



**Universidade de São Paulo**

**Biblioteca Digital da Produção Intelectual - BDPI**

---

Outros departamentos - FM/Outros

Comunicações em Eventos - FM/Outros

---

2012

# Obstructive sleep apnea is common among patients referred for coronary artery bypass grafting and can be diagnosed by portable monitoring

---

CORONARY ARTERY DISEASE, PHILADELPHIA, v. 23, n. 1, supl. 2, Part 3, pp. 31-38, JAN, 2012  
<http://www.producao.usp.br/handle/BDPI/32565>

*Downloaded from: Biblioteca Digital da Produção Intelectual - BDPI, Universidade de São Paulo*

# Obstructive sleep apnea is common among patients referred for coronary artery bypass grafting and can be diagnosed by portable monitoring

Naury de Jesus Danzi-Soares<sup>a</sup>, Pedro Rodrigues Genta<sup>a</sup>, Flávia Baggio Nerbass<sup>a</sup>, Rodrigo Pinto Pedrosa<sup>a</sup>, Flávia Souza Nunes Soares<sup>a</sup>, Luis Antonio Machado César<sup>b</sup>, Luciano F. Drager<sup>a</sup>, Robert Skomro<sup>c</sup> and Geraldo Lorenzi-Filho<sup>a</sup>

**Background** Obstructive sleep apnea (OSA) is common among patients with coronary artery disease. However, OSA remains largely under recognized. The lack of clinical suspicion and difficulties to access full polysomnography (PSG) are limiting factors. The aim of this study was to evaluate, among patients referred to coronary artery bypass grafting (CABG): (i) the prevalence of OSA, (ii) the association of OSA with clinical symptoms, (iii) the performance of overnight unattended portable monitoring (PM) as an alternative method for the diagnosis of OSA.

**Methods** Consecutive patients referred for CABG were evaluated by standard physical evaluation and validated questionnaires (Berlin questionnaire and Epworth Sleepiness Scale) and underwent full PSG and PM (Stardust II).

**Results** We studied 70 consecutive patients (76% men), age  $58 \pm 7$  years (mean  $\pm$  SD), BMI [median (interquartile range)]  $27.6 \text{ kg/m}^2$  (25.8–31.1). The prevalence of OSA (full PSG) using an apnea–hypopnea index of at least 5 events/h was 87%. Commonly used clinical traits for the screening of OSA such as the Epworth Sleepiness Scale and neck circumference had low sensitivities to detect OSA. In contrast, the Berlin questionnaire showed a good

sensitivity (72%) to detect OSA. PM showed good sensitivity (92%) and specificity (67%) for the diagnosis of OSA.

**Conclusion** OSA is strikingly common among patients referred for CABG. The Berlin questionnaire, but not symptom of excessive daytime sleepiness is a useful tool to screen OSA. PM is useful for the diagnosis of OSA and therefore is an attractive tool for widespread use among patients with coronary artery disease. *Coron Artery Dis* 23:31–38 © 2011 Wolters Kluwer Health | Lippincott Williams & Wilkins.

Coronary Artery Disease 2012, 23:31–38

**Keywords:** coronary artery bypass grafting, coronary artery disease, obstructive sleep apnea, polysomnography, portable monitoring

<sup>a</sup>Sleep Laboratory, Pulmonary Division, <sup>b</sup>Clinical Unit of Chronic Coronary Heart Disease, Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil and <sup>c</sup>Department of Medicine, University of Saskatchewan, Saskatoon, Canada

Correspondence to Naury de Jesus Danzi-Soares, RN, PhD, Sleep Laboratory, Pulmonary Division Heart Institute (InCor), University of São Paulo Medical School, Av. Enéas Carvalho de Aguiar, 44 CEP:05403-000, São Paulo, Brazil Tel/fax: +55 (011) 30695486; e-mail: nauryrnurse@gmail.com

Received 12 July 2011 Revised 13 September 2011  
Accepted 26 September 2011

## Introduction

Obstructive sleep apnea (OSA) is characterized by repeated episodes of partial or complete upper airway obstruction during sleep, and is associated with recurrent hypoxemia and arousals [1]. OSA is a public health problem with a prevalence ranging from 4 to 25% in the general population [2,3]. OSA is tightly linked to cardiovascular disease, and the prevalence of OSA is much higher in patients with established cardiovascular disease [4], such as hypertension [5,6], atrial fibrillation [7], and hypertrophic cardiomyopathy [8]. The estimated prevalence of OSA among patients with coronary artery disease (CAD) ranges from 40 to 45% [9–13]. Therefore, patients referred to coronary artery bypass grafting (CABG) may present a high prevalence of OSA. The recognition of OSA in patients with CAD is important since OSA may be associated with increased risk of acute myocardial infarction and stroke [4].

There is also evidence that OSA is independently associated with postoperative complications [14–17].

Despite the potential importance, OSA remains largely under-recognized among patients with established cardiovascular disease [4]. One potential limitation is the recent evidence that several clinical characteristics typical of patients with OSA may not be present in patients with established cardiovascular disease. In contrast to patients referred to sleep laboratories, OSA is not frequently accompanied by excessive daytime sleepiness among patients with hypertension [5], stroke [18], patients with long-term pacing [19], and metabolic syndrome [20]. The lack of typical symptoms may contribute to the low recognition of OSA among patients with established cardiovascular disease. A major limitation is the reduced availability and the high costs of the gold standard method

for OSA, full polysomnography (PSG) [1,21]. Portable monitoring (PM) is a promising alternative for OSA diagnosis [22]. PM is similar to a Holter monitor and precludes the necessity of a sleep technician attending the study. PM offers the advantage of being possible to be applied in multiple settings such as hospital, coronary care unit, intensive care unit, wards, as well as at home. PM has been validated earlier for the diagnosis of OSA among preselected populations referred to sleep laboratories [23–25]. However, the use of PM among populations not triaged by a sleep specialist remains unestablished.

