Sleep-disordered breathing as a risk factor for unnecessary pacemaker implantation

Anna Szajerska-Kurasiewicz^{1*}, Danuta Loboda^{1, 2*}, Tomasz Roleder^{3, 4}, Michalina Stepanik⁵, Jacek Durmala⁵, Krzysztof S Golba^{1, 2}

¹Department of Electrocardiology, Upper-Silesian Medical Center, Katowice, Poland ²Department of Electrocardiology and Heart Failure, Medical University of Silesia, Katowice, Poland

³Regional Specialist Hospital, Research and Development Center, Wrocław, Poland

⁴Department of Cardiology and Structural Heart Diseases, Medical University of Silesia, Katowice, Poland

⁵Department of Rehabilitation, Medical University of Silesia, Katowice, Poland

*Both authors equally contributed to the study

Correspondence to:

Anna Szajerska-Kurasiewicz, MD, Department of Electrocardiology, Upper-Silesian Medical Center, Ziołowa 45/47, 40–635, Katowice, Poland phone: +48 32 359 89 90, e-mail: a.szajerska.kurasiewicz@ amail.com

Copyright by the Author(s), 2022

DOI: 10.33963/KP.a2022.0011 Received:

September 23, 2021 Accepted:

January 14, 2022

Early publication date: January 14, 2022

ABSTRACT

Background: Sleep-disordered breathing (SDB) is a risk factor for bradyarrhythmia, which is reversible with positive airway pressure therapy.

Aims: The study aims to evaluate the occurrence and number of severe sinus bradycardia and advanced atrioventricular block (AVB) in patients with cardiovascular diseases and SDB risk factors.

Methods: The analysis covered 207 patients with cardiovascular diseases aged 59.4 (standard deviation [SD], 10.49) years, including 177 men (85.51%), hospitalized in the Department of Electrocardiology and the Day Stay Cardiac Rehabilitation Ward Upper-Silesian Medical Centre in Katowice, Poland. The inclusion criterion was a high risk of SDB, in particular obstructive sleep apnea (OSA), in one of the following questionnaires: the Four-Variable Screening Tool, the STOP-Bang Questionnaire, and the Epworth Sleepiness Scale. Both level-3 portable sleep tests and electrocardiogram Holter recordings were made simultaneously.

Results: SDB was confirmed in 175 (84.5%) patients, including severe in 74 (35.7%), moderate in 42 (20.3%), and mild in 59 (28.5%) participants. The dominant type of SDB was OSA, which was found in 158 (76.3%) participants. The severe SDB was a predictor of third-degree AVB (odds ratio [OR], 11.61; 95% confidence interval [CI], 1.37–98.60), second-degree AVB type 2 (Mobitz) (OR, 4.51; 95% CI, 1.17–18.08), pauses above 3 seconds (OR, 10.26; 95% CI, 2.18–48.40), and sinus bradycardia below 40 bpm (OR, 3.00; 95% CI, 1.36–6.60) during sleep.

Conclusions: SDB, with particular emphasis on OSA, is a risk factor for sinus bradycardia and advanced AVB during sleep, which may lead to a hasty qualification for pacemaker implantation. The severity of SDB determines the frequency and number of bradyarrhythmic episodes.

Key words: conduction disorders, pacemaker implantation, sleep-disordered breathing

INTRODUCTION

Sleep-disordered breathing (SDB) in the forms of obstructive sleep apnea (OSA) and central sleep apnea (CSA) increases the risk of cardiovascular (CV) complications, such as difficult-to-control arterial hypertension, heart failure, coronary artery disease, cerebrovascular accidents, arrhythmias, and sudden cardiac death, as well as sinus bradycardia and atrioventricular block (AVB) [1–4]. Nocturnal episodes of upper airway obstruction due to pharyngeal wall collapse are a cause of OSA, with older age, male sex, obesity, and large neck circumference being major risk factors [5, 6]. CSA, on the other hand, is caused by unstable ventilatory control during sleep and occurs in a significant percentage of patients with congestive heart failure [6, 7]. Apnea--related bradycardia and AVB are associated with hypervagotonia [8] and are reversible

WHAT'S NEW?

Sleep-disordered breathing (SDB) is common in patients with cardiovascular diseases and is a risk factor for nocturnal bradyarrhythmias. Even severe bradycardia and advanced atrioventricular block in patients with sleep apnea syndrome can be vagally mediated. In this case, they do not require the implantation of a pacemaker. Published on August 29, 2021, the "2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy" recommend that patients with asymptomatic nocturnal sinus bradycardia or atrioventricular block be assessed for the presence of SDB (Class I C).

with the implementation of adequate treatment, including positive airway pressure (PAP) therapy [9, 10]. Nevertheless, a failure to diagnose and treat SDB may increase the risk of adverse CV outcomes [11–13] and lead to a hasty decision to implant a cardiac pacemaker (PM).

