Sleep Breath (2013) 17:125–132
DOI 10.1007/s11325-012-0660-z

ORIGINAL ARTICLE

Actigraphic assessment of sleep in chronic obstructive pulmonary disease

Deuzilane M. Nunes · Veralice M. S. de Bruin · Fernando M. Louzada · Carina A. T. Peixoto · Antônio G. M. Cavalcante · Cláudia Castro-Silva · Pedro F. C. de Bruin

Received: 24 May 2011 / Revised: 22 November 2011 / Accepted: 23 January 2012 / Published online: 16 February 2012

Abstract
Purpose Previously, sleep in chronic obstructive pulmonary disease (COPD) has been objectively investigated only by lab-based polysomnography. The main purpose of this study was to evaluate sleep quality in COPD patients in their home environment using actigraphy. We also investigated the factors associated with sleep impairment and the relationship between objective and subjective sleep quality and daytime somnolence in these patients.
Methods Twenty-six patients with moderate to very severe COPD and 15 controls were studied by actigraphy for at least 5 days. Subjective sleep quality was evaluated by the Pittsburgh Sleep Quality Index and daytime sleepiness by the Epworth Sleepiness Scale (ESS). Dyspnea was quantified by the modified Medical Research Council (MMRC) scale.
Results COPD patients showed increased sleep latency (p=0.003), mean activity (p=0.003), and wake after sleep onset (p=0.003) and reduced total sleep time (TST; p=0.024) and sleep efficiency (p=0.001), as compared to controls. In patients, severity of dyspnea was correlated with sleep activity (r=0.41; p=0.04) and TST (r=−0.46; p=0.02) and multiple regression analysis showed that MMRC score was the best predictor of TST (p=0.02) and sleep efficiency (p=0.03). Actigraphy measures during daytime were not significantly different between patients and controls. Subjective sleep quality was poorer in patients than controls (p=0.043). ESS scores were not significantly different between the two groups. Actigraphy measures were not correlated with subjective sleep quality or daytime somnolence in both groups.
Conclusions Nocturnal sleep is markedly impaired in stable COPD patients studied by actigraphy in their home environment and this impairment is related to severity of dyspnea.

Keywords Actigraphy · Chronic obstructive pulmonary disease · Daytime somnolence · Dyspnea · Pittsburgh Sleep Quality Index · Sleep

D. M. Nunes · V. M. S. de Bruin · A. G. M. Cavalcante · C. Castro-Silva · P. F. C. de Bruin (✉)
Department of Medicine, Universidade Federal do Ceará, Rua Prof. Costa Mendes, 1608 4° Andar, CEP 60430-040, Fortaleza, Ceará, Brazil
e-mail: pfelipe@superig.com.br

F. M. Louzada · C. A. T. Peixoto
Department of Physiology, Universidade Federal do Paraná, Curitiba, Paraná, Brazil

Introduction

Disturbed sleep is a common feature of chronic obstructive pulmonary disease (COPD) and contributes to impaired quality of life in this condition [1]. Over 50% of patients with COPD report long sleep onset latency, frequent arousals during the night, and/or general insomnia [2]. Sleep-related complaints are ranked third in frequency, behind dyspnea and fatigue, by these patients [3]. Analysis of a large COPD outpatient database revealed that 21.4% of the COPD patients were diagnosed and/or treated for insomnia as compared to only 7.2% of non-COPD patients [4]. Sleep problems in COPD are probably secondary to multiple factors and tend to increase with disease progression [5]. Although the effects of disturbed sleep on daytime function have not been directly assessed, they could play a substantial role in daytime symptoms and chronic fatigue commonly found in these patients [6].

Previous studies on sleep in COPD have relied on subjective information gathered from self-administered

questionnaires [7, 8] or on objective measures from lab-based polysomnography [9, 10]. Presently, polysomnography is regarded as the gold standard test for the objective evaluation of sleep, but it is expensive and time consuming. Actigraphy is a simple tool used to assess the sleep–wake cycle. An actigraph is a small device worn around the wrist that detects movement. It provides an estimate of time asleep based on the marked decrease in activity that occurs while a subject is sleeping. Actigraphy is much less expensive than polysomnography and allows for prolonged objective monitoring of sleep, lasting from days to weeks. Actigraphy data correlate well with polysomnography, with reliability coefficients ranging from 0.89 to 0.98 for normal sleep [11–13]. Actigraphy can also help to identify sleep problems in specific conditions, such as schizophrenia [13], Alzheimer's disease [14], bipolar disorder [15] and lung cancer [16], and in patients with motor handicaps [17]. To our knowledge, there have been no previous studies using actigraphy for the assessment of sleep in ambulatory patients with COPD. The main purposes of this study were to characterize sleep abnormalities in COPD patients in their home environment using actigraphy and to compare these actigraphy findings with those of normal healthy adults. We also investigated the main factors associated with sleep impairment and the relationship between objective and subjective sleep quality, daytime activity levels, and daytime sleepiness in these patients.

