

Oral breathing and dental malocclusions

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ABSTRACT. ***Aim** Aim of the present study was to evaluate existing correlations between oral breathing and dental malocclusions. **Methods** The study was conducted on a paediatric group of 71 oral breathers selected at the Allergology and Paediatric Immunology Department of Umberto I General Hospital, University of Rome "La Sapienza". The children were selected based on inclusion/exclusion criteria. Children aged 6 to 12 years with no history of craniofacial malformations or orthodontic treatment were included. The results were compared with a control group composed of 71 patient aged 6 to 12 years with nasal breathing. After their medical history was recorded, all patients underwent orthodontic/otolaryngological clinical examinations. The following diagnostic procedures were then performed: latero-lateral projection telerradiography, orthopantomogram, dental impressions, anterior rhinomanometry before and after administering a local vasoconstrictor, nocturnal home pulse oximetry (NHPO) recording, spirometry test, skin prick test, study cast evaluation and cephalometric analysis following Tweed's principles. The intraoral examination assessed: dental class type, overbite, overjet, midlines, crossbite, and presence of parafunctional oral habits such as atypical swallowing, labial incompetence, finger sucking and sucking of the inner lip. Evaluation of the study casts involved arch perimeter and transpalatal width assessment, and space analysis. **Results** The results showed a strong correlation between oral breathing and malocclusions, which manifests itself with both dentoskeletal and functional alterations, leading to a dysfunctional malocclusive pattern. **Conclusions** According to the authors' results, dysfunctional malocclusive pattern makes it clear that the association between oral breathing and dental malocclusions represents a self-perpetuating vicious circle in which it is difficult to establish if the primary alteration is respiratory or maxillofacial. Regardless, the problem needs to be addressed and solved through the close interaction of the paediatrician, otorhinolaryngologist, allergologist and orthodontist.*

Key words: Oral breathing; Malocclusion; Craniofacial growth.

Introduction

In growing subjects, chronic obstructive airway disease is often associated with orthodontic anomalies of orthodontic interest. Although the issue has been studied extensively since the early 20th century [Ketcham et al., 1912; Tomes, 1991], the nature of the anatomical and functional relationships between respiratory and dental apparatuses makes it difficult to clarify how their respective dysfunctions interact. To date, it has been impossible to ascertain whether the craniofacial alterations are a cause or, rather, a consequence of increased resistance to oronasal airflow.

An important study published by Harvold [1981] showed the presence of cranial and muscle alteration in growing primates in which oral breathing was experimentally induced for an extended period of time.

To better understand the close association between oral breathing and dentoskeletal alterations, the development of the craniofacial district, which is a result of the interaction of genomic and epigenetic factors, must be analysed. Genomic information is needed for cell synthesis, while epigenetic factors regulate growth and development processes [Moss et al., 1962]. Therefore, a set of biophysical and biomechanical conditions must be satisfied for the craniofacial district to develop correctly: oro/nasal-pharyngeal airway patency; proper behaviour of the orofacial musculature during childhood; correct functioning of the orofacial area valves [Fränkel, 1991].

Physiological breathing plays an active role in harmonious craniofacial development, but when external factors alter its mechanism, its influence on the development of the skull leads to functional and skeletal alterations.

Breathing can be classified into 3 types:

- nasal;
- mixed (oro-nasal);
- oral

Nasal breathing corresponds to the physiological pattern, while the mixed and oral types are

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consequences of breathing alterations.

Oral breathing is a parafunctional habit whereby air passes exclusively or partially through the mouth instead of the nose, and it is accompanied by skeletal and functional alterations in the orofacial district.

The causes of oral breathing are classified as either congenital and acquired. The former comprise: choanal atresia, nostril atresia and nasal septum deviations. The latter include: outcomes of nasal fractures, rhinopharyngitis, allergic rhinitis, nasal polyposis, chronic sinusitis, chronic adenotonsillitis, chronic hypertrophic rhinitis, adenotonsillar hypertrophy, malignant and benign tumours.

