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Sleep bruxism: contemporary insights in diagnosis, etiology and management

van der Zaag, J.

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Chapter 7

Effects of pergolide on severe sleep bruxism in a patient experiencing oral implant failure.

**Jac. van der Zaag, Frank Lobbezoo, Gordon PGL van der Avoort,
Darrel J Wicks, Hans L Hamburger & Machiel Naeije.**

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Summary

In the patient described in this study, oral implants failed as a probable consequence of severe, polysomnographically confirmed sleep bruxism. As this patient had the wish to be re-implanted after this failure, we decided to try diminishing the frequency of bruxism and duration 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 sleep bruxism.

Keywords: sleep bruxism, polysomnography, occlusal stabilization splint, pergolide, case report

Introduction

Bruxism can be defined as a stereotyped oral movement disorder, characterized by daytime and/or sleep-related teeth grinding and/or clenching (1, 2). The disorder is usually held responsible for clinical problems like attrition [i.e. mechanical wear, resulting from mastication or parafunction, that is limited to contacting surfaces of the teeth (3)], pain in the masticatory muscles and/or the temporomandibular joints (4), and overload of oral implants and of their suprastructures (5), although convincing evidence for the validity of these possible causal relationships is still lacking (6–8).

Bruxism can be influenced by counselling (e.g. addressing the patient's awareness of daytime clenching; sleep hygiene instructions in case of sleep bruxism) and occlusal appliances (9). These latter devices probably function more like protectors of the remaining teeth rather than actually diminish bruxism (10). Furthermore, several pharmacological interventions for sleep bruxism have been suggested (e.g. centrally acting drugs such as diazepam and dopamine-related medications) (11). However, in the absence of definitive evidence, the appropriate treatment of bruxism is still a matter of debate.

In the present report, the efficacy of two treatment modalities on severe sleep bruxism is described in a patient experiencing oral implant failure. In this open-label study, an occlusal stabilization splint and two doses of a dopamine D1/D2 receptor agonist were tested, using polysomnographically determined outcome measures for the quantification of sleep bruxism (12, 13).

Clinical report

In July 1998, a 51-year-old man consulted the clinic of the Department of Oral Function of ACTA, Section of Prosthetic Dentistry & Oral Implantology, with a wish for oral implants in the left upper jaw to improve his aesthetics and oral function. Following the routine oral implant treatment planning protocol of the clinic, three 3i®* osseotite self-tapping fixtures*, with lengths of 13, 10 and 8.5 mm and diameters of 3.75, 3.35 and 5.0 mm, respectively, were finally placed at the former tooth sites of elements 25 to 27. After a healing phase of almost 1 year, the implants appeared to be firmly anchored, as assessed clinically and radiographically. In June 1999, a healing abutment operation was performed. Three 3i®abutments with lengths of 4, 4.4 and 6 mm, respectively, were placed on the implants.

*3i, Palm Beach Gardens, FL, USA.

One month later, a bridge was made on the implants, to the full satisfaction of both the patient and the prosthodontist.

In May 2000, the patient again consulted the clinic of the Department of Oral Function of ACTA, this time with the complaint of severe pain in the left upper jaw area. Radiographs (Fig. 1) showed that two of the three fixtures were broken, viz. those at the former tooth sites of elements## 25 and 26, while the anchorage of the third fixture was severely compromised. Consequently, we decided to remove all three fixtures, along with the suprastructure (Fig. 2).

Even though the routine oral implant treatment planning protocol [including negative answers to questions## 15.c. and 15.d. of the history questionnaire of the Research Diagnostic Criteria for Temporomandibular Disorders (14, 15) and the presence of a maximum occlusal tooth wear score of 2 (i.e. loss of clinical crown height of $\leq 1/3$) (16) – not abnormal for a middle-aged man] had not revealed signs or symptoms of severe

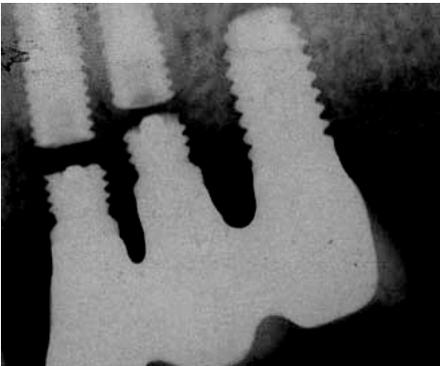


Fig. 1. Radiograph of the upper-left (pre-) molar region, showing fractured fixtures at the former tooth sites of elements# 25 and 26, and loosening of the fixture at the former tooth site of element# 27.



