Sleep Medicine 33 (2017) 160–164

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Images in Sleep Medicine

# Severe sleep apnea resistant to all treatments

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ARTICLE INFO

*Article history:*
Received 26 January 2017
Received in revised form
17 February 2017
Accepted 21 February 2017
Available online 10 March 2017

*Keywords:*
Opioid
Benzodiazepine
Adaptive servoventilator
Hypoxemia
Obstructive sleep apnea
Central sleep apnea

## 1. Introduction to the case

A 62-year-old female was referred to the Sleep Clinic with severe daytime somnolence despite sleeping 11–13 h/day, falling asleep during conversations and at meals with an Epworth Sleepiness Scale score of 24/24. She was diagnosed with obstructive sleep apnea (OSA) of unknown severity >10 years ago and underwent three titration polysomnograms (PSGs). She was using bilevel PAP in the spontaneous mode (BPAP) at 24/20 cm of water pressure (CWP) with supplemental oxygen bled in at 3 L/min and endorsed several pressure-related side effects. She had ongoing issues with imbalance, falls, cognitive impairment and chronic pain for the last three years and was increasingly homebound. During this time period, her benzodiazepine and opioid medication dosages had steadily escalated. She was taking diazepam 20 mg/day, oxycodone extended-release 80 mg four times/day and short-acting oxycodone 5 mg six times/day. Medical history was significant for chronic headache/back pain, anxiety, lacunar infarcts, cerebral amyloid angiopathy, systemic hypertension, Hashimoto's thyroiditis and diabetes mellitus. Body mass index was 33 kg/m$^2$, neck circumference 40 cm, oropharynx Friedman class IV and blood pressure was normal. Thyroid stimulating hormone was normal, bicarbonate 30 mmol/L, outside pulmonary function tests were unavailable and ejection fraction on echocardiogram was 69%. BPAP download showed very sporadic usage.

## 2. Image analysis

Split-night PSG was performed as previous PSGs could not be retrieved, revealing severe OSA and associated hypoxemia, with emergence of central sleep apnea (CSA) on positive airway pressure secondary to opioid medication (Fig. 1). Bradypnea was noted with a respiratory rate of 10 breaths per minute and an ataxic breathing pattern was seen at times both during the diagnostic and titration portions of the study (Fig. 2). Arterial blood gas (ABG) could not be obtained despite several attempts.

Our patient was prescribed continuous positive airway pressure (CPAP) at 10 CWP, as most obstructive events appeared to be eliminated at this pressure, with the understanding that it was crucial to decrease the use of opioid/benzodiazepine medications for optimal therapy and symptom control. Although CSA could develop/worsen with excessive PAP pressures, the high doses of opioid medication were thought to be a more important contributor. Full-night titration PSG was offered, but it was discussed that the chance of success was relatively low. Over the next two months, she successfully decreased diazepam to 5 mg/day, extended-release oxycodone to 80 mg/day and discontinued short-acting oxycodone. Subsequent PSG showed elimination of sleep-disordered breathing SDB/hypoxemia on CPAP at 10 CWP in the nonsupine position in non-rapid eye movement sleep (Fig. 3). Respiratory rate was 16 breaths per minute and no abnormal breathing patterns were noted (Fig. 4). While end-tidal/transcutaneous carbon dioxide monitoring was not performed, ABG obtained the morning after the follow-up PSG did not reveal evidence of significant hypoventilation ($PaCO2 = 45$ mm of Hg, range = 35–45 mm of Hg). CPAP was reset to 12 CWP as no rapid eye movement (REM) sleep was seen on titration PSG. Download five months later showed excellent usage and no residual SDB. Daytime sleepiness, although persistent, was improved from previous assessments.

