Surgical and Nonsurgical Manssagement of Obstructive Sleep Apnea

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Contents

71.1 History – 2137

71.2 Normal Sleep Stages – 2137

71.3 Sleep Apnea Syndrome – 2137
71.3.1 Classification – 2138
71.3.2 Differential Diagnosis – 2138
71.3.3 History of OSA Syndrome – 2139
71.3.4 Clinical Manifestations – 2139
71.3.5 Physical Findings – 2139

71.4 Diagnosis – 2140
71.4.1 Physical Examination – 2140
71.4.2 Cephalometric Examination – 2141
71.4.3 Computed Tomography – 2141
71.4.4 Polysomnography – 2142
71.4.5 Site of Obstruction – 2142

71.5 Medical Treatment – 2143
71.5.1 Oral Appliances – 2144
71.5.2 Continuous Positive Airway Pressure – 2145

71.6 Surgical Treatment – 2145
71.6.1 Tracheostomy – 2145
71.6.2 Nasal Surgery – 2146
71.6.3 Uvulopalatopharyngoplasty – 2146
71.6.4 Laser-Assisted Uvulopalatoplasty – 2147
71.6.5 Uvulopalatal Flap – 2148
71.6.6 Tongue Surgery – 2148
71.6.7 Orthognathic Surgery Procedures – 2149
71.6.8 Mandibular Advancement – 2149

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71.6.9 Genial Tubercle Advancement – 2149
71.6.10 Genial Advancement with Hyoid Myotomy and Suspension – 2150
71.6.11 Maxillomandibular Advancement – 2151
71.6.12 Mandibular Setbacks – 2154

71.7 Complications – 2154

71.8 Summary – 2156

References – 2156

Learning Aims

1. Obstructive sleep apnea definition, diagnosis, treatment
2. Understanding nonsurgical approaches for the management of OSA
3. Understanding surgical approaches for the management of OSA
4. Complications of treatment
5. Understanding positive surgical outcomes

71.1 History

Sleep and dreaming have been sources of mystery and fascination since biblical times. Sleep consists of inevitably recurring episodes of readily reversible relative disengagement from sensory and motor interaction with the environment [1]. The function of sleep remains a mystery, and only in recent years has there been research into specific symptom complexes and causes of sleep disorders. In 1979, the Association of Sleep Disorders Center and the Association for the Psychophysiological Study of Sleep published the first classification of sleep and arousal disorders [2].

Modern sleep research became possible in 1924 when Hans Berger [3], a German psychiatrist, described the recording of human electroencephalography. Loomis and colleagues, in 1935 [4, 5], published a quantitative description of the four levels of sleep based on electroencephalogram (EEG) characteristics. The historic discovery of a cyclic phase of sleep characterized by rapid conjugate eye movements was made by Aserinsky and Kleitman in 1953 [6]. Subsequent studies confirmed this to be a very active phase of sleep that correlated closely with dreaming [7].

71.2 Normal Sleep Stages

Normal sleep architecture includes both quiet sleep (non-rapid eye movement [non-REM] sleep) and active sleep (rapid eye movement [REM] sleep). Non-REM sleep consists of four stages that are based largely on the original criteria of Loomis and colleagues [4, 5]. Stage 2 predominates and accounts for 45–50% of total sleep time. The four stages of non-REM sleep represent progressively deeper sleep marked by the increasing appearance of high-amplitude slow waves in stages 3 and 4, which are collectively known as *delta sleep.* Non-REM sleep is characterized by a general slowing of all levels of activity. Progression through all four stages of non-REM sleep usually occurs rapidly after sleep onset. REM sleep occurs after non-REM sleep has been established, and the first REM period normally occurs after 70–90 minutes of non-REM sleep. The average duration of a period of REM sleep is approximately 20 minutes. The initial REM period of the night is usually very brief, but subsequent REM periods become longer. During an average night of REM/non-REM cycle progression, four to six REM periods normally occur at intervals of 60–90 minutes. REM sleep occupies about 20–25% of total sleep time in a healthy young adult. REM sleep EEG patterns look very similar to those seen during the wakeful state. Generalized skeletal muscle atonia (except for the ocular muscles) and absence of reflexive activity are other features unique to REM sleep. Marked physiologic changes also occur during REM sleep. Temperature, blood flow, and oxygen use in the brain are increased. Heart rate, blood pressure, and respiration show dramatic fluctuations and increase in average rate.

During sleep, the control of respiration is influenced by two systems: the metabolic control system and the behavioral control system [8]. The influences of hypoxia and hypercarbia on ventilation are the predominant components of the metabolic control system of respiration. This system predominantly controls respiration during non-REM sleep. The behavioral control system governs respiration during voluntary activities, such as swallowing or speaking, and may suppress the ventilatory response to metabolic stimuli. During REM sleep, the effects of hypoxia and hypercarbia on ventilation are much less than during non-REM sleep and the behavioral control system may predominate. With a blunted response to hypoxia and hypercarbia, irregular respirations, and decreased skeletal muscle tone of the upper airway muscles during REM sleep, an episode of partial or complete airway obstruction with apnea or hypopnea may occur.

71.3 Sleep Apnea Syndrome

The sleep apnea syndrome is a disorder characterized by abnormal breathing in sleep and sleep fragmentation. At least 30 episodes of apnea occur during 7 hours of nocturnal sleep in these patients. *Apnea* is defined as the cessation of airflow from the nostrils and mouth for at least 10 seconds. These apneic episodes can result in hypoxemia, hypercarbia, systemic and pulmonary hypertension, polycythemia, cor pulmonale, bradycardia, and cardiac dysrhythmias. Sudden death has occurred in patients with sleep apnea. Throughout the night, the alternating episodes of apnea and arousal from sleep may occur as frequently as 400–600 times, with each typical apnea episode lasting 15–60 seconds. These episodes can amount to as much as 50% of a night's sleep. The frequent disruption results in symptoms similar to those of sleep deprivation. These

include excessive daytime sleepiness, fatigue, depression, personality changes, and impotence. These dysfunctional symptoms are common primary complaints and are often the reason people seek treatment.

Epidemiologic data suggest that sleep apnea syndrome may be quite common, particularly in its milder forms. In fact, obstructive sleep apnea (OSA) is the second most common sleep disorder, insomnia being the most common. A 1993 Sleep Commission Report estimated that 20 million Americans have sleep apnea, with the majority being undiagnosed and untreated [9]. The exact prevalence is unknown, but sleep apnea syndrome may affect up to 2–3% of adult males [8]. In certain populations, the prevalence may be as high at 10%. Most patients are diagnosed after age 40, but sleep apnea can occur at any age. There is a strong male predilection, with men outnumbering women by up to 8:1 until menopause. This implies a hormonal influence. The cost for diagnosis and treatment of this sleep disorder accounts for over $50 million (U.S.) in hospital bills each year. Overall, sleep disorders and sleepiness cost the U.S. economy a minimum of $15.9 billion in direct costs each year [10].

71.3.1 Classification

Central sleep apnea, OSA, and mixed sleep apnea are the variations of apnea that occur in the syndrome. In central sleep apnea, respiratory muscle activity ceases simultaneously with airflow at the mouth and nostrils [11]. This disorder is found in patients with central nervous system (CNS) insufficiency that affects the outflow of neural output from the respiratory center to the diaphragm and other muscles of respiration. CNS disorders associated with central sleep apnea include brainstem neoplasms, brainstem infarctions, bulbar encephalitis, bulbar poliomyelitis, spinal surgery, cervical cordotomy, and primary idiopathic hypoventilation.

Patients with central sleep apnea have been treated with some success by using respiratory-stimulating drugs such as theophylline, progesterone, and acetazolamide. In severe central apnea, modalities of treatment have included phrenic nerve pacemaker implantation to ensure regular respiration during sleep and nocturnal mechanical ventilation with a negative-pressure ventilator for more severe cases. There are no simple and convenient methods of treatment for mild central apnea.

The most common type of sleep apnea by far is obstructive. This is characterized by sleep-induced obstruction of the upper airway that results in cessation of airflow with preservation of respiratory effort, respiratory center drive, and diaphragmatic contraction [11].

Mixed sleep apnea is a combination of central and obstructive apnea. This pattern begins with an episode of central apnea with no airflow detectable at the mouth and nostrils and no respiratory muscle activity. The pattern ends with an episode of obstructive apnea with cessation of airflow only at the mouth and nostrils [11].

71.3.2 Differential Diagnosis

Profound hypersomnolence is a characteristic feature of both sleep apnea and narcolepsy; hence, they are often confused. However, unlike sleep apnea, narcolepsy affects both sexes equally, with most patients experiencing the onset of symptoms around or shortly before puberty [12]. The first symptom to appear with narcolepsy is usually excessive daytime somnolence (EDS). The sleep attacks can range from mild to severe and are characterized by the sudden onset of overwhelming sleepiness that lasts 30 seconds to 20 minutes. After brief naps, the narcoleptic usually feels refreshed and relatively free from disturbing symptoms for up to 2 hours. Serious accidents, marital discord, and the inability to hold jobs frequently result from these sleep attacks. Another feature of narcolepsy is the abrupt loss of muscle control (cataplexy). Attacks can be particularly disabling because they are characteristically precipitated by emotional experiences such as laughter, anger, or excitement. Additional associated symptoms of narcolepsy include sleep paralysis and hypnagogic hallucinations. Sleep paralysis is the skeletal muscle atonia of REM sleep persisting into the awake state. Hypnagogic hallucinations are REM sleep imagery occurring while falling asleep. Patients are sometimes misdiagnosed as schizophrenic if hypnagogic hallucinations are prominent.

Diagnosis of narcolepsy is made by documenting sleep-onset REM periods during a nocturnal polysomnography [12]. In normal sleep, REM sleep is usually not seen until about 70–90 minutes into sleep. The clinical features of narcolepsy probably represent abnormal manifestations of REM sleep.

Treatment modalities for narcolepsy include behavioral therapies, CNS stimulants, tricyclic antidepressants, or monoamine oxidase inhibitors (only in resistant cases) and L-tryptophan [13].

Other disorders that may be confused with sleep apnea syndrome include sleep-related abnormal swallowing syndrome, gastroesophageal reflux, depression, alcohol or drug dependence, and sleep-related nocturnal myoclonus.

Definitions

- Apnea: cessation of airflow for more than 10 sec
- Hypopnea: decrease in airflow (greater than 50%) with oxygen desaturation >4%
- Hypopnea index: number of shallow respirations per hour
- Obstructive Sleep Apnea: more than 5 apneic events per hour
- Narcolepsy: sleep attacks characterized by the sudden onset of overwhelming sleepiness that lasts 30 seconds to 20 minutes
- Apnea Hypopnea Index (AHI): Total number of apneas and hypopneas per hour
- Respiratory Disturbance Index (RDI): Total number of respiratory-effort related arousals + hypopneas + apneas per hour

71.3.3 History of OSA Syndrome

OSA has a remarkably short history, considering the incidence and disabling symptoms of the syndrome. Burwell and coworkers published the first description of the syndrome in 1956 [14]. Their report compared an obese, somnolent, polycythemic patient with the sleepy red-faced boy, Joe, in the Charles Dickens novel *The Posthumous Papers of the Pickwick Club* (1837). However, Burwell and coworkers did not link their patient's excessive daytime sleepiness to nocturnal sleep fragmentation. In 1966, Gastaut and associates [15] were the first investigators to demonstrate repeated apneas in pickwickian patients during sleep. They correctly attributed the excessive daytime somnolence in these patients to nocturnal sleep fragmentation caused by repeated apneas.

The misdiagnosis of narcolepsy in patients with sleep apnea and the general skepticism of EDS as a valid clinical sign are the two main reasons sleep apnea syndrome was overlooked for so long.

71.3.4 Clinical Manifestations

Sleep apnea patients present with a variety of symptoms and clinical manifestations. Patients with OSA most often complain of EDS. The patients may experience serious social, economic, and emotional problems from the EDS associated with this disorder. The uncontrollable desire to sleep may predispose the patients to occupational or automobile accidents.

