Weighing the Potential Effectiveness of Various Treatments for Sleep Bruxism

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ABSTRACT

Sleep bruxism may lead to a variety of problems, but its pathophysiology has not been completely elucidated. As such, there is no definitive treatment, but certain preventive measures and/or drugs may be used in acute cases, particularly those involving pain. This article is intended to guide clinician scientists to the treatment most appropriate for future clinical studies. To determine the best current treatment, 2 measures were used to compare the results of 10 clinical studies on sleep bruxism, 3 involving oral devices and 7 involving pharmacologic therapy. The first measure, the number needed to treat (NNT), allows several randomized clinical studies to be compared and a general conclusion to be drawn. The second measure, effect size, allows evaluation of the impact of treatment relative to a placebo using different studies of similar design. Taking into account the NNT, the effect size and the power of each study, it can be concluded that the following treatments reduce sleep bruxism: mandibular advancement device, clonidine and occlusal splint. However, the first 2 of these have been linked to adverse effects. The occlusal splint is therefore the treatment of choice, as it reduces grinding noise and protects the teeth from premature wear with no reported adverse effects. The NNT could not be calculated for an alternative pharmacologic treatment, short-term clonazepam therapy, which had a large effect size and reduced the average bruxism index. However, the risk of dependency limits its use over long periods. Assessment of efficacy and safety of the most promising treatments will require studies with larger sample sizes over longer periods.

For citation purposes, the electronic version is the definitive version of this article: www.cda-adc.ca/jcda/vol-73/issue-8/727.html

Sleep bruxism, which is characterized by grinding of the teeth or clenching of the jaw, may be associated with premature tooth wear, breakage of dental fillings, temporomandibular disorders (e.g., pain or limited movement) and temporal headache upon waking. Sleep bruxism differs from bruxism while awake, which is characterized by involuntary clenching of the teeth in reaction to certain stimuli, generally without grinding; this form is related to a tic or habit. Both types of bruxism are either primary (idiopathic), in which case there is no associated medical condition, or secondary (iatrogenic), in which case there is an associated medical condition (**Fig. 1**).[1] In addition, the tooth clenching observed during wakefulness and secondary bruxism may be associated with certain medications or drugs (e.g., neuroleptics, amphetamines or antidepressants that are selective serotonin reuptake inhibitors, cocaine, methylenedioxymethylamphetamine [ecstasy]) or certain

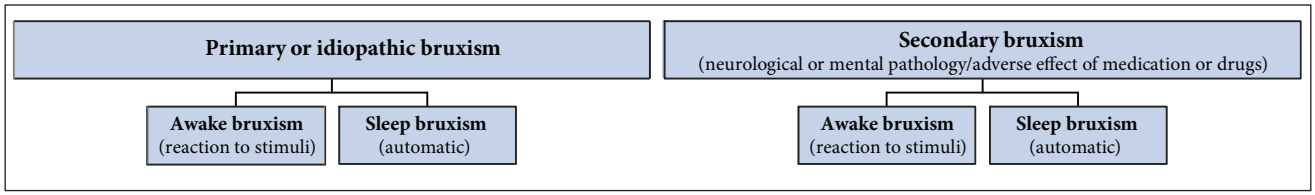

Figure 1: Hierarchical diagram of the different forms of bruxism

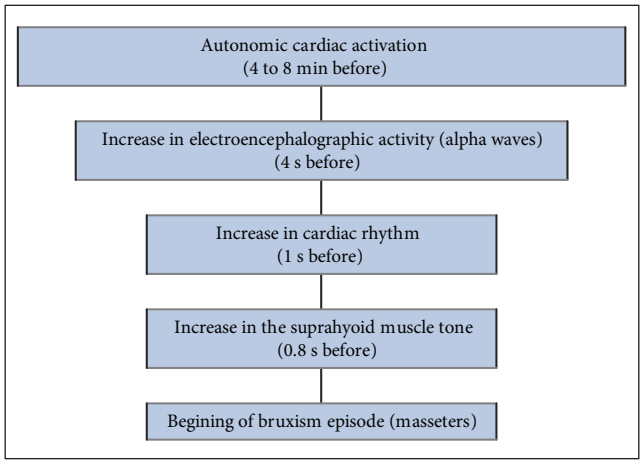

Figure 2: Sequence of physiological events preceding an episode of bruxism.

