

Evaluation of an ambulatory device, CID 102, in the diagnosis of obstructive sleep apnoea syndrome

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ABSTRACT: Diagnosis of obstructive sleep apnoea syndrome (OSAS) is usually performed during overnight polysomnography in the sleep laboratory. In an attempt to simplify the diagnostic strategy, we compared an ambulatory device, CID 102, with polysomnography during the same night in the laboratory in 50 consecutive patients referred for polysomnography.

The CID 102 device monitors oxygen saturation, heart rate, body position and tracheal breath sounds. An acoustic pressure sensor is placed on the suprasternal notch. Signals coming from this sensor are amplified and analysed in three different channels, according to their frequency and energy. CID respiratory disturbance index is defined as the number, per hour of analysis time, of apnoeas lasting more than 10 s plus episodes of desaturation by 4% or more associated with pauses lasting from 7–10 s or snores. The polysomnographic data were recorded on paper (Reega 2000, Alvar) and analysed manually. Polysomnographic apnoea-hypopnoea index (AHIp) was defined as the number of apnoeas plus hypopnoeas per hour of sleep.

The sensitivity, specificity, positive predictive value and negative predictive value of various CID respiratory disturbance index (≥ 5 , ≥ 10 , ≥ 15 and ≥ 20 per hour) in diagnosing obstructive sleep apnoea syndrome were determined. When OSAS was diagnosed as AHIp ≥ 15 , sensitivity and specificity of a CID respiratory disturbance index ≥ 5 were 73 and 62%, respectively. Positive predictive value of CID respiratory disturbance index ≥ 10 for AHIp ≥ 10 was 94%. CID 102 false negative patients had only hypopnoeas without any desaturation.

These results suggest that CID 102 may be helpful in the detection of severe respiratory disturbances during sleep but does not have the diagnostic value of polysomnography.

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Obstructive sleep apnoea syndrome (OSAS) is characterized by repetitive episodes of complete or partial upper airway obstruction. The chief clinical consequence of OSAS is excessive daytime sleepiness, which may result in social disharmony, employment difficulties and automobile accidents. Several reports also indicate that OSAS is associated with an increased risk of cardiovascular disease, such as systemic hypertension, myocardial infarction and stroke [1–3]. In a recent study on a population based sample of employed men and women aged 30–60 yrs, 9% of men and 4% of women had 15 or more apnoeas or hypopnoeas per hour of sleep, a level of disturbance that warrants consideration for treatment [4].

Given the high prevalence of OSAS in the general population, the serious consequences, and the fact that OSAS is readily treatable, the number of patients referred to sleep laboratories for investigation of sleep disorders would dramatically increase. Diagnosis of OSAS is usually performed during overnight polysomnography in the sleep laboratory. The cost and the inconvenience of polysomnography have favoured the development of

ambulatory recordings to simplify the diagnostic strategy. CID 102 (Cidelec, France) is a commercially available simple portable monitoring device, which digitally records tracheal breath sounds, arterial oxygen saturation (S_{a,O_2}) and body position [5]. The stored data are downloaded to a personal computer with appropriate software, which allows snoring events, desaturation episodes and apnoeas to be documented. As a first step in estimating the potential value of this device that may ultimately be used on the patient at home, we evaluated sensitivity, specificity, positive and negative predictive values of CID 102 *versus* polysomnography, during the same night, in the laboratory.

Methods

Subjects

We studied 50 consecutive subjects (49 males and 1 female) who underwent polysomnography in the laboratory. They were referred to us either because of suspected

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OSAS (n=11) or because they were taking part in an epidemiological study (n=39). Some of the latter subjects had no specific sleep complaints. The only criterion for selecting patients was that they were scheduled to undergo a complete nocturnal polysomnography in our laboratory. Every subject underwent pulmonary function tests, including measurements of lung volumes and flow-volume curves, and determination of arterial blood gas.

