Sleep, 19(9):S101–S110

Management of Simple Snoring, Upper Airway Resistance Syndrome, and Moderate Sleep Apnea Syndrome

*†Patrick Lévy, *‡Jean-Louis Pépin, *§Pierre Mayer, *†Bernard Wuyam and ‡Dan Veale

**Sleep and Respiration Unit, Grenoble University Hospital, Grenoble, France; †PRETA Laboratory, Physiology Department, Grenoble University, Grenoble, France; and ‡AGIR, Regional Home Care Association, Grenoble University Hospital, Grenoble, France*

Summary: The spectrum of respiratory sleep disorders has been extended in the last years to include conditions that are less well defined than severe obstructive sleep apnea (OSA). Moderate OSA, snoring, and upper airway resistance syndrome (UARS) represent three conditions in which there are still unresolved pathophysiological, epidemiological, and clinical questions. Therefore, the therapeutic approach remains unclear. We have tried to define these entities and to review the respective indications and efficacy of pharmacological treatment, weight loss, sleep posture, oral appliances, upper airway surgery, and, finally, continuous positive airway pressure (CPAP). From these data, we also aim to define strategies of treatment for moderate OSA, snoring, and UARS. However, these conditions are likely to be particularly appropriate for randomized trials comparing different modalities of treatment that may be the only way to validate these treatment strategies. **Key Words:** Snoring—Moderate OSA—Upper airway resistance syndrome—Weight loss—Sleeping position—Oral appliances—Pharmacological treatment—Surgery.

In the last decade, obstructive sleep apnea (OSA) has been identified as a common clinical condition. Recent epidemiological studies have confirmed a high prevalence of the disease in middle-aged adults (1). OSA is associated with significant neuropsychological impairment and a high cardiovascular morbidity, for which causal relationships are postulated (2). Since 1981, nasal continuous positive airway pressure (CPAP) has been the first-line therapy for OSA (3). However, there are important side effects related to CPAP use (4), and although there is no doubt that nasal CPAP is nearly the only therapeutic possibility in severe OSA, other alternatives are highly desirable in moderate OSA. This is even more important in non-apneic situations such as snoring and upper airway resistance syndrome (UARS). UARS is an abnormal increase in upper airway resistance during sleep that results in daytime sleepiness (5).

The treatment of OSA and other abnormal increases of upper airway resistance during sleep should achieve three goals: 1) alleviate symptoms, 2) reduce morbidity, and 3) decrease mortality. There is, however, an additional goal that may become more and more important in the near future: to improve quality of life. Consequently, the choice of a particular treatment for a given patient should induce the lowest possible level of side effects while the same rate of success is achieved for the three goals listed above. If we use these criteria for assessing different modes of treatment, it is obvious that the morbidity associated with the different clinical situations must be clearly established.

In this paper, we have tried to define moderate OSA, simple snoring, and UARS, and their respective morbidities. Furthermore, we have reviewed the effects of pharmacological therapy, weight loss, sleeping position, oral appliances, and surgery. Finally, we have attempted to define a strategy of management in each of these clinical situations through specific algorithms.

Accepted for publication July 1996.

Address correspondence and reprint requests to Dr. Patrick Lévy, Département de Pneumologie, Unité Sommeil et Respiration, CHU de Grenoble, BP 217 X, 38043, Grenoble, France.

§Present address: Department of Respiratory Medicine, Hôtel-Dieu de Montréal, Montréal University, Quebec, Canada.

Definitions

There are several epidemiological and clinical questions that arise when defining, respectively, sleep ap-

nea, snoring, and UARS. These three entities overlap each other. The percentage of UARS and OSA without snoring is still unknown. Although it seems that a very small subset of OSA patients do not exhibit any detectable snoring, UARS may be present in non-snorers (5), particularly in women (6). In both situations, however, the detection of snoring may be critically affected by the method used to detect it. An objective assessment of snoring is lacking in most of these studies. The description of UARS has raised the question of the number of snorers exhibiting this syndrome.

Snoring is a vibration that presumably always corresponds to some extent to an increase of upper airway resistance. However, snoring is a very frequent symptom, affecting 19% to 37% of the general population and more than 50% of middle-aged men (7). There are still some questions about the effective health risk associated with snoring without any apnea or hypopnea (8), although a continuum has been proposed from snoring to sleep apnea (9).

In contrast, one needs specific criteria to diagnose UARS. The original definition (5) included a clinical complaint [daytime sleepiness ± abnormal multiple sleep latency tests (MSLT)], flow limitation [detected by esophageal pressure (P_{es}) monitoring], and increased respiratory efforts with arousal just following the peak negative P_{es}. Although these criteria do not always seem to be appropriate [nearly 30% of the women with UARS have a normal arousal index despite significant respiratory efforts (6)], this is a clear situation apart from "simple snoring", where the upper airway resistance may be slightly or moderately increased without sufficient respiratory efforts to produce microarousals and/or generate daytime somnolence. The percentage of UARS among a population of snorers remains unknown. The only studies providing insight into this area are clinical searches for daytime somnolence in snorers, usually assessed by questionnaire. Mendelsen found that 24% of 58 patients referred for primary snoring presented with daytime somnolence (MSLT = 8.7 ± 0.9 minutes) (10). In another study, up to 50% of 118 patients with a respiratory disturbance index (RDI) of less than 5 had significant hypersomnia (11). However, the frequence of UARS is overestimated in these studies, because the patients were referred to a sleep laboratory for suspected sleep breathing disorders—an important bias of selection. Finally, simple snorers should be defined as patients with loud snoring without any excessive daytime sleepiness and with insufficient increase in upper airway resistance to result in repeated microarousals or sleep disruption.

