Effect of Widespread Sleep Apnea Screening on Progression of Atrial Fibrillation

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Sleep apnea (SA) is recognized as a predictor of incident atrial fibrillation (AF) and AF recurrence after treatment. However, data on the prevalence of SA phenotypes in patients with AF and the effect of widespread SA screening on AF outcomes are scarce. We conducted a retrospective study of patients with AF referred for SA testing between March 2018 and April 2020. The screening was performed using home sleep testing or polysomnography. AF outcomes were examined by assessment of AF progression as defined by a change from paroxysmal AF to persistent AF, change in antiarrhythmic drug, having an ablation or cardioversion. Of 321 patients evaluated for AF, 251 patients (78%) completed SA testing. A total of 185 patients with complete follow-up data and SA testing were included in our analysis: 172 patients (93%) had SA; 90 of those (49%) had primarily obstructive sleep apnea, 77 patients (42%) had mixed apnea, and 5 patients (3%) had pure central apnea. Time from AF diagnosis to SA testing was associated with AF progression; after 2 years, the risk of AF progression increased ($p < 0.008$). Continuous positive airway pressure treatment did not affect AF progression ($p = 0.99$). In conclusion, SA is highly prevalent in an unselected population of patients with AF, with mixed apnea being present in over 40% of the population. Early SA testing was associated with decreased rates of AF progression, likely because of earlier and potentially more aggressive pursuit of rhythm control. © 2022 Published by Elsevier Inc. (Am J Cardiol 2022;00:1−7)

Atrial fibrillation (AF) is the most common arrhythmia worldwide and currently affects approximately 46 million people.[1,2] Sleep apnea (SA) has been identified as an independent predictor of incident AF. Those with severe obstructive sleep apnea (OSA) have a twofold to a fivefold higher risk of developing AF, and episodes of SA have been temporally linked to AF recurrences.[3−7] OSA has been associated with reduced efficacy of cardioversion and antiarrhythmic medications and increased AF recurrence after catheter ablation.[8−15] The American Heart Association, American College of Cardiology and Heart Rhythm Society recommend SA testing if suspected clinically in patients with AF.[16] Implementation of this guidance, however, is challenging given that patients with AF often do not manifest typical symptoms of SA.[17] Previous studies examining the prevalence of SA in AF relied on screening questionnaires such as the snoring, tiredness, observed apnea, high blood pressure, body mass index (BMI), age, neck circumference, and male gender (STOP-Bang) questionnaire.[18] However, because patients with AF often do not manifest symptoms of SA, current data may underestimate the prevalence in patients with AF. In this study, we retrospectively analyzed the prevalence and distribution of SA phenotypes in an unselected population of patients with AF and examined the effect of widespread screening of SA on the progression of AF.

Methods

Beginning in March 2018, patients referred to the Arrhythmia Clinic for management of AF were referred for SA evaluation. To facilitate widespread SA screening, a clinical pathway was developed in conjunction with Sleep Medicine. This included the creation of an order set for sleep testing in the electronic medical record and automatic notification of referrals and results to select individuals within Sleep Medicine and Electrophysiology. Entry of this order set would alert a designated electrophysiology nurse practitioner who would facilitate insurance approval for home sleep testing (HST). Testing would be interpreted by one of several designated Sleep Medicine clinicians, with results then routed to the electrophysiology nurse practitioner, who would ensure communication of results to the patient and facilitate referral to Sleep Medicine if appropriate. Patients with AF who successfully completed sleep testing between March 2018 and April 2020 were included in this analysis. This study was approved by the Beth Israel Deaconess Medical Center Institutional Review Board.

Screening was performed primarily using HST. A subset of patients with inconclusive HST or those recommended for

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additional testing based on HST results were then referred for overnight polysomnography (PSG). Based on the sleep study results, patients were categorized as having primarily OSA, primarily central sleep apnea (CSA), mixed SA, or no SA. OSA was defined as recurrent episodes of partial or complete airway obstructions with thoracoabdominal effort, whereas CSA was defined as the complete cessation of breathing with no thoracoabdominal effort.[6] The apnea-hypopnea index (AHI 4%) was used to denote severity and is defined as an oxygen desaturation >4% lasting for at least 10 seconds. Patients with mixed apnea displayed characteristics of OSA and a central AHI ≥5/hour of sleep or features of periodic breathing. The severity of OSA was categorized based on previously published guidelines with mild OSA having AHI 4% of 5 to 14 events/hour, moderate OSA with AHI 4% 15 to 29 events/hour, and severe OSA with AHI 4% ≥30 events/hour.[19] For patients who completed both HST and PSG, we used the PSG results for analysis.

