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# Prevalence of malocclusion in preschool and primary school children with habitual snoring and Sleep-Disordered Breathing

ABSTRACT

***Aim*** *Our research aimed to find out whether it was possible to establish a correlation between instrumental polysomnographic variables in children with Sleep Disordered Breathing (SDB) and their clinical dentofacial records.*

***Materials and methods*** *197 children, 116 (59%) male and 81 (41%) female, age range 0 to 12 years, with a clinical history highly suggestive for SDB were enrolled. All patients underwent full-night POLY-MESAM® examination and a specific clinical orthodontic evaluation. A comparison between polysomnographic variables and clinical orthodontic variables was made.*

***Results and conclusion*** *OSAS can not be diagnosed by the paediatric dentist only by performing a single clinical orthodontic examination of oral cavity, but a polysomnography wis essential and, if this should point out any pathology referring to SDB, a cranial lateral cephalometry will then be required.*

***Keywords:*** *Malocclusion; Obstructive Sleep Apnoea Syndrome; Primary snoring; Quality of life; Sleep Disordered Breathing.*

## Introduction

Nose represents the main natural passageway for air flow. But when such a way turns out to be impaired and breathing becomes too difficult, oral respiration, defined as a pathological lasting alteration in the respiratory phase, may set in. Nasopharyngeal obstruction, as a predisposing factor towards Obstructive Sleep Apnoea Syndrome (OSAS) in paediatric age, influences the child's head posture since it alters the breathing pattern [Tarvonen and Koski, 1987].

The mandible undergoes adaptation processes increasing divergence with clockwise growth of its ramus to create an oropharyngeal passageway for air; the condyle modifies its growth direction just according to mandibular postrotation [McNamara, 1981]. It is possible to record a supereruption of the molars that creates premature contact of the mandible and increases postrotation [Miles et al., 1996]. Such mechanism is often connected to mandibular retrognathism and micrognathia which worsens the already existing narrow opening of the respiratory skeletal pharyngeal spaces; these ones, if associated, for example, with a prolonged, severe adenoid and tonsil hypertrophy, represent a considerable risk of apnoea [Cozza et al., 1990].

Under normal conditions tongue muscles influence and regulate the upper jaw morphogenesis. Conversely, when breathing through the mouth, the tongue assumes a forward and downward position in order to move its dorsum and the tonsillar tissue away from the posterior pharyngeal wall and it does not perform its modelling function on the palate; for this reason the teeth and maxilla are deprived of their natural support. The consequent "stretching" of lips and cheeks causes a centripetal force that inhibits a correct transverse maxillary growth and predisposes towards lip incompetence [Caprioglio et al., 1994]. Keeping the mouth open favours all these modifications that account for the onset and persistence of the so-called *adenoid facies* [Defabjanis, 2003]. This results in a characteristic face shape and profile: long narrow face, mouth-opened appearance, reduced dimension of the upper lip, small and hypotonic nostrils because of short use, narrow palate, contracted maxillary arches with frequent skeletal asymmetries and both skeletal and dental malocclusion [Bresolin, 1984].

For what concerns skeletal maxillomandibular structures, patients with OSAS fall chiefly within Class I with an increase of ANB angle and consequent decrease of SNB angle, as pointed out by cephalometric examination [Zucconi et al., 1999].

It seems that the earlier the causative factor, i.e. oral breathing, occurs the more severe the skeletal alterations become. It is therefore essential that such patients be treated as soon as possible. The relationship between the way of breathing and the development of the stomatognathic system is not simply a cause-and-effect link but rather a whole of complex interrelationships between environmental and genetic factors. Many experimental studies on primates reveal that a correct growth is connected to nasal breathing, whereas an impaired growth is correlated to oral respiration [Yamada et al., 1997].

The absence of a single direct causal link deserves notice, but above all it is important to stress the presence of a vicious circle that must be interrupted through a different diagnostic approach targeted to each clinical picture.

The particular growth of the craniofacial region, which ensues from a narrow passageway of the upper respiratory tract, may be explained according to the "soft tissues stretching" hypothesis [Solow and Kreiborg, 1977; Solow et al., 1996], which suggests the idea of a connection of six factors linked to form a vicious circle. According to this hypothesis obstruction of the upper airways, such as obstruction due to adenotonsillar hypertrophy, would lead to a change in head position causing hyperextension as a counterbalance aimed to warrant an adequate air flow. Consequently cephalic hyperextension would stretch face and neck soft tissues giving rise to forces able to modify facial morphology.

