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CLINICAL INVESTIGATIONS

The prevalence of obstructive sleep apnea in patients with atrial fibrillation

Asmaa M. Abumuamar[1] | Paul Dorian[2] | David Newman[3] | Colin M. Shapiro[4]

[1]Institute of Medical Science, Faculty of Medicine, University of Toronto, Toronto, Ontario

[2]Division of Cardiology, St. Michael's Hospital, University of Toronto, Toronto, Ontario

[3]Division of Cardiology, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, Ontario

[4]Department of Psychiatry, Toronto Western Hospital, University Health Network, University of Toronto, Toronto, Ontario

Correspondence
Asmaa M. Abumuamar, MD, MSc, The Sleep and Alertness Clinic, 790 Bay Street, Suite 800, Toronto, ON M5G 1N8 Canada
Email: asmaa.abumuamar@mail.utoronto.ca

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Background: Obstructive sleep apnea (OSA) is a systemic disorder associated with significant cardiovascular complications. OSA may play a role in the initiation and worsening of atrial fibrillation (AF). This study aimed to determine the prevalence and clinical predictors of OSA in patients with AF.

Hypothesis: OSA is underdiagnosed in a large number of patients with AF and may not be predicted by conventional clinical indices.

Methods: Consecutive nonselected patients with AF were recruited from different arrhythmia clinics in Toronto, Ontario, Canada. Patients with previous diagnosis and/or treatment of OSA were excluded. Patients underwent 2 consecutive nights of ambulatory sleep testing with full electroencephalogram recording. OSA was defined as an Apnea-Hypopnea Index (AHI) score ≥ 5 per hour of sleep.

Results: 123 patients with AF were recruited, with 100 patients included in the final analysis. OSA was detected in 85% of these patients. 27% of patients with normal overall AHI had an increased AHI during rapid eye movement sleep. Only age and male sex were independent predictors of the presence of OSA in these patients.

Conclusions: OSA is common and often undetected in patients with AF, especially in nonobese and/or female patients. Patients may have a normal overall AHI but an abnormal AHI during rapid eye movement sleep. The clinical relevance and therapeutic implications in this subgroup should be further investigated. The clinical features of OSA are not reliable predictors of OSA in patients with AF. A low threshold for detection of OSA, with sleep studies, in these patients may be merited.

KEYWORDS

ambulatory sleep testing, atrial fibrillation, obstructive sleep apnea, OSA predictors, OSA prevalence, OSA screening

1 | INTRODUCTION

Obstructive sleep apnea (OSA) is a potentially serious sleep disorder associated with significant economic and healthcare consequences. OSA is characterized by recurrent complete or partial upper-airway obstructions during sleep leading to an absent or reduced airflow associated with ineffective continuous respiratory effort.[1] These obstructive events are terminated by brain arousal and a surge of the sympathetic nervous system outflow. Thus, the pathophysiological correlates of OSA involve hypoxia, hypercapnia, sleep fragmentation, and the impact of repeated bursts of neurohumoral sympathetic tone.

Symptoms of OSA include loud snoring, episodes of apneas or choking, dry mouth, sore throat, and morning headache. Patients with OSA may experience excessive daytime sleepiness and fatigue, depression,[2] and attention deficit.[3] In addition, OSA is associated with adverse health consequences, including a high readmission rate of patients with heart failure compared with the general population.[4]

OSA affects 25% of middle-aged males and 9% of middle-aged females,[5] with an increased prevalence in patients with cardiovascular disorders.[6,7] The relationship between OSA and atrial fibrillation (AF) is complex and involves several metabolic, inflammatory, autonomic, and neurohumoral factors.[8] The prevalence of OSA in patients with AF is

variably reported. Holmqvist et al. reported OSA, based on medical records, in 18% of a large cohort of 10 132 patients with AF.[9] Gami et al detected OSA in 49% of patients with AF undergoing electrical cardioversion.[10] OSA has been recently recognized as a potential risk factor for the development and progression of AF.[11,12] The presence of comorbid HTN and obesity may constitute additional factors that influence the progression of AF in patients with OSA.[8] Independently of relevant covariates, patients with OSA have a higher incidence of AF after coronary artery bypass graft surgery.[13] In addition, OSA is associated with increased frequency of AF in patients with coronary artery disease,[13] heart failure,[14] and hypertrophic cardiomyopathy.[15] Moreover, moderate to severe OSA is a strong predictor of AF recurrence after electrical cardioversion[16] and ablation procedures.[17]

Given the negative impact of OSA on AF and the potential for a reduction in AF burden with the treatment of OSA,[8] we aimed to determine the prevalence of OSA in patients with AF presenting to outpatient specialized arrhythmia clinics and to investigate possible clinical predictors of OSA in this population.

