pii: jc-00061-16 http://dx.doi.org/10.5664/jcsm.6138

JCSM
Journal of Clinical Sleep Medicine

CASE REPORTS

Positive Airway Pressure-Induced Conversion of Atrial Fibrillation to Normal Sinus Rhythm in Severe Obstructive Sleep Apnea

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Accumulating data implicate obstructive sleep apnea (OSA) as a predisposing factor to the development of atrial fibrillation (AF), the latter representing the most common sustained cardiac arrhythmia. The postulated mechanisms leading to atrial arrhythmogenesis in OSA include alterations in intrathoracic pressures, intermittent hypoxemia, and autonomic nervous system fluctuations. Although these OSA-related pathophysiologic pathways may result in atrial structural and electrical remodeling, thereby predisposing to AF, there are data to suggest that the immediate influences of respiratory events may trigger arrhythmic events. This case demonstrates an immediate reversal of AF to normal sinus rhythm with optimal continuous positive airway pressure (CPAP) therapy in the background of severe OSA. These findings of immediate benefit of reversal of OSA pathophysiology on cardiac arrhythmia suggest OSA may have acute influences on cardiac electrophysiology.

Keywords: atrial fibrillation, continuous positive airway pressure, obstructive sleep apnea

Citation: Walia HK, Chung MK, Ibrahim S, Mehra R. Positive airway pressure-induced conversion of atrial fibrillation to normal sinus rhythm in severe obstructive sleep apnea. *J Clin Sleep Med* 2016;12(9):1301–1303.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common, underdiagnosed condition characterized by repetitive upper airway collapse during sleep that is associated with atrial fibrillation (AF), the latter representing the most common sustained cardiac arrhythmia. OSA is postulated as a predisposing AF risk[1,2] due to OSA-mediated pathophysiological influences, including intermittent hypoxemia, hypercapnea, intrathoracic pressure alterations , autonomic nervous system fluctuations, and upregulation of inflammation leading to atrial arrhyrhmogenesis. In fact, immediate temporal associations of respiratory events of OSA and paroxyms of AF have been described.[3] Furthermore, exposure to OSA-related respiratory events can lead to chronic cardiac remodeling. Whether treatment of OSA would exert benefit acutely and/or over the long term remains unclear.

