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

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Chapter 1

# General Introduction

**Introduction**

Besides bacteriologic (e.g., caries and periodontal infections) and chemical (e.g., erosion) factors, mechanical factors (e.g., trauma and parafunctional activities like bruxism) are an important threat to the oral health. Dentists are often faced with patients presenting problems that may be due to bruxism, 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 (De Leeuw 2008; Lobbezoo *et al* 2006a; Lobbezoo *et al* 2006b; Abe *et al* 2009). Clearly, bruxism is an important disorder, from both a clinical and a research point of view. The latter is illustrated by the enormous amount of articles published on this topic the last forty years. Indeed, a recent literature search, performed by our group, resulted in 2,354 papers dealing with bruxism, thus showing that there is great interest in this topic.

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. During wakefulness, it is primarily characterized by clenching, tapping of the teeth, or jaw bracing without tooth contact (Lobbezoo *et al* 2009). When it occurs during sleep, the condition is often denoted in the literature as sleep bruxism (SB) (Thorpy 1990; AASM 2005, 2007). There are several definitions of awake and sleep-related bruxism in the literature. A definition, often used in clinical daily dental practice, is the one formulated in The Glossary of Prosthodontic Terms (2005), where bruxism is defined as the parafunctional grinding of teeth, and as an oral habit consisting of involuntary rhythmic or spasmodic nonfunctional gnashing, grinding, or clenching of the teeth in other than chewing movements of the mandible, which may lead to occlusal trauma. Another commonly used definition, given by the American Academy of Orofacial Pain (AAOP), is that bruxism is a diurnal or nocturnal parafunctional activity including clenching, bracing, gnashing, and grinding of the teeth (De Leeuw, 2008). A third definition is focused on SB. In 2005, the American Academy of Sleep Medicine (AASM) published the second edition of the International Classification of Sleep Disorders (ICSD-2), in which SB is described as an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive (intense) sleep electroencephalographic (EEG) arousal activity. In many SB studies, the association with EEG arousal activity is mentioned, but these arousals are not taken into account when scoring SB using the ICSD-2. In this thesis, in which the main focus is on SB, the definition of the AASM (2005), given above, will be

used, except for the scoring of EEG arousals. This definition is preferred over the more dental ones suggested in The Glossary of Prosthodontic Terms and by the AAOP for its unequivocal and operational nature (Lobbezoo *et al* 2009).

SB is a common condition. Two large-scale studies with solid study designs came to almost the same prevalence estimations for SB. Lavigne and Montplaisir (1994) reported a general population prevalence of 8.0%; Ohayon *et al* (2001), of 8.2%. This figure is the same for men and women. It should be noted, however, that these prevalence data were derived from self-reports, of which the reliability and validity may be questionable (see below). Despite this potential shortcoming, the concordance between the findings in the studies by Lavigne and Montplaisir (1994) and Ohayon *et al* (2001) increases the face validity of these figures.

As stated above, the topic of this thesis is limited to SB. Many questions about SB are still unanswered (e.g., in relation to the etiologic factors and the management strategies) (Huynh *et al* 2006; Lobbezoo *et al* 2009). In addition, in contrast to awake bruxism, SB is well-quantifiable by polysomnography (PSG; a sleep recording that captures, among others, electrical brain activities [EEG], electrical muscle activities [electromyography, EMG], and body movements) (Gallo *et al.* 1997; Walters *et al.* 2007). In this chapter, the diagnosis, the etiologic factors, and the management strategies of SB are introduced. The chapter ends with a synopsis of the different chapters and the aims of this thesis.

**Diagnosis**

A proper quantification of SB is important to diagnose and monitor the condition, and to evaluate its management (Lavigne *et al* 1996; Walters *et al* 2007; Lavigne *et al* 2008). In general, a SB patient reports to a dental professional with certain signs or symptoms that are possibly related to bruxism, like tooth wear or temporomandibular pain. This can be a reason for further examination, e.g., by using a combination of the following diagnostic tools: 1. questionnaires, inquiring about parafunctional activities during sleep and/or a report of grinding sounds by a bed partner or parent; 2. a clinical examination of dental wear, masseter muscle hypertrophy, and/or temporomandibular pain; and 3. an electromyographic or polysomnographic examination (Lavigne *et al* 2008). Except for polysomnography, the main disadvantage of all above-mentioned techniques is that they do not provide a detailed insight into SB in relation to sleep, so that the sleep aspects of SB cannot be assessed. The

latter is important to ensure that the studied condition is actually sleep-related bruxism according to the definition of the AASM (2005).