We evaluated the risk of AF progression in those with SA. AF progression was defined as 1 or more of the following: a change from paroxysmal AF to persistent AF, a change in antiarrhythmic drug, having an ablation, or having cardioversion at any time after the date of SA testing during follow-up. Demographics, co-morbidities,

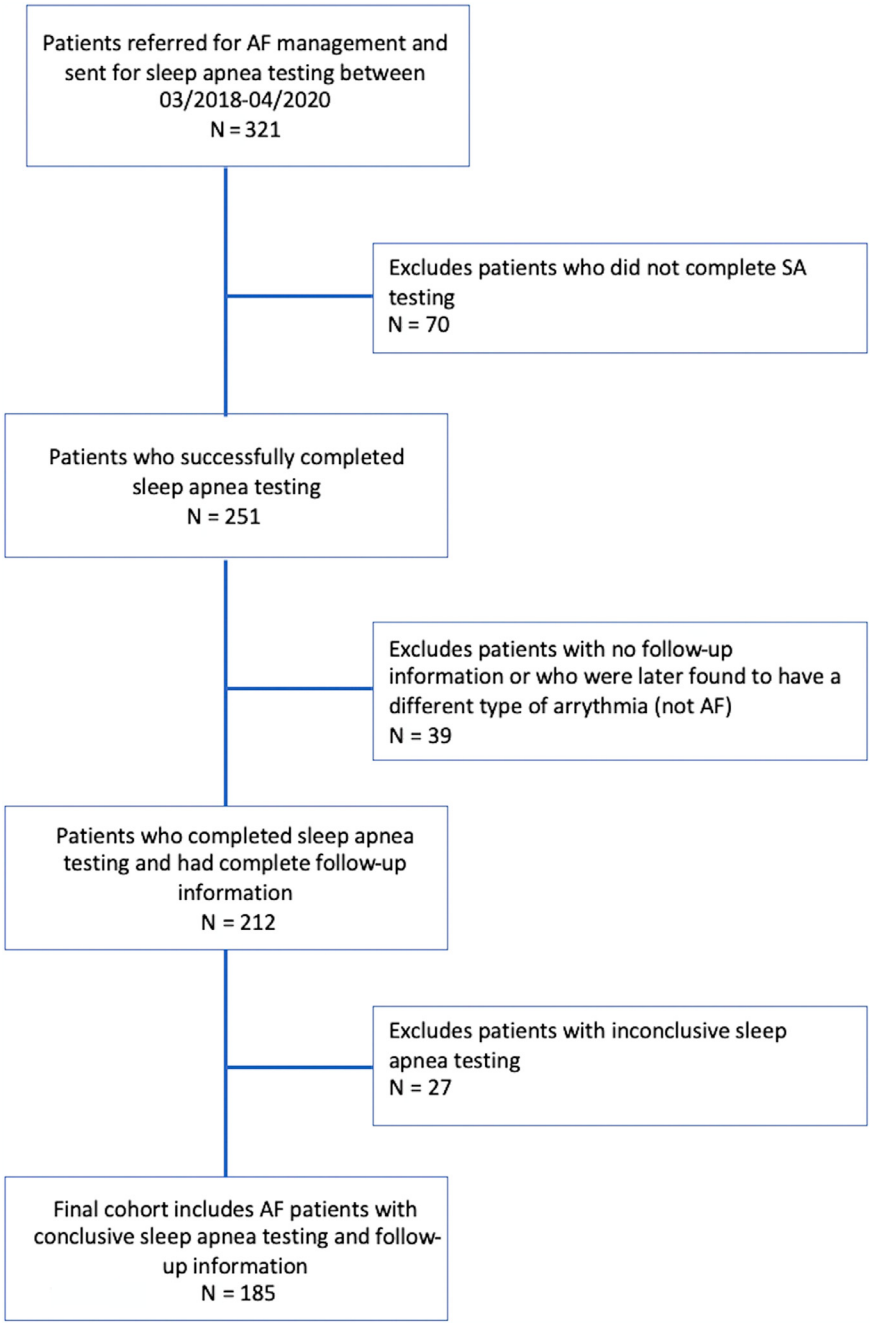

Figure 1. Study cohort.

echocardiographic data, details of AF history and management, and HST and PSG reports were obtained through manual review of the electronic medical records. The end of our follow-up period was August 1, 2021. Data are presented as number (%) or median and interquartile range. Categorical outcomes were compared using chi-square or Fisher's exact test. Continuous variables were compared using Wilcoxon rank sum test. All tests were two-sided, and $p < 0.05$ was considered statistically significant. Analyses were performed using SAS 9.4 (SAS Institute Inc, Cary, North Carolina).