The purpose of this study was to evaluate the prevalence of bradycardia and advanced AVB in patients with CV diseases and SDB risk factors and to analyze the relationship of SDB severity with the occurrence and number of bradyarrhythmias.

METHODS

Questionnaires assessing the risk of SDB were conducted in a group of 443 consecutive patients with CV diseases hospitalized in the Department of Electrocardiology or the Day Stay Cardiac Rehabilitation Ward of the Upper-Silesian Medical Center in Katowice, Poland. In the further study, we included only those patients who were at high risk for SDB in terms of at least one of the following scales: the 4-Variable Screening Tool [14], the STOP-Bang Questionnaire [15], the Epworth Sleepiness Scale (ESS) [16], and who agreed to take the level-3 portable sleep test (L3PST) and electrocardiogram (ECG) Holter recording. The exclusion criteria were previously implanted PM and current SDB treatment with PAP therapy or intraoral appliances.

The Institutional Review Board of the Medical University of Silesia in Katowice, Poland, approved the study (no. KW/0022/KBI/77/18 and no. PCN/0022/KBI/77/I/18/20), and all patients signed informed consent.

Each eligible patient underwent L3PST using an Alice NightOne (Philips Respironics, Bothell, WA, USA) device [17]. The analysis was performed according to the recommendations of the American Academy of Sleep Medicine (AASM) [18], evaluating the following parameters: (1) the number of episodes of obstructive, central, and mixed sleep apnea, defined as the complete cessation of the airway flow for ≥ 10 seconds with or without preserved respiratory effort; (2) the number of hypopnea episodes, defined as the reduction in the airway flow by \geq 90%, with a duration of \geq 10 seconds, followed by a decrease in the hemoglobin oxygen saturation of $\geq 4\%$; (3) the number of apneas and hypopneas per hour of total recording time (respiratory event index [REI]) [events/hour]; (4) the mean duration of respiratory events (seconds); (5) the minimum and mean arterial oxygen saturation estimated by pulse oximetry

 (SpO_2) during sleep [%]; and (6) the total sleep time with oxygen saturation under 90% (TST90) [%].

ECG Holter recordings were performed between 10 p.m. and 6 a.m. The occurrence and number of the following events were analyzed: (1) episodes of third- or second-degree type 2 (Mobitz) AVB; (2) pauses longer than 3 seconds; and (3) sinus bradycardia episodes, defined as the heart rate slowing below 40 bpm. The study was conducted in accordance with the 2017 International Society for Holter and Non-Invasive Electrocardiology and the Heart Rhythm Society expert consensus statement on ambulatory ECG and external cardiac monitoring/telemetry [19].

Statistical analysis

The obtained results were analyzed using MedCalc 20.008 software (MedCalc Software Ltd, Ostend, Belgium). Quantitative parameters were characterized using the arithmetic mean and SD or median, interquartile range (IQR) and sample range, depending on the normality of the distribution assessed by the Kolmogorov-Smirnov test. The analysis of quantitative variables was conducted by assessing the significance of differences for two or more independent samples using: (1) the one-way analysis of variance with post hoc comparisons used the Tukey-Kramer test or (2) the Kruskal Wallis H-test with Conover post hoc analysis, according to the result of Levene's test for equality of variances. Qualitative data were expressed by incidence as percentages and compared using the Chi² test or Fisher's exact test if the number of expected frequencies in the subgroups was less than five. The logistic regression analysis was used to estimate the odds ratio (OR) of the incidence of bradycardia, AVB, or pauses in the SDB groups. The optimal cut-off value for REI, minimum SpO₂, and TST90 for predicting the combined incidence of bradycardia and advanced AVB during sleep were determined by receiver-operating characteristic (ROC) curve analysis. P < 0.05 was used as the limit of the significance level in the tests conducted.

RESULTS

Based on the questionnaires, a high risk of SDB was found in 282 out of 443 respondents (63.7%). Diagnostic L3PST and ECG Holter recordings were performed in 207 participants at high risk for SDB, including 177 (85.5%) men and 30 (14.5%) women — these patients constituted the study group. The mean (SD) age of the participants Table 1. Risk factors and the prevalence of cardiovascular diseases in patients with high vs. low to moderate risk of sleep-disordered breathing based on the Four-Variable Screening Tool, the STOP-Bang Questionnaire, and the Epworth Sleepiness Scale

