



Case Report

Insomnia: A Relative Contraindication to Upper Airway Stimulation

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Abstract

Both Obstructive Sleep Apnea (OSA) and insomnia are common sleep disorders. A subset of patients have both OSA and insomnia. Upper Airway Stimulation (UAS), also known as hypoglossal nerve stimulation, has emerged as a novel surgical treatment for OSA in patients intolerant of Continuous Positive Airway Pressure (CPAP). Upper airway stimulation is designed to increase upper airway muscle tone during sleep, thereby alleviating obstructive events. Stimulation of the hypoglossal nerve activates the genioglossus muscle leading to anterior protrusion of the tongue and soft palate during inspiration. This form of therapy may worsen difficulties with sleep initiation or maintenance in patients with underlying insomnia. This report highlights the importance of assessing for co-morbid insomnia during the preoperative evaluation of patients considering upper airway stimulation.

Keywords: Insomnia; Obstructive sleep apnea; Upper airway stimulation

Introduction

Obstructive Sleep Apnea (OSA) affects a significant portion of the adult population. Insomnia, defined as difficulty with initiating or maintaining sleep with associated daytime symptoms, is also a common sleep disorder. A subset of patients have both OSA and insomnia.

Many patients do not tolerate Continuous Positive Airway Pressure (CPAP) which is considered to be the gold-standard treatment for OSA [1]. Over the past thirty years, numerous surgeries have been developed to enlarge the upper airway but fail to address the underlying loss of muscle tone associated with sleep onset which is pivotal to the

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pathogenesis of obstructive sleep apnea. In contrast, Upper Airway Stimulation (UAS), also known as hypoglossal nerve stimulation, is designed to increase upper airway muscle tone, thereby alleviating obstructive events. Stimulation of the hypoglossal nerve activates the principal upper airway dilator, the genioglossus muscle. Due to mechanical coupling of the palatoglossus muscle, which insinuates itself into the intrinsic tongue musculature, there is also an increase in the retropalatal airway with each anterior tongue movement. Therefore, upper airway stimulation leads to an increase in both the retroglottal and retropalatal airway during sleep. In 2014, Inspire (TM) was the first upper airway stimulation device to be approved by the Food and Drug Administration [2]. Current indications for UAS include: CPAP intolerance, age 22 years or older, BMI <35, Apnea Hypopnea Index (AHI) 15-65 and absence of complete concentric collapse of the velopharynx during drug induced sleep endoscopy.

The Hypoglossal Nerve Stimulator consists of three components: (1) the Nerve Stimulator, (2) the Neurogenerator and (3) the Respiratory sensor. During sleep, the respiratory sensor detects a change in intrathoracic pressure with inspiration leading to stimulation of the hypoglossal nerve via the neurogenerator and nerve stimulator electrode. This leads to a stiffening of the tongue as well as anterior displacement of both the tongue and soft palate.

A 5-year follow up study on patients enrolled in the initial Stimulation Therapy for Apnea Reduction (STAR) trial was published in 2018 [3]. The median AHI decreased from 32 to 14 at 5-years with good durability of effect. The response rate, defined as at least a 50% decrease in AHI and a postoperative AHI < 20 was 75% and 44% had resolution of their sleep apnea with an AHI<5. In addition, quality of life measures were significantly improved at 1 and 5 years following implantation. Nightly use was 86% and 80% at 1 and 5 years, respectively. Serious complications related to UAS were rare, although tongue abrasion and discomfort with stimulation required adjustment in settings or use of a dental guard in up to 20% of patients [3,4]. Post-operative morbidity is low with an immediate return to a normal diet and typically only mild incisional pain.

To date, over 3000 UAS devices have been implanted. The future of upper airway stimulation includes the potential expansion of current surgical indications and identification of patient factors which may affect surgical outcomes. Many of such studies have been recently reported and others are underway [4-8]. In contrast to studies focusing on expansion of indications, very little has been reported regarding identification of additional exclusion criteria including the possible interaction of various sleep disorders on UAS therapy as a treatment for OSA. This report identifies insomnia, an additional common sleep disorder, as a potentially relative contraindication to UAS therapy.

Insomnia is defined as difficulty with initiating or maintaining sleep with associated daytime symptoms including fatigue, daytime sleepiness, cognitive difficulties, mood disturbance, and social or work dysfunction. There are several types of insomnia: primary

insomnia, also referred to as psychophysiological insomnia, and secondary insomnia due to a mental disorder or medical condition [9].

Psychophysiological insomnia is seen in approximately 15% of patients evaluated in a sleep center and 2% of the general population [10]. The key feature of psychophysiological insomnia is heightened mental or physical arousal related to sleep and learned associations and behaviors that interfere with sleep for a period of greater than one month. Increased mental arousal is often described as an overactive mind or “racing thoughts” which interfere with initiation or maintenance of sleep. Heightened somatic tension associated with bedtime can also hinder sleep initiation. Polysomnographic findings include an increased sleep latency greater than 30 minutes. Additional findings include an increase in wake after sleep onset, reduced sleep efficiency and an increase in stage 1 non-rapid eye movement sleep (NREM 1), also known as light sleep.

