



UvA-DARE (Digital Academic Repository)

Sleep bruxism: contemporary insights in diagnosis, etiology and management

van der Zaag, J.

Publication date
2012

[Link to publication](#)

Citation for published version (APA):

van der Zaag, J. (2012). *Sleep bruxism: contemporary insights in diagnosis, etiology and management*.

General rights

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, Singel 425, 1012 WP Amsterdam, The Netherlands. You will be contacted as soon as possible.

Chapter 5

Review article: Principles for the management of bruxism.

**Frank Lobbezoo, Jac. van der Zaag, Maurits KA van Selms, Hans L
Hamburger & Machiel Naeije.**

J Oral Rehabil. 2008;35(7):509-523. Review.

Summary

The management of bruxism has been the subject of a large number of studies. A PubMed search, using relevant MeSH terms, yielded a total of 177 papers that were published over the past 40 years. Of these papers, 135 were used for the present review. Apparently, research into bruxism management is sensitive to fashion. Interest in studying the role of occlusal interventions and oral splints in the treatment of bruxism remained more or less constant over the years: between 1966 and 2007, approximately 40–60% of the papers dealt with this subject. The percentage of papers that dealt with behavioural approaches, on the other hand, declined from >60% in the first 2 decades (1966–1986) to only slightly >10% in the most recent decade (1997–2007). In the latter period, >40% of the papers studied the role of various medicines in the treatment of bruxism, while in the preceding decade (1987–1996), only approximately 5% of the studies dealt with the pharmacological management of bruxism. Unfortunately, a vast majority of the 135 papers have a too low level of evidence. Only 13% of the studies used a randomized clinical trial design, and even these trials do not yet provide clinicians with strong, evidence-based recommendations for the treatment of bruxism. Hence, there is a vast need for well-designed studies. Clinicians should be aware of this striking paucity of evidence regarding management of bruxism.

Keywords: bruxism, management, behavioural approaches, biofeedback, oral appliances, occlusion, medication, nutrition, study design, review.

Introduction*

Bruxism is an oral movement disorder that is characterized by grinding or clenching of the teeth. The disorder may occur during sleep as well as during wakefulness, and has an estimated prevalence in the general adult population of approximately 8–10% (1).

The aetiology of bruxism has a multifactorial nature. In the past, peripheral factors like occlusal discrepancies and deviations in orofacial anatomy have been considered the main causative factors for bruxism. Nowadays, such factors are known to play only a minor role, if any. Recent focus is more on central factors. Psychosocial factors like stress and certain personality characteristics are frequently mentioned in relation to bruxism. Further, it has been shown that bruxism is part of a sleep arousal response. In addition, bruxism appears to be modulated centrally by various neurotransmitters. Finally, pathophysiological factors like smoking, diseases, trauma, genetics and the intake of alcohol, caffeine, illicit drugs and medications may be involved in the aetiology of bruxism (1–3).

Bruxism should be diagnosed along multiple axes, viz. questionnaires, an oral history taking (including a bed partner's report of grinding sounds), an extra-oral and intra-oral inspection for clinical signs of bruxism, and, in some cases, an electromyographic (EMG) recording of the activity of the masticatory muscles or even a polysomnographic (PSG) recording of the sleeping patient. Any single one of these diagnostic tools should not be used in isolation, because patients may not be aware of the presence of bruxism, the clinical signs of bruxism may reflect a problem in the past rather than one in the present, and EMG and PSG only give a random indication of a disorder that fluctuates over time (1, 3, 4).

A host of dental problems have been ascribed to bruxism, such as attrition (i.e. mechanical wear, resulting from parafunction, and limited to the contacting surfaces of the teeth), hypertrophied masticatory muscles, fractures / failures of restorations or dental implants, headache and pain in the masticatory system (temporomandibular disorder pain; TMD pain) (1, 5, 6). Treatment of bruxism is indicated when the disorder causes any one of these possible consequences. Unfortunately, there is a striking paucity of high-quality evidence regarding management of bruxism. Here, a focused overview is given of the various occlusal, behavioural and pharmacological management approaches for bruxism. To demonstrate the completeness of the review, the details of the literature search strategy used are also provided.

*Based on a lecture given at the JOR Summer School 2007 sponsored by Blackwell Munksgaard and Medotech.

Search strategy

A literature search was performed on 28 June 2007, using the National Library of Medicine's Medical Subject Headings (MeSH) Database and PubMed. MEDLINE was searched with the following query: 'Bruxism / therapy' or 'Bruxism / drug therapy' or 'Bruxism / surgery' or 'Bruxism / prevention and control' or 'Bruxism / rehabilitation', restricted to 'Major Topic headings only' (MAJR) and using 'English' and 'Human' as search limits. This strategy yielded 177 papers, 29 of them being reviews. Of the 29 review papers, nine were used for the present paper for the additional value of their reasoning. Five papers could not be traced by the institutional library, while a total of 17 papers were omitted for various reasons. For example, six of these papers dealt with the repair of tooth surface loss caused by bruxism; not with the management of the disorder itself. The overview of the literature given here is therefore primarily based on 135 papers (177 papers, minus 20 excluded reviews, minus 5 untraceable papers, minus 17 omitted papers). The overview is supplemented with 15 papers that did not show up in the literature search, but that were nevertheless deemed important for the completeness of the overview. These papers were traced using the reference lists of already included papers.

The remaining 135 papers, which were published between 1966 and 2007, show that research on bruxism treatment is sensitive to fashion. Interest in studying the role of occlusal interventions and oral splints in the treatment of bruxism remained more or less constant over the years: during the entire period, approximately 40–60% of the papers dealt with this subject. The percentage of papers that dealt with behavioural approaches, on the other hand, declined from >60% in the first 2 decades (1966–1986) to only slightly >10% in the most recent decade (1997–2007). In the latter period, >40% of the papers studied the role of various medicines in the treatment of bruxism, while in the preceding decade (1987–1996), only approximately 5% of the studies dealt with the pharmacological management of bruxism. These time trends are illustrated in Fig. 1.

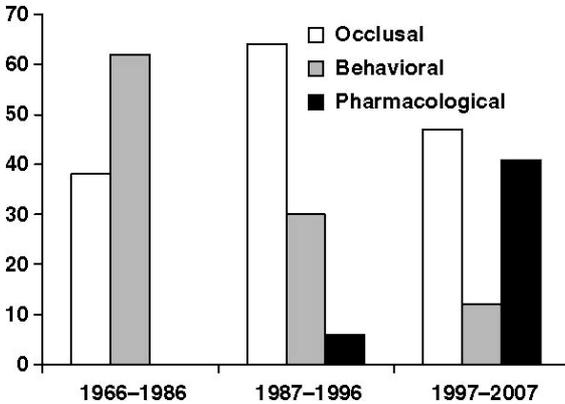


Fig. 1. Time trends in bruxism treatment. Percentage of papers, published in the three specified periods, which deal with occlusal, behavioural, or pharmacological interventions of bruxism.

The type of material represented by the included papers was classified according to the PubMed Publication Types as an indication of the scientific strength of the papers. Figure 2 shows the percentage distribution of the publication types within the set of 135 included papers. Clearly, a vast majority of the 135 papers have a low level of evidence. Only 13% of the studies used a randomized clinical trial design, and even most of these were more designed as experimental trials than as true clinical trials. In addition, it is not always clear whether bruxism during wakefulness, sleep bruxism, or both were studied. Further, the use of indirect or equivocally defined outcome measures for the quantification of bruxism is commonly encountered in the set of included papers. Publication type, bruxism type(s) and outcome measures are therefore part of this review as to indicate quality of the evidence.

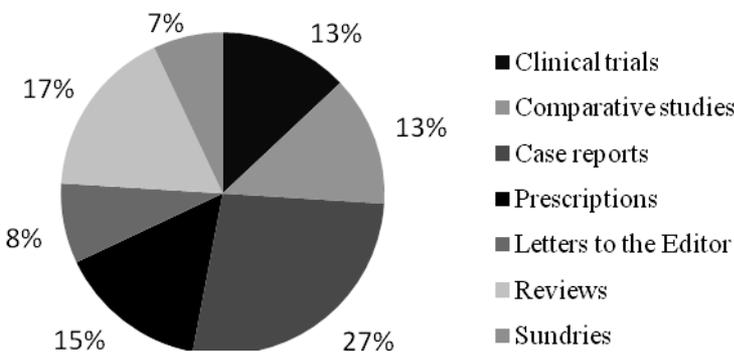


Fig. 2. Percentage distribution of the publication types within the set of included papers.