Material and methods

Subjects

Twenty-six patients with clinically stable COPD were consecutively recruited from the Respiratory Outpatient Clinic at the University Hospital of the Federal University of Ceará, Brazil. Inclusion criteria for the study group were age 40 years or older, medical diagnosis of COPD stage II to IV according to the GOLD criteria [18], positive smoking history, and a clinically stable condition, as assessed by the attending physician. Patients who had a history of disease exacerbation 4 weeks prior to the study or were currently on oral steroids, hypnotic-sedative medication, or nocturnal oxygen therapy were excluded. Fifteen age-matched non-smoking subjects without evidence of pulmonary disease or other serious chronic medical condition, recruited among patients' caregivers and family members, were studied as controls. None of the participants in the study were involved in shift-work or had any serious disability apart from COPD. The study protocol was approved by the local Research Ethics Committee (COMEPE 209-08) and written informed consent was obtained in all cases.

Study design and measurements

This was a cross-sectional study of the sleep–wake cycle in patients with moderate to very severe COPD and control subjects, assessed by wrist actigraphy in their home environment. Lung function was evaluated by spirometry [18, 19]. The severity of dyspnea was quantified by the modified Medical Research Council (MMRC) dyspnea scale, an ordinal scale based on degree of physical activity that precipitates dyspnea. Scores range from 0 (only gets breathless with strenuous exercise) to 4 (too breathless to leave the house or becomes breathless when dressing or undressing). The MMRC scale is the most commonly used instrument to measure dyspnea during activities of daily living, because of its simplicity, ease of administration, and validation in COPD [20, 21].

Individuals completed a sleep log and wore an activity monitor (Motionlogger, Ambulatory Monitoring Inc., Ardsley, NY, USA) on their non-dominant wrist for five to seven consecutive 24-h periods, including a weekend, to record activity levels at 1-min intervals (zero crossing mode). Activity data were used to calculate (Action W-2 software; Ambulatory Monitoring) the following parameters: sleep onset time (the first of at least three consecutive minutes with an activity frequency count of 0); sleep offset time (the final activity frequency count of 0 before waking in the morning); total sleep time (TST; sleep duration minus the sum of the durations of all awakenings); mean activity level; sleep latency (time in minutes to sleep onset); wake after sleep onset (WASO; total time awake after the first sleep onset period); percent sleep (percent minutes scored as sleep); and sleep efficiency (TST/sleep duration×100). The bedtime and wake time from subject's daily sleep log were used to guide the analysis of the actigraph data recordings.

Subjective sleep quality was assessed by the Pittsburgh Sleep Quality Index (PSQI), a seven component scale, each one dealing with a major aspect of sleep. Components are weighted equally on a 0–3 scale, with a global score ranging from 0 to 21. A PSQI score above 5 has a sensitivity of 89.6% and specificity of 86.5% in differentiating good from poor sleepers [22]. Daytime somnolence was evaluated by the Epworth Sleepiness Scale (ESS), a validated questionnaire that measures subject's expectation of dozing in eight hypothetical situations. Dozing probability ratings range from 0 (none) to 3 (high probability). A total score of 10 or more was considered to be indicative of excessive daytime sleepiness [23, 24].

Statistical analysis

Analyses were carried out by the Statistical Package for Social Sciences V16.0 (SPSS Inc, Chicago, IL, USA). Data are reported as mean±SD and the level of significance was

set at $p<0.05$. For actigraphy variables, mean values from 5 to 7 days were obtained for each participant and then a mean for the group (COPD vs. control) was calculated. Data were examined for normality using the Kolmogorov–Smirnov test. For normally distributed variables with homogeneity of variance, unpaired Student's test was performed. For those variables that did not meet the homogeneity of variances requirement, nonparametric Mann–Whitney test was used. Chi-square test was used to compare COPD and controls regarding the presence of poor sleep quality (PSQI>5), excessive daytime sleepiness (ESS≥10), and low objective sleep efficiency (TST/sleep duration<85%). Pearson correlation coefficients were calculated to evaluate the relationship between actigraphy measures, PSQI scores, ESS scores, and clinical variables. Spearman's correlation coefficients were used in order to express the relationships between MMRC and other variables. Stepwise multiple regression was used to evaluate how variables predicted the following sleep actigraphy measures: TST, sleep efficiency, sleep onset latency, and WASO. Variables considered as potential predictor factors were MMRC score, age, body mass index (BMI), and forced expiratory volume in 1 s (FEV1).

Results

Demographic and clinical characteristics of 26 patients with COPD and 15 controls are shown in Table 1. COPD was classified as moderate (stage II) in 13 patients (50.0%), severe (stage III) in ten (38.5%), and very severe (stage IV) in three (11.5%). Twenty-five patients reported regular use of pulmonary medication, including long-acting beta-2 agonists (n=20), short-acting beta-2 agonists (n=9), inhaled steroids (n=6), anticholinergics (n=4), and methylxantines (n=4). Four COPD patients (15.4%) were current smokers. Fifteen patients (57.7%) and five controls (33.3%) were retired.

