Diagnostic Accuracy of a Portable Electromyography and Electrocardiography Device to Measure Sleep Bruxism in a Sleep Apnea Population: A Comparative Study

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Article

Diagnostic Accuracy of a Portable Electromyography and Electrocardiography Device to Measure Sleep Bruxism in a Sleep Apnea Population: A Comparative Study

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Abstract: Background: The gold standard for the diagnosis of sleep bruxism (SB) and obstructive sleep apnea (OSA) is Polysomnography (PSG). At the end of the apnea episodes there is frequently a final hyper motor muscle activity that could act as a confusion factor in the diagnosis of SB with the electromyography portable devices. The aim of this study was to compare the concordance on the number of episodes of SB in a population with OSA suspected, between the diagnosis obtained by PSG, analyzed manually by a sleep expert and that obtained manually and automatically by a portable electromyography (EMG) and electrocardiography (EKG) device. Methods: Twenty-two subjects underwent one night of polysomnographic study with simultaneous recording with the EMG-EKG device. Variables referring to the number of SB episodes and SB index measured with both tools, and analyzed in the manual and automatic modes, were compared. Masticatory muscle activity was scored according to published criteria. After PSG testing, patients were segmented by severity of OSA according to the American Academy of Sleep Medicine (AASM) criteria. ANOVA and the Bland–Altman plot were used to quantify the agreement between both methods. The concordance was calculated through the Intraclass Correlation Coefficient (ICC). Results: The total events of SB per night in the PSG study were on average (8.41), lower than the one obtained with EMG-EKG manual (14.64) and automatic (22.68) analysis. SB episodes mean number decreases from non-OSA group to OSA group with both PSG (5.93, $p<0.05$) and EMG-EKG analyses (automatic = 22.47, manual = 13.93). However, this decreasing was in a minor proportion with the automatic EMG-EKG analysis mode (from 23.14 to 22.47). The ICC based on the number of SB episodes in the segmented sample by severity degree of OSA along the three tools (PSG, manual EMG-EKG and automatic EMG-EKG) shows an acceptable agreement in non-OSA (0.61) and mild OSA (0.53 *) groups. However, it is insufficient in the moderate (0.24) and severe (0.23) OSA groups: the EMG-EKG automatic analysis measure 14.27 units more than PSG. The results with the manual EMG-EKG analysis improved this correlation, but are not good enough. Conclusion: There is an acceptable concordance between the results obtained in the PSG manual analysis and those obtained by the EMG-EKG device with automatic and manual analysis for the diagnosis of SB, but only in patients without OSA or with mild OSA. In patients with moderate or severe OSA, apneas could act as a confusion factor in the diagnosis of SB with electromyographic portable devices, although further study is needed.

Keywords: bruxism; electromyography; sleep apnea; polysomnography; sleep bruxism; sleep wake disorders

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1. Introduction

Bruxism is a repetitive activity of the masticatory musculature, characterized by grinding, clenching of the teeth and/or sustained jaw thrust without tooth contact. In addition, bruxism can occur during sleep (sleep bruxism) and/or during wakefulness (awake bruxism) (1). Sleep Bruxism (SB) also occurs concomitantly or secondarily to other sleep disorders, such as Obstructive Sleep Apnea (OSA) (2–8). OSA consists of recurrent episodes of partial or total upper airway obstruction, accompanied by sleep fragmentation caused by arousals and commonly accompanied by snoring (9), in addition to other complications (hypertension, arrhythmias, cardiovascular disease, etc.) (10,11). This increases the difficulty in achieving a high diagnostic yield with electromyography (EMG) devices, and the criteria for neurophysiological analysis of these recordings vary among different studies (12–18). Differentiating between masticatory muscle activity (MMA), rhythmic masticatory muscle activity (RMMA), sleep-related oromotor activity (OMA) and recognized bruxing activity is a challenge during the polysomnography (PSG) analysis itself. These types of activity are not always properly recognized by the algorithms programmed into the automatic analysis mode of EMG devices and not all EMG devices offer manual analysis mode. Moreover, the criteria used for the manual mode of these recordings are not uniform (12,19–28). PSG studies in sleep laboratories include electroencephalography (EEG), electrooculogram (EOG), electrocardiogram (EKG), EMG recordings (of the masticatory muscles and tibial muscles), and thoracoabdominal movements recordings. It is also includes oronasal flow and oxygen saturation, allowing a definitive evaluation of SB and the detection of other disorders such as OSA, parasomnias, or restless legs syndrome (29–31).

However, the cost of PSG is high and requires very sophisticated instruments and highly specialized personnel, making its application unfeasible in dental clinics, and in particular in the general dental practice. Therefore, in recent years, portable ambulatory instruments have been developed, providing information similar to PSG but more affordable and easier to handle. Their validity is still under discussion and requires further research, but they can be very useful as a clinical approach to SB evaluation (32)

EMG-EKG is a three-channel Holter-type device designed to detect the surface EMG signal of the two masseter muscles and the heart rate (HR) by EKG. This EKG capability is what differentiates this device from other portable devices and supports its efficacy. The reliability of EMG-EKG has been proven with very good diagnostic yield (19,33,34). However, these studies have not been developed in an OSA population. There are also many patients with undiagnosed OSA. Knowing that OSA is a disease frequently concomitant with SB, we considered the need to assess the reliability of the ambulatory EMG for this type of population.

