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Chapter 2

Time-variant nature of sleep bruxism outcome variables using ambulatory polysomnography: implications for recognition and therapy evaluation.

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Summary

The aim of this study was to quantify the time-variant nature of sleep bruxism (SB) and to discuss its consequences. Six clinically diagnosed bruxers and six non-bruxers participated. Four ambulatory polysomnographic (PSG) recordings were obtained for every participant. As SB outcome variables, the number of episodes per hour of sleep (Epi h^{-1}), the number of bursts per hour (Bur h^{-1}) and the bruxism time index (BTI: the percentage of total sleep time spent bruxing) were established. To quantify the time-variant nature of SB, standard errors of measurement (SEMs) were calculated. For the non-bruxers, the SEMs for Epi h^{-1}, Bur h^{-1} and BTI were 1.0, 5.7 and 0.1. For the bruxers, the respective values were 2.1, 14.9 and 0.4. In the discussion, arguments are given that because of the time-variant nature of the PSG recordings, cut-off bands around cut-off points might be useful for the recognition of SB.

Keywords: bruxism, variability, recognition, diagnosis, therapy evaluation

Introduction

Sleep bruxism (SB) is a stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep (1). The disorder has a prevalence that varies from about 20% in children to about 3% in persons over 60 years of age (2, 3). It has recently been argued that SB is mainly regulated centrally (e.g. by psychological stress, sleep arousal and ⁄or disturbances in the central dopaminergic system) and not peripherally (e.g. by deviations in dental occlusion and⁄or in the orofacial anatomy), although the aetiology of SB is still not fully understood (4, 5).

Sleep bruxism is generally regarded a possible cause for occlusal tooth wear in the form of attrition [i.e. mechanical wear resulting from mastication or parafunction, which is limited to the contacting surfaces of the teeth (6)] and for temporomandibular pain (7, 8). Since these consequences of SB can have a serious nature, treatment of the disorder is often indicated (9).

A proper diagnosis of SB and a reliable way of monitoring possible treatment effects are then important issues (10–12).

To objectively recognize SB, the use of polysomnography (PSG) is often recommended. 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 (13). The discriminative power of these criteria was recently confirmed by Rompré et al. (14). However, the fluctuating character of SB (12) will inevitably also be reflected in fluctuations in the outcome variables of the PSG recordings. This complicates the recognition of SB with the use of PSG cut-off points and the monitoring of possible treatment effects.

Therefore, the aims of the present study were: firstly, to quantify the time-variant nature of SB, using ambulatory PSG recordings; and secondly, to discuss its consequences for the recognition of this disorder and for the monitoring of possible treatment effects.

Materials and methods

Participants

Initially, 13 participants were included in the study, 10 of them being women. For private reasons that were unrelated to the study, one female participant dropped out of the study

after the first PSG recording. Thus, a total of 12 persons completed the entire protocol. Two men and four women, with a mean (±SD) age of 29.0(±7.1; 22–42) years, were allocated to the non-bruxer group; one man and five women, with a mean (±SD) age of 36.5(±11.5; 23–53) years, to the group of clinically diagnosed bruxers.

Bruxers were recruited by means of an announcement in a local newspaper, and from among the patients attending the clinic of the department of Oral Function of the Academic Centre for Dentistry Amsterdam (ACTA). Non-bruxers were recruited from among the employees and students of ACTA. To be included in the group of bruxers, participants should fulfil all of the following criteria: being at least 18 years of age; having a natural dentition with signs of tooth clenching [i.e. hyperkeratotic ridges in the cheeks, tongue scalloping, and ⁄or incisal impressions in the lips (15)]; having a history of tooth grinding sounds for at least three nights per week during the last 6 months [adapted from Kato et al. (16); and having occlusal tooth wear to at least the degree of exposed dentine [i.e. grade two (17)]. Non-bruxers were included when they were at least 18 years of age; with a natural dentition; and without signs of clenching, a recent history of grinding sounds, and an occlusal tooth wear grade of two or more. Participants, bruxers and non-bruxers alike, were excluded when they fulfilled one or more of the following criteria: suffering from epilepsy or any sleep disorder other than SB [determined on the basis of the first PSG recording and the Sleep Disorders Questionnaire (SDQ), see Procedure]; using any medication that has a known influence on sleep structure or SB [e.g. selective serotonin re-uptake inhibitors or anti-Parkinson medication (18)]; or being diagnosed with temporomandibular pain (19, 20). The scientific and ethical aspects of the protocol were reviewed and approved by the board of the Netherlands Institute for Dental Sciences, and written informed consent was obtained from all participants.