Subjective sleep quality assessment showed that 15 patients with COPD (57.7%) and four controls (26.7%) were poor sleepers (p=0.055). On average, PSQI score was significantly worse in the COPD group than in controls (p=0.043). Excessive daytime sleepiness (ESS≥10) was detected in ten patients and two controls. Mean ESS score was not significantly different between the COPD and control groups (8.27 vs. 6.07, p=0.12) (Table 1).

Actigraphy data are summarized in Table 2. During nighttime, the COPD group showed significantly increased sleep latency (39.49 vs. 11.19, p=0.003), mean activity (42.1 vs. 21.2, p=0.003), and WASO (96.09 vs. 49.26, p=0.003) and reduced TST (280.84 vs. 360.15, p=0.024) and sleep efficiency (73.1 vs. 87.7, p=0.001) as compared to controls. During daytime, we were unable to demonstrate any significant differences in actigraphy variables between the two groups.

Table 1 Demographic and clinical characteristics, subjective quality of sleep and daytime sleepiness of 26 patients with COPD and 15 normal controls

	COPD	Controls	p
Age (years)	66.96±8.54	63.00±10.66	0.20[a]
Men/women	19/7	8/7	0.20
BMI (kg/m^2)	24.81±3.92	24.38±2.55	0.70[a]
Smoking history (pack-years)	54.36±32.28	–	–
FEV1 % predicted	47.62±16.04	–	–
COPD stage (II/III/IV)	13/10/3	–	–
MMRC (0/1/2/3/4)	2/8/9/5/1	–	–
PSQI global score	6.96±3.5	4.80±2.4	0.043[a]
Poor quality sleep (PSQI>5) No/yes	11/15	11/4	0.055[b]
ESS	8.27±4.4	6.07±3.9	0.12[a]
Daytime somnolence (ESS≥10) No/yes	16/10	13/2	0.09[b]

BMI body mass index, *FEV1* forced expiratory volume in 1 s, *MMRC* modified Medical Research Council dyspnea scale; *COPD stages*: II—moderate, III—severe, and IV—very severe; *PSQI* Pittsburgh Sleep Quality Index, *ESS* Epworth Sleepiness Scale

[a] Unpaired Student's *t* test

[b] Chi-square test

Results from correlation analyses between actigraphy measures, subjective sleep quality, daytime somnolence, and demographic and clinical characteristics are presented in Table 3. The COPD group showed a negative correlation between age and nighttime sleep onset (r=−0.43; p=0.03) and a trend for a positive correlation between age and daytime percent sleep (r=0.36; p=0.07). During nighttime, MMRC dyspnea score was positively correlated with mean activity (r=0.41; p=0.04) and negatively correlated with TST (r=−0.46; p=0.02). No significant correlations were found between the degree of airway obstruction, as assessed by post-bronchodilator percent predicted FEV1, and actigraphy variables. In controls, there was a negative correlation between age and both sleep onset (r=−0.68; p=0.005) and sleep efficiency (r=−0.57; p=0.03) during nighttime and a positive correlation between age and mean activity (r=0.62; p=0.01) and WASO (r=0.61; p=0.02) during nighttime. There was no correlation between PSQI score and actigraphy variables, both in patients and controls. No correlations were also found between PSQI score and MMRC dyspnea score or percent predicted FEV1. In the COPD group, there were no correlations between ESS scores and actigraphy-derived parameters, while a positive correlation was found between ESS and age (r=0.67; p<0.001). No significant correlations were demonstrated between ESS and PSQI

Table 2 Actigraphy variables in 26 patients with moderate to very severe COPD and 15 normal controls

	COPD	Controls	*p*
Actigraphy variables—nighttime			
Sleep onset time (h:min)	22.37±1.06	23.03±0.43	0.178[a]
Sleep offset time (h:min)	05.39±0.57	05.59±1.04	0.295[a]
Total sleep time (min)	280.84±110.6	360.15±67.7	0.024[b]
Mean activity (accel/min)	42.1±24.1	21.2±13.7	0.003[b]
Sleep latency (min)	39.49±43.5	11.19±14.6	0.003[b]
WASO (min)	96.09±43.5	49.26±44.6	0.003[b]
Sleep efficiency (%)	73.1±13.7	87.7±10.9	0.001[b]
Actigraphy variables—daytime			
Total sleep time (min)	54.01±44.2	49.88±42.1	0.771[a]
% sleep	5.3±4.4	4.9±4.1	0.745[a]
Mean activity (accel/min)	183.96±42.8	200.54±31.1	0.197[a]

WASO wake after sleep onset
[a]Unpaired Student's *t* test
[b]Mann–Whitney test

score or percent predicted FEV1 in COPD patients. In control subjects, ESS was negatively correlated with both sleep offset time (r=−0.62; p=0.01) and TST (r=−0.62; p=0.01) and positively correlated with sleep latency (r=0.66; p=0.01) during nighttime.

