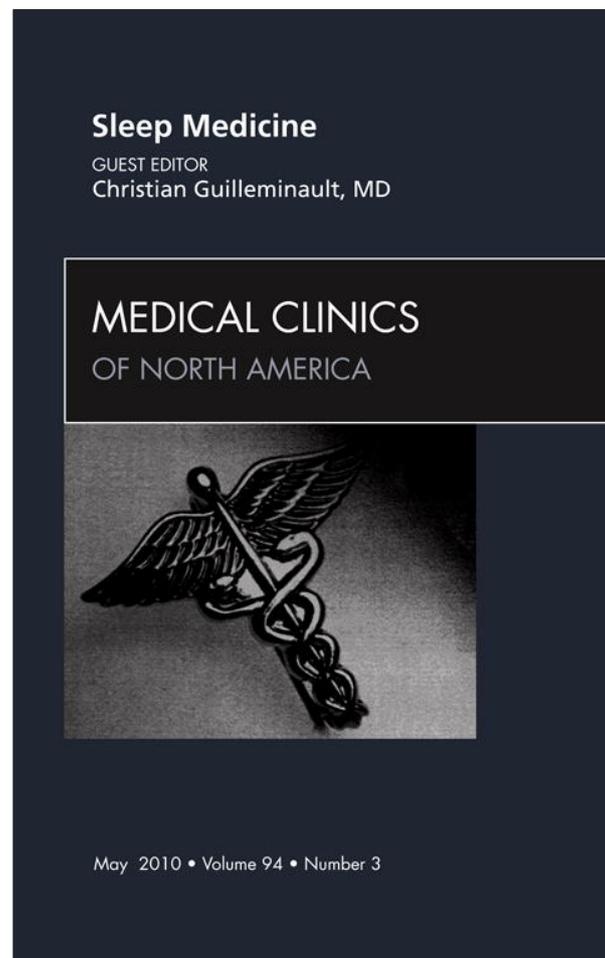


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Surgical Options for the Treatment of Obstructive Sleep Apnea

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KEYWORDS

• Obstructive sleep apnea • Sleep apnea syndromes • Surgery

Obstructive sleep apnea (OSA) is a highly prevalent condition characterized by increased nocturnal airflow resistance resulting in repetitive episodes of pharyngeal collapse during sleep.¹ Approximately 20% of adults in the United States have OSA (defined as an apnea-hypopnea index (AHI) $\geq 5/h$) with up to 10% having moderate to severe disease (AHI $\geq 15/h$).^{2,3} In addition, between 3% and 10% of children have OSA (AHI $\geq 1/h$).⁴⁻⁷ Obesity, male gender, advancing age, and mandibular-maxillary insufficiency are well-characterized risk factors.⁸ OSA predisposes to increased cardiovascular and cerebrovascular morbidity and mortality, and is associated with excessive daytime sleepiness and neurocognitive underperformance.⁸ Untreated, the 15-year mortality for adults with severe disease is approximately 30% with adjusted mortality hazards ratios of 1.4, 1.7, and 3.8 for mild, moderate, and severe disease, respectively (P -trend = 0.004).²

Conventional nonsurgical OSA therapy necessitates indefinite positive airway pressure (eg, continuous positive airway pressure [CPAP] or bilevel therapy) that works by pneumatically stenting open the upper airway, thus preventing apneas and hypopneas during sleep.⁹⁻¹¹ CPAP is an effective treatment modality for OSA, improving symptoms (eg, excessive daytime sleepiness, quality of life) and reducing cardiovascular mortality.^{12,13} Unfortunately, more than 50% of patients with OSA are intolerant of and ultimately reject CPAP therapy.^{14,15} Common complaints include mask discomfort and leak, rhinorrhea, conjunctivitis, dry mouth, nasal congestion, aerophagia, claustrophobia, and chest wall discomfort.⁶ Individuals intolerant of CPAP therapy have a 10% absolute increased mortality risk (compared with adherent subjects) at 5 years.^{16,17}

Contract/Grant Support: None.

Conflict of interest: None.

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Med Clin N Am 94 (2010) 479–515

doi:10.1016/j.mcna.2010.02.001

0025-7125/10/\$ – see front matter. Published by Elsevier Inc.

medical.theclinics.com

Effective surgical therapies for OSA predate the first reported use of CPAP by Sullivan and colleagues⁹ in 1981 and Rapoport and colleagues¹⁸ in 1982. Tracheostomy was employed as early as 1969¹⁹ and Kuo and colleagues²⁰ in 1979 (and later Bear and Priest²¹ in 1980) reported the results of mandibular advancement for the treatment of OSA. In 1952, Ikematsu²² began removing excessive oropharyngeal tissue to alleviate snoring and reported the results of his palatopharyngoplasty with partial uvullectomy in 152 habitual snorers in 1962. In the late 1970s, Fujita and colleagues²³ adapted Ikematsu's procedure and introduced the uvulopalatopharyngoplasty as a new surgical approach to treat OSA.²⁴ Because the anatomic cause of OSA is heterogeneous with most OSA patients having multiple concurrent pharyngeal abnormalities,^{25–27} surgical procedures have evolved to address specific anatomic airflow limitations and to augment the effectiveness of existing procedures. This review describes the pathophysiology of OSA, the rationale for surgery, and the various surgical techniques used to treat OSA.

PATHOPHYSIOLOGY OF OSA

Patients with OSA have nocturnal airflow restriction resulting from upper-airway collapse between the naso- and hypopharynx.²⁸ During normal breathing, contraction of the diaphragm results in an increased thoracic volume that generates negative intrapleural pressure drawing air down to the alveoli. During a normal negative pressure inspiration, upper-airway reflexes phasically activate pharyngeal muscles (eg, genioglossus, tensor palatini, geniohyoid, stylohyoid) to dilate and stiffen the upper airway to maintain patency.^{29–32} Pharyngeal dilator muscle activity is reduced in normal and OSA individuals during sleep.^{30,33} However, patients with OSA have anatomically smaller upper airways and diminished pharyngeal dilator tone resulting in clinically significant airflow limitation (eg, apneas and hypopneas) during nocturnal negative pressure inspiration.^{28,34,35} Most individuals with OSA have multiple pharyngeal abnormalities²⁵ with anatomic airway narrowing primarily in the lateral dimension.^{36,37}

In addition, patients with OSA are often obligatory mouth breathers during sleep.³⁸ Nasal breathing (compared with mouth breathing) is more efficient because the nasal cavity has a more constant resistance (compared with the oral cavity) and because stimulation of nasal receptors is involved in activating the pharyngeal dilators.³⁹ In normal individuals, a transition from nasal to oral breathing results in a greater risk of pharyngeal collapse because of greater negative inspiratory pressures needed to overcome increased airway resistance.³⁹ Experimental nasal obstruction^{40–42} or inhibiting the nasopharyngeal reflex (by applying topical anesthesia)⁴³ causes nocturnal apneas, hypopneas, and oxygen desaturation in normal individuals.

OSA is in part a neurologic disorder of the upper airway.^{30,33,44,45} Pharyngeal collapse is often caused by abnormal activation of pharyngeal dilator muscles from dysfunctional pharyngeal reflexes.⁴⁶ In patients with nocturnal upper-airway resistance, repetitive vibratory trauma (eg, snoring) and tremendous swings in pharyngeal pressures (caused by apneas and hypopneas) during sleep results in pathologic injury to the pharyngeal dilator muscles and nerves.^{33,47,48} This irreversible damage predisposes the upper airway to inspiratory collapse during sleep.^{45,49–51}

RATIONAL FOR OSA SURGERY

The aim of OSA surgery is to eliminate airway collapse and reduce airway resistance during sleep without causing impairment to the normal functions of the upper airway and associated structures. Indications for surgery depend on: (1) the severity of OSA

and comorbid medical conditions; (2) the severity of symptoms (eg, excessive daytime sleepiness); and (3) the anatomic location(s) causing obstruction. General indications for surgery include moderate-severe OSA, severe excessive daytime sleepiness (even when the AHI is $\leq 20/h$), OSA with comorbid conditions (eg, arrhythmias, hypertension), OSA with anatomic airway abnormalities, and failure of medical OSA management.⁵² Upper-airway abnormalities amenable to surgery include those within the nasal cavity (eg, deviated septum, polyps, hypertrophic turbinates, collapsible nasal valves), nasopharynx (eg, stenosis, adenoids), oropharynx (eg, palatine tonsils, elongated uvula, redundant mucosal folds, low hanging palate, webbing), and hypopharynx (eg, lingual tonsils, large tongue base, redundant aryepiglottic folds) (**Table 1**). Relative contraindications to surgery include morbid obesity (except for bariatric surgery and tracheostomy), severe or unstable cardiopulmonary disease, active alcohol/illicit drug abuse, older age, unstable psychological problems, or unrealistic expectations from surgical therapy.

