کارگاه‌های آموزشی مرکز اطلاعات علمی

مقاله نویسی علوم انسانی

اصول تنظیم قراردادها

آموزش مهارت های کاربردی در تدوین و چاپ مقاله
Obstructive Sleep Apnea

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Abstract:
Sleep apnea, and particularly obstructive sleep apnea, is a common disorder that is characterized by a repetitive, partial or complete cessation of airflow, associated with oxyhemoglobin desaturation and an increased effort to breathe. In recent years, orthodontists have been interested and involved in the management of this disorder since it has been shown that oral appliance therapy can be an effective treatment approach for obstructive sleep apnea (OSA). The purpose of this article is to present a review of OSA by describing this disorder, its diagnosis, currently available treatment modalities and, in particular, the role of oral appliance therapy in its treatment.

Key Words: Obstructive sleep apnea; Diagnosis; Treatment modalities; Oral appliances

INTRODUCTION
Obstructive sleep apnea is characterized by a repetitive cessation of airflow because of an upper airway obstruction, despite simultaneous respiratory efforts made during sleep. Upper-airway obstructions in OSA can be either partial or complete and often result in (possibly severe) oxygen desaturation. When a complete or partial airway obstruction is manifested by a complete cessation or substantial reduction (i.e., >50%) in oronasal airflow, of at least 10 seconds, the respiratory event is defined as apnea or hypopnea, respectively. Thus, this disorder is also referred to as obstructive sleep apnea-hypopnea syndrome (OSAHS) [1]. Among patients with OSAHS, however, the sites of obstruction and narrowing differ greatly. The retropalatal (posterior to the soft palate) and retroglossal (posterior to the base of tongue) regions are commonly affected sites, with multiple sites of obstruction and narrowing not being uncommon [2].

According to the recommendations of the American Academy of Sleep Medicine, OSAHS is defined by a combination of symptoms (such as excessive daytime sleepiness) and laboratory findings. Laboratory findings should demonstrate a Respiratory Disturbance Index (RDI) of five or more obstructed breathing events per hour of sleep. These events include any combination of apneas, hypopneas, and Respiratory Effort Related Arousals (RERAs). On the basis of the RDI, OSAHS may be classified as mild (RDI 5-15), moderate (RDI 15-30), or severe (RDI > 30). However, since the detection of RERAs requires more sensitive diagnostic monitoring techniques, the number of breathing events is usually quantified by the number of apneas and hypopneas per hour of sleep (i.e., Apnea-Hypopnea Index; AHI) [1].

In order to more precisely describe OSA, one should differentiate between three different
types of sleep apnea. First, obstructive sleep apnea, which is the result of an airway blockage, is usually due to the collapse of soft tissue at the rear of the throat during sleep. This is the most common type of apnea. Second, central sleep apnea is caused by a failure on the part of the brain to control breathing. In this case, there is no physical blockage but the result is still an inability to breathe. Third, mixed sleep apnea occurs because of a combination of a physical blockage and a stoppage in breathing brought on by a lack of brain signals [3].

History
From a historical point of view, in 1837, Charles Dickens undoubtedly described the first sleep apnea patient in "The Posthumous Papers of the Pickwick Club" [4] in which he wrote "... and on the box sat a fat and red-faced boy, in a state of somnolency" or "Joe - damn that boy, he's gone to sleep again." In 1889, Hill describes a child "who breathes through his mouth instead of his nose, snores, restless at night and suffers from headache at school." In 1918, Sir William Osler coined the term "Pickwickian" to refer to obese, hypersomnolent patients [5]. In 1956, Burwell, in describing several obese, hypersomnolent patients with respiratory and cardiac failure, employed the term "Pickwickian Syndrome." Finally, in 1978, Dr. John Remmers explained why the upper airway collapses during sleep, leading to obstructive sleep apnea. However, it was not until 1981 when Sullivan et al treated sleep apnea patients with continuous positive airway pressure (CPAP).

