ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 3, Issue – 6, December - 2020, Page No. : 114 - 120

Positioning Obstructive sleep apnea-hypopnea syndrome in Orthodontic practice: A Review

[1]Dr. Rony T Kondody, MDS .Department of Orthodontics & Dentofacial Orthopaedics at ESIC Dental College and Hospital, Gulbarga.

[2]Dr. Arshad Hussain, Reader, Department of Orthodontics & DentofacialOrthopaedics at Al-Badar Rural Dental College & Hospital, Gulbarga.

[3]Dr. Safiya Sana, Reader, Department of Orthodontics & DentofacialOrthopaedics at Al-Badar Rural Dental College & Hospital, Gulbarga.

[4]Dr Rekha Reddy, MDS, Al-Badar Rural Dental College & Hospital, Gulbarga.

Corresponding Author: Dr. Rony T Kondody, MDS .Department of Orthodontics & Dentofacial Orthopaedics at ESIC Dental College and Hospital, Gulbarga.

Citation of this Article: Dr. Rony T Kondody, Dr. Arshad Hussain, Dr. Safiya Sana, Dr Rekha Reddy, "Positioning Obstructive sleep apnea-hypopnea syndrome in Orthodontic practice: A Review", IJDSIR- December - 2020, Vol. – 3, Issue - 6, P. No. 114 – 120.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

This review aimed to identify and summarize the importance of positioning obstructive sleep apnea in orthodontic practice. The objectives were to understand the influence of various craniofacial factors influencing the development of this condition. The orthodontic and dentofacial orthopedic specialty involves not only concerned with moving teeth and correction of malocclusion but also focuses on the management of various orofacial disorders including screening, diagnosis, and management of obstructive sleep apnea. OSA requires a thorough clinical examination as well as overnight testing to determine presence and severity before initiating treatment. Polysomnography (PSG) remains the most reliable method in the early diagnosis of obstructive sleep apnea syndrome. In the case of moderate to severe OSA, CPAP is still the mainstay for the treatment. However, oral appliances appear to be an effective non-surgical alternative for many individuals with either snoring alone or mild conditions of OSA. Early assessment, along with coordinated efforts made by the attending physician and orthodontist involved, a significant number of patients with OSA can be treated successfully with oral appliances

Keywords: oral appliances, obstructive sleep apnea, Polysomnography

Introduction

Sleep-disordered breathing (SDB) is characterized by increased upper airway resistance, which temporarily interrupts pulmonary ventilation, oxygenation, and sleep quality represents as respiratory disorders from snoring to

various degrees of obstructive sleep apnea (OSA).[1] The orthodontic and dentofacial orthopedic specialty involves not only concerned with moving teeth and correction of malocclusion but also focuses on the management of various orofacial disorders including screening and management.

As a specialist in the field of study of the science of oro-facial growth and development combined with in-depth knowledge of various oral appliances, orthodontists are well suited to collaborate and work for hand in hand with other health practitioners in the early diagnosis and treatment of obstructive sleep apnea.[2]

Obstructive sleep apnea/hypopnea syndrome (OSAHS) is considered to be a common clinical condition that affects a large number of populations worldwide.[3] It is characterized by recurrent events of upper airway obstruction during sleep which may involve awakening with reduction (hypopnea) or complete cessation (apnea) of airflow in the presence of respiratory movements. According to the study of the American Association of Sleep Medicine, an accurate diagnosis requires at least five episodes of apnea/hypopnea per hour of sleep combined with clinical signs and symptoms, the most important of which are excessive daytime sleepiness and loud snoring.[4]

By convention, apneas which are lasting for 10s or more duration have been considered to be clinically significant important in various patients, whereas they are the condition of apnea which may last for 15–20s or last as long as 1–3 min.[5-7]

It can be classified as central sleep apnea, obstructive sleep apnea, and mixed apnea which a combination of central and obstructive.[8]

- In **central sleep apnea**, the airway is not blocked but the brain or central nervous system fails to signal the muscles to breathe due to the instability in the respiratory control center.
- **In obstructive sleep apnea**, repetitive episodes of upper airway blockage are seen during sleep, which results in reduce in oxygen distribution to vital organs, and cause heart rhythm irregularities.

