

# Recurrent Syncope in a patient with Sleep Apnea and Narcolepsy resolved after Permanent Pacemaker Implant

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## Abstract

Concomitant narcolepsy and sleep apnea (SA) can occur together. We present a case in which a patient presented with recurrent episodes of syncope and falls, which were found to be due to narcolepsy and Obstructive Sleep Apnea (OSA). She was initially treated with continuous positive airway pressure (CPAP), but continued to have recurrent episodes. Her symptoms were resolved with the implantation of a permanent pacemaker (PPM) after diagnosing an intermittent high degree AV nodal block.

**Keywords:** syncope; narcolepsy, sleep, apnea, pacemaker, atrio, ventricular, block, sinus, arrest

**Short title:** Syncope in a Patient with SA and Narcolepsy resolved after PPM

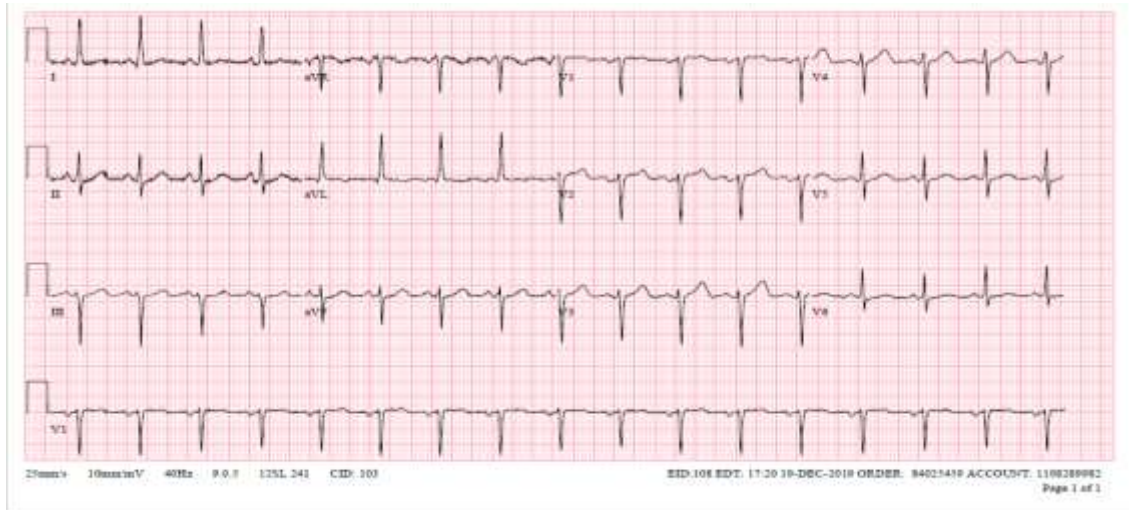
## Introduction

OSA is characterized by apneic/hypopneic episodes caused by repetitive collapse of the upper airway during sleep. SA is treated with CPAP which prevents these episodes by maintaining positive pharyngeal transmural pressure while stabilizing the upper airway through increased end-expiratory lung volume [1, 2]. Narcolepsy is defined as daytime sleepiness associated with cataplexy, hypnagogic hallucinations and sleep paralysis. It is usually treated with lifestyle modifications such as improved sleep hygiene and health maintenance, but may also require pharmacologic therapy [3, 4]. We present a case of a 41 year old female with a history of OSA, Obesity Hypoventilation Syndrome (OHS), and narcolepsy who presented to the hospital for recurrent episodes of syncope with falls. She was found to have episodes of daytime narcolepsy with intermittent episodes of sinus arrest and third degree atrio-ventricular block (AVB). The patient was compliant with her CPAP at night, however, was still noted to have significant bradyarrhythmias. During hospitalization, she underwent implantation of a PPM with resolution of her syncopal episodes.

## Case Presentation

This is a case of a 41 year old female with a past medical history (PMHX) of narcolepsy, OHS, OSA (on home CPAP) and hypertension (HTN) who was admitted to the hospital with recurrent episodes of syncope and falls. She was advised to make lifestyle modifications previously for her narcolepsy, but her symptoms persisted. She reported an inability to maintain her work due to her recurrent falls with episodes of syncope.