Prevalence and risk factors
OSA affects at least 4% of men and 2% of women and more than 10% of the population over the age of 65 [6]. However, it is estimated that, in the general population, approximately 80 to 90% of patients meeting the criteria of at least moderate OSAHS remain undiagnosed [1]. In addition, another study suggested that if an AHI of 5 were used as the upper limit of normal, then the incidence of sleep-disordered breathing would be 9% for women and 24% for men. If an AHI of 15 were defined as the upper limit, then the incidence of OSA would be 5 and 12% in women and men, respectively. This demonstrates that, when using the AHI alone in a middle-aged population, the prevalence of undiagnosed OSA is surprisingly high [7]. In the UK, sleep disorders affect about 770,000 people; OSA is one of the most common sleep disorders which occurs at a frequency similar to type 1 diabetes and twice that of severe asthma [3].

The increased risk of OSAHS in males has been attributed to gender differences in airway morphology for instance, fat distribution and craniofacial dimensions, and the protective effects of female hormones on upper-airway patency. This latter hypothesis is confirmed by the fact that the menopausal state entails a risk for developing OSAHS. Moreover, an increase in age can be considered as a contributing factor to OSAHS [8]. OSAHS also appears to be more common in several endocrine disorders, like hypothyroidism, acromegaly, Cushing Syndrome, and diabetes mellitus. Further risk factors for OSAHS include familial aggregation, an overweight condition and obesity, central body fat distribution, large neck girth, genetics, smoking, menopause, alcohol use before sleep, nighttime nasal congestion and craniofacial and upper airway abnormalities like retro- or micro-gnathia, macroglossia, adenotonsillar hypertrophy, long face and inferior positioning of the hyoid bone [1,2,8]. In addition, tendencies toward a reduced cranial base length and angle, a large ANB angle, a steep mandibular plane, elongated maxillary and mandibular teeth, narrowing of the upper airway, a long and large soft palate, and a large tongue have also been reported [2]. Besides gender and age, obesity is probably
the most important risk factor for OSAHS. It is hypothesized that obesity can influence breathing during sleep by inducing hypoxemia and altering the upper airway structure and function. Finally, several intoxications may cause predisposition to upper-airway obstruction during sleep, including the use of tobacco, alcohol, and respiratory depressant or sedative medication [1].

**Pathophysiology**

Normal sleep can be divided into rapid eye movement (REM) and non-rapid eye movement (NREM) stages. Following the onset of sleep in normal subjects, sleep descends from the NREM into the REM stage after approximately 90 minutes. As the night progresses, more time is spent in REM sleep and less in NREM. Although muscle tone is usually decreased in all states of sleep, it is essentially absent during REM sleep, except for extraocular muscle movements. This muscle relaxation does not cause significant reduction in respiratory flow volumes in normal subjects. However, oropharyngeal muscle flaccidity, in combination with anatomical and other predisposing factors, and a higher negative airway pressure during inhalation among patients with OSA lead to obstruction of the upper airway. This, in turn, causes progressively increasing efforts to inspire against a closed airway, associated with hypoxemia, progressive hypercapnia and acidemia. These disturbances in blood chemistry result in catecholamine production and a subsequent cardiopulmonary sequel. Respiratory efforts against a closed airway result in an arousal from sleep, the regaining of muscle tone and the reestablishment of the airway. This cycle of sleep–obstruction–arousal–sleep, is repeated throughout the night and causes alterations in the normal REM/NREM sleep cycle, often causing relative sleep deprivation that culminates in daytime sleepiness [5,9].

