Sleep bruxism

Associations and comorbid conditions

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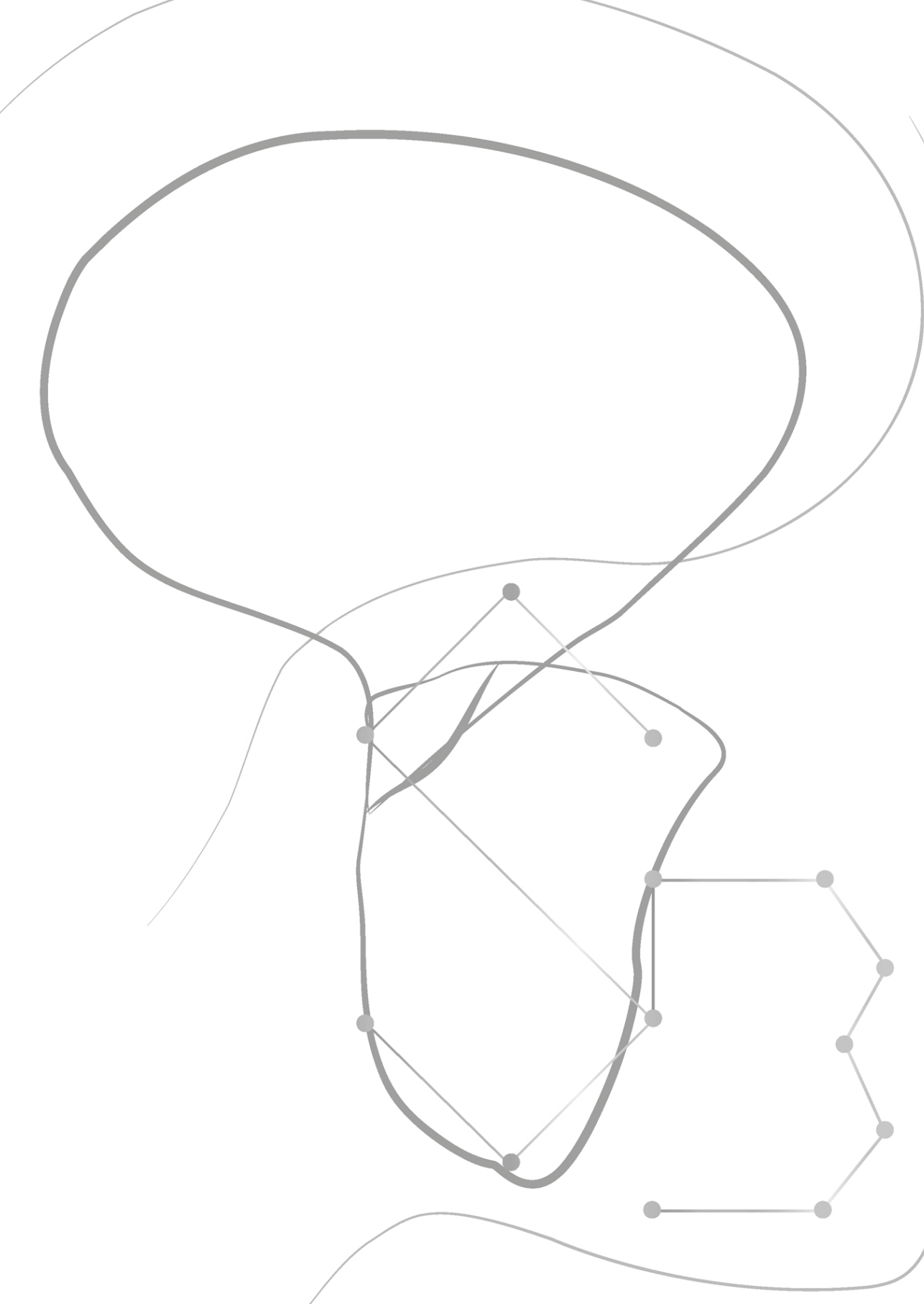

CHAPTER 1

GENERAL INTRODUCTION

SLEEP BRUXISM (SB)

Bruxism has been defined as "a repetitive jaw-muscle activity that can occur during sleep and wakefulness".[1] This single definition of generic bruxism has recently been separated into two definitions: one for sleep bruxism (SB) and another one for awake bruxism (AB).[2]

