SLEEP APNEA

"... and it happened suddenly while he slept"
How often have you heard those words uttered following the death of a close friend or relative? Soon the event is brushed aside as a "sudden heart attack." With the growing recognition in recent years of sleep-related breathing disorders such as obstructive sleep apnea, the health professional may now be able to understand, anticipate, and perhaps prevent sudden death from sleep-induced airway obstructions.

Obstructive sleep apnea (OSA) is a potentially fatal disorder affecting nearly 7 million adult males in the United States. Its recognition has been due primarily to a rapid growth in sleep research over the past three decades. By definition, apnea is a cessation of airflow at any level of the respiratory tract, most notably in the nasal and oral pharynx, lasting at least 10 seconds.69

The classic description of OSA dates back to 1956 when Burwell described it as a "pickwickian" syndrome. The clinical features of the syndrome include marked obesity, a short and wide neck, daytime sleepiness, periodic noisy respiration, hypertension, and cardiac abnormalities. Today the typical OSA patient is a male in the fifth or sixth decade of life, of short stature, and usually markedly overweight. This syndrome is pernicious and has been implicated as a proximate cause of automobile and industrial accidents, as the victim readily falls asleep at the wheel of his automobile or while operating dangerous machinery. Daytime hypersomnia is very common because the extreme restlessness of multiple obstructive apneas makes refreshing nighttime sleep impossible.

DEVELOPMENT OF THE AIRWAY

A child's airway is unique in both its structure and function. Because the infant's larynx is positioned higher to allow for simultaneous suckling and nasal breathing, obstruction in the nose or nasopharynx may severely affect the infant who by nature is an obligate nasal breather. With growth, the larynx descends and allows for more normal adult breathing patterns.7 Symptoms of OSA have been reported as early as the neonatal period and can develop and worsen over time until a full-blown syndrome is seen in childhood.

Children with OSA present with symptoms similar to those of adults with the condition, such as daytime somnolence, nighttime snoring, and diaphoresis. However, they may also have abnormal daytime behavior ranging from aggressiveness and hyperactivity to pathologic shyness and social withdrawal. They may have hearing problems, morning headaches, frequent upper respiratory infections, failure to thrive, and obesity.

Short stature and low weight have been described in a pediatric OSA population.4,5 Goldstein et al found that eliminating obstructive apnea resulted in an increase in growth, thereby attributing the short stature to a decrease in the produc-
tion of growth hormone which is usually secreted during sleep. Small stature could thus be misinterpreted as a primary congenital anomaly when it may actually be secondary to sleep apnea.

Multiple etiologies of OSA in children have been demonstrated, including narrowing of the upper nasal airway (choanal atresia), pharyngeal hypoplasia, lymphoid tissue hypertrophy, micrognathia, hypotonia, and iatrogenic obstruction of the pharynx. Pediatric patients with craniofacial anomalies often exhibit upper airway obstruction. The structural problems associated with airway obstruction in these patients may be due to choanal atresia (Treacher Collins syndrome), malformations of the cranial base and midface (Crouzon and Apert syndromes), or mandibular hypoplasia (Pierre Robin anomaly and hemifacial microsomia) (Fig. 58–1).
In the adult OSA population, males are affected 10 to 20 times more often than females. As in the pediatric population, the most common features of OSA in adults are excessive daytime sleepiness, disturbed sleep, and loud snoring. If unrecognized and untreated, sleep apnea may have serious and potentially life-threatening consequences. Common medical complications associated with this disorder include pulmonary hypertension, right-sided heart failure, nocturnal hypoxemia, cardiac arrhythmias, and systemic hypertension. Moreover, the excessive daytime somnolence presents serious economic hardships. The patient may also experience deterioration of memory and judgment, confusion, automatic behavior, and personality changes. The degree of impairment may range from mild, e.g., falling asleep while watching television or reading, to severe, e.g., occupational or motor vehicle accidents.

Almost all apneic patients are told by their parents that they snore loudly at night. It has been reported that more than 70 per cent of adult OSA patients snored heavily throughout childhood, suggesting possible airway complications in early childhood. Marked sinus arrhythmia has been noted in OSA patients. A progressive sinus bradycardia during apnea with an abrupt reversal to sinus tachycardia at the onset of ventilation has been described. Because this pattern is so characteristic of sleep apnea syndromes, A Holter monitor is used as a screening technique in populations considered at risk. Systemic and pulmonary arterial pressures rise in association with episodes of sleep apnea but return to normal once breathing is resumed. However, the severity of the arterial pressure values varies with frequency of apneic events.

Most studies have reported that the site of obstruction in OSA patients is the oropharynx. Guilleminault in 1978 found that the posterior and lateral pharyngeal walls invaginate and may be the only abnormal finding (Fig. 58-2). In a fluoroscopic and computer tomographic study of the pharynx in OSA patients in 1983, Suratt and coworkers concluded that airway collapse occurs initially between the soft palate, tongue, and posterior pharyngeal wall. They also found that the airway posterior to the soft palate was significantly narrower in OSA patients than in controls. In 1983, Bohlman studied 33 apneic adults and 12 normals and found significant narrowing of the pharyngeal airway in all OSA patients and no narrowing in the controls. In 11 patients the narrowing was at a single level, and in 22 patients two or more levels were affected. In 1983 Haponik showed that the site of obstruction varied between the nasopharynx, oropharynx, and hypopharynx.

