

Use of Electromyographic and Electrocardiographic Signals to Detect Sleep Bruxism Episodes in a Natural Environment

Original

Use of Electromyographic and Electrocardiographic Signals to Detect Sleep Bruxism Episodes in a Natural Environment / T., Castroflorio; Mesin, Luca; G. M., Tartaglia; C., Sforza; D., Farina. - In: IEEE JOURNAL OF BIOMEDICAL AND HEALTH INFORMATICS. - ISSN 2168-2194. - STAMPA. - 17:6(2013), pp. 994-1001. [10.1109/JBHI.2013.2274532]

Availability:

This version is available at: 11583/2520489 since: 2021-08-21T18:14:46Z

Publisher:

IEEE / Institute of Electrical and Electronics Engineers

Published

DOI:10.1109/JBHI.2013.2274532

Terms of use:

This article is made available under terms and conditions as specified in the corresponding bibliographic description in the repository

Publisher copyright

IEEE postprint/Author's Accepted Manuscript

©2013 IEEE. Personal use of this material is permitted. Permission from IEEE must be obtained for all other uses, in any current or future media, including reprinting/republishing this material for advertising or promotional purposes, creating new collecting works, for resale or lists, or reuse of any copyrighted component of this work in other works.

(Article begins on next page)

Use of Electromyographic and Electrocardiographic Signals to Detect Sleep Bruxism Episodes in a Natural Environment

T. Castroflorio¹, L. Mesin², G.M. Tartaglia¹, C. Sforza¹, D. Farina³

Abstract—Diagnosis of bruxism is difficult since not all contractions of masticatory muscles during sleeping are bruxism episodes. In this paper, we propose the use of both EMG and ECG signals for the detection of sleep bruxism. Data have been acquired from 21 healthy volunteers and 21 sleep bruxers. The masseter surface EMGs were detected with bipolar concentric electrodes and the ECG with monopolar electrodes located on the clavicular regions. Recordings were made at the subjects' homes during sleeping. Bruxism episodes were automatically detected as characterized by masseter EMG amplitude greater than 10% of the maximum and heart rate increasing by more than 25% with respect to baseline within 1 s after the increase in EMG amplitude above the 10% threshold. Further, the subjects were classified as bruxers and non-bruxers by a neural network. The number of bruxism episodes per night was 24.6 ± 8.4 for bruxers and 4.3 ± 4.5 for controls ($P < 0.0001$). The classification error between bruxers and non-bruxers was 1% which was substantially lower than when using EMG only for the classification. These results show that the proposed system, based on the joint analysis of EMG and ECG, can provide support for the clinical diagnosis of bruxism.

Index Terms—bruxism, surface EMG, masseter muscle, cardiac activation, concentric electrode

INTRODUCTION

Sleep bruxism (SB) is an oral parafunction characterized by grinding or clenching of the teeth during sleep that is associated with an excessive (intense) sleep arousal activity (1). Sleep bruxism should be distinguished from the awake bruxism that is mainly the result of emotional tension or

psychosocial disorders, and forces the subject to respond with a prolonged contraction of the masticatory muscles (2). Bruxism while awake is commonly characterized by clenching, while SB has a combination of clenching and grinding (3,4).

Large scale epidemiologic surveys on self reported SB showed a prevalence of 8% in the adult population, an equally distribution of the disorder in men and women, and a tendency to decline with increasing age, both in North America and Europe (5,6). The pathophysiology of SB is still unclear. Scientific data supporting an association between SB and stress, anxiety, and the hypothalamic-adrenal axis are not definitive (7,8).

Several types of rhythmic oromotor activities can be present during sleep, occurring in almost 60% of the adult population (9), while the percentage of people fulfilling the diagnostic criteria for a SB diagnosis is much smaller. Thus, rhythmic oromotor activities may be considered a normal sleep-related motor behavior (7,10). Morphologic, psychosocial or pathophysiological factors contribute to an increase in frequency, duration, and intensity of these physiological muscle activities to pathological levels. The diagnosis of SB based on these activities is currently based on the “bruxism generator model” (10).

A well defined oromotor activity has proven to constitute the basic pattern of SB. This unique and complex motor pattern is called “rhythmic masticatory muscle activity” (RMMA) (8,10,11, 12). During a RMMA, the electromyogram (EMG) of masticatory muscles presents bursts of periodic activity at a frequency of approximately 1 Hz and amplitude of approximately 10% of the maximum voluntary clenching activity while awake (data for the masseter muscle) (11). In contrast to mastication, sleep-related RMMA are characterized by co-contraction of opening and closing jaw muscles and these purposeless movements rarely last more than 8 s (12).