The prevalence of OSA has not been systematically evaluated among patients referred for CABG. We reasoned that these patients undergo an extensive preoperative evaluation and that this would be a window of opportunity to recognize and establish OSA diagnosis. The aim of this study was to determine among consecutive patients referred to CABG: (i) the prevalence of OSA, (ii) the association with clinical symptoms that may help the recognition of OSA, and (iii) the performance of the type III PM device (Stardust II) for OSA diagnosis.

## Methods

### Study design

All patients underwent a detailed clinical evaluation including biochemical analysis and echocardiography followed by a full PSG and PM on separate nights before surgery. All patients first performed PSG and then PM, as detailed below. All sleep studies were scored blindly by an experienced technologist and interpreted by a sleep medicine physician.

### Participants

We studied consecutive patients with severe CAD, above 40 years of age referred for CABG and evaluated in a single outpatient clinic at the Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil. We excluded patients with previous diagnosis of OSA, history of stroke with severe disability, and patients with clinical instability, defined as clinical admission for decompensated heart failure or changes in medication over the last month and patients on use of supplemental oxygen. All procedures were carried out in accordance with institutional guidelines. The protocol was approved by the System of Scientific Documentation and Committee of Analysis of Research Projects, from the Clinics Hospital, University of São Paulo Medical School (number 168/06) and a written informed consent was obtained from all participants.

### Clinical evaluation

In the preoperative period, all participants underwent a detailed history and physical examination, including measurement of blood pressure, abdominal and neck circumferences. Large neck circumference ( $\geq 43.2$  and

$\geq 40.6$  cm, for men and women) was suggestive of OSA as described earlier [26]. The Berlin questionnaire and the Epworth Sleepiness Scale were used to evaluate clinical signs and symptoms suggestive of OSA.

### Berlin questionnaire

The Berlin questionnaire classifies patients as having a low or high risk for OSA. The classification is based on responses in three symptom categories concerning snoring, tiredness, and the presence of comorbidities. Briefly, being positive in category 1 is defined as persistent symptoms ( $> 3$ – $4$  times/weeks) in two or more questions about snoring. Category 2 is defined as positive by the presence of persistent tiredness ( $> 3$ – $4$  times/weeks). Category 3 is defined as positive by the presence of hypertension or a BMI of at least  $30 \text{ kg/m}^2$ . To be considered at high risk for OSA, a patient has to be positive in at least two symptom categories [27].

### Epworth Sleepiness Scale

The Epworth Sleepiness Scale was used to evaluate subjective excessive daytime sleepiness. Briefly, the patient rates the probability of dozing of 0–3 in eight different conditions, and a score above 10 points represents the presence of excessive daytime sleepiness [28].

### Laboratory evaluation

Blood samples were drawn for determination of fasting glucose, total cholesterol, low-density lipoprotein, high-density lipoprotein, triglycerides, and creatinine. Two-dimensional echocardiography with M-mode recording was obtained according to the American Society of Echocardiography [29]. Systolic dysfunction was defined by a left ventricular ejection fraction of 45% or less.

### Polysomnography

The standard PSG performed in the sleep laboratory overnight included electroencephalography (C3/A2, C4/A1, O1/A2, O2/A1), electrooculography, submental and anterior tibialis electromyography, pulse oximetry, measurements of airflow thermistor and nasal pressure, body position detector, snoring sounds detector, and measurements of rib cage and abdominal movements during breathing (XactTrace, EMBLA Medical Devices, Broomfield, Colorado, USA). Sleep stages were scored according to the criteria proposed by Rechtschaffen and Kales [30]. Hypopnea was defined as a 50% in airflow lasting at least 10 s associated with oxygen desaturation of greater than 3% or with an arousal. Apnea was defined when cessation of airflow lasted at least 10 s and was further classified based on presence or absence of respiratory effort as central, obstructive, or mixed [31]. The apnea–hypopnea index (AHI) was calculated as the total number of respiratory events per hour of sleep. The classification of severity of OSA was defined according to AHI as mild (5–14.9), moderate (15–29.9), and severe ( $\geq 30$  events/h) [1].

### Portable monitoring

All participants underwent an overnight study with a standard four-channel recording device (Stardust II, Respironics Inc., Murrysville, Pennsylvania, USA). This device records nasal pressure, thoracic excursion (as measured by a piezoelectric crystal), body position, pulse oximetry, heart rate, and is classified as type 3 in accordance with the AASM recommendations [25]. PM sleep studies were unattended and performed on the ward, in the preoperative period 2–3 days before surgery. Hypopnea was defined as a 50% or discernible decrement in airflow lasting at least 10 s with a 3% reduction in oxygen saturation. Apnea was defined when cessation of airflow lasted at least 10 s and was further classified as central, obstructive, or mixed based on presence of respiratory effort [31]. The total recording time was used as the denominator to calculate the AHI [1].

