Sleep Breath (2012) 16:535–542
DOI 10.1007/s11325-011-0539-4

ORIGINAL ARTICLE

The sleep/wake state scoring from mandible movement signal

Frederic Senny · Gisele Maury · Laurent Cambron · Amandine Leroux · Jacques Destiné · Robert Poirrier

Received: 30 March 2011 / Revised: 26 May 2011 / Accepted: 31 May 2011 / Published online: 11 June 2011

Abstract
Purpose Estimating the total sleep time in home recording devices is necessary to avoid underestimation of the indices reflecting sleep apnea and hypopnea syndrome severity, e. g., the apnea–hypopnea index (AHI). A new method to distinguish sleep from wake using jaw movement signal processing is assessed.
Methods In this prospective study, jaw movement signal was recorded using the Somnolter (SMN) portable monitoring device synchronously with polysomnography (PSG) in consecutive patients complaining about a lack of recovery sleep. The automated sleep/wake scoring method is based on frequency and complexity analysis of the jaw movement signal. This computed scoring was compared with the PSG hypnogram, the two total sleep times (TST_{PSG} and TST_{SMN}) as well.
Results The mean and standard deviation (in minutes) of TST_{PSG} on the whole dataset (n=124) were 407±95.6, while these statistics were 394.2±99.3 for TST_{SMN}. The Bland and Altman analysis of the difference between the two TST was 12.8±57.3 min. The sensitivity and specificity (in percent) were 85.3 and 65.5 globally. The efficiency decreased slightly when AHI lies between 15 and 30, but remained similar for lower or greater AHI. In the 24 patients with insomnia/depression diagnosis, a mean difference in TST of −3.3 min, a standard deviation of 58.2 min, a sensitivity of 86.3%, and a specificity of 66.2% were found.
Conclusions Mandible movement recording and its dedicated signal processing for sleep/wake recognition improve sleep disorder index accuracy by assessing the total sleep time. Such a feature is welcome in home screening methods.

Keywords Mandible movements · Sleep/wake state recognition · Portable monitoring · Sleep apnea

F. Senny (✉) · A. Leroux · J. Destiné
Electronic Department, Montefiore Institute,
University of Liège (ULg),
Building B28, Grande Traverse, Sart-Tilman,
B4000 Liège, Belgium
e-mail: F.Senny@ulg.ac.be

G. Maury
Pneumology, CHU Mont-Godinne,
Yvoir, Belgium

L. Cambron · R. Poirrier
Neurology Department, Faculty of Medicine,
University of Liège (ULg),
B35, Sart-Tilman,
Liège, Belgium

Introduction

To diagnose an obstructive sleep apnea/hypopnea (OSA) syndrome requires a full-night polysomnography (PSG) in a sleep laboratory. Epidemiologic study reported that more than 24% of adult males and 9% of adult females suffer from the OSA [1]. Drawbacks of PSG are, for instance, a long waiting list for patients and a high time cost for the technical staff. The latter has to carefully place the sensors to record many physiological signals like brain (EEG), chin muscle (EMG), eye (EOG), and cardiac (EKG) activities, thoracic/abdominal movements, and nasal airflow and afterwards to analyze them visually according to standard rules in order to compute relevant indices reflecting sleep disorder severity. One of the indices of the OSA is the apnea–hypopnea index (AHI), the ratio between the number of apneas and hypopneas, scored in respiration signals, and the total sleep time (TST), computed from EEG, EMG, and EOG traces [2, 3].

Portable monitoring (PM) devices have been developed to make easier and reduce the cost of OSA diagnosis and have been classified into three types (numbered 2 to 4, the attended PSG being type 1): type 2 PM is the home PSG, type 3 PM records four channels, and type 4 PM records one channel, but neither an expert is available at home nor EEG traces are recorded for the two latter types [4, 5]. In types 3 and 4, one to four cardiorespiratory biosignals are recorded for the detection of sleep apneas and hypopneas. However, the index is computed over the total recording time and this leads obviously to underestimation of the index compared with the gold standard PSG [4, 5]. To overcome the index underestimation problem and to allow an accurate diagnosis among the wider family of sleep disorders, sleep periods should be recognized from available data recorded by a type 3 or 4 PM by a dedicated method.

Despite the lack of sleep staging and microstructure analysis, actigraphy has been used in patients with a variety of sleep disorders to distinguish sleep from wake periods [6–16]. In this paper, the signal of interest is mandible movement, recorded by a type 3 PM, of which three assets have been highlighted in [17–24]. First, patterns in this signal are related to sleep apneas/hypopneas. These patterns are characterized by an increasing mandible lowering going with increasing oscillating movements during the sleep event, which is terminated by one or several discontinuous and ample movements. Thus, an automated method processing mandible movements on their own has been developed and shown to be effective in detecting and classifying sleep apneas/hypopneas [21]. Second, arousals that often follow sleep events appear in the mandible movement signal as the discontinuous and ample movements mentioned above [21, 22]. Third, differences in mandible movement behavior have been observed between wake, normal sleep, and disturbed sleep (by apneas, hypopneas, snoring, and respiratory effort-related arousal events): mandible movements are quicker, less structured, and aperiodic (like speech compared with snoring); moreover, the mouth is less opened in the wake state than during respiratory events [23, 24]. Nevertheless, it does not mean that the mandible always moves or that periodic patterns do not occur in the wake state: periodic patterns are assumed to be characterized by movements faster in wake (e.g., mastication) than in sleep (e.g., snoring), while quiet wake characterized by very few mandible movements is clearly difficult to distinguish from quiet sleep. An automated analysis of the mandible movement signal to distinguish sleep from wake gave a sensitivity and a specificity of, respectively, 85.1% and 76.4%, which were similar to other published data about the actigraphy method [24].

