CRANIOFACIAL RESEARCH CONNECTION JOURNAL

MAXILLOFACIAL SURGERY IN THE MANAGEMENT OF OBSTRUCTIVE SLEEP APNEA SYNDROME _ Genioplasty

Literature Review

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P.M. Cota, A.C.D. Filho, G. Vieira, Thereza-Bussolaro C. Maxillofacial Surgery in the. Management of Obstructive Sleep Apnea_genioplasty Syndrome. Craniofacial Research Connection Journal. 2021;1(16-25)

Accepted for publication: 29/03/2021

ABSTRACT: Obstructive sleep apnea is a disturbance in breathing patterns while sleeping associated with a high morbidity and mortality rate. Several anatomical and neuromuscular factors have been linked to obstructive sleep apnea-hypopnea syndrome (OSAHS). Surgical treatments include soft tissue surgery, hypoglossal stimulating nerve, and orthognathic surgery. Bimaxillary orthognathic surgery improves both symptoms and quality of life. Genioplasty, a less invasive type of orthognathic surgery, has been shown as effective in reducing the apnea-hypopnea index by 45.7%. The present study aims to review the literature about the management of OSAHS through maxillofacial surgery, focusing on the effect of genioplasty in patients with OSAHS. Therefore, we sought to answer the research question: Can genioplasty improve OSAHS patients' Apnea-Hypopnea Index (AHI) or oxygen saturation (sat. 02)? Pubmed and ScholarGoogle databases were searched. Twelve studies were found reporting improvements in patients undergoing genioplasty. Genioplasty showed to be effective in surgical management for patients with OSAHS.

KEYWORDS: Sleep Apnea, Obstructive. Orthognathic Surgery. Genioplasty.

INTRODUCTION

Sleep-related disordered breathing (SRDB) encompass diagnostic categories such as snoring, upper airway resistance syndrome and obstructive sleep apnea-hypopnea syndrome (OSAHS).[1]

Prevalence of OSAHS in adults varies from 14% in men and 5% in women, however, this number is higher in a convenience population, such as obese patients with indications for bariatric surgery, and stroke victims.[2]

Likewise, the lack of diagnosis in many individuals who are not aware of their condition can mislead the prevalence of the disease, generating an alert about the possibility of increasing this prevalence in the future.[3]

OSAHS is associated with a high rate of morbidity and mortality[2], and anatomical and functional factors predispose to the development of OSAHS.[2] The severity of the disease is classified based on the number of respiratory intermittences that the individual has per hour of sleep, the so-called rates of apnea-hypopnea (AHI).[4,5] This classification criterion, for now, follows the light limit for AHI≥ 5, moderate for AHI ≥ 15, and severe for AHI ≥ 30. [5]

In addition to all deleterious effects on the individual's health, untreated OSAHS have an economic impact on society, as they increase public health costs, decrease the individual's productivity and, at the same time, increase the incidence of motor vehicle accidents.[1] The OSAHS management includes a variety of conservative and surgical treatments.[6] Among conservative treatments, there are functional muscle therapy, CPP continuous respiratory pressure device, and mandibular repositioning devices. Among surgical treatments, there are soft tissue surgery, hypoglossal stimulating nerve and orthognathic surgery.[5]

Orthognathic surgery improves quality of life as well as symptoms in syndromic patients, which can be observed in cases of bimaxillary surgical advancement.

Genioplasty is a less invasive orthognathic surgery than bimaxillary surgery and has been shown to be effective in the management of patients with OSAHS, reducing the apnea-hypopnea index by 45.7% .[7]

It has been used to improve functional or aesthetic deformities and in general, the indication of the procedure is based on the type and extent of the deformity, which can be considered prominent (prognathia), deficient (retrognathia), or asymmetric.1

This study aimed to review the literature on the approach of OSAHS by oral maxillofacial surgery and the effect of genioplasty in patients with OSAHS and to seek an answer to the research question: orthognathic surgery of chin advancement can improve the Apnea-Hypopnea (AHI) of patients with OSAHS?

METHODS

For the literature review, a data search was performed on the Pubmed / Medline and Google Scholar platforms, until November 16, 2020. Inclusion criteria were original case reports and review papers, as well as monographs and theses. Papers in Portuguese and in English were included. search terms were adapted for each database.