Almost all patients or their bed partners give a history of heavy, loud snoring that has usually been present for several years before the EDS was noted. The snoring is produced from the passage of air through the oropharynx causing vibrations of the soft palate. Typically, the snoring is interrupted periodically by apneic episodes that last 30–90 seconds. Bed partners usually describe an episode in which the snoring stops and the patient seems to stop breathing for a period of time. A loud snort followed by a hyperventilation usually signals an end to the apneic episode.

Other common presenting complaints are morning headaches and nausea that result from the hypercarbia that develops with the hypoventilatory episodes. Depression, personality changes, and intellectual deterioration may also develop.

The systemic hypertension that is a common finding in OSA may be related to the catecholamine release triggered by the systemic hypoxemia. In more advanced severe cases, pulmonary hypertension, polycythemia, and cor pulmonale may develop and become life-threatening. However, most patients do not manifest these disturbances because their ventilation during wakeful periods is sufficient to prevent these complications of chronic hypoxia.

A prominent sinus dysrhythmia is commonly associated with the apneic episodes. The extent of bradycardia is directly proportional to the severity of the oxygen desaturation. The greatest degree of cardiac slowing occurs in obstructive apneas in which a Mueller Maneuver (forced inspiration against a closed glottis) is performed. Increased vagal efferent tone mediates the bradycardia. This typically can manifest itself as a first-degree heart block and premature ventricular contractions.

The development of severe and life-threatening medical complications from the apneic events clearly depends on the frequency, duration, and degree of hypoxemia and associated hypertensive response.

Attempted inspiration against upper airway obstruction may produce increased negative thoracic pressure and pulmonary hypertension/edema.

71.3.5 Physical Findings

A major feature of OSA is obesity. The increased body weight correlates with increased frequency of apnea and the severity of hypoxemia. However, the morbidly obese, somnolent, hyperventilating patient with cor pulmonale represents only a small number of sleep apnea patients. Lower body mass index (BMI) patients with OSA often have more abnormal cephalometrics than obese people [16, 17].

Obstruction can occur at a number of points in the airway. Physical examination of these patients may reveal hypertrophy of the tonsils or adenoids, retrognathia,

micrognathia, macroglossia, deviation of the nasal septum, a thick short neck, or tumors in the nasopharynx or hypopharynx. Both primary and secondary medical conditions are associated with OSA, owing to their effects on the upper airway anatomy. These may include temporomandibular joint disorders, myxedema, goiter, acromegaly, and lymphoma.

All newly diagnosed patients should be evaluated for hypothyroidism. A screening test for thyroid stimulating hormone (TSH) is inexpensive and can be easily treated with hormone replacement therapy. This directly reverses apnea frequency independent of changes in weight and pulmonary function [18].

Most patients with classic OSA have no identifiable craniofacial anomaly. However, there does appear to be a significant subpopulation of sleep apnea patients with craniofacial anomalies [19, 20]. Lowe and colleagues [21] found several alterations in craniofacial form in subjects with OSA that may reduce the dimensions of the upper airway and subsequently impair stability of the upper airway. These include bimaxillary retrognathism, a steep occlusal plane, overerupted maxillary and mandibular teeth, proclined incisors, a steep mandibular plane, a large gonial angle, increased upper and lower facial heights, a posteriorly placed pharyngeal wall, and an anterior open bite in association with a long tongue [21]. Bacon et al. [19] evaluated 32 patients with sleep apnea by cephalometry and demonstrated an anteroposterior shortening of the cranial base, a posterior facial compression with narrowing of the pharyngeal airway, and an increased lower facial height. Rivlin and associates [22] reported on nine OSA patients with posterior displacement of the mandible. The number of apneas correlated with the total posterior displacement [22]. Gungor et al. compared 32 patients with cephalometric tracings with differences in midface length to be significantly shorter and upper lip E-plane length was significantly longer in the OSA group, maxillary length was slightly longer with shorter mandibular lengths, upper incisors were significantly protrusive and the hyoid to mandible distance was greater [23].

71.4 Diagnosis

71.4.1 Physical Examination

A diagnostic evaluation includes a thorough history and physical examination, fiberoptic endoscopy, radiologic evaluation, and polysomnography. Little additional information can be gained from routine laboratory tests. Except in severe cases, pulmonary function tests, electrocardiogram (ECG), arterial blood gases, and chest radiographs are often normal during wakefulness in sleep apnea patients.

Other diagnostic tests that may aid in evaluating sleep apnea patients include a complete blood count (CBC), serum electrolytes, and thyroid function tests. Secondary polycythemia may be revealed by a CBC, and nocturnal carbon dioxide retention may be reflected by increased bicarbonate levels. Hypothyroidism, a contributing cause of sleep apnea, may be identified from thyroid function studies.

After a complete history is obtained from the patient and his or her bed partner, a complete clinical examination of the mouth, nasal, pharyngeal, and laryngeal areas is performed. The emphasis of the clinical examination should be the identification of anatomic abnormalities that may contribute to or produce obstruction during sleep. The nose is examined for a deviated nasal septum and enlargement of the turbinates. Micrognathia, retrognathia, and macroglossia may be noted on examination of the oral cavity. Occasionally, masses or tumors in the nasopharynx or hypopharynx may be noted. In the pharynx, adenotonsillary hypertrophy, a long soft palate, a large base of the tongue, and excess pharyngeal mucosa are potential causes of obstruction. The larynx is examined for vocal cord webs and paralysis of the vocal cords. OSA patients may present with any combination of these anatomic abnormalities.

After topically anesthetizing the nasal cavity and pharynx, a fiberoptic endoscope is introduced through the nose. In sequential fashion, the nasopharynx, oropharynx, hypopharynx, and larynx are examined. The appearance and position of the soft palate, base of tongue, and lateral pharyngeal walls are evaluated. Changes in the position of the base of the tongue such as forward movement with protrusion of the mandible are noted. The appearance of the pharyngeal airway and degree of pharyngeal wall collapse is noted while the patient performs a modified Müller maneuver. To accomplish this maneuver, the patient attempts to inspire with the mouth and nose closed. Increased negative pressure in the pharynx will demonstrate the point of collapse.

Unfortunately, these are all static observations in an awake patient. Croft and Pringle first proposed drug-induced sleep endoscopy (DISE) as an effective technique for evaluating OSA [24]. This technique can be performed in the outpatient setting with the use of medication-induced sleep in conjunction with nasoendoscopy for evaluation of the upper airway. As such, new classification systems such as the VOTE (Velum, Oropharynx, Tongue base, and Epiglottis) classification system by Kezirian and colleagues give improved diagnosis and decision planning for surgical procedures [25].

71.4.2 Cephalometric Examination

A lateral cephalogram is routinely obtained in the radiologic evaluation of sleep apnea patients (Fig. 71.1). Cephalometric analysis is performed to identify any skeletal and soft tissue abnormalities that may exist. The advantages of cephalometry are its easy access, low cost, and minimal radiation exposure. However, it should be recognized that there are obvious limitations of evaluating a three-dimensional area with a two-dimensional lateral cephalometry.

Mandibular or maxillary position can be evaluated by a number of methods, including the SNA (sella-nasion–A point) and SNB (sella-nasion–B point) angles. Patients with skeletal deficiencies are more likely to have obstruction at the base of the tongue or at the level of the soft palate. Riley and colleagues [27, 28] determined that OSA patients had an inferiorly positioned hyoid bone, a longer-than-normal soft palate, and a narrowing at the base of the tongue. The position of the hyoid bone is determined by drawing a perpendicular line from the mandibular plane (MP) through the hyoid bone (H). The mean MP-H distance for normal subjects is 15.4 ± 3 mm (see Fig. 71.1). The position of the hyoid bone is important because it serves as a central anchor for the muscles of the tongue and thereby partly determines tongue position. Soft palate length is measured from a line drawn from posterior nasal spine (PNS) to the tip of the soft palate shadow (P). The mean PNS-P distance in normal subjects is 37 ± 3 mm. Posterior airway space (PAS) is determined by a line drawn from point B through the gonion (Go) intersecting the base of the tongue and the posterior pharyngeal wall. Figure 71.2 demonstrates change in PASs after maxillomandibular advancement (MMA). Mean PAS in normal subjects was determined to be 11 ± 1 mm. Lower face height is measured from the anterior nasal spine (ANS) to the menton (Me). There is no absolute value for this measurement in OSA patients. However, some studies have shown an increased lower face height and a shortened cranial base with OSA patients [19].

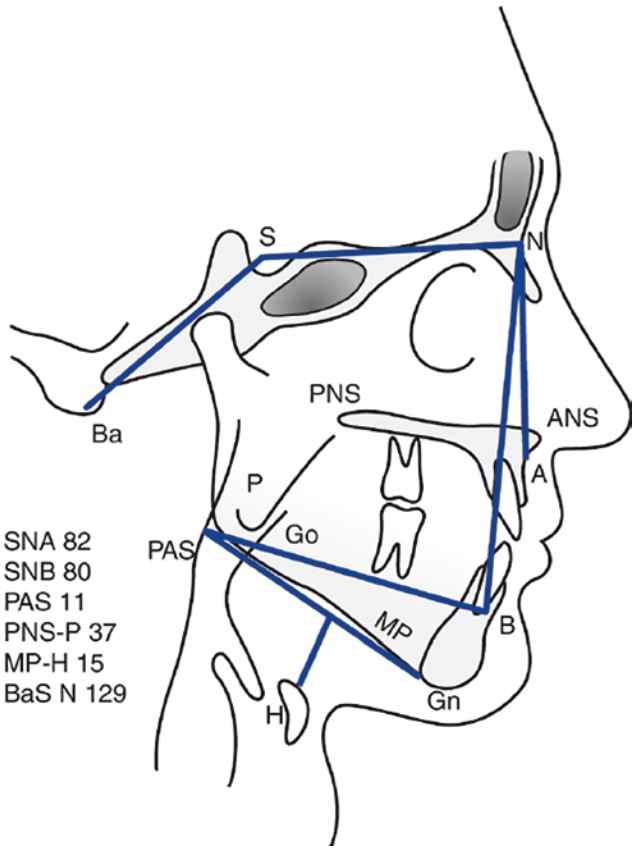


Fig. 71.1 Cephalometric screening used for initial evaluation of patients with obstructive sleep apnea syndrome. A A point, ANS anterior nasal spine, B B point, Ba basion, Gn gnathion, Go gonion, H hyoid, MP mandibular plane, N nasion, P palate, PAS posterior airway space, PNS posterior nasal spine, S sella, SNA sella-nasion–A point, SNB sella-nasion–B point. (Adapted from Tiner and Waite [26], p. 1535)

71.4.3 Computed Tomography

Computed tomography (CT) is an alternative to cephalometry and has been used to provide a quantitative assessment of the upper airway at various levels. With three-dimensional CT reconstructions, Lowe and coworkers [29] found OSA patients with larger tongue surface areas and smaller airway surface areas. Haponik and associates [30] found significantly decreased cross-sectional areas of the nasopharynx, oropharynx, and hypopharynx in OSA patients when compared with control subjects by using CT scanning. Some authorities believe that the airway can be assessed only by a CT scan. However, Riley and colleagues [28, 31] compared patients who had three-dimensional CT scans and found a statistically significant correlation between the PAS measured on the lateral cephalogram and the volume of the pharyngeal airway measured on CT scans. Waite and Villos [32] demonstrated by helical CT analysis that MMA increases both anteroposterior and lateral dimension of the airway at all levels from nasopharynx to hyoid. Many studies are currently being done to determine the effects of patient position and changes in airway. A cephalogram and a CT scan are static evaluations at a fixed time of a dynamic system and they should be viewed as only part of the overall evaluation of the patient. As technology improves, computational fluid dynamics can simulate the upper airway during performance of the Muller Maneuver. This theoretically gives more information during the event. However, this approach is more complex, and current modeling with

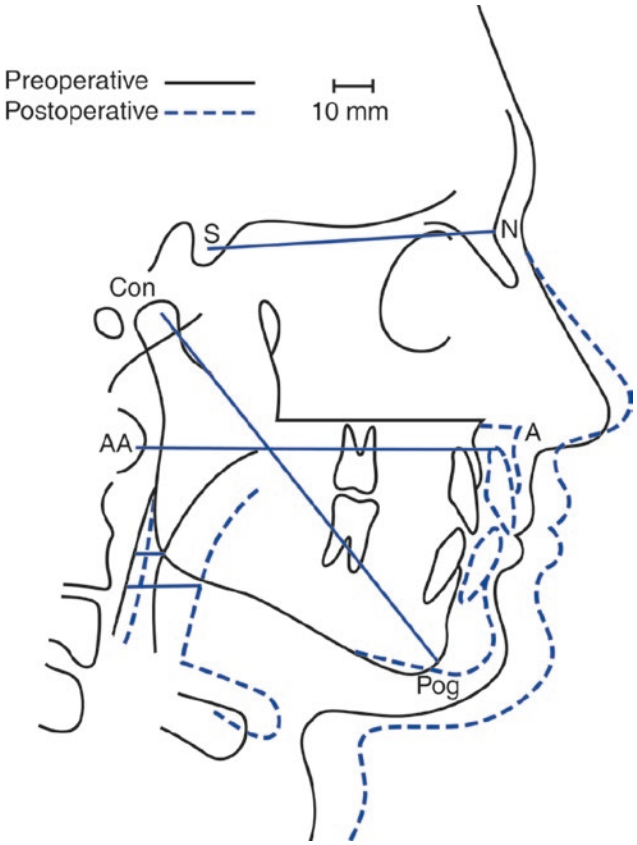


Fig. 71.2 Change in posterior airway space after maxillary and mandibular advancement. A A point, AA anterior edge of atlas, Con condylion, N nasion, Pog Pogonion, S sella. (Adapted from Tiner and Waite [26], p. 1536)

three dimensional imaging of anatomical cross sections is more efficient.

