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**Update on the use of portable monitoring system for the diagnosis of sleep apnea in specific population**

Treptow E *et al.* Update on the use of portable monitoring system

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**Abstract**

The prevalence and severity of obstructive sleep apnea (OSA) is higher in specific population: children, elderly, obese and patients with pulmonary and cardiovascular diseases, compared to the general population. OSA is associated with greater morbidity and mortality in these patients. Although full-night polysomnography is still the gold standard diagnostic sleep study for OSA, it is a time consuming, expensive and technically demanding exam. Over the last few years, there is growing evidence on the use of portable monitors (PM) as an alternative for the diagnosis of OSA. These devices were developed specially for sleep evaluation at home, at a familiar environment, with easy self-application of monitoring, unattended. The use of PM is stablished for populations with high pre-test probability of OSA. However, there is a lack of studies on the use of PM in age extremes and patients with comorbidities. The purpose of this review is to present the studies that evaluated the use of PM in specific population, as well as to describe the advantages, limitations and applications of these devices in this particular group of patients. Although the total loss rate of recordings is variable in different studies, the agreement with full-night polysomnography justifies the use of PM in this population.

**Key words:** Polysomnography; Portable monitoring; Home unattended portable monitoring; Out-of-center sleep testing; Elderly; Children; Cardiovascular diseases; COPD; Obesity

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**Core tip:** This is the first review that evaluated the use of Portable Monitoring as an alternative method for diagnosis of obstructive sleep apnea in specific population. Additionally, we present the physiopathological background, technical considerations and clinical implications on the use of PM in age extremes and patients with comorbidites. We also describe the advantages, limitations and applications of these devices in children, elderly, patients with cardiovascular or respiratory diseases and obese patients.

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**INTRODUCTION**

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of airflow cessation or reduction despite evidence of continuing respiratory effort. Previous studies of the general population estimated a prevalence of OSA of 2% to 5% in women and 3% to 7% in adult males[1,2]. However, due to the burden of obesity worldwide and the higher life expectancy observed in the last years, those previous studies probably underestimate the actual prevalence of OSA. In an epidemiological study conducted in Brazil[3], that included 1042 inhabitants, OSA was observed in 32.8% of the participants. The prevalence of OSA was higher among males, overweight and obese patients and increased with age. Peppard *et al*[4] also observed the highest prevalence among older subjects. The authors estimated the current prevalence of moderate to severe sleep disordered breathing (SDB) of 17% among 50 to 70 years old man and of 9% among 50 to 70 years old women. The prevalence and impact of OSA may be even higher in specific comorbid medical conditions as cardiovascular and respiratory disorders.

Polysomnography (PSG) is the standard diagnostic test for OSA. It is a time consuming, expensive and technically demanding exam and requires that the patient sleep in an unfamiliar environment. Even if taken into consideration the initial prevalence described in the studies, the demand for PSG is higher than the capacity of sleep centers for performing the tests[5] and many patients remain undiagnosed or untreated[6]. Therefore, in the last few years, portable monitoring (PM) systems were proposed as an alternative diagnostic method. PM is cost-effective, diminishes the time on the waiting list and allows sleep studies to be done at the patient’s home.

In 1994, the American Sleep Disorders Association (ASDA), a precursor to the American Academy of Sleep Medicine (AASM) classified sleep studies into four types (Table 1)[7,8]. Full-night PSG (type 1) is the gold-standard diagnostic test for OSA and must be attended by a technician able to intervene if there is an equipment malfunction or sensor disconnection during the recording time. The most evidence on the use of PM comes from studies with type 3 monitors, which requires a minimum of 4 signals: ventilation (at least two channels of respiratory movement; or respiratory movement and airflow), oxygen saturation, and ECG or heart rate. It is an unattended exam that be applied in sleep laboratories, hospitals, clinics and at home. Recently, Collop *et al*[9] proposed a new method for the classification of sleep studies accordingly to the sensors used to evaluate the following parameters: sleep, cardiovascular, oximetry, position, effort and respiratory (Table 2).

