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ORIGINAL ARTICLE

Home diagnosis of obstructive sleep apnoea in coronary patients: validity of a simplified device automated analysis

Carole Planès · Michel Leroy · Najette Bouach Khalil · Rami El Mahmoud · Franck Digne · Florence de Roquefeuil · Bernadette Raffestin

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Abstract
Introduction Our aim was to evaluate a type 3 portable simplified device as a screening tool for obstructive sleep apnoea (OSA) in coronary patients.
Materials and methods In 50 patients selected independently from sleep complaints, we compared the number of respiratory events per hour of valid recording time counted automatically by the device and the number counted manually per hour of sleep on polysomnography performed at home during the same night.
Results Five patients were excluded because of technical failures. Estimated OSA prevalences (95% confidence interval) for apnoea/hypopnoea index (AHI) cut-offs ≥5, ≥15, and ≥30 by polysomnography were 0.93 (0.81–0.98), 0.69 (0.53–0.81), and 0.27 (0.15–0.42), respectively. The device would have correctly diagnosed 75% of patients with severe OSA (AHI ≥30 by polysomnography) and would have classified the remaining 25% as having moderate OSA.
Discussion This ambulatory device may prove valuable in reducing the costs of diagnosing and managing OSA in coronary patients.

Keywords Coronary artery disease · Obstructive sleep apnoea · Ambulatory diagnosis

C. Planès · M. Leroy · N. Bouach Khalil · B. Raffestin (✉)
Service d'Explorations Fonctionnelles Multidisciplinaire,
Hospital Ambroise Paré, Assistance Publique-Hôpitaux de Paris,
Université de Versailles,
9 Av Charles de Gaulle,
92104 Boulogne, France
e-mail: bernadette.raffestin@apr.aphp.fr

R. El Mahmoud · F. Digne · F. de Roquefeuil
Service de Cardiologie, Hospital Ambroise Paré,
Assistance Publique-Hôpitaux de Paris, Université de Versailles,
Boulogne-Billancourt, France

Introduction

Many studies suggest that obstructive sleep apnoea (OSA) significantly increases cardiovascular morbidity and mortality and that treatment with continuous positive airway pressure (CPAP) not only improves symptoms and daytime function but also reduces the cardiovascular risk [1]. Epidemiological data indicate an increased prevalence of OSA among patients with coronary artery disease (CAD) [2–7] and poorer long-term outcome in CAD patients with OSA, compared to those without OSA. Compared to CAD patients without OSA, those with OSA had higher 5-year mortality rates [8] and an increased risk of stroke [9]. In a small group of CAD patients, we previously reported a reduction in new cardiovascular events after treatment of OSA [10]. The high prevalence of OSA and adverse prognostic significance of untreated OSA among CAD patients suggests a need for OSA screening in this patient population.

The American Thoracic Society [11] and the American Academy of Sleep Medicine [12] recommend supervised polysomnography in the sleep laboratory for diagnosing OSA and for initiating CPAP. However, because OSA is common, following this recommendation would require far more sleep laboratories than are currently available. The cost and the inconvenience of polysomnography have prompted the development of clinical predictive algorithms and overnight home monitoring techniques aimed at facilitating the diagnosis of OSA [13]. CID102L8 (Cidelec France) is a commercially available, level 3, portable, monitoring device that digitally records and analyse tracheal breath sounds, nasal flow from pressure obtained with a nasal cannula, thoracic and abdominal movements, body position, arterial oxygen saturation, peripheral arterial tone and heart rate from a pulse photoplethysmogram, and

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wrist actimetry. The stored data are downloaded to a personal computer using software that automatically counts snoring events, desaturation episodes, apnoeas, hypopnoeas, flow limitation episodes, and autonomic activation events. Although CID102L8 refers to the level 3 monitoring device not including neurophysiologic data, there is an upgraded version of CID102L8 which allows the recording of neurophysiologic data in addition to the respiratory parameters and may serve in the ambulatory setting as a type 2 device, comprehensive portable polysomnography.

