Alternative devices for obstructive sleep apnea

Daniel A. Barone, MD

Summary

Obstructive sleep apnea (OSA) is a relatively common condition which is most often treated with continuous positive airway pressure (CPAP). Given the compliance issues associated with CPAP, it is important for neurologists (and anyone who treats OSA) to be aware of what other treatment devices exist for this disease. This article reviews mandibular advancement devices, nasal expiratory airway pressure devices, hypoglossal nerve stimulators, and oral pressure therapy devices in terms of their mechanism of action, efficacy, and practicality.

As neurologists, it is important to be familiar with the diagnosis and treatment of obstructive sleep apnea (OSA); untreated, it can increase the risk of developing cardiovascular, cerebrovascular, metabolic, and gastroesophageal diseases. The modality most often prescribed for OSA is continuous positive airway pressure (CPAP). CPAP works by using air pressure to splint the airway open, thus preventing the repeated episodes of upper airway collapse which occur in OSA. Despite being effective, its weakness lies in the realm of patient compliance. In as many as 43%–86% of cases,¹ patients cannot tolerate CPAP, for a variety of reasons including feelings of claustrophobia and overall discomfort. Fortunately, new modalities have recently been developed, and it is instructive for us to examine the newer and perhaps lesser-known devices and treatments available for OSA.

Mandibular advancement devices

Mandibular advancement devices (MAD), although having been around since the 1900s, have only recently been thought of as a potential treatment for OSA; in 2006, the American Academy of Sleep Medicine (AASM) added them to CPAP as acceptable treatment for mild to moderate OSA, and for severe OSA in cases when CPAP has failed (i.e., from patient noncompliance).² These devices protrude the mandible anteriorly, which then has the effect of increasing the size of the upper airway. The tongue is anchored to the mandible, and this protrusion moves the tongue base away from the back wall of the oropharynx (which is a common site of collapse in those with OSA). The authors of one review article reported that subjective daytime sleepiness (as measured by the Epworth Sleepiness Scale [ESS]) was

Weill Cornell Center for Sleep Medicine, New York, NY.

Correspondence to: dab9129@med.cornell.edu

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decreased by MAD as compared to placebo devices.³ In another review article, it was found that MAD resulted in successful treatment in 19%–75% of patients with OSA when an apnea-hypopnea index (AHI) of <5 was the target, and in 30%–94% of patients with OSA when an AHI of <10 was considered.⁴ Typical side effects of MAD are jaw discomfort, tooth tenderness, excessive salivation, or temporary bite changes, and, as per a recent review article, are reported in slightly more than half of the patients.³ CPAP and MAD had a similar effect on AHI, but the authors found that CPAP may produce a better outcome than MAD with regards to complaints of sleepiness, even in the milder cases.³ Despite these potential problems, MAD are a typically well-tolerated and effective alternative to CPAP. Depending on third-party reimbursement, cost to the patient can be up to \$3,000, but is typically much less (\$0–600).² The AASM has recommended that follow-up be conducted every 6 months for the first year of use with a MAD, then annually after that; the devices tend to deteriorate or become maladjusted, and regular follow-up can help address this.

Nasal expiratory positive pressure devices

Nasal expiratory positive pressure devices (nasal EPAP), which were first introduced in 2008, have recently garnered a fair amount of media attention. These devices are essentially adhesive patches, which are applied over each nostril before sleep. The patches contain small one-way valves, which provide little resistance to inspiration, but increased resistance during expiration.⁵ This has the effect of generating positive pressure in the nasopharynx, which then travels down to the oropharynx where it acts as pressure split in a manner similar to CPAP. The increased airway pressure, along with an increased expiratory time, either alone or together, makes the upper airway less vulnerable to collapse on subsequent inspiration.⁵ Several studies have been designed to evaluate the effectiveness of these devices, and they have shown remarkable results. In one study, median AHI was reduced from 15.7 (moderate) to 4.7 (normal), and ESS decreased from 11.1 ± 4.2 (excessive sleepiness) to 6.0 ± 3.2 (normal); 34 of 51 patients were still using the EPAP devices at the end of 12 months.⁶ In another study, 47 of 59 CPAP-intolerant participants were able to tolerate the devices at home. Twenty-four demonstrated efficacy on repeat polysomnography and were restudied 6 weeks later; the average AHI dropped from 31.9 ± 19.8 (severe) to 16.4 ± 12.2 (mild to moderate).⁵ Unfortunately, the clinical trial experience does not always translate to real-life experience, and, as can be seen in the discussion of said articles, the issues of short duration,⁵ many exclusion criteria, and self-reported adherence⁷ do not fully evaluate how patients will do in real life. In those who do respond favorably, however, nasal EPAP devices can be a great “backup,” especially in the case of travel (when patients are less likely to bring their CPAP unit, for example). The typical cost to the patient is around \$2 per night.