Occlusal approaches

Two categories of occlusal management strategies for bruxism can be distinguished: ‘true’ occlusal interventions and occlusal appliances.

‘True’ occlusal interventions

This category, that includes approaches like occlusal equilibration, occlusal rehabilitation and orthodontic treatment that is aimed at ‘achieving harmonious relationships between occluding surfaces’, still gives rise to a great deal of controversies among dental clinicians and researchers. Protagonists usually claim success of such approaches on the basis of their own clinical experience. In the literature, however, no high-quality evidence that supports the use of these irreversible techniques can be found: most of the papers on that subject are prescriptions (i.e. sets of statements, directories, or principles that describe an individual’s approach to a clinical problem), comparative (singlecohort) studies, letters to the editor and case reports. As an example of a prescription, Butler (7) described an occlusal adjustment procedure for the treatment of bruxism, amongst others, however, without a proper theoretical basis. Similarly, Frumker (8) formulated a set of principles for a successful occlusal treatment, on the basis of an unfounded idea that the better the occlusal anatomy and function, the easier the bruxers ‘relieve tension in the masticatory and associated musculature’. In an experimental comparative study, Holmgren and Sheikholeslam (9) tried to substantiate the effects of occlusal adjustment on the myo-electrical activity of the jaw-closing muscles. However, their brief, daytime EMG recordings of postural activity and maximal voluntary clenching cannot be interpreted in terms of bruxism.

A number of letters to the editor and case reports have been published with the objective of presenting convincing descriptions of the efficacy of occlusal interventions in the management of bruxism, by means of either occlusal equilibration (10) or occlusal rehabilitation with composite resin materials (11). In 1973, however, Stephens (12) expressed his awareness of the lack of science in this domain full of controversies and suggested that occlusal adjustment is indicated only as part of a periodontal treatment plan when trauma from occlusion is present – a suggestion that was generally followed by periodontists, especially in combination with the use of occlusal splints (13). More recently, in several letters to the editor, serious concerns and doubts have been expressed regarding occlusal interventions in adult bruxers (14, 15) as well as in young bruxers with a mixed dentition

(16). Of all authors, Greene et al. (17) are the most explicit by stating that occlusal adjustment ‘... further mutilate[s] the dentition beyond what the bruxism itself has performed. This is a classical example of misuse of an irreversible procedure with no evidence of its therapeutic value’.

As opposed to the afore-mentioned ‘low-quality evidence’ prescriptions, comparative studies, letters and case reports, only one study did use a randomized clinical trial (RCT) design (18). In that study, the effectiveness of an orthodontic technique (viz. buccal separators) in relieving bruxism activity was evaluated, and no differences between the active treatment and control conditions were observed. In a letter to the editor regarding this publication, the developer of the buccal separator technique failed to provide new, convincing evidence in favour of his technique (19).

In short, there is no support in the literature for the use of ‘true’ occlusal interventions like equilibration, rehabilitation and orthodontic alignment in the management of bruxism. In view of the current insights into the aetiology of bruxism, viz. that the disorder is mainly regulated centrally – not peripherally (2, 20), future research on this category of management strategies for bruxism seems redundant.

Occlusal appliances

The second category of occlusal management strategies for bruxism contains the frequently used occlusal appliances. According to an article by John Sedgwick in Newsweek of 4 December 1995, approximately 3.6 million ‘mouth guards’ are being manufactured in the USA on an annual basis. This represents a total cost of at least \$1 billion per year, indicating that insight into the efficacy of such appliances is important not only from a dental point of view, but also from an economic one.

As for occlusal equilibration and orthodontic treatment, a vast majority of the scientific papers that deal with the role of occlusal splints in the treatment of bruxism are prescriptions, case reports, comparative studies and case–control studies. Most prescriptions describe clinical and technical procedures for the manufacture of various types of splints. These splints have different names [e.g. occlusal bite guard (modified), bruxism appliance, bite plate, night guard (retainer), occlusal device] and slightly different appearances and properties, but in essence most of them are hard acrylic-resin stabilization appliances, mostly worn in the upper jaw (21–31). When a hard occlusal splint is intolerable for the patient, Taddey (32) suggests the use of a thin plastic shell like the one used to apply home

bleaching solutions. He claims that this solution works through a mechanism related to bio-feedback (see below); however, no evidence for the efficacy of plastic shells in the treatment of bruxism is provided. Three more prescriptions describe the manufacture of soft-resin bruxism appliances (33–35). Although the concept of soft splints is appealing, hard splints are generally preferred over soft splints for practical reasons (e.g. soft splints are more difficult to adjust than hard ones), to prevent inadvertent tooth movements, and because hard splints are suggested to be more effective in reducing bruxism activity than soft splints (36).

Four prescriptions describe splints that do not require a contribution of a dental laboratory. The first one describes an ‘in-office’ procedure for the manufacture of a regular hard acrylic-resin bruxism device that reduces the delay in starting a bruxism treatment, because no dental laboratory is involved (37). A similar prescription describes the chair-side manufacture of a composite splint (38). The third prescription describes the chair-side adjustment of the so-called ‘Nociceptive Trigeminal Inhibition (NTI) Clenching Suppression System’ – a small anterior splint that is supposed to be effective, amongst others, in the management of bruxism (39). Although in a randomized crossover trial by Baad-Hansen et al. (40) an inhibition in masseter muscle activity during sleep was found for the NTI splint as compared with a ‘regular’ hard occlusal splint, no evidence for the NTI splint’s long-term efficacy or safety is available so far. Finally, the fourth prescription describes the scientifically unsupported concept of the pre-fabricated and chair-side adjustable ‘Bruxism ‘S’ Splint’ that can be used in combination with active orthodontic treatment (41). More research is needed to assess the efficacy and safety of such unconventional, chair-side solutions before their application in dental practice can be recommended.

The case reports that deal with occlusal splints in the management of bruxism usually describe success in extreme and / or special-category patients. Bodenham (42) successfully treated an athlete for sports-related (daytime) clenching by means of a ‘bite guard’ (hard acrylic-resin splint) in the lower jaw, while Jones (43) successfully treated a 5-year-old girl with sleep bruxism and related headache by means of a hard maxillary splint – a treatment that must obviously have an as-short-as-possible duration, as to prevent gross disruption of the orofacial growth and development. Two case reports describe the application of bimaxillary soft splints for heavy bruxers (44, 45). This solution reportedly has the advantage of grinding sound reduction. Its durability, however, can be questioned. An interesting case is described by Cassisi et al. (46). Using a longitudinal design, these authors showed that the number of EMG bruxism events per hour of sleep reduced in their

35-year-old female bruxer when a hard occlusal splint or a palatal (non-occlusal) splint was worn as compared with a no-splint condition. On the basis of their case study, the authors suggest that more research is needed using groups of bruxism patients and a proper study design.

Several studies assessed the efficacy of occlusal splints in groups of bruxism patients, using a comparative, single-cohort ‘pre-treatment – post-treatment’ design and/or a case–control design. Although these are not the strongest designs to assess the efficacy of treatment modalities (for that purpose, RCT with long-term evaluations are required), such studies are frequently performed and the conclusions of these studies are stronger – and thus more valuable – than those of case reports. In an early study by Clark et al. (47), it was shown that occlusal splint treatment resulted in a decrease in nocturnal EMG activities in about half of the patients, while in about a quarter of the patients, no change or – in the remaining quarter – even an increase in EMG activity was observed. Using nocturnal EMG recordings as well, Hiyama et al. (48) found a significant reduction in bruxism activity while wearing an occlusal splint in all of their six study participants. An interesting single-cohort study was performed by Okeson (36). He compared, in a group of ten bruxers, the efficacy of hard versus soft occlusal splints. Both types of splints were worn by each of the ten participants, using a fixed order. It was shown that while the hard splint reduced nocturnal EMG activity in eight of 10 bruxers, the soft splint yielded an increase in bruxism activity in half of the bruxers and a decrease in only one of the remaining five participants. This suggests that hard splints are more effective in reducing bruxism activity than soft splints. Nevertheless, the use of soft splints is still common, at least in general dental practices in Sweden, despite the lack of scientific support for their efficacy and effectiveness (49).