Parameters of SDB integrated risk assessment by the use of	Number of patie	P-value ^a	
scales	Group with a high risk of SDB	Group with a low to moderate risk of SDB	
Groups size, n (%)	282 (63.7)	161 (36.3)	
Male sex, n (%)	239 (72.6)	90 (27.4)	<0.001
Loud and frequent snoring, n (%)	188 (84.3)	35 (15.7)	<0.001
Observed stop breathing or choking/gasping during sleep, n (%)	124 (96.1)	5 (3.9)	<0.001
Excessive daytime sleepiness (>10 points in ESS), n (%)	54 (94.7)	3 (5.3)	<0.001
Diagnosed hypertension, n (%)	236 (66.3)	120 (33.7)	0.006
Age older than 50 years, n (%)	240 (64.0)	135 (36.0)	0.72
Neck circumference ≥43 cm (males), n (%)	156 (85.2)	27 (14.8)	<0.001
Neck circumference ≥41 cm (females), n (%)	27 (87.1)	4 (12.9)	<0.001
BMI >30 kg/m², n (%)	147 (79.0)	39 (21.0)	<0.001
BMI >35 kg/m², n (%)	67 (91.8)	6 (8.2)	<0.001
Cardiovascular diseases, n (%)			
Coronary artery disease, n (%)	225 (61.8)	139 (38.2)	<0.001
History of myocardial infarction, n (%)	196 (61.1)	125 (38.9)	<0.001
Difficult-to-control blood pressure, n (%)	102 (80.3)	25 (19.7)	<0.001
Atrial fibrillation, n (%)	38 (67.9)	18 (32.1)	0.65
History of ischemic stroke, n (%)	7 (58.3)	5 (41.7)	0.62
HFmrEF, n (%)	54 (64.3)	30 (35.7)	0.86
HFrEF, n (%)	20 (52.6)	18 (47.4)	0.35

^aP-value for differences between groups

Abbreviations: BMI, body mass index, difficult-to-control blood pressure — systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg on antihypertensive treatment; ESS, Epworth Sleepiness Scale; HfmrEF, heart failure with mid-range ejection fraction; HfrEF, heart failure with reduced ejection fraction; OSA, obstructive sleep apnea; SDB, sleep-disordered breathing

Table 2. Distribution of slee	p-disordered breathing and	heart rate parameter	s in the study group
			/ / /

Test variable	SDB severity			<i>P</i> -value ^a	
	None (A)	Mild (B)	Moderate (C)	Severe (D)	
Group size, n (%)	32 (15.5)	59 (28.5)	42 (20.3)	74 (35.7)	
REI	2.67 (1.64)	9.66 (2.75)	20.81 (4.86)	53.67 (16.79)	< 0.001
Mean SpO ₂ , %	92.78 (2.54) A≠D	92.59 (1.70) B≠D	92.24 (2.17) C≠D	90.01 (3.74)	< 0.001
Minimal SpO ₂ , %	84.26 (8.34) A≠D	83.56 (5.61) B≠D	81.81 (6.71) C≠D	74.31 (10.24)	< 0.001
TST90, %	14.14 (24.95) A≠D	10.19 (17.31) B≠D	14.98 (21.91) C≠D	29.43 (25.32)	< 0.001
Mean duration of respiratory events, sec	23.22 (8.31)	23.58 (6.30)	22.60 (5.17)	25.28 (8.26)	0.31
Mean HR	59.51 (9.03)	59.52 (7.41)	59.33 (9.10)	61.29 11.34)	0.63
Minimal HR	47.19 (8.61)	48.23 (6.14)	45.59 (9.31)	43.53 (8.32)	0.009

All data are given as mean (standard deviation [SD])

^aP-value for differences between groups

Abbreviations: HR, heart rate estimated by pulse oximetry; REI, respiratory event index; SDB, sleep-disordered breathing; SpO₂, arterial oxygen saturation estimated by pulse oximetry; TST90, total sleep time with oxygen saturation under 90%

was 59.38 (10.37) years, the mean body mass index (BMI) 32.49 (7.25) kg/m², and the neck circumference 44.05 (3.81) cm. The parameters used to assess SDB risk, CV comorbidities, and their occurrence and number in the survey group are given in Table 1.

Based on L3PST, SDB was confirmed in 175 (84.5%) out of 207 subjects. The OSA found in 158 (76.3%) participants was the predominant type of SDB. Central apnea episodes predominated in 10 (4.8%) cases, whereas mixed apnea episodes predominated in seven (3.4%) cases. Mild SDB (REI, 5–14 events/hour) was found in 59 (28.5%), moderate SDB (REI, 15–30 events/hour) in 42 (20.3%), and severe SDB (REI, >30 events/hour) in 74 (35.7%) patients. SDB was not confirmed in 32 (15.5%) cases despite the high risk. The recorded L3PST parameters are shown in Table 2.

