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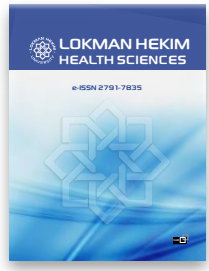

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REVIEW

Local and Systemic Factors Affecting the Eruption of Primary and Permanent Teeth in Children

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Abstract

Tooth eruption is a biological process influenced by local and systemic factors. These factors impact the development and eruption timing of primary and permanent teeth, potentially leading to early or delayed eruption. While local factors generally affect the eruption of a single primary or permanent tooth, systemic factors can influence primary dentition, permanent dentition, or all eruption periods, causing eruption problems in both dentitions. Consequently, various clinical and radiographic findings may arise. Dentists can diagnose and assess these influencing factors through clinical and radiographic evaluations, enabling them to identify existing conditions or diseases. This review aims to compile the local and systemic factors affecting the eruption of primary and permanent teeth.

Keywords: Local factors; Systemic factors; Tooth eruption

Tooth eruption is defined as the movement of a tooth from its position within the alveolar bone to its functional position in the oral cavity. This process continues until contact is made with the opposing arch's tooth. It comprises five stages: Pre-eruptive, intraosseous, mucosal penetration, pre-occlusal, and post-occlusal.[1] The eruption movement of teeth begins with the initiation of root formation.[2] Experimental studies indicate that the coronal follicle, periodontal traction, root elongation, pulpal growth, bone development, periodontal ligament pressure, pulpal pressure, and vasculature all play a role in tooth eruption. The mechanism of tooth eruption and the regulation of this process are primarily determined by systemic and local factors.[1]

Tooth eruption is controlled by biological mechanisms that depend on coordinated signaling between the dental follicle, alveolar bone, and adjacent tissues. The dental follicle plays a central role by secreting cytokines such as colony-stimulating factor-1 and regulating the receptor activator of nuclear factor kappa-B ligand/osteoprotegerin balance, which control osteoclast differentiation and bone resorption required for the eruptive pathway. Disruption of these regulatory mechanisms can delay or prevent normal eruption, highlighting the biological complexity of this process.[1,3]

Literature Search Strategy

To provide a comprehensive review of these contributing factors, this review was prepared by selecting relevant literature through searches of PubMed, Google Scholar, and Scopus using the keywords "tooth eruption," "eruption disorders," "local factors," "systemic factors," "primary teeth,"

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and "permanent teeth." Studies published in English between 2010 and 2024 were included if they provided clinical, radiographic, or etiological data related to local or systemic factors affecting tooth eruption. The exclusion criteria of this review were as follows: Studies that did not directly address the tooth eruption process, those focusing solely on treatment procedures or surgical interventions, animal model and *in vitro* experimental studies, and publications published before 2010 were excluded. These criteria were applied to ensure that only studies providing relevant, evidence-based, and up-to-date information related to the etiological factors influencing the eruption of primary and permanent teeth in children were included in the review.

Local and systemic factors affecting the tooth eruption process are summarized collectively in Figure 1. These factors are discussed in the text under their respective subheadings.

Local Factors Affecting the Eruption of Primary Teeth

Fibrotic Mucosa and Eruption Cyst

Changes in the oral mucosa overlying an erupting tooth may delay eruption in children. In particular, habitual chewing on hard objects can increase mucosal fibrosis, further contributing to eruption delay.[1] An eruption cyst is a type of developmental cyst considered the soft-tissue counterpart of a dentigerous cyst. Evidence indicates that it occurs in both primary and permanent teeth, most commonly in the permanent first molars and maxillary permanent incisors. Although its exact etiology is unknown, factors such as trauma, infection, and limited space are thought to contribute to its development.[4] Typically asymptomatic, eruption cysts do not cause pain or sensitivity in children unless secondary infection occurs. They do not present any radiographic findings. However, in cases where there is a delay in tooth eruption or an increase in lesion size, enucleation or marsupialization of the cyst may be necessary.[5]

Natal and Neonatal Teeth

Teeth present in the mouth at birth are called natal teeth, while those that erupt within the first 30 days after birth are referred to as neonatal teeth.[3,6] Natal teeth resemble primary teeth in shape and size but are generally smaller and have a yellowish color.[7] Natal teeth are most commonly found in the region of the mandibular primary central incisors, but they can also occur in the maxillary primary incisors and the regions of both mandibular and maxillary primary molars.[8] Although the exact etiology is

