

# Controlled Assessment of the Efficacy of Occlusal Stabilization Splints on Sleep Bruxism

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**Aims:** To assess the efficacy of occlusal stabilization splints in the management of sleep bruxism (SB) in a double-blind, parallel, controlled, randomized clinical trial. **Methods:** Twenty-one participants were randomly assigned to an occlusal splint group ( $n = 11$ ; mean age =  $34.2 \pm 13.1$  years) or a palatal splint (ie, an acrylic palatal coverage) group ( $n = 10$ ; mean age =  $34.9 \pm 11.2$  years). Two polysomnographic recordings that included bilateral masseter electromyographic activity were made: one prior to treatment, the other after a treatment period of 4 weeks. The number of bruxism episodes per hour of sleep (Epi/h), the number of bursts per hour (Bur/h), and the bruxism time index (ie, the percentage of total sleep time spent bruxing) were established as outcome variables at a 10% maximum voluntary contraction threshold level. A general linear model was used to test both the effects between splint groups and within the treatment phase as well as their interaction for each outcome variable. **Results:** Neither occlusal stabilization splints nor palatal splints had an influence on the SB outcome variables or on the sleep variables measured on a group level. In individual cases, variable outcomes were found: Some patients had an increase (33% to 48% of the cases), while others showed no change (33% to 48%) or a decrease (19% to 29%) in SB outcome variables. **Conclusion:** The absence of significant group effects of splints in the management of SB indicates that caution is required when splints are indicated, apart from their role in the protection against dental wear. The application of splints should therefore be considered at the individual patient level. *J OROFAC PAIN* 2005;19:151–158

**Key words:** bruxism, occlusal stabilization splint, polysomnography, randomized clinical trial, sleep

Sleep bruxism (SB) has classically been described by the American Sleep Disorders Association (ASDA) as a stereotyped movement disorder, characterized by grinding or clenching of the teeth during sleep.<sup>1</sup> More recently, Kato et al<sup>2</sup> after thoroughly reviewing the literature, specified that SB is a parasomnia and an oral parafunctional activity during sleep that is characterized by either jaw clenching (tonic activity) and/or repetitive phasic jaw muscle activity that produces tooth grinding.

SB is a common disorder. Its prevalence has been reported in questionnaire studies to be the highest in childhood (about 14%) and decreases from about 8% in younger adults to about 3% in elderly people.<sup>3,4</sup> The discussion about the etiology of SB encompasses peripheral factors (eg, occlusal discrepancies, orofacial morphology) and central factors such as neuropathophysiology (eg, sleep arousal, altered brain chemistry) and psychology (eg, stress, personality).<sup>2,5</sup> Nowadays, the central factors seem to be more

prominent.<sup>2,5</sup> In the clinical setting, questionnaires, oral history taking, and a clinical examination are most commonly used to establish the presence of SB.<sup>6,7</sup> The main disadvantage of these techniques, however, is that the presence of SB may be difficult to establish.<sup>7,8</sup> For that reason, for the diagnosis of SB for research purposes, the use of polysomnography is recommended.<sup>6,7,9</sup>

Bruxism is usually held responsible for clinical problems such as attrition (ie, mechanical tooth wear resulting from mastication or grinding activities)<sup>10</sup> and pain in the masticatory muscles or temporomandibular joints (ie, painful temporomandibular disorders [TMD]).<sup>6,11-13</sup> Convincing evidence for the validity of these possible causal relationships is still lacking.<sup>14,15</sup> Nevertheless, the relationships seem plausible from a clinical point of view. In bruxism research, the presence of both tooth wear and TMD should therefore be taken into account.

Treatment modalities that have been suggested to be appropriate for the management of SB include medication, sleep hygiene, relaxation, behavioral therapy, and occlusal stabilization splints.<sup>16</sup> Splints have already been used for a long time, not only for the management of SB but also for that of (painful) TMD. As for the application of splints in the management of SB, previous studies have shown variable results.<sup>16</sup> This suggests that the main effect of splints is probably limited to a protection against attrition.<sup>16</sup> However, there is a lack of well-designed, controlled, randomized clinical trials with regard to the efficacy of splints in reducing SB activities. Therefore, the aim of the present study was to evaluate the effects of splints on the frequency and duration of SB. Furthermore, since SB is also described as a parasomnia,<sup>1,2,7,17</sup> the effect of splints on sleep variables was also tested in this double-blind, parallel, controlled, randomized clinical trial.