All adult OSA patients should be offered a nonsurgical treatment option (eg, CPAP) before proceeding to surgery. Even in patients electing to proceed directly to surgery, a trial of CPAP therapy may be helpful as this is a noninvasive means to determine the expected extent of symptom abatement after surgery. Preoperative CPAP is indicated in patients with severe OSA (AHI $>40/h$ with severe nocturnal oxygen desaturation $<80\%$) and should be continued postoperatively until 2 weeks before the postoperative polysomnogram.⁵³

In children, early recognition of OSA and prompt correction of anatomic upper-airway abnormalities is paramount. By the age of 4 years, 60% of the adult craniofacial skeleton is attained, with 90% by age 12 years.^{54–56} Children with pharyngeal obstruction (eg, tonsillar hypertrophy, turbinate enlargement) protect the patency of the airway by sleeping in the prone or side position with an extended, flexed head, and an anteriorly displaced tongue.⁵⁷ Anterior displacement of the tongue is associated with narrower upper and shorter lower dental arches,^{57–61} posterior displacement of the mandible,^{60,62} with resultant development of mandibular retrusion, increased overjet, and facial height^{63–68} (all known risk factors for OSA).^{58,69} Thus, early recognition

Table 1**Anatomic location of pharyngeal obstruction in relationship to surgical procedure**

Anatomic Treatment Effect	Anatomic Obstruction	Surgical Procedure
1. Bypass upper airway	Collapsed airway	Tracheostomy
2. Soft tissue removal	Nasal cavity	Polypectomy, radiofrequency ablation of the turbinates
	Nasopharynx	Adenoidectomy
	Oropharynx	Tonsillectomy Uvulopalatopharyngoplasty Laser-assisted uvulopalatoplasty
	Hypopharynx	Midline glossectomy Tongue base reduction
3. Skeletal/soft tissue modification	Nasal cavity	Septoplasty
	Oropharynx	Rapid maxillary expansion
	Hypopharynx	Mandibular advancement Genioglossal advancement Hyoid myotomy suspension
	Oro- and hypopharynx	Maxillomandibular advancement

and surgical correction of significant airway obstruction in children is necessary to prevent abnormal craniofacial development and the irreversible damage to the pharyngeal dilator reflexes that potentially can lead to the lifelong consequences of OSA.

In adults and children, preoperative upper airway assessment is necessary to determine the level of obstruction (anywhere between the nose and epiglottis) and plan the optimal surgical approach. Preoperative fiberoptic endoscopy (performed with a Müller maneuver) and cephalometric analysis are helpful to classify the type of airway obstruction and identify obstructions of the hypopharynx.^{26,70} Computed tomography may have added benefit.⁷¹ The anatomy of the upper airway is classified into 3 general obstructive types (Fujita classification): (1) type 1: narrow oropharynx (eg, large tonsils, enlarged uvula, pillar webbing) with normal palatal arch position; (2) type 2: low arched palate with relatively large tongue; further subdivide into 2a (predominantly oropharyngeal abnormality) and 2b (abnormality involves oro- and hypopharynx); (3) type 3: hypopharyngeal obstruction (eg, retrognathia, floppy epiglottis, enlarged lingual tonsils) with normal oropharynx.^{72,73} The type of obstruction is often modified whether nasal obstruction concurrently exists. Surgical procedures address specific upper-airway abnormalities (eg, uvulopalatopharyngoplasty for type 1, genioglossus advancement for type 3, maxillomandibular advancement for combined type 1, 2 and 3). Preoperative pharyngeal anatomy, OSA severity, and patient preference (eg, recovery time, prolonged facial paresthesias, and malocclusion) are all contributing factors influencing the surgical decision.

SURGICAL SUCCESS

Various surgical procedures are now available to increase the posterior airspace and treat OSA in CPAP intolerant patients. However, no surgical treatment is 100% effective. Similar to previous reviews of OSA surgery,^{74,75} we defined surgical success as an AHI less than 20 and a reduction in AHI of 50% or more after surgery.⁷⁶ Where possible, we provide surgical cure rates (defined as an AHI <5/h in adults and <1/h in children).⁷⁷

PHARYNGEAL SURGICAL PROCEDURES

Individual surgical procedures are described in the following sections for the treatment of OSA organized by the treatment effect on the anatomic airway obstruction (eg, bypassing the upper airway obstruction, removal of soft tissue structures, or skeletal (or soft tissue) modification) (see **Table 1**).

Procedures that Bypass the Upper Airway Obstruction

Tracheostomy

In 1965, Valero and Alroy⁷⁸ reported improvement in nocturnal oxygenation in a patient with progressive respiratory failure secondary to traumatic micrognathia. Kuhlo and colleagues in 1969¹⁹ followed by Lugaresi and colleagues in 1970^{79,80} were the first to effectively treat OSA (or Pickwickian syndrome) by means of a tracheostomy. By bypassing the upper airway, tracheostomy is purported to be curative for OSA.⁸¹ Although many studies purport resolution of airway obstruction after tracheostomy,^{82–85} relatively few studies report pre- and posttracheostomy polysomnography parameters (eg, AHI) (**Table 2**).^{81,86–91}

The largest case series (n = 50) reported complete resolution of obstructive apneas after tracheostomy.⁸¹ Of 9 studies evaluating 61 patients, tracheostomy was highly effective at eliminating obstructive apneas (apnea index went from

Table 2
Efficacy of tracheostomy for OSA^a

Study	Demographics		Apnea Index ^b				AHI (REM) ^c				Apnea-Hypopnea Index (NREM) ^c				
	N	Age (y)	Pre-Trach	Post-Trach	P-value ^d	Follow-up (mo)	Pre-Trach	Post-Trach	P-value ^d	Pre-Trach	Post-Trach	P-value ^d	Pre-Trach	Post-Trach	P-value ^d
Haapaniemi et al, 2001 ⁸⁶	7	53.4 ± 9.8	60.9 ± 30.7	-	-	-	-	-	-	-	-	-	-	-	-
Kim et al, 1998 ⁸⁷	23	47.0 ± 12.4	-	-	-	-	58.5 ± 34.1	26.0 ± 31.2	-	92.5 ± 39.1	19.8 ± 26.3	-	-	-	-
Fletcher, 1989 ⁹⁵	1	-	51.0	114.0	0.0	-	-	-	-	-	-	-	-	-	-
Fletcher et al, 1987 ⁸⁸	8	55.4 ± 6.8	9.0 ± 3.2	84.6 ± 38.7	0.0 ± 0.0	-	-	-	-	-	-	-	-	-	-
Guilleminault et al, 1981 ⁸¹	4	-	30.0 ± 6.9	94.5 ± 19.8	0.4 ± 0.5	-	-	-	-	-	-	-	-	-	-
Sugita et al, 1980 ⁹⁰	1	40.0	3.0	77.0	0.0	-	-	-	-	-	-	-	-	-	-
Weitzman et al, 1980 ⁹⁶	10	47.5 ± 2.4	0.3 ± 0.4	96.1 ± 21.9	1.1 ± 3.3	-	79.0 ± 18.6	26.5 ± 25.2	-	113.7 ± 23.0	25.8 ± 25.4	-	-	-	-
Motta et al, 1978 ¹⁰¹	6	47.0 ± 4.0	7.5 ± 6.3	73.0 ± 12.2	0.0 ± 0.0	-	-	-	-	-	-	-	-	-	-
Weitzman et al, 1978 ¹⁰⁰	1	67.0	0.5	96.7	4.1	-	-	-	-	-	-	-	-	-	-
Summary	61	49.3 ± 9.9	19.0 ± 26.3	88.4 ± 25.7	0.5 ± 1.9	<0.001	63.8 ± 31.9	26.2 ± 29.2	<0.001	98.9 ± 36.0	21.6 ± 25.8	<0.001	21.6 ± 25.8	<0.001	<0.001

Abbreviations: N, number; NREM, nonrapid eye movement sleep; REM, rapid eye movement sleep; Trach, tracheostomy.

^a Mean (or percent) ± standard deviation. - denotes not reported.

^b The apnea index is the average number of obstructive apneas per hour during sleep.

^c The AHI is the average number of obstructive apneas and hypopneas per hour during sleep.

^d P-value calculated via an extended t-test and evaluates pre- and posttracheostomy measures.

88/h before to 0.5/h after tracheostomy; $P < .001$) (see **Table 2**). However, patients may have persistent hypopneas with a surgical success rate of approximately 73% (see **Tables 2** and **3**). Rodman and Martin⁹² reported persistent (although generally improved) obstructive apneas and oxygen desaturations in 3 morbidly obese patients after tracheostomy caused by kinking of the tracheostomy tube during sleep or external obstruction by the patient's own soft tissues. Haapaniemi and colleagues⁸⁶ reported that although obstructive apneas improved after tracheostomy (mean follow-up 5.1 years), most patients had persistent oxygen desaturations with many having oxygen dip indexes ($\geq 4\%$) of $\geq 15/h$. Fletcher and Brown⁹³ reported persistent REM-associated desaturations after tracheostomy in patients with OSA with concomitant chronic obstructive pulmonary disease. Despite improvements in obstructive apneas after tracheostomy, emergence or worsening of central apneas is frequently observed, although generally resolves within 3 to 6 months.^{80,81,89,94–98}

Tracheostomy is effective at preventing OSA-related arrhythmias,^{98–100} reducing pulmonary artery pressures,^{80,81,101} and improving hypertension^{81,88,100–103} and diabetes¹⁰² in patients with OSA. Many (but not all)⁸⁶ studies have reported near complete resolution of nocturnal symptoms and daytime sleepiness.^{80,81,84,90,91,96,100,101,103–106} A retrospective analysis by He and colleagues¹⁰⁷ suggested a mortality benefit of tracheostomy (0% vs 38% mortality at 8 years) compared with no OSA therapy. Partinen and colleagues^{108,109} found similar mortality benefits (0% vs 11% at 5 years) after tracheostomy.