Other factors have been associated with OSA, including retrognathia, micro-

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**FIGURE 58-2.** Pharyngeal airway collapse commonly seen in OSA patients (nasendoscopic and diagrammatic representation): A, Left, normal pharyngeal airway lumen as seen through an endoscope; right, note collapse of the airway after Muller maneuver by patient. B, Schematic representation of anteroposterior, lateral, and coronal projections of pharyngeal airway collapse often demonstrated in the OSA patient.
gnathia, myxedema, adenotonsillar hypertrophy, elongated and thickened soft palate and uvula, deviated nasal septum, macroglossia, and neoplasm.\(^{27,72}\)

The mechanisms of obstruction are not yet fully understood. The patency of the upper airway is controlled partially by the central nervous system and is maintained by adjustment of muscular tone of the pharyngeal muscles. The forces that contribute to the tendency toward collapse during inspiration are (1) the surrounding atmospheric pressure (a constant), (2) the weight of the soft tissue of the neck (varies in individuals), (3) the local compliance of the airway walls (varies depending on fatty infiltration, edema, and other factors), and (4) the negative pressure inside the lumen of the airway during inspiration. Normally, airflow varies inversely with resistance and directly with the pressure developed between the airway and the airway opening. Guilleminault has shown that a decrease in muscle tone and/or neck fat increases resistance.\(^{20}\) This "Bernoulli" effect also contributes to collapse: If the volume of airflow is constant, the velocity of air at the constriction decreases. Thus, if there is a narrowing of the airway at one point, the tendency to collapse increases during inspiration.

DIAGNOSING OBSTRICTIVE SLEEP APNEA

Since sleep apnea has a multisystemic effect upon a patient's well-being, a team approach is necessary to diagnose and treat it. Because of this, sleep-wake centers have evolved. A sleep disorder center may have any or all of the following specialists: neurologist, otolaryngologist, cardiologist, psychologist, nutritionist, orthodontist, oral and maxillofacial surgeon, and a technical support staff. This multidisciplinary approach provides insight into the many factors that are ultimately responsible for sleep apnea.

Polysomnography (PSG) (multiphysiologic sleep recording) is the benchmark for determining whether sleep apnea exists. A number of physiologic variables are monitored during sleep and yield information regarding the patient's sleep-wake state and cardiac and respiratory function.

The basic components of PSG measurement include polygraphic recordings by means of the electroencephalogram (EEG), electro-oculogram (EOG), and chin electromyogram (EMG). Recordings of nasal and oral airflow, oxygen saturation, end-tidal carbon dioxide values, thoracic and abdominal respiratory movements, leg muscle activity, penile tumescence, and esophageal acidity are all measured (Fig. 58 - 3).

The following factors are usually noted during a typical sleep study: (1) number and length of apneas, (2) sleep latency, (3) time to first REM period, if any, (4) sleep stages, and (5) number of sleep arousals. In normal patients positive findings such as hypoventilation and apneas alone do not justify diagnosis of obstructive apnea. The frequency of events is of utmost importance in the final diagnosis. Therefore, the apnea index (AI) was developed to measure the severity of apnea. By definition, the apnea index is the total number of apneic episodes during sleep divided by the sleep time in minutes and multiplied by 60. Studies have shown that an AI of up to 5 is within normal limits, and anything greater than 5 is abnormal. Sleep-related hypopneas have been documented in most patients during sleep studies. This often compounds the problem, so an apnea-hypopnea index (AHI) has been developed and, as with AI, an AHI of up to 5 is normal in the adult.\(^{19}\) In addition to PSG, another examination in a typical sleep study is the multiple sleep latency test (MSLT), which provides an objective evaluation of excessive daytime sleepiness.\(^{19}\)

The otolaryngologist at the center evaluates the patient for any head and neck pathology, usually with the aid of an endoscope for determining the site of the obstruction. The otolaryngologist may be able to perform a Müller maneuver. This procedure has been shown by Sher et al to be highly diagnostic.\(^{58}\) In the
FIGURE 58–3. POLYSOMNOGRAPHY (PSG). MULTIPHYSIOLOGIC PARAMETRIC TESTS UTILIZED TO DOCUMENT SLEEP APNEA. A, Normal PSG examination results. Note regular airflow and EKG, minimal muscular activity, and adequate oxygen saturation. B, Abnormal PSG. Note cardiac dysrhythmias and interrupted airflow with hyperactive muscular activity consistent with snoring and airway obstruction; also note oxygen desaturation after obstructive episodes.

maneuver, the patient is instructed to occlude the nasal airway by gently squeezing the external nares with two fingers and to make a vigorous inspiration with the mouth closed. This provides general information about the degree of obstruction at specific points along the pharyngeal airway and the propensity for dynamic occlusion of the pharyngeal airway during sleep. The degree of obstruction at two levels, the soft palate and base of the tongue, is measured from 1+ (minimal movement) to 4+ (total collapse of the airway). This should be done in both upright and supine positions.
Electromyography and nasopharyngeal endoscopy have illustrated mechanisms of upper airway obstruction such as decreased activity in muscles of the upper airway, pharyngeal collapse, and active contraction of the velopharyngeal sphincter muscles. Brouillette and coworkers used fluoroscopy for the same purpose of establishing obstructive sites, and others used somnofluoroscopy to predict treatment success.

During examination, the soft palate may be found to be an obstructing factor. Performing a Müller maneuver helps identify other areas of collapse, and a lateral cephalogram can provide considerable additional information. A uvulopalatopharyngoplasty (UPPP) is designed to eliminate the mucosal and muscular redundancy in the velopharynx. In this procedure the oropharynx is enlarged to two dimensions, effectively elevating the palate away from the posterior pharyngeal wall (Fig. 58-4).

Computed tomography offers another mechanism to evaluate the airway of the OSA patient. Crumley studied the cross-sectional dimension of the airway with cine scans, providing a dynamic view of the airway during various phases of respiration in sleeping patients.

The patency of the human airway has also been evaluated through the use of cephalometrics. Initial airway evaluations utilizing this technique focused on normal growth and development in the nasopharyngeal region. Further study of the nasopharyngeal area centered on measurements of the soft palate, pharyngeal wall, and tongue position.

