Effect of Continuous Positive Airway Pressure on an Atrial Arrhythmia in a Patient with Mild Obstructive Sleep Apnea

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CASE REPORTS

Cardiac arrhythmias have been reported in up to half of individuals with obstructive sleep apnea (OSA) and have been proposed to be one of the factors contributing to their increased mortality. Several studies have demonstrated evidence of an association between OSA and a number of cardiac arrhythmias. The mechanisms of arrhythmogenesis in OSA may be due to enhanced vagal output triggered by a combination of apnea and hypoxemia. This case demonstrates

a dose-dependent reduction in atrial ectopy with increasing continuous positive airway pressure (CPAP) in the setting of mild sleep apnea.

Keywords: Continuous positive airway pressure, atrial arrhythmia

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Obstructive sleep apnea (OSA) is a common condition with high rates of morbidity and mortality as it has been associated with hypertension, ischemic heart disease, heart failure and cardiac arrhythmia.¹ Patients with OSA may be predisposed to arrhythmias because of OSA-associated hypoxemia, acidosis, apneas, and arousal, and also due to alterations in intrathoracic pressures leading to cardiac mechanical structural changes. Continuous airway pressure (CPAP) is the mainstay of treatment for OSA, especially moderate and severe OSA. There have been a few studies suggesting that CPAP may reduce arrhythmias,¹⁻³ but mechanisms are speculative.

We present a case of mild sleep apnea in which CPAP was effective in immediately reducing arrhythmias during the sleep study, better explained by pressure effects than by elimination of hypoxia or event number *per se*.

REPORT OF CASE

A 79-year-old African American female with symptoms of snoring, difficulty staying asleep, and daytime sleepiness was referred for a split-night polysomnogram for the evaluation of sleep apnea. She slept for 6 hours on average at night, and the Epworth Sleepiness Scale was 6/24. Past medical history included hypertension, bladder cancer, coronary artery disease (normal ejection fraction), GERD, and hyperlipidemia. Medications included simvastatin, hydrochlorothiazide, esomeprazole, lisinopril, and metoprolol. The diagnostic portion of the study revealed an apnea hypopnea index (AHI) of 13.9, and a lowest oxygen saturation of 91%. The treatment portion of the study revealed an optimal pressure of CPAP 9 cm H₂O, reducing the event rate to < 5/h.

The diagnostic portion of the study revealed frequent atrial ectopy with no apparent temporal relationship with respiratory events (see **Figures 1A, B**). CPAP was titrated from 4 cm H₂O to 9 cm H₂O. At a pressure of 5 cm H₂O, the frequency

of atrial ectopy decreased compared to baseline (**Figure 1C**). At a pressure of 7 cm H₂O, the ectopy decreased even further (**Figure 1D**). At a pressure of 9 cm H₂O, the atrial ectopic beats were nearly eliminated (see **Figure 1E**). The lowest oxygen saturation was 91% under all conditions, while the mean oxygen saturation improved from 92% at baseline to 96% at a pressure of 9 cm H₂O. The average heart rates at baseline, CPAP of 5 cm H₂O, CPAP of 7 cm H₂O, and CPAP of 9 cm H₂O were 66 bpm, 65 bpm, 61 bpm, and 59 bpm respectively. The average number of ectopic beats in 1 minute at baseline, CPAP of 5 cm H₂O, CPAP of 7 cm H₂O, and CPAP of 9 cm H₂O were 16/min, 14/min, 12/min, 9/min respectively.

DISCUSSION

Cardiac arrhythmias are reported in as many as half of patients with OSA and are one of the factors contributing to mortality.⁴ Several studies have shown modest associations between OSA severity and a number of cardiac arrhythmias.^{4,5}

This case illustrates a dose-dependent reduction in atrial ectopy with CPAP therapy in the setting of mild sleep apnea. The improvement in atrial ectopy with CPAP was immediate and evident at even low levels of CPAP, before substantial increases in oxygen saturation above the baseline of 92%. One can consider three potential mechanisms by which CPAP improve the degree of atrial arrhythmia—reversal of autonomic nervous system dysfunction, reversal of sleep apnea-related mechanical atrial stretch, and/or mild improvement in oxygenation. The patient was taking a β -blocker medication, suggesting that the benefits of CPAP may have been mediated more by reduced vagal drive or improved oxygenation rather than sympathetic nervous system surges. The OSA-arrhythmia relationship may be similar to that noted between OSA and hypertension, in which elevated blood pressure persists during wakefulness; and in this case, arrhythmogenic potential persists and/or progresses over

Figure 1A—Diagnostic portion of the sleep study

30-sec epoch showing the diagnostic portion of the study including respiratory channels.

Figure 1B—Diagnostic portion of the sleep study

EKG channel (lead II) in the 30-sec epoch in the diagnostic portion of the study, without CPAP (ectopic beats 16/min).

Figure 1C—Continuous positive airway pressure 5 cm water pressure

EKG channel (lead II) in a 30-sec epoch with CPAP at 5 cm H₂O (ectopic beats 14/min).

Figure 1D—Continuous positive airway pressure 7 cm water pressure

EKG channel (lead II) in a 30-sec epoch with CPAP at 7 cm H₂O (ectopic beats 12/min).

Figure 1E—Continuous positive airway pressure 9 cm water pressure

EKG channel (lead II) in a 30-sec epoch with a CPAP at 9 cm H₂O (ectopic beats 9/min).

time into wakefulness.⁴ The idea is that in response to long-standing repetitive exposures, there occurs chronic autonomic nervous system imbalance and/or cardiac structural remodeling leading to atrial arrhythmogenesis. However, in this case there was an immediate improvement in the frequency of atrial ectopy with CPAP treatment.

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