ACTA SCIENTIFIC DENTAL SCIENCES

Volume 9 Issue 1 January 2025

Review Article

Risk in Sleep Apnea Hypopnea Syndrome

Bibiana Fabado Martínez, Otto Aleman Miranda*, Marta Belén Cabo Pastor, Maria Celia Haya Fernández and Cristina Ribes Vallés

Private Practice, USA

***Corresponding Author:** Otto Alemán Miranda, Private Practice, USA.

Received: November 18, 2024

Published: December 19, 2024

Abstract

An exhaustive bibliographic review was carried out on the main risks and complications of sleep apnea and hypopnea syndrome, highlighting the multiple damages that this condition can cause to affected individuals.

Keywords: Sleep Apnea; Hypopnea; Syndrome; Dyspnea

Introduction

Sleep medicine, as a regulated specialty, was born at the beginning of the 20th century, specifically in the 1930s, marked by technological growth that enabled a more objective approach to the study of sleep (Velayos, 2009). Finding a scientific definition of sleep is not an easy task. Each author presents his particular vision to concisely describe the intricacies of this phenomenon.

Mediano., *et al*. in 2021, describe it as "a physiological process and biological need that is characterized by a transient, periodic, and reversible decrease in consciousness." With less possibility of reacting to the environmental stimuli that surround human beings. Sleep occupies a third of human life and the functions it performs are essential for health and daily performance (Abrams, 2015; Heslop., *et al*. 2002).

During sleep, processes such as memory consolidation, metabolic and endocrine regulation, immune activation and elimination of toxins occur (Abrams, 2015) Insufficient sleep can cause or exacerbate cardiovascular diseases (Heslop., *et al*. 2015), type 2 diabetes mellitus, metabolic syndrome (Cappuccio., *et al*. 2010), psychiatric diseases (Clement-Carbonell., *et al*. 2021; Glozier., *et al*. 2010) and cancer (Haus., *et al*. 2013) [1-3].

Objective

To describe the main risks and complications of SLEEP APNEA HYPOPNEA SYNDROME.

Literature search methods Scientific information was collected through a search using the following descriptors in English: The Medical Subject Headings (MeSH): "SLEEP APNEA HYPOPNEA SYNDROME, risk in APNEA.

Analysis strategy

The search was based solely on SLEEP APNEA HYPOPNEA SYNDROME

In development.

The National Sleep Foundation states that the hours of sleep required by humans decrease as age increases (Table 1).

EDAD	HORAS DE SUEÑO RECOMENDADAS
Newborns (0-3 months)	14 - 17 hours
Infants (4-11 months)	12 - 15 hours
Toddlers (1-2 years)	11 - 14 hours
Pre-schoolers (3-5 years)	10 - 13 hours
Schoolchildren (6-13 years)	9 - 11 hours
Adolescents (14-17 years)	8 - 10 hours
Young adults (18-25 years)	7 - 9 hours
Middle-aged adults (26-64 years)	7 - 8 hours
Elderly (≥65 years)	7 - 8 hours

Table 1: Hours of sleep by age.

International classification of sleep disorders

Sleep disorders are a very large and heterogeneous group of processes. There are many diseases that have a sleep disorder as one or more of their symptoms. In fact, it is difficult to find any disease that does not alter nighttime sleep or the tendency to sleep during the day. Since sleep medicine burst onto the clinical scene, various official classifications have been proposed for sleep disorders (Gallego Pérez-Larraya., *et al*. 2007). The first classification of sleep disorders was based on core developments based on the main symptom. This first approach advanced towards a greater importance of the disease to the detriment of the symptom as a classification basis. The most up-to-date classifications recover the initial trend that is once again based on symptoms. The first classification was developed in 1979 by the Association of Sleep Disorders Centers (ASCD). Of all the classifications that have been developed over the years, it is worth highlighting the International Classification of Sleep Disorders (ICSD-2) of 2005, where seven large groups are proposed: insomnia, respiratory disorders, hypersomnias, circadian rhythm disorders, abnormal movements, isolated symptoms, normal variants and parasomnias that group the eighty different entities defined [1-3].

The most recent classification was published by the American Academy of Sleep Medicine in 2014 under the direction of Sateia MJ. The third edition of the International Classification of Sleep Disorders (ICSD) includes 7 categories of sleep disorders (Table 2).

International Classification of Sleep Disorders-Third Edition
Insomnia
Sleep-related breathing disorders
Central hypersomnias
Circadian sleep-wake rhythm disorders
Parasomnias
Sleep-related abnormal movements
Other sleep disorders

Table 2: International Classification of Sleep Disorders, 2014.

The third edition of the ICSD also includes diagnostic criteria for obstructive sleep apnea in adults, which are as follows (A and B or C to meet the criteria): A. The presence of one or more of the following:

- The patient complains of drowsiness, unrefreshing sleep, fatigue, or symptoms of insomnia.
- The patient awakens holding his or her breath, gasping, or choking.
- The bed partner or other observer reports snoring, interruptions in breathing, or both during the patient's sleep.
- The patient has been diagnosed with hypertension, mood disorder, cognitive dysfunction, coronary artery disease, cerebrovascular disease, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus [1-3].

B. Polysomnography (PSG) or home sleep test (OCST) demonstrates: 1. Five or more obstructive respiratory events (obstructive or mixed apneas, hypopneas, and effort-related arousals) per hour of sleep during a PSG or per hour on monitoring outside a sleep center. 3. Sleep-related breathing disorders. Sleep-related breathing disorders are characterized by altered breathing during the normal development of the sleep pattern [4-6].

Due to continuous interruptions, patients report excessive daytime sleepiness. Thus, disorders in which respiratory movement is diminished or absent in an intermittent or cyclical manner are linked to serious cardiovascular consequences due to lack of oxygen (Gallego., *et al*. 2007). All respiratory disorders have consequences for both the patient and their family if they are not treated (Battagel., *et al*. 2005).

Currently, the most important respiratory disorders are included within what many authors have called "Obstructive Respiratory Disorders during Sleep" (ORDS) which encompass the variety of pathological manifestations from simple snoring to the most severe cases of Sleep Apnea Hypopnea Syndrome (SAHS). Among the most prominent sleep respiratory disorders are: primary snoring (PR), increased upper airway resistance syndrome (AURRS) and sleep apnea hypopnea syndrome (SAHS) (Fernández Julián., *et al*. 2002). ORDs can be conceptually defined as a clinical development that encompasses primary snoring, relatively harmless, where the resistance to the passage of airflow in the upper airways (UAV) is scarce; increased upper airway resistance syndrome (AURRS) defined as an intermediate state; and sleep apnea-hypopnea syndrome (SAHS), where the obstruction is complete (apnea) or partial (hypopnea) and results in gasometric alterations (hypoxemia and hypercapnia) and nocturnal and diurnal symptoms, with substantial health compromise (Martínez Font, 2018) [5,6].

3.1 Primary or simple snoring. The International Classification of Sleep Disorders (ICSD 780.53.1) manual defines primary or simple snoring as the presence of severe respiratory sounds produced in the upper airways during sleep, without episodes of apnea, hypoventilation, desaturations or related arousals or evidence of insomnia or hypersomnia related to snoring. RP is not in any case a physiological event. Its presence reveals a variable degree of

obstruction of the upper airways, an anatomical variation that in turn will generate turbulence in the air column and the vibration of anatomical structures, originating the sound that is identified as snoring, which in turn can have multiple variations in terms of tone and intensity (Santamaría., *et al*. 2014). Riley and Powell, in 2000, classified snoring into

- **Simple or benign snoring:** when, in addition to not bothering companions, it is not accompanied by objective evidence of upper airway resistance. - Habitual loud snoring or social snoring: when it bothers the companion and/or people who live in the house, but without upper airway resistance. This type of snoring is also usually considered simple [5-7].
- **Snoring accompanied by SAHS:** Although its prevalence in the general population is difficult to estimate, it may occur in more than 50% of adult individuals, being more frequent in men than in women (Santamaría., *et al*. 2014). The prevalence of snoring increases with age and body mass index (BMI), although some authors consider its relationship with neck diameter to be more specific. Sleeping position is another relevant variable: snoring increases in supine position, and is attenuated in lateral decubitus, due to the effect of gravity on the soft parts of the upper airways. This problem increases with the consumption of muscle relaxants, benzodiazepines and alcohol, by producing hypotonia in the oropharyngeal muscles. Snoring is also associated with certain pathologies, such as gastroesophageal reflux and hypothyroidism (Friedman, 2009; Flemons, 2002), habits such as smoking and physiological conditions such as pregnancy (O'Brien., *et al*. 2013) [7-9].