An important step towards the consolidation of the use of PM in the diagnosis of sleep apnea was achieved in 2009[10,11]. The Centers for Medicare and Medical Services (CMS) approved the use of PM as a diagnostic tool and allowed continuous positive airway pressure (CPAP) coverage when clinical evaluation and unattended PM systems were used for the diagnosis of OSA. Also, a workshop sponsored by the American Thoracic Society (ATS), the American College of Chest Physicians (ACCP), AASM and the European Respiratory Society (ERS) was held to determine the research priorities for incorporating ambulatory management of adults with obstructive sleep apnea into healthcare systems[12]. One of the main points discussed in this workshop was the need to include specific population in the studies with PM. There is still a lack of evidence evaluating diverse ethic groups, elderly, children and comorbid conditions such as chronic pulmonary obstructive disease (COPD), asthma, heart failure, obesity and neuromuscular disorders.

The Portable Monitoring Task Force of AASM[13] recommends PM as an alternative to PSG for the diagnosis of OSA in patients with a high pretest probability of moderate to severe OSA and does not recommend the use in patients with comorbid medical disorders or in specific population (elderly and children). However, the need for earlier diagnosis, particularly in the presence of comorbidities and the evidences of PM as a cost-effective method[13–17] led to many studies evaluating specific population in the last years. Therefore, the aim of this review is to describe the recent findings, as well as the advantages and limitations of the use of PM in patients with comorbidities and particular population that we have chosen to call “specific population”.

**ELDERLY POPULATION**

***Physiopathological background***

The prevalence of sleep apnea increases with age[3,4] due to several mechanisms: sleep pattern, hormones, respiratory system changes and a higher prevalence of comorbidities. Total sleep time (TST) sleep efficiency, REM sleep and slow-wave sleep decrease in older subjects (18). Sleep becomes more superficial and fragmented and this may contribute to the development of SDB[18,19]. After menopause, the prevalence of sleep apnea in women is similar to men, mainly due to the effects of lower levels of sexual hormones[18,20]. Estrogen is associated with peripheral body fat deposition and lower levels predispose an increase in central fat. It is also related to temperature regulation in the body. Hot flashes are common during menopause and increase arousals during sleep. Progesterone is a respiratory stimulant and lower levels result in impairment of the ventilatory control. Age is associated with a decrease pulmonary vital capacity (PVC), increased pharyngeal wall fat deposit and impairment of the control of pharyngeal dilator muscles that may result in diminished airway patency during sleep[18,21].

***Technical considerations***

Cognitive impairment, Alzheimer’s disease and dementia contribute to difficulties when considering the use of PM at home in the eldery. Therefore, it is important a careful explanation of the exam for the patient as well as for a caregiver that may help during the application of the sensors. It is recommended verbal and written instructions and a brief practical demonstration[22]. Whenever possible, leg electromyography (EMG) monitoring should be included in the sleep studies due to the higher prevalence of period leg movement in this population[22,23].

***Clinical evidence of the use of PM in elderly population***

Morales *et al*[24], enrolled 452 participants with average age of 71.4 years, who presented sleepiness complaints, and evaluated a risk score for apnea and the use of PM ResCare Auto Set (Resmed, Sydey, Australia) at home. This PM measured airflow with the use of a nasal cannula (PM type IV). The results of PM used alone, in the absence of clinical data, had limited discriminatory power for the finding of severe cases of OSA. Therefore, the combination of sleep apnea symptoms, neck circumference, age, sex and the recording of airflow and respiratory effort were more accurate in identifying severe OSA in older adults. The agreement between PSG and the unattended sleep study was better in patients presenting more than 20.9 events/h.

PM type 3 Stardust II (Philips Respironics, Inc., PA, United States) was used to evaluate 43 patients over 65 years with high pre-test probability of OSA[22]. The receiver operator curve (ROC) of PM *vs* PSG were greater than 0.83 in all cutoffs analyzed. However, the specificity of the use of PM was above 60% only when in patients with moderate and severe OSA. There was a loss of data of 10.5% mainly due to download failures and oximetry record loss. The authors concluded that PM had a good diagnostic agreement with attended PSG and may become an affordable and comfortable alternative method for the diagnosis of OSA in this population.