Prevalence

Obstructive sleep apnea syndrome condition affects an average of 2% of adult females and 4% of adult males, increasing as of the fifth decade of life.[9] OSA with or without daytime sleepiness can be observed in a total of 9% of women and 24% of men. The majority of cases remain undetected which corresponds to about 80% to 90% of adult patients. About 2% of preschool ages children are affected to varying degrees.[10]

Pathogenesis of Obstructive Sleep Apnea / Hypopnea Syndrome (OSAHS)

The distinctive feature of obstructive sleep apnea syndrome is that there will be upper pharyngeal airway space occlusion, mainly at the level of the oropharynx.[11] The resulting airway obstruction will lead to progressive asphyxia which will continue to remain until there is brief arousal from sleep. The immediate reason leading to the collapse of the upper airway and associated spaces in case OSA is the production of critical subatmospheric pressure during inspiration which may exceed the maximum potential of the dilator and abductor muscles of the airway to maintain the stability of the pharyngeal airway.

The size of the upper pharyngeal airway space is reduced, either due to fat deposition or it can be because of the compression of the pharynx by superficial fat masses.[12] High-frequency vibration due to snoring also affects the soft tissues of the palate and pharynx that may affect the size of the airway lumen which may aggravate the narrowing by producing edema of the soft tissues.[13-15,]

Chronic persistent sleeping is a common symptom associated with obstructive sleep apnea, caused by the interplay between multiple factors including sleep-related issues in the tissues, enlarged tonsils, macroglossia, a retrognathic mandible, and also various medications include sedatives and allergic drugs.[16,17]

Upper pharyngeal airway space is also being affected by variation in hormonal level, impaired neuromuscular tone, rostral fluid shifts, and genetic predisposition which shows a wide range of clinical presentation that needs multidisciplinary and interdisciplinary management approach.[2]

Familial and Genetic Predisposition

Familial aggregation is identified as a source in some cases of obstructive sleep apnea which was first recorded and identified by Strohl and coworkers.[18] Which was supported by the studies of Redline S and Tishler PV who later confirmed the influence of various factors like inheritance and genetic.[19] Genome-wide association scans found out the susceptibility loci that show that linkage patterns for the disorder may differ among whites and African Americans.[20,21]

Even though, some authors suggested the influence of obesity, definitive conclusions on genetic underpinnings require further in-depth investigation to define the genetic basis of the disease.[22]

The importance of heritability has been demonstrated to influence anatomical structures like the lateral pharyngeal walls volume, tongue, and other associated soft tissue structures in and around craniofacial areas.[23] Inherited abnormalities as well as various other factors may influence to initiate the occurrence of disordered breathing which is more severe during sleep.[24,25] Along with other multiple factors, genetic determinants of obesity and regional fat distribution are also important factors in the development and pathogenesis of this condition.

Diagnosis

Detailed medical and sleep history should be obtained prior, and a physical examination should be performed before initiating any treatment in a suspected case of OSA. History of various factors that may predispose to loud and deep snoring, apneas, choking episodes, excessive daytime sleepiness, nocturia, severe headaches, sleep fragmentation, or decreased or reduced concentration should be examined'.

During clinical examination, attention should also be given to the presence of physical characteristics, such as large neck circumference (>17 inches in men, >16 inches in women), an increased body mass index (30 kg/m2), macroglossia, tonsillar hypertrophy, and enlarged/elongated uvula.[26]

Obstructive sleep apnea can be diagnosed or identified by polysomnography. It shows a pattern of obstructive hypoventilation, which is defined as at least 25% of total sleep time with hypercapnia (PaCO2 .50 mm Hg) associated with at least 1 of the following conditions. The conditions include (1) snoring, (2) paradoxical thoracoabdominal motion, (3) flattening of the inspiratory nasal pressure waveform.[27]

Management of Obstructive Sleep Apnea

The therapeutic approach in treating a case of obstructive sleep apnea requires multidisciplinary and interdisciplinary communication between various healthcare professionals, along with regular follow-up.

Basic Measures

- In a mild case of obstructive sleep apnea, weight loss measures help in relieve constriction of the airway, and also it could improve health and quality of life.
- Avoid alcohol and medication such as anti-anxiety drugs and sleeping pills

- Sleep on your side or stomach rather than on the back which will prevent the tongue and soft palate to rest against the back of the throat.
- Keep the nasal passage open while sleeping by use of saline nasal sprays or use of nasal decongestant.