The purpose of this study was to assess the value of automated respiratory parameter analysis by the type 3 simplified automated device as a tool for OSA screening in patients with CAD. In a group of CAD patients selected independently from sleep complaints, we compared the number of respiratory events per hour of valid recording time counted automatically by the simplified device to the number counted manually and reported by hour of sleep during unattended full polysomnography performed at home on the same night. We measured the sensitivity and specificity of the portable simplified device for identifying patients with severe OSA requiring treatment.

Materials and methods

Study population

We included in this ancillary study the 50 patients (49 men) who, after undergoing percutaneous coronary intervention following an acute coronary event, were taking part in a study assessing prevalence of OSA in coronary patients. In brief, this study was performed from April 2004 to July 2007 to examine whether proximal coronary ectasia, when observed in patients with severe coronary atherosclerosis, could be related to OSA. A sleep study was performed in 25 patients with coronary ectasia, and 25 controlled coronary patients matched for age and body mass index. Results ruled out the hypothesis since no difference in OSA prevalence and severity was observed between the two groups. No attempt was made to select the patients based on a high suspicion of sleep apnoea or previous diagnosis of sleep apnoea, but participants completed the Epworth sleepiness scale questionnaire. Diagnosis of OSA was based on overnight polysomnography performed at home in each patient. Our institutional review board approved the study which complies with the Declaration of Helsinki, and all participants gave their written informed consent.

Polysomnography

In the evening, a technician went to the patient's home to place the various leads and sensors needed for polysomnography. Polysomnographic data were recorded using the extended version of the CID102L8 (CIDELEC Ste Gemmes-sur-Loire, France) which records neurophysiologic signals including two electroencephalogram (EEG) leads (C4O2 and FP2T4), electrooculogram, and a submental electromyogram. Respiratory parameters included nasal flow assessed with a nasal cannula connected to a pressure sensor, thoracic and abdominal movements with piezo belts, and oxygen saturation measurement (SaO2) by pulse oximetry (Biochem-Ox2000, Waukesha, WI, USA) with storage of the signal at 1-s intervals. An electrocardiogram (ECG) was recorded, as well as body position and wrist actimetry. Breath sounds were recorded by a microphone placed over the sternal notch in front of the trachea. The CID102L8 microphone has a flat frequency response in the 50–20,000 Hz range. Signals from the microphone are analysed and processed according to frequency and energy as previously described [14]. Pressure variations in the airtight cavity between the skin and microphone are related to increasing deformation of the suprasternal notch during obstructed inspirations.

Interpretation of the polysomnogram

The sleep recording was interpreted by an experienced examiner who was unaware of the study and of the clinical status of the patients. Sleep stages were classified according to the criteria of Rechtschaffen and Kales [15], and arousals were scored according to the American Sleep Disorders Association criteria [16]. Apnoea was defined as the absence of nasal flow and tracheal breath sounds for longer than 10 s. Apnoea was classified as obstructive when accompanied with thoracic and abdominal movements, and as central otherwise. Hypopnoea was defined as a noticeable change in airflow associated with either an at least 3% decrease in SaO2 or an arousal on the EEG trace. The apnoea-hypopnoea index (AHI) was calculated as the total number of apnoeas and hypopnoeas per hour of sleep. Results of the full polysomnography were obtained by manual analysis only.

Automated analysis of respiratory events by CID102L8, the simplified recording not including neurophysiologic data

