Comparison of Various Treatments for Sleep Bruxism Using Determinants of Number Needed to Treat and Effect Size

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Purpose: Sleep bruxism (SB) is associated with temporomandibular pain, headaches, tooth wear, and disruption of the bed partner's sleep. The aim of this report was to compare SB treatments from various experimental studies to guide the selection of a treatment for a large sample size study. ***Materials and Methods:*** After a literature search, randomized controlled studies of 7 pharmacologic treatments and 3 oral devices were included. The number needed to treat (NNT) was calculated from raw data from the sleep laboratory at the Hôpital du Sacré-Coeur, Montréal or from published articles when sufficient data were available. The effect size (ES) was calculated for all included studies. In the most effective treatments, the NNT ranged from 1 to 4, while a high ES was above 0.8. ***Results:*** The treatments with the best NNT and ES results were the mandibular advancement device (MAD) and clonidine. The NNT (± 95% CI) and ES were 2.2 (1.4 to 5.3) and 1.5 for the MAD, and 3.2 (1.7 to 37.3) and 0.9 for clonidine, respectively. An NNT of 3.8 (1.9 to –69.4) and an ES of 0.6 were observed with the occlusal splint, with a reduction of 42% in the SB index. NNT could not be calculated for clonazepam, although the ES was 0.9. ***Conclusion:*** Although the NNT and ES results seem to indicate that the MAD and clonidine are the most promising experimental treatments, both treatments were associated with side effects (ie, discomfort for the MAD; REM suppression and morning hypotension for clonidine). The occlusal splint and clonazepam seem to be acceptable short-term alternatives, although further longitudinal, large sample size randomized controlled trials in SB management are needed. *Int J Prosthodont 2006;19:435–441.*

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The pathophysiology of sleep bruxism (SB) is not yet fully understood. First described in 1907,[1] it is currently classified as a movement disorder of stereotypical mandibular movements, characterized by tooth grinding or jaw clenching during sleep.[2] SB may be responsible for tooth wear, temporomandibular dysfunction (eg, pain or movement limitation), headaches, and a tooth-grinding noise that can interfere with a bed partner's sleep.[2–4] The mean prevalence of tooth-grinding awareness is 8% in the general adult population.[2]

In daily practice, clinicians are required to make decisions on the most appropriate treatment for tooth grinding/SB. A variety of experimental studies have explored different SB treatments to investigate its pathophysiology and establish the best available treatment. These studies are biological investigations with common limiting factors, including a small sample size, an acute single dose of medication, or a short-term oral device treatment. Moreover, not all pharmacologic studies have a crossover design or a washout period between treatments.

The concept behind number needed to treat (NNT) is the translation of results obtained from randomized clinical trials into clinical answers and guidelines.[5,6] In order to provide a patient with the best outcome, the benefits of a treatment should be weighed against any harmful effects. The NNT method enables comparisons between various trials from which a general outcome is extracted. In other words, the NNT is the number of patients who need to receive treatment A, in comparison to treatment B (generally a placebo), for 1 patient to obtain a determined benefit or adverse outcome event.[6,7] The lower the NNT, the more beneficial the treatment. NNT is calculated from the reciprocal of the absolute risk reduction, from which the number needed to treat to benefit (NNTB) is extracted.[5,8] This method can also generate the number needed to treat to harm (NNTH) through similar calculations of adverse outcomes.[8]

Another index that helps evaluate the impact of a treatment is the effect size (ES). This compares the impact of the treatment relative to a placebo within different trials. ES is the ratio of the mean of the difference between the treatment and the placebo over the mean SD of the difference.[9] The advantage of ES is that it is easily applied to data found in the literature, while to calculate the NNT, the raw data of baseline and treatment and placebo nights are needed.