The purpose of this paper is the evaluation of the sleep/wake automated scoring computed from the mandible movement trace recorded by the Somnolter device (NOMICS, Belgium), a type 3 PM which records nasal airflow, blood oxygen saturation, and body position signals in addition to the mandible movement signal. A study is currently carried out to compare the automatic AHI provided from the Somnolter signal analysis to the PSG.

Materials and methodology

Subjects and recordings

Between 2009 and 2010, all the patients who underwent a polysomnography in the Sleep Laboratory of the University Hospital of Liège, Belgium and who slept in the same conditions (room, devices, and technical staff presence) were considered in the study. All these patients were suspected of any kind of sleep disorder; only those coming for positive pressure (CPAP) titration were excluded. This study followed the principles of the Declaration of Helsinki; the patients were informed of the aim of the study and gave oral agreement.

The PSG (S7000 or N7000 polysomnographs, EMBLA Medcare, Denver, USA) included neurophysiology signals: a three-channel electroencephalography (EEG, C3-A2, C4-A1, FZ-CZ), left and right electrooculography (EOG), submental electromyography (chin EMG) for sleep staging and arousal scoring, and two (left and right) tibial EMG for periodic leg movement evaluation. Cardiorespiratory signals comprised ECG, nasal cannula/pressure transducer (NAF; Protech, Mukilteo, USA), chest and abdominal inductance plethysmography belts, a plethypulse, a blood oxygen oximeter (SpO_2, Nonin, Plymouth, USA), a snoring sound detection (piezoelectric sensor from EMBLA), and a body position marker (body position sensor Protech).

Jaw movements and the portable monitoring device

The Somnolter device (NOMICS, Liège, Belgium) was placed the same night as the PSG. The Somnolter is a type 3 PM that records at least nasal airflow (Protech nasal canula), SpO_2 (Nonin), built-in body position, and mandible movement signals. It could also record thoracic movements through the respiratory impedance plethysmography belt. The recording of this mandible movement signal was performed by a distance meter based on the principle of magnetometry. The sensors were composed of two coils and capacitors, each embedded in a small cylinder (7 mm diameter; 25 mm main axis). They were disposed, parallel to each other, perpendicular to the midline of the face, and fixed with plasters, one in the dimple above the chin and the other on the forehead (Fig. 1). They were connected to an electronic circuit by two cables. The electronic circuit

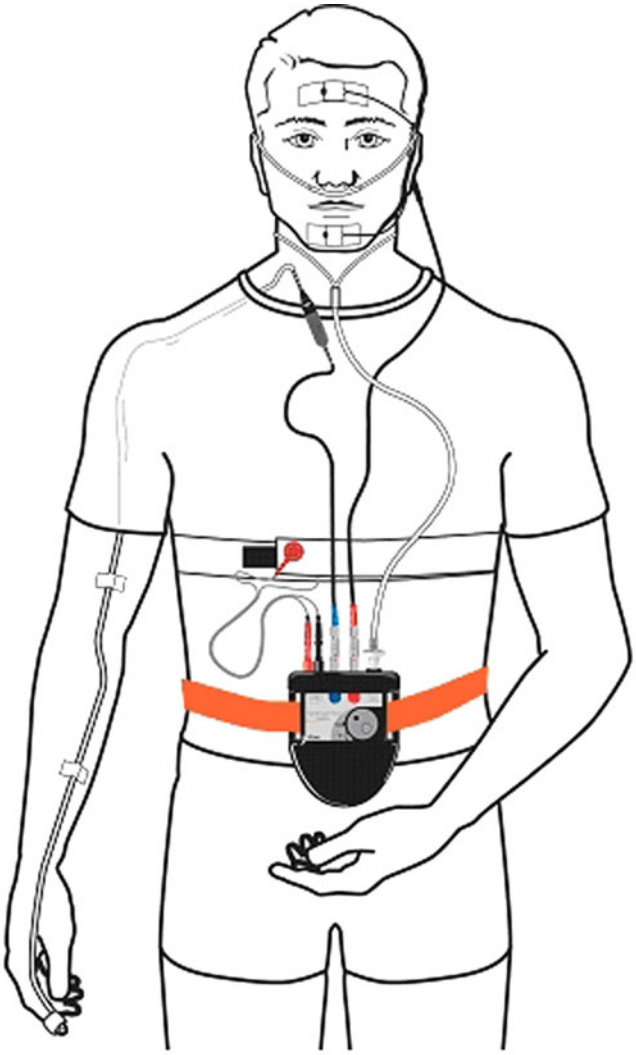

Fig. 1 Placement of the sensors measuring the midsagittal jaw movement and the ambulatory Somnolter device, which records the midsagittal jaw movements, the nasal airflow, the arterial oxygen saturation, and the body position. The device is also able to record the thoracic movements

converted distance into voltage. The signal was digitalized with a sampling frequency of 10 Hz. Physical calibration was done by asking the patient to first close his/her mouth and then to open it fully.

