Underlying Mechanisms for Coexisting Central and Obstructive Sleep Apnea: Nocturnal PaCO₂ and Poor Sleep Quality Are Key Issues

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Established Facts

- Treating coexisting central and obstructive sleep apnea is a challenging issue.

Novel Insights

- Hypocapnia, body position and quality of sleep are key mechanisms for coexisting central and obstructive sleep apnea.
- Polysomnography with transcutaneous PCO₂ nocturnal recordings help physicians during the diagnosis procedure and for proposing the appropriate treatment strategy.

Key Words

Central sleep apnea · Obstructive sleep apnea · Poor sleep quality · Nocturnal PaCO₂ · Servoventilation

Abstract

Coexisting central and obstructive sleep apnea is a challenging clinical situation. We report a case exhibiting an overnight shift from obstructive to central events. The central sleep apnea component was related to sleep instability, hyperventilation and low nocturnal PaCO₂. Both types of respiratory events were successfully treated with adaptive servoventilation.

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Introduction

Coexisting central (CSA) and obstructive sleep apnea (OSA) is a growing concern [1, 2] as most of the patients referred for sleep studies suffer from comorbidities such as cardiovascular and/or neurologic disorders which increase the likelihood of both central and obstructive events [3]. Managing these complex sleep breathing disorders is challenging in terms of treatment strategy. Knowledge of the underlying pathophysiological mechanisms is crucial to understand why central and obstructive events coexist and to provide the most appropriate type of ventilatory support.

Case Report

A 59-year-old obese (BMI = 33.1) male subject treated for hypercholesterolemia and type II diabetes was referred to our sleep laboratory for snoring, nocturnal choking and gasping as well as poor sleep quality. The patient was a night shift worker complaining of morning headaches and daytime fatigue. Sleepiness (Epworth) and depression (Beck) scores were in the normal ranges (5/24 and 5/63, respectively). The patient's medications were aspirin and metformin. He had no history of smoking, alcohol consumption or illicit drug use. Physical examination revealed a normal cardiopulmonary and neurological status. His blood pressure was 139/87 mm Hg, and his heart rate was 69 beats/min. Polysomnography showed a severe sleep breathing

Fig. 1. Polysomnography summary showing hypnogram, pulse oximetry, PtcCO₂ and body position sensor (from top to bottom). Typical respiratory patterns are highlighted with typical obstructive events (mean PtcCO₂: 42 mm Hg; left) and central events (mean PtcCO₂: 39.2 mm Hg; right).

Fig. 2. Percentage of central and obstructive AHI during the full night (full polysomnography) as well as the first and second part of the night.

disorder (apnea hypopnea index, AHI: 61.3 events/h) with two distinct patterns (fig. 1; table 1). During the first part of the night, the respiratory events were mainly obstructive (AHI: 41 obstructive events/h) and occurred in the nonsupine position with an oxygen desaturation index of 45/h and a mean nocturnal transcutaneous PCO_2 (PtcCO_2) of 42.1 mm Hg. Albeit for the microarousals ending the obstructive events, the sleep macrostructure was relatively preserved. During the second half of the night, the patient exhibited mainly central apneas (AHI: 76.5/h) while predominantly in the supine position with a mean PtcCO_2 of 39.2 mm Hg and an oxygen desaturation index of 73.5/h (fig. 2). Sleep fragmentation and instability was obvious, with a limited amount of stage 2 sleep, reduced slow-wave sleep and no REM sleep. A cardiologic evaluation including an exercise test reasonably ruled out significant systolic cardiac failure. Magnetic resonance brain imaging was normal. Blood gases (PaO_2 : 82 mm Hg; PaCO_2 : 39 mm Hg) were in the normal range. Continuous positive airway pressure was initially used to treat both OSA and CSA [4] but was not effective in controlling central events (residual central AHI: 34.3/h). A control sleep study confirmed that CPAP was ineffective, with a residual central index of 25/h in the supine or lateral position.

Treatment with adaptive servoventilation (AutosetCS™ settings: positive expiratory pressure: 4 cm H_2O , pressure support: 3–10 cm H_2O) [5] was then applied and achieved a full correction of both OSA and CSA (built-in software AHI: 1.1/h).

Table 1. Details of the full night, as well as the first and second parts of the night recordings

	Full polysomnography	First part: mainly obstructive events	Second part: mainly central events
TST, min	528	329	198
REM sleep, % TST	5.7	8.4	0.4
NREM sleep stage, % TST			
N1	19.5	9.1	40.1
N2	66	69.2	59.5
N3	8.8	13.2	0
Sleeping position, % TST			
Supine	36.1	4.2	100
Nonsupine	63.9	95.8	0
AHI, /h	61.3	53.5	76.9
Central events, /h	34.8	12.5	76
Supine position	28.1	4.2	76
Nonsupine position	6.2	9.3	0
Obstructive events, /h	26.5	41	0.8
Supine position	0.3	0	0.8
Nonsupine position	26.2	39.2	0
Median PtcCO_2 , mm Hg	42	42	38.9
ODI >3%, /h	56.1	47.5	73.5
Mean saturation, %	92.6	93.2	90.4
Average desaturation, %	8	5.1	11.8

TST = Total sleep time; ODI = oxygen desaturation index.

Discussion

Excessive ventilation during sleep inducing low PaCO_2 brings patients close to their apnea threshold and induces central apneas and periodic breathing [2, 6, 7]. Our case illustrates that the overnight shift from OSA to CSA events was associated with a reduction in mean transcutaneous PtcCO_2 . A decrease in PtcCO_2 results from an increase in ventilation that can be explained by several factors.

First, the decrease in PtcCO_2 and shift in the apnea type may be linked to an overnight deterioration in cardiac function leading to hyperventilation. Our patient did not suffer from overt cardiac failure but had diastolic hypertension and diabetes, which are both frequently associated with diastolic cardiac failure, which is frequently underdiagnosed and associated with CSA.

Second, we cannot exclude an overnight fluid shift from the legs to the neck that can affect upper airway mechanics and lead to OSA, whereas fluid shift to the lungs can provoke hyperventilation, hypocapnia and CSA [8].

Third, poor sleep quality and rapid transitions between wakefulness and sleep (i.e. the possibility of arous-

al responses with hyperventilation) may be an additional factor. In patients with increased chemoresponsiveness, an increased central gain will promote and perpetuate respiratory instability and central events. This mechanism contributes to CSA in cardiac failure but is also a trigger for central apneas in any situation associated with chronic hyperventilation and/or sleep fragmentation [9].

Our case illustrates that central apneas occur concurrently with sleep instability and low PaCO₂, both at sleep initiation and during the second part of the night. Figure 1 also depicts the central role of the sleeping posi-

tion with illustrative evidence that the lateral sleeping position reduced the severity of CSA [10]. The respective contribution of unstable sleep, cardiac function and fluid shift is difficult to identify, but our clinical case clearly shows that hypocapnia, body position and quality of sleep are key mechanisms for CSA and OSA.

Financial Disclosure and Conflicts of Interest

None of the authors have any conflicts of interest related to the study.

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