## Materials and Methods

### Population Sample

A total of 27 participants, 8 men and 19 women, fulfilled the inclusion criteria and entered the study. Two of them canceled both appointments for sleep registrations and 2 others cancelled the second appointment for unrelated private reasons. For another 2 participants, 1 of the registrations failed due to technical problems. Thus, a total of 5 men and 16 women, with a mean ( $\pm$  SD; range) age of 34.8 years ( $\pm$  12.2; 18 to 68 years), com-

pleted the entire protocol. Four men and 7 women with a mean age of 34.2 years ( $\pm$  13.1; 21 to 68 years) entered the occlusal splint group; 1 man and 9 women with a mean age of 34.9 years ( $\pm$  11.2; 18 to 55 years) were designated the palatal splint group.

Participants were recruited from patients attending the Department of Oral Function, Section Oral Kinesiology, of the Academic Centre for Dentistry in Amsterdam (ACTA) or the Department of Clinical Neurophysiology of the Slotervaart General Hospital in Amsterdam. An announcement in a local newspaper was also used to recruit participants. Participants gave their informed consent prior to the experiment. The Medical Ethics Committee of the Slotervaart General Hospital approved the procedures.

Each participant (or his or her partner) had to confirm a recent history of tooth-grinding sounds for at least 3 nights per week during the last 6 months.<sup>9</sup> Participants had to have tooth wear to at least the degree of exposed dentine (ie, grade 2).<sup>18</sup> All participants who met these criteria, were at least 18 years of age, and signed the informed consent were included.

Excluded were all participants with a medical contraindication, such as epilepsy or any sleep disorder other than SB, or the use of any medication with a known influence on sleep structure or SB (eg, selective serotonin re-uptake inhibitors, anti-Parkinson's disease medication). Also excluded were participants who had more than 2 posterior teeth missing (except for teeth missing for orthodontic reasons and third molars), or who had a dental prosthesis. Finally, a TMD pain diagnosis was also used as an exclusion criterion.

### Experimental Procedure

To exclude a TMD pain diagnosis, all participants underwent an examination by an experienced dental practitioner (JZ) at the Clinic for Oral Kinesiology at ACTA, which consisted of a TMD examination according to the procedures suggested by the Research Diagnostic Criteria for TMD.<sup>19</sup> In addition, the presence of bruxism was clinically established by means of an inspection of the soft and hard intraoral tissues. A medical examination was performed at the Slotervaart General Hospital by an experienced neurologist (HLH).

If the participant was eligible to enroll in the study protocol, he or she underwent a first polysomnographic (PSG) recording at the hospital's sleep laboratory. After this first recording, the participants were randomly assigned using the

block randomization method<sup>20</sup> to either the occlusal splint group or the palatal splint group. A second experienced dentist (FL) inserted the splints, without mentioning the type of splint and its expected mechanism, and to deal with the patient adherence to the treatment, repeatedly emphasized to the participants the necessity of wearing their splint 24 hours a day, except during eating. After a 4-week period of splint therapy, a second sleep recording was obtained with the splint in situ.

The occlusal splint was a hard acrylic stabilization type of splint, worn in the maxilla, with full coverage of the occlusal surfaces. It had a thickness of about 1.0 mm at the level of the first molar. The palatal splint was made of the same hard acrylic resin and had palatal coverage only. After the second sleep recording, the patients were informed about the nature of their treatment. Patients with a palatal splint were offered an occlusal splint, which was accepted by all of them.

According to the hospital's protocol for sleep studies, all PSG recordings took place in a quiet, dark, single room. The recordings were performed with silver chloride surface electrodes, using a Biosaca sleep-recording unit (Ortivus). The montage protocol consisted of the following recordings:

- Electroencephalography: C<sub>3</sub>A<sub>2</sub>, O<sub>2</sub>A<sub>1</sub>
- Electromyography (EMG): Right and left masseter muscles, submental area, and right anterior tibialis muscle
- Electro-oculography: Right and left
- Oxygen saturation (Sa O<sub>2</sub>)
- Heart rate (electrocardiogram)
- Body position
- Sound (piezo-electric device)

The EMG signals of the masseter muscles were recorded with a sampling frequency of 256 Hz per channel. Hardware filters were set at 50 Hz notch, 3 Hz high pass, and 100 Hz low pass.