The occurrence of RMMA can be identified during awake and sleep stages by using multiple physiological measurements supplemented with audio and video recordings (8,11,12). A major disadvantage of this equipment is the high cost and amount of time needed for manual/visual scoring (13). Furthermore, these systems are mainly used in laboratory settings, thus providing information that may not be representative of oral behaviors as they occur in the natural environment (at home). Finally, the scoring of masticatory muscle activity under these conditions is mainly based upon subjective evaluation and skill of the examiner (13). Portable EMG devices (14,15) may solve some of these limitations. However, because bruxers exhibit many non-pathological orofacial activities during sleep (16), the analysis of the EMG

¹ Department of Human Morphology and Biomedical Sciences “Città Studi”, Università degli Studi di Milano, Milan, Italy

² Department of Electronics, Politecnico di Torino, Turin, Italy

³ Department of Neurorehabilitation Engineering, University Medical Center Göttingen, Georg-August University of Göttingen, Göttingen, Germany

Corresponding Author:

Tommaso Castroflorio

Department of Human Morphology, University of Milan, Milan, Italy

Via Mangiagalli 31, 20133 Milano, Italy

Tel +39 011 19507750; fax: +39 011 19508071

E-mail: tcastroflorio@libero.it

There is no conflict of interests in the preparation of this manuscript. The Bruxoff device was provided by Spes Medica (Genova, Italy). The authors do not have any financial relation with Spes Medica.

© 2013 IEEE. Personal use of this material is permitted. Permission from IEEE must be obtained for all other uses, in any current or future media, including reprinting/republishing this material for advertising or promotional purposes, creating new collective works, for resale or redistribution to servers or lists, or reuse of any copyrighted component of this work in other works

tends to substantially overestimate SB episodes. Furthermore some diagnostic devices based on dental contact detection have been introduced with promising results (17,18,19).

Interestingly, recent studies show that the SB event is preceded by a sudden shift in autonomic cardiac and respiratory activity, and by a specific brain activation (12) that suggests overactivation of the autonomic system as closely related to the pathogenesis of SB (8, 11, 12). The hypothesis of the study is that heart rate could be used in addition to EMG monitoring to improve the accuracy in automatic detection of bruxism events. In this study, we exploit this idea and propose a system based on the joint analysis of EMG and ECG for accurate detection of SB episodes and thus for the automatic diagnosis of bruxism. The possible improvement in the detection of SB episodes with respect to the classic approach based exclusively on EMG is investigated. The signals are recorded by a compact portable device (Bruxoff®, Spes Medica, Battipaglia, Italy), that was used in a natural environment by the subjects of the study without technical support during use (after appropriate training).

MATERIALS AND METHODS

Subjects

The study was performed on 46 subjects. The subjects were selected among patients referring to a private dental clinic, in an urban area of the northern Italy, for the treatment of discomfort, fatigue, or pain of the jaw muscles, and between patients referring for habitual dental control. Each subject was classified as bruxer or non-bruxer by an expert clinician, on the basis of the clinical diagnostic criteria for SB, as described by the American Academy of Sleep Medicine (1). According to these criteria, a subject is identified as a bruxer if: 1) he/she reports or is aware of tooth grinding sounds or tooth clenching during sleep; 2) one or more of the following signs is present: a) abnormal teeth wear; b) discomfort, fatigue, or pain of the jaw muscles and locking of the jaws on awakening; c) hypertrophy of the masseter muscles on voluntary forceful clenching; d) jaw muscle activity cannot be better explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

With these criteria, 25 bruxer subjects (mean age \pm SD: 31.05 ± 8.42 years; 8 men and 14 women) were selected as study group from 57 patients referring to the dental clinic in the period September 2010 - January 2011; and 21 healthy age- and gender-matched subjects (mean age \pm SD: 32.89 ± 9.73 years, 10 men and 12 women) were selected to constitute the control group. Both bruxers and non-bruxer subjects were also screened for temporomandibular disorders (TMD) according to the research diagnostic criteria for TMD (RDC/TMD) (20).

Exclusion criteria for both sleep bruxers and control individuals were: 1) presence of prosthodontic rehabilitations, 2) missing teeth, 3) periodontal disease, 4) according to Rompré et al. (8), Group II and/or Group III TMDs (discal and/or articular TMDs) (20) as a primary complaint to facilitate the clinical selection of candidate bruxer subjects according to the diagnostic criteria for SB, 5) a medical history of neurological disorders, mental disorders, or sleep

disorders (e.g., apnea, periodic leg movements, insomnia). All the subjects were unmedicated at the time of recording, and were not under the effect of alcohol, nicotine or caffeine. According to Rompré et al. (8) subjects exhibiting a high value of pain on the VAS scale (question 7 of the RDC/TMD) were classified as low level bruxers, subjects exhibiting a low value of pain were classified as high-level bruxers, while subjects exhibiting no pain were classified as healthy.

After the selection procedure, four bruxers were excluded from the analysis because they did not complete the experimental procedures. One of these four subjects did not tolerate the presence of the wires of the recording device during sleeping and the other three interrupted the recordings after less than 4 hours of sleep. Therefore, the results are presented for 21 patients and 21 controls. The procedures were approved by the local ethic committee. 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.