Sinus bradycardia and advanced AVB were recorded in 42 (20.1%) subjects, including 39 (18.4%) patients with SDB and only three patients without SDB (1.4%); P = 0.07. Sinus bradycardia <40 bpm was confirmed in 29 (18.6%), type 2 (Mobitz) second-degree AVB in nine (5.8%), third-degree AVB in seven (4.5%), and pauses >3 seconds in 12 (7.7%) patients. In 15 (8.6%) cases, two or three types of the foregoing disorders occurred jointly in the same patients. The incidence of bradyarrhythmias was not dependent on the type (obstructive vs. central vs. mixed) of respiratory events recorded. Pauses >3 sec-

Table 3. The ratio of patients with sinus bradycardia and nocturnal advanced atrioventricular block according to the severity of sleep-disor-
dered breathing

Type of rhythm	SDB severity				<i>P</i> -value ^a
or conduction disturbances	None (A)	Mild (B)	Moderate (C)	Severe (D)	
Groups size, n (%)	32 (15.5)	59 (28.5)	42 (20.3)	74 (35.7)	
Sinus bradycardia <40 bpm, n (%)	2 (6.7)	3 (6.0) B≠D	8 (20.5)	18 (26.9)	0.009
2 nd -degree AVB type 2 (Mobitz), n (%)	1 (3.3)	0 (0.0)	2 (5.1)	7 (10.4)	0.09
3 rd -degree AVB, n (%)	0 (0.0)	0 (0.0)	1 (2.6)	6 (9.0)	0.04
Pauses >3 sec, n (%)	0 (0.0) A≠D	0 (0.0) B≠D	2 (5.1)	10 (14.9)	0.004
At least one of the above, n (%)	3 (10.0) A≠D	3 (6.0) B≠D	10 (25.6)	26 (38.8)	<0.001

^a*P*-value for differences between groups $\neq -P < 0.05$

Abbreviations: AVB, atrioventricular block; SDB, sleep-disordered breathing

Table 4. The number of episodes of sinus bradycardia and nocturnal advanced atrioventricular block according to the severity of sleep-disordered breathing

Type of rhythm	SDB severity				0
or conduction disturbances	None (A)	Mild (B)	Moderate (C)	Severe (D)	P-value [®]
Groups size, n (%)	32 (15.5)	59 (28.5)	42 (20.3)	74 (35.7)	-
Sinus bradycardia <40 bpm	0 (0.0–0.0) range 0–23 A≠D	0 (0.0–0.0) range 0–384 B≠D	0 (0.0–0.0) range 0–920	0 (0.0–0.0) range 0–1033	0.01
2 nd -degree AVB type 2 (Mobitz)	0 (0.0–0.0) range 0–2	0 (0.0–0.0) range 0–2	0 (0.0–0.0) range 0–1135	0 (0.0–0.0) range 0–2133	0.15
3 rd -degree AVB	0 (0.0–0.0) range 0–0 A≠D	0 (0.0–0.0) range 0–0 B≠D	0 (0.0–0.0) range 0–27	0 (0.0–0.0) range 0–14	0.04
Pauses >3 seconds	0 (0.0–0.0) range 0–0 A≠D	0 (0.0–0.0) range 0–0 B≠D	0 (0.0–0.0) range 0–400	0 (0.0–0.75) range 0–100	0.008

^a*P*-value for differences between groups, \neq -*P* <0.05, all data are given as median, interquartile range (in parenthesis), and sample range Abbreviations: see Table 3

Table 5. The relative odds of the incidence of sinus bradycardia below 40 bpm, advanced atrioventricular block, and pauses above 3 seconds in patients with severe sleep-disordered breathing

Dependent variable	Independent variable: severe sleep-disordered breathing		
	Odds ratio (95% CI)	<i>P</i> -value ^a	
Sinus bradycardia <40 bpm	3.00 (1.36–6.56)	0.007	
Second-degree AVB type 2 (Mobitz)	4.51 (1.13–18.08)	0.03	
Third-degree AVB	11.61 (1.37–98.60)	0.03	
Pauses >3 seconds	10.26 (2.18–48.40) 0.003		

^aP-value for differences between groups

Abbreviations: CI, confidence interval; other — see Table 3

onds were found more often in the group of patients with episodes of atrial fibrillation (AF) during sleep compared to the rest of the studied population (n = 4, 33.3% vs. n = 8, 4.6%; P < 0.001). In patients with severe SDB, the occurrence and the number of bradyarrhythmias were significantly higher than in patients without SDB or with mild SDB (Tables 3 and 4).

The OR for the occurrence of the foregoing disorders in a patient with severe SDB was 3.00 (95% Cl, 1.360–6.598) for sinus bradycardia <40 bpm, 11.61 (95% Cl, 1.366–98.599) for third-degree AVB, 4.51 (95% Cl, 1.126–18.075) for second-degree AVB type 2 (Mobitz), and 10.26 (95% CI, 2.176–48.397) for pauses >3 seconds, respectively (Table 5). The percentage of conduction disturbances in the group with moderate SDB was also high, so the cut-off point for REI in predicting sinus bradycardia <40 bpm or advanced AVB turned out to be >22 events/h (sensitivity, 78.57%; specificity, 65.97%) (Figure 1A). The relationship between minimal SpO₂ (cut-off point, \leq 75%; sensitivity, 54.76%; specificity, 86.52%), TST90 (cut-off point, >5.42%; sensitivity, 80.95%; specificity, 52.82%), and the incidence of the foregoing disorders was also visible (Figure 1B–C).



