Content available at: https://www.ipinnovative.com/open-access-journals

IP Indian Journal of Orthodontics and Dentofacial Research

Journal homepage: https://www.ijodr.com/

Review Article

Orthodontic perspectives of obstructive sleep apnea

Nazia Beg[1]*, Astitav Mittal[2], Mohit Makkar[1]

[1]*Dept. of Orthodontics and Dentofacial Orthopaedics, Swami Devi Dyal Hospital and Dental College, Panchkula, Haryana, India*
[2]*Maulana Azad Medical College (MAMC), Delhi, India*

ARTICLE INFO

Article history:
Received 08-02-2023
Accepted 15-07-2024
Available online 02-09-2024

Keywords:
Obstructive sleep apnea
Orthodontics
Sleep disorder
Mouth breathing
Crossbite
Growth

ABSTRACT

OSA is defined as a condition in which there is repetitive partial or complete collapse of the pharynx during sleep. An apnea is defined as an almost complete (at least 90%) cessation of airflow, and hypopnea is defined as a reduction in nasal pressure amplitude of at least 50% and/ or a reduction in thoracoabdominal movement of 50% or more for a minimum of 10 seconds. Often, patients first seek an otorhinolaryngologic (ENT) evaluation rather than presenting to a sleep center. Normal sleep involves air passing through and going directly down to the lungs. With an obstructed airway, the structures in the back of the throat (the tongue, the tonsils, and/or adenoids) occlude the airway due to an inadequate motor tone of the tongue and/or airway dilator muscles, and thus, prevent the air from passing. The aetiology is multifactorial, and the main risk factors in children include adenotonsillar hypertrophy, obesity, neuromuscular disorders and craniofacial anomalies. Adenotonsillar hypertrophy is the most commonly reported aetiological factor. Therefore, the treatment of choice for paediatric OSA is adenotonsillectomy. In adulthood, the use of oral appliances is a treatment option in the management of OSA

1. Introduction

The term "obstructed sleep apnea" (OSA) refers to a disorder when the pharynx repeatedly collapses during sleep, either partially or completely. Obstructive sleep apnea (OSA) is a term used to describe OSA that causes excessive daytime sleepiness.[1] A hypopnea is defined as a decrease in nasal pressure amplitude of at least 50% and/or a decrease in thoracoabdominal movement of 50% or more for a minimum of 10 seconds. An apnea is defined as an almost complete (at least 90%) cessation of airflow. Christan Guilleminault et al. first developed the term "obstructed sleep apnea syndrome" (OSAS) in 1976. They classified it as the presence of daytime sleepiness and at least 30 obstructive apneas every night, each of which lasted at least 10 seconds.[2] Instead of using apneas each night, the apnea index (AI), which measures apneas per hour of sleep, was quickly adopted. It is grouped into the following categories by the American Academy of Sleep Medicine (AASM):

Mild (5 events per hour)

1. Moderate (15-30 events per hour)
2. Severe (> 30 events per hour)

Some studies used the oxygen desaturation index (ODI),[3] which was defined as the number of desaturations per hour of sleep and is categorized into :

1. Mild OSA (5-14 desaturation per hour of sleep)
2. Moderate OSA (15-29 desaturations per hour of sleep)
3. Severe OSA (>30 desaturations per hour of sleep

* *Corresponding author.*
E-mail address: naziabeg3005@gmail.com (N. Beg).

https://doi.org/10.18231/j.ijodr.2024.031

1.1. Risk factors for OSA

1. Excess weight and measures of obesity
2. Gender distribution
3. Age
4. Ethnicity
5. Craniofacial morphology
6. Familial / genetic factors

1.2. Diagnosis

1. Medical History
2. Physical examination
3. Visualization of the upper airway
4. The Mueller manoeuvre
5. Lateral roentgenography
6. Polysomnography
7. Portable devices
8. The multiple sleep latency test (MSLT)

1.3. Clinical features

The symptoms of OSA are as follows:

1. Loud irregular snoring
2. Snorts, gasps, and other unusual breathing sounds during sleep
3. Long pauses in breathing during sleep
4. Excessive daytime sleepiness
5. Fatique
6. Obesity
7. Changes in cognitive functions such as alertness, memory, personality, or behavior.
8. Impotence
9. Morning headaches

Following are the clinical conditions predisposed by OSA

1. Mouth breathing
2. Posterior crossbite
3. Decreased overbite
4. Class I Malocclusion

1.4. Mouth breathing

It has been suggested that mouth breathing in children causes adenotonsillar hypertrophy and dental malocclusions. The most frequent craniofacial defects linked to OSA in children have been identified by various cephalometric investigations, although there is little information available on the prevalence of malocclusion in paediatric OSA patients. Additionally, it has been found that people who have craniofacial abnormalities are more likely to develop OSA. In this way, pediatric orthodontic examinations and treatment may prevent the emergence of OSA in adulthood.(Figure 1).

