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Chapter 17

Orthodontic Considerations in Obstructive Sleep Apnea — State of the Art

Hakima Aghoutan, Sana Alami, Samir Diouny and Farid Bourzgui

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/59086

1. Introduction

Obstructive Sleep Apnea (OSA) was described as early as 1837 in "The Posthumous Papers of the Pickwick Club". Dickens, a British author, described "Joe", the main character, as a fat boy who falls asleep easily and involuntarily (Figure 1). [1]

In The Pickwick Papers (c. 1836–1837)

Figure 1. Artist (Hablot Knight Browne- Phiz) rendering of Joe, Charles Dickens' character.

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Later on, Osler (1914) used the term "Pickwickian syndrome" to describe obese and sleepy patients, in homage to Dickens' character "Joe". As early as 1956, Bickelmann et al [2] reported that the "Pickwickian syndrome" was associated with extreme obesity and alveolar hypoventilation.

Gastaut's research group described three different types of apnea, namely, obstructive apnea central apnea, and mixed apnea. In 1973, Guilleminault introduced the apnea-hypopnea index (AHI), which refers to the total number of apnea and hypopnea episodes per hour of sleep, and proved, along with Dement, that obesity is not a prerequisite for OSA. In 1977, Guilleminault and Dement used the term "sleep apnea syndrome", in association with hypertension and electrocardiographic pathologies. [3]

Recently, much research on OSA has been conducted with a view to help elucidate the characteristic features of OSA. Sleep is a process through which the body restores energy used during the day. Not much is known about its biological purpose, but its evaluation can be undertaken by muscle and brain electrical activity, and ocular movement. Good-quality sleep entails several functions; these include physical recovery, biochemical refreshment, memory consolidation and psychological well-being. [4]

In adults, sleep is regulated by a cycle of five periods. The first four periods belong to non-rapid eye movement sleep (light and deep stage) and the fifth period is named the rapid-eye-movement (REM) or paradoxical sleep (active stage). The progression from the first stage to the RAM constitutes one sleep cycle. Generally, there are four to six sleep cycles per night; during which activities of the brain, muscles, and the cardio-respiratory system fluctuate (Figure 2, 3). [4]

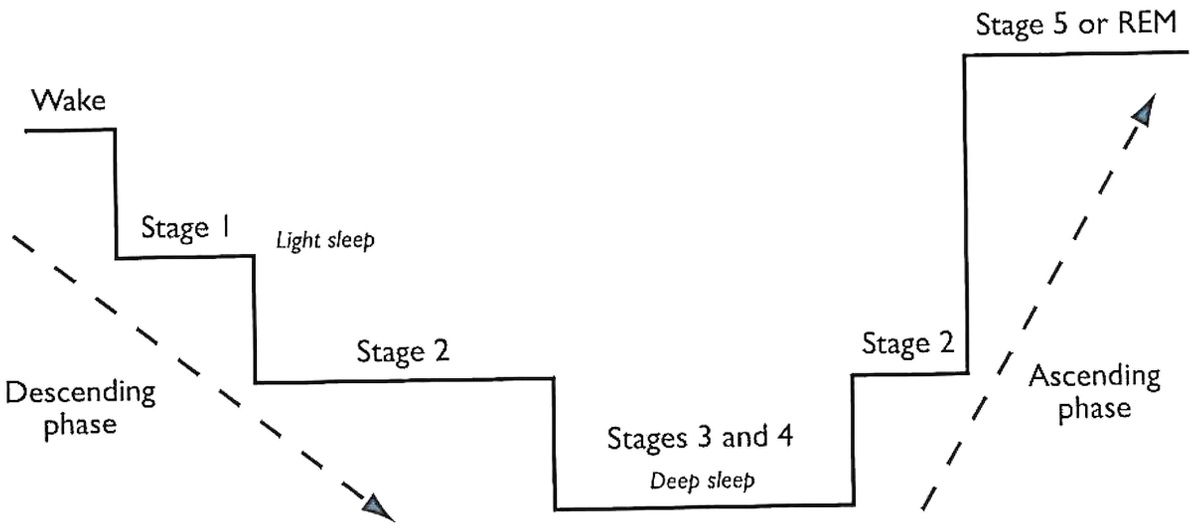

Figure 2. A sleep cycle (non-REM to REM stages). [4]

During these sleep stages, several sleeping disorders can occur. International classification of Sleep Disorders (ICSD-3), revisited in 2014, includes the following broad categories: [5]

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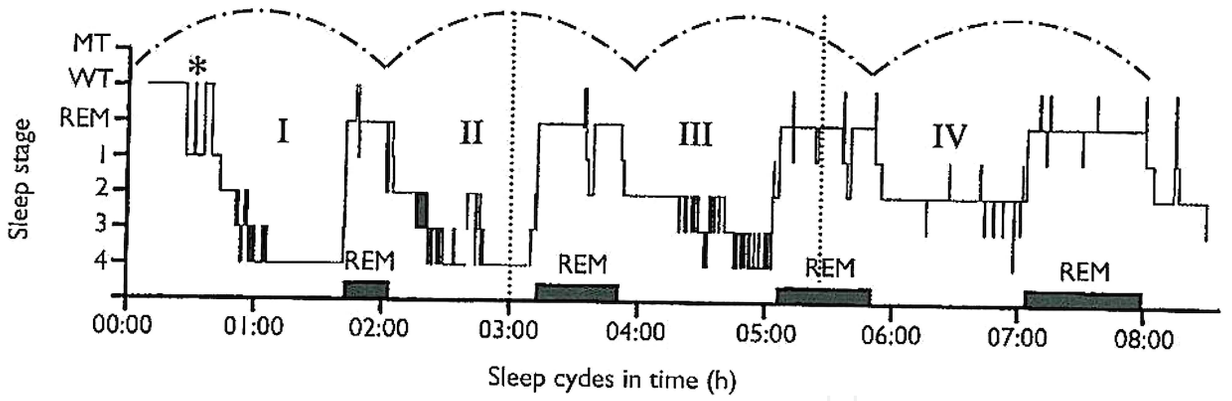

Figure 3. Consecutive wave of non REM to REM sleep cycles (I to IV). Throughout the night, REM becomes longer than slow wave sleep (stage 3and 4). MT: movement time, WT: wake time. [4]

- Insomnia
- Sleep related-breathing disorders
- hypersomnomlence Central disorders
- Circadian rhythm sleep-wake disorders
- Parasomnias
- Sleep related-movement disorders

Sleep apnea is the most common sleep disorder related to breathing. There are 3 types of sleep apnea: Obstructive, central, and mixed (a combination of both forms). Obstructive sleep apnea (OSA) is caused by partial or complete obstruction at multiple levels of the upper airway, producing reduction (hypopnea), or cessation (apnea) of airflow. Due to the lack of adequate alveolar ventilation, oxygen saturation may drop and partial pressure of CO2 may occasionally increase. Snoring and sleep fragmentation are common with OSA that can be graded as mild, moderate and severe.[6] Adults and children are equally affected. However, the prevalence, etiology and pathophysiology of the disorder differ from one group to another. It is important to note that the physiopathology and etiology of OSA are poorly understood.

Furthermore, OSA is associated with neuropsychological impairment, sexual dysfunction, metabolic and cardiovascular co-morbidities; and causes an increase in mortality. Quality of life and economic potential are also affected: snoring affects the sleeping pattern of the partner, and frequent arousals at night result in relative sleep deprivation and can cause excessive daytime sleepiness, loss of concentration and motor vehicle accidents.[4]-[7] Therefore, OSA is regarded as a public health condition and increases the consumption of health care resources.

Continuous positive airway pressure (CPAP) is considered a golden standard treatment; oral appliances and surgical procedures for upper airway soft tissues and maxilla-mandibular advancement are other alternatives. Hence OSA treatment requires a multidisciplinary management. [8] Orthodontists, sleep specialists and surgeons should all be involved in

managing and treating OSA. This chapter gives a comprehensive account of the literature on OSA and underlines the role of orthodontists in managing OSA with a view to improve the physical, mental and social status of patients diagnosed with OSA.