During the last decade, a series of investigations have been published that support a new conception of RP, according to which this entity does not have the classically described harmless character, but on the contrary, it causes a significant deterioration of the cognitive and academic functions of those who suffer from it, also generating behavioral alterations and even metabolic disorders, especially in the pediatric population. (Bourke., *et al*. 2011; Biggs., *et al*. 2011). 3.2 Increased upper airway resistance syndrome (RAVAS) [1-3].

This concept was introduced by Guilleminault and refers to the increase in resistance of the upper airways without a number of apneas-hypopneas or a decrease in oxygen saturation sufficient for the diagnosis of SAHS, but with significant daytime sleepiness due to the fragmentation of sleep by repeated micro-awakenings in relation to the inspiratory efforts necessary to not give up and keep the airway open (Guilleminault, 1993). This syndrome also causes neurocognitive alterations, attention deficit and hyperactivity (Capote., *et al*. 2002). One of the greatest inconveniences for its diagnosis is that the symptoms of SAHS are similar to those of SAHS and the symptoms that occur cause overlapping of the diagnosis (Fernández Julián., *et al*. 2002b). 3.3 Sleep apnea-hypopnea syndrome (SAHS) [8-10].

This syndrome is characterized by repeated obstructions of the upper airway that prevent the normal flow of air to the lungs. Patients with SAHS, in a single night, repeatedly stop breathing while sleeping, which reduces the flow of oxygen to vital organs (hypoxia), with serious health risks. Blockages can be complete (apneas) characterized by interrupting the flow of air for a time equal to or greater than 10 seconds or partial (hypopneas) with a reduction of approximately 50% of the air. They are followed by sudden respiratory efforts that resume breathing in the form of loud snoring and cause electroencephalographic microarousals of which the patient becomes unaware (Duarte., *et al*. 2012) [10-12].

The clinical picture of breathing pauses, snoring and microawakenings is completed with oxyhemoglobin desaturation and daytime hypersomnolence as a consequence of fragmented and non-restorative sleep (Zamarrón Sanz., *et al*. 2001). The most frequent place where the airway closure will occur is between the base of the tongue and the posterior and lateral pharyngeal wall (Clark., *et al*. 1993). Snoring as a symptom is the main reason for consultation among those who are carriers of SAHS (Hirotaka., *et al*. 2006) [13-15].

3.3.3 Concept of SAHS. In the literature review, it is worth highlighting the disparity of terms used to refer to the apnea syndrome (OSAS, SAOS, SAS…). It was Guilleminault who introduced the concept of sleep apnea in 1973 to define patients with obstructive apneas during sleep and excessive daytime sleepiness. For years, the meanings used by various authors have been very varied. Today, SAHS is considered a clinical entity in its own right and a differential diagnosis must be made with other sleep respiratory disorders. According to the Spanish National Consensus on the apnea-hypopnea syndrome of 2005, it defines SAHS as "a condition of excessive

sleepiness, cognitive-behavioral, respiratory, cardiac, metabolic or inflammatory disorders secondary to episodes of upper airway obstruction during sleep [15-17].

Following the recommendation of the Spanish National Consensus on SAHS, with the support of the Spanish Society of Pulmonology and Thoracic Surgery (SEPAR) and the Latin American Thoracic Association (ALAT), the term SAHS is used because it is considered the most complete and least exclusive (Lloberes., *et al*. 2011). 3.3.4 Epidemiology. Sleep-disordered respiratory disorders of the upper respiratory tract are quite common in the general population (Bondemark and Lindaman, 2000). Specifically, SAHS is considered the most common today (De Ruiter., *et al*. 2018). Common snoring has an approximate prevalence of 25% in the general population, but not all of these patients have SAHS (Maguire., *et al*. 2010) [16-18].

In SAHS, the prevalence in adult patients examined is between 2 and 14% (Alessandri-Bonetti., *et al*. 2016). And it has been observed in 40-50% of patients with congestive heart failure (Eskafi., *et al*. 2004). Differences are found between sexes since men tend to suffer from this pathology more than women and as age increases, the prevalence also increases (Bondemark and Lindaman, 2000), reaching a prevalence in men over 60 years of age of 30-60% (Knappe., *et al*. 2017). The fact that it is more frequent in men has been associated with the distribution of fat, height and collapsibility of the airways, neurochemical mechanisms and also sexual hormones (Gasparini., *et al*. 2013). There are also differences between ethnic groups. In Asians, it seems that craniofacial morphology influences the development of this pathology more than obesity. However, in Hispanics and Americans, the most decisive risk factor is obesity (Gasparini., *et al*. 2013) [18-20].

The epidemiology of SAHS in the child population is no less important. There are important differences in the prevalence data for simple snoring in children in the various works in the literature (it ranges from 1.5 to 27% due to the difficulty in defining when snoring is occasional and when it is habitual). An average prevalence of 10% is accepted (Cruz Navarro I, 2014). Regarding SAHS, the American Academy of Pediatrics (AAP) in its Clinical Practice Guideline (CPG) on the diagnosis and management of pediatric SAHS accepts a prevalence ranging from 1.2 to 5.7% (when the diagnosis is made based on nocturnal PSG in a sleep laboratory) or from 0.8 to 2.8% when it is carried out according to home or outpatient PSG (Marcus., *et al*. 2012). 3.3.5 Etiopathogenesis. 3.3.5.1 [21-23].

Etiopathogenesis in adults. The pathogenesis of snoring and airway narrowing remains unclear. It has been proposed that the skeletal pattern, muscle tone and soft tissues are very important when it comes to maintaining the airway open during sleep (Bondemark and Lindaman, 2000). Obesity is one of the main risk factors for developing SAHS. It is estimated that 50% of patients with obesity (defined as a body mass index (BMI) ≥30 kg/m2) have a diagnosis of OSAHS, while up to 70% of patients diagnosed with OSAHS are obese (Labarca., *et al*. 2014).

The interaction of central obesity is associated with increased ectopic fatty tissue, especially in the cervical subcutaneous tissue, which can be estimated clinically by measuring the cervical circumference using a measuring tape, just below the Adam's apple. This clinical parameter has a high specificity for the diagnosis of OSAHS in the population (Labarca., *et al*. 2021). In a Chilean study, a cervical circumference of 43 centimeters or more (shirt size XL or 17 inches or more) is associated with a diagnosis of OSAHS with a specificity of 88% (Labarca., *et al*. 2020) [22-24].

Morphological research has shown that weight gain is associated with significant fat infiltration in the parapharyngeal and tongue regions, causing the hyoid to move caudally and lengthen the airways in patients, which is correlated with a higher number of respiratory events (Wang., *et al*. 2020). At the molecular level, adiponectin is worth highlighting. Under normal conditions, high concentrations of adiponectin are associated with a lower cardiovascular risk, however, in obese patients with SAHS, concentrations are significantly decreased, which translates into a higher risk of developing cardiovascular diseases (Labarca., *et al*. 2020). Patients with SAHS present a clinical phenotype highly suggestive of Metabolic Syndrome (MS), sharing common factors such as sedentary lifestyle, age, sociocultural determinants and even genetic predisposing factors. MS corresponds to the group of cardiovascular risk factors, determined as the "Adult Treatment Panel III" (ATP III) and which includes arterial hypertension, insulin resistance, anthropometric markers of abdominal obesity and dyslipidemia (Heinzer., *et al*. 2015) [24,25].

The accumulation of visceral fat, mainly in the abdomen and neck, together with systemic inflammation and the phenomena of intermittent chronic hypoxia and sleep fragmentation, would be the main predisposing factors for the development of both pathologies, favoring metabolic imbalance (Heinzer., *et al*. 2015). Chronic

intermittent hypoxia associated with the pro-inflammatory state described above has a negative impact on glycemic homeostasis. It has been proven that in patients with SAHS, the pancreas presents a sustained secretion of insulin, in response to hypoxia and hyperglycemia, and this is found in both obese and non-obese individuals, demonstrating that intermittent chronic hypoxia has a role in stimulating and directly damaging glucose metabolism, which triggers the risk of developing insulin resistance and type 2 diabetes mellitus (Labarca., *et al*. 2020; Wang., *et al*. 2015) [25,26].