**Diagnosis**

OSA is diagnosed using a combination of a sleep history, supporting questionnaires, a clinical examination of the upper airway, and overnight sleep monitoring by polysomnography (PSG) [10]. An overnight PSG is the “gold standard” for making the diagnosis and it includes the following physiologic measures [7]:

1. Electroencephalogram (EEG) evaluates the various stages of sleep, confirms the presence of sleep during the study, and determines sleep latency (time it takes to fall asleep).
2. Electrooculogram (EOG) confirms REM sleep by detecting eye movement activity.
3. Electromyography (EMG), usually obtained from sub-mental musculature, evaluates muscle tone during sleep.
4. Electrocardiogram (EKG) provides important information about cardiac rhythm abnormalities during sleep.
5. Airflow measurement, either a nasal or an oral airflow detector, establishes and quantifies airflow during respiration.
6. Thoracic and abdominal respiratory effort detectors monitor, detect and quantify thoracic and abdominal respiratory movement, which should correlate with the airflow during the normal respiratory cycle.
7. Blood oxygen saturation detector, usually a pulse oximeter, monitors oxygen saturation during respiratory events.

Besides PSG, the Epworth sleepiness scale (ESS) questionnaire elicits the likelihood of falling asleep in eight different situations [11]. In this evaluation, a score of 10 or more is considered sleepy. A score of 18 or more is very sleepy. If you score 10 or more on this test, you should consider whether you are obtaining adequate sleep, need to improve your sleep hygiene and/or need to see a sleep specialist (Table I.).

Scales that objectify sleepiness are the Multiple Sleep Latency Test and the Maintenance of Wakefulness Test [1].
As a guideline, to make a diagnosis, an individual with an AHI or RDI of more than 5, in combination with daytime sleepiness and/or cardiovascular disease, is considered to have OSA and must seek treatment [11].

OSAHS should be discriminated from central sleep apnea syndrome, Cheyne-Stokes respiration and several other conditions characterized by excessive sleepiness, including narcolepsy, insufficient sleep, periodic leg movements, non-respiratory arousal disorders, and alcohol or drug abuse. Moreover, OSAHS should be distinguished from simple snoring, which is associated with a physiological number of airway obstructions and the absence of OSAHS-related symptoms [1].

**Signs and symptoms**

An individual affected by OSA might suffer from the following signs and symptoms; Excessive daytime sleepiness is one of the most common complaints. Another most common complaint is nightly snoring interrupted by pauses in breath. Other signs and symptoms can be listed as loss of energy, fatigue, restless sleep, high blood pressure, an overweight condition, personality change, depression, irritability, forgetfulness, sexual dysfunction, nocturia, and Trouble concentrating [3].

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Epworth Sleepiness Score</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Controls</td>
<td>5.9 (2.2)</td>
<td>2 - 10</td>
</tr>
<tr>
<td>Primary Snoring</td>
<td>6.5 (3.0)</td>
<td>0 - 11</td>
</tr>
<tr>
<td>OSA</td>
<td>11.7 (4.6)</td>
<td>4 - 23</td>
</tr>
<tr>
<td>Narcolepsy</td>
<td>17.5 (3.5)</td>
<td>13 - 23</td>
</tr>
<tr>
<td>Idiopathic Hypersomnia</td>
<td>17.9 (3.1)</td>
<td>12 - 24</td>
</tr>
<tr>
<td>Insomnia</td>
<td>2.2 (2.0)</td>
<td>0 - 6</td>
</tr>
<tr>
<td>PLMD</td>
<td>9.2 (4.0)</td>
<td>2 - 16</td>
</tr>
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</table>

**Sequels**

As mentioned before, OSA can cause daytime somnolence which in turn leads to problems with daytime functioning, such as motor vehicle crashes, psychosocial problems, decreased cognitive function, and a reduced quality of life. Furthermore, patients with OSA may also suffer from various systemic disorders, including hypertension, coronary artery disease, myocardial infarction, congestive heart failure, stroke, diabetes and the metabolic syndrome, and sudden death [9, 13-15].