Procedure

At intake, a set of questionnaires was administered [containing, amongst others, a standard Dutch medical questionnaire and the Dutch version of the SDQ (21, 22)], an oral history was taken, and a clinical examination was performed. Following the intake procedure, the selected bruxers and non-bruxers were invited to sleep with an ambulatory PSG unit (Monet*) for home recordings during four nights. The mean interval between successive

recordings was approximately 3 weeks. The montage protocol consisted of the following leads:

1. Electroencephalography (EEG; C3A2;O2A1);
2. Electromyography (EMG; right and left masseter muscle; submental area);
3. Electro-oculography (EOG; right and left).

Each masseter EMG signal was recorded at 512 Hz and was appropriately filtered (hardware; 50 Hz notch; 3 Hz high pass; 100 Hz low pass). No audio/video recordings were carried out. The entire montage was performed in our laboratory.

Data analysis

All PSG recordings (12 participants x 4 nights = 48 recordings) were analysed with REMBRANDT* software. The analyses consisted of two parts: a sleep analysis and a bruxism analysis. The sleep analysis was performed to exclude sleep disorders other than SB in addition to the results of the SDQ, to determine any abnormalities in sleep structure, and to enable the sole inclusion of masticatory muscle activities during actual sleep in the analysis of SB. Using 30-s epochs, all sleep analyses were carried out automatically according to the criteria of Rechtschaffen & Kales (23). An experienced sleep scientist manually checked all analyses. Finally, the following standard sleep variables were determined: total sleep time (min), percentage of non-REM (rapid eye movements) sleep, percentage of REM sleep, and sleep efficiency (i.e. sleep time as percentage of the total time in bed).

The analyses of SB were performed in four steps, using an automatic bruxism analyzing tool incorporated in the REMBRANDT software. The algorithms of this tool are comparable with those described by Gallo et al. (24). The first step of the tool consisted of EMG sample rate conversion (to down-sample the signal to approximately 100 Hz). During the second step, the EMG signals were rectified and low-pass filtered (time constant 0.1 s) to locate areas of increased amplitude. During the third step, periods of elevated EMG activity, using an EMG threshold of 10% of the maximum voluntary EMG activity of the left and the right masseter muscles were detected. Finally, during the fourth step, three SB outcome variables were derived during actual sleeping periods only, viz., the number of bruxism episodes per

*Medcare Automation BV, Amsterdam, The Netherlands.

hour of sleep (Epi h^{-1}) and the number of bruxism bursts per hour of sleep (Bur h^{-1}) according to Lavigne et al. (13, 25), and the bruxism time index (BTI; i.e. the total time spent in bruxing divided by the total sleep time, times 100%) according to van der Zaag et al. (26).Thus performed, all analyses were manually checked. Since no statistically significant differences in SB outcome variables were found between the analyses of the EMGs of the left and the right masseter muscles, the results of both analyses were averaged. These average values were used in the statistical analyses described below.

Statistics

The two groups of participants (i.e. bruxers and nonbruxers) were compared for possible age differences, using an independent-samples t-test. Possible associations between gender and group were assessed with a chi-squared test. The standard sleep variables as well as the SB outcome variables were analysed for within-subject effects (i.e. the four recordings: 'Night effect') as well as for between-groups effects (i.e. bruxer versus non-bruxer: 'Group effect'), using a General Linear Model (GLM) with repeated measures. General Linear Model was preceded by Mauchly's Test of Sphericity as to assess normality of the variables studied.