There is an increased risk of developing cardiovascular and metabolic diseases when there is coexistence of SAHS and type 2 diabetes mellitus. In addition, the risk of suffering major cardiovascular events, such as unstable angina, myocardial infarction, stroke, heart failure and cerebrovascular mortality, is increased (Koo., *et al*. 2018; Adderley., *et al*. 2020). Observational research has shown that SAHS increases the incidence of type 2 diabetes mellitus, especially in patients with moderate to severe SAHS (Wang., *et al*. 2013). This group presents a 71% higher risk of incidence of type 2 diabetes mellitus, independently of the other risk factors for type 2 diabetes mellitus, including BMI and waist circumference (Nagayoshi., *et al*. 2016; Xu., *et al*. 2019). Metabolic fatty liver disease is a disease that is currently called MAFLD by its acronym in English (metabolic dysfunction-associated fatty liver diseases) (Eslam., *et al*. 2020) and corresponds to a group of diseases that are characterized by the deposit of fat in the liver (hepatic steatosis) which may in turn be associated with inflammation (steatohepatitis), different degrees of fibrosis and cirrhosis in more advanced cases (Younassi., *et al*. 2016) [26-28].

Its association with SAHS is very common since several common pathophysiological processes coexist with the aforementioned metabolic diseases. In 2019, Parikh., *et al*. published that suffering from SAHS could increase the risk of developing MAFLD up to 3 times. In addition, in 2020, Umbrol., *et al*. estimated a prevalence of MAFLD of up to 90% in patients with SAHS. Both authors state that severity negatively influences the evolution of fatty liver with a greater tendency towards inflammation and hepatic fibrosis. It is worth highlighting the meta-analysis carried out by Jullian-Desayes., *et al*. in 2020, where including 2,120 subjects from two cohort studies, 75 % showed hepatic steatosis using non-invasive tests, with SAHS being a significant risk factor for developing hepatic steatosis, as well as male sex, obesity and type 2 diabetes mellitus. Table 3 shows the profile of patients at high risk of developing SAHS below [27-29].

Patients at high risk of developing sleep apnea-hypopnea syndrome (SAHS)
Obesity (BMI>35)
Congestive heart failure
Atrial fibrillation
Refractory hypertension
Type 2 diabetes
Arrhythmia
Stroke
Professional vehicle drivers

Table 3: Risk factors for developing SAHS.

In non-overweight patients, craniofacial abnormalities such as micrognathia, retrognathia, and macroglossia are also risk factors that predispose to OSAHS. These pathologies contribute to restricting the airways. The loss of muscle tone that occurs during sleep contributes to the collapse (Clark., *et al*. 1993). Other orofacial risk characteristics that may predispose to OSAHS are enlarged palatine tonsils, widened uvula, high-arched palate, deviated nasal septum, increased anterior facial height, short and pronounced anterior cranial base, inferior displacement of the hyoid bone, a long soft palate, and decreased airway space (Cohen-Levy., *et al*. 2009). Another risk factor for SAHS is alcohol consumption, as it relaxes the muscles of the airway, making it more prone to obstruction (Cohen-Levy., *et al*. 2009). 3.3.5.2. Etiopathogenesis in children. Anatomical and functional factors in the child population are especially important when analyzing the etiology and risk factors. Thus, it is a combination of both. It is mostly due to a mechanical problem, which causes an imbalance between the constricting and dilating forces of the airway during sleep (Rey de Castro, 2007; Dorta Jiménez., *et al*. 2009; Villa Asensi., *et al*. 2009). The most common cause of SAHS in the child population is obstruction secondary to hyperplasia of the palatine tonsils and adenoids. Its peak incidence is between 3 and 6 years of age. It is suggested that adenotonsillar hyperplasia probably does not only act on respiratory disorders during sleep, but also precipitates the pathology in children with previous functional alterations of the airways (Rey de Castro, 2007; Asensi., *et al*. 2009; Muzumdar., *et al*. 2008) [29-31].

Other predisposing risk factors for SAHS in childhood are craniofacial abnormalities (micrognathia and retrognathia) and different dysmorphogenetic syndromes such as Pierre-Robin, Crouzon,

Treacher-Collins and Apert (Coromina., *et al*. 2006; Villa-Asensi, 2009; Muzumdar H., *et al*. 2008).

3.3.6 Maxillofacial and occlusal alterations in patients with SAHS. As a general rule, patients with SAHS tend to be critical oral breathers, determined by adenoidal and/or tonsillar hyperplasia, and the adenoid facies or tired face is observed (Coromina., *et al*. 2006). The adenoid facies is characterized by an increased and narrow lower facial height, small and upturned nose, open mouth, half-open lips, narrow alar base and retrognathia (Figure 1 and 2). Intraorally, it is related to a narrow and high maxilla, posterior crossbite and class II malocclusion (distoclusion) (Figure 3). In addition, the patient presents dental malocclusion, exposed upper incisors, short upper lip, thick and inverted lower lip and hypoplasia of the maxillary sinuses (Solano., *et al*. 2010) [31-33].

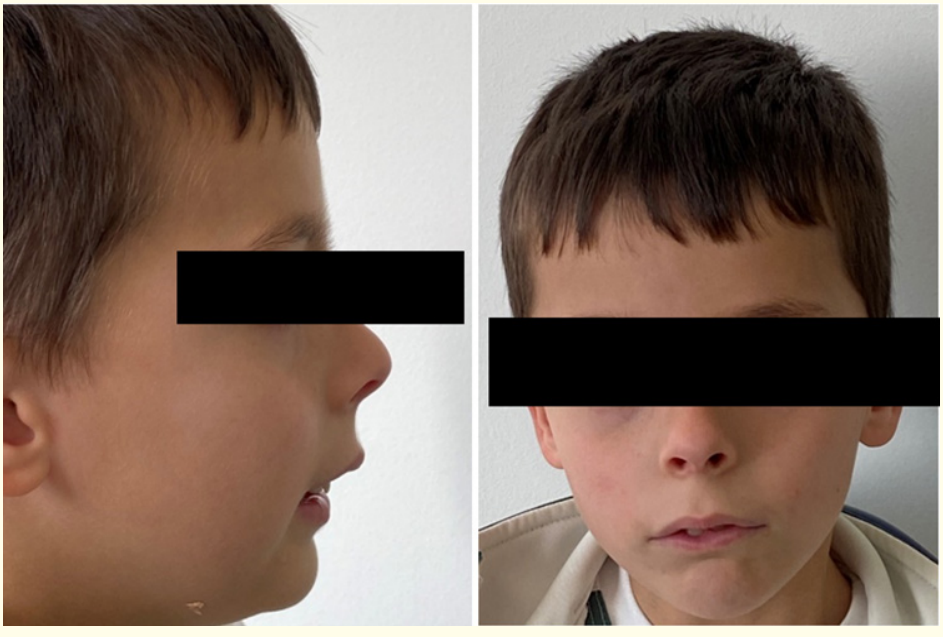

Figure 1,2: Extraoral photographs of a patient who is an oral breather.

Adenoidal hypertrophy is considered the main conditioning factor for oral breathing, since the patient is forced to keep the mouth open permanently in order to breathe, which determines that he adopts significant postural changes such as lowering the jaw, extending the head and positioning the tongue downwards, so that the jaw will be more backwards and downwards, that is, a clockwise rotation of the mandible with the inhibition of its growth (Coromina., *et al*. 2006; Suárez., *et al*. 2005; Solano., *et al*. 2010). Dentally, the lower incisors, by not having contact with their upper antagonists, move vertically upwards (egression) and backwards (distoclusion), so that the lower lip follows them, which is placed between the upper and lower incisors and pushes from the palatal surface of the upper incisors forwards (vestibuloversion). It can also be characterized by a short upper lip that accentuates the concavity of the lower lip, and a protruding and often erythematous lower lip (Solano., *et al*. 2010) (Figure 4 and 5). [1-3].