Earlier investigations focused primarily on speech-related problems. In a dissertation written in 1949, Hixon presented an extensive analysis of morphology and function of the oronasal and pharyngeal areas. He measured lateral cephalometric radiographs of persons with normal and nasal speech taken during rest and phonation of various vowels. Pharyngeal growth examination, velopharyngeal valving competence, tongue height, growth of the soft palate, posterior pharyngeal wall movement, and soft palate function were other areas evaluated using cephalometrics to give a better understanding of pharyngeal growth and function.

In the 1960s, work by Wildman, Engman, Bushy, Schweiger, and Chierici used cephalometrics to study craniofacial soft tissue anatomy as it related to skeletal

**FIGURE 58-4. THE POSTOPERATIVE EFFECTS OF UVALOPALATOPHARYNGOPLASTY (UPPP) AS SEEN ON CEPHALOMETRIC RADIOGRAPHS.** Note in these two UPPP patients how the operation has shortened as well as thickened their uvulas. There appear to be minimal changes in their anteroposterior pharyngeal airway widths.
landmarks. Nasopharyngeal dimension and adenoidal tissue evaluation were studied in the 1970s to examine the effect of adenoidal and tonsillar size on respiration and craniofacial and nasopharyngeal development. With the development of sleep-wake centers came a considerable increase in cephalometric literature on the airway and OSA. Hyoid bone position, soft palate length, mandibular body length and position, and soft tissue dimensions (e.g., tongue, soft palate, adenoids, tonsils) have all been evaluated and found to play significant roles in OSA. Recently, Djupesland evaluated 25 OSA patients and 10 controls cephalometrically. Findings indicated an increased soft palate length and thickness, closer contact between the tongue and soft palate, a more inferiorly positioned hyoid bone, and a reduced nasopharyngeal and oropharyngeal airway space anteroposteriorly in OSA patients.

Laniado assessed the craniofacial soft tissue characteristics of an adult patient population with documented OSA, and for the first time indices of apnea severity were correlated with craniofacial morphology.

Recently, Trieger has proposed that the typical middle-aged, obese male with OSA may have yet a different etiology, unrelated to any skeletal abnormality of the jaws. On cephalometric radiography, the oral and hypopharyngeal airway spaces appear not only narrowed but also clouded, suggesting some infiltrative process. The increased thickness of the posterior pharyngeal wall has been well documented by cephalometric studies. The nature of this thickening, previously assumed to be due to fat deposits, is found on histologic examination to be lymphedema. This suggests some interference with lymphatic drainage of the nasopharynx, oropharynx, and hypopharynx which normally flows into the superior vena cava and then into the right atrium of the heart. Lymphedema would imply an increased hydrostatic pressure leading to a transudation of colloid and protein coming out of the capillaries and into the adjacent tissues. This edema suffuses the tissues of the head and neck and narrows the airway. A significant number of patients with OSA have been shown to have right atrial and ventricular dysfunction with a decreased ejection fraction and evidence of hypokinesis (66 per cent) despite enlargement (55 per cent). The right atrium is also seen to be enlarged and to be the major source of an observed increase in a recently described hormone — atrial natriuretic peptide or factor (ANF). This peptide follows a circadian rhythm similar to that of cortisol and renin. Its effects are to produce a diuretic and natriuretic action in the kidney in response to an increased pressure and stretch of the right atrium.

Krieger et al have shown that ANF is markedly increased during sleep in patients with OSA. The introduction of continuous positive airway pressure (CPAP) reverses both the diuresis and the sodium excretion rapidly. CPAP is believed also to decrease cardiac preload and, in effect, unburden the heart. Thus the ANF serum levels may serve as a helpful guide to the effectiveness of treatment of OSA.

One may speculate on what initiates the train of events leading to OSA. In the orthognathic patient it may well be related to the initial hypoxemia associated with the decrease in ventilation secondary to significant obesity. With hypoxemia there is an increased tension in the vasculature of the lungs (mainly mid-sized vessels) and pulmonary artery leading to pulmonary hypertension. The right ventricle hypertrophies to compensate, as does the right atrium. This impedes the forward flow of blood and lymph into the heart and causes lymphedema of the pharyngeal airway. The entire process is greatly facilitated in the presence of any contributing anatomic abnormality such as enlarged tonsils or subglottic or sublingual masses, which may encroach on the airway. In the nonobese patient with a significant anatomic obstruction of the airway, a similar chain of events is created because respiratory exchange is diminished and hypoxemia ensues.

There are data to show that weight loss can provide significant benefit for the patient with OSA. Perhaps this improves ventilation and decreases the workload of the heart, with a secondary gain of better forward flow of blood and lymph, thus
opening up the airway. Further studies may prescribe methods of enhancing heart function, in addition to an aggressive program of weight control to help manage the patient with OSA. Various surgical methods directed only toward removing an obvious anatomic factor contributing to obstruction have not been as uniformly successful as anticipated. Persistence of OSA is significantly associated with nocturnal premature death, undoubtedly related to hypoxia and lethal cardiac dysrhythmia.

## TREATMENT OF OBSTRUCTIVE SLEEP APNEA

Treatment of the sleep apnea patient is directed toward identifying the locus (loci) of obstruction. An obvious example is hypertrophic tonsils obstructing the airway, for which a tonsillectomy is recommended. Most often, however, the diagnosis is not that simple. There are often multiple factors involved, e.g., obesity and a compromised airway secondary to retrognathia, or any combination of factors. Most patients with OSA do not have readily identifiable pathology on standard otolaryngologic examination. Rojewski et al reported that in an evaluation of 200 patients with OSA, only three patients were found to have an anatomic problem that could be surgically corrected.

The treatment of the OSA patient can be subdivided into medical and surgical modalities of therapy. Since the pathogenesis of sleep-disordered breathing has not been fully elucidated, therapy in most cases continues to be difficult because the etiology remains obscure. Thus the application of medical and surgical treatment modalities can further be divided into general measures recommended for the management of all cases of sleep-disordered breathing and specific measures recommended for particular disorders.