2. OSA epidemiology

2.1. Prevalence

Due to various definitions of respiratory events and differences in study design, contradictory variable prevalence rates of OSA are reported. The American Academy of Sleep Medicine published the first guidelines to standardize the definition of OSA; however, the standardization of OSA definition only expanded the diagnostic criteria.[1] According to the Wisconsin sleep cohort study, the estimated prevalence of moderate to severe sleep breathing disorder in the United States for the period of 1988–2011 ranged from 3% to 17% in adults depending on sex and age; OSA seemed to affect especially middle-aged and elderly men and has increased substantially over the last two decades in the US. [9] In Morocco, however, OSA prevalence ranges from 5, 4% to 7, and 9% in the general population. [10] De Backer (2013) reported that epidemiological studies investigating the prevalence of OSA are all biased because there is a lack of a uniform definition. He also indicated that the prevalence of an AHI of >5 events per hour in the general population (without taking into account symptoms of sleepiness) has been estimated to be 24% in the male population. When symptoms of sleepiness are also taken into account, this prevalence goes down to 4% in males and 2% in females. [11]

2.2. Risk factors

In the literature on OSA, most researches agree that risk factors for OSA include obesity, upper airway and craniofacial abnormalities, gender, age, alcohol consumption and cigarette smoking. [12] Obesity, particularly central or upper body fat distribution, with increased neck circumference (collar size) is a main risk factor for OSA. But this association may be less important with the elderly. In non-obese patients, craniofacial abnormality like micrognathia and retrognathia may also be considered as a risk factor leading to OSA. [13]

Aging is also associated with higher OSA prevalence. Still, it is not clear if OSA in the elderly compared to middle-aged adults manifests itself the same way; middle age and over-weight adult men seem to have the highest prevalence of OSA. However, after menopause, prevalence seems to be the same for both women and men. [1]Some studies have examined craniofacial features among different ethnic groups; their objective was to investigate whether ethnicity differences had an effect on the prevalence of OSA. These studies reported an increased risk of OSA among African-Americans, Latinos and Asians. [14]-[17] Wong et al. (2005) claimed that the hyoid bone was located more caudally in Chinese subjects and may be a severity indicator in this population. [18] In addition, OSA prevalence seems to be much higher in patients with cardiac or metabolic disorders than in the general population. Other factors such as heredity, hormonal change, sedative hypnotics and supine sleep position have also been described as risk conditions for developing OSA. [11, 12]

2.3. Mortality and morbidity

Epidemiologic data have shown a strong association between untreated obstructive sleep apnea and incident cardio and cerebrovascular morbidity and mortality. [19, 20] These co-morbid conditions may be due, in part, to common risk factors (i.e. obesity and hypertension), and also to hypoxemia-hypercapnia, which can lead to vascular dysfunctions. [21] In an18-year mortality follow-up conducted on the population-based Wisconsin Sleep Cohort sample (n = 1522), Young et al. found a significant mortality risk with untreated sleep breathing disorder (SBD). They underscored the need for early diagnosis and treatment of SBD, indicated by frequent episodes of apnea and hypopnea, regardless of sleepiness symptoms.[20]A recent review of OSA in adults reported an increased risk of morbidity and mortality associated with OSA, which reached its peak at 55 years of age. [12], This association seems to disappear after 70 yrs. [22]Sampaio et al., 2012 suggested that women revealed more psychological morbidity associated with OSAS. Therefore, it seems extremely important to look at women as potential patients for sleep apnea. [23] However, Gozal and Kheirandish-Gozal highlighted the potential interaction between gene polymorphisms, organ vulnerability, and the phenotypic expression of OSA and suggested that it should be identified and incorporated into future prediction schemes of morbidity risks associated with OSA. [24]

3. OSA pathophysiology

In recent years, the understanding of the pathophysiology of sleep-breathing disorder has improved. Central nervous system regulation of breathing is now recognized as a significant contributor to the pathogenesis of OSA. To understand the pathophysiologic mechanisms that contribute to OSA, an overview of anatomical and physiological aspects of upper airway is in order.

3.1. Upper airway anatomy and physiology

The upper airway is a complex, multifunctional, and dynamic neuro-mechanical system. It is defined as the passageway for gas and food, beginning at the mouth and nose and ending at the epiglottis and vocal cords. It is composed of bony structures (maxilla, mandible and hyoid bone) and soft tissues (tonsils, soft palate, tongue, uvula, pharyngeal muscle, para-pharyngeal fat pads and lateral wall of the pharynx). The mandible and hyoid bone are the principal craniofacial bone structures that determine the dimensions of the upper airways. Soft tissues form the walls of the upper airways and they are supported by bone structures.[1, 25] The upper airways are typically divided into three segments: The nasopharynx (end of the nasal septum to the margin of the soft palate), the oropharynx (free margin of the soft palate to the tip of the epiglottis), divided into the retropalatal and retroglossal regions, and the hypopharynx (tip of the epiglottis to the vocal cords) (Figure 4).

The pharynx has several functions that enter into competition with each other; it requires patency and closure.[1, 4] It serves the neurological (speech, taste, smell), but also gastro-intestinal and respiratory system (chewing, swallowing, breathing).Speech and swallowing

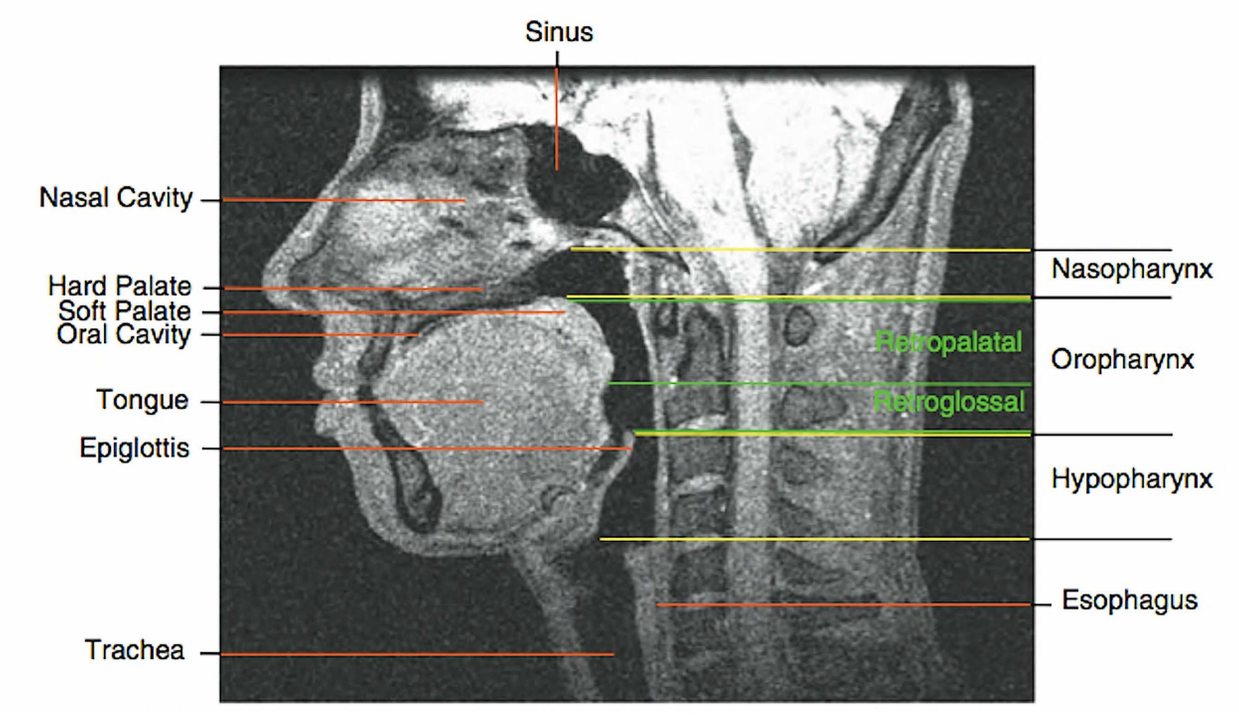

Figure 4. Sagittal magnetic resonance imaging of airway and division of oropharynx. (Clete A. Kushida)

require that the upper airway be collapsible. However, during breathing, the pharynx must remain patent.

Oropharynx and hypopharynx compose the collapsible portion of the pharynx. Due to the absence of bone and cartilage in these segments, their lumen patency, during awakening and sleep, depends heavily on muscle activity and intrinsic airway collapsibility, which is dictated by a combination of passive mechanical properties and active neural mechanisms.

During inspiration, negative intra-thoracic pressure is transmitted to the upper airways, resulting in a reduction in the transverse area of the pharynx. [25] The permeability of the upper airways is maintained through the balance between opposing forces from factors that collapse the airway and those that promote its patency. This is called "the balance of pressure concept" and involves the following determinants (Figure 5): [1,4, 26]

- The baseline pharyngeal area, determined by both craniofacial and soft tissue structures;
- The compliance or collapsibility of the airway;
- The negative intraluminal pressure within the airway (intraluminal pressure), transmitted from inspiratory muscles (the diaphragm, the external intercostal muscles....), that tends to narrow the airway;
- The pressure acting on the outside surface of the pharyngeal wall (tissue pressure), which also tends to collapse the airway such as compression by the lateral pharyngeal and submandibular fat pad and a large tongue confined to a small oral cavity;

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- The positive extra-luminal pressure from the abduction force of the pharyngeal dilator muscles, which is directed outwards, and functions to increase cross-sectional area.