Multiple regression analysis was used to identify the independent variables considered as indicators of the effect measured by the actigraphy parameters, in COPD patients. Models were built taking into account problems of confounding factors and colinearity and the following variables were ultimately retained: MMRC score, age, and BMI. The MMRC score explained 17.4% (adjusted R^2) of the overall TST variance and 11.0% of the sleep efficiency variance (Table 4).

Discussion

These results show that nocturnal sleep is markedly impaired in clinically stable patients with moderate to very severe COPD studied by actigraphy in their natural home environment. During nighttime, sleep onset latency was found to be more than three times higher and mean activity and WASO almost twice as high in patients with COPD than in age-matched controls. We are not aware of earlier reports on the use of actigraphy for the evaluation of the sleep–wake cycle in ambulatory patients with COPD. In a pilot study by Shilo and coworkers, eight patients under intensive care with respiratory failure due to exacerbation of chronic bronchitis or pneumonia were assessed by actigraphy for

Table 3 Correlation coefficients for sleep–wake variables derived from actigraphy and demographic and clinical characteristics of 26 patients with COPD and 15 normal controls

	Age		BMI		PSQI global score		ESS		FEV1 % predict	MMRC
	COPD	Controls	COPD	Controls	COPD	Controls	COPD	Controls	COPD	COPD
Nighttime										
Sleep onset time	−0.43*	−0.68*	−0.11	−0.29	−0.04	0.09	−0.04	−0.36	−0.02	0.26
Sleep offset time	−0.28	−0.15	−0.19	−0.27	0.11	0.08	0.12	−0.62*	−0.17	−0.25
Total sleep time	0.17	−0.16	−0.02	−0.24	0.12	0.13	0.25	−0.62*	0.07	−0.46**
Mean activity	−0.11	0.62*	−0.06	0.25	−0.07	−0.14	−0.24	0.34	−0.21	0.41**
Sleep latency	−0.30	0.29	0.09	0.17	−0.17	−0.25	−0.16	0.66*	−0.10	0.32
WASO	0.31	0.61*	−0.18	0.19	0.18	−0.08	−0.005	0.22	0.02	0.29
Sleep efficiency	−0.13	−0.57*	0.15	−0.22	−0.05	0.13	0.11	−0.41	0.01	−0.38
Daytime										
Total sleep time	0.34	0.007	−0.11	−0.17	0.24	−0.10	0.22	−0.26	−0.07	−0.16
% sleep	0.36	0.06	−0.10	−0.16	0.24	−0.09	0.21	−0.28	−0.07	−0.18
Mean activity	−0.17	0.22	0.05	0.16	−0.32	−0.09	−0.03	0.52*	−0.06	0.26

WASO wake after sleep onset, *FEV1* forced expiratory volume in 1 s, *MMRC* modified Medical Research Council
*p<0.05, Pearson correlation; **p<0.05, Spearman correlation

Table 4 Results of the stepwise multiple regression analyses of 26 patients with COPD with actigraphy parameters as dependent variable

Outcome/ predictors	Coefficient	SE	*p* value	Partial *R*	Adjusted R^2
Total sleep time			0		
Constant	380.09	44.67			
MMRC score	−55.50	22.56	0.02	0.21	0.17
Sleep efficiency					
Constant	69.36	17.59	0.001		
MMRC score	−7.57	3.35	0.03	0.22	0.11
Sleep onset latency					
Constant	1,551.38	99.4	0		
MMRC score	16.4	13.6	0.24	0.22	0.15
WASO					
Constant	127.67	58.34	0.04		
MMRC score	13.20	10.14	0.20	0.10	0.01

MMRC modified Medical Research Council, *WASO* wake after sleep onset

three consecutive 24-h periods to measure the beneficial effect of melatonin administration on sleep quality [25].

This is the first study to confirm objectively the prior finding from lab-based polysomnography that COPD patients have marked sleep disruption, including prolonged sleep latency, decreased total sleep time, and increased number of nocturnal arousals [10, 26–30]. The causes of disturbed sleep in COPD are not fully understood. Nocturnal hypoxia may be one cause, due to a combination of low baseline oxygen saturation and alterations in respiratory muscle function and ventilation, particularly during REM sleep [31, 32]. However, it has been estimated that less than 5% of COPD patients exhibit significant nocturnal desaturation [7]. Moreover, studies on the effects of oxygen supplementation on quality of sleep have produced controversial results [3]. Sleep-related complaints in COPD patients are more likely related to a combination of factors. COPD is commonly found in middle-aged or older individuals who have been long-time smokers and may suffer from a variety of conditions associated with smoking or aging. It is recognized that, for the general population, the frequency and severity of sleep complaints increase with age and the same is probably true for patients with COPD [33]. In the present study, older age was related to late sleep onset and more daytime sleep among COPD patients. Gender is another factor that deserves consideration. Healthy women, despite better objective sleep quality measures, present more sleep-related complaints than men [34]. Surprisingly, the literature on the influence of gender on sleep in COPD is scarce, despite the recognition that psychiatric illness, a major cause of disrupted sleep, is twice as common in female patients with COPD [35]. In the present study, no significant gender-related differences in sleep quality were found. This is a complex issue and deserves further investigation.

