THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION
JADA

Maxillomandibular advancement surgery for obstructive sleep apnea syndrome

JEFFREY R. PRINSELL

J Am Dent Assoc 2002;133;1489-1497

COVER STORY

Maxillomandibular advancement surgery for obstructive sleep apnea syndrome

JEFFREY R. PRINSELL, D.M.D., M.D.

ABSTRACT

Background. Although maxillomandibular advancement, or MMA, surgery is highly successful, the indications for and staging of MMA in the treatment of obstructive sleep apnea syndrome, or OSAS, have not been settled upon.

JADA CE CONTINUING EDUCATION ARTICLE 1

Types of Studies Reviewed. The author presents a retrospective review of several published case series with inclusion criteria of 20 or more patients who underwent MMA and received documented preoperative and postoperative diagnostic polysomnography. Protocols of MMA as a primary vs. secondary operation, with and without adjunctive procedures in a site-specific approach, are compared and discussed.

Results. As an extrapharyngeal operation that enlarges and stabilizes the entire velo-orohypopharyngeal airway, MMA, which can be safely combined with adjunctive nonpharyngeal procedures, may circumvent the staging dilemmas associated with multiple, less successful, segmental, invasive, pharyngeal procedures. In accordance with current goals and guidelines governing OSAS surgery, MMA does not need to be limited to severe OSAS cases as a last resort after other procedures have failed but, rather, is also indicated as an initial operation for (velo-oro)hypopharyngeal narrowing.

Conclusions. MMA is a highly successful and potentially definitive primary single-staged surgery that may result in a significant reduction in OSAS-related health risks, as well as financial savings for the health care system.

Clinical Implications. The diagnosis and management of OSAS requires a multidisciplinary team approach, including a working relationship between the dentist and sleep physician. General dentists and dental specialists who participate in the management of snoring and OSAS cases should have some knowledge of basic sleep medicine.

Obstructive sleep apnea syndrome, or OSAS,[1] is a potentially life-threatening medical disorder[2-4] that affects approximately 18 million people in the United States,[5] more commonly in males.[6] It is caused by repetitive collapse and blockage of the upper airway, or UA, usually behind the tongue base (orohypopharynx) and sometimes the soft palate (velopharynx) while asleep.[7-10] Snoring and daytime hypersomnolence are the two most common symptoms of OSAS. It also may cause memory loss, morning headaches, irritability, depression, decreased sex drive and impaired concentration. Left untreated, OSAS typically worsens progressively with advancing age, weight gain or both, and may result in hypertension, strokes, myocardial infarction, anoxic seizures and sudden death while asleep. OSAS also may result in motor vehicle accidents due to drowsiness while driving.[11,12]

Maxillomandibular advancement is a highly successful and potentially definitive primary single-staged surgery.

Comprehensive evaluation of and treatment planning for OSAS cases require a multidisciplinary team approach. Conservative treatments include positional therapy (for example, a tennis ball–like device is used to promote nonsupine sleep); a reduction in late-evening consumption of sedative-hypnotic medications or alcoholic beverages; weight loss; nasal continuous positive airway pressure, or nCPAP; and oral appliances. However, these therapies have associated compliance issues.

Although it is known that weight loss may lessen OSAS,[13] it is not known what degree of weight reduction is necessary, particularly because OSAS also occurs in

BOX 1

GOALS OF OSAS* SURGERY.†

- Safe with minimal morbidity
- Minimal pain, disfigurement and dysfunction
- Therapeutic—immediate and long-term
- Cost-effective
- Comprehensive—ideally addressing all sites of obstruction in one operation

* OSAS: Obstructive sleep apnea syndrome.
† Adapted with permission of the publisher from Prinsell.[26]

nonobese people.[14] Noncompliance is a common problem in that many patients have difficulty losing weight or in maintaining weight loss.[15] The universal convenience of nCPAP, which is generally considered the gold-standard treatment for OSAS, is that it pneumatically splints open the entire UA with a high degree of therapeutic efficacy,[16,17] thus eliminating the need to identify specific sites of obstruction. However, many patients experience difficulty tolerating a lifetime of required nightly use of continuous compressed air delivered by machine via a nasal mask.[18-22]

Oral appliances, or OA, a site-specific therapy, are quite effective for treating mild-to-moderate cases of OSAS when selected appropriately to enlarge and stabilize the "correct" area of UA obstruction (that is, orohypopharyngeal narrowing).[23,24] An adjustable (vs. fixed) OA allows controlled titration to achieve optimal mandibular advancement, within the comfort limits of temporomandibular joint laxity. When used in combination with nCPAP, OA may lower required positive airway pressures to more tolerable therapeutic levels and, thus, improve nCPAP compliance. Orally delivered positive airway pressure may circumvent the need for conventional nCPAP in selected cases of intractable nasal or velopharyngeal obstruction. A trial of OA that is therapeutic but not tolerable may, nevertheless, predict the success of mandibular advancement surgery.