Unfortunately, tracheostomy has several problems including patient dissatisfaction (eg, psychosocial aspects), perioperative complications (eg, wound infection, tissue necrosis, bleeding), recurrent bronchitis, granulation tissue, trachea-innominate fistula formation, and stoma stenosis (often requiring surgical revision).^{105,110–115} Perioperative mortality is higher in obese individuals than in nonobese individuals.¹¹⁶ Permanent tracheostomy (either tube¹¹¹ or tube-free¹¹⁷) is currently used in highly select cases with severe OSA who are intolerant of CPAP (and poor candidates for other surgical procedures). A temporary tracheostomy is occasionally used before other OSA procedures (eg, uvulopalatopharyngoplasty, bariatric surgery) to protect the airway, particularly in morbidly obese subjects.¹¹⁸

Closure of a permanent tracheostomy (after resolution of OSA by other surgeries or weight loss¹¹⁹) may be associated with a relatively high complication rate ($\sim 30\%$), especially when done with a 3-layer as opposed to a de-epithelialization technique.^{120,121} In addition, long-term tracheostomy may cause pharyngeal tissue obstruction (eg, granulation tissue, tracheomalacia) that may predispose to OSA after closure.^{122,123}

Procedures that Remove Soft Tissue

Laser-assisted uvulopalatoplasty (LAUP)

This office-based procedure (similar to uvulopalatopharyngoplasty, but omitting tonsillectomy) uses a CO₂ laser to shape the soft palate and is an effective surgical technique for snoring ($\sim 90\%$ success), but has limited OSA efficacy (see **Table 3**).¹²⁴ Two randomized trials of LAUP found no significant change in the AHI after surgery compared with those randomized to no surgery.^{125,126} A meta-analysis of these 2 studies found no statistically significant difference in daytime sleepiness (measured by the Epworth sleepiness scale) between surgery and control groups (mean difference -1.4 ; 95% confidence interval [CI] -5.0 – 2.2).¹²⁷ More worrisome is that LAUP may worsen OSA in up to 21% of patients.¹²⁸ LAUP is not approved by the American Academy of Sleep Medicine to treat OSA.¹²⁹ In addition, complications are common

Table 3
Comparison of surgical efficacy for OSA^a

Study	Demographics			AHI ^b			Ref.
	N	Studies	Age (y)	Before Surgery	After Surgery	% Change	
Bariatric surgery ^f	437	16	38.8 ± 14.9	53.3 ± 38.2	15.3 ± 18.7	-72.6 ± 60.6	338,339
GA	91	4	-	53.9	17.3	-67.8	134
HS	101	4	-	38.7	25.0	-33.0	134
HS and GA (or mortised genioplasty)	328	7	-	33.5	15.2	-58.0	134
LAUP	72	3	-	-	-	-	77
MMA	627	22	44.4 ± 9.4	63.9 ± 26.7	9.5 ± 10.7	-85.0 ± 18.2	266
Midline glossectomy	74	5	-	53.0	24.2	-54.4	134
Radiofrequency ablation (tongue)	394	11	-	37.0	23.4	-35.7	134
RME							
Children	88	3	7.1 ± 0.7	10.9 ± 4.7	0.8 ± 1.3	-91.0 ± 20.2	320
Adults ^d	10	1	27.0 ± 0.6	19.0 ± 1.3	7.0 ± 1.3	-63.2 ± 7.1	321
Tonsillectomy	1,079	23	6.5	18.6	4.9	-73.7	177,178
Tongue base suspension	77	6	-	29.0	16.3	-32.9	134
Tracheostomy ^e	33	2	47.2 ± 10.4	98.9 ± 36.0	26.2 ± 29.2	-79.2 ± 25.8	87,96
UPPP	992	37	48.1	60.0	-	-38.2	75,77
Multimodality surgery ^g	1,978	58	46.2	48.0	-	-60.3	74

Abbreviations: BMI, body mass index; GA, genioglossus advancement; HS, hyoid suspension; LAUP, laser-assisted uvulopalatoplasty; MMA, maxillomandibular advancement; N, number; RME, rapid maxillary expansion; UPPP, uvulopalatopharyngoplasty.

^a Plus-minus values are mean (or percent) ± standard deviation. - denotes not reported.

^b The AHI is the average number of apneas and hypopneas per hour during sleep.

^c Surgical success defined as the percent of subjects with an AHI <20/h and a ≥50% reduction in the AHI after surgery. Surgical cure defined as an AHI <5/h after surgery. For tonsillectomy, surgical cure was defined as an AHI <1/h after surgery.

^d Surgically assisted RME involved horizontal osteotomies.

^e The AHI for tracheostomy reported during non-REM sleep only and included only obstructive apneas and hypopneas.

^f A statistical significant reduction in BMI was noted at 18 months after bariatric surgery (pre-BMI 53.9 ± 15.7 vs post-BMI 37.8 ± 14.8 kg/m²; P<.001).

^g Multimodality surgery refers to simultaneous nasal, palate, and/or base of tongue surgery for OSA.

including early postoperative pharyngeal edema,^{130,131} with up to 59% complaining of persistent side effects (27% difficulty swallowing; 27% globus sensation in throat) after LAUP.¹²⁷

Midline glossectomy

Surgical removal of the center portion of the tongue base (usually via laser) was proposed by Fujita and colleagues¹³² and Woodson and Fujita¹³³ in 1991 for the treatment of OSA in patients with hypopharyngeal obstruction. A review of 5 case series (n = 74) showed a surgical success rate of approximately 50% (see **Table 3**).¹³⁴ Postoperative bleeding and pharyngeal edema requiring protective tracheostomy is not uncommon after surgery.^{132,133}

Radiofrequency ablation of the tongue

Radiofrequency ablation uses a probe to precisely direct temperature-controlled radiofrequency energy to heat (between 60 and 90°C) and ablate target tissues without causing collateral damage to adjoining structures.¹³⁵ Radiofrequency treatment of the tongue base does not require general anesthesia, but usually requires multiple treatment sessions over several weeks, and is successful at eliminating snoring.^{135–137} Eleven case series describing 394 patients with OSA (mean AHI 37/h) undergoing radiofrequency ablation of the tongue reported a surgical success rate of only 36% (see **Table 3**).¹³⁴ Statistically significant improvements in subjective daytime sleepiness and health-related quality of life were observed in most, but not all studies.^{135,138–145} Radiofrequency ablation of the tongue is generally considered adjunctive (not primary) OSA treatment in select patients.¹³⁴

Radiofrequency ablation of the turbinates (and other nasal procedures)

A relationship between nasal obstruction, mouth breathing and symptoms of OSA was first described in the 1800s.^{146–150} Increased nasal resistance may result from septal deviation, turbinate hypertrophy, chronic nasal congestion, polyps, or collapsible valves. Various procedures include polypectomy, radiofrequency ablation of the turbinates, alar valve or rim reconstruction, and septoplasty (eg, straightening of the septum). Nasal surgery is generally not curative,^{151,152} but can improve the AHI, and is often used in a multimodality surgical approach or to decrease CPAP pressure requirements.^{153–155} In addition, surgical correction of nasal obstruction improves health-related quality of life in patients with OSA.¹⁵⁶

Inferior turbinate enlargement is a frequent cause of nasal obstruction. Radiofrequency ablation is a highly successful surgical procedure producing volumetric inferior turbinate reduction. Radiofrequency ablation heats the hypertrophied turbinates causing scar tissue with resulting shrinkage over 1 to 3 weeks.¹⁵⁷ This procedure is generally performed in the outpatient setting with minimal discomfort beyond nasal stuffiness lasting 3 to 5 days.¹⁵⁸

Radiofrequency volumetric soft palate tissue reduction (somnoplasty)

Somnoplasty involves directed radiofrequency energy to ablate and reduce soft tissues of the palate.^{157,159–161} Decreased snoring occurs via scar-induced stabilization of the soft palate. Although symptom (eg, snoring) improvement after surgery is reported,^{136,162} evidence for improvement of OSA is lacking.^{163–165} A recent randomized placebo-controlled trial in patients with mild OSA found no statistically significant improvement in the AHI or symptoms after somnoplasty.¹⁶⁶

Tonsillectomy

Tonsillectomy is one of the most common surgical procedures in children.^{167,168} OSA is a frequent indication for tonsillectomy and is considered first-line therapy for

children with OSA.^{167,169} Surgical tonsillectomy techniques vary, but generally complete resection of the tonsils with adenoidectomy (if necessary) is preferred (**Fig. 1**).¹⁷⁰ Partial intracapsular tonsillectomy (eg, tonsillotomy) has been found to reduce postoperative morbidity (eg, pain),^{171–175} but postoperative objective measures of efficacy (eg, AHI reduction) are lacking.¹⁷⁶