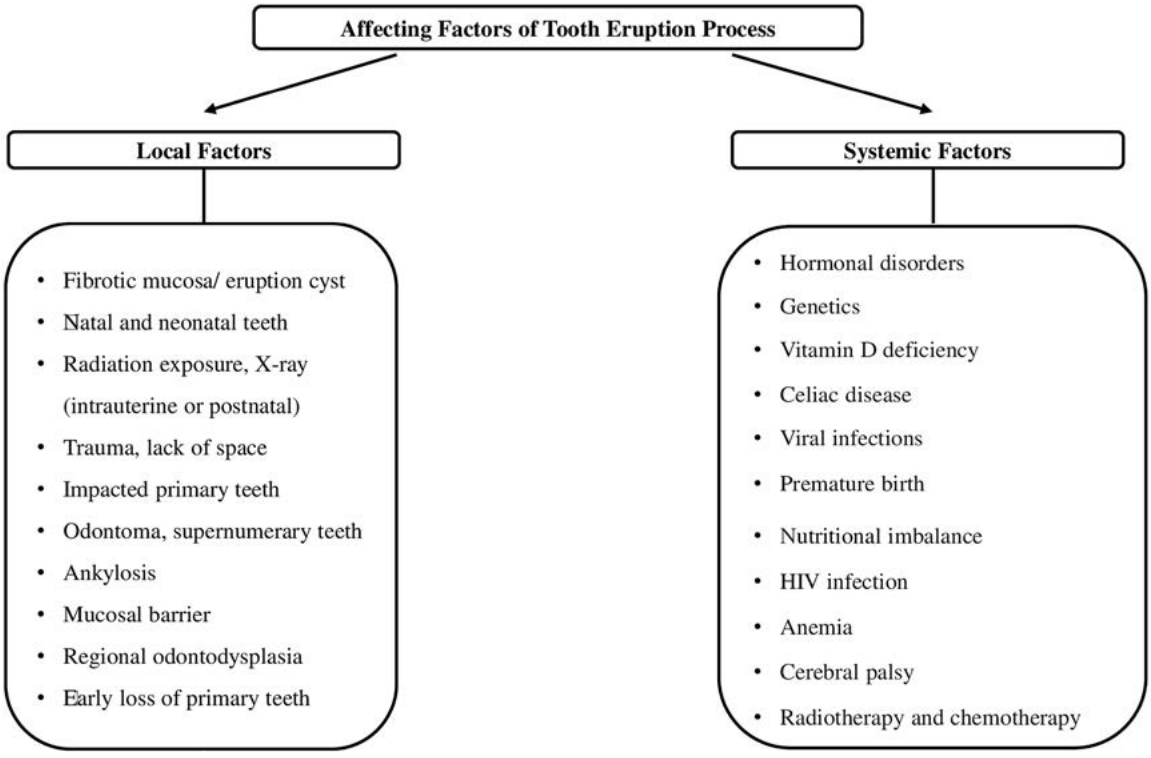

Figure 1. Local and systemic factors affecting the tooth eruption process.

not fully understood, factors such as infection, high fever, trauma, malnutrition, endocrine disorders, and kidney infections during pregnancy are believed to contribute to the formation of natal and neonatal teeth.[6,7] In addition, natal and neonatal teeth can occur in association with 50 different syndromes.[1] Some of these syndromes include Ellis-Van Creveld syndrome, Hallermann-Streiff syndrome, Jadassohn-Lewandowsky syndrome, Rubinstein-Taybi syndrome, Pierre-Robin syndrome, Craniofacial dysostosis, Steatocystoma multiplex, Sotos syndrome, Wiedemann-Rautenstrauch syndrome, Meckel-Gruber syndrome, Down syndrome, Walker-Warburg syndrome, and Ectodermal dysplasia.[1,7,8]

X-rays

Animal studies have shown that exposure of the fetus to radiation during intrauterine life can lead to various disturbances in the development and eruption of both primary and permanent teeth. The potential damage to the fetus from radiation exposure during pregnancy varies based on the dose, duration, and the stage of development of the fetus. Exposure to radiation in the early stages of pregnancy can result in the complete absence of enamel and dentin, enamel defects, and a reduction in dentin apposition in certain areas of the tooth. Additionally, it can lead to the formation of short, curved, and dilacerated tooth roots. While low doses of radiation exposure in the fetus during intrauterine life may cause delays in tooth eruption in children, the same dose applied postnatally can accelerate tooth eruption. However, high doses of radiation administered during postnatal life can delay tooth eruption and may even cause pauses in the eruption mechanism.[1,9]