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http://dx.doi.org/10.1016/j.sleep.2017.02.016
1389-9457/

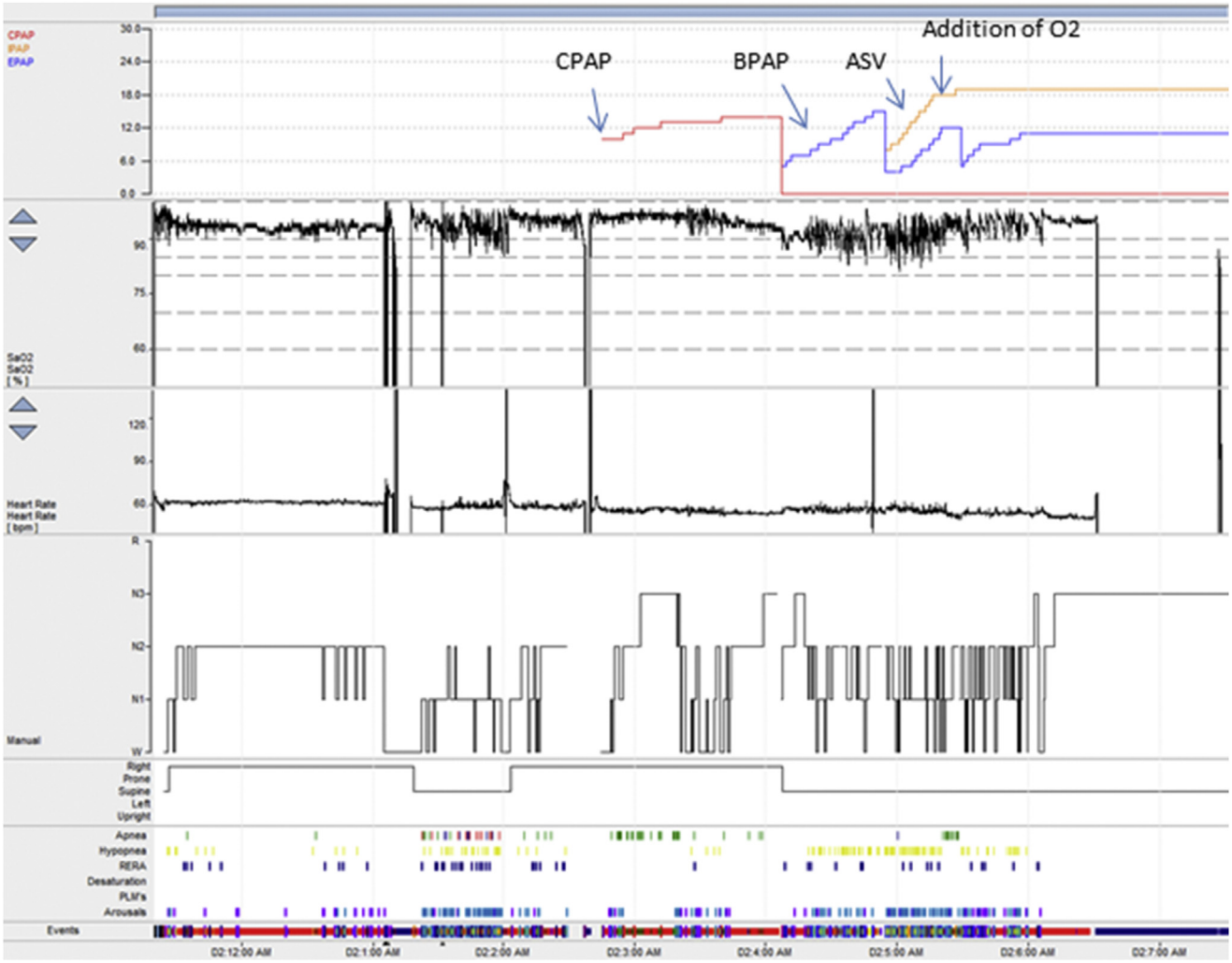


**Fig. 1. Hypnogram showing diagnostic and titration portions of the split-night polysomnogram**. Apneas marked in red are obstructive and those in green are central. Overall apnea-hypopnea index (AHI) was 25/h, respiratory disturbance index (RDI) was 37/h, with a central apnea index (CAI) of 4/h during the diagnostic study, and an oxyhemoglobin saturation nadir of 85%. No rapid eye movement (REM) sleep was observed. On CPAP up to 14 cm water pressure, AHI increased to 32/h with a CAI of 28/h. As CPAP failed to control disordered breathing events, the patient was switched to BPAP; AHI was 81/h, with mostly central apneas. In view of persistent central sleep-disordered breathing events on BPAP up to 19/14 cm water pressure, an adaptive servoventilator device was tried; at the maximal settings, AHI was 28/h with persistent central events. No improvement was noted with addition of oxygen for significant hypoxemia during the last h of the study. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)