BOX 2

GUIDELINES FOR OSAS* SURGERY.†

SURGICAL PREREQUISITES

- Clinically significant OSAS (AHI‡ > 15 or AI§ > 5, LSAT¶ < 90 percent and EDS#)
- Conservative treatments (such as CPAP**) not applicable, unsuccessful or intolerable
- Patient's condition medically and psychologically stable
- Patient willing to proceed with surgery (informed consent)

FOR SPECIFIC SITES/SEGMENTAL AREAS THAT ARE DISTINCTLY IDENTIFIABLE

- Treat with appropriate procedures that address these specific sites
- If a staged approach is recommended, treat the most severe site first

FOR DIFFUSELY COMPLEX OR MULTIPLE SITES THAT ARE NOT READILY DISTINGUISHABLE

- Perform skeletal advancement procedures first to enlarge and stabilize pharyngeal airway as a
  - primary single-stage definitive treatment or to
  - minimize the risk of postoperative edema-induced airway embarrassment associated with subsequent pharyngeal surgery
- Perform pharyngeal soft-tissue procedures second, if still necessary, for clinically significant residual OSAS

* OSAS: Obstructive sleep apnea syndrome.
† Adapted with permission of the publisher from Prinsell.[26]
‡ AHI: Apnea hypopnea index.
§ AI: Apnea index.
¶ LSAT: Lowest oxyhemoglobin desaturation.
\# EDS: Excessive daytime sleepiness.
** CPAP: Continuous positive airway pressure.

Surgery generally is indicated when applicable conservative therapies are unsuccessful or not well-tolerated, as well as for patients who have an identifiable underlying surgically correctable abnormality that is causing the OSAS.[25] Surgery can provide definitive treatment, thus eliminating patient compliance issues, but only if performed competently, both in terms of technical skill and on the correct site or area of UA obstruction. The goals and guidelines of OSAS surgery are listed in Box 1 and Box 2, respectively.[26]

DIAGNOSTIC MODALITIES

Polysomnography, a recording of physiological measurements during sleep, is used to establish the diagnosis of OSA, a sleeping breathing disorder characterized by repetitive obstructive apnea and hypopnea events that result in arterial oxyhemoglobin desaturations. Apnea and hypopnea are defined as a cessation and diminishment, respectively, of airflow for 10 or more seconds. The degree of severity of OSA is quantified by an apnea index, or AI, and, more commonly, by an apnea hypopnea index, or AHI.

AI is the number of apneas per hour of sleep. AHI, also referred to as the respiratory disturbance index, or RDI, is the number of apnea and hypopnea events per hour of sleep. An AHI or RDI greater than 10 is considered pathologic. The quality of sleep, in terms of the amount of rapid eye movement, or REM, sleep and the deeper stages (for example, stages 3 and 4) of non-REM sleep, may be adversely shortened by the frequent arousals associated with OSA. Polysomnography also is instrumental in diagnosing other disorders, such as central sleep apnea, whose neurogenic apnea events occur in the absence of the diaphragmatic respiratory effort that characterizes OSA.

Methodical and meticulous examination of the entire UA is needed before proceeding with site-specific surgery to identify and rank the site or sites of disproportionate anatomy, which may vary from patient to patient and contribute to OSAS. The Fujita classification of airway obstruction divides the velohypopharynx into descriptive levels:

- type I is retropalatal, posterior to the soft palate or velopharyngeal;
- type III is retrolingual, posterior to the tongue base or hypopharyngeal;
- type II is both retropalatal and retrolingual, or velohypopharyngeal.[27]

Clinical examination alone is inadequate to evaluate the UA. One must look beyond the curtain of the soft palate, the window of the velo-orohypopharyngeal inlet and the horizon of the tongue base to see the entire UA. Several reputable diagnostic imaging modalities (for example, nasopharyngolaryngoscopy, computed tomography, magnetic resonance imaging, cephalometry) are available. Each modality has advantages and disadvantages, the selection of which depends on numerous factors.

If lateral cephalometry is to be used for UA imaging, then two modifications are recommended.[28] First, because pharyngeal volume fluctuates with phases of respiration, cephalometry (Figure 1) should be standardized by obtaining films at both end-tidal volume and during a modified Mueller maneuver (that is, forced inspiration against a closed mouth and nose, to simulate the UA collapse associated with negative inspiratory forces generated during OSA events). Second, because the most critical site of hypopharyngeal closure may vary, the cephalometric posterior airway space should be measured at the most narrow level of hypopharyngeal collapse, rather than at a level determined by skeletal landmarks.[28]

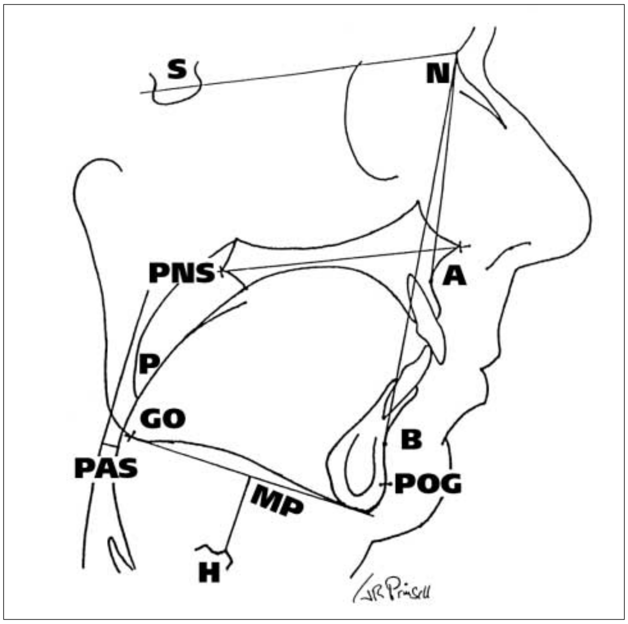

Figure 1. Lateral cephalometric analysis. SNA: Sella-nasion-A point of maxilla (mean ± standard deviation, 82 ± 2 degrees). SNB: Sella-nasion-B point of mandible (80 ± 2 degrees). PNS-P: Posterior nasal spine–soft palate tip (35 ± 3 millimeters). PAS: Posterior airway space (11 ± 1 mm). MP-H: Mandibular plane–hyoid (15 ± 2 mm). GO-POG: Gonion-pogonion (84 ± 5 mm). (Reprinted with permission of the publisher from Prinsell.[28])