Insomnia due to a mental disorder affects approximately 3% of the population [11]. The insomnia is typically more prominent than sleep disturbances usually associated with the mental disorder. It is most common in women and middle age adults. The key feature of this type of insomnia is that it begins with the onset of the mental disorder and its course waxes and wanes in concert with alterations of the underlying mental disorder, most commonly anxiety and major depressive disorder. In general, difficulty initiating sleep is observed in younger patients with anxiety disorder. Difficulty maintaining sleep, as demonstrated by early morning awakenings, is more common in the elderly. Polysomnographic features include an increased sleep latency and decreased sleep efficiency. Increased nocturnal arousals are observed as well as an increase in NREM 1 and NREM 2 sleep and a decrease in NREM 3 and Rapid Eye Movement (REM) sleep, also known as deep sleep.

Insomnia due to a medical condition is identified in 4% of patients seen in sleep centers and is more common in the elderly [12]. The onset of this type of insomnia is associated with onset of the diagnosed medical condition and the course of the two disorders is closely aligned. Numerous medical and neurologic conditions can lead to this often chronic form of insomnia. The most common causative medical conditions are those that result in pain. Other medical conditions leading to insomnia include those that limit mobility and those associated with difficulty breathing, such as nocturnal asthma. The following case report exemplifies the potential interplay between insomnia and UAS therapy for treatment of OSA.

Case Study

A 66 year old male was evaluated in the Otolaryngology clinic for review of surgical treatment options for his OSA. Recent polysomnography revealed an apnea hypopnea index of 25 and an oxygen saturation nadir of 85%. The patient reported a several year history of worsening sleep quality associated with snoring, frequent nighttime arousals/awakenings, non-refreshing sleep and excessive daytime sleepiness. Despite repetitive attempts at CPAP use under the supervision of a sleep physician the patient remained CPAP intolerant. Past medical history was significant for OSA, essential hypertension and moderate to severe arthritis of his shoulders with associated chronic pain. Past surgical history was significant for tonsillectomy and arthroscopic shoulder surgery x 2. Current medications included Lisinopril, Hydrochlorothiazide and Naproxen. Physical examination was significant for a body mass index 32, Friedman tongue position III,

mild macroglossia, moderate elongation of the soft palate and surgically absent tonsils.

The patient underwent drug induced sleep endoscopy and was found to have partial anterior-posterior collapse at the velopharynx (no complete concentric collapse), no oropharyngeal collapse, partial collapse at the base of tongue and no epiglottic collapse and was deemed a candidate for upper airway stimulation therapy. The device was activated one month following uneventful implantation and a formal titration study was completed three months later. The recommended setting was 1.7 Volts (within the typical range previously reported) with a residual AHI of 3.

At the time of his six month follow up visit, the patient reported worsening of sleep quality and daytime sleepiness with use of his device. Although his spouse reported resolution of his snoring and apneic events while using the device, a repeat polysomnogram was performed to insure that his current settings were still optimal for control of his OSA. This study confirmed that his current settings were adequate. On follow-up, the patient reported increased difficulty maintaining sleep on the nights he used his UAS device. Although he reported no difficulty initiating asleep with use of the delay timer (set for 30 minutes), he experienced difficulty resuming sleep following frequent awakenings due to pain. Despite relief of his pain with a change in body position, he reported difficulty re-initiating sleep due to the repetitive tongue movement associated with UAS device use. Review of his device revealed an average nightly use of only 3.5 hours. The patient was instructed to turn his device off and back on to reset the delay timer during these episodes of prolonged wakefulness after sleep onset.

At the time of his next three month follow up visit, review of the device revealed an average nightly use of just 1.5 hours and the patient reported he had recently discontinued use of the device altogether. The patient reported that he felt like he had a better sleep quality and subsequently less daytime sleepiness following nights when he did not use his UAS device. Ultimately, due to the frequency of his nighttime arousals from pain and additional sleep disruption by having to repetitively reset his device the patient decided to discontinue UAS therapy. Prior to making a final decision regarding explanation of the device, the patient was referred back to his Orthopedic surgeon and pain clinic to determine if additional medical or surgical treatment options for his chronic shoulder pain were available.

Discussion

In summary, the overall efficacy of upper airway stimulation is high, with approximately 70% responding to therapy and surgical morbidity is low in comparison to traditional structural surgery for OSA. As with any relatively new procedure many questions remain including ideal patient selection, long-term compliance rates and durability of results. Both surgeons and sleep medicine specialists need to be aware of the potential interaction between UAS therapy for the treatment of OSA and pre-existing insomnia. Regardless of subtype, the key features of insomnia capable of interfering with successful UAS therapy include prolonged sleep latency and frequent nocturnal arousals. Future research on the effect and treatment of insomnia on UAS therapy is needed. At this time indications for UAS include only: CPAP intolerance, age 22 years or older, BMI <35, Apnea Hypopnea Index (AHI) 15-65 and absence of complete concentric collapse of the velopharynx during drug induced sleep endoscopy, with

few specified contraindications. It appears that untreated insomnia may be a relative contraindication to upper airway stimulation. Additional prospective research on the effect and treatment of insomnia on UAS therapy is needed.

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