Obstructive sleep apnea syndrome (OSAS) in children and the contribution of orthodontist in the treatment

Amel Belkhiri

DOI: https://doi.org/10.22271/oral.2021.v7.i2d.1210

Abstract
Obstructive sleep apnea syndrome (OSAS) in children is a pathology that can lead to serious complications. It is therefore imperative to detect this disease as early as possible in order to be able to treat it. The narrowing of the airways responsible for OSAS is frequently the result of enlarged lymphoid tissue in children. Surgical removal of the adenoids and tonsils is the first line of treatment. However, this surgical treatment is not sufficient in young patients with maxillary hypodevelopment with narrow, arched palate, and mandibular retrusion or hyperdivergence. With two types of devices (mandibular propulsion orthosis, distractor), the orthodontist can effectively contribute to the treatment of pediatric OSAS.

Keywords: Obstructive sleep apnea syndrome, children, diagnosis, polysomnography, treatment, rapid palatal expansion, mandibular propulsion orthosis

1. Introduction
Obstructive Sleep Apnea Syndrome (OSAS) is a frequently occurring childhood malady, whose incidence is largely underestimated. OSAS is thought to affect between 2 to 3.5% of children, with a peak incidence between three and five years [2] and a very slight male preponderance near puberty [15]. This syndrome is caused by intermittent, partial, or complete obstruction of the upper airways (pharynx and larynx) during sleep. This results in a decrease (hypopnea) or stop (apnea) of breathing for at least 10 seconds. When it is complete and prolonged, the obstruction of the airways causes breathing efforts which can lead to brief awakenings called "micro awakenings" which disrupt the quality of sleep [9]. OSAS causes serious complications without treatment: pulmonary dysfunction, heart failure, neurocognitive deficit with delayed learning, even disturbances in mood and attention and a break in the growth curve. It is therefore essential that patients with OSAS are identified and treated.

2. Risk factors for children in the obstructive sleep apnea syndrome
OSAS is associated with certain conditions such as asthma, ear, nose, and throat infections (recurrent ear infections, rhinitis, sinusitis), obesity, drepanocytic anemia, and certain congenital afflictions such as trisomy 21, neuromuscular problems and maxillo-facial malformations [12, 15]. In the case of major skeletal malformations, responsible for a three-dimensional narrowing of the middle or lower facial mass massive facial, severe OSAS is found, in particular for orofacial clefts. Much more discrete maxillofacial anomalies, far from these syndromic dysmorphoses, have been associated with OSAS. They can simply involve a decrease in space such as retrognathia (jaw retracted), brachygnathia (short jaw), endognathia (narrow jaw in the transverse direction) and mandibular hyper divergence characterized by a vertical lengthening of the mandible. They generate a descent and / or a retreat of the lingual mass, inserted at the level of the chin.
symphysis, which reduces the oropharynx. The dysmorphosis of these young patients is frequently accompanied by dental malocclusions, because on the one hand the insufficient development can cause a lack of space for the teeth or be accompanied by dental lags, and on the other hand the child adopts a lingual and mandibular posture of compensation which is at the origin of secondary anomalies.

3. Symptomatology of OSAS
Usually, OSAS is first discerned through assessment of clinical symptoms, a medical history, a morphological and functional evaluation of the upper airways, and finally by the indispensable polysomnographic examination, which, alone, can provide an irrefutable objective confirmation of the diagnosis.

3.1 Nocturnal symptoms

3.1.1 Snoring
Symptom most commonly associated with OSAS. It takes place when the airways are partially obstructed. When air passes through this limited space, it vibrates the soft tissues of the throat, uvula and soft palate. These vibrations create a sound that we call snoring. It is the first call sign, both for parents and for the medical team. In 96% of OSAS cases, this snoring is found, but it should be noted that simply noisy breathing, or a complete absence of snoring, may accompany certain confirmed OSAS [10].

3.1.2 Pauses in respiration (apnea)
An apnea is defined as a total cessation of air flow for at least 10 seconds. In children, the pathological threshold is determined from one apnea per hour of sleep. OSAS is accompanied by frequent apneas, unknown to the patient, which end up worrying those around him. These apneas can cause sudden awakenings with sometimes a feeling of suffocation.