Polysomnography

The overnight sleep study began at the patient's usual bedtime, usually around 10.00–11.00 p.m., and was terminated after final awakening or around 7.00 a.m. The polysomnographic data were recorded with a 16-channel polygraph (Reega 2000, Alvar) at a paper speed of 15 mm·s⁻¹. Sleep was assessed with four electroencephalograms (EEG), two electro-oculograms (left and right) and submental electromyogram (EMG). Respiration was monitored with thoracic and abdominal piezo sensors (Nihon Khoden), diaphragmatic EMG, and oronasal thermocouples. An electrocardiogram (EKG) was also recorded using three skin electrodes.

Polysomnography data were analysed manually, the scorer being blinded to the results of CID 102 recording. Sleep staging was established according to the standard criteria of RECHTSCHAFFEN and KALES [6]. Apnoea was defined as cessation of oronasal airflow for more than 10 s. Hypopnoea was defined as a reduction of oronasal airflow to at least 50% of the value prevailing during preceding normal breathing, for at least 10 s, followed by transient EEG arousals. An arousal was defined as an episode lasting 3 s or longer, in which there was a return of alpha activity associated with increased EMG activity.

Desaturation was not a criterion for scoring either apnoea or hypopnoea. Apnoea was considered as obstructive when oronasal airflow was absent, while abdominal or thoracic movements and diaphragmatic EMG were present. Central apnoea was scored if oronasal airflow, respiratory movements and diaphragmatic EMG were absent. Mixed apnoea consisted of a central event followed by an obstructive one. Polysomnographic apnoea index was defined as the number of apnoeas per hour of sleep, and polysomnographic apnoea-hypopnoea index as the number of apnoeas plus hypopnoeas per hour of sleep.

Recordings with ambulatory device

CID 102 (CIDELEC; Sainte-Gemmes sur Loire, 49130 France) is a commercially available digital portable recording device monitoring tracheal breath sounds, body position and oxygen saturation. A personal computer (Note-Book) is used for data analysis.

Detection of snores and apnoeas are based on the analysis of tracheal breath sounds. A sound pressure transducer (Sennheiser Electronic KGD 3002, Wedemark, Germany)

is placed on the suprasternal notch facing the trachea. This transducer has a frequency response range of 50–20,000 Hz. Signals coming from this sensor are amplified and analysed in three different channels, according to their frequency and energy. Signals recognized as normal breath sounds (50–2,000 Hz, <80 dB) are recorded on a first channel. Snores (20–200 Hz, >76 dB) are recorded on a second channel. Very low frequency pressure variations (0.01–0.5 Hz) are analysed on a third channel. These latter reflect the intrathoracic depression related to respiratory movements and, therefore, allow the apnoea type to be defined: central, obstructive or mixed. Attenuation of transducer signal below 50 Hz is corrected by specific amplifiers (one for 0.01–0.5 Hz and the other for 20–50 Hz). On each channel, the power signal is compressed and integrated between silences which correspond to zero flow periods. Saturation signal comes from a finger pulse oximeter (Biochem-Ox2000, Waukesha, WI, USA) and is stored sequentially in memory every 1 s.

The software computes number and amplitude of snores. Apnoeas corresponding to silences of at least 10 s duration are computed as respiratory events whether or not they are associated with oxygen desaturation. Oxygen desaturation episodes to be computed as respiratory events must follow either silences of at least 7 s duration or snoring episodes. Respiratory events are, therefore, counted as apnoeas of 10 s or more duration plus episodes of desaturation by 4% or more, associated with either silences lasting from 7–10 s or snores. When apnoea ≥ 7 s or episodes of snores are detected, the top of the Sa_o₂ value at the beginning of the event is determined and considered as the baseline value. A desaturation is counted when a single decrease of Sa_o₂ by 4% or more occurs within 50 s of the beginning of the event and ends between 10–50 s after the end of the event. Episodes of desaturation which are not associated with apnoea of at least 7 s duration or snores are not computed as respiratory events. Apnoea characteristics (central, obstructive or mixed) are determined.

