Review
Management of sleep bruxism in adults: a qualitative systematic literature review

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SUMMARY This paper updates the bruxism management review published by Lobbezoo et al. in 2008 (J Oral Rehabil 2008; 35: 509–23). The review focuses on the most recent literature on management of sleep bruxism (SB) in adults, as diagnosed with polysomnography (PSG) with audio–video (AV) recordings, or with any other approach measuring the sleep-time masticatory muscles’ activity, viz., PSG without AV recordings or electromyography (EMG) recorded with portable devices. Fourteen (N = 14) papers were included in the review, of which 12 were randomised controlled trials (RCTs) and 2 were uncontrolled before–after studies. Structured reading of the included articles showed a high variability of topics, designs and findings. On average, the risk of bias for RCTs was low-to-unclear, whilst the before–after studies had several methodological limitations. The studies’ results suggest that (i) almost every type of oral appliance (OA) (seven papers) is somehow effective to reduce SB activity, with a potentially higher decrease for devices providing large extent of mandibular advancement; (ii) all tested pharmacological approaches [i.e. botulinum toxin (two papers), clonazepam (one paper) and clonidine (one paper)] may reduce SB with respect to placebo; (iii) the potential benefit of biofeedback (BF) and cognitive–behavioural (CB) approaches to SB management is not fully supported (two papers); and (iv) the only investigation providing an electrical stimulus to the masseter muscle supports its effectiveness to reduce SB. It can be concluded that there is not enough evidence to define a standard of reference approach for SB treatment, except for the use of OA. Future studies on the indications for SB treatment are recommended.

KEYWORDS: sleep bruxism, management, bruxism, treatment, oral appliances, pharmacology, biofeedback, cognitive–behavioural therapy

Accepted for publication 24 May 2015

Introduction
Bruxism is an umbrella term grouping different motor phenomena. Recently, an international expert group reached consensus to define it as follows: ‘bruxism is a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Bruxism has two distinct circadian manifestations: it can occur during sleep (indicated as sleep bruxism) or during wakefulness (indicated as awake bruxism)’ (1). Over the past few years, as part of an ongoing strategy to summarise the available findings on the argument, potential clinical consequences of bruxism have been systematically reviewed as far as its effects on the temporomandibular joints and jaw muscles as well as on natural teeth and restored implant-supported dentitions are concerned (2–4). On the other hand, evidence is gaining
weight in support of the concept that a certain amount of bruxism-related motor activities is not necessarily pathological (5, 6).

Based on that, a need emerged to define the best strategies to manage bruxism in the clinical setting. A 2008 review performed on the topic by Lobbezoo et al. (7) pointed out that most papers are inconclusive due to the low methodological quality. Consequently, along with a recommendation for designing higher-quality studies, the authors suggested that a common sense ‘triple-P’ approach, based on a combination of oral appliances (i.e. ‘plates’), counselling/behavioural strategies (i.e. ‘pep talk’) and centrally acting drugs (i.e. ‘pills’), is the most suitable strategy to manage bruxism within the current evidence of a central aetiology of the condition (7).

Since that time, knowledge on bruxism has likely been improved, especially as far as the aetiology and clinical relevance of the various bruxism-related motor phenomena are concerned. In addition, other literature reviews were performed on selected bruxism management topics (8–11). Notwithstanding that, findings still offer a fragmental picture and the need for a state-of-the-art summary has emerged.

Considering that, this paper aims to update the bruxism management review published by Lobbezoo et al. in 2008 (7), by assessing the most recent literature on the topic. In an attempt to increase the validity of our report, and given the absence of widely accepted standards for an awake bruxism diagnosis, the review focuses on the management of sleep bruxism (SB) in adults, as diagnosed with PSG or sleep-time EMG of the masticatory muscles. Based on that, all clinical trials, cohort studies (i.e. before–after case series) or before–after case reports that fit with the topic of SB management were included in the review.

Materials and methods

Search strategy

On 15 March 2015, a systematic search in the medical literature was performed to identify all peer-reviewed English language papers that were relevant to the review’s topic, viz., sleep bruxism management in adults. With the aim to be as inclusive as possible, as a first step in the search strategy, the keyword term ‘bruxism’ was used to start browsing the literature indexed in the two most qualified medical databases (i.e. National Library of Medicine’s Medline and Scopus) to retrieve lists of potentially relevant papers. The literature search was limited to papers that were added to the databases later than the date of Lobbezoo et al.’s search, viz., 28 June 2007.