Manual scoring rules

Each PSG recording was manually scored by one of the authors (LC and RP). The sleep stages were defined every successive 30-s epochs, according to the Rechtschaffen and Kale scoring rules [3]. The manual hypnogram is called PSG hypnogram. The arousals were reported, as recommended by the American Sleep Disorders Association [25]: an acceleration of the EEG frequencies in NREM sleep, associated with an increase of chin EMG amplitude in REM sleep, occurring at least during 3 s. Breathing events were scored according to the American Academy of Sleep Medicine (AASM) Task Force proposals [26]. An apnea was defined as a cessation of airflow amplitude, below 20% of the reference value during 10 s or more. A hypopnea was scored each time a reduction of airflow (10 s or more) occurred below 70% of the reference value, provided that it was associated with either an oxygen desaturation of more than 3% or an arousal.

Recordings were classified, according to the AHI, as mild if AHI<15, moderate if 15<AHI<30, and severe in case of AHI≥30. OSA syndrome is diagnosed if an AHI≥5 is associated to excessive daytime sleepiness or two or more symptoms (choking or gasping during sleep, frequent awakenings, not well refreshed in the morning, fatigue, disturbed concentration) [2].

Jaw movement processing

The jaw movement signal processing especially focuses on separating high jaw activity wake from healthy sleep (no jaw movement most likely) and respiratory events, characterized by oscillating jaw movements and a more opened mouth. It uses a wavelet-based complexity measure to compute local and contextual features computed on a 1,024 sample sliding window without overlap (i.e., 102.4 s) and multilayer perceptrons to take the final decision [24]. The Somnolter analysis software has the same analysis basis, but some post-processing were added: (1) small sleep regions were deleted (less than three windows, i.e., 204.8 s), (2) if periodic jaw movements (period within 10 and 60 s) were found from the fast Fourier transform (a 3,096-sample Kaiser sliding window was used such that 1,024 samples were overlapped by the next sliding window), the central part of the Kaiser window was scored as sleep, and (3) the sleep/wake scoring was finally scaled to fit the standard 30-s epoch. This latter sleep/wake scoring is called the SMN hypnogram. Figure 2 sketches the jaw movement behavior at the sleep onset where high jaw activity (wake is the gray part in the chin EMG trace) vanishes and lets place to low jaw activity (sleep is the white part in the chin EMG trace).

Statistical data analysis

Data from PSG and Somnolter were converted into European Data Format files [27] for synchronization and statistical analysis under the MATLAB environment [28]. Since both PSG hypnogram and SMN hypnogram had the same time base but did not exactly began at the same moment, a time START (lit off) and a time STOP (lit on) were defined for all patients and the results were computed only within this range. Intraclass correlation (ICC) was computed under the R software [29] to measure the strength

