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

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## Chapter 8

### **General Discussion**



The general aim of this thesis was to give a contemporary insight in the diagnosis, etiology, and management of sleep bruxism (SB).

### Diagnosis

During the past 15 years, researchers worldwide have adopted the research diagnostic criteria for sleep bruxism (RDC/SB) as proposed by Lavigne et al. (1996). These criteria mainly consist of frequency indices, i.e., numbers of oromotor events per hour of sleep. For these indices, cut-off values were proposed above which one could be considered an SB patient. This approach is useful for a gross indication of the SB condition of an individual. Furthermore, it enables the comparison between individuals as well as between groups of patients; even when these groups are studied in different laboratories. As to further improve the diagnosis of SB, the group of Lavigne continues developing their criteria for an SB diagnosis. For example, they published a study challenging the threshold above which an oromotor event is considered an SB event (Lavigne et al, 2001). Thus, the recommended threshold value decreased from 20% to 10% of the maximum voluntary contraction level. Nowadays, this 10% threshold is widely accepted and also used in this thesis. More recently, Rompré et al. (2007) distinguished low, moderate, and high frequencies for SB, on the basis of which new cut-off criteria will be proposed in the future. Such criteria will be useful when applied in SB patients with co-morbid temporomandibular pain (TMP), since it is a common observation in the literature that the presence of TMP yields lower PSG-established SB indices (for a review, see Manfredini and Lobbezoo, 2010).

Despite these improvements of the RDC/SB (Lavigne et al, 1996), such an approach falls short when one is interested in the actual bruxism intensity, i.e., the frequency, duration, and intensity of the oromotor events as developed by an individual SB patient while asleep. For example, an index of 10 bruxism episodes per hour of sleep in one individual can still be different from the same index found in another individual regarding the actual duration of the constituent oromotor events. To partly overcome this shortcoming, in **Chapter 6** of this thesis, the so-called bruxism time index (BTI), i.e., the sleep time spent bruxing, was introduced. If this index is used together with the indices developed by Lavigne et al., one does not only know the number of SB events per time unit, but also the

duration of these events. This gives a better insight into the actual oromotor activities that were recorded during the individual patient's sleep.

Regardless of the technique that is used to quantify SB, the interpretation of sleep recordings is always hampered by the fact that SB is a fluctuating rather than a stable condition over time. This phenomenon of variability over time was first studied by Lavigne et al. (2001), who concluded on the basis of a retrospective analysis of polysomnographic data that in some SB patients, the variability is significant, especially in sleep stages 1 and 2, and should thus be taken into account. This recommendation was further elaborated in **Chapter 2** of this thesis, in which the time-variant nature of SB was assessed prospectively. The study yielded recommendations for both diagnosis and therapy evaluation of SB in which the variability in SB outcome measures can play a role. In short, the study shows that for the recognition of SB on the basis of sleep recordings, cut-off bands around cut-off points, rather than cut-off points should be used, while for the determination of a significant change in SB in an individual patient after therapy, an SB outcome measure should have a value that is equal to or greater than the so-called smallest detectable difference. It should be kept in mind that the values of the cut-off bands and of the smallest detectable differences depend upon the protocol used (e.g., the number of recordings used and the population studied) as well as upon setting in which they are determined. It is thus recommended to establish these values for every sleep laboratory and patient population. Most importantly, however, for therapy evaluation, the decision whether or not a treatment is effective in an individual SB patient remains the ultimate responsibility of the clinician.

The above-outlined approach can be applied to other disorders that use cut-off criteria for diagnostic and therapy evaluation purposes as well. It is therefore recommended that laboratories studying conditions like periodic limb movements during sleep (PLMS), restless legs syndrome, and obstructive sleep apnea (Aarab et al, 2009) adopt these quantification techniques that respect the time-variant nature of the condition of interest. Finally, it should be emphasized that the accuracy of diagnostic procedures can only be judged if reliable and valid techniques are being used. In other words, before introducing diagnostic procedures into everyday clinical practice, a rigorous evaluation of these procedures is needed to reduce the risk of incorrect decisions. Bossuyt et al. (2003) published the so-called Standards for Reporting of Diagnostic Accuracy (STARD), which describes a checklist and flow diagram for authors of reports on this topic. This remains to be done for the recognition of SB using the above-outlined approaches.

## Etiology

As indicated in the review described in **Chapter 3**, multiple factors can be associated with SB; notably central factors of pathophysiological and psychosocial nature. So far, most evidence for these associations is derived from cross-sectional studies, and these factors can thus be interpreted only as risk indicators (i.e., potential factors that are derived from cross-sectional studies and can therefore only suggest associations; Beck, 1998). For the determination of risk factors (i.e., factors that are derived from longitudinal studies and can directly indicate the possibility of a disease occurring; Beck, 1998), longitudinal studies are required. Unfortunately, such studies are scarce and more research is needed to further elucidate the true nature of the etiology of SB. Most promising seem studies assessing the genetic background of SB. Especially Finish studies that use twin-cohort study designs are currently contributing to the growing body of knowledge on this topic. For example, Rintakoski et al. (2010) showed that genetic factors account for about half of the phenotypic variance in liability to SB in young adults. Importantly, the fact that also in this study, only young, otherwise healthy subjects were included, underlines the commonly felt need to expand our research efforts to patient samples of various ages and with co-morbidities so that findings can be better generalized. Recently, Van Selms et al. (in preparation) reported that in adolescents, the self-reported prevalence rates of SB are significantly higher than those reported in the literature for adults. SB is thus age-dependent, and the factor ‘age’ should be taken into consideration as predictor in epidemiological studies (Lavigne and Montplaisir, 1994). Similarly, co-morbid conditions like TMP, sleep apnea, general movement disorders like M. Parkinson, and psychological conditions like stress, depression, and anxiety are commonly associated with SB (e.g., Pierce et al, 1995; Sjöholm et al, 2000; Ohayon et al, 2001; Lavigne et al, 2005). It is therefore necessary to study SB populations with various combinations of such factors being present, as to enable a better generalization of findings and to further unravel the connections between these etiological factors in SB.