***Clinical implications***

Gooneratne *et al*[25] evaluated, on a longitudinal cohort study, 289 older patients, followed for an average of 13.8 years and with mean age of 78 ± 6.4 years. The authors observed a mortality hazard ratio of 1.5 in older adults with excessive daytime sleepiness (EDS). The combined risk factors of EDS and SDB - defined in this study as apnea hypopnea index (AHI) over 20 events/h of sleep - increased the mortality hazard ratio to 2.3. Another study[26], evaluated healthcare costs for 2 years in elderly and middle-aged patients with OSA matched with healthy controls. The results showed that the costs were 2.02 and 1.81 times as high in elderly and middle-aged patients with OSA, respectively, in comparison to their controls. Possible reasons implicated in higher costs in elderly population are greater morbidity, medication effects, depression and anxiety. Therefore, the use of PM in elderly may provide earlier diagnosis and reduce morbidity and mortality in this population.

**CHILDREN**

***Physiopathological background***

Multiple factors have been implicated in the development of OSA in children[27]. Although adenotonsillar hypertrophy is the leading cause of OSA in this population, other anatomical factors, increased upper airway collapsibility and obesity are responsible for the burden of OSA in children, with the peak prevalence from 2 to 8 years old. Anatomical factors such as nasal obstruction, macroglossia, micrognathia, abnormalities of the cranial base and midfacial hypoplasia result in a decrease of the upper airway patency. The increase in upper airway collapsibility is evaluated by the critical closing pressure (Pcrit) - the pressure in which the upper airway collapses. This pressure is higher in children with OSA compared to those that only present primary snoring[27–29]. Finally, obesity results in increased work of breathing and fat deposition in the upper airway.

***Technical considerations***

One of the challenges on the use of PM in children is the tolerability of the sensors during the exam. The most common cause of discomfort is usually the thermistor or nasal cannula[30,31]. Also, body movements may interfere with the signals acquired[32]. A limitation of the these type of sleep studies in children is the lack of evaluation of end-tidal carbon dioxide pressure (PCO2), an important measure for detecting hypoventilation[31,33] that is an important criteria in children.

***Clinical evidence of the use of PM in children***

Studies have demonstrated conflicting results on the use of PM in children[30-32,34]. The type of sleep study used, the setting (home vs laboratory), different definitions for SDB and the diverse age range evaluated may account for these results.

Goodwin *et al*[31]evaluated the use of PM in 157 children enrolled in the cohort entitled Tucson Children’s Assessment of Sleep Apnea study (TuCASA). The sleep evaluation consisted of a Type II monitoring performed at home and the equipment was placed and calibrated by technicians. The failure rate was 9%, mostly due to problems with pulse oximetry. High quality data was obtained in more than 90% of the exams and the majority of patients did not experience any discomfort. The authors concluded that the use of an unattended sleep study is feasible in children ages 5 to 12 years.

In 2002, a study with only 12 participants aged 3 to 6 years[32] compared the use of PM in the laboratory setting with PSG. The PM measured the following signals: thermistor, laryngeal microphone, an ECG lead, a belt for thoracic effort, two belts for abdominal effort, body position and a pulse oximeter. PM underestimated the incidence of OSA and overestimated the number of central apnea cases. Although the sensitivity increased with the increase of respiratory disturbance index (RDI), specificity was low in most RDI cutoffs. Furthermore, these two studies failed to compare data using AASM guidelines (measures of airflow with nasal cannula, and interpretation according to pediatric criteria). Based on these results, PM should be reserved for highly suspected OSA in children when treatment is urgently needed.

Poels *et al*[30] used a PM that measured respiratory movement, air flow (thermistor), heart rate, oxygen saturation and body position to evaluate 24 children aged 2 to 7 years and candidates for adenotonsillectomy. This study was meant to reflect daily practice, as the placement of the sensors was done by the caregivers. The definition criteria for a successful recording was those with 390 minutes of artifact-free signal present in 3 traces simultaneously within the TST. Only 7 out of 24 exams fulfilled this criteria. Another limitation was that from the initial 53 eligible children, only 45% of the caregivers agreed to participate in the study.

Rosen *et al*[35] compared type III PM with laboratory PSG in 55 children with 8 to 11 years, and found that PM had a 88% sensibility and 98% specificity to diagnose OSA, using a criteria of AHI > 5.

Hamada e Iida[36] evaluated the effects of adenotonsillectomy in 48 children aged 2 to 11 years, using a home type III PM. The AHI decreases from 20.6 ± 16.6 to 4.4 ± 2.1/h. However, there were no significant improvement of oxygen saturation nadir (76.7 ± 17.1 e 80.8 ± 14.6%). Complete cure was noted in 75.6% of the children. The author recognized that oxygen saturation values were inconsistent, probably due to an adult size of the probe, not appropriate for children.