These associations may be due in part to risk factors common to all these conditions; they may also reflect a role of OSA in the etiology of these conditions [9]. Whether or not OSA has a mechanistic role in the metabolic syndrome, it is clear that people with diabetes mellitus should be questioned about OSA symptoms. In a longitudinal population study, 31 persons with moderate or worse OSA had 3 times the adjusted odds of developing new hypertension compared with persons without OSA. In studies of patients with congestive heart failure, the prevalence of OSA has ranged from 11 to 37% [9].

Enhanced sensitivity to carbon dioxide may predispose some patients with heart failure to the development of central sleep apnea [16]. These findings raise the question of whether routine screening for OSA should be performed in patients with congestive heart failure. Obstructive sleep apnea is also highly prevalent in patients with stroke (43-72%) and transient ischemic attacks [9]. Patients with coronary heart disease and obstructive sleep apnea are endangered by apnea-associated ischemia and these ischemic episodes lead to activation of the CNS and additional fragmentation of sleep. Patients with nocturnal ischemia should be screened for underlying sleep apnea even if nitrate therapy fails [17]. People with obstructive sleep apnea have a peak in sudden death from cardiac
causes during sleeping hours, which contrast strikingly with the nadir of sudden death from cardiac causes during sleeping hours in people without obstructive sleep apnea and in the general population [15].

**Treatment modalities**

Generally, treatment options for OSA can be categorized into surgical interventions and non-surgical methods. Surgical techniques, usually use for treatment of OSA, are tracheotomy, uvulopalatopharyngoplasty (UPPP), genigolossus advancement with hyoid myotomy (GAHM), maxillo-mandibular advancement, laser assisted uvulopalatoplasty (LAUP), and radiofrequency volumetric tissue reduction techniques like somnoplasty.

Prior to the development of nasal continuous positive airway pressure, the primary modality for managing severely affected patients was formal tracheotomy. This technique provided an absolute solution to the problem and was the most predictable surgical intervention. Currently, this option is only used in the most extremely affected patients with severe daytime somnolence or cardiovascular sequels [7]. However, one study concluded that tracheotomy effectively treated patients with uncomplicated OSA, but was much less effective in treating patients with OSA and cardiopulmonary decompensation [18].

The introduction of uvulopalatopharyngoplasty (UPPP) by Fujita et al in 1981 represented the first specialized surgical procedure, other than tracheotomy, for the management of OSA (Fig. 1) [7]. The success rate of UPPP alone in all patients with OSA is in the range of only 40-50% [1,7,19]. UPPP is theoretically more successful if performed only on those patients with narrowing or collapse in the region of the oropharynx and velum [6]. Thus, the success rate is even lower (5%) when the level of collapse is in the retroglossal area [19]. The most common adjunctive procedure for obstruction in the hypopharynx and tongue base region is GAHM. This procedure expands the hypopharyngeal region at the tongue base by advancing the genial tubercle and hyoid bone [7]. Maxillomandibular advancement provides maximal enlargement of the retrolingual and some enlargement of the retropalatal airway by means of Le Fort I and a mandibular sagittal split advancement osteotomy. Successful management of OSAHS has been reported in approximately 60% of patients following UPPP and/or GAHM and in 90% of patients after maxillo-mandibular advancement [1].

Some studies also emphasized the significant impact that transverse expansion of the
maxillary and mandibular arches can have on patients with OSA [20-22].

The introduction of laser assisted uvulopalatoplasty (LAUP) was initially met with excitement and optimism in regards to its potential effectiveness on OSA. Unfortunately, while it has been shown that it is a worthwhile option for snoring, it only produces minimal results for mild OSA [7].

Radiofrequency ablation of the soft palate and tongue base has also been recently introduced as an alternative for OSA (Fig. 2). Preliminary reports using this technique have been encouraging [7].