Medical modalities include treatment by a reduction of the risk factors that can precipitate apneic episodes, e.g., avoidance of alcohol or other central nervous system depressants. Inhalation therapy, with continuous positive airway pressure, and airway patency devices are helpful. Surgical treatment techniques include adenoid and tonsil removal if they are enlarged. To correct skeletal deformities that compromise the oropharyngeal airway, tracheostomy, UPPP, maxillomandibular surgery, anterior superior suspension of the hyoid bone, and hyoidoplasty may be undertaken.

Prior to initiating any form of treatment, all patients should undergo a complete and thorough history, physical examination, and laboratory workup. From this a definitive diagnosis can often be reached. Various therapeutic recommendations and modifications are inherent in treatment of the OSA patient. Overall, successful treatment of the OSA patient requires continual follow-up and reassessment of the whole patient and not just the upper airway complication. A multidisciplinary approach achieves the best results.

The results of the UPPP treatment remain variable. Only 50 per cent of patients respond with nearly total remission of their symptoms. Failures appear to be related to airway narrowing in the hypopharynx in addition to the oropharynx. Investigators agree that the variability of results may be determined by the anatomic site of airway constriction. Patients showing the greatest benefit with the UPPP are those in whom the Müller maneuver suggests occlusion only at the level of the velopharyngeal sphincter. Patients with multiple sites of obstruction were less relieved by surgery alone. Therefore, patients with hypopharyngeal obstruction such as a large tongue base, an omega-shaped epiglottis, and redundant aryepiglottic folds did not benefit from UPPP. According to Sher and coworkers, the ideal patient for UPPP is one with a 3 to 4+ collapse at the level of the soft palate and no collapse in the lower pharynx.

Another treatment modality is CPAP (Fig. 58–5). This provides pressurized air
through a face mask to keep the airway open. The minimum pressure at zero flow required to maintain upper airway patency ranges from 4 to 15 cm of H2O.47,54,63

In a cephalometric study comparing pre- and post-treatment effects of UPPP and CPAP, Moore noted the following: (1) the skeletal and soft tissue craniofacial features of these two groups were statistically the same in all 70 variables measured prior to treatment.45 Moreover, he found that the post-treatment effects were very comparable. The irony of these findings tends to further obscure the underlying etiologic factor(s) of OSA.

Weight loss is usually recommended to a majority of patients. In Laniado’s study correlating numerous variables to apnea severity indices, body mass index (BMI) was consistently the most highly correlated variable when it was entered into the equation.38 Subjectively, all patients feel that their apnea is less severe after they have lost weight. As a primary form of therapy, however, it is not uniformly effective.60

Tracheostomy is usually the last resort in order to establish a definitive airway. On occasion, it may be the most effective and most urgent treatment, especially if the patient is developing frequent, life-threatening dysrhythmias in association with hypoxic episodes. It bypasses the occlusion of the upper airway which occurs during sleep and establishes a more direct patent airway.16 This continues to be the only consistently effective treatment, except for patients in whom clinically distinct upper airway abnormalities can be identified and corrected, i.e., adenotonsillar hypertrophy, tumor impinging on the airway, and micrognathia.29 Permanent tracheostomy, however, is associated with a number of potential complications and significant psychosocial problems.8

Other alternatives for patients, particularly those with a skeletal abnormality such as retrognathia or micrognathia, are maxillofacial surgery, orthognathic surgery, or intraoral appliance therapy. These procedures can aid in increasing the pharyngeal diameter and correcting the posterior and inferior displacement of the tongue into the pharyngeal cavity. Initially, an orthodontic repositioning appliance such as a modified Herbst is used to open the airway (Fig. 58–6). The widening of the pharynx in an anteroposterior dimension is confirmed cephalometrically, and sleep studies are repeated in 3 to 6 months if there is any improvement. If the results are encouraging and the patient is amenable to surgery, a surgical advancement of the jaw(s) can be performed. OSA has been treated by orthodontic appliances, maxillofacial surgery, or a combination of these procedures.21,42,49,59 With orthodontic appliances alone it was found that an anterior repositioning of the mandible caused the genioglossus muscle to come forward, the tongue to acquire a more proper posture, and the hypopharyngeal space to open.21 Elimination of wedging of the soft palate against the nasopharyngeal wall was also achieved in a
study of 16 patients using a Herbst appliance. Pancherz found a distinct improvement in 15 patients with snoring and sleep, while one patient could not tolerate the appliance.\(^\text{44}\)

Orthodontic appliances have also been used to help predict the success of surgical mandibular advancements.\(^\text{1,42}\) Meir-Ewert and Brosig used a protracting device called the Esmarch prosthesis to pull the mandible 3 to 5 mm forward during sleep on 26 OSA patients.\(^\text{42}\) Thirteen were excluded because of a poor-fitting appliance or resolution of the sleep apnea by other forms of therapy (e.g., weight reduction) prior to appliance treatment. However, following all-night PSGs with and without the appliance on the remaining 13 individuals, an average improvement of 61 percent AI 92 per cent in apnea length, and 75 per cent in apnea time was found. Also, snoring was reduced in all patients and disappeared completely in some. Even though micrognathia has been cited in the literature as a common etiology of OSA, few cases of sleep apnea associated with mandibular retrognathism have been reported. Valero and Alroy in 1965 reported a case of a 55-year-old Arab man with sleep apnea and mandibular retrognathism secondary to a traumatic injury at age 3.\(^\text{67}\) A tracheostomy was performed and the sleep apnea symptoms disappeared. Blokzijl reported two patients with sleep apnea and mandibular retrognathism who were treated with tracheostomies with good results.\(^\text{2}\) Lugaresi et al and Imes et al documented one case each of patients presenting with sleep apnea and mandibular retrognathism who were treated successfully by tracheostomy.\(^\text{27,40}\)

In 1970, Piecuch reported on the use of costochondral rib grafts to the temporomandibular joint areas of a 5-year-old girl who presented with mandibular hypoplasia and upper airway obstruction, manifested by apneic periods during sleep. The grafts were successful and the respiratory problems were alleviated following surgery. This finding suggested the possibility that mandibular retrognathia may be a contributing factor in some cases of sleep apnea.

