Obstructive Sleep Apnea: Therapies Other Than CPAP

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Abstract
Nasal application of continuous positive airway pressure (CPAP) is the standard form of therapy for treating obstructive sleep apnea (OSA). Common difficulties associated with CPAP therapy include sense of dryness in the mouth, rhinorrhea, nasal congestion and dryness, mask discomfort, claustrophobia, irritation from device noise, aerophagy, chest discomfort and partner’s intolerance. Therefore, many patients are unable to or unwilling to comply with the use of CPAP. This article discusses the various non-CPAP approaches that have been investigated in the management of OSA, which include behavioral therapy (weight loss and positional therapy), pharmacological treatment, mandibular advancement techniques and surgery. However, none of these has been shown to be superior to CPAP. In clinical practice, only selected patients will benefit from therapies other than CPAP.

INTRODUCTION
Sleep apnea is characterized by temporary cessation of breathing during sleep. It may be either central or obstructive origin. Obstructive sleep apnea (OSA) is characterized by periodic collapse of the upper airway during sleep. The prevalence of OSA has been reported to be present in 2% in middle-aged women and 4% in middle-aged men. The long-term effects of OSA include hypertension, cardiovascular disease, stroke and higher mortality. Nasal application of continuous positive airway pressure (CPAP) was first reported to treat OSA in 1981. Since then, it has become the most effective and widely used treatment for OSA. CPAP acts as a pneumatic splint to force the upper airway open during sleep, and thus prevents OSA. The common difficulties associated with CPAP therapy include sense of dryness in the mouth, rhinorrhea, nasal congestion and dryness, mask discomfort, claustrophobia, irritation from device noise, aerophagy, chest discomfort and partner’s intolerance. Many patients are unable to or unwilling to comply with the use of CPAP. Up to 30% of patients with OSA reject CPAP treatment as a long-term treatment option. Among those who accept CPAP, compliance is highly variable and often thought to be suboptimal.

Therapies other than CPAP that have been used in the management of sleep apnea include,
1. Behavioral therapy (weight loss and positional therapy)
2. Pharmacological treatment
3. Mandibular advancement techniques
4. Surgery

BEHAVIORAL THERAPY
Weight loss
This includes weight loss and positional therapy. Obesity has been shown to be associated with OSA. An increase in the body mass index of one standard deviation was associated with a four-fold increase in the risk of having an apnea-hypopnea index (AHI) of more than five per hour. Obesity can reduce the size or change the shape of the upper airway, which may promote airway occlusion. MRI studies have shown an increased deposition of fat in the upper airway of individuals with OSA. The upper airway has been shown to be more collapsible in patients with OSA. Peppard et al., have shown in normal volunteers that for each percentage of change in weight there was approximately a 3% change in the AHI (a 10% reduction in weight was associated with a 26% reduction in the AHI). For subjects with a mild increase in the AHI (<15 per hour), a 10% increase in weight was associated with a six-fold increase in the chance of developing moderate to severe sleep disordered breathing (AHI > 15 per hour).

Several studies have examined the short term effect of weight loss in OSA and have recently been reviewed. Significant weight loss is associated with improvement in OSA and total resolution in some patients. However, the
amount of weight loss achieved does not always correlate with the extent of improvement in the AHI. Furthermore, in most such studies, the patients had an AHI of more than 30 per hour. In clinical practice, weight loss is often considered in patients with mild or moderate disease who are reluctant to try or are non-compliant with CPAP. To assess the long term efficacy of weight loss in the reduction of AHI, 216 obese patients with sleep apnea were enrolled for a weight reduction program. One hundred and one patients who lost 10% or more of their initial weight underwent a follow up sleep study, which revealed that 34 patients had an AHI < 10 per hour with resolution of symptoms. Of the 24 patients who were followed for 5 to 11 years, 11 patients regained 50% of their initial weight loss and OSA recurred in eight of these patients. Furthermore, OSA recurred in 7/13 patients who maintained their weight loss.

In summary, weight loss can reduce AHI in the short term with resolution of AHI and symptoms in some patients with OSA. Long term data regarding the effect of weight loss on AHI are limited. In the long term, OSA may recur due to increase in weight or even despite maintaining weight loss.