Non-surgical methods for treating OSA are elimination of associated risk factors, pharmacologic agents, nasal continuous positive airway pressure (nCPAP), and oral appliances. Although elimination of risk factors usually requires additional treatment, they should always be considered due to their facilitating effect on OSA management. Successful control of obesity, improvement in sleep-awake patterns, avoidance of stimulants in the evening, changing the sleep position from supine to lateral or more upright in supine-dependent upper-airway obstruction and avoidance of alcohol, tobacco and respiratory depressants or sedative medication can help patients cope with OSA [1].

In selected patients, pharmacological agents such as tricyclic antidepressants, serotonergic agents, wake–promoting agents (modafinil) and steroids may be able to alleviate the symptoms of OSA.

Nasal continuous positive airway pressure was
first introduced in 1981 as a non-surgical treatment and, since then, has become widely used for this condition. Nasal CPAP delivers positive airway pressure through a tightly sealed nasal mask. A constant pneumatic pressure is maintained in the upper airway, prevents collapse during nocturnal respiration [7]. Currently, a variety of devices and nasal masks from different manufacturers are available (Fig. 3).

Although, poor compliance (less than 50%) is the major drawback of nCPAP, when implemented properly, it remains the treatment of choice for OSA [7]. Several side effects, including allergy to the face mask, air leaks, abrasions on the ridge of the nose, dry nose or mouth in the morning and nasal congestion, have been reported due to nCPAP [1]. Nevertheless, CPAP is considered a first-line treatment for symptomatic patients with sleep apnea-hypopnea syndrome (SAHS), although some patients with milder SAHS can be expected to transfer to mandibular repositioning splints due to CPAP intolerance. Future treatment recommendations may also vary with current rapid changes in both mandibular repositioning splints and CPAP technology [23].

From a historical perspective, in 1934, Pierre Robin was the first to use a monobloc functional appliance to treat airway obstruction in infants with micrognathia. His method was not well received until 1985, when Meier-Ewert and coworkers described an intraoral protraction device for the treatment of sleep apnea [24]. Currently, a variety of oral appliances are available on the market. Oral appliances (OAs) are categorized as palatal lifting devices, tongue posture trainers, labial shields, tongue repositioning devices, and mandibular repositioning appliances [1,25]

Palatal lifting devices are infrequently used having poor results and patient compliance. Tongue posture trainers have limited efficiency and are not routinely utilized. Labial shields are also not commonly prescribed and are unpopular.

A tongue repositioning device (TRD) is a custom-made soft acrylic appliance that covers the upper and lower teeth (Fig. 4). It has an anterior plastic bulb which exerts negative pressure to hold the tongue in a forward position inside the bulb. This device is used particularly in patients with large tongues or inadequate healthy teeth. It has been shown that TRDs can make a 50% reduction in AHI [24,25].

Although, there are many different types of mandibular repositioning appliances (MRA), the role of each one is to advance the mandible. In terms of construction design, mandibular advancement appliances (MAA) are predominantly derived from functional orthodontic devices [26]. MRAs can be either one piece (monobloc) or two pieces (bibloc), and are either custom-made or prefabricated (Fig. 5). Furthermore, they may be adjustable (titratable) or nonadjustable [1,24]. Some types of OAs, which are either available on the market or can be seen in the articles, are Herbst appliance, Twin Block appliance, PM positioner, Karwetzky activator, Klearway appliance, EMA, TAP, Somnofit, Somnoguard [24, 25,27-29].

MRA mechanisms of action are somewhat complicated. Forward displacement of the
mandible has two effects: firstly, it moves the base of the tongue anteriorly, which in turn increases the retroglossal airway space; secondly, anterior movement of the tongue not only decreases the gravitational effect on the soft palate, but also results in anterior movement of the soft palate via the palatoglossal arches leading to the reduction in the collapsibility of the velopharynx [25, 29, 30].

