Obstructive Sleep Apnea — An Orthodontic Concern

Earle F. Cote

With invited discussion by T. Arthur Babineau

A description of the obstructive sleep apnea syndrome and its many ramifications, with a case report on the diagnosis and treatment of a patient whose condition was relieved by orthodontics and orthognathic surgery.

KEY WORDS: · AIRWAY · APNEA · RESPIRATION · SLEEP APNEA · · SNORING · TONGUE ·

Chronic, persistent snoring is a common symptom that increases in prevalence throughout the lifespan, with well over 50% of individuals over the age of sixty reporting it. Snoring is caused by an interplay between a variety of factors, including sleep-related loss of muscle tone in the tissues supplied by the glossopharyngeal nerve, anatomical obstruction of the nasal passages, large tonsils, large tongue, a retrognathic mandible, obesity, alcohol, sedative medication, allergies, and certain medical conditions.

The snoring sound is produced by the vibration of the soft palate or other oropharyngeal tissues. It can become a medical concern because it is a key symptom of obstructive sleep apnea syndrome (OSA).

Cessations of breathing for ten seconds or longer are termed *apneas* (from the Greek – *without breath*). When thirty or more apneic episodes occur in the course of seven hours of sleep, resulting in excessive sleepiness during the waking hours, a person is described as having sleep apnea syndrome. This condition may begin at any age, but the incidence increases with age.

Normal Respiration

Normal respiration requires that air be displaced from the external environment into the lungs, where it can contact the alveolar membrane to make oxygen available for gaseous exchange with the blood stream. The ostensibly simple function of the upper (supralaryngeal) airway is to permit the unimpeded movement of humidified air to the tracheobronchial tree.

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Apnea results from partial or complete interference with this process. The most commonly encountered impedence of airflow is upper airway obstruction, which is most often secondary to morphologic, pathologic, or functional abnormalities of the upper airway.

The supralaryngeal airway is very susceptible to obstruction elicited by skeletal muscle hypotonicity associated with sleep, and the most obvious manifestation of such upper airway obstruction is snoring. Although snoring has been the subject of countless humorous literary references, it a medical enigma that is now well recognized as an important sign of the potentially lethal condition that we call obstructive sleep apnea.

It has been shown that snoring may range from a totally benign, inconsequential social nuisance to a primary sign of a medical condition associated with serious cardiopulmonary and behavioral sequelae (GUILLEMINAULT AND DEMENT, 1978).

Until recently, sonorous snoring and excessive daytime sleepiness have been given little attention by the medical community. The superficially trivial nature of these most common symptoms associated with severe upper airway obstruction has made them appear less than worthy of careful scrutiny by a profession dealing largely in life-and-death matters, so the pathogenesis of these symptoms has been given little attention.

This has changed with the recent proliferation of clinical sleep laboratories throughout the United States, where careful attention to the details of sleep behavior has finally discovered the importance of sleep-related breathing disorders.

Symptoms

The most common initial complaints in sleep apnea are excessive daytime sleepiness, irritability and/or depression, and snoring (REMMERS ET AL., 1978). In some instances, patients may also present with cardiac involvement.

Obstructive sleep apnea should always be suspected in an individual with a history of loud, sonorous snoring combined with any of these symptoms.

Snorers may also show other types of breathing disturbance, including disruption of the central nervous system stimuli that initiate a breath. This differs from obstructive sleep apnea, where a breath is initiated but air exchange cannot occur because air movement is blocked by collapse or other blockage of the throat.

It is crucial to establish the type and frequency of apnea in each patient with suspected sleep apnea, because treatment for obstructive apnea will not be effective in central apnea.

Treatment for sleep apnea can reduce snoring dramatically. However, even a dramatic reduction of snoring may not necessarily result in improvement in sleep apnea, so careful monitoring is essential.

The Sleep Evaluation

In 1964, airway occlusion during sleep was described in obese patients with daytime somnolence and snoring by GASTAUT and his colleagues. Via the simultaneous monitoring of airflow and thoracic breathing movements, these investigators identified the occurrence of repeated episodes of upper airway obstruction during sleep. Upper airway obstruction was identified in these studies by the cessation of airflow at the nose and mouth with *no* interruption of thoracic breathing movements.

These investigations clearly establish the existence of a sleep-related breathing disorder as a primary pathophysiologic entity. This discovery is especially significant for individuals with apparently nor-

mal respiration during waking hours in whom sleep can induce a potentially lethal alteration in respiratory function.

It remained, however, for later investigations to disclose the ubiquity of this syndrome and the multiplicity of cardiopulmonary and behavioral sequelae.

Sleep Monitoring

Sleep monitoring consists of the simultaneous recording of a number of physiologic variables during sleep. The electroencephalogram, the electro-oculogram, and the electromyogram are necessary to determine sleep stages. The cardiopulmonary measures that are recorded include airflow at the nose and mouth, thoracic breathing movements, oxygen saturation via ear oximetry, and electrocardiographic activity.

These measures allow the categorization of the various states of sleep and identification of distinctions among three types of apnea — obstructive, central, and mixed.