SURGICAL STAGING

Although many surgical procedures and protocols have been reported,[29] most are limited to specific sites or segmental areas; consequently, they often are subtherapeutic, especially in cases involving more severe OSAS in the setting of diffusely disproportionate UA anatomy. Diagnostic dilemmas such as identifying and ranking, in terms of severity, the often multiple sites of obstruction, as well as knowing when and how to prioritize and combine surgical procedures in one or more stages, may be influenced and perhaps biased by the surgeon's education, training and

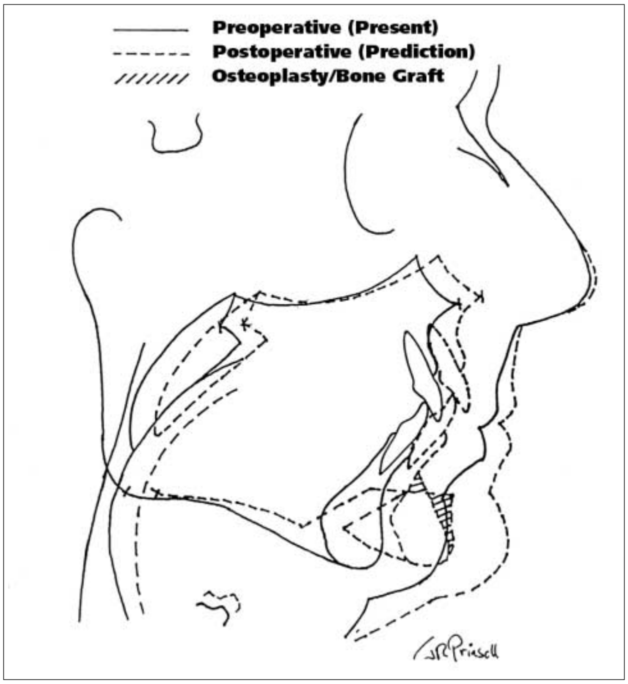

Figure 2. Lateral cephalometic prediction of maxillomandibular advancement. (Reprinted with permission of the publisher from Prinsell.[28])

experience.

One approach is to perform certain procedures in a stepwise manner according to a methodical protocol (for example, perform uvulopalatopharyngoplasty, or UPPP,[30] first ; if unsuccessful, a hyoid suspension; if that fails, a mandibular advancement). However, this may result in unnecessary additional surgery, which may be painful, dysfunctional, expensive, subtherapeutic and, ultimately, a deterrent for patients to pursue definitive surgical treatment.

A comprehensive literature review[29] showed that the overall success of UPPP, the most commonly performed OSAS surgery, was only 41 percent (137 of 337 patients from 37 independent case series), based on the criteria of postoperative AHI less than 20, AI less than 10, or a greater than 50 percent reduction in either value. Furthermore, surgery directly on the pharyngeal tissues (for example, the soft palate) may produce severe pain, hemorrhage and life-threatening airway edema in the immediate postoperative period, as well as permanent functional impairment, such as velopharyngeal insufficiency (that is, oronasal reflux of air and liquids), nasopharyngeal stenosis, voice changes and dysphagia,[29,31-34] and, in cases of snoring amelioration, may produce silent apnea.[35] Palatal scarring also may limit the amount of maxillary and obligatory (if no change is planned in the occlusal relationship) mandibular advancement and, thus, compromise the therapeutic potential of secondary jaw (telegnathic[36]) surgery.

Riley and colleagues[37] reported an overall success rate—based on a postoperative AHI of less than 20 or a greater than 50 percent reduction in AHI—of 61 percent (145 of 239 patients) and a success rate of only 42 percent (44 of 104 patients with severe OSAS) for phase I surgery, which consisted of UPPP, genioglossus advancement with hyoid myotomy suspension or both. It is disconcerting that only 26 percent (24 of 94 patients) who experienced failed phase I surgery elected to proceed with phase II of their protocol, even though maxillomandibular surgery was known to be highly successful.

MAXILLOMANDIBULAR ADVANCEMENT SURGERY

Maxillomandibular advancement, or MMA, surgery (Figures 2 and 3) pulls forward the anterior pharyngeal tissues attached to the maxilla, mandible and hyoid to structurally enlarge the entire velo-orohypopharynx, as well as to enhance the neuromuscular tone of the pharyngeal dilator musculature (for example, soft palate and tongue base) via an extrapharyngeal operation, with minimal risks of postoperative edema-induced UA embarrassment or pharyngeal dysfunction. Although the surgical techniques are similar, the goals and criteria for success differ for MMA as telegnathic surgery vs. orthognathic surgery (that is, UA opening to treat OSAS vs. dentofacial skeletal deformity correction to treat malocclusion, respectively). MMA is the most effective (excluding tracheostomy) and acceptable surgical treatment for OSAS, with success rates of 96 percent,[38] 97 percent,[39] 98 percent[37] and 100 percent.[28]

Riley and colleagues[37] reported the largest MMA series, in which 98 percent of patients with OSAS (89 of 91) were treated successfully, based on a postoperative AHI of less than 20 or a greater than 50 percent reduction in AHI. However, it is perhaps misleading that MMA was labeled a phase II procedure in that 67 (74 percent) of the 91 patients did not participate in phase I of their two-phase protocol. Waite and colleagues[38] reported improvement—based on a mean postoperative AHI of 15—in 96 percent of patients (22 of 23) in whom MMA was performed as a primary procedure, often in combination with adjunctive procedures.