Questionnaires provide subjective information, which has the limitation that participants can under- or overestimate the condition of interest, especially when sleeping alone (Koyano and Tsukiyama 2009). Notwithstanding these limitations, questionnaires are widely used for the assessment of SB, both in the clinical setting and in research. For example, Van der Meulen et al (2006) described a questionnaire with four bruxism questions (awake and sleep-related grinding and clenching). The authors' conclusion was that this questionnaire is useful for research and clinical purposes (van der Meulen *et al* 2006).

For the quantification of tooth wear as an indirect measure of SB, multiple tools are available (e.g., Wetselaar *et al* 2009). However, the clinician or researcher should realize that tooth wear is unreliable for quantification of the current status of the disorder, because tooth wear is a accumulation of dental tissue loss over time, as well as a mixture of all types of mechanical and chemical degradation (i.e., attrition, abrasion, erosion, and abfraction). It is therefore not always obvious which type of tooth wear is actually present. Hence, for the diagnosis of SB, tooth wear should only play an additional role (Carlsson *et al* 2003).

To objectively recognize and quantify SB, the use of polysomnography is recommended as the gold standard (Walters *et al* 2007; Lavigne *et al* 2008). In 1996, Lavigne *et al* proposed PSG cut-off criteria for the recognition of SB, to be used in combination with an overall evaluation of the patient. The proposed cut-off criteria were at least 4 so-called bruxism episodes of SB events per hour of sleep, or at least 25 bruxism bursts per hour of sleep. A bruxism burst should have a duration of at least 0.25 seconds, and can be either part of an episode or occur as an isolated burst. A bruxism episode is defined as a series of at least 3 bruxism bursts $> 0.25$ seconds but $< 2$ seconds (phasic), or 1 burst $> 2$ seconds (tonic), or a combination of these two (mixed), with a maximum interval between bursts of 3 seconds. Next to this, a recent history of tooth-grinding sounds for at least 3 nights per week during the last 6 months has to be present (Kato *et al* 2001a). This later criterion has again a subjective nature, which may hamper the valid recognition of SB. Further, the fluctuating character of SB (Lavigne *et al* 2001a) will inevitably be reflected in fluctuations in the outcome variables of the PSG recordings. In treatment studies, a finding of a difference in measured outcome variables between two recordings may thus be due to a natural fluctuation in SB or to a real therapeutic effect. This complicates the recognition of

SB and the monitoring of possible treatment effects with the use of PSG cut-off points. Therefore, in the study described in **Chapter 2** of this thesis, a thorough analysis of the time-variant nature and its implications for diagnosis and therapy evaluation by statistical means is given.

**Etiology**

Although the literature regarding the etiology of SB is difficult to interpret, which is, at least in part, due to existing disagreements about the definition and diagnosis of the condition, there is consensus about the multifactorial nature of SB. In general, four types of etiologic or causal factors (i.e., factors that produce or predispose), viz., morphologic, psychosocial, physiologic/biologic, and exogenous ones, are being described (Lobbezoo *et al* 2009). Morphologic factors include the anatomy of the orofacial skeleton and the morphology of the dental occlusion/articulation. There is increasing evidence that these factors play only a minimal role in the etiology of SB, if at all (Lobbezoo and Naeije 2001; Lobbezoo *et al* 2001; Lobbezoo *et al* submitted). Psychosocial factors, such as anxiety, stress, and personality, receive increasing attention and are probably important contributors to the etiology of SB (e.g., Ohayon *et al* 2001; van Selms *et al* 2004). Trauma, genetics, sleep-disordered breathing (e.g., obstructive sleep apnea), endogenous neurochemicals (e.g., dopamine, acetylcholine), and sleep-related arousals, are all considered to be centrally acting initiators and/or aggravators of SB and are part of the group of physiologic/biologic factors (Lavigne *et al* 2005; Lobbezoo *et al* 2006a; Lobbezoo *et al* 2009). Possible exogenous etiologic factors that may lead to a worsening of SB are, among others, the use of medications (e.g., selective serotonine reuptake inhibitors), the presence of neurological diseases (e.g., coma), the use of drugs (e.g., amphetamine, ecstasy), smoking (nicotine), and the intake of alcohol and caffeine (Lavigne *et al* 2008). A detailed overview on the etiology of SB is given in the review in **Chapter 3**.