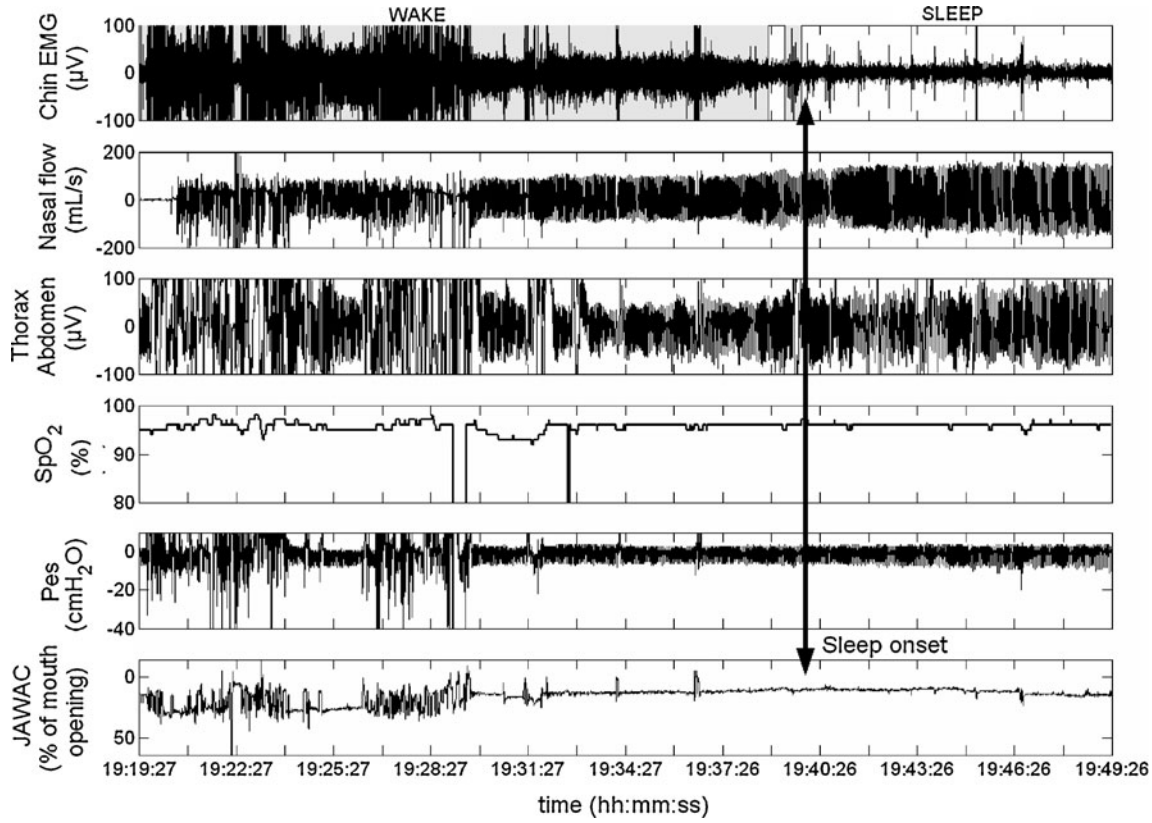

Fig. 2 At sleep onset, the jaw activity (JAWAC signal) lowered, thus separating two main behaviors of the JAWAC signal: high and chaotic jaw movement during (active) wake, highlighted as the *gray parts* in the chin EMG signal, compared with low and stable jaw movement during (quiet) sleep, the *white part* in the chin EMG

of the relation between two measurements (TST_{PSG} and TST_{SMN}). This relation was completed by the Bland and Altman test. Sensitivity (Se), specificity (Sp), and positive and negative likelihood ratios (LR+ and LR−) of the epoch-by-epoch comparison were also computed. All these tests were applied to several sets of recordings based on two criteria: final diagnosis made afterwards (i.e., sleep disorders breathing, insomnia, etc.) and the AHI only.

The sleep latency from both methods was also compared. Two definitions of sleep latency were used: sleep latency was the time in minutes between the START time and the first sleep period (including sleep stage 1) lasting one sleep 30-s epoch or 15 consecutive minutes of sleep (noted as SL and SL_15, respectively). The first definition comes from the standard AASM rules. The second takes into account the wider analysis window of the automatic method and a more relevant duration of the first sleep period leading to a value arbitrarily set at 15 min. The sleep latencies coming from PSG data are noted subsequently as SL_{PSG} and SL_15_{PSG}, while the sleep latencies from Somnolter data are noted as SL_{SMN} and SL_15_{SMN}.

Results

The database was made of 133 recordings. Nine of them were excluded due to a partial loss of data on the computer (n=1), poor quality of PSG data (n=3), a fault in jaw movement recording (n=4), and a battery problem (n=1). It means that 4% (5 out of 133) of the recordings were excluded due to a technical problem about the PM, which is acceptable. From the remaining 124 recordings, there were 32 women and 92 men, with a mean age of 50.8±12.4 years and BMI of 29.5±5.4 kg/m^2. As mentioned in Table 1, an

Table 1 Anthropometric information and main diagnosis about the patients

No. of recordings	Anthropometric data				Diagnosis		
	No. of males	No. of females	Age	BMI	OSAHS	Insomnia/depression	Other pathologies
124	92	32	50.8±12.4	29.5±5.4	68	27	29

Table 2 Performance of the automated sleep/wake scoring according to the diagnosis and the AHI assessed by the Bland and Altman analysis, the ICC (TST_{PSG}, TST_{SMN}), and the sensitivity/specificity (epoch-by-epoch comparison) methods

Diagnosis	Number	Bland and Altman TST_{PSG}–TST_{SMN} (minutes)		ICC	Sensitivity/specificity (%)	
		Bias [95% CI]	Standard deviation		Se	Sp
Overall	124	12.8* [−11.6, 37.2]	57.3	0.82	85.3	65.5
OSAHS	68	12.6 [−17.9, 43.0]	56.7	0.81	85.8	64.3
Insomnia	27	−3.3 [−59.4, 53.0]	58.2	0.85	86.3	66.2
Other pathologies	29	25.3* [−30.4, 81.0]	59.7	0.80	83.8	70.1
AHI<15	49	6.8 [−33.8, 47.4]	48.3	0.89	86.9	67.9
15<AHI<30	32	13.4 [−35.0, 61.8]	64.0	0.77	83.1	63.3
AHI≥30	43	12.4 [−24.3, 49.1]	56.0	0.78	85.8	63.5

*$p<0.05$, statistically significant difference

OSA syndrome was diagnosed in 68 of them (54.8%), insomnia/depression/anxiety was diagnosed in 27 of them (21.7%), while 29 others (23.5%) had other sleep disorders (e.g., periodic limb movement, circadian rhythm disorder, etc.).