Recordings

The participants completed a questionnaire concerning awareness of sleep bruxism, anxiety, sleep habits, stress, nervousness, fatigue, current facial pain intensity, fatigue of jaw muscles, and painful jaw upon awakening. A pain evaluation numerical rate scale (question 7 of the RDC/TMD: "How would you rate your facial pain on a 0 to 10 scale at the present time, that is right now, where 0 is "no pain" and 10 is "pain as bad as could be"?") was scored by each subject at the moment of the delivery of the portable device to self report the pain intensity in the craniofacial region.

A portable device (Bruxoff®, Spes Medica, Battipaglia, Italy) with three channels was used for recording. Two channels were used to acquire surface EMG bilaterally from the masseter, and the third channel was used to acquire the electrocardiogram (ECG). The three signals were sampled at 800 Hz, with 8 bit resolution. The data were stored on a MicroSD card as a binary file. The surface EMG channels were filtered between 10 and 400 Hz with gain 4300. The ECG channel was filtered between 15 and 160 Hz with gain 700. Surface EMGs from the masseter muscle of both sides were detected with disposable bipolar concentric electrodes (Code®, Spes Medica, Battipaglia, Italy) (21), with a radius of 16 mm and with detection site made of AgCl (Fig.1).



Fig.1: The Bruxoff® and the CoDe® (Spes Medica, Battipaglia, Italy) electrode used in this study for the detection of myoelectric signals from the masseter muscles. This electrode was chosen to avoid any orientation problem. At the top a schematic representation of the electrode location over the masseter muscle is shown. Black line: gonial angle-cantus line used as anatomical landmark.

These electrodes were chosen to permit an easy application by the subject, avoiding the electrode orientation problem and reducing EMG crosstalk (21, 22).

The ECGs were detected with two disposable monopolar electrodes located bilaterally on the clavicular region. EMG and ECG signals were recorded during two consecutive nights (at least 4 hours of sleep per night). The subjects used the device and mounted the electrodes at their homes without technical assistance, after prior training. The first night was a familiarization session, while the recordings during the second night were used for the data analysis.

At the beginning of the recording, the subjects were asked to perform three maximum voluntary clenching (MVC) lasting 3 s each and separated by 10 s of rest. The greatest of the MVC measures was used for normalizing the EMG values as a percent of MVC.

Signal analysis

The amplitude of the surface EMG and the heart rate (HR) were extracted from the raw EMGs and ECGs. The raw EMGs were digitally low-pass filtered (Butterworth filter of order 5) between 10 and 300 Hz in order to remove high frequency noise and the amplitude was estimated as the average rectified value (ARV) from intervals of 1 s duration.

From the EMG ARV during the MVC and during bruxism episodes (see below for the description of the method of detection of SB), an asymmetry index was calculated as follows:

$$As = \frac{ARV_{DX} - ARV_{SX}}{ARV_{DX} + ARV_{SX}} \% \quad (20)$$

where ARV_{DX} / ARV_{SX} is the amplitude of the EMG of the right/left masseter muscles, respectively. The asymmetry index ranges from +100% to -100%; positive values indicate a stronger right side muscular activity, while negative values indicate a stronger left side muscular activity. The asymmetry index was calculated in order to demonstrate if EMG signals could be recorded on only one side or on both sides of the face.

The raw ECG was digitally low-pass filtered (Butterworth filter of order 5) with cut-off frequency 100 Hz. The QRS complex was enhanced by a matched filter, using as a template the spike triggered average of QRS waves identified by thresholding a portion of the signal with high signal-to-noise ratio. The HR was estimated every second from intervals of 10-s duration preceding the time instant of interest by detecting the QRS events. The HR values were then restricted to the range 6-240 pulses per minute (ppm) to exclude outliers due to false positives or false negatives. Finally, values of HR exceeding the mean value (within the 10 s) by more than 30% were excluded since they were also considered outliers. The HR at the specific time instant of interest was then estimated by linear regression of the remaining values, after exclusion of

the outliers. EMG ARV and HR were used for identifying bruxism episodes. Bruxism episodes were detected when the EMG ARV (average over the two masseters) was greater than 10% MVC and the HR increased by more than 25% within 1 s from the increase of EMG ARV above the 10% MVC (12). A cut-off of 8-s duration was used for bruxism episodes to avoid the recording of long EMG activities that could reflect awakening with or without sleep stage shifts (12). Phasic, tonic and mixed contractions from both the masseter muscles were identified. According to Lavigne et al. (11), phasic episodes were characterized by brief, repetitive contractions with three or more consecutive EMG bursts lasting 0.25-2 s each, while tonic episodes were characterized by sustained contractions lasting more than 2 s and less than 8 s (see above). Contractions lasting more than 8 s were classified as a short awakening (12), while mixed episodes were contractions lasting less than 8 s and characterized by both short (less than 2 s) and long contractions (more than 2 s, but less than 8 s). The first and the last hour of sleep were not considered for the analysis to avoid the inclusion of voluntary contractions.