Neurophysiological data were disregarded, and respiratory events were automatically counted with a computerised algorithm from the same recording of nasal flow, breath sounds, thoracic and abdominal movements, O_2 saturation, body position, and actimetry. Absence of breath sounds for at least 10-s duration was counted as an apnoea. Apnoea was classified as obstructive when accompanied with thoracic and abdominal piezo belt movements, deformation

of the suprasternal notch, or snoring upon return of breathing. Central apnoeas are apnoeas without thoracic or abdominal movements, suprasternal pressure variations, or an increase of inspiratory over expiratory energy in the few breaths preceding or following apnoeas. Hypopnoea was defined as at least 30% decrease in nasal flow or heavy snoring on each breath with an at least 3% decrease in SaO_2. Periods of nasal flow flattening not followed by O_2 desaturation but accompanied with an increase in inspiratory over expiratory energy with a ratio equal to or greater than 2 on the breath sound analysis were quoted as flow limitations. Transient flattening of the pulse photoplethysmogram with a decrease in the RR interval on the ECG was counted as an indicator of autonomic activation. The automated-analysis report supplied the total numbers of obstructive, mixed, and central apnoeas; hypopnoeas; and flow limitation episodes. These numbers were given per hour of total validated analysis time since the simplified device does not allow sleep assessment. The software excluded periods during which sound-pressure signals were inadequate, as well as periods with SaO_2 less than 40% suggesting oximeter clip detachment. Although it is possible to edit the automated scoring and to review it manually, the present results were obtained by automated analysis only.

Statistical analysis

Results are given as mean±SD (min–max value) or as the median and interquartile range (25–75%). Least-square regression was used to evaluate relationships between the number of respiratory events per hour of validated recording time by automated analysis and the number per hour of sleep by manual scoring of polysomnogram. Agreement between the two methods was also assessed using the Bland and Altman analysis. Sensitivity, specificity, positive predictive value, negative predictive value, and negative likelihood ratio of the CID102L8 automated respiratory analysis for diagnosing sleep apnoea, using manual interpretation of the polysomnogram as the reference, were calculated. *P* values of 0.05 or less were considered statistically significant.

Results

Of the 50 patients, four were excluded because the quality of the EEG recording was considered inadequate for manual sleep scoring. Of these four patients, two also had poor-quality recordings of respiratory parameters. Another patient was excluded because of oximetry failure. Thus, in this unattended setting, technical failure rates were 6% (3/50) for respiratory parameters and 8% (4/50) for neurophysiologic data.

Age, anthropometric characteristics, Epworth sleepiness scale scores, and polysomnography results in the 45 patients kept in the study are shown in Table 1. Total sleep time was at least 120 min in 44 of 45 patients, and total valid recording time was 447.0±750.8 (209–480)min. None of the patients with AHI values greater than 15 according to full polysomnography had predominantly central apnoeas; the highest percentage of central apnoea was 33% in a patient whose AHI was 18.

The AHI (events per hour of sleep) by full polysomnography correlated closely with the number of apnoeas plus hypopnoeas per hour of automated-analysis time by the simplified device (r^2=0.78; Fig. 1a). Plotting the AHI difference between the two methods against their mean showed slight underestimation of respiratory parameters by the simplified device automated analysis, with a mean AHI difference (automated-analysis polysomnography) of −3.4± 77.5 (Fig. 1b). Scatter of the differences between the two methods showed no evidence of increasing with the number of respiratory events.

A flow decrease with an EEG arousal but no desaturation can be classified as hypopnoea by manual polysomnography scoring. In contrast, a flow decrease without at least 3% desaturation is not counted as a hypopnoea by the automated-analysis software of the simplified device but may be taken as a flow limitation episode if associated with an increased of inspiratory over expiratory energy ratio of the breath sound. We therefore compared the automatically counted number of apnoeas plus hypopnoeas plus flow limitation episodes per hour of valid recording time obtained with the simplified device and the manually counted number of apnoeas plus hypopnoeas per hour of sleep. As shown in Fig. 2a, agreement between the two methods was not improved (r^2=0.66). Moreover, adding flow limitation episodes to apnoeas and hypopnoeas led to overestimation of respiratory events, compared to manual polysomnography scoring, the mean difference (automated-

Table 1 Anthropometric values and results of polysomnography of the 45 study subjects

Characteristics	Values
Age (years)	63.4±11.6 (37–81)
Body mass index	26.4±3.9 (20–38)
Epworth sleepiness scale	8±8.3 (0–22)
Total sleep time (min)	346±62 (105–438)
Apnoea-hypopnoea index	23.8±15.3 (2–67)
% of time below 90% O_2 saturation	2 (0–6)[a] (0–54)
Arousal index	28.6±12.5 (11–72)