2. Materials and Methods

Twenty-two (n=22) participants underwent a full-night PSG testing with simultaneous recording of the Bruxoff® EMG-EKG device (OT Bioelettronica, Italy). Procedures were conducted following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement and checklist (35). The study protocol was approved by the Ethics Committee of the Hospital Clínico San Carlos in Madrid, (C.P. - C.I. 14/380-E). Written informed consent was obtained from all participants and all procedures were conducted according to the Declaration of Helsinki. Variables referring to the number of SB episodes and SB index (episodes / hour), measured with both tools and analyzed in their manual and automatic modes, were compared. Masticatory muscle activity was scored according to published criteria (24,31). After PSG testing, the sample was segmented by severity of OSA according to AASM criteria (24).

2.1. Sample Selection:

The participants of the study are adult patients attended by the Sleep Unit (Clinical Neurophysiology Department) of San Carlos University Hospital (Madrid, Spain) who underwent an earlier screening according to the suspicion of OSA and SB, the latter by self-referred tests (Paesani

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modified test) and physical examination (36). Exclusion criteria were major neurological disorders, psychiatric disorders, other sleep disorders, psychoactive medication, or edentulism. The clinical examination (tooth wear, masticatory muscle myalgia, TMJ arthralgia, hard tissue, soft tissue, and masseter and/or temporal hypertrophy) was performed according to Diagnostic criteria for the temporomandibular disorders (DC/TMD) and the American Academy of Orofacial Pain (AAOP) criteria and conducted by a dentist with ability in orofacial pain (37,38). Finally, to the patients which did not incur in the exclusion criteria and had a positive SB screening, a PSG diagnosis was performed by an experienced clinical neurophysiologist with specific training in SB. EMG-EKG with artifacts or other technical problems were excluded. As a result, a sample of 22 subjects with an average age of 46.55 ± 10.06 was achieved, including 15 men and 7 women. A concordance between EMG-EKG device and the PSG (Gold Standard) design was used with six participants without OSA and sixteen with OSA. The sample of OSA patients was segmented by the degree of severity in three groups: Mild OSA = 7, Moderate OSA = 3, Severe OSA = 6.

2.2. PSG recordings

The full-night monitoring recordings in the Sleep Laboratory (minimum of 8 hours in bed) were performed using a Deltamed Coherence 5.0 system. PSG recordings were made according to the AASM recommendations (24), including: six EEG derivations; right and left EOG; submental, masseter and leg EMG; nasal cannula/pressure and oronasal thermal flow; thoracic and abdominal respiratory effort bands; snoring; body position sensor; pulse-oximetry; audio and video recordings. Impedance values were checked and adjusted (< 5 Ω), and standard calibrations were performed. All PSG recordings were manually reviewed according to international criteria (24). In the SB and OSA group the diagnosis was confirmed by PSG performed by a sleep expert, following blind masking with respect to the clinical examination.

2.2.1. PSG Sleep Bruxism Analysis

SB events were estimated through rhythmic (RMMA; Figure 1), and non-rhythmic masticatory muscle activity (MMA) recorded with EMG on the masseter muscles (surface electrodes). Published criteria for SB episodes in PSG were followed [25]. For the calculation of dichotomous variables, the presence of > 4 RMMA-MMA/SB episodes/h was considered. For the calculation of quantitative variables, the type of SB event is decided: phasic event (three or more EMG burst, at least 0.25 seconds and up to 2.0 seconds), tonic event (at least one EMG burst > 2.0 seconds), and mixed event (both types) (31,39). Increased muscle tone following an apnea episode, which is part of the AASM criteria definition of arousal (24), as well as sleep related oromotor activity (OMA; Figure 2) different from RMMA-MMA/SB were excluded to avoid possible confounding bias. All isolated SB events, independent of respiratory events, were accepted according to EMG criteria, regardless of whether accompanied by arousals.