Statistical Analysis

Data obtained from a total of 42 subjects (21 bruxers and 21 controls) were analyzed. The non-parametric Mann-Whitney test was used to compare the asymmetry index, the number of SB episodes per night, and the number of SB episodes per hour between controls and bruxers.

The Spearman correlation test was used to investigate the relation between self-reported pain and the number of SB episodes and between the total number of masseters' contractions and the number of SB episodes (Prism 5, Graphpad Inc.). The significance level was set at $P < 0.05$. Results are reported as mean and SD.

Diagnosis of Bruxism by a Neural Classifier

The features obtained by processing EMG and ECG signals were: number of contractions, number of bruxism episodes, number of tonic, phasic or mixed contractions (both for contractions associated or not associated to bruxism), contractions associated to short awakenings (following bruxism contractions or other contractions), mean ARV, mean ARV during bruxism episodes, average time in which the masseter muscles were contracted during sleep, and duration of sleep. All these variables were used to classify the subjects involved in the experiments in three classes (11): non-bruxer (associated to the class 0), low-frequency bruxer (number of SB episodes per hour comprised between 2 and 4) (class 1), and high-frequency bruxer (more than 4 SB episodes per hour) (class 2). The classification was also performed using only the variables which could be obtained by recording only the EMG, without ECG, for assessing the improvement in diagnostic yield due to the inclusion of ECG, which is the main novelty of the proposed approach. The variables only related to EMG were: number of contractions, contractions associated to short awakenings, mean ARV, average time in which the masseter muscles were contracted during sleep, and duration of sleep. To reduce redundancy in the feature space, the algorithm described in (24) was used to determine the

interdependencies between candidate variables computing the Partial Mutual Information (PMI). PMI represents the information between a variable and the classification output that is not already present in the previously selected features. The features with maximal PMI were iteratively selected from the set of candidates. Using the PMI selection method, the candidate features were ordered for decreasing values of mutual information with the classification output. This also allowed to identify the most discriminative features for diagnosis.

The classifier was based on a set of multilayer perceptrons (MLP). A single hidden layer was used, which is sufficient to approximate any nonlinear function (universal approximation property) (25). Different MLPs had a different number of inputs, a number of neurons in the hidden layer in the range 4 – 20 (with sigmoidal activation function) and a single output neuron (with linear activation function). Different MLP topologies were trained by modifying iteratively the weights and the bias in order to reduce the error in fitting the desired output, using the quasi-Newton algorithm with a number of iterations in the range 10-50. Once trained, each ANN was used to classify new data, which were not used for training. Output values lower than 0.5 were associated to the class 0 (non-bruxers), values larger than 1.5 to the class 2 (high-frequency bruxers), and values in between to the class 1 (low-frequency bruxers). The data were divided into training (70% of data), validation (15% of data), and test sets (15% of data), choosing randomly the subjects associated to each set, for 10 times. The ANN with best generalization performance (i.e., with minimum error in classifying the validation data) was chosen as optimal. A first indication of the performance of the optimal ANN was provided by the error in the test set. Moreover, the topology of the optimal ANN was further tested on 100 random choices of training (75% of data) and test sets (25% of data) and an indication of the average classification performance was provided.

RESULTS

The control group reported no complaints, signs or symptoms of bruxism, while 13 bruxers of 21 presented abnormal wear of the teeth, 20 reported discomfort, fatigue or pain of the jaw muscles, and six showed hypertrophy of the masseter muscles on voluntary forceful clenching. The VAS for the patient group was 5.3 ± 1.5 . EMG data showed that bruxers exhibited significantly more orofacial motor activity during sleep than control subjects. Fig. 2 shows an example of EMG and ECG signals obtained during a bruxism (left) and not-bruxism masseter contraction. The number of masseter contractions per night was 168.7 ± 85.7 for bruxers and 50.5 ± 52.5 for controls. Although the number of masseter contractions was correlated with the number of SB episodes ($R^2=0.64$, $P < 0.0001$) (Fig. 2), the number of contractions identified as bruxism episode was a small fraction of the total contractions.

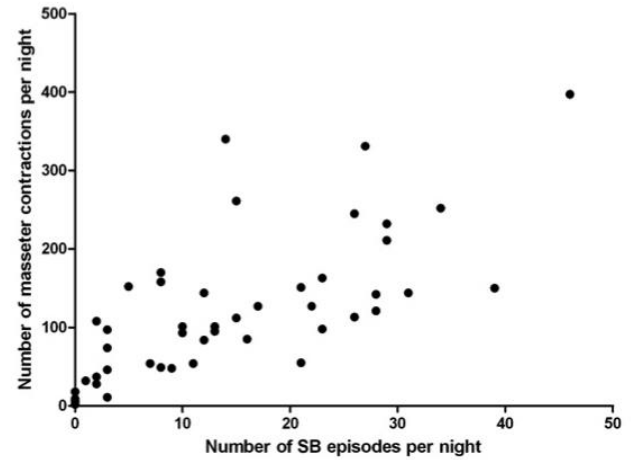


Fig. 2: Scatter plot between the total number of masseter contractions and the number of SB episodes per night in all the investigated subjects. The Spearman test revealed a significant correlation between these two variables Table I. Descriptive statistics of the sleep bruxism episodes in the analyzed groups. Data a ($R^2=0.64$, $P < 0.0001$).