SB is defined as "a masticatory muscle activity during sleep that is characterized as rhythmic (phasic) or non-rhythmic (tonic) and is not a movement disorder or a sleep disorder in otherwise healthy individuals".[2] The prevalence of self-reported SB varies from 8.0% to 31.4% in the adult general population.[3] There is no difference in SB prevalence between both sexes, and the prevalence decreases with age.[3]

When assessing SB on polysomnographic recordings, episodes of rhythmic masticatory muscle activities (RMMA; one of the biomarkers of SB) frequently occur in relation to sleep micro-arousals.[4,5] The majority of RMMA events are preceded by a cascade of physiological events.[6,7] During sleep, abrupt shifts in the frequency of electroencephalographic (EEG) activity occur, that may or may not be accompanied by increases in heart rate or muscle tone, and are commonly denoted as arousals.[4,8,9] It has been noted that during light sleep, most SB episodes are preceded by a microarousal occurring 3-15 seconds before consecutive changes in heart rate and muscle tone.[5] The physiological activation begins with a rise in sympathetic-cardiac activity (4-8 minutes before RMMA), EEG activity (4 seconds before RMMA), heart rate (1 second before RMMA), muscle tone (0.8 second before RMMA), and, finally, RMMA.[5]

SB has a multifactorial etiology, including biological, psychosocial, and lifestyle factors. Examples of biological factors are genetics and sleep arousal.[10] Neurotransmitters, such as dopamine and serotonin, appear to play a role in SB genesis as well.[11] Variants in the HTR2A gene, that encodes one of the receptors for serotonin, were shown to be differentially represented in SB and control groups.[12] In addition, medications that alter dopaminergic and serotonergic neurotransmission, can aggravate SB events.[13] Besides these biological factors, psychosocial factors like stress, depression, and anxiety have been reported as risk factors for SB. However, the association between psychosocial factors and SB is inconclusive.[11,14,15] Finally, lifestyle factors, such as smoking, drinking alcohol, and caffeine intake, are associated with SB as well.[16-18] Use of nicotine, such as cigarette smoking and alcohol intake, may induce bruxism.[13] In addition, heavy smoking and heavy drinking increase the chance of having SB.[17,18]

SB and awake bruxism (AB)

AB is defined as "a masticatory muscle activity during wakefulness that is characterized by repetitive or sustained tooth contact and/or by bracing or thrusting of the mandible and is not a movement disorder in otherwise healthy individuals".[2] Its prevalence varies from 22.1% to 31.0% in the adult general population.[3]

SB and AB have a similar genetic origin, and they are associated with several similar risk factors.[19] However, SB and AB behave differently. SB is mainly characterized by grinding activity, while AB shows mainly clenching activity.[20,21] Both are also associated with common psychosocial factors, such as stress, depression, and anxiety,[15] but they are associated with these psychosocial factors with different degrees of certainty.[2] While the association between SB and psychosocial factors is controversial, many studies supported the association between AB and psychosocial factors.[14] All this leads to the question whether SB and AB are different entities. Therefore, **chapter 2** assesses the main characteristic of SB and AB, and **chapters 2** and **3** investigate whether SB and AB are associated.

SB, AB, and temporomandibular disorders (TMD)

Temporomandibular disorders (TMD) are one of the possible negative consequences of SB and AB (for more details, see **box 1**).[2] Both SB and AB have been considered as risk factors for TMD in adolescent and adult populations.[22] Even though SB is considered as a risk factor for TMD, the association between SB and TMD is still controversial.[23] The co-occurrence of SB and AB increased the chance of having painful TMD.[24] However, the exact role of psychosocial factors as well as that of SB and AB in TMD remains unclear. Therefore, **chapter 3** assesses the association of SB and AB with TMD and psychosocial factors.