The data are mean±SD (min–max value) except for percent of time below 90%, which is reported as the median and min–max value

[a] Interquartile range

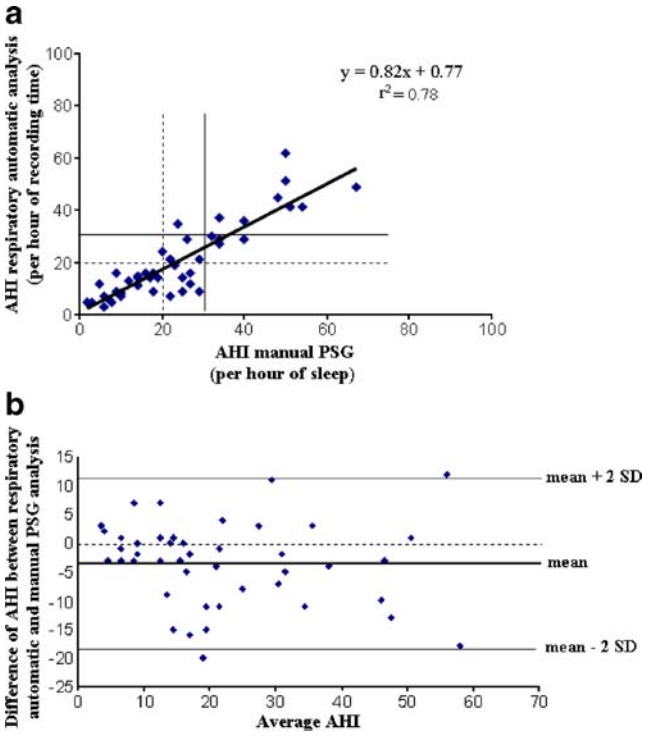

Fig. 1 **a** Apnoea/hypopnoea index (AHI) determined by the simplified device automated analysis and by manual scoring of full polysomnography recordings. **b** Plot of the difference between the two AHI values against their mean

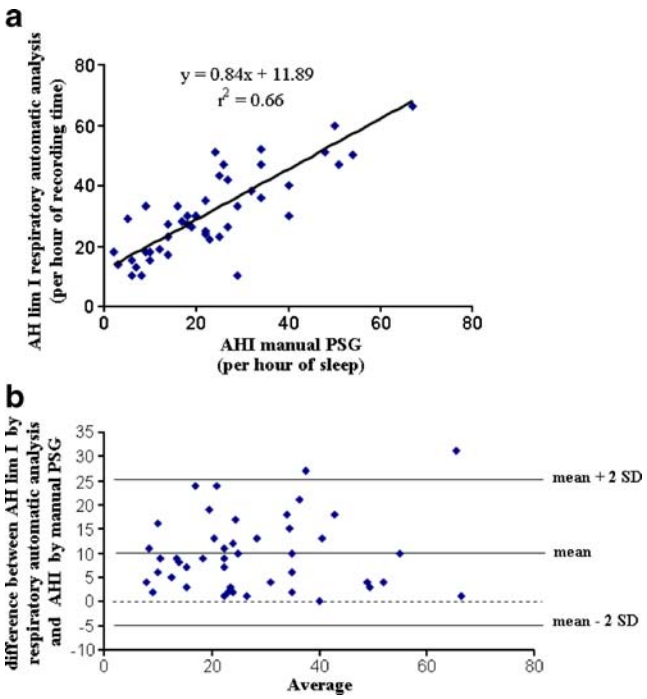

Fig. 2 **a** AHI plus flow limitations per hour determined by the simplified device automated analysis and AHI by manual scoring of full polysomnography recordings. **b** Plot of the difference between the two values against their mean

analysis polysomnography) being 10±7.5 total respiratory events per hour (Fig. 2b).