Hochban and colleagues[39] reported a 97 percent success rate, based on the relatively rigid criterion of postoperative AHI of less than 10, in a series of 38 consecutive patients with OSAS in whom MMA was performed as a primary surgery, without any adjunctive procedures. In their series of carefully selected cases, which included only healthy nonobese patients with specific cranioskeletal deformities and pharyngeal narrowing, a stepwise algorithm of staged procedures was "not justified" according to the authors.

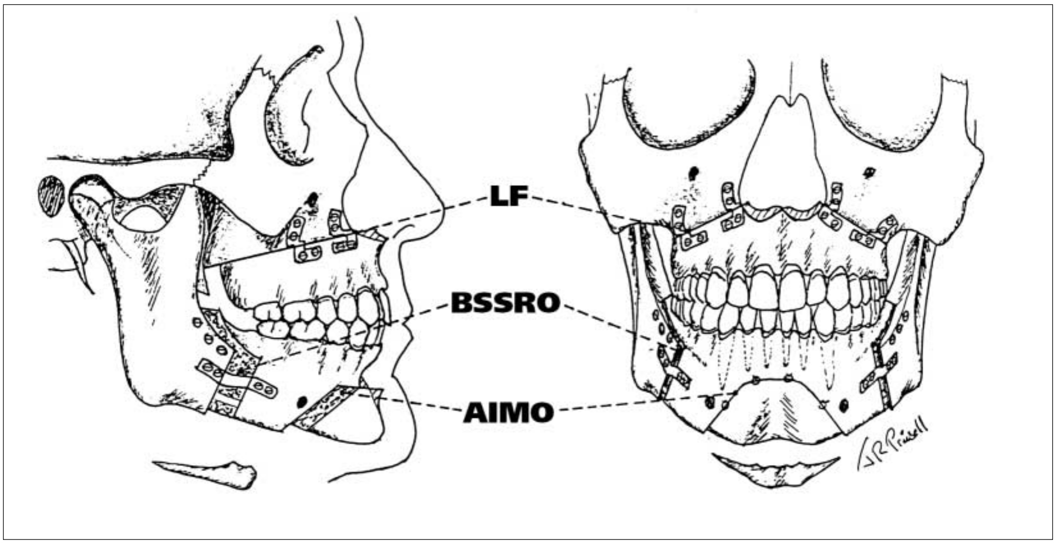

Figure 3. Maxillomandibular advancement. LF: LeFort I osteotomy with bone graft and rigid internal fixation for advancement of the maxilla with anterosuperior repositioning of the soft palate. BSSRO: Bilateral sagittal split ramus osteotomies with rigid internal fixation for mandibular advancement. AIMO: Anterior inferior mandibular osteotomy with bone graft and osteoplasty for additional advancement of the genial tubercles with attached muscles, tongue base and indirect hyoid suspension. (Reprinted with permission of the publisher from Prinsell.[28])

Prinsell[28] reported a 100 percent success rate, based on a postoperative AHI of less than 15, an AI of less than 5, or a reduction in AHI and AI of greater than 60 percent, in 50 consecutive cases in which MMA incorporated an anterior interior mandibular osteotomy and occasional concomitant nonpharyngeal adjunctive procedures as a single-stage operation. Independent polysomnography (performed and interpreted by sleep physicians rather than the surgeon) revealed a surgical therapeutic efficacy equal to that of nCPAP (Table). The mean body mass index lowered from 30.7 to 28.6, and mean blood pressure normalized from 138.9/89.9 millimeters of mercury to 123.9/80.2 mm Hg. The mean hospital stay was only 1.6 nights. No episodes of postoperative hemorrhage or edema-induced UA embarrassment occurred. Patients who experienced residual neurosensory deficits, the most common complication of MMA, reported significant resolution and their quality of life was not adversely affected. All patients reported improvement in OSAS symptoms (for example, daytime hypersomnolence) and accepted changes in facial appearance, as predicted by preoperative computer imaging, as esthetically pleasing (Figure 4).[26]

INDICATIONS FOR MAXILLOMANDIBULAR ADVANCEMENT

Box 3 lists the indications and contraindications for MMA.[26] To be considered for OSAS surgery, including MMA, the four surgical prerequisites listed in box 2 should be satisfied. The primary anatomical criterion for MMA is hypopharyngeal (that is, retrolingual) narrowing (Fujita type III airway obstruction), which can be measured by the cephalometric ETV PAS of less than 9 mm.[26] (Specific measurements for other imaging modalities as inclusion criteria for MMA have not been published.) In the setting of hypopharyngeal narrowing, coexistent velopharyngeal narrowing (Fujita type II obstruction), as well as nonpharyngeal sites (for example, septal deviation, turbinate hypertrophy, sinus polyps, mandibular lingual tori, cervicofacial lipomatosis) also may be treated with MMA. Patients with sites of obstruction in the absence of hypopharyngeal narrowing, such as sole velopharyngeal (retropalatal) narrowing (Fujita type I obstruction), should not receive MMA but may be considered for other procedures.[28]

In terms of surgical staging for selected cases of diffusely complex or multiple sites of velo-orohypopharyngeal obstruction, including coexistent soft-palatal dysmorphism and mild-to-