71.4.4 Polysomnography

Nocturnal polysomnography remains the gold standard for establishing the diagnosis of sleep apnea, quantitating its severity, and determining the success of treatment modalities. The study is performed in a sleep laboratory and the patient's sleep is monitored overnight. At least 4 hours of total sleep time must be recorded for a diagnostic study. The components of the polysomnogram include the EEG, electrooculogram (EOG), electromyogram (EMG), and ECG (lead V2). Sleep staging and architecture are determined by the EEG, EOG, and EMG tracings. Potentially lethal cardiac dysrhythmias are detected by the ECG. Oxygen saturation is measured by ear oximetry. A 5% or greater decrease in arterial oxygen saturation from baseline is significant during episodes of apnea or hypopnea. Respiratory effort and breathing pattern are measured using respiratory inductive plethysmography or by measuring intrathoracic pressure changes with an esophageal balloon catheter. The distinction between an episode of central apnea and obstructive apnea is made by correlating airflow at the nose and mouth with movement of the abdominal and thoracic respiratory muscles. Central apnea occurs if both airflow and respiratory muscle movement stop simultaneously. An episode of obstructive apnea occurs when airflow at the mouth and nose ceases, but respiratory muscles in the abdomen and thorax continue to move dysfunctionally.

Of particular interest are the number of respiratory events (apneas and hypopneas), the number of oxygen desaturations below 90%, and the lowest oxygen desaturation. The respiratory disturbance index (AHI) can be calculated from these data:

$$\text{AHI} = \frac{\text{Hypopneas} + \text{Apneas}}{\text{total sleep time}(\text{mins})} \text{x}\,60$$

An AHI greater than 5 is considered abnormal and an AHI greater than 20 is considered clinically significant, because EDS usually does not occur below this level. OSA also becomes clinically significant when oxygen desaturation events fall below 85%.

Respiratory Disturbance Index (RDI) is another calculation that can be used to determine severity of OSA. Similarly to AHI, it includes apneas and hypopneas in its calculation, but also includes respiratory effort related arousals (RERAs). These are flow limitations resulting in arousals from sleep. It is felt that AHI alone leads to underdiagnosis of OSA when compared to the use of RDI.

$$\text{RDI} = \frac{\text{RERAs} + \text{Hypopneas} + \text{Apneas}}{\text{total sleep time}(\text{mins})} \text{x}\,60$$

71.4.5 Site of Obstruction

After a complete presurgical evaluation, the Fujita classification can categorize the site of obstruction. Each patient is grouped: type I, oropharynx; type II, oropharynx and hypopharynx; and type III, hypopharynx. In a review of 40 OSA patients, Riley and coworkers [33] found the majority of patients had a type II obstruction (soft palate and base of tongue).

The mandible, base of tongue, hyoid, and pharyngeal wall are intimately related by their muscular and ligamentous attachments. The mandible is related to the base of the tongue by the genioglossus muscle. The tongue, through multiple muscular and connective tissue attachments, is related to the hyoid bone and to the mandible in such a way that retraction of the mandible results in a narrowing of the airway and posterior movement of

the tongue. Compensatory mechanisms exist in non–sleep-apneic patients to prevent occlusion of the airway. However, in sleep-apneic patients, these mechanisms do not exist or are unable to compensate adequately.

Obstruction of the upper airway is primarily prevented by the action of the pharyngeal dilating muscles contracting in phase with respiration. Reduced muscle tone is normal and prominent during REM sleep. However, OSA patients may have a significant reduction in muscle activity during non-REM sleep so that the pharynx becomes narrower and airway resistance increases. In patients with abnormal skeletal development, the reduction in size of the resting airway may predispose them to upper airway obstruction during sleep.

The patency of the upper airway is determined by a balance between the pharyngeal musculature and the negative oropharyngeal pressures that are generated from resistance to airflow in the nasopharynx. Because the airway of OSA patients is unstable even at rest, any structural narrowing of the airway will eventually hinder the muscular component of the balance. Collapse of the airway in OSA is primarily a result of high intraluminal negative pressures associated with hypotonic pharyngeal wall musculature and disproportionate anatomy in either the oropharynx or the hypopharynx or both. Disproportionate anatomy includes any combination of large base of tongue, long soft palate, narrow mandibular arch, shallow palatal arch, or retrognathic mandible.

71.5 Medical Treatment

Once the diagnosis has been confirmed, the treatment approach for sleep apnea is determined by the severity of the physiologic derangements and the predominant type of apnea. Regardless of the predominant type of apnea present, all patients should be cautioned that certain drugs may precipitate or exacerbate OSA. Alcohol and other CNS depressants have been shown to aggravate sleep apnea and even to precipitate apnea and oxygen desaturations in normal persons [34].

Weight loss and nasal continuous positive airway pressure (CPAP) are the initial modes of therapy that should be initiated in obese patients with moderate OSA. A study of 16 patients who lost an average of 20 kg showed fewer apneas, reduced oxygen desaturations, and less daytime sleepiness than a control group of patients who did not lose weight [35]. Many patients can relate weight gain in preceding years to an increase in severity of their OSA symptoms. Unfortunately, weight loss by dietary measures is seldom sustained, and OSA symptoms recur with weight gain. Riley and associates [36] found that 47 of 50 OSA patients who were between 20% and 100% overweight at the time of diagnosis had regained all the weight they had initially lost 5–7 years later.

The role of oxygen therapy in the treatment of sleep apnea is controversial. In a study by Motta and Guilleminault [37], the administration of oxygen increased the duration of apneic episodes and led to worsening of acidosis and hypercarbia during both REM and non-REM sleep. It is unknown how many of their patients had chronic obstructive lung disease. Other studies have shown that supplemental oxygen therapy consistently reduced the severity of oxygen desaturation and decreased the frequency of apnea [38, 39].

The combined experience of these reports suggests that oxygen therapy limited to a flow rate of 2 L/min can be used safely in most OSA patients and will produce beneficial effects on respiration. The dangers of profound hypoxemia are greater than the concerns of prolonged apnea, acidosis, and hypercarbia. The effects of oxygen therapy on a patient with severe airway obstruction or chronic respiratory acidosis should be monitored with oximetry or polysomnography.

Several drugs have been used in the treatment of OSA syndrome with variable results. The carbonic anhydrase inhibitor acetazolamide stimulates respiration by producing a metabolic acidosis. This drug reduced the number of apneas and decreased the severity of oxygen desaturations in a group of patients with central sleep apnea [40]. However, in several cases, acetazolamide given to patients with mild OSA produced more frequent OSAs of longer duration [41]. Therefore, acetazolamide is probably not indicated in the management of OSA syndrome.

Some patients with OSA benefit from the respiratory stimulant effect of progesterone, especially those with the obesity-hypoventilation syndrome [42–45]. Progesterone increases alveolar ventilation and improves oxygenation, but its effect on frequency of apnea is limited. Major side effects that limit its long-term use include decreased libido, alopecia, and impotence.

The tricyclic antidepressant protriptyline is the most effective and best-studied drug for the treatment of OSA [46]. In a study of 12 patients, Smith and colleagues [47] showed a reduction in apnea frequency and oxygen desaturation during non-REM sleep, in addition to a decrease in REM sleep. Protriptyline produces its beneficial effect by a preferential stimulation of upper airway muscle tone and by decreasing the percentage of time spent in REM sleep, thereby reducing the more severe REM-related apneas. Anticholinergic side effects such as dry mouth, constipation, urinary retention, and impotence are frequent and limit its use.

71.5.1 Oral Appliances

The use of a variety of prosthetic devices is another approach to treatment. The nasopharynx and the posterior tongue are the two anatomic areas of concern. Insertion of a nasopharyngeal airway has been used to prevent upper airway occlusion at the level of the soft palate [48]. The American Sleep Disorders Association recommends that oral appliances may be used in patients with primary snoring, mild OSA, or in those with moderate to severe OSA who refuse or are intolerant of nasal CPAP [49]. Common side effects of oral appliance therapy include excessive salivation, xerostomia, soft tissue irritations, transient discomfort of the teeth and temporomandibular joint (TMJ), and temporary minor occlusal changes. Uncommon, more serious complications include permanent occlusal changes and significant TMJ discomfort.

Removable anterior repositioning splints have been used somewhat successfully to temporarily advance the mandible while passively bringing the tongue forward with it [49–51]. The optimal amount of forward movement is between 50% and 75% of the patient's maximum protrusive distance. An important design feature of these devices is that the appliance must maintain the mandible in the forward position while the patient is asleep.

A tongue-retaining device (TRD) that pulls the tongue forward without moving the mandible forward has also been used successfully in some patients with mild to moderate OSA [52, 53]. The TRD functions by placing the tongue into a cup or bubble positioned between the anterior teeth. Surface adhesion holds the tongue in place and the appliance requires that the patient's jaw remains partially open. One disadvantage of the TRD is that the tongue is not always held forward because surface tension of the tongue in the bubble is lost after a time. The TRD and mandibular anterior repositioning splints both force nasal breathing, which can be difficult for patients with inadequate nasal airways.

Arguably, the most researched oral appliance is the Klearway titratable appliance. It features a maxillary and mandibular component connected with an adjustable screw mechanism (Fig. 71.3). The components are made of a thermoactive acrylic resin that is slightly soft at body temperatures and very compliant at high temperatures. This property decreases major tooth discomfort and considerably increases retention in those patients who have lost a significant number of teeth. A unique feature of the Klearway appliance is that it permits both lateral (1–3 mm) and vertical (1–5 mm) jaw movement during sleep, which reduces the risk of TMJ and jaw muscle discomfort. This movement also facilitates oral breathing in patients with nasal airway obstruction. The screw mechanism of the appliance allows for an 11-mm anterior movement of the mandible with a total of 44 incremental steps of 0.25 mm. In a study of 38 patients with moderate to severe OSA by Lowe and coworkers [54], the Klearway appliance reduced the RDI to less than 15/hr. in 80% of the moderate group and in 61% of the severe group.