Many experimental pharmacologic trials have studied methods to reduce SB and examined its neurochemical aspects. To the authors' knowledge, the first substance reported to affect SB, L-dopa, was used for a patient with Parkinson's disease who presented secondary SB.[10] L-dopa is a precursor of dopamine, adrenaline, and noradrenaline. Studies in 1980 and 1999 linked urinary catecholamines to SB in both adults and children.[11,12] As a result, 2 independent experimental randomized crossover trials were performed with L-dopa and bromocriptine, which is a dopamine D2 agonist.[13,14] In 1996 and 1997, 2 case reports suggested that propranolol, a beta-adrenergic blocker, reduced SB.[15,16] Subsequently, an experimental randomized crossover trial was designed in the laboratory at the Hôpital du Sacré-Coeur, Montréal to study the influence of propranolol on SB.[17] The results showed that this medication did not influence SB, and thus another experimental randomized crossover trial was performed with clonidine, an alpha 2 agonist.[17]

Serotonin reuptake inhibitors—usually antidepressive medications—were reported to provoke or aggravate SB.[18,19] Independent experimental randomized crossover trials have been performed for SB using L-tryptophan and amitriptyline, a serotonin precursor and tricyclic antidepressant.[20–22] In addition, clonazepam, a benzodiazepine, was studied in SB subjects.[23] Other pharmacologic treatments are used as preventive measures, such as the botulinum toxin, which paralyzes masseteric muscles. Muscle relaxants are used as well; however, according to PubMed and Medline searches, there are no randomized controlled clinical studies published on this treatment.

Whereas pharmacologic treatments do not completely eradicate SB and can be associated with side effects, the dental splint is a commonly used preventive measure. This approach was developed in the 1940's for masticatory or temporomandibular dysfunctions, although according to PubMed and Medline, publications of experimental trials started to bloom in the early 1970s. Some of these trials were done with various dental splints to study their mechanical impact on SB.[24–26] An occlusal splint in the superior maxilla, worn at night for 2 weeks without pain by SB subjects, only decreased SB by 40%, but did eliminate the noise from tooth grinding and prevent tooth wear.[24]

Further, idiopathic SB is often associated with peripheral sensory inputs, such as dental malocclusion, and with cognitive-behavioral factors, such as stress, anxiety, or personality.[3,4] Nevertheless, there is little established evidence concerning the effect of behavioral SB treatments and hypnosis. Relaxation seems to have more effect on tooth clenching while awake than on SB.

The aim of this analysis was to help establish the status of current knowledge on putative treatments for SB and the best avenues for further investigation. The ultimate objective was *(1)* to guide future dose-dependent randomized controlled pharmacologic trials and longitudinal randomized controlled studies of oral devices, and *(2)* to assist clinicians in the decision-making process by providing comprehensive access to comparative and balanced information.

Materials and Methods

Included Studies

A literature search was performed by 2 coauthors using PubMed and Medline. Every published, experimental, randomized, double-blind study involving electromyographic (EMG) recordings with placebo and drug treatments given to SB subjects was included to allow a homogenous comparison. Some studies recorded the EMG signal along with the polygraphic sleep recordings, while others used ambulatory devices. The studies included were either found in the literature or had been conducted at the sleep laboratory at the Centre de Recherche du Sommeil, Hôpital du Sacré-Coeur, Montréal. A recent review of drugs and SB was also consulted.[19]

Excluded Studies

Case reports and open studies were excluded.

Number Needed to Treat

The NNT is the number of patients who need to be treated with a specific treatment in order for 1 patient to receive a benefit (NNTB) or harm (NNTH) compared to placebo.[7,8] The NNT was not calculated for all included trials, because raw individual data and data from baseline nights were needed. Since the studies included had a crossover design, the NNT was calculated accordingly.[7] The NNT is calculated as the reciprocal of the absolute risk difference, with N as the number of subjects whose SB index (SB episodes/hour) decreased under placebo or treatment, and n as the number of SB subjects included in the study. The value of N is calculated according to mean SB indexes decreased by over 25% between placebo and treatment, because it has been previously reported that the mean variability of the SB index over time is 25.3%.[27]

$$\text{NNT} = \frac{1}{(\text{N improved on treatment} / n) - (\text{N improved on placebo} / n)}$$

The lower the NNT value, ie, NNTB, which can range from 1 to infinite, the larger the treatment effect. NNT is considered beneficial from 1 to 4. At the opposite end of the spectrum, a negative treatment effect will translate into a negative NNT, ie, NNTH. If the treatment effect is null, then the NNT is infinite.