CID respiratory disturbance index (RDI) is defined as the number of respiratory events per hour of total analysis time. The program excludes periods during which signals coming from the sound pressure transducer are insufficient. Periods of Sa_o₂ levels below 40% are also excluded, since they may correspond to oximeter clip detachment. The RDI is calculated after subtracting these periods from the total duration of the study. A compact record is obtained and includes, for each minute recorded, body position (patient lying on the left or on the right side, or on the back), acoustic level of snores, cumulative duration of apnoeas, maximum and minimum of saturation and heart rate (fig. 1).

The software also calculates total number of snores, number of snoring episodes with more than 10 s duration, and cumulative duration of snoring episodes. A snoring episode is considered to end when there is more than 5 s between two snores.

A few hours before the beginning of the overnight study, the acoustic pressure sensor was taped to the skin on the suprasternal notch and the body position sensor

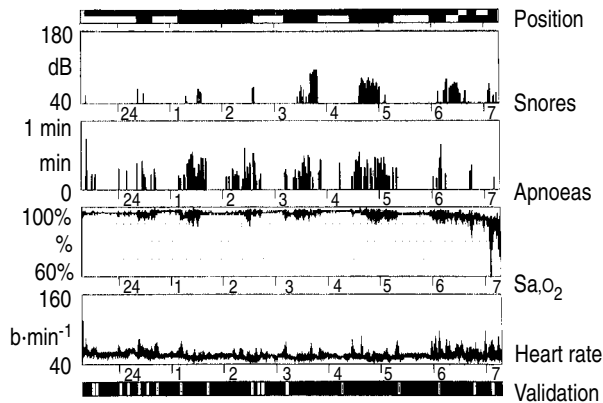


Fig. 1. – Representative example of an overnight record obtained with CID 102. For each minute recorded, the figure represents from top to bottom: body position (periods with the patient lying on his back (■), right side (▒), and left side (□)); acoustic level of snores (dB); cumulative duration of apnoeas (min); maximum and minimum of O₂ saturation (%); heart rate (beats·min⁻¹); periods of validation (closed box). The patient snores and demonstrates apnoeas and O₂ desaturation when he is lying on his back.

on the lower part of the sternum. The patient was then free to move until the beginning of polysomnography. Once polysomnography started, the probe of the oximeter was attached to the finger and the sensors plugged into the device, which was then automatically switched on 15±1 min later. Storage of data ended when the memory had been exhausted, or when the device was manually turned off. The device had sufficient memory to record continuously for 8 h.

Statistical analysis

Relationships between parameters derived from CID 102 analysis and those derived from polysomnography were evaluated using a nonparametric test (Spearman's rank correlation analysis). Total numbers of apnoeas obtained with each method were compared according to the method of BLAND and ALTMAN [7].

The sensitivity (true positive/true positive + false negative), specificity (true negative/true negative + false positive), positive predictive value (true positive/true positive + false positive) and negative predictive value (true negative/true negative + false negative) of various apnoea-hypopnoea indices were calculated for different cut-off values of CID respiratory disturbance index. A p-value less than 0.05 was considered to be statistically significant.

Results

Patients' characteristics and results of polysomnography are shown in table 1. Nine patients were obese with a body mass index (BMI) ≥30 kg·m⁻². Only two of the patients had an arterial oxygen tension below 9.3 kPa (Pa_aO₂ ≤70 mmHg) and one had an arterial carbon dioxide tension above 5.7 kPa (Pa_aCO₂ ≥43 mmHg) when awake. Only four patients had significant obstructive pulmonary disease with forced expiratory volume in one

Table 1. – Patients characteristics and results of polysomnography

Parameter	
Age* yrs	52±11
BMI* kg·m ⁻²	27±3
Total sleep time* min	369±72
Number of patients with AHI	
≥5	42
≥10	36
≥15	26
≥20	18

*: data are presented as mean±SD. BMI: body mass index (weight/height²); AHI: apnoea-hypopnoea index.