Fig. 2. The removed suprastructure plus its three fixtures.

parafunctional activities, the dental team suspected the presence of bruxism as a possible cause for the fractures, especially because manufacturing defects were excluded by the laboratory of the Department of Basic Dental Sciences of ACTA, Section of Material Sciences, and no other known risk factors for implant failure were obviously present (e.g. smoking). For that reason, and because this patient had the wish to be re-implanted, the patient was referred to the Section of Oral Kinesiology of the departmental clinic. There, a preliminary diagnosis of mainly sleep-related bruxism was established on the basis of the combined outcome of an extensive history taking [including several positive answers to a 12-item oral parafunctions questionnaire (17)] and clinical examination, that did not only include an assessment of occlusal wear (see above) but also a thorough inspection of the oral soft tissues (uncovering signs of tooth clenching, like hyperkeratotic ridges in the cheeks, tongue scalloping, and incisal impressions in the lips) and hard tissues (uncovering wear facets that intermaxillary matched in several eccentric jaw positions).

To confirm this preliminary diagnosis of mainly sleep-related bruxism as well as to evaluate the efficacy of the subsequent treatment modalities (see below), six overnight polysomnographic (PSG) recordings were performed between October 2000 and May 2002. Possible daytime bruxism was not monitored, because history taking did not indicate the presence of that disorder. The interval between successive PSG recordings varied from about 1 month to almost 1 year (see Table 1, first column). The recordings took place in a quiet, dark, single room of the sleep laboratory of the Department of Clinical Neurophysiology of the Slotervaart General Hospital in Amsterdam, where a Biosaca sleep-recording unit[†] was used with the following montage protocol:

- Electroencephalography (EEG; C3A2;O2A1);
- Electromyography (EMG; right and left masseter muscle; submental area; right anterior tibialis muscle);
- Electro-oculography (EOG; right and left);
- Electrocardiography (ECG; heart rate);
- Oxygen saturation (SaO₂);
- Body position.

[†]Ortivus AB, Täby, Sweden.

Table 1. Summary of the polysomnographic (PSG) recordings: recording dates, related treatment modalities, and sleep bruxism outcome measures

Event	Date / Duration	Epi/h	BTI (%)
PSG 1	27 October 2000	25.4	8.2
Stabilization splint	1 month		
PSG 2	24 November 2000	24.1	3.4
Washout	2 months		
Pergolide 0.3 mg	2 months		
PSG 3	23 March 2001	4.7	0.7
Pergolide 0.5 mg	1 month		
PSG 4	18 April 2001	9.7	1.3
Washout	1 month		
PSG 5	17 May 2001	5.7	0.8
Follow-up	1 year		
PSG 6	2 May 2002	11.0	1.7

Epi/h, number of sleep bruxism episodes per hour of sleep; BTI, bruxism time index.

Each masseter EMG signal was appropriately filtered (50 Hz notch; 3 Hz high pass; 100 Hz low pass) and was digitized at 256 Hz. The PSG recordings were analysed with Compumedics Replay sleep analysing software[‡]. All sleep analyses were performed according to Rechtschaffen and Kales (18), using 30-s epochs. The resulting hypnograms (i.e. schematic summaries of the sleep structure of single PSG recordings) were judged to have a normal structure by an experienced neurologist who specializes in sleep disorders (HLH). The analysis of sleep bruxism was performed according to the criteria introduced by Lavigne et al. (12). As sleep bruxism outcome measures, the number of bruxism episodes per hour of sleep (Epi/h) and the bruxism time index [BTI; i.e. the total time spent in bruxing divided by the total sleep time, times 100% (13)] were determined and averaged

[‡]Compumedics, Abbotsford, VIC., Australia.

over both sides, using a 10% maximum voluntary contraction (MVC) threshold level and a custom-made program [JAWS v1.441+ masseter muscle analysing software, Aalborg University (19)].

