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Case of the Month

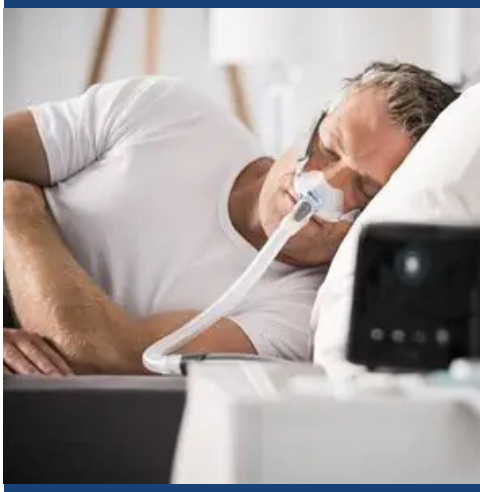
Obstructive Sleep Apnoea and the Pitfalls of Home Sleep Testing on CPAP

Abstract

Obstructive Sleep Apnoea (OSA) is a common condition seen in both the primary care and ENT settings.
The patients typically presenting with snoring, witnessed apnoeas, and non-restorative sleep. This case describes an adult patient with longstanding OSA established on continuous positive airway pressure (CPAP) therapy, who experienced a deterioration in sleep quality despite ongoing adherence. A home sleep polygraphy study performed while the patient was wearing CPAP appeared to show normal breathing during sleep, leading to uncertainty regarding the underlying cause of symptoms. This article highlights the diagnostic challenges associated with interpreting sleep studies , and emphasises the importance of a multidisciplinary approach to the management of these patients.

Introduction

Obstructive Sleep Apnoea is characterised by recurrent episodes of upper airway collapse during sleep. These events contribute to intermittent hypoxia, sleep fragmentation, and impaired daytime functioning.
While laboratory polysomnography remains the diagnostic gold standard, the increasing use of home sleep polygraphy [Learning Box 1] has introduced both opportunities and pitfalls. In particular, sleep studies conducted while a patient continues to use CPAP can mask ongoing or recurrent OSA and provide misleading reassurance to clinicians. This case illustrates how symptoms, examination findings, and sleep study interpretation must be integrated carefully to avoid misdiagnosis or delayed treatment optimisation.



Learning Box 1

Home Sleep Apnea Testing (HSAT):
A portable diagnostic device used to monitor oxygen levels, airflow, and respiratory effort during sleep, usually at home. It is less comprehensive but still effective in diagnosing moderate to severe OSA.

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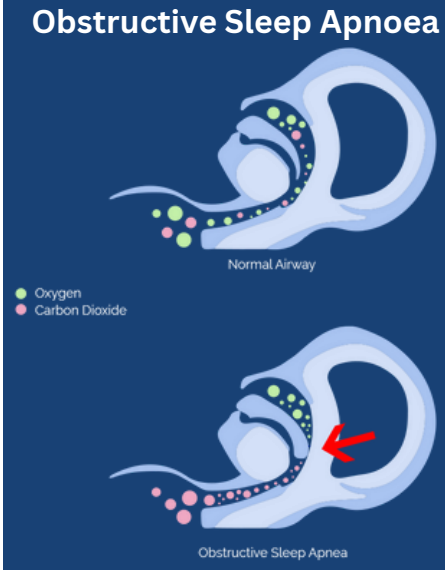
Case Presentation

Clinical History

An adult patient with a known history of OSA had been using CPAP consistently for several years and initially reported excellent improvement in symptoms. However, over the course of late 2024 and early 2025, the patient described increasingly unrefreshing sleep, persistent fatigue, cognitive fog, and a general sense of reduced wellbeing. These symptoms emerged despite apparently appropriate nightly use of CPAP. The patient also noticed nasal congestion and intermittent difficulty breathing nasally, and reported limited benefit when switching between fixed-pressure and auto-adjusting CPAP modes.

Examination

ENT examination revealed a mildly deviated nasal septum, congested nasal mucosa, and non-obstructive tonsils. These findings suggested that nasal factors might be contributing to reduced CPAP comfort and potentially impacting sleep continuity. A nasal steroid spray was prescribed to improve nasal airflow and alleviate mucosal congestion.



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Sleep Study Findings

A home respiratory polygraphy assessment was performed. [Learning Box 2}. The study estimated a total sleep time of nearly eight hours and recorded an Apnoea–Hypopnoea Index (AHI) of 5.1 events per hour. Oxygen saturation remained mostly stable, with a mean SpO_2 of 95% and a nadir of 90%, and there was no time spent below 90%. Snoring was detected in only a very small proportion of the night. The study did, however, document a Periodic Limb Movement Index of 26 per hour. The overall interpretation suggested normal breathing during sleep with moderate periodic limb movements.