Based on title and abstract assessment, the studies were selected for potential inclusion independently by two of the authors (D.M, F.L.), who also performed data extraction and quality assessment, with any disagreements resolved by discussion to reach consensus. All authors contributed to the search expansion by checking for potential additional papers in the Google Scholar database, in the reference lists of relevant papers and in their own personal databases and institutional libraries.

The criteria for admittance in the systematic review were based on the type of study, and the inclusion was restricted to clinical investigations on humans, assessing the effectiveness of any treatment approaches to SB, as diagnosed with PSG or sleep-time EMG of the masticatory muscles. Based on that, all clinical trials, cohort studies (i.e. before–after case series) or before–after case reports that fit with the topic of SB management were included in the review.

Systematic assessment of papers

The methodological characteristics of the selected papers were summarised according to a format which enabled a structured summary of the articles in relation to four main issues, viz., ‘P’ – patients/problem/population, ‘I’ – intervention, ‘C’ – comparison and ‘O’ – outcome (PICO) (12).

For each article, the study population (‘P’) was described based on the criteria for inclusion, the demographic features of the sample and the sample size. The intervention (‘I’) section included details of the management approach under investigation, along with information on the study design and the approach to SB diagnosis. The comparison criterion (‘C’) was based on the description of the control condition(s) and features of the passive or active control group(s). The study outcome (‘O’) was evaluated in relation to a brief summary of the main study’s findings.
Quality assessment of randomised controlled trials (RCTs)

The methodological quality and risk of bias of included RCTs were assessed in accordance with the Cochrane Handbook (13) and the guidelines of the Cochrane Handbook of Systematic Reviews of Interventions 4.2.6 (14).

The guidelines recommend the explicit reporting of the following individual elements for RCTs: random sequence generation and allocation sequence concealment (selection bias); blinding of participants and personnel (performance bias); blinding of outcome assessment (detection bias); completeness of outcome data (attrition bias); selective outcome reporting (reporting bias); and other sources of bias. Each item was judged as being at low (A), unclear (B) or high (C) risk of bias, based on the specifications provided below:

1 Randomisation: graded as adequate (A), unclear (B) or inadequate (C). Adequate (A) included any one of the following methods of randomisation: computer generated or table of random numbers, drawing of lots, coin-toss, shuffling cards or throw of a dice. Inadequate method of randomisation (C) utilising any of the following: case record number, date of birth or alternate numbers were judged as inadequate (quasirandomised studies).

2 Concealment of allocation: graded as adequate (A), unclear (B) or inadequate (C). Allocation concealment means that the process of allocating participants or actually placing them to the different groups to which they have been randomly assigned must be concealed from the person recruiting participants into the trial. Adequate (A) methods of allocation concealment would include either central randomisation or sequentially numbered sealed opaque envelopes. This criterion was considered inadequate (C) if there was an open allocation sequence and the participants and trialists could foresee the upcoming assignment.

3 Blinding of participants and personnel: graded as yes (A), unclear (B) or no (C). This bias arises from systematic differences in the way that care is provided or from exposure to factors other than the intervention that is being studied. Such performance bias occurs during the treatment and/or delivery of the intervention(s). It arises as the result of differences in the way that the intervention is delivered to the different study groups; that is, not only does the intervention differ between groups, the method of delivering it also differs.

4 Blinding of outcomes assessment: graded as yes (A), unclear (B) no (C). It refers to whether persons assessing the outcome of care were aware of which treatment the participant received. Such detection bias can be minimised when the outcome assessor is blind to participant groups. A lack of blinding can exaggerate the estimated effect of treatment.

5 Handling of withdrawals and losses (i.e. completeness of outcome data): graded as yes (A), unclear (B) or no (C). It is judged based on the presence of a clear description of the difference between the two treatment groups of losses to follow-up.

6 Outcome reporting: graded as adequate (A), unclear (B) or inadequate (C). It is possible that only some outcomes are included in the trial report (i.e. selective reporting), meaning that some of the outcomes have been omitted from the report and thus leading to an inadequate outcome reporting.