### Data Analysis

Sleep was analyzed in 30-second periods according to Rechtschaffen and Kales,<sup>21</sup> and the absence of sleep disorders other than SB (eg, sleep apnea, periodic limb movements in sleep) was confirmed with the use of Compumedics Pro Fusion analyzing software by a neurologist who specializes in sleep disorders (HLH) or an experienced sleep scientist (DJW). All bruxism data were analyzed by the same investigator (JZ), who was blind to the

presence or absence of an occlusal splint or a palatal splint. Total sleep time, percentage of sleep time spent in each sleep stage, sleep efficiency (%), and the number of arousals per hour of sleep (arousal index) were calculated as sleep outcome measures based on scoring rules published by ASDA.<sup>22</sup>

Bruxism analyses were performed according to the criteria of Lavigne et al,<sup>23,24</sup> and a custom-made program (JAWS v1.441+ masseter muscle analyzing software, Aalborg University)<sup>25</sup> was used at a 10% maximum voluntary contraction (MVC) threshold level.<sup>2,17</sup> The raw signals were rectified and filtered with a high pass of 3 Hz and a low pass of 10 Hz. As outcome variables for bruxism, the number of episodes per hour of sleep (Epi/h) and the number of bursts per hour of sleep (Bur/h) were determined.<sup>23</sup> In addition, the bruxism time index (BTI) was calculated. This index describes the total time spent in bruxing divided by the total sleep time and multiplied by 100%.<sup>26</sup> Only bruxism outcome variables detected during sleeping periods were used in the analyses. Events such as fragmentary myoclonuses and body movements were excluded.

### Statistical Analysis

Shapiro-Wilk tests, together with an assessment of histograms and normal QQ plots, were used to screen all variables for a normal distribution. If a non-normal distribution was found, a logarithmic transformation was performed in order to achieve a normal distribution.

To determine whether differences were present in baseline characteristics of the participants at the onset of the study, independent samples t tests were performed for differences in age and initial bruxism between the 2 groups. Similarly, chi-square tests were performed to test for differences in gender distribution between the 2 groups.

A general linear model (GLM) with repeated measures was used for further statistical analyses of a factor within the 4-week treatment period and between factors (ie, the type of splint used) as well as their interaction. If a normal distribution could not be achieved, even after logarithmic transformation, nonparametric tests (viz, Wilcoxon signed rank tests) were used.

Finally, the individual values of the bruxism outcome variables used in this study (Epi/h, Bur/h, and BTI) were also examined. A change (increase or decrease) of at least 50% with the occlusal or palatal splint was arbitrarily chosen to be a clinically significant response to the treatment. Chi-

**Table 1** Descriptive Statistics of the Standard Sleep Variables, Bruxism Variables, and BTI as Derived Before and After a 4-week Treatment Period for Both Splint Types

Variable	Before treatment		After 4-week treatment period	
	Occlusal splint	Palatal splint	Occlusal splint	Palatal splint
Sleep				
Total sleep time (min)	461.95 ± 21.12	423.70 ± 25.12	445.55 ± 29.19	436.95 ± 33.83
Sleep stage (%)				
1	6.36 ± 1.52	6.56 ± 1.03	3.66 ± 0.68	7.40 ± 2.26
2	55.51 ± 2.75	61.58 ± 1.95	60.65 ± 2.45	62.67 ± 2.61
3	8.81 ± 1.79	6.65 ± 0.78	7.52 ± 0.89	5.34 ± 0.96
4	8.47 ± 2.25	4.58 ± 1.44	6.02 ± 1.49	5.61 ± 2.04
REM	20.85 ± 1.32	20.61 ± 1.23	22.15 ± 1.35	18.96 ± 2.54
Sleep efficiency (%)	81.30   90.30   94.60	74.25   84.70   93.65	78.20   91.60   95.40	78.88   89.15   93.10
Arousal index	29.53 ± 6.38	15.30 ± 5.01	31.23 ± 5.60	18.33 ± 7.81
Bruxism				
Epi/h	6.22 ± 1.06	7.41 ± 2.27	11.11 ± 3.67	10.57 ± 4.57
Bur/h	55.37 ± 10.85	52.51 ± 14.77	99.76 ± 32.50	94.93 ± 45.10
BTI	1.36 ± 0.25	1.28 ± 0.45	2.09 ± 0.55	1.91 ± 0.82

For normally distributed variables, means ± SEM are given. One variable was not normally distributed. For that variable, the 25th, 50th (ie, the median), and 75th percentiles are given.

square tests were performed between both groups to check for any group differences.