Tonsillectomy is curative (AHI <1/h) in 60% of pediatric cases of OSA (see **Table 3**).^{177,178} Higher presurgery AHI and body mass index (BMI, calculated as weight in kilograms divided by the square of height in meters) are risk factors for residual disease after tonsillectomy.^{179,180} In complicated cases of pediatric OSA (eg, morbid obesity, severe OSA), tonsillectomy is curative in 39% of children compared with 74% in uncomplicated cases.¹⁷⁸ Children with residual OSA after tonsillectomy may benefit from rapid maxillary expansion.¹⁸¹ Tonsillectomy improves quality of life parameters (short- and long-term) in children with OSA,^{182,183} with improvements in behavior scores¹⁸⁴ and sleep disturbances.^{185,186}

Self-limited pain and swelling of the throat is common after tonsillectomy. Risks for postoperative complications include younger age (<24 months), increased severity of OSA, craniofacial abnormalities, obesity, poor functional status (eg, hypotonia, failure to thrive) and cor pulmonale.^{182,187} Life-threatening complications are rare,¹⁸⁸ but postoperative respiratory failure requiring mechanical ventilation (usually transiently during postsurgery recovery) occurs in approximately 30% of children.^{187,189} However, children left intubated electively after tonsillectomy have higher complication rates.¹⁹⁰ Hemorrhage, dehydration, and pulmonary edema occur in approximately 9% of cases.¹⁹¹

Uvulopalatopharyngoplasty

Fujita and colleagues²³ and Conway and colleagues²⁴ adapted Ikematsu's surgical snoring procedure²² and reported his uvulopalatopharyngoplasty (UPPP) results for treating OSA in 1980. This operation enlarges the oropharyngeal airway lumen by

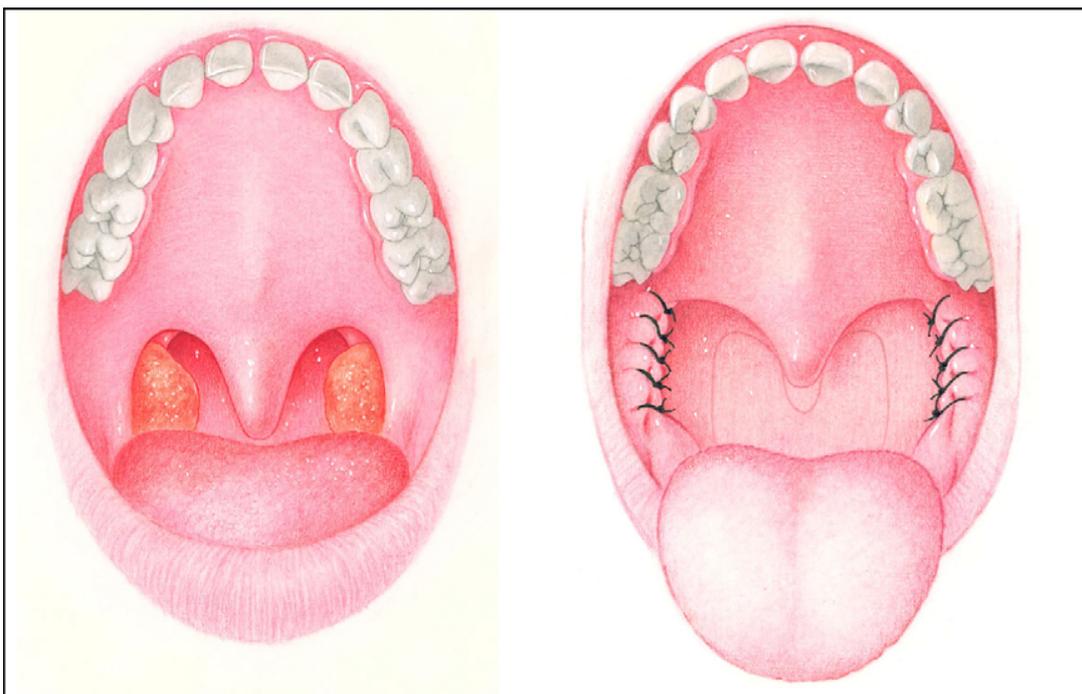


Fig. 1. Tonsillectomy. The primary treatment of OSA in children with tonsillar enlargement is tonsillectomy usually with concurrent adenoidectomy. To prevent collapse and improve OSA success, it is preferable that the lateral pharyngeal walls are sutured.

excising redundant tissues from the soft palate, tonsillar pillars, and uvula (**Fig. 2**). UPPP is currently the most widely performed OSA pharyngeal surgical technique in adults.⁷⁷ Several variations of the UPPP have been proposed including the methods of Fujita and colleagues,^{23,192,193} Simmons and colleagues,¹⁹⁴ Fairbanks,¹⁹⁵ Dickson and Blokmanis,¹⁹⁶ Friedman and colleagues,¹⁹⁷ and Powell and colleagues¹⁹⁸ (uvulopalatal flap surgery). Uvulopalatal flap surgery (**Fig. 3**) reduces the risk of nasopharyngeal incompetence and is associated with less postoperative pain, but is contraindicated in patients with excessively long or bulky soft palates (or uvulas).^{199–201} Woodson and Toohill²⁰² developed transpalatal advancement pharyngoplasty, which combines a UPPP with removal of the posterior hard palate (via a curvilinear palatal incision), with subsequent advancement of the mucoperiosteal flap and suturing to the alveolar mucoperiosteum (**Fig. 4**). This technique is associated with a decrease in retropalatal collapsibility and an increase in the retropalatal airspace compared with traditional UPPP, and may provide higher surgical success and cure rates.^{201,203}

There are no known randomized controlled trials of UPPP that assess pre- and post-surgery AHI,^{74,204,205} and many studies do not report objective postsurgery sleep data.²⁰⁶ One randomized trial found no statistically significant difference in the oxygen desaturation index between the surgery and conservative management groups.^{204,207} UPPP is highly effective for eliminating snoring, with success rates between 70% and 90%.¹⁹³ However, several meta-analysis have reported surgical success rates for OSA between 40% and 60%, and a surgical cure rate (an AHI <5/h) of only 16%

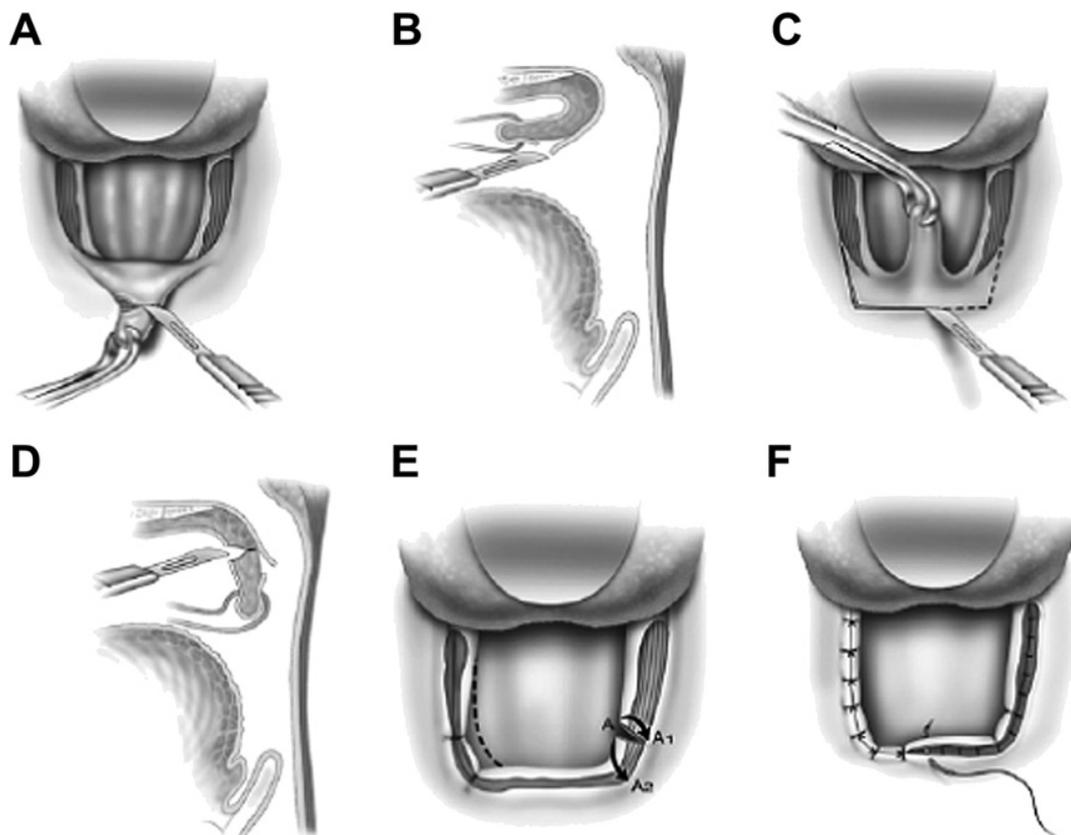


Fig. 2. Uvulopalatopharyngoplasty. This operation enlarges the oropharyngeal airway lumen by excising redundant tissues from the soft palate, tonsillar pillars, and uvula. Shown is Friedman's submucosal uvulopalatopharyngoplasty technique (A–F). (Reproduced from Friedman M, Schalch P. Surgery of the palate and oropharynx. *Otolaryngol Clin N Am* 2007;40:835; with permission from Elsevier.)



