CPAP in Obstructive Sleep Apnea and Atrial Flutter-Fibrillation

Is This Truly Two for the Price of One?

To the Editor:

There is growing research interest in the association between atrial fibrillation (AF) and obstructive sleep apnea (OSA), which are independently common diseases that have a serious impact on health, quality of life, and health-care resources. In many cases, both these diseases show a parallelism in that appropriate early diagnosis and treatment have demonstrated positive clinical benefits.

However, studies are promoting positive interactions of CPAP for preventing and reducing recurrences of AF, despite the lack of clear evidence-based analysis and guidelines. We read with interest the article by Bazan et al in CHEST (May 2013) in this area of growing interest. We would like to comment on some aspects of their study that are relevant to clinical practice.

First, there are some functional and structural cardiac changes that were not evaluated in this study and could potentially influence the results. The relationship between the duration of CPAP use and echocardiographic findings were not discussed but can provide a better understanding of whether CPAP could influence AF and recurrences. Additionally, echocardiographic factors such as left atrial diameter variations, severity of mitral valve regurgitation, and left ventricular hypertrophy were not measured, but these are potentially strong predictive factors for AF and recurrences.

Second, the authors did not describe correlations between OSA, efficacy of antiarrhythmic drugs, and CPAP therapy and AF recurrence. Third, there were no data on CPAP compliance and duration of CPAP use among patients who had and did not have AF recurrences following ablation therapy for atrial flutter. Fourth, the authors presented no data on other causal factors for AF. In one study, OSA was a strong predictor of recurrent AF after an ablation procedure; BMI, ejection fraction, left atrial size, and hypertension did not affect the outcomes postablation for atrial flutter. Finally, other relevant measurements such as levels of N-terminal pro-B-type natriuretic peptide or B-type natriuretic peptide were not provided.

We believe that the study by Bazan et al provides valuable information with regard to OSA and AF recurrences, but mechanisms that influence this association remain complex. Further studies that look at cardiac structural and functional modifications to identify triggers or promoters of atrial flutter recurrences are necessary.

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We sincerely appreciate the comments of Drs Esquinas and Pravinkumar regarding our article in CHEST on obstructive sleep apnea (OSA), atrial flutter (AF), and reduction of new-onset atrial fibrillation (AFib) by CPAP treatment. We hypothesized about a selective physiopathologic interaction between OSA and AF (as suggested by an 82% prevalence of OSA in patients with AF), in which pulmonary hypertension during apnea episodes or other mechanisms would induce right atrial overload and/or remodeling, thus favoring the occurrence of AF. We further theorized a beneficial impact of CPAP early in the atrial remodeling process leading to AFib (before any AFib documentation), independent or not of AF being documented.

In our study, no serial echocardiography was performed to evaluate the structural changes induced by CPAP. Therefore, hemodynamic mechanisms through which CPAP presumably protects from AFib remain unclear.

REFERENCES


Response

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