

Original articles

Decreased amount of slow wave sleep in nocturnal bruxism is not improved by dental splint therapy

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Abstract

Objective : To test the efficacy of dental treatment of bruxism on sleep quality, using slow wave sleep as the primary outcome parameter.

Methods : The study design consisted of an open label, unpaired comparison between normals and patients and a paired comparison between pre- and post-treatment patient recordings. Twenty patients suffering from bruxism (13 male, 7 female, mean age 35 years) and 6 normal volunteers (3 male, 3 female, mean age 30 years) participated in the study. Polysomnographic recordings were performed in a sleep laboratory in a general hospital both before and after treatment. The treatment was derived from a model that ascribes bruxism to a dental malocclusion, and consisted solely of dental therapy (Jeanmonod 1988).

Results : The untreated bruxism group had worse sleep than normals when comparing slow wave sleep (21% versus 32% slow wave sleep percentage in sleep period time) during the second polysomnographic recording, after one night adaptation. Therapy did not improve sleep quality; bruxism patients showed only minor, non-significant differences in sleep quality when comparing pre- and post-treatment recordings.

Keywords : Bruxism; polysomnography; slow wave sleep; dental treatment; dental splint.

Introduction

Sleep bruxism is «a stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep» and should be distinguished from daytime parafunctional jaw muscle activities, such as teeth clenching, bracing, or gnashing». It is considered a parasomnia «because it is considered an undisable physical phenomenon that includes skeletal muscle activity that is present during the sleep» (Lavigne, 2000). Bruxism behavior in itself might be relatively common and even be considered normal behavior. It should only be considered pathological when there is severe tooth damage, sleep disturbance or pain, or perhaps even when the noise is sufficient to disturb bedpartners (Bader, 2000).

The pathophysiology of bruxism is not fully known. Several possible causes are cited in the liter-

ature. Sleep bruxism might be caused by dental occlusion problems, psychological influence, or central neurological causes (Bader, 2000).

Most authors today no longer adhere to the occlusal model of bruxism (Lobbezoo *et al.*, 2000). Occlusal therapy has not yet been proven to be a good therapy for sleep bruxism (Tsukiyama *et al.*, 2001). A particular French school, however, still uses a form of occlusal therapy for bruxism (Jeanmonod, 1988). Despite the lack of clinical trials, dentists who use the bruxism therapy according to Jeanmonod consider it to be clinically effective.

This study was designed to evaluate one aspect of therapeutic efficiency of the bruxism therapy according to Jeanmonod.

The evaluation of a treatment for a given disease is not complete without evaluating its effect on important complications of the disease. If bruxism has an influence on sleep quality, then a successful bruxism therapy should improve sleep quality. For this reason, we examined the influence of splint therapy for bruxism according to Jeanmonod (1988) on the macro architecture of sleep.

Sleep bruxism is often linked to sleep stages 1 and 2. Bruxism events occur less frequently in stages 3 and 4. Therefore, a comparison focusing on slow wave sleep (stages 3 + 4) and light sleep (stages 1 + 2), seemed an interesting approach to the study of sleep macro architecture in bruxism.

The commonly used term for sleep stages 3 and 4 is "slow wave sleep". Slow wave sleep is regarded as an indicator of sleep depth or sleep intensity. (Borbély and Acherman, 2000). It correlates with subjective appreciation of sleep.

Materials and Methods

The study protocol was approved by the Antwerp OCMW (community hospitals) Institutional Review Board, and carried out at the polysomnographic unit of the Middelheim General Hospital.

Patients : Twenty patients and 6 normal volunteers were included in the study after having given