All statistical tests were performed with SPSS 12.0 software. Probability levels of  $P < .05$  were considered statistically significant.

## Results

No baseline group differences were found regarding gender ( $\chi^2 = 2.007$ ;  $P = .157$ ), age ( $t = 0.134$ ;  $P = .895$ ), and initial bruxism outcome variables ( $0.158 \leq t \leq 0.494$ ;  $.627 \leq P \leq .876$ ).

### Sleep

All hypnograms (baseline and second PSG recordings) were judged to have a normal structure. Table 1 shows the mean values of the standard sleep variables for both the before-treatment and after-treatment conditions for both splint types. No statistically significant between-subject effects of splint type on the sleep variables were found, nor were there any significant within-subject effects on sleep of the 4-week treatment period (Table 2). Finally, no statistically significant interactions between splint type and treatment period could be observed.

### Sleep Bruxism

During all first-night (baseline) PSG recordings, the 21 participants showed sleep bruxism scores that fulfilled the research criteria for an SB diag-

nosis according to Lavigne et al.<sup>17,23</sup> The mean values of the SB outcome measures, derived before and after a 4-week treatment period with either an occlusal splint or a palatal splint, are shown in Table 1. As for the sleep variables, no significant group effects could be observed on the SB variables in regard to either splint type (between-subject) or treatment period (within-subject). Also no interaction between these 2 effects was found. However, there was a slight tendency ( $F = 3.804$ ,  $P = .066$ ; Table 2) toward a longer total duration of bruxism, as expressed by the BTI after the 4-week treatment period (before, about 1.3%; after, about 2.0%; Table 1), regardless of the type of splint.

Some of the patients showed an increase (33% to 48% of the cases) or decrease (19% to 29%) of at least 50% in their individual bruxism outcome variables, or showed no such change (33% to 48%) (Table 3). No difference was found in the number of patients with an increase or decrease between the occlusal splint and the palatal splint group ( $0.597 \leq \chi^2 \leq 1.499$ ;  $.473 \leq P \leq .742$ ).

## Discussion

The aim of the present study was to investigate the efficacy of an occlusal stabilization splint on SB and on sleep variables in a double-blind, parallel, controlled, randomized clinical trial. Despite a slight tendency toward a longer total duration of bruxism (BTI), no significant group effects of splint therapy on the SB variables were found. This corroborates

**Table 2** GLMs and Wilcoxon Signed Rank Tests of the Effects of Splint Type (Occlusal versus Palatal), Treatment Phase (Before Treatment versus After) and, in Case of a GLM, of Their Interactions on the Various Sleep Variables as Well as on Epi/h, Bur/h, and BTI

Variable	Effect	Test statistic	P
Sleep			
Total sleep time (min)	Splint type	0.459	.506
	Treatment phase	0.008	.931
	Interaction	0.678	.420
Sleep stage (%)			
1	Splint type	1.345	.260
	Treatment phase	0.593	.451
	Interaction	2.147	.159
2	Splint type	1.752	.201
	Treatment phase	3.253	.087
	Interaction	1.376	.255
3	Splint type	2.432	.135
	Treatment phase	1.742	.203
	Interaction	0.000	.992
4	Splint type	0.831	.373
	Treatment phase	0.392	.539
	Interaction	2.345	.142
REM	Splint type	0.852	.368
	Treatment phase	0.014	.906
	Interaction	1.019	.325
Sleep efficiency (%)	Splint type	-0.915	.360
	Treatment phase	-0.521	.602
Arousal index	Splint type	2.553	.127
	Treatment phase	0.818	.377
	Interaction	0.065	.802
Bruxism			
Epi/h	Splint type	0.024	.879
	Treatment phase	0.055	.817
	Interaction	0.121	.732
Bur/h	Splint type	0.074	.788
	Treatment phase	0.039	.846
	Interaction	0.003	.956
BTI	Splint type	0.035	.854
	Treatment phase	3.804	.066
	Interaction	0.017	.897

For normally distributed variables (test statistic = F), GLMs were used. For not normally distributed variables (test statistic = Z), Wilcoxon signed rank tests were used.

