

2ND PLACE FOR HIGHEST SCORE DURING THE ABSTRACT REVISION PROCESS FOR CASE REPORTS.

CASE REPORT

30. Avoiding Pacemaker: Resolution of Sinus Node Dysfunction through CPAP Therapy



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► https://www.youtube.com/watch?v=hJIcIJ1w8oM&list=P_LhqNq3xJClbafO0Y5bvBcgMmXpgzJxd44&index=5&t=14584s

Background: Sleep apnea-hypopnea syndrome (SAHS) is a common yet underdiagnosed sleep-related breathing disorder with significant cardiovascular implications. Besides hypertension, heart failure, and ischemic disease, SAHS has been increasingly associated with arrhythmias and conduction abnormalities. Sinus node dysfunction (SND) is among the rhythm disturbances most frequently reported, with clinical manifestations ranging from bradycardia to prolonged sinus pauses. The pathophysiology involves recurrent nocturnal hypoxemia, sympathetic-vagal imbalance, and intrathoracic pressure fluctuations, which together promote electrical instability. Traditionally, pacemaker implantation has been considered the treatment of choice for symptomatic or severe SND. However, growing evidence suggests that adequate management of SAHS with continuous positive airway pressure (CPAP) may reverse rhythm disorders, highlighting the importance of identifying reversible causes before committing to invasive procedures.

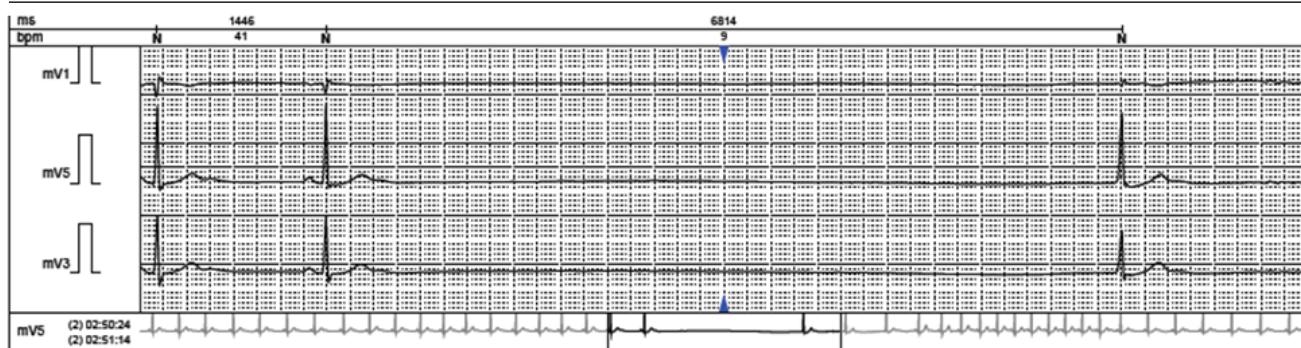
The Case: We report the case of a 34-year-old man with morbid obesity (BMI 43.6) and no cardiovascular history, who presented with severe daytime sleepiness (Epworth score 17). Physical examination

was unremarkable, with no structural heart disease. Baseline electrocardiogram showed sinus bradycardia at 56 bpm. Holter monitoring revealed sinus node dysfunction with nocturnal pauses up to 6.8 seconds. Based on these findings, dual-chamber pacemaker implantation was initially considered. Simultaneous overnight polysomnography confirmed severe SAHS, with an apnea-hypopnea index (AHI) of 70 events/hour, oxygen desaturation index of 52/hour, and a nadir oxygen saturation of 56%. Given the temporal overlap between apneic events and sinus pauses, an alternative strategy was pursued. The patient was started on CPAP therapy and received weight loss counseling.

At one-month follow-up, he reported complete resolution of daytime symptoms. Repeat polysomnography demonstrated significant improvement, with AHI reduced to 38/hour, desaturation index to 19/hour, and minimum O₂ saturation improved to 70%. Holter monitoring confirmed normalization of sinus rhythm, with a mean heart rate of 68 bpm and no further pauses. Pacemaker implantation was therefore avoided.

Conclusion: This case highlights the complete reversibility of sinus node dysfunction after treatment of severe SAHS with CPAP therapy. It underscores the need to systematically consider sleep-disordered breathing in patients with unexplained bradyarrhythmias, particularly in younger individuals without cardiac comorbidities. Identifying SAHS as an underlying cause may dramatically alter the therapeutic strategy, preventing unnecessary device implantation and its long-term complications. Beyond improving quality of life, CPAP should be recognized as a potential disease-modifying therapy for rhythm disturbances. Early screening, accurate diagnosis, and prompt initiation of noninvasive ventilation may represent a decisive step in redefining the management of SND in the context of sleep apnea.

Figure 1. Night Time Pause of 6,8 s. (the Pause was Preceded by Apnea for 52 Seconds and O₂ Saturation Decrease to 56%).



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