CLINICAL VIGNETTE

Central Sleep Apnea Post Hypoglossal Nerve Stimulator Implantation

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History

A 61-year-old male with moderate obstructive sleep apnea (OSA) underwent hypoglossal nerve stimulator (HGNS) implantation after being intolerant to positive airway pressure (PAP) therapy and mandibular advancement device (MAD). His respiratory event index (REI) of 27/hr was titrated to CPAP of 7 cm H2O with no central events noted. The HGNS device is implanted to reduce the apneic events in individuals with OSA by stimulating the hypoglossal nerve relieving upper airway obstruction. The unit contains three parts: a lead sensing chest wall movement, a processor which interprets the movement and estimates end expiration, and a stimulation cuff which encases the appropriate subbranch(es) of the hypoglossal nerve to stimulate tongue protrusion and prevent obstruction. The cuff has 3 electrodes which can be configured as a cathode (-) or anode (+). The device is usually activated about a month after implantation and requires subsequent lab titration a few months later.

Physical Examination Findings

The patient was afebrile with a heart rate of 60 beats/min, blood pressure of 131/69 mm Hg and oxygen saturation of 98%. His hypoglossal nerve stimulation scars were well healed. The rest of his physical exam was unremarkable.

Diagnostic Studies

Following activation of the device, the patient presented for voltage titration in the sleep lab. An initial bipolar (+-+) electrode configuration was used. This limits the electrical activity to local area around the cuff. The voltage titration was increased to 1.2V. However, residual central apneas started to occur at a voltage of 1.1V, with simultaneously no airflow nor movement of chest wall (Figure 1). The overall response was unanticipated given the patient's original diagnosis of obstructive sleep apnea.

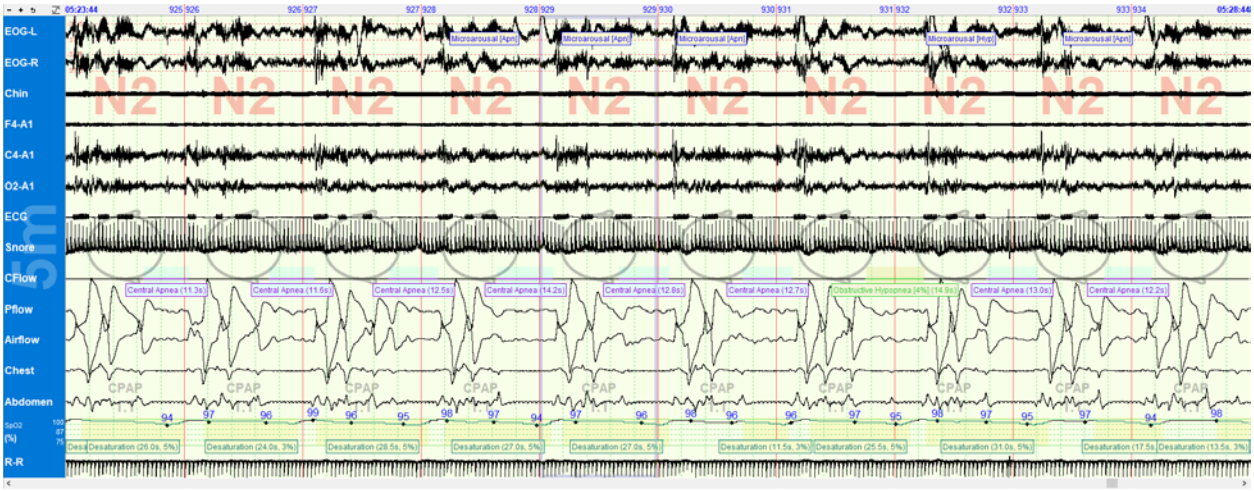

Figure 1: Polysomnogram with hypoglossal nerve stimulation titration

Clinical Course

Awake endoscopy with advanced programming noted good response to stimulation at the tongue base, but poor response at the soft palate. The configuration was changed to a monopolar configuration with a range of 0.6-1.6V, which allows for a widened electric field. Follow-up polysomnography (PSG) titration with new settings demonstrated good control of OSA with an apnea-hypopnea index (AHI) of 2.08 events/hr (normal <5 events/hr) at 1V (Figure 2). Unfortunately, the patient could

not tolerate the new settings and was eventually explanted. He was started on BIPAP (max IPAP 25, min EPAP 7, max PS 8, min PS 4) which improved symptoms with residual AHI of 0.6/hr and 100% compliance on modem check.