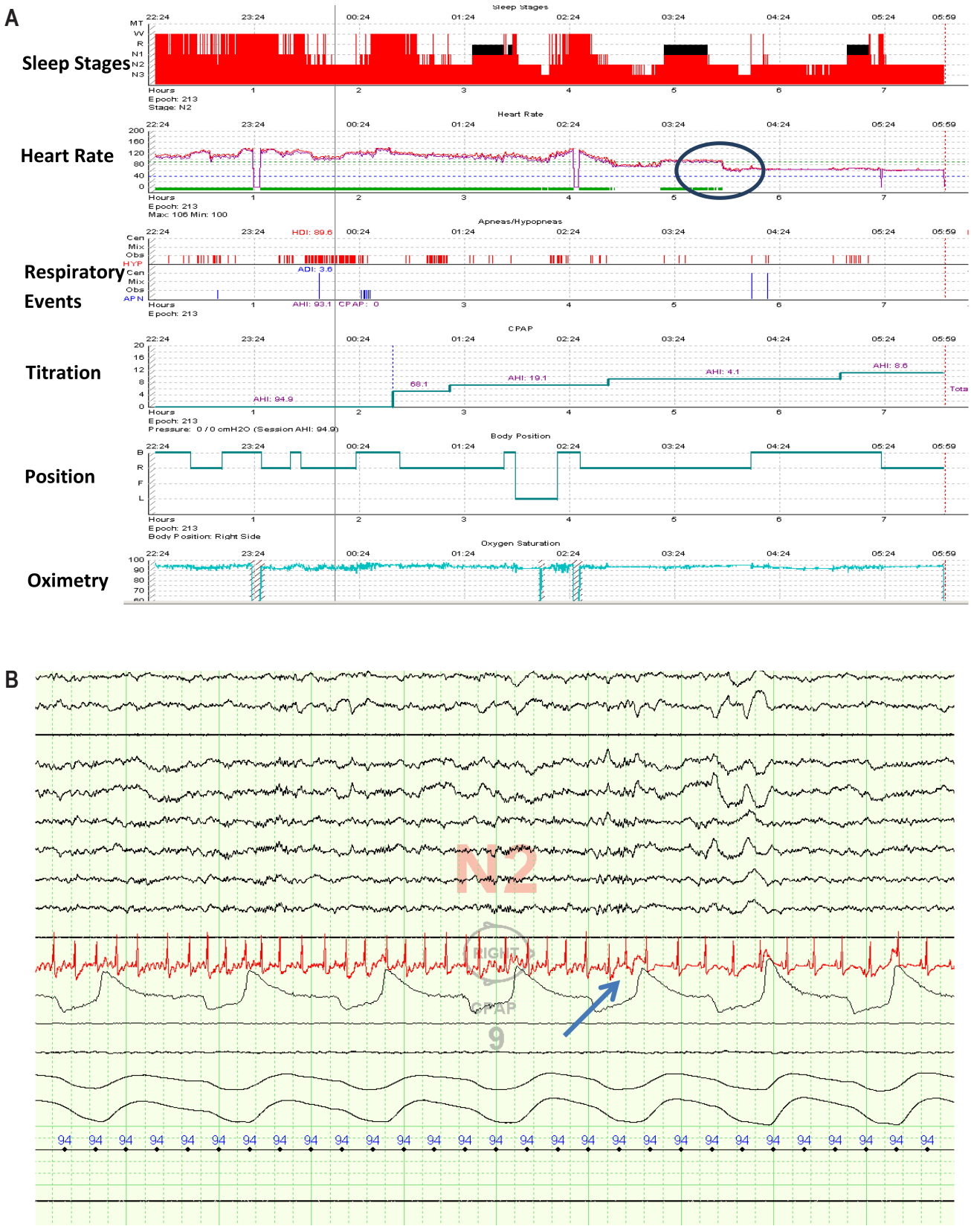
Although a recent meta-analysis identified that patients treated with continuous positive airway pressure (CPAP) had a 12% decreased risk of AF[4] after ablation or cardioversion, there are limited data on the acute effect of CPAP atrial rhythm changes in AF. We present a case of severe OSA and AF with rapid venrtricular rate in which CPAP appeared to immediately convert AF to normal sinus rhythm during the polysomnogram.

REPORT OF CASE

A 53-y-old man (body mass index, 38.3 kg/m^2) with OSA of unknown severity and undergoing auto CPAP with unknown compliance was referred for a split-night polysomnogram. His history included diabetes mellitus, hyperlipidemia, hypertension, obesity, AF, dilated cardiomyopathy, and systolic heart failure. Three months prior to presentation cough and shortness of breath developed, and the patient was found to be in AF with rapid ventricular rates (130 bpm); echocardiogram showed a left ventricular ejection fraction of 20% to 25%. A stress test suggested apical ischemia, but left heart-cardiac catheterization showed only mild coronary luminal irregularities. The patient was to undergo transesophageal echo (TEE)-guided direct current (DC) cardioversion, but because of concern about left atrial thrombus, rivaroxaban was begun and he underwent DC cardioversion after a repeat TEE 2 to 3 weeks later. However, he reverted to AF after a few days. Another DC cardioversion on amiodarone again resulted in early recurrence of AF, which persisted. The most recent echocardiogram showed left ventricular ejection fraction of 37 ± 5%, normal right ventricular systolic function, left atrial cavity volume 88.0 mL, and left atrial volume index 40.2 mL/m^2 consistent with severe left atrial dilatation. Medications included meloxicam, glucophage, pravastatin, carvedilol, lisinopril, omeprazole, furosemide, and rivaroxaban. The diagnostic portion of the sleep study revealed an apnea-hypopnea index (AHI) of 90.6 events/h, consisting of 9 apneas (8 obstructive and 1 central) and 71 hypopneas with nadir oxygen saturation of 88%. The titration revealed an optimal CPAP setting of 9 cm H_2O, during which the AHI was reduced to < 5 events/h with a nadir oxygenation of 89% and average saturation of 93%.