**Table 3** Number of Participants Showing an Increase, No Change, or a Decrease in Individual Bruxism Outcome Values for the Palatal and Occlusal Splint Groups

	Increase $\geq$ 50%			No change			Decrease $\geq$ 50%		
	Epi/h Same Other	Bur/h Same Other	BTI Same Other	Epi/h Same Other	Bur/h Same Other	BTI Same Other	Epi/h Same Other	Bur/h Same Other	BTI Same Other
Occlusal splint	4 1	4 -	4 1	3 -	3 1	3 -	3 -	3 -	3 -
Palatal splint	3 -	3 -	3 2	4 -	4 2	4 -	1 2	1 -	1 -
Total	7 1	7 -	7 3	7 -	7 3	7 -	4 2	4 -	4 -

Each number under "same" represents, per splint type (occlusal or palatal), the number of individuals who had the same degree of change (more than 50% increase, no change, or more than 50% decrease) for every bruxism outcome variable. Each number under "other" represents the number of individuals who had another degree of change for at least 1 of the bruxism outcome variables.

the conclusions of (non-PSG) studies (eg, Kydd and Daly,<sup>27</sup> Holmgren et al<sup>28</sup>). However, other authors have found significant effects (eg, Clark et al,<sup>11</sup> Okkerse et al<sup>29</sup>). For the individual bruxism variable outcomes, some patients had an increase of at least 50%, whereas others showed a decrease of 50% or more. This stresses the difference that may exist between statistical significance in group data and clinical significance on the individual level.<sup>30</sup> Finally, no influence of splint usage on the sleep variables was found. In addition, normal sleep<sup>31</sup> was not disturbed by SB. This is in accordance with most results found in the literature (eg, Reding et al,<sup>32</sup> Sjöholm et al,<sup>33</sup> Lavigne et al,<sup>17,23</sup> Lobbezoo et al<sup>34</sup>).

For the diagnosis and evaluation of bruxism, many techniques have been described in the literature, eg, the use of questionnaires, oral history data, EMG recordings, the quantification of tooth wear (either clinically or on dental study casts), or a combination of these techniques.<sup>35-38</sup> The main disadvantage of these techniques is that they do not provide a detailed insight into bruxism in relation to sleep, so that SB cannot be assessed. Therefore PSG recordings are recommended as a validated tool for the diagnosis of SB, with a good to very good sensitivity and specificity.<sup>7,23</sup> Disadvantages of PSG recordings are the difficulty of recruiting patients for sleeping in a sleep laboratory, which resulted in 4 dropouts in the current study, and an increased risk of technical failures, which resulted in 2 dropouts in the current study.

In the present study, the efficacy of stabilization splints in the management of SB was established using only 2 different points in time. In general, the efficacy of a treatment modality can be interpreted only if there is some information about the magnitude of the natural fluctuation in the outcome variables. In a study by Lavigne et al,<sup>24</sup> it was shown that the naturally occurring variability of SB over time does not result in different polysomnographically established diagnoses at different points in time. This means that the outcomes of evaluation studies to treatment modalities for SB over a certain period of time are not seriously hampered by natural fluctuation. Thus, 2 measurement times seemed sufficient. Furthermore, ethical and compliance aspects of having patients sleep for multiple nights in a sleep laboratory setting must be taken into account.

Another possible methodological shortcoming of the present study may be the small number of participants in both groups. Power analyses done on the present results show a need for at least 84 participants per group ( $\beta = 80\%$ ); even larger samples

would be needed to study discrimination between muscle contraction types (ie, phasic, tonic, and mixed episodes). However, it is difficult to recruit a sufficient number of patients to participate in sleep laboratory studies. Furthermore, the high cost of PSG recordings is a practical limitation on the total number of participants.