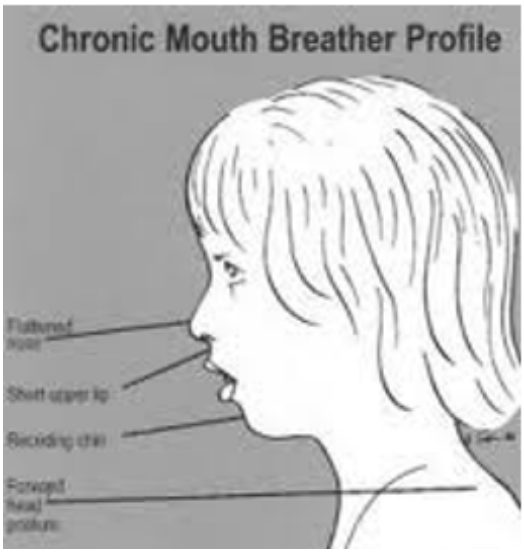

Figure 1: Mouth breathing

1.5. Posterior crossbite

The presence of a posterior crossbite is related to the altered equilibrium between the tongue and cheeks and is related to mouth breathing's low and anterior position of the tongue, which results in a lack of internal pressure and causes a reduction in the upper arch's transversal growth with the development of lateral and posterior teeth. On the other hand, mouth breathing is associated with decreased nose prominence and width dimensions compared to normal children. These facial characteristics could lead to a reduced upper airway space resulting in obstructive apnoea events (Figure 2).

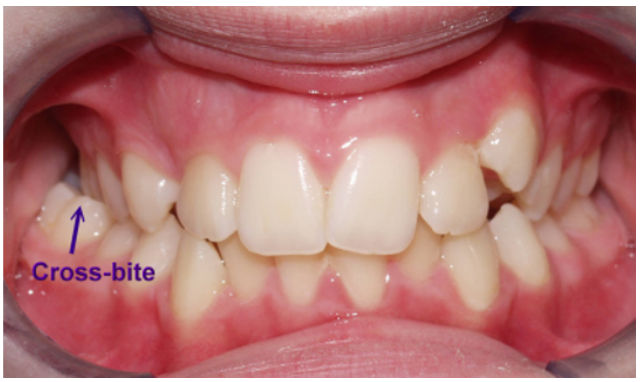

Figure 2: Posterior crossbite

Narrow maxillary dentition as a predisposing factor for OSA because a narrow upper dental arch is also thought to diminish the oropharyngeal volume available for the tongue. Reduced overbite, a shorter upper dental arch and shorter lower dental arch, crowding in the mandibular arch, and Class II malocclusions have all been linked to OSA patients

from an orthodontic standpoint. Small upper dental arches are hypothesized to reduce the amount of oropharyngeal space available for the tongue, making small maxillary dentition a risk factor for OSA. From an orthodontic perspective, crowding in the mandibular arch, Class II malocclusions, a reduced overbite, a shorter upper dental arch, and a shorter lower dental arch have all been linked to OSA patients. Lateral crossbite due to maxillary skeletal constriction were found to be approximately 30–50% in OSA patients compared to 10 per cent which was found in the general population.

1.6. Decreased overbite

Decreased over bite (Figure 3) and an increased overjet is significantly associated with OSA. Both reduced overbite and increased overjet are associated with the vertically oriented craniofacial growth pattern that is a typical sign of OSA in children. Overjet was also discovered to be related to the severity of OSAS in individuals who were not obese, which raised the possibility that malocclusion may be a significant factor in the onset of sleep apnea and hypopnea.

In addition to the above mentioned dental features, the associations of malocclusion classes and obstruction of the upper airways have been suggested. In patients with Class II malocclusions, mouth breathing and nasopharyngeal airway blockage are more common. The Balters' viewpoint holds that Class II malocclusions are caused by the tongue being held back, which disturbs the cervical region. The respiratory function is impeded in the region of the larynx and there is thus a faulty deglutition and mouth breathing. Additionally, it was discovered that Class II malocclusion was the most typical finding in OSA. People with skeletal class II and a more retrognathic mandible who did not respond well to the operation were this way. Findings of Class II malocclusion in OSA appear consistent enough to give strong consideration in the aetiology/pathology of this disorder.