Fig. 3. Uvulopalatal flap. A modification of Fujita's uvulopalatopharyngoplasty involves retracting and advancing the uvula superiorly under the soft palate without removal.

(see **Table 3**).^{74,75,134} A recent retrospective analysis of the Mayo Clinic experience found a similar UPPP cure rate of 24%.²⁰⁸ Predictors of surgical cure in this analysis included younger age, lower preoperative BMI and AHI. Unfortunately, most patients with initial improvement in AHI after UPPP have recurrence within 5 years of therapy.²⁰⁹ Fortunately, UPPP likely confers a mortality benefit in CPAP intolerant

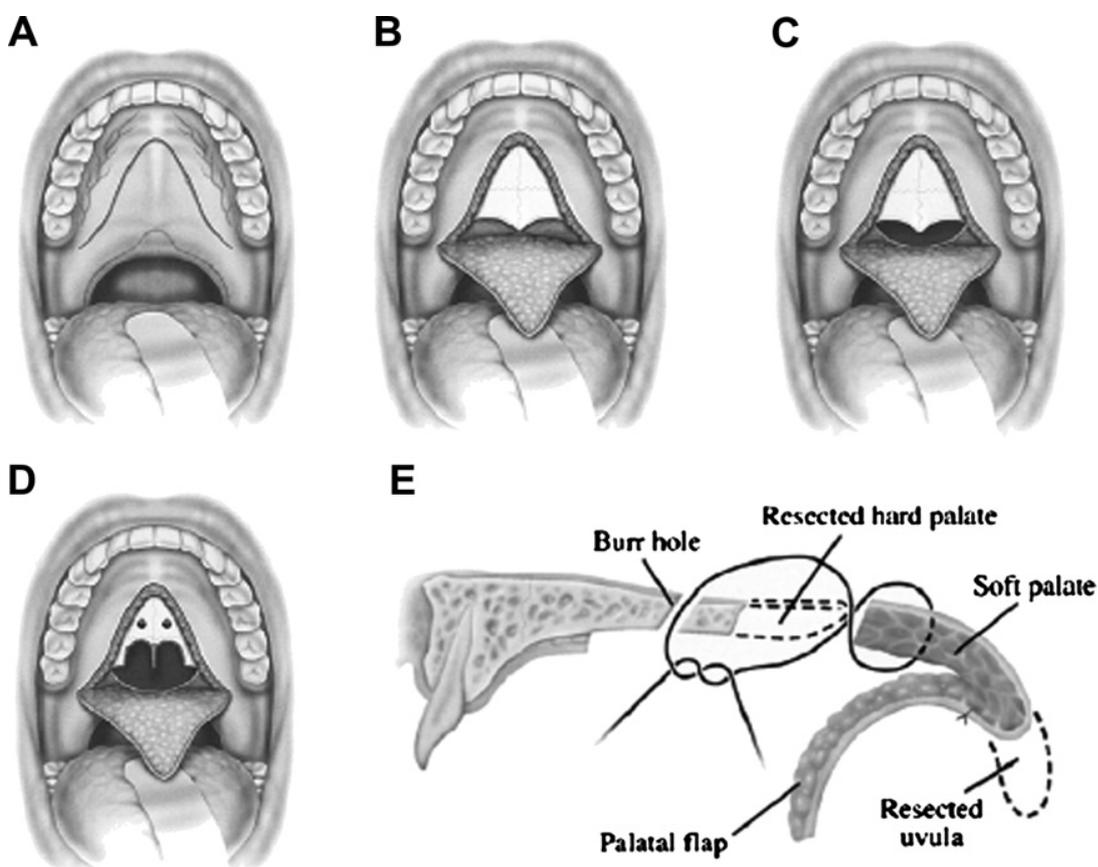


Fig. 4. Transpalatal advancement pharyngoplasty. Woodson's transpalatal advancement pharyngoplasty combines a uvulopalatopharyngoplasty with soft palate advancement. (Reproduced from Friedman M, Schalch P. Surgery of the palate and oropharynx. Otolaryngol Clin N Am 2007;40:840; with permission from Elsevier.)

patients (compared with no treatment), even when most patients do not obtain surgical cure.^{210–212} However, because UPPP is likely to eliminate snoring but will often leave residual OSA causing silent apnea, all patients must have postoperative sleep studies to rule out persistent disease.

UPPP is generally more effective at reducing apneas than hypopneas,^{75,193} and is most effective in patients with primarily oropharyngeal obstruction (as opposed to hypopharyngeal abnormalities).^{70,193} However, using fiberoptic endoscopy to select patients with predominantly soft palate pharyngeal collapse during a Müller maneuver has shown variable improvement in surgical success (45%–85%).^{213–216} Although the efficacy to cure OSA is suboptimal, UPPP may be useful in lowering positive airway pressure requirements, thus improving CPAP compliance in select patients.²¹⁷ However, UPPP may promote air leak during future CPAP therapy,^{218,219} although a recent study disputes this finding.²²⁰ Approximately 70% of patients are satisfied after UPPP.^{221,222}

Early postoperative complications include wound dehiscence, hemorrhage, infection, and transient velopharyngeal incompetence (eg, nasal regurgitation and hypernasal speech).¹⁹⁵ Late postoperative complications include pharyngeal discomfort (eg, dryness, tightness), postnasal secretions, dysphagia, inability to initiate swallowing, odynophagia, nasopharyngeal stenosis, taste and speech disturbances, tongue numbness, and rarely permanent velopharyngeal incompetence. Up to 30% of patients complain of persistent although generally mild dysphagia.^{223–226} A systematic review reported a serious complication rate of 2.5% with 30 deaths (~0.2% mortality) and persistent side effects in 58% (31% nasal regurgitation, 13% voice changes, 5% taste disturbances) of patients after UPPP.¹²⁷ Voice changes are generally mild.²²⁷ A recent study noted that health-related quality of life measurements were better in patients with post-UPPP side effects compared with CPAP users (independent of compliance) with side effects.²²⁸

Procedures that Modify or Advance the Skeletal or Soft Tissue Structures

Genioglossus advancement

In the mid-1980s, Riley and colleagues^{229,230} first described genioglossus muscle advancement (GA) to improve the posterior airspace (eg, base of tongue). Their initial technique (a modified horizontal mandibular osteotomy) was later improved in 1986 to include a limited inferior parasagittal mandibular osteotomy (**Fig. 5**).^{230,231} Advancing the geniotubercle forward of the mandible positions the genioglossus and geniohyoid muscles anteriorly, thus enlarging the retrolingual space.²³² Variations of this procedure include mortised genioplasty, circle genioplasty, and standard genioplasty.^{233,234} Four case series describing 91 patients with severe OSA (mean AHI 54/h) undergoing GA as sole treatment report a surgical success rate of 67% (range 39%–79%) (see **Table 3**).¹³⁴ GA is generally used within a multimodality approach to treat base of tongue obstructions.

Hyoid myotomy and suspension

In the mid-1980s, Riley and colleagues^{229,230,235} developed a hyoid suspension procedure to improve the posterior (retrolingual) airspace (**Fig. 6**). The hyoid bone is located in the anterior neck below the mandible and is involved in maintaining upper airway patency.^{236,237} Several protocols have been described including hyoid to mandibular suspension (hypomandibular), hyoid to thyroid cartilage suspension (thyrohyoid), and hyoid expansion.²³⁸ Hyoid suspension is generally used within a multimodality approach^{238–240} with a surgical success rate (performed with previous or concurrent palate surgery) of approximately 50% (see **Table 3**).¹³⁴ However, there

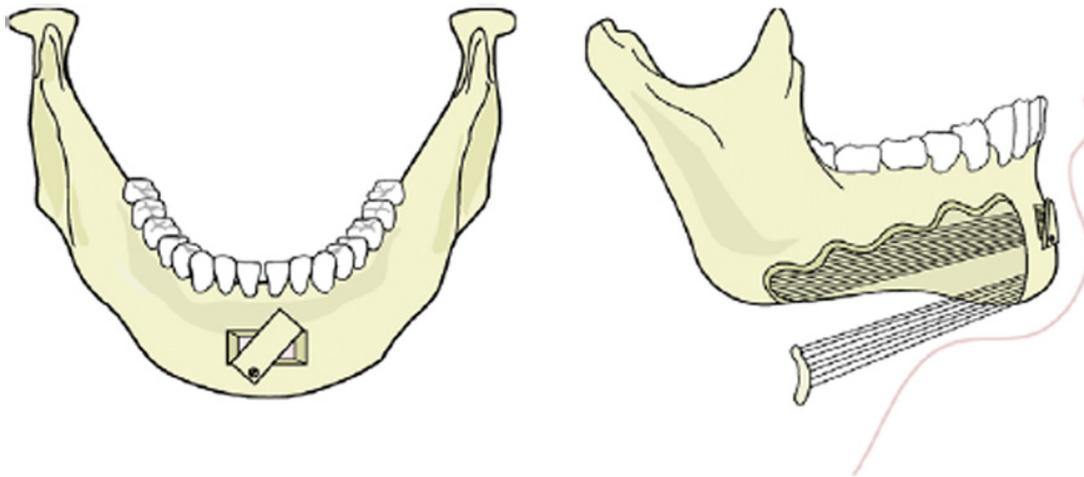


Fig. 5. Mandibular osteotomy with genioglossus advancement. Limited inferior parasagittal mandibular osteotomy (eg, a rectangular window in the symphyseal bone with advancement, rotation, and immobilization of the geniotubercle) with advancement of the genioglossus and geniohyoid muscles. (Reproduced from Li KL. Hypopharyngeal airway surgery. *Otolaryngol Clin N Am* 2007;40:848; with permission from Elsevier.)