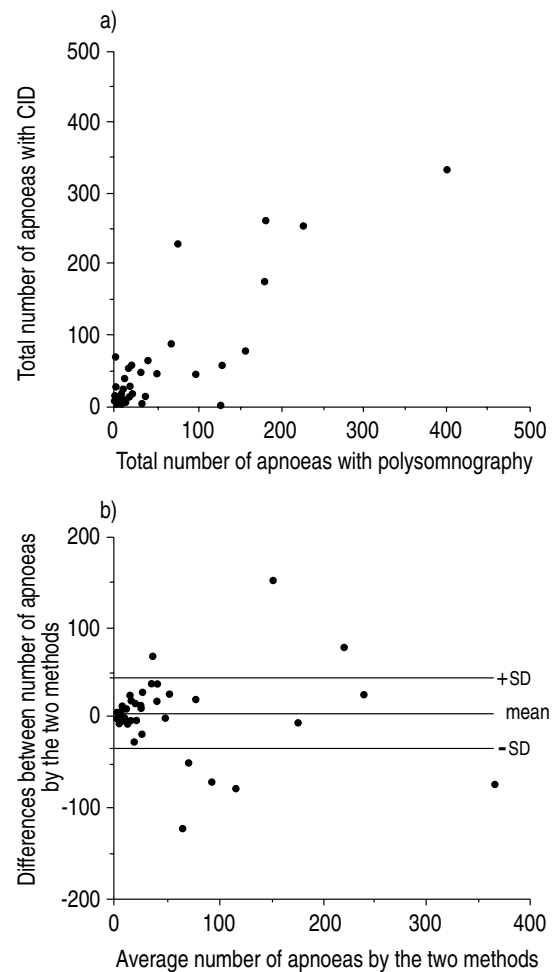


Fig. 2. – a) Relationship between total numbers of apnoeas measure with the CID 102 device and with polysomnography (p<0.01, rank coefficient correlation r=0.68). b) Values of the difference in numbers of apnoeas versus average number of apnoeas obtained with the two methods (mean±SD=3.53±39.8).

second/vital capacity (FEV₁/VC) <70% of predicted value. Only 17 of the 26 patients with apnoea-hypopnoea index of at least 15 had 5 or more 4% desaturations per hour.

Total sleep time on polysomnography was lower than total analysis time with CID 102 (369±72 vs 404±71 min; p<0,05). Relationship between total numbers of apnoeas recorded by polysomnography and CID 102

was significant ($p < 0.001$; rank correlation coefficient $r = 0.68$) (fig. 2a). Plot of the difference between the two methods against their mean demonstrates no consistent bias between the two methods, since mean difference of numbers of apnoeas was only 3.53 (fig. 2b). However, scatter of the difference as estimated by standard deviation of the difference ($SD = 39.8$) was large and appears to increase with number of apnoeas. In most of the patients, CID respiratory disturbance index was far lower than polysomnography apnoea-hypopnoea index, although both values were correlated ($p < 0.001$; rank coefficient correlation $r = 0.63$) (fig. 3). Numbers of obstructive apnoeas detected by the two methods were also poorly correlated ($r = 0.46$; $p < 0.05$).

Since the polysomnographic criterion for diagnosis of OSAS is not yet established, the results were initially analysed with OSAS defined as polysomnographic apnoea-hypopnoea index ≥ 5 , ≥ 10 , ≥ 15 and ≥ 20 for various