Box 1. TMD is a group of conditions affecting the temporomandibular joints and masticatory muscles.[25] Prevalence of TMD is approximately 16.3%-25.6% in adult general population samples.[26,27] The prevalence is higher in women, peaks around middle age, and declines with ageing.[26,27] Its signs and symptoms include pain and dysfunction of the masticatory system.[25] TMD pain is one of the main reasons for patients to seek treatment.[28] TMD is a multifactorial condition that adopts the biopsychosocial model for diagnosis and treatment.[29] The biological factors include demographic variables, like age and sex, and oral behaviors, such as SB and AB.[30] Psychosocial factors also play an important role in the etiology of TMD.[31] TMD has a positive association with insomnia and OSA as well.[32,33] Diagnostic criteria for TMD (DC/TMD) have been developed, consisting of a biological axis (axis I) and a psychosocial axis (axis II).[29]

Assessment of SB and AB

There are three ways for the assessments of SB and AB, viz., self-report, clinical examination, and electromyography. The diagnostic grading of bruxism is based on these three approaches, namely possible, probable, and definite, respectively.[2] Self-report is obtained by questionnaires, the patient's complaints, and the report from a sleep partner.[2] Patient's complaints, such as muscle fatigue and temporal headache, could point towards patients with SB.[34] Self-report is widely used in the clinical setting and in epidemiological research. Clinical examination considers the clinical features related to bruxism, such as masseter hypertrophy, linea alba buccalis, i.e., ridging on the buccal mucosa, and indentations on the tongue or lip.[2] Tooth wear and repetitive failure of restorations, prostheses, and dental implant are also clinical diagnostic indications for bruxism. However, none of them form a direct confirmation of current SB activity.[34]

The Standardized Tool for the Assessment of Bruxism (STAB) yields a protocol to assess SB and AB in a comprehensive manner.[35] The protocol considers both self-report and clinical evaluation. The newly developed BruxScreen is proposed for screening purposes, epidemiological study, and clinical application. The aim of this tool is to assess bruxism effectively according to the A4 principle, i.e., accurate, applicable, affordable, and accessible. It contains a modification of the Oral Behaviours Checklist (OBC) and other self-report questionnaires. In addition, the BruxScreen includes a clinical assessment form for the clinician to investigate intra- and extra-oral signs of bruxism, including tooth wear. This screener focuses only on the assessment of SB and AB, while the original OBC focuses on a wide variety of oral behaviors, including singing, yawning, etc.[2] The BruxScreen has already been tested for its face validity and is ready to be applied for further psychometric testing, followed by large-scale epidemiological research and clinical application.[36]

Notwithstanding these important recent developments in the assessment of SB and AB, the gold standard for the assessment of SB is still polysomnography (PSG),[2] while the gold standard of AB assessment remains electromyography (EMG) together with ecological momentary assessment (EMA).[2] EMG activity with an amplitude of 10-20% of the maximum voluntary contraction is considered as an RMMA episode.[37,38] Alternatively, an RMMA episode may be regarded as an EMG activity that surpasses two times the amplitude when relaxing.[39,40] Each EMG burst must be separated by less than two seconds in the same RMMA episode, and the new RMMA episode is counted after at least three seconds of stable baseline EMG.[40,41] There are three different types of an RMMA episode, namely, phasic, tonic, and mixed types. A phasic episode contains at least 3 EMG bursts, and each burst lasts 0.2-2 seconds. A tonic episode is an EMG burst that lasts more than 2 seconds. A mixed episode is a combination of phasic and

tonic episodes.[37] The 1996 criteria, formulated by Lavigne et al., described as the cut-off criteria of SB at least 4 episodes/hour or 25 bursts/hour of sleep, and at least 2 episodes with grinding sounds.[37] Rompré et al. (2007) suggested a lower cut-off point of 2 episodes/hour for moderate SB.[42] However, these cut-off points were developed for research purposes, and clinical consequences related to SB could not be identified by using these cut-off points.[43] Therefore, the question has been posed whether cut-off points should actually be implemented.