For example, for the bruxers only approximately 15% of the contractions were bruxism episodes. The number of bruxism episodes per night and the number of bruxism episodes per hour were approximately three times greater in bruxers than in controls (Table I) ($P < 0.0001$). There was no association (Spearman test) between the number of SB episodes per night or per hour and pain grading on the VAS scale.

TABLE I

	Bruxers	Controls	P (Mann-Whitney)
Age (Mean \pm SD)	31.05 \pm 8.42	32.89 \pm 9.73	ns
Sex	14 F / 8 M	12 F / 10 M	
Number of masseter contractions	168.7 \pm 85.7	50.5 \pm 52.5	<0.0001
Number of SB episodes per night	24.63 \pm 8.42	4.31 \pm 4.50	<0.0001
Hours of sleep	7.74 \pm 1.53	7.11 \pm 1.51	ns
Number of SB per hour	3.21 \pm 1.09	0.60 \pm 0.64	<0.0001
SB Asymmetry Index	57.1 \pm 29.8%	24.5 \pm 33.1%	<0.01
MVC Asymmetry Index	32.6 \pm 27.4%	46.7 \pm 24.6%	ns

Descriptive statistics of the sleep bruxism episodes in the analyzed groups. Data are reported as Mean \pm SD

The masseter muscle activation during SB episodes was more asymmetrical for bruxers ($As=57.1 \pm 29.8\%$) than for controls ($As=24.5 \pm 33.1\%$) ($P < 0.01$). However, the masseter activity had a similar index of asymmetry between bruxers ($As=32.6 \pm 27.4\%$) and controls ($As=46.7 \pm 24.6\%$) during the MVC measures at the beginning of the recording session. The optimal ANN used for the diagnosis had 11 input features, used 14 hidden neurons for the classification, and was trained for 10 iterations (indeed, often a number of iterations between 10 and 20 was sufficient to get a perfect classification of the

training data; thus, a low number of iterations was selected to avoid overfitting). The selected input features ordered by PMI were the followings: number of bruxism episodes, ARV of EMG during bruxism episodes, number of mixed contractions, number of awakenings, number of tonic contractions, number of mixed contractions not associated to bruxism episodes, number of phasic contraction, duration of sleep, average activation time, number of phasic contractions not associated to bruxism episodes, and total number of contractions. This ranking indicates the characteristics most relevant for the diagnosis.

Considering the first 10 random choices of training, validation and test sets, the classification error in the validation set was $15 \pm 8\%$ and the error on the test set was $31 \pm 15\%$. To further test the optimal ANN, data were randomly divided into training (75% of data) and test set (25% of data), for 100 random choices, as explained in Methods. Table II summarizes the results by providing the confusion matrix of the classification.

TABLE II

		Estimation		
		Class 0	Class 1	Class 2
Diagnosis	Class 0	80%	19%	1%
	Class 1	27%	60%	13%
	Class 2	1%	50%	49%

Classification of the optimal ANN using EMG and ECG

A control subjects (diagnosis: class 0, top row of the table) was correctly identified as non-bruxer with a probability of 80%. A control subject would be erroneously classified as low-frequency bruxer with 19% probability and as a high-frequency bruxer with a probability of only 1%. A high-frequency bruxer was incorrectly classified as a non-bruxer with probability of only 1%, although he/she would not be differentiated as high- or low-frequency bruxer (approximately equal probability for these two classes). These classifications results used features that needed the HR for their calculation. The classification results were repeated when using only EMG, for comparison.

When only EMG derivations were used, the optimal ANN had 3 input variables (with the following order of PMI: number of contractions, average activation time, number of awakenings), 12 hidden neurons, and was trained for 10 iterations. Considering the first 10 random choices of training, validation and test sets, the classification error was $45 \pm 19\%$ in the validation set and $43 \pm 15\%$ in the test set, both greater

than when using the ECG. The classification with only EMG was also further tested on 100 random choices of training (75% of data) and test sets (25% of data). Table III provides the confusion matrix of the classification. This table can be compared to Table II when also ECG is used for classification (all other conditions are the same, see also Fig.3).