2 | METHODS

2.1 | Study design

This is a prospective cohort study. Ethical approval was obtained from the University Health Network and St. Michael's Hospital research ethics boards. Male and female patients age > 18 years with documented AF were included. Exclusion criteria included previous diagnosis and/or treatment of OSA, a sleep study within 6 months prior to recruitment, and inability to provide an informed consent.

2.2 | Ambulatory sleep testing

Consecutive unselected patients were recruited from 2 major arrhythmia clinics in Toronto, Ontario, Canada. All patients, regardless of relevant symptoms or clinical features of OSA, underwent an unattended ambulatory sleep monitoring using a device recording system that allows for a self-applied home sleep study (Somté Polysomnography [PSG] system, v2, P/N: 8023–0001-02; Compumedics Ltd., Abbottsford, Victoria, Australia).

Participants underwent 2 consecutive nights of the ambulatory home sleep recording. The standard array of the recorded signals included electroencephalogram (EEG), electro-oculogram (EOG), electromyogram, electrocardiogram, airflow, snore, respiratory effort, body position, limb movement, oxygen saturation, pulse rate, and pulse waveform.

The EEG recording system was designed and validated by Neurozone MSH Inc. (Dundas, Ontario, Canada). The system used in this study allows for identical scoring as the standard PSG. M1 and M2 electrodes were placed over the left and right mastoid process, respectively. FZ, a reference for M1 and M2, was placed in the middle of the forehead. Left and right standard EOG channels were applied. The EOG channels were referenced to the contralateral mastoid site (M1 and M2). In addition, 2 standard chin electromyogram electrodes were applied over the mentalis and submentalis muscles. For leg muscle activity, 2 standard limb-movement sensors were placed symmetrically on each leg, 2 cm apart, over the tibialis anterior muscle. Modified limb II and V1 standard electrocardiogram electrodes were applied. A nasal pressure transducer was used to monitor airflow and snore. Dual thoraco-abdominal respiratory inductance plethysmography sensors were used to monitor respiratory effort during the study. A pulse oximeter probe, with a maximum acceptable signal averaging time of ≤3 seconds at a heart rate of 80 bpm, was used to monitor oxygen saturation. Moreover, the unit includes an internal position sensor that records patients' body position during sleep.

The Esprit Nova client access software (v1.1.402) was utilized for automatic analysis of PSG data. Following this analysis, the studies were manually scored and edited by a qualified sleep technologist. The scoring of each night was done independently, and scorers were blinded to patients' identification information and to the results of the first night of the home sleep test. The sleep studies were reviewed and approved by a sleep specialist. The standard American Academy of Sleep Medicine (AASM) criteria for scoring and interpretation of PSG data were followed. The following standard criteria were used for OSA diagnosis and severity classification[18]: (1) OSA is defined as an Apnea-Hypopnea Index (AHI) score ≥ 5 per hour of total sleep time (TST); (2) mild OSA is defined as an AHI score ≥ 5 and < 15/h TST; (3) moderate OSA is defined as an AHI score ≥ 15 and < 30/h TST; and (4) severe OSA is defined as an AHI score ≥ 30/h TST. The AHI scores from the 2 nights of home sleep testing were averaged to give a final AHI, which was used for classification of OSA in this study.

2.3 | Statistical analysis

The data were analyzed using SPSS statistical software, version 23 (IBM Corp., Armonk, NY). The differences between patients with and without a diagnosis of OSA were assessed by the independent sample t test for continuous variables and the χ^2 test for categorical variables. A multiple regression analysis was performed to test the predictive ability of a set of factors to detect the severity of OSA. Age, body mass index (BMI), sex, neck circumference, and hypertension (HTN) were used as independent factors, whereas the AHI representing the severity of OSA was the dependent variable. A binary logistic regression analysis was performed to assess independent predictors of the presence of OSA. The model included age, sex, BMI, neck circumference, and blood pressure as independent variables. The diagnosis of OSA (AHI ≥5/h) was the dichotomous dependent variable. We checked multicollinearity among the predictors using collinearity statistics to ensure that the variables do not have high correlations with each other in the model. We applied the forced entry methods that test all variables in 1 block while controlling for the effect of other predictors in the model. In another model, we replaced the continuous variable age with the categorical variable age > 50 years and replaced the BMI with BMI ≥30 kg/m^2 (obesity) and a second time with BMI >35 kg/m^2.

