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Orthodontic view in the diagnoses of obstructive sleep apnea

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Abstract:

Obstructive sleep apnea is an exciting area for orthodontists to be involved. The level of awareness of sleep apnea and related health issues is growing rapidly. The demand of integrating sleep into the orthodontic practice shortly will be driven by the need of the societies as some of our patients will be shortly coming into our offices aware of sleep apnea. However, with our busy clinical orthodontic practice, the need of condense short review become more demanding. Therefore, this review will try to summarize the clinical and orthodontic observation in the diagnoses of adult obstructive sleep apnea with clinical application in orthodontic practice.

Keywords:

Adult, clinical orthodontic, diagnoses, sleep apnea, review

Introduction

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. This creates repetitive episodes of obstruction of the upper airway causing a loss of breath and oxygen, anywhere from 10 to 30 seconds or longer.^[1] When this occurs, blood oxygen levels drop, and heart rate and blood pressure rise.^[1] The brain ultimately sends a distress signal that partially or fully wakes the person and alerts the body to breathe, causing the patient to gasp for air.^[2] 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.^[3] The standard OSA severity measuring system is called the apnea-hypopnea index (AHI), which is defined as the number of apnea

plus hypopnea per hour of sleep.^[2] The AHI has been used to classify patients as either having OSA or being normal (AHI < 5 events per hour of sleep), as well as to classify the severity of OSA. The American Academy of Sleep Medicine classified OSA severity according to AHI as mild sleep apnea (AHI 5–15 events per hour of sleep); moderate sleep apnea (AHI 15–30 events per hour of sleep); severe sleep apnea (AHI >30 events per hour of sleep).^[3] The National Center for Sleep Disorders study estimated that between 50 and 70 million Americans are affected by a sleep disorder.^[1] In other Western countries, the overall prevalence of mild and moderate obstructive sleep apnea syndrome (OSAS) is estimated at 20% and 7%, respectively.^[4] In South America, OSAS was observed in 32.8% of the participants and is considered to be one of the higher prevalence.^[5] In the Middle East, 3 out of 10 Saudi men and 4 out of 10 Saudi women are at a high risk of developing OSA.^[6,7]

Clinical Observation of Obstructive Sleep Apnea and Risk Factors

There are certain behavioral and systemic factors that can contribute to sleep apnea.

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These include obesity, sleeping in the supine position, and alcohol (or any other central nervous system depressant).

There are also anatomic factors that can contribute to OSA such as maxillary or mandibular retrognathism, increased lower face height, large tongue, elongated soft palate, and an inferiorly positioned hyoid bone. This list points out how critical the role of the orthodontist can be in diagnosing OSA.^[8]

Clinical prediction models

Clinical prediction models have been developed to improve the predictive value of clinical variables. The models make use of demographic and anthropometric data combined with self-reported symptoms. In summary, the researcher found out that the best indicator factors for the presence of OSA are body mass index (BMI), neck circumference and witnessed apneic events. Interestingly, age, snoring and the presence of hypertension did not differentiate OSA patients from normal.^[9]

Orthodontic Observation

Anatomical and craniofacial observation

Craniofacial factors play an important role in the pathogenesis of obstructive sleep and particularly significant in Asian patients.^[10] The importance of considering patients' craniofacial morphology as a factor that protects or worsens OSA is confirmed by a systematic literature review carried out by Pirklbauer *et al.* in 2011.^[11] A meta-analysis of the available literature examined the possible relationship between craniofacial structure and OSAS considered the mandible body length as the most potentially significant factor associated with OSA.^[12] Patients with OSA manifest a spectrum of abnormalities of craniofacial and skeletal anatomy that appear to promote upper airway obstruction during sleep.^[13] The contributions of skeletal abnormalities vary among patients and between ethnic groups. For example, Asians have more pronounced skeletal abnormalities and are less obese than Caucasians.^[14] Young *et al.* suggested that OSA patients manifest several skeletal and soft tissue abnormalities, including abnormalities related to size and position of mandible and maxilla, narrowed nasal cavities, and tonsil hypertrophy.^[15]

