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COMMENTARY

Sleep apnea and atrial fibrillation: the spell of Groundhog Day

Commentary on Lin CH, Timofeeva M, O'Brien T, Lyons OD. Obstructive sleep apnea and nocturnal attacks of paroxysmal atrial fibrillation. *J Clin Sleep Med*. 2022;18(5):1279–1286. doi: 10.5664/jcsm.9840

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The observation of repetitive episodes of apneas, accompanied by futile efforts to breathe and progressive hypoxia that is only terminated by arousals from sleep in patients with severe obstructive sleep apnea (OSA), is dramatic. Any observer will intuitively make the hypothesis that OSA is harmful to the heart. Atrial fibrillation (AF) is one of the most common arrhythmias in clinical practice[1] and is a good candidate to be triggered by OSA.[2] There are well-documented acute and chronic events triggered by OSA that can contribute to AF. Large swings in intrathoracic pressure during obstructive events cause atrial stretch that, in turn, is a fruitful soil for AF generation. Intermittent hypoxia, arousal from sleep, and exaggerated negative intrathoracic pressure during obstructive events trigger large surges in both cardiac parasympathetic and sympathetic nervous activity that can cause acute episodes of AF in a susceptible heart.[2] Therefore, it is not surprising that, from the outset of clinical research on OSA, a high prevalence of nocturnal arrhythmias, and in particular AF, was described.[3]

One would expect that 45 years would be sufficient to clarify the link between OSA and AF. In fact, several large studies concluded that there is an independent association between sleep apnea and AF.[4,5] However, several investigators in the area may feel like the film "Groundhog Day," a movie in which a man finds himself living the same day over and over and over again. Why is that so? One major limitation is that AF may occur among patients with different heart conditions, ranging from lonely AF (patients without overt risk factors for AF), where 1 study found no clear association with OSA,[6] to patients with severe HF, where AF is associated Cheyne-Stokes–central sleep apnea.[7] In addition, it must be clearly stated whether AF is paroxysmal or sustained. Another component of confusion to any cross-sectional study relies on the fact that both OSA and AF share several risk factors such as obesity, sedentary habits, and increasing age.

In this issue of the *Journal of Clinical Sleep Medicine*, Lin et al[8] reported a small but clever study that adds meaningful information to this puzzle. The authors recruited exclusively patients with paroxysmal AF, and most of the patients (> 80%) had been diagnosed with AF for the first time. Of 152 patients with paroxysmal AF, 67 underwent polysomnography (PSG) and 20 (29.8%) had moderate to severe OSA. AF attack in sleeping hours occurred more frequently in patients with moderate to severe OSA than in patients with no or mild OSA (70% vs 26%, respectively). Due to the study design, and perhaps also because of the small number of patients, there were no significant differences between patients without and with moderate to severe OSA in terms of other risk factors for AF, such as age, body mass index, hypertension, the presence of structural heart disease, left ventricular function, left atrial size, or alcohol consumption. In addition, patients who presented with a paroxysmal AF attack during sleeping hours had 6.6 times the odds of having OSA compared with those who presented AF during waking hours. Another interesting finding that has been found in other studies in the cardiovascular field[9] is that patients with OSA were not sleepy (Epworth Sleepiness Scale = 5.5 ± 4.3). The absence of clear OSA symptoms raises 2 flags: difficulties in recognizing patients with OSA and, more importantly, we must clarify a causal relationship between OSA and AF in order to justify investigation and treatment of OSA among patients with AF.

The study of Lin et al has several limitations that were acknowledged.[8] The number of patients was small, and unfortunately, 85 out of 152 patients were excluded because they failed to undergo PSG. In order to increase the number of patients, the field would largely benefit from the use of simplified and validated methods for investigating OSA, such as portable monitoring.[10] While PSG provides a large and detailed number of channels, most PSG information such as sleep stages and arousals added very little to the study of Lin and collaborators (Table 2 in their paper).[8] In fact, there is mounting evidence that simple parameters such as the hypoxic burden are sufficient to provide relevant information regarding the relationship between OSA and cardiovascular risk.[11] Another important finding was that, out of the initial group of 152 patients, only 8 (5.2%) were using continuous positive airway pressure therapy. On the other hand, out of 66 patients who underwent PSG, 20 (30.3%) presented moderate to severe OSA. This sheds light into the real-life clinical situation where the vast majority of patients remain not diagnosed and not treated.

Going back to our film, the escape from the seemingly endless repetition of the same day is only achieved when Phil Connors is transformed into a good person, and he finally gains

Rita's love. In order to conquer the heart of the cardiovascular field we need larger studies. More importantly, we need randomized trials that may clarify whether it is worth recognizing and treating oligosymptomatic OSA among patients with AF.

CITATION

Lorenzi-Filho G, Genta PR. Sleep apnea and atrial fibrillation: the spell of Groundhog Day. *J Clin Sleep Med*. 2022;18(5):1223–1224.

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DISCLOSURE STATEMENT

The authors report no conflicts of interest.