For both the bruxers and non-bruxers, the standard error of measurement (SEM) was calculated to quantify the time-variant nature of the PSG recordings. standard error of measurement, as indicator of the average standard deviation (SD) of individual PSG outcomes, was calculated according to Fleiss and Kingman (27). To check whether the SDs depended upon the mean values of the PSG variables, linear regression analyses were performed for all the participants combined. The mean values and SDs of each participant were based upon the results of the four night-recordings. The smallest detectable difference (SDD), as the smallest difference between two PSG outcomes of an individual, which is significant at a 95% probability level (27, 28), was calculated as follows: $SDD = 1.96 \times \sqrt{2} \times SEM$. All statistical tests were performed with SPSS 12.0 software package.† Probability levels of $P < 0.05$ were considered statistically significant.

†SPSS Inc., Chicago, IL, USA.

Results

The groups of non-bruxers and bruxers did not differ with respect to age (t = 1.362; P =0.203) or gender (χ^2 =0.444; P =0.505). All 48 hypnograms were judged to have normal structures by an experienced neurologist who is specialized in sleep disorders (HLH, co-author). Table 1 shows the descriptive statistics (mean ± SD) of the standard sleep variables for the four PSG recordings, both for the non-bruxers and for the bruxers. The standard sleep variables did not differ significantly between both groups (no significant Group effects; Table 2), nor between the four nights (no significant night effects; Table 2).

Table 3 shows the descriptive statistics (mean ± SD) of the SB outcome variables, collapsed across the four PSG recordings for every non-bruxer and bruxer; Fig. 1(a–c) shows the SB outcome variables for the four PSG recordings, both for the six non-bruxers and for the six bruxers. As already indicated by the relative positions of the dashed lines (non-bruxers) and the solid lines (bruxers) in Fig. 1(a–c), the SB outcome variables Epi h^{-1}, Bur h^{-1} and BTI were significantly higher for the bruxers than for the non-bruxers (significant Group effects; Table 4). No significant differences were found in the SB outcome variables between subsequent nights (no significant night effects; Table 4).

As indications of the time-variant nature of the PSG recordings, the SEM and the SDD of the SB outcome variables for the non-bruxers and the bruxers are shown in Table 5. The mean values and SDs of the PSG variables for each participant, based upon the four-night recordings, were also calculated. Linear regressions between the PSGs' SDs and the mean values were found (P <0.0001; Fig. 2a–c). For all three PSG variables, the SD were higher when the mean PSG values were higher.

Table 1. Descriptive statistics (mean ± SD) of the standard sleep variables per polysomnographic recording (PSG 1-4) for both the non-bruxers and the bruxers

	Non-bruxers			
	PSG 1	**PSG 2**	**PSG 3**	**PSG 4**
Total sleep time (min)	439.7 ± 64.9	445.5 ± 38.5	431.3 ± 27.1	426.5 ± 33.7
Non-REM (%)	72.2 ± 6.9	73.0 ± 7.2	76.2 ± 3.0	73.1 ± 3.1
REM (%)	25.7 ± 6.8	25.6 ± 7.3	22.4 ± 2.8	25.8 ± 3.4
Sleep efficiency (%)	81.3 ± 13.9	84.7 ± 7.3	83.3 ± 5.0	89.3 ± 7.7

	Bruxers			
	PSG 1	**PSG 2**	**PSG 3**	**PSG 4**
Total sleep time (min)	463.5 ± 25.1	465.3 ± 37.7	433.2 ± 48.8	466.5 ± 37.4
Non-REM (%)	78.7 ± 4.9	77.1 ± 6.2	76.9 ± 6.5	76.2 ± 5.8
REM (%)	20.9 ± 4.9	22.5 ± 6.1	22.4 ± 6.3	23.2 ± 5.8
Sleep efficiency (%)	86.5 ± 9.5	86.7 ± 12.8	82.5 ± 10.1	90.8 ± 11.1

Table 2. General Linear Model with repeated measures of the effects of Group (non-bruxers or bruxers), night (the four polysomnographic recordings) and their interaction on the standard sleep variables (TST, percentage of non-REM sleep; percentage of REM sleep and SE)

