Sleep bruxism: contemporary insights in diagnosis, etiology and management

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Chapter 3

Review article: Bruxism: its multiple causes and its effects on dental implants – an updated review.

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Chapter 3

Summary

There is a growing interest in bruxism, as evidenced by the rapidly increasing number of papers about this subject during the past 5 years. The aim of the present review was to provide an update of two previous reviews from our department (one about the aetiology 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 brux ism 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 aetiology of bruxism and its purported relationship with implant failure.

Keywords: aetiology, bruxism, causality, dental implants, failure, overload, morphology, pathophysiology, psychology, review

Introduction

Bruxism is a movement disorder of the masticatory system that is characterized, among others, by teeth grinding and clenching, during sleep as well as during wakefulness (1, 2). Bruxism has a prevalence in the general adult population of about 10% and is usually regarded as one of the possible causative factors for, among others, temporomandibular pain, tooth wear in the form of attrition, and loss of dental implants (3). These possible musculoskeletal and dental consequences of bruxism illustrate the clinical importance of this disorder. Importantly, it should be borne in mind that there is still a lack of agreement about, for example, the definition of bruxism, which makes it sometimes difficult to unequivocally interpret the available evidence.

During the past decades, bruxism has been studied extensively, and many research papers and review articles have been published. To illustrate this, A MEDLINE search was performed on 28 April 2005, using the National Library of Medicine's (NLM) Medical Subject Headings (MeSH) Database and PubMed. The search term 'Bruxism'[MeSH Terms] OR bruxism [Text Word] yielded 1773 papers, 202 of which were reviews. When using the truncated search term bruxi*, thereby turning off automatic term mapping and the automatic explosion of MeSH terms, 1791 papers were found, 206 of them being reviews. The overlap between these two searches was 100%. A pure MeSH search on this subject (viz. 'bruxism'[MeSH]) resulted in 1588 papers, including 172 reviews. About 20–30% of the papers, found with any of these three search strategies, were published during the past 5 years; the remaining papers, between 1966 and 2000. This shows, that there is a growing interest in bruxism.

Most of the reviews that were found with the above-described search strategies have a broad nature, covering many aspects of bruxism, like its definitions, epidemiology, diagnostic procedures, aetiology/pathophysiology, concomitant disorders, clinical consequences, various therapeutic approaches and prognosis. Relatively few of the review articles focus on specific aspects of bruxism. Recently, we published two 'indepth' focused reviews: one about the aetiology of bruxism (4) and the other about the possible role of bruxism in the failure of dental implants (5). Unfortunately, in these review articles, no controllable PubMed search was described, thus leaving the readers ignorant of the completeness of the

review. Therefore, because of the above-substantiated growing interest in bruxism during the past 5 years, the aim of the present review was to provide an update of both previous reviews and to describe the details of the literature search strategies used.

Aetiology of bruxism

Summary of review by Lobbezoo and Naeije (4)

The literature, which is so far published about the aetiology of bruxism, is often difficult to interpret. In part, this is because of the persisting disagreement about the definition and diagnosis of this disorder. However, there is consensus about the multifactorial nature of the aetiology of bruxism. Besides peripheral (viz. morphological) factors, central (viz. pathophysiological and psychosocial) factors can be distinguished. In the past, morphological factors, like occlusal discrepancies and deviations in the anatomy of the bony structures of the orofacial region, have been considered the main causative factors for bruxism. Nowadays, these factors are thought to play only a small role, if at all. Recent focus is more on the pathophysiological factors. For example, bruxism has been suggested to be part of a sleep arousal response, the oral motor event either preceding or following the arousal. In addition, bruxism appears to be modulated by various neurotransmitters in the central nervous system. More specifically, disturbances in the central dopaminergic system have been described in relation to bruxism. Further, factors like medication, (illicit) drugs, genetics, trauma, and neurological and psychiatric diseases may be involved in the aetiology of bruxism. Psychosocial factors like stress and personality are frequently mentioned in relation to bruxism as well. However, research to these factors comes to equivocal results and needs further attention. Taken all evidence together, bruxism seems to be mainly regulated centrally, not peripherally.