TABLE

POLYSOMNOGRAPHIC RESULTS.*

VARIABLE	PREOPERATIVE†	nCPAP†‡	POSTOPERATIVE‡	*P* VALUE POSTOPERATIVE VS. PREOPERATIVE	*P* VALUE POSTOPERATIVE VS. nCPAP
AI§	34.5 ± 27.9	2.0 ± 4.0	1.0 ± 1.9	.001	.078
AHI¶	59.2 ± 28.4	5.4 ± 6.8	4.7 ± 5.9	.001	.306
LSAT# (%)	72.7 ± 13.6	88.6 ± 6.3	88.6 ± 3.9	.001	.450
No. of Desaturations < 90%**	118.8 ± 160.7	2.4 ± 4.7	6.6 ± 12.2	.001	.022
% Stage 3 and 4 Sleep	6.2 ± 13.6	10.1 ± 14.1	9.0 ± 13.6	.037	.210
% REM†† Sleep	9.9 ± 7.7	23.1 ± 19.5	16.5 ± 7.4	.001	.036
Sleep Efficiency (%)	83.2 ± 11.9	83.1 ± 16.7	86.7 ± 9.9	.054	.139

* Adapted with permission of the publisher from Prinsell.[28]
† Data are mean ± standard deviation.
‡ nCPAP: Nasal continuous positive airway pressure.
§ AI: Apnea index.
¶ AHI: Apnea hypopnia index.
# LSAT: Lowest oxyhemoglobin desaturation.
** Number of oxyhemoglobin desaturation events.
‡‡ REM: Rapid eye movement.

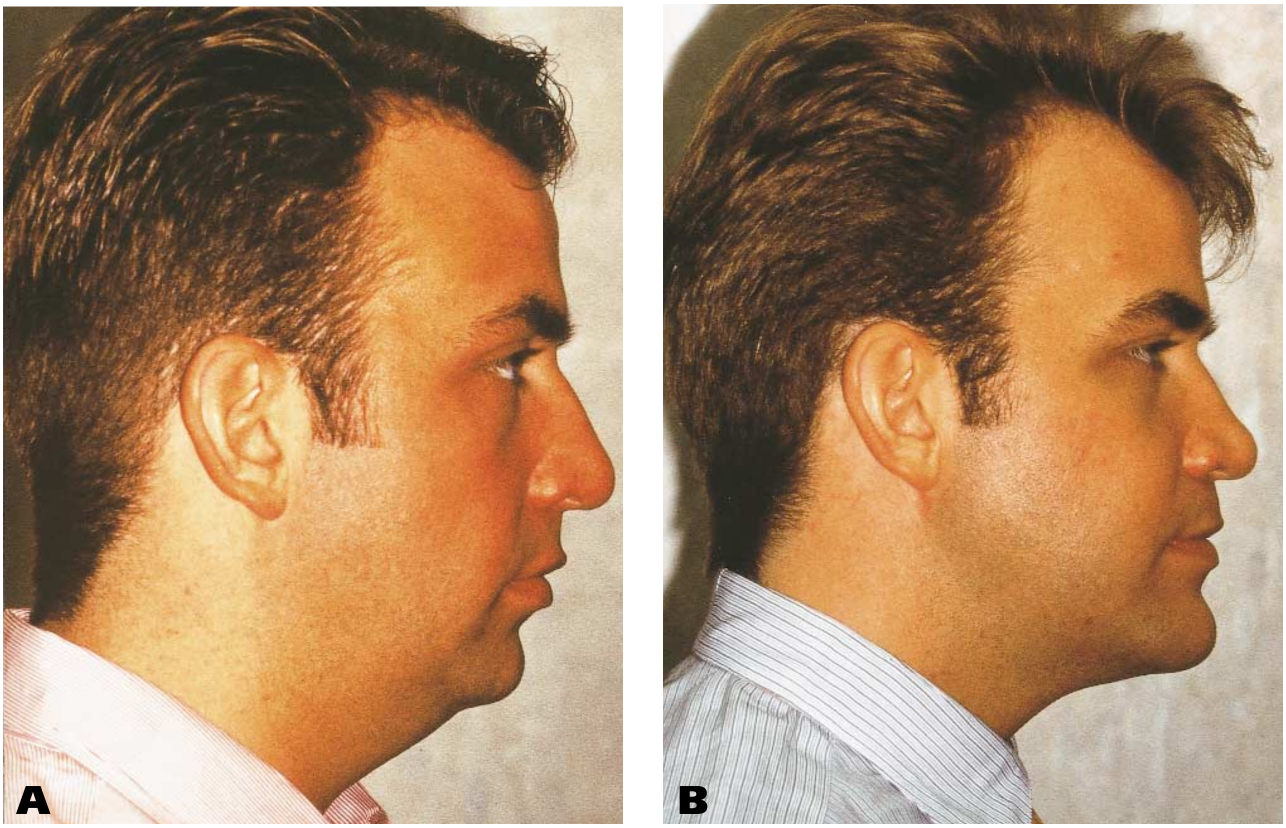

Figure 4. Preoperative (A) and postoperative (B) photographs of a patient who underwent maxillomandibular advancement surgery. (Reprinted with permission of the publisher from Prinsell.[26])

moderate tonsillar hypertrophy, MMA may be performed first. This is done to enlarge and stabilize the entire velo-orohypopharyngeal UA either to definitively treat the OSAS and obviate the need for invasive segmental pharyngeal procedures (which may be painful, dysfunctional and subtherapeutic) or to decrease the risk of postoperative edema-induced airway embarrassment after subsequent pharyngeal surgery, which may be necessary in cases of clinically significant residual OSAS (perhaps with advancing age, weight gain or both).[28]