TABLE III

		Estimation		
		Class 0	Class 1	Class 2
Diagnosis	Class 0	68%	29%	3%
	Class 1	36%	51%	13%
	Class 2	19%	73%	8%

Classification of the optimal ANN using EMG only

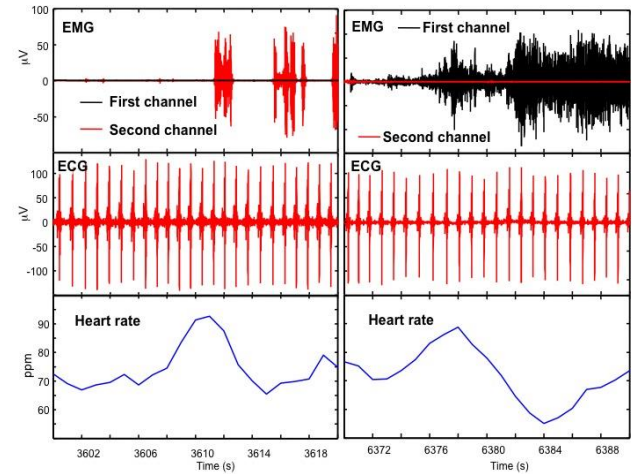


Fig. 3: Example of portions of signals (EMG, ECG) recorded during a contraction identified as a bruxism episode (left) and during another from the same subjects which was excluded, as the estimated heart rate (bottom trace) did not increase before the contraction.

The misclassification errors of the control subjects increased when only EMG was used. Moreover, the identification of a high-frequency bruxer was not possible neglecting the ECG.

DISCUSSION

The results of this study showed that it is possible to detect bruxism episodes from EMG and ECG recorded in a natural environment during sleep and that the joined analysis of EMG and ECG signals significantly improves the classification of bruxers according to the clinical diagnostic criteria.

A collection of signs and symptoms related to SB in conjunction with a complaint by a sleep partner (1) is still the most efficient and reasonable way to assess SB in clinical settings (26). The classic sleep laboratory diagnosis involve electroencephalography (EEG), EMG, ECG, oximetry,

oronasal thermistors, and nasal cannula pressure transducers with abdominal and chest belts to monitor respiratory changes (27). Furthermore, to distinguish SB episodes from other oromandibular activities, simultaneous audio-video recordings are usually performed (8,10). In addition to the cost and complexity of use of these systems, another major limitation is that the recording environment is not the habitual nocturnal one and some patients may not tolerate changes in their sleep environment, influencing the natural occurrence of SB (26). Thus, in the daily practice, sleep laboratory recordings are only recommended for complex SB patients, when unexplained findings are present (e.g., frequent breakage of teeth or dental restorations) or when tooth tapping suggests sleep-related epilepsy (26). To solve the problems of polysomnography in the diagnosis of bruxism, several portable EMG measurement devices have been previously produced to diagnose SB (28, 29). These tools were innovative because they permitted multiple night recordings in the natural environment with lower costs. However, EMG alone does not allow proper characterization of bruxism. Indeed bruxers exhibit non-pathological orofacial activities during sleep, which can constitute up to 30% of the entire set of oromotor events (16). Thus, the use of EMG to detect SB episodes is questionable. In our study a statistical correlation was found between the number of muscular contractions and the number of SB episodes per night (Fig. 2). However, the number of SB episodes represented only the 15% of the entire set of masseter contractions and the association between number of contractions and bruxism episodes was not perfect (Fig. 2). Accordingly, the classification accuracy based on only EMG in the current study was rather poor (more than 30% of bruxers were misclassified) (Table III). Finally, it is important to underline that up to 60% of the normal population, i.e., subjects not reporting sleep-related tooth grinding history, exhibits RMMA episodes, but at a low frequency (about an episode per hour of sleep) (10), as confirmed by our results in which controls exhibited 0.6 ± 0.6 SB episodes per hour of sleep (Tab. I), suggesting that portable EMG devices cannot differentiate between bruxers and non-bruxers. In general, even the most recent miniature self-contained EMG detectors (28, 29) do not discriminate SB contractions from other kinds of similar muscular activities. A multimodal approach is thus necessary for a more robust automatic diagnosis.

The present study is based on the finding that during light sleep, most SB episodes are observed in relation to brief cardiac and brain reactivation windows lasting 3-15 s, termed "micro-arousal" (MA) and recurring 8-15 times per hour of sleep in young healthy subjects (30). Lavigne (12) showed the following SB sequence: 1) a rise in sympathetic cardiac activity around 4 minutes before RMMA; 2) a rise in the frequency of EEG activity 4 s before RMMA; 3) a tachycardia starting one heart beat before RMMA; 4) an increase in jaw opener suprahyoid muscles 0.8 s before RMMA; 5) finally, RMMA EMG episodes scored as SB on masseter muscles (12). Using this information, the main contribution of the present study is the integration of information from both EMG and ECG to detect SB events. In this way, SB events can be discriminated from other muscle contractions in a relatively simple way without complicating the types of features