7 Other risk of bias: graded as yes (A), unclear (B) or no (C). This evaluation focused on the presence of any other methodological shortcomings related with the study design or SB evaluation that may have influenced the study results.

Quality assessment of uncontrolled cohort before–after studies and case series

The methodological quality of the included cohort before–after studies was assessed adopting the Critical Appraisal Skills Programme (CASP) Cohort Study Checklist (15). The CASP tool uses a systematic approach based on 12 specific questions to appraise three broad areas: an assessment of study validity, an evaluation of methodological quality and presentation of results, and an assessment of external validity. The twelve items were stated as follows:

1 Study issue is clearly focused
2 Cohort (or cases) is recruited in an acceptable way
3 Exposure (SB) is measured accurately
4 Outcome (post-treatment changes in SB variables) is measured accurately
5 Confounding factors are addressed
6 Follow-up is long and complete
7 Results are clear
8 Results are precise
Results are ‘credible’

Results can be applied to the local population

Results fit with available evidence

There are important clinical implications

Each of the questions can be answered with ‘yes’, ‘no’ or ‘can’t tell’. ‘Yes’ answers are endorsed one point, so that each study can have a maximum score of 12.

Results

Literature search outflow

The search allowed identifying 878 and 1470 citations in the Medline and Scopus databases, respectively, of which 854 were present in both databases. Thus, 1494 citations were screened for eligibility. As shown in Fig. 1, after excluding the citations that were clearly not pertinent for the review’s aim on the basis of their title and abstract (TiAb screening), 26 papers were retrieved in full text and were assessed to reach consensus as to include/exclude the papers for/from systematic assessment. Consensus decision was to exclude 12 of the 26 papers. Reasons for exclusion were the following: single case reports of patients treated for bruxism not diagnosed with PSG or EMG (N = 4) (16–19); case series of cohorts of patients treated for bruxism not diagnosed with PSG or EMG (N = 4) (20–23); studies with an absence of criteria that were used for scoring SB activity (N = 3) (24–26); and a survey paper (N = 1) (27). Thus, fourteen papers were included in the review. Of them, twelve were RCTs (28–39) and two were uncontrolled before–after studies (40, 41).

Search expansion strategies did not allow retrieving any other relevant papers, and 14 papers entered the review process.

Structured reading of papers and report of main findings

Structured reading of the included articles showed a high variability of topics and designs (Tables 1–2).

Seven papers report on the effectiveness of oral appliances (OA), either with a before–after (40) or with a RCT design (29, 32–34, 38, 39). The latter includes comparison groups treated with gabapentin (32), with palatal appliances (33), or adopting different protocols as far as the intermittent vs continuous appliance wearing (29), the different vertical dimension of occlusion (VDO) (39) and the appliance design (34, 38) are concerned. These papers account for 121 participants in total, with non-homogeneous recruitment strategies as far as the SB severity and the demographic features are concerned. Follow-up duration also varies across studies, ranging from the very few protocol days (i.e. 3–5) in a short-term crossover investigation (38) to up to 3 months in the uncontrolled before–after study (40). Similarly, the observation points are multiple, viz., more than only the baseline and end-of-treatment assessments, in a few studies only (29, 33, 39). Findings with respect to the effects of treatment protocols on SB parameters are variable and hard to interpret. The investigations comparing different OA designs and treatment regimens suggest that stabilisation splints are better than palatal splints (33); an intermittent use is superior to continuous wearing (29); a 3 mm increase in VDO is more effective than a 6 mm increase (39); a mandibular advancement appliance (MAA) with a robust advancement (75%) is superior to less marked (25%) advancement devices (38); and the restriction of mandibular movements with oral appliances does not have any major influence on jaw-muscle activity during sleep (34). Stabilisation appliances are equally effective as the neuroleptic drug gabapentin, which is only slightly superior to reduce SB events in subjects with poor sleep quality (32). The before–after study concludes that a MAA providing a 50–75% advancement significantly decreases the number of SB episodes (40).