disorders (e.g., Parkinson's disease, tardive dyskinesia, depression, major anxiety). The purpose of this article is to guide clinician scientists to the treatment(s) most appropriate for future clinical studies; therefore, published works on primary bruxism and the management of sleep bruxism were selected for analysis. Elements of this analysis have already been published in a more in-depth piece[2]; the current article constitutes a useful summary for the dentist working directly with patients.

Primary Sleep Bruxism

The prevalence of sleep bruxism awareness in the general population is 8%.[1] The prevalence of this condition decreases with age, from 14% to 20% among children 11 years of age and younger, to 13% among young adults (18–29 years), to 3% among those 60 years of age and older.[1] Sleep bruxism is usually identified by a report of tooth grinding by the affected person's sleep partner. Tooth abrasion and hypertrophy of the masseter muscles are other signs that can help to confirm the occurrence of tooth grinding; however, the validity of these signs is weak, given that grinding may have taken place months before the patient's visit, and hypertrophy could be secondary to clenching habits while the patient is awake.[1] The definitive diagnosis is based principally on the report of grinding noises during sleep and pain or tension in the facial muscles upon waking. It can be confirmed by a polygraphic recording of the muscular activities of the jaws, combined, if possible, with a simultaneous audiovisual recording to exclude nonspecific orofacial events (e.g., myoclonus, tic, swallowing, somniloquy, sighing), which represent more than 30% of orofacial events during sleep.

The pathophysiology of sleep bruxism has not yet been completely elucidated, but possible causes range from psychosocial factors (e.g., stress, anxiety) to excessive response to microarousals. Microarousals are defined as brief (3–15 seconds) periods of cortical activation during sleep, which are associated with increased activity of the sympathetic nervous system.[3] Nearly 80% of bruxism episodes occur in clusters during sleep and are associated with microarousals.[3] The grinding is preceded by a sequence of physiological events: increased sympathetic activity (at 4 minutes before grinding starts), followed by cortical activation (at 1 minute before) and increased cardiac rhythm and muscle tone of the opening of the mouth (at 1 second before)[3,4] (**Fig. 2**).

There is currently no specific, definitive treatment for sleep bruxism, although various preventive measures (e.g., occlusal splint, stress management) and certain drugs (benzodiazepines, antidepressants) can be used for acute cases, particularly those involving pain.[1] In one Canadian study, an occlusal splint for the upper maxilla, worn nightly for 2 weeks, reduced the occurrence of bruxism by 40% and prevented dental abrasion.[5] However, another study suggested that this beneficial effect disappeared after 4 weeks of use.[6] Several experimental studies have been conducted to investigate pharmacologic methods to reduce sleep bruxism and to assess its neurochemical aspects.[7] Regular use of these drugs is restricted, however, because most of them induce drowsiness (e.g., benzodiazepines and tricyclic antidepressants) or carry the risk of dependency (e.g., benzodiazepines) or hypotension (e.g., clonidine).

