Reconstruction of Airway Soft Tissues in Obstructive Sleep Apnea

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KEYWORDS

• Obstructive sleep apnea • Palatopharyngoplasty
• Glossectomy • Nasal surgery snoring • Adenotonsillectomy

Surgery for obstructive sleep apnea (OSA) is multimodal. Procedures and aims of treatment vary. Surgery, medical devices, and medical therapy each may contribute to individualized patient care. There is no single procedure or intervention that ''cures'' upper airway obstruction. Treatment varies as the disease varies. In addition, surgical treatment varies because the level of obstruction and influence on air flow occurs at multiple levels and from many structures.

Simply put, the problem in OSA centers on an airway too small for a given individual's physiology. The human larynx, unlike other mammals, has a more caudal location, away from the skull base. The result is a conduit prone to collapse and obstruct where there is soft tissue. Portions of the upper airway from the nasal tip to the glottis are surrounded by osseous or cartilaginous structures that provide some stability. The remainder of the airway is composed of soft tissue.

The flow of air through a collapsible tube is influenced by the difference in upstream, downstream, and intraluminal forces (as a Starling resistor). Multiple levels of the upper airway influence obstruction. The propensity for collapse at each level may be influenced by various tissues including muscle, mucosa, vascular tissue, and mechanoreceptors. Our understanding of how each of these components participates in the obstruction is incomplete. Despite this, for some individuals, surgery can play a critical role as part of the multimodal algorithm for treating OSA.[1,2]

The concept of surgery for sleep-disordered breathing is often misunderstood. It is not a unique procedure, or even set of procedures, but a reconstructive approach to improve airway form and function with the goal of reducing severity of disease. With this concept the goal of surgery is not limited to surgery as a first-line ''cure'' of all disease but rather a tool used also for ancillary treatment combined with other medical modalities or as salvage. Although surgical salvage treatment after medical failure is an important role for the surgeon it is not the only and may not be the primary one.

Criticism of surgery for sleep apnea and sleep-disordered breathing is often enthusiastic because in major reviews, procedures are pooled, treating all techniques as equivalent. Further, many fail to account or understand that multiple levels of the airway and therefore multiple structures may be involved. Last, assessing clinical outcomes when multiple interventions or procedures are needed especially for surgery is difficult. This article is not intended as a critical assessment of surgical outcomes but rather will focus on airway structures and the nature of the procedures applied to influence them.

STRUCTURE

A hurdle impeding the progress of surgical treatment of OSA and sleep-disordered breathing is the concept that surgery treats the soft tissues around the airway. This focus on the ''donut'' or the soft tissue and skeletal ring surrounding the airway distracts attention from the ''donut hole.'' The airway lumen is the ultimate literal and

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figurative pathway of disease. Obviously, tissue defines a lumen, but it is the nature and function of the lumen that is pathologic in most patients, not the tissues themselves. A tissue focus may, in some ways, perpetuate the widespread impression that there may be a single common method or procedure to treat the disorder. With this concept, all that is needed to fix sleep apnea is for someone to discover the secret procedure. Although appealing, because of its simplicity, the concept fails in most patients because it is not the ''palate'' that is abnormal but the associated ''palatal airway.'' The key outcome of ''reconstructive airway'' surgery is normalization of airflow and not normalization of tissue. Unfortunately, airflow, especially in the upper airway, is complex and there are few uncomplicated solutions. OSA is not just a structural disease but also involves a loss of physiologic compensation; however, a reconstructive approach may provide marked clinical improvement when applied appropriately.

Reconstructive surgery has the goal of improving form and function. Tissues may be moved, removed, or remodeled using many techniques. In the airway, acute goals include changing airway size, volume, shape, and compliance. This must be achieved while limiting morbidity or negative alteration of other functions of the upper airway, including smell, taste, swallowing, and speech. Interventions for the upper airway are still evolving, and many new ones are being assessed. Many techniques used in the past will likely be abandoned.

Current methods are stages of an evolutionary process and certainly will change. The two surgical approaches to treat OSA and related disorders include bypassing the upper airway, or reconstructing the skeletal or soft tissues. A large variety of techniques have been described, reflecting, to some degree, surgeon preferences but also the wide variability of upper airway structural anatomy that contributes to disease. The skeletal and cartilage framework not only determines much of the size, shape, and compliance of the airway but also describes the intrinsic patterns of facial growth that correlate to risk factors for sleep-disordered breathing. The position of the human larynx in the neck instead of at the skull base is important. This position is speculated to be related to changes in human cranial development and growth patterns. Cranial development, therefore, can affect both facial and airway form. A major risk factor for OSA is a longer soft tissue pharyngeal airway superior to the larynx. This structure is more susceptible to collapse from changes in body position, vascular volume, and tissue edema, and loss of muscle tone during sleep. Skull base development in OSA also creates a narrowed posterior maxillary airspace.

Soft tissue changes worsen an airway already at risk. Often, a constellation of abnormalities are markers of facial skeletal abnormalities in OSA. These may include increased distance of the hyoid bone from the mandibular plane, decreased mandibular and maxillary projection, increased vertical length of the lower anterior face, increased vertical length of the posterior airway, and increased cervical angulation. In addition, other factors contribute to OSA.