Four papers report on pharmacological management of SB. Two of them deal with botulinum toxin injections of the jaw muscles, either in a controlled (36) or in an uncontrolled setting (31). Two other papers had a crossover design, assessing the effectiveness of clonazepam (37) or clonidine (35) with respect to placebo. In total, 90 subjects took part in those studies. In general, the findings from botulinum toxin studies are supportive of its effectiveness to reduce the intensity of SB episodes, but not their frequency (31). Follow-up assessments are provided for up to 12 weeks, thus not allowing to draw conclusions on the duration of those effects. The two placebo-controlled crossover studies have an observation period limited to the three nights of the protocol regime and suggest that both the benzodiazepine clonazepam and the antihypertension drug clonidine may have SB-reducing effects (35, 37).
Two papers deal with sleep hygiene and relaxation techniques, as compared to untreated subjects (28) and with the effects of wake-time EMG biofeedback on SB parameters (30). The total number of participants amounted to 29 individuals, with very different age ranges. The studies have similar duration (3–4 weeks) and have two and three observation points, respectively. None of them followed up patients after the end of treatment. Findings suggest that a wake-time EMG-based biofeedback program aiming to reduce awake bruxism may also reduce SB events (30). A protocol comprising teaching of sleep hygiene measures as well as muscle relation techniques is not effective to reduce SB (28).

The remaining paper reports an uncontrolled series of ten patients receiving electrical stimuli to the masseter muscles (41). The protocol provided a three-night EMG recording under three conditions, viz., one without electrical stimuli vs two nights with stimuli provided at two different sensation thresholds immediately after the heart rate exceeded 110%. Findings are suggestive of the effectiveness of such electrical stimuli to suppress SB.

Quality assessment

Quality assessment of RCTs shows that, on average, the risk of bias was low-to-unclear for all the reviewed studies (Tables 3–4). Risks of bias are mainly related with the unclear report of the strategies that were adopted to blind outcome evaluations (91.6%) as well as with the unclear report of randomisation procedures and allocation concealment (66.6%). Other potential risks of bias are related with the EMG-only SB diagnosis that was adopted in 41.6% of studies, the measurement of one-night effect only (25%) and the failure to include multiple (i.e. more than two) observation points (25%). The impact of such sources of risk has been judged as ‘unclear’.

Quality assessment of the two before–after studies shows several methodological limitations, mainly due to the adoption of very small-sized convenience study samples and the failure to include multiple observation points.

Discussion

Bruxism is a phenomenon of growing interest for many specialists. Based on the need for providing evidence-based knowledge, a series of systematic literature reviews on various bruxism topics has recently been published (1–4, 42, 43). Notwithstanding that, it seems that information on the management of bruxism is still fragmental, as suggested by the common sense suggestions recommended by Lobbezoo et al. (7) in a paper summarising the principles for the management of bruxism. The present review focused on the recent literature on SB management, viz., published
Table 1. Features of the reviewed studies based on PICO-like structured reading. RCTs