Some pulmonary medications may also affect sleep. Improvements in sleep quality have been reported after the use of the anticholinergic ipratropium, but not tiotropium, in cases with COPD. Methylxantines have been found to adversely affect sleep in healthy individuals, although studies in COPD patients have not confirmed this negative effect [3]. Nicotine consumption has also been associated with sleep impairment. Increased sleep latency, sleep fragmentation, reduced sleep efficiency, decreased nocturnal oxygen saturation, and increased daytime sleepiness have all been previously described in smokers [36, 37]. In this study, the relatively small number of current smokers precluded a more detailed analysis of this potential effect.

Our results show that dyspnea is an important predictor of sleep quality, as assessed by actigraphy, in patients with moderate to very severe COPD. Dyspnea is the major limiting factor for activities of daily living in COPD, and the severity of dyspnea, as measured by the MMRC scale, correlates well with health status scores and predicts the likelihood of survival [38, 39]. Previously, nocturnal dyspnea has been linked to disturbed sleep in COPD [5]. Dyspnea has also been described as a major determinant of sleep quality in other medical conditions, such as heart failure [40]. A potentially relevant implication of our finding is that better control of dyspnea may lead to sleep improvement in COPD. It has been recognized that optimal management of dyspnea is a frequently neglected aspect in the care of patients with advanced COPD [41]. Conventional management of COPD has focused on treating bronchoconstriction and reducing hyperinflation and airway inflammation, usually with a long-acting anticholinergic agent and a combination of an inhaled long-acting beta agonist and an inhaled corticosteroid. Although these therapies are undoubtedly helpful, as disease progresses through advanced stages, dyspnea becomes refractory in the majority of cases [42]. Pulmonary rehabilitation programs can promote symptom control and, to some extent, improve dyspnea in COPD [43]. Long-term oxygen therapy is frequently prescribed in the later stages of disease for those patients who are significantly hypoxemic. However, its effectiveness in palliating dyspnea has yet to be proven [44]. Combining noninvasive intermittent positive pressure ventilation with long-term oxygen therapy can reportedly improve shortness of breath and carbon dioxide retention in at least some COPD patients [45], although it may worsen quality of life [46]. It has been increasingly suggested that opioids may have a role in the management of COPD patients with dyspnea refractory to conventional treatment, despite some serious adverse effects [47]. The impact of these and other modalities of treatment

on quality of sleep and dyspnea in COPD should be a matter for future studies.

Poor subjective sleep quality was observed in the majority of our patients, in agreement with the actigraphy findings. However, for the COPD group as a whole, we identified no significant correlations between actigraphy variables and subjective sleep quality scores. Cormick and coworkers reported an association between objective measures from polysomnography, including reduced total sleep time and increased arousal index, and subjective complaints of difficulty initiating and maintaining sleep in 16 patients with severe COPD [29]. On the other hand, several investigators have reported discrepancies between subjective data and surrogate markers of sleep and sleep disturbances [48]. Indeed, it has been suggested that objective and subjective assessments reflect different aspects of sleep and therefore may not be necessarily correlated [49]. As long as it is not clear how objective and subjective measures of sleep relate to each other, they should probably both be incorporated into clinical studies [50].

Our analyses of actigraphy data did not show significant differences in activity levels during daytime between COPD patients and controls. Recently, a systematic review of the literature found significant reductions in duration, intensity, and counts of daily physical activity in patients with COPD as compared to normal controls, although correlations between levels of physical activity and severity of disease were weak and/or non-significant [51].

This study has limitations that warrant discussion. First, our subjects were not screened for obstructive sleep apnea (OSA). Because COPD and OSA are both common chronic conditions, they should be expected to occur together, particularly in middle-aged and older male individuals. However, it has been demonstrated that the prevalence of OSA in patients with COPD is similar to that in the general population [52, 53]. It is therefore unlikely that non-exclusion of OSA cases has significantly affected our comparisons between COPD and control groups. On the other hand, this may have contributed, at least in part, to the relatively high daytime sleepiness scores found in our patients, as compared to some previous reports [7, 54]. The same observation may apply to mean PSQI score in our control group, which is similar to the mean score for healthy 80-year-olds in a previous large cohort [55]. It should also be noted that although our control subjects were appropriately matched for age, gender, and BMI, they were not specifically matched for comorbid diseases and medications. Finally, the possibility that poor sleep quality may be a causative factor for the dyspnea observed in our patients, and not the reverse, should be considered. Previously, it has been shown that one night's sleep deprivation can lead to a small but statistically significant reduction in FEV1 and forced vital capacity in a group of patients with COPD [56]. Although this reported spirometric decline was not clinically relevant, it might be speculated that chronic alterations in sleep would result in additive negative effects on respiratory function, which could become relevant along the prolonged course of this disorder. Further long-term studies on the effects of sleep disruption on respiratory function and severity of dyspnea in COPD are needed to investigate this question.