Nasal Continuous Positive Airway Pressure

Continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BiPAP) is the standard option in treating moderate to severe cases of OSA.[28] It consists of delivering, during sleep, compressed air into the airway to keep it open, by positive pressure across the airway walls, and pneumatic splinting effect.

CPAP can be administered through the oral, nasal, or oro-nasal interface. The success of treating OSA with CPAP depends greatly on individual patient acceptance and compliance. This can fall for numerous reasons including functioning noise, discomfort, feelings of claustrophobia, and skin irritation. Thus, CPAP prescription requires an explanation of benefits and medical reasons for its use. Patients should also be informed about the function and maintenance of equipment.[2]

Orthodontic Appliances

Various orthodontic treatment modalities or appliance systems have proven to be an effective method in early management of mild to moderate cases of OSA, and, at the same time, improve the associated craniofacial abnormalities. These include appliances like rapid maxillary expansion (RME)[29] which reduces the constriction of maxillary arch thereby open up the airway, mandibular advancement appliances (MAAs)[30], and orthopedic maxillary protraction appliances, etc. Usage of these appliances is truly based on the severity of the patient's condition and compliance with the treatment.[31]

The effectiveness and success of these orthodontic appliances in improving symptoms and conditions have been attributed to enlarging the reduced pharyngeal airway space as well as by increasing or widening the lateral dimension of the the velopharyngeal airway. This is accomplished by forwarding the positioning of the mandible and thereby reducing the collapsibility of the pharyngeal airway.[32]

Stimulation of various dilator muscles especially genioglossus muscle with the help of various advancement appliances has also been proved to be very effective in improving upper pharyngeal airway stabilization.[33] These also alter various neuromuscular forces that are influencing the craniofacial skeleton and dentition, thereby promoting a combination of overall skeletal growth and dentoalveolar changes.

These orthodontic appliances are suggested as an efficient alternative therapy compared to the use of CPAP in cases of mild to moderate OSA patients, for those who do not tolerate or adhere to CPAP, or those patients who refuse surgical correction. They can be also used for the treatment of patients with snoring, who do not respond to various behavioral measures or therapy such as weight loss or sleep position change.[2,34,35]

The design and construction of Mandibular advancement devices (MAD) which is used for treating various cases of OSA is solely based on a complete clinical examination of the stomatognathic system which consists of soft tissues, dental occlusion, masticatory muscles, and the temporomandibular joint, etc.[36] Mandibular advancement devices will cover the occlusal surfaces of upper and lower dentition, in turn, hold the mandible in more an advanced position concerning the resting position. This should be used until the severity there is a reduction of snoring and daytime sleepiness to an acceptable or normal level, or the patient cannot tolerate further advancement. This function basically by widening or enlarging the collapsed upper pharyngeal airway space by moving the

mandible and tongue forward and then the activation of airway dilator muscles.[2]

Craniofacial skeletal changes induced by various orthodontic appliances in treating mild to moderate cases were evaluated using cephalometric analysis which shows, changes like retroclination of the maxillary incisors, proclination of the mandibular incisors, increased lower facial height, and changes in a molar relationship. Also reducing soft-tissue edema in the pharyngeal airway area, associating orthodontic appliances seems to result in a decrease in palatal length and an increase in pharyngeal area.[37]

Surgical Measures

Surgery correction should be considered for severe cases after proper clinical diagnosis and severity assessment by various or multiple objective testing methods. It is highly recommended for patients with severe obstructing anatomical conditions like tonsillar hypertrophy and nasal obstruction which block complete cessation of the airway. Also, it can be used were other non-invasive therapies which include orthodontic appliances that fail to control or ineffective in treating mild, moderate, and severe cases of airway obstruction.[38]

The surgical management or treatment includes clinical evaluation of the pharyngeal airway for detection of various skeletal or soft tissue abnormalities like:

1. The nose and associated structures like alar cartilage deformities, septal deviations, enlarged turbinates, nasal floor constriction, etc.
2. The retroglossal area and the tongue for mandibular retrognathia, macroglossia.
3. The retropalatal area for lymphoid hyperplasia, retrusive or deficient maxilla, deep palate.