In 1980, Bear and Priest reported on a 35-year-old black male with excessive sleepiness, retrognathic mandible, and short fat neck.\(^\text{45}\) Sleep apnea secondary to upper airway obstruction was diagnosed. After the patient refused a tracheostomy, an acrylic occlusal splint was fabricated to protrude the mandible in order to determine whether surgical advancement would have lasting and positive effects on the patient’s sleep apnea. An objective measurable improvement in apneic spells was noted; therefore surgical advancement of the mandible was performed. Following surgery the patient had no further problems with excessive daytime sleepiness and no apparent nightly apnea episodes.
OUR PERSPECTIVE

The remainder of this chapter focuses on how sleep apnea patients are evaluated. Case reports are presented to illustrate diagnostic dilemmas that many sleep apnea patients present. In the past 7 years our dental department has been an active participant in our sleep-wake disorders team. In that time, we have evaluated and have acquired cephalometric radiographs on nearly 500 sleep apnea patients.

Radiographic Technique

Three cephalometric radiographs are obtained after placing 1.5 cc of barium sulfate into each nostril and 0.5 cc in the oral cavity (3.5 cc total). This barium sulfate nasal lavage facilitates evaluating and measuring the oral and pharyngeal airways. Three cephalographs are obtained:

1. A normal lateral view in centric occlusion
2. A lateral view in which the mandible is protruded to its maximum extent
3. A posteroanterior projection

Patients are instructed not to swallow or breathe during exposure of all radiographs.

Cephalographs should be obtained in a standardized fashion with a fixed cathode-to-object distance of 60 inches (5 feet) and a constant object-to-film distance. All of our measurements were taken at a 15 cm object-to-film distance. The lateral and PA cephalographs should then be traced and measured. Our cephalometric analysis uses 25 linear and angular skeletal measurements and 22 linear and angular airway soft tissue measurements (Figs. 58–7 and 58–8).

Established normative standards for the lateral projection have proven to be useful in evaluating these patients (Tables 58–1 and 58–2). The posteroanterior projection provides additional information on the mediolateral dimensions of the airway. When it is interpreted along with the lateral cephalograph, the analysis is multidimensional (Fig. 58–9). The protrusive lateral cephalograph is used to assess any dimensional changes to the anteroposterior width of the airway when

![Skeletal cephalometric measurements for assessment of OSA. Normative data for the above measurements can be found in Table 58–1.](image)
mandible is advanced. The information from this projection may suggest a possible treatment approach. However, this should not stand alone but should be compared to a direct-view nasopharyngoscopic examination by the otolaryngologist.

At a regularly scheduled treatment planning conference, a discussion of findings with other members from the team generates an appropriate treatment plan for each patient reviewed. When the treatment course of the first 250 patients screened is radiographically reviewed, one notes some interesting trends. Adults

![Image of facial anatomy with labels for Frankfort horizontal, PNS, ANS, Odontoid, C2, C3, Hyoid, and Mandible plane.]

**FIGURE 58-8.** Soft tissue cephalometric measurements for assessment of OSA. Normative data for the above measurements can be found in Table 58–2.

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Males (Means)</th>
<th>Males (SD)</th>
<th>Females (Means)</th>
<th>Females (SD)</th>
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<tr>
<td>SNA</td>
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<td>(3.70)</td>
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<td>SNB</td>
<td>80.60°</td>
<td>(3.90)</td>
<td>78.50°</td>
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<td>(2.30)</td>
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<td>(3.70)</td>
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<td>(2.20)</td>
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<td>(5.00)</td>
<td>70.40 mm</td>
<td>(6.00)</td>
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<td>(2.50)</td>
<td>7.60°</td>
<td>(2.20)</td>
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<td>(3.59)</td>
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<td>(3.70)</td>
<td>33.30°</td>
<td>(4.60)</td>
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<td>24.60°</td>
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<td>(1.10)</td>
<td>139.30°</td>
<td>(7.95)</td>
</tr>
<tr>
<td>ArGo</td>
<td>124.20°</td>
<td>(5.50)</td>
<td>126.00°</td>
<td>(6.50)</td>
</tr>
</tbody>
</table>

SD = Standard deviations.
### TABLE 58-2. NONAPNEIC POPULATION SOFT TISSUE CEPHALOMETRIC MEASUREMENTS