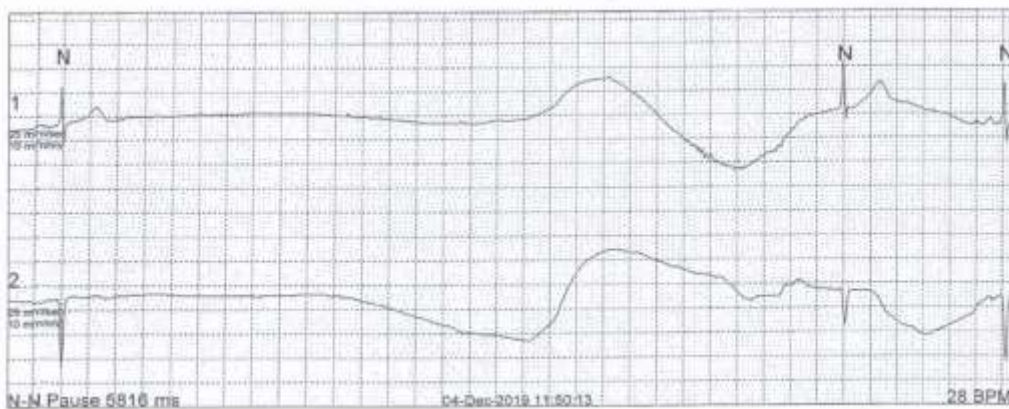
On arrival to the emergency department (ED), her vital signs were stable, and physical examination (including orthostatic blood pressure) unremarkable, with the exception of morbid obesity (weight: 159.9 kg, body mass index (BMI): 55.9). Her laboratory tests did not demonstrate any electrolyte or endocrine abnormalities. Her arterial blood gas (ABG) was significant for hypercapnia (PCO<sub>2</sub>: 65.5 mmHg; Normal 35-45 mmHg). Her 12-lead electrocardiogram (ECG) demonstrated normal sinus rhythm (NSR) and left ventricular hypertrophy (LVH), without any significant abnormalities (Figure 1). The patient was admitted to the telemetry unit for continuous cardiac monitoring and further evaluation of her recurrent syncopal episodes.



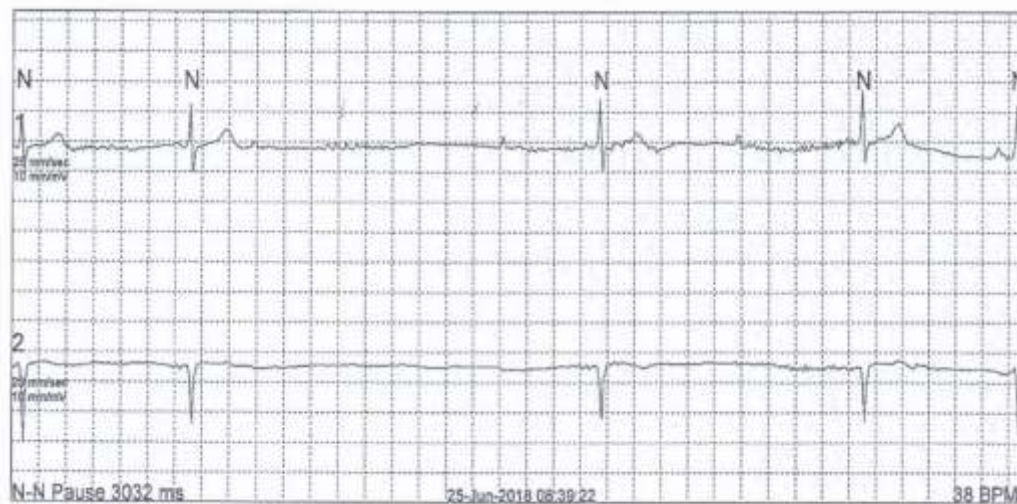
**Figure 1:** ECG on admission shows NSR, LVH, HR 101 bpm.

During her hospitalization she had an echocardiogram, which demonstrated a hyper-dynamic left ventricle, without any significant valvular abnormalities. She completed a 24-hour holter monitor, which reported basic sinus rhythm. However, there were episodes of marked bradycardia, down to 40 beats per minute (bpm), with episodes of sinus

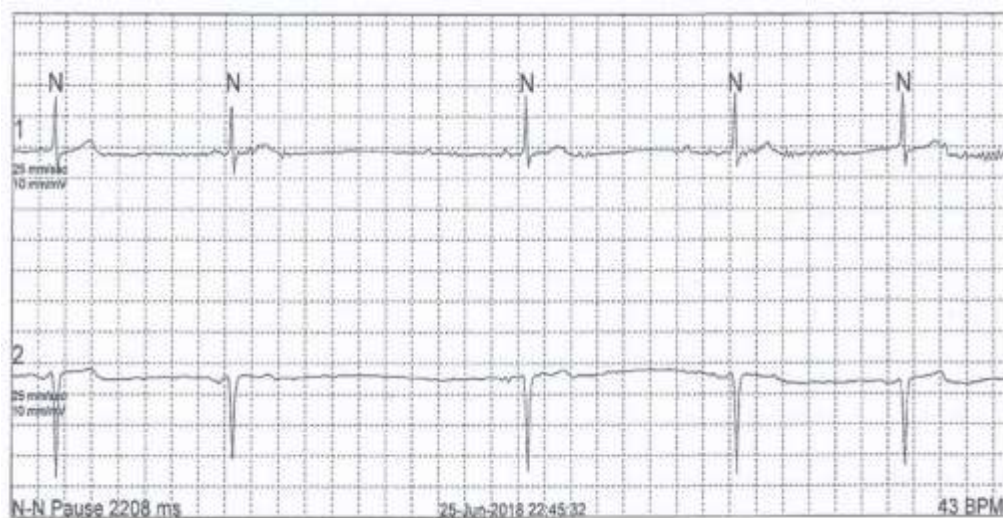
arrest/pauses during waking hours (Figure 2). The patient had a holter performed about a year prior to this admission which also revealed similar bradyarrhythmias during both daytime and nighttime hours, with the addition of intermittent third degree AVB (Figure 3, 4).