extracted from the EMG. This study demonstrated a substantial increase in diagnostic accuracy when the information from the signals was integrated. The study was performed by the use of a portable device directly applied by the patients, without technical support, in their homes. The compliance of the patients was very good with only one patient who did not tolerate the presence of the device and particularly of the wires. Other 3 patients were excluded since they interrupted the measure before 4 hours of sleep; however they also reported that their sleep was usually very poor and was not influenced by the device. Bruxers showed in this study 3 times the number of bruxism episodes per hour of sleep with respect to the control group, in accordance with the results obtained by Lavigne et al. (31) in a polysomnographic (PSG) study on 18 bruxers and 18 controls. Recently, Rompré et al. (8) identified three subgroups of bruxers who differed in sleep bruxism frequency: low, moderate, and high. Subjects in the low SB subgroup had values lower than the cut-off of 4 episode/hr proposed by Lavigne et al. (31). Bruxers recruited in our study presented characteristics that are comparable to those classified as low bruxers by Rompré et al. (8). The data from that study were collected in a sleep laboratory where severe cases are often observed: in our study, bruxers were recruited among patients of a dental clinic of a metropolitan area in the northwestern part of Italy, and in this clinical experience bruxers, in accordance with the clinical diagnostic criteria, are mainly low bruxers. This is probably due to another observation performed by Rompré et al. (8): they suggested that pain is frequently reported among sleep bruxers who display low frequencies of jaw muscle contractions. Pain is the main reason for a patient seeking care, and thus for a sleep bruxer pain, discomfort or fatigue of the jaw muscles could represent the main reason for seeking dental care.

The classification of subjects was performed by an ANN, which proved good accuracy in classifying bruxers from non-bruxers, although the differentiation between high- and low-frequency bruxers showed lower accuracy. Classification performances decreased if only EMG was used, indicating the importance of including ECG for a correct investigation of SB episodes.

In addition to automatic diagnosis, the recording of EMG during sleeping also allows the analysis of symmetry in muscle activity. In this study, controls and bruxers had a different asymmetry index, with a more asymmetrical activation of the masseter muscles of bruxers (Tab. I). The observed asymmetry was not related to differences in electrode location or differences in skin-electrode contact, since the EMGs at MVC were symmetric. The asymmetric activation is probably due to the fact that sleep bruxism has a combination of clenching and grinding (2): when clenching is prevalent, the masseter activation is probably more symmetric; when grinding is prevalent the masseter activation is probably more asymmetric with a more important muscular activation on the side of the mandibular movement. The information on muscle activation symmetry can be obtained only from bilateral recordings, which are therefore preferable over unilateral systems (28, 29).

CONCLUSION

We proposed a method for the automatic diagnosis of bruxism based on the analysis of bilateral EMG and ECG signals. The approach provides good classification results according to the SB/DC (1). The signals were recorded in a natural environment with a portable device. With respect to other portable EMG devices, the system used allows recording the masseter contractions of both sides together with the hearth rate. The joint information on EMG activity and HR provides classification results substantially better than when using EMG alone. It is concluded that the system proposed (portable device and signal processing) could be useful as a screening test for those subjects referring to the dental office with signs and symptoms of SB.

ACKNOWLEDGMENT

Authors are sincerely grateful to Enrico Castroflorio (Dept. of Biological Sciences, University of Eastern Piedmont, Alessandria, Italy), for his support in the preliminary visual analysis of the EMG and ECG signals.

