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### Sleep bruxism: contemporary insights in diagnosis, etiology and management

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## Chapter 9

### **Summary**



Bruxism is a movement disorder of the masticatory system that is characterized, among others, by teeth grinding and clenching. It can occur while awake as well as during sleep. In this thesis, the main focus is on SB. Bruxism is an important threat to oral health. Dentists are often faced with patients presenting problems that may be due to SB such as extensive tooth wear in the form of attrition (i.e., mechanical wear resulting from mastication or parafunctional activity), temporomandibular pain, masticatory muscle hypertrophy, and loss of restorations and even of dental implants. Many questions about SB are still unanswered (e.g., in relation to the etiologic factors and the management strategies). Therefore, the general aim of this thesis was to give a contemporary insight in the diagnosis, etiology, and management of SB.

The aim of the study described in **Chapter 2** was to quantify the time-variant nature of SB and to discuss its consequences. Six clinically diagnosed bruxers and six non-bruxers participated. Four ambulatory polysomnographic recordings were obtained for every participant. As SB outcome variables, the number of episodes per hour of sleep (Epi / hr), the number of bursts per hour (Bur / hr) and the bruxism time index (BTI: the percentage of total sleep time spent bruxing) were established. To quantify the time-variant nature of SB, standard errors of measurement (SEMs) were calculated. For the nonbruxers, the SEMs for Epi / hr, Bur / hr and BTI were 1.0, 5.7 and 0.1. For the bruxers, the respective values were 2.1, 14.9 and 0.4. In the discussion of the chapter, arguments are given that because of the time-variant nature of the polysomnographic recordings, cut-off bands around cut-off points might be useful for the recognition of SB.

In the review described in **Chapter 3**, the aim was to provide an update of two previous reviews from our department (one about the etiology of bruxism and the other about the possible role of this movement disorder in the failure of dental implants) and to describe the details of the literature search strategies used, thus enabling the readers to judge the completeness of the review. Most studies that were published about the etiology during the past 5 years corroborate the previously drawn conclusions. Similarly, the update of the review about the possible causal relationship between bruxism and implant failure reveals no new points of view. Thus, there is no reason to assume otherwise than that bruxism is mainly regulated centrally, not peripherally, and that there is still insufficient evidence to support or refute a causal relationship between bruxism and implant failure. This illustrates

that there is a vast need for well-designed studies to study both the etiology of bruxism and its purported relationship with implant failure.

**Chapter 4** discussed the possible association between SB and periodic limb movements during sleep (PLMS). Both motor events may have a common underlying neurophysiologic mechanism, especially in relation to the occurrence of sleep-related EEG arousals. To test this hypothesis, three research questions were assessed. First, it was assessed whether PLMS events occur more frequently in SB patients than in healthy controls. Second, the question was put forward whether the combined presence of SB and PLMS events is more common than that of isolated SB or PLMS events in a group of SB patients. Third, as to further unravel the possible role of EEG arousals in the underlying neurophysiologic mechanism of SB and PLMS, it was assessed in a group of SB patients whether combined SB/PLMS events with associated EEG arousals are more common than those without associated EEG arousals. Positive answers to these questions could suggest a common neurophysiological basis for both movement disorders. Seventeen SB patients and eleven healthy controls were polysomnographically studied. SB, PLMS, and EEG arousals were scored. An association was noted when the occurrence was within a 3-sec association zone. The PLMS index was higher in SB patients than in healthy controls ( $P = 0.000$ ). Within the group of SB patients, the combined SB/PLMS index was higher than the isolated SB index ( $P = 0.000$ ) and the isolated PLMS index ( $P = 0.010$ ). Similarly, the combined SB/PLMS index with EEG-arousal was higher than the combined SB/PLMS index without EEG-arousal in SB patients ( $P = 0.001$ ). The results of this study indicate that SB, PLMS, and EEG arousals are highly associated and probably have a common underlying neurophysiological mechanism.

In the review in **Chapter 5**, the various management strategies of bruxism were studied. 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 this 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 behavioral approaches, on the other hand, declined from more than 60% in the first two decades (1966–1986) to only slightly over 10% in the most recent decade (1997–2007). In the latter period, more than 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 (RCT) 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.

In **Chapter 6**, the efficacy of occlusal stabilization splints in the management of SB was assessed using a parallel RCT design. Twenty-one participants were randomly assigned to an occlusal splint group ( $n = 11$ ), or a palatal splint (i.e., an acrylic palatal coverage) group ( $n = 10$ ). Two polysomnographic recordings that included bilateral masseter electromyographic activity were made: one prior to treatment, the other after a treatment period of 4 weeks. Epi / hr, Bur / hr, and BTI were established as outcome variables at a 10% maximum voluntary contraction threshold level. A general linear model was used to test the effects between splint groups and within the treatment phase as well as their interaction for each outcome variable. Neither occlusal stabilization splints nor palatal splints had an influence on the SB outcome variables or on the sleep variables measured on a group level. In individual cases, variable outcomes were found: Some patients had an increase (33% to 48% of the cases), while others showed no change (33% to 48%) or a decrease (19% to 29%) in SB outcome variables. The conclusion was that the absence of significant group effects of splints in the management of SB indicates that caution is required when splints are indicated, apart from their role in the protection against dental wear. The application of splints should therefore be considered at an individual level.

In the study in **Chapter 7**, the case of a severe SB patient was reported. In this male, 51-year-old patient, oral implants failed as a probable consequence of severe, polysomnographically confirmed SB. As this patient had the wish to be re-implanted after this failure, we decided to try diminishing the frequency and duration of his bruxism first. To that end, two management strategies were used. Their efficacy was evaluated polysomnographically, yielding a total of six overnight recordings. Of the selected management strategies, the administration of low doses of the dopamine D1/D2 receptor agonist pergolide finally resulted in a substantial and lasting reduction in the bruxism outcome measures under study. This result supports the previous suggestion that central neurochemicals like dopamine may be involved in the modulation of sleep bruxism. The

case report also illustrates the importance of an extensive history taking (questionnaires as well as oral) and clinical examination of oral implant patients for the presence of severe bruxism before the implant procedure is started. In case of doubt, polysomnography may be considered to definitively confirm or rule out the presence of severe SB.