<table>
<thead>
<tr>
<th>Study first author, year</th>
<th>Population (P)</th>
<th>Intervention (I)</th>
<th>Comparison (C)</th>
<th>Outcomes (O)</th>
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<tr>
<td>Valiente, 2015 (28)</td>
<td>$N = 16$ participants (8M,8F; m.a. 39-9 ± 10-8 years; range 24–62) with recent history of TG sounds for at least three nights per week during the last 6 months and grade 2 tooth wear</td>
<td>Test group (4M,4F): sleep hygiene instructions and Jacobson’s relaxation techniques (20-min CD recorded by a psychologist) 4-week protocol PSG Two observation points (baseline and 4 weeks)</td>
<td>Control group (4M,4F): information on the condition of SB</td>
<td>For both the control group and the experimental group, no significant differences could be observed between the PSG-SB outcome measures obtained before and after the 4-week period</td>
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<td>Matsumoto, 2015 (29)</td>
<td>$N = 20$ bruxers (9M,11F; m.a.28-9 years; range 24–37) from University students and staff, diagnosed with clinical/anamnestic American Academy of Sleep Medicine (AASM) criteria</td>
<td>Test group (C): continuous use of SA covering the occlusal surfaces of the maxillary dental arch during sleep 29-night protocol EMG activity of the masseter muscle on one side (portable EMG recording unit) Six observation points (week 1, 2, 3)</td>
<td>Control group (I): intermittent use of SA (every other week, that is, at the 1st to 7th, 15th to 21st and 29th nights)</td>
<td>The intermittent use of stabilisation splints may reduce SB activity for a longer period compared with that of continuous use</td>
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<td>Sato, 2015 (30)</td>
<td>$N = 13$ male subjects (m.a. 26-8 ± 2.5 years; range, 22–31) with subjective awareness of AB</td>
<td>Test group (BF, $n = 7$): auditory BF alert signals to remind the subjects of clenching were generated during the daytime 3-week protocol One-channel portable EMG-BF device (2-day, 5-h EMG recording periods during the daytime and sleeptime) Three observation points (week 1, 2, 3)</td>
<td>Control (CO) group ($n = 6$): only EMG recordings</td>
<td>The number of tonic EMG events during sleep in the BF group significantly decreased in weeks 2 and 3, whereas that in the CO group did not show any significant change throughout the recording period EMG-BF to improve AB tonic EMG events can also provide an effective approach to the regulation of SB tonic EMG events</td>
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<td>Shim, 2014 (31)</td>
<td>$N = 24$ subjects (10M,14F; a.r. 20-2–38-7 years) with a clinical diagnosis of SB4/24 dropouts</td>
<td>Group A: 10 subjects receiving bilateral BTX-A injections (25 U per muscle) into the masseter muscles only PSG Two observation points (baseline and 4 weeks)</td>
<td>Group B: 10 subjects receiving the injections into both the masseter and temporalis muscles</td>
<td>BTX-A injection did not reduce the frequency, number of bursts, or duration for RMMA episodes in the two groups. The injection decreased the peak amplitude of EMG burst of RMMA episodes in the injected muscles ($P &lt; 0.001$, repeated measure ANOVA) in both groups.</td>
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<tr>
<td>Study first author, year</td>
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<td>Madani, 2013 (32)</td>
<td>N = 24 patients (11M, 13F; m.a. 28.3 ± 7.1 years, range 18–50) with complaint of SB (ICSD criteria)</td>
<td>Group A (m.a. 31.7 ± 9.2 years): hard SS covering the maxillary dental arch 2-month protocol PSG Two observation points (baseline and 2 months)</td>
<td>Group B (m.a. 26.1 ± 5.2 years): gabapentin – 1 capsule (100 mg) orally at bedtime for the first 3 nights, then 200 mg/night for the next 3 nights, thereafter 300 mg/night continued for 2 months</td>
<td>Significant reduction in most SB variables in both groups after treatment</td>
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<td>Takahashi, 2013 (33)</td>
<td>N = 23 (11M, 12F; m.a. 22.2 years) healthy volunteers</td>
<td>Test group: SS covering the occlusal surfaces of the maxillary dental arch Crossover design with two weeks washout between phases One-channel EMG Three 3-day observation points</td>
<td>Control group: PS not covering the maxillary teeth</td>
<td>The number of MMA events per hour decreases significantly with SS</td>
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<td>Arima, 2012 (34)</td>
<td>N = 11 subjects (4M, 26.3 ± 3.2 years; 7F, 25.9 ± 3.1 years) with self-reported SB</td>
<td>Test group: restrict-MMOA that prevented from performing mandibular movements 30-night protocol Crossover design with one of the three types of appliances (1 week each) Bilateral masseter home-EMG Six observation points (nights 1, 2, 3, 16, 23 and 30)</td>
<td>Control group: free-MMOA that allowed normal mandibular movements; or free-MOA Bilateral masseter EMG</td>
<td>The total number of phasic EMG episodes and bursts per hour of sleep is significantly reduced during any of the three combinations of oral appliances when compared with baseline values. The restriction of mandibular movements with oral appliances does not have any major influence on jaw-muscle activity during sleep</td>
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<td>Carra, 2010 (35)</td>
<td>N = 16 SB subjects (6M, 10F; m.a. 24.5 years; range 21–31)</td>
<td>Test group: single dose of clonidine (0.3 mg by mouth) 1 h before bedtime 4-night protocol PSG Crossover design</td>
<td>Control group: single dose of placebo</td>
<td>RMMA/SB decreases under clonidine</td>
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<td>Lee, 2010 (36)</td>
<td>N = 12 subjects (7M, m.a. 25 ± 2.3 years; 5F, m.a. 24.8 ± 0.8 years) with nocturnal bruxism (unspecified criteria)</td>
<td>Test group (3M,3F; 25.0 ± 2.2 years): BTX-A into each subject’s masseter muscles at three sites – 80U of BTX-A 12-week observation EMG of both masseter and temporalis muscles for three consecutive nights at home for an average of 6 hrs per night Four observation points (baseline, 4, 8, 12 weeks)</td>
<td>Control group (4M,2F; 24.8 ± 1.4 years): saline injection into each subjects’ masseter muscles at three sites – 0.8 ml of saline</td>
<td>The injection of botulinum toxin in the masseter muscle reduces the number of bruxism events during sleep for up to 12 weeks</td>
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later than Lobbezoo et al.’s review, providing both a qualitative assessment and a structured overview of included papers.