Actigraphy is not a replacement for polysomnography. It should be regarded as another means for assessing sleep in COPD particularly when sleep architecture and extensive physiological monitoring are not considered to be required. In addition, actigraphy can provide information on rest activity patterns, night-to-night variability, and circadian rhythm disorders that cannot be obtained from polysomnography. Presently, polysomnography is not routinely indicated in COPD and current recommendations include suspicion of sleep apnea or complications of hypoxemia that are not explained by the awake arterial oxygen levels, and pulmonary hypertension out of proportion to the severity of pulmonary function derangement [57]. It is obvious from a careful analysis of the available literature and from clinical practice that a significant number of COPD patients who do not meet the above criteria for polysomnography present with sleep problems that require careful investigation and therapeutic intervention. Based on the present results, we suggest that actigraphic assessment should be considered at least for some of these cases.

In summary, clinically stable patients with moderate to very severe COPD studied by actigraphy in their natural home environment show marked impairment of nocturnal sleep. Dyspnea is one of the main factors associated with sleep disruption in these patients. Objective information derived from actigraphy can be relevant for the evaluation and management of COPD patients. When sleep is impaired, treatment of the underlying respiratory condition should be optimized, and if sleep problems persist, available nonpharmacologic and pharmacologic options, particularly aiming to improve dyspnea, should be considered. There is a need for a clearer understanding of the effects of disturbed sleep on various aspects of daytime function in COPD. Actigraphy can become an important tool for the direct assessment of these patients in future studies.

Acknowledgments This work was supported in part by the Research Support Foundation of Ceará—FUNCAP and the National Research Council—CNPq of Brazil.