3.1.3 Night sweats
They are a good indication of increase of the blood level of carbon dioxide and bear witness to the hypoventilation that accompanies apnea. They can occur daily and be abundant.

3.1.4 Nocturnal mouth breathing (which is sometimes occurs in the daytime)
It is caused by an obstruction in the nasal air passageways. In healthy subjects, 70% of the inspired air passes through the nasal passages; in many children with apnea, mouth breathing will provide rescue breathing [3, 8, 15].

3.1.5 Abnormal sleeping positions
By setting their heads in a hyper-extended position thus limiting lowering of the base of the tongue children can improve air flow [15, 17]. Children also adopt this position when they nod off during long automobile trips; when they continue this behavior during waking moments it may become a chronic statural anomaly. We can also observe in children with apnea: a sleep is agitated with frequent position changes, shaking of the lower limbs, night terrors, recurrent nightmares, confusion in waking periods, bruxism, episodes of somnambulism or somniloquy (emission of sounds during sleep), repeated nocturnal arousals, nocturnal micturation or enuresis (involuntary and unconscious urination during sleep), and, with nursing infants, nocturnal crying.

3.2 Diurnal symptoms
During the day, compared to children of the same age, we can observe:
- Diurnal behavioral disorders with pseudo-hyperactivity, an increase in psychiatric disorders [16];
- Difficulty concentrating and learning, disturbances in executive functions with reduced performance on intelligence tests, difficulties in school [17]. We thus note a risk of OSAS multiplied by 6 or 9 among the last 3 pupils of the class [11];
- Drowsiness, quite rare, affecting only about 20% of children with OSAS.

4. Diagnostic
Since none of the symptoms already seen are specific, the diagnosis of OSAS is made by recording the sleep.

4.1 The polysomnography (PSG)
The polysomnography makes registrations of different stages of sleep derived from electro-encephalographic graphs and an electro-oculogram that discerns specific phases of arousal and sleep, notably REM, or Rapid Eye Movement, and also records changes in leg placement from a position sensor with electromyograms along with a night-long electrocardiogram. We can also associate a microphone (snoring) or other sensors. This examination makes it possible to detect with great precision sleep breathing disorders and to define their type (central apnea, obstructive, mixed, hypopnea). It also makes it possible to determine the structure of sleep and to quantify the time spent in the different stages of sleep.

4.2 The ventilation polygraphy (VP)
The VP measures cardio-respiratory parameters including ventilation deficit, oxyhemetics, and cardiac rate. It does not register brain activity, leg and eye movements. This examination, the sensitivity of which is slightly lower than polysomnography, however, makes it possible to confirm the diagnosis of OSAS when the clinical suspicion is strong.

4.3 The oximetry
It allows the oxygen level in the blood to be evaluated using a digital sensor. Alone is used less and less for the diagnosis of OSAS, given its significantly lower sensitivity and the inability to differentiate between obstructive and central apnea.

5. Treatment of obstructive sleep apnea syndrome in children

5.1 Surgical treatment
The treatment of choice for childhood OSAS is adenotonsillectomy, which effectively treats between 53 and 100% of cases. It will have the effects of lowering respiratory resistance, harmonizing facial growth by promoting nasal breathing, and improving behavioral and cognitive disorders if they are present. There is the problem of medium and long-term follow-up on the possibility of recurrence of OSAS. Indeed, some patients may have persistent ventilatory disorders after the operation (from 47% to 75% for the study by Tauman[14] on a population of obese children) or recurrence of symptoms a few years after being cured (for 14.5% of them according to Guilleminault et al [17]). These results underline, on the one hand, the importance of an exploration of postoperative sleep
and, on the other hand, the importance of cofactors such as obesity and craniofacial abnormalities.

5.2 Orthodontic treatment
Orthodontic devices would allow an expansion of the maxillary skeleton and an interesting mandibular anteriorization in young apneic patients with dysmorphosis [9]. They can be offered as second-line after failure of treatment with adeno-tonsillectomy, or as first-line for cases of moderate OSAS in the absence of obvious lymphoid organ hypertrophy.