LITERATURE REVIEW

Sleep Obstructive Hypopnea Apnea Syndrome (OSAHS)

OSAHS Pathophysiology

Obstructive sleep apnea syndrome (OSAHS) is a multifactorial chronic disease characterized by the presence of daytime signs and symptoms. Specifically, during sleep, patients experience five or more obstructive events per hour of sleep (≥ 5 / h), resulting in repeated partial (hypopnea) or total (apneas) upper airway obtructions, compromising airflow to the lungs. These events are diagnosed through

polysomnography, which provides the so-called apnea and hypopnea index (AHI).[6]

During sleep, a patient with apnea has pauses in breathing, snoring, choking, expiratory, restlessness, short periods of noisy hyperpnea, and jaw relaxation. For instance, family members report's when observing the patient sleeping7 The patient himself may complain of morning headache and nausea, dry mouth, and sore throat.[2]

In 2014, an update of the protocol for the diagnosis of OSA was established by the task force (TF) of the American Academy of Sleep Medicine (AASM), and the recommendation is that the diagnosis should be made through a comprehensive assessment of the patient.[8] TF establishes a careful sleep analysis and adequate follow-up altogether as recommended medical practice. Additionally, establishes polysomnography (PSG), considered the gold standard, as a diagnostic test in all those adult patients who, after a comprehensive evaluation, present risks for OSAHS.[9]

Faced with a diagnosis of OSAHS, more attention and guidance are given to conditions of cardiovascular abnormalities such as refractory heart failure, systemic arterial hypertension, angina, and nocturnal arrhythmia.10 With the progressive increase in obesity, OSAHS may contribute to the onset in these patients, alveolar hypoventilation, even during the day, development of pulmonary vascular hypertension, and acute respiratory failure. [11,12]

The effects of upper airway (UAW) obstruction includes ineffective inspiratory efforts, ventilatory pauses, high negative intrathoracic pressures, changes in arterials gases, stimulation of chemoreceptors, and baroreceptors. Which all lead to frequent arousals, increased muscle sympathetic nerve activity, and adverse cardiovascular response. Additionally, these arousals impair sleep architecture, causing daytime sleepiness and several cognitive symptoms.[3] Hypoxia and chronic alveolar and arterial hypercapnia can also lead to pulmonary vascular hypertension with repercussions in both ventricles.[13]

Craniofacial disharmony and UAW anatomy have been referred to as risk factors for OSAHS. For instance, reduced vertical dimension in the anterior region of the face, oropharyngeal space, size of the tongue and soft palate, positioning and size of the mandible, and the length of the upper airway.[14] Strong evidence has been observed for craniofacial characteristics such as reduced pharyngeal space and inferior positioning of the hyoid bone. [15]

Prevalence

A direct relationship between the degree of obesity and the prevalence of obstructive apneas has been reported.[15] prevalence of OSAHS varies between 30% - 40% in overweight individuals, while in those with body mass index (BMI) above 40 kg / m2, the prevalence increases to 90%.[16]

It has been observed that obesity increases pharyngeal collapsibility both due to the mechanical effect of soft neck tissues on the pharynx and the reduction in lung volume that occurs in these patients. In addition, deterioration of neuromuscular control has been linked to the action of adipokines.[17] In this group of individuals, differences in the distribution and metabolic activity of adipose tissue can modify the mechanical and neural components of pharyngeal collapsibility, favoring the occurrence of obstructive apneas. [18]

Conversely, the prevalence of obesity among patients with OSAHS is also high. Several mechanisms linked to sleep deprivation and the discontinuity of metabolism seem to aggravate the state of obesity in patients with OSAHS, through resistance to leptin and increased levels of ghrelin, promoting an increase in appetite and caloric intake and, through insulin resistance, leading to diabetes.[18,19]

The higher prevalence of OSAHS in adult males has been published in the literature. The hormonal difference in testosterone between the sexes has been accounted for this difference.[20] Studies show that testosterone administration increases ventilatory response to hypoxia and hypercapnia. Another point is the inverse relationship between obesity and testosterone. In other words, obese people have low testosterone levels. As well as weight loss leads to an increase in testosterone levels. Furthermore, knowing that obesity is an aggravating risk factor for OSAHS, there is an association between testosterone and apnea, but without association as a causal factor.[21]

There is evidence that the hormone level of progesterone can influence the dilating activity of the muscles of the upper airways. Pre-menopausal women have a great deal of activity in the genioglossus musculature when compared with post-menopausal women and men of the same age.[22] It has been suggested that

progesterone may have a protective role against apnea before menopause. The highest level of progesterone occurs in the luteal phase of the menstrual cycle. During this period, women experience an increase in ventilatory control, pointing to one of progesterone hormone function. Exogenous progesterone has been associated with a small but well-marked, improvement in ventilation during sleep in men and women with apnea.[6,23]