Facial structure interacts with obesity to create apnea risk. In the Wisconsin Sleep Cohort, a population-based study, two thirds of the apnea-hypopnea index (AHI) was explained by a combination of facial structure and obesity.[3] In nonobese subjects, facial structure alone explains AHI. Individuals with normal craniofacial structures require morbid obesity or other airway pathology to display OSA. Soft tissues are frequently abnormal in OSA, both primarily and also as a consequence of the disease.

There is a strong familial aggregation of apnea, and in some populations, this has been linked to soft tissue abnormalities. Genetic studies have demonstrated the inheritability of abnormal lateral wall size and tongue size in populations of patients with apnea.[4] Soft tissue abnormalities include a longer and wider soft palate, larger tongue, smaller oropalatal airspace, a posteriorly placed epiglottis, and smaller posterior airspace.

Obesity alters OSA in both direct and indirect ways. It increases the severity of OSA by worsening hypoxemia during sleep. Obesity also effects metabolism, ventilation, and lung volume to worsen apnea. Leptin and other inflammatory obesity-related cytokines may increase CO2 response, and central ventilatory sensitivity augments the tendency toward periodic breathing and increased airway instability. Increased soft tissue tongue size has also been associated with obesity. Fat distribution around the neck has long been postulated, without evidence, to compromise the airway.

ANESTHETIC CONSIDERATIONS

Patients with sleep apnea may have unique preoperative, intraoperative, and postoperative care issues.[5] Difficulty with intubation and extubation related to facial structural or ventilatory control issues exist. High-risk patients include, but are not limited to, patients with severe obesity, poor pulmonary reserve, pharyngeal tissue redundancy, hypoxemia, access to narcotic use, multiple airway surgical procedures, and

excessive sleepiness. Controversy exists as to the appropriateness of outpatient surgery and the need for intensive postoperative monitoring.[6] Postoperative management can be helped in many cases with the use of nasal continuous positive airway pressure (CPAP). Rarely, tracheotomy is required. Objective monitoring to include pulse oximetry has been advocated; however, it is critical to realize that oximetry does not measure hypoventilation, especially when assessed on an intermittent basis or when low-flow oxygen is in use. Close nursing observation is necessary in the perioperative period. Symptoms of respiratory insufficiency and hypercarbia may include increased pulse and respiratory rate, elevated blood pressure, and agitation or restlessness. Studies suggest that the stimulating and disruptive environments of the hospital provide a degree of safety and that risk may increase in quiet and unobserved areas. Risk increases with sedation, dehydration (increasing tenacious secretions), and increased doses of narcotics. Postoperative interventions for patients with apnea who have airway and nonairway surgery may include treating nasal airways, nasal CPAP, patient positioning (elevation of the head of bed), adequate hydration, corticosteroids, and other non-narcotic pain medication. Patients with sleep apnea are also at elevated risk because of significant comorbidities of hypertension, cardiac and pulmonary disease, and obesity.

AIRWAY ASSESSMENT

Airway assessment in patients at risk for OSA may include (1) identifying features that predict risk of apnea and severity of disease; (2) improving patient selection with the goal of eliminating patients at high risk of failure; and (3) identifying specific structural abnormalities to direct reconstruction. Assessment must also balance the severity of the disorder, host comorbid conditions, and anticipate clinical outcomes to determine surgical approach. Patients, especially those with high risk factors for disease and unfavorable airway structure who are unable or unwilling to proceed with limited interventions, may warrant approaches such as maxillomandibular surgery or tracheotomy.

Methods of evaluation include physical examination, endoscopy, and radiologic assessment. Inspection without instrumentation provides information about facial growth (hyoid, neck, mandible, and maxillary positioning). Significant nasal disease can be identified or suspected with limited instruments. Routine physical examination may assess the nasal valve, septal position, high arched palate, tonsil size, tongue size, and evidence of pharyngeal tissue damage. All patients deserve an adequate examination, especially if initial therapy is less than completely successful.

FRIEDMAN STAGING

The Friedman staging system for the oral cavity and oropharyngeal portions of the upper airway defines four stages based on the following: (1) tonsil size (1 to 4+); (2) a modification of the Mallampati classification (1 to 4+); (3) presence or absence of severe obesity (> or < BMI of 40 kg/m^2); and (4) major craniofacial abnormalities.[7] The Friedman staging system identifies patients at risk for apnea who present with symptoms of snoring. It also demonstrates both positive and negative uvulopalatopharyngoplasty (UPPP) predictive values.

Friedman staging groups tonsil size as ''favorable'' (tonsil grades 3 and 4, ie, large tonsils), or ''unfavorable'' (tonsil grades 1 and 2, ie, small tonsils). The Modified Mallampati classification is ''favorable'' (grades 1 and 2, visualizing the free margin of the soft palate), and ''unfavorable'' (grades 3 and 4, free margin of palate not visible) on examination. Friedman Stage I has demonstrated high success rates with limited palatal surgery alone. Friedman Stage III demonstrates less success rate with limited palatal surgery.

