Open Bite in Prematurely Born Children

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ABSTRACT

Purpose: The aims of this study were to: examine the expression of open bite in prematurely born children and discuss the etiological factors that may lead to bite it.
Methods: The subjects were 328 prematurely born (<37 gestational weeks) Caucasoid and African American children and 1,804 full-term control children, who participated in the cross-sectional study of the Collaborative Perinatal Project in the 1960s and 1970s. Dental documents, including casts and photographs, were taken once at the age of 6 -12 years in the mixed dentition. The occlusion was recorded by examining and measuring the hard stone casts. Vertical open bite was recorded only for full erupted teeth. The statistical method used was chi-square analysis.
Results: Significant differences in the incidence of anterior open bite (from left to right canine) was found between the preterm and control groups and between gender and ethnic groups. The prevalence of anterior open bite was nearly 9% in the preterm group and almost 7% in the control group. African Americans (9%) had a significantly greater incidence of open bite than Caucasians (3%; *P*<.0001). Generally, girls had a greater incidence of open bite than boys (8% vs 6%; *P*<.11). When the study groups were divided by prematurity, gender, and ethnic group, the prevalence of open bite was increased—especially in preterm African American boys compared to controls (11% vs 8%).
Conclusions: The results show differences in the development of anterior open bite between ethnic and gender groups. Premature birth may also influence dental occlusal development. Of importance are the patient's: general health condition; respiratory infections; inadequate nasal- and mouth-breathing; oral habits; and other medical problems. Preterm children may be relatively more predisposed to etiological factors for the development of anterior open bite. (J Dent Child 2007;74:165-70)

KEYWORDS: DENTAL OCCLUSION, MALOCCLUSION, DENTAL DEVELOPMENT

Preterm birth is defined as birth prior to 37 completed weeks of gestation and usually with low birth weight (<2,500 g). Prematurity naturally includes a wide variation of gestational ages and birth weights. The incidence of preterm birth varies in different populations, with the general average in 2000 being between 4% to 15%,[1] the average in the United States being approximately 12%,[2] and the average in Finland being 6%.[3] Of all preterm births:

a. 20% to 25% are induced due to medical or obstetric conditions that can affect the health of the fetus, mother, or both; and
b. 70% to 80% of the preterm births are spontaneous.[4]

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The improvement in perinatal and neonatal care, the use of prenatal corticosteroids, and postnatal surfactant treatment[5] has decreased the mortality rates of preterm infants. During the past decades, the survival of preterm infants—especially those with a birth weight between 500 to 1,000 g—has increased.[6] Despite this, the morbidity is still quite high correlated to birth weight and gestational age at birth.[7] Typical causes of neonatal morbidity include respiratory problems, intracranial hemorrhages, asphyxia, metabolic disorders, hypo- and hyperglycemia, hyperbilirubinemia, hypocalcemia, and infections.[7,8]

Prematurely born children are poorly adapted to the extrauterine environment due to the immaturity of many organs and often suffer serious metabolic derangements and infections during the neonatal period.[9] There are several systemic derangements that can also interfere with the developing teeth. According to earlier studies, it has been reported that preterm birth may also affect the develop-

ment of the dentition, including generalized and localized enamel hypoplasia, dental caries, and palatal deformities.[10,11] Premature birth has also been indicated to affect the eruption of deciduous teeth[12] and permanent dentition.[13,14] The eruption process of permanent incisors and first molars was significantly earlier in preterm children compared to controls.[14] Preterm birth may also affect the development of sagittal occlusal relationships and asymmetry, with preterm children having significantly greater prevalence of prenormal canine relationships and mesial molar occlusion.[15]

Anterior open bite is defined as an open vertical dimension between the incisal edges of maxillary and mandibular anterior teeth.[16] The upper and lower teeth are separated when the jaw is completely closed. The major causes and etiological factors of anterior open bite are:

1. forces resulting from sucking habits (tongue, finger, pacifier, lip);
2. airway obstruction;
3. inadequate nasal- and mouth-breathing;
4. allergies;
5. septum problems;
6. enlarged tonsils and adenoids; and
7. skeletal growth abnormalities.[17-19]