Another important hormone is estrogen, and its administration is associated with a plasma decrease in interleukin, which is elevated in patients with apnea. Therefore, postmenopausal women, using hormone replacement therapy (progesterone and estrogen), have a lower prevalence of OSAHS, as they have two protective mechanisms. However, a controversial result was observed in a study with a control group, which showed that the severity of the disease increased in pregnant patients, a condition characterized by an increased level of estrogen and progesterone.[24]

In a simplistic way, it could be said that the central distribution of fat also accounts for the greater predominance of males in the development of OSAHS, while peripheral adiposity and the absence of the hormone testosterone, possibly, protect women against obstructive apneas. However, in general, the effects of hormones are still uncertain and further studies with better methodological designs are needed to establish better evidence.[25]

Regarding age, a higher prevalence has been associated with older people. Aging contributes to the increase in the prevalence of OSAHS through the role in the progressive increase of upper airway compliance, with a consequent increase in the predisposition to collapse. It is interesting that, with age, the difference in prevalence between the sexes decreases.[7]

OSAHS RISk FAcTORs

Studies show that the individual's body mass reflects a lot on the anatomical components and abnormalities, which occur more frequently in individuals with large neck circumference, contributing to higher rates of OSAHS in obesity.[26] It was found that craniofacial differences including pharyngeal airways smaller, greater volume of soft tissue around the upper airways, less positioning of the hyoid bone and greater volume of the lateral pharyngeal wall can be observed in these patients.

The patency of the upper airways is also influenced by functional factors, such as leptin. In experimental models, leptin has been found to influence sleep architecture and upper airway resistance. Leptin is a peptide produced by adipose tissue that affects satiety and plays a role in metabolic regulation, including glucose and fatty acid metabolism, as well as insulin resistance. Leptin has also been suggested as a risk marker for cardiovascular disease. Syndrome patients have elevated leptin levels, with a positive correlation observed between leptin levels and OSAHS severity.[27,28]

It is proven that resistance to leptin in individuals with obesity and OSAHS makes weight loss difficult and contributes to the worsening of the syndrome.[27]

OSAHS Diagnosis

The presence and severity of OSAHS are generally determined by polysomnography (PSG), an analysis that measures cardio-respiratory parameters during sleep. Respiratory sensors detect reductions in ventilation, which are classified as apneas (almost complete cessation of airflow for 10 seconds), hypopneas (partial decrease in airflow for 10 seconds) or excitations related to respiratory effort (subtle changes in the flow of air). air due to the higher resistance of the upper airways). The apnea-hypopnea index is defined by the sleep time in hours.[29]

The Academy of Sleep Medicine classifies the severity of OSAHS according to the AHI as mild (5-15 events per hour), moderate (> 15-30 events per hour) or severe (> 30 events per hour). Notably, other methods must be considered during the evaluation of the patient.[27]

Dentist Role in OSAHS

Dentist's role in the sleep area is recent and the integration of the discipline in the education of future professionals in the area is still rare. Sleep dentistry is a relatively new area, which emerged promoting a synergy between medicine and dentistry, in which dentists bring their skills and knowledge of the stomatognathic system to deal with the problems that doctors face when having to maintain or promote the airway. unobstructed during sleep.[4]

Dentist has a significant role in the management of patients at risk of OSAHS, a role that is recognized among physicians in the field. For example, a Scottish study in 2010 interviewed doctors who specialize in sleep medicine and 100% of them responded that dentists have an important role in helping patients with snoring and sleep problems, as well as patients with OSAHS.5

There are several ways that the dentist can act in order to collaborate in the identification and referral of patients affected by the syndrome, which increasingly requires multidisciplinary management. Initially, the dentist can help in the identification and referral of suspected cases to a doctor specializing in sleep medicine, who will make the assessment and definitive diagnosis of the syndrome.22 Therefore, the dentist can use anamnesis, clinical examination, questionnaires and request / interpretation of complementary exams under the competence of the dentist, which serve to assist in the identification of patients at risk of developing, or even already affected by the disease.[29]

