م.د. محمد ابراهيم الزبيدي

# Delay eruption: LOCAL AND SYSTEMIC FACTORS THAT INFLUENCE ERUPTION

## 1-ANKYLOSED TEETH

The problem of ankylosed primary molars deserves much attention by dentists. Application of the term *submerged* molar to this condition is inaccurate, even though the tooth may appear to be submerging into the mandible or maxilla. This misconception results from the fact that the ankylosed tooth is in a state of static retention, whereas in the adjacent areas eruption and alveolar growth continue. The term infraocclusion, although commonly used today, is not preferable to ankylosis. Ankylosis should be considered an interruption in the rhythm of eruption and that a patient who has one or two ankylosed teeth is more likely to have other teeth become ankylosed. The mandibular primary molars are the teeth most often observed to be ankylosed. In unusual cases all the primary molars may become firmly attached to the alveolar bone before their normal exfoliation time. Ankylosis of the anterior primary teeth does not occur unless there has been a trauma. The cause of ankylosis in the primary molar areas is unknown, but at least three theories have been proposed.

The observation of ankylosis in several members of the same family lends support to the theory that it follows a familial pattern. Studies have reported that the condition occurs more frequently among siblings of children with the same characteristics. The occurrence is noted to have a familial tendency and is probably a non-gender-linked trait. Investigators have observed the prevalence of ankylosis to be much lower among black children than among white children.

One study observed that, in a group of children with 108 ankylosed teeth, 21 of the affected primary teeth had no permanent successors. Others also reported a higher prevalence of developmentally absent premolar teeth in patients with ankylosis, suggesting that there is a relationship between the congenital absence of permanent teeth and ankylosed primary teeth. However, later study discounted this relationship based on observation and a careful review of the literature.

Normal resorption of the primary molar begins on the inner or lingual surfaces of the roots. The resorption process is not continuous but is interrupted by periods of inactivity or rest. A reparative process follows periods of resorption. In the course of this reparative phase, a solid union often develops between the bone and the primary tooth. This intermittent resorption and repair may explain the various degrees of firmness of the primary teeth before their exfoliation. Extensive bony ankylosis of the primary tooth may prevent normal exfoliation and the eruption of the permanent successor.

# Delay eruption: LOCAL AND SYSTEMIC FACTORS THAT INFLUENCE ERUPTION

Ankylosis of the primary molar to the alveolar bone does not usually occur until after its root resorption begins. If ankylosis occurs early, eruption of the adjacent teeth may progress enough that the ankylosed tooth is far below the normal plane of occlusion and may even be partially covered with soft tissue. An epithelium- lined track, however, will extend from the oral cavity to the tooth. Ankylosis may occasionally occur even before the eruption and complete root formation of the primary tooth. In a case report, early ankylosis of a mandibular second primary molar that was not diagnosed until the patient was 10 years of age, at which time the succedaneous second premolar was lying malposed but occlusal to the unerupted primary molar. Ankylosis can also occur late in the resorption of the primary roots and even then can interfere with the eruption of the underlying permanent tooth.

The diagnosis of an ankylosed tooth is not difficult to make. Because eruption has not occurred and the alveolar process has not developed in normal occlusion, the opposing molars in the area seem to be out of occlusion. The ankylosed tooth is not mobile, even in cases of advanced root resorption. Ankylosis can be partially confirmed by tapping the suspected tooth and an adjacent normal tooth with a blunt instrument and comparing the sounds. The ankylosed tooth will have a solid sound, whereas the normal tooth will have a cushioned sound because it has an intact periodontal membrane that absorbs some of the shock of the blow. The radiograph is often a valuable diagnostic aid. A break in the continuity of the periodontal membrane, indicating an area of ankylosis, is often evident radiographically. In the management of an ankylosed tooth, early recognition and diagnosis are extremely important.

The eventual treatment may involve surgical removal. However, unless a caries problem is unusual or loss of arch length is evident, the dentist may choose to keep the tooth under observation. A tooth that is definitely ankylosed may at some future time undergo root resorption and be normally exfoliated. When patient cooperation is good and recall periods are regular, a watchful waiting approach is best.