Defining moderate sleep apnea syndrome may not be easier. The severity of OSA usually refers to the number of apneas plus hypopneas per hour of sleep, as expressed by RDI. One may consider that a RDI between 10 and 30 defines mild to moderate OSA. However, this definition is probably insufficient, because the morbidity associated with OSA is variable at the same level of RDI. This is obvious in regard to daytime sleepiness and is also expected regarding cardiovascular morbidity. The evaluation of moderate sleep apnea syndrome is even more complex because the magnitude of overnight oxygen desaturation and the duration of the disease are difficult to assess. These parameters affect the validity of intraindividual comparisons. Another factor is the night-to-night variability, which is more pronounced in mild apneics, especially in the elderly (12). Therefore, a definition of moderate sleep apnea syndrome cannot rely only on RDI. The best compromise could be the combination of the three following criteria: 1) RDI less than 30, 2) moderate sleepiness (i.e. Epworth sleepiness scale value between 9 and 12), and 3) absence of any cardiovascular morbidity related to OSA.

Thus, from a clinical point of view, the end-points of treatment should be to alleviate snoring in simple snorers and to alleviate both snoring and daytime somnolence in UARS. In moderate OSA, elimination of apneas and hypopneas, and perhaps flow limitation, should be obtained together with the suppression of snoring and excessive daytime sleepiness.

Pharmacological treatment

A huge number of drugs have been tested as treatments for OSA, with very little success (13). Theophylline, almitrine, and other ventilatory stimulants have virtually no effect. Nicotine has been recently tested using transdermal application, without significant effects either on sleep-disordered breathing or on snoring (14).

Protriptyline is probably the drug that has been used most commonly for treating OSA. Most studies report protriptyline to be effective in 50% to 70% of cases of OSA (15,16) but its usefulness is limited by anticholinergic side effects. A more recent paper compared the action of protriptyline to that of fluoxetine, a specific serotonin agonist acting as a serotonin re-uptake inhibitor (17). Both drugs reduced the percentage of rapid eye movement (REM) sleep (baseline: 17% ± 2% vs. 3% ± 1% with protriptyline and 7% ± 3% with fluoxetine). For the whole group of patients, the RDI decreased from 57 ± 9 to 34 ± 6 and 33 ± 8 with fluoxetine and protriptyline respectively. The RDI in REM sleep remained unchanged in both cases. Fifty percent of the 12 patients were considered by the authors (17) as having a definite beneficial response to one or both of these medications. Finally, tolerance to fluoxetine was better than that of protryptiline. How-

ever, these drugs should be used mainly in REM-related OSA, if at all.

Sleep apnea is more prevalent in men, and testosterone has known effects on ventilatory control. Therefore, hormonal therapy has been tested in OSA. The reduction of androgen activity was obtained in male patients using flutamide, a nonsteroidal antiandrogen that acts as a competitive inhibitor of androgen binding to the androgen receptor both centrally and peripherally and provides a complete androgen blockade by 1 week (18). In eight men with moderate to severe OSA [RDI = 41 ± 4 in non-rapid eye movement (NREM) sleep and 53 ± 4 in REM sleep], this resulted in no changes in sleep architecture and ventilatory responses to hypoxia and hypercapnia. Moreover, the reduction in sleep-disordered breathing obtained both during NREM and REM sleep was nonsignificant. The authors (18) suggested that men who had only heavy snoring or mild forms of sleep apnea might have been more sensitive to flutamide. However, there were no data in their own study (18) to support this hypothesis, and these drugs are not currently used in snoring and sleep apnea.

Logically, hormone replacement has also been tested in postmenopausal women, because women appear to be more susceptible to snoring and sleep-disordered breathing after the menopause. The results are contradictory, however (19–21). Healthy postmenopausal women were investigated in regard to sleep and respiration after placebo and after a combination of estrogen and progesterone during 1 week. The total number of apneas and hypopneas per night was reduced from 15 ± 4 to 3 ± 1 with combined progesterone and estrogen. It should be noted that these values of apneas and hypopneas correspond to those of normal subjects (RDI less than 2 events/hour of sleep at baseline) and not to those of OSA patients. The authors concluded that female hormones are important in protecting postmenopausal women against sleep-disordered breathing. Whether these results can be extrapolated to subjects with clinically significant respiratory disturbances during sleep requires further studies. A case report has also been published indicating that a probably severe OSA (RDI was not mentioned) was improved with estradiol and abolished using both estradiol and medroxyprogesterone treatment (20). However, a more recent study (21) examining the effects of short-term hormone replacement (either estrogen alone or in combination with progesterone, over a term of 50 days) only demonstrated a small and clinically insignificant reduction in RDI during REM sleep (47 vs. 58 events/hour of sleep), whereas the overall RDI did not change (40 vs. 43). The discrepancy between this last result (21) and the previous studies (19,20) might be related to the clinical characteristics of the subjects (severity of nocturnal respiratory disturbances, degree of obesity) and to the dose of hormone replacement therapy [i.e. high nonconventional concentrations of estrogen and progesterone (19) that are likely to be associated with significant side effects, precluding their clinical use].

On the whole, pharmacological treatment has little effect on apnea and hypopnea. It should be noted, however, that none of these drugs has been evaluated in an objective manner on snoring, nor tested in cases of UARS.

Weight loss

Approximately 70% of OSA patients are obese, that is, exhibiting a body mass index (BMI) of more than 28 kg/m^2 or a body weight in excess by more than 20% of the ideal weight (22). The relationship between obesity and OSA is still unclear. However, it is one of the most commonly recognized risk factors. Obesity appears to be largely determined by genetic factors that influence metabolic rate, fat storage, and eating behavior and are associated with autonomic, endocrinological, and hypothalamic function abnormalities. This is particularly true in regard to regional fat distribution (23), which may be of particular relevance to the pathogenesis of OSA. In OSA, upper body obesity may be a relatively greater risk factor than is total body fat mass; it has been shown that neck circumference is a better predictor of the presence of OSA than BMI (24). Weight loss has definite effects and results in improvement or, in exceptional cases, disappearance of sleep-related breathing disorders (25). There is also a strong influence of weight reduction on snoring frequency and intensity. Both may result from the decrease of pharyngeal collapsibility obtained with weight loss (25). Furthermore, weight loss is not only associated with a reduction in RDI and collapsibility but also with a nearly complete elimination of apnea when the critical pressure (P_{crit}) reflecting the collapsibility is lowered below −4 cm H_2O (26).