3 | RESULTS

3.1 | Sample size and population characteristics

A total of 123 patients were recruited. Of these, 19 patients refused to proceed with the sleep studies and 4 were unable to successfully

TABLE 1 Demographic characteristics of the study population

	Total, N = 123	Males, n = 85	Females, n = 38	Difference, *P* Value
Age, y	63.6 ± 13.3	62 ± 13	66 ± 13	0.1
Sex (%)	69 M, 31 F	69	31	0.000[a]
BMI, kg/m^2	28.7 ± 5.8	29 ± 5.4	28 ± 6.7	0.4
≥30 kg/m^2	34	31	41	0.3
>35 kg/m^2	12	12	12.5	0.9
Neck circumference, cm	39.7 ± 3.7	40.96 ± 3.2	36.37 ± 3	0.000[a]
>42 cm in males or > 41 cm in females	20	28	3	0.005[a]

Abbreviations: BMI, body mass index; F, female; M, male; SD, standard deviation. Data are presented as % of total or mean ± SD. The independent sample *t* test was used to compare means of continuous variables, and the χ^2 test was used to compare proportions of categorical variables.

[a] Denotes significance.

apply or use the home sleep apparatus. The final analysis included 100 patients (70 males, 30 females).

Demographic characteristics of the study sample are shown in Table 1. Patients were categorized, based on their BMI, as 2% underweight, 27% normal weight, 37% overweight, 22% obese, and 12% morbidly obese. Thirty percent of patients had permanent AF and 70% had paroxysmal AF (ie, normal sinus rhythm) at the time of the sleep study.

Clinical characteristics of the study population are shown in Table 2. The use of medications is shown in Table 3, which shows the use of β-blockers as the only main difference (87% in OSA vs 13% in non-OSA; *P* = 0.007).

3.2 | The prevalence and severity of OSA in patients with AF

OSA was detected in 85% of patients (N = 100) and was more common in males than in females (91% vs 70%, respectively; *P* = 0.006). Patients with OSA were older than patients without OSA (65 ± 13 years and 55 ± 14 years, respectively; *P* = 0.009). In addition, patients with OSA had a significantly higher BMI than did patients without OSA (29 ± 6 kg/m^2 vs 25 ± 4 kg/m^2, *P* = 0.008). Patients with OSA had a higher prevalence of obesity (defined as a BMI ≥ 30 kg/m^2) compared with patients without OSA (40% vs 8%; *P* = 0.02). For the prevalence of OSA with age subgroup and sex comparisons, see Table 4.

In all patients with OSA, the mean AHI was 23 ± 16/h (range, 5/h–90/h). Mild OSA was detected in 45% of patients, moderate OSA in 27%, and severe OSA in 28% of patients. Severity categories among females with OSA (n = 21) were 52% mild, 19% moderate, and 29% severe. In males with OSA (n = 64), 42% had mild, 30% had moderate, and 28% had severe OSA. There was no significant difference in the average AHI between male and female patients with OSA. For the average AHI with age subgroup and sex comparisons, see Table 5.

TABLE 2 Clinical characteristics of the study sample with comparison between patients with and without OSA

	Total, N = 107	OSA, n = 85	Non-OSA, n = 15	Difference, *P* Value
HTN	50	53	27	0.06
DM	10	12	7	0.5
CAD	6	7	7	0.9
History of stroke	9	9	0	0.2
HF	3	3	0	0.4
Hyperlipidemia	42	42	40	0.8
Cardiomyopathy	2	2	0	0.6
Brugada syndrome	1	1	0	0.6
Pacemaker insertion	3	4	0	0.4
Previous AF/flutter ablation	19	19	27	0.4
Cardiac valve replacement	2	2	0	0.5
Depression	9	9	6	0.2
Anxiety	4	3	7	0.2
Bronchial asthma	3	3	0	0.4

Abbreviations: AF, atrial fibrillation; CAD, coronary artery disease; DM, diabetes mellitus; HF, heart failure; HTN, hypertension; OSA, obstructive sleep apnea. Data are presented as % of total. The χ^2 test was used to compare proportions of categorical variables.