Effect Size

ES is another method for analyzing the effect of a treatment relative to placebo, by allowing comparisons to be made between studies with similar designs. The ES was readily calculated for all included studies, since this method is easily applied to published data. The ES is calculated as the ratio of the mean difference between treatment and placebo of the SB index relative to the SD of that difference.

$$\text{ES} = \frac{\text{Mean of difference between treatment and placebo}}{\text{SD of difference between treatment and placebo}}$$

The ES is considered as small (0.2), medium (0.5), or large (0.8).[9] The larger the ES, the smaller the sample size required to observe the treatment effect. Even though the ES is adjusted for sample size, the power of the study was also calculated based on paired *t* tests using Systat (Systat).

Results

Included Studies

An Internet search using PubMed and Medline for randomized controlled clinical studies produced a total of 10 studies for inclusion in the analysis (Table 1). Studies using the following pharmacologic SB treatments were included: bromocriptine, L-dopa, propranolol, clonidine, clonazepam, tryptophan, and amitriptyline (2 studies). Studies using the following oral devices were included: the mandibular advancement device (MAD), occlusal splints (2 studies), and palatal splints (2 studies).

Raw data were retrieved from 5 SB studies performed at the sleep laboratory at the Hôpital du Sacré-Coeur, Montréal using the following treatments: bromocriptine, L-dopa, propranolol, clonidine, and dental splints (2 studies).[13,14,17,24,25] The treatments were tested in randomized double-blind crossover trials with placebo. The doses of medication given were 1.25 mg of bromocriptine, which was gradually increased (during 6 days) to the maximum dose of 7.5 mg for 8 days (+ 20 mg domperidone); 2 doses of 100 mg of L-dopa (+ 25 mg benserazide) within 4 hours of each other; a single dose of propranolol (120 mg); and a single dose of clonidine (0.3 mg). The oral devices were worn during 2 weeks of habituation before polygraphic recordings, although the MAD was worn for only 1 night. These studies were performed over 8 years with a healthy SB population (19 to 39 years of age). A total of 38 subjects were recruited (21 women, 17 men), with some subjects participating in more than one study. Subjects were selected according to tooth-grinding history (> 3 nights/week) as confirmed by a polygraphic recording of habituation (night 1) and diagnosis (night 2). Experimental nights were nights 3 and 4. Sleep and SB variables were recorded using Harmonie software (Stellate).

Five additional independent studies were also analyzed with the available published data: clonazepam, L-tryptophan, amitriptyline (2 studies), and a dental splint.[20–23,26] In the single-blind controlled study with a single dose of clonazepam (1 mg) or placebo given half an hour before bedtime, 10 SB subjects (6 women, 4 men) were recruited for polygraphic recordings.[26] In the randomized double-blind study with 8 days of placebo or L-tryptophan (50 mg/kg body weight), 8 SB subjects were recruited (7 women, 1 man; 22 to 47 years of age). Subjects were recorded for electromyographic (EMG) activity for 8 nights of baseline, followed by 8 nights of either medication or placebo, and then crossed over.[20,21] In the randomized double-blind study with 1 week of placebo or amitriptyline (25 mg/night), 10 SB subjects were recruited (8 women, 2 men; age

Table 1 All Included Oral Devices and Pharmacologic Treatments Studies

Treatment	Study design	Treatment duration	Dose	Sample size	Reference
Oral device					
MAD*	Randomized, controlled, crossover	1 night		13	25
Occlusal splint*	Randomized, double-blind, controlled, crossover	2 wk		23	24,25
Occlusal splint	Randomized, double-blind, controlled, parallel	4 wk		11	26
Palatal splint*	Randomized, double-blind, controlled, crossover	2 wk		9	24
Palatal splint	Randomized, double-blind, controlled, parallel	4 wk		11	26
Pharmacologic treatment					
Amitriptyline	Randomized, double-blind	1 wk	25 mg	10	21
Amitriptyline	Randomized, double-blind	4 wk	25 mg	10	22
Bromocriptine*	Randomized, double-blind, controlled, crossover	2 wk	1.25–7.5 mg (6 d); 7.5 mg (8 d)	7	14
Clonazepam	Single-blind, controlled	Acute single dose	1 mg	10	23
Clonidine*	Randomized, double-blind, controlled, crossover	Acute single dose	0.3 mg	16	17
L-dopa*	Randomized, double-blind, controlled, crossover	Acute single dose	2 × 100 mg (before and during the night)	10	13
Propranolol*	Randomized, double-blind, controlled, crossover	Acute single dose	120 mg	10	17
Tryptophan	Randomized, double-blind	8 d	50 mg/kg	8	20

*Studies performed at the sleep laboratory at the Hôpital du Sacré-Coeur, Montréal.