Pharyngeal dilating muscles can be divided into four groups: [1, 4]

- Muscles influencing hyoid bone position such as geniohyoid and sternohyoid
- Muscles of tongue: Genioglossus is the largest and the most important muscle
- Muscles of the palate such as tensor palatini and levator palatini
- Muscles protruding the mandible, principally the pterygoid muscles

In normal individuals in awake state, the upper airway dimensions remain practically constant throughout inspiration by neuromotor mechanisms, like reflex muscle activation in response to stimuli such as sub-atmospheric pressure and hypercapnia. However, during sleep, neuromotor tone decreases and upper airway resistance increases considerably especially in sleep onset and REM stages. These physiologic variations are counteracted by a reduction of diaphragm and intercostal muscles activity and thus a decrease in inspiratory pressure. This tendency for the human upper airway to collapse predisposes it to abnormal deformation during sleep, mainly in susceptible individuals. [1,4, 27]

OSA results from a combination of structural upper airway narrowing and abnormal upper airway neuromotor tone. It is believed that the upper airways collapse more easily in OSA patients and occurs at slightly negative intra-thoracic pressures or even positive pressures. [27] Narrowing can occur in more than one site. The retropalatal or velopharyngeal region is the most common site; but the collapse usually extends to other locations. Since REM sleep is associated with greater muscle hypotonia compared to non-REM sleep, sleep-breathing disorder is more likely to occur during REM sleep. [13] In addition, the sleep-awake state in the pathogenesis of OSA is important to highlight. OSA patients, even with the most severe apnea, have generally no respiratory dysfunction during wakefulness through compensatory systems. [28]

According to recent studies on OSA pathophysiology, anatomical factors are not the whole story. The coordination between collapsing and dilating forces is an important concept and there is increasing evidence that the quantity and pattern of ventilation plays a substantial role in airway collapse [29] as well as the presence of upper airway neuropathology. [28] In addition, not all individuals with OSA have the same anatomical features. Thus, OSA patho-physiological factors are usually divided into three categories, whose complex interplay may explain the variable response to treatment:

1. Anatomic factors that effectively reduce airway caliber;
2. Non-anatomic factors that promote increased upper airway collapsibility and include: mechanical factors that are passive and related to tissues properties; and
3. neurological factors that change with the state of awakening or sleep

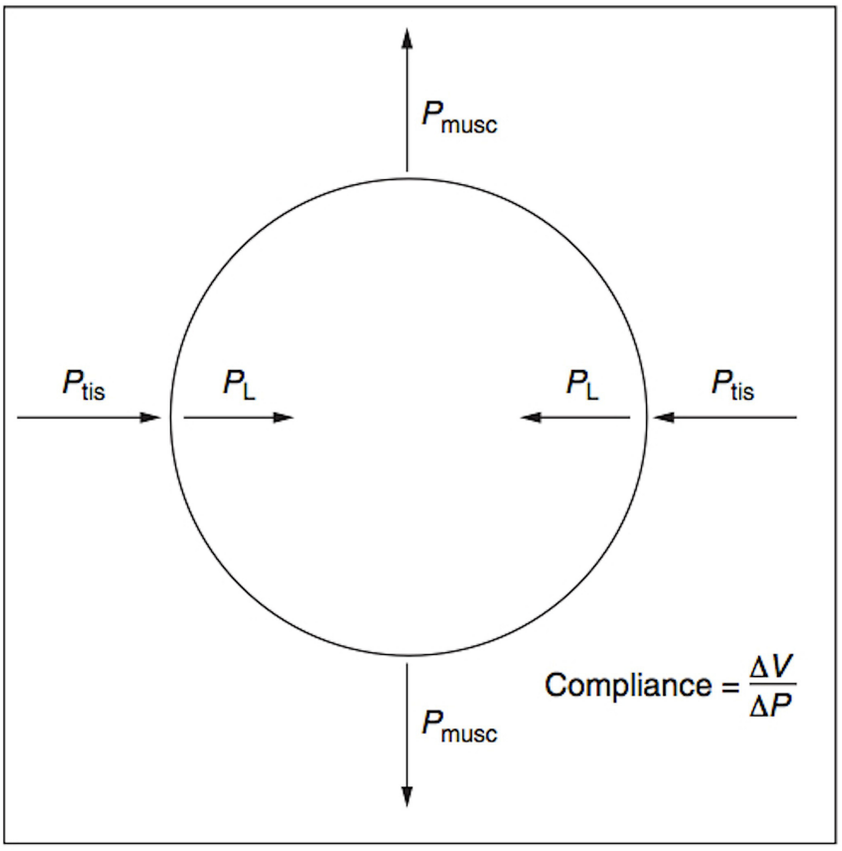

Figure 5. Determinants of upper airway caliber. PL = intraluminal pressure; Ptis = pressure in the tissues surrounding the pharyngeal wall; Pmusc = pressure exerted by the pharyngeal dilating muscles; V = change in volume; P = change in pressure. [1, 26]

3.2. Anatomic factors in OSA

There have been a number of studies comparing anatomic features of OSA patients and normal individuals. Upper airway imaging techniques such as cephalometry, acoustic reflection, nasopharyngoscopy, computed tomography and magnetic resonance imaging, have greatly improved the understanding of OSA biomechanical aspect, and guided treatment modalities.

Over the past several decades, many studies have demonstrated that patients with OSA have significant craniofacial and upper airway abnormalities when compared with age matched and sex matched controls. [17, 30]

Typical abnormalities include retroposition of the mandible and maxilla, shorter mandibular body length, longer anterior facial height, steeper and shorter anterior cranial base.... [1, 4, 13, 17]

However recent studies have shown no strong evidence for a direct causal relationship between sagittal and vertical craniofacial features and sleep-breathing disorder. In contrast, transverse width in the maxilla has a real impact with strong support for a narrow maxilla in OSA patients. [31]-[32] In addition, there is theoretical evidence that the size and the shape of the upper airway are also important and influence upper airway collapsibility.[4, 13] Imaging

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studies have shown reduced nasopharyngeal and oropharyngeal sagittal dimensions in OSA cases, associated with longer soft palate and longer airway. Indeed, the upper airway long axis of OSA patients is likely to be oriented transversely compared to the wide, elliptically shaped airway of normal controls.[33]-[35]

Lung volume is also reported to influence upper airway caliber and compliance.[13, 29] Decreased lung volume results in a caudal traction effect, which decreases the pharynx area and increases its resistance and its collapsibility due to a loss of tracheal tug.

Nasal airway pressure required to maintain airway patency is defined as the critical closing pressure (P_{crit}). [4] It has been demonstrated that P_{crit} is related to anatomical features and lung volumes, and shown to correlate with soft palate length in obese patients and airway length and hyoid-mandibular distance in non-obese patients [13]

On the other hand, the magnitude of extra luminal tissue pressure depends on the interaction of the upper airway soft tissues and the bony compartment size (Figure 6).[36] According to this model, soft tissues excess like in obesity, or restriction in bony compartment size such as retrognathia or both can lead to tissue pressure increase, thereby reducing airway caliber and predisposing to OSA. Soft tissues excess can be seen in case of tongue, soft palate and pharyngeal wall volume augmentation; but also in adenoids and tonsils lymphoid tissue hypertrophy.

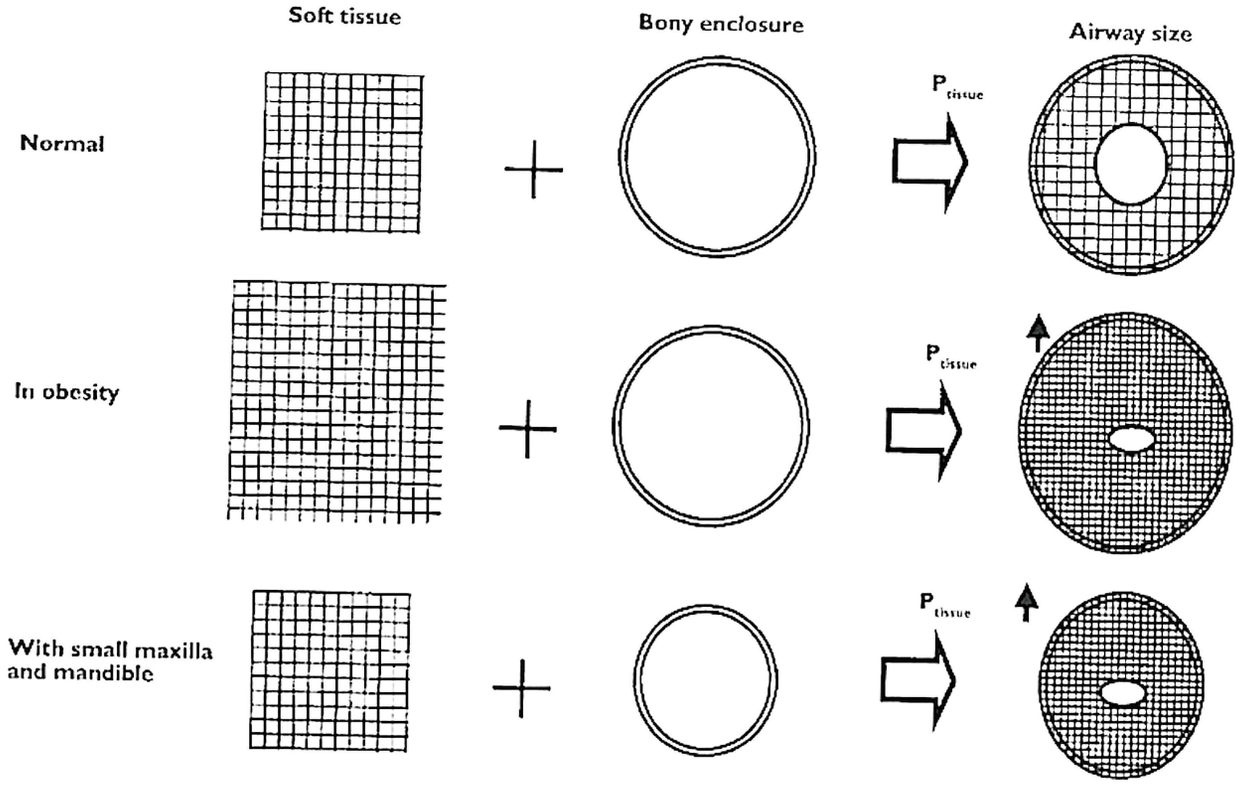

Figure 6. Figure 6: OSA pathophysiology: schematic explanation for anatomic factors interaction to regulate extraluminal tissues pressure (P_{tissue}) [36]

Despite the relationship between structural features and function, some patients with OSA do not have clear anatomic abnormalities. Evidence for a direct causal relationship between craniofacial structure and OSAs has yet to be elucidated because several methodological deficiencies in the literature and lack of research standardization methods and treatment success definitions have been highlighted.