Many studies have examined the effects of weight loss on sleep-related breathing disorders and sleep architecture (26–33). The improvement in RDI due to weight loss, however, although it is found systematically, is only partial. This is clearly the case in moderate to severe sleep apnea syndrome, and it is also found in mild to moderate sleep apnea (33). In this last study, there was no improvement in RDI when compared with modified sleeping position and the use of nasal spray (13 ± 6 vs. 14 ± 6) (33). When examining the 12 subjects who lost any amount of weight, there was a significant reduction in the mean number of snores as compared to baseline but not as compared

to the two-modality treatment night (sleeping position and nasal spray). The effect of weight loss became significant, however, for patients who lost at least 3 kg. The authors (33) concluded that weight loss plus sleeping position and nasal spray had no effect on the apnea/hypopnea index (AHI), but that the major effect of the combination of these three treatments on the frequency of snoring appeared to be related to weight loss.

Like other treatment modalities, it is important to reassess patients fully after weight loss and ensure that there is little or no residual disordered breathing. Occasionally, patients will have substantial reduction in weight and a parallel cessation of sleep apnea, but no improvement may be observed in other patients despite dramatic weight loss. Weight loss is difficult to achieve and particularly difficult to maintain. Several studies have investigated the effects of surgical procedures directed towards a reduction in BMI on respiratory disturbances (27,28,31). Jejuno-ileal shunt, gastroplasty, or gastric bypass have been advocated to ensure weight loss and maintenance of the weight reduction. However, consideration must be given to the operative risk when administering an anesthetic to a supine patient with obstructive sleep apnea syndrome (OSAS) (34), especially in morbidly obese patients, who are the best candidates for this surgery (35).

Finally, in any case weight loss should be encouraged in obese OSAS patients. A small subset of patients may be cured by a significant and maintained weight loss. Moreover, other modalities of treatment may either require some weight loss (i.e. maxillofacial surgery) or be more easily applied due to the changes in compliance and resistance of the upper airway secondary to weight reduction (i.e. nasal CPAP). Weight loss also has beneficial effects on snoring; there are no data related to weight loss and UARS.

Sleep posture

It has long been recognized that snoring patients do so most loudly in the supine position. Similarly, it has been well proven that a large proportion of unselected patients with a diagnosis of OSAS demonstrate a different rate of apneic events in the lateral compared to the supine position (36–41). Positional sleep apnea syndrome has been defined as an AHI during the time in supine sleep that is two or more times the AHI during sleep in the lateral position (41). Up to 60% of 184 unselected cases of OSA investigated in a sleep laboratory were reported to meet this criterion (42). A tennis ball sewn into the pajama top at the midthoracic level was one of the first means used to prevent sleep in supine position. At the present time, two different strategies are available: sleep position training using a posture alarm (PA) device (37) and a tongue retaining device (TRD) designed to prevent tongue retrolapse when the patient sleeps in the supine position (43).

The PA device was first tested in a small subset of patients in a preliminary study (37). The PA was designed to train the patient to avoid supine sleep by delivering an auditory beep if the patient maintained this posture for more than 15 seconds. Seven out of 10 patients had an AHI within normal limits while wearing this device. The device was further tested and compared with TRDs (43). Sixty OSA patients with positional sleep apnea were randomly assigned to one of the following groups: TRD, PA, both TRD and PA, and good health habits for sleep apnea. These good health habits included loss or maintenance of weight by controlling diet, exercise at least 20 minutes a day, no alcohol use after 6:00 p.m., and learning to avoid the supine sleep position. These instructions were given to all four groups and were the only modality of treatment in one group. The mean RDI at baseline was around 30/hour. All groups showed a marked improvement in RDI, although the mean value was not significantly improved in the PA group. When a determination was made of the number of subjects reaching "normal values" of RDI after treatment (<5.5/hour in the present study), between 53% and 60% of the patients met this criterion in the three single-treatment groups. The combined treatment group had the highest success rate (73%). The TRD was additionally useful to protect those who continued to sleep on their back part of the time. When looking at the predictive factors associated with success or failure of these treatments, five factors were identified. The severity of the original RDI, the severity of the lateral position RDI, and the initial weight were predictive of failure. The reduction of body weight obtained from the initial level to that of the post-treatment evaluation was predictive of success. Nasal patency was predictive of success only in the TRD group, because wearing a TRD obliges the patient to breath nasally (see below). A sixth factor was motivation, as illustrated by the good results (mean RDI from 27 to 8/hour after treatment, with 9 out of 15 patients reaching normal values of RDI) obtained in the health habits group, where patients were left on their own and had a strong motivation to help themselves (43).

Finally, lateral sleeping position has also been tested, together with a nasal decongestant (oxymetazoline, an alpha-agonist), in 20 "asymptomatic" male snorers (44). The mean pretreatment RDI was 18 ± 7/hour, but it should be noted that the median was at 2, whereas three patients had RDI values, respectively, at 61, 71, and 101/hour. The authors (44) observed a small improvement in RDI (14 ± 6/hour post-treatment) and no change at all in snoring frequency. In this subset

TABLE 1. *The effects of oral appliances upon snoring*

Study	No. of patients	Device	Snoring improved (%)	Comment
Bonham et al. (48)	12	MAD	73	Spouse report
Clark et al. (55)	24	Herbst MAD	YES	Subjective scale
Ichioka et al. (54)	14	MAD	100	Subjective score
Kloss et al. (1986) (77)	7	Esmarch	100	Patient report
Nakazawa et al. (1992) (78)	12	MAD	100	Patient report
O'Sullivan et al. (53)	51	MAD	100	Patient and laboratory
Schmidt-Nowara et al. (47)	68	SnoreGuard	98	Patient report
Lowe (1990) (79)	1	MAD	100	Lab measures
Lyon (1990) (80)	15	Elastomeric	100	Not specified

Table contents adapted from Schmidt-Nowara et al. (51).
Abbreviation used: MAD, mandibular advancement device, not otherwise specified.

of patients, an additional weight loss yielded no further improvement in RDI but significantly reduced the frequency of snoring (33).

