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Agreement between clinical and portable EMG/ECG diagnosis of sleep bruxism

T. CASTROFLORIO*, A. BARGELLINI*†, G. ROSSINI*†, G. CUGLIARI‡, A. DEREGIBUS*† & D. MANFREDINI§ **Department of Surgical Sciences, Specialization School of Orthodontics, Dental School, University of Torino, Torino, †Gnathology Unit, Department of Surgical Sciences, Dental School, University of Torino, Torino, ‡Unit of Medical and Genomic Statistics, Department of Brain and Behavioural Sciences, University of Pavia, Pavia, and §Department of Maxillofacial Surgery, TMD Clinic, University of Padova, Padova, Italy*

SUMMARY The aim of this study was to compare clinical sleep bruxism (SB) diagnosis with an instrumental diagnosis obtained with a device providing electromyography/electrocardiography (EMG/ECG) recordings. Forty-five ($N = 45$) subjects (19 males and 26 females, mean age 28 ± 11 years) were selected among patients referring to the Gnathology Unit of the Dental School of the University of Torino. An expert clinician assessed the presence of SB based on the presence of one or more signs/symptoms (i.e. transient jaw muscle pain in the morning, muscle fatigue at awakening, presence of tooth wear, masseter hypertrophy). Furthermore, all participants underwent an instrumental recording at home with a portable device (Bruxoff®; OT Bioelettronica, Torino, Italy) allowing a simultaneous recording of EMG signals from both the masseter muscles as well as heart frequency. Statistical procedures were performed with the software Statistical Package for the Social Science v. 20.0 (SPSS 20.0®; IBM, Milan, Italy). Based on the EMG/ECG analysis, 26 subjects (11 males, 15 females, mean age 28 ± 10 years) were diagnosed as sleep bruxers, whilst 19 subjects (7 males, 12 females, mean age 30 ± 10 years) were diagnosed as non-bruxers. The correlation between the clinical and EMG/ECG SB diagnoses was low (ϕ value = 0·250), with a 62·2% agreement (28/45 subjects) between the two approaches (kappa = 0·248). Assuming instrumental EMG/ECG diagnosis as the standard of reference for definite SB diagnosis in this investigation, the false-positive and false-negative rates were unacceptable for all clinical signs/symptoms. In conclusion, findings from clinical assessment are not related with SB diagnosis performed with a portable EMG/ECG recorder.

KEYWORDS: sleep bruxism, bruxism, clinical criteria, diagnosis

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Introduction

Sleep bruxism (SB) is a sleep-related motor phenomenon characterised by involuntary phasic (rhythmic) or tonic (sustained) motor activity in the masticatory muscles (e.g. masseter, temporalis) during sleep. It can be associated with a number of clinical problems, including oro-facial pain, tooth wear and failure of dental restorations (1, 2). Furthermore, it is considered a risk factor for complications in implant- and teeth-supported rehabilitations (3, 4).

Over the years, several strategies were proposed to diagnose bruxism (5, 6). The literature shows that the wide majority of data came from studies adopting a self-reported bruxism detection (7). Such an approach is suitable, at best, to indicate a 'possible' bruxism (8) and is in contrast with the proposed standard of reference for SB diagnosis, which require 'definite' measurements by means of polysomnography (PSG) (8–11). Nonetheless, PSG has some disadvantages (e.g. high cost, amount of time needed for manual/visual scoring, laboratory environment) (12), and it is

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mainly used for research purposes, with minor impact on the clinicians' daily routine (13). Indeed, in the clinical setting, SB diagnosis is still mainly based on clinical assessment (10).

A recent consensus panel suggested that such clinical diagnosis is able to detect, at best, a 'probable' bruxism (8). Notwithstanding that, it should be remarked that PSG/SB criteria themselves were originally validated against a set of clinical/anamnestic criteria for sleep-time bruxism (9).

Thus, there is a need to assess the relationship between the various grades of SB diagnosis, as also recently suggested in a paper on the correlation between the different clinical and anamnestic SB findings (14). Based on that, also the actual relationship between clinically based 'probable' SB and measurement-based 'definite' SB is worthy to be further investigated.

Recent studies have validated a portable device providing combined surface electromyography (EMG) and electrocardiography (ECG) measurements, which showed an excellent diagnostic accuracy with respect to PSG for the diagnosis of SB (15–17). The adoption of such portable devices could ease the assessment of the relationship between clinically diagnosed and instrumentally diagnosed SB.