Figure 3: Decreased overbite

1.7. Class I or class II malocclusion

The most frequent snoring-related results were class I malocclusions. Although there are many factors that contribute to the onset and causes of OSA, having a Class I or II malocclusion appears to be linked to a higher occurrence of the condition. Additionally, it has been noted that patients with Class I and Class II malocclusions have wider upper pharyngeal openings. (Figure 4). Examination of Aetiology of OSA reported a familial tendency of narrow, high palates in the relatives of OSA patients. A statistically significant difference was found in palatal heights between OSA and control patients at the level of the first premolar, second premolar, and molar. A palatal morphology is another dental characteristic that OSA patients regularly reported having. A predictive morphometric model for OSA that incorporates the V palatal shape also shows the potential utility of physical and dental examinations. Snoring has also been linked to palatal morphology with a V shape, a shortening of the top arch, and a closer spacing between the upper premolars.

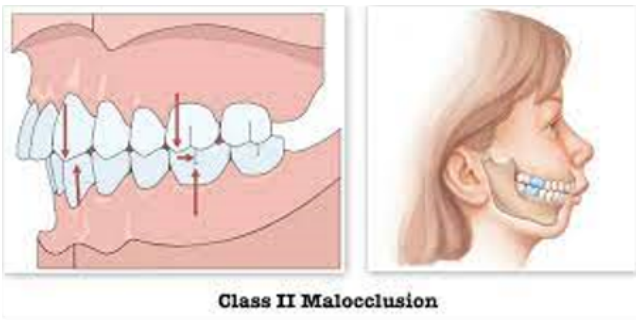

Figure 4: Class II malocclusion

In addition, when compared to controls, patients with OSA exhibited considerably smaller maxillary and mandibular arch widths and longer maxillary and mandibular arches. A narrow arch may encroach into the nasal cavity area because the roof of the mouth doubles as the nose's floor. In addition to raising nasal resistance, maxillary constriction can cause the upper oral cavity to narrow laterally and cause a low-positioned tongue, both of which limit the retroglossal region. This offers a plausible explanation for the posterior airway space constriction observed in OSA patients. Nevertheless, palatal height measurement alone was not a reliable indicator of maxillary constriction. Thus, it remains very questionable as to whether maxillary and or mandibular constriction can be a primary etiological factor in OSA.

Cephalometry antero posteriorly, both the face and anterior cranial base tend to be retruded and the cranial base angle is reduced in OSA patients, which leads to a reduction in the space available for the airway. In addition, mandibular retrusion may also occur. In the vertical plane,

increases in lower face height and maxillomandibular planes angle have been reported. Other Cephalometric features that associated with OSA are smaller than normal SNA and/or SNB; increased Frankfort mandibular plan angle and/or maxillary mandibular plane angle.

Furthermore, it has been demonstrated that orthodontic treatment options, like rapid maxillary expansion (RME) (Figure 5) and mandibular advancement, may reduce OSA symptoms in children.

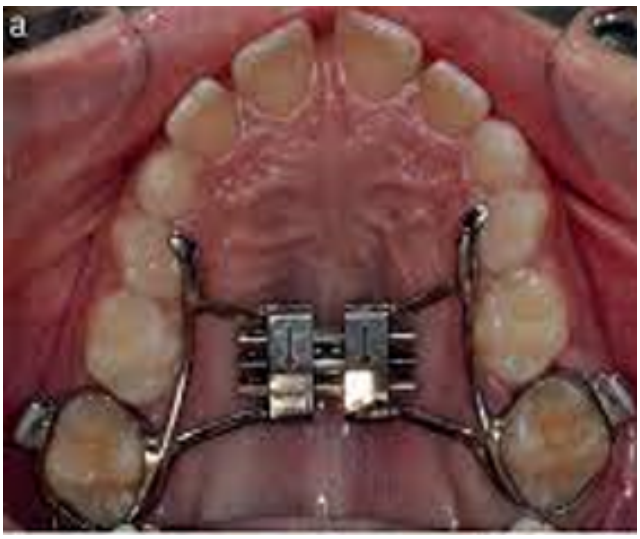

Figure 5: Rapid maxillary expansion

2. Treatment

Treatment options can be broadly divided into:[1]