Method and Comparison of Treatments

To determine which currently available treatment is best, the results of clinical studies of the treatment of sleep bruxism were compared. To ensure a homogeneous comparison, only published randomized, placebo-controlled, double-blind studies involving electromyography (EMG) recordings, identified through PubMed/MEDLINE, were considered (see **Appendix 1** at www.cda-adc.ca/jcda/vol-73/issue-8/727.html). Case studies and open clinical studies were not included. Some studies recorded the

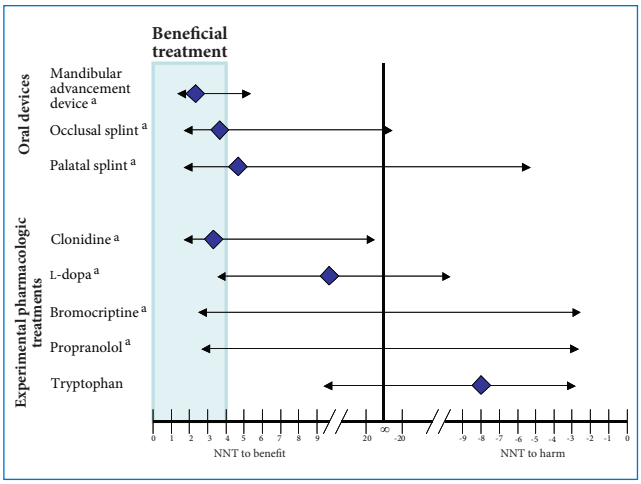

Figure 3: Number needed to treat (NNT; diamonds) calculated for studies of treatments for sleep bruxism included in the comparison. Arrows indicate 95% confidence intervals for each NNT. The shaded blue area indicates the potential benefit of the analyzed treatment.

[a]*Experimental studies conducted in the authors' sleep laboratory.*

Figure 3 is adapted from Huynh and others[2] with the permission of Quintessence Publishing Co. Inc.

EMG signal of the masseter muscles in a sleep laboratory, whereas others used portable devices.

A total of 10 studies were identified, 3 dealing with oral devices and 7 involving pharmacologic agents. Two of the 3 device studies, conducted in the authors' own laboratory, compared different device types: the occlusal splint, the palatal splint (without dental protection) and the mandibular advancement device (for snoring or light-to-moderate sleep apnea).[5,8] Data for the third study of oral devices was extracted from the published article.[6] Of the pharmacologic studies, 3 (covering bromocriptine, L-dopa, propranolol and clonidine) were conducted in the authors' laboratory[9–11] and 4 (covering clonazepam, L-tryptophan and amitriptyline) were conducted by other researchers.[12–15]

The studies were first compared in terms of the number needed to treat (NNT), which allows comparisons across randomized clinical studies and determination of an overall conclusion. The NNT is the number of patients that must receive treatment A in order that one more patient (relative to treatment B, usually placebo) will benefit (or be harmed).[16] NNT is the inverse of the absolute risk reduction. In the NNT equation, the number with improvement under treatment or placebo refers to the number of patients whose sleep bruxism index (usually expressed as number of episodes per hour) was reduced by 25% or more, n_T is the total number of patients who received the treatment and n_P is the total number of patients who received placebo. The cut-off for reduction in the sleep bruxism index was based on a previous study, in which the average variability in this index was 25.3%.[17]

$$\text{NNT} = \frac{1}{(\text{no. improved under treatment}/n_T) - (\text{no. improved under placebo}/n_P)}$$

The "NNT to benefit" ranges from 1 to infinity, whereas the "NNT to harm" ranges from –1 to negative infinity. A treatment is considered beneficial if the NNT is between 1 and 4. If there is no effect, the NNT will be infinite.

The second measure, effect size, allows evaluation of the impact of treatment relative to placebo on the basis of different studies of similar design. In practical terms, effect size is the average of the difference between the sleep bruxism index with treatment and with placebo divided by the standard deviation of this average difference.[18]

$$\text{Effect size} = \frac{\text{Average (bruxism index with treatment – bruxism index with placebo)}}{\text{Standard deviation of average difference between treatment and placebo}}$$

The effect size is categorized as small (0.2), medium (0.5) or large (0.8).[18] Therefore, the larger the effect size, the smaller the number of patients required to observe a treatment effect. The power of each study, according to sample size and effect size, was calculated with paired *t*-tests (Systat Software Inc., San Jose, Calif.).

