Home Oximetry Studies for Diagnosis of Sleep Apnea/Hypopnea Syndrome*

Limitation of Memory Storage Capabilities

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Background: Memory oximeters enable diagnostic studies for sleep apnea hypopnea syndrome (SAHS) to be performed in the home. However, memory capabilities may be limited.

Study Objectives: To compare a pulse oximeter used at home with an 8-h memory, storing data every 12 s, and in the laboratory, with on-line recording every 2 s.

Design: Prospective cohort study.

Setting: Patients’ homes and a sleep laboratory.

Patients: One hundred patients with suspected SAHS.

Measurements: Home oximetry and a laboratory full polysomnography. The number of ≥ 4% dips in pulse oximetric saturation (SpO₂) was calculated for each study. Daytime sleepiness was assessed by the Epworth Sleepiness Scale (ESS) score.

Results: The mean dips per hour were 5.3/h (range, 0 to 53/h) for home studies and 13.4/h (range, 0 to 106/h) for laboratory studies; the relationship between home and laboratory studies was as follows: home = (0.4 × laboratory) – 0.01 ± 11.2; r² = 0.64. Mean difference was 8.4/h (−2.5 to +77.9/h), which correlated with the mean of the measurements. At a cutoff point of 10/h, 52 studies were both negative and 13 studies were both positive. Nineteen home studies were false-negatives. Sensitivity was 0.41, and specificity was 1.0. In these 19 studies, 7 patients had an ESS score >10 and 4 patients had an ESS score >14. To confirm that differences were due to different sampling rates, 16 additional patients had on-line data and stored data collected simultaneously in the laboratory. Mean dips per hour were 3.2/h (range, 0.1 to 18.3/h) for the stored data and 8.34/h (0.2 to 22.8/h) for on-line data; the relationship being stored was as follows: 0.5 on-line = 1.17 × (stored) + 2.6; r² = 0.69. Mean difference was 5.2/h (0.04 to 15.4 h), which correlated with the mean of the measurements.

Conclusion: Home studies using a memory storage pulse oximeter may underestimate the number of hypoxic dips, probably due to sampling rates. Clinically significant hypoxic SAHS may therefore be missed.

Key words: polysomnography; pulse oximetry; sleep apnea

Abbreviations: CPAP = continuous positive airway pressure; ESS = Epworth Sleepiness Scale; SAHS = sleep apnea hypopnea syndrome; SpO₂ = pulse oximetric saturation

The sleep apnea hypopnea syndrome (SAHS) is an important public health problem, affecting 1 to 4% of middle-aged men and approximately 2% of middle-aged women. With increasing recognition of the syndrome, the need has arisen for a simple patient-friendly diagnostic test to cope with the increasing number of referrals. To this extent, pulse oximeters, which record pulse oximetric saturation (SpO₂) and cardiac frequency, have been shown to be useful, although pulse oximetry cannot be used to exclude SAHS.

To be useful as a diagnostic tool in the home, a pulse oximeter should have a memory storage capability. There are few studies in adults and none in children to our knowledge assessing whether pulse oximeters used for home studies adequately reflect that which is observed in the laboratory. Warley et al demonstrated, using simulated computer-generated signals, that Biox pulse oximeters are adequate for assessing SAHS. However, we have observed that there is a discrepancy between the number of falls in...
SpO₂ of ≥4% obtained from home studies and from on-line recordings in the laboratory using similar oximeters.

Materials and Methods

Patients

One hundred consecutive patients were studied. They were referred from ear, nose, and throat surgeons, primary-care physicians, and other chest physicians for assessment of suspected SAHS using full polysomnography. All completed an Epworth Sleepiness Score (ESS).⁸

Pulse Oximeter

The oximeters used in this study were the Biox 3740 (Ohmeda; UK). For the home and laboratory studies, the oximeters were identical, including the software version (version 9) and the default settings. Signal averaging defaulted to 6 s in this software version. A finger-clip oximeter probe was used in all studies. These oximeters have a memory storage capability of 8 h, storing a data point every 12 s. This memory capability was used in the home studies and some of the laboratory studies for comparison. Recordings online in the laboratory studies recorded a data point every 2 s. These oximeters also have pulse waveforms that provide an indication of signal strength. It was not possible to evaluate whether good signal strength was obtained in the home studies. In the laboratory studies, the pulse waveform was monitored throughout the night to ensure good quality signal strength.