In many bruxism studies, a first PSG recording is used solely for habituation of the participants to sleeping under experimental conditions, while the recording of the subsequent night is used for diagnosis and research purposes (see Lavigne and Manzini,<sup>7</sup> and Kato et al<sup>2,9</sup>). In that way, the so-called first-night effect (FNE) is avoided. In the present study, 2 PSG recordings made 4 weeks apart were compared. Considering the relatively long interval separating the 2 recordings, it is likely that both can be considered first-night recordings, thus minimizing the influence of a possible FNE. As reviewed by Le Bon et al,<sup>39</sup> the FNE mainly results in a decrease in the amount of rapid eye movement (REM) sleep, total sleep time, and sleep efficiency and an increase in intermittent awakenings during the first night of sleep as compared with subsequent ones. In the present study, these sleep variables did not deviate from the normal values,<sup>31</sup> which suggests that the FNE was limited, if present at all. Nevertheless, the absence of nights for habituation can still be considered a possible confounder in the present study.

To eliminate the influence of confounders such as swallowing and sleep talking in the analysis of SB, the use of an EMG detection threshold is commonly used. In the literature, a vast range of threshold levels ranging from 10% to 40% of the MVC level are suggested.<sup>23,40-42</sup> In the present study, a threshold level of 10% MVC was used. This is in line with more recent articles, in which a tendency toward a consensus on the use of 10% MVC can be noted.<sup>2,17,43,44</sup>

Occlusal stabilization splints are widely used in the management of bruxism as well as TMD-related conditions. As reviewed in detail by Dao and Lavigne,<sup>16</sup> there is no evidence for the efficacy of splints in the management of painful TMD. They therefore suggest that splints should be used as an adjunct for TMD pain management rather than a definitive treatment. In regard to the management of SB with splints, some authors have claimed that such devices have a (transient) decreasing group effect on nocturnal EMG activity (eg, Clark et al,<sup>11</sup> Okkerse et al<sup>29</sup>). The interpretation of many previous studies, however, is hampered by some methodological problems. For

example, some studies used only EMG recordings for the quantification of bruxism (eg, Clark et al,<sup>11</sup> Solberg et al,<sup>45</sup> Sheikholeslam et al,<sup>46</sup> Hiyama et al<sup>47</sup>). Okkerse et al<sup>29</sup> did use PSG recordings for the assessment of SB and found a significant decrease of parafunctional muscle activity with the bite plane in situ. However, these authors used a different type of splint. Also, they did not use a control condition, which reduces the strength of evidence considerably.<sup>48</sup> In the present study, there were no statistically significant group effects of an occlusal or palatal splint on the bruxism outcome variables measured. As already mentioned, this may be because this study had only limited power. At a group level, it can therefore be concluded that occlusal stabilization splints and palatal splints have a similar influence on the frequency and duration of SB, viz, no changes in outcome variables over a 4-week period. This is in accordance with the conclusion by Dao and Lavigne<sup>16</sup> that the use of occlusal splints as a therapy for SB is at least questionable, except for its important role in protection against tooth wear. Furthermore, this finding is consistent with the current view that, in most cases, SB is mainly regulated centrally, not peripherally.<sup>2,5</sup>

When the efficacy of stabilization splints in the management of SB is considered at the individual level, some authors have shown a decrease, no effect at all, or even an increase in muscle activity.<sup>11</sup> Sjöholm et al<sup>49</sup> published a preliminary, but similar, report of individual increases and decreases in bruxism activity with a splint in situ. This variable effect is also in accordance with the individual findings in the present study, where some patients showed an increase in their bruxism variables greater than 50%, others showed no change, and yet others showed a decrease of more than 50%. Interestingly, no difference was found in the number of patients with an increase or decrease between the occlusal splint and the palatal splint group. This suggests that the possible effects of the therapy may be independent of the type of splint used, which may be related to similar influences of both splint types on oral behaviors such as salivation and swallowing.<sup>2</sup> In short, the use of splints in SB patients in the clinical setting should be examined at an individual level, although criteria for such a decision still need to be established. The results of this study thus indicate that caution is required when a splint is indicated in the management of SB, apart from its role in the protection against dental wear.