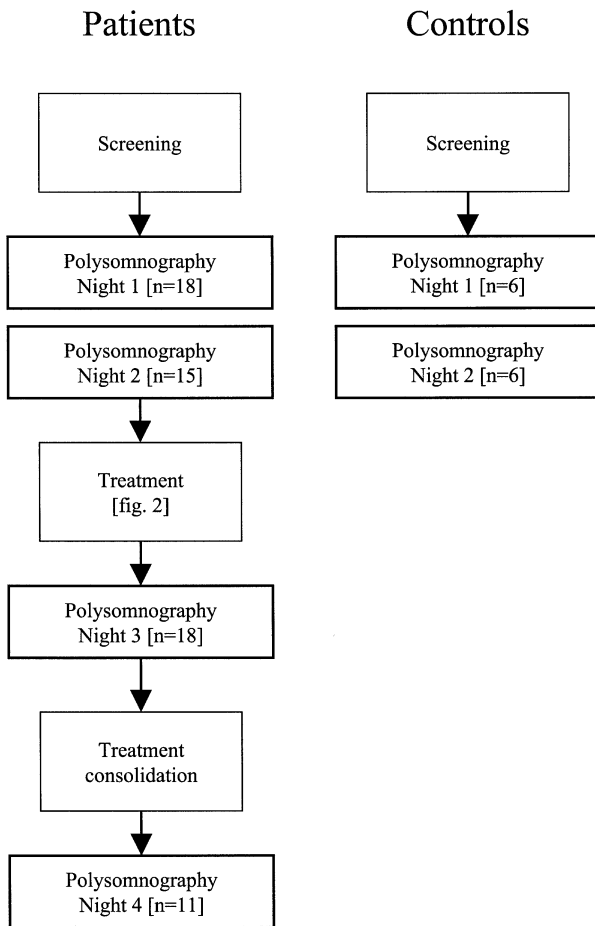


Fig. 1. — Simplified flowchart of the protocol. After screening, both patients and normal volunteers underwent polysomnographic recordings on two consecutive nights.

their informed consent. Patients and normal subjects were screened using a sleep questionnaire, concerning use of sleep medication, alcohol, tobacco and caffeine, day time and evening activities, sleep behavior and environmental factors before and during sleep.

Inclusion criteria for patients were 1) dental examination compatible with bruxism, 2) grinding sounds during sleep, reported by partner or roommate.

Exclusion criteria 1) subjects should not use benzodiazepines, antispastic drugs, amfetamines, dopamine, antidepressants or neuroleptics ; 2) subjects should not suffer from other neurological disorders ; 3) subjects should not suffer from caffeine abuse.

Normal volunteers were selected from a list of volunteers not having a clinical history or dental examination compatible with bruxism. In both groups, other sleep disorders listed in the International Classification of Sleep Disorders and the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, were excluded using questionnaire, anamnesis, heteroanamnesis and clinical examination (Buysse *et al.*, 1994). Selection of available volunteers was based on sex and age, in

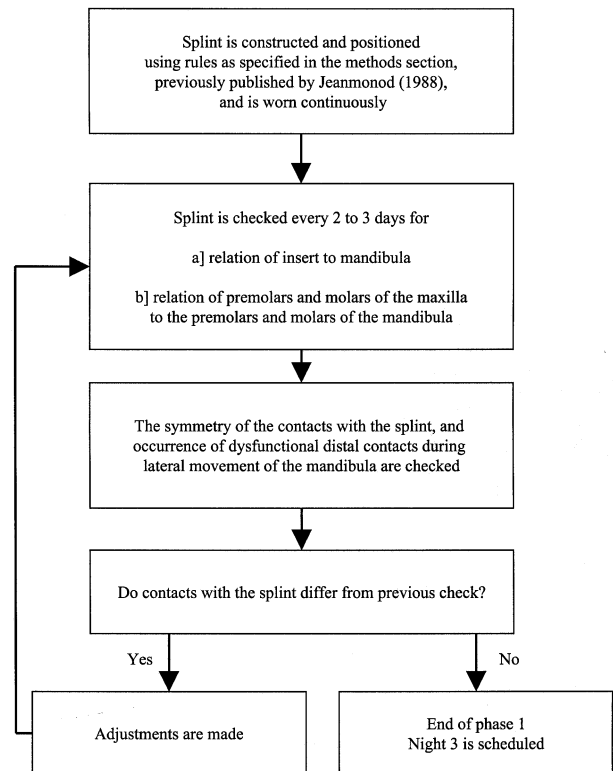


Fig. 2. — Flowchart for the primary treatment used in the study, which is based on the therapy described by Jeanmonod (1988)

order to minimize differences in these parameters between normals and patients.