Home Studies

Patients were loaned an oximeter to use overnight at home. They were instructed to behave normally, retiring to bed at their usual time and rising as normal, and they were asked to record if they had consumed alcohol and whether they had had a normal night’s sleep. They were shown how to set up the oximeter and how to disable the pulse volume and low-saturation alarms. On return, the 8-h memory was downloaded into the CNS SleepLab Laboratory system (Erich Jaeger; UK) for analysis. A download program was written in-house specifically to retrieve data from the oximeter memory, so that it could be analyzed using the CNS SleepLab analysis suite.

Laboratory Studies

Three studies were performed.

Study 1: This study was performed to determine whether any differences existed between the in-house–developed download programs on the CNS system analysis software. We studied 16 patients and compared the results from the CNS system analysis to another, previously validated, commercially available analysis package on a BBC-B computer. The memory of the oximeter was downloaded into the CNS system analysis software and into a BBC-B computer. The BBC-B produced a printout and analysis of 4% dips in SaO₂ in 8 sequential hours.⁹

Study 2: Within 3 days of the home study, patients underwent a full polysomnographic study that included EEG, electro-oculography, electromyography, and ECG recordings, thoraco-abdominal and nasal-oral airflow measurements, and pulse oximetry.¹⁰ The signals were recorded on-line using the SleepLab system. Video sound recording was made throughout the night. Patients went to bed at their normal bedtime; if they consumed alcohol on their home study night, they were allowed to consume similar quantities on the night of the study.

Study 3: To confirm the findings of the initial comparison between home and laboratory studies, a further 16 patients used two oximeters simultaneously in the laboratory, and concurrently with full polysomnography. The second oximeter was used to simulate the home study, with the data being stored in the oximeter memory.

Analysis of Sleep Studies

The analysis of the oximetry data was performed using the CNS SleepLab software. This was set to analyze the SpO₂ for dips of ≥4%. For each analysis, the total number of dips, the averaged basal SpO₂, and the lowest recorded SpO₂ were obtained. The total time for the study was obtained from the recorded download time and corrected for periods of artifact. For the laboratory study, the total time of recording, less artifact, was obtained. Dips per hour were calculated for each study by dividing the total number of dips recorded by the total time of the study.

Data Analysis

The data were analyzed using the Minitab statistics package (release 12.23).¹¹ To obtain the relationship between the number of dips per hour from the home studies and the number of dips per hour from the laboratory studies, linear regression analysis was applied, its significance being established by analysis of variance as applied to regression. To compare the two methods, the data were analyzed using method comparison analysis.¹² This compares the difference between the measures from each method to the mean of those measures. The mean difference between the two measurements indicates the bias, and the SD of the differences indicates the error between the two methods. The 95% confidence limits for the estimates of the mean difference and the limits of agreement (difference ± 2 SDs) were obtained. Differences were calculated as (laboratory study − home study). From the data obtained, the sensitivity and specificity of home oximetry studies were obtained using cutoff points of 10/h¹³ and 15/h, respectively.¹⁴ An ESS score of 0 to 10 was taken as a normal level of daytime sleepiness, while a score of >14 was regarded as significant clinical daytime sleepiness. Data are presented as mean (range) unless otherwise stated.

Results

All subjects completed the studies without difficulty. None of the subjects consumed alcohol on either of the study nights.

Study 1

In the 16 subjects studied, the mean number of hypoxic dips using the CNS SleepLab analysis suite was 23.5/h (range, 4 to 103/h), compared to 22.5/h (range, 5 to 109/h) from the BBC-B analysis. This gave a mean dips per hour of 3.2/h (range, 0.6 to 13.8/h) and 3.1/h (range, 0.7 to 14.3/h) for the CNS SleepLab and BBC-B analysis, respec-
tively. The relationship of the CNS SleepLab analysis to BBC-B analysis was as follows: CNS SleepLab = 0.03 + (1.02 × BBC-B) ± 0.37 (r² = 0.99, p < 0.001). The mean difference was 0.1/h (SD, 0.36/h). The limits of agreement were −0.62 to 0.82/h, and the 95% confidence limits were −0.1 to 0.3/h.