BOX 3

MMA* SURGERY FOR OSAS.†‡

INDICATIONS

- All surgical prerequisites satisfied (see Box 2)
- Hypopharyngeal narrowing (that is, Fujita type III) (for example, cephalometric PAS§ < 9 millimeters at ETV¶)
- VOH# narrowing (that is, Fujita type II)
- May combine with adjunctive nonpharyngeal procedures (whose postoperative edema does not affect the VOH airway)
- Sometimes even in the absence of dentocraniofacial skeletal deformities (for example, retrognathia)
- Diffusely complex or multiple sites of disproportionate upper-airway anatomy that include (VO)H narrowing (for example, ETV PAS < 9 mm) as a
  - primary single-stage definitive operation or
  - stage 1 operation to enlarge and stabilize the entire VOH to minimize risk of potential edema-induced airway embarrassment associated with subsequent pharyngeal surgery, if needed for clinically significant residual OSAS

CONTRAINDICATIONS

- Not all surgical prerequisites satisfied (see Box 2)
- Absence of hypopharyngeal narrowing (for example, ETV PAS > 9 mm) (for example, sole velopharyngeal narrowing—that is, Fujita type I)

* MMA: Maxillomandibular advancement.
† OSAS: Obstructive sleep apnea syndrome.
‡ Adapted with permission of the publisher from Prinsell.[26]
§ PAS: Posterior airway space.
¶ ETV: End-tidal volume.
# VOH: Velo-orohypopharyngeal.

MAXILLOMANDIBULAR ADVANCEMENT AND ADJUNCTIVE PROCEDURES

In general, pharyngeal soft-tissue surgery (for example, tonsillectomy and adenoidectomy, UPPP, laser-assisted uvuloplasty,[35] palatopharyngoglossoplasty,[40] uvulopalatopharyngoglossoplasty,[41] somnoplasty or radiofrequency volumetric tissue reduction of the tongue[42] and/or palate,[43] cautery-assisted palatal stiffening operation,[44] uvulopalatal flap,[45] laser midline glossectomy[46] with epiglottidectomy[47] and lingualplasty[48]) probably should not be performed concomitantly with MMA unless immediate tracheostomy, prolonged endotracheal tube intubation or autotitrating nCPAP are used for several days during the resolution of the postoperative pharyngeal edema (which can be life-threatening, particularly if compounded by coexistent hypopharyngeal narrowing).[28]

Furthermore, the additional tension on pharyngeal (such as soft palatal) wound closure produced by simultaneous skeletal (that is, maxillary advancement) surgery may lead to cicatricial scarring and UA stenosis, with resultant compromised therapeutic efficacy of MMA. In contrast, adjunctive nonpharyngeal procedures (for example, septoplasty, turbinate reduction, removal of sinus polyps and mandibular lingual tori, cervicofacial lipectomy), whose postoperative edema typically does not compromise the UA, may be performed concomitantly with MMA as a safe, comprehensive, single-stage surgical treatment of OSAS.[28]

DENTAL GUIDELINES

The diagnosis and management of OSAS requires a multidisciplinary team approach, including a working relationship between the dentist and sleep physician. Because OSAS is a relatively common and potentially life-threatening medical disorder that sometimes may be treated by the dental practitioner, it is prudent that questions regarding OSAS be included on the patient's medical history form. Patients having, at a minimum, the two key symptoms of snoring with witnessed pauses while asleep, and daytime hypersomnolence, should be referred to a sleep physician, usually via the primary care physician, to rule out or diagnose OSAS.

In accordance with a clinical protocol established by the Academy of Dental Sleep Medicine,[49-51] the diagnosis is established and the degree of severity quantitated via overnight polysomnography, typically at an accredited sleep center. If significant OSAS exists, then the sleep physician may attempt treatments including weight loss, positional therapy, a reduction in late-evening alcohol consumption or in other sedative hypnotic medications, and nCPAP, as applicable.

On the other hand, patients with simple snoring or OSAS for which applicable initial therapies by the sleep physician are either unsuccessful or intolerable should be referred for site-specific therapy such as OA and surgery. The selection, fabrication, fitting and follow-up of OA therapy should be performed by qualified dentists. Similarly, the selection, based on detailed airway examination, and staging of surgical procedures should be performed by qualified surgeons (for example, oral and maxillofacial surgeons and otolaryngologists). It is essential to refer these patients back to the sleep physician for repeated polysomnography to document the therapeutic efficacy of surgery and OA therapy.[49-51] General dentists and dental specialists who participate in the management of snoring and OSAS cases should have some knowledge of basic sleep medicine.

CONCLUSIONS

MMA satisfies the goals of surgery (Box 1) and is the most successful (excluding tracheostomy) acceptable surgical treatment for OSAS, with a therapeutic efficacy equal to that of nCPAP. As a comprehensive, safe, nondysfunctional, extrapharyngeal operation that structurally enlarges and physiologically stabilizes the entire velo-orohypopharyngeal UA, MMA may eliminate the need for—and thus circumvent the staging dilemmas associated with—multiple, segmental, less successful and invasive pharyngeal procedures. In accordance with the guidelines for OSAS surgery (Box 2) in which a site-specific approach is advocated, MMA should not be limited to cases of severe OSAS or dentocraniofacial skeletal deformities or when other surgeries have failed, but rather is also indicated as the initial surgical treatment of choice for (velo-oro)hypopharyngeal narrowing, even in the setting of relatively mild OSAS and in the absence of retrognathism.[28]