Beyond this mechanical approach, the restoration of spontaneous nasal ventilation is absolutely sought after treatment, whether surgical or orthopedic.

Care should be taken, especially for young patients who may have developed the habit of ventilating through the mouth, even in the absence of any obstacles. To avoid recurrence, it may be worthwhile to offer real rehabilitation through ventilatory exercises. These exercises can be supported by small devices called “functional educator” [Fig. 1] which guide the tongue in a high position, keeping lips joined.

5.2.1 Rapid maxillary disjunction
The association between palatal morphology (arched palate) and oral ventilation is well known to orthodontists. Its explanation is based on the morphogenetic role of the tongue which, faced with chronic nasal obstruction, has adopted a low position over the long term to allow an oral ventilation supplement. The low position of the tongue no longer stimulates the maxillary sutures or counteracts the functional centripetal pressures exerted by the cheeks, which contributes to the narrowness and depth of the palate.

When faced with a developmental insufficiency in the nasal stage, it is not uncommon to find associated narrowing of the dental arch, or even a lack of support for the cheekbones. These forms of narrow dental arches are often associated with dental crowding and an inversion of dental relations in the transverse direction (or linguoclusion); the maxillary arch should, under normal conditions, circumscribe the mandibular arch like the lid of a box.

Rapid maxillary disjunction is an orthopedic treatment that seeks to separate the median intermaxillary and interpalatal sutures, which are still fibrous in children, to “catch up” to a deficit in sutural growth. The repercussions of rapid maxillary disjunction on ventilation have been described even before the identification of OSAS in children. It would widen the nasal passages and improve nasal ventilation.

Secondly, creating space for the tongue would allow it to free up the oropharynx.

Treatment can be started from the age of four to five, when all of the temporary teeth have erupted, and when the child can be cooperative.

The MSE breaker (Maxillary Skeletal Expander) is an individual device, made from dental impressions processed in the laboratory, which is then sealed by the orthodontist on the patient’s upper molars. The device consists of anchoring systems on the teeth [Fig. 2] and a median cylinder which are connected by rigid arms. Once fixed in the mouth, the MSE breaker is activated daily, using a small wrench, from a quarter to half a millimeter, for 15 days to three weeks. The patient initially feels discomfort due to the bulkiness of the device, then tension with each turn of the key, but activation is not painful.

An expansion of 5 to 8mm is thus obtained, depending on the initial transverse deficit. It is manifested by the opening of a large space (diastema) between the upper incisors.

After the desired expansion has been achieved, the cylinder is blocked, allowing the spontaneous ossification of the disjointed suture, which stabilizes after three to six months. The median diastema usually closes spontaneously during this phase of contention.

Fig 1 : Functional educator

Fig 2: The MSE breaker.

5.2.2 Mandibular advancement devices (activators)
In adult OSAS cases, mandibular advancement devices (orthoses) advance the mandible and tongue during the night and thus reduce the risk of pharyngeal collapse during sleep. These same orthoses constitute a treatment route for pediatric OSAS [16], provided the child has insufficient development of the mandible, adopting a position set back to the maxilla.

They then seek to correct the retrusion mandibular, anteriorizing the insertions of the tongue and normalizing dental reports. These same orthoses constitute a treatment route for pediatric OSAS [16], provided the child has insufficient development of the mandible, adopting a position set back to the maxilla.

They then seek to correct the retrusion mandibular, anteriorizing the insertions of the tongue and normalizing dental reports.

The mandibular advancement devices (or the activator, or Herbst rods [Fig. 3]) is an individuated device, made by an orthodontist from dental impressions treated in the laboratory in resin or thermostatic material. It is a removable device, worn at home and during sleep, and removed at mealtimes.

Fig 3: Herbst rods.