The present research aimed to find out whether it was possible to establish a correlation between instrumental polysomnographic variables in children with Sleep Disordered Breathing (SDB) and their clinical dentofacial reports to make an orthodontic diagnosis avoiding conventional radiographic exposures.

## Materials and methods

A sample of 197 children, 116 (59%) male and 81 (41%) female, age range 0 to 12 years, with a clinical history highly suggestive for SDB were enrolled in the study during the period from January 2006 to July 2010. They were split according to their age as follows.

- First infancy (16%): 0-2 years (31 children, 18 males and 13 females).
- Childhood (68%): 3-5 years (134 children, 82 males and 52 females).
- School age (16%): 6-12 years (32 children, 16 males and 16 females).

The characteristics of this patient sample were: average age 4.12 years ± 2.08 SD, family history of snoring in 100 of 197 cases (51%), habitual snoring in 188 of 197 cases (95%), reported apnoea in 158 of 197 cases (80%), daytime mouth breathing in 169 of 197 cases (86%), recurrent upper airway infections in 118 of 197 cases (60%), daytime irritability in 87 of 197 cases (44%), excessive daytime sleepiness in 53 of 197 cases (27%), failure to thrive in 28 of 197 (14%).

All patients underwent full-night POLY-MESAM® (MAP; Martinsried; Germany) [Verse et al., 2000; Castronovo et al., 2003]. The polysomnograph for home recording was positioned at 18.00 p.m. in the Paediatric Department - Sleep Disorders Centre and then children were sent home in order to record their sleep in a quiet and friendly environment. The recording was scheduled to start at 22.00 p.m to 07:00 a.m. Children's compliance was good.

POLY-MESAM® is an ambulatory diagnosis system for early recognition of SDB. In the standard setting, it has seven channels able to measure the following signals:

- For each child the following polysomnographic parameters were taken into account as they were regarded as the most significant variables to diagnose sleep respiratory pathology [Guilleminault et al., 1984].
  1. Oxygen Desaturation Index (ODI).
  2. Mean Peripheral Oxygen Saturation (% $SpO_2$ m).
  3. Minimum Peripheral Oxygen Saturation (% $SpO_2$ min).
  4. Hand-scored snoring (MS).
  5. Heart Rate Variation Index (HRVI phase/h).
  6. Heart Rate Variation Index (HRVI %).
  7. Mean Heart Rate (MHR).
  8. Heart Rate Standard Deviation (HRSD).
  9. Mobility Index (MI).
  10. Sum of Mobility Index (Sum of MI).

  According with International guidelines these indexes are pathologic in case of: ODI 5, $SaO_2$ m≤92% and $SpaO_2$ min 75.

According with polysomnographic results, patients were grouped into four new categories: Primary Snoring, Mild OSAS, Moderate OSAS and Severe OSAS.

Each patient underwent specific clinical orthodontic evaluation. The following parameters were evaluated.

- Aesthetic analysis
  Face (F): low angle, normal angle, high angle.
  Lip Competence (LC): competent lip, incompetent lip.
  Nasolabial Angle (NLA): <90°, 90° to 105°, >105°.
- Functional analysis
  Visus Musculature (V. MUSC): hypotonic, normotonic, hypertonic.
  Deglutition (DEG): correct, incorrect.
  Mandibular midline deviation during opening and closing movement (ML dev.): present, absent.
  Premature contact and possible mandibular displacement (PREC): present, absent.
- Intraoral examination
  Angle molar Class (AMC): I, II, or III Class.
  Angle Cuspid Class (ACC): I, II or III Class.
  Mid Line (ML), checking the relation between upper maxillary arch midline and mandibular arch midline: correct, incorrect.
  Crossbite (CB): present, absent.
  Overjet (OVJ): reduced, normal, increased.
  Overbite (OVB): reduced, normal, increased.

### *Statistical analysis*

A descriptive statistical analysis of the sample was employed and sensitivity of each orthodontic variable was calculated.