1. Behavioral interventions
2. Non-surgical options
3. Surgical options

1. Behavioural interventions
   (a) Weight loss
   (b) Sleep positions
   (c) Avoidance of alcohol and smoking
   (d) Lifestyle changes
2. Non-surgical interventions
   (a) Continuous positive airway pressure
   (b) Oral appliance therapy

2.1. Continuous positive airway pressure (CPAP)

The treatment for moderate to severe OSA cases is CPAP. A new equipment called a continuous positive airway pressure machine has a mask that the patient wears over their nose. It maintains the neck open all night long and delivers a constant flow of air.[4](Figure 6)

Figure 6: Continuous positive airway pressure.

2.2. Oral appliance therapy

Orthodontic appliances should be fabricated in a way that it can be worn by the patient either in a permanent or removable manner depending upon the condition of the patient.[5] They can be favored by patients as an alternative and are preferable for non-apneic snorers who have failed conservative lifestyle (weight loss) adjustments as well as apnoeic patients who are unable to tolerate CPAP.[6] Nearly 100 different oral appliances are currently available which are divided into 3 main groups.

1. MAD's (Mandibular advancement devices)
2. TRD (Tongue retaining devices)
3. iSoft palate lifting devices

2.2.1. Tongue retaining devices

The tongue retaining device is a monobloc oral appliance that is specifically made to hold the tongue in an anterior bulb with negative pressure as you sleep, allowing it to stay forward between the anterior teeth. (Figure 7a,b).[7] These devices moderately extend the mandible by holding the tongue forward in the bulb of the device. Examples of TRD's in the market include Aveo TSD, SnorX, TRD, Deepsleep Pro,[7] etc.

Indications:

1. Lack of tooth support or edentulous arches
2. Non-apnoeic snorers
3. Mild snoring
4. Down syndrome

Adverse effects:

1. Excessive salivation
2. Difficulty in swallowing

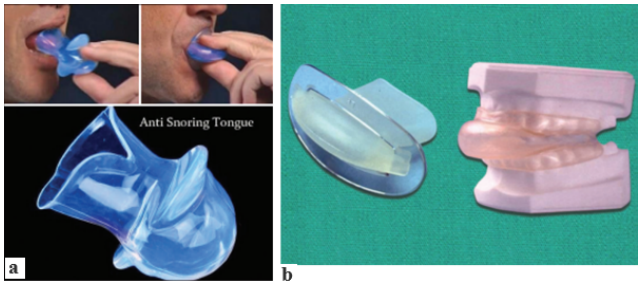

Figure 7: Tongue retaining devices.

2.2.2. Mandibular advancement appliances (MAD)

Mandibular advancement appliances change the position of the jaw and tongue by projecting the mandible forward to prevent upper airway collapse. (Figure 3) MAD's represent the main non-continuous positive airway pressure (non-CPAP) alternative for patients for OSA.[8] It aim to increase the upper airway size and reduce the risk of sleep apnoea and snoring in patients with OSA. These appliances are designed to protrude outward from the maxillary and mandibular teeth where they are attached. The designs of the device is personalized according to the dentition, materials used for fabrication, occlusal coverage, one plate (monobloc) or two separate plates device, allowable vertical mouth opening, the advancement approach and its adjustability to the advancement level (titration).[9] There are also over-the-counter Boil-and-Bite MADs that are made of flexible materials that become malleable when exposed to hot water. Fitting the gadget requires biting down over a flexible mold. However, MADs are not suitable in people wearing dentures or with severe dental issues.[10]

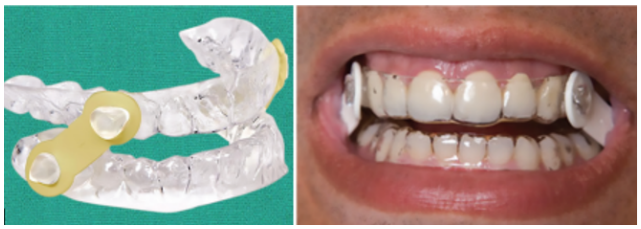

Figure 8: Mandibular advancement appliance

2.2.3. Soft palate lifting devices:[11]

The soft palate tissue is gently elevated by these Lifter devices, which have an adjustable acrylic button that extends distally to the midline of the soft palate. This stops the tissue from vibrating as you sleep. The majority of patients have trouble tolerating this device for long periods of time, however it could be helpful as a diagnostic tool if you think the obstruction of the airway is due to an excessive palatal drape.[12] Thus, the Palatal lifter significantly improves the upper airway passage dimensions, helping to terminate snoring and airway obstruction**.**