Effect	**df**	**TST**		**Non-REM (%)**		**REM (%)**		**SE**	
		F	**p**	**F**	**p**	**F**	**p**	**F**	**p**
Group	1	1.708	0.221	2.598	0.138	1.419	0.261	0.211	0.656
Night	3	1.062	0.380	0.364	0.779	0.456	0.715	1.946	0.143
Interaction	3	0.635	0.599	0.764	0.523	0.534	0.662	0.295	0.828

df, degrees of freedom; TST, total sleep time; SE, sleep efficiency.

Table 3. Descriptive statistics (mean ± SD) of the sleep bruxism outcome variables (Epi h^{-1}; the number of bursts per hour of sleep, Bur h^{-1}; and the bruxism time index, BTI) as collapsed across the four polysomnographic recordings for every participant (non-bruxer or bruxer)

	Non-bruxers					
	No. 1	**No. 2**	**No. 3**	**No. 4**	**No. 5**	**No. 6**
Epi/h	2.95 ± 1.11	2.13 ± 1.26	1.68 ± 0.90	2.68 ± 0.90	1.53 ± 0.94	1.00 ± 0.37
Bur/h	20.73 ± 7.23	11.65 ± 7.24	7.68 ± 4.13	12.90 ± 3.98	10.25 ± 6.98	6.23 ± 3.39
BTI	0.44 ± 0.21	0.24 ± 0.15	0.23 ± 0.10	0.40 ± 0.15	0.20 ± 0.12	0.13 ± 0.05
	Bruxers					
	No. 7	**No. 8**	**No. 9**	**No. 10**	**No. 11**	**No. 12**
Epi/h	7.38 ± 2.77	4.08 ± 1.63	5.40 ± 2.30	6.13 ± 1.87	4.95 ± 1.30	6.35 ± 2.10
Bur/h	57.03 ± 19.81	23.90 ± 14.75	36.38 ± 13.11	49.18 ± 11.71	40.55 ± 13.44	43.08 ± 14.92
BTI	1.35 ± 0.77	0.65 ± 0.30	0.74 ± 0.26	1.10 ± 0.31	0.87 ± 0.43	0.75 ± 0.30

Epi h^{-1}, episodes per hour of sleep; Bur h^{-1}, the number of bursts per hour of sleep; BTI, bruxism time index.

Table 4. General Linear Model with repeated measures of the effects of Group (non-bruxers or bruxers), night (the four polysomnographic recordings) and their interaction on the sleep bruxism outcome variables (Epi/h^{-1}, Bur/h^{-1} and BTI)

Effect	**df**	**Epi/h**		**Bur/h**		**BTI**	
		F	**p**	**F**	**p**	**F**	**p**
Group	1	44.159	0.000	35.319	0.000	28.760	0.000
Night	3	0.671	0.577	1.308	0.290	0.199	0.897
Interaction	3	2.802	0.057	1.689	0.190	1.156	0.343

df, degrees of freedom.

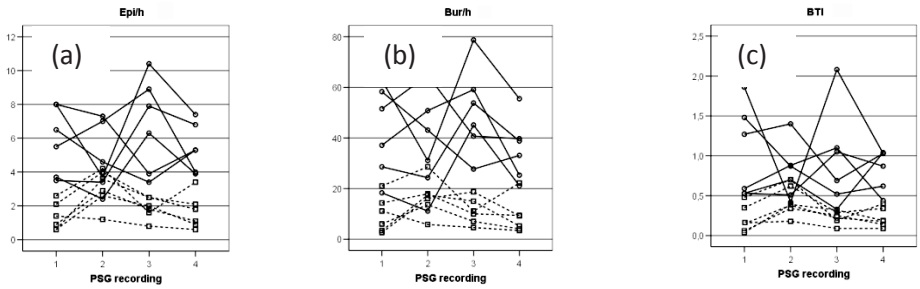

Fig. 1. Individual graphs of (a) the number of episodes per hour of sleep (Epi h); (b) the number of bursts per hour of sleep (Bur h); and (c) the bruxism time index (BTI), as derived during the four polysomnographic (PSG) recordings (1–4). Squares/dashed lines represent non-bruxers; circles/solid lines, bruxers.