<table>
<thead>
<tr>
<th>Measurements</th>
<th>Males</th>
<th>SD</th>
<th>Females</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Par-ad1</td>
<td>31.00 mm</td>
<td>(5.50)</td>
<td>27.50 mm</td>
<td>(5.35)</td>
</tr>
<tr>
<td>Par-ad2</td>
<td>27.60 mm</td>
<td>(9.10)</td>
<td>25.60 mm</td>
<td>(4.05)</td>
</tr>
<tr>
<td>PAS</td>
<td>12.45 mm</td>
<td>(9.10)</td>
<td>11.75 mm</td>
<td>(4.05)</td>
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<tr>
<td>PNS-P</td>
<td>44.15 mm</td>
<td>(13.30)</td>
<td>47.10 mm</td>
<td>(2.35)</td>
</tr>
<tr>
<td>ANS-PNS-P</td>
<td>126.20* mm</td>
<td>(7.10)</td>
<td>130.60* mm</td>
<td>(5.50)</td>
</tr>
<tr>
<td>SPW</td>
<td>11.65 mm</td>
<td>(1.60)</td>
<td>11.60 mm</td>
<td>(1.80)</td>
</tr>
<tr>
<td>NPh1</td>
<td>30.60 mm</td>
<td>(5.20)</td>
<td>28.70 mm</td>
<td>(4.50)</td>
</tr>
<tr>
<td>PPW1</td>
<td>11.60 mm</td>
<td>(3.70)</td>
<td>11.60 mm</td>
<td>(1.20)</td>
</tr>
<tr>
<td>NPh2</td>
<td>10.50 mm</td>
<td>(4.00)</td>
<td>8.30 mm</td>
<td>(2.50)</td>
</tr>
<tr>
<td>PPW2</td>
<td>9.85 mm</td>
<td>(3.70)</td>
<td>8.40 mm</td>
<td>(1.70)</td>
</tr>
<tr>
<td>OP1</td>
<td>10.90 mm</td>
<td>(3.70)</td>
<td>8.30 mm</td>
<td>(5.30)</td>
</tr>
<tr>
<td>PPW3</td>
<td>4.40 mm</td>
<td>(2.50)</td>
<td>4.25 mm</td>
<td>(1.70)</td>
</tr>
<tr>
<td>OPh2</td>
<td>11.90 mm</td>
<td>(3.90)</td>
<td>9.70 mm</td>
<td>(6.50)</td>
</tr>
<tr>
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<td>5.10 mm</td>
<td>(2.90)</td>
<td>4.25 mm</td>
<td>(1.50)</td>
</tr>
<tr>
<td>HPh1</td>
<td>16.90 mm</td>
<td>(6.50)</td>
<td>11.00 mm</td>
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<td>PPW5</td>
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<td>(.70)</td>
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</tr>
<tr>
<td>HPh2</td>
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<td>(3.70)</td>
<td>22.60 mm</td>
<td>(7.40)</td>
</tr>
<tr>
<td>PPW6</td>
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<td>(1.10)</td>
<td>5.20 mm</td>
<td>(1.00)</td>
</tr>
<tr>
<td>MP-H</td>
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<td>(5.30)</td>
<td>15.40 mm</td>
<td>(7.20)</td>
</tr>
<tr>
<td>P-NPh1</td>
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<td>(7.10)</td>
<td>35.50 mm</td>
<td>(8.30)</td>
</tr>
<tr>
<td>EP</td>
<td>57.10 mm</td>
<td>(6.50)</td>
<td>49.75 mm</td>
<td>(7.70)</td>
</tr>
<tr>
<td>Ep-P</td>
<td>19.70 mm</td>
<td>(5.70)</td>
<td>14.25 mm</td>
<td>(7.70)</td>
</tr>
</tbody>
</table>

SD = Standard deviations.

with OSA tend to have convex profile, long anterior upper and lower facial height, low-set hyoid bone, steep mandibular plane, large Y axis, obtuse cranial base, obtuse gonial angle, and constricted posterior airway space. The UPPP and CPAP populations are virtually no different in the soft tissue and skeletal cephalometric values, but when these groups are compared to the nonapneic group the differences are localized primarily in the soft tissue airway and not the skeleton. The UPPP and CPAP groups tend to show increased posterior pharyngeal wall thickness as well as a low-set hyoid bone. The patients treated with the protrusive/surgical approach tend to have maxillomandibular retrognathism and excessive vertical facial height secondary to increases in the mandibular plane angle. Other

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**FIGURE 58-9.** Analysis of anteroposterior cephalometric radiograph.
abnormal features in this group are (1) low-set hyoid and (2) smaller posterior pharyngeal airway space (particularly at the base of the tongue) secondary to a thickened posterior pharyngeal wall and compounded by a retracted lower jaw.

CASE REPORTS

CASE REPORT 1

L.L., a 46-year-old white male, was referred by the sleep-wake disorders center for evaluation of his sleep apnea. Past medical history was significant for an aspirin allergy, left eye blindness since childhood, and surgical removal of a pilonidal cyst 20 years before. Past dental history was significant for multiple dental restorations.

The patient complained of daytime somnolence ("lethargy") during the past 12 to 15 years. He reported snoring 95 per cent of the time during sleep. Initial PSG studies suggested a moderate OSA pattern. Physical examination revealed a tall, mildly obese male with notable mandibular retrognathism and a Class II dental malocclusion (Fig. 58–10). The radiographic survey documented a lower airway constriction at the base of tongue which improved markedly upon mandibular protrusion. These findings were also substantiated by nasoendoscopic examination. A modified Herbst appliance was fabricated and fit into the patient's dentition so that he would be able to fixate his mandible into a protruded posture. After 3 months, repeat PSGs documented improvement only when the appliance was activated. The patient agreed to surgical correction of his problem. He underwent 6 months of presurgical orthodontic therapy. A bilateral sagittal split osteotomy advancement of the mandible was performed.


FIGURE 58-12. Pre- and postoperative cephalometric results of Case 1. Note marked improvement in the anteroposterior dimension of the pharyngeal airway in patient L.L.

Four months later the braces were removed and retainers placed. Two and a half years later postsurgical PSG studies and cephalometric evaluation documented remarkable stability and a favorable response (Figs. 58-11 and 58-12).

**COMMENT**

This patient represents a straightforward documentation of an OSA whose etiologic mechanism clearly was secondary to his mandibular retrognathia and associated lower airway obstruction. Very few OSA patients follow such a scenario in our experience, and only 5 per cent even qualify for preliminary appliance therapy.

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**CASE REPORT 2**

R.W., a 42-year-old Caucasian male, was referred for evaluation of his sleep apnea. Past medical history was unremarkable. Past dental history included routine dental restorations, periodontal therapy, and orthodontic therapy during adolescence. This patient reported an 18-month history of a snoring problem that had worsened in the past 3 months. Polysomnographic evaluation demonstrated a moderate to severe obstructive apnea (A1 = 18.8 and AHI = 30.5).

Physical examination revealed an individual with only minimal obesity and average build. His facial profile was convex with mandibular retrognathia. He had a Class I dental and Class II skeletal relationship.