The TST_{PSG} in the whole dataset was 407±95.6, while the TST_{SMN} from the algorithm was 394.2±99.3. Table 2 sums up the bias and standard deviation in minutes from the Bland and Altman analysis of the TST (PSG versus SMN): on the whole dataset, the difference in TST was 12.8 with a standard deviation of 57.3 min, as sketched in Fig. 2 (44.0± 38.7 min when the absolute value of the difference was considered), and did not differ in OSA syndrome patients (12.6±56.7). As far as insomnia and "other pathologies" are concerned, the mean differences were, respectively, −3.3 and 25.3, while the standard deviations were 56.0 and 59.7 min. When the AHI-only criterion is considered, for mild AHI (AHI<15), the bias was 6.8 and increased with severity (13.4 and 12.4 in case of moderate and severe AHI, i.e., AHI≥15). Table 2 also provides the ICC between TST_{PSG} and TST_{SMN}: the worst correlation was found for moderate AHI (15<AHI<30; ICC=0.77), while the best correlation was achieved for mild AHI with ICC=0.89. The method correlated better when AHI was >30 (ICC=0.78) than in patients having a moderate AHI. By comparing epoch by epoch from the two hypnograms (PSG and SMN), the sensitivity and specificity were computed over all the recordings and according to the diagnosis (see Table 2). The overall sensitivity was 85.3%, while the corresponding specificity was 65.5%. Table 3 details the 95% confidence intervals (95% CI) for the sensitivity, specificity, and positive and negative likelihood ratios (Figs. 3 and 4).

The sleep latencies computed from the two methods (SL_{SMN} and SL_15_{SMN}, defined in the "Statistical data analysis" section) were compared with the sleep latency from PSG (SL_{PSG} and SL_15_{PSG}). The sleep latency from the two methods differed from PSG latency. Nevertheless, the means of SL_15_{SMN} and SL_15_{PSG} were not significantly different (mean difference of 4.2 min, average of the absolute difference was 37.8 min) despite a very large discrepancy in some cases (standard deviation of the difference and of the absolute difference were 59.6 and 46.2 min, respectively; see Fig. 5). Table 4 provides the mean and standard deviation of the difference between the sleep latency from PSG and SMN for the two definitions considered (one 30-s epoch and 15 min of sleep).

Finally, from the whole dataset ($n=124$), we considered for the AHI computation the sleep periods and TST computed from the two methods: automatically from the mandible movement signal and the manually scored sleep periods from PSG. The respiratory events were still those manually scored, but only those occurring in sleep periods were involved in

Table 3 Detailed sensitivity/specificity analysis on an epoch-by-epoch basis

Diagnosis	Se	Sp	LR+	LR−
Overall	85.3 [82.7–87.5]	65.5 [59.6–70.9]	2.47 [2.09–2.92]	0.22 [0.18–0.27]
OSAHS	85.8 [85.4–86.1]	63.5 [62.5–64.4]	2.34 [2.29–2.40]	0.22 [0.21–0.23]
Insomnia	86.2 [83.6–88.6]	66.1 [60.8–71.1]	2.55 [2.18–2.98]	0.20 [0.17–0.25]
Other pathologies	83.8 [81.2–86.2]	70.1 [64.2–75.5]	2.31 [1.94–2.74]	0.23 [0.19–0.27]
AHI<15	86.8 [84.3–89.0]	67.9 [62.1–73.2]	2.70 [2.27–3.22]	0.19 [0.16–0.23]
15≤AHI<30	83.1 [80.3–85.6]	63.3 [57.5–68.7]	2.26 [1.94–2.65]	0.26 [0.22–0.32]
AHI≥30	85.8 [83.2–88.0]	63.5 [57.3–69.2]	2.34 [1.99–2.76]	0.22 [0.18–0.27]

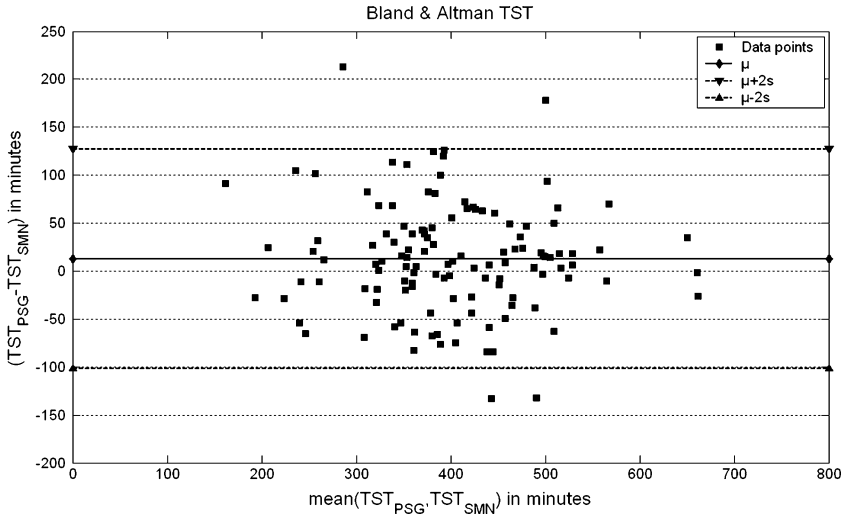

Fig. 3 Bland and Altman (*dashed lines* are limits of agreement) comparing the manual TST_{PSG} and automatic TST_{SMN} total sleep time (n=124)

the corresponding AHI computation. The AHI_SMN (= number of PSG events in SMN sleep periods/TST_{SMN}) was slightly lower than the AHI (= number of PSG events in PSG sleep periods/TST_{PSG}) but remained close to the latter: ICC was equal to 0.95, linear regression equation of $0.9\times x-1.6$, and the mean±standard deviation of the difference was 4.9±5.8 (95% CI of the bias was [−1.2, 11.0]).