Another commonly used and effective oral appliance is the Herbst appliance, which is an anterior mandibular positioning device. It consists of two full-coverage clear acrylic components snapped onto the maxillary and mandibular teeth connected with two rod and tube attachments that allow vertical opening, protrusion, limited lateral movement, and no retrusive movement. It is used only at night and advances the mandible 5 to 7 mm or at least 75% of the patient's maximum protru-

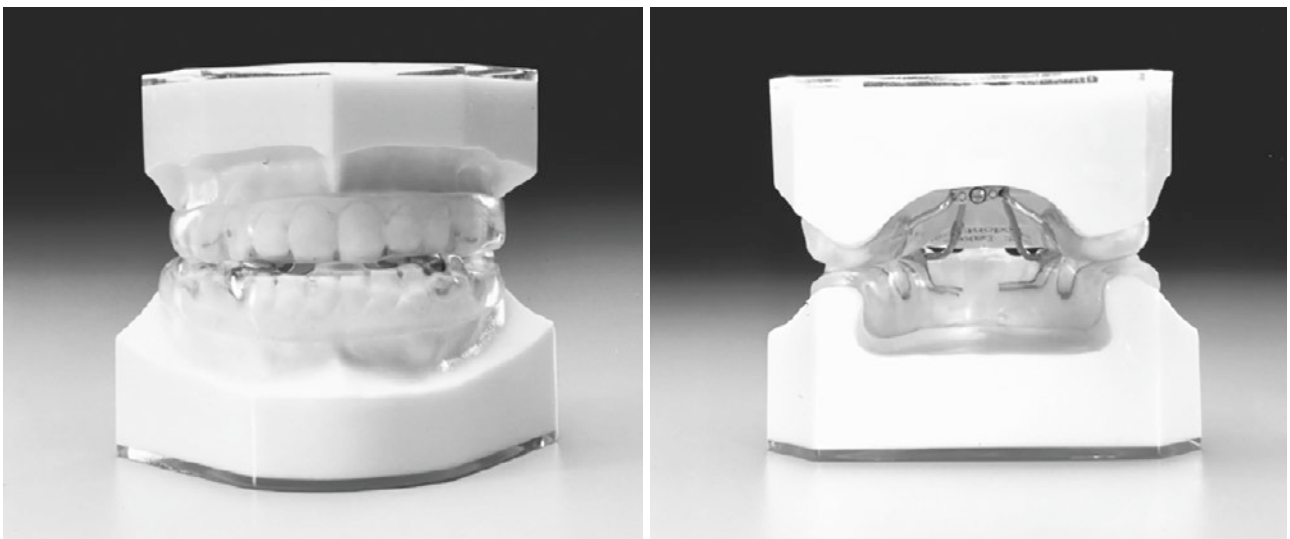


Fig. 71.3 Klearway oral appliance

sive distance. A study by Clark and associates [55] on 24 patients with mild to severe OSA using the Herbst appliance showed a significant improvement in the RDI after 4 months of appliance use in 58% of the subjects on the postappliance polysomnogram.

As with any device, compliance has been shown to be a problem with oral appliances. If appliance therapy is successful, further treatment options may include mandibular advancement surgery to achieve the same forward tongue position on a permanent basis.

71.5.2 Continuous Positive Airway Pressure

CPAP through the nose has been shown to be quite successful in treating a broad range of OSA patients and is at present the most successful nonsurgical treatment [56–58]. The nasal CPAP is administered while the patient is asleep by means of a tightfitting mask that is connected to a compressor. A CPAP of 7 to 15 cmH_2O acts as a pneumatic splint of the upper airway and prevents passive collapse of soft tissues during respiration. Stimulation of mechanoreceptors of the genioglossus muscle leading to increased airway tone has also been suggested as a mechanism of action. Sullivan and colleagues were the first to report the successful treatment of sleep apnea with nasal CPAP in 1981 [59]. In most cases, this therapy is effective in eliminating apneas and hypopneas, improving arterial oxygen saturations, reducing or eliminating excessive daytime sleepiness, and eliminating sleep disruption and fragmentation. CPAP may be combined with surgery and weight loss, or it may be used as a sole form of therapy. Although initially recommended for short-term relief of sleep apnea, the use of nasal CPAP for long-term care of patients has increased over the past few years. In recent years, bilevel positive airway pressure (BiPAP) systems that allow independent regulation of inspiratory and expiratory pressures and the newest modification in CPAP systems, auto-CPAP, have been used to more effectively treat OSA and increase tolerance and compliance [60, 61]. Auto-CPAP units adjust the CPAP throughout the night rather than delivering one fixed pressure. Optimal CPAP is delivered to the patient adjusting for positional changes, alcohol or sedative effects, sleep-state-dependent changes (REM vs. non-REM), and the effects of upper airway infections or congestion. BiPAP ($2500) and auto-CPAP ($1600) systems are more expensive than traditional CPAP ($600–$800) systems.

Despite the uniform success of this therapy, patient compliance remains a problem. Compliance rates at 12 months have been reported as low as 54% [62]. The average nightly use of CPAP is 4.8 hours and the rate of use is usually determined in the first week of use. Overall, approximately one third of patients love CPAP, one third struggle with CPAP but eventually tolerate it, and one third hate CPAP and never use it. Patient dissatisfaction results from nasal dryness and congestion, sore throat, dryness of the skin and eyes, claustrophobia, and the inability to tolerate the noise, discomfort, or mask. Careful patient selection and follow-up are essential if nasal CPAP is selected as a treatment modality.

71.6 Surgical Treatment

Surgery has been the primary form of therapy for OSA. To be successful, the surgical procedure must either bypass the obstructive area or prevent collapse of the soft tissues in the upper airway at the obstruction. Many patients and surgeons tend to view surgical treatment of OSA as a quick and permanent cure. However, a clear definition of what constitutes a cure is lacking in the literature. This problem often makes a determination of the efficacy of individual surgical procedures difficult. Only objective data obtained from a postoperative polysomnogram can be accepted as proof of efficacy for surgical procedures. Currently, the procedures used in the surgical treatment of OSA include tracheostomy, nasal surgery, uvulopalatopharyngoplasty (UPPP), and several orthognathic surgical procedures. Selection of the individual procedure is determined by the severity of the sleep apnea, the presence of a maxillofacial skeletal deficiency, the site of the obstructive segment, and the presence of morbid obesity.

71.6.1 Tracheostomy

Tracheostomy was the first efficacious surgical procedure for treating OSA, performed by Kuhlo and coworkers in 1969 [63]. It is almost 100% curative in relieving the signs and symptoms of OSA because it bypasses all the potential obstructive sites in the upper airway. After tracheostomy, there is a rapid and striking reduction in daytime somnolence and a marked improvement in sleep architecture owing to a major reduction in the frequency of arousals. Sinus dysrhythmias, bradycardia, pulmonary hypertension, hypoxemia, and apnea all improve dramatically with the procedure. Tracheostomy clearly is an effective surgical treatment for patients with OSA.

The disadvantages of a permanent tracheostomy can have a devastating effect on sleep apnea patients. Almost all patients experience psychological depression from the social and medical problems associated with a lifelong tracheostomy. The tracheostomy leaves the patient aesthetically disfigured and exposes the patient to common local complications such as bleeding, infection,

pain, and granulation tissue formation. Patients are also at increased risk for the more serious complications of tracheal stenosis or erosion into an adjacent blood vessel. Because of these disadvantages and complications, a permanent tracheostomy should be reserved for severe cases of OSA with significant cardiovascular symptoms. Simmons and coworkers [64] have suggested that tracheostomy should be the primary therapy for all patients who spend substantial time in severe oxygen desaturations below 50% and for those who have life-threatening cardiac dysrhythmias during sleep apnea. Tracheostomy may also be used as an interim treatment until adjunctive procedures to reconstruct the upper airway are completed.

71.6.2 Nasal Surgery

Significant obstruction in the nasal cavity has been shown to cause excessive daytime sleepiness, sleep fragmentation, hypopneas, and periodic breathing during sleep [65]. In most patients with moderate to severe OSA, nasal obstruction is not the major contributing factor. As a general principle, increased nasal resistance is known to increase collapsibility of the pharynx. These obstructions may be due to a deviated nasal septum, nasal polyps, or enlargement of the turbinates. A thorough nasal evaluation is critical, as this may improve adherence to positive airway pressure (PAP) treatment modalities [66]. Specifically, patients with posterior septal deviation cannot tolerate PAP, and thus nasal endoscopy is a critical portion of the nasal evaluation. In these patients, septoplasty, nasal polypectomy, inferior turbinectomies, and/or nasal valve stabilization are usually helpful as adjunctive surgical procedures in the treatment of OSA. Unless the obstruction in the nasal cavity is severe, surgical correction usually will not yield any significant improvement on a repeat polysomnography.

71.6.3 Uvulopalatopharyngoplasty

The oropharynx and soft palate can cause significant airway obstruction during sleep. At least 10% of persons older than 40 years snore regularly and significantly. Loud and intermittent snoring is found in almost all patients with OSA. In many cases, habitual snoring is present for many years before sleep apnea is diagnosed. Ikematsu [67] followed a large number of habitual snorers over several years and found that 91% of these patients had decreased oropharyngeal dimensions and longer soft palates and uvulas than normal subjects. He was able to eliminate their snoring by surgically excising the excessive soft tissue in the palatal folds and partially excising the uvula.

With minor modifications, Simmons and coworkers [64] and Fujita and associates [68] popularized the UPPP for the treatment of OSA. The procedure was designed to eliminate oropharyngeal obstruction by performing a tonsillectomy and adenoidectomy, excising the uvula, removing redundant lateral pharyngeal wall mucosa, and resecting 8–15 mm along the posterior margin of the soft palate. With this technique, there is an increased risk of velopharyngeal insufficiency.

UPPP results in symptomatic improvement in the patient and eliminates habitual snoring in almost all cases. However, reports show that significant objective improvement on the postoperative polysomnogram ranges only from 41% to 66% [62, 64, 69, 70]. This procedure only eliminates the obstruction at the level of the soft palate and does not address obstructions occurring in the hypopharyngeal and base of tongue areas. Many patients have more than one site of obstruction. If UPPP is performed when the presurgical evaluation demonstrates obstruction localized to the soft palate-tonsil area, the success rate of the surgical procedure approaches 90% treating OSA [28, 31].

The surgical technique of UPPP varies to some degree by patient and surgeon, but the basic goal is to shorten the palate and widen the PAS (Fig. 71.4). A mucosal incision is created with electrocautery on the anterior surface of the soft palate. The dissection is frequently carried laterally to include the palatine tonsil. The tonsillar bed is coagulated and hemostasis achieved. Palatal muscle is excised and mucosa from the nasopharynx is pulled forward for primary closure. Multiple interrupted resorbable sutures are placed. If the tonsil is removed, the mucosa of the anterior fauces pillar is closed to the posterior fauces pillar. This attempt to remove redundant pharyngeal tissue and stretch or tighten the posterior pharyngeal wall results in constriction. In addition, frequently by shortening the soft palate, the width of the soft palate is thickened, as demonstrated cephalometrically. Lymphoid tissue from the tonsillar fossa can be removed separately or in conjunction with the uvula (Fig. 71.5). The amount of velum to be excised is determined by the location of palatal competence and closure of the nasopharynx. These can be estimated or identified during nasopharyngoscopy. Palatal incompetence can occur but usually is of minimal concern if the patient swallows carefully. Pain with swallowing usually lasts for several weeks.

Complications from UPPP are related to changes in palatal function. Permanent velopharyngeal incompetence occurs in approximately 5% of patients and is more common during the first 2 months postoperatively.