Findings suggest that the number of recent papers on the argument that adopted an objective SB evaluation (i.e. measurement of actual masticatory muscle activity by means of PSG or EMG), even if higher than that in Lobbezoo et al.’s review (7), is still scarce. Such finding is in line with other reviews on selected bruxism management topics (8–11). In an attempt to be as comprehensive as possible, inclusion in the review was tentatively open also to before–after case series and case reports, in addition to RCTs. Notwithstanding that, we retrieved only twelve RCTs and two uncontrolled before–after studies (i.e. case series) that were eligible for inclusion. The included papers cover a wide variety of management strategies, and the lack of between-study homogeneity as far as the study design is concerned prevented us from performing any meta-analysis of data.

Quality assessment of the reviewed RCTs shows the methodology is generally acceptable, even if some areas with potential risk of bias seem to be common to several investigations. In particular, strategies for randomisation and allocation concealment are unclear in two-thirds of the studies. In addition, the adoption of only two observation points (i.e. baseline and end-of-treatment assessments) as well as the single-channel masseter recordings without full audio–video PSG evaluation are other factors that may have influenced results of several papers. As for the before–after studies, they are very small-sized and include only two observation points, thus having potential limitations in terms of their external validity. However, the choice to include only papers in the review with a definite sleep bruxism diagnosis (i.e. PSG) or its best available alternative (i.e. sleep-time EMG adopting criteria for SB activity) has likely resulted in an acceptable internal validity of the reviewed investigations (1).

Table 1. (continued)

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<tr>
<th>Study first author, year</th>
<th>Population (P)</th>
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<tr>
<td>Saletu, 2010 (37)</td>
<td>N = 21 drug-free middle-aged patients (10M,11F; m.a. 45.1 ± 12.6 years) with SB (ICSD criteria)</td>
<td>Test group: crossover study, with three consecutive (pre-drug night, placebo night and clonazepam -1 mg night) PSG</td>
<td>Control group: 21 sex and age-matched subjects without SB Non-randomised study</td>
<td>The bruxism index is significantly improved under 1 mg clonazepam (41% improvement with respect to placebo on individual change values)</td>
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<td>Landry, 2009 (38)</td>
<td>N = 12 moderate to severe SB (3M,9F; m.a. 26 ± 1.5 years)</td>
<td>Test group: MAA (25% or 75% advancement) 5-night crossover PSG</td>
<td>Control group: MOS</td>
<td>MAA are more effective than MOS to reduce SB The short-term use of a robust MAA (75%) is associated with SB decrease</td>
</tr>
<tr>
<td>Abekura, 2008 (39)</td>
<td>N = 12 healthy volunteers (4M, m.a. 25.3 years)</td>
<td>Test group: Occlusal splints at 3 mm VDO increase worn for two nights Crossover design with more than 6 nights washout between phases One-sided masseter and temporalis muscle EMG</td>
<td>Comparison group: Occlusal splints at 6 mm VDO increase worn for two nights</td>
<td>Splint with 3 mm increase in VDO is superior to 6 mm-splint in decreasing bruxism</td>
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M, males; F, females; m.a., mean age; a.r., age range; TG, tooth grinding; SA, stabilisation appliance; AB, awake bruxism; BF, biofeedback; BTX-A, botulinum toxin-A; U, unit; RMMA, rhythmic masticatory muscle activity; ANOVA, analysis of variance; ICSD, International Classification of Sleep Disorders; SS, stabilisation splint; PS, palatal splint; MMA, masticatory muscle activity; MMOA, maxillary and mandibular oral appliance; MOA, maxillary oral appliance; MAA, mandibular advancement appliance; MOS, mandibular occlusal splint; VDO, vertical dimension of occlusion.
As for the results, it seems that all tested pharmacological approaches [i.e. botulinum toxin (two papers) (31, 36), clonazepam (one paper) (37) and clonidine (one paper) (35)] may reduce SB with respect to placebo. Botulinum toxin’s effects are not surprising, and they are in line with the expected pharmacological properties of the drug. However, the fact that both studies on the argument show a reduced intensity, but not frequency, of SB episodes suggests that peripherally acting drugs do not affect the genesis of SB episodes (31, 36). Such findings are in line with clinical investigations showing that improvement in muscle pain levels after botulinum toxin injection is not unequivocally superior to placebo (44, 45) or to physiotherapy (46). On the other hand, centrally acting drugs, such as the benzodiazepine clonazepam (37) and the antihypertension clonidine (35), are both effective in reducing SB frequency. The effects of clonazepam, which has sedative and muscle relaxant properties, were to a certain extent predictable, whilst the actual action mechanism of clonidine is yet to be clarified. One hypothesis is that, as clonidine is a selective α2-agonist with sympatholytic effect and activation of the sympathetic autonomic nervous system precedes bruxism events, such medication probably interrupts the cascade of events that result in bruxism episodes (35, 47).