The most evident consequences of chronic oral breathing are represented by craniomaxillofacial alterations, mainly caused by abnormal mandible displacement and subsequent dysmorphism of the oral structures and modified posture [Bernkoff et al., 1997]. The tongue lies against the bottom of the mouth, thus allowing the passage of air via the oral route. In the event of concomitant tonsillar hypertrophy, the tongue undergoes further anteriorisation, since its base is forced to move away from the posterior pharyngeal wall due to spatial constraints.

The most frequent dysgnathia is represented by both dental and skeletal Class II, with reduction of the transverse diameter of the upper arch, ogival palate, mono- or bilateral posterior crossbite, and anterior open bite with buccally inclined upper incisors.

It must be clarified that the malocclusion is not a consequence but, rather, the cause of oral breathing through incorrect mandible and tongue positioning due to the presence of dysmorphism.

Facial morphology is typical (the so-called "facies adenoidea"), characterised by labial incompetence with a short upper lip that has accentuated lower concavity, and a protruding and often erythematous lower lip. The tongue placed between the arches and hypotonia of the alar cartilages due to limited use can also be observed.

In the most severe and complicated forms, the patient's general appearance is asthenic and longilineal, with an underdeveloped thoracic cage, sunken or keeled sternum, winged scapulas, kyphosis and rachitic traits. Listlessness, inattentiveness or drowsiness may be present and are ascribable to the sleeping disorders often associated with chronic oral breathing. Therefore, early detection of risk factors is essential in order to reduce the risk that the symptoms will become chronic and to avoid long-term complications.

Materials and methods

Aim of the study was to evaluate a paediatric population of 71 oral breathers to assess risk factors,

dentoskeletal modifications, and the relationship between chronic oral breathing and malocclusion, in order to outline a proper diagnostic-therapeutic approach through the collaboration between specialised dentists, otolaryngologists and paediatricians.

The study was conducted on a group of 71 oral breathers selected at the Allergology and Paediatric Immunology Department of Umberto I General Hospital, University of Rome "La Sapienza". For enrolment purposes we used a standardised questionnaire in order to define the condition of "chronic oral breather". Study exclusion criteria were as follows: history of orthodontic treatment, craniofacial malformations, age out of the range of 6-12 years. The results were compared with a control group composed of 71 patient aged 6 to 12 years with nasal breathing and no history of allergy.

At the first clinical evaluation, the subjects underwent a complete physical examination and anterior rhinoscopy. Each patient underwent skin allergy tests (Skin Prick Test by Lofarma Allergeni) for common food allergens (alpha-lactalbumin, beta-lactalbumin, casein, albumen and vitellus, soy, corn and fish) and inhalant allergens (Dermatophagoides pteronyssinus, Dermatophagoides farinae, Alternaria tenuis, Paritaria officinalis, Cynodon dactylon, Olea europea, Lolium perenne, cat epithelium and dander, dog epithelium and dander. Positive control test: histamine; negative control: saline solution). During the clinical evaluation, all cooperating patients underwent anterior active rhinomanometry (Rhinospir PRO 164) before and after administration of the vasoconstrictor Rinazina (naphazoline), 2 drops/nostril.

In accordance with the International Standardization Committee on Objective Assessment of the Nasal Airways, nasal breathing function was assessed separately for both nostrils by dynamic means during quiet breathing with the mouth closed. Rhinomanometric results were interpreted considering the nasal flows at 150 Pa, and compared with paediatric height-dependent reference values reported in the literature.

Nocturnal home pulse oximetry (NHPO) was recorded for all participants. The pulse oximeter (Respironics 920M), along with a diary of nocturnal awakenings, was given to the participants' parents, who were taught how to use the device. The data recorded by the device were analysed using Profox software, which automatically recorded the number and length of desaturations (defined as falls in O₂ saturation > 4 percentage points), and the time spent at each different saturation levels. Pulse oximetry was defined as "positive" if there were 3 or more desaturation clusters (referred as 5 or more desaturations over 30 recorded minutes) and at least 3

desaturations in which SaO₂ dropped below 90%. Pulse oximetry was defined as “negative” in absence of desaturation clusters and drops of SaO₂ below 90%, and “inconclusive” in absence of the criteria needed for defining a pulse oximetry testing as “positive” or “negative”.