### Statistical analysis

Data were analyzed with SPSS 17.0 (SPSS Inc., Chicago, Illinois, USA) statistical software. The sample size was determined according to previous studies [32,33]. Assuming a SD of 9 on AHI and the minimum important difference in AHI of 5 to obtain a power of 90% and an  $\alpha$  of 0.05, 69 participants were required. After checking normality with the Kolmogorov–Smirnov test, the results were expressed as mean  $\pm$  SD, median (interquartile range), or in percentages, when appropriate. Wilcoxon signed-rank test and paired Student *t*-test or Mann–Whitney *U*-tests were used for independent samples and  $\chi^2$ -tests were used to compare frequency variables between patients without and with OSA. Sensitivity, specificity, positive likelihood ratio, and negative likelihood ratio and the negative and positive predictive values for each AHI cut-off value of 5 and 15 events/h were calculated using the AHI value from PSG and PM. Bland–Altman plots was generated to assess agreement between the PSG and PM results [34]. Receiver operator curves (ROC curves) were constructed to illustrate true-positive and false-positive results with AHI cut-off values of 5 and 15 events/h. We also analyzed the agreement between PSG and PM as described earlier [32,33]. Briefly, diagnostic agreement was considered when AHI was at least 30 events/h on both systems or, if AHI less than 30 events/h on PSG, the AHI was within 10 events/h on both systems. Overestimate of AHI by PM was defined as AHI 10 events/h greater on PM than PSG (both less than 30 events/h). Underestimation of AHI by PM was defined as AHI 10 events/h less on PM than PSG (both < 30 events/h) [32,33]. A *P* value of 0.05 or less was considered statistically significant.

### Results

We prospectively enrolled 79 patients referred for CABG. Four patients withdrew from the study (two withdrew consent, one presented clinical instability and one CABG cancelled). Five patients were further excluded because

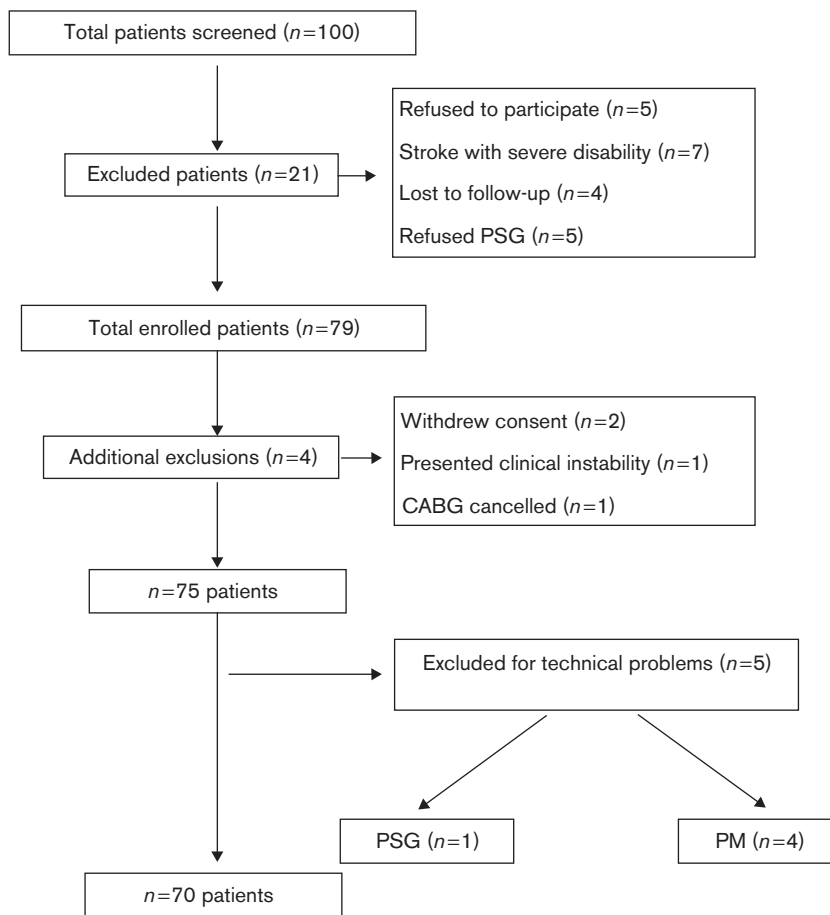
of technical problems, one being full PSG (computer malfunction) and four PM (two lost oximetry signal, one patient lost the cannula signal and one study had the data corrupted during the download process). Therefore, 70 patients (53 men, 17 women) comprised the final sample (Fig. 1). The median interval between PSG and PM studies was [median (IQR)] 4 (2–36) days. All patients were clinically stable and there were no changes in body weight between PSG and PM studies, ( $77.3 \pm 15.7$  and  $77.4 \pm 15.8$  kg, respectively,  $P = 0.96$ ). The population studied consisted predominantly of middle age, overweight Caucasians. The baseline characteristics of the entire population are presented in Table 1. Forty-two patients had no previous history of myocardial infarction, and only eight patients (11%) presented a recent acute myocardial infarction (< 3 months). The number (percentage) of patients with AHI from 0–4.9, 5–14.9, 15–29.9, and  $\geq 30$  events/h as determined by full PSG was 9 (12.9%), 23 (32.9%), 19 (27.1%), and 19 (27.1%), respectively. Therefore, the prevalence of OSA considering an AHI of at least 5, at least 15 events/h was 87 and 54%, respectively. Table 2 shows respiratory events and minimum oxygen saturation obtained during PSG and PM. Despite statistical differences, the respiratory variables were in the same range. The mean difference in AHI between the two diagnostic methods (Bland–Altman plot) was + 5.3 events/h, indicating that AHI values from PM were on average lower than those from PSG. The SD of the difference was 14.6 events/h (Fig. 2). The agreement between PM and PSG was 73%, with PM underestimating AHI in 23% and overestimating in 4% of cases.

