

Can continuous positive airway pressure therapy have antiarrhythmic properties?

Czy CPAP może mieć działanie antyarytmiczne?

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A 61-year-old obese (body mass index 36.6 kg/m²) male with diabetes mellitus was admitted to the hospital due to an exacerbation of chronic heart failure. He had poorly controlled arterial hypertension complicated by heart muscle hypertrophy and grade III retinopathy. Arterial hypertension was treated with renal denervation in the past, but the procedure did not result in any significant improvement in blood pressure (BP) control (prior to the hospitalisation the patient was receiving four hypotensive agents, including diuretic). On admission his office BP values were 170/85 mm Hg. The patient had his BP pharmacologically lowered, and was scheduled to have 24-h electrocardiography (ECG) Holter monitoring. ECG monitoring showed sinus rhythm with a mean heart rate of 56 bpm, and numerous ventricular extrasystoles. Also 975 sinus pauses lasting less than 2.55 s were recorded. The histograms depicted in Figure 1A show an association between the number of pauses per hour and the time of day. Interestingly, the pauses and most of the episodes of bradycardia were recorded in the nighttime. Few sinus pauses were also recorded during the patient's nap in the late evening. Because of obesity and daytime sleepiness the patient was scheduled to have a whole-night polygraph examination (Embletta[®] MPR PG, Ontario, Canada), along with same-time ECG monitoring. The study revealed severe obstructive sleep apnoea (OSA) with an apnoea-hypopnea index of 48.3 per hour. The apnoea episodes were in close time-association with the bradycardia episodes, and pauses were directly induced by apnoea episodes. Figure 1B depicts a fragment of the polygraph study with visible, repetitive apnoea episodes associated with desaturation. The patient received treatment with continuous positive airway pressure (auto CPAP, ResMed S9 Escape[™]). He tolerated the treatment well. The patient had good compliance — he used the CPAP for at least 5.5 h every night. After 7 and 30 nights of treatment, extended 72-h ECG monitoring was performed. This time the study showed no episodes of bradycardia with heart rate lower than 52 bpm. The number of pauses was reduced by over 90%. Modification of hypertension pharmacotherapy along with CPAP use resulted also in significant lowering of the BP values, and ambulatory BP monitoring revealed normalisation of nocturnal values, with preserved dipping profile (Fig. 2). Six months after the hospitalisation the patient is doing well, he is tolerating the CPAP treatment perfectly, has good BP control, and has not suffered any heart failure exacerbations. The present case highlights the importance of OSA in the pathogenesis of cardiac arrhythmias and resistant hypertension. OSA is associated with repetitive episodes of hypoxaemia, desaturation, and microarousals, which, along with changes in the transthoracic pressure, promotes autonomic dysregulation and cardiac arrhythmias.

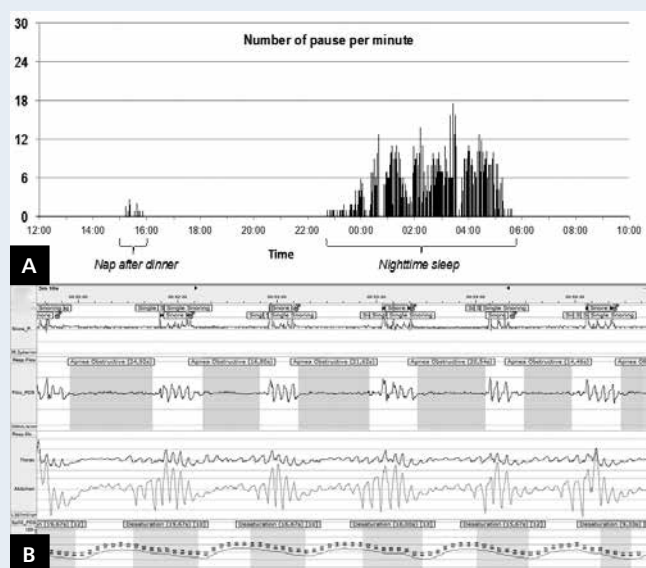


Figure 1. Daytime and nighttime distribution of electrocardiogram-proven sinus pauses (A), and potentially responsible, obstructive sleep apnoea episodes recorded in the polygraph study (B)

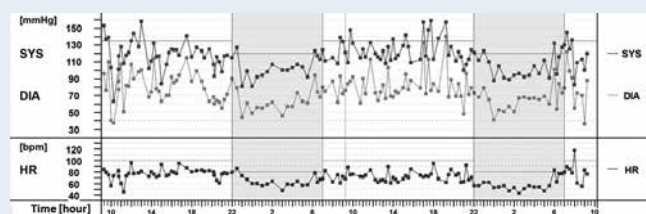


Figure 2. Results of ambulatory blood pressure monitoring after continuous positive airway pressure treatment

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