A 73-year-old male presents with hypersomnolence and witnessed pauses in breathing during sleep lasting 30-60 seconds. He was diagnosed with obstructive sleep apnea in 1996 and underwent uvulopalatopharyngoplasty (UPPP). His symptoms re-emerged 2 years later. Multiple sleep studies done since then demonstrated Cheyne Stokes breathing. He was treated with continuous positive airway pressure (CPAP), bilevel PAP (with and without backup rate), and/or supplemental oxygen but he continued to have unabated diurnal hypersomnolence and his wife noted incomplete resolution of pauses in breathing.

He had history of atrial fibrillation, coronary artery disease, and permanent pacemaker placement. A representative tracing from his diagnostic polysomnogram is shown below (display range 240 seconds). Which of the following treatments is likely to be MOST effective in treating his sleep disordered breathing?

A. Beta blockers
B. Adaptive Servoventilation
C. Automatically adjusting PAP
D. Theophylline
E. Supplemental CO₂

Disclosure Statement
This is not an industry supported study. Dr. Kakkar has indicated no financial conflicts of interest.
Adaptive servoventilation

DISCUSSION

This patient has Cheyne Stokes breathing pattern (CSB). The classic waxing and waning breathing pattern occurs due to oscillating respiratory drive, which results in ventilatory overcompensation in response to changes in arterial CO₂. Most common causes of CSB are systolic heart failure (ejection fraction <40%), stroke, and ascension to high altitude. CSB is thought to result from hyperresponsiveness of chemoreceptors to CO₂ changes, which is amplified by hypoxia and, in patients with heart failure, prolonged circulatory time. Sleep state predisposes to instability of respiratory control system due to loss of “behavioral influence” and rise in the apneic threshold of PaCO₂. Many of these patients demonstrate awake hypocarbia, predisposing them to central apnea followed by initiation and perpetuation of apnea-hyperpnea cycles. CSB in heart failure is associated with prolonged circulatory time (>45 seconds) and apnea-hyperpnea cycle length. Presence of CSB is associated with increased mortality in patients with heart failure.1

Many treatments are available for CSB. Treatment of underlying cause, like optimization of medical regimen, overdrive atrial pacing, cardiac resynchronization with biventricular pacing, and heart transplant are effective treatments in patients with heart failure. Descent to lower altitudes is effective in CSB associated with high altitude. Theophylline and acetazolamide have been shown to be effective in attenuating the CSB in short-term trials.2,3 However, long-term efficacy and safety is not proven. CPAP and bilevel PAP have been used to treat CSB.4 PAP therapy improves oxygenation, decreases collapsibility of hypopharynx, and improves hemodynamics. Added dead space or inhaled CO₂ are also effective in stabilizing the breathing pattern in CSB.5 Safety of CO₂ delivery devices, increased mechanical load, sympathetic stimulation, and increased arousal index remain concerns with these devices. Oxygen decreases the hypoxia and therefore hypocarbia resulting from compensatory hyperventilation.6

Adaptive servoventilation (ASV) continuously calculates target ventilation throughout the night (90% of recent average ventilation) and adjusts pressure support to achieve that target. Default EPAP is 5 cm H₂O, which may be adjusted in 1-2 cm increments to resolve any upper airway obstruction. The pressure support is adjusted by the device depending upon the target ventilation, has default settings of 3-10 cm, and can be adjusted. The machine has a default back-up rate of 15 breaths per minute. ASV is more effective than supplemental oxygen, CPAP, and bilevel PAP.8 Contraindications to the use of ASV include moderate to severe COPD, chronic hypercarbia (>45 mm Hg), restrictive thoracic or neuromuscular disease, and profound hypoventilation.

According to unpublished data, beta-blockers may be effective in treating the CSB, however, the efficacy is unestablished.9 Automatically adjusting PAP (Auto PAP) is contraindicated for treatment of central sleep apnea. Our patient demonstrated normal awake PaCO₂ (37 mm Hg), short apnea-hyperpnea cycle length, normal circulatory time (calculated from end of central apnea to the nadir of desaturation event immediately following the central apnea), and severe fragmentation of sleep. He was successfully treated with ASV, which led to resolution of CSB on polysomnographic recording, attenuated arousals, and improved daytime functioning.