## Results

The findings of the instrumental research, by POLY-MESAM® pointed out prevalence of different pathological patterns in the sample of 197 children suffering from SDB.

Primary Snoring was noticed in 38 children (19% of cases); OSAS was found out in 159 children (81% of cases), classified, according to the degree of severity, as mild in 75 children (38%), moderate in 48 children (25%) and severe in 36 children (18%) (Fig. 1). Diagnosis of OSAS was stated in the following values: ODI ≥5, $SpO_2$ m≤92% and $SpO_2$ min≤75 [Bearpark et al., 1995]. Afterwards, the sample distribution was evaluated according to age and the outcome was as follows (Table 1).

- Primary snoring: it was noticed in 3 children (10% of cases) during infancy, in 26 children (20%) during childhood and in 9 schoolchildren (28%).
- Mild OSAS: it was detected in 11 children (35%) during infancy, in 51 children (38%) during childhood

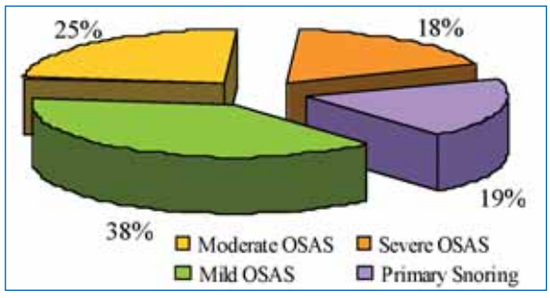


**FIG. 1 -** *Prevalence of sleep disorders of breathing in the tested sample-instrumental diagnosis.*

| Subjects N° | Primary Snor. 38 | Mild 75 | OSAS Moderate 48 | Severe 36 | Total 197 |
|---|---|---|---|---|---|
| ODI | 2.3 | 6.8 | 13.0 | 24.0 | 10.6 |
| SpO2 m (%) | 97.2 | 96.7 | 96.3 | 95.1 | 96.4 |
| SpO2 min (%) | 87.1 | 81.6 | 79.1 | 67.3 | 79.4 |
| MS | 20.9 | 43.2 | 50.3 | 73.1 | 46.0 |
| HVI phase/h | 15.8 | 18.0 | 17.7 | 20.1 | 17.8 |
| HVI % | 38.5 | 37.5 | 43.0 | 43.8 | 40.0 |
| MHR | 83.6 | 89.6 | 89.0 | 100.0 | 90.2 |
| HRSD | 13.8 | 14.1 | 13.1 | 15.6 | 14.0 |
| MI | 5.8 | 8.4 | 9.3 | 10.4 | 8.4 |
| Sum of PC | 43.4 | 61.7 | 65.9 | 86.2 | 63.6 |

**TABLE 2 -** *Mean polysomnographic values of sleep disorders of breathing classified according to severity*

and in 13 children (41%).

- Moderate OSAS: it was found out in 6 children (20%) during infancy, in 34 children (25%) during childhood and in 8 children (25%)
- Severe OSAS: it was found out in 11 children (35%) during infancy, in 23 children (17%) during childhood and in 2 children (6%)

Table 2 reports the mean values of the polysomnographic parameters according to the degree of severity of respiratory sleep disorder. As Table 2 indicates, our study found out correspondence between these data and the employed diagnostic criteria. The more severe the disease becomes the higher mean values of ODI, MS, HVI in phase/h and percent, MHR, HRSD, MI and sum of PC rise; at the same time the mean values of $SpO_2$ m and $SpO_2$ min in percent decrease.

| | | | Infancy subjects N° | | Childhood subjects N° | | School subjects N° | |
|---|---|---|---|---|---|---|---|---|
| Primary Snoring | 38 | 19% | 3 | 10% | 26 | 20% | 9 | 28% |
| Mild OSAS | 75 | 38% | 11 | 35% | 51 | 38% | 13 | 41% |
| Moderate OSAS | 48 | 25% | 6 | 20% | 34 | 25% | 8 | 25% |
| Severe OSAS | 36 | 18% | 11 | 35% | 23 | 17% | 2 | 6% |
| Total | 197 | | 31 | | 134 | | 32 | |