In a preliminary report, certain characteristics of the craniofacial structure were observed and found to be common in patients with OSAS. These include mandibular deficiency, bimaxillary retrusion, short cranial base, reduced cranial base angle and mandibular length, increased lower anterior facial height, craniocervical angulations, inferiorly positioned hyoid, and enlarged soft palate.^[16] Similarly, shorter and steeper anterior

cranial base, smaller midface, a smaller and more posteriorly positioned mandible, and larger total and upper facial height were more observable in Chinese compared to Caucasians.^[17] However, mandibular deficiency related to maxilla has been reported as the most common skeletal abnormality predisposing to OSA.^[18] Thus, deficiency leads to restricting of the space in the oropharyngeal area.^[19] As a consequence, the tongue, soft palate, and soft tissues surrounding the upper airway are displaced posteriorly that lead to constricting of the upper airway lumen. This point is supported by the fact that mandibular advancement by appliances or surgery increases the size of the pharyngeal lumen and reduces the severity of OSA.^[20,21]

Malocclusion and palatal morphology observation related to obstructive sleep apnea patients

Malocclusion and OSA are other areas of interest to the orthodontist. However, the influence of malocclusion in OSAS is still an unending issue. There has been growing interest in 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.^[22] From the orthodontic point of view, reduced overbite, narrower upper, and shorter lower dental arch; crowding in the mandibular arch; Class II malocclusion were reported as dental features that were associated with OSA patients.^[23,24] Specifically, lateral crossbite and increased overjet were seen more frequently in OSA patients. Lateral crossbite due to maxillary skeletal constriction were found to be approximately 30–50% in OSA patients^[25,26] compared to 10 per cent which was found in the general population.^[27]

Overjet was also found to be associated with the severity of OSAS in nonobese patients, and this leads the researcher to suggest that malocclusion may play an important role in the development of sleep apnea/hypopnea.^[28] In addition to the above mentioned dental features, the associations of malocclusions class and obstruction of the upper airways have been suggested. This implies that these malocclusion characteristics may have a predisposing anatomical factor for these problems. Paul and Nanda found greater prevalence of mouth breathing and nasopharyngeal airway obstruction in patients with Class II malocclusions.^[29] According to the Balters' philosophy, Class II malocclusions are a consequence of a backward position of the tongue, disturbing the cervical region. The respiratory function is impeded in the region of the larynx and there is thus a faulty deglutition and mouth breathing.^[30] In addition, Class II malocclusion was found to be the most frequent findings among OSA Malay patients when compared with the control group.^[23] This lead to an interesting question, is a patient with Class II malocclusion at a higher risk of having a higher apnea-hypopnea index

than a patient with Class I occlusion? The Regression analysis supported yes answer for this question as an increased in overjet was found to be a significant factor contributing to increased AHI in OSA patients.^[28] In addition, a research by Kim *et al.* found that people who responded poorly to the surgery were skeletal class II with a more retrognathic mandible which supported the observable fact.^[31] This led Patel and Nagle (2013) in their critically appraised summary to conclude that the findings of Class II malocclusion in OSA appear consistent enough to give strong consideration in the etiology/pathology of this disorder.^[32] We would add to Patel and Nagle statement “in some ethnicity” as we found that Class I malocclusion were the most common findings in other ethnicities with snoring. While the development and causes of OSA are multifactorial, having a Class I or II malocclusion seems to be associated with a higher incidence of OSA. Furthermore, it has been reported that the upper pharyngeal width in the subjects with Class I and Class II malocclusions with the vertical growth patterns were significantly narrower than in the normal growth pattern group.^[33]

On the other hand, the relationship between maxillary constriction and the etiology of OSA were examined and the researchers reported a familial tendency of narrow, high palates in the relatives of OSA patients.^[23] 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.^[26] Other dental features which were reported frequently in OSA patients is a palatal morphology. The V palatal shape was included as a predictive morphometric model for OSA, and the model illustrates the potential value of physical and dental examination.^[34] A palatal morphology with V shape and decrease in the upper arch length and inter first upper premolar distance were also reported to be associated with snoring in a university population.^[35] In addition, we previously found that those patients with OSA had significantly narrower maxillary and mandibular arch widths with an increase in maxillary and mandibular arch length when compared with controls.^[23] In view of the fact that the roof of the mouth is also the floor of the nose, a narrow arch can infringe on the nasal cavity space. Therefore, maxillary dimensions. In addition to contributing to high nasal resistance, maxillary constriction can contribute to the lateral narrowing of the upper oral cavity and a low tongue posture, which consequently narrow the retroglossal region.^[26] This may provide a reasonable explanation for the narrowing of the posterior airway space seen in OSA patients. Nevertheless, palatal height measurement alone was not a reliable indicator of maxillary constriction.^[25] Thus, it remains very questionable as to whether maxillary and or mandibular constriction can be a primary etiological factor in OSA.