**Figure 2:** 24 hour holter monitoring showing sinus arrest (04/12/2019).



**Figure 3:** 24 hour holter monitoring showing 3rd degree AVB during the daytime (06/25/2018).



**Figure 4:** 24 hour holter monitoring showing 3rd degree AVB during the nighttime (06/25/2018).

A computed tomography angiography (CTA) of the chest and a venous doppler of the lower extremities were performed which ruled out a pulmonary embolism (PE) and deep vein thrombosis (DVT). Telemetry monitoring also revealed periods of sinus bradycardia, with intermittent sinus pauses and AVB's during daytime hours. No other reversible etiologies for her bradyarrhythmias were determined. Due to the recurrent episodes of syncope in the presence of sinus arrest with high degree AVB, the decision was made to proceed with PPM implantation. She underwent successful placement of a dual chamber PPM without complications and was discharged home in stable condition.

Since discharge, the patient has been following up routinely at our outpatient cardiology clinic. Six months since placement of the PPM, she has not had any recurrent episodes of syncope nor falls. She has also been able to maintain her current employment without any issues.

## Discussion

Cardiac rhythm disorders have been reported in patients with OSA. Several explanations have been described, such as negative intrathoracic pressure during efforts to breathe, intermittent episodes of hypoxia, and/or surges in sympathetic activity [5]. OSA has a strong positive correlation with an elevated BMI, via chronic obstruction of the upper airways with resultant apneic and hypopneic episodes [6]. These possible explanations for arrhythmias in OSA, may also apply to our patient who was morbidly obese with a BMI of 59.9.

The most common arrhythmia identified in OSA is atrial fibrillation, which is likely related to left atrial dilatation and remodeling [5]. Bradycardia has also been reported to occur in patients with increased vagal tone [5]. More specifically, it may occur during apneic episodes, when the absence of ventilation and resultant hypoxia both stimulate an increase vagal response from the carotid body [7]. One small study involving 6 patients with OSA, found bradycardia to be a consistent arrhythmia, postulated to have been caused by a combination of apnea and hypoxemia [8]. Episodes of heart block have also been reported, especially among patients with severe OSA (greater than 60 apneic and/or hypopneic episodes per hour) [9].

Bradycardia can be present in the setting of narcolepsy. Narcolepsy is often characterized by excessive daytime sleepiness and cataplexy, with early onset rapid eye movement (REM) sleep. Narcolepsy has also been associated with decreased levels of the neuropeptide, hypocretin - 1 (Hcrt - 1) in the cerebrospinal fluid (CSF) [10]. Hcrt-1 deficiency has been

found to significantly influence the autonomic nervous system, including resting heart rate [10]. Hcrt-1 fibers are present in the lateral paraventricular nucleus, which is responsible for the inhibitory pathway for preganglionic cardiac vagal neurons [10]. It is via this inhibitory pathway, that a deficiency of hcr-1 leads to an attenuated chronotropic response during both REM and non-REM sleep [10].

Narcolepsy and OSA can be concurrent disorders. One study found that 6% of patients with a positive multiple sleep latency test (MSLT) experienced respiratory issues during sleep, suggestive of both narcolepsy and OSA coexisting [11].

The use of CPAP in our patient did not alleviate her symptoms nor suppress her arrhythmias. Our patient's OSA was neither amenable to surgical intervention, nor responsive to central nervous system (CNS) stimulants. Our patient's management was unique as there was complete resolution of symptoms after the implantation of a PPM.

## Conclusion

Cardiac rhythm disorders, specifically bradyarrhythmias, are likely a common pathology in patients with narcolepsy and OSA. The coexistence of both of these disorders should be strongly considered as a possible etiology in patients with recurrent episodes of syncope and falls. Thus, when patients with clinically significant arrhythmias, especially bradyarrhythmias, are found in conjunction with OSA and/or narcolepsy, indications for PPM implantation should be investigated if conventional therapies fail.

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## Conflict of Interest

None to declare.

## Informed Consent

The manuscript has been sufficiently de-identified to protect the patient.



## Authors Contributions

The corresponding author, FJL and AS, wrote the main manuscript. GU provided additional research data in the introduction and discussion section. AS provided the electrocardiogram (Figure 1). All authors reviewed the final manuscript.

## Data Availability

The authors declare that data supporting the findings of this study are available within the article.

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