Table 5. The mean value, the SEM and the SDD for both groups of participants (non-bruxers and bruxers) and for all three sleep bruxism outcome variables (Epi/h^{-1}, Bur/h^{-1} and BTI)

	Non-bruxers			**Bruxers**		
	Mean	SEM	SDD	Mean	SEM	SDD
Epi/h	2.0	1.0	2.6	5.7	2.1	5.7
Bur/h	11.6	5.7	15.9	41.7	14.9	41.2
BTI	0.3	0.1	0.4	0.9	0.4	1.2

SEM, standard error of measurement; SDD, smallest detectable difference.

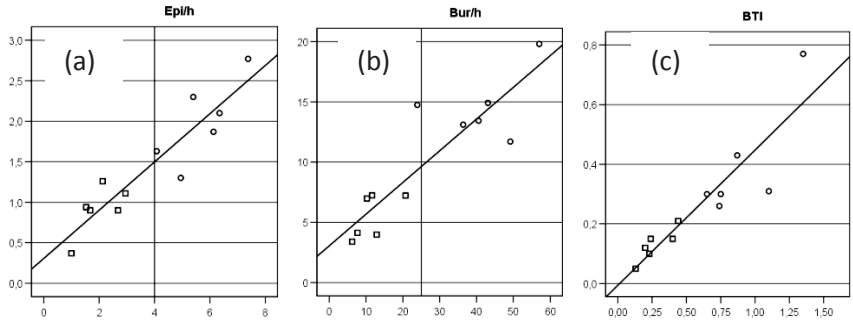

Fig 2. Linear regression between the standard deviations and average values of the polysomnographically established sleep bruxism outcome variables Epi h^{-1} (a; $R^2 = 0.84$), Bur h^{-1} (b; $R^2 = 0.77$) and BTI (c; $R^2 = 0.81$). The solid vertical lines in the graphs, at 4 Epi h^{-1} (a) and at 25 bur h^{-1} (b), represent the previously published cut-off criterion for those variables (13). Squares represent non-bruxers; circles, bruxers.

Discussion

In this study, data are given that quantify the time-variant nature of SB variables. This was performed by analysing the ambulatory PSG recordings of clinically established bruxers and non-bruxers, which were obtained during four overnight recordings.

First-night effect

A first PSG recording, used solely for habituation to sleeping under experimental conditions, should preferably precede the recording that is used for diagnosis and research purposes as to avoid the so-called first-night effect (29, 30), even though there is evidence that this effect may even last more than one night (31). If present in this study, the first-night effect would have resulted in first-night PSG recordings deviating from the other three recordings in terms of outcome variable values. However, GLM analyses did not reveal any differences between the four nights, neither on SB outcome variables nor on standard sleep variables, making the presence of the first-night effect unlikely. It may also be speculated that all the PSG recordings, being separated by relatively long time intervals, were similarly influenced by a first-night effect. However, this possibility is not likely either, because all individual PSG recordings, without any exception, were judged to display a normal sleep structure (32). In addition, the group means of the standard sleep variables fell within the range from those of normal adults (i.e. individuals who are living on a conventional sleep-wake

schedule and who are without sleep complaints) (32). The absence of an obvious first-night effect may be due to the fact that the ambulatory recordings were made at the participants' habitual sleeping environment, thereby leaving the need for habituation redundant.