However, the close association between the size of the dental arch and the improvement of OSA in children and adult after rapid maxillary expansion may provide further support for this concept.^[36-38]

Cephalometric observation

Today, the diagnosis and treatment of OSA depend on a multidisciplinary team of health professionals. The orthodontists have a fundamental role in OSA recognition and screening through radiographs that are part of their work routine. Lateral cephalometry is a readily available, inexpensive, and reliable technique for assessing the pharyngeal airways, whereby details of skeletal and soft tissue structures can be accurately measured and compared with extensive normative data.^[39] Battagel *et al.* in his cephalometric comparison of patients with snoring and OSA pointed out to various skeletal differences in both horizontal and vertical planes. For example, anteroposteriorly, 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.^[40] In addition, mandibular retrusion may also occur. In the vertical plane, increases in lower face height and maxillomandibular planes angle have been reported.^[40] Other Cephalometric features that associated with OSA are smaller than normal SNA and/or SNB; increased Frankfort mandibular plan angle and/or maxillary mandibular plan angle.^[41] However, cephalometric measurements alone are not sufficient to diagnose the severity of OSA, overnight, facility-based, and attended PSG remains the criterion standard for diagnosis of OSA.^[2,3]

Soft tissue observation and facial morphology in obstructive sleep apnea patients

Soft tissue features associated with OSA patients are narrower retropalatal and retroglossal airway space; thicker and longer soft palate; reduced angle between the uvula tip and anterior nasal spine; and greater tongue mass.^[41] Nevertheless, there is a lack of studies focusing on establishing a relationship between facial morphology and obstructive sleep apnea based on the subjective determination of facial morphological pattern and facial type. The discrepancy between the influences of the facial profile on airway measurements is still ongoing. For example, the facial morphological pattern was found to influence AHI in an ascending order along with sex, age, and BMI in Brazilian sleep patients.^[42] Clinical examination findings in south-east Asian ethnicity indicate that the most frequent finding among snorer and OSA groups were the convex profile.^[23] In the Middle East, straight profiles shape were found to more frequent.^[35] Brachyfacial type was found to be more associated with severe apnea than the dolichofacial type.^[42]

However, we can still emphasize that OSA is a multifactorial disease, the etiology of OSA is too diverse and complex to be explained by a simple relationship established between facial morphology and the development of the disease.

Conclusion and Practical Orthodontic Applications

In this review, the diagnosis of adult OSA was an overview from the orthodontic prospective, we did not go in depth with OSA treatment, which will need another short clinical review. However, we can still emphasize two points related to diagnosis and treatment planning of OSA patients. First, for obese orthodontic OSA patients with a convex type of facial divergence, the orthodontist may implement modified treatment goals. For instance, if the orthodontist was considering extraction-based therapy in such patients, less effect might be seen on the profile and on lip support. Second, the evidence of an association between facial morphology and OSA may point to therapeutic orthodontic modalities that enhance the shape of the anatomical traits of the face although such gain is limited by genetic determinism.^[42] In summary, medical history (e.g., snoring) and clinical examination allow orthodontists to identify the risk factors of OSA or signs related to OSA (obesity, allergy, nasal dysfunction, maxillary constriction, retrognathia, long uvula, mouth breathing). The orthodontist can also use validated questionnaires (Epworth; STOP-BANG; Berlin) to screen for sleep related breathing disorders (OSA and snoring) in his orthodontic patient population. Several imaging modalities (lateral and frontal cephalogram, cone beam computed tomography, magnetic resonance imaging) can assist orthodontic professionals in assessment of this condition.^[43] Orthodontic professionals need to expand their cooperation with physicians and sleep medicine specialist and should try to look for sleep lab to refer the patients for better patients care. The orthodontist should be more alert to enquire about snoring even in Class I; class II malocclusion and straight and convex profile patients especially with vertical growth patterns. When orthodontist carries out the functional, positional, and structural assessments of the dentofacial pattern, we strongly support the suggestion of inclusion the assessment of the pharyngeal structures with the orthodontic diagnosis and treatment planning.^[44]

Shortly, the demand of integrating sleep into the orthodontic practice will be driven by the need of the societies as some of our patients will be coming into our offices aware of sleep apnea, and what would be a casual, patient conversation may lead to a positive discussion about how an orthodontist could help in the process of diagnosis and treatment of this disorder.^[45]

Incorporating dental sleep medicine in our orthodontic practice is inspiring. Nevertheless, we understand that this will not happen overnight, but it is worth trying.^[45]

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Conflicts of interest

There are no conflicts of interest.

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