Recordings : Subjects underwent polysomnographic recordings from 8 pm until 7 am. Both patients and normals, according to their usual habits at home, freely chose lights out time. Subjects arrived at the sleep laboratory at least 6 hours before the start of the recording. Figure 1 shows a simplified flowchart of the protocol. All subjects underwent two consecutive recordings (night 1 and night 2). Night 2 was considered to represent baseline state for both patients and normals.

All patients underwent a third recording (night 3). During night 3, all patients wore their oral splint. No splint was worn during night 1, 2 or 4. Night 3 was scheduled when the patient's therapy, started after night 2, was considered successful according to Jeanmonod's criteria (1988) (Fig. 2). Because of the recursive nature of this procedure, time between night 2 and night 3 differed among patients. Eleven patients also underwent a fourth recordings (night 4), after consolidation of the applied treatment.

Recorded parameters were EEG (seven derivations using Fp1, Fp2, C3, C4, Cz, O1, O2), EOG, ECG, respiration effort, surface electromyogram (EMG) of the mentalis muscle and temporalis and masseter muscles on both sides. Ag/AgCl surface disc electrodes were applied to the skin with con-

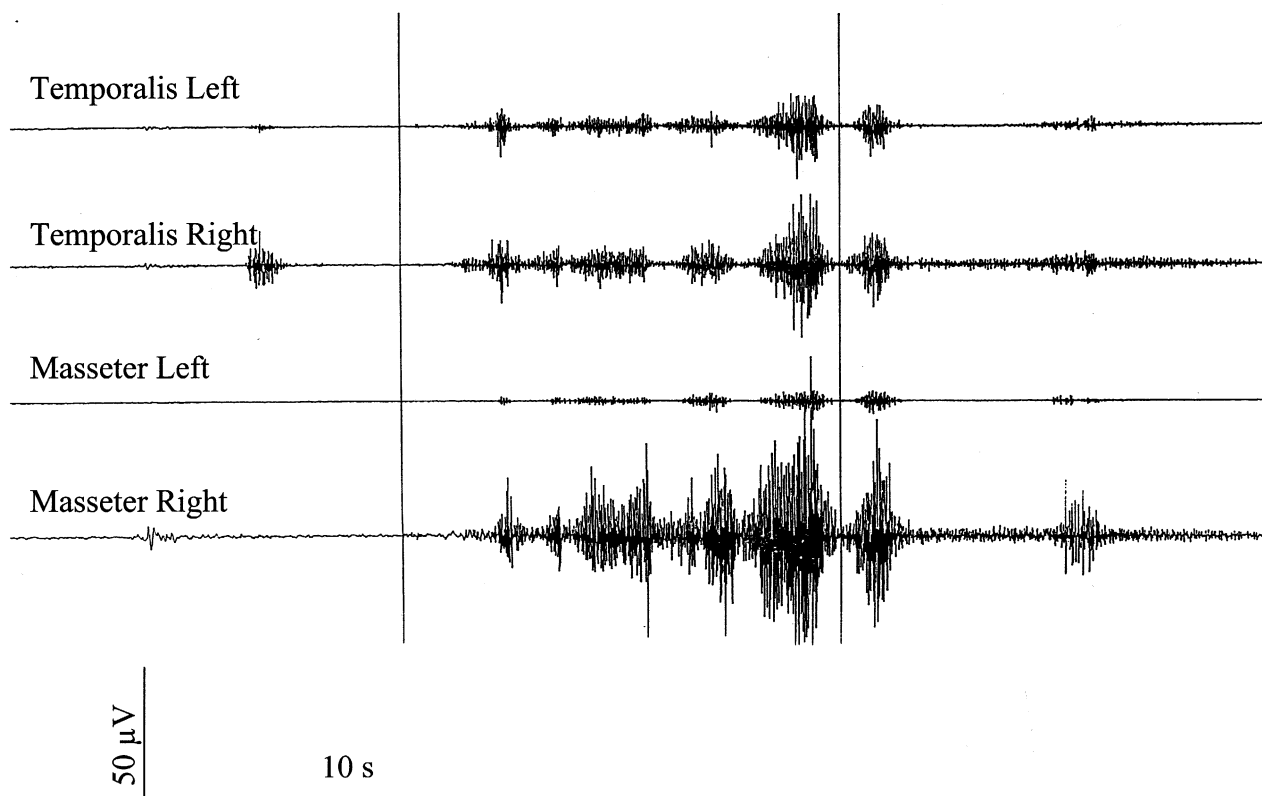


Fig. 3. — Typical bruxism event.