Results

From March 2018 to April 2020, 251 of 321 patients with AF referred for SA evaluation successfully completed SA testing. Of these, 212 had complete follow-up information, and 27 of these 212 patients had inconclusive results on HST and were excluded from the final analysis. Thus, our final cohort included 185 patients with AF with conclusive SA testing and complete follow-up information (Figure 1).

Of the 185 patients, 93% had some form of SA, 49% had OSA primarily, and 42% had mixed apnea. Only 7% of patients had no SA. The median age was 64 years, and most of our cohort were male (Table 1). In those with OSA, 66% were male compared with 81% male in those with mixed SA. Patients with mixed and central apnea had a higher BMI and AHI 4% than those with OSA. Additionally, in those who underwent PSG, the minimum oxygen saturation was lower for central and mixed apnea compared with OSA. HST diagnosed a higher percentage of OSA of any severity than PSG (47% compared with 30% overall) (Figure 2). In contrast, relative to PSG, HST underestimated the prevalence of mixed and CSA, but both tests had similar rates of no SA diagnosis. Of those diagnosed with any type of SA, 67% (all patients with moderate or greater OSA) were referred to Sleep Medicine for initiation of continuous positive airway pressure (CPAP). Patients with central and mixed sleep apnea had higher rates of CPAP initiation than patients with OSA (Table 2).

In our cohort of patients with SA, 43% of patients had no progression of AF during follow-up, whereas 57% experienced AF progression. Of the patients with progression of AF, 41% progressed from paroxysmal to persistent AF, 39% changed antiarrhythmic drugs, 47% underwent cardioversion, and 28% underwent ablation (Figure 3). We found that increased time from AF diagnosis to SA diagnosis was associated with AF progression for all patients with SA ($p < 0.008$) (Table 3). Patients with SA who had sleep testing more than 2 years after AF diagnosis were more likely to have AF progression than those who underwent SA testing within 2 years of AF diagnosis ($p < 0.008$). Because of the very small number of patients in our cohort with primarily CSA, these patients were combined with the mixed apnea group for analysis. In the unadjusted analysis, this association was significant for

Table 1
Study demographics

Variables	All patients (N=185)	No sleep apnea (N=13)	Obstructive sleep apnea (N=90)	Mixed apnea (N=77)	Pure central apnea (N=5)
Age (years)	64 (58 − 71)	61 (56 − 65)	63 (57 − 71)	68 (60 − 71)	71 (65 − 72)
Male	133 (72%)	8 (62%)	59 (66%)	62 (81%)	4 (80%)
Female	52 (28%)	5 (39%)	31 (34%)	15 (20%)	1 (20%)
Body mass index (kg/m^2)	29 (26− 34)	27 (24 − 30)	29 (25 − 33)	30 (27 − 34)	30.2 (28 − 30)
Coronary artery disease	11 (6%)	1 (8%)	3 (3%)	7 (9%)	0 (0%)
Chronic kidney disease	9 (5%)	1 (8%)	5 (6%)	3 (4%)	0 (0%)
Type II diabetes	10 (5%)	0 (0%)	6 (7%)	4 (5%)	0 (0%)
Hypertension	70 (38%)	5 (39%)	30 (33%)	33 (43%)	2 (40%)
stroke	2 (1%)	1 (8%)	1 (1%)	0 (0%)	0 (0%)
Heart failure with reduced ejection Fraction (EF ≤40%)	8 (4%)	0 (0%)	3 (3%)	5 (7%)	0 (0%)
Atrial fibrillation type at diagnosis					
Paroxysmal	153 (83%)	10 (77%)	79 (88%)	59 (77%)	5 (100%)
Persistent	32 (17%)	3 (23%)	11 (12%)	18 (23%)	0 (0%)
Echocardiographic Data					
Ejection Fraction (%)	59 (55% − 65%)	60 (55% − 65%)	57 (55%− 65%)	60 (55% − 65%)	63 (50% − 70%)
Left Atrium 4 chamber diameter (cm)	5 (5 − 6)	6 (5 − 6)	5 (5 − 6)	5 (5 − 6)	6 (3 − 6)
Sleep Testing Data					
Apnea hypopnea index 4%	12 (7 − 25)	1 (1 − 2)	9 (5 − 14)	23 (16 − 34)	51 (33 − 62)
Minimum oxygen saturation	87 (84% − 90%)	91 (91% − 92%)	89 (85% − 90%)	86 (81% − 88%)	84 (73% − 85%)
Mild obstructive sleep apnea	-	-	68 (75%)	-	-
Moderate obstructive sleep apnea	-	-	16 (18%)	-	-
Severe obstructive sleep apnea	-	-	6 (7%)	-	-

