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Anesthesia for Hypoglossal Nerve Stimulator: a Case Report and Anesthesia Implications

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Abstract

Patients with obstructive sleep apnea (OSA) are at increased risk of perioperative complications. Continuous positive airway pressure (CPAP) has been the mainstay of the treatment. Despite the success of CPAP therapy patient compliance remains a problem. Hypoglossal nerve stimulator is a relatively new option for the patients who do not tolerate CPAP therapy. This procedure has been shown to decrease the severity and symptoms of OSA in select patients. This therapy has been approved by the U.S. Food and Drug Administration since April 2014. We describe in detail anesthetic management and specific considerations of a patient with OSA needing the surgery for Hypoglossal nerve stimulator implant.

Keywords: Obstructive Sleep Apnea; Continuous Positive Airway Pressure: Hypoglossal Nerve Stimulator

Introduction

Obstructive sleep apnea (OSA) is a common disorder characterized by repetitive collapse of the upper airway during sleep, resulting in recurrent arousal [1]. OSA can lead to adverse health outcomes, including cerebrovascular disease and cardiovascular disorders such as hypertension, ischemic heart disease, arrhythmias, pulmonary hypertension, and congestive heart failure. OSA is also associated with metabolic syndrome, depression, and increased risk of accidents [2]. Risk factors for OSA include alcohol consumption, smoking, obesity, increased neck circumference, male sex, advanced age, tonsillar and adenoidal hypertrophy, macroglossia, nasal obstruction, and craniofacial abnormalities [3]. Although numerous methods have been used to restore upper airway patency during sleep, no single treatment modality has been shown to provide relief to all patients with this disorder, as all are only partially effective and/or may be poorly tolerated. So far continuous positive airway pressure (CPAP) has been the mainstay of treatment.

Hypoglossal nerve stimulation is a useful second-line therapy in patients who cannot tolerate CPAP machines or intraoral devices. The surgeon implants a device containing a neurostimulator subcutaneously in the patient's chest, with one lead attached to the patient's hypoglossal nerve (cranial nerve XII) at the base of the tongue, and one lead implanted in the patient's chest. The lead in the chest consists of a pressure sensor that detects breathing. Information about the respiration pattern is relayed to the device, which stimulates the hypoglossal nerve. When stimulated, the tongue moves forward, thus opening the airway. The patient activates the device by remote control before going to sleep. The device turns on after 20 minutes to minimize disrupting the patient's sleep onset; the device is deactivated remotely when the patient wakes up [4].

Report of Case

A written patient consent was obtained for this case report. A 34-year-old male with a diagnosis of severe OSA was scheduled for Hypoglossal Nerve Stimulator Implant surgery. The patient reported snoring, obstructive breathing and daytime somnolence. His symptoms began many years ago, unchanged since that time. He was diagnosed with OSA and treated with a CPAP machine. The patient is a non-smoker. His sleep studies were as follows.

Pre-CPAP

His initial sleep study revealed an average awake SaO2 of 95%, while asleep he had an AHI of 42/hr with a nadir SaO2 of 88%. The patient's ox hemoglobin saturation was below 90% during <1% of the night. Snoring was noted frequently. The patient slept only in the supine position during the night. There were frequent brief awakenings. Lead II on his cardiac monitor revealed what appeared to be an irregular heart rhythm. The heart rate varied from 50 to 96 bpm during the recording.

Post-CPAP

The patient used auto-CPAP during the night. At a CPAP of 6.5 cm H2O, the patient had no respiratory events. The average oxygen saturation was 97%. The lowest SaO2 was 94%. The heart rate ranged from 57 to 104 bpm during the night; it is difficult to infer whether the tachycardia (>90bpm) was actually occurring while asleep. For the majority of the night the patient was supine. Based on the results of the sleep study, patient was prescribed a CPAP of 7 cmH2O. Follow-up compliance tracking showed an average use of 7.5 hours nightly, use >4hrs 93% of nights, no leak around the mask, and AHI 1.4/hr. After a year of CPAP use, patient reported discomfort with the mask, inability to tolerate the CPAP machine, and was willing to explore the surgical option of implanting a Hypoglossal Nerve Stimulator.

Intraoperative Management

Induction

Patient's height was 176 cm, weight was 73 kg, body mass index was 26 kg/m2, and neck circumference was 39cm with a Mallampati class III airway. While placing the standard ASA monitors, care was taken not to place ECG leads on the right anterior chest because this area was to be prepped into the surgical field. The patient was pre-oxygenated with 100% oxygen for 3 minutes. Anesthesia was induced with intravenous 0.2mg glycopyrrolate, 2mg midazolam, 100mcg fentanyl, and slow titration of 120mg propofol with assurance of assisted ventilation. Oral endotracheal intubation was facilitated with 100 mg succinylcholine and accomplished using a MacGrath video-laryngoscope.