TRACHEOTOMY

Historically, tracheotomy was the first intervention for severe OSA. The procedure has high social morbidity. Long-term studies demonstrate that, although tracheotomy reduces the morbidity and mortality associated with sleep apnea, it has primary morbidity as well.[8] Obesity, a short neck, a low larynx, and the inability to extend the neck may complicate tracheotomy. Experienced centers often perform ''skin-flap'' tracheotomy techniques that reduce associated morbidity. In OSA, the airway in wakefulness is patent, and tracheotomies may be occluded during wakefulness and opened only during sleep. In perceived psychosocial implications, risks of stenosis, infection, and other potential complications, tracheotomy has limited application. With the advent of acceptable medical treatment options, tracheotomy is often reserved for severe disease, complicated airway management, perioperative airway safety, and for patients too ill for other procedures or therapies.

NASAL SURGERY

A seeming paradox has been described regarding the nose and OSA. Nasal obstruction has clearly been demonstrated as a risk factor for OSA, yet treatment does not necessarily affect the disease risk. Aside from the extraordinarily unlikely possibility that nasal disease does not impact sleep treatment, studies must be interpreted correctly. Symptomatic nasal obstruction is poorly associated with abnormal resistance, so diagnosis of structural nasal disease is difficult.

Nasal airway disease is predominantly in the nasal valve, which contributes 70% of upper airway resistance in adult humans. The nasal septum is only a limited contributor, yet all surgical treatment studies base conclusions on interventions overwhelmingly limited to septoplasty. Furthermore, as a secondary risk factor, measuring nasal obstruction independently of the primary risk factors introduces confounding variability. This makes it difficult to measure outcomes unless the primary risk factors also have been controlled. To our best knowledge, no studies have done this to date. Last, and most important, a patent and open nasal airway is important for sleep and sleep quality.[9,10] Using a metric such as AHI is potentially incorrect. Data show support that a patent nasal airway is a major predictor of successful medical and surgical treatment. Treating an abnormal nasal airway is important in effectively treating sleep-disordered breathing,[11] but this requires accurate diagnosis and an understanding of comprehensive treatment methods.

As noted previously in humans, progressive shortening of the nasal maxillary complex and elongation of the pharyngeal airway is a common theme in facial growth and development. A smaller maxilla narrows the retromaxillary space but also markedly reduces the volume of the nasal cavity. The abnormality in the nose may not be deviation or tissue hypertrophy but, ultimately, a predisposed small nasal cavity. Improvements in nasal obstruction after maxillary expansion have been reported.[12,13]

Nasal obstruction demonstrates greater positional dependence and increased obstruction in patients with OSA compared with normal subjects. The cause of this is unresolved but may result from structural and physiologic medical disorders, including vasomotor instability and increased inflammation. Nasal blockage may (1) reduce nasal afferent reflexes, which help maintain muscular tone of the upper airway; (2) augment the tendency for mouth opening, which destabilizes the lower pharyngeal airway (by posterior rotation, vertical opening, and inferior displacement of the hyoid); (3) reduce humidification, increase mucus viscosity, and increase surface tension forces; and (4) increase upstream airway resistance predisposing to downstream airway collapse.

Treating nasal obstruction may have a significant impact on snoring, OSA, central sleep apnea, and insomnia. Physiologically, treatment reduces airway resistance, reduces work of breathing, decreases ventilatory effort and arousal, and decreases cyclic breathing instability. Treatment varies according to pathologic findings and may include correction of septal deviations, inferior turbinate hypertrophy, nasal valve collapse, or removal of nasal polyps. Treatment of nasal obstruction improves daytime and nighttime subjective quality of life, sleep, and daytime performance. Controversy exists as to the safety of performing simultaneous nasal and other pharyngeal surgeries. Which sleep apnea surgeries are safe to pursue combined with nasal surgery has not been established. Criteria to consider include but are not limited to the following: (1) mild OSA; (2) no anticipated requirement of nasal packing that would preclude perioperative nasal CPAP; (3) no major medical comorbidity that will place the patient at risk (hypertension, coronary artery disease, or other vascular disease); and (4) appropriate and skilled postoperative monitoring and observation.

TONSILLECTOMY/ADENOIDECTOMY (ADULTS)

In adults, removal of nonhypertrophic palatine tonsils is generally ineffective as an isolated procedure for treating OSA. However, removal of nonhypertrophic tonsils is commonly done as a part of other pharyngoplasty procedures. Adults with enlarged tonsils, without other major airway abnormalities, and without profound morbid obesity may respond to tonsillectomy. Adenoidal enlargement in adults is uncommon and, if present, warrants referral for a causative etiology, viral infection, systemic disease, or neoplasm.