These last results, although not very impressive in regard to the effects on RDI, and others, as mentioned above, support the use of several of these treatments in combination in simple or asymptomatic snorers and mild OSA patients. They should be tested in UARS.

Oral appliances

Oral appliances are commonly used by dentists for correcting various types of dental malocclusion. In the last decade, however, 13 devices have been specifically designed to treat snoring and sleep apnea (45,46). The term "oral appliances" defines devices inserted into the mouth in order to modify the position of the mandible, the tongue, and/or other structures in the upper airway for the purpose of relieving snoring or sleep apnea. There are two groups of devices available. One group is mandibular advancing devices. These devices use traditional dental techniques to attach the device to one or both dental arches and to change by this means the mandibular posture. Although these techniques generally require a process that includes taking dental impressions and fabrication by a dental laboratory, there is at least one device made of a thermolabile material that can be directly molded to the patient's teeth (47). Some devices restrict mouth opening. Some appliances generate a posterior extension of the maxilla that is designed to modify the position of the soft palate or the tongue. The second group of oral appliances, the tongue retainers, is designed to keep the tongue in an anterior position during sleep. This is achieved by means of negative pressure in a soft plastic bulb. These devices also modify mandibular posture, particularly by downward rotation. A prefabricated device allowing molding to the patient's teeth in the clinic is available (45).

The goal of therapy with oral appliances is to modify the position of upper airway structures in order to enlarge the airway, reduce resistance, and presumably to reduce upper airway collapsibility. The effects on upper airway muscle function may also be important, due to the changes in direction of muscle fibers. Cephalometric measurements have been used to demonstrate changes in upper airway dimensions due to the use of these devices. In awake patients, the space between the soft palate and the posterior wall of the pharynx was shown to be enlarged (48). There are conflicting results concerning the effects of these devices on the retrolingual region, as reflected by measurement of the posterior airway space (PAS), which was enlarged in some reports (47), even by more than 50% (49), and not enlarged in others (48). In another study (50), there was no observable change in PAS, but the distance between the hyoid bone and the mandibular plane (MP-H) was reduced. Moreover, the baseline MP-H distance and the post-treatment MP-H distance were found to be significantly shorter in responders (AHI <10) than in non-responders. In addition, the length of the soft palate showed a significantly greater reduction in responders, compared to non-responders (50).

A review and a report on practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances were published recently by a task force of the American Sleep Disorders Association (ASDA) (51,52). The authors selected 21 publications for the review (51). There was no randomized control study—only case series with comparisons before and after treatment. The effects of oral appliances are available for 204 patients, reported in nine publications for snoring, and for 304 patients in 20 publications in regard to sleep apnea.

The effects of these oral appliances on snoring are summarized in Table 1. A high proportion of patients showed improvement, whatever the type of device. In most studies, improvement in snoring was inferred from the reports of patients or bed partners. A recent

report, however, demonstrated a significant reduction in snore frequency and intensity, although snoring was not eliminated. This improvement was also verified by a questionnaire administered to 57 patients and their 50 partners (53).

Effects on the AHI were available in 271 cases. The mean AHI values before and after treatment were 42.6 and 18.8, respectively. Although 70% of the patients had at least a 50% reduction in AHI, only 51% reached a normal value (AHI <10/hour); 39% of the patients remained at more than 20/hour. From the 14 papers providing individual data, 13% of the patients appeared to be worsened by the treatment (AHI with the oral device greater than the initial value before treatment). Predictive factors of success are difficult to establish, because the populations studied were too limited. The initial AHI was found to be a negative factor in several studies (47,50,53). It has been suggested that success would be unlikely with an AHI of more than 50 or 60. However, this has not been confirmed in other publications (51). In one study, in addition to the initial AHI, cephalometric variables (length of the soft palate, MP-H distance, PAS, and posterior facial height) were predictive of the AHI value following treatment (50).

When comparing tongue retaining devices (TRDs) and mandibular advancement devices, there is no difference in terms of efficacy in eliminating apneas and correcting oxygen saturation (51). As previously mentioned, the TRD is a useful adjunct to position training (43).

Side effects of these treatments have been reported. Excessive salivation and transient discomfort after awakening are commonly experienced with initial use and may hinder early acceptance of the device (47,53). Later complications are essentially represented by temporomandibular joint discomfort and changes in dental occlusion. These complications were relatively uncommon, however.

Compliance has been evaluated on the basis of the patients' reports and in a limited number of objective studies. A partial use of the device has been found at the initiation of treatment in some patients (45). However, after adaptation, patients seem to use their device for the entire night and almost every night (47). The main question, regarding long-term compliance, remains. Compliance has been found to be up to 100% in 14 patients followed for 3–21 months (54) and 75% in 68 patients questioned after a median of 7 months of use (47). It seems that long-term compliance is much lower, however, reaching only 52% in 24 patients followed for 3 years (55). The main reasons for discontinuing the treatment were side effects and lack of efficacy.

When summarizing the indications, one can follow the recommendations of the ASDA (52). Oral appliances are indicated for use in patients with primary snoring or mild OSA who do not respond to or are not appropriate candidates for treatment with behavorial measures such as weight loss or sleep-position change. Patients with moderate to severe OSA should have an initial trial of nasal CPAP therapy because greater effectiveness has been shown with this intervention than with the use of oral appliances. Upper airway surgery should also be considered in this subgroup of patients. Oral appliances are indicated for patients with moderate to severe OSA who are intolerant or refuse treatment with nasal CPAP or who refuse or are not candidates for upper airway surgery. Finally, UARS represents a situation in which oral appliances might have beneficial effects. However, there are no data available for this at the present time.