Examination and diagnosis of SAHS Patients with SAHS are at greater risk of having memory problems, increased daytime sleepiness, difficulty concentrating, depression, irritability, xerostomia, and nocturnal drooling. In terms of their daily life, they may have poor work performance, occupational accidents, and poor social relationships that influence the quality of life of patients with untreated SAHS. There is evidence of exacerbations of epilepsy, asthma, and hypertension in patients with untreated or undiagnosed SAHS. Last but not least, untreated patients with SAHS have two or three times more traffic accidents than other drivers (Martínez Font J, 2017). 3.3.7.1 Diagnosis in adults It should be emphasized that the diagnostic and therapeutic management of SAHS is clearly multidisciplinary. Different specialists (pediatricians, family doctors, dentists, neurologists, otorhinolaryngologists, pulmonologists, neurophysiologists, psychologists, etc.) are involved in the

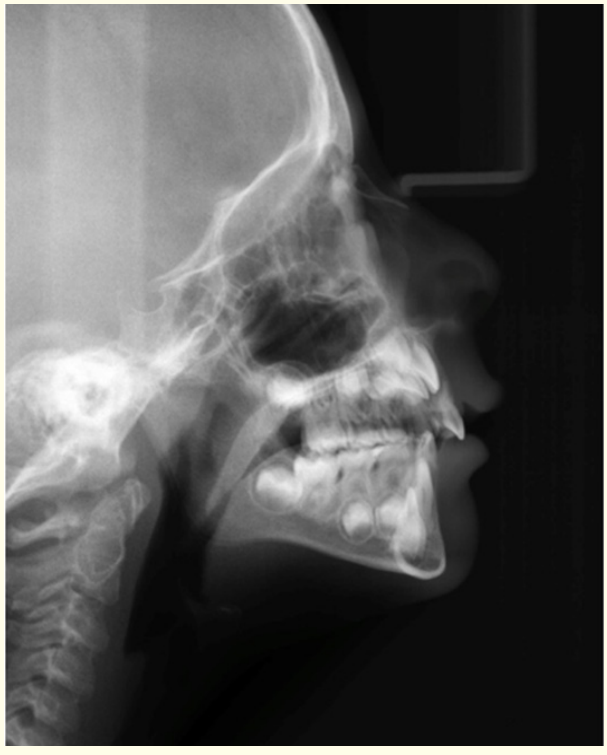

Figure 3: Lateral cranial teleradiograph of the previous patient.

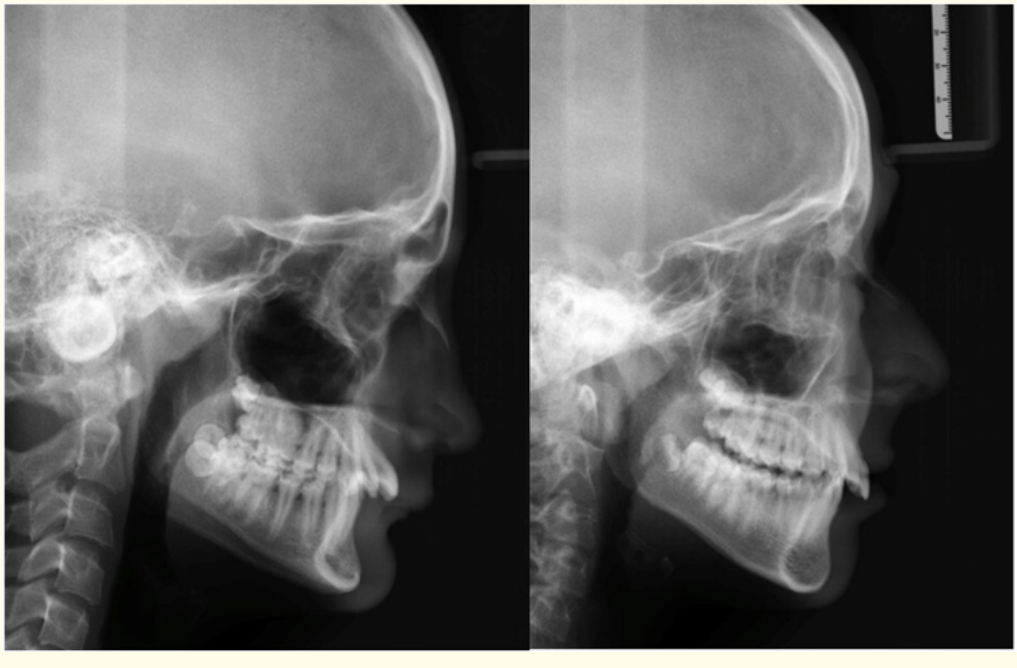

Figure 4,5: Teleradiography of patients with distoclusion 3.3.7.

care process of patients affected by sleep-related respiratory disorders (Cruz Navarro, 2014). The diagnostic criteria for SAHS are based on the signs and symptoms determined during the sleep evaluation, which includes a sleep history, a medical check-up and a sleep test [32-34].

The above is considered a "standard" within the recommendations of the American Academy of Sleep Medicine (AASM) (Epstein., *et al*. 2009). The diagnosis of SAHS begins with a sleep history that is obtained from three parts: analysis of the general health maintenance routine, evaluation of obstructive sleep apnea symptoms and finally, evaluation of patients at high risk of suffering from SAHS (Martínez Font J, 2017). Patients at high risk for SAHS include obese patients, congestive heart failure, atrial fibrillation, refractory hypertension treatment, type II diabetes, cardiac arrest, nocturnal dysrhythmias, pulmonary hypertension, professional drivers and those evaluated for bariatric surgery (Epstein., *et al*. 2009). Within the general anamnesis, special emphasis should be placed on the assessment of excessive daytime sleepiness using the Epworth scale and the possibility of its influence on daily life activities, especially when driving. The Epworth scale was designed to be answered by the patient himself. It consists of 8 questions in which different situations are presented to him and the subject must establish what possibilities he would have of falling asleep. Each situation is scored from 0 to 3 depending on the null or clear tendency to sleep, with a maximum of 24. Its reproducibility is variable and some of its questions may present sociocultural variations, but it is a universally accepted scale (Johns, 1993) [34-36].

Sleep habits, total time spent sleeping, bedtime, wake-up times, naps, etc. should also be assessed. General examination should always include BMI (BMI >30 kg/m2), increased neck circumference (>17 inches in men and >16 inches in women), blood pressure, the presence of retrognathia, overjet, macroglossia, hyoid-mandible distances, and at least one inspection of the upper airways, narrowing of the lateral tonsils, high-arched palate, tonsillar hypertrophy, elongated uvula, nasal abnormalities (polyps, deviations), and oropharynx in search of anatomical alterations, especially in patients with obesity (Eguía V., *et al*. 2007). Another characteristic that should be evaluated when there is suspicion of SAHS also includes a grade 3 or 4 in the Mallampati classification [35-37].

PSG is the gold standard and recommended method for making a correct diagnosis in patients with suspected OSAHS. It consists of a simultaneous recording of neurophysiological and respiratory variables that allow us to assess the quantity and quality of sleep, as well as to identify the different respiratory events and their cardiorespiratory and neurophysiological repercussions. (Durá Cantolla., *et al*. 2005; AASD, 1997; Kushida., *et al*. 2005). Los monitores portátiles domiciliarios están indicados en pacientes con movilidad reducida o enfermedades críticas (Epstein y cols., 2009).

Los dos métodos aceptados para la evaluación objetiva del SAHS son la polisomnografía y los monitores portátiles de uso domiciliario (Epstein y cols., 2009) [1-3].

In 1994, the AASM Practice Committee developed four levels of recordings for sleep apnea hypopnea screening. This classification of recording systems is still in use today and divides sleep studies into levels: Level I: conventional monitored polysomnography (PSG) in the sleep laboratory. Level II: unmonitored PSG, home or hospital polysomnography. Level III: portable systems and respiratory polygraphy, unmonitored, that evaluate a minimum of 4 channels. Level IV: includes simplified continuous recording systems of one or two parameters. According to AASM parameters, level II and III systems and portable recordings are indicated for the diagnosis of OSAHS as alternatives to standard PSG only in the following situations: patients with severe clinical symptoms indicative of OSAHS and laboratory PSG is not available, for patients who have impediments of any kind to not be evaluated in the sleep laboratory and for follow-up studies and evaluation of the response to the assigned therapy (Erman., *et al*. 2007). The severity of OSAHS is classified based on the patient's Apnea-Hypopnea Index (AHI) or Respiratory Disturbance Index (RDI) (Epstein., *et al*. 2009): - Mild: 5-15 events/hour - Moderate: 15-30 events/hour - Severe: >30 events/hour 3.3.7.2 Diagnosis in the pediatric population The AAP recommends screening for snoring at each child health check-up, as well as in other circumstances during daily consultations (for example, in the evaluation of tonsillitis) [35-38].

For this purpose, there is the BEARS questionnaire (Owens JA., *et al*. 2005), which assesses the main characteristics and most common sleep problems in the child population aged 2 to 18 years, increasing the probability of recognizing snoring in children.

If the BEARS questionnaire is positive for snoring, or if the patient has risk factors for sleep-disordered breathing, the AAP recommends that a detailed clinical history and examination be performed to determine whether a thorough evaluation is necessary

for the child to have OSAHS. Therefore, to carry out such a targeted clinical history, there is another questionnaire, Chervin's Pediatric Sleep Questionnaire (PSQ) for OSAHS and adapted to Spanish, which helps to differentiate primary snoring from OSAHS (with a sensitivity of 78% and a specificity of 72%) (Chervin., *et al*. 2007).