Study 2

Eighty-four patients were studied. All underwent full polysomnography within 3 days of the home studies. The mean number of 4% dips for the home study was 35.9/h (range, 0 to 268/h) compared to 106/h (range, 0 to 897/h) for the laboratory study. This gave means of 5.3/h (range, 0 to 53/h) and 13.7/h (range, 0 to 106/h) for the home study and laboratory study, respectively. The relationship of home study to laboratory study was as follows: home = (0.4 × laboratory) − 0.01 ± 11.2 (r² = 0.64, p < 0.001). The differences were significantly correlated with the mean of the measurements; the relationship was as follows: difference = 1.14 − (0.76 × mean) ± 7.6 (r² = 0.64, p < 0.001). There was therefore a significant proportional bias with larger differences occurring at higher mean values. This bias was removed when the reciprocal of the difference was taken. Before transformation, the mean difference was 8.4/h, the SD was 12.6/h, the limits of agreement were 16.8 to 33.6/h, and the 95% confidence limits were 5.6 to 11.1/h (Fig 1). After transformation, the mean difference was 0.34/h, the SD was 1.02/h, the limits of agreement were 1.7 to 2.4/h, and the 95% confidence intervals were 0.12 to 0.56/h.

Using the cutoff of 10/h for SpO₂, 52 studies were both negative and 13 both positive. Nineteen studies were negative on home study, but positive on laboratory study. These 19 studies were therefore false-negatives. The sensitivity and specificity were 0.41 and 1.0, respectively.

In the 19 false-negative studies, seven patients had an ESS score > 10, and four patients had an ESS score > 14. The mean ESS score was 11.2 (range, 3 to 20). In the 13 positive studies, the mean ESS was 10 (range, 1 to 18); in the 52 negative studies, the mean ESS score was 10.6 (range, 0 to 24). The χ² test showed that the distribution of ESS scores among the three groups was not significantly different (χ² = 4.3; degrees of freedom = 4).

The averaged basal SpO₂ values obtained from the home and laboratory studies were significantly different at 95% (86.6 to 98.2%) and 93.3% (85.4 to 96.4%), respectively (paired t test, p < 0.001). The lowest recorded SpO₂ was 85.6% (51 to 94%) and 79% (30.7 to 91.7%) for the home and laboratory studies, respectively; these were also significantly different (p < 0.001).

From the full polysomnography studies, the mean number of apneas and hypopneas was 123 (range, 25 to 943), giving an apnea-hypopnea index of 15.9 events per hour (range, 4.1 to 111.4/h). The distribution of positive, negative, and false-negative studies was similar to that obtained using oximetry alone for a cutoff level of 15/h.

From these results, the patients who had negative
findings on both home and laboratory studies and a normal ESS score did not have a trial of nasal continuous positive airway pressure (CPAP), while those who had positive findings on both studies underwent a trial of CPAP regardless of their ESS score. For the 19 false-negative studies, all patients underwent a trial of CPAP, of which six patients remained on CPAP at the end of the trial.

Study 3

Sixteen patients completed this study. The mean SpO₂ dips per hour was 3.2/h (range, 0.1 to 18.3/h) for stored data in the laboratory and 8.34/h (0.2 to 22.8/h) for the on-line recordings. The relationship of on-line data to the stored data was as follows: stored = (0.5 × on-line) − 1.17 ± 2.6 (r² = 0.69, p < 0.001). The mean difference was −5.2h and the SD was 4.4/h (Fig 2). The limits of agreement were −3.6 to 14.0/h, and the 95% confidence limits were −2.8 to 7.6/h.

Discussion

The results of this study show that home oximetry studies using the oximeters used in this study in patients with suspected SAHS significantly underestimate the number of episodes of hypoxemia during sleep, and may therefore miss more clinically significant SAHS than oximeter studies analyzed on-line in the hospital. The results suggest that this is due to the memory capability of the oximeter, in that a data point is stored every 12 s as compared to every 2 s when recordings are made on-line in the laboratory. These findings have clinical, epidemiologic, and technical implications for home oximetry studies in adults. No such studies have yet been carried out in children.

The number of hypoxemic episodes detected from the home studies was less than one half the number detected during on-line recordings. This was not due to the analysis program used as the analysis of both the CNS SleepLab and the BBC-B systems gave almost identical results (study 1). It is also unlikely that the difference can be explained by (1) differences in the conditions under which the studies were made (oximetry only vs laboratory polysomnography), since the patients were in their own beds and had less equipment attached when at home, thereby favoring better sleep and hence more apneas; (2) an order effect of performing the home studies before the polysomnography studies; study 3 shows similar results to study 2, and although some of the differences between the results in study 2 may be explained by an order effect, this is probably small in comparison to the effects of different memory storage methods used in this study; and (3) signal quality. We could not ensure that signal quality was as good as that obtained in the laboratory, using the pulse waveform as a guide. The fact that the differences remained in study 3 suggests that this may have only contributed a minor degree to the differences observed in study 2.

