the spinal cord-injured rat. Neuroscience 2007;149:813-821
34. Coderre TJ, Kumar N, Lefebvre CD, et al: Evidence that gabapentin reduces neuropathic pain by inhibiting the spinal release of glutamate. J Neurochem 2005;94:1131-1139
35. Priebe MM, Sherwood AM, Graves DE, et al: Effectiveness of gabapentin in controlling spasticity: a quantitative study. Spinal Cord 1997;35:171-175