Data presented as median (interquartile range) or number (%)

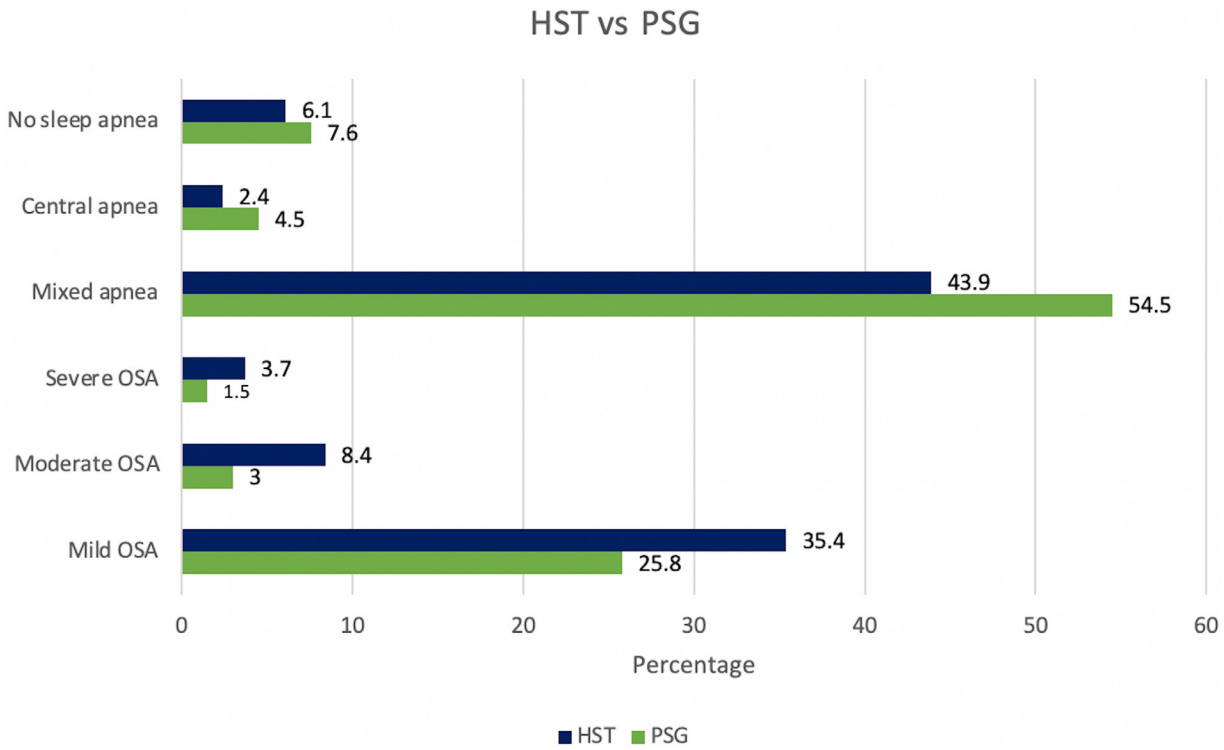

Figure 2. Home sleep testing versus polysomnography sleep testing results. Comparing HST and polysomnography results for types and severity of sleep apnea.

patients with mixed apnea but not OSA. When adjusted for age, gender, BMI, and hypertension, the association was borderline significant for the mixed apnea group. Male gender, age, BMI, hypertension, left ventricular hypertrophy and left atrial diameter were not associated with AF progression (Table 3). Additionally, CPAP initiation did not significantly affect AF progression (p = 0.99) in patients with SA.