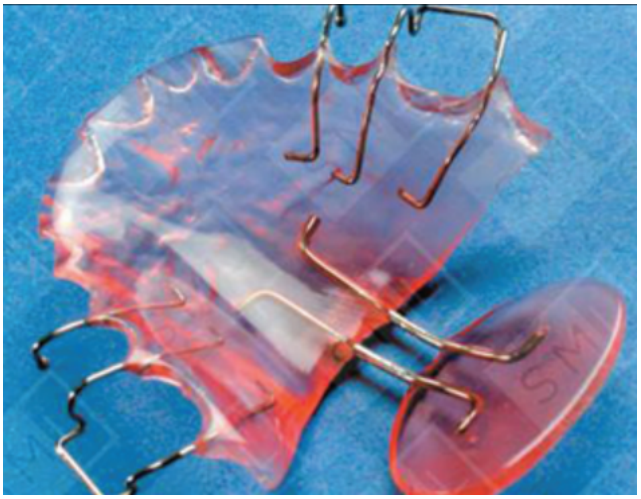

Figure 9: Soft palate lifting devices

2.3. Mechanism of action of oral appliances

Oral appliances are used only during sleep which move the lower jaw, tongue, soft palate, or uvula while preserving an open, unhindered airway.[13] It causes the tongue and jaw to push forward, preventing upper airway collapse while you sleep. (Figure 10). Mandibular advancement devices carry out an anterior and inferior movement of the jaw, generating anatomical variations in the Upper airway that enable an increase in the pharyngeal area.[14] Functional mandibular advancement causes the hyoid bone's position to shift in favor of a position that is more forward. It shifts the suprahyoid musculature into a new position of equilibrium, which favors an increase in the volume and permeability of the upper airway.[15]

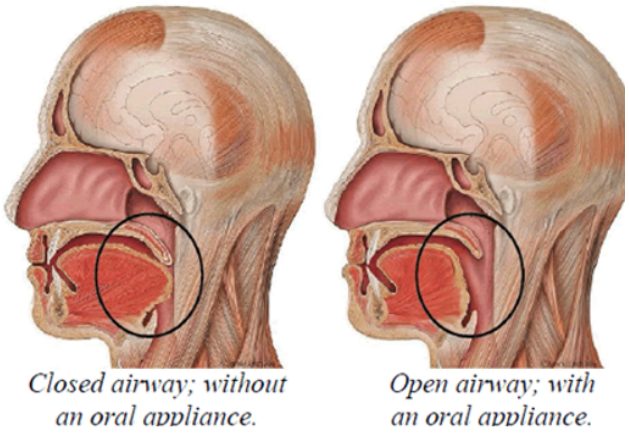

Figure 10: Mechanism of action–oral appliance therapy

Clinically, obstructive sleep apnea (OSA) is defined by the occurrence of daytime sleepiness, loud snoring, witnessed breathing interruptions, or awakening due to

gasping or choking in the presence of at least 5 obstructive respiratory events per hour of sleep.

The symptoms of OSA include snoring, disturbed sleep and neuro behavioural problems. The prevalence of OSA in children is 1% - 4% and, if left untreated, it may result in severe complications which include neurocognitive impairment, behavioural problems, failure to thrive and cor pulmonale. The aetiology is multifactorial, and the main risk factors in children include adenotonsillar hypertrophy, obesity, neuromuscular disorders and craniofacial anomalies. Adenotonsillar hypertrophy is the most commonly reported aetiological factor. therefore, the treatment of choice for paediatric OSA is adenotonsillectomy. In adulthood, the use of oral appliances is a treatment option in the management of OSA.[16] Studies in adult populations have found that a MAD has a diminished or similar effect on OSA signs and symptoms compared to CPAP but with improved tolerance and compliance to the device.[17,18] MAD's have therefore been suggested for adult patients with mild to moderate OSA or for those who cannot tolerate CPAP.[19,20]

3. Conclusion

All orthodontist should consider incorporating OSA screening into their history-taking and examination of patients. Individual orthodontist should be made to participate in the treatment and monitoring of OSA patients as appropriate and permissible under applicable laws, standards of care and insurance coverages. The decision for an orthodontist to participate in the treatment of OSA is a choice that should be made based on interest as well as training, skills, experience, laws, standards of care, and insurance coverage applicable to the orthodontist.

