# **Chapter 2 Treatment Options in Sleep Apnea**



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#### Contents

Background	14
Continuous Positive Airway Pressure (CPAP)	14
Surgery as Adjunct to CPAP	15
Tonsillectomy	16
Nasal Surgeries	17
Mandibular Advancement Devices (MADs)	18
Surgery as Alternative to CPAP	18
"Too Little Space"	19
Maxillo-Mandibular Advancement	19
Maxillary Expansion	20
"Too Much Tissue"	20
Simple Tonsillectomy	20
Lingual Tonsillectomy	21
Uvulopalatopharyngoplasty (UPPP)	21
Partial Glossectomy	
"Tissue Too Lax"	
Tongue Suspension	24
Hyoid Myotomy and Suspension	24
Radiofrequency Ablation (RFA)	
Hypoglossal Nerve Stimulation.	25
Tracheostomy	26
Summary	26
References	26

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## Background

Obstructive sleep apnea (OSA) is a disorder characterized by repeated episodes of upper airway collapse, leading to nocturnal hypoxemia and associated arousals [1, 2]. The prevalence of moderate to severe sleep apnea, defined as AHI  $\geq$ 15 and  $\geq$ 30 respectively, continues to rise in the United States with an estimated 10–17% of men and 3–9% of women having the disorder [3]. This translates to approximately 13 million adults over age 30 in the United States alone [4]. The prevalence of this disorder is significantly higher in certain populations with nearly 50% of the elderly, 55% of those with coronary artery disease, and 37% of those with type I or II diabetes meeting criteria for diagnosis [5–7].

Untreated or under-treated OSA poses a serious public health risk with numerous co-morbidities and health consequences. Most notably, OSA patients are at increased risk for cardiovascular disease and hypertension secondary to chronic nocturnal spikes in heart rate and blood pressure due to arousal and catecholamine release [1]. OSA has also been associated with increased sympathetic burden, metabolic disorders, cerebrovascular disease, insulin resistance, cancer, perioperative complications, and early all-cause mortality [1, 8, 9]. In addition to these, untreated OSA has been associated with reduction of cognition, daytime somnolence, a high prevalence of depression, and increased motor vehicle collision rates [1, 2, 5, 10].

### **Continuous Positive Airway Pressure (CPAP)**

First described in 1981, CPAP continues to be the gold standard for treatment of moderate to severe sleep apnea and is the most efficacious at improving all parameters of sleep apnea severity, including AHI, ODI and O2 nadir, when used as prescribed [11, 12]. Nasal CPAP functions as a pneumatic splint in the upper airway, offsetting the negative pressure created during inspiration and preventing airway collapse [13]. CPAP effectively improves awake performance, quality of life measures, snoring, driving performance, and neurocognitive function [13, 14]. It also reverses many of the health sequelae of OSA by significantly reducing blood pressure, resolving pathological cardiac dysrhythmias, reducing sympathetic activity, improving insulin sensitivity, and improving long-term morbidity and mortality [13].

Despite the proven efficacy of CPAP in treatment of OSA, compliance is remarkably low with up to 50% of patients failing to meet the recommended 4+ hours of usage per night and many not even filling the prescription [15–17]. Few OSA patients wear CPAP for the 6 hours or more a night required to minimize the AHI, therefore CPAP functions to reduce but not eliminate elevated AHI in most users. There are many factors playing a role in patient non-adherence, including



**Fig. 2.1** A patient requiring a soft neck collar to prevent jaw opening and leak of CPAP

lack of education, high pressures, nasal obstruction, facial discomfort, social concerns, and claustrophobia [10, 16]. Some patients cannot maintain an effective seal with CPAP without accessory devices (Fig. 2.1). Of those that do comply with CPAP, nearly a third report nasal discomfort and dry mouth [18]. Intolerance for or inadequate therapy with CPAP lead many patients to seek alternative therapies for OSA.