How to adjust the mandibular repositioning splint is a matter of controversy. Both horizontal and vertical adjustments are needed. Adjustable appliances must be titrated by the dentist in order to attain their optimum therapeutic efficacy of them. The desired patient response to an MRA should be evaluated by polysomnography. Nevertheless, different studies have suggested various guidelines for appliance adjustment [6, 12, 25, 28-34]. For instance; the amount of mandibular advancement may be 3-5 mm, ⅓, ⅔ or ¾ of the maximum protrusive position from maximum intercuspation or ½ to ¾ of the width of a premolar. It has been shown that the amount of the vertical opening has no impact on the efficacy of the MRA, but that this may cause side effects. Thus, according to a group of researchers, the vertical opening should be kept as small as possible. If it is too large, a decrease in posterior airway space, instead of an increase, may occur. Other studies propose an 8-12 mm vertical opening or a 3-5 mm distance between incisors. It is also worth mentioning that there is a significant correlation between supine oropharyngeal size and the amount of the horizontal component of mandibular position change. This suggests that a certain amount of horizontal, rather than vertical, mandibular repositioning is necessary to obtain oropharyngeal enlargement in order to reposition the tongue forward with the genioglossus muscle, the hyoid bone (geniohyoid), and the anterior and posterior wall of the oropharyngeal and hypopharyngeal area (pharyngeal constrictor) [6].

In one study, it was indicated that, despite continued therapy, the efficacy of oral appliances decreases over time [28]. Several factors could be responsible for the long term decrease in efficacy; increase in patient’s age, increase in the body mass index (BMI), alcohol consumption, change in the patient’s preferred sleeping position (supine position), adaptation of the soft tissues to mandibular advancement and loss of the initial tightness, and return of increased muscle tone of the suprathyroid musculature to normal over time can be considered as these factors. Various researchers have suggested different criteria for the successful OA therapy of OSA, including more than a 50% decrease in apnea index (AI) with a posttreatment AI of less than 10 per hour, a reduction in AHI to less than 20 per hour, a reduction in AHI by more than 50 with posttreatment AHI of less than 20 per

Fig. 5: A custom-made monobloc (A) and a prefabricated bibloc (B).
hour and, finally, a reduction in AHI to less than 10 or 15 per hour with relief of symptoms [29]. Many studies have evaluated variables that may be associated with treatment outcome [25,29]. Accordingly, the predictors of oral appliance efficacy are summarized as follows:

- Younger age
- Lower BMI
- Lower neck circumference
- Lower AHI
- Increased protrusion of the appliance
- Smaller and/or narrower oropharynx
- Smaller overjet
- Less erupted maxillary molars
- Normal mandible length
- Shorter mandibular plane to hyoid distance
- Shorter soft palate length
- Smaller upper to lower facial ratios
- Normal or reduced lower facial height
- Small soft palate and tongue
- Increased retropalatal airway space
- Larger angle of the cranial base to the mandibular plane
- Forward maxilla

According to a recent study on responders and non-responders to oral appliance therapy, the middle and inferior airway space and oropharyngeal airway cross-sectional area were significantly larger in non-responders. The position of the mandible relative to the cervical spine was the only significant skeletal variable and was larger in non-responders. Changes in BMI between the two groups were statistically significant; the reported averages were a 2.9% increase in non-responders and a 0.5% decrease in responders [35].

Oral appliances are generally indicated as first-line therapy in patients with simple snoring and mild to moderate (5 < AHI < 30) OSA and as second-line therapy for patients with moderate to severe OSA when other therapies have failed [25]. Of course, similar to other treatment options, some contraindications like an inadequate number of healthy teeth for good retention, severe TMJ problems, inadequate protrusive ability, advanced periodontal disease, and severe dental and skeletal side effects are also present [1,18].