TABLE 3 Medications of study participants with a comparison between patients with and without OSA

	Total, N = 100	OSA, N = 85	Non-OSA, N = 15	*P* Value
Antidiabetic medication	9	9	7	0.7
Anticoagulant	71	73	20	0.000[a]
ASA	20	16	27	0.2
Digoxin	7	8	0	0.2
β-Blocker	50	87	13	0.007[a]
ACEI	18	20	0	0.05
ARB	14	12	13	0.08
CCB	30	29	20	0.5
Diuretic	10	11	0	0.1
Statin	42	42	20	0.1
Antiarrhythmic	31	29	20	0.4
Benzodiazepine	7	5	13	0.1
Antidepressant	9	7	6	0.2

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; ASA, acetylsalicylic acid (aspirin); CCB, calcium channel blocker; OSA, obstructive sleep apnea. Data are presented as % of total. The χ^2 test was used to compare proportions of categorical variables.

[a] Denotes significance.

TABLE 4 Prevalence of OSA among patients with arrhythmia including age and sex comparisons

	Prevalence of OSA (AHI ≥5/h)			
	Total	Males	Females	*P* Value
All age groups	85 (N = 100)	91 (n = 70)	70 (n = 30)	0.006[a]
Age, y				
≥60	92 (n = 71)	94 (n = 48)	87 (n = 23)	0.3
<60	69 (n = 29)	86 (n = 22)	14 (n = 7)	0.000[a]
40–59	71 (n = 21)	88 (n = 16)	20 (n = 5)	0.004[a]
≥40	87 (n = 92)	92 (n = 64)	75 (n = 28)	0.02[a]
<40	63 (n = 8)	83 (n = 6)	0 (n = 2)	0.03[a]

Abbreviations: AHI, apnea-hypopnea index; OSA, obstructive sleep apnea. Data are presented as % of total. The χ^2 test was used to compare proportions of categorical variables.

[a] Denotes significance.

3.3 | REM-related apnea

The average AHI during rapid eye movement (REM) sleep for 98 patients was 22 ± 20/h (range, 0–79/h). The mean REM AHI for females was 22 ± 23/h (n = 29), whereas males had mean REM AHI of 22 ± 19/h (n = 69). There was no significant difference in the average REM AHI between males and females.

Of patients who went into REM sleep (n = 98), 81% had an AHI ≥5/h during REM sleep (REM-related apnea), including 79% of females and 84% of males. Of patients with REM-related apnea, 95% had an overall AHI ≥5/h (ie, diagnosed with OSA). Therefore, 5% of patients with REM-related apnea had a normal overall AHI. On the other hand, 27% of patients without a diagnosis of OSA (n = 15) had REM-related apneas despite a normal overall AHI.

3.4 | Clinical predictors of OSA in patients with AF

Multiple regression analyses showed that age and BMI were independent predictors of the AHI (*P* = 0.03 and *P* = 0.000, respectively). Direct logistic regression was performed to assess the ability of several factors to predict the presence of OSA (AHI ≥5/h) as described in the Methods section. The result was significant for age (*P* = 0.013), indicating that age as a continuous variable was an independent predictor of OSA (odds ratio [OR]: 1.05, 95% confidence interval: 1.01–1.09) even after adjusting for sex and BMI (*P* = 0.009). As a binary variable, age ≥ 60 years was an independent predictor of OSA (OR: 12, *P* = 0.003). In addition, male sex was a predictor of OSA (OR: 4.57, 95% confidence interval: 1.4–14.3, *P* = 0.009). Males had 5× higher risk of having OSA than did females, even after adjusting for age and BMI (*P* = 0.008).

TABLE 5 Average AHI among study participants with age and sex comparisons

	Average AHI/h			
	Total	Males	Females	Difference, *P* Value
All age groups	20 ± 17	22 ± 17	15 ± 14	0.05
Age, y				
≥60	22 ± 17	24 ± 19	17 ± 13	0.09
<60	14 ± 13	16 ± 12	7 ± 14	0.1
40–59	16 ± 14	17 ± 13	10 ± 16	0.2
≥40	21 ± 17	23 ± 14	16 ± 14	0.06
<40	9 ± 9	12 ± 8	0.45 ± 0.14	0.1

Abbreviations: AHI, apnea-hypopnea index; SD, standard deviation. Values are expressed as mean ± SD. Independent sample *t* test was used to compare means between groups.