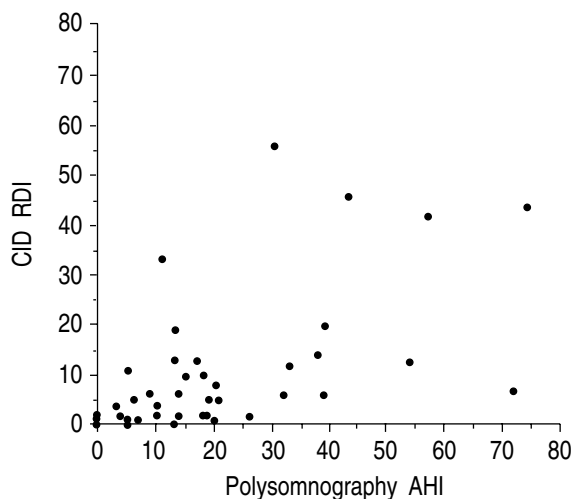


Fig. 3. — Relationship between CID respiratory disturbance index (RDI) and apnoea-hypopnoea index (AHI) of polysomnography using data from all patients ($p < 0.001$; rank coefficient correlation $r = 0.63$).

Table 2. — CID sensitivity, specificity, positive and negative predictive values

	Apnoea-hypopnoea index			
	≥ 5	≥ 10	≥ 15	≥ 20
CID RDI ≥ 5				
Sensitivity %	64	67	73	83
Specificity %	88	71	62	59
Positive predictive value	96	86	68	54
Negative predictive value	32	45	68	86
CID RDI ≥ 10				
Sensitivity %	43	47	54	61
Specificity %	100	93	83	78
Positive predictive value	100	94	78	61
Negative predictive value	25	41	63	78
CID RDI ≥ 15				
Sensitivity %	21	25	27	41
Specificity %	100	100	92	94
Positive predictive value	100	100	78	78
Negative predictive value	20	34	54	76

RDI: respiratory disturbance index.

cut-off points of CID respiratory disturbance index (table 2). For each value of apnoea-hypopnoea index taken as definition of OSAS, sensitivity increased and specificity decreased as CID respiratory disturbance cut-off value was decreased. For example, using a polysomnographic apnoea-hypopnoea index ≥ 15 to define OSAS, sensitivity of CID 102 increased from 27 to 73% when CID 102 positive cut-off value was decreased from 15 to 5. Over this range, specificity decreased from 92 to 62%. Taking respiratory disturbance index ≥ 5 as a cut-off point, CID 102 alone identified 19 of the 26 patients with apnoea-hypopnoea index ≥ 15 , a value considered for some investigators to warrant consideration for treatment. Nine patients with apnoea-hypopnoea index ≤ 15 had a CID respiratory disturbance index greater than or equal to five (false positive). In these cases, the apnoea-hypopnoea index of polysomnography ranged 5–14.

Patients with an apnoea-hypopnoea index ≥ 15 but a CID respiratory disturbance index ≤ 5 (false negative) had mostly hypopnoeas without any desaturation: apnoea index ≤ 5 in all but one. In the 18 patients with CID respiratory disturbance index ≥ 10 , 11 had severe OSAS with apnoea-hypopnoea index ≥ 20 and all but one had an apnoea-hypopnoea index of at least 10.

Number of snores detected with CID 102 was of no help in identifying patients with OSAS. Number of snores was not correlated with apnoea-hypopnoea index.

Discussion

These results suggest that CID 102, an ambulatory device based on oximetry and detection of tracheal breath sounds, may be helpful in the identification of patients with OSAS. However, this device does not have the diagnostic value of overnight polysomnography.

Several epidemiological studies suggest a high prevalence of OSAS in different population settings. Given the likely morbidity of untreated sleep apnoea, this raises questions about how OSAS should be identified. Commercially available automatic or semi-automatic ambulatory systems for monitoring nocturnal breathing and oxygenation have proliferated in recent years. Investigation of snoring to detect and diagnose sleep apnoea syndrome has also been used for many years. However, few validation studies have been published [8–14].

