Maxillary, Mandibular, and Chin Advancement: Treatment Planning Based on Airway Anatomy in Obstructive Sleep Apnea

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Surgical correction of obstructive sleep apnea (OSA) syndrome involves understanding a number of parameters, of which the 3-dimensional airway anatomy is important. Visualization of the upper airway based on cone beam computed tomography scans and automated computer analysis is an aid in understanding normal and abnormal airway conditions and their response to surgery. The goal of surgical treatment of OSA syndrome is to enlarge the velo-oropharyngeal airway by anterior/lateral displacement of the soft tissues and musculature by maxillary, mandibular, and possibly, genioglossus advancement. Knowledge of the specific airway obstruction and characteristics based on 3-dimensional studies permits a directed surgical treatment plan that can successfully address the area or areas of airway obstruction. The end occlusal result can be improved when orthodontic treatment is combined with the surgical plan. The individual with OSA, though, is more complicated than the usual orthognathic patient, and both the medical condition and treatment length need to be judiciously managed when OSA and associated conditions are present. The perioperative management of the patient with OSA is more complex and the margin for error is reduced, and this needs to be taken into consideration and the care altered as indicated.

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Surgical correction of obstructive sleep apnea syndrome (OSAS) involves understanding a number of parameters, of which the 3-dimensional (3D) airway anatomy is important. Recent advances in the visualization of the upper airway based on cone beam computed tomography (CBCT) scans and automated computer analysis have been a great aid in understanding normal and abnormal airway conditions and their response to surgery. Knowledge of the specific airway obstruction and its characteristics based on preoperative studies permits a precise surgical treatment plan directed at the areas of restriction.[1] The goal of surgical treatment of OSAS is to enlarge the velo-oropharyngeal airway by anterior/lateral displacement of the soft tissues and musculature by maxillary, mandibular, and possibly, genioglossus advancement (Fig 1). Treatment may also include the correction of transverse problems with expansion as part of the overall plan and other soft tissue procedures such as uvulopalatoplasty.[2] Review of the literature indicates that maxillary and mandibular advancement (MMA) is 75% to 100% successful in the correction of OSAS.[3-8] MMA may be performed as a stand alone procedure or as part of a staged treatment, which yields the highest success rates.[3,9] In the literature the usual advancement of the maxilla is at least 1 cm. Long-term cephalometric studies have shown good skeletal stability after MMA advancement in these cases.[6,7] However, anatomic airway changes with surgery have generally only been studied on lateral cephalometric radiographs, which are 2-dimensional (2D). Three-dimensional airway analyses permit a more refined and directed approach to correction of the airway in OSAS, yet there is little in the literature regarding the use of this modality. This article will cover the indications for and planning algorithm in the treatment of the anatomic airway in OSAS by MMA based on computerized 3D analyses as an integral part of the overall treatment plan, which must also address neuromuscular and medical factors.

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FIGURE 1. MMA and genioglossus advancement schema.

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To effectively develop a treatment plan, the surgeon should have a clear understanding of both the medical and surgical management used in OSAS.[10] The treatment of OSAS, or the relatively newer term "sleep-disordered breathing" (SDB), which includes snoring and upper airway resistance syndrome, presently is accomplished by medical and/or surgical management. Neither of these 2 modalities is always successful, but each has its place in the management of these conditions. Primary medical management of OSAS via continuous positive airway pressure (CPAP) is the main treatment modality for airway collapse.[11-14] The partially or totally obstructed upper airway in OSAS/upper airway resistance syndrome is primarily an anatomic problem secondarily influenced by neuromuscular influences and, therefore, a surgical problem.

There are 3 defined general anatomic levels (nose, palate, and base of tongue) in the upper airway to evaluate. Each may be partially or totally blocked in patients with OSAS. The surgeon's goal is to enlarge the airway by repositioning or removing the obstruction at each respective level without creating a complication. It is more difficult to treat the more distal levels of the airway from the nose to the larynx. This is due, in part, to the increasing amount of soft tissue encountered (nose vs tongue). Furthermore, the sleep patient is far more difficult to treat than is usually anticipated because the problem is frequently multifactorial and it is easy to overestimate the ability to control all the variables. A few examples are factors such as weight, age, gender, body mass index (in kilograms per square meter), skeletal and soft tissue anatomy, excessive daytime sleepiness (EDS), and the patient's subjective response. Therefore it is prudent to have a broad approach to the treatment, where all levels found to be obstructed are addressed. Failure or incomplete treatment will result if these issues are not appreciated, because OSAS is known to involve diffuse upper airway collapse during sleep and, therefore, seldom will treatment of a single site be curative.

There are 2 major rationales that should be well understood in the surgical management of OSAS. The first, "behavioral derangement," is due to EDS. EDS is the result of nocturnal arousals during sleep due to airway collapse and usually manifests as sleepiness and/or fatigue to such a degree that the subject lacks vigilance and does not function normally during the day. Symptoms may include snoring, apneas, morning headaches, fatigue, sleepiness after lunch, memory loss, irritability, poor work performance, altered family relationships, and in some cases alterations in libido. These symptoms may be minimal, where the patient denies sleepiness, or severe, to the point that the subject falls asleep driving and may cause a catastrophic accident.[12] When this level of sleepiness is experienced, concentration and vigilance are impaired and may result in dangerous pathologic sleepiness. Regardless of the number of these symptoms, there is not necessarily a direct correlation with the severity as seen on the apnea-hypopnea index (AHI).

The second rationale is "pathophysiologic derangement," which is, in part, cardiorespiratory in nature. It is well known that at some level of OSAS severity, there is an increased risk for myocardial infarction, stroke, and sudden death.[13] Three important well-known physiologic processes are involved in OSAS that predispose to these risks: hypoxemia, negative intrathoracic pressure, and disequilibrium of the autonomic nervous system.[14-17]

The indications for surgery are as follows:

- EDS
- Respiratory disturbance index (RDI) of greater than 20 episodes per hour of sleep (or in cases where the RDI is <20 associated with marked objective EDS)
- Oxygen desaturation of less than 90%
- Hypertension and/or arrhythmia
- Negative esophageal pressures more negative than negative 10-cm H_2O
- Anatomic abnormalities of the upper airway
- Failure of medical management (one of the most common rationales but not an acceptable metric by itself)

These indications are similar to those for medical management with CPAP, which is not curative and frequently not tolerated.

Patient Evaluation

The patient's history and complaints are generally associated with snoring and/or EDS (behavioral derangement) and rarely do they mention respiratory or cardiac pathology as their chief complaint. Therefore it is imperative that a basic understanding of this metric exist when one is evaluating such patients. This is especially true in light of the fact there are multiple other causes for the behavioral derangement in EDS other than OSAS. Some examples are volitional sleep deprivation, alcoholism, insomnia, and narcolepsy. Furthermore, OSAS does not develop in all snorers. However, as intermittent snoring turns to habitual snoring, the airway progressively collapses, and as a rule of thumb, if the bed partner frequently leaves the bedroom, there is about an 80% chance the patient will have some degree of OSAS.