The diagnostic portion of the study demonstrated AF with rapid ventricular rate (average heart rate of 110 bpm [range, 98–125]). At a pressure of 9 cm H_2O, conversion of AF to normal sinus rhythm occurred, which was maintained through the

Figure 1



(A) The hypnogram demonstrates (from top to bottom) sleep stages, heart rate, respiratory events, diagnostic portion and continuous positive airway pressure therapy, body position, and oxygenation. The circle shows when atrial fibrillation was reverted to normal sinus rhythm. **(B)** A 30-sec epoch is shown, when the conversion of atrial fibrillation to normal sinus rhythm occurred with continuous positive airway pressure at 9 cm H_2O.

rest of the study with a heart rate of approximately 64 bpm (range, 60–68). **Figure 1A** and **1B** show the hypnogram and 30-sec epoch highlighting the transition from AF to normal sinus rhythm with concordant optimization of sleep apnea treatment. After the sleep study OSA was treated, and upon presentation for catheter ablation 6 weeks later, he was in sinus rhythm with paroxysmal AF. He has remained in sinus rhythm since the AF ablation procedure with pulmonary vein isolation.

DISCUSSION

This case illustrates conversion of AF with optimal CPAP pressure in a middle-aged obese male patient with a severely dilated left atrium in the background of severe OSA. A candidate mechanism to be considered includes immediate reversal of negative intrathoracic pressures changes resulting in alleviation of cardiac mechanical stress and acute reduction of left atrial volume[5] with CPAP therapy. Other considerations are reversal of hypoxemia and improvement of autonomic nervous system activity with CPAP. The degree of hypoxia observed in this case, however, was not profound. It can be postulated that those with severe left atrial dilatation are more susceptible to the acute mechanical physiology induced by sleep apnea than by chronic remodeling processes in the atria alone[6] and positive airway pressure was able to effectively address.

Although evidence suggests that CPAP improves systolic and diastolic abnormalities in patients with OSA as early as 3 months with progressive improvement in cardiovascular remodeling over 1 year, our case report demonstrated acute conversion to normal sinus rhythm in response to CPAP therapy in a patient with previously persistent AF and severely dilated left atrium. Although the conversion may have been fortuitous, the patient's prior course showed refractory persistent AF with posttreatment course notable for return to sinus rhythm with paroxysms of AF, likely triggered by pulmonary vein ostial triggers that were eventually successfully targeted for ablation with pulmonary vein isolation. Therefore, subgroups of patients with AF, particularly those with characteristic underlying left atrial/pulmonary vein electrical substrates, may be more amenable to acute benefits of CPAP in OSA. Even though there are data to suggest that acute negative intrathoracic changes can lead to immediate atrial and ventricular morphologic changes that may predispose those with systolic dysfunction to AF,[7] a potential area of future investigation would be to identify imaging or electrical parameters (such as early recurrences of AF after cardioversion, as seen in this patient) that may be indicative of atrial substrates that are vulnerable to OSA-induced changes in intrathoracic pressure-induced wall stress. Moreover, further investigation of reversal of OSA mechanical stress in susceptible individuals will allow for the opportunity to implement more efficient risk stratification strategies to management of AF.

ABBREVIATIONS

AF, atrial fibrillation
AHI, apnea-hypopnea index
CPAP, continuous positive airway pressure
DC, direct current
TEE, transesophageal echo
OSA, obstructive sleep apnea

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SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication February, 2016
Submitted in final revised form April, 2016
Accepted for publication April, 2016
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DISCLOSURE STATEMENT

This was not an industry supported study. Reena Mehra has received NIH funding for which she has served as Principal Investigator (NHLBI RO1 1 R01 HL 109493, R21 HL108226). Her institution has received positive airway pressure machines and equipment from Philips Respironics for use in NIH-funded research. She has received honorarium from the American Academy of Sleep Medicine for speaking. She serves as the Associate Editor for the journal *Chest*. She has received royalties from Up to Date. Harneet Walia has received the Neurological Institute-Center of Outcomes Research Scholarship of Cleveland Clinic. Sally Ibrahim and Mina Chung have no conflicts of interest.