Discussion

This study focused on an innovative PM device, called Somnolter, which is a type 3 PM using mandible movement signal in addition to nasal airflow, SpO_2, and body position. The main problem with the PM is the underestimation of respiratory events due to no or bad assessment of sleep periods. A correct TST allows the calculation of an index per hour, defined for respiratory or neurological events. This index is essential to the severity classification of the OSA syndrome.

As suggested in [24], the inferior jaw activity signal is another easy-to-use actimeter. Thus, the basic principle is like the wrist actigraphy (WAc) signal behavior analysis: high and unstructured activity is likely related to wake, while the quiet and periodic features may occur in sleep. The complex relations existing between the two states "wake" and "sleep" and the time behavior of mandible movement signal were investigated, leading to an automated analysis of the mandible movement signal [24].

In our prospective study, the results came from the data from 124 patients, among them 32 were females (26%), 68 were diagnosed with OSA (55%), and 27 were diagnosed with insomnia/depression (26%). An important caveat in this study is the absence of a control group with subjects without sleep disturbances. The TST_{PSG} in the whole dataset was 407±95.6, while the TST_{SMN} from the algorithm was 394.2±99.3 with an ICC of 0.82. The Bland and Altman analysis of the difference between the two TST in all patients was 12.8±57.33 min, with a sensitivity and a specificity of, respectively, 85.3% and 65.5%. Globally, there is an underestimation of

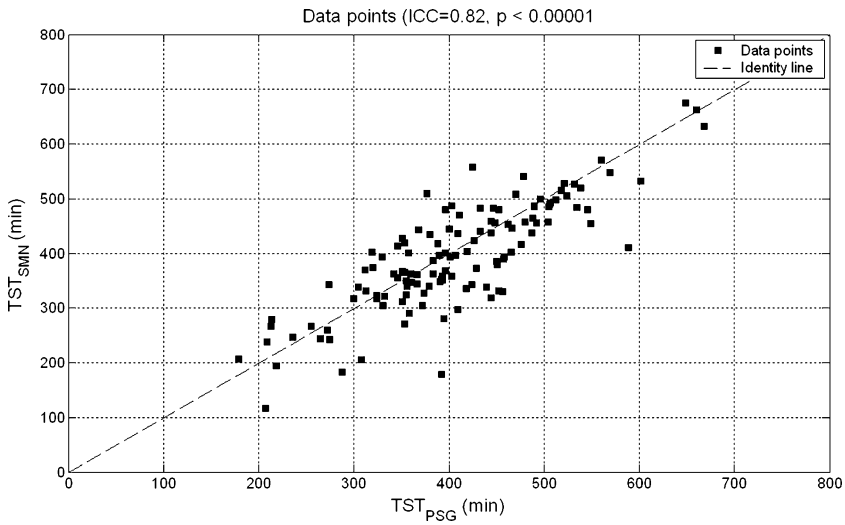

Fig. 4 Scatter plot of the data points (n=124), the automatic total sleep time (TST_{SMN}) versus the manual total sleep time (TST_{PSG}). The identity line is drawn

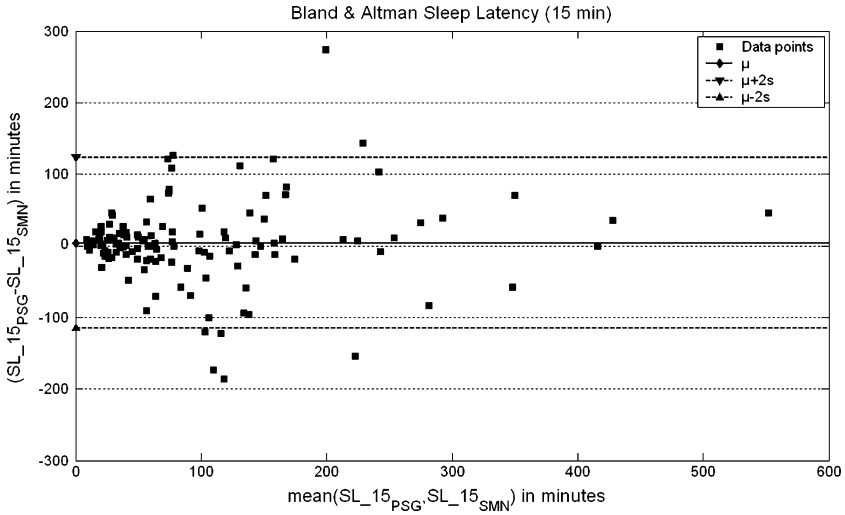

Fig. 5 The Bland and Altman plot of sleep latency (defined as the time between the START analysis and the first 15 consecutive minutes of sleep). The average and limits of agreement are also shown