Upper airway surgery

Upper airway surgery is obviously used largely for treating snoring and sleep apnea syndrome. This treatment is reviewed extensively in several other papers in this issue. There are, however, several key points for this discussion. The main characteristic of the studies reporting the effects of surgery on OSA and snoring is the lack of objective assessment, particularly regarding frequency and intensity of snoring (56). The second point is the rate of success of uvulopalatopharyngoplasty (UPPP). When defining surgical success as a reduction of 50% or more in RDI and a consequent achievement of an RDI of less than 20, a recent meta-analysis showed a response rate of 41% (57). Moreover, if success is defined by a normalization of sleep structure and an RDI of less than 10/hour, a quantitative evaluation is difficult to make from the literature. In fact, most series do not provide enough data to establish the effects of surgery on sleep (56).

Many studies have attempted to select good responders to UPPP. Indeed, the extension of upper airway collapse to the hypopharynx is associated with a poor outcome of UPPP. Many techniques have been proposed to predict the most likely anatomical level of obstruction in the pharynx. This has been done using pharyngeal endoscopy in sleeping patients during abrupt changes of pressure applied to the upper airway, therefore assessing the static properties of the pharynx (58). Pharyngeal pressure measurements have also been monitored in NREM and REM sleep, demonstrating that the upper airway collapse usually extends caudally in REM sleep and that having the collapse confined to the oropharynx preoperatively does not guarantee a successful outcome (59). As a whole, none of these techniques seem able to predict the responders to UPPP (60).

When an assessment is made of the indications for UPPP, there is a general agreement that UPPP has virtually no place in the treatment of severe OSA (61). For moderate OSA, the results are still contradictory. There are recent data demonstrating that the efficacy of UPPP may be poor, as well, in improving respiratory disturbances such as snoring frequency and intensity (62). There is a frequent dissociation, however, between subjective and objective results. In a recent paper, for example, UPPP was assessed in 155 non-apneic snorers (63). Snoring, sleep problems, excessive daytime sleepiness (EDS), and tiredness were graded using a multiple-choice questionnaire. The results at 3 (n = 105), 12 (n = 55) and 24 months (n = 49) were compared to those of non-surgically treated non-apneic snorers at 12 months. The authors found a significant reduction in snoring, EDS, and daytime fatigue in the UPPP group, whereas the symptoms remained nearly unchanged in the non-surgically treated group (63). In this study, the prevalence of EDS may be related to the presence of UARS among the heavy snorers treated with UPPP. Therefore, the discrepancy between objective and subjective results in previous studies may lie in an underestimation of the UARS if one admits that UPPP can result in a reduction of upper airway resistance, whatever the RDI. This hypothesis (64) is not supported by any objective fact, however, because at the present time there are no data concerning the assessment of UPPP in polygraphically proven UARS.

Moreover, skeletal changes that potentially increase upper airway resistance have been reported in UARS (6). In approximately 50% of women in this study, (6) exhibiting UARS, it was observed that a narrow ogival palate with a triangular chin and a variable dental overjet occurred. A class II malocclusion was present in nearly all of these subjects (141/156). In this group of women with UARS, the subjects without these anatomic features had marked obesity, with a BMI of more than 35 kg/m^2. Therefore, surgery dedicated to the correction of bony abnormalities would seem to be preferable to UPPP alone in UARS. However, there are no published data at the present time regarding the efficacy of maxillofacial surgery in UARS. Guilleminault et al. only suggested that nasal CPAP might not be an effective long-term treatment for this syndrome and that other means of treatment such as surgery might have to be considered (5).

The results of maxillofacial surgery in moderate and severe OSA are reviewed in two other papers in this issue. Maxillomandibular surgery is credited with the best surgical results available for OSA. It should be noted, however, that only one group has reported on a substantial number of patients in regard to the whole procedure (phase I, consisting of genioglossal advancement, hyoid myotomy and suspension, and soft palate surgery; and phase II, consisting of maxillomandibular osteotomy and advancement) (65,66). The rate of success of phase I [up to 79% (66)] surgery, which relies only on the experience of this group or on very limited published data (67), requires confirmation. In regard to phase II surgery, there is no doubt that the rate of success in OSA is very high (up to 90–95%). This success seems to be obtained either secondary to unsuccessful phase I surgery (65) or without phase I surgery (68). The remaining problem at the present time is to define selection criteria selection for this type of surgery. There is no clear information available in the literature on this subject, and we can only suggest from clinical experience that obesity and poor motivation for surgery might contraindicate the surgical procedure.

Continuous positive airway pressure

As mentioned above, CPAP remains the first-line therapy for OSA, even if the OSA is moderate. However, the key point is compliance. A few studies, coming mainly from the U.S., (69–71) have demonstrated low compliance and irregular use of the CPAP device. However, most of the other studies, using cumulative time meters, have found a high rate of compliance (ranging from 65% to 80%) (4, 72–76) and acceptance [about 15% of patients refuse this treatment after a single night's use in the laboratory (72,74)]. This high rate of compliance remains to be firmly established when monitoring the effective compliance (time spent at the effective pressure ± 2 cm H_2O). The differences in compliance that are observed may merely reflect the respective efficacy of technical and medical follow-up in the different countries. However, there are significant side effects that affect a majority of patients using CPAP therapy (4,76). The reason for a high compliance despite these side effects [daily use of 6.5 ± 3 hours, 88% of the patients using their device every night (4)] is obviously the clinical benefit obtained: only 1% of the patients had no subjective benefit induced by their therapy. Although compliance has not been studied in a large group of moderate OSA patients, the situation may be different in this condition, in which excessive daytime somnolence is limited.