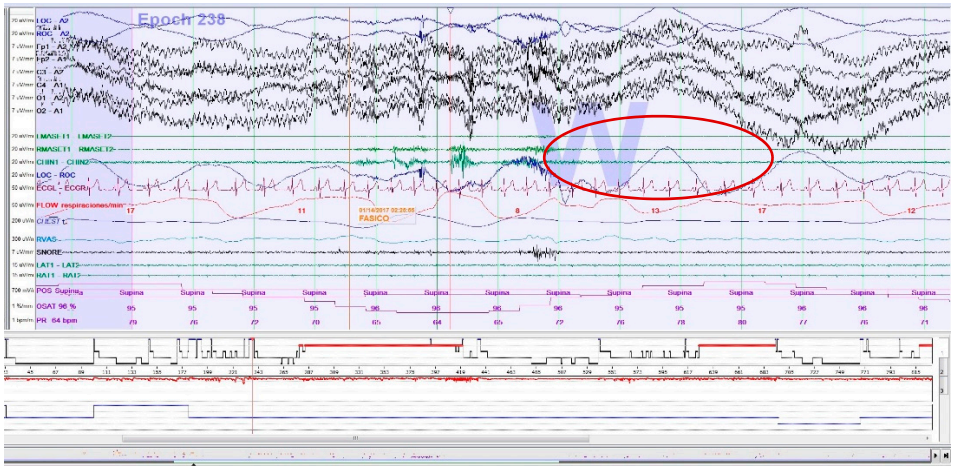

Figure 1. Epoch (30 seconds) of a PSG recording: An EMG phasic event of SB.

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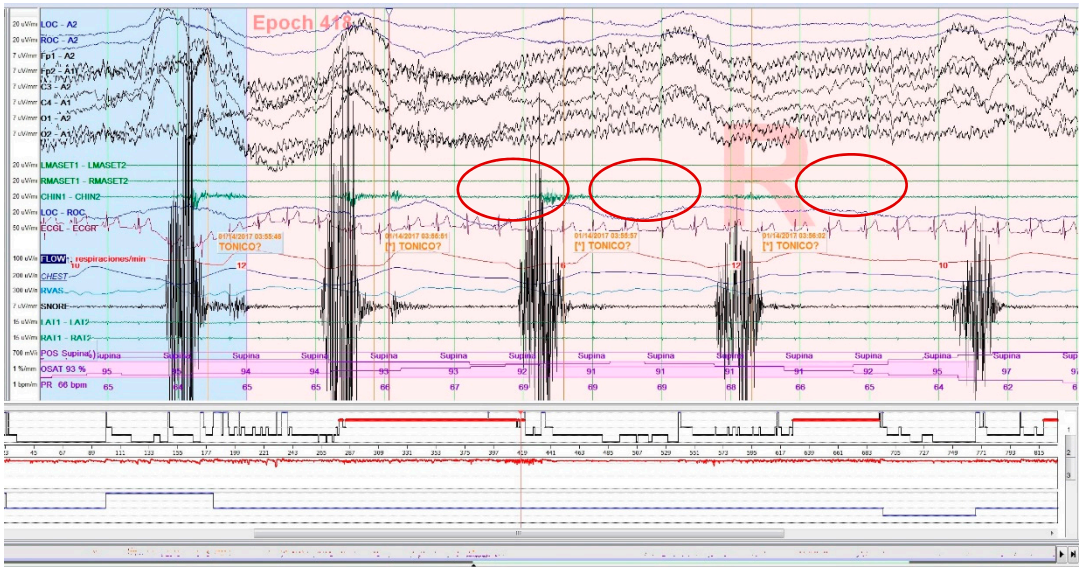

Figure 2. Epoch (30 seconds) of a PSG recording: Tonic EMG episode, corresponding to Sleep-related Oromotor activity (OMA), following snoring. It is excluded as SB episode.

2.3. Bruxoff Sleep Bruxism Analysis

Bruxmeter is the software system of the EMG-EKG device (Bruxoff®; Figure 3 and Figure S1).

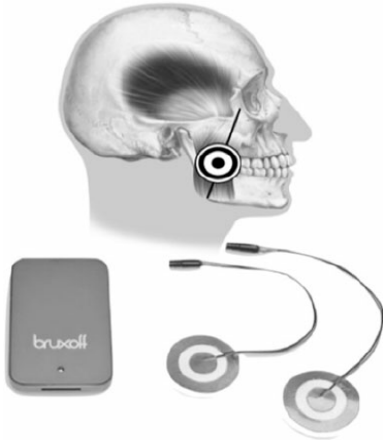

Figure 3. EMG-EKG Device.

Interpretation is performed both manually, with the investigator analyzing the raw data, and automatically, with the device's software analyzing the data to generate a diagnosis. When automatically analyzed reached a sensitivity of 91.6% (19).

The MicroSD card provided data for the diagnostic variables: bruxing event, number of bruxing events per hour of sleep (SB index), and number of bruxing events per night.

The bruxism event criteria depend on whether the analysis is performed in manual or automatic mode. Manual mode: EMG signal with peaks > 0.25s and an average amplitude of 10% of the patient's maximum voluntary contraction (MVC), being preceded 1s earlier by an increase in heart rate (HR) of 15%. Automatic mode: EMG signal with an amplitude of at least 10% of the patient's MCV, preceded by an increase in HR of 20%, 1-5s before (Figure 4).