## Surgery as Adjunct to CPAP

While CPAP remains gold standard therapy, occasionally the source of noncompliance or intolerance for a patient may be amenable to surgical correction. Often these surgical treatments allow for better airflow, reduce mouth opening, or reduced pressures, improving efficacy and lessening the discomfort of CPAP therapy, and encouraging compliance in previously intolerant patients [19].

#### **Tonsillectomy**

In patients with grade III or IV tonsils and low BMI, simple tonsillectomy has strong curative potential [20, 21]. In those who still require CPAP post-operatively, the majority require lower pressures than prescribed pre-operatively [20]. Tonsillar hypertrophy creates preferential mouth breathing during sleep, increasing mouth leak with CPAP, and contributes to CPAP intolerance [20]. Up to half of the patients intolerant of CPAP pre-tonsillectomy are able to utilize the therapy post-operatively, likely secondary to the reduced pressures and removal of an "obstructive feeling" in the pharynx [20]. Due to these findings, primary tonsillectomy is a rational approach to OSA patients with 3 or 4+ tonsils (Fig. 2.2). Repeat home sleep apnea testing can be performed after a period of healing in order to determine if additional PAP therapy is still required. Aside from pain perioperatively, the most common complication associated with tonsillectomy is post-operative bleeding which occurs in approximately 1–2% of cases, however the procedure is generally considered low risk [19, 20].

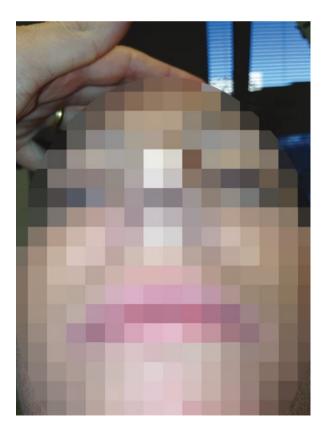
**Fig. 2.2** Three-plus tonsillar hypertrophy which can be removed as first-line therapy for OSA



## Nasal Surgeries

Obstructive nasal symptoms, secondary to inferior turbinate hypertrophy, internal nasal valve collapse, or deviated septum, are commonly reported by CPAP users and likely contribute to non-compliance [16, 22]. Nasal obstruction worsens during sleep due to supine position, which causes dependent nasal vasocongestion, and is exacerbated by CPAP usage [16, 22]. Nasal surgery is not recommended alone in the treatment of OSA due to insufficient reduction in AHI, but can be used in select patients to improve CPAP adherence by removing nasal obstruction and reducing pressure settings while improving sleep quality, and reducing nocturnal arousals and daytime sleepiness [10, 21, 23]. Even in cases where CPAP is not significantly reduced, the relief of symptoms associated with nasal obstruction is enough to allow patients to use therapy comfortably [10]. A thorough examination of the nasal passages is required for any patient prior to CPAP therapy. If there is a history of nasal congestion, or anatomic evidence of fixed (e.g., deviated septum) or dynamic (e.g., nasal valve collapse; inferior turbinate hypertrophy) blockage, the patient will benefit from application of a nightly nasal steroid spray and/or nasal breathing strips prior to sleep. Dynamic nasal valve collapse due to weakening of the upper lateral cartilages can be an overlooked source of nasal blockage if a modified Cottle

Fig. 2.3 Narrow internal nasal valves that demonstrated collapse on modified Cottle maneuver



maneuver is not performed (Fig. 2.3). If these conservative measures fail to improve nasal breathing, surgical correction prior to CPAP initiation is advised since patients are more likely to be adherent to CPAP therapy long-term if obstacles to CPAP are managed prior to therapy initiation. However, positive outcomes following nasal procedures for patients already using CPAP are highly consistent, and therefore corrective nasal surgery should be offered to any patient with obstructive nasal anatomy who is struggling with CPAP adherence [10].