Preoperative Evaluation

GENERAL WORKUP

A thorough medical and sleep history should be taken regardless of age, and it is recommended in adults that a subjective questionnaire such as the Epworth Sleepiness Scale be used.[18] The Epworth Sleepiness Scale assesses the propensity of sleep in 8 situations. It is not perfect and does not always correlate with OSAS severity but does give some information regarding the patient's waking sleepiness.

A clinical examination of the head and neck along with a 3D CBCT scan, followed by fiberoptic nasopharyngoscopy and polysomnography (PSG), is recommended. Careful evaluation of the following is suggested: nasal airway obstruction, the palatal region to include the lateral pharyngeal walls, the size and character of the tonsils (if present), oral soft tissues, malocclusion or other skeletal abnormities, and tongue and tongue base. The clinical examination is then correlated with the radiographic study and fiberoptic nasopharyngoscopy, along with data from an objective overnight PSG scan and the subjective symptoms of the patient.

The airway can be divided into 3 anatomic sections for evaluation and thus treatment of SDB; *1*) nose, *2*) retropalatal, and *3*) retroglossal (Fig 2). Airway obstruction may occur in any 1 or more of these areas simultaneously; thus a thorough knowledge of the anatomy in each area and its influence on the airway is necessary. The airway description in this chapter will deal with the internal anatomy and supporting structures.[1]

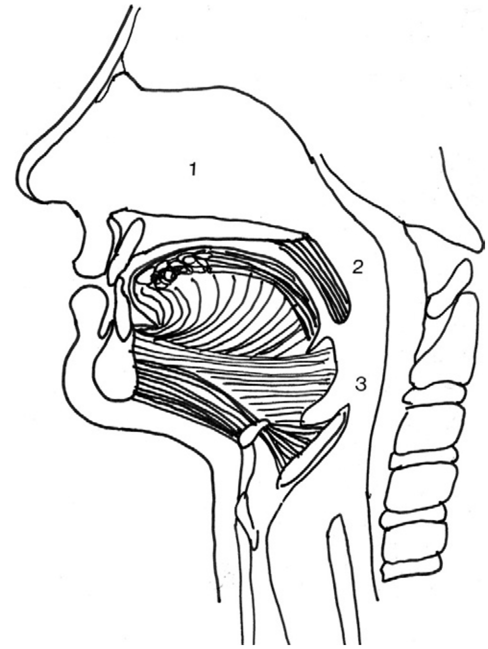

FIGURE 2. Lateral schema showing the 3 general areas of airway obstruction that may occur alone or, more frequently, in any combination: 1, nasal area; 2, retropalatal area; and 3, retroglossal area.

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The nose prepares the air for the lower respiratory tract by warming and humidifying it. Any deformity of the internal nose will affect airflow, and thus the surgeon and orthodontist should be concerned with nasal aerodynamics. The nares act as a funnel guiding the airstream toward the 2 valve areas, which is generally the narrowest area of the nose. Deformities or collapse of the alar cartilages will cause airflow reduction at the external nasal valve. The angle between the caudal end of the upper lateral cartilages and the septum is called the internal nasal valve. This angle is normally between 10° and 15°. The total nasal area at this point has been estimated at between 55 and 64 mm^2 and is normally the smallest part of the airway. Any stricture or stenosis here can cause restricted airflow or turbulence. Airflow symptoms at this level can be evaluated by the Cottle test. While the patient breathes quietly, the cheek is retracted laterally, thus opening the nasal valves; if the Cottle test improves the breathing, the result is considered positive and indicates a valve problem. Septal deviations also obstruct the nose in addition to causing airflow turbulence and secondary problems such as dryness and bleeding. The turbinates, of which the inferior is the most import with regard to airflow dynamics, are in a constant cycle. One side is congested whereas the other is decongested; however, the total nasal resistance remains relatively constant. The turbinates rapidly heat the air from 0°C to 36°C and humidify it. Enlarged inferior turbinates can obstruct the nose and greatly reduce airflow, and thus surgical reduction may be indicated. The overall width of the face and especially the maxilla also influences the nasal cavity, as the floor of the nose is the hard palate. Maxillary constriction seen with cross-bite–type malocclusions and low tongue positions will thus decrease the nasal width, negatively affecting the airflow.

Area 2 in Figure 2 is the retropalatal area. This consists of the space behind the soft palate and anterior to the posterior wall of the pharynx. Airflow dynamics here are influenced by the position of the maxilla, soft palate, and adenotonsillar structures. A retruded maxilla will decrease the available space behind the hard palate and thus the airway. This is most clearly seen in cases of midfacial retrusion such as Apert-Crouzon syndrome, and clefting but can also be found in ordinary maxillary retrusion. The length and thickness of the soft palate are also important, and either a very thick or long palate will cause obstruction. Airway problems can be seen after cleft palate surgery, especially after pharyngeal flap or sphincter-type procedures. In the adult with chronic SDB and snoring, the palate thickens, elongates, and descends, further compounding the airway problem. In adolescents, however, the most common condition is lymphoid hyperplasia with enlargement of the adenoids and tonsils. This reduces the retropalatal airway, causing obstructive sleep apnea (OSA), and is stated to be the cause of airway problems 60% of the time in this group.

Area 3, is the retroglossal region, which is most influenced by the position of the mandible and the tongue. Retrusion of the mandible carries the base of the tongue posteriorly, and with relaxation during sleep, the tongue may drop further back, obstructing the airway and leading to apneic or hypopneic spells. Posterior tongue position can also influence palatal position causing secondary obstruction at the palatal level also. Some idea of the tongue position can be gained from a lateral cephalometric radiograph; however, more complete visualization of the tongue and the airway can be gained from 3D imaging as seen on CBCT examinations and nasopharyngoscopy. The size of the tonsils also contributes greatly to airway size at this level, mainly in the transverse width.

