ORIGINAL RESEARCH PAPER

Medical Science

# RAPID MAXILLARY EXPANSION AS THE DEFINITIVE OPTION FOR THE TREATMENT OF SLEEP APNEA / HYPOAPNEA SYNDROME IN CHILDREN. LITERATURE REVIEW

KEY WORDS:

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## INTRODUCTION

Obstructive Sleep Apnea / Hypoapnea Syndrome (OSAHS) has been described as a respiratory sleep disorder, characterized by partial or total obstruction of the upper airway, which distorts normal ventilation during sleep and normal sleep patterns[1]. Its most frequent etiology in children is adenotonsillary hypertrophy[2,3,4], but other causes associated with syndromes and dentomaxillary anomalies are also recognized, such as maxillary compression.

Currently, the gold standard to confirm and evaluate the severity of OSAHS is through the polysomnographic study.[2,5,6.]

The most common treatment in children with OSAHS is adenotonsillectomy; however, other treatments have been studied over time. Less invasive treatment possibilities such as continuous positive pressure ventilation (CPAP) and rapid maxillary expansion. This last correspondence to an increase in the transverse distance of both hemiarcades, through the separation of the middle palatal suture.

It has been described that this procedure could alleviate the symptoms of childhood OSAHS[3,7].

Given all these antecedents, this review aims to collect information on the effect of rapid maxillary expansion in patients with childhood OSAHS.

## OBJECTIVES AND METHODS

The main objective of this research is to describe whether rapid maxillary expansion is a definitive treatment option for OSAHS in children.

Regarding the search methodology to carry out this review, the following keywords were used: "RAPID MAXILLARY EXPANSION", "SLEEP APNEA SYNDROME", "SLEEP APNEA", ORTHODONTIC TREATMENT ", together with the Boolean operators" AND " and "OR".

These terms were entered in different databases to carry out a completed electronic search. The databases used were Pubmed, Epistemonikos, The Cochrane Library and TripData.

## THEORETICAL FRAMEWORK

Obstructive Sleep Apnea / Hypoapnea Syndrome (OSAHS)

OSAHS has been defined by the American Thoracic Society as a breathing disorder characterized by an obstruction of the upper airway that can be prolonged partial (hypopnea) or intermittent total (obstructive apnea) and will distort normal ventilation during sleep and normal patterns of this[1].

Its incidence is highly variable, ranging from 1% to 6 to 12% have been described [2, 3, 5, 8, 9]. Its prevalence is independent of gender and generally occurs between the ages of 2 to 5 years[3,4,6].

The most frequent cause of OSAHS in children is adeno-tonsillar hypertrophy, although it has been proven that the severity of OSAHS does not correlate with the size of the tonsils or adenoids [2,3,4]. It has also been suggested that adeno-hypertrophy -Tonsillar, it would not act as the only cause of the respiratory alterations observed during sleep [5,4,10].

Other causes of OSAHS in childhood are craniofacial anomalies[2, 3, 11] and various syndromes such as: S. de Pierre-Robin, S. Cruzon or S. Down[2,3]. All these pictures can present a reduced pharyngeal space and therefore suffer an obstruction, even with normal-sized tonsils and adenoids[4].

Clinically, the sign that characterizes this syndrome in children is nocturnal snoring, with an incidence between 7 and 9% [12]. However, snoring is not always associated with daytime symptoms, nor with polysomnographic alterations, which may indicate the presence of apneas[13].

The observable nocturnal symptoms are snoring; apneas or respiratory pauses, which are preceded by an increasing ventilatory effort and which could end in an awakening; restless, non-restorative sleep and abnormal sleeping postures to keep the airway patent[2,3,4].

Daytime symptoms can be behavioral, cognitive, and character disturbances, such as hyperactivity and aggressiveness; tiredness; poor school performance; they may even develop an attention deficit syndrome[2,3,14]. On the other hand, the symptoms associated with adeno-tonsillar hypertrophy are mouth breathing, dry mouth, nasal congestion, frequent airway alterations.

Before a child with suspected OSAHS, an anamnesis should be taken to rule out other diseases that are related to sleep, as well as bad habits. An otorhinolaryngological and dental examination should be carried out to assess lymphatic tissue hypertrophy, alterations in the palate or jaw, and evaluate the pondo-stature development[4].

Polysomnography (PSG) is currently the gold standard to confirm and assess the severity of OSAHS[4,5,6.] The most

common and effective treatment in children with OSAHS is tonsillectomy or adenoidectomy. After surgical intervention, polysomnographic alterations are resolved in between 75 and 100% of cases[4,7].

Continuous positive air pressure (CPAP) should be considered an alternative to surgery when children present minimal tonsil tissue, persistence of OSAHS even after adenotonsillectomy[5] or when surgical intervention is contraindicated[1,9].

Orthodontic treatments aim to correct maxillomandibular anomalies, which could be an etiological factor for childhood OSAHS. The techniques consist of fast or slow maxillary distractions through the placement of intraoral appliances. This allows correction of ogival palates and nasal obstructions[15,16].

Untreated childhood OSAHS can produce serious consequences, mainly growth retardation, cor pulmonale and mental retardation[4,13].

***Dentomaxillary Anomalies (DMA)***

A group of anomalies in the development of the jaws, which generally occur together with dental malpositions. Its etiology is multifactorial, having genetic, congenital or systemic factors as predisposing factors as well as local factors.[2.]

DMAs are usually clinically significant variations of the normal fluctuation of growth and morphology[17] and that in most cases they result from a relative discrepancy between the size of the teeth and the bones; or a disharmony in the development of the maxillary bone bases[18].

It is estimated that 50% of the child population is affected by some type of DMA[19].