TONSILLECTOMY/ADENOIDECTOMY (PEDIATRICS)

Adenotonsillectomy is the treatment of choice for OSA in children. Traditionally, it has been considered highly effective but its actual effectiveness is uncertain. In pediatric patients with uncomplicated disease, a meta-analysis of level 4 evidence (case series) demonstrated that tonsillectomy and adenoidectomy reduced the AHI by an average of 13.9 events per hour and normalized AHI in approximately 80% of patients.[14] Outcomes

vary by population and are affected by airway and facial structure, obesity, nasal pathologic findings, allergies, and underlying medical conditions. Factors predictive of elevated AHI postoperatively in noncomplicated pediatric patients include enlarged inferior turbinates, deviated nasal septum, Mallampati scores of 3 and 4, and retropositioned mandibles. Persistent airway inflammatory disease has been identified in other groups and responds successfully to anti-inflammatory therapies, including topical nasal steroids and leukotriene inhibitors.[15]

Adenotonsillectomy has demonstrated significant improvements in behavior and school performance independent of final AHI levels.[16–18] Quality-of-life measures have been shown to improve despite the level of disease and can be maintained after surgery.[17,19,20]

Pediatric patients with sleep apnea may represent an at-risk population for sleep apnea independent of hypertrophic tonsils and adenoids. Associated problems include obesity and disproportionate facial growth and development. Identifying children at risk of persistent apnea and additional treatment may be critical. A family history of sleep apnea associated with findings of a high arched palate, long faces, or retrognathic mandible may warrant orthodontic assessment and treatment in addition to tonsil and adenoid surgery. Appropriate treatment of the nose also is important in children and should not be overlooked. This may include addressing hypertrophied turbinates, a deviated septum, or the maxillary foundation. Recent data highlight the importance of looking at multiple levels and structures in children with OSA.[21–23] The combination of adenotonsillectomy and orthodontic expansion may be necessary for some patients to achieve successful treatment.

PALATOPHARYNGOPLASTY

UPPP was first described by Fujita.[24] The procedure removes the distal soft palate, faucial tonsils, uvula, and redundant mucosa from the anterior and posterior tonsillar pillars. The posterior pillar is then sutured anteriorly and the mucosa approximated. Multiple variations of the technique have been described. Its effectiveness for sleep apnea is not related to symptomatic reduction in snoring, but instead is associated with increases in pharyngeal airway size in the retropalatal airway segment. UPPP is generally combined with other surgical procedures to treat other airway sites. The procedure may be contraindicated in patients with velopharyngeal insufficiency (VPI), submucous cleft palate, a nonpalatal level of obstruction, and in patients whose speech or swallowing may be at special risk. Aggressive resection of the palate with UPPP techniques has demonstrated no improvement in success, but does increase the risk of VPI. Side effects of UPPP are common and include mucosal dryness, sensation of oropharyngeal tightness or phlegm, pharyngeal dysphagia, and severe postoperative pain. Major complications are rare and include acute respiratory distress, VPI, rhinolalia, nasopharyngeal stenosis, and hemorrhage. Respiratory distress and fatality with UPPP are rare.

Controlled and randomized studies for sleep apnea surgery are exceedingly difficult to perform. However, such studies have been performed and do demonstrate UPPP is effective in treating physiologic measures of sleep and respiration, quality of life, risk of motor vehicle accidents, cardiovascular risks, and mortality.[23] The effectiveness of palate surgical procedures varies. Not all techniques are equivalent. Using the Friedman staging system, outcomes can be better stratified (Friedman stage 1 = 70% success, stage 2 = 40% success, and stage 3 = 10% success). Traditional UPPP is most useful in individuals with massive tissue redundancy of the pharynx that requires excision and enlarged tonsils. Avoidance of excessive removal of the distal soft palate is important to prevent incompetence between the soft palate and tongue and worsening possible nasal CPAP tolerance by increasing the potential for a mouth leak. Data demonstrate that failure of UPPP often is attributable to persistent obstruction at the retropalatal airway segment, not just obstruction in the lower pharynx, as has been often assumed.

Several methods of UPPP have now been described and compared. These techniques reconstruct the soft and hard tissue framework of the palate and not only modify the mucosa and tonsils. Described techniques include lateral pharyngoplasty and expansion sphincteroplasty. Lateral pharyngoplasty exposes and plicates the lateral pharyngeal wall muscles and superior constrictor proximal toward the hamulus to the free margin of the soft palate.[24,25] Expansion sphincteroplasty exposes, isolates, and divides the palatopharyngeus muscle on the pharyngeal wall and uses this muscle as a sling to advance and open the soft palate and pharynx (**Fig. 1**).[26] An additional technique to address obstruction by the soft palate is palatal advancement. Palatal advancement removes distal hard palate to advance the soft palate anteriorly and superiorly and appears to be most beneficial in certain patients with retropalatal obstruction (**Fig. 2**).[27] We have found that in some patients, these techniques can have significant effect in improving