A couple of studies used indirect measures for bruxism, which render these studies difficult to interpret in terms of occlusal splints being effective in the management of bruxism. Mejjias and Mehta (50) assessed the individual responses of bruxers to splint therapy. They found that their five participants all reacted favourably to the treatment, as assessed by the wear of a special bruxism monitoring device [‘Bruxcore’; see Koyano (4)] that consists of differently coloured plastic layers – an assessment technique that is easy to use but may lead to difficult-to-interpret outcomes because of the questionable paradigm that bruxism and wear are fully related, and because of the fact that the device itself may interfere with the bruxism behaviour. Using EMG recordings, Hamada et al. (51) observed significant reductions in the masticatory muscle activity of bruxers. The posttreatment

values were similar to those of healthy control subjects. In this study, however, the EMG measures were taken from voluntary daytime recordings, where direct EMG assessments of the actual bruxism behaviour are to be preferred. Also Moses (52) used daytime EMG measures to assess the restraining effect of his so-called Passivator appliance on bruxism. Consequently, this study is difficult to interpret as well. Using indirect clinical measures of bruxism only (e.g. dental and musculoskeletal pain complaints), Yustin et al. (53) found mandibular occlusal devices to be effective in the treatment of bruxism and of its associated pain complaints in most of their 86 study participants. Again, the use of indirect bruxism measures renders this study difficult to interpret unequivocally. In contrast to the findings of the above-described comparative and case-control studies, Sheikholeslam et al. (54) and Yap (55) found no effects of the occlusal splint on active nocturnal bruxism. However, these studies used awareness of bruxism behaviour (54) and wear facets on the occlusal splint (55), in combination with indirect clinical measures as outcome variables, which also renders these studies inconclusive.

Several studies used the 'higher quality' clinical trial design, although randomization to assign patients to test or control treatments/conditions (i.e. the 'true' RCT design) was employed in a few studies only. Shiau (56) observed that splint therapy did not change the length of the so-called silent period (an EMG characteristic that was believed to be related to bruxism, thus representing an indirect measure for that condition), nor were there any differences in length of the silent period between treated and untreated bruxers. Nagels et al. (57) were mainly interested in the possible effects of an occlusal splint on sleep quality in bruxers compared with normal volunteers, which turned out to be absent. Unfortunately, no indication is given regarding the effects of the splint on the bruxism behaviour itself. This study thereby falls outside the main scope of this overview. Using a cross-over design and nocturnal EMG activity as an outcome measure, Rugh et al. (58) did not observe any differences in efficacy in the treatment of sleep bruxers between two different types of hard occlusal splints, viz. one with canine guidance and another one with first molar guidance: in their eight participants, both splint types yielded variable outcomes that more or less resembled those described by Clark et al. (47) (see above). Hachmann et al. (59) demonstrated in a small-scale study that occlusal splints are effective in the treatment of bruxism in 3-to 5-year-old children: untreated bruxing children displayed increased wear facets, which were not observed in the treated group. Their conclusion that splints are thus

efficient against bruxism is, however, premature because the bruxism behaviour itself was not assessed, but only one of its possible consequences.

Two recent studies did use a ‘true’ RCT design. Using polysomnographic recordings, Dubé et al. (60) and Van der Zaag et al. (61) compared the efficacy of a hard occlusal splint versus a palatal control device [i.e. a placebo splint’, which is actually inactive in bruxers as long as it is kept thin (62)] in the treatment of sleep bruxism. While Dubé et al. (60) concluded that at two weeks, both devices reduce muscle activity associated with bruxism, Van der Zaag et al. (61) did not observe significant effects for either of the devices after four weeks of usage. The combination of the results of both studies corroborates the suggestion by Harada et al. (63) that oral appliances have only a transient effect on (EMG-determined) sleep bruxism as measured over a 6-week period. On the longer term, Ommerborn et al. (64) observed a reduction in bruxism activity after 12 weeks of occlusal splint therapy that continued into a 6-months follow-up. However, although these latter authors used a strong (RCT) study design, they did not quantify bruxism activity with EMG or polysomnography. Rather, they used the ‘Bruxcore’ bruxism monitoring device, which has several disadvantages (see above). This makes their results difficult to interpret unequivocally. Interestingly, Van der Zaag et al. (61) observed large differences between individual sleep bruxism patients. Some of them indeed showed a decrease in bruxism activity, while others showed no change or even an increase, which again is in line with the findings of Clark et al. (47). The reasons for these differences are as yet unclear.

Landry et al. (65) performed a short-term RCT to the efficacy of mandibular advancement devices (MAD; a bimaxillary appliance that is indicated for the management of snoring and sleep apnea) when compared with that of ‘regular’ maxillary occlusal splints. They observed only a moderate reduction in polysomnographically established sleep bruxism with the occlusal splint in situ, but a large decrease in bruxism activity when the MAD was worn – regardless of the amount of protrusion of the appliance. The authors could not readily explain this result, but they hypothesized, amongst others, that the fact that approximately two-thirds of their study sample reported localized pain with the MAD in situ may be responsible for the observed decrease. After all, it has been reported that in the presence of pain, bruxism activity may reduce considerably (6, 66, 67).

Given the contradictory results of the above-described studies and the scarcity of RCT on the efficacy of occlusal splints in the management of bruxism, it is prudent to limit the use of oral splints in the management of bruxism to the prevention or limitation of dental

damage that is possibly caused by the disorder (68, 69). Future research should focus on developing criteria for the clinical decision to use (or not to use) an occlusal splint in an individual bruxism patient.

Behavioural approaches

A wide variety of behavioural approaches have been tried in the management of bruxism. Here, the most widely studied one of these approaches, viz. biofeedback, will be reviewed first. In a subsequent section, the remaining behavioural techniques will be dealt with.

Biofeedback

Biofeedback uses the paradigm that bruxers can ‘unlearn’ their behaviour when a stimulus makes them aware of their adverse jaw muscle activities (‘aversive conditioning’). This technique has been applied for bruxism during wakefulness as well as for sleep bruxism. While awake, patients can be trained to control their jaw muscle activities through auditory or visual feedback from a surface EMG. For sleep bruxism, auditory, electrical, vibratory and even taste stimuli can be used for feedback.

Bruxism during wakefulness One of the early publications on the use of biofeedback in the management of bruxism during wakefulness is a prescription by Mittelman (70). He described an EMG technique that provides the daytime clencher with auditory feedback from his / her muscle activity, ‘telling the degree of muscle activity or relaxation that is taking place.’ The subtitle of Mittelman’s paper (‘It can be administered easily and inexpensively in any dental office’) suggests that the technique is ready for broad application. A similar suggestion is given in the review articles by Cannistraci (71) and Rubeling (72). Shulman (73) used a flat occlusal splint for biofeedback. The splint was inserted in the explicit understanding that the appliance serves to remind the daytime bruxer of adverse tooth contacts (i.e. contacts other than those involved in chewing and swallowing). In the author’s hands, an immediate success of approximately 50% was obtained. Kramer (74) applied a special kind of biofeedback to manage the daytime bruxism problem of an 8-year-old boy with learning difficulties: whenever a bruxism event started, the boy’s teacher pressed her finger firmly against the boy’s jaw for a few seconds. The intervention was successful in approximately 2 weeks and during the first follow-up week, the bruxism

behaviour remained at a low level. The author suggested that this approach should be implemented with special populations in educational settings. This approach resembles the one used by Blount et al. (75), who successfully treated two profoundly retarded adult bruxers for their condition with 'contingent icing', i.e. brief applications of ice to the facial area whenever bruxism occurred. Likewise, Rudrud and Halaszyn (76) used contingent massage to combat daytime bruxism. Unfortunately, strong scientific evidence for the efficacy of any of these afore-described diurnal biofeedback approaches is lacking.

In a comparative study by Manns et al. (77), the apparently successful application of auditory feedback from surface EMG activity was shown in 33 daytime bruxers with myofascial pain. The study design, however, precludes strong conclusions to be drawn from this study. Using a RCT design, Treacy (78) showed significant jaw-closing muscle activity decreases after a 4-month treatment period with a muscle activity awareness training program compared with an active control treatment and a sham treatment. Although this finding supports the efficacy of this type of biofeedback in the management of diurnal bruxism, no long-term results are given. This urges dentists to remain reserved when applying this technique, especially because another RCT failed to show significant decreases in masticatory muscle EMG levels as a result of either a biofeedback training program or a control treatment (79). Hence, more research is needed to assess the efficacy of biofeedback in the management of bruxism during wakefulness.