Based on these premises, the aim of this study was to compare the 'probable' SB diagnosis based on the clinical assessment with the instrumental SB diagnosis obtained with a portable device providing EMG/ECG recordings. The study design aims to answer the clinical research questions: 'is there a correlation between the clinical and instrumental SB diagnosis?' The null hypothesis was that purported clinical signs and symptoms of SB (i.e. transient jaw muscle pain in the morning, muscle fatigue at awakening, presence of tooth wear or shiny spots on restorations, masseter hypertrophy) are not related with instrumentally diagnosed SB. If the null hypothesis was rejected, the diagnostic value of clinical SB diagnosis could approximate the needed requirements for a 'definite' diagnosis, thus having potentially relevant clinical implications.

Materials and methods

Subjects and study design

The study was performed on 45 subjects [19 men and 26 women, mean age ± standard deviation (s.d.) 28 ± 11 years] selected among patients referring to the Gnathology Unit of the Dental School of the University of Torino. To ensure that individuals with different SB severity took part to the study, participants were initially recruited based on a clinical assessment suggesting their probable bruxism ($N = 22$, 10 males and 12 females, mean age ± s.d. 26 ± 4) or the absence of bruxism ($N = 23$, 9 males and 14 females, mean age ± s.d. 32 ± 14). Exclusion criteria were (i) presence of extensive prosthodontic rehabilitations, (ii) missing teeth, with the exception of the third molars, (iii) periodontal disease, (iv) Presence of temporomandibular joint disorders, as diagnosed with the Research Diagnostic Criteria for TMD (18), (v) medical history of neurological, mental or sleep disorders (e.g. periodic leg movements, insomnia). Furthermore, the subjects were not under medications at the time of recording and were not under the effect of alcohol, nicotine or caffeine.

In the morning hours, an expert clinician made the clinical assessment for SB based on the presence of the following diagnostic criteria:

1. transient jaw muscle pain in the morning, as confirmed by pain elicitation in the masseter muscles upon palpation, as diagnosed according to a positive palpation of at least one of the three masseter muscle sites per side described in the Research Diagnostic Criteria for TMD guidelines (18);
2. muscle fatigue at awakening, reported by the patient;
3. presence of tooth wear or shiny spots on restorations, as assessed by the presence of noticeable (at least grade 2) (19) wear spots on the incisal surfaces of the anterior teeth and/or on the guiding cusps of the posterior teeth;
4. masseter hypertrophy upon digital palpation, scored positively if the muscle volume approximately tripled upon a voluntary clench in maximal intercuspal position (9).

Based on that, the presence of one or more of the above clinical signs/symptoms was suggestive of a clinical SB diagnosis.

All participants underwent an instrumental in-home evaluation with a portable device (Bruxoff®*) allowing a simultaneous recording of EMG signals

*OTBioelettronica, Torino, Italy.

from both the masseter muscles as well as heart frequency. The three signals were sampled at 800 Hz, with 8 bit resolution. Data were stored on a MicroSD card as a binary file. The EMG and the ECG channels were filtered between 10 and 400 Hz with a gain of 4300, and between 15 and 160 Hz with a gain of 700, respectively. Masseter muscles' EMG activity was detected with disposable bipolar AgCl concentric electrodes (Code®†), with a 16 mm radius. The choice of adopting such electrodes was due to their easy applicability and design, avoiding muscle fibre electrode orientation problem and reducing EMG crosstalk (20, 21). ECG recordings were detected with a disposable bipolar electrode located on the left side of the thorax, at about 5-10 cm below the sternum.

Each participant underwent two consecutive recording nights (at least 4 h of sleep per night). The first night was an accommodation session to familiarise with the device, and only data recorded during the second night were considered for statistical analyses. The recording procedure provided that five tapping movements before sleep and after getting up in the morning were performed, to easily recognise the beginning and the end of the recordings. After the five tapping movements at the beginning of the recording session, the subjects performed three maximum voluntary clenching (MVC) on teeth. The clenches should last 3 s each and be separated by a 10-s rest. The highest MVC value was used to normalise the EMG values as a per cent of MVC. Masseter EMG bursts with duration exceeding 0·25 s were selected for oromotor activity scoring (9, 22).

Previous studies showed that the portable device has high sensitivity (92·3%) and specificity (91·6%) for SB diagnosis when the diagnostic cut-off was set at 4 SB episodes per hour (15), as suggested by the most recent PSG/SB criteria (23). In addition, a reliability study showed a good repeatability as far as the number of SB episodes per night, SB episodes per hour and heart frequency are concerned (16).