**Positional therapy**

The frequency of apnea and hypopneas is influenced by body position in up to 50% patients with OSA. The AHI is higher in the supine position and lower in the lateral position or with the head of the bed elevated to 30 to 60 degrees. The upper airway size has been shown to be larger in the seated position compared to the supine position. However, there is no increase in the upper airway size when patients with OSA move from the supine position to the lateral position. Upper airway collapsibility is also reduced in the seated position compared to the supine position. Some studies have shown a decrease in the upper airway collapsibility in the lateral position compared to the supine position. Methods for avoiding sleeping in the supine position include positional monitor that triggers an alarm, wearing a backpack with a soft ball inside, pinning a tennis ball to the patients pajama top or placing a wedge pillow lengthwise in the bed. Positioinal therapy using a backpack with a soft ball has been compared with CPAP in a randomized cross over study in patients with mild to moderate OSA (mean AHI 18 ± 5) and having an AHI in the supine position more with a soft ball has been compared with CPAP in a randomized

**Pharmacological Therapy**

**Tricyclic antidepressants**

Protriptyline is a non-sedating tricyclic antidepressant agent that was inadvertently found to reduce OSA in patients with narcolepsy. It’s mechanism of action is not clear. However, protriptyline is well known to reduce the rapid eye movement (REM) sleep. OSA is often worse in REM sleep and therefore by decreasing REM time, the apnea time and severity of oxygen desaturation may be improved. Disproportionate improvement of symptoms associated with day time somnolence after protriptyline administration in patients with OSA suggest that effects other than reduction of apneas may be of importance. Masked or coexisting depression may exist in patients with OSA, and it is possible that this part of protriptyline effect may be explained by it’s anti-depressive properties. Also, the therapeutic effect of tricyclic antidepressants on OSA is not restricted to protriptyline. Brownell et al, reported no significant change in the overall apnea index after administration of protriptyline for two weeks. The reduction of the apnea index only during REM was attributed to a reduction in REM sleep. However, in a double blind placebo-controlled study, protriptyline did not improve apneas, oxygenation or symptoms.

We currently feel that the benefits of protriptyline do not outweigh it’s side effects. Further well designed studies are required to identify the subgroup of patients who may be potentially be good responders. Patients with co-existing depression and female patients may be potentially preferable patients for this medication.

**Serotonergic agents**

Upper airway obstruction is associated with a decrease in the activity of upper airway dilator muscles such as the genioglossus. Maintenance or augmentation of activity in these muscles may prevent upper airway collapse. Serotonergic neurons exert an excitatory effect on upper airway dilator motor neurons. In an animal model of OSA, the systemic administration of serotonin antagonist led to reduction in upper airway cross-sectional area and oxygen desaturation and the administration of serotonergic agents (trazadone and L-tryptophan) was effective in treating sleep disordered breathing. In an uncontrolled study, L-tryptophan has been reported to decrease OSA in non-REM sleep. In a non-blinded uncontrolled study, fluoxetine, a selective serotonin reuptake inhibitor (SSRI), administered for 4 weeks (20 mg/day) reduced the AHI from 57 ± 9 per hour to 34 ± 6 per hour. This reduction in the AHI was observed only in non-REM sleep. In a double blind, randomized and placebo-controlled trial in patients with OSA without known psychiatric illness, paroxetine (20 mg/day), SSRI showed a significant but small reduction in the AHI (placebo 36.3 ± 24.7 per hour; treatment 30.2 ± 18.5 per hour). There was a
reduction in the obstructive episodes in non-REM sleep in the treatment group.

Serotonin seems to have a role in the maintenance of upper airway patency. Results from a double blind, randomized and placebo-controlled trial suggest that administration of SSRI lead to a reduction in the AHI in non-REM sleep. However, current data show that this reduction is not clinically significant.

**Progesterone**

Estrogen and progesterone replacement in post-menopausal women has been shown to increase upper airway muscle activity. However, there was no significant benefit in the AHI after hormone replacement therapy in post-menopausal women. Use of androgens or medroxyprogesterone has shown to be of no clinical benefit in male patients with OSA. However, medroxyprogesterone has been shown to have a beneficial effect in patients with obesity hypoventilation syndrome.