Sleep bruxism For the use of biofeedback in the management of sleep bruxism, Cherasia and Parks (80) published a prescription. Their technique used contingent arousal from sleep with actual awakenings. Although the authors are aware of the lack of validation of their technique, they stated that its potential effectiveness, ease of use and lack of risk warrant its consideration. So far, nine case reports, representing a total of 13 patients, were published in which some type of biofeedback was used to control sleep bruxism. Nissani (81) used a taste stimulus to awaken the patient. This stimulus was caused by the bruxism-related rupture of capsules, filled with an aversive substance (agreed upon with the patient, e.g. mustard, ginger, garlic, etc.) and embedded in a simple dental appliance. On the basis of a single case, the author claimed long-term success. In most of the case reports, a sound blast was applied as the aversive stimulus (82–87), although in one case study, this technique failed to be effective (88). The sound stimulus is supposed to actually wake up the patient, who is then supposed to switch off the sound and resume his / her sleep. The awakenings are a major

disadvantage of such approaches, because sleep disruption may lead to serious side effects like excessive daytime sleepiness (89). Even more subtle techniques, like the bruxism-contingent vibratory feedback system of Watanabe et al. (90) and the jaw-opening reflex feedback system that was recently developed by Jadidi et al. (91) that do not induce substantial sleep disturbance, according to the authors, might still cause significant changes in sleep architecture that yield long-term adverse reactions like daytime sleepiness (89). This concern should be taken into consideration when evaluating new biofeedback devices for the management of sleep bruxism.

An alternative approach was followed by Small (92), who used daytime biofeedback sessions in combination with an occlusal splint for night-time use to combat the sleep bruxism problem of a 36-year-old woman. A similar approach was followed by Cornellier et al. (93) in four adult bruxers. Just like the taste and sound blast methods, this approach also yielded positive (longterm) outcomes, with no sleep disruption as possible side effect. Despite this advantage, Small (92) indicated that his case report is at best suggestive and proposed better, controlled studies to test the efficacy of his approach. Obviously, this holds true for all case reports indicated and summarized here.

Over the years, several comparative studies have been published in which the efficacy of biofeedback on sleep bruxism was evaluated. Audible tones derived from EMG recordings caused a significant reduction in sleep bruxism activities in all of nine bruxers compared with control nights during which the biofeedback device was worn with an inactive bruxism warning system (94). Similar findings, with longer evaluation periods of up to three months, were reported by Clark et al. (95) and Hudzinski and Walters (96). Pierce and Gale (97) also found positive effects of nocturnal biofeedback (*viz.* a contingent, aversive tone), but reported these effects to be transient during a 6-month follow-up period. Nishigawa et al. (98) used contingent electrical lip stimulation to combat sleep bruxism, which turned out to be a promising technique for temporarily suppressing the disorder. However, the long-term effects remain to be determined, and the above-described concern regarding the risk of sleep disruption and subsequent daytime sleepiness should be assessed for this technique as well. Clearly, the comparative study design of the papers that are summarized in this paragraph precludes strong scientific conclusions to be drawn. To that end, better controlled studies are needed. In the only controlled clinical trial on the efficacy of nocturnal biofeedback, whenever a bruxism event exceeded a preset electromyographic threshold, an audible tone indeed yielded a better treatment outcome than a no-treatment

control condition (99). Unfortunately, only short-term (2-months) results are given, but not the longer-term results, which are needed for a proper assessment of the efficacy and safety of any treatment modality.

In short, despite the considerable amount of attention that researchers devoted to biofeedback [see also the review by Cassisi et al.(100)], there are serious doubts whether this is actually an effective treatment for bruxism, especially in the long-term. Further, the possible consequences of the frequent arousals, like excessive daytime sleepiness, need further attention before this technique can be applied for the safe treatment of patients with bruxism.

Other behavioural approaches

Other behavioural approaches that have been described in the literature for the management of bruxism include psychoanalysis, autosuggestion, hypnosis, progressive relaxation, meditation, self-monitoring, sleep hygiene, habit reversal / habit retraining and massed practice. In the oldest review article that was found with the present literature search strategy, Olkinuora (101) described various psychiatric treatment techniques for bruxism, such as psychoanalysis and autosuggestion. According to the author, this latter technique helps the bruxer become aware of the habit, even while asleep, by giving him / her the autosuggestion ‘I’ll wake up if I gnash my teeth’ before falling asleep. Unfortunately, this intriguing approach, which has been reviewed briefly by others as well (102), lacks scientific strength.

Another approach, hypnosis, was reviewed more than three decades ago by Goldberg (103). On top of his review, this author used three cases to illustrate the usefulness of this technique in the management of bruxism, as do several other authors (104, 105). Where these cases lack sufficient strength in terms of scientific evidence, Clarke and Reynolds (106) wrote an abstract in which they concluded on the basis of a stronger study design (*viz.* a case-control study) and by using nocturnal EMG recordings that hypnotherapy provided profound relief from problems related to nocturnal bruxism. These results were later published as a full-length paper, but now as a single-cohort study and with the addition of long-term effects as assessed with self-report (107). The conclusion remained the same and even appeared to be applicable to the follow-up period of 4–36 months. The authors, however, were fully aware of the limitations of their study and suggested some improvements to increase the strength of the evidence.

Various relaxation techniques have been described in relation to the management of bruxism. Relaxation, including meditation, is supposed to produce a sense of self-esteem and control over one's body (102). Pear (102) as well as other authors [e.g. Cannistraci and Friedrich (108)] described relaxation and meditation as part of a holistic approach, which means that awareness and 'wellness' of the whole body is being promoted. However, no information whatsoever can be found in the literature regarding the efficacy of this approach in the treatment of bruxism. Only a comparative study by Restrepo et al. (109) provided slightly stronger evidence for the positive effect of relaxation in 3-to 6-year-old children who suffer from bruxism. A drawback of that study, however, is the use of indirect measures for bruxism, which hampers an unequivocal interpretation of the outcome.

Specifically for diurnal bruxism, self-monitoring – or 'habit awareness' – has been suggested as an appropriate therapy. According to Rosen (110), bruxers gain control of daytime clenching using a self-monitoring procedure, which simply means that every time the patient notes the occurrence of clenching activity, this event is jotted down in a diary or entered in some kind of counting device. This approach would finally lead to a decrease in diurnal bruxism activity. Unfortunately, Rosen's paper is a case report and lacks scientific strength.

For nocturnal bruxism, a specific approach has been suggested, namely sleep hygiene measures (111). The objective of measures like 'avoid stimulants (e.g. caffeine, nicotine) for several hours before bedtime' and 'maintain a regular sleep schedule' is to promote better sleep. Amongst others, better sleep means that more time is being spent in the deeper sleep stages and that less arousals from sleep occur. As bruxism mainly occurs in the lighter sleep stages and in relation to arousals (1), bruxism will probably decrease. However, as yet, no well-designed studies on this behavioural treatment modality have been published.

Behavioural techniques like habit reversal / habit retraining, and massed practice have all in common that the adverse behaviour, i.e. bruxism, is actively being combated. During habit reversal, a competing activity opposite to the bruxism behaviour, but involving the same muscles, is being taught to the bruxers (e.g. opening the mouth). Two papers, describing a total of five cases, claimed success for this technique, although good-quality evidence is lacking (112, 113). Zeldow (114) published a prescription of a similar technique, which he calls 'habit retraining': the replacement of a bad habit with a good one. As the

good habit was maintaining a free-way space, it is obvious that ‘habit retraining’ is actually a variation of ‘habit reversal’.

Especially during the late sixties and early seventies of the past century, massed practice therapy for bruxism was studied relatively widely. This behavioural technique contains exaggerating the bruxism-related muscle activities, thereby making the habit punitive rather than rewarding. The first paper about this technique was a case report by Ayer and Gale (115). These authors suggested that (self-reported) bruxism may be eliminated by massed practice therapy. Comparative studies by the same authors came to the same conclusion (116, 117). They even put forward a theoretical model to explain the purported efficacy of massed practice (118). Also, Vasta and Wortman (119) described the successful application of massed practice therapy in the treatment of a single bruxer, in whom bruxism activity was assessed objectively by means of an automated time-sampling procedure. Heller and Forgione (120), on the other hand, did not observe any significant reductions in bruxism behaviour in their comparative study that used the wear of the ‘Bruxcore’ bruxism monitoring device to assess bruxism activity (see above: Occlusal appliances).

In short, the value of the above-described behavioural approaches is questionable, because they all lack a sound scientific basis: most studies so far are case reports, prescriptions and comparative studies. More well-designed research is thus needed on the use of these approaches in the management of bruxism.

Pharmacological approaches

The use of medication in the management of bruxism has been studied increasingly over the past decades. Most studies so far are case reports, but for several medicines RCT have been performed. An extensive review on the relationship between drugs and bruxism was published by Winocur et al. (121).