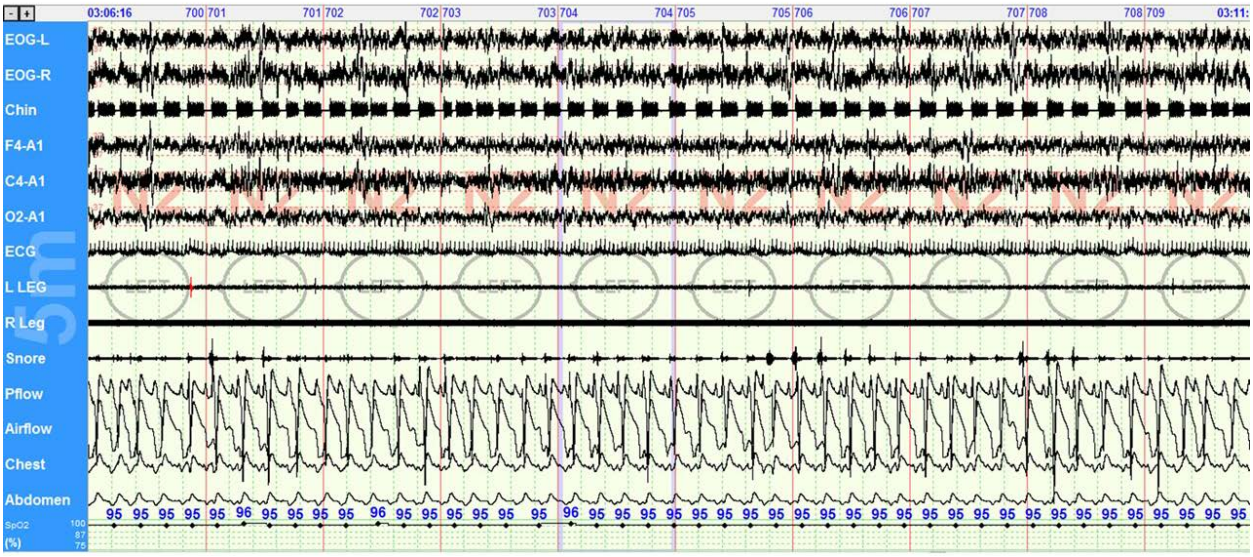

Figure 2: Polysomnography titration after adjustments in his HNS settings

Discussion

Hypoglossal Nerve Stimulation is a viable alternative treatment[1] for obstructive sleep apnea (OSA). With increasing numbers of patients implanted, increased complications have occurred.

Central sleep apnea has been reported following treatment of OSA with positive airway pressure and mandibular advancement devices.[2,3] The pathophysiology of these events is uncertain, however is speculated that a low arousal threshold leads to frequent residual arousals. High loop gain and chemoreceptor sensitivity may be the main causes.

Frequent residual arousals have been a cause of central apnea following OSA treatment.[2,4] Low arousal threshold and lingering airway obstruction result in abrupt hyperventilation as CO2 sensitivity adjusts, and awakes with subsequent O2 reduction below apneic threshold.[2,4,5] Theoretically, further improvements in airway patency would reduce arousals and interrupt this process.[2]

The pathophysiology of central sleep apnea is frequently described in terms of loop gain.[4-6] In the setting of a ventilatory disturbance, the interaction between the plant (lung and tissues), the controller (chemoreceptors), and circulation time results in ventilatory response. If the response is exaggerated, high loop gain causes an overcorrection of PaCO2 and a subsequent central apnea after crossing the apneic threshold.[4,5] In primary central sleep apnea and central apnea with Cheyne-Stokes breathing, prolonged circulation time is thought to be the significant culprit in elevating loop gain.[6] However, in central apnea following OSA treatment, changes in the controller or chemoreceptor sensitivity may play a larger role.[4]

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