On the other hand, BMI was not a predictor of OSA after adjusting for age and sex. Moreover, BMI ≥30 kg/m^2, BMI >35 kg/m^2, neck circumference, and HTN were not predictors of OSA.

4 | DISCUSSION

4.1 | The prevalence of OSA

This is the first study to assess consecutive patients referred to an arrhythmia clinic and found that the prevalence of OSA in patients with AF in this study was 85%, which is higher than previously reported.[9,10] The prevalence of OSA was 91% in males and 70% in females, which also indicates a higher prevalence of OSA in both males and females than previously reported in the literature.[19]

Age subgroup analyses detected increasing prevalence of OSA with advancing age in patients with AF, similar to previous findings in general population.[20] The prevalence of OSA was 69% in people age < 60 years, compared with 92% in people age > 60 years. In addition, patients age > 60 years had a higher AHI compared with patients age < 60 years. Increasing prevalence and severity of OSA in the older age group of patients with AF may be attributed to the effects of advanced age on the structure and function of the upper airways. These effects include atrophy of pharyngeal muscles, increased fat deposition, and lengthening of the soft palate, which all make the collapse of the upper airways during sleep more likely.[21] However, the prevalence was still high in patients age < 40 years (63%), which may indicate the presence of factors other than age that may increase the prevalence of OSA in younger patients with AF.

In this study, sex and age subgroup comparisons among patients with AF showed that males have a higher prevalence of OSA than females across all age groups. However, there was no significant difference in the prevalence of OSA between males and females age > 60 years, which may indicate protective hormonal factors in premenopausal females. Previous studies showed that the prevalence and severity of OSA in females increase after menopause.[22–24] Our finding correlates with previous findings and may suggest that menopause may be a risk factor for OSA. The role of hormone replacement therapy in the reduction of the risk of OSA is still unknown. Some studies showed a reduction in the prevalence of OSA among treated postmenopausal females[22,25]; however, other studies showed no statistically significant difference in the risk of OSA between postmenopausal females on hormone therapy and those not on hormone therapy.[26] We did not study the use of hormone therapy among females in this population. Future research may be needed to evaluate the role of treatment.

Further, there was no significant difference in the mean BMI between male and female patients with OSA. Therefore, BMI did not appear to contribute to the difference in OSA prevalence

between males and females in this population. Moreover, the severity of OSA increased with increased age and BMI. The correlation between sex and AHI disappeared after adjusting for age and BMI, suggesting that male and female AF patients, without significant difference in the BMI, had the same level of OSA severity across all age groups.

4.2 | Body mass index

Obesity is a well-known risk factor for the development and progression of OSA.[5] Approximately 60% of patients with moderate and severe OSA, and of those referred for a sleep study, are overweight.[27] Excess deposition of fat reduces the upper-airway diameter and increases the risk of OSA.[28] There is a clinical impression that a rapid weight gain is more associated with incident OSA; however, there have been no formal studies concerning this point. Evidence supports the notion that weight loss may decrease the severity of OSA in obese patients.[29] A reduction of the prevalence of OSA from 71% to 44% was reported 1 year after bariatric surgery. In addition, the severity of OSA decreased in 78% of patients. However, moderate and severe OSA persisted in 20% of patients.[30]

In our study population, obesity (BMI ≥30 kg/m^2) was present in 40% of patients with detected OSA. However, OSA was prevalent in 97% of obese and 80% of nonobese patients. The BMI correlated with the AHI. However, obesity was not a predictor of OSA. This may indicate that other risk factors play a role in the development of OSA in this group, including age,[21] sex,[31] family history of OSA/genetic factors,[32] craniofacial structural abnormalities,[33] smoking[34] and alcohol consumption,[35] and hormonal disruptions.[22] Our findings suggest that evaluation for the presence of OSA in AF patients may be warranted, regardless of obesity.