The most likely explanation for the difference between the home and laboratory studies lies in that way in which the data were collected on the 2 nights, even when the two systems were used simultaneously in the laboratory (study 3). The memory store of the oximeters used in this study is built up by collecting the lowest arterial SpO₂ during each 12-s period. In contrast, the on-line system collects data every 2 s. Although Warley et al.⁷ have shown that short sampling time periods will detect transient hypoxemic episodes, it appears that collecting data every 12 s is not sufficient to detect all the hypoxemic episodes in patients with SAHS.

These findings reinforce previous work showing that pulse oximetry cannot be used to exclude SAHS.
As well as the data storage problem shown in this study, which is particularly relevant to home studies, oximetry alone does not detect apneas and hypopneas that are not associated with hypoxemia. Furthermore, its use is restricted to patients who have a normal \( \text{SpO}_2 \) when awake, since even normal hyperventilation during sleep causes oxygen desaturation in patients with preexisting hypoxemia.

Nevertheless, many authors advocate the use of pulse oximetry in the diagnosis of SAHS, reserving more complex, more expensive, and less convenient investigations for patients in whom oximetry studies are inconclusive. Although pulse oximetry is less sensitive than polysomnography in detecting apneas and hypopneas, most cases of SAHS can be diagnosed from clinical history and oximetry. This was confirmed in our study, in which, using oximeter data alone and using a cutoff point of 10/h, no additional cases of SAHS would have been diagnosed using polysomnography rather than laboratory oximetry. Furthermore, there is increasing recognition that standard polysomnography is little or no better than oximetry alone in detecting clinically significant arousals from sleep, which are the main reason for treating sleep apnea.

To assess the possible clinical significance of the apparent false-negative home studies, we used the ESS to estimate the degree of daytime sleepiness. In the 19 false-negative studies (using a cutoff point of 10/h to diagnose SAHS), seven patients (37%) had an ESS score of > 10 and would therefore be regarded as having a high level of daytime sleepiness. Although this is consistent with the hypothesis that home oximetry missed more cases of clinically significant SAHS than on-line oximetry, these findings must be interpreted with caution, as in this study the ESS score was unable to distinguish between positive, false-negative, and true-negative findings. To further assess whether clinically significant SAHS had been missed, we determined how many patients responded to nasal CPAP. Using a cutoff point of 10/h, all of the 19 false-negative studies had a trial of CPAP, and six of these patients continued to use CPAP at the end of the trial. Thus, at least in these six patients (7% of the total), clinically significant SAHS would have been missed using home oximetry studies alone.

Since home oximetry consistently underestimates the number of hypoxic episodes compared with online oximetry, a question which arises is whether the equation relating the two (home = (0.4 \times \text{online}) + 0.01 \pm 11.2) can be used to define a different cutoff point for the diagnosis of SAHS using home oximetry. Although the equation can be used as a guide, the wide confidence limits of the equation limit its usefulness, reflecting the fact that the home oximeter system is intrinsically less accurate than the laboratory system for this purpose, because of its inadequate storage system. Furthermore, this equation only applies to oximeters that use a 12-s memory storage facility. In our view, the best solution to this problem is to use an oximeter, which has better storage capabilities.

As well as affecting the clinical management of patients, these results may also have epidemiologic implications. Studies of prevalence have used oximeters with similar memory storage, and it is likely that they have underestimated the number of individuals with hypoxic sleep apnea. Paradoxically, if this is true, it emphasizes the reason why oximetry is being used in many centers. The large number of patients being referred with suspected SAHS has made it necessary to use a system that is inexpensive and easy to use, ideally in the patient’s home.

Home oximetry, combined with a good clinical history, can have a useful place in the investigation of patients with suspected SAHS. However, the results of this study show that the data from home oximetry are dependent on the sampling rate of the memory storage system of the oximeter and are not identical to the data obtained in the sleep laboratory using on-line recordings. It is important that clinicians are aware of this when using oximetry for home assessments. Also, manufacturers need to be familiar with the clinical implications of different sampling rates used in oximeter memories, and to continue to improve them.

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