3.3. Non-anatomic factors

This category includes all factors underlying collapsibility. They are divided into pure mechanic and neurologic factors. In OSA patients, airway dilation appears less coordinated than normal subjects and intrinsic mechanical properties of airway tissues are altered (Figure 7). [30, 37]

The respiratory control pattern generator responsible for automatic control ventilation is located into the brainstem. Respiratory rhythm is regulated by chemoreceptors and neural input from the upper airway and lungs to the brainstem neuronal network.[4] Instability of ventilatory control contributes to OSA pathophysiology by leading to periodic breathing and compromising airway patency during the ventilatory cycle. [28] It has also been suggested that upper airway inflammation and trauma caused by snoring and the hypoxia caused by intermittent upper airway collapse may impair the sensory pathways (upper airway mucosa) and the activation of neuromuscular reflexes (pharyngeal dilator muscles) rendering the upper airway prone to collapse. [38]

Other factors that may contribute to OSA pathophysiology include head posture, vascular supply to the mucosa and tissues surrounding the airway and arousal threshold.

Strohl et al. (2012) [39] reported that changes in blood pressure and/or pharyngeal muscles vascularity could affect airway stability and patency. Mucosal blood flow may either help resist distortion or contribute to narrowing if engorged.

On the other hand, flexion and extension of the neck affect the mechanics of the upper airway because the axis of rotation for extension and flexion is behind the airway. Thus, altered sleep position, mainly supine, may increase upper airway collapsibility and predispose to OSA particularly in adults because of tongue base prolapse. [40] In contrast, OSA children breathe better in the supine than in the prone position; this may be true because obstruction in children occurs usually at the level of the adenoids or soft palate rather than at the level of the tongue [1]

Although arousal is known to reinstate ventilation and thus to be protective in OSA, it is not essential to terminate an obstructive event. Low arousal threshold can exacerbate instability and worsen OSA.[1, 41] However, some authors who believe, that poor sleep is a secondary cause of OSA have rejected this claim. [29]

OSA has been shown to aggregate significantly within families. Genetic factors are likely to determine upper airway anatomy, neuromuscular activity and ventilatory control stability; these factors produce the phenotype of the OSA syndrome.[1, 4, 25, 42].

In sum, it is probably reliable to conclude that, in OSA individuals, there is a multiplicity of coexisting factors interacting to varying degrees at night; and everyone has biological sus-

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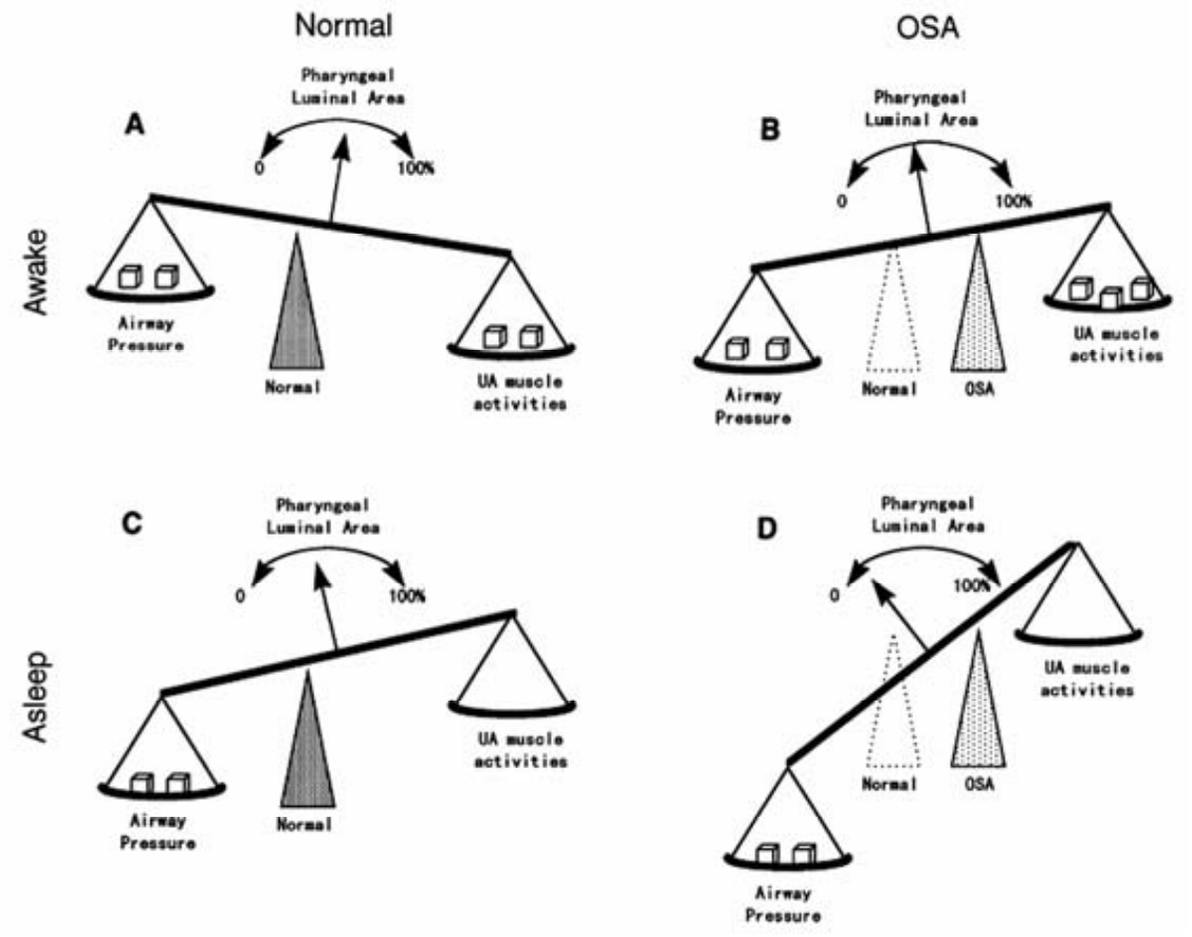

Figure 7. Schematic model proposed by Isono et al., 1997 [30] and explaining pharyngeal airway patency: When a wake, upper airway (UA) muscle activity compensates the depression forces exerted by the air, both in normal subjects (A) and the OSA (B) for which activity is most important. During sleep, activity decrease generates too much imbalance in the apneic and causes collapse (D). In panels B and D (subject with OSAS) the fulcrum that represents intrinsic properties of the pharynx, is to the right of the normal subject (A and C)

ceptibility and responds differently to environmental predisposing factors. Because OSA is a public health problem, its treatment should target the specific pathophysiologic processes that contribute to the collapse of the upper airway, in an attempt to alleviate symptoms and modify the long-term health consequences.

4. OSA diagnosis

4.1. Definitions

Aimed at maximal standardization and better care of patients, a task force of the American Academy of Sleep Medicine (AASM) has recommended terminology and standards of practice for recording sleep and breathing, and assigned evidence-based definitions for abnormal events, parameters and disorders. [43] These definitions are still valid today.

4.1.1. Respiratory events

Apnea is defined as cessation of airflow at the nose and mouth for 10 seconds or more with an arterial oxygen desaturation of 2% to 4%. Apnea is central, obstructive or mixed. The distinction between central and obstructive apnea is essential in determining the most appropriate treatment. During obstructive apnea, patients display respiratory effort without being able to ventilate because of upper airway obstruction, whereas central apnea occurs in the absence of ventilatory effort. Mixed apnea is initially started without ventilatory effort (as a 'central' pattern), and ends as obstructive with resumption of ventilatory efforts.

Hypopnea is defined as a decrease in airflow for 10 seconds or more with a concomitant drop in arterial oxygen saturation. AASM distinguish two situations of hypopnea events:

- A clear decrease (> 50%) from baseline in the amplitude of a valid measure of breathing during sleep;
- Or an amplitude reduction (< 50%) associated with either an oxyhemoglobin desaturation (> 3%) or an arousal. [4]

The exact magnitude of desaturation for a hypopnea varies in the literature. In routine clinical practice, it may not be necessary to differentiate apneas from hypopneas when both have similar pathophysiological consequences.[13] It is recommended to associate these two events in the form of an index of apnea / hypopneas (AHI).

4.1.1.1. Apnea and Hypopnea Indices (AHI)

This index, also termed respiratory disturbance index (RDI), refers to the total number of apnea and hypopnea episodes per hour of sleep. It is calculated by dividing the total number of apneas/hypopneas during a recording period by the total sleep time. AHI is usually employed to quantify OSA severity, but also to compare individual patient data with normative as well as pre-treatment and post-treatment values.