A more recent study[34], evaluated, for the first time simultaneously, the use of PM and PSG in 53 children aged 2 to 14 years with clinical suspicious of sleep apnea-hypopnea syndrome (SAHS). SAHS was defined by an obstructive apnea-hypopnea index (OAHI) of 3 or more in PSG and a RDI of 3 or more in PM. The rate of diagnostic agreement between the two methods was 84.9%. The authors concluded that PM is a valid method for diagnosis of SAHS in children.

Sárdon-Prado *et al*[37] compared type III PM in two different setting: home and laboratory, in 44 (8.3 years) and 88 (7.3 years) children. Both groups had 96.5% valid studies. The diagnosis were similar in both groups, however the study design did not allowed sensibility/specificity calculations, and there were no comparison with a gold standard (Type I sleep study).

Amorin *et al*[38] performed type III PM in 33 children, aged 10.6 ± 3.4 years, with different risk factors (adenotonsillar hypertrophy, obesity, craniofacial malformation and neuromuscular disease). Authors found 94% of valid results, adequate nasal airway in 67.7%, and adequate oxygen saturation in 96.8%. There was no comparison with a gold standard.

***Clinical implications***

Adenotonsillar hypertrophy, the most common cause of OSA in children, is a treatable condition and adenotonsillectomy is associated with improvements in quality of life, behavior and sleep parameters[27,39]. The intermittent hypoxia and arousals during the night are associated with EDS, poor academic performance, hyperactivity attention deficits and may result in behavioral and neurocognitive morbidity. Furthermore, OSA affects the metabolic and cardiovascular systems: obesity, pulmonary hypertension and systemic hypertension are more prevalent in children with SDB[27,40]. Therefore, an earlier diagnosis of OSA is mandatory in order to stablish the appropriate treatment and diminish the impact of SDB during children’s development.

**CARDIOVASCULAR DISEASE**

***Physiopathological background***

SDB are highly prevalent in patients with heart failure (HF). Whilst obstructive sleep apnea affects 53% of HF patients[41]; central apnea, in particularly Cheyne-Stokes respiration, is present in 15% to 50% of these population[41-43]. Over the last few years, studies have demonstrated the paramount role of nocturnal rostral fluid shift in the pathogenesis of SDB[44–46]. Fluid shift from the legs to the neck increase neck circumference and airway collapsibility resulting in obstructive sleep apnea. On the other hand, fluid shift to the lungs accentuate the hyperventilation already present in HF patients and reduces PCO2  below the threshold require to stimulate breathing, which results in central sleep apnea.

Furthermore, SDB affects patients with cerebrovascular disease. A meta-analysis of 2343 patients ischemic or hemorrhagic stroke and transient ischemic attack demonstrated a prevalence of SDB with AHI > 5 of 72%[47] which may be even higher in patients with acute stroke.

***Technical considerations***

Both central and obstructive apneas may be present at the same patient with cerebro-cardiovascular disease and exhibit a variation from night to night[48,49]. The overnight shift from one type of apnea to another is related to the PCO2 and the cycle length of periodic breathing which reflect cardiac function. Therefore, it is important to differentiate the type of apnea in order to offer adequate treatment for patients.

***Clinical evidence of the use of PM in cardiovascular diseases***

Quintana-Gallego *et al*[50] conducted the first study that compared PSG and unattended PM for the diagnosis of OSA in HF. A total of 75 patients, with left ventricular ejection fraction ≤ 45%, were randomly assigned for two sleep studies: PM at home and PSG. The failure rate was 9%. The majority of patients 59/75 were correctly classified with PM, with a diagnostic accuracy of 78.6%. The diagnostic accuracy of PM varied between 78% to 84%, with high sensitivities (68% to 94%) and high specificities (88% to 97%) considering AHI thresholds of 5, 10 and 15. An important finding was that the PM was able to detect different patterns of apnea (central and obstructive) in 100% of cases.