Results of Comparison between Treatments

Taking into account the NNT, the effect size and the power of the study, the following treatments reduce sleep bruxism: mandibular advancement device, clonidine and occlusal splint (**Fig. 3;** see **Appendix 2** at www.cda-adc.ca/jcda/vol-73/issue-8/727.html). However, the first 2 of these have been linked to adverse effects (see the following section), which reduces their clinical appropriateness.[8,10] The occlusal splint is therefore the treatment of choice, as it reduces grinding noise and protects the teeth from premature wear, without substantial adverse effects. The NNT could not be calculated for an alternative pharmacologic treatment, short-term clonazepam therapy, which had a large effect size and reduced the average bruxism index[14]; however, there is a risk of dependency, which limits its potential for long-term use.

Limitations

It is important to evaluate data quality before applying NNT results to clinical decision-making. The studies available for this type of comparison have certain limitations in common, specifically, small sample sizes

(7–23 patients) and short-term duration of therapy. In addition, in some studies sleep bruxism was not confirmed by means of simultaneous polygraph and audiovisual recordings; this is important because 30% of orofacial activity during sleep is not specific for bruxism. Some pharmacologic studies did not include a period of biological wash-out between treatments to prevent carryover effect.

The methods of comparison used here yield population-level measures that cannot be directly applied to individual patients.[16] Each person's medical history, including history of sleep apnea, should also be considered. In one Canadian study, aggravation of the apnea diagnosis category was observed in 4 of 10 patients with bruxism and apnea caused by wearing an occlusal splint.[19]

A variety of adverse effects have been reported, including discomfort with the mandibular advancement device (when used for just 1 night),[8] suppression of rapid eye movement sleep with clonidine and severe symptomatic morning hypotension in 20% of people with bruxism who were taking clonidine.[10] In addition, the alternative medications analyzed here, such as benzodiazepines and, more specifically, clonazepam, can engender pharmacologic dependence and drowsiness; their use must therefore be limited to short periods, in the evening, for acute cases of bruxism, and the patient must be warned not to drive after taking the drug.

Conclusion

The studies used in this analysis were designed to investigate the possible etiology and pathophysiology of sleep bruxism, as well as to indicate the most valid therapeutic approaches. Studies with larger sample sizes over longer periods will be necessary to assess the effectiveness and safety (including adverse effects) of the best treatment(s) for reducing the consequences of tooth grinding.

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At the time of article submission, Dr. Lavigne did paid consultancy work for Respironics.

This article has been peer reviewed.

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Appendix 1 Studies of treatments for sleep bruxism included in the analysis

Treatment	Study design	Duration of treatment	Dose	*n*	Muscle recordings	Reference
Oral device						
Mandibular advancement device[a]	Randomized, controlled, crossover	1 night	NA	13	Polygraphic	Landry and others[8]
Occlusal splint[a]	Randomized, controlled, double-blind, crossover	2 weeks	NA	23	Polygraphic	Dubé and others[5] Landry and others[8]
Occlusal splint	Randomized, controlled, double-blind, parallel	4 weeks	NA	11	None	van der Zaag and others[6]
Palatal splint[a]	Randomized, controlled, double-blind, crossover	2 weeks	NA	9	Polygraphic	Dubé and others[5]
Palatal splint	Randomized, controlled, double-blind, parallel	4 weeks	NA	11	None	van der Zaag and others[6]
Experimental pharmacologic treatments						
Amitriptyline	Randomized, double-blind	1 week	25 mg	10	None	Mohamed and others[12]
Amitriptyline	Randomized, double-blind	4 weeks	25 mg	10	None	Raigrodski and others[13]
Bromocriptine[a]	Randomized, controlled, double-blind, crossover	2 weeks	1.25–7.5 mg (6 days); 7.5 mg (8 days)	7	Polygraphic	Lavigne and others[9]
Clonazepam	Controlled, single-blind	Single acute dose	1 mg	10	Polygraphic	Saletu and others[14]
Clonidine[a]	Randomized, controlled, double-blind, crossover	Single acute dose	0.3 mg	16	Polygraphic	Huynh and others[10]
L-dopa[a]	Randomized, controlled, double-blind, crossover	Single acute dose	2 × 100 mg (before bed and during the night)	10	Polygraphic	Lobbezoo and others[11]
Propranolol[a]	Randomized, controlled, double-blind, crossover	Single acute dose	120 mg	10	Polygraphic	Huynh and others[10]
Tryptophan	Randomized, double-blind	8 days	50 mg/kg	8	Ambulatory	Etzel and others[15]