Search strategy

To update the review by Lobbezoo and Naeije (4) about the aetiology of bruxism, a MEDLINE search was performed on April 28, 2005, using the NLM's MeSH Database and PubMed. As search term, 'Bruxism' [MeSH] was used. The term was exploded as to include

'Sleep Bruxism', a term which is found below 'Bruxism' in the MeSH tree. The search was limited to the past 5 years and yielded 330 papers, 48 of them being reviews. On the basis of the titles, 68 research articles and 11 reviews were selected for their possible relevance to the subject of this review (viz. the aetiology of bruxism), thereby avoiding overlap with the set of references used by Lobbezoo and Naeije (4). As a next step, the abstracts (or, when not available, the full-length papers) of the 79 selected papers were read as to establish the papers' applicability to this review. Of these papers, 17 were excluded because they turned out to deal with subjects like tooth wear and myoclonus, that are outside of the main focus of this updated review. Hence, 62 papers remained for inclusion in the below-given updated review.

Updated review

As stated in the above-given summary of the review by Lobbezoo and Naeije (4), the factors that may play a role in the aetiology of bruxism can be divided into three categories: morphological, pathophysiological and psychosocial factors. Relatively few of the papers, selected for inclusion in this updated review, deal with morphological factors (approximately 10%), while only slightly more papers have the role of psychosocial factors in the aetiology of bruxism as their main focus (approximately 20%). The vast majority of the selected papers (approximately 70%) deal with possible aetiological factors that can be classified among the pathophysiological ones. These percentages corroborate the commonly observed trend in bruxism research, away from a main focus on occlusion and towards a more biomedical/biopsychosocial point of view [see, e.g. the reviews by Kato et al. (6–8); De Laat and Macaluso (9); Lavigne et al. (10); and Lobbezoo et al., (3)]. Below, the possible role of occlusal factors will be discussed first, followed by that of various psychosocial factors. Finally, several pathophysiological factors will be described in relation to their purported role in the aetiology of bruxism. As in the review by Lobbezoo and Naeije (4), in the present update, unless otherwise specified, bruxism will be considered the combination of all parafunctional clenching and grinding activities, exerted both during sleep and while awake, because these different phenomena are still not, or only inadequately, distinguished in most of the selected papers.

Occlusal factors. Several occlusal factors (e.g., large and/ or inverse overjets and overbites) were suggested to be related to self-reported bruxism in a study with children (11). In contrast to the recent insights as reviewed by Lobbezoo and Naeije (4), Griffin (12) still state that for an effective management of bruxism, establishment of harmony between maximum intercuspation and centric relation is required. However, most studies to this subject now agree that there is no, or hardly any relationship between self-reported and/or clinically established bruxism on the one hand and occlusal factors on the other hand, neither in adult samples (13–15) nor in children samples (16). Importantly, Manfredini et al. (17) state, on the basis of a review of the literature, that there is still a lack of methodologically sound studies to definitively refute the importance of occlusal factors in the aetiology of bruxism. Therefore, future research to this subject should include more objective techniques to establish the presence or absence of bruxism (e.g. electromyography or polysomnography), using the proper design for studies to cause-and-effect relationships, viz. prospective, longitudinal trials.

Psychosocial factors. Rosales et al. (18) evoked emotional stress in rats by letting them observe other rats that underwent electrical foot shocks in a neighbouring cage. Compared with rats that did not observe the foot-shocked rats, the 'observing' rats had high levels of brux-like masseter muscle activity. Although it is unknown whether this brux-like behaviour in rats is in any way related to bruxism in man, Slavicek and Sato (19) consider such behaviour in experimental animals as an emergency exit during periods of psychic overloading. The findings and suggestions of these animal studies are in line with many observations in humans that there may be a causal relationship between psychosocial factors like stress on the one hand and bruxism on the other, as reviewed by Lobbezoo and Naeije (4). Importantly, these authors state that the role of psychosocial factors in the aetiology of bruxism is far from clear, and that there is a need for more controlled studies to this subject. Since the publication of Lobbezoo and Naeije (4), several studies to this subject have been published. Unfortunately, none of them has a conclusive nature because of the absence of prospective, large-scale longitudinal trials (see above).