The sensitivity, specificity, positive and negative predictive values of PM using the AHI cut-offs ( $\geq 5$  and  $\geq 15$  events/h) are presented in Table 3. The sensitivity diminished and the specificity increased in direct relation to the increase in the AHI cut-off value. The corresponding positive predictive value also decreased. The performances of the clinical characteristics for identification of the OSA AHI at least 5 events/h and moderate-to-severe OSA (AHI at least 15 events) are described in Table 4. ROC curves of different AHI cut-offs ( $\geq 5$  and  $\geq 15$  events/h) from PM are presented in Fig. 3. The areas under the curve for each cut-off are 0.90 and 0.79, respectively.

### Discussion

This study systematically evaluated the presence of OSA in a unique population of consecutive patients evaluated for CABG and contributes to the literature by showing: (i) the prevalence of OSA is strikingly high, and 54% present moderate-to-severe OSA, (ii) several symptoms and traits typical of OSA observed in patients referred to sleep laboratories, such as excessive daytime sleepiness and large neck circumference is inaccurate to identify OSA in this population, (iii) PM is a useful tool for the diagnosis of OSA

Fig. 1



Patient recruitment flowchart. CABG, coronary artery bypass grafting; PN, portable monitor; PSG, polysomnography.

in this population. Taken together, our study suggests the widespread use of PM in patients evaluated for CABG given the high prevalence of OSA and the low specificity of clinical symptoms.

OSA is now recognized as an independent risk factor for several cardiovascular diseases, including hypertension, atrial fibrillation, and mortality due to stroke and CAD [4–7,10,13,35]. Among patients with established cardiovascular disease the prevalence of OSA is strikingly high. For instance, among patients with CAD, the estimated prevalence of OSA ranges from 40 to 45% [9]. Despite all this evidence, the clinical suspicion of OSA remains low and the vast majority of patients referred to cardiology centers remains undiagnosed. The reasons for this gap are multiple, and may include the lack of typical symptoms and physical traits that are thought to be present in patients with OSA. Supporting this hypothesis, in this study, excessive daytime sleepiness and large neck circumference showed low sensitivity (Table 4). In contrast, the Berlin questionnaire that uses a composite of three categories, including snoring,

presence of tiredness (and not excessive daytime sleepiness) and presence of obesity or hypertension presented a reasonable performance to detect OSA (Table 4). Our results are in line with several studies that evaluated consecutive patients with cardiovascular disease, including patients with stroke [18], patients referred for pace-maker implant [19], and consecutive patients with metabolic syndrome [20] and hypertension [5]. Most of these studies showed little association between OSA and typical symptoms such as excessive daytime sleepiness. These findings may help to explain the low clinical suspicion of OSA among patients seen in the cardiovascular clinic outside the sleep community.

In the context of a population with high prevalence of OSA in whom the clinical symptoms and physical traits are not particularly helpful for the triage of the patients, it is important to validate a simple test to detect OSA. PM is recommended as an alternative method for OSA diagnosis and has been validated against the gold standard PSG. However, most validation studies were performed among preselected patients referred to sleep centers

**Table 1** Baseline characteristics of the study population

	Total n=70	No OSA (AHI < 15) n=32	OSA (AHI ≥ 15) n=38	P value
Male (%)	53 (76)	21 (66)	32 (84)	0.07
Caucasians (%)	52 (74)	21 (66)	31 (82)	0.30
Age (years)	58 ± 7	56 ± 7	59 ± 8	0.06
BMI (kg/m <sup>2</sup> )	27.6 (25.8–31.1)	27.1 (25.5–30.6)	28.0 (25.8–31.7)	0.23
Neck circumference (cm)	40.0 (37.4–42.0)	38.8 (35.9–40.5)	41.0 (38.9–42.6)	0.02*
Waist circumference (cm)	100.9 ± 12.1	97.8 (90.3–106.5)	103.4 ± 11.1	0.08
ESS	7 (5–11)	7 (4–11)	7 (5–10)	0.96
ESS > 10, n (%)	17 (24)	9 (28)	8 (21)	0.44
High risk, Berlin, n (%)	49 (70)	21 (66)	28 (74)	0.46
Heart rate (beats/min)	68 (60–76)	67 (59–76)	69 (60–76)	0.71
Systolic blood pressure (mmHg)	130 (110–150)	130 (109–148)	130 (110–150)	0.73
Diastolic blood pressure (mmHg)	80 (70–84)	80 (67–80)	80 (70–87)	0.43
Comorbidities				
Smoking (%)	12 (17)	5 (16)	7 (18)	0.86
Hypertension (%)	57 (81)	26 (81)	31 (82)	0.97
Diabetes mellitus (%)	28 (40)	12 (38)	16 (42)	0.70
Stroke (%)	7 (10)	3 (9)	4 (11)	0.87
Blood samples				
Glucose (mg/dl)	105 (95–139)	105 (94–150)	104 (96–134)	0.95
Creatinine (mg/dl)	1.1 (0.9–1.2)	1.0 (0.9–1.2)	1.1 (0.9–1.3)	0.19
Total cholesterol (mg/dl)	177 (143–211)	180 (142–213)	176 (143–210)	0.71
LDL (mg/dl)	108 (85–127)	112 (83–129)	104 (85–131)	0.55
HDL (mg/dl)	37 (31–40)	36 (30–40)	37 (31–40)	1.0
Triglycerides (mg/dl)	143 (99–198)	148 (96–185)	138 (101–234)	0.58
Medications				
Statins (%)	62 (89)	31 (97)	31 (82)	0.05
β-blockers (%)	65 (93)	30 (94)	35 (92)	0.79
ACEI/ARB (%)	53 (76)	23 (72)	30 (79)	0.49
ASA (%)	46 (66)	20 (63)	26 (68)	0.60
Diuretics (%)	25 (36)	14 (44)	11 (29)	0.20
Nitrate (%)	35 (50)	18 (56)	17 (45)	0.34
Calcium channel blockers (%)	25 (36)	10 (31)	15 (39)	0.47
Oral antidiabetics (%)	4 (6)	2 (6)	2 (5)	0.86
Insulin (%)	9 (13)	3 (9)	6 (16)	0.42
Echocardiography				
Ejection fraction (%)	56 (44–65)	60 (45–66)	55 (40–65)	0.39
Ejection fraction ≤ 45%, n (%)	20 (29)	9 (28)	11 (29)	0.51