The validity of self-report, clinical examination, and EMG assessments compared with PSG has been investigated.[44] Self-reported SB has a low sensitivity but medium to high specificity.[44] There were inconsistencies between self-report and PSG: SB was established more frequently with self-report than with PSG, both in a TMD population and in the general population.[45,46] The prevalence of SB as assessed by self-report and confirmed with PSG was 5.5%, while that of self-reported SB was 12.5% in the same population.[45] Even though positive self-report by a sleep partner shows high accuracy for screening on the presence of SB,[46] self-report and clinical assessment can be screening tools for non-bruxers, but not for definitely identifying SB.[44,46] It should be noted that PSG has been suggested to be affected unfavorable by the so-called first night effect.[38] However, the study of Hasegawa et al. showed no such first night effect of PSG.[47] Another issue is the night-to-night variability that has been suggested for SB measured by EMG/PSG. Indeed, there was a high variability of the SB index measured with a portable EMG device across nights.[48] Thus, it is suggested to use a 'cut-off band' around the cut-off point for SB to take this variability into account.[38] As for awake bruxism, self-reported AB was highly correlated with the clinician's diagnosis, so self-report with clinical assessment could be an accurate tool for establishing awake clenching.[49]

Management of SB and AB

In the absence of an evidence-based management protocol for bruxism, the management of SB and AB in clinical practice follows the 'triple-P' approach, which includes pep talk, plates, and pills.[50] Pep talk, or counselling, is a common management strategy for both SB and AB. Sleep hygiene and self-monitoring trained by biofeedback from an EMG device are advised for SB and AB, respectively.[51,52] Plates refer to the frequently used occlusal appliances (hard acrylic occlusal splints) for SB management. However, the effect of occlusal splints on reducing SB activity is short-term,[53] and there are only weak evidences of their SB-reducing effect.[54] The main goal of occlusal appliances is to prevent the covered teeth and their opposing teeth from wear and restoration failures.[51] For the application of pills, or medications, amitriptyline, clonazepam, and clonidine could reduce SB.[13] However, more research on their efficacy and safety is still needed.[13] **Chapter 4** assesses whether SB and AB change after receiving counselling, with or

without additional management strategies like occlusal splints, physiotherapy, and/or psychological therapy, as part of a regular TMD treatment protocol.

SB and sleep disorders

Even though TMD, tooth wear, and restoration failure are considered as negative consequences of SB,[2] in this present decade, the view upon SB as a disorder has changed. There is increasing evidence that SB does not only play a role as a risk factor but also as a protective one. SB is considered as a protective factor against some sleep disorders like obstructive sleep apnea (OSA) and against the negative oral consequences of sleep-related gastroesophageal reflux disease (GERD).[55] Since arousal and microarousal are also related to other sleep disorders, such as restless leg syndrome/periodic limb movement and insomnia, SB has been found to be a comorbid condition of those sleep disorders (for more details on insomnia, see **box 2**).[45,56,57] Therefore, **chapters 5** and **6** of this thesis focus on the association between SB and insomnia in specific and general population samples. In addition, psychosocial factors that are commonly found in both SB and insomnia are investigated.

Box 2. Patients with the sleep disorder 'insomnia' have difficulty initiating sleep, difficulty maintaining sleep, and/or early morning awakening at least three nights a week, more than three months, and these symptoms interfere daily life activities.[58] The prevalence of insomnia is 7-23% in adult general population.[59] Insomnia has a close relationship with anxiety and depression.[60] In addition, an association between insomnia and SB has been found in the literature.[45] It has been suggested that sleep arousal forms a link between SB and insomnia.[57] The diagnosis of insomnia requires a clinical evaluation that includes sleep history, medical history, substance usage history, and psychiatric history.[61]

CONTENT OF THIS THESIS

The general aim of this thesis is to assess the associations of SB on the one hand and AB, psychological factors, TMD, and insomnia on the other hand, using self-report and instrumental techniques to establish the presence of bruxism.

Chapter 1 presents a general introduction, including the definition, epidemiology, etiology, negative consequences such as TMD, the assessment, and management of SB and AB, as well as the association between SB and sleep disorders.

Chapter 2 assesses if objectively measured SB and AB are associated with each other in the same individuals.

Chapter 3 assesses whether the associations of self-reported SB and AB with TMD are different in a TMD-patient population.

Chapter 4 presents the changes of SB and AB during TMD management in a TMD-patient population sample.

Chapter 5 focuses on self-reported SB and its associations with sleep disorders and psychosocial factors in a specific population sample.

Chapter 6 focuses on SB and its associations with sleep disorders and psychosocial factors in a general population sample.

Chapter 7 discusses the main findings of this thesis, explains the clinical implications of this thesis' findings, and provides suggestions for further research.

Chapters 8, 9 and, **10** presents summaries in English, Dutch, and Thai, respectively.

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