The results were analysed using several AHI cut-offs by polysomnography (Tables 2, 3, and 4). Only one of the patients whose AHI was less than 15 by polysomnography had 16 events per hour by the simplified device automated analysis. However, eight patients had 15 or more events by full polysomnography but between five and 15 events by the simplified device automated analysis. This underestimation of AHI by automated analysis was mostly due to a long period of recording without sleep (from 52 to 217 min).

AHI values of 30 or more by polysomnography indicates severe OSA with a high risk of cardiovascular morbidity and mortality [1] requiring treatment regardless of symptoms [17]. In our study, automated analysis of respiratory events by the simplified device detected nine of the 12 patients whose AHI was 30 or more per hour by full polysomnography. Of the three patients with a false-negative simplified device automated analysis, one had 27 events per hour of recording time and 34 per hour of sleep by polysomnography, one had 29 and 40 events, and one had 27 and 34 events. The only patient with a false-positive simplified device automated-analysis result had 35 events/hour by automated analysis and an AHI of 24 by polysomnography.

Discussion

The main finding from this study is that the limited channel type 3 portable monitor CID102L8 is reliable for detecting severe OSA in coronary patients during unattended testing at home. We found good agreement between the simplified device automated analysis and full polysomnography regarding the number of apnoeas and hypopnoeas. Sensitivity and specificity of this simplified device automated analysis for diagnosing severe OSA were acceptable. To the best of our knowledge, this is the first study using this ambulatory device for OSA screening in CAD patients comparatively to simultaneous home polysomnography.

The feasibility of monitoring breath sounds over the trachea to detect apnoeic events was established by Krumpe and Cummiskey [18]. However, few controlled studies comparing tracheal sonography to polysomnography are available. Automated analysis of tracheal sound records and continuous pulse oximetry was compared to simultaneous in-laboratory polysomnography in 129 adults who had symptoms suggesting OSA [19]. The overall prevalence of OSA was 45%, and the two diagnostic methods were well correlated, with the automated analysis having 84% sensitivity and 97% specificity.

Table 2 Prevalence of apnoea/hypopnoea ≥5 by polysomnography and binary classifier values of the automated analysis for detecting these patients

	Estimated value	95% confidence interval	
		Lower limit	Upper limit
Prevalence	0.93	0.81	0.98
Sensitivity	0.95	0.82	0.99
Specificity	0.67	0.12	0.98
Positive predictive value	0.97	0.85	1
Negative predictive value	0.5	0.09	0.91
Negative likelihood ratio	0.07	0.01	0.38

The simplified automated device, CID102L8, is an upgraded version of CID102, a simpler device that records only breath sounds and oximetry. This type 3 portable monitor also records nasal flow as assessed by nasal pressure measurements and thoracic and abdominal movements detected by piezo belts. We previously reported underestimation of AHI by CID102, although CID102 and polysomnography values were correlated [14]. The protocol of the study reported here differs markedly from the protocol used to evaluate CID102. Whereas, CID102 was compared to polysomnography in patients referred to the sleep laboratory for a clinical suspicion of sleep apnoea, the type 3 simplified automated device, CID102L8, was studied in consecutive CAD patients who did not spontaneously report sleep-related symptoms. Polysomnography was performed at the laboratory in the study of CID102 and at home in the study of CID102 L8. Most studies of simplified devices for OSA detection in preselected sleep-laboratory cohorts used in-laboratory polysomnography as the reference standard. The ambulatory polysomnography used in our study recorded only two EEG leads. Nasal flow was assessed by measuring pressure via a nasal cannula, but there was no oral thermistance for detecting mouth breathing. However, simultaneously recording tracheal breath sounds and nasal pressure is reliable for detecting apnoea, and a thermistance provides only a qualitative assessment of flow. Comparison during the same night at home avoids bias due to differences in the environment at home and at the laboratory, or to possible differences in the time spent in each body position, as well as differences introduced by night to night variability when in-home use of the ambulatory device is compared to in-laboratory polysomnography. Moreover, ambulatory polysomnography, which was used in the large-scale multicentre Sleep Heart Health study, correlates closely with data obtained by attended in-laboratory polysomnography [20]. Comparative studies of unattended home polysomnography and attended in-laboratory polysomnography found similar respiratory parameter values with the two methods [21, 22]. In the present study, EEG quality was considered satisfactory for sleep scoring in all but four patients.