A direct ventilatory stimulant effect has been demonstrated in animal studies. The results of medroxyprogesterone in uncontrolled studies in patients with OSA and hypercapnia are variable. A randomized, double blind placebo-controlled study failed to show any effect of medroxyprogesterone on sleep disordered breathing. In patients with obesity hypoventilation syndrome, medroxy progesterone (20 mg 8 hourly) reduced the day time partial pressure of carbon dioxide tension by 13 ± 2.6 mm Hg (SEM) and increased the day time partial pressure of oxygen by 12.6 ± 2.7 mm Hg (SEM). Withdrawl of medroxyprogesterone led to deterioration of the blood gas values to pretreatment levels and reinstitution caused improvement.

Currently there are no data to recommend the use of progesterone in the management of OSA. The data showing benefit of medroxyprogesterone in the treatment of obesity hypoventilation syndrome is limited by the small numbers of patients and the lack of long term results. A double blind, randomized placebo-controlled trial with long term follow up would be of tremendous value in clarifying the efficacy and safety of medroxyprogesterone in obesity hypoventilation syndrome.

**Thyroid hormone replacement**

Hypothyroidism has been associated with sleep disordered breathing. Nine out of eleven consecutive patients with hypothyroidism were reported to have OSA. However, the prevalence of hypothyroidism was only 3% in 65 consecutive patients diagnosed to have OSA. In this study, older age and obesity were associated with OSA. Rajgopal et al reported an improvement in OSA following treatment of hypothyroidism with L-thyroxine. However, Grunstein et al did not report such a favourable response of thyroid hormone replacement on OSA in patients with hypothyroidism. The mechanism of OSA in patients with hypothyroidism include obesity, hypotonia of upper airway dilator muscles caused by myopathy, narrowing of the upper airway by deposition of mucopolysaccharides and protein extravasation into the oropharyngeal tissue and impaired ventilatory control.

CPAP therapy may be started in patients with severe hypothyroidism and severe OSA and in patients with an urgent need to treat OSA in combination with thyroid hormone replacement therapy. Whether CPAP should be continued long term should be determined once euthyroid status has been achieved. It would seem reasonable to screen patients for hypothyroidism if they have OSA and clinical features of hypothyroidism.

**Acetazolamide**

As acetazolamide produces a metabolic acidosis and stimulates ventilation, it was hypothesized that it would improve sleep disordered breathing. In a blinded placebo-controlled study, there was a physiologic but no clinical improvement (reduction in AHI from 50 per hour to 26 per hour) in ten patients with OSA. Acetazolamide has been shown to increased the hypercapnic drive and not the hypoxic drive with modest improvement in both NREM and REM sleep with symptomatic improvement. In another study, Sharp et al found no improvement in patients with OSA. Two studies, have shown improvement in central sleep apnea following administration of acetazolamide. However, there was no gain in the hypoxic or the ventilator responses produced by acetazolamide. However, the hypercapnic ventilatory response was moved to a lower CO2 level. At high altitude, Hackett et al, have demonstrated a decrease in periodic breathing and central apneas following administration of acetazolamide for one day. Less than 20 patients with OSA have been studied with acetazolamide. Decrease in AHI has not been shown to be associated with clinical improvement.

**Theophylline**

Inhibition of the ventilatory depressant effect of adenosine which is elevated in peripheral blood of patients with OSA. Other mechanisms by which theophylline may stimulate ventilation include increasing in metabolic rate, stimulating hypoxic and hypercapnic ventilatory drives and by improving respiratory muscle performance. The positive chronotropic action of theophylline may improve the stability of breathing by decreasing circulation time, in congestive heart failure. It has been shown to improve central sleep apnea but not OSA when compared with placebo, however, the sleep pattern was disturbed by the overnight intravenous administration of theophylline. In patients with left ventricular systolic dysfunction, congestive heart failure and periodic breathing with central apneas, theophylline decreased central apneas from a mean value of 26 to 6 per hour.

Theophylline has been shown to be useful in central apnea and periodic breathing and does not appear to be useful in patients with OSA.