One of the oldest reports on a pharmacological approach for bruxism is the one published by Chasins (122). He concluded that the short-term administration of the muscle relaxant methocarbamol yielded ‘good control and improvement of the bruxism habit’ of approximately 40 bruxers compared with an equally sized group of untreated bruxers. Besides the fact that the study design does not meet the current standard of an RCT, bruxism was assessed solely on the basis of the patients’ reports. This makes the study difficult to interpret. In a more recent, well-designed RCT, it was shown that sleep bruxism did improve

with the frequently prescribed, non-specific muscle relaxant clonazepam (a benzodiazepine), although the maintenance of its therapeutic efficacy, its long-term tolerability and its risk of addiction need further attention (123).

Another drug that affects muscle function, by exerting a paralytic effect through an inhibition of acetylcholine release at the neuromuscular junction, is botulinum toxin. So far, its application in the management of bruxism is mainly described in case reports. Without exception, these reports claimed success of botulinum toxin in decreasing (clinically assessed) bruxism activity, especially in severe cases with co-morbidities like coma (124), brain injury (125, 126), amphetamine abuse (127), Huntington's disease (128) and autism (129). Tan and Jankovic (130) reported the results of botulinum toxin injections in 18 bruxers. In only one of their patients, (transient) dysphagia occurred as an adverse reaction. They concluded that this drug can be administered as a safe and effective treatment for severe bruxers. They also stated, however, that this treatment modality should be confined to patients who are refractory to other (conventional) treatments, and that placebo-controlled studies are needed before evidence-based recommendations can be given.

Several studies have been performed to assess the effects of serotonergic and dopaminergic medicines in the treatment of sleep bruxism. In a placebo-controlled RCT, bruxism-related nocturnal EMG activity was not influenced by the serotonin precursor L-tryptophan (131). In contrast to that negative finding, a placebo-controlled sleep laboratory RCT showed that the catecholamine precursor L-dopa exerted a modest, attenuating effect on sleep bruxism (132). Likewise, sleep bruxism activity was reduced by the administration of low doses of the dopamine D1/D2 receptor agonist pergolide in a severe bruxism case (133). The dopamine D2 receptor agonist bromocriptine, on the other hand, did not cause an exacerbation or reduction in sleep bruxism motor activity (134), although a report of two single-patient clinical trials yielded promising results for that drug (135). The effects of serotonin-related and dopamine-related drugs on bruxism therefore remain unclear.

For the use of anticonvulsant drugs in the treatment of bruxism, only case reports are available. Gabapentin was successfully applied for the treatment of a 50-yearold man who suffered from bruxism, induced by venlafaxine (an antidepressant; see below) (136). Likewise, self-reported bruxism was successfully managed with tiagabine in four of five cases described by Kast (137). Unfortunately, no RCT are available to assess the efficacy and safety of anticonvulsant drugs in the management of bruxism.

Antidepressant drugs may exert deviating effects on bruxism: either they exacerbate the condition (selective serotonin reuptake inhibitors, SSRI) or they are inert in their effects (amitriptyline). While Stein et al. (138) reported a decrease in nocturnal bruxism in two bruxers as a possible consequence of the use of the SSRI paroxetine and citalopram, most papers reported bruxism to be induced by SSRI [reviewed in detail by Lobbezoo et al. (139)]. Bostwick and Jaffee (140) described four cases of sertraline-induced bruxism, which were successfully treated with the serotonin 1A receptor agonist buspirone. Two similar cases were successfully managed with dosage manipulation by Ranjan et al. (141). These authors argued that such is a better approach than using buspirone as an antidote. Obviously, only better-designed studies can provide us with strong scientific evidence. For the efficacy assessment of the tricyclic antidepressant amitriptyline in the treatment of sleep bruxism, two RCT have been performed. Unfortunately, low doses (25 mg per night) of amitriptyline turned out to be ineffective in the management of sleep bruxism (142, 143), although some individual study participants clearly responded to the medication (144).

For two sympatholytic medicines, experimental RCT have been performed. Huynh et al. (145) found no effects of the non-selective adrenergic beta-blocker propranolol on sleep bruxism, despite the positive response to this drug in two cases of antipsychotic-induced bruxism (146). The selective alpha-2 agonist clonidine, on the other hand, does seem a promising medicine for the management of sleep bruxism, although further safety assessments are still required because severe morning hypotension was noted in approximately 20% of the participants (145). Taking the above-described evidence together, it can be concluded that although some pharmacological approaches for bruxism seem promising, they all need further efficacy and safety assessments before clinical recommendations can be made.

Miscellaneous approaches

Six papers describe management strategies for bruxism that do not readily fit either one of the above-used categories of occlusal, behavioural, or pharmacological approaches. Five of them are related to physical therapy, while one is related to a surgical procedure in the oral region. Ackerman (147) described his approach of instructing the patient with bruxism to develop his/her jaw-opening muscles. The objective is 'to develop the depressor muscles so that they will be as strong or as firm as the elevator muscles. Then, they will hold the

mandible in balance'. Ackerman expressed the hope that by adopting this philosophy, future efforts to eliminate bruxism would be more successful. Also Quinn (148, 149) suggested the use of physical rehabilitation techniques (viz. isokinetic exercises) for depressor muscle strengthening. According to this author, such exercises will assist in, amongst others, correcting bruxism. Knutson (150) claimed a rapid and complete recovery of chronic sleep bruxism after upper cervical vectored manipulation of a 6-yearold child. Unfortunately, the level of evidence of the above-summarized prescriptions and case reports, all of which used some sort of physical therapy to combat bruxism, is low. Even the positive results of a controlled trial, in which the buccinators muscles were trained by means of a special device, viz. the Pro-Fono Facial Exerciser, are inconclusive because of the ambiguous quantification of the bruxism activities (151).

DiFrancesco et al. (152) reported the results of a comparative study on a group of children with sleep-disordered breathing and bruxism, of whom a significant proportion ceased to report bruxism after adenotonsillectomy. Apart from the fact that the scientific strength of this paper is relatively low, the authors failed to provide the readers with a plausible (i.e. non-occlusal) explanation for their finding, which might very well be coincidental.

Conclusions and recommendations

From the above, it can be gathered that a vast majority of the 135 papers that constitute the basis of this review are more or less inconclusive: only 13% of them used an appropriate RCT study design. Comparative studies, case report and prescriptions are the most commonly used study designs in this literature search. Clinicians should be aware of this striking paucity of evidence regarding the management of bruxism. They should also know that nowadays, new management strategies for bruxism are being proposed by commercial companies in the absence of any scientific proof for their efficacy and safety. Hence, there is a vast need for well-designed studies on the management of bruxism.

Huynh et al. (153) used most of the above-described, well-designed RCTs to assess the number needed to treat (NNT, the number of patients who must be treated before the outcome can be expected to occur; the lower the NNT, the more beneficial the treatment) and the effect size (ES, the magnitude of the effect of a treatment relative to a placebo condition; the higher the ES, the more beneficial the treatment) for the various management strategies for sleep bruxism. They included three oral appliance studies and seven phar-

macological studies in their analyses; all of them are included in the present review [Oral appliances: Dubé et al. (60); Van der Zaag et al. (61); Landry et al. (65). Pharmacological treatments: Etzel et al. (131); Lobbezoo et al. (132); Mohamed et al. (142); Lavigne et al. (134); Raigrodski et al. (143); Saletu et al. (123); Huynh et al. (145)]. Of these treatments, the mandibular advancement device (65) and clonidine (145) seemed to be the most promising treatment approaches, yielding the lowest NNT and the largest ES. However, given the adverse reactions of these treatments (see above), the occlusal splint (60, 61) and clonazepam (123) seemed to be acceptable (short-term) alternatives. However, Huynh et al. (153) stressed that further longitudinal, large-sample size RCT are needed before evidence-based recommendations can be given.

In the absence of definitive evidence, bruxism can best be managed following the so-called ‘triple-P’ approach: Plates, Pep talk and Pills. ‘Plates’ are occlusal appliances, most commonly of the hard acrylic resin occlusal stabilization splint type. These appliances probably function more like protectors of the remaining teeth rather than that they actually diminish the bruxism behaviour. ‘Pep talk’ stands for counselling, a behavioural approach that includes addressing the patient’s awareness of the movement disorder, relaxation and lifestyle and sleep hygiene instructions. Albeit of unproven efficacy, these approaches can be applied safely in bruxism patients. ‘Pills’ represents pharmacological interventions with centrally acting drugs such as benzodiazepines. As long as definitive evidence is missing, the use of medicines in the treatment of bruxism should be confined to short periods and to severe cases in which occlusal appliances and counselling were ineffective. Such should be performed in close collaboration with medical specialists.