## ANKYLOSIS OF PRIMARY MOLARS WITH ABSENCE OF PERMANENT SUCCESSORS

Many studies emphasize the importance of the presence of a permanent successor for normal exfoliation of a primary molar. In one longitudinal study, no ankylosed primary molars without permanent successors were found to exfoliate spontaneously. However, very slow root resorption was observed for most of the ankylosed teeth. In another study, failure to carry out timed extraction of severely infraoccluded molars results in reduced alveolar bone support for the premolars was observed. However, Kurol and Olson suggest that infraocclusions and

ankylosis of primary molars do not constitute a general risk for future alveolar bone loss mesial to the first permanent molars. In their study of 119 infraoccluded primary molars next to permanent first molars, all but two of the first permanent molars showed a normal alveolar bone level mesially. Therefore the general treatment recommendation to await normal exfoliation and eruption of successors remains valid in their opinion. They suggest that, in patients in whom there is an abnormality associated with a succedaneous tooth (e.g., agenesis, ectopic eruption), early intervention is most likely required. In situations in which permanent successors of ankylosed primary molars are missing, attempts have been made to establish functional occlusion using stainless steel crowns, overlays, or bonded composite resins on the affected primary molars. Currently, bonded restorations would be the preferred choice. This treatment is successful only if maximum eruption of permanent teeth in the arch has occurred. If the adjacent teeth are still in a state of active eruption, they will soon bypass the ankylosed tooth.

### ANKYLOSED PERMANENT TEETH

The incomplete eruption of a permanent molar may be related to a small area of root ankylosis. The removal of soft tissue and bone covering the occlusal aspect of the crown should be attempted first, and the area should be packed with surgical cement to provide a pathway for the developing permanent tooth. If the permanent tooth is exposed in the oral cavity and at a lower occlusal plane than the adjacent teeth, ankylosis is the probable cause. Both Biederman and Skolnick have described a luxation technique effective in breaking the bony ankylosis. If the rocking technique is not immediately successful, it should be repeated in 6 months. A delay in treatment may result in a permanently ankylosed molar.

Unerupted permanent teeth may become ankylosed by inostosis of enamel. The close association of an infected apex with an unerupted tooth may give rise to the process. In the unerupted tooth, enamel is protected by enamel epithelium. The enamel epithelium may disintegrate as a result of infection (or trauma), the enamel may subsequently be resorbed, and bone or coronal cementum may be deposited in its place. The result is solid fixation of the tooth in its unerupted position.

### 2-TRISOMY 21 SYNDROME (DOWN SYNDROME)

Trisomy 21 syndrome (Down syndrome [DS])—that is, the presence of three number 21 chromosomes rather than the normal two (diploid)—is one of the congenital anomalies in which delayed eruption of the teeth frequently occurs. The first primary teeth may not appear until 2 years of age, and the dentition may not be complete until 5 years of age. The eruption often follows an abnormal sequence, and some of the primary teeth may be retained until 15 years of

age. In a study of 127 males and 128 females with DS, on average, six primary teeth were delayed in eruption in boys and 11 primary teeth were delayed in girls. A similar study conducted in 116 males and 124 females with DS showed delayed eruption of 13 permanent teeth in boys and eight permanent teeth in girls. These studies seem to confirm that delayed tooth eruption is common but sporadic in children with DS.

Anomalies of the eye and external ear are seen, and congenital heart defects are often present. The occurrence of DS is frequently related to maternal age. Various sources report the frequency of DS to be approximately 0.9 per 1000 births when the mother is less than 33 years of age, 2.8 per 1000 when the mother is 35 to 38 years old, and 38 per 1000 when the mother is 44 years or older, with certain populations reporting a high of 91 per 1000 in this older age group. The diagnosis of DS in a child is not usually difficult to make because of the characteristic facial pattern. The orbits are small, the eyes slope upward, and the bridge of the nose is more depressed than normal. In a study of 194 children with DS, 54% demonstrated anomalies in the formation of the external ear, characterized by outstanding “lap” ear with flat or absent helix. Mental retardation is another characteristic finding, with most children in the mild to moderate range of disability.

Many children with DS have chronic inflammation of the conjunctiva and a history of repeated respiratory tract infections. The use of antibiotics has reduced the incidence of chronic infection and has resulted in fewer deaths from infection.

Individuals with DS have a higher prevalence of periodontal disease than otherwise normal, age-matched control groups and other patients with mental disabilities and of similar age distribution. Furthermore, the reports of exaggerated immunoinflammatory responses of the tissues in patients with DS cannot be explained by poor oral hygiene alone and may be the result of impaired cell-mediated and humoral immunities and deficient phagocytic systems. The study of 10 patients with DS and aged 20 to 31 years demonstrated that young age of onset, severe destruction, and pathogenesis of disease in the periodontal tissues were consistent with a juvenile periodontitis disease pattern.