are no reliable preoperative predictors for success with hyoid suspension following UPPP.²⁴¹ Furthermore, combining genioglossus advancement with hyoid suspension marginally improves surgical success (~55%) (see **Table 3**),¹³⁴ and 1 study of hyoid suspension with radiofrequency of the tongue reported a surgical success rate of only 49%.²⁴² Excessive daytime sleepiness generally improves after hyoid suspension, albeit inconsistently.^{239,243–246}

Mandibular (or maxillary) distraction osteogenesis

Distraction osteogenesis (DO) of the mandible (and/or maxilla) involves bilateral segmental osteotomies followed by gradual distraction (via an expandable intra- or extraoral device) with subsequent ossification and bone lengthening.²⁴⁷ DO of the mandible effectively improves OSA in children with genetic craniofacial abnormalities.^{248–251} One study of 5 otherwise normal adults with OSA reported a decrease in AHI from 49/h to 7/h after mandibular (or maxillary) DO.²⁴⁷ However, this study

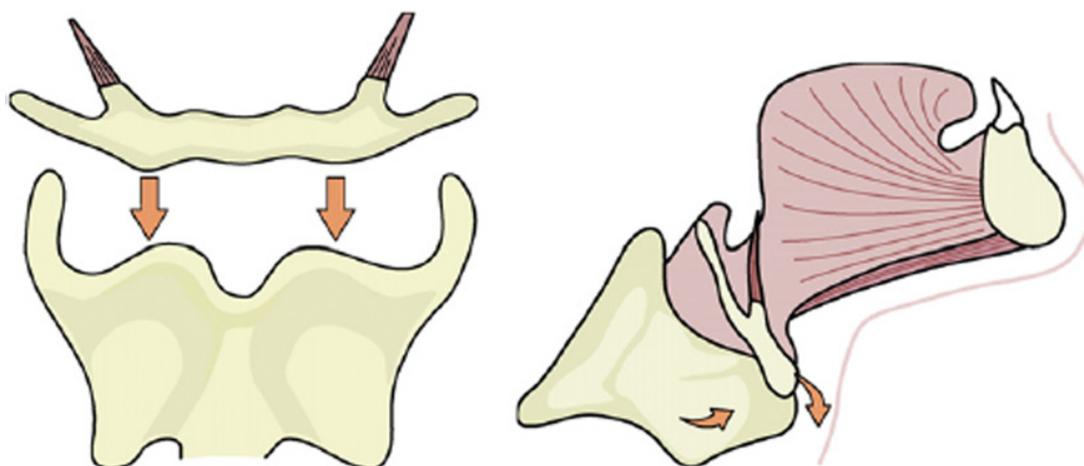


Fig. 6. Hyoid myotomy suspension. Hyoid to thyroid cartilage suspension (thyrohyoid) involves isolation of the hyoid bone that is advanced, sutured, and immobilized to the thyroid cartilage. (Reproduced from Li KL. Hypopharyngeal airway surgery. *Otolaryngol Clin N Am* 2007;40:848; with permission from Elsevier.)

reported several problems with DO including the technical difficulty of the procedure, a high risk of malocclusion, subsequent need for orthodontics because of limited control of the distractor vector, and poor patient satisfaction (eg, treatment required 4 months of stabilization via intraoral arch bars that inhibited mastication and speech).

Maxillomandibular advancement

In 1979, Kuo and colleagues²⁰ reported improvements in polysomnographic parameters and subjective sleepiness in 3 patients with OSA with retrognathia after mandibular osteotomy with advancement. Similar improvements in OSA parameters after mandibular advancement were noted by others.^{21,252,253} However, by the mid-1980s, mandibular advancement alone was largely supplanted by combined maxillary and mandibular advancement to preserve the maxilla-mandibular relationship and from the recognition that the physiologic cause for OSA is often from concomitant mandibular and maxillary deficiency.^{235,254} Mandibular osteotomy with advancement is currently relegated to the treatment of mandibular hypoplasia in syndromic children with OSA.²⁵⁵

Maxillomandibular advancement (MMA) involves Le Fort I maxillary and bilateral sagittal ramus split mandibular osteomies with advancement of the maxilla and mandible followed by rigid fixation (**Fig. 7**).²⁵⁶ Generally, the maxilla is advanced first, with the mandible advanced into occlusion. Combined MMA alleviates pharyngeal obstruction by expanding the skeletal framework that the tongue and other soft tissue

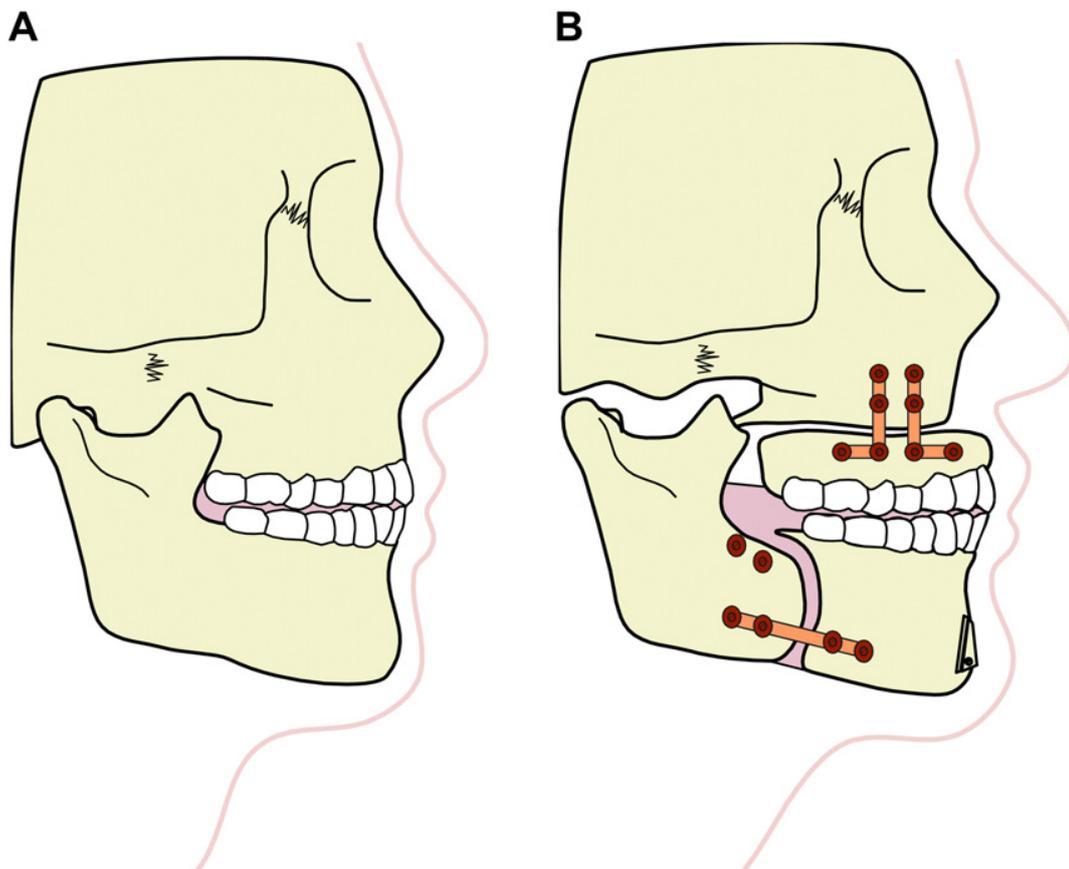


Fig. 7. Maxillomandibular advancement. Before (A) and after (B) maxillomandibular advancement surgery via a Le Fort I osteotomy (with rigid plate fixation) and bilateral sagittal split mandibular osteotomy (with bicortical screw fixation). (Reproduced and modified from Li KL. Hypopharyngeal airway surgery. *Otolaryngol Clin N Am* 2007;40:849; with permission from Elsevier.)

structures attach to resulting in reduced upper-airway restriction and collapsibility during inspiration.²⁵⁷ Mandibular advancement advances the tongue and suprahyoid muscles.²⁵⁸ Maxillary advancement pulls forward the velum and velopharyngeal muscles,²⁵⁹ increases the nasopharyngeal and hypopharyngeal spaces,^{260,261} and increases alar width with a concomitant decrease in nasal airway resistance.^{262,263} Improvements in pharyngeal obstruction after MMA occur along the entire upper airway in the lateral and anteroposterior dimensions.^{27,264}