On the other hand, the two papers on the potential benefit of biofeedback (BF) and cognitive–behavioural (CB) approaches to SB management are not supportive of their effectiveness (28, 30). Such findings are in contrast with early reports of positive effects associated with several BF and CB approaches, which led Lobbezoo et al. (7) to include ‘pep talk’ (i.e. counselling strategies) as part of a common sense approach to bruxism management. Notwithstanding that, given the relative safety and non-harmful nature of such approaches, it seems prudent to recommend their inclusion in any SB treatment protocol to maximise the effects of any multimodal approach, even if not effective as stand-alone therapies.

Studies on the effectiveness of OA provide interesting findings as well. Despite the variability in their study design, some general remarks can be suggested on the topic. First, it seems that almost every type of OA is somehow effective to reduce SB activity. This may suggest the existence of a potential ‘novelty-effect’ associated with the use of an OA, which leads to a reduction in sleep-time masticatory muscles’ activity, possibly due to the transient need for reorganising motor unit recruitment. This hypothesis may find support in the observation that intermittent OA use is more effective to reduce SB than continued use (29). However, the actual existence, clinical meaning and duration of this effect should be assessed in future studies with longer follow-up time spans. Second, it seems that OA that are designed to provide a high extent of mandible advancement (50–75%) are effective to reduce SB (38, 40). Such findings may be explained with the reduced contractile properties of masseter muscles when the mandible is advanced (48) and/or with the elimination of the amount of SB-like motor phenomena that are actually part of an apnoea-induced arousal (5). The former hypothesis contrasts with the reported lower effectiveness of OA with a markedly increased VDO (i.e. 6 mm) with respect to 3 mm-thick OA (39), as an increase in VDO is actually expected to reduce the contractile capability and efficiency of jaw-closing muscles (48). Thus, the potential mechanisms of action through which OA may reduce SB are yet to be explored in detail.

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<th>Intervention (I)</th>
<th>Comparison (C)</th>
<th>Outcomes (O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mainieri, 2014 (40)</td>
<td>19 subjects (9M,11F; m.a. 39.9 ± 12.9 years) with clinical SB</td>
<td>MAD for 3 months; 50–75% advancement</td>
<td>No control group</td>
<td>33.7% reduction in EMG episodes per hour</td>
</tr>
<tr>
<td>Sumiya, 2014 (41)</td>
<td>10 subjects (6M,4F; m.a. 26.7 ± 3.5 years) with SB awareness</td>
<td>BF (masseter EMG stimulation after heart rate increase)</td>
<td>No control group</td>
<td>Electrical stimulation can reduce the number of SB events</td>
</tr>
</tbody>
</table>