Table 2 Mean SB Activity Data for Various Randomized Trials with Mechanical or Pharmacologic Treatments

Treatment	Sample size	Units	Placebo mean (SEM)	Treatment mean (SEM)	SB index variation ((Tx-BSL)/BSL) × 100† Placebo (%)	SB index variation ((Tx-BSL)/BSL) × 100† Treatment (%)	Reference
Oral device							
MAD*	13	Epi/h	5.85 (0.95)	1.19 (0.44)	-18.32	-85.66	25
Occlusal splint*	23	Epi/h	5.41 (0.57)	3.97 (0.58)	-18.36	-42.02	24,25
Occlusal splint	11	Epi/h	–	11.11 (3.67)	–	78.62	26
Palatal splint*	9	Epi/h	4.96 (0.42)	4.45 (0.63)	-17.39	-23.04	24
Palatal splint	11	Epi/h	–	10.57 (4.57)	–	42.65	26
Pharmacologic treatment							
Clonidine*	16	Epi/h	6.11 (0.84)	3.70 (0.91)	-11.21	-48.53	17
Clonazepam	10	Epi/h	9.30 (6.50)	6.30 (3.40)	–	–	23
L-dopa*	10	Epi/h	7.03 (0.93)	5.56 (0.60)	20.50	-3.35	13
Amitriptyline (4 wk)	10	EMG activity μV.s	154321.57 (223659.03)	94113.70 (129344.92)	–	–	22
Bromocriptine*	7	Epi/h	9.04 (1.04)	9.63 (1.54)	33.04	33.63	14
Amitriptyline (1 wk)	10	EMG activity μV.s/min	1125.53 (2367.29)	755.64 (1119.03)	–	–	21
Propranolol*	10	Epi/h	5.36 (0.55)	6.52 (1.46)	-15.40	-6.15	17
Tryptophan	8	EMG activity μV.s	9108.38 (2249.36)	9640.00 (2354.73)	0.58	2.78	20

*Studies performed at the sleep laboratory at the Hôpital du Sacré-Coeur, Montréal.
†SB index variability could not be calculated for all included studies as a result of insufficient available data.
BSL = baseline.

35 ± 12 years, mean ± SD). Subjects were given 1 week of placebo or amitriptyline and then crossed over, with a washout period of 1 week.[21] Subjects were recorded for EMG activity throughout treatment. In the randomized double-blind study with 4 weeks of placebo or amitriptyline (25 mg/night), 10 female SB subjects were recruited (age 39 ± 7 years, mean ± SD). They were given 4 weeks of placebo or amitriptyline and then crossed-over, and were recorded for EMG activity throughout treatment.[22] In the double-blind parallel controlled and randomized study, 11 SB subjects received the occlusal splint and 10 SB subjects received the palatal splint, both of which were worn for 4 weeks prior to polygraphic night recordings.[26]

Table 3 NNT and ES Calculated for Various Randomized Trials with Mechanical or Pharmacologic Treatments for SB

Treatment	Sample size	NNT†	(+) 95% CI of NNT	(-) 95% CI of NNT	ES	Power	Reference
Oral device							
MAD*	13	2.17	1.37	5.25	1.46	1.00	25
Occlusal splint*	23	3.83	1.87	-69.41	0.58	0.76	24,25
Occlusal splint	11	–	–	–	0.55	0.37	26
Palatal splint*	9	4.50	1.58	-5.31	0.30	0.13	24
Palatal splint	11	–	–	–	0.28	0.12	26
Pharmacologic treatment							
Clonidine*	16	3.20	1.67	37.25	0.88	0.90	17
Clonazepam	10	–	–	–	0.88	0.70	23
L-dopa*	10	10	3.50	-11.64	0.82	0.63	13
Amitriptyline (4 wk)	10	–	–	–	0.28	0.13	22
Bromocriptine*	7	∞	2.53	-2.53	0.18	0.07	14
Amitriptyline (1 wk)	10	–	–	–	0.16	0.07	21
Propranolol*	10	∞	2.55	-2.55	0.12	0.06	17
Tryptophan	8	-8.00	9.60	-2.82	0.15	0.07	20

*Studies performed at the sleep laboratory at the Hôpital du Sacré-Coeur, Montréal.
†NNT could not be calculated for all included studies as a result of insufficient available data.