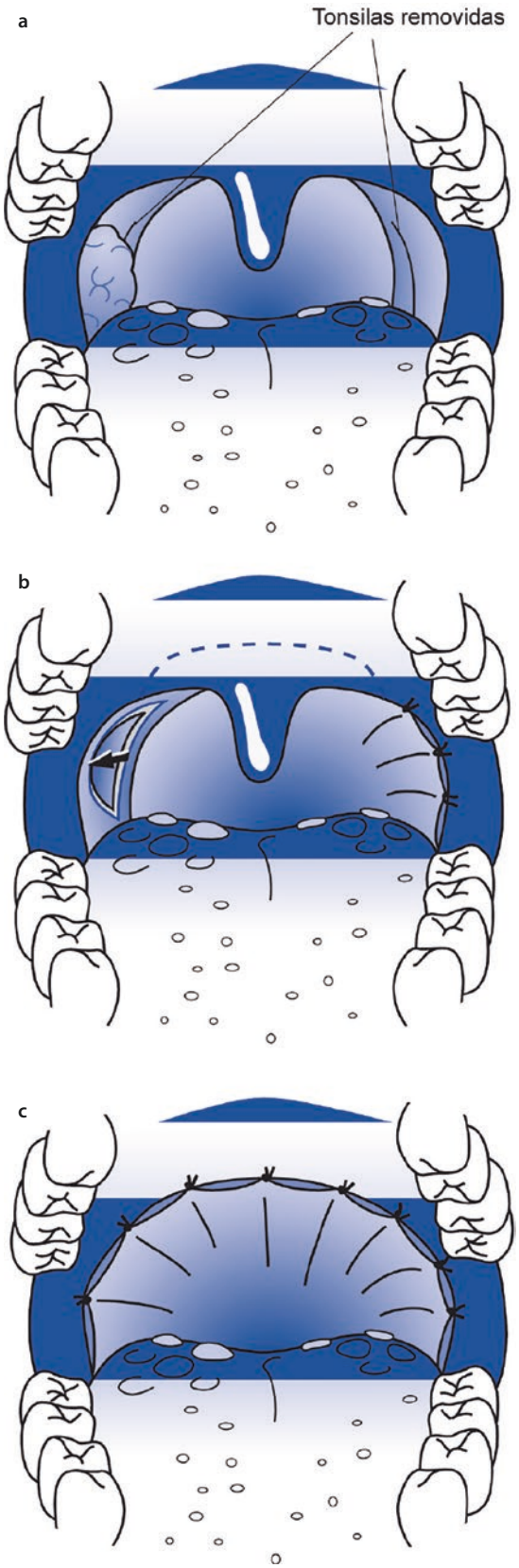


Fig. 71.4 **a–c** Uvulopalatopharyngoplasty. Tonsils and uvula are removed and the anterior pillar is closed to the posterior pillar. (Adapted from Tiner and Waite [26], p. 1540)

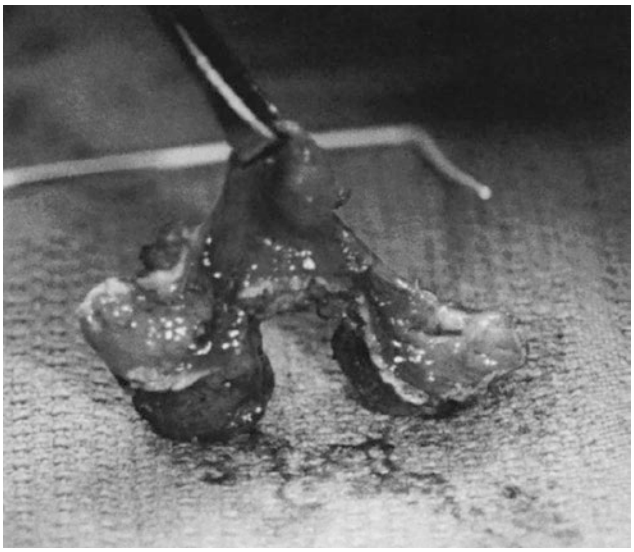


Fig. 71.5 Surgical specimen of tonsils and uvula. (Reproduced with permission from Tiner and Waite [26], p. 1541)

Patients experience occasional reflux of liquids into the nose and mild nasal air escape during speech. However, hypernasal speech and changes in the quality of the patient's speech are usually not seen. Simmons and colleagues [69] reported a 5% to 10% rate of minor wound infections that resolved with antibiotics. Palatal stenosis is definitely a risk with this operation and occurs in approximately 1% of patients. It occurs more frequently with excessive resections of the posterior tonsillar pillars and injudicious use of electrocautery. Postoperative pain after UPPP is significant, and narcotic analgesia should be used with caution to prevent sedation-induced exacerbation of OSA. Postsurgical deaths have resulted from the combination of pharyngeal edema and narcotic use.

71.6.4 Laser-Assisted Uvulopalatoplasty

In the late 1980s, Dr. Yves-Victor Kamami (Paris, France) designed a procedure to reshape and recontour the soft palate under local anesthesia with a CO_2 laser to treat snoring and selected patients with OSA syndrome [71]. He originally named the procedure "laser resection of the palatopharynx" (LRPP). Initially, the procedure was accomplished in four or five sessions spaced at monthly intervals. Over time, the procedure evolved into a one-stage technique for most patients. It consisted of two paramedian vertical incisions placed lateral to the uvula extending up toward the junction of the hard and soft palates for 2–3 cm. A second horizontal incision was placed just under the roof of the uvula, leaving a small uvula to prevent centripetal scar formation. Over a 5-year period, Kamami [71] treated 63 OSA

patients with this technique. The RDI was reduced by more than 50% in 55 patients that were classified as successful responders. The RDI improved from 41.5 to 16.9 for the average responder, and for the entire group, the average RDI improved from 41.3 to 20.3. In the early 1990s in the United States, Dr. Yosef Krespi modified the procedure and renamed it "laser-assisted uvulopalatoplasty" (LAUP). He initially used the procedure to treat loud habitual snoring. In a study of 280 patients treated in the office under local anesthesia, 84% were cured with an average of 2.7 sessions [72]. Overall results for OSA patients treated with LAUP are far less encouraging, with an average successful surgical response of 52.2% [73]. Based on these findings, the current main indications for LAUP include loud habitual snoring, upper airway resistance syndrome, and mild OSA (apnea index <20). All snoring patients who elect to undergo LAUP should be evaluated for OSA preoperatively and again postoperatively if OSA was previously diagnosed. If not, then the patient and surgeon may be lulled into a false sense of security by eliminating the snoring without eliminating the undiagnosed OSA, potentially increasing patient morbidity and mortality [74].

The most common complication after LAUP is a moderate to severe sore throat. Patients experience pain 8–10 days after surgery and reach their peak pain intensity on the fourth or fifth postoperative day. Pain control is achieved with oral analgesics and anesthetic gels. The risk for velopharyngeal insufficiency is low because the procedure is frequently done in stages and the surgeon has the opportunity to evaluate speech and soft palate function after each session. Patients are also at low risk for bleeding and infection. The great majority of patients can eat, drink, and speak almost immediately and can resume full activities the following day.

71.6.5 Uvulopalatal Flap

Given the morbidity of UPPP & LAUP procedures, the uvulopalatal flap may also be considered, which Powell et al. described in 1996 [75]. This procedure achieves anatomic results that are similar to those achieved with the UPPP/LAUP, but with less postoperative discomfort and fewer complaints of thickened secretions or foreign body sensation. This involves the reposition of the soft tissue of the palate anteriorly and anchoring them to the hard palate. The muscles are preserved, decreasing the risk for VPI.

Poor candidates for the UPF are those with a long, thick uvula and significant palatal redundancy.

71.6.6 Tongue Surgery

A variety of procedures have been developed to address tongue base obstruction. The tongue is a mobile part of the airway and can cause obstruction at the retrolingual space either due to glossoptosis or hypertrophy. Riley et al. assessed UPPP failures with cephalometric analysis and concluded that the base of tongue was the cause of persistent obstruction [28]. Schwab et al. examined upper airways of patients with OSA using magnetic resonance imaging and determined the collapse of the lateral pharyngeal wall was a significant component of sleep-related airway obstruction [76]. Thus motivation to improve surgical success rates motivated the search for surgical procedures to improve the reconstruction of these specific tongue base obstructions.

Debulking procedures such as lingual tonsillectomy, laser midline glossectomy, lingualplasty have varied results. The goals are to specifically reduce tissue, improve tension, and increase airway space. These procedures ultimately increase posterior airway space. The morbidity associated with these techniques is significant, with postoperative pain and bleeding being among the highest risk factors. The long-term efficacy and difficult access to the tongue base remain the main limiting factors for widespread acceptance. A predictable and safe reduction of the tongue is difficult to assess [77]. Alternative procedures such as radiofrequency tissue ablation and tongue base suspension sutures are more commonly utilized.

Radiofrequency tissue ablation is delivered via an insulated probe at a frequency of 465 KHz. This reduces the tongue volume by causing coagulation necrosis and secondary healing by scar and muscle contraction. The electrode is passed through multiple locations throughout the tongue. Unfortunately, most patients require multiple staged treatment sessions. The procedure can be performed under local anesthesia and in the outpatient setting; the upper airway is typically protected with either nasal CPAP or a tracheostomy.

Tongue base suspension is performed by making an incision at the lingual frenulum with a screw placed at the genioturbicle. A permanent suture is then passed through the paramedian tongue musculature along the length of the tongue, to the base of tongue and then back to the anchored screw. This allows for the entire tongue base to be pulled anteriorly, increasing posterior airway space. The advantage in this technique is that it is nonexcisional and potentially reversible.

The evolution of robotic surgery, which can allow for direct visualization through a narrow port, can allow for ease of access, improvement of visualization in order to address hypertrophic tissue, fat, and the epiglottis.

71.6.7 Orthognathic Surgery Procedures

Various orthognathic surgical procedures have been described for the treatment of OSA. The majority of patients have airway obstruction at the level of the soft palate and at the base of the tongue (type II obstruction). Orthognathic surgical procedures can change the size of the airway in several regions. Mandibular advancement and genial advancement work by changing the position of the mandible and hyoid bone with subsequent effects on the genioglossus and hyoglossus muscles. OSA patients with identifiable craniofacial anomalies can clearly benefit from a variety of these procedures. These procedures are typically performed if previous upper airway surgeries have not completely improved OSA symptoms. It should be noted that patients who complete these procedures have higher success than those treated with nasal CPAP.

Tricks and Tips

- Consider orthodontic support and consent for postsurgical malocclusion.
- Consider VSP virtual surgical planning and benefits of counterclockwise rotation.
- Recommend about 8–12 mm advancement of pogonion.
- Consider adjunctive procedures to improve upper airway such as septoplasty, turbinoplasty, and geniotomy advancement.
- Consider reinforced rigid fixation techniques and benefits of bone grafting.
- Consider difficult airway protocols and peri-operative management.

71.6.8 Mandibular Advancement

Total mandibular advancement was the first orthognathic surgical procedure used in the treatment of OSA. Kuo and colleagues, in 1979 [78], and Bear and Priest, in 1980 [79], reported complete reversal of sleep apnea symptoms in patients with horizontal mandibular deficiency treated by mandibular advancement. More recently, Alvarez and coworkers [80] reported the successful treatment of an edentulous patient with sleep apnea by mandibular and genial advancement. This increases the retrolingual space by advancing the attachments of the genioglossus or geniohyoid musculature. The exact reason for how mandibular advancement improves OSA is not clearly known, but the suspected effect is the pulling of the tongue forward off the pharyngeal wall. This effect is created by anteriorly moving the insertion of the genioglossus and geniohyoid muscles. If this were the only factor, anterior chin procedures would be equally effective as total mandibular procedures. Variations of geniotomies have been designed to maximally pull the tongue muscles forward.

A bilateral sagittal ramus osteotomy is usually the procedure of choice for total mandibular advancement. The amount of advancement is determined preoperatively from the orthognathic surgery database. Adjunctive orthodontic treatment is frequently necessary to obtain the desired occlusion and to eliminate dental compensations that would otherwise limit the amount of advancement. After advancement with the standard surgical technique, the fragments are rigidly fixed with screws or bone plates. For large advancements of 7 mm or more, long-term stability is enhanced with a 5- to 7-day course of maxillomandibular fixation and skeletal suspension wires. In advancements of 6 mm or less, maxillomandibular fixation is usually not necessary.