The triple-P approach reflects the current insight into the aetiology of bruxism, that is considered to be mainly regulated centrally; not peripherally (2). The approach also stresses that whenever bruxism treatment is indicated, the disorder should be assessed by a multidisciplinary team that includes dentists, psychologists and medical specialists. This important notion should not only be recognized by the dental discipline itself, but also by the other disciplines that are involved in this team concept [e.g. psychology (154)].

References

1. Lavigne GJ, Manzini C, Kato T. Sleep bruxism. In: Kryger M, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine*. Philadelphia, PA:Elsevier Saunders,2005:946–959.
2. Lobbezoo F, van der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants. An updated review. *J Oral Rehabil*. 2006;33:293–300.
3. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiopathology: what do we learn from sleep studies? *J Oral Rehabil*. 2008;35:476–494.
4. Koyano K. Assessment of bruxism in the clinic. *J Oral Rehabil*. 2008;35:495–508.
5. Johansson A, Johansson A-K, Omar R, Carlsson GE. Rehabilitation of the worn dentition. *J Oral Rehabil*. 2008;35:548– 566.
6. Svensson P, Jadidi F, Arima T, Baad-Hansen L Relationships between craniofacial pain and bruxism. *J Oral Rehabil*. 2008;35:524–547.
7. Butler JH. Occlusal adjustment. *Dent Dig*. 1970;76:422–426.
8. Frumker SC. Occlusion and muscle tension. *Basal Facts*. 1981;4:85–87.
9. Holmgren K, Sheikholeslam A. Occlusal adjustment and myoelectric activity of the jaw elevator muscles in patients with nocturnal bruxism and craniomandibular disorders. *Scand J Dent Res*. 1994;102:238–243.
10. Leon SP. The source of the problem. *Dent Today*. 2003;22:12.
11. Ford RT, Douglas W. The use of composite resin for creating anterior guidance during occlusal therapy. *Quintessence Int*. 1988;19:331–337.
12. Stephens RG. Occlusal adjustment in periodontal therapy. *J Can Dent Assoc (Tor)*. 1973;39:332–337.
13. Lester M, Baer PN. Survey of current therapy: bruxism splints. *Periodontal Case Rep*. 1989;11:23–24.
14. Lalonde B. Occlusal splints. *J Am Dent Assoc*. 1996;127:554, 556, 558.
15. Wessberg G. Bruxism and the bite. *Hawaii Dent J*. 2001;32:4.
16. Harnick DJ. Treating bruxism and clenching. *J Am Dent Assoc*. 2000;131:436.
17. Greene CS, Klasser GD, Epstein JB. ‘Observations’ questioned. *J Am Dent Assoc*. 2005;136:852–853.
18. Abraham J, Pierce C, Rinchuse D, Zullo T. Assessment of buccal separators in the relief of bruxist activity associated with myofascial pain-dysfunction. *Angle Orthod*. 1992;62:177–184.
19. Mintz AH. Acute TMJ versus chronic TMJ. *Angle Orthod*. 1993;63:4–5.

20. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil.* 2001;28:1085–1091.
21. Allen DL. Accurate occlusal bite guards. *Periodontics.* 1967;5:93–95.
22. Courant P. Use of removable acrylic splints in general practice. *J Can Dent Assoc (Tor).* 1967;33:494–501.
23. Greenwald AS. The bruxism appliance and its varied application: outline of procedure. *N Y J Dent.* 1968;38:443.
24. Askinas SW. Fabrication of an occlusal splint. *J Prosthet Dent.* 1972;28:549–551.
25. Glazebrook P. An equilibrated bite plate. *Probe (Lond).* 1974;16:99–100.
26. Gabriele P. A double night guard retainer. *N Y State Dent J.* 1986;52:30–31.
27. Ordene NM. A modified bruxism appliance. *N Y State Dent J.* 1989;55:40–41.
28. Perel ML. Parafunctional habits, nightguards, and root form implants. *Implant Dent.* 1994;3:261–263.
29. Davis CR. Maintaining immediate posterior disclusion on an occlusal splint for patient with severe bruxism habit. *J Prosthet Dent.* 1996;75:338–339.
30. Nassif NJ, al-Ghamdi KS. Managing bruxism and temporomandibular disorders using a centric relation occlusal device. *Compend Contin Educ Dent.* 1999;20:1071–1074, 1076, 1078, 1086.
31. Cowie RR. The clinical use of night guards: occlusal objectives. *Dent Today.* 2004;23:112, 114–115.
32. Taddey JJ. Problems and solutions. TMD patients who are gaggers. *Cranio.* 1995;13:68.
33. Anthony TH. Soft thermoplastics in bruxism appliances. *Trends Tech Contemp Dent Lab.* 1995;12:32–36.
34. Grozev L, Michailov T. Treatment of bruxism and bruxomania (clinically tested). *Folia Med (Plovdiv).* 1999;41:147–148.
35. Kalman L. Occlusal appliances: a new material. *Dent Today.* 2007;26:72–73.
36. Okeson JP. The effects of hard and soft occlusal splints on nocturnal bruxism. *J Am Dent Assoc.* 1987;114:788–791.
37. Austin D, Attanasio R. A procedure for making a bruxism device in the office. *J Prosthet Dent.* 1991;66:266–269.
38. Leib AM. The occlusal bite splint – a noninvasive therapy for occlusal habits and temporomandibular disorders. *Compend Contin Educ Dent.* 1996;17:1081–1084, 1086, 1088.
39. Boyd JP. Improving TMD treatment and protecting restorative dentistry. *Dent Today.* 1998;17:144.

40. Baad-Hansen L, Jadidi F, Castrillon E, Thomsen PB, Svensson P. Effect of a nociceptive trigeminal inhibitory splint on electromyographic activity in jaw closing muscles during sleep. *J Oral Rehabil.* 2007;34:105–111.
41. Sullivan TC. A new occlusal splint for treating bruxism and TMD during orthodontic therapy. *J Clin Orthod.* 2001;35:142–144.
42. Bodenham RS. A bite guard for athletic training. A case report. *Br Dent J.* 1970;129:85–86.
43. Jones CM. Chronic headache and nocturnal bruxism in a 5-year-old child treated with an occlusal splint. *Int J Paediatr Dent.* 1993;3:95–97.
44. Thorp PD. An appliance to be worn at night for the heavy tooth grinder. *Dent Tech.* 1975;28:144–145.
45. Maeda Y, Ikuzawa M, Mitani T, Matsuda S. Bimaxillary soft splints for unconscious hard-clenching patients: a clinical report. *J Prosthet Dent.* 2001;85:342–344.
46. Cassisi JE, McGlynn FD, Mahan PE. Occlusal splint effects on nocturnal bruxing: an emerging paradigm and some early results. *Cranio.* 1987;5:64–68.
47. 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.
48. 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.
49. Lindfors E, Magnusson T, Tegelberg A. Interocclusal appliances – indications and clinical routines in general dental practice in Sweden. *Swed Dent J.* 2006;30:123–134.
50. Mejias JE, Mehta NR. Subjective and objective evaluation of bruxing patients undergoing short-term splint therapy. *J Oral Rehabil.* 1982;9:279–289.
51. Hamada T, Kotani H, Kawazoe Y, Yamada S. Effect of occlusal splints on the EMG activity of masseter and temporal muscles in bruxism with clinical symptoms. *J Oral Rehabil.* 1982;9:119–123.
52. Moses AJ. Analysis of a functional appliance. *CDS Rev.* 1991;84:24–29.
53. Yustin D, Neff P, Rieger MR, Hurst T. Characterization of 86 bruxing patients with long-term study of their management with occlusal devices and other forms of therapy. *J Orofac Pain.* 1993;7:54–60.
54. Sheikholeslam A, Holmgren K, Riise C. Therapeutic effects of the plane occlusal splint on signs and symptoms of craniomandibular disorders in patients with nocturnal bruxism. *J Oral Rehabil.* 1993;20:473–482.
55. Yap AU. Effects of stabilization appliances on nocturnal parafunctional activities in patients with and without signs of temporomandibular disorders. *J Oral Rehabil.* 1998;25:64–68.
56. Shiau YY. The effect of the bite plane splint on the mandibular reposition in bruxers. *Taiwan Yi Xue Hui Za Zhi.* 1980;79:184–195.