Values are mean (±SD). Variables with skewed distribution are presented as median (25–75% interquartile range) or percentage.

ACEI, angiotensin-converting enzyme inhibitor; AHI, apnea-hypopnoea index; ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid; ESS, Epworth Sleepiness Scale; HDL, high-density lipoprotein; LDL, low-density lipoprotein; OSA, obstructive apnea.

\**P* < 0.05.

**Table 2** Comparison of respiratory parameters observed on polysomnography and portable monitoring

	PSG	PM	P value
Total recording time/min	451.6 ± 40.9	423.0 ± 60.7	<0.01*
Total sleep time/min	356.0 ± 70.3	–	–
AHI (events/h)	22.9 ± 20.0	17.5 ± 13.9	<0.01*
OA (events/h)	5.0 ± 7.6	7.6 ± 9.6	<0.01*
CA (events/h)	2.4 ± 8.1	1.1 ± 3.0	0.35
MA (events/h)	0.6 ± 3.7	0.3 ± 1.44	0.59
HI (events/h)	14.8 ± 12.0	8.5 ± 6.6	<0.01*
Mean SpO <sub>2</sub> %	94.3 ± 2.0	94.0 ± 2.2	0.37
Lowest SpO <sub>2</sub> %	85.0 ± 7.0	81.8 ± 8.3	<0.01*

Values are mean (±SD). Variables with skewed distribution are presented as median (25–75%, interquartile range) or percentage.

AHI, apnea-hypopnoea index; CA, central apnea; HI, hypopnoea index; MA, mixed apnea; OA, obstructive apnea; PM, portable monitoring; PSG, polysomnography; SpO<sub>2</sub>, oxygen saturation.

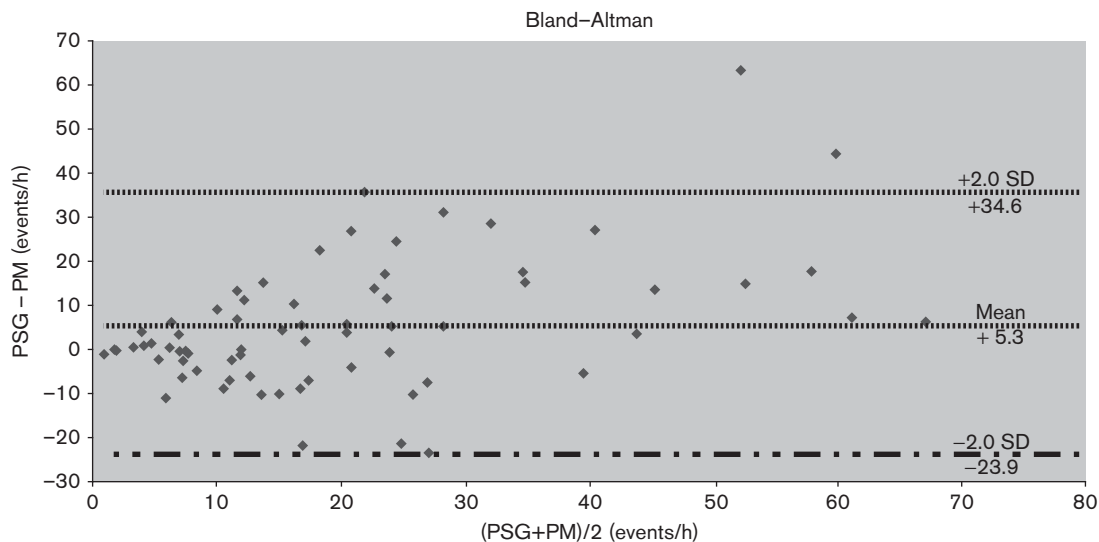
\**P* < 0.05.

[23,24,32,33,36,37]. The recent clinical guidelines of the American Association of Sleep Medicine recommend that the use of PM should be restricted to patients with a high pretest probability of moderate-to-severe OSA without significant comorbid conditions [25]. Our study therefore evaluated the performance of PM among patients that were

not evaluated and triaged by a sleep center, therefore testing the performance of PM in a clinical cardiology setting. There were several statistical differences when the mean values of respiratory parameters derived from PM and PSG (Table 2). However, the majority of the differences were probably of little clinical significance and may be at least in part explained by the night-to-night variability of AHI [36,38] or possibly reflecting the differences between equipment technologies, sampling rate, or analytical software [32]. The Bland–Altman plot indicated a substantial agreement between PM and PSG, particularly at low levels of AHI (Fig. 2). The ROC curves (Fig. 3) also showed a good performance of PM (area under the curve between 0.90 and 0.79) at all AHI cut-offs. Our study showed that PM presented acceptable sensitivity to detect OSA and acceptable specificity to exclude moderate-to-severe OSA revealing therefore a good performance of PM (Table 3).