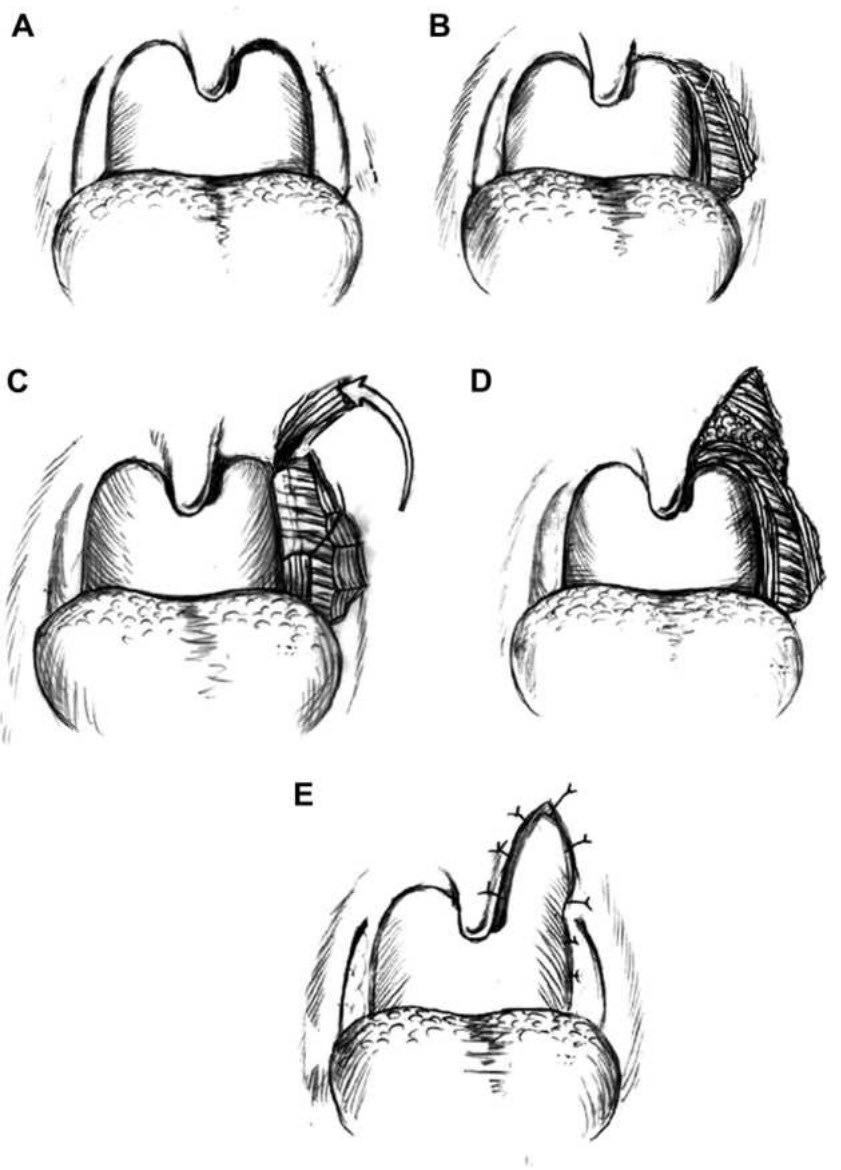

Fig. 1. Lateral pharyngoplasty is depicted. Steps for the procedure include exposure for tonsillectomy, palatopharyngoplasty (*A*). Tonsillectomy is performed preserving muscle and all mucosa (*B*). A portion of the palatopharyngeous muscle is carefully dissected from the constrictor muscle and from the medial mucosa. The muscle is elevated proximally to the approximate level of the soft palate (*C*). It is then rotated approximately 110 degrees anteriorly, laterally, and superiorly and sutured to fascia in the area of the hamulus (*D*). A dorsal palatal flap closure is shown (*E*).

the size of the ''donut hole'' without removing any tissue in some cases but rather reconstructing the tissues that are present.

SNORING

Multiple palate procedures have been advocated to treat primary snoring. Most have shown short-term effectiveness.[28] Primary snoring is not an isolated disorder and may represent a benign point in time of a progressive upper airway disease, so conservative treatments with the least side effects and complications should be considered first, if possible.[29] Snoring may be primary palatal or arise from other areas of the pharynx. Furthermore, the etiology of the snoring can be flow restriction at the same site or elsewhere in the airway. As a result, some procedures may