Within the group of physiologic/biologic etiologic factors, SB is suggested to be associated with so-called EEG arousals during sleep (Macaluso *et al.* 1998; Kato *et al.* 2001b; Kato *et al.* 2003; Lavigne *et al.* 2003; Lobbezoo *et al* 2006a), and to have associations with other sleep-related movement disorders, like Periodic Leg Movements during Sleep (PLMS) and Restless Leg Syndrome (RLS) (Chaudhuri *et al.* 2001; Walters 2007). In **Chapter 4** of this thesis, the associations between SB, PLMS, and EEG arousals

are being studied to test the hypothesis that SB and PLMS are different expressions of the same underlying neurophysiologic mechanism.

**Management**

If the consequences of SB have a serious nature, treatment of the disorder may be indicated, for instance when a patient has temporomandibular pain, the sleep is disrupted, or if extensive tooth wear is present (Winocur and Lobbezoo 2010). 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. Practically, management strategies of SB can be separated into: 1. occlusal interventions (e.g., several types of splints, selective grinding, and orthodontic treatment); 2. pharmacotherapy (e.g., several centrally seizing medications and Botulinum toxin injections); 3. behavioral/psychological interventions (e.g., sleep hygiene, biofeedback, hypnosis, and cognitive behavioral therapy); and 4. physical therapy (e.g., muscle relaxation and strengthening exercises). 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 5** of this thesis, a comprehensive review of the effectiveness of the management strategies of SB is given.

In everyday general practice, occlusal splints are often indicated. They have already been used for a long time, not only for the management of SB, but also for that of temporomandibular pain. Many previous studies to the effect of splints have shown variable results (for an overview, see Dao and Lavigne 1998*)*. In that overview, it was suggested that the main effect of splints may be limited to a protection of the teeth against attrition (Dao and Lavigne 1998). However, it remains unclear whether or not there is a causal therapeutic effect of splints in the management of SB. Therefore, in **Chapter 6** of this thesis, a placebo-controlled study to the efficacy of occlusal splints on SB was conducted.

The pharmacological management of SB is increasingly studied (for an overview, see Winocur *et al*, 2003). Many of the previous investigations focused on the central dopaminergic neurotransmitter system (e.g., Lobbezoo *et al*, 1996; Lobbezoo *et al*, 1997a, b). In a randomized clinical trial (RCT), Lobbezoo et al (1997a) found that the short-term use of L-dopa gave a 40% decrease in SB activity. However, the long-term use of L-dopa by Parkinson patients is known to aggravate SB (Magee, 1970). Bromocriptine, a dopamine D2 receptor agonist, did have a positive, deceasing effect on SB in two case reports (Lobbezoo *et al* 1997b), but did not have such an effect in a subsequent RCT (Lavigne *et al* 2001b). In

other words, there is conflicting evidence regarding the role of dopaminergic substances in the management of SB. In the case study described in **Chapter 7** of this thesis, the efficacy of two treatment modalities on severe SB is described: i.e., an occlusal splint and the D1/D2 receptor agonist Pergolide, of which two different doses were tested.

**Synopsis**

The general aim of this thesis is to give a contemporary insight in aspects of the diagnosis, etiology, and management of SB. In this chapter, SB is introduced. **Chapter 2** deals with the consequences of the time-variant nature of SB outcome variables, using ambulatory PSG as a 'gold standard' diagnostic tool for the evaluation and monitoring of SB. A thorough search of the literature resulted in a review about the etiology of bruxism, which is described in **Chapter 3.** Associations of SB with other sleep-related movement disorders like PLMS are assessed in **Chapter 4. Chapter 5** contains a comprehensive review about the management of bruxism. The effects of some of the different therapies for SB, like occlusal, pharmacological, behavioral/psychological interventions, and physical therapy, are described in **Chapters 6 and 7.** In **Chapter 6,** the question was whether occlusal splints have an effect on SB in a double-blind placebo-controlled randomized clinical trial. The central dopaminergic influence on SB was tested in **Chapter 7**. In **Chapter 8**, a general discussion is given. Finally, in **Chapter 9,** a summary in English and in **Chapter 10** in Dutch is given, along with an acknowledgments section and the curriculum vitae of the author.

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