

Nonsurgical Management of the Obstructive Sleep Apnea Patient

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The high incidence of obstructive sleep apnea (OSA) has only recently been given the attention it deserves, partially because the signs and symptoms of this particular sleep disorder were overlooked or treated with disregard for their origin. Loud snoring is a common symptom. A history of snoring usually precedes the development of other symptoms. In most cases, symptoms develop gradually over a period of years. Of the millions of people whose lives are significantly impaired by the sequelae of OSA, most go undiagnosed, with tremendous medical and economic consequences for these individuals and for society.

The primary concern with OSA is the periodic collapse of the pharyngeal airway while sleeping. During apneas caused by obstruction, the collapsed pharynx prevents airflow, which is propagated by the continued effort of the diaphragm to breathe. This causes progressive asphyxia, which increasingly stimulates breathing efforts against the collapsed airway, typically until the person is awakened. In some patients, hypopneas are more common and are caused by only a partial collapse of the pharynx. The clinical sequelae of OSA relate to the cumulative effects of exposure to periodic asphyxia and to sleep fragmentation caused by apneas and hypopneas.¹

Patients with sleep apnea often have excessive daytime sleepiness. As the disorder progresses, sleepiness becomes increasingly irresistible and dangerous, and patients develop cognitive dysfunction, inability to

concentrate, memory and judgment impairment, irritability, and depression. These problems may lead to family and social problems and job loss. Cardiac and vascular morbidity in OSA may include systemic hypertension, cardiac arrhythmias, pulmonary hypertension, cor pulmonale, left ventricular dysfunction, stroke, and sudden death.¹

The challenge for the clinician is to routinely consider the diagnosis and to incorporate several basic questions in the historical review of systems regarding daytime or inappropriate sleepiness. Recognition and appropriate treatment of OSA and related disorders will often significantly enhance the patient's quality of life, overall health, productivity, and safety on the highways.¹ This article reviews the nonsurgical options for the management of OSA.

Overview of Nonsurgical Therapies

The treatment of OSA is a complex undertaking and varies depending on which practitioner makes the diagnosis. Once the diagnosis of OSA has been established, the treatment plan is dictated by the classification of the condition (mild, moderate, or severe) and the severity of the symptoms. Regardless of which treatment is chosen, an important caution that should be observed by those who treat and those who suffer from OSA is the possible effects of drugs that depress the central nervous system and affect breathing. For example, alcohol consumed before sleeping can induce obstructive apnea in otherwise heavy snorers and cause longer periods of apnea with a greater degree of oxyhemoglobin desaturation, the severity being proportionate to the amount of alcohol consumed.² Besides alcohol, tranquilizers, sleeping pills, barbiturates, and similar drugs that have a depressing effect on the central nervous system should be avoided or used only after careful consideration of their potentially serious effects.

WEIGHT LOSS

As persons gain weight, the adequacy of the airway is compromised because of the deposition of extra adi-

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The senior author owns the patent rights and has a commercial interest in the Thornton Adjustable Positioner (TAP) and owns proprietary copyright to the Thornton Snoring Scale (TSS).

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pose tissue. This may explain why most OSA patients are overweight or obese. The degree of obesity required before OSA is established varies inversely with the degree of intrinsic inadequacy of the pharyngeal anatomy.³ Some patients are obese before developing the condition, and many can identify an increase in their symptoms with an increase in their weight. The initial mode of therapy, though often futile, is weight reduction—particularly when obesity is a contributing factor. Reducing weight may eliminate the OSA or merely decrease it, depending on what factors other than obesity are present. A study showed 16 patients with an average weight loss of 20 kg had less apnea, oxygen desaturations, and daytime sleepiness than a control group who reported no weight loss.⁴ The long-term loss of a large amount of excess weight by dietary measures alone is usually unsuccessful, and the symptoms of OSA return as the weight is regained.^{5,6} Careful consideration must be given when blaming the patient's current weight, because in some the added weight is a consequence of reduced activity secondary to the development of OSA rather than its cause.

Despite the associated problems, weight reduction remains the initial mode of therapy in obese patients with OSA. Weight gained by the obese patient was acquired over several years, and the anticipated loss of these pounds should also take an extended time. A secondary mode of treatment should be considered to benefit the patient in the interim.