Although the prevalence of OSA decreased as the AHI cut-off was raised, and we used similar AHI cut-offs for the simplified automated device and polysomnography, the sensibility and specificity of the simplified device automated analysis did not change significantly as the prevalence decreased. Among the 12 patients whose AHI by polysomnography was 30 or more, nine (75%) had results of the simplified device automated analysis indicating severe OSA. The three remaining patients had automated-analysis values greater than 25/h, indicating moderate OSA. A prospective observational study showed that the increased cardiovascular mortality and morbidity in patients with OSA of this severity is diminished by CPAP [1], indicating that CAD patients with AHI ≥30 may require immediate diagnosis followed by CPAP. However, the impact of OSA on mortality in epidemiological studies or sleep-clinic cohorts may not reflect the impact in specific populations, such as CAD patients, who are at high risk for mortality and morbidity related to their cardiovascular

Table 3 Prevalence of apnoea/hypopnoea ≥15 by polysomnography and binary classifier values of the automated analysis for detecting these patients

	Estimated value	95% confidence interval	
		Lower limit	Upper limit
Prevalence	0.69	0.53	0.81
Sensitivity	0.71	0.52	0.85
Specificity	0.93	0.64	1
Positive predictive value	0.96	0.71	1
Negative predictive value	0.59	0.37	0.78
Negative likelihood ratio	0.31	0.18	0.54

Table 4 Prevalence of apnoea/hypopnoea ≥30 by polysomnography and binary classifier values of the automated analysis for detecting these patients

	Estimated value	95% confidence interval	
		Lower limit	Upper limit
Prevalence	0.27	0.15	0.42
Sensitivity	0.75	0.43	0.93
Specificity	0.97	0.82	1
Positive predictive value	0.9	0.54	0.99
Negative predictive value	0.91	0.76	0.98
Negative likelihood ratio	0.26	0.10	0.69

disease [8, 9]. In a recent prospective study, the beneficial effect of CPAP on survival without new cardiovascular events was not confined to patients with severe OSA but, instead, extended to patients with AHI values in the 15–30 range [23].

Specificity of the automated analysis was good. In patients with moderate or severe sleep apnoea (AHI ≥15 by polysomnography), there was a single false-positive patient whose AHI was 16 by automated analysis and nine by polysomnography. The lower sensitivity of the simplified device automated analysis compared to polysomnography was partly ascribable to our method for manually scoring hypopnoea. Decreased flow through the nasal cannula was scored as hypopnoeas when followed by either desaturation (3% decrease in SaO_2) or an EEG arousal. By the simplified device automated analysis, apnoeas without substantial desaturation were easily detected, but flow decreases with arousal but no desaturation were not detected as hypopnoeas. The definition used in our study to identify hypopnoeas by polysomnography may be excessively broad. A recent study examining more specifically the significance of varying definition of hypopnoea showed that hypopnoeas with a desaturation of at least 4% are independently associated with cardiovascular disease but that those with an arousal are not [24]. Although the simplified automated device would be expected to detect sympathetic activation associated with arousal from sleep, via the analysis of the photoplethysmogram and heart rate, the number of these autonomic activations associated with respiratory events per hour of valid recording time correlated poorly with the AHI by polysomnography (data not shown). They underestimated AHI, and this underestimation increased at higher AHI values.