4.1.2. Obstructive sleep apnea syndrome

As noted previously, OSA is characterized by repeated partial or complete collapses of the upper airway during sleep, which precludes or reduces airway flow. It is associated with excessive daytime somnolence, sleep fragmentation and adverse sequelae attributable to frequent obstructive apneas or hypopneas during sleep. According to the AASM, OSA refers to an AHI ≥ 5 associated to one or both of these two criteria:

- Excessive daytime sleepiness (EDS) not explained by other factors
- Manifestation of at least two of following symptoms that should co-exist:
- Daily severe snoring
- Choking or suffocation during sleep
- Fragmented and non-restorative sleep

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- Diurnal tiredness
- Concentration difficulties
- Nocturia

However, the presence of 15 or more obstructive respiratory events per hour of sleep in the absence of sleep related symptoms is enough proof for the diagnosis of OSA due to the greater association of this severity of obstruction with important consequences such as increased cardiovascular disease risk.[44] Two indicators must be taken into account for severity estimation of OSA: AHI and the importance of diurnal hyper-somnolence after exclusion of another cause of sleepiness. Patients in normal sleep have an AHI of 5 or less. Patients with mild sleep apnea have an AHI of 5 to 15, with moderate sleep apnea typically 15 to 30 events and severe apnea 30 or more events per hour.

4.2. OSA clinical approach

Despite its high estimated prevalence, awareness of OSA remains insufficient in the community.[4] Health professionals, including orthodontists, should not disregard the risk factors of OSA and should detect and diagnose this disorder. OSA screening should be based on sleep-oriented history and physical examination in conjunction with objective tests. When diagnosed, OSA severity level must be determined for an effective treatment decision.[44]

4.2.1. Physical examination

According to the AASM, sleep history is sought to evaluate OSA symptoms and to determine patients who present high-risk levels. A sleep examination is directed at modifying the OSA probability based on the history, looking for associated or complicating disease, and excluding other potential causes for symptoms.

Clinical assessment must encompass all sleep and physical features of the patient that may provide helpful guidance for screening this condition such as:

4.2.1.1. Excessive Daytime Sleepiness (EDS)

EDS is caused by sleep fragmentation due to frequent arousals at night. It is still a very subjective symptom that overlaps significantly with other factors such as tiredness and lethargy. [4] Epidemiological studies estimate EDS prevalence at 8% to 30% in the general population. [45]Sleepiness may occur during "passive" conditions, such as watching television or, in severe forms, during "active" conditions, such as conversation or driving. Several instruments have been developed to measure EDS. Currently, the most useful instrument is the Epworth Sleepiness Scale.[46] This questionnaire provides sleep propensity measure and has good test–retest reliability. It should be described with regards to onset, situation, and chronicity of sleep problems (Figure 8). [45]Objective laboratory sleep tests, like multiple sleep latency test (MSLT) or maintenance of wakefulness test (MWT) are also used for EDS assessment, but their limits are principally related to their costs and duration.

Epworth Sleepiness Scale

Name: _____ Today's date: _____

Your age (Yrs): _____ Your sex (Male = M, Female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the **most appropriate number** for each situation:

0 = would **never** doze
1 = **slight chance** of dozing
2 = **moderate chance** of dozing
3 = **high chance** of dozing

It is important that you answer each question as best you can.

Situation	**Chance of Dozing (0-3)**
Sitting and reading _____	___
Watching TV _____	___
Sitting, inactive in a public place (e.g. a theatre or a meeting) _____	___
As a passenger in a car for an hour without a break _____	___
Lying down to rest in the afternoon when circumstances permit _____	___
Sitting and talking to someone _____	___
Sitting quietly after a lunch without alcohol _____	___
In a car, while stopped for a few minutes in the traffic _____	

THANK YOU FOR YOUR COOPERATION

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Figure 8. Figure 8. 1997 version of Epworth sleepiness scale. [47]. A score > 10 is consistent with EDS, and a score >16 indicates a high level of EDS.

4.2.1.2. Snoring and witnessed apneas with choking or gasping

The presence of snoring alone is a poor predictor of OSA. Thus, it must be correlated with other accompanying clinical features. Similarly, snoring absence does not exclude OSA. If severe, snoring can affect social relationship and become one of the main complaints of patients. Talking to the partner and family members can be very helpful; they can often report signs, such as apnea or falling asleep unintentionally (that the patient may be unaware of or deny). Therefore, patients can report awakening during choking episodes. But this is less common among females. OSA can also be associated with array of nocturnal and daytime symptoms that are not necessarily specific to this affection, but can complete its clinical pattern and give an idea about its impact on patients' functionalities. One can cite poor sleep quality, morning headaches, impaired memory, failed concentration, nocturia, and depression....[4]

4.2.1.3. Obesity

Obesity is the main predisposing factor for OSA. It is usually quantified by BMI (Body Mass Index). Increased BMI is closely correlated to OSA likelihood and severity. [4, 13] Additionally, central obesity (i.e. fat around the neck and waist), evaluated by neck circumference and hip-to-waist ratio, is simple clinical measurements that seem most predictive for SDB. There is no evidenced threshold value for these measurements, but a BMI ≥ 30 kg/m^2 and a neck circumference >17 inches in men and >16 inches in women are habitually used as critical values.[4] Moreover, a study found that waist-hip ratio is the most reliable correlate of OSA in both sexes; while neck circumference is an independent risk factor for males. [48]To establish OSA diagnosis, obesity indicators alone are not sufficient and further diagnostic testing is needed. [26]

4.2.1.4. Craniofacial examination

Clinical examination should include anatomical features of craniofacial and oropharyngeal structures as they can compromise airway patency. Particular attention should be paid to upper airway narrowing signs such as tonsillar hypertrophy especially in children, nasal obstruction, macroglossia with dental impressions at the edge of the tongue, elongated uvula or soft palate inflammation. [44, 45] Oropharyngeal crowding can be assessed using the modified Mallampati classification designed originally by anesthetists to grade intubation difficulty (Figure 9). [49]

Other conditions that should be searched for when examining potential OSA patients are skeletal abnormalities because they are high risk factors among either obese or non-obese individuals. Actually, retrognathia, micrognathia, maxilla deficiency with high arched/narrow hard palate, longer anterior facial height, cranial base abnormalities or inferior hyoid bone position should be evaluated as they may suggest the presence of OSA. Cephalometric radiographs enable health professionals to obtain quantitative measures of these features. [50]

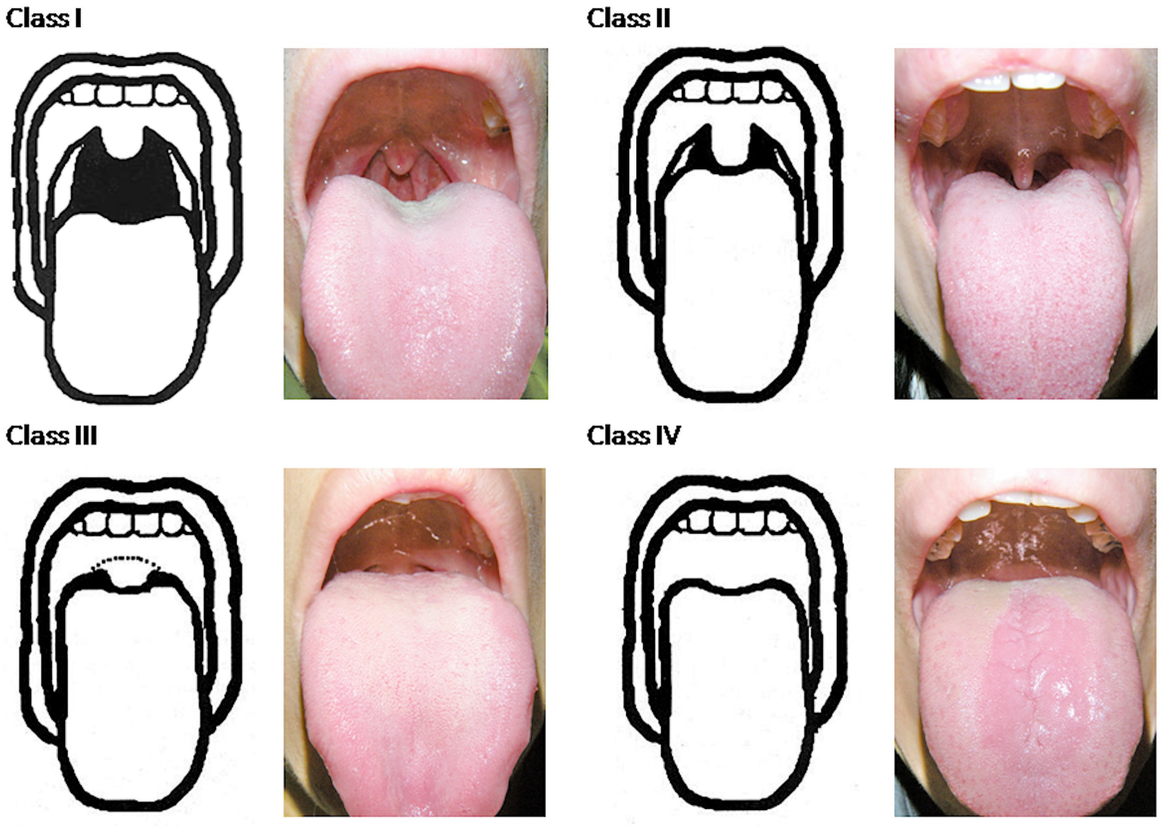

Figure 9. Modified Mallampati classification of oropharyngeal visualization. Class I: Soft palate, tonsils, pillars, and uvula, are clearly visible. Class II: Soft palate, pillars, and uvula are visible. Class III: only part of soft palate and base of uvula are visible. Class IV: Soft palate is not visible at all. 49