In another study which obtained blood samples and conducted gingival health assessments of 75 individuals with DS and aged 2 to 18 years. The extent of gingival inflammation and the antibody titers of the individuals with DS suggested that colonization of certain pathogenic organisms for periodontal disease had occurred before 5 years of age. The prevalence and extent of gingivitis were significantly higher than in normal children. The antibody titers also suggested that colonization of additional pathogenic organisms increased with age. The authors

believe that there are abnormalities in the systemic defenses that are responsible for the early onset of disease in the individuals with DS.

Dental caries susceptibility is usually low in those with DS in both the primary and the permanent dentitions. Shapira and Stabholz successfully demonstrated caries reduction and improved periodontal health during a 30-month period after initiating a comprehensive preventive oral health program for 20 children with DS. Seagriff-Curtin and associates believe that although some children with low mental ability are unmanageable for dental procedures, most are pleasant, cheerful, affectionate, and well behaved. They can often be managed in the dental office in a conventional manner. The possibility of reduced resistance to infection should be considered in the dental management of the child with DS.

## 3- CLEIDOCRANIAL DYSPLASIA

A rare congenital syndrome that has dental significance is cleidocranial dysplasia (CCD), which has also been referred to as *cleidocranial dysostosis, osteodentin dysplasia, mutational dysostosis,* and *Marie-Sainton syndrome*. Transmission of the condition is by either parent to a child of either gender, so that the disorder thus follows a true Mendelian dominant pattern. CCD can also occur sporadically with no apparent hereditary influence and with no predilection for race. The diagnosis is based on the finding of an absence of clavicles, although there may be remnants of the clavicles, as evidenced by the presence of the sternal and acromial ends. The fontanels are large, and radiographs of the head show open sutures, even late in the child's life. The sinuses, particularly the frontal sinus, are usually small.

Cephalometric analyses of 17 patients with CCD. Found that, on average, the patients exhibited mandibular prognathism caused by increased mandibular lengths and short cranial bases. The maxillae tended to be short vertically but not anteroposteriorly. The development of the dentition is delayed. Complete primary dentition at 15 years of age, resulting from delayed resorption of the deciduous teeth and delayed eruption of the permanent teeth, is not uncommon.

One of the important distinguishing characteristics is the presence of supernumerary teeth. Some children may have only a few supernumerary teeth in the anterior region of the mouth; others may have a large number of extra teeth throughout the mouth. Even with removal of the primary and supernumerary teeth, eruption of the permanent dentition, without orthodontic intervention, is often delayed and irregular. One report indicates the successful dental management of a patient with CCD over a 15- year period. The patient was first seen at 2 years of age. Treatment consisted of timed extractions of primary and supernumerary teeth and

conservative uncovering of the permanent teeth. The surgical procedures were planned according to progressive radiographic evidence of the development of the permanent teeth.

This management results in a nearly normal but slightly delayed eruption sequence. Orthodontic treatment was begun at 14 years of age, and by 16 years of age the patient displayed acceptable occlusion and normal vertical dimension, root development, and periodontal bone support.

The pediatric dentist serves as the coordinator of overall oral health care and disease prevention during an extended treatment regimen that usually includes two surgical interventions and three stages of orthodontic surgery.

Delayed eruption has also been reported in other forms of osteopetroses.

## 4- HYPOTHYROIDISM

Hypothyroidism is another possible cause of delayed eruption. Patients in whom the function of the thyroid gland is extremely deficient have characteristic dental findings.

**Congenital Hypothyroidism (Cretinism)**

Hypothyroidism occurring at birth and during the period of most rapid growth, if undetected and untreated, causes mental deficiency and dwarfism. In earlier medical and dental literature, this condition was referred to as *cretinism*. Congenital hypothyroidism is the result of an absence or underdevelopment of the thyroid gland and insufficient levels of thyroid hormone. Today it is routinely diagnosed and corrected at birth because of mandatory blood screening of newborn infants. An inadequately treated child with congenital hypothyroidism is a small and disproportionate person, with abnormally short arms and legs. The head is disproportionately large, although the trunk shows less deviation from the norm. Obesity is common.