Chervin Questionnaire The Spanish National Consensus on Childhood SAHS and the CPG on Sleep Disorders suggest a diagnostic algorithm. This algorithm includes, as a diagnostic aid, some major and some minor criteria for all habitually snoring children. A habitually snoring child is considered to be one who snores more than three nights a week for more than three consecutive weeks without presenting upper respiratory tract infection. If the habitually snoring child patient meets at least four major criteria or three major and two minor criteria, it would be an indication for referral to a Sleep/Otorhinolaryngology Unit.

Major criteria

- Learning problems
- Daytime sleepiness (falls asleep in class >1 time/week)
- ADHD behavior
- Blood pressure >85th percentile for height
- Treatment-resistant enuresis BMI>97th percentile for age
- Mallampati index >2 + adenoid hypertrophy.

Minor criteria

- Hypercholesterolemia for age
- Acute otitis media and/or recurrent upper respiratory tract infections Asthma/allergic rhinitis
- Family history of OSAHS (one parent or three close relatives)
- History of prematurity

Nocturnal PSG in children is the gold standard for diagnosing sleep-disordered breathing. Based on the results of the PSG, the ICSD proposes the following classification of SAHS: - Mild: with an apnea/hypopnea index (AHI) <5. - Moderate: with an AHI between 5 and 10. - Severe: with an AHI>10. It is currently possible to perform home PSGs with 90% valid results. 3.3.8. Treatment of SAHS Due to the high morbidity and mortality, it is necessary to perform various types of treatment, which is why it is absolutely necessary to obtain an accurate diagnosis before starting any therapy (Baptista, 2007) [38-40].

There are currently different treatment modalities for patients with SAHS depending on the degree of apnea to be treated. These range from hygienic-dietary measures to surgical treatment with its various variants, including Positive Airway Pressure (PAP), considered the treatment of choice for all types of patients with SAHS, and intraoral devices, which emerged as an alternative to the impossibility of certain patients to wear PAP (Folha., *et al*. 2015). 3.3.8.1. Hygienic-dietary measures. In adult patients, it is essential to insist on the need for a change in their nutritional and lifestyle habits, avoiding a sedentary lifestyle. When the patient's BMI is greater than 40 (morbid obesity) and sustained weight loss has not worked on several occasions, the possibility of bariatric surgery may be proposed (Epstein., *et al*. 2009; Nogueira., *et al*. 2013; Martínez-García., *et al*. 2015) [41-43].

Weight loss decreases the risk of collapsing the upper airway, improving neurological control. It is related to the reduction of cholesterol levels, increases insulin resistance and decreases blood pressure (Chen., *et al*. 2013; Frutos., *et al*. 2015). Generally, if there are higher degrees of SAHS in the supine position, it is advisable to use positional therapy to avoid falling into that position while sleeping. For this, tennis balls of a size similar to those in a pocket sewn into the medial area of the back can be used, as well as placing pillows on the back (Nogueira., *et al*. 2013; Epstein., *et al*. 2009; Frutos., *et al*. 2015). Another important measure is to avoid drinking alcohol at least 6 hours before going to bed. This increases snoring and the risk of suffering from apneas and hypopneas, due to depression in the functioning of the dilating muscles of the pharynx, depression of ventilatory resistance and hypoxia and hypercapnia. In addition, there is a potential risk of suffering from SAHS if alcohol is consumed together with hypnotics and benzodiazepines, in addition to the risk that this entails for the patient's health (Frutos., *et al*. 2015; Chen., *et al*. 2013). Tobacco consumption should also be reduced, since it increases the risk of SAHS. It increases the inflammatory response of the upper airway, which reduces the calibre and promotes collapse while sleeping (Balaguer., *et al*. 2009) [42-45].

Finally, it is important to recommend a regular schedule and an appropriate environment that favors sleep, as well as avoiding eating large amounts of food before going to sleep. Rest in a bedroom with an ideal temperature, without noise, bright lights or stimu-

lants such as electronic devices (Hoffstein, 2007; Frutos., *et al*. 2015). 3.3.8.2. Pharmacological treatment The scientific evidence on the use of drugs is limited due to the design of the studies, the differences in response to treatment, side effects and lack of cardiovascular benefits (Frutos., *et al*. 2015).

The mechanisms of action of drugs in SAHS have been aimed at increasing pharyngeal dilator muscle tone, increasing ventilatory stimulus, decreasing VAS resistance and reducing pharyngeal surface tension (Frutos., *et al*. 2015). The effectiveness of different drugs has been investigated, such as: - Tricyclic antidepressants such as protriptyline: they prevent the reuptake of serotonin and noradrenaline, reduce REM sleep and increase the tone of the upper airway, but there is research showing that there is not enough improvement (Epstein., *et al*. 2009). - Selective serotonin reuptake inhibitors such as paroxetine, not recommended for the treatment of SAHS. - Heterocyclic antidepressants such as mirtazapine, increase the risk of drowsiness and weight gain as they act as a serotonin agonist and increase its secretion. - Stimulants in ventilation such as methylexanthines, show very varied results, reduce total sleep time and its efficiency [45-47].

Opioid antagonists (naloxone and naltrexone), do not improve SAHS, more awakenings and less sleep efficiency are observed. - Medroxyprogesterone and estrogens in postmenopausal patients decrease the AHI (Frutos., *et al*. 2015). - Diuretics such as furosemide and spironolactone are associated with an increase in the caliber of the high blood pressure due to improvement in pharyngeal edema. Improvement in the AHI was observed in patients with SAHS and heart failure (Bucca., *et al*. 2007). However, Frutos., *et al*. did not obtain relevant results. - The use of clonidine has a suppressive effect on REM sleep and an improvement in apneas, but in certain patients hypopneas have a risk of becoming apneas (Frutos., *et al*. 2015). Different authors recommend reducing the use of benzodiazepines as much as possible since they increase the risk of suffering from apneas and avoiding the use of hypnotics whenever possible. If the use of sleep-inducing drugs is necessary, the drugs of choice are zolpidem, zopliclone or zaleplon (Martínez-García., *et al*. 2015; Frutos., *et al*. 2015). Other drugs that can exacerbate SAHS are corticosteroids, muscle relaxants, antiepileptics and codeine (Martínez-García., *et al*. 2015). In summary, to date, there are no drugs that work well except in patients with hypothyroidism or acromegaly in whom their treatment improves the AHI (Epstein., *et al*. 2009; Martínez-García., *et al*. 2015; Frutos., *et al*. 2015) [48-50].

Surgical treatment

Surgical techniques are indicated for patients with intolerance to PAP, or when it is contraindicated or where the use of mandibular advancement devices (MAD) has not yielded satisfactory results (Epstein., *et al*. 2009). The different surgical techniques are shown in the following table 4 (Lloberes., *et al*. 2011; Fernández-Julián., *et al*. 2015)

Surgical treatments	
Tracheostomy	
Nasal surgery	Turbinate reduction
	Septoplasty
	Rhinoplasty
	Nasal valve surgery
	Polyp removal
Oral, oropharyngeal and nasopharyngeal surgery	Uvulopalatopharyngoplasty
	Pharyngoplasty
	Tonsillectomy
	Adenoid removal
	Mandibular torus excision
	Mandibular advancement
	Palatal implants
Hypopharyngeal surgery	Tongue reduction (partial or total)
	Lingual advancement genioglossus
	Hyodes suspension
	Tongue base radiofrequency
	Tongue base suspension-retention
Laryngeal surgery	Epiglottoplasty
Maxillomandibular surgery	

Table 4: Surgical treatments for SAHS Tracheostomy can eliminate SAHS, but it does not correct central hypoventilation syndromes.

Nasal surgery alone does not resolve SAHS, however, it can be used in patients with PAP intransigence. Surgery of the base of the tongue only has a success rate of up to 40% in mild or mild-moderate SAHS (Lloberes., *et al*. 2011). The vast majority of surgical techniques are aimed at the soft palate. However, its effectiveness is only 40-50% in cases of apnea (Lloberes., *et al*. 2011; Petit., *et al*. 2002). Resections of the soft palate should be as minimally in-

vasive as possible, since if the patient develops SAHS in the future and needs treatment with PAP, its use may be a problem due to air leaks through the oral cavity (Fernández-Julián., *et al*. 2015).