POLYSOMNOGRAPHY

The PSG study is an essential part of the surgical workup and must be done before and after surgery; otherwise, the patient's PSG parameters and the surgeon's personal success rate will not be appreciated. Some of the parameters to insist on from an attended PSG study are as follows: name; age; body mass index; total sleep time of at least 240 minutes for a valid study[16]; sleep stages NREM and REM, as well as percent of sleep in each; apnea index (mean and maximum duration in seconds); hypopnea index (mean and maximum duration in seconds); awake SaO_2; lowest SaO_2; stratifications of the percent of time SaO_2 were below 90%, 80%, 70%, and so on; heart rate fluctuations (bradyarrhythmia/tachyarrhythmias); and periodic leg movements. All of these parameters may be reviewed in most any text on sleep medicine.[14] The severity as an index is summarized as the respiratory disturbance index (RDI) or, more frequently now, the apnea hypopnea index (AHI). An AHI of 5 or less is considered normal for an adult. The surgeon should be aware that the polysomnogram (PSG) study should be current (6-12 months) and, if possible, not a split-night study (one-half is diagnostic and one-half is therapeutic). Split-night studies apply continuous positive airway pressure (CPAP) for the second half of the night and may underestimate the severity of obstructive sleep apnea syndrome (OSAS). This finding is common because the total sleep time does not meet diagnostic criteria. For medical and ethical reasons, a split-night study is mandated in certain cases, especially for patients who are hypoxemic, have arrhythmias, or exhibit hypertension during the study. It is again suggested that all sleep studies for a surgical patient comprise attended overnight PSG studies. Home monitoring is acceptable in some cases but is not suggested for surgical evaluation because a majority of these types of studies lack detailed information on actual sleep parameters.

RADIOGRAPHIC STUDIES

One should avoid trying to make a diagnosis or treatment plan by using a cephalometric head film because it is only a small part of the workup and is 2D in nature. Measurements that have been commonly used for evaluating soft tissue and bony anatomy in OSAS include SNA, SNB, PNS-P, PAS, and mandibular plane to hyoid (MP-H); however, their correlation to the airway and subsequent treatment results are questionable.[19] A CBCT study should be obtained, and an airway analysis should be done. The smallest airway cross section is easily seen and has been correlated to the extent of the OSA. On the basis of this, the surgical treatment must primarily address the indicated area or areas.[19-24]

Radiation dose from CBCT scans is significantly less than other computed tomographic imaging methods such as medical computed tomography (CT) and is within the range of traditional dental imaging methods. The superior anatomic information available from 3D imaging is establishing CBCT as the preferred imaging modality for airway evaluation in the adolescent and adult populations. Medical CT imaging is best for infants and young children with SDB. As a result of 3D imaging, there is an increased awareness among practitioners regarding the relationship between craniofacial structures, airway anatomy, and obstructed SDB. Other applications using 3D imaging include evaluation of the changes in the airway resulting from surgery on the oral soft tissues and the jaws.

The accuracy of predicting airway space changes from lateral cephalometric radiographs is ± 1.5 mm. In a study by Muto et al,[19] a set-back of the mandible of 1 cm showed a 0.4-mm decrease of the airway in the anteroposterior dimension only. The usefulness of these cephalometric studies is also limited because data are obtained in only 2 dimensions. CT is superior, in terms of both accuracy and the ability to measure in 3 dimensions. Airway space measurement has been shown to be quite accurate by use of CBCT scans.[20] Multivariate analysis shows both retroglossal space (P = .027) and retropalatal space (P = .0036) to be predictive of RDI. Li et al[21] have also shown a relationship between the airway area and the likelihood of OSA. There is a high probability of severe OSA with an airway area of less the 52 mm^2, an intermediate probability if the airway is between 52 and 110 mm^2, and a low probability if the airway is greater than 110 mm^2. Lowe et al[22] showed that a majority of the constrictions occur in the oropharynx, with a mean airway volume of 13.89 ± 5.33 cm^3. Barkdull et al[24] showed a correlation between the retrolingual cross-sectional airway and OSA when this area was less than 4% of the cross-sectional area of the cervicomandibular ring. Schendel and Hatcher[25] have shown that measurement of the 3D airway using a semi-assisted software program (Vultus, Atlanta, GA) from CBCT data is as accurate as manual segmentation, reliable, and fast. The difference in measured volume of a phantom-filled cone of known volume was ± 0.15 mL. The Vultus program easily identified the smallest airway area and the largest area while analyzing the airway in successive slices. In addition, we have verified the intraexaminer reliability of this system to a mean of 0.26 cm^3 out of a total airway volume of 19.5 cm^3. Hakan and Palomo[26] have shown that other Digital Imaging and Communications in Medicine viewing systems such as Dolphim3d (Dolphin Imaging, Chatsworth, CA) are less accurate. Imaging of the upper airway by use of CBCT data is thus valuable in identifying the exact location and nature of the obstruction in OSA together with the other parameters, helping to refine the surgical approach. Incorporation of this into daily practice will allow practitioners to readily evaluate and screen their patients for anatomic related obstructed SDB (Fig 6). This is especially important in the adolescent population, where many already seek orthodontic treatment for dentofacial deformities associated with obstructed SDB and where radiographs are already routinely obtained.

FIBEROPTIC EXAMINATION

The use of nasopharyngolaryngoscopy can further help to identify regions (nose, retropalatal area, and tongue base) in the airway that may be a part of nocturnal obstruction. In addition, it is useful to rule out other causes of upper airway obstruction such as tumors, cysts, lingual tonsils, and laryngeal pathology.

PREOPERATIVE MANAGEMENT

Regardless of age, all subjects should have an appropriate medical workup because many of these patients have increased medical, anesthesia, and surgical risks. In addition, it is imperative that the patient's health status be maximized to the extent possible before surgery. The patient's overall nutritional status, including biochemical analyses, should be assessed because many of these individuals have reduced regenerative capacity due to the long-standing illness and associated medical conditions. These conditions can also cause skeletal atrophy, trabecular bone loss, and root resorption during treatment.

One of the most important issues in all OSAS patients who undergo surgery is the preoperative and postoperative airway. Clinical assessments, including all studies, should be used to decide whether a tracheotomy should be performed to protect the airway perioperatively. Should you have any concern about the airway, a tracheotomy is usually indicated. It is recommended that it not be done at the same time as a maxillomandibular advancement (MMA) procedure for 2 reasons: first, the patient's initial problem is the airway, which is already compromised, and second, combining a tracheotomy with airway surgery will significantly increase the risk of infection.