5.3 Other therapeutic alternatives
Therapeutic alternatives are proposed for patients who are not candidates for ODF, or in whom an obstruction persists: drug treatment (local anti-inflammatory drugs, or antihistamines),

http://www.oraljournal.com
revision surgery (intervention on the basis of the tongue, etc…), management of obesity or replacement therapy, by setting up continuous positive airway pressure CPAP [14]. It is a small, portable “compressor” that fits next to the bed and delivers positive airway pressure through a hose and a nasal or face mask. This device induces an increase in the pressure inside the pharynx and thus prevents its obstruction during inspiration.

This treatment makes it possible to normalize nocturnal breathing and suppress micro arousals in almost all patients suffering from obstructive apnea. Its undesirable effects remain significant and include among others: sensations of suffocation, skin lesions on the support areas of the mask, rhinitis, nasal and oral dryness and conjunctivitis in the event of an air leak in the eyes. The alteration in body image due to wearing a mask on the face is also not to be overlooked.

6. Conclusion
Collecting the symptoms of OSA in children, which are sometimes very discreet, neglected or unrecognized by parents, is essential to lead to polysomnography, emphasizing the role of the clinician, and the quality of his anamnesis. The orthodontist is included in the development of the therapeutic plan after the diagnosis of OSA, and in the control of the restoration of physiological nasal ventilation.

7. References
1. Ali NJ et al. Snoring, sleep disturbances and behaviour in 4-5year olds. Arch Dis Child 1993;68:360-366.
2. Cohen-Levy J. Traitements orthodontiques dans le syndrome d’apnées obstructives du sommeil pédiatrique. Médecine du sommeil 2011;8:61-68.
3. Fitzpatrick MF. Effect of nasal or oral breathing route on upper airway resistance during sleep. Eur Respir J 2003;22:827-832.
4. Gozal D. Sleep disordered breathing and school performance in children. Pediatrics 1998;102:616-620.
5. Guilleminault C, Stoohs R. Chronic snoring and obstructive sleep apnea syndrome in children. Lung 1990;168(Suppl.):909-912.
6. Guilleminault C, Pelayo R, Leger D, Clerk A, Bocian RC. Recognition of sleep-disordered breathing in children. Pediatrics 1996;98:871-882.
7. Guilleminault C, Li KK, Khramstov A, Pelayo R, Martinez S. Sleep disordered breathing: surgical outcomes in prepubertal children. Laryngoscope 2004;14:132-137.
8. Halbower AC et al. Childhood obstructive sleep-disordered breathing. Chest 2007;132:2030-2041.
9. Heinzer R, Aubert JD. Le syndrome d’apnées obstructives du sommeil. Forum Med Suisse 2007;7:686-691.
10. Lumeng JC, Chervin DR. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc 2008;5:242-252.
11. Lumeng JC et al. Epidemiology of pediatric obstructive sleep apnea. Proc Am Thorac Soc 2008, 242-252.
12. Marcus CL, Rosen G, Ward SL, Halbower AC, Sterni L, Lutz J et al. Adherence to and effectiveness of positive airway pressure therapy in children with obstructive sleep apnea. Pediatrics 2006;117:442-445.
13. Pételle B, Fleury BF, Cohen-Lévy J. Traitement chirurgical du syndrome d’apnées du sommeil. Rev Orthop Dento Faciale 2009;43:317-333.
14. Tauman R, Gulliver TE, Krishna J, Montgomery-Downs HE, O’Brien LM, Ivanenko A et al. Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. J Pediatr 2006;149:803-808.
15. Seailles T, Couloigner V, Cohen-Lévy. Savoir dépister le syndrome d’apnées obstructives du sommeil. Rev Orthop Dento Faciale 2009;43:261-277.
16. Villa MP, Bernkopf E, Pagani J, Broia V, Montesano M, Ronchetti R. Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion. Am J Respir Crit Care Med 2002;165:123-127.
17. Wilson SL. Upper airway patency in the human infant: influence of airway pressure and posture. J Appl Physiol 1980;48:500-504.
18. Zuconi M, Caprioglio A, Calori G. Craniofacial modifications in children with habitual snoring and obstructive sleep apnoea: a case-control study. Eur Respir J 1999;13:407-411.