The use of PM in patients evaluated for CABG may be of particular importance not only because of the high prevalence, but also because of the potential clinical importance of recognizing OSA in the preoperative

Fig. 2



Bland–Altman plot of apnea–hypopnea index from polysomnography (PSG) and portable monitoring (PM).

Table 3 Sensitivity, specificity, positive predictive value and negative predictive value, positive likelihood ratio (LR+) and negative likelihood ratio (LR–) for different cut-offs of AHI from the polysomnography and portable monitoring

	Sensitivity	Specificity	PPV	NPV	LR+	LR–
AHI ≥ 5 events/h	0.92 (0.81–0.97)	0.67 (0.31–0.91)	0.95 (0.85–0.99)	0.54 (0.24–0.81)	2.75 (1.09–6.96)	0.12 (0.05–0.32)
AHI ≥ 15 events/h	0.66 (0.49–0.80)	0.78 (0.60–0.90)	0.78 (0.60–0.90)	0.66 (0.49–0.80)	3.01 (1.50–6.02)	0.44 (0.28–0.69)

AHI, apnea–hypopnea index; NPV, negative predictive value; PPV, positive predictive value.

Table 4 Performances of the clinical characteristics for identification the OSA for AHI ≥ 5 and AHI ≥ 15 events/h

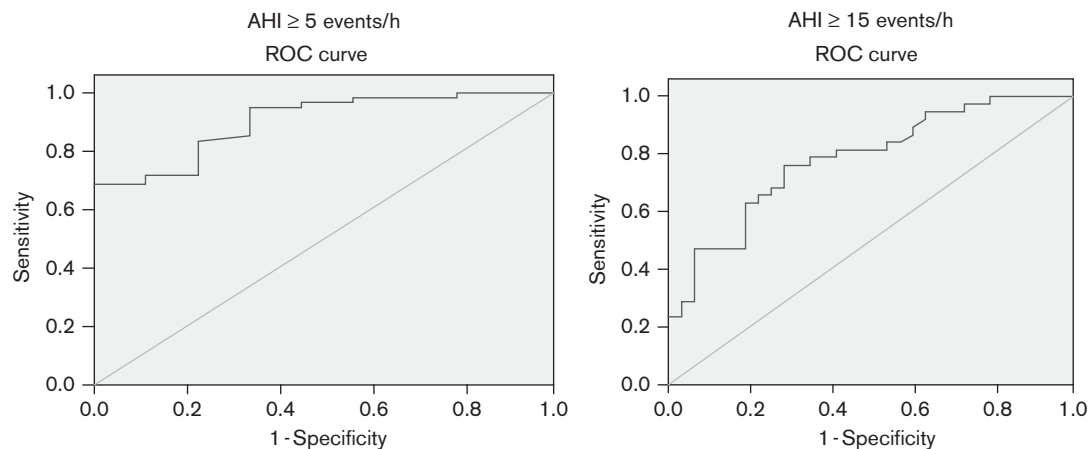
	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	LR+	LR–
AHI ≥ 5 events/h						
ESS	27	89	94	15	2.40	0.83
Berlin	72	44	90	19	1.30	0.63
BMI	34	79	91	15	1.50	0.80
Large neck	20	100	100	16	> 10	0.80
AHI ≥ 15 events/h						
ESS	21	71	47	42	0.73	1.11
Berlin	74	34	57	52	1.12	0.77
BMI	37	72	61	49	1.30	0.90
Large neck	26	94	83	52	4.21	0.79

AHI, apnea–hypopnea index; ESS, Epworth Sleepiness Scale; LR, likelihood ratio; NPV, negative predictive value; OSA, obstructive sleep apnea; PPV, positive predictive value; large neck, neck circumference ≥ 43.2 or 40.6 cm, for men and women, respectively.

period [14–17]. OSA can contribute to cardiovascular disease by multiple mechanisms, including intermittent hypoxemia, increase of the sympathetic activation with surges in blood pressure, that in conjunction with simultaneous changes in intrathoracic and cardiac transmural pressures may trigger or contribute to cardiac ischemia, arrhythmia, and CAD [4,7,9]. There is evidence that OSA contributes independently to atherosclerosis progression [39] that can be partially reverted with the treatment of OSA with continuous positive airway pressure [40]. Continuous positive airway pressure can

also reduce the appearance of new cardiovascular events among patients with CAD [41], and reduce cardiovascular mortality among patients referred to sleep studies [42]. There is also some evidence suggesting that patients with OSA have a higher incidence of postoperative complications [14–17]. The use of PM in this population offers potential advantages over PSG: easier access, no need to transfer patients to another facility (many cardiology wards do not have access to PSG), and lack of PSG technical supervision may result in overall reduction of health care costs in this population.

Fig. 3



Receiver operating characteristic (ROC) curve of apnea-hypopnea index (AHI) cut-offs ( $\geq 5$  and  $\geq 15$  events/h) for polysomnography and portable monitoring (AUC=0.90 and 0.79, respectively). AUC, area under the ROC curve.