Also, the function of masticatory muscles can significantly influence craniofacial growth. Reduced muscle function and weak masticatory muscles may cause changes in the craniofacial morphology. It has been suggested that the form of the face partly depends on the strength of the muscles.[20] It has also been suggested that the prevalence of malocclusions in physically and/or mentally handicapped children has increased and that especially seriously mentally retarded children have the highest prevalence and the most severe malocclusions compared to controls.[21] Children with cerebral palsy have also been found to have an increased prevalence of malocclusions,[22] often associated with incompetent lip closure and a drooling habit. A higher frequency of drooling (23%) was found in children with open bite compared to children with normal bite. Problems with speech and mastication have been attributed to open bites.[23,24] Morphological malocclusion like crossbite and anterior open bite may be a potential factor for temporomandibular joint dysfunction (TMD).[25] Some of the open bites resolve spontaneously without treatment during the transition period from primary to permanent dentition, but complex open bites that do not resolve by the end of the mixed dentition period may require orthodontic and/or surgical treatment. Identification of the etiological factors and background of open bite improves the effectiveness and success of treatment.

METHODS

The subjects consisted of 328 preterm and 1,804 fullterm control children, for a total of 2,132. There were 60 Caucasian children (40 boys and 20 girls) and 268 African American children (140 boys and 128 girls) in the preterm group and 803 Caucasian children (408 boys and 395 girls) and 1,001 African American children (477 boys and 524 girls) in the control group. To maintain practical proportions between the preterm and control children in the statistical comparisons, the limit for prematurity was placed at 36 gestational weeks for Caucasians and 35 gestational weeks for African Americans. The mean gestational age was 33.7 weeks for the Caucasian preterm boys and 40.4 weeks for the controls, with corresponding figures of 34.6 weeks and 40.6 weeks for the Caucasian girls, 31.7 and 39.8 weeks for the African American boys, and 32.2 and 39.9 weeks for the African American girls.

All children were among the 60,000 participants in the Collaborative Perinatal Study of the National Institute of Neurological Disorders and Stroke (NINDS), study planned and organized by Pearce Bailey, M.D., Director. The dental examinations were performed in the 6 collaborating medical centers (Buffalo, NY, Richmond, Va, Portland, Ore, Philadelphia, Pa, Providence, RI, and Johns Hopkins, Md) headed by profesor Richard H. Osborne. Medical background data were obtained from the pregnancy's first registration up to the seventh year of age, including anamnestic information on the mother's health and background.[26] The duration of gestation was considered to be the time elapsing between the first day of the last menstrual period (LMP) reported by the woman and delivery.

This period was computed in days and then transposed to weeks and rounded to the nearest week. The date of the LMP was ascertained by a special interviewer, and the duration of gestation was also based on the history and physical findings and an estimate by the obstetrician, which was confirmed at each prenatal visit. Data concerning the type of delivery—including birth weight, birth length, and head circumference—were obtained within 1 hour of delivery by an observer using calibrated scales.[26] The measurements were repeated at 4, 8, and 12 months and at 3, 4, and 7 years of age.

The dental examinations were carried out cross-sectionally, with dental casts made and photographs taken in a standardized fashion at the age of 6 to 12 years in 95% of the cases. Alginate impressions were taken at each cooperating center, and plaster casts were made.[27] All the casts were checked and trimmed at the Department of Anthropology, University of Wisconsin, Madison, Wis. The arch dimensions and occlusal variables—including the molar and canine sagittal relationships (189 variables)—were recorded by examining and measuring the hard stone casts by a modified version of the methods used by Björk et al in a 1964 study[28] and by Laine and Alvesalo in a 1986 study.[29] The level of intraexaminer error in the analysis of open bite was estimated as percentage reproducibility of the same occlusal status in double determinations. Double determinations were performed on 70 dental casts. The reproducibility in the analysis of molar and canine occlusions was 95% and 83%, respectively.