MMA as a potentially definitive primary single-stage surgical treatment of OSAS, particularly when performed in a relatively young adult population, may result in a significant reduction in OSAS-related health risks (for example, hypertension, cardiovascular dysrhythmias, stroke and myocardial infarction, as well as hypersomnolence-induced injuries such as those caused by motor vehicle accidents, and neuropsychiatric disorders such as depression and cognitive dysfunction) that, when projected over an average normal lifetime, should result in considerable financial savings for the health care system.[52-54]

Nevertheless, MMA should not be used indiscriminately, as it is technically difficult to perform and laden with potential morbidity. Although skeletal osteotomies, when healed to a bony union, are stable with no significant relapse, and the relatively long-term results of MMA are promising,[28,55] the effect of expected continued normal aging (that is, laxity) of the rejuvenated pharyngeal soft tissues on UA patency during sleep in regard to possible progessive worsening of residual OSAS (with or without associated weight gain) is unknown. Additional studies are warranted, with larger numbers of cases and longer follow-up periods. ■

Dr. Prinsell is in private practice as an oral and maxillofacial surgeon, on the faculty of the Atlanta School of Sleep Medicine and a consultant with several Atlanta-area sleep centers. Address reprint requests to Dr. Prinsell at 1950 Spectrum Circle, Suite B-300, Marietta, Ga. 30067, e-mail "drprinsell@mindspring.com".

1. Guilleminault C, Partinen M, eds. Obstructive sleep apnea syndrome: Clinical research and treatment. New York: Raven Press; 1990:xv-xvii.
2. Guilleminault C. Natural history, cardiac impact, and long-term follow-up of sleep apnea syndrome. In: Guilleminault C, Lugaresi E, eds. Sleep/wake disorders: Natural history, epidemiology, and long-term evolution. New York: Raven Press; 1983:107-25.
3. Partinen M, Jamieson A, Guilleminault C. Long-term outcome for obstructive sleep apnea syndrome patients: mortality. Chest 1988;94:1200-4.
4. He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea: experience in 385 male patients. Chest 1988;94(1):9-14.
5. National Commission on Sleep Disorders Research. A report of the National Commission on Sleep Disorders Research: Wake up America—A national sleep alert. Vol. 2. Washington: National Commission on Sleep Disorders; 1995:10.
6. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med 1993;328:1230-5.
7. Kuna ST, Sant'Ambrogio G. Pathophysiology of upper airway closure during sleep. JAMA 1991;266:1384-9.
8. Sullivan CE, Grunstein RR, Marrone O, et al. Sleep apnea—pathophysiology: upper airway and control of breathing. In: Guilleminault C, Partinen M, eds. Obstructive sleep apnea syndrome: Clinical research and treatment. New York: Raven Press; 1990:49-69.
9. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. J Appl Physiol 1978;44:931-8.
10. Tangel DJ, Mezzanotte WS, White DP. Influence of sleep on tensor palatini EMG and upper airway resistance in normal men. J Appl Physiol 1991;70:2574-81.
11. Wu H, Yan-Go F. Self-reported automobile accidents involving patients with obstructive sleep apnea. Neurology 1996;46:1254-7.