Figure 1. The cut-off point for A. respiratory event index; B. minimal arterial oxygen saturation; C. total sleep time with oxygen saturation under 90% in predicting sinus bradycardia <40 bpm or advanced atrioventricular block during sleep

Abbreviations: REI, respiratory event index; SpO₂, arterial oxygen saturation estimated by pulse oximetry; TST90, total sleep time with oxygen saturation under 90%, cut points are marked°

DISCUSSION

The presented study confirmed the high percentage of SDB, especially OSA, in the Polish population with CV diseases. The high prevalence of moderate to severe SDB has already been described in cardiac patients, estimating it at 30% to 80%, depending on the type of CV disease [7, 20–22]. This percentage is higher than in the general population in Poland, where it is reported to be 8.5% for women and 19.8% for men [23], and in the United States, 9% for women and 17% for men [6].

Common CV and SDB risk factors such as older age, male sex, obesity, and metabolic syndrome are responsible for such a high rate of SDB, predominately OSA, in patients with CV diseases [6, 10, 20, 24]. Similarly, in the surveyed group, most patients at high risk of SDB were men aged >50 years, with a BMI >30 kg/m² or higher. A high risk of SDB was significantly more likely to coexist with coronary artery disease, history of myocardial infarction, and difficult-to-control blood pressure, that warrants greater attention to this group of CV diseases.

Obesity and hypertension, a large neck circumference, and a history of loud/frequent snoring or pauses in breathing observed by household members are the basis of SDB risk assessment using prediction algorithms [14, 15]. The questionnaires used in our study, especially the STOP-Bang Questionnaire, allowed us to confirm SDB in 84.5% of those with a positive test, which is close to the positive predictive value for this test reported by Pivetta et al. [15]. At the same time, severe bradyarrhythmias were confirmed in as many as 20.1% of patients at high risk of SDB. However, a relatively small percentage (12.9%) of the surveyed CV patients reported increased daytime sleepiness. The diagnostic problem resulting from the low sensitivity of the ESS in patients with bradycardia and implanted PM was previously described by Garrigue et al. [25], who reported that a score >10 on the ESS scale was achieved by as little as 22.8% of those with the apnea-hypopnea index (AHI) >10 events/h. In addition, Velasco et al. [26] did not confirm the usefulness of the Berlin questionnaire in predicting nocturnal bradycardia. In this respect, the evaluation of other questionnaires may be useful in the bedside diagnosis of SDB-related bradyarrhythmias.

In the presented group, REI (>22 events/h) and desaturation (≤75%) were found to be predictors of nocturnal bradyarrhythmias. Patients with severe SDB appeared to be particularly at risk, which translated into a 3-fold-higher sinus bradycardia risk, an 11-fold-higher risk of third--degree AVB, a 5-fold-higher risk of second-degree AVB type 2 (Mobitz), and a 10-fold-higher risk of pauses >3 seconds in these patients. These findings are consistent with publications by other authors [2, 10, 27, 28]. Sinus bradycardia was found in approximately 1.5%-20.0% and advanced AVB in 8-20% of SDB patients [2, 9, 10, 28], compared to 1% in the healthy elderly population [29]. The incidence of bradycardia, pauses, and advanced AVB increases with higher AHI [10, 28, 30] and lower SpO₂ during apnea episodes [2, 27, 28, 30]. The incidence of bradyarrhythmias is also dependent on the sleep phase and found more frequently in the sleep phase with rapid eye movement as a result of longer duration of respiratory episodes and deeper desaturations [27, 28].

In the study group, as many as 67.9% of patients at high risk of SDB were diagnosed with AF, and pauses >3 seconds were found significantly more often in patients with episodes of AF recorded during sleep. The coexistence of sinus bradycardia during sleep and paroxysmal AF (tachycardia-bradycardia syndrome, T-B syndrome) occurred in nine patients (4.3%). It is worth noting that SDB is a significant and reversible risk factor of new-onset AF, T-B syndrome, and AF recurrence despite adequate pharmacological treatment or with the use of ablation techniques [2, 20, 31–33].

In the European Multicenter Polysomnographic Study by Garrigue et al. [25] conducted in patients with PM implantation, as many as 58% of patients with sinus node dysfunction and 68% with AVB had previously undiagnosed SDB. Among the study participants, 21.4% had severe SDB with an AHI >30 events/h. In turn, Marti-Almor et al. [33], in a REgistry of Sleep APnea monItoring and Atrial Fibrillation in pacemakeR patients (RESPIRE study), found severe OSA, defined as a respiratory disturbance index of \geq 20, in 31.1% of patients with PM capable of detecting SDB. The high sensitivity and specificity of PM with transthoracic impedance sensor in the diagnosis of severe SDB were confirmed by Defaye et al. [34] and may improve the initial diagnosis of OSA in the group of patients with permanent cardiac pacing.