MMA is the most effective craniofacial surgery (in adults) for the treatment of OSA.^{76,265} A recent meta-analysis of 22 studies (627 subjects with OSA) determined that MMA is highly effective with a mean decrease in AHI from 64/h to 11/h ($P < .001$) with pooled surgical success and cure (AHI $< 5/h$) rates of 86% and 43%, respectively (see **Table 3**).²⁶⁶ Predictors of increased surgical success include younger age, lower preoperative AHI and BMI, and greater degree of maxillary advancement.²⁶⁶ Furthermore, MMA maintains its efficacy at long-term follow-up.^{267,268} Following MMA, most patients report improvements in health-related quality of life, depression, excessive daytime sleepiness, memory impairment, and hypertension.^{267,269–279} Candidates for MMA include adults and adolescents (after the cranial sutures have completely ossified) with maxillomandibular insufficiency or those who have failed previous therapeutic interventions for OSA.^{256,265} In addition, MMA is successful in patients with obesity or with severe OSA.²⁶⁵

MMA is generally safe with no reported deaths and a major complication rate of only 1.0% (mostly cardiac causes).²⁶⁶ Between 0% and 15% of MMA patients experience mild surgical relapse without apparent symptoms or worsening of the AHI.^{267,280–282} However, relapse is not associated with the degree of mandibular advancement.^{281,283,284} Mild malocclusion occurs in up to 44% of patients and is generally treatable with prosthetics or minor occlusional equilibration.^{267,285} Transient facial paresthesia (ie, inferior alveolar nerve neurosensory deficits) after MMA is common (~100%), although most cases (86%) resolve within 1 year.²⁶⁶ Velopharyngeal insufficiency or mild speech and swallowing deficits are rarely reported after MMA.^{272,280,286,287} Patients completing sequential phase I (UPPP) and phase II (MMA) surgery generally report less pain after MMA compared with phase I surgery.^{287,288} The average hospitalization time is less than 1 week with most patients returning to work within 4 to 10 weeks after surgery.^{275,288}

After MMA, most patients report a positive perception of facial aesthetics.²⁸⁹ Li and colleagues^{289,290} noted 6 months after surgery that 50% of patients report a younger facial appearance, 36% report a more attractive appearance, and only 9% report a less attractive facial appearance. In this same study, all patients (100%) reported satisfaction with the surgical outcome. Three other studies reported no patients (0%) were bothered by postoperative facial aesthetics.^{272,274,286} Modified MMA techniques, particularly using counterclockwise rotation and pre- or postsurgical orthodontics, have been developed to prevent maxillary protrusion and improve aesthetics.²⁹¹

Maxillomandibular expansion

Surgically assisted maxillomandibular expansion (MME; limited osteotomy at Le Fort I level and midline maxilla followed by expansion) may be an effective therapy for OSA in adults (**Fig. 8**).^{292,293} One study ($n = 6$) reported improvements in excessive daytime sleepiness and OSA (AHI from 13/h to 5/h) at a mean follow-up of 18 months after an average mandibular and maxillary expansion of 9.5 and 10.3 mm, respectively.²⁹³ The investigators concluded that nonobese adolescents or young adults with mild OSA and who require orthodontic treatment are ideal candidates for MME.

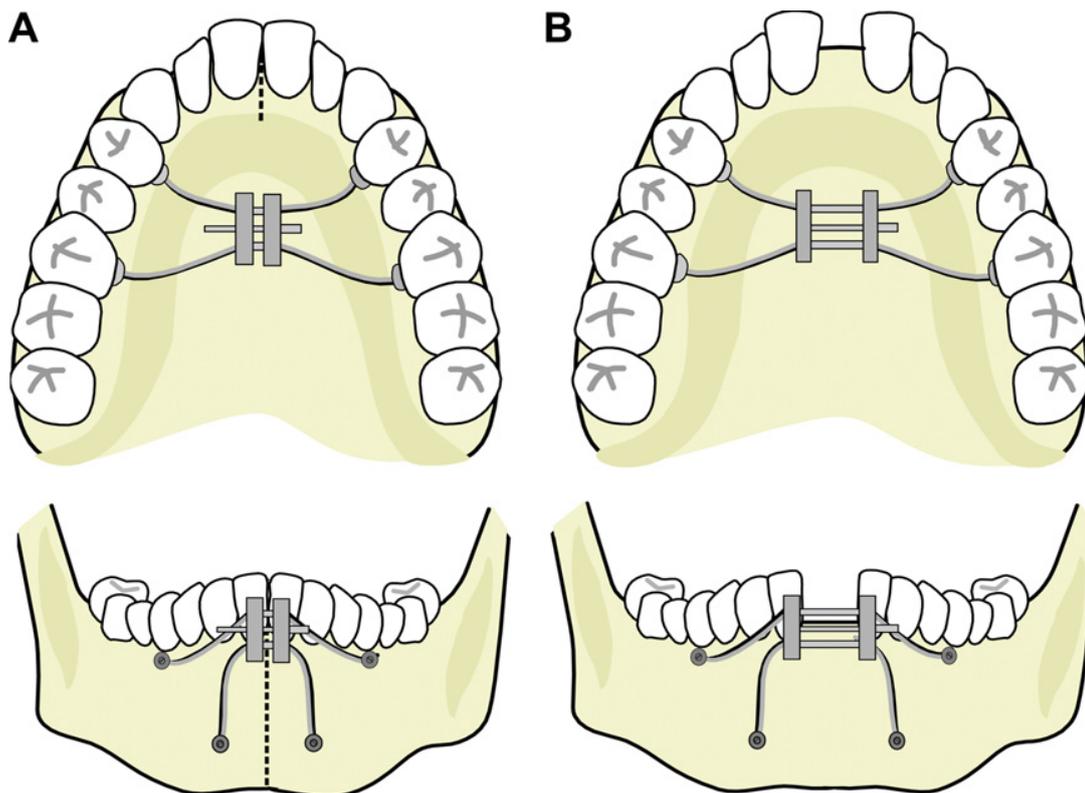


Fig. 8. Maxillomandibular expansion. Before (A) and after (B) surgically assisted maxillo-mandibular expansion with Le Fort I osteotomy and pterygomaxillary (midline) dysjunction followed by expansion using an orthodontic screwlike device.

Pillar palatal implants

This minimally invasive procedure involves inserting matchstick size rigid polyester implants via a hollow needle delivery tool into the soft palate.²⁹⁴ Pillar implants improve snoring by stiffening the soft palate, but their effect on OSA is less clear and the long-term benefits on OSA are unknown.^{295–298} In a prospective nonrandomized trial of 25 patients with mild-moderate OSA (mean AHI 16.2/h), the surgical success and cure rates were 40% and 28%, respectively.²⁹⁶ Friedman and colleagues²⁹⁵ in a randomized trial of 62 nonobese patients with mild-moderate OSA (mean AHI 23.5/h), found a statistically significant improvement in AHI after pillar implants (compared with placebo procedure), although the mean AHI after surgery was still within the moderate range (mean AHI 15.9/h) with a surgical success rate of 45%. Complications are rare, but include infrequent postinsertion extrusion.

Rapid maxillary expansion

In 1860, Angell²⁹⁹ reported the first use of rapid maxillary expansion (RME) to correct a transverse maxillary deficiency. RME is currently a common orthodontic procedure to correct dental crowding and to ensure a normal mandibular-maxillary relationship.³⁰⁰ RME expands the mid-palatal suture via a screw-type orthodontic appliance resulting in an increase in the upper transverse width.³⁰⁰ RME induces normal tongue positioning via palatal widening and flattening, downward and forward displacement of the maxilla, widening of the nasal vault (with subsequent decreased resistance and improvement in nasal breathing) and transforms a class III to a class I prognathoid position.^{301–312} After 2 to 4 weeks of expansion, a 2- to 6-month retention period is necessary while ossification between the expanded mid-palatal suture line is completed.³⁰⁴ The suture line in prepubertal children is cartilaginous and easily

separated, but horizontal osteotomy is often required in adults (whose suture line is generally ossified) before RME.³¹³

Children without known OSA often report quieter nighttime breathing, reduced snoring, and improved sleep quality after RME.^{303,314,315} In 1996, Palmisano and colleagues³¹⁶ reported the first use of RME to successfully treat OSA (AHI went from 22/h to 4/h) in a 22-year-old with maxillary constriction and a class I malocclusion. Subsequently, 3 studies evaluating RME in children with OSA ($n = 88$; mean expansion 6.2 ± 2.1 mm)^{181,317–319} reported a mean decrease in AHI from 11/h to 0.8/h after RME ($P < .001$) with subjective improvements in snoring, excessive daytime sleepiness, and behavioral problems (see **Table 3**).³²⁰ One study of 10 adults with OSA who received surgically assisted RME (mean expansion 12.1 mm) reported statistically significant improvements in AHI (19/h to 4/h; $P < .05$) with a 70% cure rate (AHI < 5 /h).³²¹

SINGLE-STAGE MULTIMODALITY APPROACH

Pharyngeal surgeries are often combined to address airway obstruction(s) at multiple levels (eg, nose, palate, tonsils, hypopharynx). Multilevel surgery may improve surgical success compared with single-site therapy.^{322–324} The most common single-stage multimodality procedure combines UPPP with a second procedure designed to improve the hypopharyngeal airway (eg, genioglossal advancement, hyoid suspension, base of tongue resection [uvulopalatopharyngoglossoplasty]). Lin and colleagues⁷⁴ in a meta-analysis of 58 studies emphasized the benefits of a multimodality approach reporting a surgical success rate of 66% (see **Table 3**).