**Anti-hypertensive agents**

Systemic hypertension is common in patients with sleep apnea. Cilazapril, an angiotensin converting enzyme inhibitor and metoprolol, a beta-blocker have been shown to reduce AHI by about 30%. Clonidine has been shown to reduce
AHI in REM sleep. Whether the reduction in sleep apnea is because of decrease in blood pressure or due to the direct effect of the drug is not clear. However, there are also data showing that anti-hypertensive treatment may lead to an increase in sleep apnea and hence the effect of anti-hypertensive treatment on sleep apnea is not clear.65

Atrial pacing

Atrial overdrive pacing has been reported to substantially reduce the number of episodes of central and obstructive apnea in patients with pacemakers that had previously been implanted for the treatment of the sick sinus syndrome or the bradycardia-tachycardia syndrome.66 However, further studies are needed to elucidate the mechanisms involved in achieving these reductions and to assess the precise role of cardiac pacing in preventing symptoms, disability, and death in the general population of patients with sleep apnea syndrome.66

Opioid antagonist and nicotine

These stimulate ventilation through generalized cortical stimulation. An increased opioid activity has been demonstrated in the cerebrospinal fluid of patients with OSA.67 Although doxapram infusion has been shown to reduce the duration of apnea and the severity of oxygen desaturation, it did not reduce the number of episodes of apnea or oxygen desaturation. Nicotine gum chewed before bedtime has been shown to reduce the number of apneic episodes in the first two hours of sleep.68 Transdermal nicotine patches did not influence the apnea episodes but was found to reduce the lowest oxygen saturation during sleep.69

Modafinil

The role of modafinil, a non-amphetamine wake promoting medication has been assessed in two randomized, double blind, placebo-controlled trials. In a study of 157 patients with OSA who were compliant for CPAP therapy, treatment with CPAP and modafinil (400 mg per day for 4 weeks) significantly improved both the subjective (Epworth sleepiness score) and objective (multiple sleep latency test) measures of day time sleepiness compared to CPAP and placebo.70 In another study, CPAP and modafinil improved alertness as assessed by the maintenance of wakefulness test after 2 weeks of therapy. However, there was no subjective or objective improvement in the day time sleepiness.71 Therefore, modafinil may be considered as adjunctive therapy in patients who are compliant with CPAP therapy but have persistent day time sleepiness. Although, modafinil has also been shown to improve objective measures of sleepiness in untreated patients with OSA, the concern is that it may not prevent the cardiovascular consequences of OSA as it does not eliminate upper airway obstruction. Due to lack of controlled trials with long term follow up, modafinil is currently not recommended in untreated patients with OSA.

Summary

Currently, there is no effective pharmacological treatment of OSA. Different drugs seem to affect different aspects of sleep apnea, such as central or obstructive or those occurring in REM or non-REM sleep. Stimulation of ventilation may be helpful in some patients with central apnea, periodic breathing or hypercapnia. Modafinil might be helpful as an adjunctive drug to CPAP in selected patients with OSA who continue to have day-time sleepiness despite use of CPAP.

ORAL APPLIANCE THERAPY

Oral devices may be helpful in the management of OSA by improving the upper airway patency by increasing the cross-sectional area or by decreasing the upper airway collapsibility by increasing the muscle tone. A tongue-retaining device allows the tongue to remain in a forward position between the anterior teeth by holding the tongue in an anterior bulb with negative pressure, during sleep. This pulls the tongue forward to enlarge the volume of the upper airway and reduce upper airway resistance. It also increases the genioglossus muscle activity in patients with OSA.72,73 Mandibular repositioning devices have been shown to increase the upper airway muscle tone in the genioglossus.74 Just the mere presence of an intraoral appliance has been shown not to have an impact on the AHI or oxygen desaturation.75 Thus, mandibular advancement is required for the appliance to improve OSA. The imaging modalities to assess the upper airway size include upright lateral cephalometry, CT scan, MRI or videodendoscopy.

Tongue repositioning device

It is a custom made soft acrylic appliance that covers the upper and lower teeth and has an anterior plastic bulb. It uses negative suction pressure to hold the tongue in a forward position within the bulb. In a study of 16 patients, 50% patients had a reduction in the AHI by 50% and in 69% patients the AHI was reduced to less than six per hour.76

MANDIBULAR ADVANCEMENT DEVICE VERSUS CPAP

Prospective cross-over studies

In a study of 23 male patients with OSA (mean pretreatment AHI 34 per hour), the mean decrease in the AHI with the device was 39% compared to 60% with CPAP.77 CPAP was more effective in improving the quality of sleep and the AHI. However, symptoms of excessive day-time sleepiness were equally improved in both groups. In two other studies, comparing a fixed position MAD and a partially adjustable MAD with CPAP, the mean reduction in AHI with the fixed MAD was 48% compared to 62% with CPAP and with the partially adjustable device was 55% compared to 70% with CPAP.78,79