The problem of an SDB-induced bradyarrhythmia incidence is strongly emphasized by the 2018 US [35] and the 2021 European [36] guidelines. Among patients with sinus bradycardia or AVB during sleep, it is recommended that the risk of SDB should be assessed and that polysomnography be performed if indicated (class I). According to the US guidelines, the diagnostics for SDB should also be considered in patients with a previously implanted PM who are at risk (class IIa).

At the same time, both guidelines [35, 36] pay attention to the high efficacy of PAP therapy, not only in reducing the incidence of respiratory events [37] but also in reducing CV risk [11–13]. The resolution of up to 80%–90% of bradyarrhythmias within three to six months after initiation of OSA treatment with continuous PAP therapy was confirmed by other authors [9, 10]. Additionally, an improvement in rhythm control with a 42% reduction in AF recurrence in patients treated with PAP has been documented [32], which reduces the need for multi-drug therapy and the risk of T-B syndrome. Thus, in patients with OSA-related bradycardia, it is recommended to implement PAP therapy with concomitant weight loss (class I in the United States) to avoid unnecessary PM implantation. In our presentation of 42 patients with bradyarrhythmias, seven (16.7%) had strong AASM recommendations to PAP therapy for OSA with REI ≥5 events/hour accompanied by increased daytime sleepiness, and an additional 25 (59.5%) had conditional recommendations to PAP therapy for OSA with REI ≥15 events/hour, regardless of symptoms [37]. However, when implementing such treatment for cardiac conduction diseases, it is worth remembering that adherence to PAP therapy is dependent on many factors and may be insufficient, especially among mildly symptomatic patients [38]. When SDB is treated unsystematically, bradycardia and drops in SpO₂ associated with respiratory events can form a dangerous duo and promote the incidence of ventricular arrhythmias [2, 3, 31].

Limitations of the study

Patient selection for the study was based on questionnaires assessing mainly OSA risk, and the study was conducted using the L3PST dedicated to the assessment of this SDB type. Thus, the group of patients with CSA was not adequately represented, and the results could not be extrapolated to patients with this type of SDB. Nevertheless, the percentage of patients with congestive heart failure for whom CSA was characteristic in the high-risk SDB group was only 7.1%. Unfortunately, because of the small female subgroup, caused by both the rarer incidence of SDB in females and the specificity of the surveys used, the data for males and females were analyzed together. Due to the relatively small number of cases of bradyar-rhythmias in the groups, the confidence intervals for the OR of nocturnal bradyarrhythmias in patients with severe SDB were wide, despite sufficient test reliability. Another limitation of the study was the lack of follow-up with patients with diagnosed OSA-related bradyarrhythmias suitable for PAP therapy.

CONCLUSIONS

The SDB, specifically OSA, is common in the population with CV diseases. It constitutes a risk factor for vagally-mediated nocturnal sinus bradycardia, advanced AVB, and pauses longer than 3 seconds. The severity of SDB determines the prevalence and number of these disorders. Due to the reversible nature of SDB-related bradyarrhythmias, each patient should be evaluated for SDB risk before deciding to implant a PM and, if warranted, should undergo polysomnography or at least L3PST and PAP therapy.

Article information

Conflict of interest: None declared.

Open access: This article is available in open access under Creative Common Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, allowing to download articles and share them with others as long as they credit the authors and the publisher, but without permission to change them in any way or use them commercially. For commercial use, please contact the journal office at kardiologiapolska@ptkardio.pl.

REFERENCES

- Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. Am J Respir Crit Care Med. 2001; 163(1): 19–25, doi: 10.1164/ajrccm.163.1.2001008, indexed in Pubmed: 11208620.
- Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol. 1983; 52(5): 490–494, doi: 10.1016/0002-9149(83)90013-9, indexed in Pubmed: 6193700.
- Gami AS, Olson EJ, Shen WK, et al. Obstructive sleep apnea and the risk of sudden cardiac death: a longitudinal study of 10,701 adults. J Am Coll Cardiol. 2013; 62(7): 610–616, doi: 10.1016/j.jacc.2013.04.080, indexed in Pubmed: 23770166.
- Salman LA, Shulman R, Cohen JB. Obstructive sleep apnea, hypertension, and cardiovascular risk: epidemiology, pathophysiology, and management. Curr Cardiol Rep. 2020; 22(2): 6, doi: 10.1007/s11886-020-1257-y, indexed in Pubmed: 31955254.
- Gottlieb DJ, Punjabi NM. Diagnosis and management of obstructive sleep apnea: a review. JAMA. 2020; 323(14): 1389–1400, doi: 10.1001/jama.2020.3514, indexed in Pubmed: 32286648.
- Peppard PE, Young T, Barnet JH, et al. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol. 2013; 177(9): 1006–1014, doi: 10.1093/aje/kws342, indexed in Pubmed: 23589584.
- Arzt M, Woehrle H, Oldenburg O, et al. Prevalence and predictors of sleep-disordered breathing in patients with stable chronic heart failure: the SchlaHF registry. JACC Heart Fail. 2016; 4(2): 116–125, doi: 10.1016/j. jchf.2015.09.014, indexed in Pubmed: 26682790.
- Somers VK, Dyken ME, Mark AL, et al. Parasympathetic hyperresponsiveness and bradyarrhythmias during apnoea in hypertension. Clin Auton