ducting paste, and held in position with small pads, soaked in collodion and dried with pressurized air. Temporalis and masseter muscle recordings were obtained through disposable Ag/AgCl electrodes. Signals were digitized in the headbox (Schwarzer, OSG, Belgium), optically transferred to a digital recording and analysis station (Brainlab, OSG, Belgium) and stored on digital media (DAT tape) in 10 s epochs for off-line analysis. Signals were recorded with an epoch length of ten seconds per screen, because this made it easier for the recording technicians to notice and resolve artifacts. Signals were later reviewed on screen for manual scoring of sleep stages according to Rechtschaffen and Kales' criteria (1968). Sleep stages were scored with an epoch length of 10 s, because the software did not allow a change in epoch length after the recording. Data were read into Excel (Microsoft, USA) in order to perform calculations and statistical analysis.

Analysis: In order to confirm the presence of bruxism activity, restricted to the patient population, polysomnographic recordings in bruxism patients and normals were visually scored for EMG findings. Bruxism events were defined as high frequency signals, with an amplitude of at least twice that of the preceding signal, and a duration of at least 1 second, occurring at the same time on at least two of the recorded muscles (masseter and temporalis on both sides). Fig. 3 shows an example

of a typical nocturnal bruxism event in one of the patients' polysomnographic recordings.

Primary endpoint was slow wave sleep percentage (time spent in stage 3 or 4 divided by total sleep time).

Parameters used in the exploratory evaluation of sleep quality were sleep period time (time from first occurrence of 3 minutes of stage 2 to final awakening in the morning), total sleep time (total time actually spent sleeping during the recording), sleep continuity index (total sleep time divided by sleep period time), movement time (total time in which EEG recordings were disturbed by movement artefacts), duration of separate sleep stages (expressed as an absolute number in seconds, and as a percentage relative both to sleep period time and to total sleep time). An epoch was visually scored as 'movement' when characteristic artifacts were superimposed on the traces for more than 50% of the epoch.

Dental treatment: Patients underwent therapy, which was started according to the principles of Jeanmonod (1988), aiming for a relaxation of the masticatory muscles. The basic principle in achieving this relaxation is that the space between upper and lower teeth (free way space) must never be eliminated. The free way space is defined as the physiologically occurring space between upper and lower teeth in a normal individual at rest. Relaxation is obtained by placing a splint, which is

checked twice a week for premature contacts. The splint is constructed in dental acrylic, using casts of upper and lower dentition. It is held in position using four clamps on the upper dental arch. The splint is positioned without suppressing the free-way space. This means that there are no contacts between upper and lower dental arch in rest position, and that there are no contacts between the splint, which is secured to the upper dental arch, and the lower dental arch.

Then, the patient is instructed to close his mouth. There should be a symmetrical contact between the splint and the lower canines, or between the four incisors, or between the two central incisors and the two canines, or between the two canines and the two lateral incisors, or between the four incisors and the two canines. This contact should occur at the same time on both sides of the dentition, in a symmetrical way. A premature contact exists when upper and lower dental arch make contact at one single point during occlusion. When this was noted, the splint was corrected and the patient was seen again after a new period of adaptation.

Therapy was considered successful when there was no further change in the position of the mandible relative to the splint. Jeanmonod (1988) used the term 'myo-déterminé' for this condition, indicating the assumption that this occlusion state is determined by a resting equilibrium position of the chewing muscles. The treatment protocol is illustrated in a flowchart in figure 2.

During the consolidation treatment, standard dental procedures were used to achieve remodeling of the patient's teeth. The purpose of the consolidation treatment was to maintain the 'myo-déterminé' occlusion, which was achieved after the primary treatment (Fig. 2) in a given patient, without the dental splint which is worn during the first phase of the treatment.

Some patients agreed to take part in the first part of the study, which encompasses reversible interventions, but not in the latter part. Therefore only eleven night four recordings were performed.

Statistics : Unpaired Student's t test was used for comparison between normal and patient groups, and paired Student's t test was used for comparisons of repeated measures. A Bonferoni correction for multiple comparisons was used in the exploratory analysis.