SLEEP POSITION

Both snoring and sleep apnea are usually worse while sleeping in the supine position. The contribution of sleep position has been recognized as more than a trivial matter in the manifestation of OSA. One study has shown that in a group of 24 unselected patients with a diagnosis of OSA, the Apnea + Hypopnea Index (AHI) was twice as high when the patients slept in the supine position as it was when they slept in the lateral decubitus position.⁷ This finding of a substantial positional effect has led to many home and commercial remedies intended to train apneic patients who have a marked worsening of their condition while in the supine position to avoid this sleep posture. The two most common home devices are a tennis ball placed in a sock and sewn in the midline of the pajama top, and a pillow attached to the sleeper's back by a belt around the waist. Whenever the patient becomes supine, the tennis ball causes enough discomfort to make them reposition, whereas the pillow, if large enough, completely prevents the supine position. An example of a more technical approach would be a gravity-activated position monitor/alarm worn on the chest that emits an auditory signal when the patient remains in the supine position for more than 15 seconds. A study

using this device on 10 male patients diagnosed with OSA associated with the supine sleep position showed a significant decrease in the number of apneic events as well as the number of episodes of significant O₂ desaturations. While wearing the alarm, the apnea index of seven patients remained within or near normal limits.⁸ Data from this as well as other studies also suggested that a treatment based on changing sleep position might be selectively effective for those who were close to their ideal weight. It appears that training patients to avoid the supine position may have some validity as a noninvasive treatment when considered as a single therapy or in combination with others.

PHARMACOLOGIC OPTIONS

Pharmacologic agents can, on occasion, be used with some degree of success in the treatment of OSA by increasing glossopharyngeal neurologic activity or decreasing rapid eye movement (REM) sleep. Among the medications most often used are protriptyline and theophylline. Because these drugs cannot be used for all patients, the search for the ideal drug continues. Moreover, some patients cannot tolerate or simply refuse this treatment option.

The most common and best studied medication for OSA is protriptyline, a tricyclic antidepressant.⁹ It was initially introduced for treatment of OSA based on its ability to reduce the frequency of apnea and oxygen desaturations during non-REM sleep, while at the same time suppressing REM activity, the stage in which apneas tend to last the longest. It was later found also to specifically increase the tone of the upper airway muscles.¹⁰ However, severe anticholinergic side effects such as dry mouth, urinary retention, constipation, and impotence are evident in approximately half of the patients taking this drug, thus limiting its use.¹¹

Theophylline is another medication from which some patients may benefit. In a study of 12 patients, Mulloy and McNicholas¹² showed a decrease in the total number of apneas and hypopneas, as well as in the AHI, but at the same time sleep quality deteriorated significantly. Unfortunately, there is no reliable method of predicting which patients with OSA are likely to benefit most from theophylline, although it is believed that it may be those with mild disease.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

Nasal CPAP therapy was introduced by Sullivan et al¹³ in 1981 for the treatment of OSA. Nasal CPAP is produced by a high-flow blower that delivers a continuous stream of room air into a sealed nasal mask that the patient wears while sleeping. The positive pressure created in the circuit pneumatically splints the pharyngeal airway open by preventing the soft palate and

tongue from occluding it.¹⁴ This therapy abolishes hypopneas, apneas, oxygen desaturations, and apnea-related sleep fragmentation in most patients. The result is rapid restoration of normal sleep and reduction of daytime sleepiness.¹⁵ Patients should be observed in a sleep laboratory to determine the optimal CPAP level. Depending on several factors, this will vary from patient to patient and time to time.

The objective of nasal CPAP is to provide sufficient pressure in the collapsible segment of the upper airway to counteract the inspiratory suction pressure. At any instant of the inspiratory cycle, there is a pressure gradient down the entire airway from the nose to the alveoli. The further down the airway, the more negative is the intrairway pressure. In the extrathoracic airway, a transmural pressure gradient during inspiration tends to constrict the airway. In the oropharyngeal muscular tube, this suction pressure is sufficient to close the airway during sleep in patients with OSA. In some patients, the upper airway closes during sleep without a transmural pressure gradient. To maintain upper airway patency, these patients depend entirely on the presence of a sufficient tone in the upper airway musculature. Nasal CPAP effectively places the entire airway in a higher range of static "atmospheric" pressure so that the whole respiratory cycle takes place above atmospheric pressure. At no stage does the oropharyngeal transmural gradient become negative. This provides a pressure splint to the segment vulnerable to closure.²

The major concern with nasal CPAP therapy is long-term compliance, because continued use of the device requires considerable patient commitment. Patients who suffer the most from this disorder are more likely to be consistent with its use. When studies were performed that covertly monitored actual CPAP use, compliance was found to be less than what was self-reported by the patients.¹⁶ This is why attempts are being made to improve the device to increase its use. Examples of this would include a ramp feature, which starts at a lower pressure and increases gradually after the patient falls asleep, and a bilevel CPAP (BiPAP), which has different expiratory and inspiratory pressures.