4.3 | Under-recognition of OSA in patients with AF

The high prevalence of OSA detected in this study may indicate that OSA is under-recognized in patients with AF. It should be noted that these patients were consecutively recruited from arrhythmia clinics and are not presenting with sleep complaints to a sleep clinic. In addition, our results suggest that OSA may be underestimated in nonobese and in female patients with AF. Several factors may play a role in the under-recognition of OSA in this specific population. Previous studies showed that patients with cardiac disorders often do not display the usual signs and symptoms of OSA.[36] In addition, there is no formal screening tool for OSA applied as a part of routine management of patients with cardiac arrhythmia. Another general factor is the challenges associated with obtaining PSG, the gold-standard method for diagnosis of OSA. In addition, the diagnosis of OSA often requires a referral by a primary-care physician to a sleep specialist.

There are valid reasons to consider detection of OSA as a prelude to treatment in patients with AF. OSA may have detrimental effects on the prognosis of AF. OSA has been found to increase the severity of symptoms and the rate of hospitalization of AF patients, and to decrease the response to antiarrhythmic medications compared with patients without OSA.[9] In addition, OSA has been found to increase the recurrence of AF after catheter ablation.[17] It has been recommended that OSA should be evaluated with PSG prior to surgical ablation.[37] Early detection and treatment of OSA may decrease the recurrence and severity of AF.[9]

To address the challenges of in-laboratory PSG, the use of portable sleep monitors is becoming more popular. To be comparable to the gold-standard PSG, sleep-monitoring devices should provide full sleep data, including EEG monitoring. The EEG recording provides analysis of sleep architecture and estimates the total sleep time and intervening awakenings. These parameters are important for an accurate estimation of the AHI, which represents the severity of OSA.

We detected 27% of patients with a normal overall AHI who have an elevated AHI during REM sleep. These patients, in particular, would be missed without an EEG-based sleep study. REM sleep is characterized by decreased ventilatory response to hypoxia and hypercapnia, periods of decreased respiratory rate, and motor suppression of respiratory and nonrespiratory muscles. These changes lead to increased upper-airway resistance and increase the severity of respiratory events during REM sleep. In addition, REM sleep is characterized by bursts of sympathetic nervous system activity, which may increase the risk of cardiovascular complications including arrhythmia and myocardial infarction. OSA during REM sleep has been linked to poor glycemic control.[38] Therefore, treatment of OSA should cover those periods during REM sleep, which increase progressively during sleep cycles. Future research is needed to investigate the clinical relevance and the therapeutic implications in patients with increased AHI during REM sleep but normal overall AHI, particularly in patients with cardiovascular comorbidities.

In this study, the use of a portable sleep-monitoring device with EEG recording provided an accurate estimation of the prevalence of OSA in patients with AF. Ambulatory sleep testing permitted screening of a large number of patients with AF for OSA, regardless of subjective symptoms or objective signs of OSA in this population. Ambulatory sleep testing may provide an affordable and practical solution to aid in the early detection and treatment of OSA.

4.4 | Study limitations

This study was subjected to selection and observational bias. Although consecutive nonselected patients were recruited, these patients were recruited from specialized arrhythmia clinics with generally younger patients, and 19% with prior ablation. The generalizability of our findings warrants further study. Similar to any sleep laboratory dataset, a number of home sleep recordings were reported as inconclusive or technically inadequate. In our sample, this occurred in 3% of subjects. In addition, we used a nasal pressure transducer (with square root transformation of the signal) to monitor airflow, which is limited to nasal airflow, leaving mouth flow undetected. The AASM recommends the use of a nasal pressure transducer (with or without square root transformation of the signal) for identification of hypopneas and as an alternative to oronasal thermal sensors for detection of apneas. The use of both oronasal thermal sensors and nasal pressure devices may yield more accurate results.

5 | CONCLUSION

OSA is very common and underdetected in both male and female patients with AF. The only independent predictors of OSA were age and male sex. A low threshold for evaluation of OSA in patients with AF may be merited irrespective of the BMI. Application of a technically adequate device for home sleep testing is recommended. This may provide an alternative reliable and affordable method for early detection of OSA in patients with AF.

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CONFLICTS OF INTEREST

Dr. Colin Shapiro has shares in Neurozone MSH Inc., the company that provided the sleep testing system. The authors declare no other potential conflicts of interest.

ORCID

Asmaa M. Abumuamar http://orcid.org/0000-0002-0994-7976

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