The numerous advantages of MRAs have been addressed in literature [36,37]. Oral appliances are simpler to use, more tolerable for patients, and portable when compared to CPAP devices. In addition, this treatment is completely non-invasive and even reversible, with a low complication rate in comparison to surgical procedures. Furthermore, oral appliance therapy is a cost-effective treatment. Despite the above mentioned advantages, side effects of oral appliance therapy are very common. Fortunately, they are usually minor and generally no serious complications have been observed [1,25,37]. Nevertheless, these side effects include oral discomfort, TMJ pain, muscle stiffness, excessive salivation or mucosal dryness, a temporary bite change in the morning, and minor dental and skeletal changes related to mandibular advancement by Cl 2 functional appliances (e.g. a decrease in the overjet and overbite, retroclination of the upper front teeth and proclination of the lower anterior teeth). After a mean (SD) of 7.3(2.1) years of OA use, one study, using lateral cephalometric analysis, showed significant ($P<0.01$) changes in many variables, including increases in the mandibular plane and ANB angles, decreases in overbite and overjet, retroclined maxillary incisors, proclined mandibular incisors, increased lower facial height, and distally tipped maxillary molars with mesially tipped and erupted mandibular molars [38]. Using study-model analysis, the same researchers reported significant occlusal changes in 85.7% of the patients in their study. Interestingly, for almost half of these patients, tooth movement was considered favorable. The group with unfavorable changes had significantly smaller overbite and overjet in pretreatment measurements and un-favorable changes were more likely in the Class I craniofacial subgroup [39]. Occasionally, an
oral appliance can worsen apnea severity. The reason for the increase in AHI with the appliance cannot be determined from a review of the patient data [25,40,41].

Patient-reported compliance with MRA therapy is generally high, with studies reporting regular use in 75 to 100% of patients, although long-term compliance has been reported to decrease over time [1]. In a 5.5-year follow-up study on 450 patients treated with mandibular advancement devices (MADs), about half of the patients were still using their MADs after 5 years [42]. Discontinuation of MRA treatment is generally related to its side effects, complications, or the lack of perceived benefits.

Treatment choices for OSA have been compared in a number of studies. Regarding nasal continuous positive airway pressure (nCPAP), as opposed to mandibular repositioning devices (MRDs), a variety of studies concluded that mandibular advancement devices are better tolerated in mild to moderate sleep apnea and are preferred to nasal CPAP [23,25,36].

Moreover, a simple non-adjustable oral appliance is no substitute for nasal positive airway pressure in patients with obstructive sleep apnea. In addition, CPAP is more effective in treating snoring and improving AHI and oxygenation. However, MRDs have lower side effects and are preferred by patients. Nevertheless, CPAP is the preferred first–line therapy for OSA patients who have significant functional impairment and even for patients with mild OSA (AHI= 5-15 per hour). In another study, the writers compared the Herbst appliance with Twin Block (Fig.6) [27]. They concluded that both are effective in treating OSA in respect to AHI, snoring and arterial blood oxygen saturation. However, while Herbst is more effective in ameliorating daytime sleepiness, both equally improve the quality of life. In addition, the side effects of both appliances are minor and subside with time. However, the cost of Twin Block is less than that of Herbst. Other experts also compared the Herbst appliance with Monobloc and reached the conclusion that AHI decreases to less than 10 in 75% of patients with Monobloc and in 67% of the patients with the Herbst appliance [25]. Furthermore, both reduce sleepiness and snoring.

On the other hand, patients feel that Monobloc is more effective in reducing symptoms and prefer it for long–term therapy.

CONCLUSION
Obstructive sleep apnea can best be treated via a team approach in which orthodontists and physicians (possibly otorhinolaryngologists) are involved. The physician’s role is to make the diagnosis, determine whether the patient is "medically" suitable for oral appliance therapy and follow up the patient by means of polysomnography. The orthodontist’s responsibility is to evaluate the patient’s "dental" suitability for oral appliance treatment and conduct a long-term dental follow-up which includes optimizing the appliance, monitoring
its retention and assessing its effectiveness.

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کارگاه‌های آموزشی مرکز اطلاعات علمی

مقاله نویسی علوم انسانی

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