sleep time except for insomnia/depression (n=27; 21.7%) where the mean TST difference is negative (−3.3±58.2 min), corresponding to an overestimation of the TST with the Somnolter. In other cases, TST was underestimated. The main point of interest is the sleep breathing disorder well known as OSA whose severity could come down to the AHI. OSA is the main respiratory sleep disorder searched in case of daytime sleepiness and is associated to health and socioeconomic repercussion. Considering the AHI criterion exclusively, the search for diagnosis devices easy to use and with excellent accuracy characteristics is growing. With the Somnolter PM device and compared to PSG, measures of agreement for TST in patients having a mild (AHI<15), moderate (15<AHI<30), and severe (AHI<30) AHI were, respectively, 6.8±48.3, 13.4±64, and 12.4±56 min. Sensitivities were, respectively, 86.8%, 83.1%, and 85.8% for mild, moderate, and severe AHI, while specificities were, respectively, 67.9%, 63.3%, and 63.5%.

The method used in this paper have comparable results with the ones from the WAc approach, a convenient and accepted way to assess the TST in PM [6, 7]. The WAc method is considered as useful, cost-effective, and noninvasive, but has some drawbacks, like its ineffectiveness in the diagnosis of some sleep disorders such as sleep breathing disorders or periodic limb movements [8–10]. Good performance has been reported in studies about the assessment of the TST in a normal population [11], in an OSA population [12], in an old population [13], and in an insomniac population [14, 15].

The mandible movement signal and its analysis suffer from the same drawbacks as WAc, i.e., (1) a lack of sensitivity in wake recognition because quite activity does not necessarily imply sleep-related brain activity and (2) a decrease in performance when sleep is disturbed [8–10, 16]. For example, the results for patients having an AHI between 15 and 30 were inferior to those from patients with lower or greater AHI (see above), but fortunately, the decrease in sensitivity of sleep recognition was only a few percent and it remained >80% (lower boundary of the 95% CI). One reason of such loss in efficiency could be the smaller number of moderate AHI patients (n=32) compared with the mild and severe populations (n=49 and n=43, respectively). Another reason could be that such patients have a more complex mandible movement signal behavior, the periodic feature could be unclear, at least less straight forward than in mild or severe cases. Furthermore, short and repeated respiratory events are mostly accompanied by respiratory (micro)arousals, which are high activity on mandible movement [21, 22]. Sleep fragmentation, in particular, macrofragmentation could lead to TST underestimation. The chosen sample windows are broad (102.4 s) and few quiet sleep epochs (30 s) could be considered as sleep in our analysis. Lastly, we have no knowledge about bruxist's mandible behavior and results from the automated signal analysis in such a case.

Table 4 Sleep latency assessment when the sleep latency is defined as one sleep 30-s epoch and 15 consecutive minutes of sleep data (mean difference [95% CI]±standard deviation)

Diagnosis	Sleep latency (30-s epoch)	Sleep latency (15 min)
Overall	33.1* [14.7, 51.7]±77.4	4.2 [−20.4, 28.8]±59.6
OSAHS	36.1* [6.6, 65.4]±88.7	−1.4 [−32.4, 34.8]±59.6
Insomnia	29.7* [−4.7, 64.1]±68.2	8.8 [−44.5, 62.1]±65.7
Other pathologies	33.3* [−6.0, 72.0]±59.5	11 [−33.3, 55.1]±33.9
AHI<15	29.9* [2.9, 56.7]±64.0	9.8 [−24.1, 43.7]±51.8
15≤AHI<30	35.0* [6.6, 63.4]±62.7	2.7 [−41, 46.4]±63.0
AHI≥30	36.3* [−2.7, 75.3]±99.9	−1.3 [−53.1, 50.3]±66.4

*p<0.05, statistically significant difference

Improvement of the method is desired, maybe with a multisignal approach or by the inclusion of a criterion (a post-processing) about effort. The periodicity test could be more versatile and much more pattern oriented (i.e., time behavior) than frequency tracking. To reduce the source of error, START and STOP times were defined for all patients and the results were computed only within this range. This is a focus on acquiring time between lights off and when there is light.

Mandible movement signal has two promising features over WAc: the detection of arousals and the delineation/classification of sleep apneas and hypopneas [24]. Further studies will focus on (1) the assessment of the sleep/wake mandible method on a healthy population, (2) a multisignal approach (including mandible movements) to identify the respiratory events, and (3) the evaluation of a full automated analysis based on mandible movements that computes an AHI.

Conclusions

Mandible movement recording and its dedicated signal processing for sleep/wake recognition improve sleep disorder index accuracy by assessing the TST. Such a feature is welcome in home screening methods

Acknowledgement The authors would like to thank the students from the University of Liège who helped us in this study.