4. Source of Funding

None.

5. Conflict of Interest

None.

References

1. Parati G, Lombardi C, Narkiewicz K. Sleep apnea: epidemiology, pathophysiology, and relation to cardiovascular risk. *Am J Physiol Regul Integr Comp Physiol*. 2007;293(4):1671–83.
2. Smith PL, Gold AR, Meyers DA. Weight loss in mildly to moderately obese patients with obstructive sleep apnea. *Ann Intern Med*. 1985;103(6):850–5.
3. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The report of an American Academy of sleep medicine task force. *Sleep*. 1999;22(5):667–89.
4. Weaver TE, Chasens ER. Continuous positive airway pressure treatment for sleep apnea in adults. *Sleep Med Rev*. 2007;11(2):99–111.
5. Lowe AA. Oral appliance for the treatment of snoring and obstructive sleep apnea. *J Can Dent Assoc*. 1999;65(10):571–4.
6. Behrents RG, Shelgikar AV, Conley R, Flores-Mir C, Hans M, Levine M, et al. Obstructive sleep apneaand orthodontics: An American Association of orthodontists white paper. *Am J Orthod Dentofacial Orthop*. 2019;156(1):13–28.
7. Oza V. Dental appliances and mandibular advancement devices in obstructive sleep apnea. *Int J Head Neck Surg*. 2019;10(2):31–3.
8. Apoorva S, Shaashi H, Selvaraj S. Oral appliances: An odds - on in obstructive sleep apnoea management. *J Acad Dent Educ*. 2021;7(2):41–5.
9. Brooks D, Horner RL, Kozar LF, Render-Teixeira CL, Phillipson EA. Obstructive sleep apnea as a cause of systyemic hypertension. Evidence from a canine model. *J Clin Invest*. 1997;99(1):106–9.
10. Jacobowitz O. Advances in Oral Appliances for Obstructive Sleep Apnea. *Adv Otorhinolaryngol*. 2017;80:57–65. doi:10.1159/000470865.
11. 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(6):501–10.
12. Robin P. Glossoptosis due to atresia and hypertrophy of the mandible. *Am J Dis Child*. 1934;48:541–7.
13. Ramar K, Dort LC. Clinical Practice Guideline for the Treatment of Obstructive Sleep Apnea and Snoring with Oral Appliance Therapy: An Update for 2015. *J Clin Sleep Med*. 2015;11(7):773–827.
14. Rodríguez-Lozano FJ, Sáez-Yuguero MR, Tovar EL, Fenoll AB. Sleep apnea and mandibular advancement device. Revision of the literature. *Med Oral Patol Oral Cir Bucal*. 2008;13(9):549–54.
15. Chan AS, Sutherland K, Schwab RJ, Zeng B, Petocz P, Lee RW, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax*. 2010;65(8):726–32.
16. Kushida CA, Morgenthaler TI, Littner MR, Alessi CA, Bailey D, Coleman-Jr J, et al. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliamnces . An update for. *Sleep*. 2005;29(2):240–3.
17. Marklund M, Verbraecken, Randerath W. Non-CPAP therapies in obstructive sleep apnea: Mandibular advancement device therapy. *Eur Respir J*. 2012;39(5):1241–7.
18. Ahrens A, McGrath C, Hägg U. A systematic review of the efficacy of oral appliance design in the management of obstructive sleep apnea. *Eur J Orthod*. 2011;33(3):318–24.
19. Phillips CL, Grunstein RR, Darendeliler MA, Mihailidou AS, Srinivasan VK, Yeeb J, et al. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: a randomized controlled trial. *J Respir Crit Care Med*. 2013;187(8):879–87.
20. Sutherland K, Vanderveken OM, Tsuda H, Marklund M, Gagnadoux F, Kushida CA, et al. Oral appliance treatment for obstructive sleep apnea: an update. *J Clin Sleep Med*. 2014;10(2):215–7.

Author biography

Nazia Beg, Senior Resident https://orcid.org/0000-0003-1147-0772

Astitav Mittal, Student

Mohit Makkar, Student

Cite this article: Beg N, Mittal A, Makkar M. Orthodontic perspectives of obstructive sleep apnea. *IP Indian J Orthod Dentofacial Res* 2024;10(3):171-176.