Table 2
Current atrial fibrillation and sleep apnea management by sleep apnea type

Variable	No sleep apnea (N=13)	Obstructive sleep apnea (N=90)	Mixed apnea (N=77)	Central apnea (N=5)	p-Value
Atrial fibrillation Management					
Beta blockers use	8 (62%)	59 (66%)	59 (77%)	5 (100%)	0.18
Calcium channel blockers use	1 (8%)	13 (14%)	6 (8%)	0 (0%)	0.49
Antiarrhythmic drug use	5 (39%)	28 (31%)	28 (36%)	2 (40%)	0.66
Amiodarone	0 (0%)	7 (25%)	10 (36%)	0 (0%)	
flecainide	4 (80%)	12 (43%)	11 (39%)	1 (50%)	
Sotalol	0 (0%)	5 (18%)	3 (11%)	0 (0%)	
dofetilide	0 (0%)	1 (4%)	3 (11%)	0 (0%)	
Propafenone	0 (0%)	1 (4%)	1 (4%)	0 (0%)	
Other/unknown	1 (20%)	2 (7%)	0 (0%)	1 (50%)	
Direct oral anticoagulant	11 (85%)	72 (80%)	60 (78%)	2 (40%)	0.01
Vitamin K antagonist	0 (0%)	5 (6%)	8 (10%)	0 (0%)	0.42
Any Ablation	6 (46%)	40 (44%)	38 (49%)	4 (80%)	0.46
Any Cardioversion	7 (54%)	34 (38%)	39 (51%)	1 (20%)	0.35
Sleep Apnea Management					
CPAP prescription	-	45 (50%)	64 (83%)	4 (80%)	0.003
Sleep medication	-	1 (1%)	11 (14%)	2 (40%)	0.002

CPAP = continuous positive airway pressure.
Data presented as number (%).
*Data obtained from PSGs only.