#### Mandibular Advancement Devices (MADs)

Mandibular advancement devices (MADs) are an appropriate alternative therapy for select patients unable to tolerate CPAP [16]. They function to reposition the tongue and/or lower jaw, increasing the retroglossal airway space [1]. The ideal candidate for MAD therapy is one with lower BMI and mild to moderate OSA (AHI  $\leq$  30) predominantly associated with supine position [16]. In general, 50% of maximal anterior jaw distraction is needed to manage mild to moderate OSA, whereas 75% of maximal distraction may be required to treat more severe OSA. MADs are not as efficacious at treating OSA as CPAP, but are more comfortable for patients and allow greater adherence to therapy [1, 16]. Greater comfort, adherence, device longevity, and fewer side-effects are present with custom-fitted MADs compared to off-the-shelf boil and bite appliances. Contraindications to MAD therapy include missing teeth, poor dentition, and TMJ dysfunction [16]. The most commonly reported side effects include TMJ discomfort, tooth pain, sialorrhea, or xerostomia [16]. Another important note is that many insurance companies do not cover the cost of MADs and the expense of the device, especially custom-fitted MADS fabricated by a dental sleep specialist, may be prohibitive for some patients [1].

#### Surgery as Alternative to CPAP

If CPAP or other first-line therapies are intolerable or fail to treat OSA, upper airway surgery can be an option for select candidates. OSA is a disease that can be caused by airway collapse at multiple locations in the upper airway, with a majority of patients having obstructions at more than one level [24]. Surgical treatment can be primary therapy in patients who have severe anatomic obstruction (tonsillar, turbinate, or uvular hypertrophy) that is best corrected surgically [16]. Surgical treatment of airway collapse for OSA is not a "one-size-fits-all" method and thus requires individualized assessment and surgical planning for each patient.

In examining the patient who has failed first-line therapies, it is important to note external anatomy and body habitus that may be effecting collapse in a patient [16]. Parameters such as severity of OSA, BMI, neck size, craniofacial structure, nasal patency, tongue position, palatal anatomy, and tonsil size are important in

determining which surgical options would be most effective for the patient [16, 20, 25]. Polysomnograms are employed in the diagnosis of OSA and in determining the severity of disease pre- and post-operatively [16]. These can be performed in a sleep center or at home.

Next, it is essential to identify the extent of collapse at the different levels of the airway. There is no current gold-standard for determining level of obstruction. The "Nose-VOTE" scoring system is commonly used and identifies collapse or obstruction at the level of the nose, velopharynx, oropharynx, tongue, and epiglottis [26]. This system allows prioritization of specific levels of the airway amenable to surgical correction and can be employed in both awake endoscopy and drug-induced sleep endoscopy (DISE) [25]. Awake endoscopy has historically been the tool utilized to determine levels of airway collapse. In most patients with OSA, the upper airway behaves differently in awake and sleep states [25]. DISE under light propofol sedation provides another layer of information by allowing better visualization of the airway collapse patterns during sleep [16, 25]. DISE provides the sleep surgeon with additional information such as site, severity, and pattern of collapse which can be combined with the findings of sleep apnea testing to formulate a rational treatment strategy. After thorough evaluation of the patient, there are three major categories of airway collapse underlying the disease state: (1) "too little space" or a craniofacial problem, (2) "too much tissue" or a hypertrophy/obesity problem, and (3) "tissue too lax" or increased tissue collapsibility.

## "Too Little Space"

Oropharyngeal crowding and collapse can be due to compression of the collapsible soft tissues of the upper airway into a reduced craniofacial or jaw structure. These patients are often younger in age, and non-obese. Physical examination may reveal retrognathia; high-arch maxilla; narrow mandibular arch; open or cross-bite; and Class II or III malocclusion. The tongue may appear to fill the oropharynx (Modified Mallampati III or IV), however the tongue is of normal size within a small jaw structure (relative macroglossia). There are several alternative therapies to CPAP for patients with the above findings.