Upper airway obstruction causes a cessation of airflow with concomitant continuation of thoracic breathing movements.

In central apnea there is simultaneous cessation of both airflow and thoracic breathing movements. Mixed apnea is the term applied to a condition in which a episode of central apnea lasts 10 seconds or longer, followed by obstructive apnea.

Studies have confirmed that patients with OSA may experience several hundred episodes of upper airway obstruction, associated with severe hypoxemia, during one night of sleep. It is not uncommon for arterial oxygen partial pressure to drop into the middle values after 20 seconds of an episode of obstructive apnea. Although average duration is approximately 25 to 30 seconds, obstructions are frequently noted to exceed one minute.

Investigations have shown that pulmonary artery pressure increases concomitantly with the increases in hypoxemia. This increase in pulmonary artery pressure is a result of the pulmonary vascular constriction that occurs with hypoxemia. It is this sequence of events that results in a marked increase in the load on the right side of the heart, which can ultimately result in right ventricular hypertrophy and failure.

Cardiac arrhythmias also often occur with obstructive events. The most commonly encountered are bradycardia and tachycardia. Bradycardia is noted concurrently with the obstructive event, and tachycardia at the termination of the apneic event and the resumption of airflow.

Pathophysiology

The pathophysiology of upper airway function during sleep has now been evaluated in considerable detail. Electromyographic studies of the genioglossus muscle during sleep have revealed a precise relationship between the respiratory cycle and the activity of this muscle, with inspiration associated with a phasic burst of activity. This produces a slight advancement of the tongue, maintaining airway patency during inspiration.

These studies focus considerable attention on the tongue in the pathogenesis of airway occlusion during sleep. It has been hypothesized that individuals with OSA have impaired genioglossal function, allowing the prolapse of the tongue against the posterior pharyngeal wall with inspiratory effort during sleep.

The situation now appears to be more complicated — evidence suggests that an invagination of the pharyngeal walls and a general hypotonia of the dilating muscles of the upper airway can also be involved in allowing airway occlusion during sleep.

The nasal airway can also play an important role in total airway occlusion. Nasal obstruction increases resistance to air flow, which in turn results in increased inspiratory effort and greater negative pressure in the pharyngeal airway. This suction increases the likelihood of collapse of the pharyngeal airway, which lacks the cartilagenous support afforded the trachea. A recent report describes the marked improvement of OSA in three patients following septoplasty for a deviated nasal septum.

Other investigators have ascribed OSA to an instability induced in the ventilatory control system by sleep, with periodic oscillations in the genioglossus and diaphragm resulting in airway occlusion at the nadir of these oscillations. Decreases in esophageal pressure as well as in the activity of the diaphragm and genioglossus have been noted before and after the onset of airway occlusion. This strongly suggests the presence of a periodic oscillation in neural respiratory drive in patients with OSA.

Case Report

The subject of this report knew how it felt. He remembers being in the middle of a business meeting, trying valiantly to stay awake, but finding it almost impossible. He also remembers many near misses on the road while driving home because of the difficulty that he had keeping his eyes open. Even an extra cup of coffee each night before getting into his car didn't help.

He would fall asleep in the barbershop chair and at the dinner table, and would use his weekend trying to catch up on the sleep that he didn't even know that he had lost.

His real problem was an enigma. He always thought that his sleepiness was normal, but as he became older (48 at the time of intervention), it continued to get worse. He was fortunate in that he had a wife who cared enough to help solve the mystery of his sleepiness. She had read an article on a condition called sleep apnea, and treatment was sought.

A preliminary study and evaluation revealed nothing neurologically wrong, so more definitive diagnostic testing was arranged at a sleep center. A clinical and radiographic evaluation at the center confirmed a diagnosis of sleep apnea.

His heartbeat, brainwaves, eye movements and muscle movements were monitored by electrodes. His blood oxygen level and his oxygen and carbon dioxide emission levels were measured, and a video camera recorded how he slept.

The first night of the study he stopped breathing 160 times, for a total of 70 minutes without breathing. The next night 183 breath cessations, ranging in length from 10 to 45 seconds, added up to 87 minutes without air flow.

These findings proved that he slept only a few minutes at a time during the night, and the combined findings indicate a serious case of apnea (Fig. 1).

Because sleep disorders come into play while the sufferer is not conscious, most people who have it don't even know that they have a problem. Yet it is estimated that from 5 to 10% of the population suffers from this sleeping disorder.

Since the patient must be asleep for a correct diagnosis, the family physician who sees the patient only during the day is at a great disadvantage. It is usually a spouse or a roommate who will be the first to notice the problem and complain to the victim about the snoring.

Body weight can play a key role in this condition; in fact, researchers have found weight reduction to be a most effective treatment for apnea in many individuals.