Airway, Airflow, and Surgery

Common nasal surgical procedures include septoplasty and inferior turbinate reduction by resection or radiofrequency. Airway collapse and a decreased internal nasal angle are treated by spreader grafts or other rhinoplasty techniques to expand and stabilize the cartilaginous structures of the nose. These changes can easily be seen on a nasal examination with a speculum or nasal endoscope but are difficult to quantify without using rhinometry. Subjective changes, though, are readily noticed by the patient. Studies of these procedures have mainly looked at the change on PSG with occasional lateral cephalometric support. Sophisticated 3D airway studies are absent except for that of Fairburn et al.[27] Maxillomandibular surgery is performed when there is clinical and radiographic evidence of jaw retrusion in adolescents or adults. In addition, MMA surgery is performed in those individuals who may have a normal dentofacial skeleton but who have not completely responded to the other surgical treatments and cannot tolerate CPAP. The rule of thumb here has been an advancement of 1 cm. Studies show generally good results as previously stated; however, these have been mainly based on different criteria for cure based mainly on PSG, and 3D airway studies are generally few.[28-32] Two-dimensional anatomic airway changes on lateral cephalometric films in individuals undergoing bimaxillary advancement show pharyngeal depth increases in the magnitude of 48% of the maxillary advancement (Li et al[21]). In addition, these studies show anatomic differences between individuals with OSA and normal subjects. However, these studies are limited because of their 2D basis.[20,33-38] Three-dimensional studies of airway changes after bimaxillary surgery are also limited but do show that the surgically induced change is not only in the anteroposterior dimension: important changes also occur in the lateral dimension. Fairburn et al examined 20 patients with OSA who underwent bimaxillary advancement. The patients underwent preoperative and postoperative PSG and CT scans. The authors found that bimaxillary advancement enlarges the entire velopharynx by elevating the tissues attached to the jaws and hyoid. Airways with a decreased ratio of lateral airway dimension to anteroposterior dimension are more prone to collapse.

Although the airway and airflow are coupled, very little attention has been given to the role of airflow in studies of OSAS. The dynamics of airflow in the pharyngeal airway may play an important role in understanding etiologic factors of airway collapsibility in OSAS. An ongoing study to assess the characteristics of airflow in OSAS is being undertaken by Stanford University Sleep Center (Palo Alto, CA) and the Department of Aerospace Engineering, University of Cincinnati (Cincinnati, OH). The study focuses on functional 3D CT imaging and computational fluid dynamics (CFD) modeling using Reynolds-averaged Navier-Stokes equations with a k-ω turbulence model.[39] It should be noted that CFD modeling is used in many different fields but has only recently been applied for studying the flow associated with OSAS.[40-42]

As a result of this approach, a virtual 3D airway model and the corresponding airflow through the airway passages can be extracted. The modeling approach is used to quantify and compare the flow variables in the pharyngeal airway before and after medical or surgical treatment. The specific flow variables calculated include axial velocity, wall static pressure, and wall shear stress.

These flow variables could be associated with periodic events of airway collapsibility. As the airway narrows during sleep, the airway lumen becomes smaller and axial velocity increases at the minimum cross-sectional area. Static pressures decrease at the obstruction, and downstream flow separation causes an abnormal increase in airflow turbulence levels. Further downstream, this separated flow may impinge on the pharyngeal airway walls, generating large wall shear stresses at the reattachment region, which over time may have the potential to cause high cycle fatigue of the lumen wall.

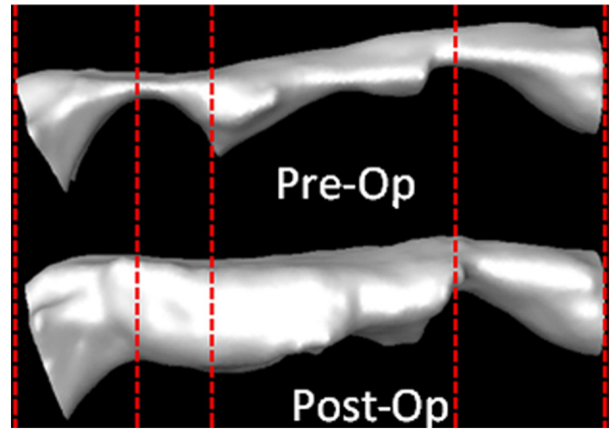

FIGURE 3. Side views of pretreatment and posttreatment pharyngeal airway models and comparison of flow variables along airway. Flow data in the pharyngeal airway were calculated by CFD during inspiration at a flow rate of 15 L/min for pre- and post-OSAS surgical treatment conditions. *Vertical red lines 1* through *5* are pre- and post-location markers. Location marker 1 represents the inlet level of the airway at the posterior nasal choanae. The outlet of the airway is at location marker 5 at the base of the epiglottis. These location markers 1 to 5 show changes from before to after treatment for each of the 3 flow variables.

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CFD modeling can generate precise data on OSAS airflow characteristics, which can help to gain insight into the possible etiology of airway collapsibility in OSAS.[43-45] CFD data were obtained and evaluated preoperatively and postoperatively for a single subject with OSAS.[1] This subject underwent bimaxillary advancement surgery for severe OSAS.

Figure 3 shows the reconstructed pharyngeal airway models before and after treatment. End-inspiratory and end-expiratory data were calculated for different flow rate conditions (5, 10, 15, and 30 L/min), but because of limited space in this presentation, only end-inspiratory data for the flow rate of 15 L/min are shown.

Preoperatively, the CFD data showed that there was a large increase in axial velocity (Fig 4A) and the wall shear stress values (Fig 4C) at the minimum cross-sectional areas (locations 2 and 4) in Figures 3 and 4. Large static pressure variations were found as well along the pharyngeal airway length, as depicted in Figure 4B. As inspiratory flow continues below the obstruction site to the supraglottic re-