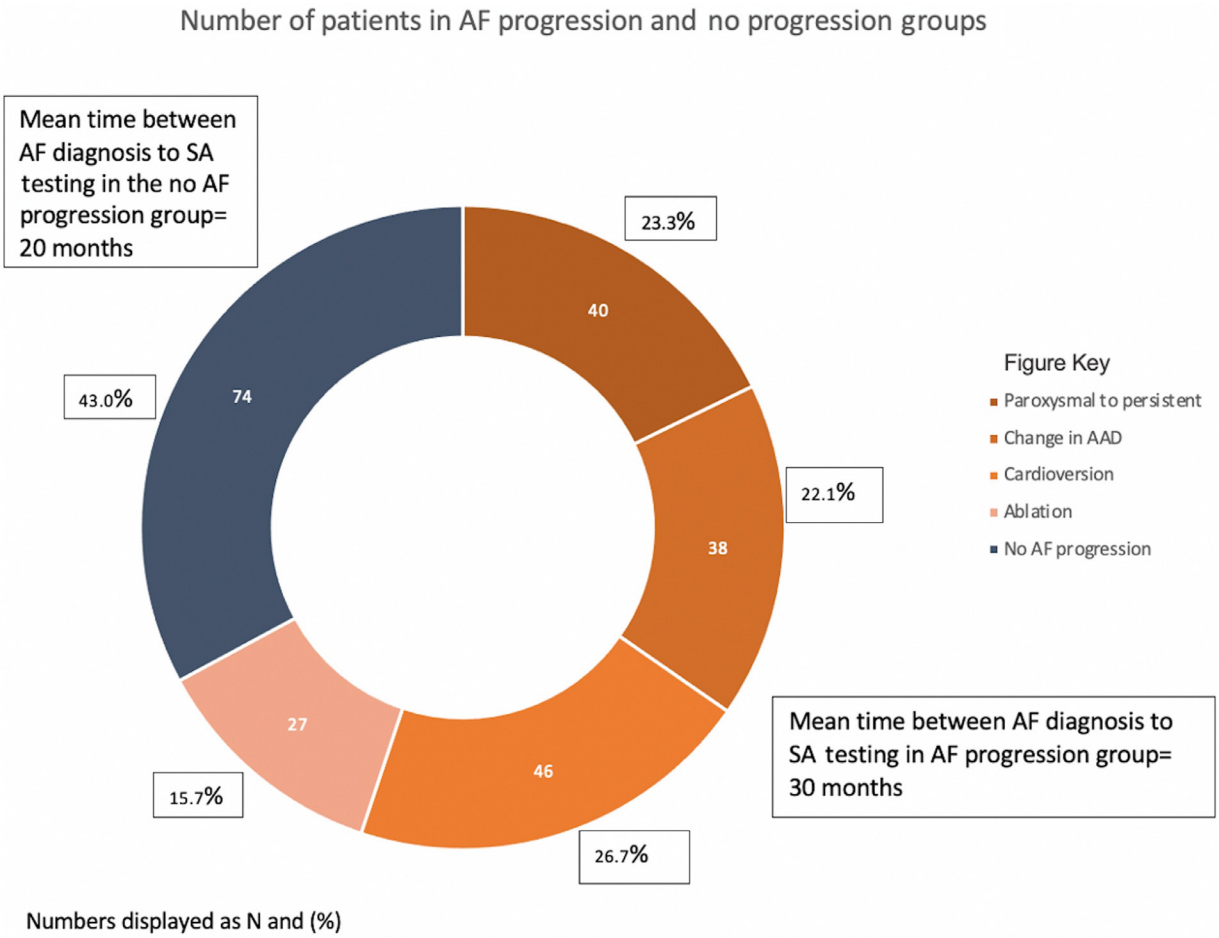

Figure 3. Atrial fibrillation progression proportions in the sleep apnea population. The breakdown for AF progression in each criterion evaluated, including change of atrial fibrillation type from paroxysmal to persistent, change in antiarrhythmic drugs, having cardioversion, or an ablation. AAD = antiarrhythmic drug.

Discussion

In this study, we found an exceedingly high prevalence of SA in an unselected population of patients with AF who underwent SA screening as part of a general management strategy for AF. Overall, 93% of patients in our cohort had some form of SA. Traaen et al[20] reported a prevalence of >80% in their cohort of patients with AF. A meta-analysis found a pooled prevalence of 78%, with individual prevalence ranging from 46% to 92%.[3] Kadhim et al[3] characterized the prevalence of sleep-disordered breathing in different AF subpopulations, with persistent AF having a prevalence of 83% (95% confidence interval [CI] 72% to 93%) and mixed AF having a prevalence of 76% (95% CI 65% to 86%). The higher prevalence of sleep-disordered breathing in our study may be because we did not rely on the presence of SA symptoms or positive questionnaire responses as a requirement for testing. Several previous studies investigating the prevalence of sleep-disordered breathing in patients with AF used questionnaires such as STOP-Bang, designed to evaluate for OSA or self-reported symptoms.[3] This is especially problematic in patients with AF as it has been reported that patients with AF with sleep-disordered breathing exhibit nonspecific symptoms or are asymptomatic.[17]

Our study also documented a significant proportion of patients with AF with mixed apnea. Most previous studies linking SA with AF have identified OSA as a predictor for both incident AF and AF recurrence after treatment. Still, few studies have linked mixed or predominantly CSA and AF. This is likely due in part to the method of testing. SA questionnaires are limited in their ability to assess for respiratory effort, a hallmark of CSA.[21] Using HST and PSG to screen for SA allowed us to better phenotype sleep-disordered breathing, and we found that mixed and complex SA was highly prevalent in our cohort of patients with AF. Although computational methods of endotyping high loop gain, the driver of hypocapnic CSA, would have been more precise,[22–25] these were not available, and it is unlikely that significantly more high loop gain apnea would have been detected given the already high prevalence detected by conventional methods. Although the prevalence of CSA in heart failure patients is well-documented, there are limited prevalence estimates in patients with AF, with a handful of studies finding CSA to be an independent predictor of incident AF.[21,26] One study reported a 16-fold higher prevalence of AF in patients with idiopathic CSA than in patients with OSA,[26] and Grimm et al[27] reported a significant association between AF and severe CSA in a group of patients

Table 3
Clinical variables associated with atrial fibrillation progression in patients with sleep apnea