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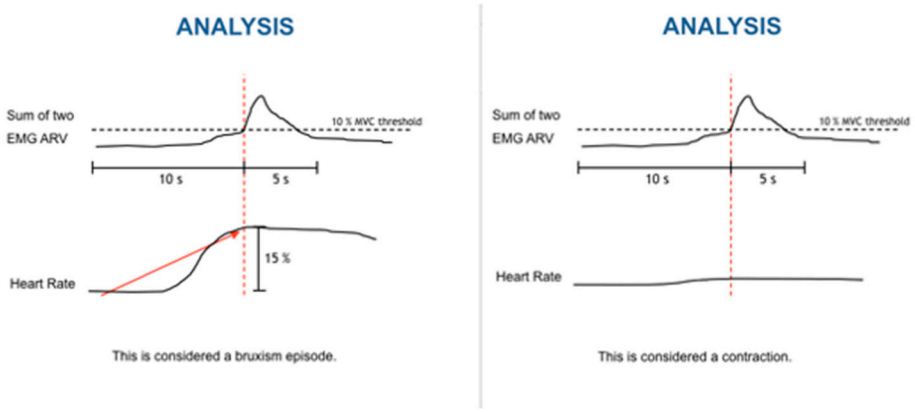

Figure 4. Algorithm used by bruxmeter software to detect SB episodes in automatic mode.

2.4. Statistical analysis

The variables used were Apnea Hypopnea Index (AHI), SB Index, number of apnea events, number of hypopnea events, and number of SB events. Descriptive variables as means and standard deviations was used. Continuous variables with a normal distribution were analyzed by the t-test. In addition, the sample was segmented according to the degree of severity of OSA and according to the types of SB episodes. The Analysis of variance (ANOVA) between non OSA group and several degree severities of OSA was calculated. Spearman correlation for the apnea and hypopnea episodes and SB episodes was used. The Bland–Altman plot were used to quantify the agreement between both methods (PSG and EMG-EKG). The concordance was calculated through the Intraclass Correlation Coefficient (ICC). All calculations were performed with the SPSS v24.0 statistical package (SPSS Inc., Chicago, IL). The p values equal to or less than 0.05 were considered statistically significant.

3. Results

The descriptive sleep data (Table 1) shows a sample with predominantly overweight. The time on sleep stages is inside normal values except for the augmented proportion of N1 stage (25.49 ± 16.32). The oximetry data means are compatible with a partial sleep apnea population pulse oximetry affecting values. The mean of sleep efficiency (81.18 ± 14.18) is in the average value for the PSG testing in a sleep laboratory.

Table 1. Data of Sleep (n=22).

	Mean (S.D.)
Physical data	
Age	46.55 (11.06)
BMI	27.23 (5.38)
Sleep data	
SPT (min)	411.55 (27.31)
TST (min)	330.05 (62.42)
SLT (min)	13.86 (26.87)
Sleep Efficiency (%)	81.66 (14.89)
WASO (min)	56.30 (47.86)
Awakes (number)	44.05 (25.16)
Sleep stage distribution	
N1/SPT (%)	25.12 (16.60)

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	Mean (S.D.)
N2/SPT (%)	43.85 (9.37)
N3/SPT (%)	15.99 (10.91)
R/SPT (%)	15.43 (5.92)
Pulse oximetry data	
Mean (%)	93.45 (2.98)
Max (%)	98.32 (1.04)
Min (%)	81.09 (11.03)
CT90 (%)	12.94 (23.65)
Sleep apnea data	
No. Apneas	106.18 (161.17)
No. Hypopneas	30.32 (31.24)
No. Apneas + Hypopneas	136.50 (172.87)
IAH	25.25 (32.83)

BMI Body Mass Index, SPT sleep period time, TST total sleep time, SLT sleep latency time, WASO wake time after sleep onset, CT90 total time lower 90% O2Sat.

The total events of SB per night in the PSG study were on average (8.41), lower than the one obtained with EMG-EKG device manual analysis (14.64) and automatic (22.68). The tonic SB episodes predominately against phasic SB episodes along the PSG and manual EMG-EKG analysis (Table 2). The Spearman correlation between the apnea and hypopnea episodes and the SB episodes is negatively [r = - 0.402 (p = 0.06)] in the total of the sample, this means that when the number of apnea episodes increase, the number of SB episodes decrease with PSG recordings.

Table 2. Data of Sleep bruxism (n=22).

	Mean (S.D.)	t
Polysomnography		
No. Episodes / night	8.41 (10.85)	3.63
No. Episodes / hour	1.49 (2.05)	3.39
No. Phasic episodes	2.00 (4.48)	2.09
No. Tonic episodes	5.55 (7.06)	3.68
No. Mixed episodes	0.86 (1.67)	2.42
Automatic Bruxoff		
No. SB episodes	22.68 (16.02)	6.64
No. Episodes / hour	3.92 (2.71)	6.78
No. Phasic episodes	5.82 (5.37)	5.06
No. Tonic episodes	5.77 (6.90)	3.87
No. Mixed episodes	1.23 (1.87)	3.06
Manual Bruxoff		
No. Episodes / night	14.64 (10.76)	6.37
No. Episodes / hour	2.54 (1.95)	6.13
No. Phasic episodes	5.27 (4.50)	5.49

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	Mean (S.D.)	t
No. Tonic episodes	8.05 (7.82)	4.82
No. Mixed episodes	1.32 (2.00)	3.07

Total SB events along the TST total sleep time, excluded the Sleep-related Oromotor Activity (OMA) with the EMG-EKG device and the Gold Standard (PSG, Manual EMG-EKG, and Automatic EMG-EKG). $p<0.05$.