The mean chronological age at which the dental casts were taken was 8.8 years in the preterm group and 8.5 years in the controls. The occlusal variables were compared between the preterm and control groups, each divided by sex and race. Vertical open bite was recorded only for full erupt-

ed teeth. The statistical method used was chi-square analysis. The Research Committee of the Faculty of Medicine at the University of Oulu, Oulu, Finland, accepted the study plan.

RESULTS

The results showed, that the African American children (9%) had a greater incidence of anterior open bite than the Caucasian children (3%; P<.0001; Table 1.). Generally, girls had a greater incidence of open bite than boys (8% vs 6%; P<.11; Figure 1).

Table 1. The Incidence of Anterior Open Bite in Caucasian and African American Children

Group	Open bite% (N)	Total (N)
Caucasians	3% (26)	852
African Americans	9% (118)	1,260
Pearson chi-square	28.16	*P*<.0001

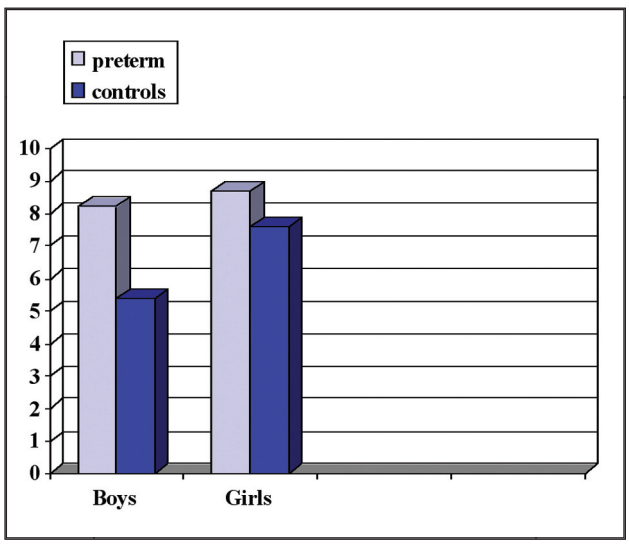

Figure 1. Anterior open bite in preterm and control boys and girls (African American and Caucasian children combined).

A greater prevalence of anterior open bite was found in the prematurely born children in approximately 9% in the preterm group and nearly 7% in the control group. Especially in the preterm African American boys, the prevalence of anterior open bite was greater compared to control boys (11% vs 8%; P<.0001; Table 2.). The same trend existed among preterm Caucasian girls. When the African American and Caucasian children were combined, the prevalence of anterior open bite was greater compared to controls for preterm boys (8% vs 5%) and for preterm girls (9% vs 8%; Figure 1. The statistical method used for the comparisons was the chi-square analysis.

Table 2 . The Prevalence of Anterior Open Bite in Preterm and Control Children Divided by Sex, Race, and Prematurity

Group	Preterm (N)	Controls (N)
Caucasian Boys	0% (39)	3% (404)
Caucasian Girls	5% (20)	4% (388)
African American Boys	11% (143)	8% (470)
African American Girls	9% (129)	10% (518)
Chi-square value	35.97	*P*<.0001

DISCUSSION

According to the literature, the prevalence of open bite in the general population at the age of 8 to 11 years is 4%.[30] Racial differences exist concerning the prevalence of different types of malocclusions: In United States, the incidence of open bite is approximately 16% in the African American population and 4% in the Caucasian population,[31] decreasing until adolescence. The results of this study support previous studies concerning racial differences and the prevalence of open bite, with African American children having a significantly higher prevalence of anterior open bite compared to Caucasians.

In a study by Karjalainen in 1999,[32] anterior open bite was detected in 18% of 3-year-old Finnish children and a greater incidence was found in children having non-nutritive sucking habits. It has been suggested females have a greater prevalence of oral habits at all ages than males,[19] especially thumbsucking. Children with thumbsucking and tongue-thrusting habits had a significantly higher percent of open bite. The gender differences in the prevalence of open bite was also seen in this study.

Open bite has been reported in mouth-breathers and is related to a downward and forward posturing of the tongue. A study by Linder-Aronson in 1970[33] concerned the complex relationship between respiratory pattern and facial growth and development. The lowered mandible may permit an overeruption of the teeth, rotation of the mandible in a clockwise manner, and more vertical and backward direction, causing elongation of the lower anterior facial height, open bite, and retrognathia. In 1991, Hultcranz[34] found an increased incidence of open bite in children with tonsillar obstruction. Children with allergies, septum deviations and nasal airway obstructions are often mouth-breathers and predisposed to developing open bite.