NA = not applicable
[a]Experimental studies conducted in the authors' sleep laboratory
Appendix 1 was adapted from Huynh and others[2] with the permission of Quintessence Publishing Co. Inc.

Appendix 2 Measures of effectiveness of treatments for sleep bruxism determined from analysis of published studies

			Mean value of bruxism severity (SE)					
Treatment	*n*	Units for bruxism severity	With placebo	With treatment	NNT[a] (± 95% CI)	Effect size	Power of study	Reference
Oral device								
Mandibular advancement device[b]	13	Episodes/h	5.85 (0.95)	1.19 (0.44)	2.17 (1.37 to 5.25)	1.46	1.00	Landry and others[8]
Occlusal splint[b]	23	Episodes/h	5.41 (0.57)	3.97 (0.58)	3.83 (–69.41 to 1.87)	0.58	0.76	Dubé and others[5] Landry and others[8]
Occlusal splint	11	Episodes/h	NA	11.11 (3.67)	Insufficient data	0.55	0.37	van der Zaag and others[6]
Palatal splint[b]	9	Episodes/h	4.96 (0.42)	4.45 (0.63)	4.50 (–5.31 to 1.58)	0.30	0.13	Dubé and others[5]
Palatal splint	11	Episodes/h	NA	10.57 (4.57)	Insufficient data	0.28	0.12	van der Zaag and others[6]
Experimental pharmacologic treatments								
Clonidine[b]	16	Episodes/h	6.11 (0.84)	3.70 (0.91)	3.20 (1.67 to 37.25)	0.88	0.90	Huynh and others[10]
Clonazepam	10	Episodes/h	9.30 (6.50)	6.30 (3.40)	Insufficient data	0.88	0.70	Saletu and others[14]
L-dopa[b]	10	Episodes/h	7.03 (0.93)	5.56 (0.60)	10 (–11.64 to 3.50)	0.82	0.63	Lobbezoo and others[11]
Amitriptyline (4 weeks)	10	EMG activity (μV.s)	154,321.57 (223,659.03)	94,113.70 (129,344.92)	Insufficient data	0.28	0.13	Raigrodski and others[13]
Bromocriptine[b]	7	Episodes/h	9.04 (1.04)	9.63 (1.54)	∞ (–2.53 to 2.53)	0.18	0.07	Lavigne and others[9]
Amitriptyline (1 week)	10	EMG activity (μV.s/min)	1,125.53 (2,367.29)	755.64 (1,119.03)	Insufficient data	0.16	0.07	Mohamed and others[12]
Propranolol[b]	10	Episodes/h	5.36 (0.55)	6.52 (1.46)	∞ (–2.55 to 2.55)	0.12	0.06	Huynh and others[10]
Tryptophan	8	EMG activity (μV.s)	9,108.38 (2,249.36)	9,640.00 (2,354.73)	–8.00 (–2.82 to 9.60)	0.15	0.07	Etzel and others[15]

NNT = number needed to treat, CI = confidence interval, NA = not available, EMG = electromyography
[a]NNT was not calculated for studies with insufficient data (i.e., absence of baseline nights)
[b]Experimental studies conducted in the authors' sleep laboratory
Appendix 2 combines information originally published as Tables 2 and 3 in Huynh and others[2]. Adapted with the permission of Quintessence Publishing Co. Inc.