When we compared the variables of SB between the OSA (n =7) and non OSA (n= 15) group, we obtained an increase of SB episodes from PSG analyses (13.71) to manual (16.14) and automatic (23.14) EMG-EKG analyses in the non OSA group, respectively. The SB episodes number mean decreased from non OSA group to OSA group with both PSG (5.93, $p<0.05$) and EMG-EKG analyses (automatic = 22.47, manual = 13.93). However, this decreasing was in minor proportion with the automatic EMG-EKG analysis mode (from 23.14 to 22.47) (Table 3.)

The phasic episodes were considerably lower on the OSA group with PSG analysis compared to EMG-EKG results ($p<0.05$) (Table 3). Segmenting the sample by the degree severity of OSA, the severe OSA patients had less SB episodes than moderate or mild OSA patients with both PSG and EMG-EKG recordings ($p>0.05$). The tonic episodes predominate against phasic episodes. The phasic episodes decrease considerably from non OSA to OSA patients with PSG analysis compared to EMG-EKG analysis (Table 4).

Although the ICC [0.55 ($p<0.05$)] based on the number of SB episodes accounts in all the subjects along the three tools (PSG, manual EMG-EKG and automatic EMG-EKG) shows an acceptable agreement, a wide dispersion can be observed with the Bland-Altman representation (Table 5). The EMG-EKG automatic analysis measures 14.27 units more than PSG. The results with the manual EMG-EKG device analysis improved (measures 6.22 units more than PSG), but were not good (Figure 5). The limits agreement of both EMG-EKG automatic and manual analysis are out of the desirable limits of the S.D. The ICC based on the number of SB episodes accounts in the segmented sample by severity degree of OSA along the three tools (PSG, manual EMG-EKG and automatic EMG-EKG) shows an acceptable agreement in Non OSA (0.61) and mild OSA (0.53, $p<0.05$) groups. However, there is insufficient in moderate (0.24) and severe (0.23) OSA groups (Table 5).

Table 3. Sleep bruxism data with segmented sample (n=22).

	Non OSA (SD) N=15	OSA (SD) N=7	F
Sleep Bruxism PSG			
Total episodes	13.71 (13.76) *	5.93 (8.64) *	4.58
Phasic episodes	4.43 (7.39) *	0.87 (1.52) *	10.10
Tonic episodes	8.14 (8.57)	4.33 (6.20)	1.83
Mixed episodes	1.14 (1.86)	0.73 (16.62)	0.25
Ep. /hour	2.11 (2 07)	1.20 (2.07)	0.74
SB Automatic Bruxoff			
Total episodes	23.14 (11.69)	22.47 (18.07)	0.95
Phasic episodes	7.14 (6.25)	5.20 (5.04)	0.331
Tonic episodes	5.00 (4.65)	6.13 (7.97)	2.76
Mixed episodes	1.43 (1.81)	1.13 (1.95)	0.00
Ep. /hour	4.38 (2.38)	3.70 (2.90)	0.80
SB Manual Bruxoff			
Total episodes	16.14 (10.73)	13.93 (11.08)	0.20
Phasic episodes	6.43 (6.47) *	4.73 (3.39) *	5.07
Tonic episodes	8.,57 (8.26)	7 80 (7.89)	0.03
Mixed episodes	1.14 (1.86)	1.40 (2.13)	0.18
Ep. /hour	3.15 (2.35)	2.26 (1.74)	1.19

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Total SB events along the TST total sleep time, excluded the Sleep-related Oromotor Activity (OMA) with sample segmented (Non OSA, OSA). *p<0.05.

Table 4. n=22.