CID 102 is an ambulatory device which can be used during unattended studies at home. Processing of data retrieval and automated analysis is simple and requires only 10–30 min, thus saving technician time as compared with the 4–6 h interpretation time of polysomnography. To avoid possible night-to-night variability in respiratory disturbances [15], CID 102 device was evaluated *versus* polysomnography during the same night in the laboratory. CID respiratory disturbance index and polysomnography apnoea-hypopnoea index were correlated, but, in most patients, CID index was lower than polysomnographic index. Part of the difference in index between the two techniques is certainly due to the difference in definition of nonapnoeic events: desaturations with snoring or short apnoeas with the CID method and decreased oronasal expiratory temperature followed by

an EEG arousal with polysomnography. In our definition of hypopnoea during polysomnography, we chose not to require coincident desaturation but rather transient EEG arousal. Indeed, hypopnoea, even when not followed by significant O₂ desaturation, may be important to identify. When associated with transient EEG arousals, they may cause sleep fragmentation [16] and its clinical consequence, diurnal sleepiness. They may also be associated with blood pressure rises through increased sympathetic tone [17]. Desaturations observed during oximetry are markers for, but not measurements of, sleep-disordered breathing. Since detection of apnoea by CID 102 is based on absence of breath sound, the device cannot detect hypopnoea defined as a simple decrease of breath flow. The strategy for identifying hypopnoea with CID 102 is based on O₂ desaturation following either pauses shorter than 10 s or periods of snoring. If we had required that a decrease of Sa_o₂ followed the decrease in airflow in order to identify hypopnoea during polysomnography, the correlation between CID 102 and polysomnography indices, as well as sensitivity, would have been improved.

CID 102 does not monitor EEG activity and does not allow an assessment of total sleep time, but only of total registered time. In an attempt to compensate for this lack of sleep information, periods most likely to be associated with wakefulness, such as those during which there are frequent changes of body position, may be subtracted. Movements of patients during awakening are often associated with insufficient energy signals coming from the sound transducer. The program excludes such periods during the validation of the registered data by the software. The patient was also asked to record, in a diary, periods of awakening. Nevertheless, the analysis time used to compute CID index overestimated total sleeping time.

It has previously been demonstrated that recording of tracheal breath sounds allows apnoea to be accurately detected when compared with pneumotachography [18]. However, comparison between total numbers of apnoeas recorded with polysomnography and CID 102 also demonstrated discrepancy for some patients. This may be related to apnoeas of borderline duration (10 s), which could have been counted as apnoea with one method and not with the other. Hypopnoeas with severe reduction of ventilation may have been detected as apnoeas by CID 102. Moreover, CID 102 may recognize two apnoeas of shorter duration instead of one long apnoea when a loud snorting due to futile respiratory efforts occurs in the middle of the event. Since it was not possible to synchronize exactly paper recording of polysomnography and CID 102 digital recording, we could not examine event by event assessment of apnoea by both thermistance and detection of tracheal breath sounds.

CID 102 is supposed to characterize the type of apnoea. For the manufacturer, very low frequency pressure variations reflect intrathoracic depression related to respiratory movements. In our study, correlation between number of obstructive apnoeas determined by the two methods was poor. Therefore, CID 102 does not seem to be reliable for characterizing the type of apnoea.

To be clinically useful a monitoring device must be sensitive (able to correctly identify those patients with the disease) and specific (able to correctly identify those without the disease), without compromising the simplicity of implantation and cost-effectiveness. The aim of this study was to determine whether CID 102, although it may measure different events from those assessed on polysomnography, may lead to the same diagnosis as polysomnography. CID respiratory disturbance index underestimates polysomnography apnoea-hypopnoea index. Therefore, it is not surprising that an acceptable sensitivity of the device is obtained for a CID positive cut-off value lower than the apnoea-hypopnoea index. In our population, a CID 102 positive cut-off value of 5 made it possible to identify 73% of patients with an apnoea-hypopnoea index of at least 15. All patients with a CID respiratory disturbance index ≥ 15 had at least 10 apnoeas plus hypopnoeas per hour of sleep. False negative patients had only hypopnoeas without any desaturation. There is no consensus about airflow recording techniques or definition of hypopnoea. Authors have variously included either oxygen desaturation or arousal, or both [19]. In some laboratories, these false negative patients would have been considered as true negative patients. Moreover, there is no evidence that treatment is indicated in patients with only hypopnoeas, except in those with severe clinical symptoms. Increased mortality in OSAS has been reported only in patients with 20 or more apnoeas per hour of sleep [20]. In our study, taking 5 as a positive cut-off value of CID, the eight patients with 20 or more apnoeas per hour were all detected with CID 102.