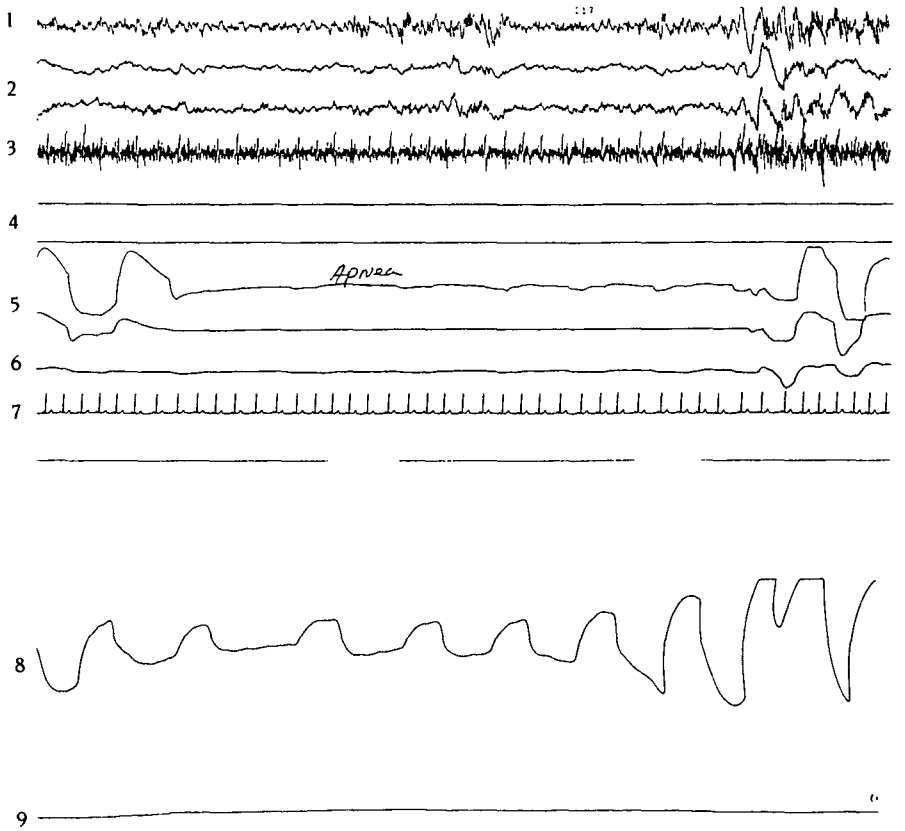

Figure 1. A small section of the polysomograph sleep recording (reduced in size), showing an episode of apnea. From top to bottom, the recordings show:

1. Electroencephalogram (EEG), showing brain activity.
2. Electro-ophthalmograms (EOG), showing left and right eye movements.
3. Electromyogram (EMG), with a sensor on the tip of the chin to detect jaw movement.
4. Leg movement recordings (left and right).
5. Nasal sensors to detect air movement through left and right nostrils.
6. Mouth sensor to detect oral air flow.
7. EKG heart recording.
8. Plethysmograph, with a sensor around the chest to detect chest expansion (respiratory effort).
9. Oximeter, recording oxygen saturation of the blood (percent).

A 10% weight loss can eliminate apnea in half of the people treated.

Weight was not a factor in the case reported here; his weight was well within the recommended guidelines for a person his size.

Treatment Options

What options are available for sleep apnea victims like this?

He could have a tracheotomy, with a surgical hole in his neck that is opened during the night and plugged for normal breathing during the day. A major problem encountered with this approach, in addition to the inspiration of raw unconditioned air, has been the psychological depression that these patients often experience.

Another procedure that has been used is the uvulopalatopharyngoplasty (UPPP), the reconstruction of the throat by resecting the posterior margins of the soft palate and redundant mucosa on the lateral pharyngeal walls. Unfortunately, the success rate for this approach to sleep apnea is only 40% because obstructions at other sites are not affected. Exact identification of the involved structures is imperative.

Sleep posture can also affect this condition. Our patient was encouraged to sleep on his side to reduce the number of apneas. This is the easiest recommendation for a patient to follow, but it was not effective in this case.

The other options above were not suggested, but another approach was recommended by the sleep center.

A continuous positive airway pressure machine (CPAP) is a new device with a mask that fits snugly over the sleeper's nose. It sends a continuous stream of air under positive pressure that is adjusted for each person using it to hold the throat open through the night.

When tested at the sleep center using the CPAP machine, our patient had only three apneic episodes during the night. This was a remarkable improvement on the almost 200 that he experienced when he was first evaluated. Even though this machine improved his breathing during sleep and eliminated his snoring, he found it impossible to use because the noise made him feel as if he were in an air tunnel. An alternative solution was needed.

At this time he was made aware of a corrective approach using an appliance similar to a bionator. His symptoms suggested that his problem was caused by his tongue slipping back into his throat, blocking the air passage while he was sleeping. His short mandible limited tongue space and geniohyoid length, predisposing to this kind of functional limitation.

An appliance was constructed for him to wear while sleeping. This was designed to bring his mandible and tongue forward, opening up the lower pharynx to allow unrestricted breathing.

It worked. For the first time, he experienced a full night of unrestricted sleep. This was later confirmed at the sleep center. "I feel great! It's as if I had been reborn," he remarked.