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Nunes DM, Mota RM, de Pontes Neto OL, Pereira ED, de Bruin VM, de Bruin PF (2009) Impaired sleep reduces quality of life in chronic obstructive pulmonary disease. Lung 187:159–163
2. George CF (2000) Perspectives on the management of insomnia in patients with chronic respiratory disorders. Sleep 23(Suppl 1): S31–35, discussion S36-38
3. Stege G, Vos PJ, van den Elshout FJ, Richard Dekhuijzen PN, van de Ven MJ, Heijdra YF (2008) Sleep, hypnotics and chronic obstructive pulmonary disease. Respir Med 102:801–814
4. Vallarino C, Rajagopalan R, Mini L (2005) Prevalence of insomnia in patients with chronic obstructive pulmonary disease in a large database. Value Health 8(3):322
5. Weitzenblum E, Chaouat A (2004) Sleep and chronic obstructive pulmonary disease. Sleep Med Rev 8:281–294
6. Breslin E, van der Schans C, Breukink S, Meek P, Mercer K, Volz W, Louie S (1998) Perception of fatigue and quality of life in patients with COPD. Chest 114:958–964
7. Lewis CA, Fergusson W, Eaton T, Zeng I, Kolbe J (2009) Isolated nocturnal desaturation in COPD: prevalence and impact on quality of life and sleep. Thorax 64:133–138
8. Nunes DM, Mota RM, Machado MO, Pereira ED, Bruin VM, Bruin PF (2008) Effect of melatonin administration on subjective sleep quality in chronic obstructive pulmonary disease. Braz J Med Biol Res 41:926–931
9. Kryger M, Roth T, Wang-Weigand S, Zhang J (2009) The effects of ramelteon on respiration during sleep in subjects with moderate to severe chronic obstructive pulmonary disease. Sleep Breath 13:79–84
10. Saaresranta T, Irjala K, Aittokallio T, Polo O (2005) Sleep quality, daytime sleepiness and fasting insulin levels in women with chronic obstructive pulmonary disease. Respir Med 99:856–863
11. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollak CP (2003) The role of actigraphy in the study of sleep and circadian rhythms. Sleep 26:342–392
12. Cole RJ, Kripke DF, Gruen W, Mullaney DJ, Gillin JC (1992) Automatic sleep/wake identification from wrist activity. Sleep 15:461–469
13. Martin J, Jeste DV, Caliguiri MP, Patterson T, Heaton R, Ancoli-Israel S (2001) Actigraphic estimates of circadian rhythms and sleep/wake in older schizophrenia patients. Schizophr Res 47:77–86
14. Singer C, Tractenberg RE, Kaye J, Schafer K, Gamst A, Grundman M, Thomas R, Thal LJ (2003) A multicenter, placebo-controlled trial of melatonin for sleep disturbance in Alzheimer's disease. Sleep 26:893–901
15. Jones SH, Hare DJ, Evershed K (2005) Actigraphic assessment of circadian activity and sleep patterns in bipolar disorder. Bipolar Disord 7:176–186
16. Du-Quiton J, Wood PA, Burch JB, Grutsch JF, Gupta D, Tyer K, Lis CG, Levin RD, Quiton DF, Reynolds JL, Hrushesky WJ (2010) Actigraphic assessment of daily sleep-activity pattern abnormalities reflects self-assessed depression and anxiety in outpatients with advanced non-small cell lung cancer. Psychooncology 19:180–189
17. Laakso ML, Leinonen L, Lindblom N, Joutsiniemi SL, Kaski M (2004) Wrist actigraphy in estimation of sleep and wake in intellectually disabled subjects with motor handicaps. Sleep Med 5:541–550
18. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P, Fukuchi Y, Jenkins C, Rodriguez-Roisin R, van Weel C, Zielinski J (2007) Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 176:532–555
19. American Thoracic Society (1995) Standardization of spirometry, 1994 update. Am J Respir Crit Care Med 152:1107–1136
20. Ferris BG (1978) Epidemiology standardization project (American Thoracic Society). Am Rev Respir Dis 118:1–120
21. Camargo LA, Pereira CA (2010) Dyspnea in COPD: beyond the modified Medical Research Council scale. J Bras Pneumol 36:571–578
22. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ (1989) The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 28:193–213
23. Johns MW (1991) A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 14:540–545
24. Bertolazi AN, Fagondes SC, Hoff LS, Pedro VD, Menna Barreto SS, Johns MW (2009) Portuguese-language version of the Epworth sleepiness scale: validation for use in Brazil. J Bras Pneumol 35:877–883
25. Shilo L, Dagan Y, Smorjik Y, Weinberg U, Dolev S, Komptel B, Shenkman L (2000) Effect of melatonin on sleep quality of COPD intensive care patients: a pilot study. Chronobiol Int 17:71–76
26. Calverley PM, Brezinova V, Douglas NJ, Catterall JR, Flenley DC (1982) The effect of oxygenation on sleep quality in chronic bronchitis and emphysema. Am Rev Respir Dis 126:206–210
27. Fleetham J, West P, Mezon B, Conway W, Roth T, Kryger M (1982) Sleep, arousals, and oxygen desaturation in chronic obstructive pulmonary disease. The effect of oxygen therapy. Am Rev Respir Dis 126:429–433
28. Wynne JW, Block AJ, Hemenway J, Hunt LA, Flick MR (1979) Disordered breathing and oxygen desaturation during sleep in patients with chronic obstructive lung disease (COLD). Am J Med 66:573–579
29. Cormick W, Olson LG, Hensley MJ, Saunders NA (1986) Nocturnal hypoxaemia and quality of sleep in patients with chronic obstructive lung disease. Thorax 41:846–854
30. Krachman SL, Chatila W, Martin UJ, Nugent T, Crocetti J, Gaughan J, Criner GJ (2005) Effects of lung volume reduction surgery on sleep quality and nocturnal gas exchange in patients with severe emphysema. Chest 128:3221–3228
31. Krachman S, Minai OA, Scharf SM (2008) Sleep abnormalities and treatment in emphysema. Proc Am Thorac Soc 5:536–542
32. Braghiroli A (2002) Nocturnal desaturations in COPD: still an open question? Sleep Breath 6(1):25–6
33. Bellia V, Catalano F, Scichilone N, Incalzi RA, Spatafora M, Vergani C, Rengo F (2003) Sleep disorders in the elderly with and without chronic airflow obstruction: the SARA study. Sleep 26:318–323
34. Silva A, Andersen ML, De Mello MT, Bittencourt LR, Peruzzo D, Tufik S (2008) Gender and age differences in polysomnography findings and sleep complaints of patients referred to a sleep laboratory. Braz J Med Biol Res 41:1067–1075
35. Laurin C, Lavoie KL, Bacon SL, Dupuis G, Lacoste G, Cartier A, Labrecque M (2007) Sex differences in the prevalence of psychiatric disorders and psychological distress in patients with COPD. Chest 132:148–155
36. Jaehne A, Loessl B, Barkai Z, Riemann D, Hornyak M (2009) Effects of nicotine on sleep during consumption, withdrawal and replacement therapy. Sleep Med Rev 13:363–377
37. Casasola GG, Alvarez-Sala JL, Marques JA, Sánchez-Alarcos JM, Tashkin DP, Espinós D (2002) Cigarette smoking behavior and respiratory alterations during sleep in a healthy population. Sleep Breath 6(1):19–24
38. Nishimura K, Izumi T, Tsukino M, Oga T (2002) Dyspnea is a better predictor of 5-year survival than airway obstruction in patients with COPD. Chest 121:1434–1440
39. Hajiro T, Nishimura K, Tsukino M, Ikeda A, Oga T, Izumi T (1999) A comparison of the level of dyspnea vs disease severity