Res. 1992; 2(3): 171–176, doi: 10.1007/BF01818958, indexed in Pubmed: 1498563.

- Simantirakis EN, Schiza SI, Marketou ME, et al. Severe bradyarrhythmias in patients with sleep apnoea: the effect of continuous positive airway pressure treatment: a long-term evaluation using an insertable loop recorder. Eur Heart J. 2004; 25(12): 1070–1076, doi: 10.1016/j.ehj.2004.04.017, indexed in Pubmed: 15191779.
- Abe H, Takahashi M, Yaegashi H, et al. Efficacy of continuous positive airway pressure on arrhythmias in obstructive sleep apnea patients. Heart Vessels. 2010; 25(1): 63–69, doi: 10.1007/s00380-009-1164-z, indexed in Pubmed: 20091401.
- Marin JM, Carrizo SJ, Vicente E, et al. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. Lancet. 2005; 365(9464): 1046–1053, doi: 10.1016/S0140-6736(05)71141-7, indexed in Pubmed: 15781100.
- Myllylä M, Hammais A, Stepanov M, et al. Nonfatal and fatal cardiovascular disease events in CPAP compliant obstructive sleep apnea patients. Sleep Breath. 2019; 23(4): 1209–1217, doi: 10.1007/s11325-019-01808-4, indexed in Pubmed: 30848437.
- Wickwire EM, Bailey MD, Somers VK, et al. CPAP adherence reduces cardiovascular risk among older adults with obstructive sleep apnea. Sleep Breath. 2021; 25(3): 1343–1350, doi: 10.1007/s11325-020-02239-2, indexed in Pubmed: 33141315.
- Takegami M, Hayashino Y, Chin K, et al. Simple four-variable screening tool for identification of patients with sleep-disordered breathing. Sleep. 2009; 32(7): 939–948, indexed in Pubmed: 19639757.
- Pivetta B, Chen L, Nagappa M, et al. Use and performance of the STOP-Bang questionnaire for obstructive sleep apnea screening across geographic regions: a systematic review and meta-analysis. JAMA Netw Open. 2021; 4(3): e211009, doi: 10.1001/jamanetworkopen.2021.1009, indexed in Pubmed: 33683333.
- Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991; 14(6): 540–545, doi: 10.1093/sleep/14.6.540, indexed in Pubmed: 1798888.
- Rosen IM, Kirsch DB, Carden KA, et al. Clinical Use of a Home Sleep Apnea Test: An Updated American Academy of Sleep Medicine Position Statement. J Clin Sleep Med. 2018; 14(12): 2075–2077, doi: 10.5664/jcsm.7540, indexed in Pubmed: 30518456.
- 18. American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specification. Version 2.6. Am Acad Sleep Med 2020. Available online: American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specification. Version 2.6. Am Acad Sleep Med 2020. https://aasm.org/clinical-resources/scoring-manual/. [Access: September 2021].
- Steinberg JS, Varma N, Cygankiewicz I, et al. 2017 ISHNE-HRS expert consensus statement on ambulatory ECG and external cardiac monitoring/telemetry. Ann Noninvasive Electrocardiol. 2017; 22(3): e55–e96, doi: 10.1111/anec.12447, indexed in Pubmed: 28480632.
- Szymański FM, Płatek AE, Karpiński G, et al. Obstructive sleep apnoea in patients with atrial fibrillation: prevalence, determinants and clinical characteristics of patients in Polish population. Kardiol Pol. 2014; 72(8): 716–724, doi: 10.5603/KP.a2014.0070, indexed in Pubmed: 24671917.
- Gonzaga C, Bertolami A, Bertolami M, et al. Obstructive sleep apnea, hypertension and cardiovascular diseases. J Hum Hypertens. 2015; 29(12): 705–712, doi: 10.1038/jhh.2015.15, indexed in Pubmed: 25761667.
- Seiler A, Camilo M, Korostovtseva L, et al. Prevalence of sleep-disordered breathing after stroke and TIA: A meta-analysis. Neurology. 2019; 92(7): e648–e654, doi: 10.1212/WNL.00000000006904, indexed in Pubmed: 30635478.
- 23. Pływaczewski R, Bednarek M, Jonczak L, et al. Sleep-disordered breathing in a middle-aged and older Polish urban population. J Sleep Res. 2008;

17(1): 73–81, doi: 10.1111/j.1365-2869.2008.00632.x, indexed in Pubmed: 18275557.