## Maxillo-Mandibular Advancement

Maxillo-mandibular advancement (MMA) is a skeletal surgery designed to enlarge the airway at the velopharynx and oropharynx [27]. MMA functions by advancing the anterior pharyngeal tissues attached to the hyoid, mandible, and maxilla [1]. It is the ideal surgical treatment for patients with significant retrognathia, and/or malocclusion [16]. In these patients, MMA is nearly curative and has been shown to reduce AHI by 87% while improving facial profile and jaw occlusion [16, 27]. Despite its efficacy, MMA is rarely performed due to the length of procedure, the level of technical skill required, and the innate risks of dental malocclusion and nerve injury [16, 27]. The operation requires a substantially longer recovery time than other surgical treatments of OSA, sometimes requires a peri-operative trache-otomy, and is often not well covered by insurance [1, 16].

## **Maxillary Expansion**

Maxillary expansion is utilized to treat patients with OSA and associated transverse maxillary deficiencies. Transverse maxillary deficiencies, secondary to asymmetric development of the mandible and maxilla, manifest as dental malocclusion, narrow and high palatal vault, and elevated nasal floor [28, 29]. These abnormalities increase nasal airflow resistance and are associated with increased risk of nasal obstruction, especially in patients with septal deviation or hypertrophic inferior turbinates [29]. Maxillary expansion alleviates nasal obstruction by increasing the size of the nasal cavity and may reduce pharyngeal obstruction by increasing space in the oral cavity allowing better positioning of the tongue [29]. Recent meta-analysis found that maxillary expansion reduces AHI by more than half and significantly reduces sleepiness [29].

#### "Too Much Tissue"

A common cause of moderate to severe OSA is too much tissue in the oropharyngeal airway due to adenotonsillar hypertrophy or obesity with increased tongue and pharyngeal fat deposits. In general, these patients have onset of OSA in adolescence and early adulthood which is accompanied by increasing weight gain leading to obesity (BMI  $\geq$ kg/m<sup>2</sup>). On examination, these patients may have hypertrophy of the adenoid, palatine or lingual tonsil, and tongue enlargement (Modified Mallampati III or IV) within a normal jaw structure (acquired macroglossia). Often, impressions of the teeth can be observed scalloping the edges of the enlarged tongue. On DISE, these patients have a base of tongue that fills the oropharyngeal airway with folding of the tongue as evidence by a midline raphe.

## Simple Tonsillectomy

Hypertrophic tonsils can play a major role in the development of OSA, as mentioned previously, and simple removal of the tissue can be curative in a select group of patients [20]. Patients with hypertrophic tonsils should be evaluated for collapse at other levels, potentially necessitating multi-level treatment. The ideal candidate for simple tonsillectomy is one with large, grade III or IV, hypertrophic tonsils and low BMI (<25) [20]. In this patient population, tonsillectomy alone is an effective treatment for OSA and has been shown to significantly improve AHI, nocturnal oxygen saturations, and sleepiness [20].

#### Lingual Tonsillectomy

The base of tongue is a common location of airway obstruction, seen in up to 70% of patients with OSA [30]. Lingual tonsillar hypertrophy (LTH) is one source of this obstruction and may be easily missed on physical examination unless accompanied by fiberoptic laryngoscopy [31]. In addition to airway obstruction, it can cause dysphagia, otalgia, cough, and globus sensation [31]. Underlying causes of LTH are unknown, but it has been associated with obesity, GERD, smoking, and previous palatine tonsillectomy [32]. There are numerous reported approaches to lingual tonsillectomy such as with diode laser, coblation, microdebridement, harmonic scalpel, and more recently using trans-oral robotic surgery (TORS) [31–33]. Historically this procedure was rarely performed due to difficulties in surgical access, risk of massive intra-operative bleeding, and risk of post-operative airway compromise secondary to laryngeal edema [31, 33]. Surgical approach with TORS allows better visualization of the anatomy and increases the surgeon's ability to move freely, which may allow better control of intra-operative complications and reduce procedure-related morbidity [31, 32]. Regardless of technique used, lingual tonsillectomy can be curative in patients with sole obstruction at this level if the tonsils are entirely removed [31, 33].