57. Nagels G, Okkerse W, Braem M, Van Bogaert PP, De Deyn B, Poirrier R et al. Decreased amount of slow wave sleep in nocturnal bruxism is not improved by dental splint therapy. *Acta Neurol Belg.* 2001;101:152–159.
58. Rugh JD, Graham GS, Smith JC, Ohrbach RK. Effects of canine versus molar occlusal splint guidance on nocturnal bruxism and craniomandibular symptomatology. *J Craniomandib Disord.* 1989;3:203–210.
59. Hachmann A, Martins EA, Araujo FB, Nunes R. Efficacy of the nocturnal bite plate in the control of bruxism for 3 to 5 year old children. *J Clin Pediatr Dent.* 1999;24:9–15.
60. Dubé C, Rompre PH, Manzini C, Guitard F, De Grandmont P, Lavigne GJ. Quantitative polygraphic controlled study on efficacy and safety of oral splint devices in tooth-grinding subjects. *J Dent Res.* 2004;83:398–403.
61. Van der Zaag J, Lobbezoo F, Wicks DJ, Visscher CM, Hamburger HL, Naeije M. Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. *J Orofac Pain.* 2005;19:151–158.
62. Hasegawa K, Okamoto M, Nishigawa G, Oki K, Minagi S. The design of non-occlusal intraoral appliances on hard palate and their effect on masseter muscle activity during sleep. *Cranio.* 2007;25:8–15.
63. Harada T, Ichiki R, Tsukiyama Y, Koyano K. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. *J Oral Rehabil.* 2006;33:482–488.
64. Ommerborn MA, Schneider C, Giraki M, Schafer R, Handschel J, Franz M et al. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. *Eur J Oral Sci.* 2007;115:7–14.
65. Landry ML, Rompre PH, Manzini C, Guitard F, De Grandmont P, Lavigne GJ. Reduction of sleep bruxism using a mandibular advancement device: an experimental controlled study. *Int J Prosthodont.* 2006;19:549–556.
66. Lavigne GJ, Rompre PH, Montplaisir JY, Lobbezoo F. Motor activity in sleep bruxism with concomitant jaw muscle pain: a retrospective pilot study. *Eur J Oral Sci.* 1997;105:92–95.
67. Arima T, Arendt-Nielsen L, Svensson P. Effect of jaw muscle pain and soreness evoked by capsaicin before sleep on orofacial motor activity during sleep. *J Orofac Pain.* 2001;15:245–256.
68. Dao TT, Lavigne GJ. Oral splints: the crutches for temporomandibular disorders and bruxism? *Crit Rev Oral Biol Med.* 1998;9:345–361.
69. Attanasio R. Bruxism and intraoral orthotics. *Tex Dent J.* 2000;117:82–87.
70. Mittelman J. Biofeedback: new answer to dental pain. It can be administered easily and inexpensively in any dental office. *Dent Manage.* 1976;16:21–22, 26–27.

71. Cannistraci AJ. A method to control bruxism: biofeedback-assisted relaxation therapy. *J Am Soc Prev Dent*. 1976;6:12–15.
72. Rubeling RR Jr. Treating patients through biofeedback therapy. *Dent Stud*. 1979;57:57–62.
73. Shulman J. Teaching patients how to stop bruxing habits. *J Am Dent Assoc*. 2001;132:1275–1277.
74. Kramer JJ. Aversive control of bruxism in a mentally retarded child: a case study. *Psychol Rep*. 1981;49:815–818.
75. Blount RL, Drabman RS, Wilson N, Stewart D. Reducing severe diurnal bruxism in two profoundly retarded females. *J Appl Behav Anal*. 1982;15:565–571.
76. Rudrud E, Halaszyn J. Reduction of bruxism by contingent massage. *Spec Care Dentist*. 1981;1:122–124.
77. Manns A, Miralles R, Adrian H. The application of audiostimulation and electromyographic biofeedback to bruxism and myofascial pain-dysfunction syndrome. *Oral Surg Oral Med Oral Pathol*. 1981;52:247–252.
78. Treacy K. Awareness/relaxation training and transcutaneous electrical neural stimulation in the treatment of bruxism. *J Oral Rehabil*. 1999;26:280–287.
79. Wieselmann-Penkner K, Janda M, Lorenzoni M, Polansky R. A comparison of the muscular relaxation effect of TENS and EMG-biofeedback in patients with bruxism. *J Oral Rehabil*. 2001;28:849–853.
80. Cherasia M, Parks L. Suggestions for use of behavioral measures in treating bruxism. *Psychol Rep*. 1986;58:719–722.
81. Nissani M. Can taste aversion prevent bruxism? *Appl Psychophysiol Biofeedback*. 2000;25:43–54.
82. Heller RF, Strang HR. Controlling bruxism through automated aversive conditioning. *Behav Res Ther*. 1973;11:327–329.
83. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. *J Behav Med*. 1980;3:385–397.
84. Moss RA, Hammer D, Adams HE, Jenkins JO, Thompson K, Haber J. A more efficient biofeedback procedure for the treatment of nocturnal bruxism. *J Oral Rehabil*. 1982;9:125–131.
85. Feehan M, Marsh N. The reduction of bruxism using contingent EMG audible biofeedback: a case study. *J Behav Ther Exp Psychiatry*. 1989;20:179–183.
86. Watson TS. Effectiveness of arousal and arousal plus overcorrection to reduce nocturnal bruxism. *J Behav Ther Exp Psychiatry*. 1993;24:181–185.
87. Foster PS. Use of the Calmset 3 biofeedback / relaxation system in the assessment and treatment of chronic nocturnal bruxism. *Appl Psychophysiol Biofeedback*. 2004;29:141–147.

88. Piccione A, Coates TJ, George JM, Rosenthal D, Karzmark P. Nocturnal biofeedback for nocturnal bruxism. *Biofeedback Self Regul.* 1982;7:405–419.
89. Roehrs T, Carskadon MA, Dement WC, Roth T. Daytime sleepiness and alertness. In: Kryger M, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine*. Philadelphia, PA: Elsevier Saunders, 2005:39–50.
90. Watanabe T, Baba K, Yamagata K, Ohyama T, Clark GT. A vibratory stimulation-based inhibition system for nocturnal bruxism: a clinical report. *J Prosthet Dent.* 2001;85:233–235.
91. Jadidi F, Castrillon E, Svensson P. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. *J Oral Rehabil.* 2008;35:171–183.
92. Small MM. Treatment of nocturnal bruxism: a case study. *Biol Psychol.* 1978;6:235–236.
93. Cornellier V, Keenan DM, Wisser K. The effects of EMG biofeedback training upon nocturnal and diurnal bruxing responses. *Int J Orofacial Myology.* 1982;8:11–15.
94. Kardachi BJ, Clarke NG. The use of biofeedback to control bruxism. *J Periodontol.* 1977;48:639–642.
95. Clark GT, Beemstervoer P, Rugh JD. The treatment of nocturnal bruxism using contingent EMG feedback with an arousal task. *Behav Res Ther.* 1981;19:451–455.
96. Hudzinski LG, Walters PJ. Use of a portable electromyogram integrator and biofeedback unit in the treatment of chronic nocturnal bruxism. *J Prosthet Dent.* 1987;58:698–701.
97. Pierce CJ, Gale EN. A comparison of different treatments for nocturnal bruxism. *J Dent Res.* 1988;67:597–601.
98. Nishigawa K, Kondo K, Takeuchi H, Clark GT. Contingent electrical lip stimulation for sleep bruxism: a pilot study. *J Prosthet Dent.* 2003;89:412–417.
99. Casas JM, Beemsterboer P, Clark GT. A comparison of stress-reduction behavioral counseling and contingent nocturnal EMG feedback for the treatment of bruxism. *Behav Res Ther.* 1982;20:9–15.
100. Cassisi JE, McGlynn FD, Belles DR. EMG-activated feedback alarms for the treatment of nocturnal bruxism: current status and future directions. *Biofeedback Self Regul.* 1987;12:13–30.
101. Olkinuora M. A review of the literature on, and a discussion of studies of bruxism and its psychogenesis and some new psychological hypotheses. *Suom Hammaslaak Toim.* 1969;65:312–324.
102. Pear JH. Holistic care concepts, bruxism and necrotizing ulcerative gingivitis. *Dent Hyg (Chic).* 1982;56:24–29.
103. Goldberg G. The psychological, physiological and hypnotic approach to bruxism in the treatment of periodontal disease. *J Am Soc Psychosom Dent Med.* 1973;20:75–91.
104. Somer E. Hypnotherapy in the treatment of the chronic nocturnal use of a dental splint prescribed for bruxism. *Int J Clin Exp Hypn.* 1991;39:145–154.