Taking into account these limitations of the evidence published during the past 5 years, the following view on the role of stress and other psychosocial factors in the aetiology of bruxism emerges from the selected papers. Following from cross-sectional (case–control) studies, bruxers differ from healthy controls in, among others, the presence of increased levels of hostility (20) as well as in the presence of depression and stress sensitivity (13, 17).

Bruxing children are apparently more anxious than non-bruxers (21), while 50-year-old bruxers more frequently report, among others, being single and having a higher educational level (22). A series of papers about the presence of bruxism and psychosocial factors among the employees of the Finnish Broadcasting Company describes that self-reports of bruxism may reveal, among others, ongoing stress in normal work life (23) and dissatisfaction with one's work shift schedule (24). Therefore, Ahlberg et al. (25) state that factors like perceived stress should be taken into account when treating bruxism-related temporomandibular pain. A multi-national, large-scale population study to sleep bruxism revealed 'highly stressful life' as a significant risk factor (26). Finally, in a longitudinal case study by Van Selms et al. (27), it was demonstrated that daytime clenching could significantly be explained by experienced stress, although both experienced and anticipated stress were unrelated to sleep-related bruxism as recorded with ambulatory devices (27, 28). Taken the findings of all these studies together, the body of evidence for a possible causal relationship between bruxism and various psychosocial factors is growing, though not yet conclusive. Hence, there remains a need for more, well-designed studies to this subject.

Pathophysiological factors. As mentioned above, the vast majority of the selected papers for this updated review deal with possible pathophysiological factors. Many of these are sleep-related. While Nagels et al. (29) report a significantly lower percentage slow wave sleep in bruxers than in healthy controls, other authors report macrostructural sleep quality and architecture to be normal in bruxism patients (28, 30). Interestingly, and in contrast to one's expectations, experimental deprivation of slow wave sleep (this sleep stage being the one during which the least bruxism activity reportedly occurs) did not significantly influence sleep bruxism (31). In contrast to these macrostructural sleep studies, in a study to sleep microstructure, the sleep of bruxers was found to be characterized by a low incidence of K-complexes and K-alphas (30). This illustrates the importance to include microstructural analyses of sleep in future studies to sleep bruxism.

In relation to sleep quality and architecture, bruxism and habitual snoring were found to be closely related (32). Ohayon et al. (26) even report an increased risk of reported sleep bruxism in the presence of loud snoring and obstructive sleep apnoea syndrome (OSAS). According to Sjoholm et al. (33), these relationships are because of the disturbed sleep of habitual snorers and OSAS patients. However, if these relationships indicate a true physiological association is still unknown.

As already summarized by Lobbezoo and Naeije (4), sleep bruxism may be considered part of an arousal response. During the past 5 years, several papers were published on this subject. First of all, Kato et al. (34), using a case–control design, found evidence for the suggestion that sleep bruxism is an oromotor manifestation secondary to the microstructural sleep event 'micro-arousal' (i.e. an abrupt change in the frequency of cortical EEG that is occasionally associated with motor activity). Similarly, experimentally induced micro-arousals were followed by masticatory motor events in all sleep bruxers in another study by Kato et al. (35). Based on a review of the literature, Kato et al. (8) suggest a sequence of events from autonomic (cardiac) changes and brain cortical activation (sleep arousal) to the genesis of sleep-related masticatory muscle activities (bruxism). Interestingly, associations have also been observed between bruxism activities on the one hand and a supine sleeping position, gastroesophageal reflux, episodes of decreased esophageal pH, and swallowing on the other (36–39). The exact temporal relationship of these factors to bruxism is, as yet, unknown. Future studies should therefore aim at unravelling an all-embracing sequence of events.