The radiographic survey demonstrated a markedly constricted pharyngeal airway in the AP plane. The airway width improved noticeably when he protruded his mandible. The patient was fit with a modified Herbst appliance so that he could fixate his mandible in a forward position during sleep. After 4 months of wearing his appliance, repeat PSGs were performed with and without the appliance in place. The studies documented marked improvement in both A1 and AHI; however, there was no difference between the studies. A follow-up nasendoscopy study revealed that the patient was obstructed in the upper airway at the level of the soft palate, whereas the initial study had suggested an upper and a lower obstruction pattern.

The patient was referred for a UPPP to eliminate the upper airway constriction. Four months after palatal surgery; PSG studies showed virtual elimination of the apneic episodes with an A1 of 0.8 and an AHI of 4.03.

**COMMENT**

Considering his recent history of snoring, the patient most likely presented in the early stages of OSA. There were no definitive etiologic factors identified. Preliminary evaluation and cephalometric analysis suggested that orthognathic surgery may have been necessary to successfully treat the patient. The follow-up PSGs clearly demonstrated that the appliance was useful, since the apnea was eliminated. Unexpectedly the apnea did not reappear when the protrusive component was eliminated.

The use of the protrusive appliance appears to eliminate certain factors, i.e., mandibular retropositioning and a constricted airway. Once the obstructive cycle was interrupted, it was obvious that precipitating factors were still localized in the upper airway and the patient would benefit in the long term from a UPPP procedure. Three and one-half year postoperative follow-up suggests good stability in results.
CASE REPORT 3

Patient O.K., a 29-year-old white male, was referred for evaluation of his sleep apnea. He had a history of cardiac enlargement and mild pulmonary hypertension. A “permanent” tracheostomy had been performed 2 years earlier. PSG studies confirmed the presence of severe OSA with an AI of 69.5 and an AHI of 141. His past dental history was significant in that at age 11 he underwent 1.5 years of active orthodontic treatment. This consisted of two maxillary bicuspid extractions and retraction of his upper anterior teeth to meet a deficient lower arch. The development of increased snoring and obstruction was noted shortly after orthodontic therapy began.

Physical examination revealed a moderately obese male with mild facial retrognia and an indwelling tracheostomy tube. His occlusion was Class I cusp and Class II molar with bimaxillary retrusion and an obtuse nasolabial angle.

Cephalometric radiographs taken in centric relation and in a protrusive relationship showed only slight improvement in hypopharyngeal airway. Segmental dental arch bars were applied and the patient was instructed in the application of elastic traction to effect a protruded mandible during sleep. A repeat PSG study was then carried out and documented improvement. In order to maintain his occlusion and correct his bimaxillary retrusion, two jaw surgeries were performed.

In 1988, both maxilla and mandible were surgically advanced 8 mm. Six months later repeat cephalometric radiographs showed sustained improvement, and his sleep apnea was markedly improved as determined by PSG (AI = 4 and AHI = 5.3.) However, the patient continued to complain of persistent poor sleep. A UPPP and reduction of base of tongue tissues were carried out 7 months later. A subsequent PSG showed no further improvement in AI and AHI.

Subjective complaints continued and a protrusive appliance for nighttime use was fabricated by the orthodontist. This was inadvertently broken soon after it was made, and the patient was referred to the prosthodontist. Two years after orthognatic surgery, the anteroposterior measurement of the oropharyngeal airway at C3 measured 11 mm compared to a preoperative measurement of 6 mm (Fig. 58–13). Despite this evident improvement, the patient sought further surgery and visited other major sleep centers. Four years postoperatively, PSG results continue to show a stable result with the AI = 1.0 and the AHI = 3.1. Six years postoperatively, subjective distress was reported by the patient. Unfortunately, he has also developed rampant caries but has repeatedly failed to keep scheduled appointments.

**COMMENT**

This patient has been followed at our center over the past 8 years for OSA. We suspect that his bimaxillary retrognathia underlies his long history of snoring and sleep disturbance and was a result of the orthodontic treatment, whereby his maxillary anterior teeth were retracted to meet his deficient lower dentition and produced clockwise rotation of the mandible with further oropharyngeal airway compromise. His obesity and history of pulmonary hypertension and right ventricular hypertrophy further compromised his airway by producing pharyngeal lynchphedema.

Increases in the airway size were accomplished transiently with positioning devices and later with orthognatic surgery. His persistent complaints caused him to seek further surgical treatment when all diagnostic parameters indicated that the obstructive sleep apnea problem has resolved. Psychiatric therapy has been of very limited help so far.

**FIGURE 58–13.** Pre- and postoperative cephalometric results of Case 3. Note marked improvement in the anteroposterior dimension of the pharyngeal airway in patient O.K.
CASE REPORT 4

M.S., a 6-year-old white female, was referred by the Center for Craniofacial Disorders to evaluate her apnea and help diagnose her craniofacial syndrome. Past medical history revealed that soon after her birth the patient developed "Robin"-like symptoms with glossoptosis and apnea. A glossoptomy was performed to maintain a patent oral airway. Subsequent to the breakdown of this procedure, a tracheostomy was performed. Frequent respiratory and tracheostomy-related problems continued over a 5-year period. The patient presented with the classic signs of sleep apnea: noisy breathing, frequent gagging and gasping, restlessness, and a knee-chest position. During the day she was extremely sleepy. Past dental history included a significant history of decay with multiple restorations placed. A palatal cleft was repaired. Physical examination revealed a well-nourished, rather obese, and significantly dysmorphic female. The patient presented with a short lower face, low-set ears, malar deficiency, and severe facial convexity. The radiographic survey demonstrated a small posterior airway secondary to an acute cranial base angle and retropositioned mandible.

Initial plans were to place the patient in orthodontic appliances and Class II elastics to help in protruding the mandible and assisting with opening her pharyngeal airway. This was done and the patient was followed for 6 months. Reports from her mother indicated that she was no longer snoring and was sleeping better. Soon after, an inverted osteotomy was performed to advance her mandible approximately 12 mm into a Class III relationship. Radiographs taken postoperatively at various times showed relative stability of the bony units with some relapse in the width of the airway space (Fig. 58–14). However, over the past 4 years the patient and parents report minimal airway problems. At this time the patient will be evaluated for orthodontic therapy in preparation for final maxillomandibular orthognathic surgery.