Advantages of CPAP

The immediate response to nasal CPAP is dramatic. Typically, within moments of its application, the patient begins to have long periods of uninterrupted sleep, marked rebound in stage 3 to 4 nonrapid eye movement (NREM) sleep occurs, and both the frequency and duration of this stage of sleep increase dramatically. In addition, there are extraordinarily long periods of REM sleep characterized by high-density phasic activity. After CPAP therapy, arousal responses to a variety of

stimuli are markedly depressed during the first few nights of treatment. The sleep rebound continues over a number of nights. By the seventh to tenth night the sleep pattern, distribution, and arousability return to more normal levels. The daytime function of patients is typically transformed. The loss of daytime somnolence is the most characteristic improvement, but other dramatic changes also are seen. Quantitative tests of neuropsychiatric function often show a reversal from a pattern of cognitive deficits, described as unequivocally "brain damaged" to normal.²

ORAL APPLIANCES

Mandibular advancement appliances (MAA), and other devices that alter the relative position of the upper and lower jaws and pharyngeal soft tissues, have a role in treating OSA. They can be divided into the following three basic categories: tongue-retaining devices (TRD), fixed MAAs, and adjustable MAAs. Each of these can be effective; however, one must understand how each works and, in turn, be able to select the one that will benefit the patient the most as well as ensure a high degree of compliance.

The TRD uses suction, caused by placing the tongue into a cup or bubble positioned between the anterior teeth, to hold the tongue in an anterior position while the patient is sleeping and preventing it from falling back and being drawn downward by the negative pressure of inspiration. It is effective as long as the suction seal is maintained, which usually is less than half the night. In one of Cartwright and Samelson's studies,¹⁷ 14 patients with OSA showed a significant improvement in their sleep quality, which was indicated by fewer apneic episodes, with shorter duration, on the evenings that the TRD was worn. Another study reported that even when the TRD was only worn for half the night, the patient reached the same level of effectiveness as with uvulopalatopharyngioplasty (UPPP); however, a patent nasal airway must be present for this treatment to be effective.^{18,19}

The MAA moves the mandible forward several millimeters, while passively bringing the tongue with it, keeping the pharyngeal airspace patent during sleep. The optimum amount of anterior advancement of the mandible is in the range of between 50% and 100% of the patient's maximum protrusive movement. Maximum protrusion will increase in most patients as the muscles and their attachments become more elastic. This forward position can be maintained by using a one-piece, or fixed, appliance that holds the maxilla and mandible together, with retention being provided by clasps, acrylic or a thermoplastic polymer. Anterior breathing holes may be necessary for some patients to allow oral respiration, especially for those with restricted nasal airflow.

A more ideal design for the appliance is the nonfixed MAA, which involves the construction of separate devices for the maxilla and mandible. A separate appliance for each arch makes fitting easier and makes it more difficult to dislodge, because removal of the appliance is in a different path than mouth opening. Connecting the upper and lower appliances is accomplished with intra-arch elastics and buccal tube and rod attachments (Herbst appliance), or a single hook and latch in the anterior region (Thornton Adjustable Positioner). Because of their versatility and ease of adaptation, these appliances are more effective than the one-piece appliance, especially for patients with active bruxism or those who feel restricted. Such patients may not be able to tolerate the rigid fixation of their jaws with the one-piece design. The objective of the design is to restrict all retrusive movement while still allowing the patient to move the mandible forward and side to side, as well as to open the mouth if necessary.

A review of 20 publications reporting the effects of oral appliances on OSA in more than 300 patients showed an improvement in average AHI with a dental appliance. When statistics were provided, the decrease in AHI was always significant ($P < .05$), and the mean AHIs before and with treatment were 42.6 and 18.8, respectively, an average reduction of 56%.²⁰ An improvement in oxygen saturations was also noted, and the time of sleep with oxygen saturation $<90\%$ was reduced from 4.4% to 3.1%.²¹