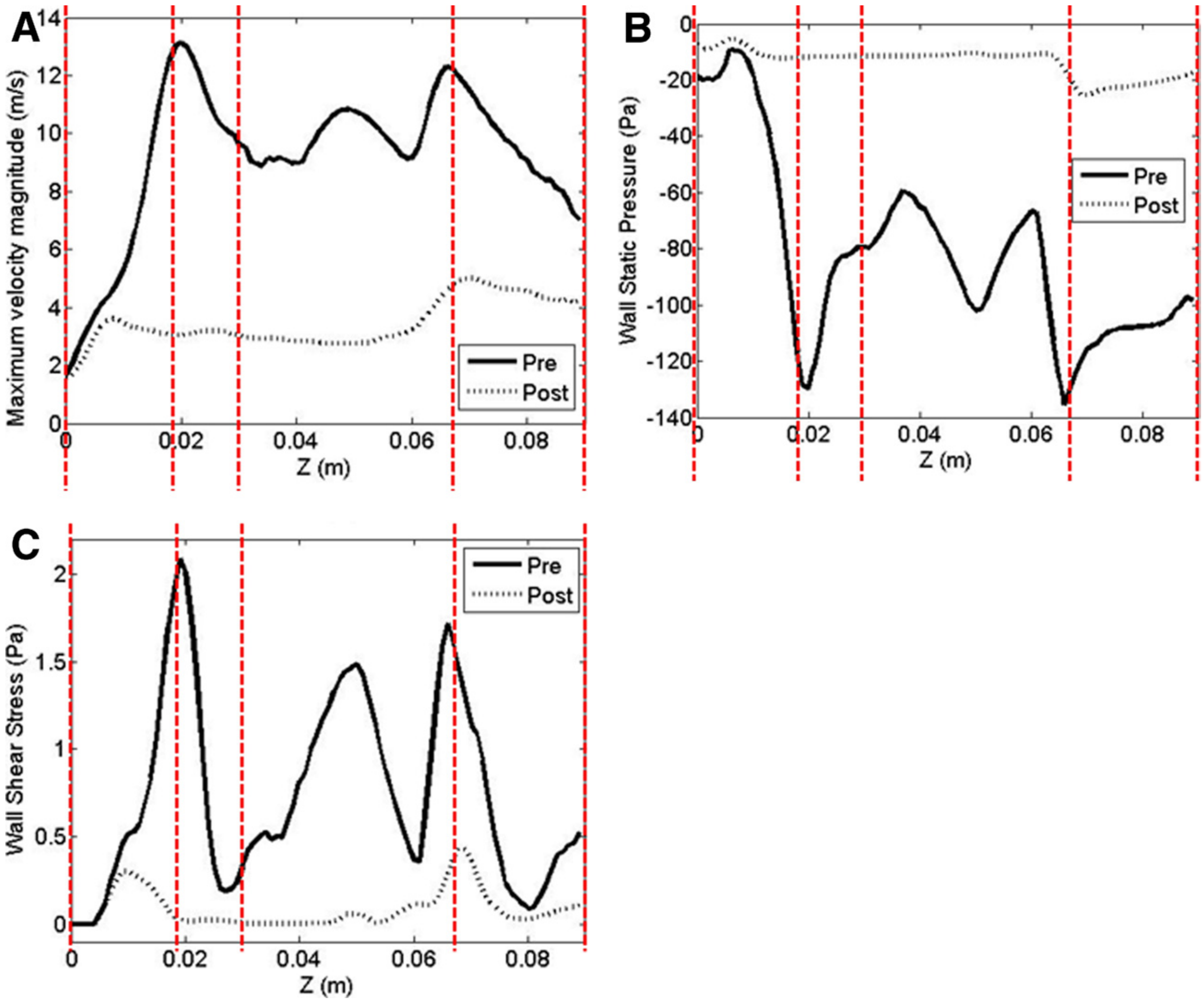

FIGURE 4. *A,* Pretreatment and posttreatment axial velocity. At the narrow sites of the airway at location numbers 2, 3, and 4, the peak velocity values were significantly higher before treatment compared with after treatment. Velocity magnitude is presented in meters per second. (Z, airway length in meters.) *B,* Pretreatment and posttreatment wall static pressure. Pressure drops were found for the same flow rate (15 L/min) before treatment compared with after treatment. Wall static pressure is measured in pascals. (Z, airway length in meters). *C,* Pretreatment and posttreatment wall shear stress. Pretreatment wall shear stress values were significantly higher than the posttreatment conditions. Wall shear stress is measured in pascals. (Z, airway length in meters.)

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gion, flow separation is observed with recirculation upward enhancing turbulence in the jet-like shear layers. Further downstream, the higher-velocity jet reattaches to the pharyngeal wall, an interaction that may cause large fluctuations in the wall shear stress and the pressure forces that act on the pharyngeal wall.

Postoperatively, the variations in flow variables along the pharyngeal airway length (z) almost fully disappeared, and both airway resistance (based on the pressure difference) and wall shear stresses were significantly reduced compared with the pretreatment case (Fig 4). The flow becomes more streamlined and smooth, the larger cross-sectional areas associated with the surgery being favorable to a laminar-like flow situation rather than a turbulent one. A laminar airflow is less traumatic than a turbulent airflow, its interaction with the airway lumen being minimized. The volumetric airway changes from before to after surgery were easily seen, with these changes playing a critical part in the pharyngeal airway and airflow dynamics, as shown in Figures 3 and 4.

The subject's severe OSAS was completely resolved after treatment. The pharyngeal airway after treatment showed improved contour and volume that dramatically changed airflow characteristics. This preliminary study will require continued research using static and compliance imaging of the upper airway from the nasal inlet to the outlet at the base of the epiglottis. A better understanding of airflow characteristics in OSAS may, in the future, provide a link to the etiology of this syndrome.

Decision Algorithm

All factors are then considered in the decision for surgery. Mild OSAS symptoms are usually associated with minimal obstruction at one of the levels, such as the nose. Soft tissue procedures such as tonsillectomy are performed before the decision for major skeletal surgery, and the patient is then retested afterward. A small anatomic airway, less than 50 mm^2, in the retropalatal area is best treated by maxillary advancement alone or in combination with mandibular advancement depending on the extent of the malocclusion. A severe Class III malocclusion may be corrected solely by maxillary surgery, for example. A small airway at the retrolingual location will most likely need a significant mandibular advancement. An airway closer to 100 mm^2 may need only a genioglossus geniohyoid advancement. This is usually performed as a segmental osteotomy as shown in Figure 1; however, in cases with associated microgenia, a full advancement genioplasty may be performed as long as the central portion of the osteotomy actually is high enough to capture the genial tubercle. It is our impression that airways, which are round in appearance, respond less to the surgery and will need larger advancements than elliptical airways with more lateral extension. Multiple levels of obstruction will necessitate bimaxillary surgery. In addition, younger and thinner patients respond better than older and obese patients, in whom there is more soft tissue involvement and laxity. If the airway problem is major, the septal and/or turbinate corrections are usually combined with the jaw surgery. If the airway problem is mild to moderate, these surgeries may be done beforehand to judge the response before embarking on major jaw surgery.

Surgical Technique

LIMITING ANESTHESIA RISKS

A laryngeal examination done indirectly or fiberoptically will give the surgeon and the anesthesiologist a chance to assess the possible difficulty of intubation and extubation. To further limit the perioperative risks, the surgeon may wish to be at the bedside of the patient on intubation and extubation. Consideration should be given to extubation in the operating room and will require the anesthesiologist to have the patient sufficiently awake at the end of the procedure.

SURGICAL PREPARATION

Nasopharyngeal intubation is accomplished with a Royal Aircraft Establishment (RAE) tube and fixed superiorly over the forehead and cranium. Because most MMA cases are done with cranial bone grafts, the tube is sutured to the scalp on the side opposite from which the graft is taken. Procurement of the cranial graft is the first procedure. Hypotensive anesthesia is necessary to reduce blood loss and improve visualization in surgery. This means a systolic pressure below 100 and a mean arterial pressure (MAp) of around 70 mmHg. Because hypotensive agents are needed, an arterial line is recommended. In addition, we use a Foley catheter and sequential compression garments, and the head is placed higher than the heart. We routinely run light on intravenous (IV) fluids to limit postoperative edema. If bimaxillary advancement is planned for an adult, we request 2 U of autologous blood before surgery.