Results

Table 1 shows a summary of the PSG recordings, including the recording dates, the related treatment modalities, and the sleep bruxism outcome variables. The initial bruxism outcome measures (i.e. those obtained during PSG 1) had high values, with about 25 bruxism events per hour of sleep and more than 8% of the total sleeping time spent in bruxing. This confirmed the preliminary (clinical) diagnosis of sleep bruxism, for which at least four bruxism events per hour of sleep are needed (12).

Given the severity of both the implant-related complication (viz. implant fracture) and the recorded sleep bruxism, it was decided to try diminishing the bruxism frequency and duration before the patient's wish to be re-implanted was considered. Two management strategies were used, following a trial-and-error approach. First of all, a hard acrylic occlusal stabilization splint was made in the upper jaw, with a thickness of about 1 mm at the level of the first molars. As to facilitate habituation to wearing the splint, the appliance was worn 24 h day⁻¹, except during eating, for about 1 month. With the splint in situ, PSG 2 was made, yielding a comparable number of sleep bruxism events per hour of sleep as obtained during PSG 1. The BTI of the second PSG recording, however, was considerably smaller than the one obtained during the first PSG recording, suggesting shorter bruxism events with a splint in situ than without such an appliance.

As the dentist in charge (JZ) was unsatisfied with the treatment outcome with the occlusal stabilization splint, another approach was subsequently followed, namely a pharmacological one. After 2 months of no treatment at all (washout of the splint effect), pergolide[§] [a dopamine D1/D2 receptor agonist that is usually prescribed to, e.g. patients with Parkinson's disease (20) or restless legs syndrome (21)] was administered at bedtime in 0.05 mg tablets, along with domperidone, a peripheral D2 receptor antagonist, to reduce possible adverse effects like nausea. During the first week, the dose of pergolide was gradually increased from 0.05 mg to 0.3 mg, using 0.05 mg increments, as to give the patient time to adapt to the medication. At the end of a 2-month period of pergolide/domperidone usage, a third polysomnographic recording was made (PSG 3).

[§]Permax, Lilly, France

With respect to PSGs 1 and 2, there was a substantial decrease in both bruxism outcome measures: Epi/h now had a value of about 5; BTI, of 0.7%. Encouraged by this effect, the dose of pergolide was gradually and incrementally increased to 0.5 mg. PSG 4, which was recorded 1 month after PSG 3, showed slightly higher bruxism outcome measures with Epi/h almost reaching a value of 10; and BTI, of 1.3%.

As by then, the patient experienced several adverse reactions to the usage of pergolide (*viz.* nausea, headaches, tiredness, and poor sleep), despite the simultaneous use of domperidone, it was decided to discontinue the medication. Thus, the dose was reduced to zero in 2 weeks time. Another 2 weeks later, *i.e.* after an appropriate washout period, PSG 5 was obtained. Interestingly, the bruxism outcome measures remained low (Epi/h = about 6; BTI = about 1%). Hence, no more management strategies were tried, and one year later, a sixth polysomnographic recording (PSG 6) revealed an Epi/h value of 11 and a BTI of about 2%, values that were comparable with those obtained during PSG 5. Unfortunately, however, for private reasons that were unrelated to the above-described management strategies and evaluations, the patient finally declined re-implantation.

Discussion

Before the patient's wish to be re-implanted could be fulfilled, it was deemed necessary to first manage the sleep bruxism, thereby hopefully preventing another failure. As outlined in the Introduction, several management strategies have been suggested for the treatment of bruxism, like relaxation training, behavioural therapy, sleep hygiene measures, medication and occlusal stabilization splints (9, 11). The latter option was tried first, because of its non-invasive, reversible nature and its protective properties (10). Polysomnography showed that the number of bruxism episodes per hour of sleep (Epi/h) was unchanged without and with a splint *in situ*. The considerable reduction in BTI in this patient was an unexpected finding, because in a previous study by Van der Zaag *et al.* (13), patients with a substantial decrease in BTI always showed a substantial decrease in Epi/h as well. Taking the unchanged Epi/h value as the main treatment outcome, the dentist responsible (JZ) considered the splint therapy unsuccessful in preventing bruxism in this patient. Therefore, an alternative approach was sought.