Our study has several potential limitations. Our patients were overweight but not overtly obese, therefore the prevalence of OSA in populations with higher obesity prevalence rates is likely higher. The number of technical problems related to PM was relatively small (5.3%) and in the same range of previous studies (3–18%) [25]. The low number of PM technical problems may be due to the fact that the studies were performed on the ward and scored by an experienced sleep technician and interpreted by a qualified sleep medicine physician; however, it is possible that the failure rate of PM when performed at home would be greater. Therefore, our data support only the use of PM in the in-patient setting, not at home. In contrast, we have recently reported similar technical performance using the same device at home in patients with hypertrophic cardiomyopathy [8]. Despite the presence of low ejection fraction in a significant proportion of our patients, we found a low prevalence of central sleep apnea. Therefore, our study does not fully allow elucidation of the use of PM in the setting of central sleep apnea. However, Quintana-Gallego *et al.* [43] have earlier validated the use of PM in patients with stable congestive heart failure in whom a significant proportion had central sleep apnea. In our study, patients who had a diagnosis of OSA were referred for follow-up in the sleep clinic for specific evaluation and treatment after surgery. Therefore, further studies are necessary to clarify the importance of OSA detection and treatment in patients with established CAD in the pre- and postoperative period of CABG.

In summary, this study showed that OSA is extremely common among patients referred to CABG and that clinical characteristics are not adequate signs to identify OSA in this population. We further showed that type 3 PM is useful tool for the diagnosis of OSA in consecutive patients with severe CAD evaluated for CABG. Further studies are needed to determine the best management

and the impact of treatment of OSA in patients with CAD evaluated for CABG.

### Acknowledgements

Naury J. Danzi-Soares contributed to study design, data collection and analysis, and manuscript draft; Pedro R. Genta contributed to data analysis; Flávia B. Nerbass contributed to data collection; Rodrigo P. Pedrosa contributed to data analysis; Flávia S. Nunes Soares contributed to study design and data collection; Luis A. M. Cesar contributed to data collection; Luciano F. Drager contributed to study design and manuscript draft; Robert Skomro contributed to manuscript draft; Geraldo Lorenzi-Filho contributed to study design, data analysis, and manuscript draft.

This study was supported by grants from the FAPESP. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

### Conflicts of interest

There are no conflicts of interest.

### References

- 1 Flemons WW, Buysse D, Redline S, Strohl K, Wheatley J, Douglas N, *et al.* Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999; **22**:667–669.
- 2 Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993; **328**:1230–1235.
- 3 Tufik S, Santos-Silva R, Taddei JA, Bittencourt LR. Obstructive sleep apnea syndrome in the São Paulo Epidemiologic Sleep Study. *Sleep Med* 2010; **11**:441–446.
- 4 Somers VK, White DP, Amin R, Abraham WT, Costa F, Culebras A, *et al.* Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation scientific statement from the