71.6.9 Genial Tubercle Advancement

A rectangular osteotomy apical to the teeth, but maintaining the inferior border of the mandible, allows the genial tubercles with their muscular attachments to be maximally advanced with minimal cosmetic change (Fig. 71.6). A modified vestibular mucosal incision is

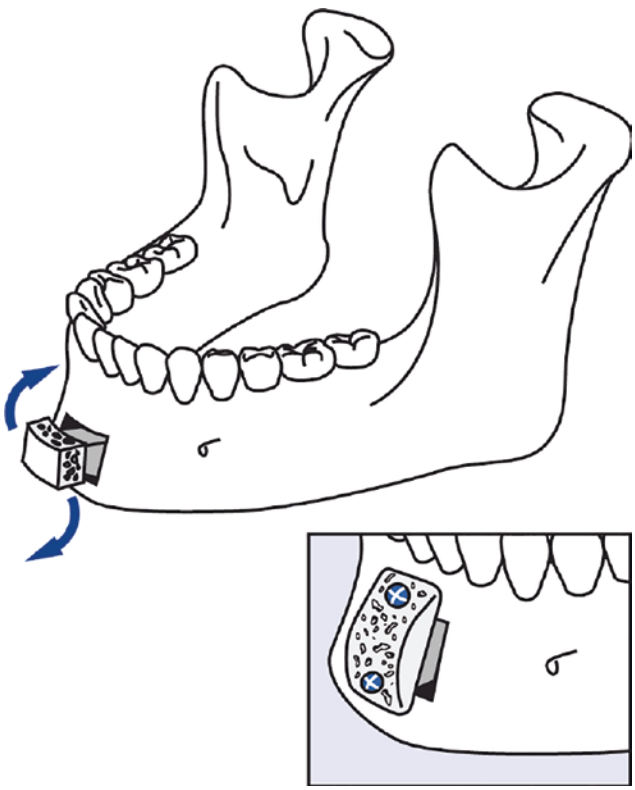


Fig. 71.6 Genial tubercle advancement. The outer table of the symphysis is removed and the inner table is secured with 2-mm screws. (Adapted from Tiner and Waite [26], p. 1542)

made in the anterior mandible. Periosteum is reflected down to the inferior border. An oscillating saw is used to make parallel horizontal cuts that include the genial tubercle. The osteotomy is designed in a shape similar to a drawer so that it can be pulled outward with the genial muscles. The bone must be broad enough cuspid to cuspid to be rotated 90° and set on top of the buccal cortex. The outer cortical and cancellous bone of the rectangle can then be removed and the inner cortex rigidly fixed with bone screws. Any hemorrhage from the cancellous bone should be controlled.

This procedure does not change the aesthetic chin or advance the anterior belly of the digastric muscle, which may be helpful in suspending the hyoid. In contrast to this procedure, a horizontal sliding geniotomy does advance the genial tubercles and the anterior belly of the digastric muscle.

71.6.10 Genial Advancement with Hyoid Myotomy and Suspension

In 1984, Riley and associates [81] described an alternative technique, in which an inferior mandibular osteotomy and an associated hyoid myotomy and suspension were used in the treatment of OSA (Fig. 71.7). This technique is similar to a horizontal mandibular osteotomy, which is commonly used for advancement genioplasty. The osteotomy is designed to include the genial tubercle on the inner cortex of the anterior mandible, where the genioglossus muscle attaches. Advancement of this segment is thought to tighten the attached musculature, preventing decrease in tone and thereby decreasing posterior repositioning of the tongue and affecting the hypopharyngeal airway. In conjunction with the osteotomy, a hyoid myotomy and suspension can be used to advance the hyoid anteriorly, allowing the epiglottis and base of tongue to advance anteriorly. The body and greater cornu of the hyoid are isolated through a submental incision. The infrahyoid muscles are transected, taking care to remain on the hyoid bone at all times to avoid injury to the superior laryngeal nerves (see Fig. 71.7a). This allows the hyoid bone to be pulled anteriorly and superiorly. Strips of fascia or nonresorbable suture are passed around the body of the hyoid and attached to the intact portion of the anterior mandible to complete the hyoid suspension.

In 1989, Riley and colleagues [82] published a review of 55 patients with OSA who were treated with inferior mandibular osteotomy and hyoid suspension. Forty-two patients had obstruction at both the oropharynx and the hypopharynx and received concomitant UPPP and inferior mandibular osteotomy with hyoid myotomy and suspension. The remaining six patients were determined

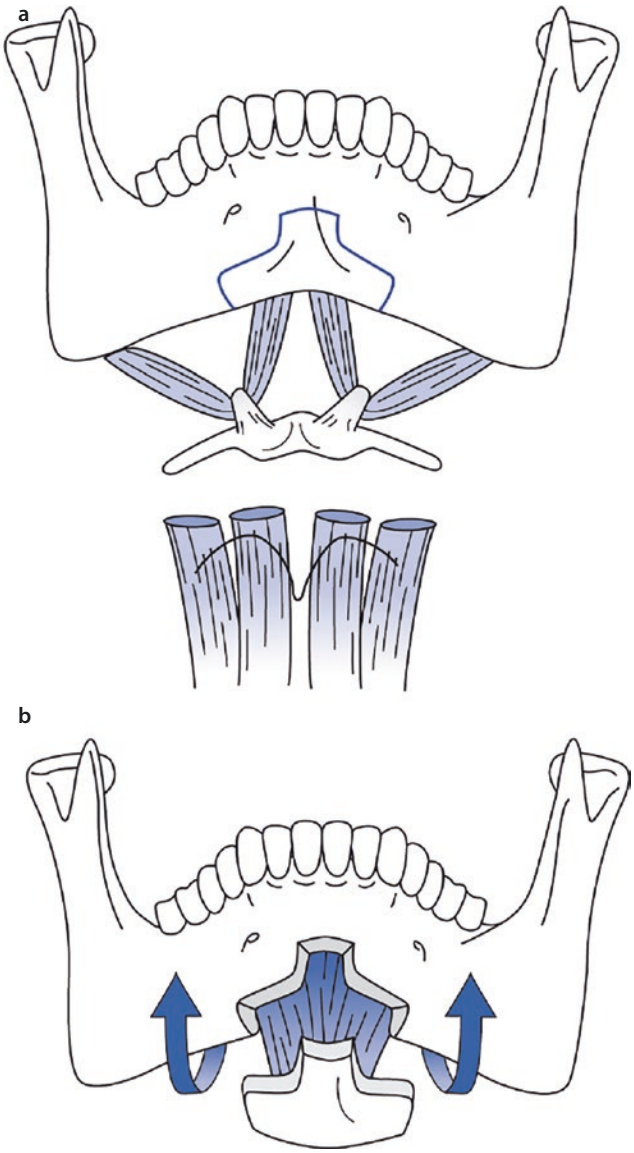


Fig. 71.7 Inferior mandibular osteotomy and hyoid myotomy. **a** Omohyoid, sternohyoid, and thyrohyoid muscles released (see Fig. 71.8 for more detail on muscular relationships). **b** Inferior segment is advanced anteriorly and locked on the anterior mandible. (**a**, **b**, Adapted from Tiner and Waite [26], p. 1543)

to have obstruction localized to the base of the tongue and underwent the osteotomy and hyoid suspension only. All patients were reevaluated by polysomnography 6 months after surgery. Thirty-seven patients (67%) were considered to be responders to surgery based on the polysomnogram results. Genioglossus advancement ranged from 8 to 18 mm with a mean of 13 mm. All responders to surgery showed significant improvement in their RDI and oxygen desaturation events. Eighteen patients (33%) were considered nonresponders and failed to show significant improvement by polysomnography. The presence of preexisting chronic obstructive

pulmonary disease (COPD) was found to be a determining factor in increasing the risk of failure.

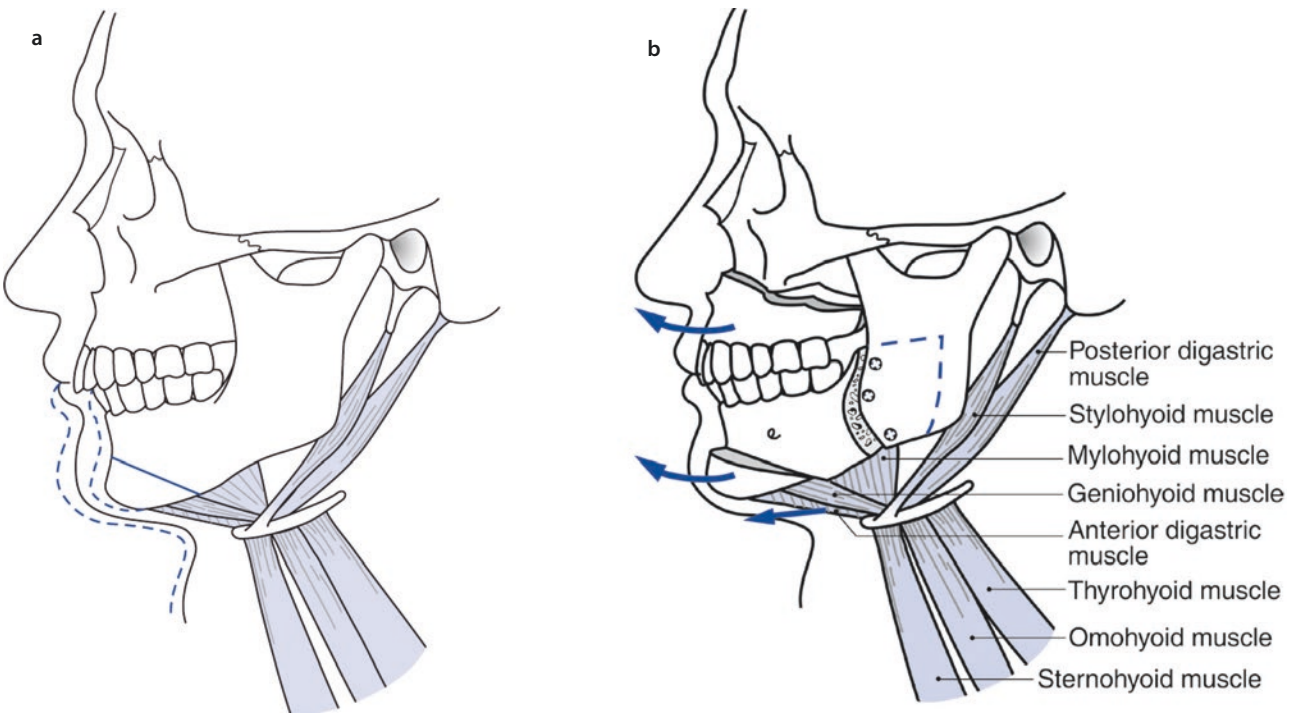
In 1994, Riley and colleagues [83] reported on a new modified technique for hyoid suspension that fixed the hyoid to the thyroid cartilage instead of the anterior margin of the mandible. When this modified technique was performed with inferior mandibular osteotomy, in lieu of the original hyoid suspension technique, the surgical response rate (with or without UPPP) was raised to 79.2%. The five nonresponders in this study of 24 patients achieved postoperative RDI values close to levels at which they would have been considered surgical responders.

Long-term follow-up of these patients has shown that the indication for this procedure is limited. Patients with normal pulmonary function, normal skeletal mandibular development, the absence of obesity, and moderate OSA are candidates for treatment with inferior mandibular osteotomy with hyoid myotomy and suspension.

The most serious reported complication from a hyoid suspension has been severe aspiration in one patient, in which the thyrohyoid membrane was totally sectioned [33]. Other complications have included wound infections, transient sensory disturbances of the mental nerve, and mandibular fracture. An advantage to hyoid suspension is that it circumvents the need for maxillomandibular fixation and does not affect the occlusion.

