EDITORIAL COMMENTARY

A dream within a dream

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Recently, a wealth of observational data has identified a strong link between sleep apnea (SA) and its contribution to cardiac arrhythmogenesis. Several mechanisms have been proposed to explain this association, including repetitive hypoxia causing oxidative stress, autonomic nervous system imbalance, and direct mechanical and hemodynamic alterations that ultimately induce remodeling of cardiac structures as well as changes to its electrophysiologic properties.[1] Common risk factors are thought to underpin this phenomenon; thus the prevailing focus of research has been on the effect of SA and its treatment, including risk factor management, on improving arrhythmia outcomes. Current literature, however, has mainly been on atrial fibrillation, with less attention to ventricular arrhythmias (VA).

In this issue, Goel and colleagues[2] described an intriguing case of a patient who experienced SA improvement following the ablation and suppression of premature ventricular contractions (PVC). A significant reduction in PVC burden from 40% to 2% was seen after ablation, as confirmed by a 2-week event recorder, with a concomitant remarkable reduction in periodic breathing percentage, apnea-hypopnea index, and overall symptomatic improvement. What is fascinating is the fact that this occurred without any change in the patient's cardiac function, continuous positive airway pressure therapy, and other risk factors for SA.

The plausible explanation put forth by the authors is the immediate hemodynamic improvement seen with PVC suppression. Firstly, reduction of pulmonary capillary wedge pressure could lead to less pulmonary congestion, thus allowing for better oxygenation and less hypoxia. Secondly, the rise in cardiac output with amelioration of electromechanical dyssynchrony caused by the presence of frequent PVC could also attribute to SA improvement. Small studies on cardiac resynchronization therapy in patients with heart failure (HF) have resulted in clinical improvement of central SA, although admittedly this occurred mainly in cardiac resynchronization therapy responders.[3,4]

Apart from these mechanical changes, another hypothetical possibility is the restoration of neurohumoral imbalances involving the sympathetic nervous system and natriuretic peptide systems. There exists a bidirectional relationship between neurohumoral systems and VA. Sympathetic activation can increase the likelihood of VA by enhancing automaticity, triggering early afterdepolarizations, and facilitating reentry with the release of norepinephrine and other neurotransmitters.[5] On the other hand, VA themselves can activate the neurohumoral systems, creating a vicious cycle that sustains and exacerbates it. This complex interplay has long been studied in the HF population; and with a significant overlap between HF and SA, this is another interesting avenue for future research.

The authors ought to be congratulated for recognizing the serendipitous moment when their patient's SA parameters improved after a seemingly unrelated procedure. At the end of the day, the dream of any physician is to improve the overall well-being of their patients. To make this dream a reality, the only way forward is to wake up from our slumber and rise to the challenge of improving our understanding of the complex interactions between different physiological systems and disease processes to deliver high-quality, patient-centered care.

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