Randomized cross-over studies

In a study of 20 patients with mild to moderate OSA (AHI 5 to 30), CPAP was more effective at improving snoring, AHI, and oxygenation. Although patients reported greater ease of use and higher compliance with the MAD, Only 30% of patients had an AHI of less than 10 per hour with the use of the MAD.80 In a study of 48 patients with OSA (11 to 43 per hour), CPAP was more effective than the oral device for
improving AHI and subjective ratings of day-time function, even in patients with milder forms of OSA (AHI 5 to 15 per hour). Therefore, many surgical techniques, simple and complex have been attempted with the aim of curing OSA (Table 1). However, the use of active appliance resulted in a significant decrease in the AHI from 30 to 14 per hour and increase in the minimum oxygen saturation from 87 to 91%. The control plate had no significant effect on the AHI or the minimum oxygen saturation. A randomized, controlled, crossover study comparing a MAD to a placebo to assess the effect of oral appliance therapy on symptoms of OSA, the treatment of upper airway obstruction in a patient with Pickwickian syndrome. Uvulopalatopharyngoplasty was first described by Fujita in 1964 and it was performed in greater numbers for the treatment of OSA in the 1980's. The surgical procedures that have evolved either aim at reducing or ablating the soft tissue in the upper airway or repositioning the soft tissue through skeletal alteration.

A major limitation in the interpretation of the data for the role of surgery in OSA is the application of different criteria to define surgical success by different authors. These include,

1. Post-operative reduction in RDI or AI by 50% from the preoperative values
2. Reduction in the preoperative RDI by at least 50% to a post-operative AHI of less than 20 or 10 per hour
3. Reduction in the preoperative RDI by at least 50% to a level of 10 apneas per hour
4. Postoperative RDI and AI are both decreased by 60% from preoperative values to post operative AI of less than 10 per hour
5. Postoperative RDI is less than 15 per hour and AI is less than five per hour

It is clear that the definition for surgical success should be clearly defined so that data reporting is uniform to allow proper interpretation. Ideally, preoperative and postoperative PSG should be performed and the assessment criteria should include RDI, sleep architecture, time spent below 90% SpO2 and symptoms.

In children having OSA, surgery to correct underlying anatomical abnormalities often results in an improvement in symptoms and in some cases, it is curative. In a study of 40 children who underwent adenoidectomy and/or tonsillectomy (aged 4 to 12 years), there was a highly significant improvement in polysomnographic scores following surgery in all patients. In a larger study in 134 children also undergoing adenoidectomy and/or tonsillectomy, there was a significant improvement in the AHI and the lowest oxygen saturation in 78% children. The unimproved children tended to have smaller tonsils, narrower epipharyngeal airspace and

**Table 1: Surgical procedures for OSA**

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<thead>
<tr>
<th>Procedures in which the soft tissue is either removed or ablated</th>
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<tr>
<td>1. UPPP (Uvulopalatopharyngoplasty)</td>
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<tr>
<td>2. Laser assisted uvulopalatoplasty (LAUP)</td>
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<tr>
<td>3. Uvulopalatopharyngo-glossectomy (UPPGP)</td>
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<td>4. Laser midline glossectomy</td>
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<td>5. Radiofrequency ablation of tongue base</td>
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<td>6. Reduction of tongue base with hyoepiglottoplasty</td>
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<table>
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<tr>
<th>Procedures in which soft tissue is repositioned through skeletal alteration</th>
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<tr>
<td>1. Mandibular advancement (MA)</td>
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<tr>
<td>2. Maxillomandibular advancement (MMA)</td>
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<tr>
<td>3. Transpalatal advancement pharyngoplasty</td>
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<tr>
<td>4. Genioglossal advancement</td>
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<td>5. Hyoid myotomy and suspension</td>
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</table>

**Surgery**

The sites of obstruction in OSA can be described as retropalatal or retrolingual. Therefore, from the surgical point of view the obstruction in pharynx may be classified either as only retropalatal, only retrolingual or both retropalatal and retrolingual. Compromise of the upper airway may be caused by soft tissue obstruction or by structural abnormalities in the upper airway. UPPP which was introduced in the early 1980’s had a high failure rate. It mainly addresses the soft tissue structures and influences only the retropalatal space. With time the narrowing of the retrolingual space has also been identified as a significant contributing factor to OSA. Therefore, many surgical techniques, simple and complex have been attempted with the aim of curing OSA (Table 1). However, the only technique that so far offers a 100% cure of OSA is tracheostomy, which bypasses the upper airway altogether.