Crucially, follow-up communication confirmed that this home sleep study had been performed while the patient continued to use their CPAP device. This has major implications for interpretation, as CPAP exerts a therapeutic effect during the study and can substantially alter respiratory indices.

Learning Box 2

Polysomnography (In-Lab Sleep Study): This is the most comprehensive method of diagnosing OSA. It measures multiple physiological parameters during sleep, including:

Brain activity (EEG)
Eye movement (EOG)
Muscle activity (EMG)
Heart rate (ECG)
Airflow (nasal and oral sensors)
Oxygen saturation (pulse oximetry)
Respiratory effort (chest and abdominal bands)

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Discussion

The key difficulty in this case arises from the influence of CPAP on the outcomes of the sleep study. When a patient undergoes sleep monitoring while using CPAP, the device actively prevents upper airway obstruction and reduces apnoeic and hypopnoeic events. This can lead to a false impression of normal breathing during sleep, even if the underlying OSA is inadequately treated or has recurred. As a result, such studies cannot reliably exclude persistent OSA, nor can they provide meaningful information about the adequacy of CPAP settings.

Despite the reassuring AHI [Learning Box 3] and oxygenation profile reported by the home study, the patient continued to experience symptoms highly suggestive of disrupted sleep, including significant daytime fatigue, cognitive slowing, and unrefreshing sleep. This discrepancy underscores the importance of prioritising clinical symptoms over numerical indices in cases where the study has been influenced by ongoing therapy. Potential explanations for the mismatch include suboptimal CPAP pressure, mask leak, poor mask fit, the emergence of treatment-related central apnoeas, coexisting sleep disorders such as periodic limb movements, and nasal obstruction limiting sleep quality or CPAP comfort. Any of these factors may produce impaired sleep despite superficially normal respiratory parameters.

Learning Box 3

Apnea-Hypopnea Index (AHI):
The Apnea-Hypopnea Index (AHI) is a key diagnostic criterion used to classify the severity of OSA based on the number of apneas (complete cessation of airflow) and hypopneas (partial reduction in airflow) that occur per hour of sleep.

- Mild OSA: AHI = 5 to 15 events per hour
- Moderate OSA: AHI = 15 to 30 events per hour
- Severe OSA: AHI > 30 events per hour

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Nasal symptoms played a significant role in this case. Nasal congestion and anatomical narrowing can reduce CPAP tolerance, encourage mouth breathing, and increase upper airway resistance, all of which may compromise effective treatment. In such scenarios, nasal steroid therapy is a reasonable first step, and further nasal surgical management may be also be considered to optimise nasal airflow and optimise CPAP delivery .

Given the persistence of symptoms and the limited interpretative value of a sleep study conducted on CPAP, the clinicians recommended repeating the evaluation without CPAP. A full laboratory polysomnography study would allow an accurate reassessment of the patient's untreated sleep architecture, OSA severity, and any coexisting sleep disorders. In parallel, a review of CPAP interfaces and pressure settings at a specialist sleep centre was advised, with consideration of alternative delivery systems such as nasal prongs or smaller mask designs. In such cases its important to work in parallel with a respiratory consultant.
A multidisciplinary approach is crucial in complex OSA. Management within a specialist sleep centre enables coordinated input from respiratory and ENT specialists, addressing both physiological and anatomical contributors to disease. This collaboration supports accurate reassessment, optimisation of CPAP therapy, and consideration of alternative treatment strategies, improving symptom control and treatment adherence.

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Conclusion

This case demonstrates the potential pitfalls of relying on home sleep polygraphy when conducted while a patient is using CPAP therapy. Although the study appeared to show normal breathing during sleep, it did not reflect untreated OSA severity or the effectiveness of CPAP. Persistent symptoms—especially in a patient with previously well-controlled OSA—should prompt clinicians to consider whether CPAP therapy is truly effective, whether nasal or anatomical factors are interfering with treatment, and whether additional sleep pathology may be present.

For GPs and ENT surgeons, this case reinforces the need for careful interpretation of home sleep studies, close attention to patient-reported symptoms, and a willingness to repeat diagnostic assessment when guided by clinical suspicion. A full in-lab polysomnography conducted without CPAP, combined with optimisation of nasal airway and CPAP interface, is essential to ensuring that patients with OSA receive treatment that is both effective and sustainable.

Finally, this case highlights the value of a multidisciplinary team (MDT) approach—integrating primary care, sleep medicine, and ENT expertise—to ensure comprehensive assessment, appropriate investigation, and long-term optimisation of OSA management.

Any further questions or learning points please contact Ms Ali.

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