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## References

1. American Sleep Disorders Association (ASDA). Parasomnias. In: Thorpy MJ (ed). *International Classification of Sleep Disorders: Diagnostic and Coding Manual*. Rochester: ASDA, 1990:142–185.
2. Kato T, Thie NMR, Huynh N, Miyawaki S, Lavigne GJ. Topical review: Sleep bruxism and the role of peripheral sensory influences. *J Orofac Pain* 2003;17:191–213.
3. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: Prevalence and association among Canadians. *Sleep* 1994;17:739–743.
4. Laberge L, Tremblay RE, Vitaro F, Montplaisir JY. Development of parasomnias from childhood to early adolescence. *Pediatrics* 2000;106:67–74.
5. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 2001;28:1085–1091.
6. Lobbezoo F, Montplaisir JY, Lavigne GJ. Bruxism: A factor associated with temporomandibular disorders and orofacial pain. *J Back Musculoskeletal Rehabil* 1996;6:165–176.
7. Lavigne GJ, Manzini C. Sleep bruxism and concomitant motor activity. In: Kryger M, Roth T, Dement WC (eds). *Principles and Practice of Sleep Medicine*, ed 3. Philadelphia: Saunders, 2000:773–785.
8. Marbach JJ, Raphael KG, Janal MN, Hirschhorn-Roth R. Reliability of clinician judgements of bruxism. *J Oral Rehabil* 2003;30:113–118.
9. Kato T, Thie NMR, Montplaisir JY, Lavigne GJ. Bruxism and orofacial movements during sleep. *Dent Clin North Am* 2001;45:657–684.
10. The glossary of prosthodontic terms. *J Prosthet Dent* 1999;81:44–110.
11. Clark GT, Beemsterboer PL, Solberg WK, Rugh JD. Nocturnal electromyographic evaluation of myofascial pain dysfunction in patients undergoing occlusal splint therapy. *J Am Dent Assoc* 1979;99:607–611.
12. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. *Adv Neurol* 1988;49:329–341.
13. Holmgren K, Sheikholeslam A, Riise C, Kopp S. The effects of an occlusal splint on the electromyographic activities of the temporal and masseter muscles during maximal clenching in patients with a habit of nocturnal bruxism and signs and symptoms of craniomandibular disorders. *J Oral Rehabil* 1990;17:447–459.
14. Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? *J Orofac Pain* 1997;11:15–23.