The Bruxoff software (Bruxmeter software®*) scores automatically the presence of SB events based on the following features: mean masseter EMG amplitude at least 10% of maximum voluntary clenching activity, preceded (1-5 s interval) by an approximately 20% increase of heart rate (beginning 1 s before RMMA onset) (9, 23). Oromotor activity during wakefulness before falling asleep was excluded from scoring.

The procedures were approved by the Ethic Committee of the Lingotto Dental School, University of Turin, Italy. All individuals gave their informed consent in accordance with the Helsinki Declaration and understood that they were free to withdraw from the experiment at any time.

Statistical analysis

The design of statistical analyses aimed to answer the underlying clinical research question of the study, viz., assessing the correlation between clinical and instrumental bruxism diagnosis.

The frequency of the presence of various clinical signs/symptoms as well as of a positive SB diagnosis with the Bruxoff device was previously described in "subjects and study design" section. Contingency tables were created to compare the Bruxoff findings (columns) and the clinical variables (rows). The correlation between the clinical findings and the instrumental diagnosis was assessed by means of ϕ coefficient, which is a measure of the degree of association between two binary variables. Such coefficient is similar to the correlation coefficient in its interpretation: ϕ values range from −1·0 to + 1·0, indicating different levels of negative or positive correlation. As a general rule for correlation analyses, values higher than 0·7 are considered supportive of a strong positive correlation (24).

In addition, a *t*-test for unpaired samples was performed to compare the mean SB index, as derived with the Bruxoff device, of subjects having or not having the various clinical findings.

All statistical procedures were performed with the software Statistical Package for the Social Science v. 20.0 (SPSS 20.0®‡). For each analysis, a P-value<0·05 was set.

Results

Based on the Bruxoff software analysis, 26 subjects (11 males, 15 females, mean age 28 ± 10 years) were diagnosed as sleep bruxers, whilst 19 subjects (7 males, 12 females, mean age 30 ± 10 years) were diagnosed as non-bruxers. The correlation value between the clinical

†Spes Medica, Battipaglia, Italy.

‡IBM, Milan, Italy.

Table 1. Cross-tabulation of sleep bruxism (SB) diagnosis based on either clinical or EMG/ECG findings

	EMG/ECG diagnosis	
	No SB	SB
Clinical diagnosis		
No SB	14	7
SB	10	14

and EMG-ECG SB diagnoses was low (φ value = 0·250), with a 62·2% agreement (28/45 subjects) between the two approaches (kappa = 0·248) (Table 1).

The frequency of positive clinical items in the study sample ranged between 31·1% for facial pain and muscle stiffness and fatigue at awakening to 42·2% for tooth wear or shiny spots on restorations and up to 53·3% for masseter hypertrophy. The correlation values with SB were low for each clinical sign/symptom, ranging from $\phi = -0{\cdot}045$ to 0·196 (Table 2), and the agreement with instrumental SB ranged between 46·6% (21/45 subjects) for muscle stiffness at awakening and 60% (27/45) for masseter hypertrophy. Assuming instrumental EMG/ECG diagnosis as the standard of reference for definite SB diagnosis in this investigation, the false-positive and false-negative rates were unacceptable for all clinical signs/symptoms (Table 3).

The average SB index was different between subjects having or not having masseter muscle hypertrophy ($P = 0{\cdot}033$), whilst there were not any significant differences for the other clinical signs/symptoms, with P values ranging from 0·351 to 0·645 (Table 4).

Based on the above, the null hypothesis that purported clinical signs and symptoms of SB (i.e. jaw pain, masseter muscle hypertrophy, tooth wear or shiny spots on restorations, morning stiffness in the jaw muscles) are not related with instrumentally diagnosed SB could not be rejected.

Table 2. Prevalence of the different clinical signs/symptoms in subjects with or without EMG/ECG diagnosed sleep bruxism (SB) and levels of correlation with SB

	Prevalence in SB (%)	Prevalence in non-SB (%)	Correlation value (ϕ)
Transient jaw muscle pain in the morning	33·3	28·6	0·051
Muscle fatigue at awakening	29·2	33·3	−0·045
Tooth wear	41·7	42·9	−0·012
Masseter hypertrophy	62·5	42·9	0·196

Discussion

The aim of this study was to compare the clinical diagnosis of SB, viz., a so-called probable bruxism, with SB diagnosis based on EMG/ECG recordings obtained with a validated portable device.

Over the years, several clinical signs and symptoms have been proposed as markers of SB. They include, among the others, the presence of transient jaw muscle pain in the morning, a feeling of fatigue or stiffness in the jaw muscles at awakening, abnormal tooth wear and masseter muscles' hypertrophy.