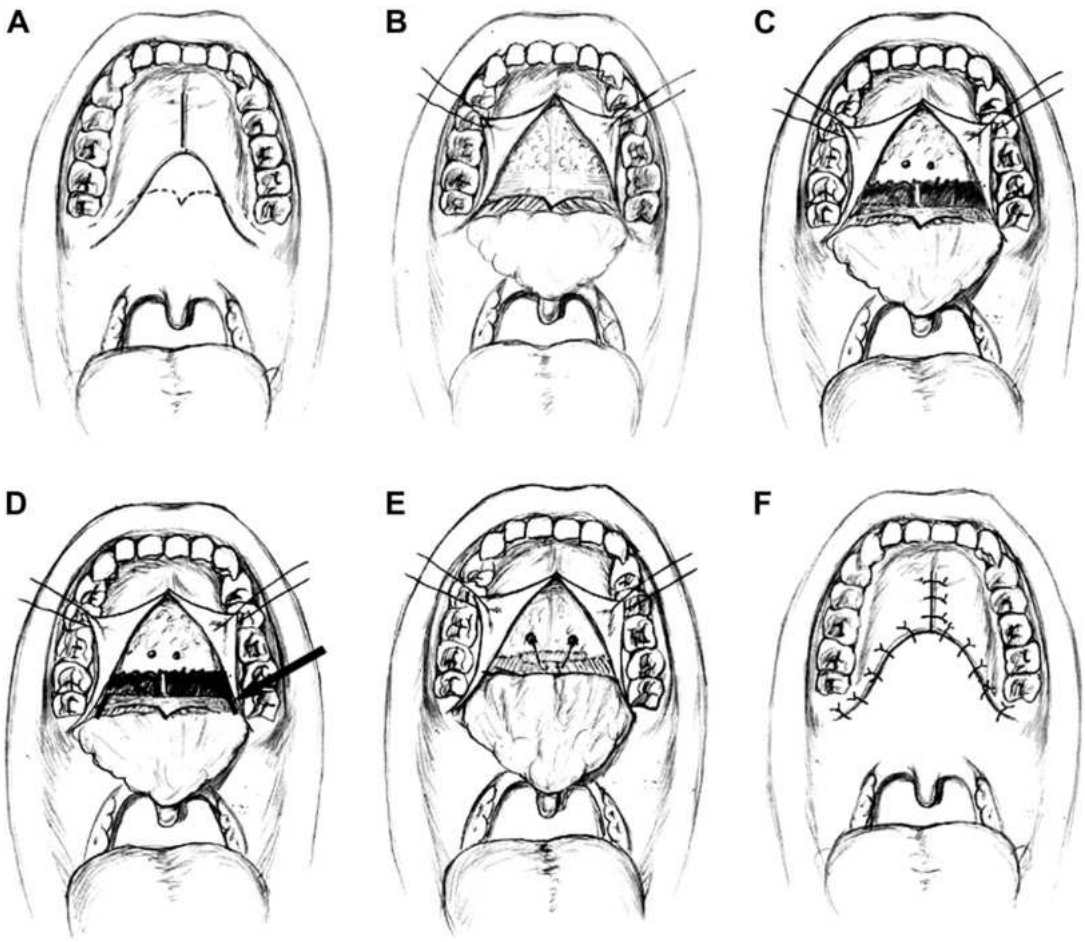

Fig. 2. Steps of palatal advancement are shown. Incisions for palatal flap are shown and allow exposure of the distal hard palate and medial to the greater palatine foramen (*A*). Two lateral flaps and a posterior flap are elevated (*B*). An osteotomy is performed and 8 to 10 mm of distal palate is removed with preservation of mucosa. More proximal through and through drill holes for later suture placement are also performed (*C*). Nasal mucosa is incised as are the tensor tendon medial to the hamulus. This allows mobilization of the soft palate (*D*). The soft palate and osteotomized segment are pulled anteriorly and attached (*E*). Flaps are closed with preservation of all mucosa. Occasionally a small amount of soft tissue debulking of the posterior flap may be helpful (*F*).

not address the source of flow restriction but may change the dynamics of tissue flutter and quality of sound emitted from the tissue.

Laser-assisted uvulopalatoplasty (LAUP) incorporates CO2 laser-enabled hemostatic surgery of the palate under local anesthesia.[30] The palate, velum, and uvula could be resected with healing by secondary intent. Two vertical trenches in the soft palate, lateral to the uvula, of variable width and length at free margin of the distal soft palate are created and the uvula reduced. Surgery may be single stage or ''titrated'' to improve snoring or the appearance of velopharyngeal dysfunction. LAUP is associated with severe pain and common complaints of pharyngeal dryness. Serious complications are infrequent. Palatal scarring initially increases tension and reduces snoring, but long-term data (5 years) suggest recurrence of snoring is common. Airway narrowing may occur and worsen sleep apnea. LAUP has not been effective for sleep apnea in clinical trials.[31] Alternatives to the use of lasers also have been described.[32,33] Various less expensive cutting and ablational tools have been used to shorten the palate, remove mucosa, and reduce the uvula and direct healing by secondary intension. All of these tools likely create scar and reduce snoring with variable effectiveness. Failure may be from persistent flow limitation, softening of scar, or flutter at nonpalatal airway sites. All