4.2.1.5. Associated comorbidities

A clinical examination should not ignore respiratory, cardiovascular, and neurologic systems. In this area, medication history must be taken into account especially with regard to drugs that are associated with OSA (Barbiturates, Benzodiazepines...), those that sedate and/or decrease respiratory drive (Antihistamines, Antispasmodics, Anxiolytics, Muscle relaxants...) and those that impair sleep onset or maintenance (Anticholesterol agents, Appetite suppressants, Benzodiazepines, Caffeine, Nicotine, Diuretics...). Furthermore, since hypertension is described as independently associated with OSA, blood pressure has been integrated into several clinical prediction rules for sleep apnea. [22, 44]

4.2.2. Objective testing

To establish OSA severity, objective testing is required. There are two accepted methods: laboratory polysomnography (PSG) and home testing with portable monitors (PM)

Polysomnography is the golden standard method for diagnosing OSA. It records sleep-breathing pattern and oxygen saturation overnight via a minimum of 12 channels of physiological signal such as electroencephalogram, electrocardiogram, electromyogram, oronasal airflow, electrooculogram, respiratory effort, body position and oxygen saturation. This

examination provides AHI by monitoring apnea and hypopnea occurrence. Clinical interpretation of OSA severity is based, in addition to AHI, on factors like oxygen desaturation and sleep fragmentation degrees. In general, a single night PSG is sufficient to make an appropriate OSA diagnosis. However, some variability can be identified in recordings between the first and the second night of a PSG, a phenomenon known as the "first night effect". This may be due to factors such as sleep position and alcohol [44, 51]

Unlike PSG that is expensive and labor intensive, PM is performed at home and thus offers greater convenience for patients. Nonetheless, this procedure has some limits related to the lack of supervision, which can affect its reliability, but also to the impossibility to detect other sleep disorders such central apnea or nocturnal epilepsy. The choice between PSG and PM should take into consideration resource limitations and pre-test clinical evaluation. Thus, PSG could be performed if PM is technically inadequate or fails to establish OSA patients with a high pre-test probability.[44]

Furthermore, numerous imaging modalities are available for 2D or 3D craniofacial and airway study. They have potential usefulness in understanding the pathogenesis of sleep- breathing disorder, and planning of treatment (adenoidectomy, orthognathic surgery), but their routine use in the evaluation and diagnosis of OSA is limited. All diagnosis components previously studied (clinical examination and diagnostic testing) should be discussed with patients to establish a program including risk factors, consequences, but also treatment options/outcomes of OSA in the context of disease severity and patients' expectations.[44]

5. Treatment of OSA syndrome

Therapeutic approach of OSA requires interdisciplinary communication among healthcare professionals and long-term management with a regular follow-up. Patient adherence to therapy, potential side effects and further stability of results must be continually monitored. On the other hand, outcomes assessment should be performed after all therapy has been undertaken. The criteria used to determine successful treatment of OSA varies widely. A task force of AASM have reported some indicators for assessment of treatment results; these include resolution of sleepiness, OSA specific quality of life measures, patient and spousal satisfaction, adherence to therapy, avoidance of factors worsening disease, obtaining an adequate amount of sleep, practicing proper sleep hygiene and weight loss for overweight/obese patients. Objectively, clinicians strive to achieve at least 50% reduction in the baseline AHI in addition to reduction in AHI to <5 events per hour or <10 events per hour. However, less stringent definitions can be adopted. Treatment modalities of OSA can be divided into surgical and non-surgical treatment to which adjunctive therapies can be associated. Less invasive treatment should be selected whenever possible. Also, patients must be advised about surgical success rates and complications, the availability of alternative options and their levels of effectiveness. [52, 53]

5.1. Non-surgical treatments of OSA

This category includes continuous positive airway pressure (CPAP), behavior modifications, and oral appliances.

5.1.1. Continuous positive airway pressure (Figure 10)

First described by Sullivan in 1981, CPAP was to become the golden standard of moderate to severe OSA treatment. [54].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 applied through oral, nasal or oro-nasal interface; and the optimal level of positive airway pressure is determined by full-night, attended in-laboratory PSG. Successful therapy with CPAP depends greatly on individual patient acceptance and compliance that can fall for numerous reasons including functioning noise, discomfort, feelings of claustrophobia, and skin irritation. Thus, CPAP prescription requires explanation of benefits and medical reasons for its use. Patients should also be informed about the function and maintenance of equipment. According to the American college of Physician (ACP), moderate quality evidence has showed that CPAP improves sleep measurement in patients with at least moderate OSA (AHI > 15events/h), and there are no data to determine which patients benefit most from specific treatment strategies. [55] However, OSA remains at present the preferred treatment for OSA, as it could effectively reduce AHI and arousal index scores, and increase the minimum oxygen saturation. Finally, if CPAP use fails, based on objective monitoring and symptom evaluation, more efforts should be implemented to improve PAP use or consider alternative therapies.

Figure 10. CPAP device requiring the use of mask interface, sealed tubing and flow generator providing airflow. [56]

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5.1.2. Behavior modifications or conservative treatments

Behavior strategy includes all practices that enhance life routines and hygiene. It involves weight loss (ideally to a BMI of 25 kg/m2 or less), positional therapy, and avoidance of smoking, alcohol and sedatives 3h before sleep. Weight loss has been shown to improve AHI in obese patients with OSA. It is recommended for all overweight OSA patients and should be combined with a primary treatment for OSA. Sleeping in the supine position can affect airway size and patency with a decrease in the area of the lateral dimension of upper airway. Positional therapy keeps the patient in a non-supine position by positioning device like alarm, pillow, back-pack or tennis ball is an effective secondary therapy or can be a supplement to primary therapies for OSA in patients who have a low AHI in the non-supine position. To ascertain treatment outcomes, indicators, such as self-reported compliance, objective position monitoring, are used. However, studies argue that CPAP is still superior to positional therapy in reducing the severity of sleep apnea and increasing the oxygen saturation level during sleep in patients with positional OSA. [50]-[57]

5.1.3. Oral appliances (figure 11-13)

Pierre Robin was the first orthodontist to have used oral appliances (OAs) in the 1900s for glossoptosis. Since the 80s, these oral devices were used as a non-invasive treatment for OSA. This therapy has proven to be effective in reducing the apnea and hypopnea index, improving oxygen saturation during sleep, and reducing snoring. OAs are recommended as an alternative therapy to CPAP for mild to moderate OSA patients with CPAP adverse effects or for those who do not tolerate or adhere to CPAP or those who refuse surgery. They are also appropriate for patients with primary snoring, who do not respond to treatment with behavioral measures such as weight loss or sleep position change. [44, 50]-[58]

Both Mandibular advancement devices (MADs) and tongue-retaining devices were described (TRD). But MADs are the most commonly used and evaluated in the literature. Orthodontists must indicate the most appropriate design of MADs for each patient, depending on dental history and complete examination of the stomatognathic system (soft tissues, dental occlusion, masticatory muscles and the temporomandibular joint). MADs cover the upper and lower teeth and hold the mandible in an advanced position with respect to the resting position. The appliance is constructed, adjusted, and gradually titrated (advanced forward) over several weeks until the snoring and daytime sleepiness are reduced to an acceptable level, or the patient cannot tolerate further advancement. They are worn during sleep and they act by enlarging obstructed upper airway by moving the mandible and tongue anteriorly and then the activation of airway dilator muscles. Craniofacial changes induced by OA were evaluated using cephalometric analysis. Significant modifications were reported: Retroclination of the maxillary incisors, proclination of the mandibular incisors, increased lower facial height, and changes in molar relationship. Loss of edema, caused by snoring and repetitive apneas, associating OAs seems to result in palatal length decrease and pharyngeal area increase. OAs have some side effects: Dry mouth, excessive salivation, jaw discomfort, myofacial pain and tooth grinding. However, they are frequently reported as mild, acceptable, and transient.

Another inconvenience of OAs is the time needed for titration, which makes it a second choice for severe or high symptomatic OSA treatment. [58]

The Academy of Dental Sleep Medicine suggested the use of cephalograms as a diagnostic aid at the initial dental examination of every patient receiving OA treatment. In addition, some cephalometric predictors like longer maxilla, shorter soft palate and decreased distance between mandibular plane and hyoid bone have been related to successful MAD treatment of OSA. [4]

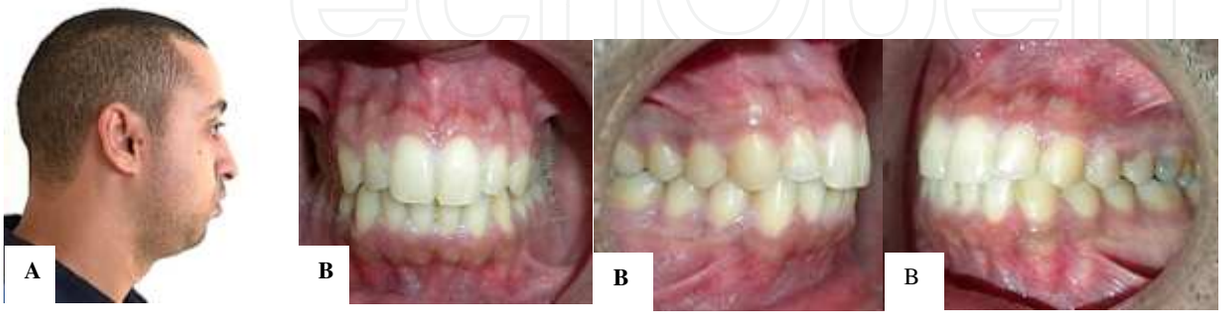

Figure 11. A 27-year-old man with mild OSA: initial profile view (A), and initial occlusal views (B). We can note a severe retrognathia compensated with a class I dental occlusion.