in indicating the health-related quality of life of patients with COPD. Chest 116:1632–1637
40. Gau FY, Chen XP, Wu HY, Lin ML, Chao YF (2011) Sleep-related predictors of quality of life in the elderly versus younger heart failure patients: a questionnaire survey. Int J Nurs Stud 48(4):419–28
41. Lanken PN, Terry PB, Delisser HM, Fahy BF, Hansen-Flaschen J, Heffner JE, Levy M, Mularski RA, Osborne ML, Prendergast TJ, Rocker G, Sibbald WJ, Wilfond B, Yankaskas JR (2008) ATS End-of-Life Care Task Force. An official American Thoracic Society clinical policy statement: palliative care for patients with respiratory diseases and critical illnesses. Am J Respir Crit Care Med 177(8):912–27
42. Elkington H, White P, Addington-Hall J et al (2005) The health-care needs of chronic obstructive pulmonary disease patients in the last year of life. Palliat Med 19:485–91
43. Nici L, Donner C, Wouters E, Zuwallack R, Ambrosino N, Bourbeau J, Carone M, Celli B, Engelen M, Fahy B, Garvey C, Goldstein R, Gosselink R, Lareau S, MacIntyre N, Maltais F, Morgan M, O'Donnell D, Prefault C, Reardon J, Rochester C, Schols A, Singh S, Troosters T (2006) ATS/ERS Pulmonary Rehabilitation Writing Committee. American Thoracic Society/European Respiratory Society statement on pulmonary rehabilitation. Am J Respir Crit Care Med 173 (12):1390–413
44. Booth S, Wade R, Johnson M et al (2004) The use of oxygen in the palliation of breathlessness. A report of the expert working group of the Scientific Committee of the Association of Palliative Medicine. Respir Med 98:66–77
45. Clini E, Sturani C, Rossi A, Viaggi S, Corrado A, Donner CF, Ambrosino N (2002) Rehabilitation and Chronic Care Study Group, Italian Association of Hospital Pulmonologists (AIPO). The Italian multicentre study on noninvasive ventilation in chronic obstructive pulmonary disease patients. Eur Respir J 20(3):529–38
46. McEvoy RD, Pierce RJ, Hillman D, Esterman A, Ellis EE, Catcheside PG, O'Donoghue FJ, Barnes DJ, Grunstein RR (2009) Australian trial of non-invasive Ventilation in Chronic Airflow Limitation (AVCAL) Study Group. Nocturnal non-invasive nasal ventilation in stable hypercapnic COPD: a randomized controlled trial. Thorax 64(7):561–6
47. Rocker G, Horton R, Currow D, Goodridge D, Young J, Booth S (2009) Palliation of dyspnoea in advanced COPD: revisiting a role for opioids. Thorax 64(10):910–5
48. Argyropoulos SV, Hicks JA, Nash JR, Bell CJ, Rich AS, Nutt DJ, Wilson SJ (2003) Correlation of subjective and objective sleep measurements at different stages of the treatment of depression. Psychiatry Res 120:179–190
49. Vgontzas AN, Bixler EO, Kales A, Manfredi RL, Tyson K (1994) Validity and clinical utility of sleep laboratory criteria for insomnia. Int J Neurosci 77:11–21
50. Zhang L, Zhao ZX (2007) Objective and subjective measures for sleep disorders. Neurosci Bull 23:236–240
51. Vorrink SN, Kort HS, Troosters T, Lammers JW (2011) Level of daily physical activity in individuals with COPD compared with healthy controls. Respir Res 12:33
52. Sanders MH, Newman AB, Haggerty CL, Redline S, Lebowitz M, Samet J, O'Connor GT, Punjabi NM, Shahar E, for the Sleep Heart Health Study (2003) Sleep and sleep-disordered breathing in adults with predominantly mild obstructive airway disease. Am J Respir Crit Care Med 167:7–14
53. Bednarek M, Plywaczewski R, Jonczak L, Zielinski J (2005) There is no relationship between chronic obstructive pulmonary disease and obstructive sleep apnea syndrome: a population study. Respiration 72:142–149
54. McNicholas WT, Calverley PMA, Lee A, Edwards JC (2004) Long-acting inhaled anticholinergic therapy improves sleeping oxygen saturation in COPD. Eur Respir J 23:825–831
55. Buysse DJ, Reynolds CF 3rd, Monk TH, Hoch CC, Yeager AL, Kupfer DJ (1991) Quantification of subjective sleep quality in healthy elderly men and women using the Pittsburgh Sleep Quality Index (PSQI). Sleep 14:331–338
56. Phillips BA, Cooper KR, Burke TV (1987) The effect of sleep loss on breathing in chronic obstructive pulmonary disease. Chest 91:29–32
57. Celli BR, MacNee W (2004) Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. Eur Respir J 23:932–946