

# PREMATURE EDENTATIONS IN CHILDREN AND THEIR IMPACT ON FACIAL GROWTH

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## ABSTRACT

We report a case in which a significant mandibular edentation in primary teeth lead to hypoplasia of the maxilla and to a forward mandibular sliding. Mandibular growth is no longer controlled and thus real prognathism sets in.

**Key Words:** premature edentation in primary teeth, facial growth, and paediatric prosthesis

## INTRODUCTION

Premature edentation is considered to be the loss of teeth at least 1 year before the physiological root resorption.

Etiology of edentation can be caused by several factors: congenital tooth absence (hypodontia, total anodontia, partial anodontia) multiple extractions (teeth with complicated caries, especially the “baby-bottle” caries, in diseases with high bacterial risk (congenital or acquired cardiopathies, immunodeficiency through chemotherapy, medullar aplasia), complex periodontitis (Papillon-Lefevre syndrome, neutropenia, etc.) and traumatic injuries.<sup>1-5</sup>

### Premature edentation and its impact on facial growth

#### *Skull base*

Premature extractions disrupt significantly local tooth growth, as well as maxillary and even facial growth. Mid-face morphology is known to be linked to the front, middle, and back connecting pillars and to be held together by the malar-zygomatic belt, undergoes mastication pressures which radiate towards the base of the skull.<sup>6-8</sup>

#### *Dental arches*

Premature loss of support in primary dentition causes loss of the occlusal support, with reduction of occlusal height and/or forward mandibular sliding (through bilateral edentation).<sup>4,9-13</sup>

#### *Alveolar bones*

Significant edentation impacts negatively on normal alveolar growth (the alveolar bone appears and lives together with the tooth).

#### *Condylar cartilages*

Unilateral mastication, caused by unilateral edentation, leads to muscular imbalance, which, at its turn, is sometimes responsible for abnormal condylar morphology; this asymmetry produces permanent disorders in mandibular movements.<sup>6,14</sup>

## CASE REPORT

At the time of admission, the seven-year-old patient had already suffered early loss of several primary teeth through “baby-bottle” caries (around the age of five she lost her incisors, canines and the most primary molars) The premature loss of the incisors and of the canines, as well as of the teeth in the lateral support region of the mandible, generated a forward mandibular sliding. This gliding prevented maxillary growth and led to micrognathism and malocclusion class III.

The orthodontic treatment was started with a Wunderer activator that she wore for approximately one year, later replaced with a removable appliance

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(radial expansion and bilateral bite planes) and in the mandible, with removable appliance; the latter functions as a space maintainer for guiding the eruption of the permanent teeth. A chin cap was also introduced to prevent mandibular growth.

Figures 1-5 present frontal and side views of the patient's face, as well as images of the occlusion 2 years after initiating the treatment.



Figure 1



Figure 2



Figure 3



Figure 4



Figure 5

## **DISCUSSION**

In the case presented above, we believe that had treatment with paediatric prosthesis been initiated earlier no forward mandibular sliding would have occurred, since, according to the patient's parents, there is no hereditary predisposition; moreover, acquired functional factors of forward mandibular sliding were excluded (dental crossbite, traumas).

According to Baeyaert et al, significant molar mandibular edentation can lead to a nearly permanent low lingual position, which produces hypoplasia of the maxilla because intermaxillary suture growth is not sufficiently stimulated.<sup>15</sup> The overall aspect of the profile resembles that in the Crouzon syndrome (concave profile and false ocular protrusion). Mandibular growth is no longer controlled and thus real prognathism sets in.

There is no doubt that premature edentation does not always lead to such ample modification.

To support this statement the "cybernetic model" can be employed. According to this model, hormonal control of the condylar cartilage plays an important part in so far as it affects cell multiplication directly and indirectly. At the same time we feel we need to point out that the normal osteo-articular and neuromuscular development is predetermined genetically.<sup>14</sup>

## **CONCLUSIONS**

Unlike the system of linear prediction of somatic

growth, cranio-facial development occurs in “growth crises”.

The three-dimensional cranial growth stops at puberty, but tooth eruption influences the rate growth of the face and of the facial skeleton. Thus, the maxilla and mandible continue their growth even after puberty (2-3 mm and 8 mm respectively)

Despite the fact that the maxilla and the mandible are region of major variability, the term “facial puzzle” is inadequate. Therefore a long-term prediction of quantitative and qualitative growth should be viewed with caution.<sup>6-8</sup>

There is no doubt that extended edentation in children alter facial architecture, thus affecting their psychological profile as well. In such cases it is therefore essential that an orthodontic and prosthetic assessment be made periodically, over a long period of time.<sup>16-19</sup>

Demars and Fremault assert that paediatric prosthesis, in addition to their prosthetic function, is the optimal solution to the problem of extended edentations since masticatory pressures are transmitted to mid-face and then to the cranial base through the malar-zygomatic belt and, in this way stimulate these segments.<sup>16</sup>

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