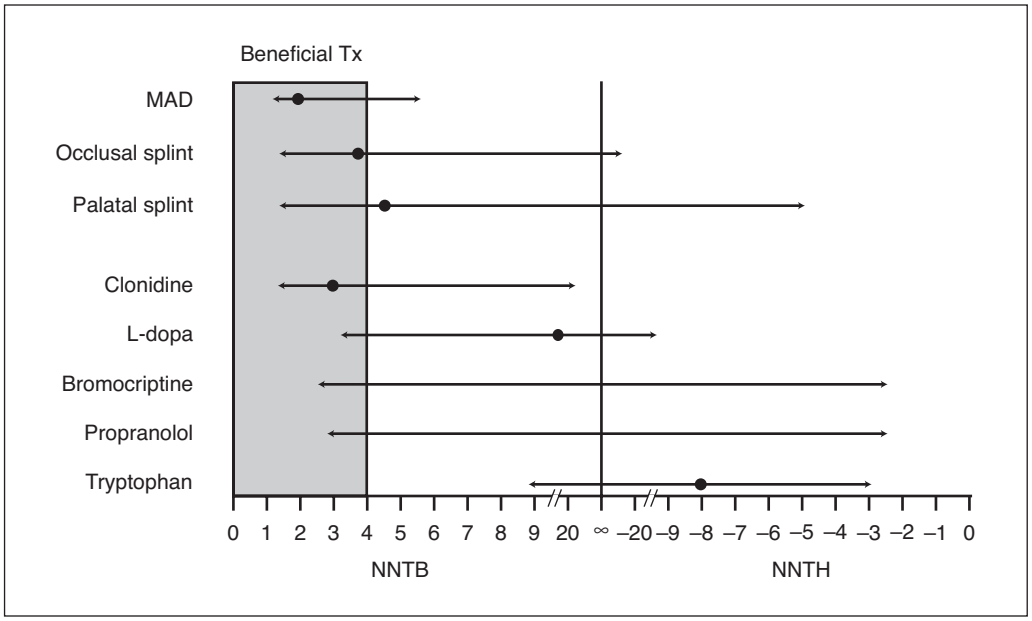

Fig 1 Forest plot of NNT calculated for various randomized crossover trials with mechanical or pharmacologic treatments for SB. The arrows show the ±95% confidence intervals.

Data Analysis

From the 6 studies completed at the sleep laboratory at the the Hôpital du Sacré-Coeur, Montréal, sleep variables[28] and oromotor/SB activity[29] were analyzed according to validated criteria. Data for the occlusal splint were pooled from 2 experimental studies performed at the sleep laboratory using oral devices, to increase the sample size and study power. In order to calculate the NNT[7,8] and ES,[9] the SB index (SB episode/hour) was used (Tables 2 and 3, Fig 1).

From the 5 published studies, various data were retrieved from the literature.[20–23,26] In the clonazepam study, EMG activity was recorded along with the polygraphic recordings.[23] The NNT for clonazepam could not be calculated, because individual and baseline night data were not available in the article. In the L-tryptophan study, EMG activity was recorded with portable EMG recorders, and the results were used to calculate the NNT and ES (Table 2).[20] In the 1-week and 4-week amitriptyline studies, recorded EMG activity was used to calculate the ES (Table 3).[21,22] In the

case of the 2 amitriptyline studies, NNT calculations could not be performed since the trial design did not include a baseline recording. In the occlusal and palatal splints study, EMG activity was recorded along with polygraphic night recordings.[26] The NNT for these oral devices could not be calculated because individual and baseline night data were not available. However, the ES was calculated from the SB data.

Number Needed to Treat

Among the pharmacologic experimental treatments, clonidine had the lowest NNT (3.2), while bromocriptine and propranolol had an infinite NNT (Fig 1, Table 2). Tryptophan had a negative treatment effect, with an NNTH of 8. Regarding the oral devices, the NNT was 2.2 for MAD, 3.8 for the occlusal splint, and 4.5 for the palatal splint (Fig 1, Table 2).

Effect Size

Among the pharmacologic experimental treatments, clonidine, clonazepam, and L-dopa had a large ES ($>$ 0.8), while all other pharmacologic treatments had a relatively small ES ($<$ 0.3) (Table 3). Regarding the oral devices, MAD had the largest ES (1.5), while the palatal splint had the lowest ES (0.28 to 0.3) (Table 3). Both studies using occlusal splints showed an ES of 0.55 to 0.58 (Table 3).