Figure 12. A MAD device was indicated for night.

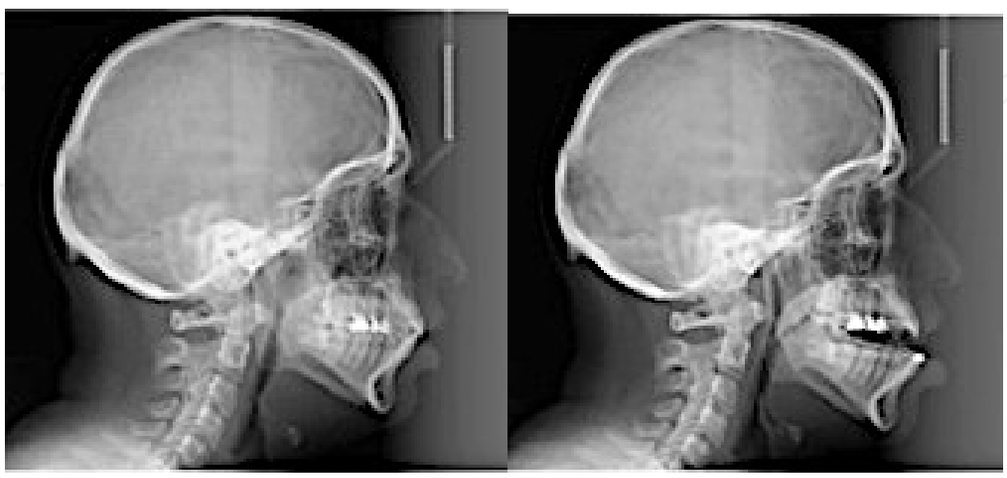

Figure 13. Lateral Cephalograms before (left) and after (right) oral appliance positioning showing change in hyoid bone position and slight enlargement of retroglossal area of pharynx

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5.2. Surgical treatments of OSA

Surgical management was the first therapeutic modality employed to treat SDB by placement of a tracheotomy tube to bypass upper airway obstruction in Pickwickian patients. Currently, there are numerous surgical approaches to upper airway treatment in OSA, which consist of upper airway tissue reduction or reconstruction at different levels. OSA surgical management often involves several procedures that can be at times multi-phased or a combination of multi-level simultaneous surgeries. The selection of the most adequate surgery entails a meticulous preoperative multidisciplinary assessment and rests on the surgeon's experience. [59]

OSA surgery should be determined after clinical diagnosis and severity assessment by objective testing. It is recommended for patients who are medically and psychologically able to tolerate the operation ; primary surgery is advocated in mild OSA and severe obstructing anatomy feasible to treat surgically such as tonsillar hypertrophy and nasal obstruction; surgery is recommended secondarily in cases of ineffective treatment or intolerance to the other non-invasive therapies in mild, moderate and severe OSA. Surgical treatment involves evaluation of three anatomic sections of the airway for detection of collapse-related abnormalities namely:

1. the nose (alar cartilage deformities, septal deviations, enlarged turbinates, nasal floor constriction),
2. the retropalatal area (lymphoid hyperplasia, retrusive maxilla, long palate) and
3. the retroglossal area and the tongue (mandibular retrognathia).

Thus, surgical procedures can be classified as intra-pharyngeal or skeletal. Intra-pharyngeal surgery includes all procedures directed towards soft tissues of upper airway, the most common being uvulopalatopharyngoplasty (UPPP). Hard tissues surgery includes maxillo-mandibular advancement (MMA) (Figures 14-16) and genioglossus advancement (GGA) (Figure 17)

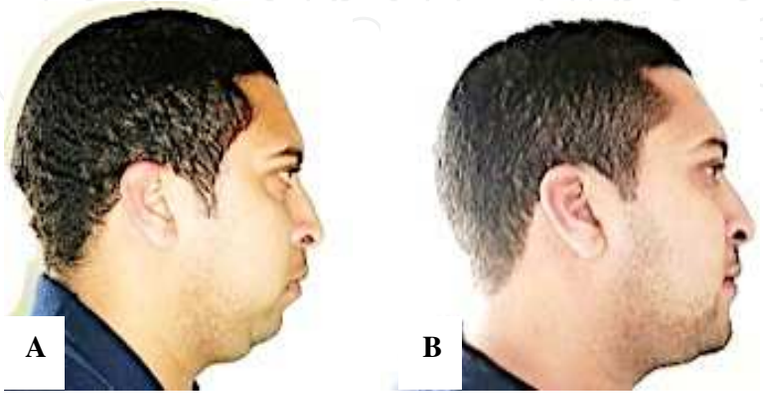

Figure 14. Profile views of a 34-year-old man with severe OSA. A: before treatment, B: after OSA management including orthognathic surgery (mandibular advancement osteotomy)

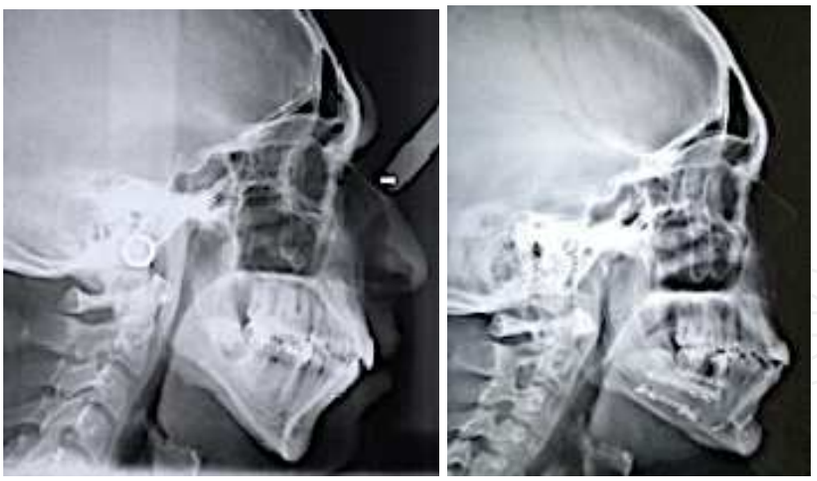

Figure 15. lateral cephalograms showing posterior airway space enlargement Before treatment (left) and after surgical mandibular advancement (right)

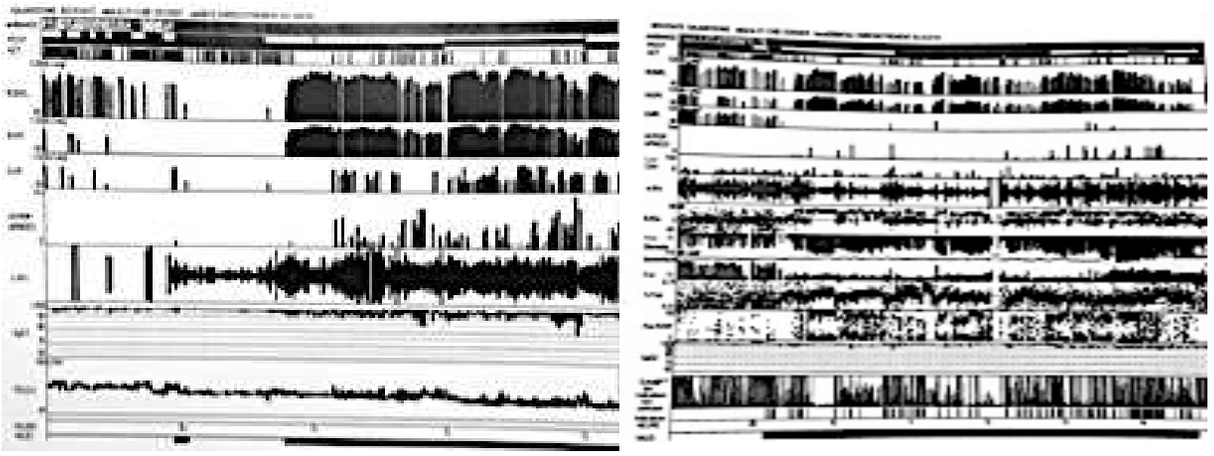

Figure 16. PSG registration: before treatment (left) and after mandibular advancement and adenoidectomy (right).

5.2.1. Sleep parameters evaluation for this clinical case

Before treatment: total number of obstructive apnea events: 51, number of total hypopnea events: 30, AHI: 30/h, desaturation index: 27/h. BMI: 24 Kg/m^2 (Severe OSA).

After treatment: total number of obstructive apnea events: 22, total number of hypopnea events: 108, AHI: 22, desaturation index: 2/h, BMI: 25 Kg/m^2. Moderate OSA.