#### Uvulopalatopharyngoplasty (UPPP)

UPPP, first described by Fujita in 1981, is one of the earliest surgical treatments described for OSA [34]. The procedure involves excision of redundant soft tissue at the level of the tonsils, posterior soft palate, and uvula with closure of the tonsillar

**Fig. 2.4** Uvulopalatopharyngoplasty (UPPP) demonstrating currently favored technique of maintaining uvular mass in the midline of the soft palate



pillars to widen the oropharyngeal airway [16, 27] (Fig. 2.4). It is one of the most commonly performed sleep surgeries, alone or combined with a multi-level approach, in the treatment of OSA and has been shown to improve mortality [35]. Post-operative polysomnography results are variable with reported AHI reductions from 33–52% with generally improved success rates when combined in a multi-level approach [16, 21]. Regardless, UPPP has been shown to significantly improve daytime sleepiness, driving performance, depression, sexual function, ventricular function, and serum lipids [5, 36]. There is extensive reporting of temporary and permanent complications associated with UPPP including difficulty swallowing, voice changes, oral pain, taste disturbance, globus sensation, and velopharyngeal insufficiency [34]. The most common complaint is foreign body sensation which may be reduced with newer uvular sparing techniques [34]. Because of these adverse effects, numerous modifications to surgical technique have been made over the years to try to reduce morbidity [16].

## Partial Glossectomy

Retroglossal collapse is present in up to 75% of patients with OSA and is most prominent in those with severe disease (AHI >30) and obesity [16, 24]. The physical exam of a patient with macroglossia can show lateral indentations from dentition



Fig. 2.5 Acquired macroglossia from Beckwith-Wiedemann Syndrome casing OSA before (a) and after partial glossectomy (b)



**Fig. 2.6** Middle third of base of tongue removed with transoral robotic surgery (TORS)

and often have Friedman tongue position of III or IV [16]. The acquired macroglossia is particularly pronounced in patients with certain metabolic disorders such as Beckwith-Wiedemann syndrome; acromegaly; or amyloidosis (Fig. 2.5). In patients with moderate OSA (AHI 15–30), the SMILE (submucosal minimally-invasive lingual excision) technique can be used. This procedure involves the use of a coblator and is generally less morbid than formal midline glossectomy [16]. In patients with severe OSA (AHI >30), a formal posterior midline glossectomy is more appropriate [16]. This procedure was historically very difficult due to challenges with surgical line-of-sight, but advances in TORS have allowed the tongue base to be much more accessible [16] (Fig. 2.6). Glossectomy, alone or in multi-level approach, is associated with significant improvement in AHI, nocturnal oxygen levels, daytime sleepiness, and snoring [24, 30]. The most common complication reported with glossectomy is loss of or change in taste, which resolves in most patients over several weeks [24].

#### "Tissue Too Lax"

In certain cases, OSA may result from increased levels of passive airway collapsibility and reduced neuromuscular tone during sleep. In general, OSA of this variety begins in middle-age and after, and is associated with lower BMI and normal tongue size (Modified Mallampati I or II). In these patients, most obstructive events will be recorded during supine sleep or REM sleep due to tongue prolapse into the oropharynx [16]. It is difficult to identify this type of collapse on awake endoscopy due to its dynamic nature, making DISE a much more sensitive diagnostic tool for this group of patients [16].

#### **Tongue Suspension**

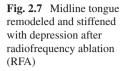
Functional collapse of the base of tongue during sleep is attributable to reduced neuromuscular tone [16]. Tongue base suture suspension utilizes a suture loop secured to a titanium screw on the lingual aspect of the mandible, effectively advancing the tongue and reducing collapse [16]. This procedure is ideal for non-obese patients without macroglossia and lower levels of OSA (AHI  $\leq$ 30) and can be an effective adjunct when combined with another procedure such as UPPP [16, 21]. It has been shown to significantly reduce AHI, sleepiness, and snoring and improve nocturnal oxygenation [16, 21, 37]. There is some evidence that this type of procedure does not provide long-term results due to tongue mobility and eventual stretching of the suture material [16, 37].