REFERENCES

- [1] AASM. International Classification of Sleep Disorders. 2nd ed. Westchester: American Academy of Sleep Medicine; 2005.
- [2] D Manfredini, F Lobbezoo. Role of psychosocial factors in the etiology of bruxism. *J Orofac Pain* 23:153-166; 2009
- [3] F Lobbezoo, M Naeije. Bruxism is mainly regulated centrally, not peripherally. *J Oral Rehabil* 28:1085-1091; 2001
- [4] GJ Lavigne, T Kato, A Kolta, BJ Sessle. Neurobiological mechanisms involved in sleep bruxism. *Crit Rev Oral Biol Med* 14:30-46; 2003.
- [5] GJ Lavigne, JY Montplaisir. Restless legs syndrome and sleep bruxism: prevalence and associations among Canadians. *Sleep* 17:739-743; 1994
- [6] MM Ohayon, KK Li, C Guilleminault. Risk factors for sleep bruxism in the general population. *Chest* 119:53-61; 2001
- [7] F Lobbezoo, HL Hamburger, M Naeije. Etiology of bruxism. In: Paesani DA (ed.). *Bruxism. Theory and practice*. New Malden (UK): Quintessence Publishing Co, Inc, 2010: 53-65
- [8] PH Rompré, D Daigle-Landry, F Guitard, JY Montplaisir, GJ Lavigne. Identification of a sleep bruxism subgroup with a higher risk of pain. *J Dent Res* 86:837-842; 2007
- [9] F Lobbezoo, GJ Lavigne. Do bruxism and tempromandibular disorders have a cause-and-effect relationship? *J Orofac Pain* 11: 15-23; 1997
- [10] GJ Lavigne, S Khouri, S Abe, T Yamaguchi, K Raphael. Bruxism physiology and pathology: an overview for clinicians. *J Oral Rehab.* 35:476-494; 2008
- [11] GJ Lavigne, H Tuomilehto, G Macaluso. Pathophysiology of sleep bruxism. In: GJ Lavigne, PA Cistulli, MT Smith (eds.). *Sleep medicine for dentists. A practical overview*. Hanover Park (IL-USA): Quintessence Publishing Co, Inc, 2009:117-124.
- [12] GJ Lavigne, N Huynh, T Kato, K Okura, K Adachi, D Yao, B Sessle. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. *Arch Oral Biol.* 52:381-384; 2007
- [13] M Farella, S Palla, LM Gallo. Time-frequency analysis of rhythmic masticatory muscle activity. *Muscle Nerve*; 39:828-836; 2009
- [14] LM Gallo, SS Gross, S Palla. Nocturnal masseter EMG activity of healthy subjects in a natural environment. *J Dent Res*; 78:1436-1444; 1999
- [15] M Farella, A Michelotti, G Carbone, LM Gallo, S Palla, R Martina. Habitual daily masseter activity of subjects with different vertical craniofacial morphology. *Eur J Oral Sci*; 113:380-385; 2005
- [16] KM Dutra, FJ Jr Pereira, PH Rompré, N Huynh, N Fleming, GJ Lavigne. Orofacial activities in sleep bruxism patients and in normal subjects: a controlled polygraphic and audio-video study. *J Oral Rehabil*; 36:86-92; 2009
- [17] Clauss, J.; Wolf-Dieter, S.; Wolf, B. In-vivo Monitoring of Bruxism with an Intelligent Tooth Splint—Reliability and Validity. In *Proceedings of the World Congress on Medical Physics and Biomedical Engineering*, Munich, Germany, 7–12 September 2009; doi: 10.1007/978-3-642-03891-4_29.
- [18] Kim, J.H.; Mc Auliffe, P.; O'Connell, B.; Diamond, D.; Lau, K.T. Development of wireless bruxism monitoring device based on pressure-sensitive polymer composites. *Sens. Actuators A Phys.* 2010, 163, 486–492.
- [19] Diaz Lantada, A.; Gonzalez Bris, C.; Lafont Morgado, P.; Sanz Maudes, J. Novel system for bite-force sensing and monitoring based on near field communication. *Sensors*, 2012, 12(9), 11544-11556.
- [20] SF Dworkin, L.LeResche Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord.*;6:301-55; 1992
- [21] D Farina, C Cescon. Concentric-ring electrode systems for noninvasive detection of single motor unit activity. *IEEE Trans Biomed Eng*;48:1326-1334; 2001
- [22] T Castroflorio, D Farina, A Bottin, MG Piancino, P Bracco, R Merletti. Surface EMG of jaw elevator muscles: effect of electrode location and inter-electrode distance. *J Oral Rehabil*, 32:411-417; 2005
- [23] M Naeije, RS McCarroll, WA Weijs. Electromyographic activity of the human masticatory muscles during submaximal clenching in the inter-cuspal position. *J Oral Rehabil.*;16(1):63-70; 1989 Jan
- [24] A Sharma. Seasonal to interannual rainfall probabilistic forecasts for improved water supply management: 1 - A strategy for system predictor identification. *Journal of Hydrology.*;239:232-239; vol. 2000
- [25] S Haykin. *Neural Networks: A Comprehensive Foundation*. Prentice Hall; 1999
- [26] K Koyano, Y Tsukiyama. Clinical approach to diagnosis of sleep bruxism. In: Lavigne GJ, Cistulli PA, Smith MT (eds.). *Sleep medicine for dentists. A practical overview*. Hanover Park (IL-USA): Quintessence Publishing Co, Inc, 2009:109-116.
- [27] D Paesani. Diagnosis of bruxism. In: DA Paesani (ed.). *Bruxism. Theory and practice*. New Malden (UK): Quintessence Publishing Co, Inc, 2010: 21-40.
- [28] T Shochat, A Gavish, E Arons, N Hadas, A Molotsky, P Lavie, A Oksenberg. Validation of the BiteStrip screener for sleep bruxism. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*; 104:e32-e39; 2007
- [29] F Jadidi, E Castrillon, P Svensson. Effect of conditioning electrical stimuli on temporalis electromyographic activity during sleep. *J Oral Rehabil*;34:152-159; 2007
- [30] L Parrino, A Smerieri, M Rossi, MG Terzano. Relationship of slow and rapid EEG components of CAP to ASDA arousals in normal sleep. *Sleep.*;24:881-885; 2001
- [31] GJ Lavigne, PH Rompré, JY Montplaisir. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. *J Dent Res.*;75:546-552; 1996