References

1. Wickwire EM, Collop NA (2010) Insomnia and sleep-related breathing disorders. Chest 137(6):1449–1463
2. The American Association of Sleep Medicine Task Force (1999) Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 22(5):667–689
3. Rechtschaffen A, Kale A (1968) A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. National Institutes of Health, Washington
4. Flemons WW, Littner MR, Rowley JA (2000) Home diagnosis of sleep apnea: a systematic review of the literature: an evidence review cosponsored by the American Academy of Sleep Medicine, the American College of Chest Physicians, and the American Thoracic Society. Chest 124:1543–1579
5. The American Thoracic Society, the American College of Chest Physicians, and the American Association of Sleep Medicine (2004) Executive summary on the systematic review and practice parameters for portable monitoring in the investigation of suspected sleep apnea in adults. Am J Respir Crit Care Med 169:1160–1163
6. Russo MB, Labutta A, Vo R, Black J, Campell W, Greene J, McGhee J, Redmond D (2005) Human biovibrations: assessment of human life signs, motor activity, and cognitive performance using wrist-mounted actigraphy. Aviat Space Environ Med 76(7):C64–C74
7. American Sleep Disorders Association (1995) Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. Sleep 18(4):285–287
8. Sadeh A, Acebo C (2002) The role of actigraphy in sleep medicine. Sleep Medicine Reviews 6(2):113–124
9. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moocroft W, Pollak CP (2003) The role of actigraphy in the study of sleep and circadian rhythms, American Academy of Sleep Medicine Review Paper. Sleep 26(3):342–392
10. Tahmasian M, Khazaie H, Sepehry AA, Russo MB (2010) Ambulatory monitoring of sleep disorders. J Pak Med Assoc 60 (6):480–487
11. Elbaz M, Roue GM, Lofaso F, Quera Salva MA (2002) Utility of actigraphy in the diagnosis of OSA. Sleep 25(5):527–531
12. Hedner J, Pillar G, Pittman SD, Sou D, Grote L, White DP (2004) A novel adaptive wrist actigraphy algorithm for sleep–wake assessment in sleep apnea patients. Sleep 27(8):1560–1566
13. Lötjönen J, Korhonen I, Hirvonen K, Rekola M, Myllymäki M, Partinen M (2001) Sleep/wake detection using an active security device. Sleep 24(suppl, No. Abstract 708.R):A399
14. Lichstein KL, Stone KC, Donaldson J, Nau SD, Soeffing JP, Murray D, Lester KW, Aguillard RN (2006) Actigraphy validation with insomnia. Sleep 29(2):232–239
15. Sánchez-Ortuño MM, Edinger JD, Means MK, Almirall D (2010) Home is where sleep is: an ecological approach to test the validity of actigraphy for the assessment of insomnia. J Clin Sleep Med 6 (1):21–29
16. Paquet J, Kawinska A, Carrier J (2007) Wake detection capacity of actigraphy during sleep. Sleep 30(10):1362–1369
17. Van de Graaf WB (1988) Thoracic influence on upper airway patency. J Appl Physiol 65:2124–2131
18. Hollowel DE, Surrat PM (1991) Mandible position and activation of submental and masseter muscles during sleep. J Appl Physiol 71(6):2267–2273
19. Poirrier R, Chakar B, Lacroix A, Dive D, Hansen I, Frank G (1997) Relationship between mouth opening and intrathoracic pressure during sleep apnea. In: Somnologie Suppl. of the 5th World Congress on Sleep Apnea, number 2, page 37, 14–18 Feb
20. Miyamoto K, Özbek MM, Lowe AA, Sjöholm TT, Love LL, Fleetham JA, Ryan CF (1999) Mandibular posture during sleep in patients with obstructive sleep apnoea. Archives of Oral Biology 44:657–664
21. Senny F, Destiné J, Poirrier R (2008) Midsagittal jaw movement analysis for the scoring of sleep apneas and hypopneas. IEEE Trans Biomed Eng 55(1):87–95
22. Poirrier R, Cambron L, Maquet P, Ansay P, Destiné J, Senny F (2005) Maxillo-mandibular distance recording as a marker of non apneic sleep-disordered breathing. Sleep Medicine 6(Suppl)
23. Miyamoto K, Özbek MM, Lowe AA, Sjöholm TT, Love LL, Fleetham JA, Ryan CF (1998) Mandibular posture during sleep in healthy adults. Archives of Oral Biology 43(4):657–664
24. Senny F, Destiné J, Poirrier R (2009) Midsagittal jaw movements as sleep/wake marker. IEEE Trans Biomed Eng 56(2):303–309
25. ASDA (American Sleep Disorders Association and Sleep Research Society) Atlas Task Force (1992) EEG arousals: scoring rules and examples. Sleep 15:173–184
26. The Report of an American Academy of Sleep Medicine Task Force (1999) Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 22:667–689
27. European data format (edf). Available at http://www.edfplus.info/. Accessed 26 May 2011
28. MATLAB. Available at http://www.mathworks.com. Accessed 26 May 2011
29. R Development Core Team (2011) R: a language and environment for statistical computing, reference index version 2.13.0. R Foundation for Statistical Computing, Vienna. ISBN 3-900051-07-0. Available at http://www.R-project.org. Accessed 26 May 2011