Certain neurochemical factors, medications and (illicit) drugs were described in detail in relation to bruxism by Lobbezoo and Naeije (4). During the past 5 years, the body of evidence of their role has been growing gradually, although its conclusive nature is still controversial (40). Several papers that were selected for this updated review deal with the influence of selective serotonine reuptake inhibitors (SSRIs) on bruxism. SSRIs have an indirect influence on the central dopaminergic system, which is the system that is thought to be involved in the genesis of bruxism (4). Lobbezoo et al. (41) state, that SSRIs may cause bruxism after long-term usage. The case reports of Jaffee and Bostwick (42), Wise (43) and Miyaoka et al. (44) corroborate this statement for the use of venlafaxine, citalopram and fluvoxamine, respectively. Another case report describes severe bruxism in relation to an addiction to amphetamine, which can be explained through amphetamine's disturbing influence on the dopaminergic system (45). In line with this report, the amphetamine-like medications that are used in the management of attention deficit hyperactivity disorder (ADHD), like methylphenidate, have bruxism as a possible side effect, as shown in a case–control study by Malki et al. (46). Also, the amphetamine-like substance XTC reportedly has bruxism as a side effect (47). Based on a study with rats, Arrue et al. (48) give a possible explanation for this side effect of XTC, viz. the XTC-induced reduction of the jaw-opening reflex. Finally, bruxism was found more frequently in heavy drug addicts (49) as well as in

smokers (25, 26). According to Ohayon et al. (26), smokers are at higher risk than non-smokers of reporting sleep bruxism, as are drinkers of alcohol and caffeine. In short, all of the above-summarized papers corroborate the conclusion of Lobbezoo and Naeije (4), viz. that disturbances in the central dopaminergic system can be linked to bruxism. However, as stated by Winocur et al. (40), more controlled, evidence-based research on this under-explored subject is needed. Further, it should be noted that information about dopaminergic substances in relation to the aetiology of bruxism is more readily available than that about other neurochemicals. Thus, although it may seem from the available evidence that mainly the dopaminergic system plays a role in the aetiology of bruxism, the lack of focus on other substances in the literature as well as the presence of many possible interactions between dopamine and other neurochemicals indicates the need for more research.

As already reviewed by Lobbezoo and Naeije (4), it remains unclear whether or not bruxism is, to a greater or lesser extent, genetically determined. In their review, Hublin and Kaprio (50) take the stand that genetic effects have a significant role in the origin of bruxism, although the exact mechanisms of transmission are still unkown. Bruxism was also shown to share a common genetic background with sleeptalking, another parasomnia (51). Recent publications thus favour the role of genetics in the aetiology of bruxism. As stated before, however, the exact genetic mechanisms still need to be unravelled in future studies.

Finally, many of the papers that were selected for possible use in this updated review deal with diseases and trauma in relation to bruxism. To start with trauma, brain damage was described as a possible cause for bruxism in the case series and case report by Millwood and Fiske (52) and Pidcock et al. (53), respectively. Further, a host of diseases of mainly neurological and psychiatric nature has been linked to the aetiology of bruxism, viz. basal ganglia infarction (54), cerebral palsy (55, 56), Down syndrome (57), epilepsy (58), Huntington's disease (59, 60), Leigh disease (61), meningococcal septicaemia (62), multiple system atrophy (63), Parkinson's disease (64), post-traumatic stress disorder (65, 66) and Rett syndrome (67). With the exception of the study by Rodrigues dos Santos (55) on cerebral palsy, which has a case–control design, all other references in the afore-given list of diseases in relation to the aetiology of bruxism are case series or case reports. This indicates, that a lot of well-designed research still needs to be performed to further evaluate the nature of the relationships that were found between bruxism on the one hand and diseases and trauma on the other.

Taken all the above evidence together, it can be concluded that most papers that were published during the past 5 years about the aetiology of bruxism have a corroborative nature in relation to the review by Lobbezoo and Naeije (4). The most promising developments that yield new points of view on this subject can be found in the research on sleep-related aetiological factors, especially sleep arousal. This factor has been studied in well-designed experiments and yielded an interesting model for the genesis of sleep bruxism. Future research should try to further elaborate, test and validate this model. Preferably, this should be performed by taking into account other promising aetiological mechanisms, like psychosocial and neurochemical ones.