	Non OSA (SD) N=6	Mild OSA (SD) N=7	Moderate OSA (SD) N=3	Severe OSA (SD) N=6	F
Sleep Bruxism PSG					
Total episodes	16 (13.55)	5.57 (6.13)	10.33 (17.89)	3.17 (4.66)	1.83
Tonic episodes	9.50 (8.52)	5.85 (2.21)	6.67 (11.54)	2.17 (2.86)	1.18
Phasic episodes	5.17 (7.80)	0.57 (1.13)	1.67 (2.88)	0.67 (1.21)	1.52
Mixed episodes	1.33 (1.97)	0.43 (0.78)	2.00 (3.46)	0,33 (0.82)	0.97
Ep. /hour	2.46 (2.03)	0.93 (1.01)	2.67 (4.61)	0.80 (0.70)	1.40
SB Automatic Bruxoff					
Total episodes	24.50 (12.19)	26 (20.44)	18 (24.26)	19.33 (12.13)	0.26
Tonic episodes	4.83 (5.07)	6.71 (5.67)	8 (13)	4.50 (8.12)	0.21
Phasic episodes	7.67 (6.68)	7.29 (5.31)	2.33 (3.21)	4.00 (4.56)	1.06
Mixed episodes	1.67 (1.86)	0.86 (1.21)	1.33 (1.52)	1.27 (2.86)	0.72
Ep. /hour	4.68 (2.45)	4.34 (3.24)	2.6 (3.55)	3.33 (2.19)	0.50
SB Manual Bruxoff					
Total episodes	16.83 (11,58)	16.14 (10.30)	14 (18.19)	11.00 (8.22)	0.32
Tonic episodes	9 (8,96)	8.29 (6,39)	7.67 (11.59)	7.00 (8.44)	0.61
Phasic episodes	6.5 (7.09)	6.71 (3,86)	4 (2.64)	3.00 (1.55)	0.92
Mixed episodes	1.33 (1,96)	1.14 (1,86)	2.33 (4.04)	1.00 (1.26)	0.29
Ep. /hour	3.33 (2,52)	2.6 (1,41)	2.03 (2.65)	1.95 (1.71)	0.55

Total SB events along the TST total sleep time, excluded the Sleep-related Oromotor Activity (OMA) with sample segmented (Non OSA, mild OSA, moderate OSA and severe OSA). p>0.05.

Table 5. ICC data with segmented sample by severity degree of OSA. SB episodes (n=22).

Non OSA N=6	Mild OSA N=7	Moderate OSA N=3	Severe OSA N=6
0.61	**0.53 ***	**0.24**	**0.23**

Agreement, ICC: Total SB episodes per night along the three tools (PSG, manual EMG-EKG and automatic EMG-EKG) with segmented sample by the degree severity of OSA. *p<0.05.

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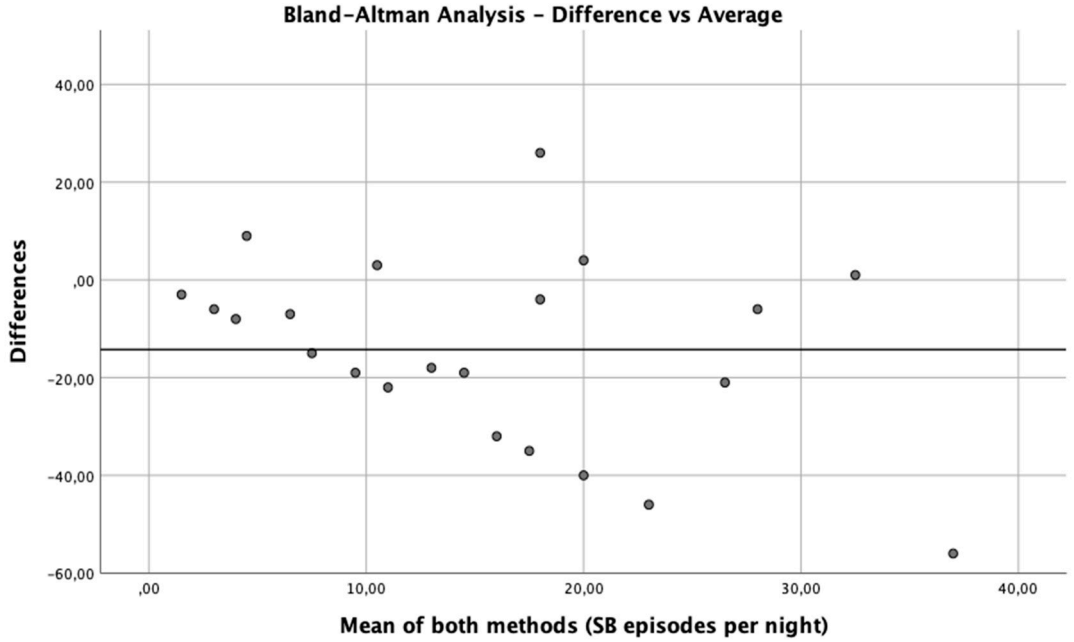
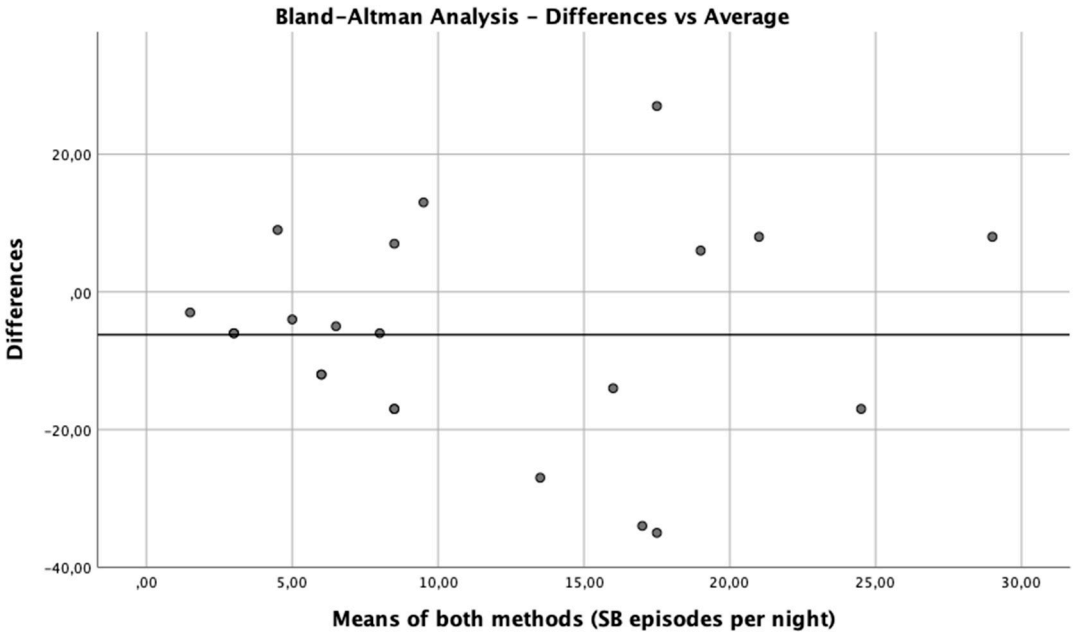