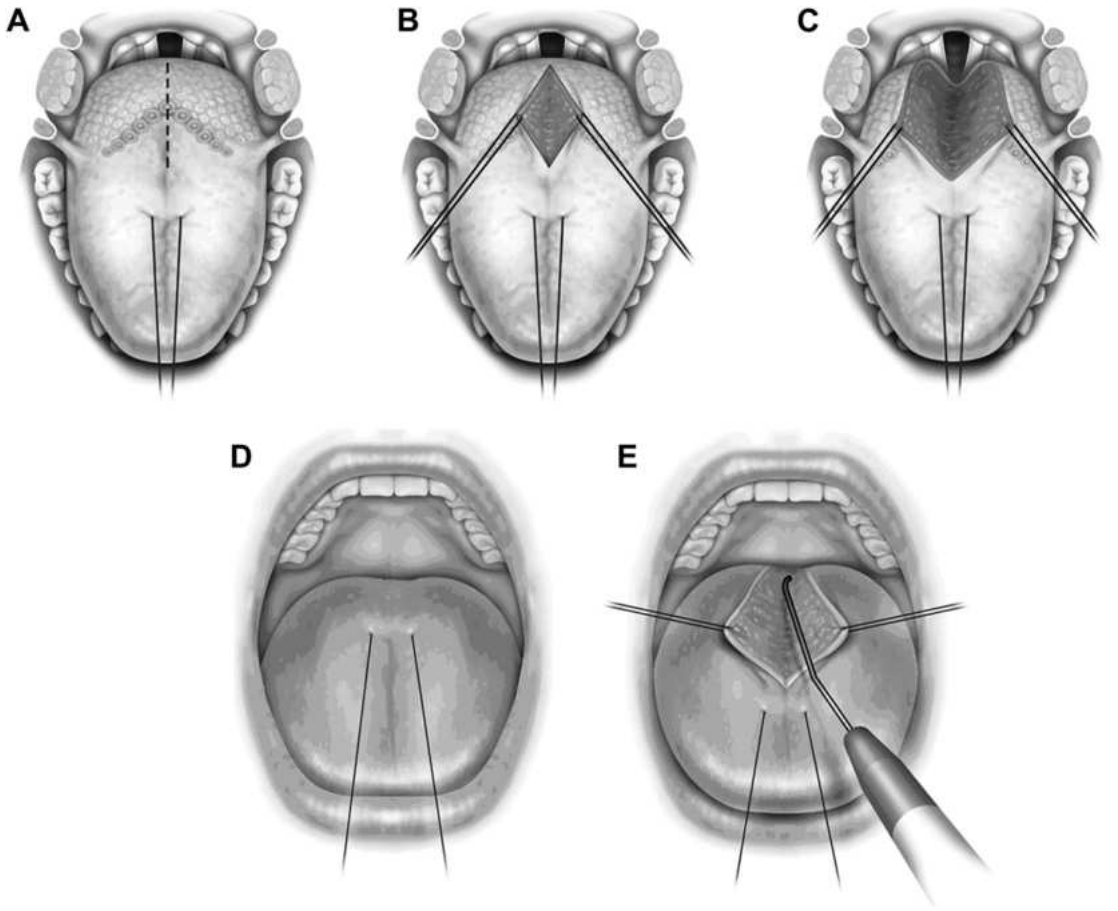

Fig. 3. A method of posterior midline glossectomy is depicted (*A–E*). A traction suture is placed, the location of midline incision is marked, and relative position of the lingual artery and the dorsum of the tongue is identified using ultrasound (*A*). The midline incision deepened and widened and carried back towards the valecula (*B* and *C*). Closure is shown (*D*).

patients, following snoring surgeries, should be cautioned about the risk of recurrence of snoring and the possible later development of overt sleep apnea.

To avoid the extensive thermal damage created to all three layers of the soft palate, as well as chronic inflammation, ulceration, and loss of seromucinous glands, alternative approaches to create palatal stiffening have been developed. Techniques using ablational radiofrequency result in less pain in the treatment of snoring compared with laser.[34] Sclerotherapy agents are used to create scar in the mucosa of the soft palate and treat primary snoring.[35] Agents are injected into the submucosa of the soft palate creating scar and tissue slough. The procedure has less pain than laser, and long-term results for snoring are better than 70%, with few major complications. Alternatively, palatal implants have been developed and demonstrate effectiveness for the treatment of primary snoring in selected populations.[36,37]

BASE OF TONGUE TECHNIQUES

Hypopharyngeal techniques include partial glossectomy, ablational glossectomy, mandibular advancement, limited mandibular osteotomies, tongue suspension,[38,39] hyoid suspension, lingual tonsillectomy, and supraglottoplasty. Some evidence-based medicine is available.[40] Ablational radiofrequency of the tongue base can be performed in the office-based setting under local anesthesia, either alone or in combination with other pharyngeal procedures. Complications, including tongue abscess, infection (cellulitis), tongue weakness, changes in speech and swallowing, and acute airway obstruction and edema, are rare. Both studies that are randomized, blinded, and controlled, as well as studies that

are uncontrolled have demonstrated effectiveness in reducing the severity and improving the disease-specific quality of life. Effectiveness also has been demonstrated in longer-term studies. These procedures are usually not used independently to definitively treat OSA but are combined with other procedures that reconstruct other segments of the upper airway.

Lingual tonsillar hypertrophy may cause or contribute to sleep apnea. If a patient's tonsils are enlarged, removal may have a marked effect on both sleep and apnea. Historically, difficult exposure and removal of tonsils in patients with sleep apnea, caused by structurally small underlying anatomy and concerns about airway edema, bleeding, and pain, made the threshold to remove these tonsils high. Only in more severe cases was it considered. Newer surgical techniques combining endoscopes and excisional radiofrequency allow easier removal of lingual tonsil tissue. This reduces the threshold of removal and provides a lower morbidity method of enlarging the hypopharyngeal airway when lingual tonsils are present.