COMMENT

During the diagnostic phase of her craniofacial evaluation at our center, it was determined that this patient had Treacher Collins syndrome, which is often associated with the Pierre Robin sequence. Preliminary screening of the patient suggested that both her cranial base defect and her mandibular morphology contributed to her apnea. Through the process of elimination this supposition was verified prior to committing ourselves to any further irreversible therapy. It is evident that further surgery will be necessary and will be directed so as not to eliminate the improvement in her airway but also to balance facial and jaw relationships.

CASE REPORT 5

J.R., a 14-year-old hispanic male with achondroplasia, was referred to our department by the Center for Craniofacial Disorders for evaluation of his orthodontic needs. Past medical history was significant for growth hormone therapy administration and OSA. A tracheostomy was carried out at the age of 12. Past dental history included routine dental care.

Physical examination documented that the patient was 3' 6" tall and weighed 50 lbs. Both measurements were below the 95th percentile and also below average for achondroplasia. His profile was concave with frontal bossing and midfacial hypopla-
sia. The radiographic survey revealed a short and narrow pharyngeal airway secondary to an acute cranial base, inferiorly positioned sella, short posterior cranial base, inferiorly positioned posterior nasal spine, and a short mandibular ramus. The small airway was further compromised by a lower-set soft palate—uvula which contributed to lower airway obstruction. A palatal lift appliance was fabricated to test the hypothesis that the uvula was the cause of the obstruction and, in addition, to validate the need for a UPPPP. After 5 months of appliance wear and monitoring by the parents, a repeat PSG survey was performed with and without the appliance in place while the tracheostomy was closed. The results showed that when the appliance was in place his apnea diminished significantly.

**Inferiorly Positioned Posterior Nasal Spine**

A UPPPP was performed on the patient. Within months the tracheostomy was decanulated and since that time (nearly 8 months) the patient has been free from apnoreic episodes.

**Comment**

Our initial plan for treating this patient’s apnea involved considering a midface advancement. This movement would have the net effect of elevating and advancing the soft palate—uvula. Moreover, it would also take into consideration the patient’s abnormal facies. However, the patient and his parents were not interested in changing his face. The patient was psychologically well adjusted and felt no need for facial changes. Perhaps a desire for facial improvement will be expressed as he matures. In so far as his airway needs are concerned, a UPPPP should provide the same effect as the maxillary advancement surgery.

The placement of an intraoral appliance proved to be extremely useful in defining the location of the obstruction.

**Summary**

The causes of OSA are multiple. There are a number of procedures that are beneficial and can decrease the threat to life secondary to hypoxemia during obstructive sleep episodes or secondary to daytime somnolence. Efforts must be directed toward identifying the locus or loci where the respiratory activity is frustrated. This may be due to conditions such as hypertrophic tonsils and adenoids or hypertrophic soft palate and uvula, narrowing at the base of the tongue, masses compressing or compromising the orohypopharynx and retrognathia and micrognathia. On occasion correction of one area of compromise may reveal other sites of obstruction.

By bypassing the nasoorohypopharyngeal areas of obstruction can be accomplished with a classic tracheostomy. This was a mainstay of therapy in the past and may still be indicated in special situations when the apnea is life-threatening and the obstruction cannot be clearly identified and overcome.

In recent years, the nighttime administration by face mask of compressed air under low pressure has significantly improved the outlook by reducing episodes of obstructive apnea. It is becoming the standard against which other therapeutic measures are judged. The level of air pressure needs to be determined by titration and observation of its effects. It appears that breathing a continuous flow of air at low pressure prevents the pulmonary alveoli from collapsing completely. This “pneumatic stent” makes it easier to expand these alveoli with the next breath rather than requiring an increased force that serves to constrict the upper airways (Bernoulli’s principle). However, sleeping with a face mask has some inherent difficulties.

It has been shown that a sizable number of people snore, which is evidence of some respiratory flow impedance. However, a smaller number of people develop OSA. Having diagnostically ruled out developmental and congenital abnormalities of jaw growth and airway compromise, we are left with a group of patients whose airways are functionally narrowed because of retrograde lymphedema. These patients probably have a primary cardiac etiology. This is reflected in the
finding that elevated serum levels of a recently discovered hormone (atrial natriuretic peptide) are present in OSA patients; many of these patients are also significantly obese and hypertensive. The administration of continuous positive air pressure lowers the level of atrial natriuretic peptide. The cells that secrete this hormone do so in response to dilatation of the walls of the right atrium. The hormone's action is primarily on the kidney and serves to facilitate the excretion of sodium and water.

Better methods to enhance weight and blood pressure reduction are needed to improve cardiac function by decreasing preload and afterload. This would result in an improvement of sleep apnea, according to our working hypothesis. Other measures need to be found which will effectively diminish the workload of the heart and enhance apnea-free respiration.

Performing an assortment of manipulative or surgical procedures in the absence of specifically diagnosed airway obstructions has resulted in only a small record of improvement. On the other hand, accurate diagnosis results in a high yield when the appropriate corrective procedure is done.

ACKNOWLEDGMENTS: The authors would like to thank Dr. Michael Thorpy, Director of Einstein/Montefiore Center for Sleep-Wake Disorders, and his staff for their assistance. We would also like to thank Dr. Robert Shprintzen, Director, Einstein/Montefiore Center for Craniofacial Disorders, for his advice and assistance in putting this chapter together; Dr. Mohsen Montahemi for providing assistance in the treatment of Case 1; Dr. Larry Wolford for providing assistance in the treatment of Case 4; Dr. Michael Stewart for his assistance with the illustrations; and Margaret Mark for her help in putting it all together.

REFERENCES