SB recording systems

In the present study, ambulatory PSG recordings were used. Alternative techniques are ambulatory EMG recordings or, at the other extreme, sleep laboratory PSG recordings. As reviewed extensively by Lavigne et al. (29), each system has its own advantages and disadvantages. Ambulatory surface EMG recordings have the benefit of their easy use and low cost. However, they do not allow for the recognition of bruxism during actual sleep. For that, multichannel sleep laboratory recordings are needed that include, amongst others, EEG, submental EMG and EOG leads, as well as audio/video control to distinguish sleep bruxism episodes from other, non-specific, orofacial activities (12–14, 25). The presently used system is a multichannel, ambulatory PSG recorder. Although this system has the disadvantage of lacking audio/video control, it is a relatively low-cost technique that allows monitoring over several nights in the patient's natural environment that yields reliable and high-quality recordings (29, 33). For these reasons, we decided to use this technique in the present study instead of the high-cost, highly controlled, but unnatural sleep laboratory setting.

Time-variant nature of SB

Variability in SB outcome measures, as observed in the present study, has been reported before. Lavigne et al. (12) reported a mean coefficient of variation for SB Epi h^{-1} of approximately 25%. This percentage is based upon a retrospective assessment of a total of 37 PSGs, with 2–8 recordings per participants, which were obtained with an inter-recording interval that varied from 2 months to 7.5 years. In a longitudinal case study, a coefficient of variation for SB Epi h^{-1} of approximately 22% was found over 29 nights (34). Using the present prospective design, a mean coefficient of variation (i.e. SEM /mean x 100%) of approximately 37% was obtained for the bruxers. This coefficient is slightly higher than those obtained in previous PSG studies (12, 34). An even higher night-to-night variability in

SB outcome measures has been observed using ambulatory EMG recordings (35, 36). The differences between studies in SB variability may be related, amongst others, to differences in patient characteristics as well as to the fact that without the use of audio/video control, non-SB orofacial activities may have been included in the outcome measures (see above). Importantly, all studies suggest that variability in SB outcome measures should be taken into consideration when SB is being diagnosed or when the condition's treatment is being evaluated. Lavigne et al. (12) concluded that by estimating the night-to-night variability in SB patients will help avoiding misleading conclusions.

Consequences for the recognition of SB

Polysomnographic recordings recognize or deny the presence of SB when the SB outcome variables' values lie above or below these variables' cut-off points (13). However, how does the use of these points relate to the time-variant nature of the disorder? If the 'true' value of a SB outcome variable lies close to the cut-off point of that variable, the recorded SB outcome variable may for one night be above this point and for another night below this point. Do these PSG recordings then confirm or deny the presence of the SB disorder, or is it impossible to draw conclusions when the SB outcome variable lies too close to the cut-off point? This suggests that not cut-off points should be used for the recognition of SB, but rather cut-off bands. Single-night PSG outcomes outside these cut--off bands then deny or confirm the presence of the disorder, while PSG outcomes lying within the cut-off bands do not permit conclusions to be drawn.

What is the width of such a cut-off band? To explain the principle of these bands, the cut-off points of 4 Epi h^{-1} and 25 Bur h^{-1} that were previously suggested by Lavigne et al. (13) will be used as an example. Suppose that the chances of a single-night PSG recording of a non-bruxer to indicate the presence of SB should be smaller than 5% (false positive) and that the chances of a PSG recording of a bruxer to deny the presence of SB should also be smaller than 5% (false negative). Then, under the assumption that the single-night recordings are normally distributed, the cut-off band is the 90% probability interval around the cut-off point, with its upper and lower limits set at a distance of 1.645 times the SD from that point. The linear regressions, which were found between the SDs and mean values of the SB outcome variables Epi h^{-1} and Bur h^{-1} (Fig. 2a,b), predict that at these variables' cut-off points of 4 and 25, the best estimates for the SD of these outcome

variables are 1.50 and 9.63, respectively. Based upon these estimates, the cut-off band for Epi h^{-1} would then range from 1.5to 6.5
(i.e. 4 ± 1.645 x 1.50); that for Bur h^{-1}, from 9 to 41 (i.e. 25 ± 1.645 x 9.63). For single-night PSG recordings that yield SB outcome variables above or below these cut-off bands, the PSG recordings indicate that SB is present or absent. For single-night PSG recordings yielding SB outcome variables within the cut-off band, the PSG recordings do not allow conclusions to be drawn about the presence or absence of SB. When decisions are not based upon single-night PSG recordings but upon the average of n recordings, the cut-off bands will become narrower, since the SD of the average of n recordings is that of a single-night recording divided by √n. Further, when using a 95% instead of a 90% probability level, the cut-off bands would range from 1 to 7 (i.e. 4 ± 1.96 x 1.50) for Epi h^{-1} and from 6 to 44 (i.e. 25 ± 1.96 x 9.63)for Burh^{-1}.