Advantages of Oral Appliances

The MAAs appear to offer significant benefits for many patients with habitual snoring and OSA. They increase the airway space, stabilize the mandible in an anterior and closed position, advance the tongue, and increase genioglossus muscle activity.²² The advantages of oral appliance therapy (OAT) are simplicity, reversability, and cost-effectiveness. It may also become the primary treatment in patients who are unable to tolerate nasal CPAP or who are poor surgical risks. OAT offers the most logical way to initiate treatment in most cases of OSA, because it is readily accepted by most patients and can supplement other treatments in the small percentage of instances in which the dental devices alone do not bring sufficient relief.²³

An advantage of the nonfixed MAAs over other MAAs is the ability to systematically pinpoint the exact mandibular position that benefits each patient the most. It is possible to start at 50% or 75% of maximum protrusion at the initial appointment and then gradually advance the mandible until all signs and symptoms not only improve but completely disappear. When using a fixed MAA one is forced to pick an arbitrary forward position and gamble on what results might be achieved.

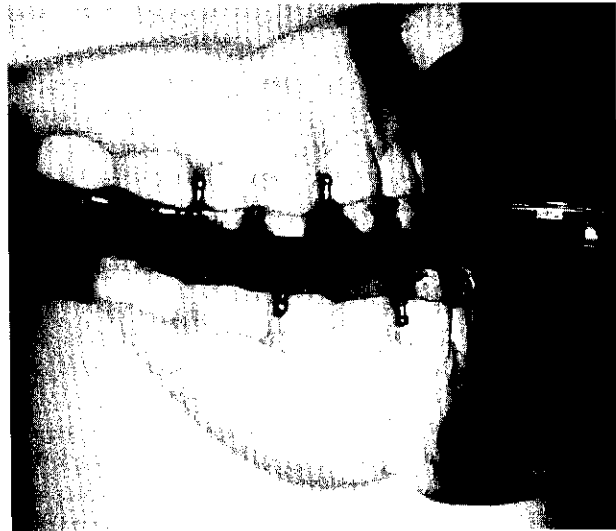


FIGURE 1. One of the TAP oral appliance designs used to treat patients with OSA.

The position at which each patient experiences maximum benefit is unique to that individual and, at this time, cannot be determined before constructing the appliance.

Experience With Oral Appliances

Most of our clinical experience has been with the Thornton Adjustable Positioner (TAP) (Figure 1), which may be more accurately described as a device that limits the mandible from retruding or opening while asleep. More than 300 of these have been delivered during the past 3 years to patients with chronic, heavy snoring who have been observed not to breathe for short periods during sleep, or for whom the shear noise has disrupted their homes and even their relationships. Some of the patients were referred from sleep laboratories with OSA, but most were patients who had heard of the appliance and wanted it simply to help their snoring. A significant number of these patients had already tried several options, including everything from inexpensive devices and other home remedies purchased at the drug store to CPAP and to surgery, which included (uvulopalatopharyngoplasty), (laser-assisted uvulopalatoplasty) and correction of a deviated septum.

Each patient had a thorough examination of the oral cavity and temporomandibular joint, and was asked to complete the Epworth Sleepiness Scale (ESS) and the Thornton Snoring Scale (TSS) which measures the effect of snoring on the quality of life of the patient and the outcome of treatment. An overnight pulse oximeter study was used to rule out OSA for those patients who had not had a sleep study performed. An investigation conducted by Series et al²⁴ showed that home nocturnal

oximetry interpretation was shown to be 95% correct, with a specificity of 98.2% and a sensitivity of only 47.7%.²⁴ The test is excellent in its negative predictive value for OSA.

After adequate information had been collected and the patients were educated about their disorder and the TAP, alginate impressions of each arch were taken, without any need for interocclusal records, and sent to the laboratory for fabrication. At the next appointment, the appliance was fitted, with elimination of all posterior contacts and positioning of the mandible to approximately 75% of maximum protrusion for most patients. The early appliances were made from acrylic, which required relining for adequate retention and adding acrylic at chairside to advance the mandible. The appliance has since evolved into two thin acrylic pieces that cover the occlusal surfaces of all the teeth. A thermoplastic polymer or orthodontic clasps are used for retention. An adjustable screw in the anterior region allows the patient or their bed partner the freedom to adjust the appliance by titrating to the most effective position, particularly on those nights when their symptoms or snoring are worse because of allergies, alcohol use, medications, or overexertion. This feature eliminates patient inconvenience, as well as valuable chair time for the dentist. Once the mandible has been correctly positioned, a follow-up home nocturnal oximetry study is done and the patient is placed on 6-month recall, with other appointments scheduled by the patient as needed.