- American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. *Circulation* 2008; **118**:1080–1111.
- 5 Drager LF, Genta PR, Pedrosa RP, Nerbass FB, Gonzaga CC, Krieger EM, *et al.* Characteristics and predictors of obstructive sleep apnea in patients with systemic hypertension. *Am J Cardiol* 2010; **105**:1135–1139.
  - 6 Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 2000; **342**:1378–1384.
  - 7 Gami AS, Pressman G, Caples SM, Kanagala R, Gard JJ, Davison DE, *et al.* Association of atrial fibrillation and obstructive sleep apnea. *Circulation* 2004; **110**:364–367.
  - 8 Pedrosa RP, Drager LF, Genta PR, Amaro AC, Antunes MO, Matsumoto AY, *et al.* Obstructive sleep apnea is common and independently associated with atrial fibrillation in patients with hypertrophic cardiomyopathy. *Chest* 2010; **137**:1078–1084.
  - 9 Prinz C, Bitter T, Piper C, Horstkotte D, Faber L, Oldenburg O. Sleep apnea is common in patients with coronary artery disease. *Wien Med Wochenschr* 2010; **160**:349–355.
  - 10 Peker Y, Kraiczi H, Hedner J, Loth S, Johansson A, Bende M. An independent association between obstructive sleep apnea and coronary artery disease. *Eur Respir J* 1999; **14**:179–184.
  - 11 Moore T, Rabben T, Wiklund U, Franklin KA, Eriksson P. Sleep-disordered breathing in men with coronary artery disease. *Chest* 1996; **109**:659–663.
  - 12 Hagenah GC, Gueven E, Andreas S. Influence of obstructive sleep apnea in coronary artery disease: a 10-year follow-up. *Resp Med* 2006; **100**:180–182.
  - 13 Peker Y, Hedner J, Kraiczi H, Loth S. Respiratory disturbance index: an independent predictor of mortality in coronary artery disease. *Am J Respir Crit Care Med* 2000; **162**:81–86.
  - 14 Liao P, Yegneswaran B, Vairavanathan S, Zilberman P, Chung F. Postoperative complications in patients with obstructive sleep apnea: a retrospective matched cohort study. *Can J Anaesth* 2009; **56**:819–828.
  - 15 Hwang D, Shakir N, Limann B, Sison C, Kalra S, Shulman L, *et al.* Association of sleep-disordered breathing with postoperative complications. *Chest* 2008; **133**:1128–1134.
  - 16 Chung SA, Yuan H, Chung F. A systemic review of obstructive sleep apnea and its implications for anesthesiologists. *Anesth Analg* 2008; **107**:1543–1563.
  - 17 Kaw R, Golish J, Ghamande S, Burgess R, Foldvary N, Walker E. Incremental risk of obstructive sleep apnea on cardiac surgical outcomes. *J Cardiovasc Surg* 2006; **47**:683–689.
  - 18 Arzt M, Young T, Peppard PE, Finn L, Ryan CM, Bayley M, *et al.* Dissociation of obstructive sleep apnea from hypersomnolence and obesity in patients with stroke. *Stroke* 2010; **41**:129–134.
  - 19 Garrigue S, Pépin J-L, Defaye P, Murgatroyd F, Poezevara Y, Clémenty J, *et al.* High prevalence of sleep apnea syndrome in patients with long-term pacing: the European multicenter polysomnographic study. *Circulation* 2007; **115**:1703–1709.
  - 20 Drager LF, Lopes HF, Maki-Nunes C, Trombetta IC, Toschi-Dias E, Alves MJ, *et al.* The impact of obstructive sleep apnea on metabolic and inflammatory markers in consecutive patients with metabolic syndrome. *PLoS One* 2010 **5**:12065.
  - 21 Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med* 2002; **165**:1217–1239.
  - 22 Chesson AL Jr, Berry BB, Pack A. Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults. *Sleep* 2003; **26**:907–913.
  - 23 Ng SS, Chan TO, To KW, Ngai J, Tung A, Ko FW, *et al.* Validation of Embletta portable diagnostic system for identifying patients with suspected obstructive sleep apnea syndrome (OSAS). *Respirology* 2010; **15**:336–342.
  - 24 To KW, Chan WC, Chan TO, Tung A, Ngai J, Ng S, *et al.* Validation study of a portable monitoring device for identifying OSA in a symptomatic patient population. *Respirology* 2009; **14**:270–275.
  - 25 Collop NA, Anderson WM, Boehlecke B, Claman D, Goldberg R, Gottlieb DJ, *et al.* Portable Monitoring Task Force of the American Academy of sleep medicine, clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. Portable Monitoring task force of the American Academy of Sleep Medicine. *J Clin Sleep Med* 2007; **3**:737–747.
  - 26 Epstein LJ, Kristo D, Strollo PJ, Friedman N, Malhotra A, Patil SP, *et al.* Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 2009; **5**:263–276.
  - 27 Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med* 1999; **131**:485–491.
  - 28 Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; **14**:540–545.
  - 29 Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, *et al.* American Society of Echocardiography's Nomenclature and Standards Committee; Task Force on Chamber Quantification; American College of Cardiology Echocardiography Committee; American Heart Association; European Association of Echocardiography, European Society of Cardiology. Recommendations for chamber quantification. *Eur J Echocardiogr* 2006; **7**:79–108.
  - 30 Rechtschaffen A, Kales A. *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects.* Los Angeles, CA: BIS/BRI UCLA; 1968. pp. 1–57.
  - 31 Iber C, Ancoli-Israel S, Chesson AL, Quan SF. *The AASM manual for the scoring of sleep and associated events, rules, terminology and technical specifications.* Westchester, IL: American Academy of Sleep Medicine; 2007.
  - 32 Santos-Silva R, Sartori DE, Truksinas V, Truksinas E, Alonso FFFD, Tufik S, *et al.* Validation of a portable monitoring system for the diagnosis of obstructive sleep apnea syndrome. *Sleep* 2009; **32**:629–636.
  - 33 White DP, Gibb TJ, Wall JM, Westbrook PR. Assessment of accuracy and analysis time of a novel device to monitor sleep and breathing in the home. *Sleep* 1995; **18**:115–126.
  - 34 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986; **1**:307–310.
  - 35 Punjabi NM, Caffo BS, Goodwin JL, Gottlieb DJ, Newman AB, O'Connor GT, *et al.* Sleep-disordered breathing and mortality: a prospective cohort study. *Plos Med* 2009; **6**:e1000132.
  - 36 Tonelli de Oliveira AC, Martinez D, Vasconcelos LF, Gonçalves SC, Lenz MC, Fuchs SC, *et al.* Diagnosis of obstructive sleep apnea syndrome and its outcomes with home portable monitoring. *Chest* 2009; **135**:330–336.
  - 37 Skomro RP, Gjevre J, Reid J, McNab B, Ghosh S, Stiles M, *et al.* Outcomes of home-based diagnosis and treatment of obstructive sleep apnea. *Chest* 2010; **138**:257–263.
  - 38 Bittencourt LR, Suchecki D, Tufik S, Peres C, Togeiro SM, Bagnato MC, *et al.* The variability of the apnea-hypopnea index. *J Sleep Res* 2001; **10**:245–251.
  - 39 Drager LF, Bortolotto LA, Lorenzi MC, Figueiredo AC, Krieger EM, Lorenzi-Filho G. Early signs of atherosclerosis in obstructive sleep apnea. *Am J Respir Crit Care Med* 2005; **172**:613–618.
  - 40 Drager LF, Pedrosa RP, Diniz PM, Diegues-Silva L, Marcondes B, Couto RB, *et al.* The effects of continuous positive airway pressure on prehypertension and masked hypertension in men with severe obstructive sleep apnea. *Hypertension* 2011; **57**:549–555.
  - 41 Milleron O, Pillière R, Foucher A, de Roquefeuil F, Aegerter P, Jondeau G, *et al.* Benefits of obstructive sleep apnea treatment in coronary artery disease: a long-term follow-up study. *Eur Heart J* 2004; **25**:728–734.
  - 42 Marin JM, Carrizo SJ, Vicente E, Agustí AG. Long-term cardiovascular outcomes in men with obstructive sleep apnea-hypopnea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005; **365**:1046–1053.
  - 43 Quintana-Gallego E, Villa-Gil M, Carmona-Bernal C, Botebol-Benhamou G, Martínez-Martínez A, Sánchez-Armengol A, *et al.* Home respiratory polygraphy for diagnosis of sleep-disordered breathing in heart failure. *Eur Respir J* 2004; **24**:443–448.