12. George CF, Smiley A. Sleep apnea and automobile crashes. Sleep 1999;22:790-5.
13. Smith PL, Gold AR, Meyers DA, Hanonick EF, Bleecker ER. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. Ann Intern Med 1985;103:850-5.
14. Rubinstein I, Colapinto N, Rotstein LE, Brown IG, Hoffstein V. Improvement in upper airway function after weight loss in patients with obstructive sleep apnea. Am Rev Respir Dis 1988;138:1192-5.
15. Johnson D, Drenick EJ. Therapeutic fasting in morbid obesity. Arch Intern Med 1977;137:1381-2.
16. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. Lancet 1981;18:862-5.
17. Sanders MH, Moore SE, Eveslage J. CPAP via nasal mask: a treatment for occlusive sleep apnea. Chest 1983;83(1):144-5.
18. Nino-Murcia G, McCann CC, Bliwise DL, Guilleminault C, Dement WC. Compliance and side effects in sleep apnea patients treated with nasal continuous positive airway pressure. West J Med 1989;150(2):165-9.
19. Waldhorn RE, Herrick TW, Nguyen MC, O'Donnell AE, Sodero J, Potolicchio SJ. Long-term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. Chest 1990;97(1):33-8.
20. Kribbs NB, Redline S, Smith PL, et al. Obstructive monitoring of nasal CPAP usage in OSAS patients. Sleep Res 1991;20:270-1.
21. Kribbs NB, Pack AI, Kline LR, et al. Obstructive measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. Am Rev Respir Dis 1993;147:887-95.
22. Reeves-Hoche MK, Meck R, Zwillich CW. Nasal CPAP: an objective evaluation of patients compliance. Am J Respir Crit Care Med 1994;149(1):149-54.
23. Schmidt-Nowara W, Lowe A, Weigand L, Cartwright R, Perez-Guerra F, Menn S. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. Sleep 1995;18:501-10.
24. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. American Sleep Disorders Association. Sleep 1995;18:511-3.
25. Practice parameters for the treatment of obstructive sleep apnea in adults: the efficacy of surgical modifications of the upper airway. Report of the American Sleep Disorders Association. Sleep 1996;19(2):152-5.
26. Prinsell JR. Maxillomandibular advancement (MMA) in a site-specific treatment approach for obstructive sleep apnea: a surgical algorithm. Sleep Breath 2000;4(4):147-54.
27. Fujita S. Midline laser glossectomy with linguoplasty: a treatment of sleep apnea syndrome. Op Tech Otolaryngol HNS 1991;2: 127-31.
28. Prinsell JR. Maxillomandibular advancement surgery in a site-specific treatment approach for obstructive sleep apnea in 50 consecutive patients. Chest 1999;116(6):1519-29.
29. Sher AE, Schechtman KB, Piccirillo JF. The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. Sleep 1996;19(2):156-77.
30. Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg 1981;89:923-34.
31. Fairbanks DN. Uvulopalatopharyngoplasty complications and avoidance strategies. Otolaryngol Head Neck Surg 1990;102:239-45.
32. Haavisto L, Suonpaa J. Complications of uvulopalatopharyngoplasty. Clin Otolaryngol 1994;19:243-7.
33. Zohar Y, Finkelstein Y, Talmi YP, Bar-Ilan Y. Uvulopalatopharyngoplasty: evaluation of postoperative complications, sequelae and results. Laryngoscope 1991;101:775-9.
34. Esclamado RM, Glenn MG, McCulloch TM, Cummings CW. Perioperative complications and risk factors in the surgical treatment of obstructive sleep apnea syndrome. Laryngoscope 1989;99:1125-9.
35. Practice parameters for use of laser-assisted uvulopalatoplasty. Standards of Practice Committee of the American Sleep Disorders Association. Sleep 1994;11:744-8.
36. Moore K. Site-specific versus diffuse treatment/presenting severity of obstructive sleep apnea. Sleep Breath 2000;4(4):145-6.
37. Riley RW, Powell NB, Guilleminalt C. Obstructive sleep apnea syndrome: a review of 306 consecutively treated surgical patients. Otolaryngol Head Neck Surg 1993;108(2):117-25.
38. Waite PD, Wooten V, Lachner J, Guyette RF. Maxillomandibular advancement surgery in 23 patients with obstructive sleep apnea syndrome. J Oral Maxillofac Surg 1989;47:1256-61.
39. Hochban W, Conradt R, Bradenburg U, Heitmann J, Peter JH. Surgical maxillofacial treatment of obstructive sleep apnea. Plast Reconstr Surg 1997;99:619-26.
40. Djupesland G, Schrader H, Lyberg T, Refsum H, Lilleas F, Godlibsen OB. Palatopharyngoglossoplasty in the treatment of patients with obstructive sleep apnea syndrome. Acta Orolaryngol 1992;492(supplement):50-4.
41. Miljeteig H, Tvinnereim M. Uvulopalatopharyngoglossoplasty (UPPGP) in the treatment of the obstructive sleep apnea syndrome. Acta Orolaryngol 1992;492(supplement):86-9.
42. Powell NB, Riley RW, Troell RJ, Blumen MB, Guilleminault C. Radiofrequency volumetric reduction of the tongue: a porcine pilot study for the treatment of obstructive sleep apnea syndrome. Chest 1997;111:1348-55.
43. Powell NB, Riley RW, Troell RJ, Li K, Blumen MB, Guilleminault C. Radiofrequency volumetric tissue reduction of the palate in subjects with sleep-disordered breathing. Chest 1998;113:1163-74.
44. Wassmuth Z, Leonard D, Mair E. Cautery-assisted palatal stiffening operation for the treatment of obstructive sleep apnea (abstract). Otolaryngol Head Neck Surg 1998;119:144-5.
45. Powell N, Riley R, Guilleminault C, Troell R. A reversible uvulopalatal flap for snoring and sleep apnea syndrome. Sleep 1996;19: 593-9.
46. Fujita S, Woodson BT, Clark JL, Wittig R. Laser midline glossectomy as a treatment for obstructive sleep apnea. Laryngoscope 1991;101:805-9.
47. Mickelson SA, Rosenthal L. Midline glossectomy and epiglottidectomy for obstructive sleep apnea syndrome. Laryngoscope 1997;107:614-9.
48. Woodson BT, Fujita S. Clinical experience with lingualplasty as part of the treatment of severe obstructive sleep apnea. Otolaryngol Head Neck Surg 1992;107(1):40-8.
49. Lowe A. Dental appliances for the treatment of snoring and obstructive sleep apnea. In: Kryger M, Roth T, Dement W, eds. Principles and practice of sleep medicine. 2nd ed. Philadelphia: Saunders; 1994:723.
50. Rogers R. Sleep disordered breathing, part II. In: Clarks clinical dentistry. Vol. 1. St. Louis: Mosby–Year Book; 1996:12-4.
51. Strauss A. Oral devices for the management of snoring and obstructive sleep apnea. In: Fairbanks D, Fujita S, eds. Snoring and obstructive sleep apnea. 2nd ed. Philadelphia: Raven Press; 1994:237.
52. Ronald J, Delaive K, Roos L, Manfreda J, Bahammam A, Kryger MH. Healthcare utilization in the 10 years prior to diagnosis in obstructive sleep apnea syndrome patients. Sleep 1999;22:225-9.
53. Bahammam A, Delaive K, Ronald J, Manfreda J, Roos L, Kryger MH. Healthcare utilization in males with obstructive sleep apnea syndrome two years after diagnosis and treatment. Sleep 1999;22:740-7.
54. Kapur V, Blough DK, Sandblom RE, et al. The medical cost of undiagnosed sleep apnea. Sleep 1999;22:749-55.
55. Riley RW, Powell NB, Li KK, Troell RJ, Guilleminault C. Surgery and obstructive sleep apnea: long-term clinical outcomes. Otolaryngol Head Neck Surg 2000;122:415-21.