Clinical Results of Oral Appliance Therapy

Although no blinded controlled study has been completed, our clinical results warrant further investigation into the effectiveness and limitations of OAT. In a follow-up phone survey of 208 of 300 patients who had worn the appliance for over 6 months, only 4 said that they felt the appliance had not improved their snoring; 81% were still wearing it an average of 25 days per month. Those no longer wearing it mentioned reasons such as weight loss or no longer having a sleep partner. Only 8 patients mentioned experiencing pain that prevented them from wearing it, and none of them had contacted our office about the situation. Three of the 300 patients had not been able to use the appliance; each of them had no specific complaint regarding pain or discomfort, only that it was difficult mentally to sleep with something in their mouth.

Finally, dental morbidity was determined by a follow-up examination. Some of the side effects experienced by these patients included tooth and jaw tenderness after the initial delivery or after an adjustment which usually subsided within a few days. A temporary bite change occurred in almost all patients, which lasted an average of 15 to 30 minutes each morning

immediately after removal of the appliance. A permanent posterior open bite occurred in 9 of the 300 patients within the first 6 months of wear. There were no associated temporomandibular disorders, and all 9 had a greater range of motion with less joint noise. All 9 were equilibrated with no further evidence of instability. It should be noted that these 9 patients were men with a history or evidence of nocturnal bruxing.

Disadvantages of Surgical Treatment

Because the source of the obstruction involves anatomic structures, surgery has been used as a treatment for some of these patients. Currently, some of the procedures used in the surgical treatment of OSA include tracheostomy, UPPP, and orthognathic surgery. These surgical procedures can be accompanied by various complications and limited success rates, and involve considerable expense.

Tracheostomy was the first surgical procedure to be used for the treatment of OSA. It is curative because it bypasses all the obstructive upper airway sites. Although improvement of the manifestations of sleep apnea is dramatic, patients may have a new set of problems develop related to the tracheostomy. These include complications, such as bleeding, stoma narrowing, and granulation tissue formation leading to hemoptysis and tracheal obstruction requiring surgical resection. Postoperative wound infection and recurrent purulent bronchitis requiring hospitalization or antibiotics have also occurred. Associated psychological problems have included depression, substance abuse, and marital problems.²⁵ With the advent of better-tolerated options, tracheostomy is now reserved for patients with morbid illness.

UPPP was first proposed by Fujita et al¹⁸ as an alternative to tracheotomy in the treatment of OSA. However, objective results determined by repeated sleep studies have shown variable results, with less than ideal success rates. In a study of 155 patients the procedure was only about 50% effective in curing or considerably improving OSA.^{26,27} Complications included bleeding, infection, swallowing difficulty, impaired speech, nasal reflux, dry mouth, increased gag reflex, and recurrence of snoring.^{26,28}

Orthognathic surgery should be considered in selected patients with specific anatomic characteristics in whom simpler therapies have failed. Intraoperative and immediate postoperative complications include hemorrhage, infection, airway obstruction, and anesthetic complications. Others involve transient anesthesia of the cheek and chin, altered dental occlusion, and prolonged maxillomandibular fixation. However, the one consideration least related to the patient's health, but often the most important determining factor, is cost. In addition to the cost of anesthesia services,

hospital stay, and surgical fees, the inclusion of techniques such as somnolineuromyography, three-dimensional magnetic resonance imaging, or dynamic computed tomography cephalometrics preoperatively, can make the expense reach large proportions. Therefore, the standpoint of cost alone, surgical treatment is substantially more burdensome.²⁹

Conclusion

There are many different nonsurgical modes of treatment for obstructive sleep apnea. Some can be used independently of one another; others can be used as part of an overall treatment plan. What is important is that the practitioner develop a new paradigm for diagnosis and treatment of this disorder. This paradigm must recognize the available modalities and prescribe a logical sequence for treatment. Oral appliance therapy should become a part of this paradigm. Most patients seek treatment for their snoring because of social repercussions, and only a few recognize the life-threatening consequences of OSA. OAT offers a cost effective, user-friendly method for combating this condition.