**TABLE 1 -** *Distribution of sleep disorders of breathing in the tested sample according to age.*

| Aesthetic analysis | | | | | |
|---|---|---|---|---|---|
| F | | LC | | NLA | |
| low angle | 11% | competent lip | 75% | < 90° | 7% |
| normal angle | 73% | incompetent lip | 25% | 90° to 105° | 82% |
| high angle | 16% | | | >105° | 11% |
| **Functional analysis** | | | | | |
| V.MUSC | | DEG | | MI. dev | | PREC | |
| hypotonic | 18% | correct | 72% | present | 10% | present | 11% |
| normotonic | 78% | incorrect | 28% | absent | 90% | absent | 89% |
| hypertonic | 4% | | | | | | |
| **Intraoral examination** | | | | | | | | | |
| AMC | | ACC | | ML | | CB | | OVJ | | OVB | |
| Class I | 74% | Class I | 72% | correct | 80% | yes | 25% | red. | 10% | red. | 18% |
| Class II | 22% | Class II | 22% | incorrect | 20% | no | 75% | norm. | 61% | norm. | 64% |
| Class III | 4% | Class III | 6% | | | | | incr. | 29% | incr. | 18% |

**TABLE 3 -** *Prevalence of facial features on clinical orthodontic examination.*

Table 3 shows the frequency rates of face characteristics observed during orthognatodontic assessment and divided into their respective analyses: aesthetic, functional and intraoral; it is possible to note how these frequency rates of face features maintain their percentage close to the mode and how, on the other hand, pathologic aspects represent only a low percentage in the tested sample. The data relating to respiratory sleep disorders, grouped according to severity, and clinical dentofacial reports were crosschecked (Table 4). As Table 4 shows, a worsening of the clinical orthodontic pattern did not mean a concomitant increase in the degree of severity of

| | F | | LC | | NLA | | V.MUSC | | DEG | | MI.dev | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Total (197 children) | low angle | 11% | competent | 74% | < 90° | 7% | hypotonic | 18% | correct | 72% | present | 10% |
| | normal angle | 73% | incompetent | 26% | 90°-105° | 82% | normotonic | 78% | incorrect | 28% | absent | 90% |
| | high angle | 16% | | | > 105° | 11% | hypertonic | 4% | | | | |
| Primary snoring (38 children) | low angle | 13% | competent | 74% | < 90° | 3% | hypotonic | 16% | correct | 63% | present | 13% |
| | normal angle | 66% | incompetent | 26% | 90°-105° | 89% | normotonic | 84% | incorrect | 37% | absent | 87% |
| | high angle | 21% | | | > 105° | 8% | hypertonic | 0% | | | | |
| Mild OSAS (75 children) | low angle | 12% | competent | 79% | < 90° | 7% | hypotonic | 16% | correct | 66% | present | 12% |
| | normal angle | 75% | incompetent | 21% | 90°-105° | 84% | normotonic | 76% | incorrect | 34% | absent | 88% |
| | high angle | 13% | | | > 105° | 9% | hypertonic | 8% | | | | |
| Moderate OSAS (48 children) | low angle | 10% | competent | 69% | < 90° | 4% | hypotonic | 21% | correct | 83% | present | 6% |
| | normal angle | 75% | incompetent | 31% | 90°-105° | 79% | normotonic | 79% | incorrect | 17% | absent | 94% |
| | high angle | 15% | | | > 105° | 17% | hypertonic | 0% | | | | |

**TABLE 4 -** *Correlation of levels of severity of SDB with clinical orthodontic assessment.*

| F | LC | NLA | V.MUSC | DEG | MI.dev | PREC | AMC | ACC | ML | CB | OVJ | OVB |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 26,39% | 24,87% | 17,76% | 21,82% | 27,91% | 9,64% | 11,16% | 22,84% | 20,81% | 19,79% | 24,36% | 39,59% | 36,04% |

**TABLE 5 -** *Calculation of orthodontic variables sensitivity.*

respiratory sleep disorders. Sensitivity of each analysed orthodontic parameter was calculated (Table 5). Face symmetry, lip incompetence, deglutition, canine and molar class, crossbite, overjet and overbite are the parameters with the highest sensitivity. A score was computed by using all three parameters that are the most important ones for both their sensitivity and accuracy in the clinical examination of the children sample: crossbite, overjet and overbite; if we consider the sum of these three parameters and at least one of them turns out to be pathologic from a clinical point of view, we have 61.42% of probability to find out a pathologic patient from an instrumental point of view.