During anamnesis, the dentist must be attentive to the patients' responses, as symptoms such as daytime sleepiness, tiredness, fatigue, indisposition, reduced attention, reduced memory, depression, decreased reflexes and a sensation of loss of organizational capacity are common complaints that they should serve as a warning for the possible diagnosis of obstructive apneas, when associated with complaints related to night sleep. The dentist can also evaluate the individual using the Epworth Sleepiness Scale, which is based on his or her chances of napping. [30]

During a patient's clinical examination, the dentist has the opportunity to assess craniofacial changes. First, more generally, the dentist sees the patient's oropharynx. In this way, it is possible to assess the size of the tongue and also use the Mallampatti classification, which assesses the patient regarding his predisposition to the disease through the anatomy of the oral cavity.[22]

The evaluation of the maxillomandibular relationship during clinical examination, and complementary imaging, such as cephalometry and tomography of the head, are of fundamental importance in dentistry surgery. And, in the presence of changes, the patient should be referred to the service of oral and maxillofacial surgery for evaluation.[3]

Types of treatment and oral and maxillofacial surgery

The management of patients with OSAHS will depend on the degree of severity of the disease, which can be mild, moderate or severe.6,24

Management options include conservative (non-surgical) and surgical methods for treatment. Some examples of non-surgical methods are behavioral modification, weight loss, medication (pharmacotherapy), continuous positive airway pressure, oral appliance therapy (for example, use of tongue retention devices or use of orthodontic advancement devices or mandibular). Surgical procedures include, for example: tracheostomy, uvulopalatopharyngoplasty, laser-assisted uvulopalatoplasty, stimulation of the genioglossal nerve and orthognathic surgery (rapid assisted maxillary expansion, maxillomandibular advancement, genioplasty).[26,29]

Behavioral factors must be modified; avoiding alcohol and sedatives is recommended for all patients. For some patients, weight loss favorably affects airway patency, minimizing apneic events and snoring. Avoiding the supine position during sleep can reduce the frequency of sleep apnea events in some patients. The role of pharmacotherapy for OSAHS remains uncertain and the effectiveness of the proposed measures with pharmacotherapeutic treatments has not been established.[29]

Among the conservative methods, there are the positive pressure devices (CPAP), which are composed of an air compressor, a tube and a mask that, injecting air into the airways, keeps the pharynx walls apart and prevents the collapse of the airway. It has its main indication in cases of moderate or severe apneas.[31]

The dentist can act in the management of cases of OSAHS, either conservatively or non-conservatively (oral and maxillofacial specialists). For example, the dentist may be called upon by the sleep specialist to make intraoral appliances, which are plates attached to the teeth, which articulate with each other, advancing the mandible and thereby moving the tissues away from the throat, avoiding snoring and sleep apnea. These plaques are easy to adapt, indicated in cases of primary snoring

(without apnea) and in mild and moderate obstructive apneas. These plaques require frequent monitoring, as they can develop important occlusal changes in patients, and have been described as the most conservative alternative in the treatment of snoring and mild sleep apnea.[21]

Orthognathic surgery has emerged as one of the most effective interventions in the treatment of severe OSAHS. Surgical techniques for correcting maxillary and mandibular deficiency are well known and have been used successfully to treat facial skeletal deformities. It is known that mandibular advancement surgery also causes an advance in the muscles of the tongue and the suprahyoid region, as well as advancing the maxilla leads to the repositioning of the soft palate and pharyngeal fleece muscles.32 This conduct leads to an increase in retrolingual space and retropalatal airway, thus improving the pharynx permeability more widely. Maxillomandibular advancement surgery has been shown to be the most efficient surgical treatment for OSAHS.[33] Orthognathic surgery, through mandibular, maxillary or bi-maxillary advances, increases the space of the upper airways (nasopharyngeal and bucopharyngeal spaces). Indicated for moderate and severe cases of OSAHS, orthognathic surgery decreases or zeroes the apnea index, and can be complemented with other surgeries to increase airway perfusion; it can be a cure for OSAHS, for many individuals, bringing quality of life and self-esteem.[17]

Genioplasty / Mentoplasty / Genioglossal Advancement or Genial Tuber Advancement

Surgery in the mentum region also called genioplasty, mentplasty, genioglossal advancement, genital tubercle advancement, or mental advancement is a less invasive orthognathic surgery than bimaxillary surgery, and has been shown to be effective in the management of patients with OSAHS, reducing the rate of hypopnea apnea by 45.7% .3 Genioplasty is a powerful tool, capable of restoring the facial balance between skeletal, soft tissues and dental components of the face.[28,31,35]

A systematic literature review by Song et al., (2017) 3 concluded that chin surgeries can improve both AHI and oxygen saturation.