References

1. Wiegand L: Disease-a-Month, Obstructive Sleep Apnea, vol XI, no 4. St. Louis, MO, Mosby, 1994, pp 203-204
2. Sullivan CE, Issa FG: Obstructive sleep apnea. *Clin Chest Med* 6:633, 1985
3. Sher AE: Obstructive sleep apnea syndrome: A complex disorder of the upper airway. *Otolaryngol Clin North Am* 23:593, 1990
4. Smith PL, Gold AR, Meyers DA: Weight loss in mild to moderately obese patients with obstructive sleep apnea. *Ann Intern Med* 103:850, 1985
5. Johnson D, Drenick E: Therapeutic fasting in morbid obesity. *Arch Intern Med* 137:1381, 1977
6. Bennet W: Dietary treatments in obesity. *Ann N Y Acad Sci* 499:350, 1987
7. Cartwright R: Effect of sleep position on sleep apnea severity. *Sleep* 7:110, 1984
8. Cartwright RD, Lloyd S, Lillie J, et al: Sleep position training as treatment for sleep apnea syndrome: A preliminary study. *Sleep* 8:87, 1985
9. Cohen RB: Obstructive sleep apnea: A mandibular positioning device for treatment and diagnosis of an obstruction site. *Compendium* 16:618, 1995
10. Bonora M, St John WM, Bledsoe TA: Differential evaluation of protriptyline and depression by diazepam of upper airway respiratory motor activity. *Am Rev Respir Dis* 131:45, 1985
11. Brownell LG, Wast P, Sweatman P, et al: Protriptyline in obstructive sleep apnea: A double blind study. *N Engl J Med* 307:1037, 1982
12. Mulloy E, McNicholas WT: Theophylline in obstructive sleep apnea: A double blind evaluation. *Chest* 101:753, 1992
13. Sullivan CE, Issa FG, Berthon-Jones M: Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1:862-5, 1981
14. Sullivan CE, Berthon-Jones M, Issa FG: Remission of severe obesity-hypoventilation syndrome after short-term treatment during sleep with nasal continuous positive airway pressure. *Am Rev Respir Dis* 128:177, 1983
15. Hoffstein V, Viner S, Mateika S: Treatment of obstructive sleep apnea with nasal continuous positive airway pressure. *Am Rev Respir Dis* 145:841, 1992
16. Rauscher H, Popp W, Wanke T: Acceptance of CPAP therapy for sleep apnea. *Chest* 100:1019, 1991
17. Cartwright RD, Samelson CF: The effects of a nonsurgical treatment for obstructive sleep apnea. *JAMA* 248:705, 1982
18. Fujita S, Conway W, Zorick F, et al: Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: Uvulopalatopharyngoplasty. *Otolaryngol Head Neck Surg* 89:923, 1981
19. Fujita S, Conway W, Zorick F: Evaluation of the effectiveness of uvulopalatopharyngoplasty. *Laryngoscope* 95:70, 1985
20. Schmidt-Nowara W, Lowe A, Wiegand L, et al: Oral appliances for the treatment of snoring and obstructive sleep apnea: A review. *Sleep* 18:501, 1995
21. O'Sullivan RA, Hillman DR, Mateljan R, et al: Mandibular advancement splint: An appliance to treat snoring and obstructive sleep apnea. *Am J Respir Crit Care Med* 1995 (in press)
22. Lowe AA: Dental appliances for the treatment of snoring and/or obstructive sleep apnea, in Kryger MH, Roth T, Dement WC: *Principles and Practice of Sleep Medicine* (ed 2). Philadelphia, PA, Saunders, 1994, p 733
23. George PT: Treatment of snoring and obstructive sleep apnea with a dental device. *General Dentistry* July-Aug:294, 1993
24. Series F, Marc I, Cormier Y, et al: Utility of nocturnal home oximetry for case finding in patients with suspected sleep apnea hypopnea syndrome. *Ann Intern Med* 119:449, 1993
25. Conway WA, Victor LD, Magilligan DJ, et al: Adverse effects of tracheostomy for sleep apnea. *JAMA* 246:347, 1981
26. Simmons FB, Guilleminault C, Miles LE: The palatopharyngoplasty operation for snoring and sleep apnea: An interim report. *Otolaryngol Head Neck Surg* 92:375, 1984
27. Guilleminault C, Hayes B, Simmons FB: Palatopharyngoplasty and obstructive sleep apnea syndrome. *Bull Eur Physiopathol Respir* 19:595, 1983
28. Hernandez SF: Palatopharyngoplasty for the obstructive sleep apnea syndrome: Technique and preliminary report of results in ten patients. *Am J Otolaryngol* 3:229, 1982
29. Anand VK, Ferguson PW, Schoen LS: Obstructive sleep apnea: A comparison of continuous positive airway pressure and surgical treatment. *Otolaryngol Head Neck Surg* 105:382, 1991