The Sleep Events, Arrhytmias, and Respiratory analysis in congestive heart failure (SEARCH) study[51] evaluated the use of a cardiorespiratory system that records 2 leads of ECG, pulse oximetry and respiratory impedance [Clear Path System (CPS), Nexan Inc., Alpharetta, GA] in 50 patients with HF and LVEF ≤ 35%. The study was performed in two nights: (1) simultaneous monitoring of PSG and CPS and (2) CPS at home. Failures of recording were due to technical errors with CPS, severe pacemaker interference and premature patient withdrawal. Predictive accuracy for RDI thresholds of 5, 10 and 15 ranged from 73% to 75% in the first night. When comparing CPS between nights, the same RDI event rates were observed in 69% to 80% of sessions for the different cutoffs. The majority of patients (74%) preferred home testing to PSG in the sleep laboratory.

Smith *et al*[52] compared simultaneous monitoring of PM and PSG to PM at home in 20 patients with HF and LVEF < 45%. Although there was a good agreement in outcomes between PM at the same night as PSG (kappa coefficient 0.63, *P* < 0.001), the agreement between PM and PSG measured in different nights was poor (kappa coefficient 0.27, *P* = 0.06). A negative result of PM of home was not able to exclude the diagnosis of SDB. The authors reported several possible explanations for the different results: diverse calculation for RDI (PSG measures RDI during TST, whilst PM measures RDI during total time in bed); night-to-night variation in respiratory events[53]; the small sample size; the amount of time spent supine and poor sleep efficiency in HF patients which may underestimate the event rate by PM.

OSA is implicated in the progression of coronary artery disease (CAD)[54–56]. Oxidative stress, endothelial disfunction, inflammation, impairment of coagulation factors and increased levels of soluble adhesion molecules are features of OSA and predispose to atherosclerosis. Therefore, an early diagnosis in this population is essential to prevent the consequents of CAD: angina, acute coronary syndrome and cerebrovascular accident. Takama *et al*[57] evaluated PSG and PM performed at the same night. The PM (Morpheus; Compumedics, Melbourne, Australia) measured airflow (thermistor), oxygen saturation, heart rate and respiratory movements (chest and abdominal bands). The specificity and sensitivity of diagnosing SDB using PM were 865 and 81% respectively. However, there was a tendency for higher AHI values obtained by PSG in comparison to PM.

Danzi-Soares *et al*[58] evaluated 79 patients with severe CAD referred for coronary artery bypass grafting. PSG and PM were performed on separate nights before surgery. The PM (Stardust II, Respironics Inc., Murrysville, Pennsylvania, United States) was performed at the hospital, as an unattended sleep study. The failure rate was 11% due to computer malfunction, problems with pulse oximetry, nasal cannula and data corrupted during download process. The agreement between PM and PSG was 73%, with PM underestimating AHI in 23% and overestimating in 4% of the cases.

***Clinical implications***

# The impact of OSA on primary and secondary prevention of cardiovascular disease is under evaluation on two large prospective trials that aim to provide better understanding of the use of CPAP in OSA[59,60]. The sleep apnea cardiovascular endpoints (SAVE) study[59] was designed to determine whether CPAP treatment can reduce the incidence of serious cardiovascular events in patients with established cardiovascular disease. Meanwhile, the impact of CPAP on patients with acute coronary syndrome (ACS) and Nonsleepy OSA: The ISAACC Trial[60], aims to determine if CPAP treatment reduces the incidence of cardiovascular events (cardiovascular death, nonfatal myocardial infarction, nonfatal stroke, hospitalization for heart failure, and hospitalization for unstable angina or transient ischemic attack) in patients with ACS.

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# CHRONIC OBSTRUCTIVE PULMONARY DISEASE

# *Physiopathological background*

# The coexistence of OSA and chronic pulmonary diseases, especially chronic obstructive pulmonary disease (COPD) is called the overlap syndrome[61–65]. Several physiopathological features of COPD may predispose OSA development: smoking, which results in local upper airway inflammation, chronic use of corticosteroids that may promote truncal obesity and upper airway edema and poor sleep quality. Moreover, the worsening of hypoxemia during the night, especially during rapid eye movement (REM) sleep. As described by Douglas *et al*[64]: “The major cause of REM hypoxemia is hypoventilation, with additional contributions from alteration in ventilation/perfusion matching and functional residual capacity (FRC) reduction”. Intermittent hypoxemia during the night is associated with an increase in pulmonary hypertension and cardiovascular events in this population.