15. Lobbezoo F, van der Zaag J, Visscher CM, Naeije M. Oral kinesiology. A new postgraduate programme in the Netherlands. *J Oral Rehabil* 2004;31:192–198.
16. Dao TTT, Lavigne GJ. Oral splints: The crutches for temporomandibular disorders and bruxism? *Crit Rev Oral Biol Med* 1998;9:345–361.
17. Lavigne GJ, Rompré PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. *J Dent Res* 2001;80:443–448.
18. Lobbezoo F, Naeije M. A reliability study of clinical tooth wear measurements. *J Prosthet Dent* 2001;86:597–602.
19. Dworkin SF, LeResche L. Research Diagnostic Criteria for Temporomandibular Disorders: Review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 1992;6:301–355.
20. Spilker B. *Guide to Clinical Trials*. New York: Raven Press, 1991.
21. Rechtschaffen A, Kales A. *A Manual of Standardized Terminology: Techniques and Scoring System for Sleep Stages of Human Subjects*. Los Angeles: UCLA Brain Information Service/ Brain Research Institute, 1968.
22. American Sleep Disorders Association. Arousals scoring rules and examples: A preliminary report from sleep disorders atlas task force of the American Sleep Disorders Association. *Sleep* 1992;15:173–184.
23. Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: Validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res* 1996;75:546–552.
24. Lavigne GJ, Guitard F, Rompré PH, Montplaisir JY. Variability in sleep bruxism activity over time. *J Sleep Res* 2001;10:237–244.
25. Rasmussen C, Nielsen KD, Arima T, et al. An automatic system for selective and standardized sleep deprivation [abstract]. *J Sleep Res* 1998;7:221.
26. van der Zaag J, Lobbezoo F, Wicks DJ, Hamburger HL, Naeije M. Self-reported jaw pain on mouth opening is associated with a reduced bruxism time index. *J Oral Rehabil* (in press).
27. Kydd WL, Daly C. Duration of nocturnal tooth contacts during bruxing. *J Prosthet Dent* 1985;53:717–721.
28. Holmgren K, Sheikholeslam A, Riise C. Effect of a full-arch maxillary occlusal splint on parafunctional activity during sleep in patients with nocturnal bruxism and signs and symptoms of craniomandibular disorders. *J Prosthet Dent* 1993;69:293–297.
29. 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.
30. Forssell H, Kalso E. Application of principles of evidence-based medicine to occlusal treatment for temporomandibular disorders: Are there lessons to be learned? *J Orofac Pain* 2004;18:9–22.
31. Carskadon MA, Dement WC. Normal human sleep: An overview. In: Kryger M, Roth T, Dement WC (eds). *Principles and Practice of Sleep Medicine*, ed 2. Philadelphia: Saunders, 1994:16–25.
32. Reding GR, Zepelin H, Robinson JE Jr, Zimmerman SO. Nocturnal teeth-grinding: All-night psychophysiological studies. *J Dent Res* 1968;47:786–797.
33. Sjöholm T, Lehtinen I, Helenius H. Masseter muscle activity in diagnosed sleep bruxists compared with non-symptomatic controls. *J Sleep Res* 1995;4:48–55.
34. Lobbezoo F, Soucy JP, Montplaisir JY, Lavigne GJ. Striatal D2 receptor binding in sleep bruxism: A controlled study with iodine-123-iodobenzamide and single photon emission computed tomography. *J Dent Res* 1996;75:1804–1810.
35. Gallo LM, Lavigne GJ, Rompré PH, Palla S. Reliability of scoring EMG orofacial events: Polysomnography compared to ambulatory recordings. *J Sleep Res* 1997;6:259–263.
36. Khan F, Young WG, Daley TJ. Dental erosion and bruxism. A tooth wear analysis from south east Queensland. *Aust Dent J* 1998;43:117–127.
37. Ahlberg J, Rantala M, Savolainen A, et al. Reported bruxism and stress experience. *Community Dent Oral Epidemiol* 2002;30:405–408.
38. Raphael KG, Marbach JJ, Klausner JJ, Teaford MF, Fischhoff DK. Is bruxism severity a predictor of oral splint efficacy in patients with myofascial face pain? *J Oral Rehabil* 2003;30:17–29.
39. Le Bon O, Staner L, Hoffmann G, et al. The first-night effect may last more than one night. *J Psychiatr Res* 2001;35:165–172.
40. Okeson JP, Philips BA, Berry DTR, Cook Y, Paesani D, Galante J. Nocturnal bruxing events in healthy geriatric subjects. *J Oral Rehabil* 1990;17:411–418.
41. Velly-Miquel AM, Montplaisir JY, Rompré PH, Lund JP, Lavigne GJ. Bruxism and other orofacial movements during sleep. *J Craniomand Dis* 1992;6:71–81.
42. Ikeda T, Nishigawa K, Kondo K, Takeuchi H, Clark GT. Criteria for the detection of sleep-associated bruxism in humans. *J Orofac Pain* 1996;10:270–282.
43. Gallo LM, Lavigne GJ, Rompré PH, Palla S. Optimization of threshold values for automated analysis of EMG recordings [abstract]. *J Dent Res* 2002;81(Special Issue A):316.
44. van Selms MKA, Lobbezoo F, Wicks DJ, Hamburger HL, Naeije M. Craniomandibular pain, oral parafunctions, and psychological stress in a longitudinal case study. *J Oral Rehabil* (in press).
45. Solberg WK, Clark GT, Rugh JD. Nocturnal electromyographic evaluation of bruxism patients undergoing short-term splint therapy. *J Oral Rehabil* 1975;2:215–223.
46. Sheikholeslam A, Holmgren K, Riise C. A clinical and electromyographic study of the long-term effects of an occlusal splint on the temporal and masseter muscles in patients with functional disorders and nocturnal bruxism. *J Oral Rehabil* 1986;13:137–145.
47. Hiyama S, Ono T, Ishiwata Y, Kato Y, Kuroda T. First night effect of an interocclusal appliance on nocturnal masticatory muscle activity. *J Oral Rehabil* 2003;30:139–145.
48. Jacob RF, Carr AB. Hierarchy of research design used to categorize the “strength of evidence” in answering clinical dental questions. *J Prosthet Dent* 2000;83:137–152.
49. Sjöholm T, Lehtinen I, Polo O. The effect of mouth guard on masseter muscle activity during sleep [abstract]. *J Sleep Res* 2002;11(suppl 1):209.