Another surgical technique option is the insertion of palatal implants. It has a success rate of 79.3 % but the risk of this technique is the extrusion of the implant which occurs 2 to 17 % of the time (Savage., *et al*. 2007). The advancement of facial skeletal structures by orthognathic surgery is an effective treatment for SAHS. It allows the pharyngeal air space to increase and makes it less prone to collapse. For this reason, it decreases the AHI, increases the minimum oxygen saturation and improves the quality of sleep. It can be performed by means of a combined advancement of the maxilla, mandible and/or chin, or by individual advancement of one of them. The selection of the technique depends on the comprehensive diagnosis, correct planning and the interests of the patient. The retroposition of the bony structures implies a decrease in the pharyngeal airspace, an element that must be taken into account in the correction of skeletal Angle class III (García-Menéndez., *et al*. 2020). Another alternative is bariatric surgery, which is indicated for individuals with a BMI≥40 kg/m2 or a BMI≥35 kg/m2 with a significant risk of comorbidity and ineffective diet control (Epstein., *et al*. 2009).

In a recent published study where modified repositioning pharyngoplasties were performed for the treatment of SAHS, after a comprehensive study of the patient, they obtained a significant decrease in the AHI from 29.1 ± 18.3 to 12.3 ± 12, with no significant changes in BMI. According to Sher's criteria, in 65.4% of patients there was a decrease in HIA by half and <20/h, and 42.3% of the total had an HIA<10/h. The most frequent complication was partial extrusion of the suture (Carrasco Llatas., *et al*. 2021). An important aspect is the surgical risk of patients with SAHS who must be operated on under general anesthesia, since they present greater preoperative complications. These complications are due to the effects of anesthetics on ventilation control and on the tone of the VAS muscles. The application of nasal CPAP after extubation and placing the patient in a semi-sitting position are general recommendations for the postoperative period (Hilman., *et al*. 2004; Rennotte., *et al*. 1995). 3.3.8.4. Positive airway pressure treatment Sullivan, in 1981, described PAP treatment for the first time, to address SAHS through the use of nasal and oronasal masks. This PAP pressure was shown to prevent apneas and improve symptoms during the day. Therefore, today it is considered the "gold standard" treatment for patients with moderate-severe SAHS (Anitua., *et al*. 2005; Cortés-Reyes., *et al*. 2017; Epstein L., *et al*. 2009; Durán-Cantolla., *et al*. 2013).

It consists of a pneumatic splint composed of a fan and a suction valve. The fan takes in ambient air and generates a constant flow through a flexible tube connected to a face mask (Balanta-Melo, 2015; Torres-Valdez, 2011). PAP treatment is based on the collapsibility of the upper airway and not on the AHI (Bonnet., *et al*. 1992). PAP treatment increases the volume of the airway, decreases the thickness of the lateral walls of the pharynx and the chronic edema secondary to the vibration process that the patient suffers during snoring. In addition, it corrects ventilatory instability by increasing the PCO2 reserve and reducing the range of exaggerated response to hypocapnia present in patients with SAHS (Torres-Valdez, 2011). The air flow, in addition to creating nasal pressure, prevents rebreathing. A minimum flow is required to renew the air in the circuit that the patient breathes. This is 4 cm of H2O (Yetkin., *et al*. 2008). PAP treatment must be a mandatory step before indicating any surgical treatment, as well as to be able to predict, based on the pressures used during calibration, which patients will improve with MAD (Torres-Valdez, 2011).

There are different PAP modalities. One of them is continuous positive airway pressure (CPAP), which provides a single constant pressure throughout the respiratory cycle previously selected by titration with PSG. It is the treatment of choice for SAHS and its effectiveness is superior to that of other therapies (Nogueira., *et al*. 2013). Its objective is to maintain a constant positive pressure throughout the respiratory cycle, decreasing inspiratory work (Cortés-Reyes., *et al*. 2017). The hours of use are controlled by a counter, they have a leak compensation system, room air filters and the possibility of adapting thermo-humidifiers. The choice of interface (nasal mask, bucconasal mask or nasal pillows) should be customized for each patient to avoid leaks (Nogueira., *et al*. 2013) [48-50].

In addition to CPAP, there are other modalities such as self-titrating positive airway pressure (APAP), bi-directional positive airway pressure devices (BPAP), and anaesthetic devices (APAP).

Efficacy of positive airway pressure treatment PAP has been shown to be an effective method for treating SAHS in 95% of pa-

tients. It prevents drops in SaO2 and electroencephalographic arousals secondary to respiratory events and normalises sleep (Anitua., *et al*. 2005; Durán-Cantolla., *et al*. 2013; Cortés-Reyes., *et al*. 2017). It reduces symptoms such as daytime hypersomnolence (Torres-Valdez, 2011; Durán-Cantolla., *et al*. 2013) and risk factors associated with comorbidities; improves quality of life (Torres-Valdez, 2011; Anitua., *et al*. 2005) and neurocognitive function (Torres-Valdez, 2011) [48-50].

It shows satisfactory and significant results in reducing arterial and pulmonary pressure in hypertensive patients (Torres-Valdez, 2011) in addition to maintaining the AHI at normal levels (Alía García., *et al*. 2010; Balanta Melo, 2015). At the metabolic level, it improves the levels of inflammation markers, corrects oxidative stress, promotes insulin sensitivity and dyslipidemia (Lloberes., *et al*. 2011; Torres-Valdez, 2011; Cortés-Reyes., *et al*. 2017,). On the nervous system, it improves neurocognitive function, increases life in patients who suffer from cerebrovascular accidents (Lloberes., *et al*. 2011; Torres-Valdez, 2011; Anitua., *et al*. 2005), reduces the risk of traffic accidents (Torres-Valdez, 2011) and reduces depression of the nervous system (Torres-Valdez, 2011; Cortés-Reyes., *et al*. 2017). At the cardiovascular level, it corrects cardiac function, decreases the risk of cardiovascular consequences (Lloberes., *et al*. 2011), alleviates hypertension (Durán-Cantolla., *et al*. 2013; Cortés-Reyes., *et al*. 2017), improves fibrinogen and plasminogen activator levels by correcting hypoxia-hyperoxia episodes (Lloberes., *et al*. 2011), corrects arrhythmias (Cortés-Reyes., *et al*. 2017), and eliminates atrial fibrillation, bradycardia and sinus pauses (Cortés-Reyes., *et al*. 2017; Lloberes., *et al*. 2011; Torres-Valdez, 2011). It reduces accidents and mortality (Anitua., *et al*. 2005; Nogueira., *et al*. 2013; Durán-Cantolla J., *et al*. 2013). 3.3.8.4.2. Adverse effects of treatment with positive airway pressure No research has been described to evaluate the long-term side effects of prolonged use of PAP in patients with SAHS.

Only short-term effects have been published, which usually appear in the first few days and are transient, such as local skin erosions, tissue pressure ulcers, nasal congestion and epistaxis, conjunctivitis, irritation of the upper airway, rhinorrhea, transient deafness, headache, insomnia, air leaks in the mask, difficulty in expiration, aerophagia, chest discomfort, dry pharynx, cold, lack of motivation and anxiety. In addition, it can cause gastric and intestinal distension due to the entry of air flow into the digestive tract (Lloberes., *et al*. 2011; Cortés-Reyes., *et al*. 2017; Balanta Melo, 2015). Other research claims that PAP can cause clinical or functional deterioration in asthmatic patients (Lafond., *et al*. 2006; Ciftci., *et al*. 2005).

Adaptation to PAP improves with good management of the problems that may arise when starting therapy. It is recommended that it be performed by a sleep unit, as well as a complete otorhinolaryngological examination to detect anatomical alterations that advise evaluating surgical alternatives (Terán-Santos, 2017). 3.3.8.5. Treatment with MAD It has been shown that its effectiveness is not as effective as PAP, oral appliances are indicated in patients with mild to moderate SAHS who prefer oral appliances to CPAP, or who do not respond well or are not candidates or fail treatment with PAP, as well as hygienic-dietary measures (Epstein., *et al*. 2009; Kushida., *et al*. 2005). 3.3.8.5.1.

Pre-treatment dental evaluation Prior to the manufacture of a MAD, it is important to perform a pre-treatment dental evaluation. This includes dental history and oral examination, paying special attention to the occlusion, periodontal status, dental mobility, parafunctional habits, wear facets, dental sensitivity and amount of overjet and overbite of the patient. It is also important to analyze the dental and skeletal midlines as well as the health of the temporomandibular joint (Martínez Font., *et al*. 2017) [48-50].