71.6.11 Maxillomandibular Advancement

Combined advancement of the maxilla and mandible (bimaxillary surgery) with or without hyoid suspension is the most recent and efficacious surgical procedure for the treatment of OSA. The surgical technique includes a standard Le Fort I osteotomy in combination with a mandibular sagittal split osteotomy for advancement of the maxilla and mandible. A concomitant inferior mandibular osteotomy with or without hyoid myotomy and suspension, as previously described, is also performed. The goal is to advance the complex as far anteriorly as possible. This may result in a significant facial change, which is most often favorable (◘ Figs. 71.8 and 71.9). Several authors have described the use of MMA in treating large series of OSA patients [84–89]. In a series of 23 patients, Waite and coworkers [84] performed a high sliding horizontal geniotomy without the hyoid myotomy and suspension. All patients were reevaluated by polysomnography at 6 weeks postoperatively. The surgical success with MMA was 65% based on a postsurgical RDI of less than 10. Riley and associates [85] reported the largest series of OSA patients treated with MMA in which 98% (89 of 91) were successfully treated based on a postoperative RDI of less than 20 with at least a 50% reduction in the RDI compared with the preoperative study. It should be noted that 67 of the 91 patients (74%) did not receive phase 1 therapy based on their two-phase



◘ **Fig. 71.8** Schematic drawings of preoperative **a** and postoperative **b** two-jaw advancement, genial advancement, and hyoid suspension. (**a**, **b** Adapted from Tiner and Waite [26], p. 1544)

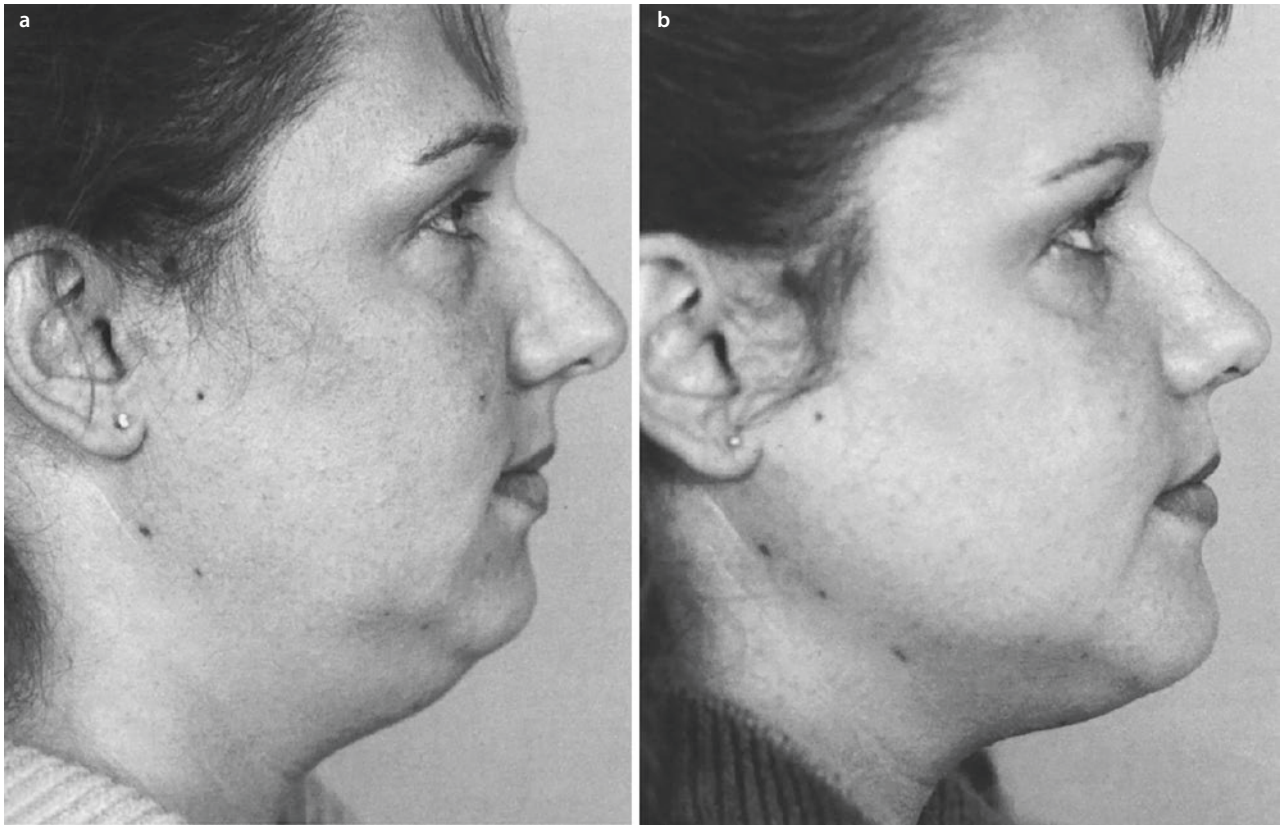


Fig. 71.9 Preoperative **a** and postoperative **b** photographs of a patient with obstructive sleep apnea syndrome who underwent two-jaw surgery with a genial advancement. (**a**, **b** Reproduced with permission from Tiner and Waite [26], p. 1545)

protocol for reconstruction of the upper airway. Despite this, the MMA was labeled a phase 2 procedure. In 1997, Hochban and colleagues [86] reported a 98% success rate on 38 OSA patients consecutively treated with a 10-mm MMA as the primary surgery, without any adjunctive procedures. Their criteria for success were based on the more rigid postoperative RDI of less than 10. Patient selection for MMA was based on subjective symptoms of excessive daytime sleepiness, an RDI of greater than 20, and specific craniofacial characteristics determined cephalometrically. Only two patients, who were morbidly obese, were treated surgically. Based on their excellent results, the authors concluded that a stepwise algorithm of staged surgical procedures was not justified. In a series of 50 OSA patients consecutively treated with MMA, Prinsell [87] reported a 100% success rate based on a postoperative RDI of less than 15, an apnea index (AI) of less than 5, or a reduction in the RDI and AI of greater than 60%. In this series, occasional concomitant nonpharyngeal procedures and an anterior interior mandibular osteotomy were accomplished with the MMA as a single-stage operation. In 1999, Lee and coworkers [88] proposed a three-stage protocol for the surgical treatment of OSA patients. All 35 patients in their series had type II obstruction with collapse at the oropharyngeal and hypopharyngeal areas. Stage 1 reconstruction consisted of a UPPP and inferior sagittal osteotomy with genioglossus muscle advancement, or an anterior mandibular osteotomy. If stage 1 was unsuccessful, patients advanced to stage 2, which consisted of MMA with rigid fixation. A hyoid myotomy and suspension was the sole component of stage 3 reconstruction. Based on postoperative polysomnography, 69% (24 of 35) were considered surgical respondents based on an RDI of less than 20. Of the 11 stage 1 failures, three elected to proceed to stage 2 reconstruction with MMA. All patients who underwent MMA had a postoperative RDI of less than 10, indicating a 100% response rate. No patient required stage 3 reconstruction. Bettega and associates [89] treated 51 consecutive OSA patients according to the Stanford two-step surgical procedure. Forty-four patients had phase 1 surgery with a success rate of 22.7% (10 of 44). Twenty patients underwent MMA as part of phase 2 in the protocol. Of these, 75% (15 of 20) were considered to be surgical responders based on a postoperative RDI

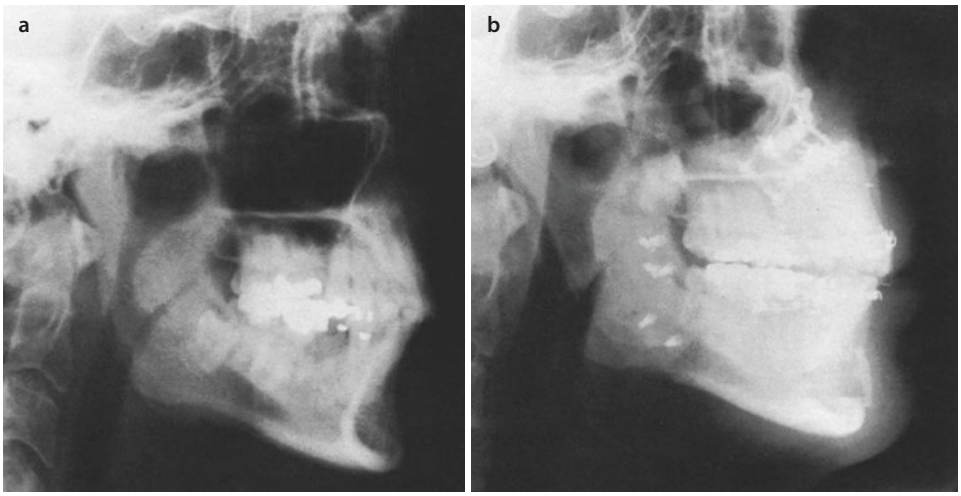


Fig. 71.10 Preoperative **a** and postoperative **b** radiographs of a two-jaw surgery and genial tubercle advancement for sleep apnea. (**a**, **b** Reproduced with permission from Tiner and Waite [26], 1992; p. 1546)

of less than 15 and at least a 50% reduction in the RDI. Of the five failures, three had postoperative RDIs of less than 20.

The PAS consistently increases with MMA. However, there is no direct relationship between the gain in PAS and the remission of sleep apnea. MMA is effective for patients who have obstruction at the base of the tongue. This surgical treatment is the most efficacious procedure for expanding the pharyngeal airway and improving or eliminating OSA. It remains the best current alternative to tracheostomy [90]. Indications for this procedure include severe mandibular deficiency (SNB <74 degrees), morbid obesity, severe OSA (RDI >50, oxygen desaturations <70%), hypopharyngeal narrowing, and failure of other forms of treatment [91]. The success rate of MMA appears to increase when adjunctive procedures such as UPPP, partial glossectomy, septoplasty, or turbinectomies are included in the treatment plan. This lends support to the theory that most OSA patients have multiple levels of obstruction.

Adjunctive orthodontic therapy is usually indicated in patients selected for MMA. Presurgical orthodontics improves the postoperative occlusion and eliminates preexisting dental compensations that would otherwise limit the amount of advancement. Maximum advancement of the facial skeleton and maintenance of a functional occlusion and acceptable aesthetics are the goals of surgical-orthodontic correction.

The osteotomies are rigidly fixed with miniplates and bicortical screws (Fig. 71.10). With large advancements (>7 mm), skeleton suspension wires and a short course of maxillomandibular fixation (1 week) can be used to reduce surgical relapse. Potential complications of MMA include surgical relapse, nonunion, bleeding, malocclusion, infection, unfavorable changes in facial appearance, and permanent or temporary sensory disturbances of the inferior alveolar and infraorbital nerves.

The long-term skeletal stability of MMA has been shown to be quite good. Louis and colleagues [92] showed a mean relapse of 0.9 ± 1.8 mm among 20 maxillary advancement patients who underwent MMA for OSA. The mean follow-up period was 18.5 months (range 6–29 months). When the patients were divided into three groups reflecting small (≤6 mm), medium (7–9 mm), and large (≥10 mm) advancements, there was no statistical difference in the measured relapse among the groups. Rigid fixation was achieved with four miniplates and no bone grafts were used in any of the maxillary advancements. Nimkarn and coworkers [93] reported on 19 OSA patients who underwent MMA with simultaneous genioplasty and found relatively stable long-term (>12 months) surgical stability of the maxilla and mandible. Maxillary and mandibular advancement was stable over the long term in both the vertical and the horizontal planes. With the exception of gonion in the vertical plane, there was no statistically significant correlation between the amount of surgical advancement and the amount of postsurgical instability.

Treatment planning MMA for OSA should consider the benefits of counterclockwise rotation and maximizing the advancement of pogonion. Minor advancements of the maxilla, A point, and incisors may allow for about 10 mm of advancement at the chin. Intuitively, one would think that advancement of the mandible is key to pulling the tongue forward and opening the posterior airway.

71.6.12 Mandibular Setbacks

In a small number of patients, a mandibular setback procedure can be the initiating factor in the development of OSA. Riley and associates [94] reported on two women who developed OSA syndrome after mandibular osteotomies for correction of class III malocclusion and skeletal prognathism. Neither patient had any symptoms of sleep apnea before surgery. Postoperatively, both patients began to snore loudly. Evaluation by polysomnography confirmed the presence of OSA syndrome. A comparative examination of the preoperative and postoperative lateral cephalograms of each patient showed a more inferiorly positioned hyoid bone and a narrowing of the pharyngeal airway as a result of the mandibular setback procedure.

In an attempt to identify those patients potentially at risk for OSA, all patients who are planned for mandibular setback procedures should be questioned preoperatively and postoperatively about the presence or absence of snoring, excessive daytime sleepiness, or observed apneas during sleep. Although the vast majority of patients who undergo mandibular setbacks are able to adapt to the changes in the skeletal and muscular apparatus, a subset of patients may be at risk for developing overt signs of OSA after mandibular setbacks.

71.7 Complications

Surgical complications in treatment of OSA may be severe and different from those related to routine orthognathic surgery. Sleep apnea patients are often older and more obese and, by the nature of their disease, have difficult airways. Obesity is a known co-morbidity associated with multiple medical issues such as hypertension, diabetes, heart disease, pulmonary dysfunction, poor intravenous access, and difficult airway management.

Respiratory failure is the major complication of airway surgery. Respiratory obstruction is a common cause of death in sleep apnea, especially after UPPP. Anesthesia protocols exist for management of the sleep apnea patient owing to physical characteristics such as obesity, short neck, small jaw, large tongue, difficult laryngoscopy, and poor mask ventilation. Jet ventilation and fiberoptic intubation are valuable techniques [95].