# *Technical considerations*

# The basis of noninvasive SpO2 measurements relies on pulsatile blood flow and is affected by poor peripheral arterial blood flow[66]. Furthermore, COPD patients often present anemia and changes in hemoglobin structure such as carboxyhemoglobinemia that may affect oxygen saturation acquisition. During PSG, the monitoring equipment may cause sleep disturbances such as arousals and body movement artifacts which interfere on the pulse signal[67]. Although the limited number of signals measured in PM could minimize this effect, the accuracy of oximetry signal in these devices is usually lower than in PSG and the possible incorrect placement of the sensor at home environment might result in sensor disconnection during the recording time.

# *Clinical evidence of the use of PM in COPD*

# The only study that evaluated the use of PM in COPD patients presented negative results[68]. Oliveira *et al*[68] evaluated 72 patients with COPD in Global Initiative for Chronic Obstructive Lung Disease (GOLD)’s stages II and III. Patients were randomly assigned for PSG and PM at the same night at the sleep laboratory and for PM at home. Only 26 patients could be included in the final analysis due to several reasons for recording losses: oximetry and air flow measurements, problems with download and patient exacerbation. The total loss rate was 61%. The authors concluded that there is insufficient data to support the use of PM in this population.

# *Clinical implications*

Many studies have demonstrated a higher survival in overlap syndrome in patients treated with CPAP[69–73]. Machado *et al*[69] compared the surveillance of patients with overlap syndrome treated with CPAP to patients that did not accept the treatment or were not adherent. Kaplan-Meier survival curves indicated significantly higher survival in CPAP-treated patients compared to the nontreated group (*P* < 0.0001), with 5 year survival estimates of 71% and 26%, respectively. The benefits of CPAP are associated with improvement in respiratory mechanics and a reduction in cardiovascular morbidity and mortality.

**OBESITY**

# *Physiopathological background*

# Obesity remains the main risk factor for the development of OSA and is the most important reversible risk factor. Obesity results in increases in neck circumference and fat deposition around the upper airways, tongue, soft palate and uvula. Moreover, a combination of increased abdominal fat mass and the recumbent position during sleep determines a reduction in FRC[74,75]. In morbidly obese patients the FRC may become so marked that the FRC presents values close to residual volume. The decrease in FRC also affects the ventilatory control system[76]. Hormonal changes are also implicated in the development of OSA in obesity. Leptin is a hormone responsible for the control of appetite and has inhibitory effects in respiratory drive. Its levels are elevated in obesity, suggesting a leptin resistance.

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# *Technical considerations*

The pattern of ventilation in obese individuals is characterized by increased work of breathing, higher respiratory rate compared to lean subjects and lower respiratory volumes. Taken together, all these factors can have an impact on the accuracy of measurements from airflow, respiratory and abdominal movements. Although the adipose tissue and poor tissue perfusion may prevent adequate penetration of light, Dumitrache-Rujinski *et al*[77] compared a group of obese patients to morbid obese patients (BMI > 40 kg/m2) and concluded that the use of oximetry is accurate in both groups. It is also important to use belts with the right size, in order to minimize any discomfort during the exam.

***Clinical evidence of the use of PM in obesity***

Lesser *et al*[78] evaluated the utility of PM for screening OSA in 29 obese (BMI ≥ 95th percentile for age/gender) healthy subjects between 9 and 18 years old with high pre-test probability of SDB. The study consisted of a simultaneous performance of PSG and PM. The Apnea Link Plus (PesMed Corporation, Poway, CA) is a type III of PM that records nasal airflow, respiratory effort, pulse rate, and pulse oximetry. The sensitivity of PM in diagnosing OSA was 100% at obstructive apnea hypopnea index (OAHI) > 1.5; 85.7% at OAHI > 5; and 100% at OAHI > 10. The specificity of the PM increased at higher cutoffs, with the best results when an OAHI cutoff of 10 was used.

Fredheim *et al*[79] compared the simultaneous use of Apnea Link (ResMed Corporation, Poway, California, United States) to Embletta. A total of 105 subjects, with a mean BMI of 43.6 were included in the study. The sensitivity of the PM was 93% for an AHI cutoff 5 events/h and 94% for an AHI cutoff 15 events/h. The specificity was 71% for an AHI cutoff 5 events/h and 94% for an AHI cutoff 15 events/h. The authors concluded that the PM has a high diagnostic accuracy for diagnosing OSA in obese patients.