For the orthodontic evaluation, which was conducted by operators of University of L’Aquila, the subject underwent an intraoral examination; latero-lateral projection telerradiography; orthopantomogram; alginate impressions of the dental arches; evaluation of the study casts and cephalometric analysis (Tweed’s method). The intraoral examination assessed: dental class type, overbite, overjet, midlines, crossbite, and presence of parafunctional oral habits such as atypical swallowing, labial incompetence, finger sucking and sucking of the inner lip.

Evaluation of the study casts involved arch perimeter and transpalatal width assessment, and space analysis.

I) Arch perimeter

The arch is divided into 4 sectors:

1. right posterior sector, extending from the mesial face of the first permanent molar to the canine mesial face.
2. right anterior sector, extending from the canine mesial face to the interincisive line.
3. left posterior sector, extending from the canine mesial face to the distal face of the first permanent molar.
4. left anterior sector, extending from the interincisive line to the canine mesial face [Fisk et al., 1966].

II) Arch width

Defined as the distance between anterior and posterior landmarks.

Anterior anatomical landmarks:

- distal pit of the transverse sulcus of the first deciduous molar;
- deepest point of the transverse sulcus of the first deciduous molar;
- distobuccal cusp apex of the first deciduous molar;
- buccal contact point between first and second premolar.

Posterior anatomical landmarks:

- point of intersection between the transverse and buccal sulci of the first molar;
- distobuccal cusp apex of the first lower molar.

III) Transpalatal width

The transpalatal width according to McNamara is calculated as the distance between the two upper permanent molars at the contact point between lingual sulcus and gingival margin.

- Narrow dental arch < 31 mm.
- Neutral dental arch = 31-35 mm.
- Wide dental arch > 35 mm.

- Statistical methods.

The collected data were analysed by descriptive analysis. Moreover, the associations, expressed as relative risk (RR), were assessed by multiple logistic regression models among the different skeletal alterations and the presence of explicative variables considered as co-factors in the skeletal alterations onset.

Parents/guardians were required to sign the informed consent form.

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The employed tests, χ^2 and Fischer, are bidirectional and with a significance level of 5%.

Results

Causes of chronic oral breathing and snoring

Our sample shows a considerable prevalence of atopia (70.4%) and hypertrophic adenoids (47.9%). Tonsillar hypertrophy is observable in 25 children (35.2%).

The prevalence of hypertrophic adenoids is significantly lower among atopic compared to nonatopic subjects (36% vs. 76.2%; $p < 0.05$).

None of the subjects exhibited severe nasal obstruction during the rhinomanometric examination: 26% of the subjects showed moderate obstruction, in 42% it was mild, and in 32% no nasal obstruction was observed.

The prevalence of nasal obstruction was slightly lower (independently of the degree of severity) in atopic compared with nonatopic subjects (26% vs. 33%), but the figure is not statistically significant.

The prevalence of obesity in the study sample was 11.3% (8/71), whereas 16.9% (12/71) of the children were overweight, 67.6% (48/71) were of normal weight and 4.2% (3/71) were underweight. Obesity was significantly higher among female subjects (75% females vs. 25% males).

NHPO read as positive in 4 children (5.6%), inconclusive in 29 (40.8%) and negative in 38 (53.5%).

Positive pulse oximetry tended to be more frequent among atopic compared to nonatopic subjects (6% vs. 4.7%), but the figure is not statistically significant.