Thus, various surgical procedures that can be used can be further classified based on the anatomical area involved like intra-pharyngeal or skeletal region. The intra-pharyngeal surgical procedure is mainly uvulopalatopharyngoplasty (UPPP), which is directed for the correction of soft tissue defects of the upper pharyngeal airway space. Hard tissue or skeletal surgery which are widely used are maxillo-mandibular advancement (MMA) and genioglossus advancement (GGA) procedures.[2,39,40]

Conclusion

Obstructive sleep apnea syndrome is one of the most common breathing disorder which is seen, with severe potential pathophysiological consequences. It severely affects the quality of life, which in turn shows increased morbidity and mortality in affected individuals. Assessment and early systematic diagnosis along with coordinated efforts made by the attending physician and orthodontist involved need a thorough and proper clinical examination as well as overnight testing to determine various levels severity of conditions before the start of treatment.

Polysomnography is still considered to be the most reliable method which is used in the early detection of obstructive sleep apnea syndrome. In moderate conditions, continuous positive air pressure is the mainstay along the use of orthodontic appliances appears to be an effective non-surgical alternative. However, very severe conditions require early surgical intervention so that further complications can be minimized.

Reference

1. Nazarali N et al. Mandibular advancement appliances for the treatment of pediatric obstructive sleep apnea: a systematic review. Eu J Orthod 2015;37(6):618–626
2. Behrent RG et al.Obstructive sleep apnea and orthodontics: An American Association of Orthodontists White Paper. Am J Orthod Dentofacial Orthop 2019;156:13-28

3. Epstein LJ et al. Clinical guideline for the evaluation, management, and long-term care of obstructive sleep apnea in adults. Adult obstructive sleep apnea task force of the American Academy of Sleep Medicine. J Clin Sleep Med. 2009; 5:263–76.
4. Jureyda and David W. Shucard Obstructive Sleep Apnea—An Overview of the Disorder and Its Consequences. Semin Orthod 2004;10:63-72.
5. Shepard JW Jr. Cardiopulmonary consequences of obstructive sleep apnea. Mayo Clin Proc 1990;65:1250-9.
6. KalesA, Caldwell, Cadieux RJ, Vela-BuenoA, Ruch LG, Mayes SD. Severe obstructive sleep apnea – II: Associated psychopathology and psychosocial consequences. J Chronic Dis 1985;38:427-34.
7. Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. Am J Cardiol 1983;52:490-4.
8. Partinen M, Guilleminault C. Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. Chest 1990;97:27-32.
9. Clark GT, Sohn JW, Hong CN. Treating obstructive sleep apnea and snoring: assessment of an anterior mandibular positioning device. J Am Dent Assoc. 2000; 131:765–71
10. Bixler EO et al. Prevalence of sleep-disordered breathing in women: effects of gender. Am J Respir Crit Care Med 2001;163: 608-613.
11. Victor LD. Obstructive Sleep Apnea in Primary Care. Dearborn, Michigan: Oakwood Hospital; 1997
12. Krol RC, Knuth SL, Bartlett D Jr. Selective reduction of genioglossal muscle activity by alcohol in normal human subjects. Am Rev Respir Dis 1984;129:247-50.
13. Issa FG, Sullivan CE. Alcohol, snoring, and sleep apnea. J Neurol Neurosurg Psychiatry 1982;45:353-9.
14. Wittig R. Clinical evaluation of excessive daytime sleepiness. In: Victor LD, editor. Clinical Pulmonary Medicine. Boston: Little, Brown; 1992. p. 439-60.
15. 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
16. Gottlieb DJ et al. Relation of sleepiness to respiratory disturbance index, the Sleep Heart Health Study. Am J Respir Crit Care Med 1999;159: 502-507.
17. Cistulli PA. Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment. Respirology 1996;1:167–174.
18. Strohl KP, Saunders NA, Feldman NT, Hallett M. Obstructive sleep apnea in family members. N Engl J Med 1978;299:969–973
19. Redline S, Tishler PV. The genetics of sleep apnea. Sleep Med Rev 2000;4:583–602.
20. Palmer LJ, Buxbaum SG, Larkin EK, Patel SR, Elston RC, Tishler PV, Redline S.Whole genome scan for obstructive sleep apnea and obesity in African-American families. Am J Respir Crit Care Med 2004;169:1314– 1321.
21. Palmer LJ, Buxbaum SG, Larkin E, Patel SR, Elston RC, Tishler PV, Redline S. A whole-genome scan for obstructive sleep apnea and obesity. Am J Hum Genet 2003;72:340–350.
22. Pack AI, Gislason T, Hakonarson H. Linkage to apnea-hypopnea index across the life-span: Is this a viable strategy?. Am J Respir Crit Care Med 2004;170:1260–1261
23. Schwab RJ, Pasirstein M, Kaplan L, Pierson R, Mackley A, Hachadoorian R, Arens R, Maislin G, Pack AI. Family aggregation of upper airway soft