Oliveira *et al*[80] enrolled 58 obese patients candidates for bariatric surgery in a study that performed simultaneous sleep studies with PM (Stardust II, Philips Respironics, Inc. United States) and PSG and PM at home. Although the results demonstrated good agreement between methods analysed by Bland-Altman graphics, intraclass correlation coefficients and kappa coefficients, the failure rate was 45%. Inadequate flow recordings, oximetric recordings, problems with downloading data and the failure of a patient to start the device were responsible for these losses, mostly when PM was performed at home.

***Clinical implications***

In patients with obesity candidates for bariatric surgery, the treatment of OSA with CPAP before and following the surgery is safe and minimizes the risk of respiratory complications such as pulmonary atelectasis and pneumonia[81,82]. After surgery, weight loss determines changes in CPAP pressure requirements and these patients should be closely followed especially in the first years after the procedure.

**CONCLUSION**

Full-night PSG remains the gold standard for the diagnosis of OSA**.** However, the use of unattended PM is an adequate alternative for earlier recognition of sleep disturbances. There is growing evidence of its importance particularly in patients with comorbidities (Table 3). Specific population, as described in this review, may present even higher prevalence of OSA than the general population; difficulties to attend to a sleep laboratory and lower tolerability to use the equipment required for PSG. Furthermore, the presence of OSA is associated with higher morbidity and mortality in these patients. Therefore, although there is no standardization of PM devices, the majority of studies have shown that the use of PM is safe, has good agreement with PSG and may be used not only for the diagnosis of OSA, but also for evaluation of autotitration at home and response after treatment.

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**Table 1 Sleep evaluation studies for the diagnosis of sleep disordered breathing**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
|  | **Type 1**  **full attended polysomnography** | **Type 2**  **full unattended polysomnography** | **Type 3**  **limited channels devices** | **Type 4**  **continuous single or dual bioparameter recording** |  |
| **Number of channels** | Minimum 7 | Minimum 7 | Minimum 4 | Minimum of 1 |  |
| **Parameters** | EEG, EOG, chin EMG, ECG, airflow, respiratory effort, oxygen saturation | EEG, EOG, chin EMG, ECG, airflow, respiratory effort, oxygen saturation | Ventilation (at least two channels of respiratory movement or respiratory movement and airflow), heart rate or ECG, oxygen saturation | Usually oxygen saturation and/or airflow |  |
| **Body position** | Documented or objectively measured | May be objectively measured | May be objectively measured | Not measured |  |
| **Leg moviment** | EMG or motion sensor desirable | EMG or motion sensor desirable | May be recorded | Not recorded |  |
| **Personnel** | In constant attendance | Not in attendance | Not in attendance | Not in attendance |  |

EEG: Electroencephalogram; EOC: Electrooculogram; EMG: Electromyogram; ECG: Electrocardiogram. Adapted from Ferber *et al*[8].

**Table 2 Scoper categorization**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Sleep** | **Cardiovascular** | **Oximetry** | **Position** | **Effort** | **Respiratory** |
| S1 – 3 EEG channels**a** + EOG + chin EMG | C1 – More than 1 ECG lead | O1 – Oximetry (ear or finger) with recommended sampling**b** | P1 – Video or visual position measurement | E1 – Two RIP belts | R1 – Nasal pressure and thermal device |
| S2 – Less than 3 EEG channels with/without EOG or chin EMG | C2 – Peripheral arterial tonometry | O1x – Oximetry (ear or finger) without recommended sampling | P2 – Nonvisual position measurement | E2- One RIP belt | R2 – Nasal pressure |
| S3 – Sleep surrogate: *e.g.,* actigraphy | C3 – Standard ECG measure  ( one lead) | O2 – Oximetry with alternative site |  | E3 – Derived effort | R3 – Thermal device |
| S4 – Other sleep measure | C4 – Derived pulse | O3 – Other oximetry |  | E4 – Other effort measures | R4 – End-tidal CO2 |
|  | C5 – other cardiac measures |  |  |  | R5 – Other respiratory measures |

EEG: Electroencephalogram; EOC: Electrooculogram; EMG: Electromyogram; RIP: Respiratory inductance plethysmography; **a**Frontal, central and occipital; **b**3 s averaging and a minimum of 10 Hz sampling rate. Adapted from Collop *et al*[9].