Other devices that could have accomplished this effect would include a removable Herbst appliance or a tongue restraining device.

Our patient now faced a big decision. The appliance worked, verifying the fact that moving the jaw forward was a solution to his problem, but did he want to wear this appliance for the rest of his life? After considerable reflection, he decided to undergo a surgical procedure to improve the anatomic relationships.

The surgery consisted of a LeForte I procedure with 5mm advancement of the maxilla with some maxillary impaction, combined with a 15mm advancement of the mandible to properly relate it with the new maxillary position.

During the presurgical orthodontic preparation, the removable orthodontic posturing appliance had to be constantly cut away and realigned as teeth were moved.

For mechanical and anatomic reasons, rigid fixation was not used in the mandible. The splint was designed with an anterior opening to allow breathing in case intermaxillary fixation was required, and this did become necessary. Provision was made for all conceivable possibilities, especially since we were dealing with a sleep apnea patient.

Surgical Changes

Cephalometric evaluation of the radiographs shown in Figures 2 and 3 shows significant changes in several measurements:

	Before	*After*
S-N-A	70	75
S-N-B	62	68
A-N-B	8	7
MP/FH	37	40

The smallest change was in A–N–B, since both A and B points were moved in the same direction by the surgery.

Facial Appearance Changes

The photographs in Figure 4 show a reduction of the nasolabial angle and increased projection of the nasal tip. The alar base width increased from 38mm to 43 mm, indicating an increase in the cross-sectional area of the nasal valve. The orbital area appears more rounded. Lip competency is improved.

Functional Changes

The success of the combined orthognathic procedure is demonstrated in Figure 5, and by the following report based on the postsurgical sleep evaluation:

> "On this night there was not one single apnea as compared to the 161 apneas he had prior to surgery. There were on this night, 10 obstructive hypopneas (partial apneas) which ranged in length from 17 to 36 seconds and had a mean duration of 28.4 second. His SaO_2 averaged 96% during both wakefulness and sleep and never fell below 92% in association with his hypopneas, compared to a low of 84% prior to surgery."

Since the surgery, the patient has experienced increased energy, alertness and happiness.

Soft Tissue Airway

Cephalometric soft tissue measurements from a point at the juncture of the base of the tongue and the tip of the epiglottis to the posterior pharyngeal wall were increased from 8mm to 15mm as a result of the mandibular advancement.

The upper airway, as measured at the narrowest point from the soft palate to the posterior wall of the nasopharynx, increased from 4.5mm to 6mm, presumably as a result of the maxillary advancement.

It should also be noted that the 5mm maxillary advancement permitted an additional 5mm advancement of the mandible.

Hyoid Changes

Recognizing that hyoid bone measurements are very sensitive to posture, and attempting to standardize head and neck posture in the cephalostat as much as pos-