There is a close relationship between OSA and difficult intubation, as both are associated with upper airway abnormalities and morphological changes. Predictors of difficult laryngoscopy and intubation such as high Mallampati score, decreased thyromental distance, increased neck circumference, and crowded oropharynx are common in OSA. OSA, especially severe OSA, also predicts difficult laryngoscopy and difficult intubation, independent of obesity. Similarly, patients with a history of difficult intubation have a higher incidence of OSA [5]. The equipment for management of a difficult airway should be in place before induction of general anesthesia. A laryngeal mask airway and fiberoptic devices may be useful. Two-person mask ventilation may be needed to attain adequate ventilation. An oropharyngeal or nasopharyngeal airway (or supraglottic airway) may help maintain airway patency until the patient is successfully intubated.

Positioning

The endotracheal tube is secured to the left side of the mouth and a gauze packing is placed between the molar teeth of the left side ensuring adequate space to visualize tongue movement during intraoperative assessment of tongue muscles activation. The endotracheal tube should be well secured as this is a shared airway and reintubation after an inadvertent extubation can be challenging intraoperatively. The operating room table is then positioned such that the patient head is 180 degrees from the anesthesiologist and the anesthesia machine.

Maintenance

Maintenance of anesthesia was achieved by remifentanil infusion titrated to the rate of 0.05-0.2 mcg/kg/min. and one MAC of desflurane with FiO2 of 50%. No muscle relaxants are used after intubation to facilitate hypoglossal nerve integrity monitoring intraoperatively.

Surgical Procedure

The submandibular incision was made within the crease on the right side of the neck. The mylohyoid muscle was retracted anteriorly and hypoglossal nerve identified. The cuff electrode of the hypoglossal nerve stimulator implant was placed distally to encase the branches innervating the protusor muscle. Intraoperative testing confirmed the activation of genioglossus muscle and forward tongue protrusion. A second incision was placed on the right side of the chest wall 3cm below the clavicle and a pocket was created above the pectoralis muscle. The electrodes from the hypoglossal nerve stimulator were tunneled in the sub-platysmal plane down towards the sub-clavicular pocket. A third incision was created in the axillary line below the inferior edge of the pectoralis muscle to create a space between the external and internal intercostal muscles in the fifth intercostal space. The pleural respiratory sensor was placed in the intercostal space pocket and the sensing lead electrode was tunneled below the subcutaneous tissue towards the sub-clavicular pocket. A diagnostic evaluation was performed to confirm good respiratory sensing, as well as effective hypoglossal nerve stimulation by visualization of tongue protrusion.

Nerve Integrity Monitoring (NIM)

Bipolar NIM monitoring was used to detect the activity of the hypoglossal nerve fibers during intraoperative electrical stimulation testing. Two color coded channels are used in this procedure. Blue inclusion channel is placed on the right side of midline deep in the tissues of mandible targeting the genioglossus muscles including oblique and horizontal section. These muscles are the protrusors and stiffeners of the tongue. Red exclusion channel is placed 5cm from the tip of the tongue dorsum superficially below the mucous membrane targeting the hyoglossus and styloglossus muscles. These muscles are the retractors of the tongue. [6]

Emergence

We extubate patients with OSA "wide awake." All the anesthetic agents were discontinued and oropharynx carefully suctioned. Awake extubation is generally safer as the return of airway tone, reflexes and respiratory drive allows the patient to maintain their own airway. Patient was extubated in a head-up position and remained semi-upright during the early recovery period. Supplemental oxygen is provided after extubation.

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We administer dexamethasone and a 5HT3 antagonist as an antiemetic prophylaxis.

Postoperative Management

Patients with obstructive sleep apnea are at high risk of developing postoperative complications and should be closely monitored in the postoperative anesthesia care unit (PACU). Society of Anesthesia and Sleep Medicine Guidelines on Preoperative Screening and assessment of Adult patients with OSA recommends development of institutional protocol for patients with known or suspected OSA. Our PACU protocol for OSA includes identification with a blue colored OSA band for the patient, blue OSA sign at the head of the patient bed, and a room-air challenge test to categorize the severity of the risk based on the initial room air challenge test.

Up-regulation of central opioid receptors and recurrent hypoxemia in OSA is associated with increased analgesic sensitivity to subsequent morphine administration. As a result, analgesic requirement should be continuously evaluated [7]. Alternative medications such as acetaminophen, tramadol, ketorolac, ketamine and dexmedetomidine should be used when appropriate to reduce narcotic use [8]. Due to the dissection both in the neck area and proximal to the pleural area, these patients should be watched for the neck hematoma and development of pneumothorax in the PACU. It is our routine practice to obtain a chest X-ray in these patients in the PACU.

Discussion

Obstructive sleep apnea is a widespread disorder with increasing prevalence worldwide. If untreated OSA results in significant morbidity treatment pathways can be non-surgical therapies and surgical therapies. Unilateral therapeutic electrical stimulation of the hypoglossal nerve is feasible and a potential therapeutic option for OSA. This therapy has been approved by the U.S. Food and Drug Administration since April 2014. This procedure has been shown to decrease the severity and symptoms of OSA in select patients [9]. Patients considered for hypoglossal nerve stimulator are those who have demonstrable CPAP noncompliance and/or intolerance, moderate to severe OSA, BMI<35kg/m2, no severe co morbidities and drug induced sedated endoscopy indicating absence of complete concentric collapse at the soft palate. This procedure can present various perioperative challenges and it is imperative to have clear goals and a plan for perioperative management of these patients.

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