Dental alterations

The results indicate reduced transverse diameter of the upper maxilla in 72.5% of the cases and a

| Characteristics | Cases n(%) | Controls n(%) | P value |
|------------------------------|------------|---------------|----------|
| Skeletal Class I | 51.56% | 78.87% | <0.05* |
| Skeletal Class II | 43.75% | 18.31% | <0.05* |
| Skeletal Class III | 4.69% | 2.82% | <0.05* |
| Transpalatal width <31mm | 72,5% | 26,76% | <0.05* |
| Vertical dimension increased | 72,4% | 19,72% | <0.05* |
| Vertical dimension reduced | 24,14% | 8,45% | <0.05* |
| Cross-bite | 34,33% | 15,49% | <0.05* |
| Open-bite | 15,38% | 25,35% | <0,001** |
| Deep-bite | 29,23% | 0 | <0,001** |

*chi2 test **fischer's exact test

TABLE 1 - Dentoskeletal alteration.

| Control case | Dpl absent | Dpl present | Total |
|----------------|------------|-------------|-------|
| Oral breathers | 10% | 90% | 100 |
| Control group | 77.46% | 22.54% | 100 |

TABLE 2 - Presence of DPL.

| Control case | Dpl absent | Dpl present | Total |
|----------------|------------|-------------|-------|
| Oral breathers | 7 | 63 | 70 |
| Control group | 55 | 16 | 71 |
| Total | 62 | 79 | 141 |

Person $\chi^2(1)=65.1195$ $pr=0.000$

prevalence of 43.75% of skeletal Class II

The vertical dimension increased in 72.5% of the cases; the prevalence of crossbite was 32.5%; bite alterations were also observed (Table 1).

There was a 90% prevalence of atypical swallowing (Table 2); 57.4% of the cases exhibited sucking of the lower lip and 29.2% showed labial incompetence.

The cross-analysis revealed that transpalatal width reduction is associated with skeletal Class II in 32.1% of the cases, skeletal Class III in 3.5% and crossbite in 37% of the subjects ($p<0,001$).

In 75% of the patients showing palatal width reduction there was also an increase in vertical dimension, whereas it was reduced in 25% of the cases. Atypical swallowing was exhibited by 86.5% of the subjects ($p<0,001$).

The skeletal Class II pattern (43.7%) was correlated not only with reduced transpalatal width in 75% of the cases, but also with an increase in vertical dimension (77.8%), which in 14.3% of the subjects was associated with an open bite, as widely confirmed by the literature, and in 22.2% with a deep bite.

Discussion

Many studies attest to the correlation between upper airway obstruction and craniofacial modifications. In

particular, a study conducted by Harvold in 1981 observed that when a growing primate is experimentally induced to be an oral breather for an extended period of time, it always develops muscle and cranial alterations.

It is important to note that this is a two-way problem: just as oral breathing can determine malocclusive patterns by affecting growth and craniofacial development, it is equally true that the various types of malocclusion can favour oral breathing.

Our study shows that a chronic oral breather develops a dysfunctional malocclusive pattern.

We considered the most frequent causes of chronic oral breathing in paediatric patients: tonsillar hypertrophy, chronic atopia and rhinitis, and obesity. Our sample showed a considerable prevalence of atopia (70.4%), a figure that is much higher with respect to the general paediatric population (10-15%) [Cavagni et al., 2006; Carr et al., 2007].

These data are chiefly attributable to selection bias, since the children enrolled in the study were referred to us by a Special Paediatric Allergology Unit.

As expected, and in accordance with the literature [Benninger et al., 2007], the prevalence of hypertrophic adenotonsillar tissue among the group of

oral breathers was extremely high. Surprisingly, the prevalence of skin test positivity to inhalers was significantly higher in children without adenotonsillar hypertrophy than among those presenting this condition (88% vs. 12%; $p < 0.01$). Furthermore, given the high prevalence of chronic rhinitis among school-age children, this should always be investigated as a cause of oral breathing and snoring. In our study, in fact, 26% of the children with moderate to severe rhinomanometry-assessed nasal obstruction failed to show a statistically significant correlation between the obstruction and atopia. While the effect of obesity on snoring is fundamental and undisputed in adults, its role in childhood is still being debated, and in our sample the prevalence of obesity was similar to that of the general population (11.3% vs. 11.1%) [Nathaniel Marshall, 2007]. The relationship between obesity and gender instead proved to be statistically significant ($p < 0.01$), with a higher prevalence of females among obese subjects (out of a total of 8 obese patients, 6 are females, 75%, and 2 are males, 25%), which is concordant with the literature [Chan, 2006].