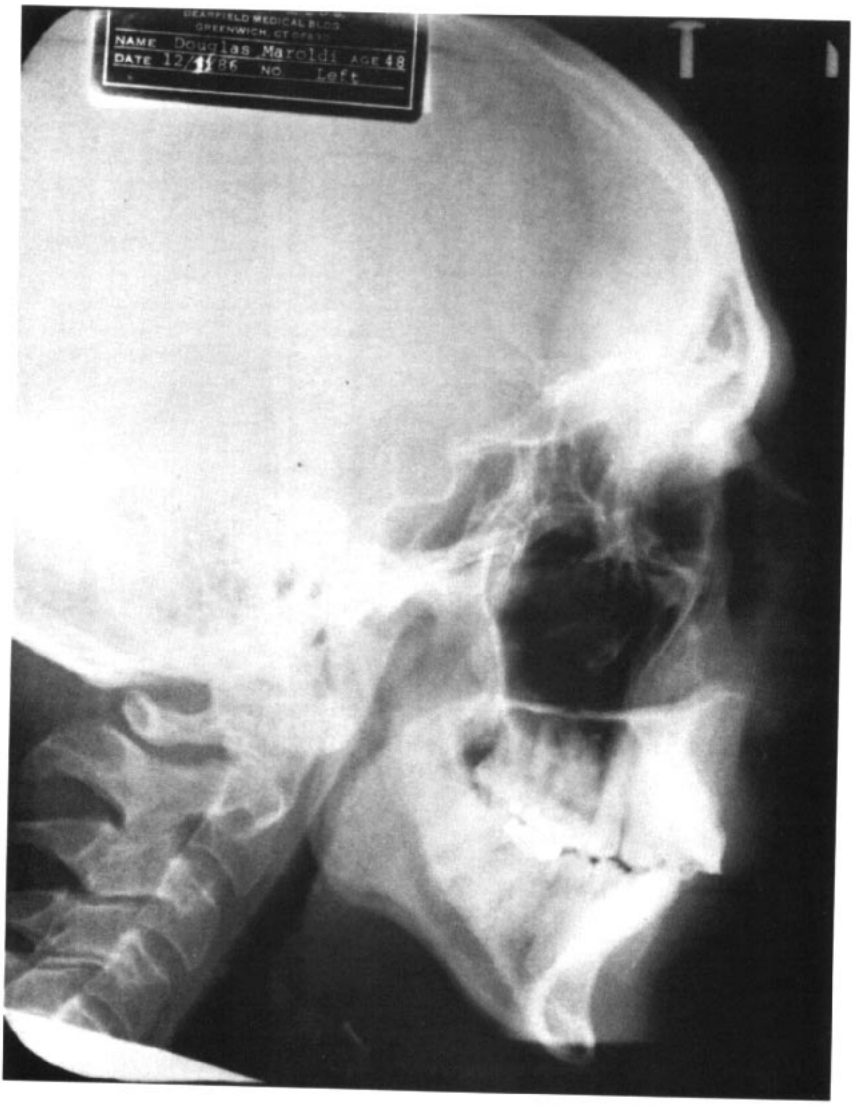

Figure 2
Pretreatment lateral cephalograph

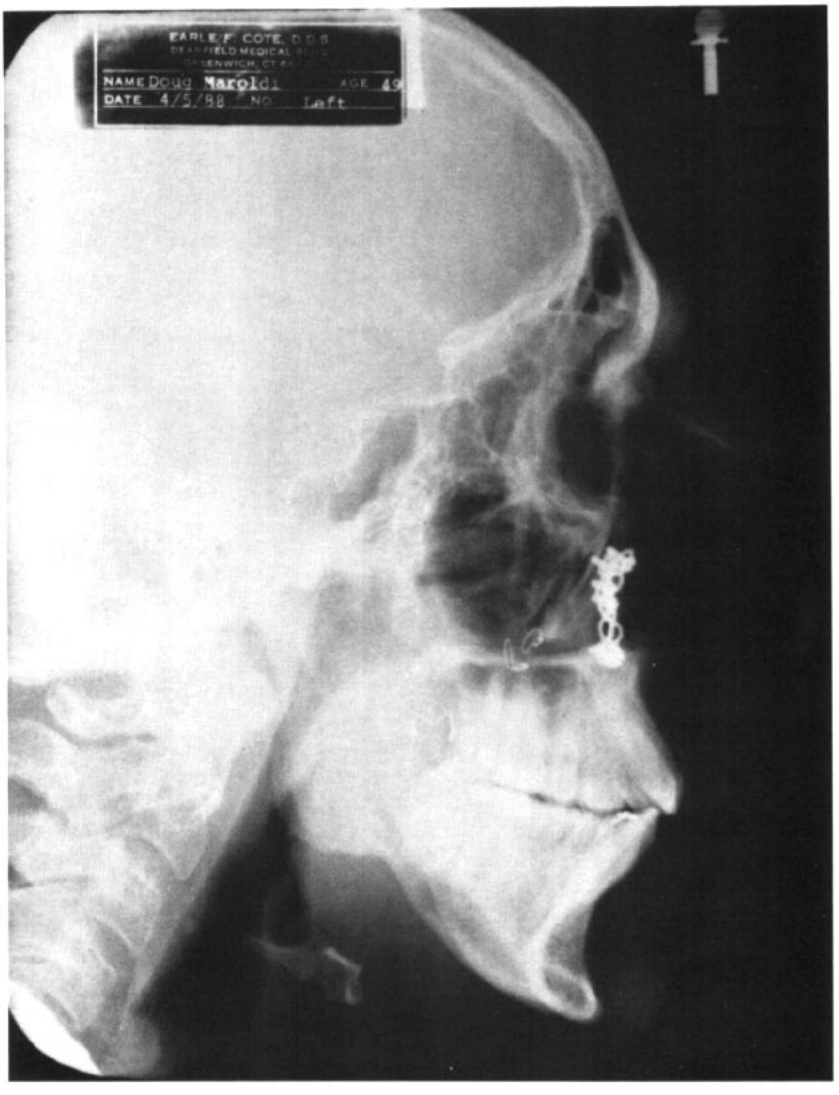

Figure 3
Posttreatment lateral cephalograph.
Note the changes in airway and tongue position. The extended head position that is typical for this facial pattern is also reduced.