Glossectomy may treat lower pharyngeal disease by modifying the shape and size of the soft tissue tongue base, hypopharynx, and supraglottic tissues. Multilevel procedures can also be done.[41,42] Early simple glossectomy procedures improved severe OSA, allowing tracheotomy decannulation. Nonetheless, with the advent of nasal CPAP, the morbidity of major tongue base resections relegated glossectomy to a limited population of patients who were not candidates for other procedures. Midline glossectomy and lingualplasty are partial glossectomies to enlarge the lower pharyngeal airway and treat OSA. In severe obstructive sleep apnea syndrome (OSAS), following UPPP failure, lingualplasty reduces AHI to fewer than 20 events per hour in 70% of patients.[43] Many studies predate the wide application of "evidence-based medicine." Using laser, complication rates approach 25% and include bleeding, severe odynophagia, tongue edema, and taste changes. For this reason, more aggressive glossectomy techniques have not been commonly performed. These procedures directly involve the airway in patients with preexisting airway risk, so the risk was considered high, and a perioperative tracheotomy was historically often performed, resulting in major morbidity for the procedure.

Chabolle and colleagues[44] reported hyoepiglottoplasty using a cervical approach to reduce tongue base size and to reposition the hyolingual complex. Patients were selected based on having soft tissue abnormalities of the pharynx (defined by an abnormal soft tissue hyolaryngeal complex on lateral cephalometric radiographs) and not having craniofacial abnormalities (sella-nasion-subspinale angle <79, and sella-nasion-supramentale angle <77). Glossectomy is performed under a direct vision following a suprahyoid pharyngotomy, with isolation and identification of the hypoglossal nerves and lingual arteries. Partial glossectomy is performed medial to the tonsilar folds and posterior to the circumvallate papillae. Average tongue base reduction was 24 cm^3. Following glossectomy, the hyoid and epiglottis are suspended anteriorly to the mandible. An 80% success rate was reported in 10 consecutive patients. Short-term complications were high (50%) as a result of infection and patients required tracheotomy. Both precluded widespread use. The procedure supports that effective tissue reduction can be an effective treatment. Newer technologies have been described that allow staged glossectomy using endoscopic techniques.[45] Plasma surgical excision allows glossectomy under local or general anesthesia and does not require tracheotomy. The procedure can be performed as outpatient surgery in select patients (**Fig. 3**).

Hyoid movement may stabilize the hypopharyngeal airway including the epiglottis. The hyoid may be suspended anteriorly and superiorly to the mandible or anteriorly and inferiorly to the thyroid cartilage. Hyoid myotomy may be performed with other surgeries such as UPPP or genioglossal advancement.[46,47] Hyoid myotomy is performed transcervically via a neck skin incision. Fascia lata or suture may be used for mandibular suspension. Thyrohyoid suspension has been described with suture or wire. Alternatively, hyomandibular suspension creates a vector of pull that is perhaps more favorable to repositioning the epiglottis and vallecular tongue base, expanding the hypopharyngeal airway lumen. Controlled studies are lacking.

EPIGLOTTIDECTOMY

Removal of the epiglottis has been performed with both midline glossectomy and lingualplasty. In both adults and children, removal of an abnormal epiglottis has improved OSA and upper airway obstruction.[48] Partial resection of the epiglottis with a CO2 laser has been described as a safe procedure in adults.

SUMMARY

Surgery for obstructive sleep apnea is part of a multimodal algorithm that includes medical therapy. Influences on the flow of air through the lumen of the upper airway can be from a variety of structures and different levels. Various surgical

techniques have been developed focusing on modifying either the skeletal anatomy or the soft tissue structures of the upper airway. Reconstructive airway surgery focuses on normalizing airflow while limiting morbidity and other functions of the upper airway.

Successful outcomes for any surgery can be dependent on several factors. A primary factor is understanding the intended goal of the procedure. In patients with sleep apnea, a specific surgery is rarely intended to be curative but rather ancillary to other methods of therapy. However, some procedures have been shown to independently improve quality of life as well as the acceptance of medical therapy. Additional factors that influence outcomes include choosing the correct procedure and the proper execution of the technique. Airflow in obstructive sleep apnea is influenced by multiple levels. It is important to understand that some patients may need multiple procedures to address various levels and structures. Last, in some patients obstructive sleep apnea may coincide with other disorders of sleep that can significantly affect their symptoms. Surgery should not be expected to improve excessive daytime sleepiness in patients where their apnea is "cured" but they continue to have significant sleep restriction or insomnia.

Focusing on improving airflow by reconstructing the soft tissues has led to the development of some newer techniques such as expansion sphincteroplasty, lateral pharyngoplasty, and palatal advancement. Some of these techniques are modifications of older procedures but with a new emphasis on modifying tissues to improve airflow. In some pediatric patients, more traditional procedures like adenotonsillectomy are sufficient although there are emerging data suggesting additional therapies may also be needed. Clinical outcomes assessing individual techniques in a multilevel disease where multiple interventions are used is difficult. Despite this, some reconstructive techniques of the soft tissues in the upper airway of patients with obstructive sleep apnea can be very beneficial.

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