There are only few data available in regard to CPAP use in UARS. The only results (11) published at the present time concerned snorers with EDS. The subjects were presumably UARS patients, but the authors (11) did not provide polysomnographic data to establish UARS with the usual criteria (see also Krieger et al., this issue). Rauscher et al. (11) studied 118 consecutive patients. Half of them reported significant daytime sleepiness. Only 19% of these 59 hypersomnolent pa-

tients accepted nasal CPAP for home therapy. The pressure needed to abolish snoring was 7.3 ± 1.6 cm H_2O which reduced the number of arousals from 20 ± 10 to 5 ± 3. Although compliance was low (2.8 ± 1.5 hours), the treatment resulted in a significant reduction of the sleepiness score. The long-term acceptance and mean rate of use was studied by Krieger et al. in non-apneic snorers (RDI less than 15) (this issue). The acceptance of therapy was greater than 60% at 3 years, with a mean rate of daily use of CPAP at 5.6 ± 1.4 hours/day. These conflicting results illustrate that further studies are needed to firmly establish CPAP compliance in polysomnographically proven UARS.

Strategies of treatment

Using the definitions suggested above for asymptomatic snoring, moderate OSA, and UARS, we would like to suggest different strategies of treatment using the various means of treatment we have reviewed.

In cases of simple or asymptomatic snoring, one can successively propose these different steps: 1) sleeping position control, weight loss achievement, 2) oral appliance use, and 3) surgical procedure. It might be unexpected that UPPP is not considered as the first-line therapy. However, this reflects the lack of objective results on snoring intensity and frequency. On the other hand, phase I maxillofacial surgery could be envisaged in this situation. There are, however, no data available at the present time.

Moderate OSA remains difficult to define. We suspect that RDI does not adequately reflect the severity of the disease. A combination of low RDI, moderate daytime sleepiness or tiredness, and absence of related cardiovascular morbidity may define moderate OSA. In this condition, one can suggest the following strategy: 1) a CPAP trial is recommended; 2) in cases of primary or secondary failure of CPAP therapy (patients refusing CPAP or becoming non-compliant), oral appliances should be tried; and 3) UPPP should probably no longer be recommended in OSA. Maxillofacial surgery needs further evaluation in this specific condition, particularly regarding phase I.

Upper airway resistance syndrome is still a clinical situation that is difficult to identify and to treat adequately. Few data are available in the literature at the present time. Therefore, we can only suggest various means of treatment that should be prospectively evaluated in the future: 1) oral appliances may potentially reduce airway resistance and thus appear as a potential means of treatment of UARS; 2) maxillofacial surgery, although it could be considered unacceptable by some patients, represents a logical method of treatment; and 3) CPAP therapy can be effective in UARS. There is, however, little experience with CPAP therapy in this condition. Although compliance has been found low by some authors, others have obtained better results.

Conclusions

In the past decade, we have identified, with increasing frequency, patients with respiratory sleep disorders that differ from the classical description of severe OSA. Snoring and UARS have been identified as individual entities. However, the morbidity in these different clinical conditions remains largely unknown. Thus, it is difficult to recommend treatment. One may suggest that these conditions, as well as moderate OSA, are favorable to prospective studies using untreated groups of patients; in cases of severe OSA, however, this would be considered unethical.

REFERENCES

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230–5.
2. Hla KM, Young TB, Bidwell T, Palta M, Skatrud GB, Dempsey J. Sleep apnea and hypertension: a population-based study. *Ann Intern Med* 1994;120:382–8.
3. Sullivan CE, Berthon-Jones M, Issa FG, Eves L. Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nose. *Lancet* 1981;1:862–5.
4. Pépin JL, Leger P, Veale D, Langevin B, Robert D, Lévy P. Side effects of nasal continuous positive airway pressure in sleep apnea syndrome (study of 193 patients in two French sleep centers). *Chest* 1995;107:375–81.
5. Guilleminault C, Stoohs R, Clerk A, Cetel M, Maistros P. A cause of excessive daytime sleepiness: the upper airway resistance syndrome. *Chest* 1993;104:781–7.
6. Guilleminault C, Stoohs R, Kim Y, Chervin R, Black J, Clerk A. Upper airway sleep-disordered breathing in women. *Ann Intern Med* 1995;122:493–501.
7. Lugaresi E, Cirignotta F, Coccagna C, Piana C. Some epidemiological data on snoring and cardiocirculatory disturbances. *Sleep* 1980;3:221–4.
8. Strollo PJ, Sanders MH. Significance of treatment of nonapneic snoring. *Sleep* 1993;5:403–8.
9. Lugaresi E, Mondini S, Zucconi M, Montagna P, Cirignotta F. Staging of heavy snorers disease: a proposal. *Bull Eur Physiopathol Respir* 1983;19:590–4.
10. Mendelsen WB. Sleepiness and hypertension in obstructive sleep apnea. *Chest* 1992;101:903–9.
11. Rauscher H, Formanek D, Zwick H. Nasal continuous positive airway pressure for nonapneic snoring? *Chest* 1995;107:58–61.
12. Mosko SS, Dickel MJ, Ashurst J. Night-to-night variability in sleep apnea and sleep-related periodic leg movements in the elderly. *Sleep* 1988;11:340–8.
13. Strohl KP, Cherniack NS, Gothe B. Physiologic basis of therapy for sleep apnea. *Am Rev Respir Dis* 1986;134:791–802.
14. Davila DG, Hurt RD, Offord KP, Harris CD, Shepard JW. Acute effects of transdermal nicotine on sleep architecture, snoring, and sleep-disordered breathing in nonsmokers. *Am J Respir Crit Care Med* 1994;150:469–74.
15. Brownell LG, West P, Sweatman P, Acres JC, Kryger MH. Protriptyline in obstructive sleep apnea: a double-blind trial. *N Engl J Med* 1982;307:1037–42.
16. Smith PL, Haponik EF, Allen RP, Bleecker ER. The effects of protriptyline in sleep-disordered breathing. *Am Rev Respir Dis* 1982;127:8–13.
17. Hanzel DA, Proia NG, Hudgel DW. Response of obstructive