Importantly, it must be stressed that the above-suggested cut-off bands can only be used when all data that are needed to construct them, are being derived from the same population sample. In other words, the cut-off bands which are described above, and which are based on a mixture of previous data (13) and present ones, should be considered illustrations of a principle rather than readily applicable criteria.

Consequences for the evaluation of SB therapy

For the evaluation of SB treatment, the natural fluctuations of SB are also important to take into account. If, after treatment, the SB outcome variable of a single PSG recording drops down below the above-described lower limit of its cut-off band, this suggests that at the time of the recording, the participant is not a bruxer anymore and the treatment can thus be considered successful. If the SB outcome variable drops down but not far enough to reach the non-bruxer values, it should be kept in mind that only changes equal to or greater than the SDD are statistically significant. Smaller changes may have nothing to do with the treatment itself but may simply be the result of the time-variant nature of the disorder. In this respect, it should also be realized that a statistically significant effect of a treatment modality does not automatically imply clinical relevance. Whether a change is clinically relevant, remains to be established by the responsible clinician and must be based upon medical grounds; not only on statistical ones.

Future research

For future research in which bruxers and non-bruxers are being compared using PSG recordings, it may be better to only include participants whose SB outcome variables are outside their respective cut-off bands. Only then, there is certainty about the actual status (non-bruxer or bruxer) of the participants. In this respect, it should be noted that before this suggestion can be adopted as a clinical or research tool, the upper and lower limits of the cut-off bands should be further tested in a larger population. In addition, the generalizability of such a tool can be improved when a less homogeneous population (in term of, e.g. co-morbidities) is used as compared with the current population as well as that of related studies (12–14, 25).

Conclusion

In short, the present study provides a quantitative insight into the time-variant nature of SB, and it suggests that, as a consequence, PSG cut-off bands around cut-off points might be useful for the recognition of SB.

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References

1. American Academy of Sleep Medicine. International classification of sleep disorders. 2nd ed. Westchester: American Academy of Sleep Medicine; 2005.
2. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep. 1994;17:739–743.
3. Laberge L, Tremblay RE, Vitaro F, Montplaisir JY. Development of parasomnias from childhood to early adolescence. Pediatrics. 2000;106:67–74.
4. Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil. 2001;28:1085–1091.
5. Lavigne GJ et al. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476–494.
6. The Academy of Prosthodontics. The glossary of prosthodontic terms. 7th edn. J Prosthet Dent. 1999;81:39–110.
7. Lobbezoo F, van der Zaag J, Visscher CM, Naeije M. Oral kinesiology. A new postgraduate programme in the Netherlands. J Oral Rehabil. 2004;31:192–198.
8. Svensson P, Jadidi F, Arima T, Baad-Hansen L. Relationships between craniofacial pain and bruxism. J Oral Rehabil. 2008;35:524–547.
9. Lobbezoo F, van der Zaag J, van Selms MKA, Hamburger HL, Naeije M. Principles for the management of bruxism. J Oral Rehabil. 2008;35:509–523.
10. Rugh JD, Solberg WK. Electromyographic studies of bruxist behavior before and during treatment. J Calif Dent Assoc. 1975;3:56–59.
11. Rugh JD, Harlan J. Nocturnal bruxism and temporomandibular disorders. Adv Neurol. 1988;49:329–341.
12. Lavigne GJ, Guitard F, Rompré PH, Montplaisir JY. Variability in sleep bruxism activity over time. J Sleep Res. 2001;10:237– 244.
13. Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res. 1996;75:546–552.
14. Rompré PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res. 2007;86:837–842.
15. Carlsson GE, Magnusson T. Management of temporomandibular disorders in the general dental practice. Chicago, IL: Quintessence Publishing Co Inc; 1999:36–37.
16. Kato T, Thie NM, Montplaisir JY, Lavigne GJ. Bruxism and orofacial movements during sleep. Dent Clin North Am. 2001;45:657–684.