Unfortunately, multimodality surgery does not always guarantee increased efficacy. A meta-analysis of hypopharyngeal surgery by Kezirian and Goldberg¹³⁴ concluded combination procedures such as genioglossus advancement with hyoid suspension or tongue radiofrequency treatment with tongue stabilization have lower surgical success rates and poorer AHI improvement compared with the same procedures performed alone.

STAGED SURGICAL PROTOCOL

The Riley-Powell-Stanford surgical protocol was developed to address the multilevel airway abnormalities that often contribute to OSA (**Fig. 9**).³²⁵ Phase 1 consists of interventions directed at the site(s) of obstruction in the nasal, pharyngeal, or hypopharyngeal regions (eg, UPPP for oropharyngeal obstruction, genioglossus advancement for hypopharyngeal obstruction).³²⁶ Approximately 6 months after surgery, repeat polysomnography is performed and patients who do not obtain surgical success (or cure), proceed to phase 2 surgery consisting of MMA.³²⁷ The Stanford group reports a staged protocol surgical success rate of 95%.³²⁵

However, the appropriateness of the staged protocol has been questioned. Wagner and colleagues²⁷⁴ noted that two-thirds of their MMA failures had previous phase 1 surgery (eg, UPPP). Others have proposed that MMA should be performed first with UPPP (or other palatal and hypopharyngeal surgeries) performed in those patients with residual OSA.²⁶⁸ A review by the American Sleep Disorders Association found insufficient evidence to assess the efficacy of a staged versus primary MMA surgical approach.⁷⁵ A recent meta-analysis of MMA found that patients with previous UPPP before MMA were less likely to obtain surgical cure (25% vs 45%; $P = .002$) compared with those without previous surgery following MMA.²⁶⁶ However, this finding was likely confounded by greater obesity and more severe OSA in patients with previous palatal surgery. The investigators concluded that, “further research is needed to identify

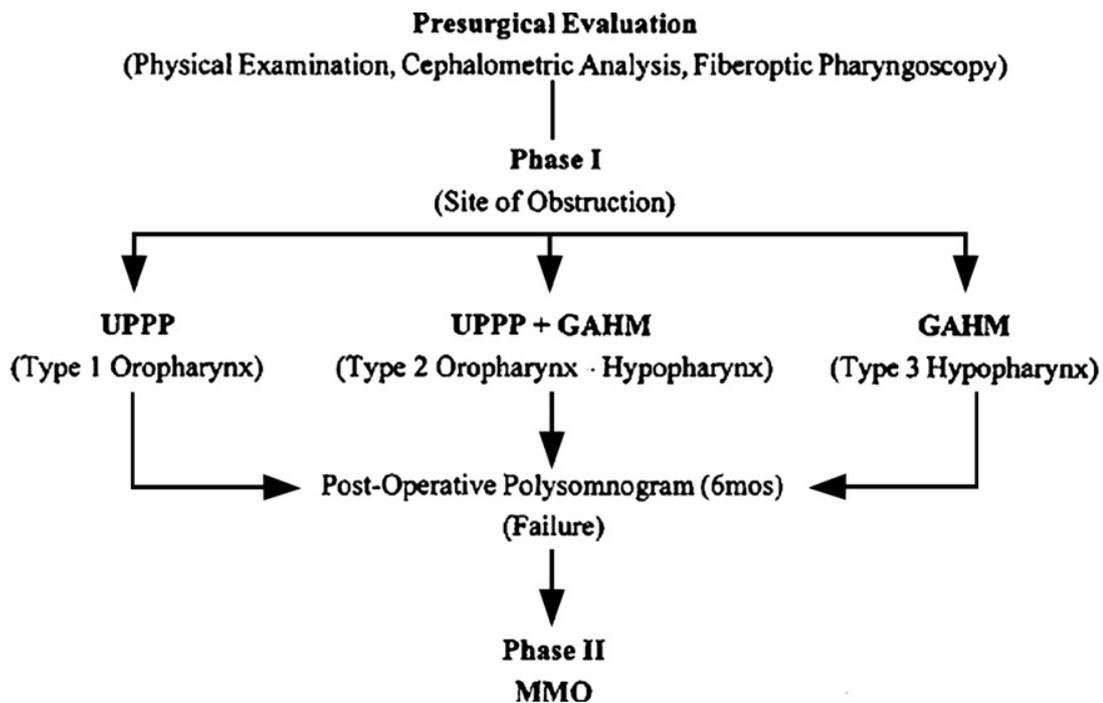


Fig. 9. Riley-Powell-Stanford surgical staged protocol. (Reproduced from Riley RW, Powell ND, Li KK, et al. Surgery and obstructive sleep apnea: long-term clinical outcomes. *Otolaryngol Head Neck Surg* 2000;122:416; with permission from Mosby-Year Book, Inc.)

preoperative patient and clinical characteristics to select those patients who would benefit most from a staged versus primary MMA surgical approach.”²⁶⁶

BARIATRIC SURGERY

Approximately 65% of adults in the United States are overweight (BMI >25 kg/m²) and more than 30% are obese (BMI >30 kg/m²).³²⁸ Surgically induced weight loss was first performed in 1967³²⁹ and is now a preferred weight reduction modality for morbidly obese individuals (BMI ≥ 40 kg/m²) with more than 100,000 procedures performed annually in the United States.³³⁰ Bariatric surgery is generally safe, results in marked and sustained weight loss, and is associated with improved mortality compared with conventional weight-loss strategies.^{331–333} Procedures are classified as predominantly malabsorptive (eg, biliopancreatic diversion, duodenal switch, jejunoileal bypass), predominantly restrictive (eg, vertical banded gastroplasty, adjustable gastric banding, sleeve gastrectomy, intragastric balloon), or combined malabsorptive and restrictive (eg, Roux-en-Y gastric bypass, sleeve gastrectomy with duodenal switch).³³³ Candidates for bariatric surgery should fulfill the 1991 National Institutes of Health guideline criteria that includes a BMI ≥ 40 kg/m², or a BMI ≥ 35 kg/m² with associated comorbidity (eg, OSA).^{334,335}

Obesity is a leading cause of OSA with an estimated 40% prevalence in obese persons (BMI ≥ 30 kg/m²).³³⁶ A 10% increase in BMI results in a 32% increase in the AHI.³³⁶ Mild to moderate weight reduction can improve sleep apnea and daytime sleepiness.^{336,337} Two recent meta-analyses have evaluated the effectiveness of bariatric surgery to treat OSA.^{338,339} Holty and colleagues³³⁹ found OSA to be highly prevalent (79%) among bariatric candidates (of these 76% had moderate to severe disease), but exceedingly underdiagnosed (only 30% preoperatively). There were no identifiable presurgical symptoms or clinical findings predictive of polysomnographically confirmed OSA.³³⁹ Greenberg and colleagues³³⁸ noted that after surgically

induced weight loss (BMI went from 55 to 38 kg/m²), the AHI improved from 55 to 16/h (see **Table 3**). However, more than 50% of bariatric recipients with preoperative OSA have residual disease despite weight loss.³³⁹ Predictors of greater AHI reduction (or OSA cure) included younger age, but not symptom improvement (eg, excessive daytime sleepiness) or the degree of BMI change.^{338,339} In addition, initial improvements in AHI appeared to wane at follow-up despite maintained weight loss.³³⁹

SUMMARY

OSA is a prevalent condition associated with increased morbidity and mortality. Although CPAP is the preferred treatment, poor compliance is common. Fortunately, several surgical treatments exist to address a variety of pharyngeal abnormalities. Case series suggest that MMA has the highest surgical efficacy (86%) and cure rate (43%). Morbidly obese individuals may benefit from bariatric surgery, although less than 50% are cured after surgically induced weight loss. Soft palate surgical techniques are less successful, with UPPP having an OSA surgical success and cure rate of 50% and 16%, respectively. Patients may benefit from a multimodality surgical approach. In conclusion, individuals intolerant of CPAP may benefit from surgical therapies that address their particular airway obstruction(s). However, further research is needed to more thoroughly assess clinical outcomes (eg, quality of life, morbidity), better identify key preoperative patient and clinical characteristics that predict success, and confirm long-term effectiveness of surgical modalities to treat OSA.

ACKNOWLEDGMENTS

We thank Kasey K. Li, MD, DDS, for graciously providing the figures illustrating tonsillectomy, uvulopalatal flap, and maxillomandibular expansion procedures.

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