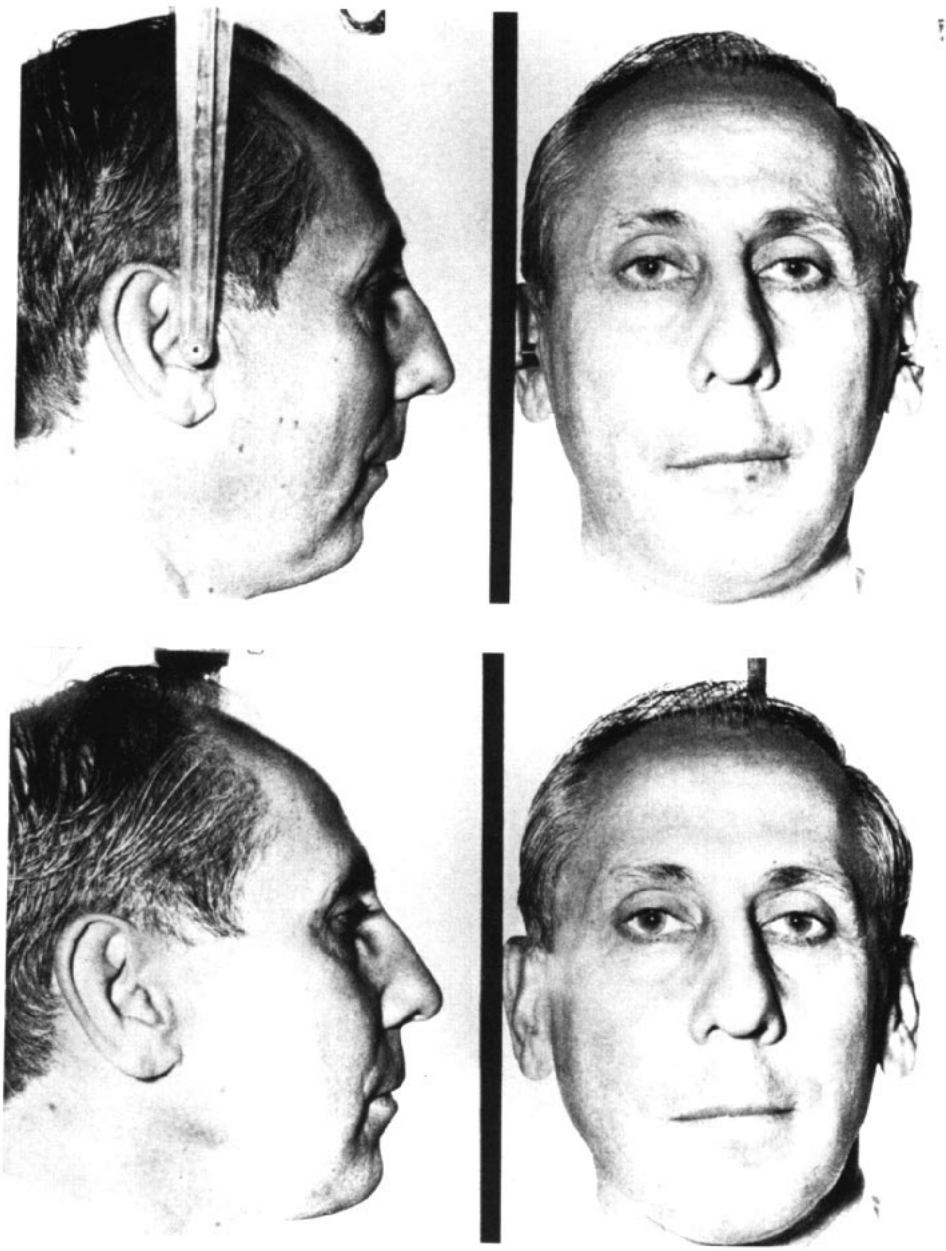

Figure 4
Pretreatment (top) and posttreatment (bottom) facial photographs.

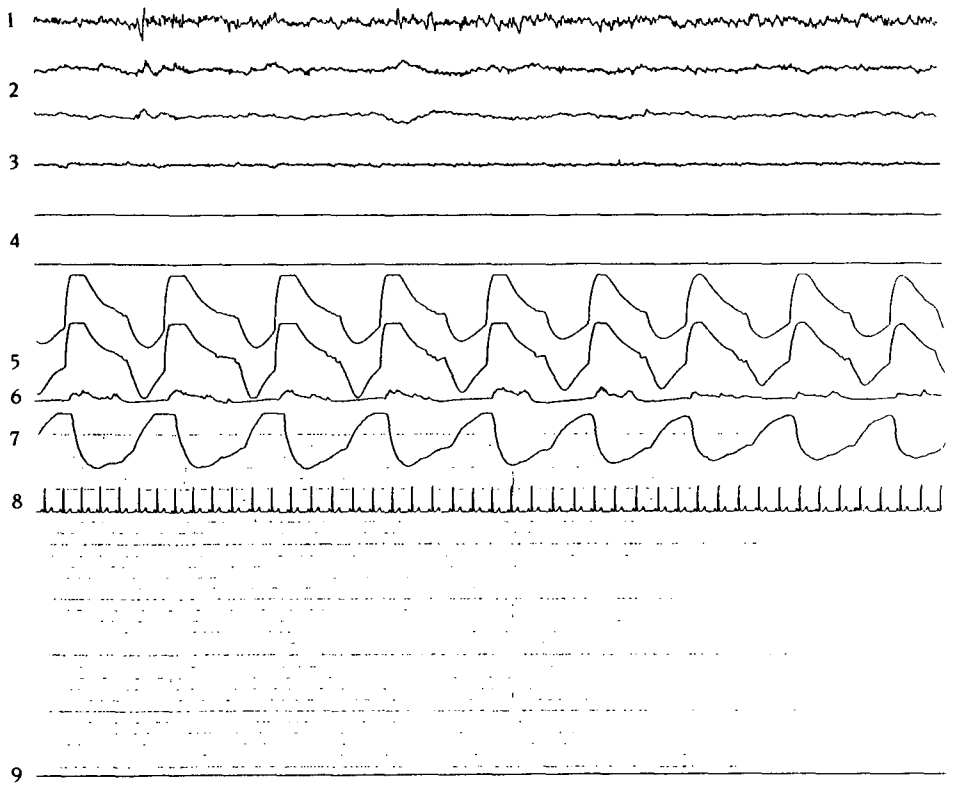

Figure 5. Posttreatment polysomograph. This segment shows only a few seconds, but is representative of the recordings throughout the night. From top to bottom, the recordings show:

1. Electroencephalogram (EEG), showing brain activity.
2. Electro-ophthalmograms (EOG), showing left and right eye movements.
3. Electromyogram (EMG), with a sensor on the tip of the chin to detect jaw movement.
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5. Nasal sensors to detect air movement through left and right nostrils.
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8. EKG heart recording.
9. Oximeter, recording oxygen saturation of the blood (percent).

sible, the following evaluations of hyoid position were made.

- Dropping a perpendicular to the lower border of the film from the inferior border of the third cervical vertebra, and measuring horizontally to the posterosuperior margin of the hyoid body, the distance was found to have increased from 35mm before surgery to 43mm after surgery.
- No satisfactory dimension could be found for evaluating vertical change because of the curvature of the neck and the fact that the mandibular border was moved by the surgery. However, all superimpositions indicate that the hyoid bone has been shifted upward and forward with the advancement of the mandible.
- A genioplasty advancement which would have moved the hyoid even farther forward was refused by the patient because he did not want to change the appearance of his chin.

— Summary and Conclusions —

Apnea is characterized by extremely loud snoring, with pauses in breathing and sometimes pronounced body and arm movements. The victim may wake up suddenly with choking sensations, gasping for air, or in a sweat. Other symptoms may be frequent napping during the day, especially in inappropriate places (like meetings, or while driving), memory problems, lack of concentration, high blood pressure. . . . Many of these symptoms can also be caused by many other conditions, so diagnosis may be difficult.

Orthodontic diagnosis may discover anatomic conditions that could cause this condition. Enlarged tonsils or adenoids in a lateral cephalometric radiograph, or maxillary width deficiency and narrow nasal cavity in a P-A radiograph, are indications for questioning the patient about other symptoms.

If obstructive sleep apnea is suspected, a medical consultation is in order. The patient may also be helped in pursuing a sleep test at one of about a hundred accredited sleep centers in the United States. About 10% of these centers are operated by Ph.D's and the others by M.D.'s. This field was first explored in laboratories by Ph.D.'s and psychologists who uncovered the clinical problems that are now being addressed by various medical disciplines.

The professional identification of an Accredited Clinical Somographer is A. C. P. The American Sleep Disorders Center, 604 Second St. S. W., Rochester MN 55902 [(507) 287-6006] can provide the names of accredited individuals.

It is important to distinguish between *central* and *mixed* sleep apnea.

CENTRAL sleep apnea involves a cessation of respiratory *effort*, as well as reduced air flow. This condition is relatively rare.

MIXED sleep apnea might involve two hundred obstructive and twenty kinds of central apneas, so some A. C. P.'s group them together as mixed apneas. The belief is that the central apneas in this category are a secondary effect of the reduced air flow caused by the obstruction rather than a primary etiologic factor.

There is no cessation of respiratory effort in obstructive sleep apnea; the effort is simply rendered ineffective by the obstruction. It is in this condition that the orthodontist may best participate in relieving or curing the symptoms.

The Author owes special thanks to Steven Brody, D.D.S. (Oral and Maxillofacial Surgeon), Walter A. Camp, M.D., (Neurologist), and Robert Watson, Ph.D., A.C.P. for their professional assistance; and to the patient for making his records available for publication.

— Invited Commentary* —

T. Arthur Babineau

I would like to begin this critique by complimenting Dr. Cote in three areas. First, for his selection of subject matter. As I reviewed the literature, I became increasingly fascinated by the subject. Secondly, I would like to compliment him on his preparation. We all know how busy Earle is and yet, despite his many commitments, the paper is extremely well prepared and written, and it arrived in my hands very well in advance of this meeting. I thank Earle for respecting the time needed for careful preparation of a critique. Finally, Earle is to be complimented on his presentation. He has been known for his humor, his dramatic flair, and his ability to ad lib. In presenting this paper he has enhanced the written document with beautiful demonstrations of all three characteristics.

It is unfortunate that time did not allow him to give an historical perspective of this syndrome. Sleep apnea syndrome offers an excellent example of the historical process whereby a series of investigators describe a syndrome.

In the "Posthumous Papers of the Pickwick Club" (1837), Charles Dickens described an incredibly fat boy named Joe who had persistent somnolence (DICKENS, 1837). Dickens's description may have been influenced by a presentation of W. WADD (1810) entitled, "Cursory Remarks on Corpulence: by a Member of the Royal College of Surgeons." The British physician CATON (1889) and his French colleague LAMACQ (1897) both observed that "narcoleptics" may suffer from obstructive airways during sleep that lead to "periodic states of suffocation."