Without adequate hormonal therapy, the dentition of the child with congenital hypothyroidism is delayed in all stages, including eruption of the primary teeth, exfoliation of the primary teeth, and eruption of the permanent teeth. The teeth are normal in size but are crowded in jaws that are smaller than normal. The tongue is large and may protrude from the mouth. The abnormal size of the tongue and its position often cause an anterior open bite and flaring of the anterior teeth. Tooth crowding, malocclusion, and mouth breathing cause a chronic hyperplastic type of gingivitis.

Although untreated congenital hypothyroidism is rare, even in developing countries, a case report documented the condition discovered in a 19-year-old boy. The patient presented with a complete caries-free primary dentition and partially erupted maxillary first permanent molars. All

primary teeth showed some abrasion. At a subsequent oral examination 1 year and 9 months after appropriate l-thyroxine therapy was initiated, several primary teeth had exfoliated, permanent incisors and first molars had erupted, and radiographs showed additional development of other permanent teeth.

**Juvenile Hypothyroidism (Acquired Hypothyroidism)**

Juvenile hypothyroidism results from a malfunction of the thyroid gland, usually between 6 and 12 years of age. Because the deficiency occurs after the period of rapid growth, the unusual facial and body patterns characteristic of a person with congenital hypothyroidism are not present. However, obesity is evident to a lesser degree. In untreated juvenile hypothyroidism, delayed exfoliation of the primary teeth and delayed eruption of the permanent teeth are characteristic. A child with a chronologic age of 14 years may have a dentition in a stage of development comparable with that of a child 9 or 10 years of age.

## 5- HYPOPITUITARISM

A pronounced slowing of the growth of the bones and soft tissues of the body will result from a deficiency in secretion of the growth hormone. Pituitary dwarfism is the result of an early hypofunction of the pituitary gland. Again, early diagnosis is routine because of the mandatory blood screening of newborn infants for congenital hypothyroidism.

An individual with pituitary dwarfism is well proportioned but resembles a child of considerably younger chronologic age. The dentition is essentially normal in size.

Delayed eruption of the dentition is characteristic. In severe cases the primary teeth do not undergo resorption but instead may be retained throughout the life of the person. The underlying permanent teeth continue to develop but do not erupt. Extraction of the deciduous teeth is not indicated because eruption of the permanent teeth cannot be ensured. Some degree of cognitive disability often occurs.

## 6- ACHONDROPLASTIC DWARFISM

Achondroplastic dwarfism, also diagnosed at birth, demonstrates a few characteristic dental findings. Growth of the extremities is limited because of a lack of calcification in the cartilage of the long bones. Stature improvements have been reported with surgical lengthening of the limbs and also with growth hormone therapy. The head is disproportionately large, although the trunk is normal in size. The fingers may be of almost equal length, and the hands are plump. The fontanels are open at birth. The upper face is underdeveloped, and the bridge of the nose is

depressed. Although the etiology of achondroplastic dwarfism is unknown, it is clearly an autosomal-dominant disorder, although sporadic spontaneous mutations occur. There is some evidence that the condition is more likely to occur when the ages of the parents differ significantly. In contrast to DS, the increased age of the father may be related to the occurrence of the condition.

Deficient growth in the cranial base is evident in many individuals with achondroplastic dwarfism. The maxilla may be small, with resultant crowding of the teeth and a tendency for open bite. Chronic gingivitis is usually present. However, this condition may be related to the malocclusion and crowding of the teeth.

## 7- OTHER CAUSES

Delayed eruption of the teeth has been linked to other disorders, including fibromatosis gingivae, Albright hereditary osteodystrophy, chondroectodermal dysplasia (Ellis-van Creveld syndrome), de Lange syndrome, frontometaphyseal dysplasia, Gardner syndrome, Goltz syndrome, Hunter syndrome, incontinentia pigmenti syndrome (Bloch-Sulzberger syndrome), Maroteaux-Lamy mucopolysaccharidosis, Miller-Dieker syndrome, progeria syndrome (Hutchinson-Gilford syndrome), and familial hypophosphatemia.

Of additional interest is the effect of bisphosphonate therapy on children with osteogenesis imperfecta. Bisphosphonates inhibit the ability of osteoclasts to resorb bone. Indeed, one study demonstrated that children with osteogenesis imperfecta treated with bisphosphonates had an associated mean delay of 1.67 years in tooth eruption. Finally, a well-laid-out diagnostic algorithm for delayed tooth eruption is shown in Figure 1.