Results

Obtained recordings : Twenty patients and six normals were included in the protocol. Technical failure of a backup device resulted in loss of 11% of recordings.

Nine patients did not give consent for the consolidation treatment after night 3, because it involves permanent remodeling of the dental sur-

faces using standard dental techniques, and were therefore not invited to return for a fourth recording.

This led to a total number of usable recordings of 18 for night 1, 15 for night 2, 18 for night 3 and 11 for night 4 in the patient group, and 6 for night 1 and 6 for night 2 in the normal volunteer group (Fig. 1).

Primary endpoint : Normal volunteers had 32% slow wave sleep (SEM 4%) during total sleep time in their second night in the sleep laboratory. Bruxism patients had 21% slow wave sleep (SEM 2%) during night two, which was recorded pre-treatment. This difference was statistically significant ($p=0.0141$). During night three, recorded post-treatment, bruxism patients showed 18% slow wave sleep (SEM 2%). This was not significantly different from the pre-treatment level.

Exploratory analysis : Duration of the primary treatment (between night 2 and 3) was between 3 and 5 weeks. This variation occurred because of the recursive nature of the procedure between night 2 and night 3 recordings (Fig. 3). All patients reported a subjective improvement after treatment (Fig. 3), before night 3 recordings.

All patients showed bruxism events during night 2 polysomnographic recording. The mean number of events was 71 per night (SEM 13, mean duration 5.5 s).

Patients and normal subjects did not show abnormalities on clinical neurological examination.

Patients and normal subjects did not differ significantly in their lights-out times (mean time 23 : 25 for normal volunteers, 22 : 41 for patients). Other macroscopic sleep parameters were not significantly different when compared in normal volunteers, and bruxism patients (table 1).

Sleep continuity index decreased from 86% to 77%. This difference was not statistically significant.

After consolidation treatment (night 3 versus 4), sleep continuity index increased from 77% to 84%, regaining the pretreatment level, while slow wave sleep percentage in total sleep time was slightly decreased. Both changes appear to be caused by the increase in total sleep time (22010 s versus 23940 s). These three phenomena were not statistically significant. Total duration of movement increased, but did not reach the pre-treatment level (350 s, 210 s and 250 s for nights 2, 3 and 4).

Discussion

This study mainly focuses on sleep quality. Sleep quality is used to compare untreated bruxism patients to normal volunteers, and to investigate a potential influence of an occlusodontal treatment on bruxism patients. Slow wave sleep was selected as a primary endpoint.

Table 1

Investigated parameters of sleep quality for each group during night 1 and 2. Mean values (in seconds) and standard error of the mean (SEM) are displayed

	controls				patients			
	mean	SEM	mean	SEM	mean	SEM	mean	SEM
Sleep period time (s)	30640	2550	24530	2910	25250	1950	27060	1120
Total sleep time (s)	20530	3940	22190	3040	21660	1700	23160	980
Sleep continuity index	70%	15%	91%	5%	87%	2%	86%	2%
Delta percentage in sleep period time	21%	4%	32%	6%	20%	2%	21%	2%
Delta percentage in total sleep time	30%	5%	35%	7%	24%	2%	24%	2%
Stage four percentage in sleep period time	14%	4%	19%	6%	10%	2%	11%	1%
Stage four percentage in total sleep time	20%	6%	21%	6%	12%	2%	12%	2%

Table 2

Investigated parameters of sleep quality in the bruxism group for night 2, 3 and 4. Mean values (in seconds) and standard error of the mean (SEM) are displayed

	night 2		night 3		night 4	
	mean	SEM	mean	SEM	mean	SEM
Sleep period time (s)	27060	1120	28930	1200	28630	1370
Total sleep time (s)	23160	980	22010	1430	23940	1280
Sleep continuity index	86%	2%	77%	4%	84%	2%
Delta percentage in sleep period time	21%	2%	18%	2%	18%	2%
Delta percentage in total sleep time	24%	2%	23%	2%	22%	2%
Stage four percentage in sleep period time	11%	1%	10%	1%	9%	1%
Stage four percentage in total sleep time	12%	2%	12%	2%	11%	2%

Most studies that use polysomnographic recordings in bruxism patients focus on the motor activity associated with the bruxing events (Wrubble *et al.*, 1989 ; Sjöholm *et al.*, 1992 ; Pierce *et al.*, 1995) Sleep quality is not often used as a parameter in the evaluation of therapies for bruxism. Lobbezoo *et al.* (1997) found a moderate effect of L-dopa on sleep bruxism. No differences in sleep parameters were reported. The number of bruxing episodes decreased with 1 to 2 per hour under treatment (Lobbezoo *et al.*, 1997). This treatment effect was statistically significant, a fact which does not necessarily imply a clinical relevance.