It is well established that central neurochemicals like dopamine are involved in the pathophysiology of bruxism (8, 22, 23). Medicines like the catecholamine precursor L-dopa

(24) and the dopamine D2 agonist bromocriptine (25) seem to be useful in diminishing bruxism activity during sleep although for the latter substance, another report from the same group revealed no such effect (26). Among others for this latter reason, Winocur et al. (11) concluded in an extensive literature review, that the effect of dopamine-related drugs on bruxism remains unclear and that more controlled, evidence-based research on this under-explored subject is needed. Furthermore, it should be noted that information about dopaminergic substances in relation to bruxism is more readily available than that about other neurochemicals. This lack of focus on other substances in the literature as well as the presence of many possible interactions between dopamine and other neurochemicals also indicates the need for more research on this subject.

In this patient, the dopamine D1/D2 agonist pergolide was prescribed on the basis of the clinical experience of our neurologist (HLH). While bromocriptine mainly targets dopamine D2 receptors that are expressed in neurones of the midbrain, caudate and limbic systems, pergolide also targets dopamine D1 receptors that are expressed in neurones of the striatum (27). The clinical implications of activating these two dopamine receptor subtypes, however, are as yet unclear (27). Pergolide was selected for its purported scarcity of adverse reactions in comparison with, e.g. bromocriptine. Using low doses of 0.3–0.5 mg (where 1–5 mg is the usual therapeutic dose used for Parkinson's disease), a substantial effect was obtained, reducing Epi/h with about 60–80% and BTI with about 85–90%. This supports the suggestion that central neurochemicals like dopamine are involved in the modulation of sleep bruxism. Unfortunately, the patient reported a gradual increase in the occurrence of adverse reactions over a 3-month period, so that the medication had to be discontinued. Therefore, pergolide cannot be recommended for the routine use in the management of sleep bruxism, the more so because the use of pergolide has recently been associated with cardiac valvulopathy (28).

An interesting aspect of the treatment with pergolide is the observation that after a 1-month washout period as well as after a 1-year follow-up, the bruxism outcome measures were still low. As it is believed that bruxism shows only a limited variability over time (*viz.* Epi/h has a coefficient of variation of approximately 25% over a period of up to 7.5 years) (29), it can be speculated that this observation is indeed the consequence of the use of pergolide. Maybe, pergolide breaks a vicious cycle that maintains bruxism. To the authors' knowledge, such long-term observations of lasting effects have not been reported before.

Future studies (viz. randomized clinical trials with a long-term follow-up) are therefore needed to further explore this speculation.

Pergolide was combined with domperidone, a peripheral D2 receptor antagonist, to reduce possible adverse effects like nausea. Interestingly, domperidone was recently shown to suppress stress-related increases in cortisol levels, which suggests that this medicine may be beneficial in the treatment of stress (30). As there is growing evidence for a possible causal relationship between psychological stress and bruxism (31, 32), it cannot be excluded that the observed efficacy of pergolide in reducing this patient's sleep bruxism activity is also partly because of the co-administration of domperidone.

This case report indicates that fracture of oral implants can indeed occur as a complication of implant procedures, even though its incidence is reportedly low: in a systematic review of prospective longitudinal studies, Berglundh et al. (33) reported implant fractures in <1% of all implants during a 5-year period. In the patient of the present report, sleep-related bruxism was the most likely cause, given the extremely high values of the bruxism outcome measures and the exclusion of manufacturing defects (see Clinical report). In previous studies of sleep bruxism patients, the average number of bruxism episodes per hour of sleep varied between about 5 (12) and about 7 (13, 22, 24) while in the present study, more than 25 episodes were recorded before treatment. Notwithstanding the conclusion of Lobbezoo et al. (7) that there is still no proof for the suggestion that bruxism can cause a sufficiently high overload of oral implants and of their suprastructures to cause implant fracture, this study indicates that in severe cases, it can.

Besides suggesting the (lasting) efficacy of the dopamine D1/D2 receptor agonist pergolide in the management of sleep bruxism, the present 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 severe sleep bruxism.

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