Powell et al. have created a two-phase directed protocol (Powell-Riley surgical protocol) for surgical treatment of upper airway obstruction at several levels in order to avoid unnecessary surgery. Phase I surgery is designed essentially to treat the upper airway soft tissue (nose, palate, and tongue base) without dental occlusion or facial skeleton modifications. Clinical response is assessed, after adequate healing, four to six months following surgery by PSG. Persistent OSA requires phase II surgery indications. Phase II surgery refers to maxilla-

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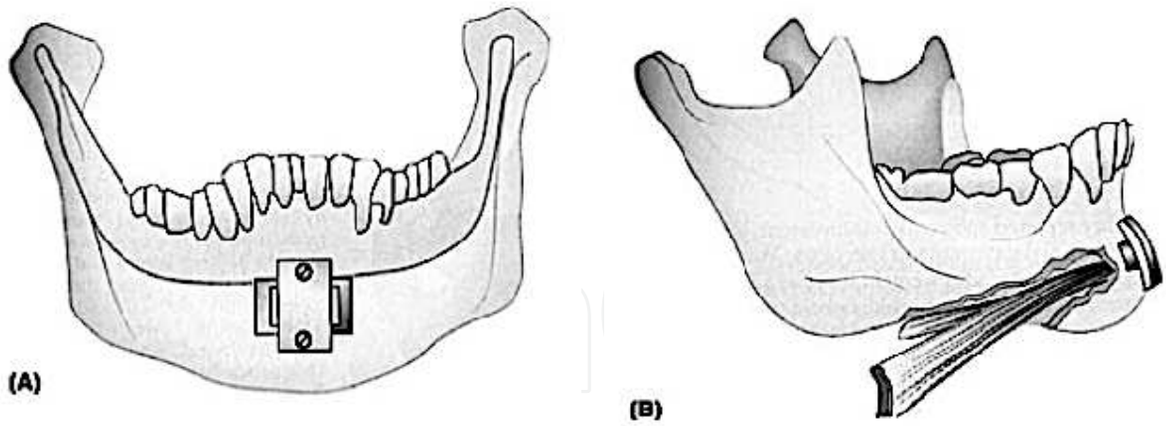

Figure 17. Genioglossus advancement technique. (A): rectangular osteotomy is created in the anterior mandible. (B) : The genial tubercle and the attached genioglossus muscle are advanced anteriorly. The bony fragment is rotated 90° to overlap the inferior border of the mandible and secured to the mandible with a titanium screw. [59]

mandibular advancement osteotomy, which physically creates more space for the tongue, thus enlarging the posterior airway space. [59]

UPPP has been developed to alleviate isolated obstructing tissues of the soft palate, lateral pharyngeal walls, and tonsils. However, according to ACP, it does not reliably normalize AHI when treating moderate to severe OSA, as a sole procedure. Furthermore, with regards to MMA, there is a need for more understanding of the relative risks and benefits of MMA compared to other treatment modalities. CPAP or OAs should generally be suggested ahead of MMA if the patient is consenting. These recommendations do not corroborate with other findings having reported a success rate of 89% obtained by physically expanding the facial skeletal framework and increasing tissue tension, which decreases velopharyngeal and suprahyoid musculature collapsibility. [53, 61]

Complications of maxilla-mandibular advancement surgery have been reported, including side effects such as neurosensory deficit, infection, bleeding, or temporomandibular joint problems; but patients' satisfaction is reported to be as high as 95%. Finally, long-term stability depends on the body mass index, the amount of skeletal advancement, and the skill and experience of the surgeon.[60, 61]

The palatal implant is a new treatment option for snoring that emerged in 2003. It is composed of polyethylene terephthalate, a biocompatible material, and inserted into the soft palate to reduce vibration and collapsibility by stiffening the soft palate, thus reducing palatal flutter and snoring. Additional stiffening of the palate is achieved by fibrosis and formation of capsule in response to the inflammatory reaction. Studies have showed that they may be effective in some patients with mild obstructive sleep apnea, who cannot tolerate or do not adhere to positive airway pressure therapy, or in whom oral appliances have been considered and found to be ineffective or undesirable. However, at the present time, it is difficult to predict if it will be a reliably effective intervention or not. [55, 59, 62]

5.3. Adjunctive treatment

5.3.1. Pharmacological therapy

A wide range of medication targeting OSA treatment has been explored in the literature. Except for hypothyroidism or acromegaly in which medication can improve AHI, there are no really effective pharmacotherapies for OSA. Topical nasal corticosteroids can be used in patients with OSA and concomitant rhinitis especially in children, and thus may be a useful adjunct to primary therapies for OSA. In addition, Modafinil, a psychostimulant, is recommended for the treatment of residual excessive daytime sleepiness despite effective PAP treatment and absence of other evident causes for their sleepiness. [44, 63]

A Cochrane review issued in 2013 showed insufficient evidence to recommend any systemic pharmacological treatment for OSA; drug therapy needs to be targeted depending on the presence or absence of obesity and the predominance of OSA in a particular sleep stage. The review also reported that among all drugs evaluated, Donepezil is the most promising for further research. [64]

5.3.2. Bariatric surgery

Bariatric surgery consists of a variety of operative techniques performed to promote weight reduction such reducing gastric banding, gastric and jujenoileal bypass or gastroplasty. It is often recommended for treatment of morbid obesity, particularly when associated with other medical complications (BMI ≥ 35 kg/m2) or those with a BMI ≥ 35 kg/m2 when dietary efforts fail at weight control. Therefore women seem likely to be candidates for this method of weight loss. [44, 65]

6. Obstructive sleep apnea syndrome in orthodontic practice

As cited above, OSA is associated with numerous craniofacial abnormalities. Orthodontics improvement of dento-facial morphology may have a positive impact on OSA components. Orthodontic professionals should provide treatment for OSA patients as well as diagnose potential OSA patients. Medical history and clinical examination allows orthodontists to identify the risk factors of OSA or signs related to OSA (obesity, allergy, nasal dysfunction, maxillary constriction, retrognathia, long uvula, mouth breathing...) or record some symptoms reported by patients. Moreover, several imaging modalities (lateral and frontal cephalogram, cone beam computed tomography, MRI.) can assist Orthodontic professionals in assessment of this condition.

Orthodontic management of OSA syndrome could be provided to children as a preventive and interceptive modal or to adults by an interdisciplinary management. A significant number of children suffering from respiratory problems and obstructive sleep apnea have nasal obstruction associated with a narrow maxilla that may increase nasal resistance and alter the tongue posture, leading to a narrowing of the retroglossal airway and OSA. Maxillary

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expansion with orthopedic appliances is very effective in these cases allowing for an increase of nasal cavity dimension. It can be combined with adenotonsillectomy for best results in children with OSA associated with adenotonsillar hypertrophy. [4, 66, 67] Among adults, this expansion can be attained by RME or surgically assisted RME and has been reported to reduce snoring and hyper-somnolence.

Maxillomandibular advancement can also be provided either by surgery or orthopedic systems as therapeutic or preventive measure in OSA cases. A good finishing of dental occlusion is desirable. On the other hand, It has been suggested [68] that the improvement observed in the respiratory symptoms with surgical MMA, namely apnea/hypopnea episodes, should be correlated with SNA increase after surgery which may help maxillofacial surgeons establish selective criteria for the surgical approach to sleep apnea syndrome patients. Mandibular advancement in case of retrognathia can be accomplished by oral appliances in adulthood, functional appliance therapy in younger patients, mandibular distraction osteogenesis or osteotomies, and is among the most frequently used approaches in OSA management.

Orthodontists can also have a role in the treatment of OSA consequences especially those with nocturnal bruxism, which differs from stress-related bruxism. Sleep bruxism has been shown to be prevalent in children, and correlated with sleep disturbances (microarousals). It is characterized by rhythmic masticatory muscle activity and may be related to the patients' attempt to improve airway patency during episodes of oxygen desaturation via co-activation of jaw opening and closing muscles. Its management requires use of night splints and restorative dentistry.

In brief, although the bi-directional cause and effect relationship between OSA and craniofacial abnormalities remains to be proven, early identification and treatment of dentofacial disorders may enhance OSA management with respect to preventive and curative approaches. Interdisciplinary professional communication is crucial for the success of global OSA management.

7. Conclusion

OSA is a common breathing disorder, which affects all age groups. It is a serious public health problem. Because of its potential pathophysiological consequences, it associates alteration of quality of life, decreased economic potential and increased morbidity and mortality in affected patients. Assessment of OSA requires a thorough clinical examination as well as overnight testing to determine PSA presence and severity before initiating treatment. Polysomnography remains the most common and reliable test for OSA diagnosis. Additionally, several imaging modalities can be used for upper airway structure and function during wakefulness and sleep. Treatment modalities of OSA are aimed at increasing life expectancy, decreasing disease problems and improving the quality of life. CPAP is still the mainstay for treatment of moderate to severe OSA. However, medical or surgical alternatives can be used in case of failure or non-compliance of the patients.

OSA is also a condition that orthodontists may encounter in their daily practice; thus, they are in a better position to diagnose and treat it using a multidisciplinary approach and management.

Author details

Hakima Aghoutan[1*], Sana Alami[1], Samir Diouny[2] and Farid Bourzgui[1]

*Address all correspondence to: hakimadental@yahoo.fr

1 Department of Dento-facial Orthopedics, Faculty of Dental Medicine. Hassan II Ain Chok University. Casablanca, Morocco

2 Chouaib Doukkali University, Faculty of Letters and Human Sciences, El Jadida, Morocco

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