## Discussion

Observing the distribution of all 197 children according to sex no significant difference was pointed out between males (116 children equal to 59%) and females (81 children equal to 41%) confirming the studies quoted in literature [Carroll, 2003; Schechter, 2002].

The most representative age in the sample was the period between 3 and 5 years, equal to 68% of the tested cases (134 children). This result can be superimposed on the growth curve of adenoid and tonsil tissue which reaches its peak during the same period, confirming the important role played by adenotonsillar hypertrophy in OS AS aetiology during paediatric age (Fig. 2).

It appears clear that severe OSAS is, in proportion, more frequent in younger children (11 cases equal to 35% during infancy) and decreases later progressively as age rises. On the other hand primary snoring tends to increase with age (3 cases equal to 10% during infancy compared to 9 cases equal to 28% in childhood). A normal clinical dentofacial aspect is present in most of the tested patients, but some orthodontic anomalies come out with a significant prevalence with regard to general population (e.g. crossbite is present in 25% of the examined cases compared to 8-10% of general paediatric population).

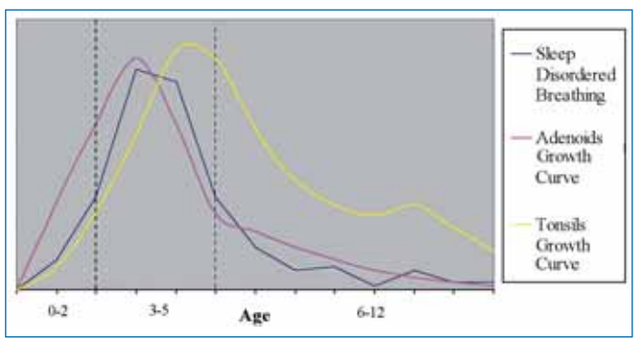


**FIG. 2 -** *Adenotonsillar growth curve and age distribution of the tested sample according to age.*

Therefore there are definite dentofacial aspects in children with SDB, but these are present in low percentage in the surveyed sample.

The crosscheck of the degree of SDB severity and possible associated craniofacial anomalies did not point out any significant relationship between the prevalence of anomalies and the severity of respiratory sleep disorder. This highlights the lack of significant correlation between the examined polysomnographic variables and the clinical dentofacial analysis, as stated in scientific literature [Gross, 1994]. This can be due to the fact that in our sample severe OSAS was most frequent in infants, in whom it was more difficult to evaluate possible craniofacial anomalies because of the still immature development of the face. It results from the obtained score that if we analyse three variables contemporaneously, crossbite, overjet and overbite, and at least one of them is present in the young patient, he or she has 60% probability to suffer from SDB. In children with SDB a single clinical orthodontic parameter does not have any sensitivity in our sample, whereas checking three different parameters in the same patient raises sensitivity percentage considerably.

## Conclusion

This work aimed to make an early diagnosis of malocclusion in young children, avoiding an instrumental diagnostic technique, surely more invasive, such as cephalometric examination. At the same time the purpose of our research was also to provide an effective treatment as early as possible in order to reduce both malocclusion and respiratory sleep disorders. Our results show that it is not possible to gather enough information from one single clinical dentofacial examination to make a diagnosis of SDB.

In literature many studies about influence of obstructive apnoea on dentofacial development are available, but data are still controversial [Cheng, 1988].

As a matter of fact head posture, combined with oral breathing, is supposed to be an important variable in morphologic dentofacial development, but a definite cause-and-effect relationship cannot be identified yet [Ellingsen, 1995]. Not all children with OSAS present facial anomalies, as well as malocclusive patterns, typical of OSAS, may be noticed also in children not affected by SDB. Consequently a paediatric dentist cannot make any diagnosis of OSAS only by performing a single clinical examination of the oral cavity, but a polysomnography will be essential and, if this should point out any pathology referring to SDB, a following orthodontic assessment by means of cranial lateral cephalometric examination might be needed.

The paediatric dentist who deals with respiratory sleep disorders cannot avoid a close collaboration with other specialists such as ENT specialist, paediatrician, neurologist

and orthodontist. Only a simultaneous interaction of reciprocal competencies can optimise management of children with OSAS.

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