The future

The genioglossus muscle is the largest dilator of the upper airway; when activated, it causes the tongue to protrude and the anterior pharyngeal wall to stiffen.⁸ It has been considered a

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potential therapeutic target for OSA, and researchers have tried to demonstrate a therapeutic effect with a variety of submental, intraoral, and IM stimulating electrodes.⁸ In these studies, improvements in upper airway diameter, pharyngeal collapsibility, air flow, and, most importantly, AHI were reported.⁸ Unfortunately, the fact that such stimulation tends to induce arousal has precluded their use in the clinical setting.⁸ Next-generation respiratory-triggered implantable devices have recently been designed and have been engineered to provide intermittent electrical impulses to the hypoglossal nerve via an implanted cuff electrode.⁹ These devices monitor respiration, via implanted thoracic leads, by sensing changes in motion of the chest wall. Electrical stimulation to the hypoglossal nerve is then provided cyclically during inspiration (which represents the most vulnerable period with regard to upper airway narrowing and collapse). When stimulated, the hypoglossal nerve causes the genioglossus muscle to contract, which results in an anterior displacement of the base of the tongue and an enlargement of the upper airway. The hypoglossal branches that innervate the genioglossus contain mostly efferent fibers, with minimal afferent input; this allows for activation of the genioglossus with less possibility of arousal.⁸ In one study, there was a significant improvement from baseline to 6 months in AHI (43.1 ± 17.5 [severe] to 19.5 ± 16.7 [moderate]) and ESS (12.1 ± 4.7 [excessive sleepiness] to 8.1 ± 4.4 [borderline sleepiness]).⁸ Another recent study presented initial data suggesting that upper airway stimulation can be effective and safe in certain patients with moderate to severe OSA who are unable or unwilling to use CPAP.⁹ However, like all surgical treatments, this is subject to unpredictable results, potential for adverse events, and likely large expense; fortunately, it will be just one of several alternatives to CPAP available in the near future.

Another new modality, oral pressure therapy (OPT), was recently cleared by the Food and Drug Administration for patient use. OPT functions as an oral vacuum, which pulls the soft palate forward and stabilizes the tongue. As with the other treatments previously discussed, this increases the size of the airway and theoretically reduces apnea. The available literature consists of 2 studies: one concerned with mechanism of action¹⁰ and another demonstrating efficacy and safety.¹¹ The first study included 5 adults with mean AHI 38.8 ± 25.0 (severe) and, as a result of OPT, demonstrated an increase in multiple upper airway dimensions. Clinically, AHI was significantly reduced to 4.9 ± 3.0 (normal).¹⁰ The second study included 30 subjects with mild to severe OSA, and objective improvement was noted in 14 of them, with a reduction in AHI from 31.0 (severe) to 5.7 (mild). Two subjects withdrew secondary to oral tissue discomfort and irritation, but no serious or severe adverse events were reported.¹¹ Clearly the initial data are impressive, and OPT certainly seems safe, but as with all new modalities, real-world experience needs to be ascertained and more extensive clinical trials need to be performed. The manufacturer reports that this promising new device should be widely available this year.

This has not been an exhaustive list of all potential alternative treatments for OSA; for example, more conservative measures such as weight loss have not been discussed. Rather, it was meant to serve as a review of the newer and, in some cases, lesser known options available to treat OSA (currently or in the near future). As this condition becomes more recognized, both within the public domain and in the medical community itself, all physicians will need to become aware of consequences of OSA, as well as the ingenious treatments that have been developed to combat it.

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