Obstructive sleep apnea (OSA) syndrome is a common breathing disorder, affecting ~5% of North American adults, the prevalence in men being almost twice that of women.1 The diagnosis is suspected by history and often body habitus, but requires confirmation with a formal sleep study. Polysomnography is the gold-standard study for the diagnosis of OSA. It determines the severity of OSA by measuring the apnea-hypopnea index (AHI), which is the number of apnoic and hypopnoic episodes that occur during 1 h. The condition is associated with increased cardiovascular morbidity and mortality, somnolence, neurocognitive dysfunction, mood disorders, and an increased risk of motor vehicle accidents; despite this, it is usually under-diagnosed (20–30% depending on clinical scenario).2–8 Researchers have demonstrated an increased incidence of cardiac arrhythmias among patients with OSA.9 Interestingly, however, there has been no systematic effort to identify the prevalence of OSA among patients with cardiac arrhythmias. Early reports described an increased association between OSA and bradyarrhythmias.10 This led to an intense focus on the role of pacemakers as a potential treatment for OSA—more than 10 well-conducted randomized trials addressing this issue have been published in the last 5 years.11–15 These trials have not borne out the initial enthusiasm for this treatment modality,16 although, interestingly, Bradley and colleagues16,17 have recently speculated a mechanism whereby increased cardiac output triggered by atrial overdrive pacing could improve upper airway patency. This may result in reductions in lung to chest receptor circulation time and left ventricular filling pressure, thereby stabilizing breathing by reducing the loop gain and preventing the hyperventilation that initiates central sleep apnea (CSA).

Of note, the Sleep Heart Health Study did not demonstrate a significant association between bradycardia and OSA.9 Atrial fibrillation (AF), an important risk factor for stroke and heart failure, in contrast, is strongly associated with OSA [odds ratio (OR) 2.19, 95% confidence interval (CI) 1.40–3.42].18 Patients with untreated OSA are at higher risk for AF recurrence at 1 year after electrical cardioversion (82% in untreated patients vs. 42% in treated patients; *P* = 0.013).19 In a substudy of the Sleep Heart Health Study,9 individuals with severe sleep apnea (AHI > 30) had four times the odds of having AF (OR 4.02, 95% CI 1.03–15.74) and three times the odds of having non-sustained ventricular tachycardia (OR 3.40, 95% CI 1.03–11.20), compared with individuals without OSA, even after adjusting for possible confounding factors. Another cohort study of 3542 patients has shown that obesity and OSA are independent risk factors for AF (body mass index: per 1 kg/m², HR 1.07, 95% CI 1.05–1.10, *P* < 0.013).20 This increased risk for AF among individuals with OSA may account, at least in part, for the significantly increased risk of stroke among patients with OSA (HR 1.97, 95% CI 1.12–3.48).21 The mechanisms linking OSA with supraventricular and ventricular arrhythmias remain somewhat speculative. However, certain points can be highlighted.

(i) Impaired autonomic nervous control has been demonstrated in patients with OSA, manifesting as increased sympathetic tone and/or decreased parasympathetic tone. Decreased baroreflex sensitivity, reduced vagal input, and impairment of the parasympathetic components of the heart rate variability have been demonstrated in patients with OSA.22,23

(ii) A persistent increase in sympathetic tone, as occurring in OSA, has been shown to generate abnormal electrical remodelling of the atrium, facilitating supraventricular arrhythmias, and AF in particular.24 Specifically, electrical remodelling may create some degree of intertrial block, contributing to the genesis of atrial arrhythmias.25

(iii) A strong association between OSA and hypertension has been reported extensively.6,26 As well, the association between hypertension and AF is well recognized.27,28
Although purely speculative, the link between OSA and AF could merely be due to the distortion of the atrial anatomy that occurs during hypertension.

To demonstrate a causative relationship between OSA and cardiac arrhythmias, prospective trials demonstrating a reduction in the incidence or recurrence of AF with effective treatment of OSA are required. In the interim, the multidisciplinary nature of OSA should be recognized, and physicians from diverse disciplines including cardiologists should equip themselves with the necessary training to recognize this disorder in all its guises. Special attention should be paid to detect cardiac arrhythmias in patients with OSA, who have structural heart disease. The autonomic imbalance associated with OSA may be the trigger for complex ventricular arrhythmias. In the setting of cardiac abnormalities, this may represent a life-threatening situation. Adequate treatment of OSA with continuous positive airway pressure may result, in selected cases, in a resolution of the arrhythmia problem. Similarly, the cardiologist and internist must be vigilant to seek the presence of OSA in patients with cardiac arrhythmias because this population may be at higher risk of developing stroke and cardiovascular events, and early treatment of OSA may have a positive impact on reducing cardiovascular morbidity.

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References