tissue structures in normal subjects and patients with sleep apnea. Am J Respir Crit Care Med 2006;173:453–463.

24. Pillar G, Schnall RP, Peled N, Oliven A, Lavie P. Impaired respiratory response to resistive loading during sleep in healthy offspring of patients with obstructive sleep apnea. Am J Respir Crit Care Med 1997;155:1602–1608.
25. Redline S, Leitner J, Arnold J, Tishler PV, Altose MD. Ventilatory control abnormalities in familial sleep apnea. Am J Respir Crit Care Med 1997;156:155–160.
26. Al-Falahi Z, Williamson J, Dimitri H. Atrial fibrillation and sleep apnoea: guilt by association? Heart Lung Circ 2017;26:902
27. American Academy of Sleep Medicine. The AASM manual for the scoring of sleep and associated events: rules, terminology, and technical specifications: version 2.5: Darien, Ill; 2018.
28. Sullivan CE, Issa FG, Berthon-Jones M, et al. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet 1981; 1: 862-5
29. Pirelli, P., Saponara, M. and Guilleminault, C. Rapid maxillary expansion in children with obstructive sleep apnea syndrome. Sleep 2004; 27:761–766.
30. Tauman, R., Gulliver, T.E., Krishna, J., Montgomery-Downs, H.E., O'Brien, L.M., Ivanenko, A. and Gozal, D. Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. J Pediatrics 2006;149, 803–808
31. Hiyama, S., Suda, N., Ishii-Suzuki, M., Tsuiki, S., Ogawa, M., Tsuiki, S., Ogawa, M., Suzuki, S. and Kuroda, T. Effects of maxillary protraction on craniofacial structures and upper-airway dimension. Angle Orthod 2002;72, 43–47. 15.
32. Chan, A.S., Phillips, C.L. and Cistulli, P.A. Obstructive sleep apnoea–an update. Internal Med J 2010; 40;102–106.
33. Tsuiki, S., Ono, T. and Kuroda, T. Mandibular advancement modulates respiratory-related genioglossus electromyographic activity. Sleep Breath 2000;4:53–58
34. Epstein LJ, Kristo D, Strollo PJ et al. Clinical Guideline for the Evaluation, Management and Long-term Care of Obstructive Sleep Apnea in Adults. Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. J Clin Sleep Med 2009; 5(3): 263-76
35. Prabhat KC, Goyal L, Bey A e al. Recent advances in the management of obstructive sleep apnea; the dental perspective. J Nat Sci Biol Med. 2012; 3(2): 113-18
36. An update on mandibular advancement devices for the treatment of obstructive sleep apnoea hypopnoea syndrome. J Thorac Dis 2018;10(1): 48–56.
37. De Almeida FR, Lowe AA, Sung JO. Long-term sequelae of oral appliance therapy in obstructive sleep apnea patients: Part 1. Cephalometric analysis. Am J Orthod Dentofacial Orthop 2006; 129: 195-204
38. Sesso DM, Powell NB, Riley RW. Upper Airway Surgery in the Adult. In Kushi- da CA. (ed.) Obstructive sleep apnea: diagnosis and treatment. Informa Healthcare USA, Inc. 2007;191-215
39. Jacobson RL, Schendel SA. Treating obstructive sleep apnea: The case for surgery. Am J Orthod Dentofacial Orthop 2012; 142: 434-42
40. Li KK. Maxillomandibular advancement for obstructive sleep apnea. J Oral Maxillo- fac Surg 2011; 69 (3): 687-94.