The first tracheostomy was performed by Kuhlo for the
more poorly developed maxillary and mandibular protrusion than the improved children.\textsuperscript{99} Substantial tonsillar hypertrophy rarely causes OSA in adults. In the carefully selected patient, tonsillectomy may be considered for the treatment of OSA.\textsuperscript{91}

**Uvulopalatopharyngoplasty (UPPP)**

This enlarges the retropalatal airway by excision of the tonsils (if present), trimming and reorientation of the posterior and anterior tonsillar pillars, and excision of the uvula and posterior portion of the palate. A meta-analysis of 37 papers indicate a reduction in the postoperative AHI by 38%.\textsuperscript{90} The results are better in patients in whom the predominant site of obstruction is retropalatal. Complications include velopharyngeal insufficiency (2%), bleeding (1%), nasopharyngeal stenosis (1%) and death due to upper airway obstruction (0.2%). The true prevalence is difficult to estimate as many reports do not comment on the presence or the absence of the postoperative complications.\textsuperscript{92}

**Laser-assisted uvulopalatoplasty (LAUP)**

This enlarges the retropalatal airway by ablation of the uvula and posterior margin of the soft palate with carbon dioxide laser. LAUP is often performed under local anesthesia. The success rate reported with this procedure ranges from 0 to 87%. However, the definition of success rate also varied in these studies to include a postoperative reduction in the RDI or AI by 50% with or without reduction of the AHI to less than 20 per hour.\textsuperscript{93-98} LAUP by producing a raw surface, involves circumferential scarring, the severity of which depends on the velopharyngeal axial configuration; therefore it can result in a diminished velopharyngeal air space and decreased distensibility.\textsuperscript{99} In a study of 174 patients (100-UPPP and 74 LAUP), laser-assisted surgery of the palate was concluded to be significantly inferior to conventional UPPP from the an anatomical viewpoint.\textsuperscript{99}

**Mandibular advancement**

Sagittal mandibular osteotomies are performed to effect anterior mobilisation of the insertion of the tongue at the genioglossus tubercle and thus enlarge the retrolingual space. There must be a significant antecedent mandibular deficiency and dental malocclusion to permit the requisite degree of anterior movement of the mandible and the mandibular teeth.\textsuperscript{100,101} Although, the reports of this technique are few, they demonstrate the potential influence of mandibular deficiency as a cause of OSA and furthermore that mandibular advancement could treat OSA.

**Maxillomandibular advancement**

The degree of mandibular advancement performed to treat OSA without maxillary advancement would lead to prognathism and dental malocclusion. Performing maxillary advancement permits mandibular advancement in patients with OSA having maxillomandibular deficiency but not maxillomandibular disproportion. The maxilla and the mandible are both advanced by sagittal osteotomies, which enlarge the retrolingual as well as some retropalatal space. It also improves the tension and collapsibility of the suprahypoid and velopharyngeal musculature.\textsuperscript{102} Thirty-eight consecutive patients either with maxillary and/or mandibular deficiency or dolichofacial type in combination with narrow posterior airway were treated with 10 mm maxillomandibular advancement. A retromolar sagittal split osteotomy and Le Fort I osteotomy was performed. OSA considerably improved in all patients and there was no significant difference when compared to the results of nasal CPAP. The AHI was reduced to less than 10 per hour, oxygen saturation increased and the sleep quality was better.\textsuperscript{103}

Therefore, successful surgical outcomes of OSA are dependent on careful patient selection. A combination of treatment may have to be used to address the retropalatal and the retrolingual airway space. There is concern that LAUP may worsen OSA. Controlled trials are needed to find the value of the surgical approach in the management of OSA.

**SUMMARY**

Various therapeutic approaches have been attempted in the management of OSA. However, none of these has been shown to be superior to CPAP. In clinical practice, only selected patients will benefit from therapies other than CPAP. CPAP remains the standard option to treat OSA.

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Announcement

10th Biennial Congress of the Trans-Pacific Allergy and Immunology Society (TPAIS), 21st - 23rd November, Oberoi Towers, Mumbai, India to be held jointly with 9th Asian Research Symposium in Rhinology (ARSR), 19th-21st November, 2004
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