(A)



(B)

Figure 5. Bland- Altman analysis. **A)** Automatic analysis of PSG recordings versus automatic EMG-EKG device analysis. Limits agreement = + 34.77, - 3.87 (Bias = -14.27). **B)** Analysis of PSG recordings versus manual EMG-EKG device analysis. Limits agreement = + 26.72, - 3.68 (Bias = -6.22).

4. Discussion

Some authors describe the possibility that there is a subtype of patients with subclinical or mild OSA and that such EMG activity could play a protective role against OSA (18). We must bear in mind that OSA and SB share structures that play a fundamental role with protective functions during sleep. Also, there are inter-individual differences. Therefore, it is essential to clarify the PSG criteria for the evaluation of SB and its comorbidities, in order to design quality studies and avoid biases in that evaluation (24,31,40). Different authors suggest that SB studied with PSG in patients with OSA

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usually occurs close to Apnea-Hypopnea (AH) events (12,17,40). However, many of them have been conducted with incomplete diagnostic tools to analyze all neurophysiological components. In our sample, the correlation between the apnea and hypopnea episodes and the SB episodes is negatively in the total of the sample, this suggested that when the number of apnea episodes increase, the number of SB decrease with PSG recordings. Authors like Yap, indicate that AH and SB events are probably epiphenomena in adult patients with coexisting OSA and SB, where SB events were subsequent to AH events, featured predominantly alluding to a specific form of secondary SB triggered by sleep micro-arousals. Nevertheless, we consider that this kind of activity could act as a confusion factor and maybe should be consider as an AH final expected hyper motor activity and not as secondary SB, if there is not a minimum window of time after AH and the EMG hyper motor activity. Likewise, the refinement of ambulatory devices depends on the correlation and concordance obtained with the gold standard (PSG). Improving the ability to avoid bias with automatic analysis of portable EMG devices is essential to avoid overestimation of the disease. In our sample the total events of SB per night in the PSG study were lower than the one obtained with EMG-EKG device manual analysis and automatic. The SB episodes number mean decreases from non OSA group to OSA group with both PSG and EMG-EKG analyses. However, this decreasing was in minor proportion with the automatic EMG-EKG analysis mode. Our results are in line with the Martynowicz findings, where the relationship between OSA and SB depends on the degree of severity of OSA (41). It should be considered that studies on this relationship are scarce and present different methodological designs and goals. Okeson or Sjöholm have no found differences in terms of SB in OSA versus non OSA patients, but the sample was not segmented by the degree severity of OSA, or severe OSA patients were not included in the sample, respectively (22,25). On the other hand, Okura indicate that OSA patients with SB have a unique phenotype of OSA and also emphasize the distinct relationship of respiratory events with RMMA and non-specific masticatory activity (NSMA) (42). In our sample, the agreement between PSG and EMG-EKG device shows an acceptable value in non OSA and mild OSA groups. However, there is insufficient in moderate and severe OSA groups. The exclusion or inclusion of the EMG event following the respiratory event (which we have discarded with PSG analysis) could explain the results variability and perhaps lead an overestimation of SB in moderate and severe OSA patients with EMG portable devices when include that hyper motor activity. Saito conducted another study in which obtained a positive and significative correlation between OMA and AHI (14). As Kato pointed in 1999, perhaps the OMA activity constitutes a bias if it is not clearly excluded of the neurophysiological analyses (43).