م.د. محمد ابراهيم الزبيدي

# Delay eruption: LOCAL AND SYSTEMIC FACTORS THAT INFLUENCE ERUPTION

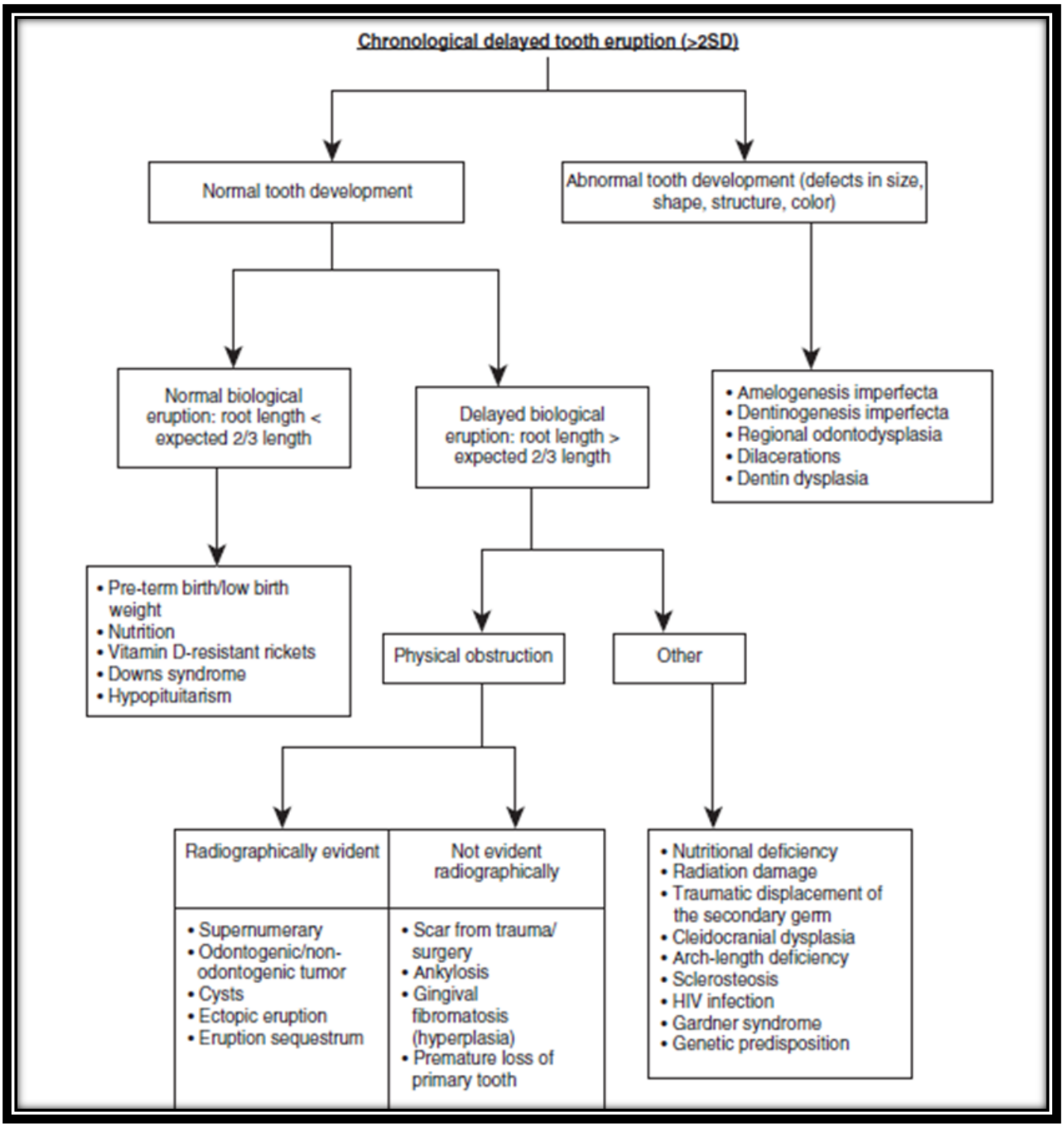


Figure 1 Diagnostic algorithm of delayed tooth eruption. (From Suri L, Gagari E, Vastardis H: Delayed tooth eruption: pathogenesis, diagnosis, and treatment. A literature review, *Am J Orthod Dentofacial Orthop* 126:435, 2004.)