SURGICAL TECHNIQUE

The Le Fort I surgical technique is generally similar to that published by other authors with several exceptions.[10] The maxilla is approached first, and a horizontal vestibular incision is made from the first molar on 1 side to the opposite first molar. A subperiosteal dissection is then accomplished, exposing the anterior face of the maxilla back to the pterygoid process and the piriform rims anteriorly. The infraorbital nerve is visualized and attention taken to not retract forcefully directly on the nerve. The anterior nasal spine is removed with the muscles attached and the nasal floor elevated; this includes separating the septum from the maxilla. At this time, we prefer to fracture the pterygoid plates while the maxilla is still stable. Next, the osteotomy cuts are marked at least 5 mm above the apices of the teeth and generally somewhat higher to accommodate the plates. The maxilla is then mobilized by an anterior downfracture and posterior downfracture. Sufficient mobility must be obtained to advance the jaw passively into the desired position, which in many adult patients with OSA is 1 cm or greater. The maxilla is rigidly fixed in the new position with titanium miniplates. An intermediate splint is frequently beneficial here to align the maxilla and prevent midline discrepancies and sideways and vertical yaw of the jaw. Next, attention is turned to the mandible. An incision is made along the external oblique ridge from midramus height to lateral to the first molar. The tissues are then elevated at the subperiosteal level to expose the lateral border of the mandible and anterior aspect of the ramus only. This is the so-called modified sagittal split technique.[46] The temporalis muscle must be stripped from the coronoid process high enough to access the medical ramus above the lingual process. The medial ramus is then exposed to just above and behind the lingual process, which can easily be identified with a nerve hook and is always at the same level as the mandibular occlusal plane. Retraction is obtained and an osteotomy made 4 to 5 mm above the lingual process with a saw or bur to just behind the lingual. It is not necessary to carry the cut any further back because the split will usually occur at this level anyway and it is safer to end the cut at this level. The osteotomy is carried along the anterior aspect of the coronoid process following the external oblique ridge, where they connect to just distal to the first molar tooth or where the ridge turns down and the bone becomes thinner. A vertical osteotomy is then made from this osteotomy inferiorly through the inferior border of the mandible, usually in the area of the antegonial notch. It is extremely important here to cut completely through the inferior border from lateral to medial; otherwise, the odds of a bad split increase significantly. The split is then accomplished with osteotomes and spreaders. Careful attention must be given to the position of the inferior alveolar nerve during this process because it frequently can be found in the proximal fragment and must be gently teased out to prevent injury to it. The muscles are detached from the distal fragment at this time. A similar procedure is then accomplished on the opposite side of the mandible. The mandible is then brought into occlusion with the maxilla. An occlusal splint is beneficial with some built-in overcorrection. The mandible is then plated or fixed with screws or a combination of the two. Rigid fixation is mandatory because the advancements are always quite large over 1 cm and sometimes around 2 cm. In the larger advancements (>1 cm) we prefer to use distraction in the mandible. Slower advancement allows fine tuning of occlusion and limits the force on the condyles. When distraction is used, the rate is 1 mm/d with no latency period. Distraction of the sagittal split segments is easily done, and the activating wires can be placed intraorally.[47] The maxillary bone grafts are then placed in the advancement gap and either wedged in or secured with screws. The maxillary vestibular incision is closed with a nasal muscle cinch suture and a V-Y advancement of the vestibular tissues. This prevents nasal widening and thinning of the lips, which is a common complication of bimaxillary advancement.[46-48] The mandible soft tissues are then closed. Intermaxillary fixation is not used because of the postoperative airway issues and the fact that rigid internal fixation is applied. Postoperative training elastics are used, however, to guide the jaws into the appropriate occlusion.

POSTOPERATIVE MANAGEMENT

Medical management of the postsurgical patient with OSAS is more complicated than the usual orthognathic patient. A very important care concept is frequently missed in the postoperative period. A majority of OSAS patients, once in the intensive care unit (ICU), appear stable, but usually, these findings are noted while the patient is awake. The problem is that the patient's condition may dramatically change when he or she is overly sedated or asleep. This is when a typical OSAS patient's condition may become pathologic and he or she have airway problems.[32]

It is recognized that the surgeon will select and use his or her familiar routine for recovery and follow-up. We suggest the use of an ICU or monitored bed postoperatively and careful use of analgesics and antihypertensives. The use of a patient-controlled analgesia pump is not recommended in sleep patients because the sensitivity to "any" respiratory depressant drug is so variable that total apnea can be the outcome even in minute doses. A logical and safe method to control pain in the ICU is the administration of small IV doses at frequent intervals by an ICU nurse who is constantly evaluating any attenuation of respiratory rates. The use of nasal CPAP after surgery, especially when the patient is sleeping, is very helpful to maintain the airway and control edema and tends to lessen the use of narcotics. After MMO surgery, bilateral nasal trumpet airways must be used to apply CPAP. If not used, air emphysema will be seen in the face and neck and is most unpleasant. Use of CPAP may also decrease patient anxiety because, if properly fitted, the patient knows from preoperative experience that CPAP will protect his or her airway when sleeping.[14,18,19] Usually the patient may be transferred out of the ICU or step-down unit on the first postoperative day. Pain can be controlled with oral liquid analgesics or intramuscular injections because IV medication is seldom necessary after the first postoperative day. The patient is encouraged to be out of bed ambulating and taking oral liquids on the first postoperative day. The patient is seen by a nutritionist for diet instructions before discharge. Discharge is up to the surgeon and patient, and it usually depends on pain control and oral intake. The young adult or adolescent patient is usually easier to manage because such patients do not have the overlying medical problems seen in the adult OSA patient. The main concern is airway management the first postoperative night. To assist in this, we do not apply intra-arch elastics until 1 to 2 days postoperatively. Management of the pediatric patient with OSA and craniofacial abnormalities is complex and beyond the scope of this article.

FOLLOW-UP

Follow-up is again up to the individual surgeon. Because most surgeries for OSAS may create some degree of pain, swelling, and airway concerns, it is suggested to have frequent follow-up. It should be kept in mind that surgical edema will usually peak at 72 hours, at which point the patient may notice the effect of swelling and become concerned about his or her airway. This finding can in some cases be quite significant. The usual hospital stay is 2 to 3 days for an adult OSAS MMO procedure. Our patients are seen at day 3 or 4 in the office and again weekly until fully healed. Those with CPAP are encouraged to continue use of the device while sleeping until 1 to 2 weeks before the follow-up full-attended PSG study, which is done at 4 to 6 months. This time interval allows for weight stabilization and neurologic equilibration.[20] A full PSG study can be done as early as 4 to 6 weeks after surgery in the pediatric population. Should further surgical or medical treatment be necessary, these plans can then be made. If the outcomes of the PSG study and EDS are resolved, routine follow-up is at 6 months and then yearly as necessary.

FIGURE 5. Pretreatment facial views. *A*, Lateral facial view. *B*, Frontal facial view.