In our sample the tonic episodes predominate against phasic episodes. The phasic episodes decrease considerably from non OSA to OSA patients with PSG analysis compared to EMG-EKG analysis. Other studies indicated that the phasic episodes could have a protective role against OSA (18,44). However, our results may suggest that tonic episodes may also play a protective role, but our study design doesn't analyze the risk or protection factor, it is just a correlation between different instrumental tools.

We obtained an acceptable ICC based on the number of SB episodes accounts in all the subject along the three tools (PSG, manual EMG-EKG and automatic EMG-EKG). Other studies obtained better diagnostic yield values, but these studies did not research the possible bias of the OSA activity for the SB estimations or found no association between AHI and RMMA index (19,33). Additionally, if the portable device is not able to identify the sleep stage, and an event fitting the criteria for RMMA occurs during wake time it would be scored as a SB event leading again to an overestimation (45). Overestimating SB would mean overestimating its association with other sleep disorders. In addition, it is essential to complement the instrumental diagnosis of SB with the clinical examination and the patient's self-referred tests, as this would allow to assess the sequelae of SB (1). The clinical consequence of SB is the true indicator of the need for treatment (32,46). In any way, definitive EMG ambulatory evaluation of SB should be increasingly implemented in the clinical setting and not only in research, as it is the only reliable and objective measure that bruxing activity is present and active. Similarly, EMG is a useful tool for proper follow-up as a measure of the efficacy of certain therapeutic approach strategies. The use of EMG on a daily and reliable basis would mean being able to

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implement this tool in the same way that, for example, a periodontal chart is performed for the staging of periodontal disease and its evolution. The combined use of respiratory polygraphy with EMG also allows a complete screening of both entities (SB-OSA), being also used for the follow-up of patients who are users of a mandibular advancement device. It would be interesting to use portable respiratory polygraphs that include EMG in masseters, like the one used by Winck (21). The EMG-only devices have not sufficient diagnostic yield for SB in populations which OSA has not been previously discarded. Including masseter and temporalis muscles EMG montage in sleep units as routine would be useful to improve the knowledge about SB-OSA relationship. The bruxing activity is considered as a continuum, so instruments that allow the recording of several nights in a less costly way, such as EMG, should be refined. Therefore, the determination of new correlations and updated cut-off points is important (32). It would be desirable that all the EMG portable devices designs and software to comply with the recommendations of the SENIAM project (Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles) criteria, which has resulted in European recommendations for sensors and sensor placement procedures and signal processing methods for Surface ElectroMyography (SEMG). The EMG-EKG portable device used for this study complies those recommendations. However not all the EMG portable devices share a similar protocol (47–49). Once the performance of portable EMG has been improved, it could be used for concordance studies against other types of novel tools that are emerging due to the obvious evolution of technology, big data, and artificial intelligence (50). Such studies would allow them to be performed longitudinally and more fluently than with PSG in a sleep lab.

In the case of studies on dental materials used in oral rehabilitations in bruxism patients, there are large biases due to not objectively measuring SB. With the improvement of EMG, increasing its use by clinicians and researchers in the different fields of dentistry, a large part of these biases could be avoided. The results show that manual analysis of SB events is more reliable than automatic analysis in our sample. Training in this type of analysis and calibration among professionals, as is done for example with DC/TMD exploration for temporomandibular disorders, would provide great advantages for professionals who handle this type of patient (37). It would be advisable to perform a basic OSA screening of all patients with suspected SB. In the case of EMG-EKG device, manual analysis of bruxing events in these patients shows greater reliability than automatic analysis. Likewise, there are already EMG devices that are used for the management of SB through biofeedback (51). Further research along these lines could provide new non-invasive, reversible, and inexpensive management methods for the patient. It would be useful to reproduce studies with a similar design and increasing the sample size to confirm these results. This would provide data to improve the automatic analysis algorithms of portable devices for the SB evaluation.

Limitations

The patient attending the sleep unit may suffer from "laboratory" effects on the first night, but it was not feasible for us to perform more than one night of PSG recording. The groups are not balanced due to the low sample size and there is a predominantly OSA population. The simultaneous placement of the surface electrodes of the portable device and polysomnography could generate interference and a poorer quality of signal reception. We tried to improve this limitation with smallest surface electrodes for the EMG-EKG device than those normally includes the package of the EMG and EKG electrodes.

5. Conclusions

- There is an acceptable concordance between the results obtained in the PSG manual analysis and those obtained by the EMG-EKG device with automatic and manual analysis for the diagnosis of SB, but only in patients without OSA or with mild OSA.
- In patients with moderate or severe OSA, apneas could act as a confusion factor in the diagnosis of SB with electromyographic portable devices.

- It would be recommended to clarify the analysis scores to differentiate the muscle activity consecutive to the apnea episode from the masticatory muscle activity that meets the criteria for SB, especially for the programming of portable devices algorithms.

Supplementary Materials: The following supporting information can be downloaded at: www.mdpi.com/xxx/s1, Figure S1: EMG-EKG Device

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