Discussion

Relative to the NNTB, ES, and power of each study, the treatments showing the greatest decreases in SB were the MAD, clonidine, and the occlusal splint. On the other hand, according to the same indexes, bromocriptine, tryptophan, and propranolol resulted in the least changes to SB.

The aim of this study was to help clarify the range and comparative efficacy of SB treatments by making a comparative index. Within our analysis, the trials included allowed a relatively homogenous comparison, since the populations studied were relatively similar. Although the MAD and clonidine treatments had the lowest NNTB, highest ES, strongest study power, and most significant reductions of SB, this does not mean they are the best SB treatments. Patients who wore the MAD for only 1 night complained of discomfort, while clonidine suppressed REM sleep and caused some symptomatic morning hypotension. Since an acute single dose of 0.3 mg was given to healthy young subjects, whereas the therapeutic dosage ranges from 0.2 mg to 0.6 mg in divided doses, further dose-dependent trials should be done with clonidine to reduce the side effects. The next best mechanical treatments seem to be the occlusal and palatal splints, although the palatal splint does not protect the teeth. As for alternative pharmacologic treatments, although the NNT could not be calculated, an acute single dose of 1 mg of clonazepam, whereas the usual initial dose is 1.5 mg/day up to maintenance doses of 8 to 10 mg/day (evening) in divided doses, had an ES of 0.9 and reduced the mean SB index from 9.3 to 6.3 per hour of sleep.[23] On the other hand, the NNTB of L-dopa was high (10), which indicates that it does little to improve SB. Bromocriptine and propranolol both had an NNT of infinity, which translates into an absence of effect, either positive or negative, on SB. These 2 medications also had a small ES and study power. This study found only 1 NNTH, which was for tryptophan.

As a general rule, the results and conclusions of a study are only as good as the experimental design and data, which is why it is important to evaluate the data before applying the NNT results to decision-making in clinical practice. This study found no published data on mechanical or pharmacologic SB treatments in large studies. Of course, not all randomized clinical studies are accessible through PubMed and Medline databanks.[30] However, because of the multicultural backgrounds of the authors of this study, the literature search included English-, French-, and Japanese-language studies. Furthermore, meta-analyses of small trials cannot substitute for large trials, and may be misleading.[31] It has been shown that in the absence of a large randomized controlled trial, the meta-analysis of small trials would lead to the implementation of an ineffective treatment 32% of the time and to the dismissal of a valuable treatment 33% of the time, when the meta-analysis and the large study do not agree.[31] Thus, the small sample size of all analyzed trials (7 to 23 subjects) is a limiting factor.

Another limitation is the trial design. Although only randomized crossover trials were included, some trials did not include polygraphic sleep recordings done in a sleep laboratory, EMG recordings analyzed with simultaneous audio-video recordings to confirm SB, habituation, and baseline recordings, or a washout period between pharmacologic treatments and placebo to avoid a carry-over effect.[13,20–22,26]

The clinical application of the results from NNTB and ES calculations to an individual patient should be attempted with caution and with full knowledge of the patient's medical history and all available alternative treatments. Although the occlusal splint is commonly used to treat SB, it should not be used for an SB patient who also suffers from sleep apnea. It was observed that in 4 of 10 SB/apnea patients, the occlusal splint caused aggravation of the diagnosis category (OSA).[32] Furthermore, the apnea-hypopnea index increased more than 50% in 5 of 10 SB/apnea patients,

and also increased snoring.[32] When the case of a patient is not comparable to the trial sample, given the inclusion and exclusion criteria, a correction factor must be added to calculate the NNT.[5] As a result, it is important to regard the NNT as a population parameter, not as an individual index.[7]

Conclusions

Clinically, the occlusal splint remains the SB treatment in which the benefits outweigh the side effects in healthy SB patients. Experimentally, the many pharmacologic studies conducted on SB subjects provide clinicians with pieces of the SB treatment puzzle and enhance the overview of SB etiology and pathophysiology, which has yet to be fully understood. Further randomized controlled trials with long durations (ie, months, years) and large sample sizes are needed for both oral devices and pharmacologic treatments.

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