Sensitivity, specificity and positive predictive value of CID 102 could have been different if we had evaluated the device during sequential home monitoring and laboratory polysomnography. Several studies using portable monitoring devices have reported a proportion of failures among unattended studies [11, 12]. In general, the incidence of failures is likely to increase with increases in the number of sensors used to detect OSAS. The CID 102 device requires only three sensors and no failure was observed during our study. However, although CID 102 was evaluated using the lowest possible amount of technician participation and the highest involvement of automatic analysis, the study was an attended one. The next step in CID 102 validation should include home investigations, eventually on several successive nights to determine the variability of symptoms.

Our data suggest that CID 102 is more sensitive than a simple nocturnal oximetry in selecting patients with OSAS. Only 17 (65%) of the 26 patients with apnoea-hypopnoea index ≥ 15 had 5 or more 4% desaturations per hour. This low sensitivity of oximetry alone in our study may be due to the fact that most of the patients had normal lung function and no baseline hypoxaemia. Among patients with similar frequencies and durations of nocturnal apnoeic events, there is considerable variability in the degree of nocturnal arterial haemoglobin desaturation. The amount of apnoea or hypopnoea related desaturation depends on several factors, including baseline Sa_o₂ level, functional residual capacity, apnoea or

hypopnoea duration, apnoea type. The number of 4% desaturations could be very different from one patient to another with the same apnoea-hypopnoea index. The value of nocturnal oximetry alone in the evaluation of subjects with suspected OSAS is controversial. In the study by GYULAY *et al.* [13], when 5 or more episodes of 4% desaturations were required to identify patients with OSAS (apnoea-hypopnoea index ≥ 15), home oximetry had a sensitivity of 50% and a specificity of 90%. In this latter study, 93% of the OSAS patients spent at least 1% of the time at S_{a,O_2} below 90%. However, specificity decreased to 53% with the use of this criterion. The high sensitivity (98%) of ambulatory oximetry reported by SERIES *et al.* [14] was based on the overall recognition of recurrent decreases in S_{a,O_2} without defining the magnitude of these decreases. However, the low specificity of this method (48%) made it necessary to perform a complete polysomnographic recording to confirm or rule out the diagnosis of OSAS.

Recording from CID 102 positional sensor makes it possible to detect whether there is a position in which the patient snores more frequently and in which apnoeas occur. There is evidence that many unselected patients who sustain a sleep apnoea diagnosis demonstrate a differential rate of apnoeic events when these are calculated separately by time in each position [21]. The results presented in figure 1 illustrate such a patient, who snores and demonstrates apnoeas and desaturations only when he is lying on his back.

In summary, CID 102 is a simple device for diagnosis of OSAS. Such an ambulatory technique does not have the diagnostic value of overnight polysomnography but may help to limit the number of such investigations. According to the recommendations of the American Sleep Disorders Association for the use of portable recordings in the assessment of OSAS [22], a CID respiratory disturbance index of 15 or more should be sufficient to confirm diagnosis and shorten delay in initiation of therapy in patients with severe clinical symptoms. However, in patients with excessive diurnal sleepiness, CID 102 does not allow the exclusion of breathing abnormalities, such as hypopnoea without desaturation, and other sleep disorders, such as periodic limb movements. Moreover, the performance of the device during unattended home monitoring needs further evaluation.

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