Obesity, hypertension, COPD, myocardial infarction, and stroke are more common in the patient with OSA [96]. The surgeon should anticipate these medical problems and be prepared to manage appropriately. Standard intravenous access and central lines are much more difficult to obtain in the obese patient. Large obese surgical patients are more difficult owing to intraoral access, and obesity is often associated with increased infection and poor healing. Hypotensive anesthesia may be contraindicated and postoperative blood pressure management more complicated in the presence of pain control. Renal perfusion is often reduced in this group and antihypertensive medication will complicate fluid management. Older patients with hypertension and poor cardiac function need closer management of fluid status than the routine healthy orthognathic patient. People with COPD and low baseline oxygen saturations may desaturate quickly if oversedated postoperatively, and poor oxygen saturation may contribute to poor healing, dehiscence, and bone nonunion. Coronary artery disease, especially stents, arrhythmias, and strokes, is often treated with anticoagulation or antiplatelet therapy. Extensive upper airway surgery such as MMA or tongue base or palate surgery is at a great risk for hemorrhage.

Patient positioning on the operating room table for large obese patient is clearly a challenge. Many sleep apnea patients just do not fit the average operating room table and careful positioning with padded support must be provided. Aggressive arm slings that wrap the shoulder upward often produce chronic pain and postoperative musculoskeletal pain.

Deep venous thrombosis has a higher association with extended surgery time, abdominal girth, and obesity. Sequential compression devices should be used on all OSA patients and maintained until adequate ambulation [97, 98]. Antiplatelet therapy or low-dose heparin is not recommended owing to the continued bleeding in the maxilla, sinus, and nasal area. Soft tissue ecchymosis may create airway obstruction.

Malocclusion and inferior alveolar nerve paresthesia are the two most common postoperative complaints. MMA surgery is much different than orthognathic surgery because the intention is to maintain the same occlusion but move both the upper and the lower dentition forward, thereby increasing the upper airway space. Large skeletal changes are often necessary to open the airway but also pose greater challenges in maintaining the intricate relationship of intercuspation. Even a small percentage of relapse in a large advancement will result in a nonfunctional malocclusion. Patients with presurgical abnormal occlusion but good dentition should be considered for presurgical orthodontics. Patients must know in advance the risk of malocclusion and realize orthodontics is not considered medically necessary. Because the magnitude of skeletal change is greater than with orthognathic surgery, the skeletal fixation techniques must be modified.

Paresthesia may be more problematic in this patient subset owing to the greater skeletal change and the age distribution. Older age groups typically do not adjust to nerve injury as well as younger patients. Most nerve

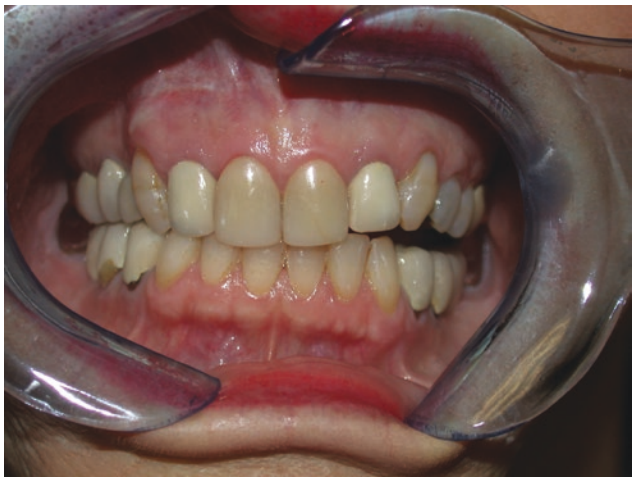


Fig. 71.11 Postoperative malocclusion of the posterior dentition due to a deep curve of Spee and an extensive prosthetic bridge

injuries improve slowly with time, but low-dose medical therapy with clonazepam, amitriptyline, or gabapentin can be very beneficial. Microneurosurgery in this patient group is unknown. Even complaints of paresthesia in the maxilla are reported that may be due to larger Le Fort advancements causing delayed infraorbital reinnervation and/or larger defects in the lateral wall of the sinus associated with chronic sinusitis. Most patients will tolerate mild paresthesia for the benefit of better breathing.

Typical patients that present for MMA are middle aged and, therefore, often have unique dental considerations, such as periodontal disease, missing teeth, impacted teeth, implants, and special prosthodontic factors. These factors may prevent presurgical orthodontics, but also complicate treatment. Dental implants and extensive bridges do not behave like independent teeth with physiologic movement (Fig. 71.11). Sometimes, malocclusion can be corrected by equilibration, new restorations, or even simple extractions. Periodontal disease, bone loss, and calculus can often be the cause of surgical wound infections. The geniotomy incision is the most likely to dehisce and become infected, perhaps owing to age-related periodontal factors.

Skeletal relapse is a common problem in all surgery, but in orthognathic surgery, this is often corrected by the orthodontist. Up to 20% relapse is reported in orthognathic surgery, which would be only 1 mm if the skeletal change was 5 mm. But in MMA surgery, the skeletal change is often 10 mm and a 20% relapse would be 2 mm, which may be beyond the envelope of treatment in orthodontics or equilibration. Larger skeletal changes imply larger bone gaps and less bone contact. Larger bone gaps also imply longer bone plates with greater flexibility. Therefore, the surgeon must use techniques

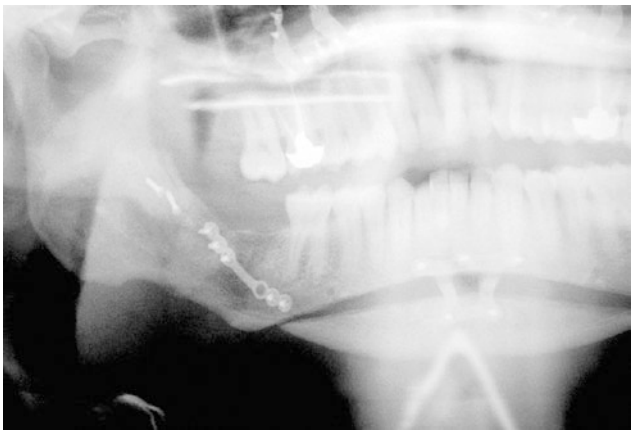


Fig. 71.12 Plate fracture in the early postoperative period

that reduce relapse such as bone grafting and reinforced plating materials.

Hardware failure and plate fracture are not uncommon in skeletal surgery (Fig. 71.12). Routine fixation systems are not designed for MMA surgery, and thin flexible plates used in larger bone defects will bend and flex, ultimately leading to stress fractures. This can be worsened by the surgeon bending and work-hardening the plate to fit an osteotomy gap. Large maxillary advancements anatomically do not have good bone apposition, and chronic movement with mastication will cause delayed healing, nonunion, or stress fracture of the plating system. Prebent OSA plates are stronger in the gap area and control the amount of advancement. Prebent OSA plates do not require the surgeon to bend the plate to fit the osteotomy step and are, therefore, stronger. Bone grafting the lateral wall of the maxilla is advisable owing to the limited bone contact and prevention of soft tissue ingrowth. This will improve stability, increase healing, and reduce relapse [99].

Mandibular fixation of the sagittal split osteotomy can be done in several different ways. Bicortical position screws are probably the strongest, but when they fail, the entire construct is lost. Bicortical screws may compress the inferior alveolar nerve and, if rotation of the segments occurs, there will be poor adaptation of the proximal and distal segments. Monocortical plates are perhaps kinder to the TMJ and nerve but more flexible. Stronger, stiffer alloys are available that improve the flexibility of the plate. Mandibular fixation in OSA patients usually requires a modification because the advancement is greater, there is less bone apposition, and the patients are stronger and larger. Double monocortical plate per side or superior bicortical position screws with a monocortical plate will increase the engineering support (Figs. 71.13 and 71.14). Fixation of the osteotomy is ultimately the surgeon's choice based on multiple factors.

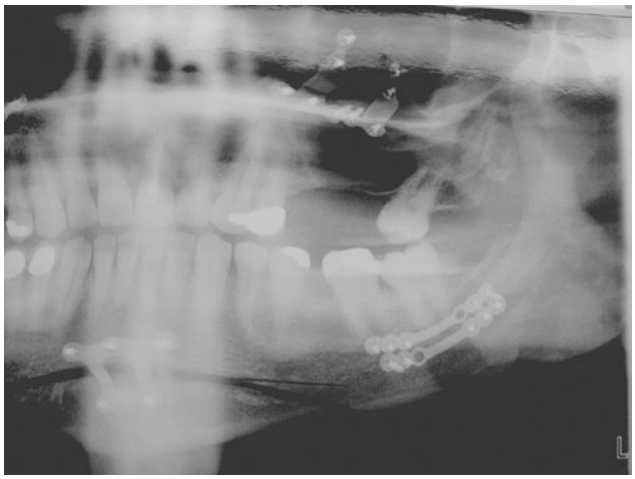


Fig. 71.13 Double monocortical fixation increases strength in large advancements

Table 71.1 Results of maxillomandibular advancement for obstructive sleep apnea[a]

Results	RDI	Desaturation[b]	Patients (*N*)	Percent of Total
Excellent	≤10	0	20	28.2
Good	≤10	≤20	26	36.6
Satisfactory	≤10	>20	15	21.1
Poor	>20	>20	10	14.1

RDI respiratory disturbance index
[a]Maxillomandibular advancement surgery results for 71 obstructive sleep apnea syndrome patients classified by polysomnography
[b]Number of oxygen desaturations <90%

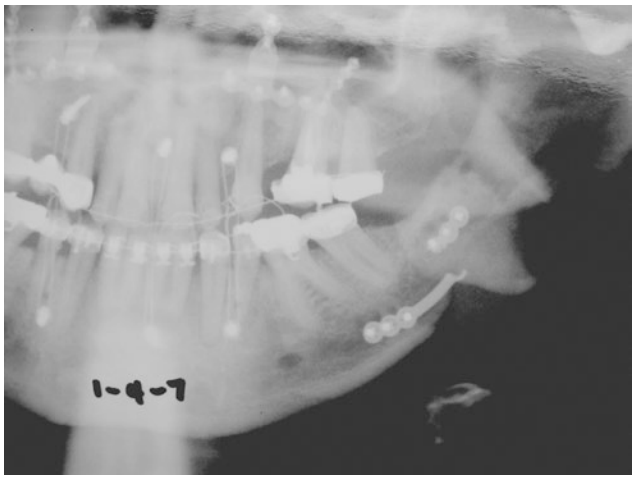


Fig. 71.14 Combination of both monocortical and bicortical fixation screws

71.8 Summary

Because the OSA syndrome is a complex disorder, the type of treatment selected should be tailored to the individual patient based on the relative risks and benefits of the therapy and the severity of the disease. Although a subset of the patients who present with OSA has an identifiable craniofacial anomaly, care must be used in choosing a simple mechanistic therapy. The success of the chosen therapy should be evaluated both subjectively and objectively. There is no clear agreement on what constitutes a cure of sleep apnea. Most authors use the RDI in assessing severity of disease and success of treatment. However, all agree that the potentially significant physiologic consequences that can be life-threatening result from hypoxemia. In some cases, patients after treatment have no oxygen desaturations below 90%, but in terms of RDI, they are considered not cured and are deemed treatment failures.

A more reasonable approach would be to define the concept of success in terms of "excellent," "good," "fair," and "poor" and to avoid using the term "cured" in assessing treatment outcomes. A widely cited meta-analysis by Sher et al. [100] describes surgical success as 50% reduction in AHI or a 50% reduction in the AI with a postoperative AI of <10.

The classic definition of surgical success should not overshadow the importance of impact from the patient's perspective. These terms could be quantitatively approached assigning lowest oxygen desaturation and RDI parameters to each one. In Table 71.1, the results of 71 patients treated by MMA are assessed by these criteria. In managing patients with severe sleep apnea, a "cure" is seldom achieved with a single surgical or medical treatment (tracheostomies excluded). However, MMA may significantly improve a patient to the point that nonsurgical therapies are more efficacious, if needed at all.

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