DISCUSSION

Twelve studies were found, and a total of 224 patients achieved improvement in OSAHS, either in the AHI and / or by Sat 02. Six31,36,37,46,45,43 studies were carried out in the USA, one in Brazil6, one in China40, one in the Czech Republic42, one in Iran38, one in Turkey39 and one in Spain44; the years of publication ranged from 1981 to 2016. Table 1 shows the summary of the studies found: In the studies by Johnson et al., the polysomnographic data of nine obese patients with OSAHS were evaluated before and after mandibular sagittal surgery and lower osteotomy with advancement of the genioglossus, the average oxygen saturation increased from 74.4 ± 4.1% to 87.2 ± 1.4%, a statistically significant increase ($p = 0.01$).[36]

Riley et al. bring the results of treatments based on polysomnographic data, these include analysis of the Index of respiratory disorders and less oxyhemoglobin desaturation. The group was obese, with an average BMI of 29.6 ± 5.8 kg and had severe OSAHS, the mean age of patients was ± 51 years. Altogether, 15 patients, 13 men and 2 women. Based on post-surgical polysomnography criteria, 12 out of 15 patients (75%) experienced improvement in daytime sleepiness and marked improvement in breathing disturbance during sleep.[37]

In the data collected by Tabrizi et al., One can see the evaluative results of 10 individuals (4 men, 6 women) who underwent mandibular osteotomy. The average age of the subjects was 37 years. The mean of the preoperative AHI was 19.9 ± 2.60 and, 1 year after the operation, it was 12.2 ± 2.25. The mean change in AHI was 8.4 ± 2.67.

Tabela 1: Sumário dos resultados

Autor	País	Ano	Titulo	N
Johnston C. et al	EUA	1981	Obstructive sleep apnea in Treacher-Collins syndrome.	9
Rilley, Powel, Guilleminault	EUA	1993	Obstructive sleep apnea syndrome: a review of 306 consecutively treated surgical patients	6
Johnson et al	EUA	1994	Uvulopalatopharyngoplasty and Inferior Sagittal Mandibular Osteotomy with Genioglossus Advancement for Treatment of Obstructive Sleep Apnea	2
Riley et al	EUA	1994	Evaluation of Mandibular Wing Osteotomy in Obstructive Sleep Apnea Cases with Retrognathia	15
Yin et al	China	1994	Mandibular advancement for the treatment of micrognathia with obstructive sleep apnea	15
Lee et al	EUA	1999	Staged Surgical Treatment of Obstructive Sleep Apnea Syndrome	35
Fooltan et al	República Tcheca	2007	Genioglossus advancement and hyoid myotomy in treating obstructive sleep apnoea syndrome – A follow-up study	31
Santos Jr et al	Brasil	2007	Genioplasty for genioglossus muscle advancement in patients with obstructive sleep apnea-hypopnea syndrome and mandibular retrognathia.	2
Tabrizi et al	Irã	2013	Evaluation of Mandibular Wing Osteotomy in Obstructive Sleep Apnea Cases with Retrognathia.	10
Garcia et al	Espanha	2014	Genioglossus muscle advancement: A modification of the conventional technique.	1
Kusçu et al	Turquia	2015	Sole effect of genioglossus advancement on apnea hypopnea index of patients with obstructive sleep apnea	17
Chan & Ducic	EUA	2016	A Simplified, Reliable Approach for Advancement Genioplasty	81
Total SAHOS pacientes manejados com sucesso pela genioplastia				224

N-Número de pacientes; EUA-Estados Unidos da América

Data evaluation showed no correlation between age and AHI. The SAP mean was 9.93 ± 1.01 in the pre-operation and 10.3 ± 1.09 in 1 year after the operation. The data evaluation showed no significant change after the operation. The surgical technique chosen was genioplasty, which was performed in conjunction with maxillomandibular advancement. It is believed that advancing the mandibular base can pull geniohyoid muscles forward in a position that improves airway patency in patients.[38]

CONCLUSION

Based on the studies found in this review, patients with OSAHS can be managed by oral and maxillofacial surgery, in a multidisciplinary way, improving quality of life, as well as improving, in various symptoms in patients with OSAHS who did not obtain control by conventional non-surgical treatments. It was shown that genioplasty in its most varied techniques has been shown to be effective in decreasing AHI and improving 02 saturation in patients with OSAHS.

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