sleep apnea to fluoxetine and protriptyline. *Chest* 1991;100: 416–21.
18. Stewart DA, Grunstein RR, Berthon-Jones M, Handelsman DJ, Sullivan CE. Androgen blockade does not affect sleep-disordered breathing or chemosensitivity in men with obstructive sleep apnea. *Am Rev Respir Dis* 1992;146:1389–93.
19. Pickett CK, Regensteiner JG, Woodard WD, Hagerman DD, Weil JV, Moore LG. Progestin and estrogen reduce sleep-disordered breathing in postmenopausal women. *J Appl Physiol* 1989;66:1656–61.
20. Franklin K, Lundgren R, Rabben T. Sleep apnea syndrome treated with oestradiol and cyclic medroxyprogesterone. *Lancet* 1991;338:252–3.
21. Cistulli PA, Barnes DJ, Grunstein RR, Sullivan CE. Effect of short term hormone replacement in the treatment of obstructive sleep apnoea in postmenopausal women. *Thorax* 1994;49:699–702.
22. Guilleminault C. Clinical features and evaluation of obstructive sleep apnea. In: Kryger MH, Roth T, Dement WC, eds. *Principles and practice of sleep medicine.* Philadelphia: WB Saunders, 1994.
23. Bouchard C. Genetic aspects of human obesity. In: Björntorp P, Brodoff BN, eds. *Obesity.* Philadelphia: JP Lippincott, 1992: 343–51.
24. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy and the obstructive sleep apnoea syndrome. *Eur Respir J* 1990;3:509–14.
25. Suratt PM, McTier R, Findley LJ, Pohl SL, Wilhoit SC. Changes in breathing and the pharynx after weight loss in obstructive sleep apnea. *Chest* 1987;92:631–7.
26. Schwartz AR, Gold AR, Schubert N, et al. Effect of weight loss on upper airway collapsibility in obstructive sleep apnea. *Am Rev Respir Dis* 1991;144:494–8.
27. Harman AM, Wynne JW, Block AJ. The effect of weight loss on sleep-disordered breathing and oxygen desaturation in morbidly obese men. *Chest* 1982;82:291–4.
28. Peiser J, Lavie P, Ovnat A, Charuzi I. Sleep apnea syndrome in the morbidly obese as an indication for weight reduction surgery. *Ann Surg* 1984;199:112–5.
29. Smith PL, Gold AV, Meyrs DA, Haponik EF, Bleecker ER. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Ann Intern Med* 1985;103:850–5.
30. Pasquali R, Colella P, Cirignotta F, et al. Treatment of obese patients with obstructive sleep apnoea syndrome (OSAS): effect of weight loss and interference of otorhinolaryngoiatric pathology. *Int J Obesity* 1990;14:207–17.
31. Sugerman HJ, Fairman RP, Sood RK, Engle K, Wolfe L, Kellum JM. Long-term effects of gastric surgery for treating respiratory insufficiency of obesity. *Am J Clin Nutr* 1992;55:597s–601s.
32. Suratt PM, McTier RF, Findley LJ, Pohl SL, Wilhoit SC. Effect of very low calorie diets with weight loss on obstructive sleep apnea. *Am J Clin Nutr* 1992;56:182s–4s.
33. Braver HM, Block AJ, Perri MG. Treatment for snoring: combined weight loss, sleeping on side, and nasal spray. *Chest* 1995;107:1283–8.
34. Rennotte MT, Baele P, Aubert G, Rodenstein DO. Nasal continuous positive airway pressure in the perioperative management of patients with obstructive sleep apnea submitted to surgery. *Chest* 1995;107:367–74.
35. National Institutes of Health Consensus Group. Gastrointestinal surgery for severe obesity: NIH consensus. Development Conference Consensus Statement 1991. *Am J Clin Nutr* 1992;55: 615s–9s.
36. Cartwright R. Effect of sleep position on sleep apnea severity. *Sleep* 1984;7:110–4.
37. Cartwright R, Lloyd S, Lilie J, Kravitz H. Sleep position training as treatment for sleep apnea syndrome: a preliminary study. *Sleep* 1985;8:87–94.
38. McEvoy RD, Sharp DJ, Thornton AT. The effects of posture on obstructive sleep apnea. *Am Rev Respir Dis* 1986;133:662–6.
39. Phillips BA, Okeson J, Paesani D, Gilmore R. Effect of sleep position on sleep apnea and parafunctional activity. *Chest* 1986;90:424–9.
40. George CF, Millar TW, Kryger MH. Sleep apnea and body position during sleep. *Sleep* 1988;11:90–9.
41. Cartwright RD, Diaz F, Lloyd S. The effects of sleep posture and sleep stage on apnea frequency. *Sleep* 1991;14:351–3.
42. Lloyd S, Cartwright RD. Physiologic basis of therapy for sleep apnea. *Am Rev Respir Dis* 1987;136:525–6.
43. Cartwright RD, Ristanovic R, Diaz F, Caldarelli D, Alder G. A comparative study of treatments for positional sleep apnea. *Sleep* 1991;14:546–52.
44. Braver HM, Block AJ. Effect of nasal spray, positional therapy and the combination thereof in the asymptomatic snorer. *Sleep* 1994;17:516–21.
45. Cartwright RD, Samelson C. The effects of a nonsurgical treatment for obstructive sleep apnea: the tongue retaining device. *JAMA* 1982;248:705–9.
46. Lowe AA. Dental appliances for the treatment of snoring and/or obstructive sleep apnea. In: Kryger M, Roth T, Dement W, eds. *Principles and practice of sleep medicine,* 2nd edition. Philadelphia: WB Saunders, 1994:722–35.
47. Schmidt-Nowara WW, Mead TE, Hays MB. Treatment of snoring and obstructive sleep apnea with a dental orthosis. *Chest* 1991;99:1378–85.
48. Bonham PE, Currier GF, Orr WC, Othman J, Nanda RS. The effect of a modified functional appliance on obstructive sleep apnea. *Am J Orthod Dentofacial Orthop* 1988;94:384–92.
49. Johnson LM, Arnett GW, Tamborello JA, Binder A. Airway changes in relationship to mandibular posturing. *Otolaryngol Head Neck Surg* 1992;106:143–8.
50. Eveloff S, Rosenberg CL, Carlisle CC, Millman RP. Efficacy of a Herbst mandibular advancement device in obstructive sleep apnea. *Am J Respir Crit Care Med* 1994;149:905–9.
51. Schmidt-Nowara W, Lowe A, Wiegand L, Cartwright R, Perez-Guerra F, Menn S. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. *Sleep* 1995;18:501–10.
52. American Sleep Disorders Association. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. *Sleep* 1995;18:511–3.
53. O'Sullivan R, Hillman DR, Mateljan R, Pantin C, Finucane KE. Mandibular advancement splint: an appliance to treat snoring and obstructive sleep apnea. *Am J Respir Crit Care Med* 1995;151:194–8.
54. Ichioka M, Tojo N, Yoshizawa M, et al. A dental device for the treatment of obstructive sleep apnea: a preliminary study. *Otolaryngol Head Neck Surg* 1991;104:555–8.
55. Clark GT, Arand D, Chung E, Tong D. Effect of anterior mandibular positioning on obstructive sleep apnea. *Am Rev Respir Dis* 1993;147:624–9.
56. Rodenstein DO. Assessment of uvulopalatopharyngoplasty for the treatment of sleep apnea syndrome. *Sleep* 1992;15:S56–62.
57. Sher AE, Schechtman KB, Piccirillo JF. The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. *Sleep,* 1996 (in press).
58. Launois SH, Feroah TR, Campbell WN, et al. Site of pharyngeal narrowing predicts outcome of surgery for obstructive sleep apnea. *Am Rev Respir Dis* 1993;149:182–9.
59. Shepard JW, Thawley SE. Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. *Am Rev Respir Dis* 1990;141:1350–5.
60. Liistro G, Aubert G, Rodenstein DO. Management of sleep apnoea syndrome. *Eur Respir J* 1995;8:1751–5.
61. Walker EB, Frith RW, Harding DA, Cant BR. Uvulopalatopharyngoplasty in severe idiopathic obstructive sleep apnoea syndrome. *Thorax* 1989;44:205–8.
62. Miljeteig H, Mateika S, Haight JS, Cole P, Hoffstein V. Subjective and objective assessment of uvulopalatopharyngoplasty for treatment of snoring and obstructive sleep apnea syndrome. *Am J Respir Crit Care Med* 1994;150:1286–90.
63. Janson C, Hillerdal G, Larsson L, et al. Excessive daytime sleepiness and fatigue in nonapnoeic snorers: improvement after UPPP. *Eur Respir J* 1994;7:845–9.
64. De Backer W, Van de Heyning P. Is the role of UPPP in nonapnoeic snorers underestimated? *Eur Respir J* 1994;7:843–4.
65. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea

syndrome: a review of 306 consecutively treated surgical patients. *Otolaryngol Head Neck Surg* 1993;108:117–25.
66. Riley RW, Powell NB, Guilleminault C. Obstructive sleep apnea and the hyoid: a revised surgical procedure. *Otolaryngol Head Neck Surg* 1994;111:717–21.
67. Johnson NT, Chinn J. Uvuvlopalatopharyngoplasty and inferior sagittal mandibular osteotomy with genioglossus advancement for treatment of obstructive sleep apnea. *Chest* 1994;105:278–83.
68. Hochban W, Brandenburg U, Peter JH. Surgical treatment of obstructive sleep apnea by maxillomandibular advancement. *Sleep* 1994;17:624–9.
69. Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993;147:887–95.
70. Reeves-Hoche MK, Meck R, Zwillich CW. Nasal CPAP: an objective evaluation of patient compliance. *Am J Respir Crit Care Med* 1994;149:149–54.
71. Engleman HM, Martin SE, Douglas NJ. Compliance with CPAP therapy in patients with the sleep apnoea/hypopnea syndrome. *Thorax* 1994;49:263–6.
72. Waldhorn RE, Herrick TW, Nguyen MC, O'Donnell AE, Sodero J, Potolicchio SJ. Long-term compliance with nasal continuous positive airway pressure therapy of obstructive sleep apnea. *Chest* 1990;97:33–8.
73. Rolfe I, Olson LG, Saunders NA. Long-term acceptance of continuous positive airway pressure in obstructive sleep apnea. *Am Rev Respir Dis* 1991;144:1130–3.
74. Krieger J. Long-term compliance with nasal continuous positive airway pressure (CPAP) in obstructive sleep apnea patients and nonapneic snorers. *Sleep* 1992;15:s42–6.
75. Stokes Dickins QS, Jenkins NA, Mrad R, Schweitzer PK, Walsh JK. Nasal continuous positive airway pressure in the treatment of obstructive sleep apnea. *Op Tech Otolaryngol Head Neck Surg* 1991;2:91–5.
76. Hoffstein V, Viner S, Mateika S, Conway J. Treatment of obstructive sleep apnea with nasal continuous positive airway pressure: patient compliance, perception of benefits and side effects. *Am Rev Respir Dis* 1992;145:841–5.
77. Kloss W, Meier-Ewert K, Schäfer H. Zur Therapie des obstruktiven Schlaf-Apnoe-Syndroms. *Fortschr Neurol Psychiatr* 1986: 54:267–71.
78. Nakazawa Y, Sakamoto T, Yasutake R, et al. Treatment of sleep apnea with prosthetic mandibular advancement (PMA). *Sleep* 1992:15:499–504.
79. Lowe A, Fleetham J, Ryan F, Mathews B. Effects of a mandibular repositioning appliance used in the treatment of obstructive sleep apnea on tongue muscle activity. In: Issa FG, Suratt PM, Remmers JE, eds. *Sleep and respiration.* New York: Wiley-Liss, 1990:395–405.
80. Lyon HE, Phillips B, Theiss BL. Treatment of snoring and obstructive sleep apnea. *Compend Contin Educ Dent* 1990;XIII(5): 416–20.