In the eight patients who had AHI values by polysomnography in the 15–30 range and AHI by the simplified device automated analysis less than 15, this underestimation of AHI by the device was in part due to a considerable difference between the valid recording time and the total sleep time. Despite actimetry assessment, the simplified automated device, CID102L8, and most of the other type 3 ambulatory devices without neurophysiologic data, cannot differentiate wake from sleep and therefore reports the number of events per unit of recording time. This may decrease sensitivity and spuriously increase specificity. This problem may be partially overcome by asking patients to keep a log indicating approximate durations of sleep periods and periods spent awake. In patients who snore loudly, periods without snoring should suggest wakefulness. The main adverse consequence of not assessing sleep via neurophysiological monitoring is AHI underestimation. However, this latter diagnostic strategy requires more technical expertise and is labour-intensive and time-consuming.

In the present study, prevalence of OSA in this group of CAD patients without sleep-related complains was surprisingly high. This could be due to age, severity of coronary disease, and to the fact that all but one patients were men. However, these patients rarely seek medical advice because of sleep-related complaints [7]. As shown by the results of the Epworth questionnaire, most of the CAD patients screened for OSA in the present study did not report daily sleepiness, a symptom suggestive of OSA. Daytime tiredness is more commonly reported but less specific and can also be related to the cardiovascular condition. Therefore, whether routine screening is warranted in this population deserves consideration, and the AHI cut-off that should be used to decide when treatment is appropriate should be determined. At present, there is no rationale for selecting a specific AHI cut-off. Recently, strategies based on a clinical score and simplified home testing followed by an evaluation of symptom improvement with auto-CPAP therapy have been developed. However, many patients are actually referred to the sleep laboratory because of co-morbidities, and an improvement in quality of life is not the main health benefit resulting from OSA treatment. Available evidence suggests that treatment may be in order in patients at risk for cardiovascular disease. Sleep apnoea is associated with an increase in the cardiovascular risk. Furthermore, several studies suggest that in patients with CAD, OSA may further increase the risk for subsequent development of cardiovascular events and ischaemic complications. Increases in the incidence of major cardiac events and in the angiographically proven restenosis rate following percutaneous coronary intervention for acute

coronary syndrome were recently reported in patients with OSA [7]. CPAP treatment of patients with OSA improves not only daytime function and quality of life but also hypertension [25], cardiac failure [26], and nocturnal angina [27]. Given that asymptomatic OSA may be associated with adverse health outcomes and that these can be prevented by CPAP treatment, CAD patients for OSA should be screened for OSA using a simple and reliable diagnostic tool. The simplified automated device, CID102L8, may be well-suited to the diagnosis of OSA in coronary patients since it differentiates between obstructive and central apnoea [28].

The simplified automated device, CID102L8, was easy to use in our study, with a failure rate of only 6% for respiratory parameter monitoring. However, one of the limitations of the present study is that the device was set up at home by a technician, not the patient. Moreover, we did not study event-by-event agreement between automated analysis and manual scoring, given the difficulty of maintaining blinded manual scoring of polysomnography. In everyday practise, a technician at the hospital uses adhesive tape to secure the ECG leads, actimeter, body position sensors, and microphone to the skin in the appropriate positions, checks signal quality and amplitude, and provides instructions to the patient about starting the recording at home at bedtime after placing the nasal cannula and oximeter. This procedure requires approximately 30 min of technician time. Downloading the recording to a personal computer takes less than 15 min. Body position is detected by the device, which can therefore determine the number of respiratory events in each position.

Based on predictive values for our home automated-analysis results, the probability of having severe OSA is high in patients whose automated-analysis AHI is greater than 30 and considerably decreased in patients whose automated analysis is less than 15. Thus, the portable type 3 simplified device CID102L8 may prove useful for diagnosing moderate-to-severe OSA in high-risk populations such as patients with CAD. However, a negative result in a patient with symptoms suggesting OSA, such as daytime sleepiness, does not rule out this diagnosis or the presence of other sleep disorders warranting in-laboratory polysomnography. Nonetheless, this simplified ambulatory device may prove valuable for reducing the cost of diagnosing and managing OSA.

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Conflicts of interest None of the authors have a financial relationship with CIDELEC, the company that developed and sells the ambulatory device.

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