Dental implants and bruxism

Summary of review by Lobbezoo et al. (5)

Bruxism is generally considered a clinical problem, which may have detrimental consequences for dental, periodontal and musculoskeletal tissues. Bruxism has also been suggested to cause excessive (occlusal) load of dental implants and their suprastructures, which may ultimately result in bone loss around the implants or even in implant failure. Not surprisingly, bruxism is therefore often considered a contraindication for implant treatment, although the evidence for this is usually based on clinical experience only. So far, studies to the possible cause-and-effect relationship between bruxism and implant failure do not yield consistent and specific outcomes. This is partly because of the large variation in the literature in terms of both the technical aspects and the biological aspects of the study material. Although there is still no proof for the suggestion that bruxism may cause an overload of dental implants and of their suprastructures, Lobbezoo et al. (5) conclude that a careful approach is nevertheless recommended. There are a few practical guidelines as to minimize the chance of implant failure. Besides the recommendation to reduce or eliminate bruxism itself, these guidelines concern the number and dimensions of the implants, the design of the occlusion and articulation patterns, and the protection of the final result with a hard occlusal stabilization splint.

Search strategy

For an update of Lobbezoo et al. (5) about the possible role of bruxism in the failure of dental implants, a MeSH search strategy was performed, using the following query: 'Bruxism' [MeSH] AND ('Dental Implants' [MeSH] OR 'Dental Abutments' [MeSH] OR 'Dental Prosthesis, Implant-Supported' [MeSH] OR 'Dental Implantation' [MeSH]). This query yielded 41 papers, four of them being reviews. Of these 41 papers, 16 were already included in the paper by Lobbezoo et al. (5). Another 13 papers were judged as non-applicable for use in the current review. Of the remaining 12 papers, the titles suggested a possible relevance to the subject of this review (viz. the role of bruxism in implant failure). In addition to this search, the titles of the papers from the above-described MeSH search ('Bruxism' [MeSH]) over the past 5 years (see Aetiology of bruxism – Search strategy) were judged, which yielded another two papers of which the titles suggested their possible relevance to the subject of the current review. Hence, 14 papers were selected on top of the papers that were already included in the review by Lobbezoo et al. (5). As a next step, the abstracts of these 14 papers were read as to establish the papers' applicability to this review. Three papers turned out not to deal with dental implants after all, while two other papers mainly dealt with prevalence rates of biomechanical problems and of bruxism itself in dental implant patients. These five papers were further disregarded. The remaining nine papers were included in the below-given updated review, regardless of them being research papers, case report, or reviews.

Updated review

The nine papers that were selected for this update using the above-described search strategy could be classified as follows: one editorial (68); three (sets of) expert opinions (69–71); two case reports (72, 73); one (prospective) case series (74) and two non-systematic reviews (75, 76).

Without exception, these publications' conclusions regarding causality and their practical guidelines for the use of dental implants in bruxism patients fit into the picture as sketched by Lobbezoo et al. (5). For example, on the basis of a (non-systematic) review of the literature, Jacobs and De Laat (75) also conclude that there is no direct causal relation between bruxism and implant failure. Further, Engel and Weber (74) corroborate the recommendation of Lobbezoo et al. (5) to proceed carefully when planning implant

procedures in bruxists. In line with this recommendation, Tagger-Green et al. (76) state that good clinical examinations and correct treatment plans (i.e. taking into account factors like location and size of the implants) may reduce the risk of implant failure. In the case report by Ganales et al. (73), the predictability of the clinical results following optimal treatment planning is illustrated in a bruxist receiving dental implants. The recommendation of Lobbezoo et al. (5) to protect the final treatment result in bruxers with implants by means of a hard stabilization splint for night-time use (night guard), as to minimize (or even negate) the lateral destructive forces, is also given in an anonymous editorial (68) as well as in a case report (72). Further, support for this recommendation can be found in the (sets of) expert opinions of Schneider et al. (70) and Gittelson (71).

Despite the apparent lack of evidence, it may be good clinical practice to adopt the conclusions and practical guidelines of Lobbezoo et al. (5). The recommendation for future research to specifically address the possible relationship between bruxism and dental implant failure, using high-quality study designs, still holds out firmly against time, the more so because most of the above-included papers have a low strength of evidence according to the grading system of the Oxford Centre for Evidence-Based Medicine.

Conclusion

The aim of this review was to provide an update of the reviews by Lobbezoo and Naeije (4) and by Lobbezoo et al. (5). From both updates, it followed that the conclusions of these previous reviews are left unchanged. In other words: 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 both the aetiology of bruxism and to its purported relationship with implant failure. Evidence-based information about these subjects would be welcomed in the dental clinic, where the causes and consequences of bruxism still frustrate (and fascinate) dentists.

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