Open bite and tongue-thrust are also attributed to abnormal tongue function. Resting position of the tongue rather than the intermittent force and tongue thrust may plays a role in the etiology of open bite. Proffit et al[35] suggested that therapy for tongue function is most effective when combined with orthodontic therapy and that speech therapy may be combined with orthodontic treatment.

It has been shown in the literature that bite forces and facial morphology have a correlation.[36,37] It has been re-

ported that open bite patients often show weak occlusal force and/or long face and that long face individuals show weaker occlusal force than short face individuals.[37,38] The influence of muscular environment in general dental development has also been emphasized in a study by *Ghafari et al* in 1988.[39] Their study show the prevalence of open bite and posterior crossbite being higher in children with myopathies. On the contrary, it has been assumed that a weak occlusal force may be a promoting factor for, rather than the cause of open bite.[40]

In younger children, the major cause of anterior open bite is non-nutritive sucking habits. Sucking habits are quite normal until the age of 3 years, but the persistence of these habits after that age significantly increases the development of undesirable effects on dental arch form and occlusion.[41] A prolonged sucking habit causes anterior open bite, proclination and protrusion of the maxillary incisors; and posterior crossbite. The amount of malocclusions has reported to increase with duration of the habit. About half of the children with a finger-sucking habit still suck at 7 years of age.[42] The effect of a finger-sucking habit on the vertical dimension of the occlusion is mainly caused by reduced anterioralveolar growth.[43] Spontaneous correction will often happen when the habit ceases early enough, alveolar process accelerates in growth, and upper incisors erupt to establish incisal contact. To minimize the risk of developing occlusal abnormalities, the ideal age to discontinue a child's non-nutritive sucking habit may be at two years of age.[44]

The role of early feeding on occlusion appears quite unclear based on published literature.[32,41] Breast-feeding and bottle-feeding involve different orofacial muscles and may lead to differences in growth of the maxilla and dental arches. Breast-feeding has been found to have positive effects on the development of an infant's oral cavity—including improved shaping of the hard palate, resulting in fewer malocclusions and proper tooth alignment.[45] The forceful motion of breast-feeding has also been suggested to encourage mandibular development.[46] Early bottle-feeding indicates a pattern of low facial muscle activity, which may affect the development of alveolar ridges and the hard palate, leading to posterior crossbite. Breast-feeding seems to have a protective effect on the development of a posterior cross-bite in the deciduous dentition. Children with non-nutritive sucking habits and who were bottle-fed had more than double the risk for posterior crossbite.[47] Due to early adaptation to the extrauterinal life, preterm children are often too weak to be breast-fed and must be tube-fed or bottle-fed at first. The early activity of muscles, however, may activate the development of the mandible in preterm children.[48]

The incidence of cerebral palsy (CP) and minor neurological disorders are related to gestational age and may vary between 5% and 19%[49,50] in extremely low birth weight infants, according to different studies. The pathophysiological relationship between amelogenesis imperfecta and open bite malocclusion still remains unknown.[51] It has been reported that preterm children have more dental defects and generalized and localized enamel hypoplasias both in the deciduous and permanent dentition.[11] Preterm children are also predisposed to several systemic derangements, respiratory problems, and other infections. Additionally, the head posture may be altered due to difficulties with breathing, which can also interfere with the developing teeth and may help explain the increased prevalence of open bite in some preterm children.

CONCLUSIONS

This study's results show racial and gender differences in the development of occlusion and anterior open bite. A greater prevalence of anterior open bite was found in African Americans compared to Caucasians and in prematurely born children compared to fullterm children. The results support earlier studies concerning racial differences in malocclusions. Premature birth may also influence the dental occlusal development. Preterm children may be more predisposed to etiological factors that influence the development of anterior open bite, respiratory infections, inadequate nasal- and mouth-breathing, oral habits and other medical problems.

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