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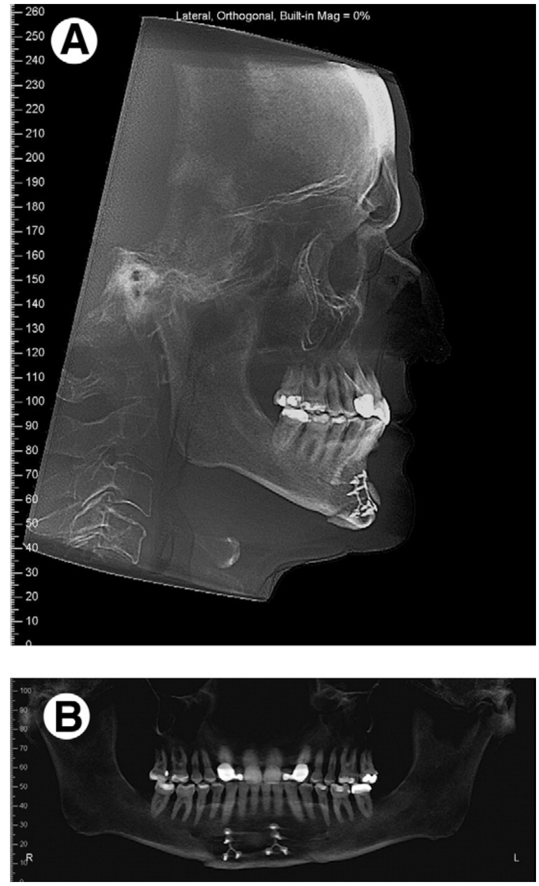

FIGURE 6. Pretreatment radiographs. *A*, Lateral cephalogram. *B*, Panoramic radiograph.

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WEIGHT IN OSAS

Obesity is considered an important associated factor in the onset and severity of SDB. An elegant study by Peppard et al[49] nicely outlines the cardiovascular risks associated with SDB for weight gain and weight loss. Excess body weight was found to be positively associated with SDB and relative to a stable weight, where a 10% weight gain predicted a 32% increase in the AHI. This same 10% gain in weight predicted a 6-fold increase in the odds of moderate to severe SDB developing. Conversely, a weight loss of 10% predicted a 26% decrease in the same index (AHI). Unfortunately, weight loss programs in patients with SDB have failed miserably. Even with the use of sophisticated behavioral modification programs and bariatric surgery, failures are common. Because there can be measurable improvement in patients with SDB when weight is lost, there has been keen interest in environmental, mechanical, genetic, and hormonal factors that may affect weight in these subjects. It should be stressed to each and every overweight OSAS patient that weight loss is an important part of the pretreatment or posttreatment management because even modest weight loss may improve surgical outcomes.

Clinical Case

A 54-year-old man was seen in consultation for his OSA, which was debilitating, prevented him from functioning as a pilot, and severely affected his daily life. The OSA was so severe that it significantly affected his health and, if left untreated, may have been life-threatening. Weight loss, changes in his sleep positioning, eliminating alcohol, mandibular positioning devices, uvular surgery, and genioglossus muscle advancement were ineffective in controlling his sleep apnea because of an anatomically small airway quantified by a CBCT scan. The Vultus analysis showed a smallest surface area of 59.5 mm^2, which—according to the literature mentioned previously—falls in the region of moderate to possibly severe sleep symptoms. Overnight PSG showed moderate OSA with an AHI of 21/h, which increased to 49.4/h in rapid eye movement (REM) sleep. This was associated with bradycardia/tachycardia and an oxygen desaturation to 82%. Sleep stages were also disrupted, with 75% of the time spent in stage 2.

Clinical facial and oral examination showed a Class III–type malocclusion with maxillary insufficiency and mild skeletal mandibular asymmetry with chin deviation to the right (Figs 5-7). The patient was minimally concerned with facial or dental esthetics but was most interested in correcting the sleep apnea. Maxillary and mandibular arches were misaligned with midline discrepancy, and old fixed bridges were present in the maxillary anterior, replacing missing lateral incisors. There was also a history of clicking and locking of the temporomandibular joint with late reciprocal meniscus displacement bilaterally, which was currently stable. Intermittent myofascial pain was present upon awakening from nocturnal bruxism.