MAD, mandibular advancement device; BF, biofeedback.
Table 3. Quality assessment of RCTs based on the Cochrane Handbook of Systematic Reviews of Interventions. Reasons for risk of bias are provided

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Valiente, 2015 (28)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only two observation points)</td>
</tr>
<tr>
<td>Matsumoto, 2015 (29)</td>
<td>Low</td>
<td>Low</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (no PSG – only EMG diagnosis)</td>
</tr>
<tr>
<td>Sato, 2015 (30)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only two observation points)</td>
</tr>
<tr>
<td>Shim, 2014 (31)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only two observation points)</td>
</tr>
<tr>
<td>Madani, 2013 (32)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only two observation points)</td>
</tr>
<tr>
<td>Takahashi, 2013 (33)</td>
<td>Low</td>
<td>Low</td>
<td>High (crossover design with different appliance design)</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (no PSG – only EMG diagnosis)</td>
</tr>
<tr>
<td>Arima, 2012 (34)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>High (crossover design with different appliance design)</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (no PSG – only EMG diagnosis)</td>
</tr>
<tr>
<td>Carra, 2010 (35)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only one-night effect)</td>
</tr>
<tr>
<td>Lee, 2010 (36)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (no PSG – only EMG diagnosis)</td>
</tr>
<tr>
<td>Saletu, 2010 (37)</td>
<td>High (controlled case–control design)</td>
<td>High (controlled case–control design)</td>
<td>Low</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only one-night effect)</td>
</tr>
<tr>
<td>Landry, 2009 (38)</td>
<td>Low</td>
<td>Low</td>
<td>High (crossover design with different appliance design)</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (only one-night effect)</td>
</tr>
<tr>
<td>Abekura, 2008 (39)</td>
<td>Unclear</td>
<td>Unclear</td>
<td>High (crossover design with different appliance design)</td>
<td>Unclear</td>
<td>Low</td>
<td>Low</td>
<td>Unclear (no PSG – only EMG diagnosis)</td>
</tr>
</tbody>
</table>
Finally, the only investigation providing an electrical stimulus to the masseter muscle to suppress its sleep-time activity supports the effectiveness of such kind of stimulation to reduce SB (41). This finding is in line with papers adopting different protocols of contingent electrical stimulation (CES) of the temporalis muscle, either with a RCT (24, 25) or a before–after design (26), which were not included in this review due to the lack of adoption of criteria that were used for scoring SB activity.

In general, findings from the reviewed literature suggest that evidence-based recommendations on SB management at the individual level are not yet available. In particular, it must be remarked that none of the reviewed papers focused on the indications for treatment. Such an approach contrasts with recent recommendations to consider SB as a phenomenon, and not a disorder per se (1, 4). Motor activities grouped under the umbrella term ‘bruxism’ do not necessarily have a pathological relevance and are not necessarily treatment-demanding conditions (49). This means that overtreatment of unspecific SB phenomena may be a concern until the relationship with clinical symptoms and consequences is fully clarified for each motor activity. Until now, an approach aiming to manage SB as a whole via the reduction of muscle activity did not lead to clear conclusions as far as the reduction in clinical pain levels are concerned (26, 44, 45).

Thus, from a clinical viewpoint, it is important that the investigations on SB management focus on the motor activities that are associated with clinical consequences, also targeting symptoms as a treatment goal. The study of the triangle bruxism – pain – psychosocial factors may contribute considerably to understand which SB phenomena should be viewed as treatment-demanding conditions. Based on that, it must be pointed out that current evidence does not support the existence of a standard of reference protocol for SB treatment. Thus, it is still recommendable that SB management is provided with caution within the framework of a conservative, ‘multiple-P’ approach (i.e. plates, pep talk, pills, psychology, physiotherapy).

Conclusions

The present literature review on SB management provides an update with respect to the last paper on the argument (i.e. the 2008 review by Lobbezoo et al.) (7)
and suggests that there is not enough evidence to define a standard of reference approach for the treatment of this phenomenon, with the exception of the use of oral appliances. Future studies focusing on the indications for SB treatment are recommended to provide a tailored approach to subjects with SB.

## Conflicts of interest

The authors declare they do not have any conflict of interests.

## Funding

The authors declare they did not receive any financial support for this manuscript.

## References


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