**Table 3 Summary of manuscripts of the use of portable monitoring in specific population**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Ref.** | **Year** | **Population** | **Sample Size** | **Device** | **Protocol** |  | **Protocol** |
| Morales *et al*[24] | 2012 | Elderly  mean age 71.4 yr | 452 | Rescare AutosetTM (ResMed, Sydney, Australia) | PM at home  PSG at sleep lab |  | PM at home  PSG at sleep lab |
| Polese *et al*[22] | 2013 | Elderly  > 65 yr | 43 | Stardust II ® (Phillips Respironics, Inc., Murrysville, PA, USA) | PM home  PM + PSG at sleep lab |  | PM home  PM + PSG at sleep lab |
| Goodwin *et al*[31] | 2001 | Children  5 to 12 yr | 157 | Compumedics PS-2 System (Abbotsford, Victoria, Australia) | PM at home |  | PM at home |
| Zucconi *et al*[32] | 2003 | Children  3 to 6 yr | 12 | POLY-MESAM; MAP; Martinsried, Germany | PSG at sleep lab  PM at sleep lab |  | PSG at sleep lab  PM at sleep lab |
| Poels *et al*[30] | 2003 | Children  2 to 7 yr | 24 | Embletta PDS; Flaga hf Medical Devices, Reykjavik, Iceland) | PM at home |  | PM at home |
| Rosen *et al*[35] | 2003 | Children  8 to 11 yr | 55 | PT-2 System SensorMedics, Yorba Linda, California, United States | PM home  PSG lab |  | PM home  PSG lab |
| Alvarez *et al*[34] | 2008 | Children  2 to 14 yr | 53 | Edentec Monitoring System (Edentrace II, Model 3711; Edentec Corporation, Nellcor Puritan Bennett, Eden Prairie, Minnesota, United States) | PSG + PM at sleep lab |  | PSG + PM at sleep lab |

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| Sardón-Prado *et al*[37](abstract) | 2006 | Children | 132 | Not informed | PM at hospital  PM at home |
| Amorim *et al*[38] (abstract) | 2004 | Children  Mean age 10.6 yr | 31 | Not informed | PM at home |
| Quintana-Gallego *et al*[50] | 2004 | Heart failure  LVEF ≤ 45% | 75 | Apnoescreen II; Erich Jaeger Gmbh and CoKg, Wuerzburg, Germany | PSG at sleep lab  PM at home |
| Abraham *et al*[51] | 2006 | Heart failure  LVEF ≤ 35% | 50 | ClearPath System Nx-301; Nexan Inc, Alpharetta, GA | PSG + PM at sleep lab  PM at home |
| Smith *et al*[52] | 2007 | Heart failure  LVEF ≤ 45% | 20 | Embletta; Flaga, Iceland | PSG + PM at sleep lab  PM at home |
| Takama *et al*[57] | 2010 | CAD patients | 83 | Morpheus; Compumedics, Melbourne, Australia | PSG + PM at sleep lab |
| Danzi-Soares *et al*[58] | 2011 | CAD patients | 79 | Stardust II, Respironics Inc., Murrysville, Pennsylvania, United States | PSG + PM at sleep lab |
| Oliveira *et al*[68] | 2012 | COPD patients | 72 | Stardust II ® (Phillips Respironics, Inc., Murrysville, PA, United States ) | PSG + PM at sleep lab  PM at home |
| Lesser *et al*[78] | 2012 | Obese pediatric patients  (9-18 yr ) | 25 | ApneaLink Plus (PesMed Corporation, Poway, CA) | PSG + PM at sleep lab |

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| Fredheim *et al*[79]  (abstract) | 2014 | Morbidly obese patients | 105 | ApneaLink | PM  PM + Embletta |
| Oliveira *et al*[80] | 2014 | Obese patients | 58 | Stardust II ® (Phillips Respironics, Inc., Murrysville, PA, United States) | PSG + PM at sleep lab  PM at home |

PM: Portable monitoring; PSG: Polysomnography; LVEF: Left ventricle ejection fraction; CAD: Coronary artery disease; COPD: Chronic obstructive pulmonary disease.