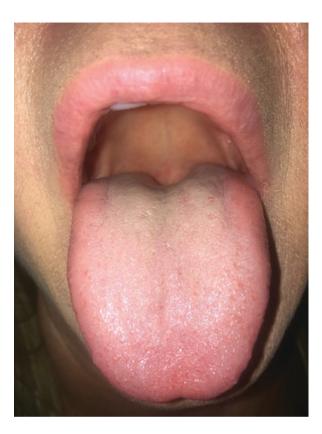
#### Hyoid Myotomy and Suspension

Most hypopharyngeal obstructions occur at the base of the tongue, but up to 20% of patients will have significant retro-epiglottic collapse [16]. Hyoid myotomy and suspension effectively advances the supraglottic structures away from the posterior pharyngeal wall, increasing the size of the airway and preventing collapse [16] (Fig. 2.4). Advancement of the hyoid bone in the anterior-superior direction provides tension on the hyoepiglottic ligament in the central hypopharynx and on the stylohoid muscles of the lateral hypopharynx. It is most effective in patients who have significant epiglottic retroflexion on awake or sleep endoscopy [16]. Hyoid suspension is commonly performed with another procedure and significantly improves AHI and success rates compared to the other procedure alone [16, 21, 27]. Even when performed alone, hyoid suspension significantly improves severity of OSA [38]. Complications are very rare, but tend to involve lingual edema or neck seromas [16, 38].

## Radiofrequency Ablation (RFA)

RFA is a form of electrical energy that can penetrate deep into tissues with a high level of precision and control [39]. The applied energy causes coagulation necrosis and inflammation in the tissue leading to fibrotic stiffening of the area and tissue reduction [16, 39]. The stiffening effect counteracts the natural collapsibility of the mucosalized, tubular airway. One advantage of RFA is that it can be used at several different levels of the upper airway including the nasal turbinates, soft palate, and base of tongue [39] (Fig. 2.7). Treatment of the palate has a 60% success rate and of





the tongue with up to 83% success [21]. Patients often require multiple treatments to achieve effective results [39]. RFA can be used alone, but is frequently combined with other procedures for greater effect [16, 21, 39]. RFA is associated with minimal risk and is a low cost option for patients [16]. The effect of the procedure is expected to lessen over time due to tissue remodeling of the scar tissue, however the procedure can be repeated if needed due to its low morbidity and pain.

## Hypoglossal Nerve Stimulation

Nerve stimulation therapy involves the implantation of a device that induces protrusion of the genioglossus muscle during sleep in accordance with breathing patterns [1, 21]. In a highly select patient population, the therapy significantly reduces AHI, oxygen desaturation index, and systolic blood pressure [2]. This procedure is discussed in detail elsewhere within this text.

## Tracheostomy

Surgical tracheostomy was the earliest treatment of OSA and is highly effective reducing AHI, sleepiness, and mortality [1]. Tracheostomy has high morbidity and is associated with significant changes in lifestyle [21, 40]. Despite its efficacy in treating OSA, due to the significant impact on quality of life, it is reserved as a last line of therapy only for those who have failed all other treatments, have emergent loss of airway, or have severe cardiopulmonary disease such as chronic congestive heart disease [1, 16, 40].

## Summary

Obstructive sleep apnea is a multifactorial disorder that requires an individualized treatment plan. CPAP is first line therapy in most cases of moderate-to-severe OSA and currently represents the gold standard of treatment, but is often not tolerated by patients and has a high rate of non-adherence. In these patients, evaluation by a sleep surgeon is an appropriate next step to find effective treatment options as untreated OSA has significant health consequences. Full workup should include history, physical exam, polysomnography, awake endoscopy, and occasionally drug-induced sleep endoscopy (DISE) to effectively identify underlying causes and levels of obstruction or collapse. Therapy should be directed towards the specific levels of obstruction in the patient and may include one or multiple operations.

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- 2 Treatment Options in Sleep Apnea
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