In 1918, SIR WILLIAM OSLER coined the term "Pickwickian" after Dickens's description of Joe in referring to obese, hypersomnolent patients. In 1939, KERR AND LAGEN noted that a significant cardiocirculatory problem could develop in such patients, leading to cor pulmonale and cardiac failure. In 1956, BURWELL AT AL. reviewed the principal clinical features of the Pickwickian syndrome.

Between 1959 and 1962, ALEXANDER ET AL. (1959) and ALEXANDER ET AL. (1962) defined a "Joe" type of Pickwickian syndrome characterized only by obesity and hypersomnolence.

Finally, in 1964 GASTAUT ET AL., mentioned by Dr. Cote, reported on the presence of repetitive obstructive apnea during sleep in the obese "Pickwickian." But as the literature indicates, symptoms of this syndrome have been described as much as 130 years prior to the Gastaut publication.

There is much speculation about the exact pathogenesis of the disorder. As pointed out, numerous studies have investigated the pathophysiology of sleep apnea but much remains to be learned.

According to KALES ET AL. (1984), genetic and endocrine factors have been linked to obstructive sleep apnea. They state that several studies have shown an association between sleep apnea and hypothyroidism. They further state that in most cases of sleep apnea, anatomic abnormalities of the upper airway are not found on a "routine" clinical inspection.

Occasionally, gross anatomic factors such as mandibular malformation, micrognathia, tonsillar and adenoidal hypertrophy, nasal septal deviation, and acromegaly play a major contributory role. Even allergies have been associated with a considerable increase in sleep apneic episodes. In light of some activities planned for this meeting, it may be

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* *Presented at the Annual Meeting of the Eastern Component of the Edward H. Angle Society.*

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of interest to note that people with benign chronic snoring often develop obstructive sleep apnea after ingestion of alcohol. Could this be a cause for some of the somnolence at scientific presentations?

Dr. Cote mentioned weight reduction as a most effective nonsurgical treatment for sleep apnea in certain cases. It is interesting to note that two-thirds of more than 1000 sleep apneics seen at Stanford Sleep Research Clinic were overweight. However, according to LOMBARD AND ZWILLICH (1985), weight loss is unsuccessful in most cases.

In an article published in the Medical Clinics of North America in November 1985, CHRISTIAN GUILLEMINAULT describes a technique using cephalometrics to evaluate the posterior airway space by drawing a line from point "B" through Gonion. This line will intersect the base of the tongue and posterior pharyngeal wall, providing points for measuremt of the posterior airway space (PAS). A normal mean value of 11.5mm is reported.

He also reports that the CT Scanner has confirmed that the light shadows seen in the posterior airway space on cephalometric radiographs are related to redundant tissue in the hypopharynx.

The soft palate is evaluated by a line constructed from the posterior nasal spine to the tip of the soft palate contour, and the position of the hyoid bone is determined by a line drawn perpendicular to the mandibular plane through the hyoid. Appropriate measurements on the cephalometric radiographs will indicate whether the airway below the base of the tongue is abnormally small, how long the soft palate is, and alsi identify retrognathia.

Using this method of cephalometric evaluation, Guilleminault and associates have diagnosed and performed surgery on numerous patients. The surgical treatment varied from maxillary and/or mandibular osteotomies to hyoid bone myotomies and alteration of the hyoid suspension.

STANLEY THAWLEY (1985), in his article "Surgical Treatment of Obstructive Sleep Apnea," states that total mandibular advancement surgery has been successful in a limited number of patients. Fortunately in the case of the patient reported here, the diagnosis was correct and the surgery was successful. However, Thawley states that the number of patients receiving this procedure is not as large as those receiving the UPP (Uvulopalatopharyngoplasty), and fewer postoperative patients have been studied extensively to objectively document the clinical improvement.

He also describes an operation performed by Dr. Riley at Stanford University in which an anterior inferior genial osteotomy is performed on the mandible to include the genial tubercle on the inner cortex. This is the site of attachment for the genioglossus muscle, and with this procedure the anterior-inferior portion of the mandible is advanced forward while maintaining the general continuity of the mandible. Only the part of the mandible with the attached genioglossus muscle is advanced more anteriorly. The bone is held in place by attaching it with pieces of fascia to the remaining intact mandible.

This theoretically should pull the tongue forward and improve the hypopharyngeal airway, but provides no additional tongue space. Riley has reported short-term results for five patients treated with this procedure, reporting that all improved clinically with fewer symptoms of obstructive sleep apnea. This procedure has the advantages of obviating the need for intermaxillary fixation, and it does not affect the dental occlusion.

In concluding, I would like to compliment Dr. Cote once again for a fine description and demonstration of a subject not too familiar to many of us that gives new importance to our therapeutic armamentarium. It has been by far the most interesting paper that I have had the privilege of critiquing.

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