- Pływaczewski R, Bieleń P, Bednarek M, et al. Influence of neck circumference and body mass index on obstructive sleep apnoea severity in males [article in Polish]. Pneumonol Alergol Pol. 2008; 76(5): 313–320, indexed in Pubmed: 19003760.
- Garrigue S, Pépin JL, Defaye P, et al. High prevalence of sleep apnea syndrome in patients with long-term pacing: the European Multicenter Polysomnographic Study. Circulation. 2007; 115(13): 1703–1709, doi: 10.1161/CIRCULATIONAHA.106.659706, indexed in Pubmed: 17353437.
- Velasco A, Hall C, Perez-Verdia A, et al. Association of high-risk scores for obstructive sleep apnea with symptomatic bradyarrhythmias. J Cardiovasc Med (Hagerstown). 2014; 15(5): 407–410, doi: 10.2459/JCM.0b013e3283630d07, indexed in Pubmed: 23867909.
- Zwillich C, Devlin T, White D, et al. Bradycardia during sleep apnea. Characteristics and mechanism. J Clin Invest. 1982; 69(6): 1286–1292, doi: 10.1172/jci110568, indexed in Pubmed: 7085875.
- Koehler U, Becker HF, Grimm W, et al. Relations among hypoxemia, sleep stage, and bradyarrhythmia during obstructive sleep apnea. Am Heart J. 2000; 139(1 Pt 1): 142–148, doi: 10.1016/s0002-8703(00)90321-1, indexed in Pubmed: 10618575.
- Fleg JL, Kennedy HL. Cardiac arrhythmias in a healthy elderly population: detection by 24-hour ambulatory electrocardiography. Chest. 1982; 81(3): 302–307, doi: 10.1378/chest.81.3.302, indexed in Pubmed: 7056104.
- Roche F, Xuong AN, Court-Fortune I, et al. Relationship among the severity of sleep apnea syndrome, cardiac arrhythmias, and autonomic imbalance. Pacing Clin Electrophysiol. 2003; 26(3): 669–677, doi: 10.1046/j.1460-9592.2003.00116.x, indexed in Pubmed: 12698666.
- Mehra R, Benjamin EJ, Shahar E, et al. Sleep Heart Health Study. Association of nocturnal arrhythmias with sleep-disordered breathing: The Sleep Heart Health Study. Am J Respir Crit Care Med. 2006; 173(8): 910–916, doi: 10.1164/rccm.200509-1442OC, indexed in Pubmed: 16424443.
- Mujović N, Marinković M, Mihajlović M, et al. Risk factor modification for the primary and secondary prevention of atrial fibrillation. Part 2. Kardiol Pol. 2020; 78(3): 192–202, doi: 10.33963/KP.15240, indexed in Pubmed: 32189488.
- Marti-Almor J, Marques P, Jesel L, et al. Incidence of sleep apnea and association with atrial fibrillation in an unselected pacemaker population: Results of the observational RESPIRE study. Heart Rhythm. 2020; 17(2): 195–202, doi: 10.1016/j.hrthm.2019.09.001, indexed in Pubmed: 31493591.
- 34. Defaye P, de la Cruz I, Martí-Almor J, et al. A pacemaker transthoracic impedance sensor with an advanced algorithm to identify severe sleep apnea: the DREAM European study. Heart Rhythm. 2014; 11(5): 842–848, doi: 10.1016/j.hrthm.2014.02.011, indexed in Pubmed: 24561163.
- 35. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS Guideline on the Evaluation and Management of Patients With Bradycardia and Cardiac Conduction Delay: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation. 2019; 140(8): e382–e482, doi: 10.1161/CIR.00000000000628, indexed in Pubmed: 30586772.
- Glikson M, Nielsen JC, Kronborg MB, et al. ESC Scientific Document Group. 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Eur Heart J. 2021;42(35): 3427–3520, doi: 10.1093/eurheartj/ehab364, indexed in Pubmed: 34455430.
- Patil SP, Ayappa IA, Caples SM, et al. Treatment of Adult Obstructive Sleep Apnea with Positive Airway Pressure: An American Academy of Sleep Medicine Clinical Practice Guideline. J Clin Sleep Med. 2019; 15(2): 335–343, doi: 10.5664/jcsm.7640, indexed in Pubmed: 30736887.
- Mehrtash M, Bakker JP, Ayas N. Predictors of continuous positive airway pressure adherence in patients with obstructive sleep apnea. Lung. 2019; 197(2): 115–121, doi: 10.1007/s00408-018-00193-1, indexed in Pubmed: 30617618.