To facilitate the study of DMA, they have been grouped according to similar characteristics, thus emerging different classifications such as the classification according to Angle, Fisher, Simon and Modified Biogenetics.

The modified biogenetic classification[20], divides the DMA into intramaxillary and intermaxillary anomalies.

Among the intermaxillary anomalies are the anomalies in the transverse direction, which are all those anomalies in the horizontal plane, and that are independent of the intermaxillary relationship in the sagittal and vertical planes. In general, they are due to the lack of growth of the jaws.

There are two types of abnormalities in the transverse direction: the cross bite and the scissor bite.[21].

Within of the etiopathogenesis, two etiological factors are recognized, genetic and bad habits[22.] According to the established diagnosis, two therapeutic objectives can be set: control of bad habits and maxillary expansion. The treatment of these types of anomalies must be early. It is recommended to perform the treatment in the first phase mixed dentition, to achieve an increase in the length of the arch, and also less recurrence is achieved.

***Rapid Maxillary Expansion (RME)***

RME, or disjunction, is performed through the separation of the middle palatal suture, achieving an increase in the apical base, and secondarily the space available for the teeth[21].

In rapid maxillary expansion, important histological changes do not occur at the level of the anchoring teeth, due to the speed with which this procedure is performed, which does not allow the biological reaction of the alveolar bone. Where there are significant changes is in the mid-palatal suture; both halves of the bone appear separate and, between them, distended collagen fibers and a large number of osteoblasts. After disjunction, new bone tissue is deposited in the area of the mid-palatal suture, restoring its integrity at 12 months.

**DISCUSSION**

In this review, nine studies were analyzed, of which one is a randomized clinical trial[23] and 8 are non-randomized clinical trials[6,24,25,26,27,28,29,30]. All of these used children with OSAHS, which was diagnosed by polysomnography (AHI> 1). In most studies the children underwent EMR, and in some they underwent EMR and AT. In addition, the AHI, symptoms, sleep parameter and oxygen saturation were evaluated before, after and in the long term of maxillary expansion.

In 2008[23], Christian Guilleminault aimed to investigate whether EMR can be offered as an alternative to TA surgery, as well as whether both therapies are required, and if so, to determine the order in which they should be applied.

In 2009[24] a study was carried out by Silvia Miano et. al, where children with OSAHS were treated by EMR for 12 months. AHI decreased significantly after expansion and induces a reduction in the awakening index.

In 2010[25], Paola Pirelli evaluated children who presented oral breathing, snoring, histories of nocturnal apnea and clinical signs of malocclusion (maxillary compression). After EMR and after 4 months of treatment, AHI decreased and SaO2 increased.

In 2011 María Villa et. Al[26], evaluated 14 children of which 10 underwent EMR. The diagnosis of OSAHS was by PSG. After 12 months of expansion after removal of the device, the AHI decreased significantly and the SaO2 improved.

In 2012, Pirelli[27] performed an evaluation and diagnostic confirmation of OSAHS using PSG. EMR was performed first and AT second stage. Finally, the evaluation after the second stage of treatment showed a complete remission of symptoms in most of the patients, a normalization of the AHI and an increase in SaO2.

In 2013, María Pía Villa et al[28] carried out a study whose objective was to evaluate the result of surgical (TA) and orthodontic treatment. Some underwent AT, others underwent EMR, and still others underwent both therapies. OSAHS resolved completely in 4 of the 5 children who underwent both treatments.

In 2015, Paola Pirelli et al[29] wanted to evaluate the long-term efficacy of EMR in a group of children with OSAHS. A PSG and CT were performed prior to EMR, after treatment and 12 years later. At the end of the expansion, changes were reported in the AHÍ and changes in the initial SaO2 of 78.9% and final of 95.1%.

In 2015, Villa[30] conducted a study with children who presented signs of malocclusion in conjunction with adenotonsillary hypertrophy and symptoms of OSAHS. It included the follow-up of patients who underwent EMR after 12 months of treatment, a significant decrease in AHI could be observed, as well as in the number of awakenings and with an increase in total sleep time and SaO2.

Finally, in 2017[6], Buccheri recruited 11 patients with clinical signs of malocclusion and symptoms of OSAHS; twelve months after the application of the intraoral device, the AHI decreased, resulting in 5 fully healed patients (AHI <1), 5 moderate improvements ( AHI <5) and 1 patient without any changes after treatment.

The RME in most studies, reduces the symptoms of OSAHS, as well as the AHI parameter. Improvements in total sleep time,

SaO2, and decreased arousals have also been shown. In addition, it was observed that, to achieve resolution of OSAHS, patients needed to be intervened by means of RME and Tonsiloadenectomy therapy, with no differences in the order in which they were performed.

Therefore, there is consensus among the studies that the treatment of OSAHS cannot be performed solely by RME, but must be planned in an interdisciplinary manner. It should be evaluated on a case-by-case basis and determining the etiology involved, to define the treatment to use.

## CONCLUSIONS

RME is an effective procedure for the treatment of transverse DMA and has recently been considered for the treatment of OSAHS, since it causes irreversible changes in the oral cavity and airways. It has been shown to decrease the symptoms of OSA, as well as the parameter that defines it, which is the AHI. But this decrease does not in all cases achieve the resolution of the syndrome. Improvements in total sleep time, SaO2 and decreased awakenings have also been found.

Maxillary expansion could be considered a definitive treatment for OSAHS, only and when the clinical conditions present as an isolated transverse DMA without hypertrophy or another etiological factor. Even so, the evidence reviewed presents a high risk of bias, therefore it is necessary to carry out more studies focused on randomized clinical trials, long-term prospective observational studies, and systematic reviews with meta-analyzes. These should be aimed at comparing treatments in patients with or without hypertrophy, and their response to single or combined maxillary expansion with other treatments such as Tonsiloadenectomy.

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