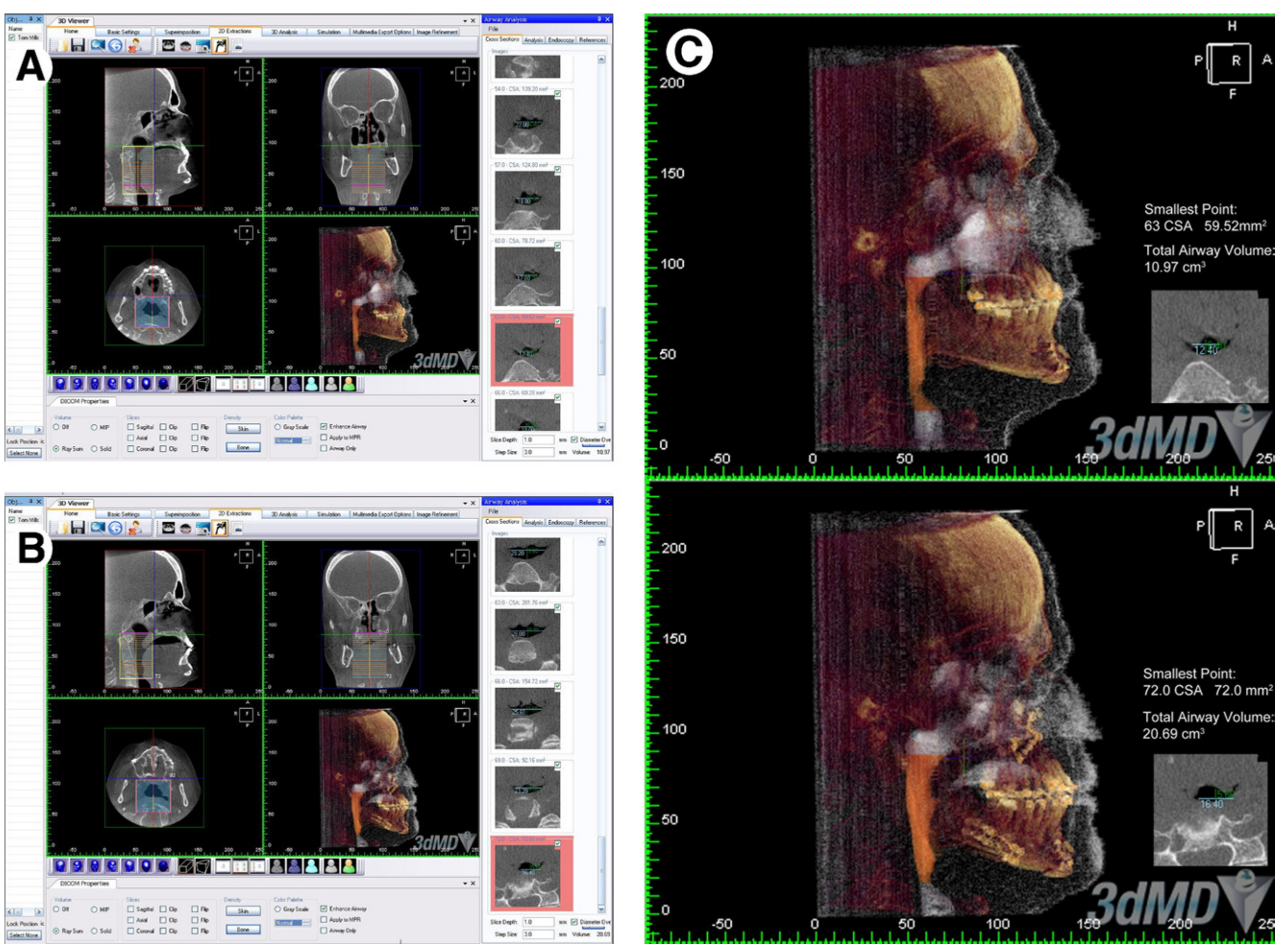

FIGURE 7. Pretreatment and posttreatment Vultus airway analysis. *A*, Pretreatment. Total airway volume was 10.97 cm^3, and smallest airway area was 59.5 mm^2. *B*, After treatment. Total airway volume was 20.69 cm^3, and smallest airway area was 72 mm^2 after 12-mm MMA. *C*, The smallest airway area presurgery is 56.52 mm^2 with a total airway volume of 10.97 cm^3 above and postsurgery is 72 mm^2 with an airway volume of 20.69 cm^3 according to the Vultus airway analysis.

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CBCT imaging showed adaptive temporomandibular joint condylar changes.

Presurgical orthodontic therapy consisted of a short course of treatment to prepare the teeth for surgery and for the prescribed final occlusion. Before the application of orthodontic appliances, a diagnostic setup was performed to ascertain the therapeutic occlusion. Impressions of the teeth were taken, poured in stone, and mounted on a semiadjustable articulator in an unstrained repeatable physiologic centric relation position. Centric relation was established disengaging the malocclusion with short-term flat-plane splint therapy and manual manipulation bite registration techniques. Teeth surfaces on the casts were altered and then duplicated in the mouth with minimal restorative dental procedures to ensure ideal postsurgical occlusal interdigitation. An arch form was chosen with a template based on the patient's mandibular cancellous and alveolar bone and arch form for optimal periodontal health postorthodontic therapy. Edgewise appliances were then bonded to the surfaces of the teeth with custom bases to ensure the expression of the intended orthodontic therapeutic occlusion dictated by the diagnostic setup. Rectangular nitinol (0.016 × 0.022-inch) wire was placed for torque control and bodily movement. Bodily teeth movement is preferred to minimize root resorption from tipping. Light orthodontic pressures and forces are used to achieve optimal tooth movement in the shortest possible time with a minimal amount of tissue damage. Rectangular nickel titanium wires were engaged in 0.018 rectangular slots by use of RMO synergy brackets (Rocky Mountain Orthodontics, Denver, CO) ligated to the central bracket wing only to minimize the friction, during the leveling stage.

The magnitude of orthodontic forces has been show to influence the severity of root resorption, and light orthodontic forces are advocated. Traditionally, heavy forces of up to 3,000 to 4,000*g* have been used in orthodontics. Burstone[50] and Ricketts[51] showed that 50 to 75*g* of force was adequate to successfully move incisors. The pressure necessary for bodily movement in adults and concluded that 197g/cm^2 was optimal.[52]

An accelerated presurgical phase of treatment was advocated because of the health advantages gained by normalizing the airway and patient oxygen saturation during sleep in lieu of prolonged presurgical orthodontics as traditionally done. Rectangular elgiloy (0.016 × 0.022-inch) passive wires (Rocky Mountain Orthodontics) were placed presurgically with hooks placed interdentally for intermaxillary traction. Postoperative orthodontic treatment was minimal based on the exactness of the preoperative setup.

FIGURE 8. *A,* Face after treatment. *B,* Profile after treatment.

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A bimaxillary advancement of 12 mm was then performed with the patient under general anesthesia and the patient admitted for postoperative care; cranial bone was used to graft the maxillary defect (Figs 8, 9). The surgical technique was performed as outlined previously. Postoperatively, he did well, and there were no complications. He was discharged on postoperative day 4. The entire orthodontic and surgical treatment period took less than 9 months. A PSG scan was done at postoperative month 3. The AHI was then 5, and he reported physical improvement. The smallest airway measured 72 mm^2, and the volume increased to 20.69 cm^3 from a preoperative value of 10.97 cm^3.

In summary, bimaxillary surgery plays an important role in correction of OSA that is refractive to medical management or where medical management is not

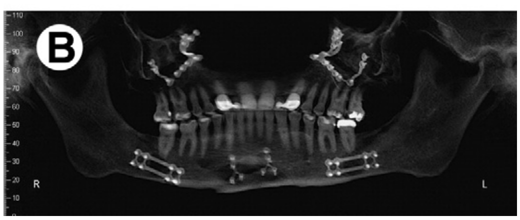

FIGURE 9. *A,* Lateral cephalometric radiograph after treatment. *B,* Panoramic radiograph after treatment.

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tolerated and in those patients desiring a definitive correction of the problem. The older adult patient usually requires MMA of at least 1 cm based on the literature and is complex to manage because of the longstanding associated soft tissue abnormalities at multiple levels. They may also be medically compromised, which complicates the perioperative period. Treatment of the young adult or adolescent patient is more direct and aimed at correcting the skeletal malformation but may be complicated by existing temporomandibular joint problems such as condylysis or rheumatoid arthritis. Distraction osteogenesis is playing a larger role in the treatment of these patients who require large advancements (0.1 cm). The widespread use of CBCT scans and the recent development of automated airway analysis such as Vultus provides the surgeon more refinement in treatment planning because the exact site or sites of obstruction can be readily visualized. Surgical correction can thus be more individually tailored for each patient.

Orthodontists, in preparing patients for jaw surgery in the treatment of OSA, tend to focus on the mechanical aspects of the presurgical and postsurgical occlusion. Specialists are acutely attuned to the nuances of occlusion; often, the biochemistry of the patient and its importance in healing are neglected. Calcium homeostasis, parathyroid hormone levels, vitamin D metabolites, and the patient's biochemistry, in general, should be monitored to reduce the metabolic causes of bone loss and tooth resorption.

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