In our study, pulse oximetry testing was used to screen for breathing disturbances. The pulse oximetry parameters did not correlate with any of the risk factors studied and were not affected by atopia.

Predictably, in our oral breather group the main stomatognathic alterations affected the upper maxilla, which in 72.5% of cases exhibited significant transverse diameter reduction, thus showing palate hypodevelopment that must unquestionably have repercussions on the lower arch.

Hence, the mandible can undergo 2 types of displacement: clockwise rotation and retropositioning, thus causing a skeletal Class II pattern (43.75%), and monolateral deviation, leading to a crossbite (34.33%).

Clockwise rotation of the mandible not only causes mandibular retropositioning, but also determines increased divergence (77.8%), which in 22.2% of the patients was correlated with a deep bite and in 78.9% with atypical swallowing.

Mandibular retrusion and deep bite entail an intraoral reduction of the volume available for the tongue, which is thus forced to shift upward and backward, since forward movement is impeded by the incisor "wall".

The upthrust on the median palatine suture determines both palatal and nasal structures deformations (respectively, ogival palate and aquiline nose with a tendency toward septal deviation).

The backward shift of the tongue is impeded by the presence of hypertrophic tonsillar tissue that, even if only partially obstructive, will then cause complete obstruction due to pressure exerted by the root of the tongue.

The backward shift of the tongue leads to lingual dyskinesia, which is manifested by complex atypical

swallowing [Schindler, 1990].

Complex atypical swallowing is defined as deglutition with diverging arches. In this case, labial and mimic muscle contraction, lack of contraction of the mandibular elevator muscles, and tongue interposition between the dental arches during swallowing can be observed. The incidence of complex atypical swallowing does not decrease with age, as is instead the case with the simple type [Piemonte, 1997].

These results show that lingual behaviour due to malocclusion leads to respiratory obstruction.

In 14.3% of the cases, clockwise rotation of the mandible is associated with an open bite. This malocclusive pattern manifests itself with the loss of the anterior seal, labial incompetence in 29.2% of cases, reduced divergence, reduced overbite and increased overjet. Swallowing is defined as atypical (87.5%) because of the need for an anterior seal that, in this case, is provided by tongue interpositioning.

With this type of occlusal pattern, the open bite can account for 2 different aetiologic causes, namely:

- a. secondary to thumb-sucking or sucking on pacifier or feeding bottle past the normal age;
- b. secondary to adenotonsillar hypertrophy that forces the child to breath with his/her mouth open.

In both cases the result is oral breathing and lingual dyskinesia with subsequent craniofacial alterations.

When the tongue assumes an anteriorised position, it cannot exert its remodelling effect on the palate, which will be underdeveloped, in turn decreasing horizontal growth of the nasal structures and leading to reduced patency.

Conclusion

The results indicate that the effects of this close association will lead to a dysfunctional malocclusive pattern:

- a. Physiognomy.
- b. Oral breathing.
- c. Malocclusions.
- d. Otitis.
- e. Breathing disorders during sleep.
- f. Headache.
- g. Dizziness.
- h. Mood disorders.
- i. Back pain.

This dysfunctional malocclusive pattern makes it clear that the association between oral breathing and dental malocclusions represents a self-perpetuating vicious circle in which it is difficult to establish if the primary alteration is respiratory or maxillofacial. Regardless, the problem needs to be addressed and solved through the close interaction of the paediatrician, otorhinolaryngologist, allergologist and orthodontist.

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