The presence of at least one of the above clinical signs or symptoms, together with reported tooth grinding during sleep, has been used to validate PSG-SB criteria (9, 23). As those validation studies, none of these signs and/or symptoms has been directly associated with ongoing SB (5). Nonetheless, the

Table 3. False-positive and false-negative sleep bruxism (SB) findings based on the presence of clinical signs/symptoms and their agreement with SB diagnosis

	False-positive SB findings (%)	False-negative SB findings (%)	Agreement (%)
Transient jaw muscle pain in the morning	42·9	51·6	51·1
Muscle fatigue at awakening	50·0	54·8	46·6
Tooth wear	47·4	53·8	48·8
Masseter hypertrophy	37·5	42·9	60

Table 4. Sleep bruxism index of subjects with and without the different clinical signs/symptoms

	SB index of positive subjects (%)	SB index of negative subjects (%)	P-value
Transient jaw muscle pain in the morning	5·0 ± 3·4	4·1 ± 2·8	0·351
Muscle fatigue at awakening	4·1 ± 3·1	4·5 ± 3·0	0·645
Tooth wear	4·8 ± 3·6	4·08 ± 2·5	0·431
Masseter hypertrophy	5·2 ± 3·4	3·3 ± 2·1	0·033

evaluation of their presence is still considered the best available approach to diagnose SB clinically (8,10).

Results of this investigation show that such clinical criteria do not correlate with an instrumental SB diagnosis. Indeed, none of them is significantly related with SB findings based on the EMG/ECG recordings, with the minor exception of a higher SB index in subjects with masseter hypertrophy. This implies that the resulting clinical diagnosis had a very poor agreement ($k = 0{\cdot}248$) with the definite SB diagnosis. Of course, as a main limitation of this study, it must be remarked that, for an actual diagnosis of definite SB to be made, full PSG recordings should have been required. Anyhow, their adoption is unlikely to change the study findings. Indeed, the EMG/ECG recorder adopted in this investigation showed an excellent correlation with PSG findings in a previous study (15) and was thus introduced in the research setting to ease data gathering.

Despite seemingly discouraging, data from this investigation are actually in line with the fragmental literature on the relationship of SB with pain and tooth wear. In general, the literature suggested that the proposed PSG cut-off values for SB were suitable for discriminating between patients with and without tooth wear (25), whilst they were not suitable to intercept subjects who are at risk for developing pain in the jaw muscles (26–30).

Our findings are open to interesting considerations. Indeed, at a first glance, it could be concluded that a clinical SB diagnosis is not acceptable, so that even the recently defined 'probable' bruxism is far from being 'probable'. On the other hand, despite quantitative recordings are without any doubts the standard requirement for a definite SB diagnosis, it emerged that several issues need to be clarified concerning the interpretation of bruxism measurements. Indeed, it seems that neurologically driven criteria drawn from PSG studies are not related with the clinical consequences of SB, especially as far as muscle fatigue and pain are concerned.

A possible explanation for such lack of relationship is that EMG adaptations to pain in the jaw muscles may lead to a reduced muscle activity (i.e. less SB) in patients with pain (31–33). This means that even those types of bruxism activities (e.g. prolonged, high intensity, isometric contractions such as in the case of mandible thrusting) that are plausible risk factors for muscle pain are likely to be detected as such only in the early stages of pain onset, before protective adaptations turn in to reduce muscle activity (13).

A recent review suggested how to refine some concepts underlying a potentially 'ideal' SB diagnosis (13). Based on that, this study's findings support the view of SB as a variegated motor phenomenon, and not as a disorder *per se*. Thus, until the different motor activities that are currently grouped together under the umbrella term 'bruxism' are not properly discriminated based on their EMG features, it is unlikely that we are able to get deeper into the clinical picture.

In short, taken together, our findings suggested that currently proposed clinical diagnostic criteria for SB are not evidence based.

Conclusions

This study showed that findings from clinical assessment are not related with SB diagnosis performed with a portable EMG/ECG recorder. Further studies on larger and more representative samples are needed to get a deeper insight into the relationship between an instrumental SB diagnosis and the purported clinical signs/symptoms.

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Conflict of interest

The authors do not have any financial conflict of interests or relationship with any financial organisation that may be interested in the contents of this manuscript. The authors declare that all them have contributed to conceptualise and perform the investigation as well as to manuscript's writing and revision before submission.

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Correspondence: Andrea Bargellini, Department of Surgical Sciences, Specialization School of Orthodontics, Dental School, University of Torino, Via Nizza 230, 10126 Torino, Italy.
E-mail: bargelli@ipsnet.it