To test clinical relevance of a bruxism treatment, one could investigate long-term effects on dental attrition. Another way of evaluating the clinical relevance of a therapy for bruxism, is evaluating the sleep quality before and after treatment.

Subjective improvement is often used to evaluate sleep quality. Since this is per definition not an objective parameter, and since treatment in our study was unblinded, we tried to use a more objective parameter, which could be evaluated blinded to treatment.

We found a decreased amount of slow wave sleep in treated and untreated bruxism patients, compared to normal volunteers.

A number of studies report on sleep quality in bruxism.

In 24 patients with a mean age of 35, all suffering from sleep bruxism, slow wave sleep account-

ed for 23% of total sleep time (Bader, 1997). No control group was examined.

Boutros *et al.* (1993) analyzed five bruxing patients, and found no abnormalities in sleep pattern. No controls were examined in this study.

Dettmar *et al.* (1987) examined five individuals showing distinct signs of tooth wear, and recorded no abnormalities in sleep profiles. No control group was used (Dettmar *et al.* 1987).

Some studies did examine sleep quality in bruxing patients, as compared to a normal control group.

Sleep variables in 31 normal volunteers showing rhythmic masticatory muscle activity, selected from a group of 82 normal volunteers, and 33 bruxers were recently compared by Lavigne *et al.*, (2001). Normal volunteers showing rhythmic masticatory muscle activity had 13.1 % slow wave sleep, while bruxers had 12.1%. This difference was not statistically significant.

A group of six bruxism patients showed no differences in conventional sleep variables, including amount of slow wave sleep, when compared to six controls. However, significantly more transient arousals, characterized by EEG desynchronization were found in the bruxism patients. The authors feel that this discrepancy might have been caused by the standard approach to sleep scoring, in which relatively long epoch lengths of 30 seconds are used (Macaluso *et al.*, 1998). In our study, an epoch length of 10 seconds was used. In combi-

nation with the higher number of patients who participated in the patient-control comparison (table 1), this might have increased the sensitivity of our study.

In an older study, Reding *et al.* (1968) performed polysomnography on 40 nocturnal bruxists and 18 control subjects and found normal cyclic variations in the hypnogram, and relative proportions of sleepstages which were within normal expectations, leading to the conclusion that sleep EEG records of nocturnal teeth grinders are essentially normal.

Lavigne *et al.* (1996) applied polysomnography in 18 patients suffering from bruxism, and 18 asymptomatic volunteers, and found no difference in sleep parameters.

The mean age of the subjects was comparable to our subjects. Perhaps inclusion criteria were less stringent in Lavigne's group, since presence of toothwear was not mandatory when either morning masticatory muscle pain, or masseteric muscle hypertrophy on palpation were present. Two out of eighteen patients did not show any signs of toothwear. All patients in our study showed toothwear on dental examination. In Lavigne's study, criteria for sleep efficiency were not focused on slow wave sleep.

The difference in the amount of slow wave sleep we found was not observed in these studies. Patient selection might be one possible explanation for the fact that we did find a difference in amount of slow wave sleep between patients and normals. All of the patients who were included in our study not only showed tooth wear compatible with bruxism, but also reported that family members or roommates had heard bruxing sounds during sleep.

Another explanation is the size of our group of normals, which is relatively small compared to our patient group.

When we look at the effect of the parameter we selected, slow wave sleep, on the occlusodental therapy according to Jeanmonod, we found no difference before and after treatment. This therapy, which resulted in subjective improvement in all patients included in this study, therefore did not improve sleep macro-architecture.

There did appear to be a small decrease in sleep quality when we compare pre- and post treatment recordings in our patients, with treated patients having less slow wave sleep. These differences, however, were not statistically significant.