The specialist must confirm that the patient has enough teeth, between 6 and 10 teeth per arch, and has no limitations in protrusion (>5 mm) or opening (>25 mm) movement, since the mechanism of action of the MADs is based on advancing the mandibular position to avoid a subsequent collapse of the upper jaw and thus raise its caliber by stimulating the tension of the infrahyoid muscles. If the patient presents a wear greater than 20% of the clinical crowns, it indicates severe bruxism and would complicate treatment with MADs, as well as moderate to severe TMJ pain would contraindicate treatment with these devices. Completely edentulous patients are not candidates for the use of MADs but can be treated with lingual devices (Almeida., *et al*. 2009).

Action of the DAM. 3.3.8.5.2 Mechanisms of action The DAM cover the upper and lower teeth and the mechanism of action is to advance the mandible and achieve an anterior lingual repositioning (Campos López., *et al*. 2017; Simón Pardells., *et al*. 2012) thanks to the superior displacement of the hyoid bone, lateral displacement of the parapharyngeal fat pads and anterior displacement of the muscles of the base of the tongue. It also tenses the palatoglossus

and palatopharyngeus muscles, elevates the vertical dimension by activating the genioglossus muscle and the lateral walls of the velopharyngeal area. The soft palate moves ventrally and increases the caliber of the retromandibular space (Ferguson., *et al*. 1997; Hamoda., *et al*. 2018).

These modifications are associated with a reduction in extraluminal pressure and an increase in neuromuscular activation of the upper airway by neurosensory stimulation, decreasing pharyngeal collapsibility (Cortés-Reyes., *et al*. 2017; Balanta Melo, 2015).

These devices have several advantages since they are comfortable and easy to manipulate by the patient, they are non-invasive, they are reversible, easy to manufacture by a specialist prosthetist and, as a general rule, they are well accepted by the patient (Cobo Plana., *et al*. 2002). The MADs act like the Herbst type orthopedic devices but on the adult population where bone growth cannot be stimulated and they are worn in the mouth for 6 to 8 hours a day (Guilleminault, 1976). The recommended percentage of progress is 50% for snoring and 70% for SAHS. It has been shown that increasing the degree of mandibular advancement improves the patient's clinical situation, such that for every 2 mm of protrusion there is a 20% benefit in both the number and severity of desaturations (Cobo Plana., *et al*. 2002; Martínez Gomis., *et al*. 2004).

Types There are currently many types of MAD on the market. Their differences lie in the manufacturing material, location of the connecting rods, degree of adjustment, amount of increase in vertical dimension or lateral movement of the mandible. There are also laboratory devices (Figure 6-9) or prefabricated one-piece or two-piece, adjustable or non-adjustable, made of a more rigid or soft material, but what has been shown is that devices made to measure for the patient have fewer failures (Hamoda., *et al*. 2018; Hoffstein, 2007) [48-50].

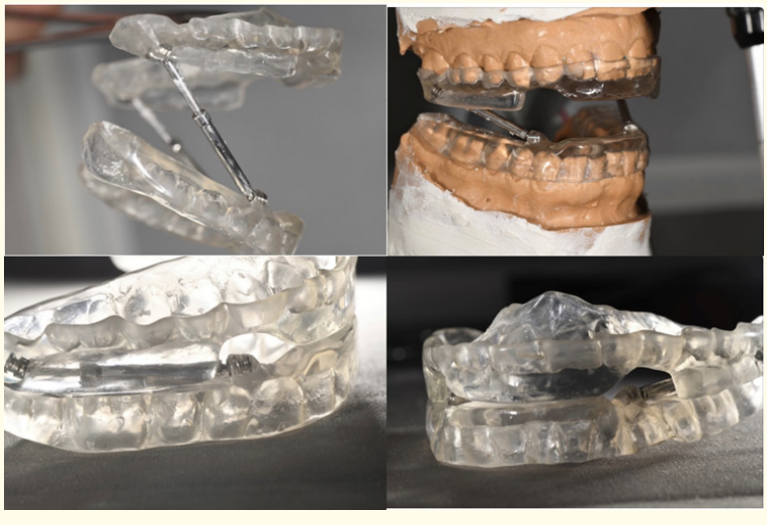

Figure 6-9: Laboratory-made MADs made to measure for the patient.

DT: José Peidró. The devices must be adjusted by professionals in oral health, TMJ, dental occlusion and associated structures (Kushida., *et al*. 2005). 3.3.8.5.4 Risks of using MADs The adverse effects of MADs, because they have only recently come into use, have not yet been able to be investigated in the long term and are only focused on the development and prevalence of temporomandibular joint disorders (TMD) (Parres., *et al*. 2016; Pantin., *et al*. 1999). When using a MAD, the mandible is in a more anterior position, which will affect the structures of the oral cavity such as muscles, teeth and occlusion and even the temporomandibular joint (TMJ) (Parres., *et al*. 2016; Hamoda., *et al*. 2018).

The risks of using MAD include the development of signs and symptoms of TMD, occlusal changes associated with the use of MAD and other minor risks (Redondo de Mena., *et al*. 2019). A) Signs and symptoms of TMD The most relevant risks are TMJ arthralgia, which according to various authors is due to a remodeling

of the articular disk, but which patients accept and occurs over the first hours or days after starting. To remedy this risk, it is proposed to perform a progressive advancement of the mandible (Martínez Gomis., *et al*. 2004; Alía García., *et al*. 2010; Chen., *et al*. 2013). Joint noises in the TMJ in patients who use MAD occur in most cases when the device is removed and they occlude again, producing contact with the molars and premolars [48-50].

This can trigger problems such as capsulitis, but a very low percentage evolves into a joint blockage (Martínez Gomis., *et al*. 2004; Parres., *et al*. 2016). To date, there is still not enough research to determine the relationship between the degree of protrusion caused by the MAD and the risk it may cause to the TMJ, although it is expected that the greater the protrusion and the more hours the device is used, the greater the risk (Doff., *et al*. 2012). During the first month, between 7 and 66% of patients with MAD suffer from muscle pain and mandibular fatigue (Martínez Gomis., *et al*. 2004). Joint sensitivity and muscle pain are the most common side effects, so the presence of these symptoms must be ruled out initially during treatment.

Headache can also appear secondary to SAHS (Parres., *et al*. 2016; Clark., *et al*. 1993). B) Changes in occlusion It is estimated that between 14 and 26% of patients treated with MAD have reduced their overbite by more than 1mm after 2-3 years (Marklund., 2006). It is related that the greater the amount of mandibular advancement, the greater the decrease in overbite and overjet (Martínez Gomis., *et al*. 2004). Posterior open bite is also related to the occlusal changes produced by the use of MAD. This is due more to a long-term mandibular postural advancement, which leads to remodeling of the articular surfaces of the TMJ and intrusion and extrusion of the teeth over time (Balanta Melo., 2015) [48-50].

There is a relationship between the increase in the patient's vertical dimension and greater changes in occlusion (Anitua., *et al*. 2005; Clark., *et al*. 1993). C) Minor side effects Various side effects have been described, such as pain, sensitivity or pressure at the initial stages of using DAM, but which decrease over time (Martínez Gomis., *et al*. 2004; Chen., *et al*. 2013). Effects have also been published with excess salivation, which decreases over the days, xerostomia due to the increase in the patient's vertical dimension with labial incompetence (Martínez Gomis., *et al*. 2004; Chen., *et al*. 2013). At the beginning of using DAM, the patient may report a feeling of suffocation and nausea. Gingival irritation and lesions of the oral mucosa, transient (Martínez Gomis., *et al*. 2004). Finally, fractures of the DAM, the telescopic arm or loss of retention of the same can also occur (Alía García., *et al*. 2010). In conclusion and given the relevance that SAHS presents for the general population, the benefits of the use of DAM and PAP are much greater than the risks that SAHS triggers, so it is the duty of all health professionals to pay special attention to these patients, with a correct diagnosis, treatment and personalized and close follow-up, educating them in good hygienic-dietary habits [48-50].

Conclusion

There are many complications and risks to which a patient suffering from sleep apnea or hypopnea is exposed. In this work, the main ones were described so that professionals and the general public who wish to review them have easy access.

Acknowledgements

To my wife for all her unconditional professional and personal support.

Bibliography

1. Abrams R. "Slepp deprivation". *Obstetrics and Gynecology Clinics of North America* 42.3 (2015): 493-506.

2. Alessandri-Bonetti G., *et al.* "The Effects of Mandibular Advancement Device on Pressure Pain Threshold of Masticatory Muscles: A Prospective Controlled Cohort Study". *Journal of Oral and Facial Pain and Headache* 30 (2016): 234-240.

3. Alía García E., *et al.* "Efficacy and adverse effects of intraoral appliances in the treatment of obstructive sleep apnea". *Científica Dent Rev Científica Form Contin* 7.2 (2010): 19-26.