105. LaCrosse MB. Understanding change: five-year follow-up of brief hypnotic treatment of chronic bruxism. *Am J Clin Hypn.* 1994;36:276–281.
106. Clarke JH, Reynolds PJ. Hypnosis for treatment of nocturnal bruxism. *J Dent Res.* 1989;68:402.
107. Clarke JH, Reynolds PJ. Suggestive hypnotherapy for nocturnal bruxism: a pilot study. *Am J Clin Hypn.* 1991;33:248–253.
108. Cannistraci AJ, Friedrich JA. A multidimensional approach to bruxism and TMD. *N Y State Dent J.* 1987;53:31–34.
109. Restrepo CC, Alvarez E, Jaramillo C, Velez C, Valencia I. Effects of psychological techniques on bruxism in children with primary teeth. *J Oral Rehabil.* 2001;28:354–360.
110. Rosen JC. Self-monitoring in the treatment of diurnal bruxism. *J Behav Ther Exp Psychiatry.* 1981;12:347–350.
111. Morin CM. Psychological and behavioral treatments for primary insomnia. In: Kryger M, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* Philadelphia, PA: Elsevier Saunders, 2005:726–737.
112. Rosenbaum MS, Ayllon T. Treating bruxism with the habit-reversal technique. *Behav Res Ther.* 1981;19:87–96.
113. Blore D. Grinding down. *Nurs Times.* 1995;91:46–47.
114. Zeldow LL. Treating clenching and bruxing by habit change. *J Am Dent Assoc.* 1976;93:31–33.
115. Ayer WA, Gale EN. Extinction of bruxism by massed practice therapy. Report of case. *J Can Dent Assoc (Tor).* 1969;35:492–494.
116. Ayer WA, Levin MP. Elimination of tooth grinding habits by massed practice therapy. *J Periodontol.* 1973;44:569–571.
117. Ayer WA. Massed practice exercises for the elimination of tooth-grinding habits. *Behav Res Ther.* 1976;14:163–164.
118. Ayer WA, Levin MP. Theoretical basis and application of massed practice exercises for the elimination of tooth grinding habits. *J Periodontol.* 1975;46:306–308.
119. Vasta R, Wortman HA. Nocturnal bruxism treated by massed negative practice. A case study. *Behav Modif.* 1988;12:618–626.
120. Heller RF, Forgione AG. An evaluation of bruxism control: massed negative practice and automated relaxation training. *J Dent Res.* 1975;54:1120–1123.
121. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I. Drugs and bruxism: a critical review. *J Orofac Pain.* 2003;17:99–111.
122. Chasins AI. Methocarbamol (robaxin) as an adjunct in the treatment of bruxism. *J Dent Med.* 1959;14:166–170.

123. Saletu A, Parapatics S, Saletu B, Anderer P, Prause W, Putz H et al. On the pharmacotherapy of sleep bruxism: placebo-controlled polysomnographic and psychometric studies with clonazepam. *Neuropsychobiology*. 2005;51:214–225.
124. Van Zandijcke M, Marchau MM. Treatment of bruxism with botulinum toxin injections. *J Neurol Neurosurg Psychiatry*. 1990;53:530.
125. Ivanhoe CB, Lai JM, Francisco GE. Bruxism after brain injury: successful treatment with botulinum toxin-A. *Arch Phys Med Rehabil*. 1997;78:1272–1273.
126. Pidcock FS, Wise JM, Christensen JR. Treatment of severe post-traumatic bruxism with botulinum toxin-A: case report. *J Oral Maxillofac Surg*. 2002;60:115–117.
127. See SJ, Tan EK. Severe amphetamine-induced bruxism: treatment with botulinum toxin. *Acta Neurol Scand*. 2003;107:161–163.
128. Nash MC, Ferrell RB, Lombardo MA, Williams RB. Treatment of bruxism in Huntington's disease with botulinum toxin. *J Neuropsychiatry Clin Neurosci*. 2004;16:381–382.
129. Monroy PG, Da Fonseca MA. The use of botulinum toxin-a in the treatment of severe bruxism in a patient with autism: a case report. *Spec Care Dentist*. 2006;26:37–39.
130. Tan EK, Jankovic J. Treating severe bruxism with botulinum toxin. *J Am Dent Assoc*. 2000;131:211–216.
131. Etzel KR, Stockstill JW, Rugh JD, Fisher JG. Tryptophan supplementation for nocturnal bruxism: report of negative results. *J Craniomandib Disord*. 1991;5:115–120.
132. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. *Mov Disord*. 1997;12:73–78.
133. Van der Zaag J, Lobbezoo F, van der Avoort PGGL, Wicks DJ, Hamburger HL, Naeije M. Effects of pergolide on severe sleep bruxism in a patient experiencing oral implant failure. *J Oral Rehabil*. 2007;34:317–322.
134. Lavigne GJ, Soucy JP, Lobbezoo F, Manzini C, Blanchet PJ, Montplaisir JY. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. *Clin Neuropharmacol*. 2001;24:145–149.
135. Lobbezoo F, Soucy JP, Hartman NG, Montplaisir JY, Lavigne GJ. Effects of the D2 receptor agonist bromocriptine on sleep bruxism: report of two single-patient clinical trials. *J Dent Res*. 1997;76:1610–1614.
136. Brown ES, Hong SC. Antidepressant-induced bruxism successfully treated with gabapentin. *J Am Dent Assoc*. 1999;130:1467–1469.
137. Kast RE. Tiagabine may reduce bruxism and associated temporomandibular joint pain. *Anesth Prog*. 2005;52:102–104.
138. Stein DJ, Van Greunen G, Niehaus D. Can bruxism respond to serotonin reuptake inhibitors? *J Clin Psychiatry*. 1998;59:133.

139. Lobbezoo F, van Denderen RJA, Verheij JGC, Naeije M. Reports of SSRI-associated bruxism in the family physician's office. *J Orofac Pain*. 2001;15:340–346.
140. Bostwick JM, Jaffee MS. Buspirone as an antidote to SSRI-induced bruxism in 4 cases. *J Clin Psychiatry*. 1999;60:857–860.
141. Ranjan S, Chandra PS, Prabhu S. Antidepressant-induced bruxism: need for buspirone? *Int J Neuropsychopharmacol*. 2006;9:485–487.
142. Mohamed SE, Christensen LV, Penchas J. A randomized double-blind clinical trial of the effect of amitriptyline on nocturnal masseteric motor activity (sleep bruxism). *Cranio*. 1997;15:326–332.
143. Raigrodski AJ, Christensen LV, Mohamed SE, Gardiner DM. The effect of four-week administration of amitriptyline on sleep bruxism. A double-blind crossover clinical study. *Cranio*. 2001;19:21–25.
144. Melis M. Dr. Melis comments on Raigrodski, et al.'s article in the January 2001 issue of *Cranio*. *Cranio*. 2001;19:149.
145. Huynh N, Lavigne GJ, Lanfranchi PA, Montplaisir JY, De Champlain J. The effect of 2 sympatholytic medications propranolol and clonidine on sleep bruxism: experimental randomized controlled studies. *Sleep*. 2006;29:307–316.
146. Amir I, Hermesh H, Gavish A. Bruxism secondary to antipsychotic drug exposure: a positive response to propranolol. *Clin Neuropharmacol*. 1997;20:86–89.
147. Ackerman JB. A new approach to the treatment of bruxism and bruxomania. *NY State Dent J*. 1966;32:259–261.
148. Quinn JH. Mandibular exercises to control bruxism and deviation problems. *Cranio*. 1995;13:30–34.
149. Quinn JH. Treating bruxism and clenching. *J Am Dent Assoc*. 2000;131:723.
150. Knutson GA. Vectored upper cervical manipulation for chronic sleep bruxism, headache, and cervical spine pain in a child. *J Manipulative Physiol Ther*. 2003;26:E16.
151. Jardini RS, Ruiz LS, Moyses MA. Electromyographic analysis of the masseter and buccinator muscles with the pro-fono facial exerciser use in bruxers. *Cranio*. 2006;24:29–37.
152. DiFrancesco RC, Junqueira PA, Trezza PM, De Faria ME, Frizzarini R, Zerati FE. Improvement of bruxism after T & A surgery. *Int J Pediatr Otorhinolaryngol*. 2004;68:441–445.
153. Huynh NT, Rompre PH, Montplaisir JY, Manzini C, Okura K, Lavigne GJ. Comparison of various treatments for sleep bruxism using determinants of number needed to treat and effect size. *Int J Prosthodont*. 2006;19:435–441.
154. Billups AJ. Mouthguards, nightguards, palliatives and collaboration. *Va Dent J*. 1992;69:19–21.