When comparing recordings before treatment, and after treatment consolidation, no difference in slow wave sleep was found. There were no significant differences in sleep quality when comparing night 3 (after treatment, before consolidation treatment) versus night 4 (after consolidation treatment). The tendency to worsening of sleep quality during night 3 might be caused by the dental splint the patients wore at night during the first part of the

therapy (starting after night 2, and stopped after night 3). This splint was worn during night 3 recordings, but not during the other recording nights.

Using sleep parameters in the evaluation of therapies for sleep bruxism is a logical step. This approach was also used in therapies based on a central neurological model of bruxism.

The effect of bromocriptine on sleep bruxism was recently investigated (Lavigne *et al.*, 2001) in a group of seven bruxism patients, with an age range of 23-39 years. A randomized double blind cross over design was used. The authors originally intended to include 14 patients, but stopped the trial prematurely when no effect was demonstrated on an interim analysis.

Bromocriptine did not decrease bruxism episodes during sleep. The treatment did not have an effect on striatal D2 binding, which was examined by SPECT. There was no effect of the treatment on sleep variables. Stage 3 and 4 sleep percentage was 15.4 % in the placebo group, and 12.9 % in the treated group. This difference was not statistically significant.

There are several possible explanations why we did not find an effect of the occlusodental therapy according to Jeanmonod on macro architecture of sleep.

Recent work in this field suggests that nocturnal bruxism has a central neurologic cause, and is associated with sleep abnormalities such as arousal reactions (Yap, 1998). In this model, the sleep abnormalities are not caused by bruxism. Rather, the two phenomena have a common central neurologic cause.

A somewhat older view on the pathophysiology of bruxism is the occlusodental one.

In 1968, Reding published on this topic, stating that the basic pathophysiological mechanism of bruxism is the occurrence of teeth grinding due to the forceful contact of occlusal tooth surfaces, often accompanied by loud clicking or grating sounds. This leads to damage to the teeth and their supporting structures, which can be detected by dental examination.

The "occlusal interference" theory states that premature tooth contacts, or abnormalities in the way the patient bites lead to bruxism. Improper tooth contacts may affect periodontal mechanoreceptors and elicit reflex excitation of the jaw closing muscles. Other studies provided arguments against this hypothesis. Rugh and Harlan, for instance, failed to elicit bruxism by placing high restorations in patient's mouths (Rugh and Harlan, 1988).

In a model where malocclusion causes bruxism, in turn leading to sleep disturbances, one would expect an occlusal therapy for bruxism to improve sleep quality. If occlusion is not an important factor in the disease, then a therapy designed to improve

occlusion would not be effective. This view is supported by the current literature on bruxism, in which morphological features are no longer considered as the main causative factors in the disease (Lobbezoo *et al.*, 2000). The fact that the bruxism therapy in our study did not improve sleep quality might thus be in favor of the central neurological model for bruxism.

Another explanation could be that the occluso-dental model is correct, but that the therapy according to Jeanmonod is not effective in improving occlusion. This possibility must of course be taken into account, since the reverse was not yet proven by clinical trials.

A third possibility is that the occluso-dental model is correct, and that the therapy according to Jeanmonod is effective in improving occlusion, but that we did not use the right parameters to evaluate sleep quality. This is a sound explanation in view of the recent literature in which bruxism is associated with arousal reactions (Lavigne *et al.*, 2001). This possibility will undoubtedly be taken into account in further studies on the occluso-dental model.

The design of the study should also be considered as a possible cause. Perhaps limitations in the design caused a false negative study. The main analysis was a comparison between slow wave sleep before and after treatment. This test was sufficiently powered to detect a difference of 3% in slow wave sleep. It would be interesting to obtain a power calculation for the previously cited studies in which no significant difference in sleep macro architecture was found between bruxism patients and normal volunteers.

In summary, several factors can account for the fact that this treatment did not improve macro-architecture of sleep. Overall, the approach in which a therapy for bruxism is evaluated through the effect of the therapy on a possible complication of the condition remains interesting.

Conclusions

Patients suffering from bruxism showed poor sleep quality, when compared to normal volunteers, using slow wave sleep as primary outcome parameter.

A bruxism treatment in two stages, based on the dental pathophysiological model, did not show significant improvement on slow wave sleep.

These findings provide some further support for a central cause of bruxism.

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