	Atrial Fibrillation progression(N=98)	No atrial fibrillation progression(N=74)	Crude risk ratio (95% CI)	Adjusted risk ratio * (95% CI)	p-Value
Duration of atrial fibrillation diagnosis to sleep apnea testing					
All sleep apnea					**0.008**
≤ 24months	49 (50%)	52 (70%)	reference	reference	
>24 months	49 (50%)	22 (30%)	1.4 (1.1 − 1.8)	1.5 (1.1 − 1.9)	
Obstructive sleep apnea	*N=48*	*N=42*			
≤ 24months	28 (58%)	30 (71%)	reference	reference	
>24 months	20 (42%)	12 (29%)	1.3 (0.89 − 1.9)	1.3 (0.84 − 2.0)	
Mixed apnea†	*N=50*	*N=32*			
≤ 24months	21 (42%)	22 (69%)	reference	reference	
>24 months	29 (58%)	10 (31%)	1.5 (1.1 − 2.2)	1.3 (0.99 − 1.8)	
Male sex	68 (69%)	57 (77%)			**0.27**
Age	64 (57 − 70)	66 (59 − 72)			**0.09**
Body mass index (kg/m^2)	28 (25 − 34)	30 (27 − 35)			**0.12**
Hypertension	32 (33%)	33 (45%)			**0.11**
Left Atrium 4 chamber diameter (cm)	6 (5 − 6)	5 (5 − 6)			**0.43**
Left ventricular hypertrophy	28 (29%)	22 (30%)			**0.97**
CPAP	64 (65%)	49 (66%)			**0.99**

CI = confidence interval; CPAP = continuous positive airway pressure.
Data presented as median (interquartile) or n (%).
* Risk ratios adjusted for age, BMI, gender, and hypertension.
† Includes patients with pure CSA (n = 5).

with left ventricular dysfunction (odds ratio [OR] 5.21; 95% CI 1.67 to 16.27, p = 0.01). The relatively small number of patients with primary CSA in our cohort may also be secondary to greater use of HST than PSG and the lack of a significant population of patients with AF with clinical heart failure. The American Academy of Sleep Medicine recommends consideration of PSG for those with a high probability of sleep-disordered breathing other than OSA. Still, there is a lack of clear guidance regarding the appropriateness of using HST for broad SA screening in patients with AF.[28,29] Given the frequent and clinically important intersection of AF and heart failure, it would be important to understand whether initial referral of patients with both conditions to PSG over HST is warranted.

In our study, we found a high prevalence of mixed and central apnea in a population of patients with AF. Additionally, we found an association between the duration of AF diagnosis to SA testing and AF progression. Our analysis found that patients tested for SA within 2 years of AF diagnosis had less AF progression than those tested more than 2 years after AF diagnosis. This association was significant for the entire cohort, but when comparing OSA and mixed apnea, the association was insignificant for the OSA group. Although the association for patients with mixed apnea became borderline significant after adjusting for variables, this may be because of the relatively small numbers of patients with mixed apnea. The relation for all patients between AF diagnosis and SA testing suggests an important role in the early identification of SA in patients with AF. CPAP treatment was not associated with decreased progression of AF in our cohort. We believe there are several implications of this finding. First, CPAP adherence and details related to treatment duration were unavailable. Second, optimizing CPAP treatment may take months to years; thus, follow-up may not have been sufficiently long to observe a clinical benefit. Finally, earlier testing for SA may reflect a more aggressive pursuit of rhythm control and may have simply identified patients in an earlier stage of AF.

Our study had several limitations. First, our findings may be affected by selection bias of patients referred for management in our Arrhythmia Clinic. This population could be inherently more complex with higher rates of SA, and thus, generalizability may be limited. Another limitation is that the time of AF diagnosis and AF onset may not be the same. Additionally, the widespread use of HST in this study may also be a limitation. The gold standard for SA testing is PSG. Previous studies have shown that HST can provide accurate results. Although, the ability of HST, using the current standard scoring guideline, which is heavily weighted to the obstructive designation, to diagnose various phenotypes of SA is limited relative to PSG. Finally, our study had a relatively small sample size; larger prospective studies are needed.

In conclusion, obstructive and mixed SA is highly prevalent in an unselected population of patients with AF. Duration of more than 2 years from AF diagnosis to SA testing was associated with higher rates of AF progression in patients with SA. Our results highlight the importance of early screening and management of sleep-disordered breathing in patients with AF to slow or prevent AF progression.

Disclosures

The authors have no conflicts of interest to declare.

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