17. Lobbezoo F, Naeije M. A reliability study of clinical tooth wear measurements. J Prosthet Dent. 2001;86:597–602.
18. Winocur E, Gavish A, Voikovitch M, Emodi-Perlman A, Eli I. Drugs and bruxism: a critical review. J Orofac Pain. 2003;17:99–111.
19. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord. 1992;6:301–355.
20. Visscher CM, Lobbezoo F, de Boer W, van der Zaag J, Verheij JGC, Naeije M. Clinical tests in distinguishing between persons with or without craniomandibular or cervical spinal pain complaints. Eur J Oral Sci. 2000;108:475–483.
21. Douglass AB, Bornstein R, Nino-Murcia G, Keenan S. Creation of the ASDC Sleep Disorders Questionnaire. Sleep Res. 1986;15:117.
22. Sweere Y, Kerkhof GA, de Weerd AW, Kamphuisen HAC, Kemp B, Schimsheimer RJ. The validity of the Dutch Sleep Disorders Questionnaire (SDQ). J Psychosom Res. 1998; 45:549–555.
23. Rechtschaffen A, Kales A. A manual of standardized terminology: techniques and scoring system for sleep stages of human subjects. Los Angeles, CA: UCLA Brain Information Service/Brain Research Institute; 1968.
24. Gallo LM, Lavigne G, Rompré PH, Palla S. Reliability of scoring EMG orofacial events: polysomnography compared with ambulatory recordings. J Sleep Res. 1997;6: 259–263.
25. Lavigne GJ, Rompré PH, Poirier G, Huard H, Kato T, Montplaisir JY. Rhythmic masticatory muscle activity during sleep in humans. J Dent Res. 2001;80:443–448.
26. van der Zaag J, Lobbezoo F, Wicks DJ, Visscher CM, Hamburger HL, Naeije M. Placebo-controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain. 2005;19:151–158.
27. Fleiss JL, Kingman A. Statistical management of data in clinical research. Crit Rev Oral Biol Med. 1990;1:55–66.
28. Kropmans TJ, Dijkstra PU, Stegenga B, Stewart R, de Bont LGM. Smallest detectable difference in outcome variables related to painful restriction of the temporomandibular joint. J Dent Res. 1999;78:784–789.
29. Lavigne GJ, Manzini C. Sleep bruxism and concomitant motor activity. In: Kryger M, Roth T, Dement WC, eds. Principles and practice of sleep medicine. Philadelphia, PA: WB Saunders Co; 2000:773–785.
30. Kato T, Thie NM, Huynh N, Miyawaki S, Lavigne GJ. Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain. 2003;17:191–213.
31. Le Bon O, Staner L, Hoffmann G, Dramaix M, San Sebastian I, Murphy JR et al. The first-night effect may last more than one night. J Psychiatr Res. 2001;35:165–172.

32. Carskadon MA, Dement WC. Normal human sleep: an overview. In: Kryger M, Roth T, Dement WC, eds. Principles and practice of sleep medicine. Philadelphia, PA: WB Saunders Co; 1994:16–25.
33. Fry JM, DiPhillipo MA, Curran K, Goldberg R, Baran AS. Full polysomnography in the home. Sleep. 1998;21:635– 642.
34. Dal-Fabbro C, Tufik S. A linear study of a man with sleep bruxism for 30 consecutive nights – preliminary reports. J Sleep Res. 1996;5(Suppl. 1):41–82.
35. Rugh JD. Electromyographic analysis of bruxism in the natural environment. In: Weinstein P, ed. Advances in behavioral research in dentistry. Seattle, WA: University of Washington Press; 1978:67–83.
36. Funch DP, Gale EN. Factors associated with nocturnal bruxism and its treatment. J Behav Med. 1980; 3:385–397.