In **Chapter 4** of this thesis, it was shown that sleep-related movement disorders like SB and PLMS both are highly associated with EEG arousals and are thus probably the result of a common underlying neurophysiological mechanism. This conclusion is corroborated by the fact that also the pharmacological management strategies of both movement disorders show similarities. For example, both SB and PLMS can be managed with dopaminergic agents (e.g., pergolide, a D1/D2 receptor agonist, which was also applied

successfully in the severe SB case described in **Chapter 7** of this thesis). It is well established that centrally acting neurochemicals like dopamine are involved in the pathophysiology of bruxism, and that dopamine agonists show greater efficacy than placebo in the treatment of movement disorders like PLMS (Lobbezoo et al, 1996, 2006; Scholz et al, 2011). So far, SB and PLMS have been studied as separate entities. However, based on the above arguments, it is recommended that both movement disorders are assessed in combination in future research. This will further improve our insight into the etiology of both conditions. Possibly, such research will yield an overall index for sleep-related movement disorders or even new, common management strategies.

### **Management**

So far, an evidence-based treatment of SB with proven efficacy and acceptable safety is not available (see the review in **Chapter 5** of this thesis). Many previous studies on this topic do not meet the quality criteria for properly performed treatment efficacy studies: randomized controlled trials (RCTs) – being the gold-standard design for such studies – rarely meet all criteria formulated in the so-called Consolidated Standards of Reporting Trials (CONSORT; Altman et al, 2001). It is therefore recommended, in the absence of evidence, to follow the so-called ‘Triple-P Approach’, suggesting to manage SB patients with any combination of Pep-talk (viz., counseling, such as sleep hygiene instructions), Plates (viz., occlusal stabilization splints; see **Chapter 6**), and Pills (viz., medication; see **Chapter 7**). Of these approaches, medication has the most causal nature – which is to be preferred over ‘palliative’ strategies – and the research efforts on the use of medication in SB are increasing. However, medicines like clonazepam, a generally acting benzodiazepine (Saletu et al, 2005, 2010), but also other promising, more causally acting candidates like pergolide (see **Chapter 7**), should preferably be reserved for acute crises of SB that cannot be managed with strategies like sleep hygiene instructions and/or splints. Safety issues related to the use of such medicines (e.g., adverse effects of clonazepam on blood pressure) prevent these drugs from being recommended for widespread clinical usage in general dental practices. Clearly, for such medicines to be prescribed, consultation with a medical doctor or specialist is required.

As mentioned in the General Introduction of this thesis, a management approach could be: 1. managing SB as a neurophysiologic phenomenon; 2. managing the etiological factors of SB; or 3. managing the harmful effects of SB. Whenever possible, the choice for

one or another management strategy is based upon the most predominant etiology in an individual patient (Winocur and Lobbezoo, 2010). In **Chapter 6** of this thesis, it was shown that when an occlusal stabilization splint is worn on a nightly basis for a period of four weeks, no significant improvement in SB could be observed as compared to the pre-treatment phase. This study had an RCT design and fully met the CONSORT criteria (Altman et al, 2001). This is evidenced by the fact that the study was included in a meta-analysis of the Cochrane database (Macedo et al, 2008). Interestingly, another high-quality RCT on the efficacy of splints in the management of SB was performed by Dubé et al. (2004), however over a two-week period. The fact that they did observe a significant improvement in SB suggests that the effect of splints has a transient nature. In the four-week study described in **Chapter 6**, some SB patients also showed an improvement over the study period, while on the other hand other participants did not benefit from their splint or even got their SB condition worsened. This further underlines the necessity for individually tailored management strategies. As a matter of fact, this focus on individuals rather than on groups of patients is increasingly seen in dentistry and medicine and represents the New Thinking of the 21<sup>st</sup> century (for reviews, see Chan and Ginsburg, 2011; Stohler and Zubieta, 2010).

An occlusal stabilization splint was also tried as one of the treatment options for the case described in **Chapter 7** of this thesis. In that severe SB patient, pharmacological treatment with pergolide finally resulted in an acceptable and lasting decrease in SB. Interestingly, while the splint had no effect on the number of SB events per hour of sleep, the bruxism time index (see above) decreased significantly, the change in the value of this index being greater than its smallest detectable difference (see **Chapter 2**). This finding can only be explained as shorter SB events with the splint in situ. Possibly, due to the central origin of SB, in some patients, the number of SB events that is initiated cannot be influenced with a peripherally acting treatment, while the duration of the events can be modulated by sensory influences from the orofacial area (Kato et al, 2003).

### **Conclusions**

The studies in this thesis provide further evidence that sleep bruxism is a centrally initiated condition, which can be modulated by peripheral sensory influences from the orofacial area. The diagnosis, etiology, and management should be assessed on an individual basis rather than on the level of patient groups. Sleep bruxism is highly associated with other sleep-

related movement disorders, suggesting that these conditions are possibly the results of the same underlying neurophysiological mechanism.

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