4. Almeida FR and Lowe AA. "Principles of oral appliance therapy for the management of snoring and sleep disordered breathing". *Oral and Maxillofacial Surgery Clinics of North America* 21.4 (2010): 413-420.

5. Darien IL. "American Academy of Sleep Medicine; 2014. An American Sleep Disorders Association Review. The indications for polysomnography and related procedures". *Sleep* 20 (1997): 423-487.

6. Anitua E and Durán-Cantolla J. "BTI APNIA, intraoral device APNIA: diagnosis and treatment of sleep apnea and snoring". Vitoria: Teamwork Media Spain (2005).

7. Balaguer C., *et al.* "Smoking and Sleep Disorders". *Archivos de Bronconeumología* 45.9 (2009): 449-458.

8. Balanta-Melo J. "Craniofacial changes with long-term use of mandibular advancement devices for the treatment of obstructive sleep apnea: A systematic review". *Revista Estomatologia* 22 (2015): 35-45.

9. Baptista PM. "Surgery as treatment for obstructive sleep apnea". *Anales del Sistema Sanitario de Navarra* (2007): 30.

10. Battagel JM and Kotecha B. "Dental side-effects of mandibular advancement splint wear in patients who snore". *Clinical Otolaryngology* 30 (2005): 149-156.

11. Biggs S., *et al.* "Working memory in children with sleep-disordered breathing: Objective versus subjective measures". *Sleep Medicine* 12 (2011): 887-891.

12. Bondemark L and Lindman R. "Craniomandibular status and function in patients with habitual snoring and obstructive sleep apnoea after nocturnal treatment with a mandibular advancement splint: a 2-year follow-up". *European Journal of Orthodontics* 22.1 (2010): 53-60.

13. Bonnet M., *et al.* "The Atlas Task Force: EEG arousals: Scoring rules and examples". *European Journal of Ophthalmology* 22 (2000): 53-60.

14. Bourke R., *et al.* "Neurobehavioral function is impaired in children with all severities of sleepdisordered breathing". *Sleep Medicine* 12 (2011): 222-229.

15. Bucca CB., *et al.* "Diuretics in Obstructive Sleep Apnea with Diastolic Heart Failure". *Chest* (2007).

16. Campos López J and Campos Peña A. "Treatment of sleep apnea-hyponea syndrome with CPAP and mandibular advancement device". *Seville* (2017).

17. Cappuccio FP., *et al.* "Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis". *Diabetes Care* 33.2 (2010): 414-420.

18. Carrasco Llatas M., *et al.* "Modified repositional pharyngoplasty for the treatment of OSAHS: how we do it and our results". *Acta Otorrinolaringológica Española* 72 (2021): 152-157.

19. Chen H and Lowe AA. "Updates in oral appliance therapy for snoring and obstructive sleep apnea". *Sleep Breath* 17.2 (2013): 473-486.

20. Chervin RD., *et al.* "Pediatric sleep questionnaire: prediction of sleep apnea and outcomes". *Archives of Otolaryngology--Head and Neck Surgery* 133 (2007): 216-222.

21. Ciftci TU., *et al.* "Effect of nasal continuous positive airway pressure in uncontrolled nocturnal asthmatic patients with obstructive sleep apnea syndrome". *Respiratory Medicine* 99.5 (2005): 529-34.

22. Clark GT., *et al.* "Effect of anterior mandibular positioning on obstructive sleep apnea". *American Review of Respiratory Disease* 147 (1993): 624-629.

23. Clement-Carbonell V., *et al.* "Sleep Quality, Mental and Physical Health: A Differential Relationship". *International Journal of Environmental Research and Public Health* 18.2 (2021): 460.

24. Cobo-Plana J., *et al.* "Illustrious General Council of Colleges of Dentists and Stomatologists of Spain" (2002): 417-427.

25. Cohen-Levy J., *et al.* "Treatment of obstructive sleep apnea syndrome in adults by mandibular advancement device: the state of the art". *International Orthodontics* 7.3 (2009): 287-304.

26. Coromina J and Estivill E. "The snoring child. The child with sleep apnea obstructive sleep apnea syndrome". Madrid: EDIMSA (2006).

27. Cortés-Reyes E., *et al.* "New perspectives in the treatment of obstructive sleep apnea-hypopnea syndrome". *Revista Colombiana de Anestesiología* 45 (2017): 62-71.

28. Cruz Navarro I. "The snoring child (SAHS)". *Revista de Pediatría de Atención Primaria* 23 (2014): 89-100.

29. De Ruiter MHT., *et al.* "Durability of treatment effects of the Sleep Position Trainer versus oral appliance therapy in positional OSA: 12-month follow-up of a randomized controlled trial". *Sleep Breathing* 22 (2018): 441-450.

30. Dorta Jiménez B and Herreros RO. "Update on the OSA Syndrome Obstructive Sleep Apnea in Children". *Rev Spanish Association of Child and Adolescent Psychiatry* 26 (2009): 4.

31. Doff MHJ., *et al.* "Long-term oral appliance therapy in obstructive sleep apnea syndrome: A controlled study on temporomandibular side effects". *Clinical Oral Investigations* 16.3 (2012): 689-697.

32. Duarte ER., *et al.* "Treatment of snoring and sleep apnea syndrome with a removable mandibular advancement device in patients without TMD". *Dental Press Journal of Orthodontics* 17 (2012): 90-96.

33. Durán-Cantolla J. "Cols and Spanish Sleep Group (GES). National Consensus on SAHS". *Archivos de Bronconeumología* 41 (2005): 12-29.

34. Eguía VM and Cascante JA. "Sleep apnea-hypopnea syndrome. Concept, diagnosis and medical treatment". *Anales del Sistema Sanitario de Navarra* 30.1 (2007): 53-74.

35. Epstein LJ., *et al.* "Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults". *Journal of Clinical Sleep Medicine* 15 (2009): 263-276.

36. Erman MK., *et al.* "Validation of the Apnealink™ for the evaluation, management and long-term care of obstructive sleep apnea in adults". *Journal of Clinical Sleep Medicine* 3.4 (2007): 387-392.

37. Eskafi M., *et al.* "Use of a mandibular advancement device in patients with congestive heart failure and sleep apnoea". *Gerodontology* 21 (2004): 100-107.

38. Eslam M., *et al.* "A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement". *Journal of Hepatology* 73.1 (2020): 202-209.

39. Ferguson KA., *et al.* "A short-term controlled trial of an adjustable oral appliance for the treatment of mild to moderate obstructive sleep apnea". *Thorax* 52.4 (1997): 362-368.

40. Fernández Julián E., *et al.* "Chronic snoring and UPPP". *Acta Otorrinolaringológica Española* 53 (2009): 269-280.

41. Fernández-Julián E., *et al.* "Snoring and sleep apnea syndrome -hypopneas during sleep. In: Sleep medicine treatise. 1a. Madrid: Editorial Médica Panamericana (2015): 478-492.

42. Flemons WW. "Clinical practice. Obstructive sleep apnea". *The New England Journal of Medicine* 347.7 (2002): 498-504.

43. Frutos J de., *et al.* "Medical treatment of sleep apnea-hypopnea. In: Sleep medicine treatise. 1a. Madrid: Editorial Médica Panamericana (2015): 439-444.

44. Folha GA., *et al.* "Validity and reliability of a protocol of orofacial myofunctional evaluation for patients with obstructive sleep apnea". *European Journal of Oral Sciences* 123.3 (2015): 165-172.

45. Gallego Pérez-Larraya J., *et al.* "Classification of sleep disorders". *Anales del Sistema Sanitario de Navarra* 30 (2007): 19-36.

46. García Menéndez M., *et al.* "Orthognathic surgery in the treatment of obstructive sleep apnea syndrome". *Revista Cubana de Estomatología* 57.1 (2020): e1644.

47. García Urbano J. "Snoring and obstructive apnea. Solutions to sleep problems. 1st edition. Madrid: Ripano (2010).

48. Gasparini G., *et al.* "OSAS treatment with oral appliance: assessment of our experience through the use of a new device". *European Review for Medical and Pharmacological Sciences* 17 (2013): 385-391.

49. Glozier N., *et al.* "Short sleep duration in prevalent and persistent psychological distress in Young adults: the DRIVE study". *Sleep* 33.9 (2010): 1139-1145.

50. Guilleminault C., *et al.* "A cause of excessive daytime sleepiness. The Upper Airway Resistance Syndrome". *Chest* 104 (1993): 781-787.