Systemic Factors Affecting the Eruption of Primary Teeth

Hormonal Disorders

Hypothyroidism

Hypothyroidism affects the functions of all organs, primarily the cardiovascular, gastrointestinal, and nervous systems. In addition, thyroid hormone plays a crucial role in growth and development in children.[10] Congenital hypothyroidism is referred to as cretinism and is characterized by abnormal dwarfism.[11] Cretinism is characterized by thick lips, macroglossia, malocclusion, a flat nose, a narrow forehead, and delayed tooth eruption. Thick lips and macroglossia develop due to subcutaneous mucopolysaccharide accumulation, while the impact of hypothyroidism on craniofacial and dental tissues can result in the impaction of the mandibular permanent second molars.[12] In children with cretinism, primary dentition may begin around the ages of 3–4, while permanent dentition may start between the ages of 12–20.[11]

Hypopituitarism

Hypopituitarism refers to the hypofunction of the pituitary gland, typically caused by a pathological formation in or around the gland. Its most prominent feature is a delay in the growth of all tissues. Hypopituitarism during childhood leads to pituitary dwarfism. In pituitary dwarfism, due to developmental delays in the skeletal and genital systems, bone age lags significantly behind chronological age, and sexual development is halted. In these patients, delays in the formation and eruption of primary teeth are observed.[11]

Hypoparathyroidism

In hypoparathyroidism, teeth may exhibit cemental dysplasia, hypoplastic enamel, short and rounded roots, hypodontia, delays in tooth eruption or failure of eruption, and widening of the periodontal ligament.[13]

Genetics

Several genetic syndromes have been reported to affect the eruption of primary teeth, leading to alterations in the timing, sequence, or pattern of eruption during early childhood. These conditions often involve developmental disturbances that influence both craniofacial and dental structures. Syndromes such as Down syndrome, Ellis–Van Creveld syndrome, Hallermann–Streiff syndrome, and Goltz syndrome are among the most frequently associated with delayed or abnormal eruption of primary dentition, mainly due to their systemic and skeletal manifestations. To provide a clearer understanding, the features related to these syndromes are summarized in Table 1.[3,14–17]

Vitamin D Deficiency

Vitamin D deficiency causes rickets in children and osteomalacia in adults. Rickets is described as a pediatric condition characterized by skeletal deformities and dwarfism due to a deficiency in mineralization of bone and cartilage tissue. The clinical signs of rickets include pale skin, sweating, hair loss, bone deformities, and muscle hypertrophy. In these patients, the gonial angle is increased, and the palate becomes deeper. Changes in teeth associated with rickets include delays in the eruption and shedding of primary teeth, alterations during the eruption process, systemic hypoplasia, and anomalies in the shape and size of teeth.[18]

Table 1. Syndromes associated with eruption disorders in the primary dentition.[3,14–17]

Condition	Features
Down syndrome	Congenital heart defects, as well as eye and external ear anomalies associated with Down syndrome, begin to develop during the 6th to 8th weeks of intrauterine life.[5] Delays in the eruption of primary teeth and changes during the eruption process can be observed, particularly in the maxillary and mandibular primary anterior teeth and primary first molars. While in a normally developing healthy child, the primary central incisors erupt first and the primary second molars erupt last, children with Down syndrome may exhibit variations in this eruption sequence. The eruption of the first tooth typically occurs between 12 to 14 months; however, this timeline can extend up to 2 years, and the complete eruption of primary teeth in children with Down syndrome may not be achieved until they are 4 to 5 years old.[3,14] It has also been reported that some primary teeth in these children may persist in the mouth until the age of 14 to 15.[3]
Elis-van Creveld syndrome	Ellis-Van Creveld syndrome is a genetic disorder inherited in an autosomal recessive manner, with its exact prevalence not fully known. The characteristic features of the syndrome include short ribs, polydactyly, growth retardation, ectodermal anomalies, and congenital heart defects. The most commonly observed oral manifestations are hypodontia and delayed eruption. In some patients, natal teeth and early erupting primary teeth observed within the first two months after birth may be present. Additionally, dental transpositions, enamel hypoplasia, taurodontism, multiple frenula, or frenulum anomalies are among the other oral findings that can be seen.[15]
Goltz syndrome	Goltz syndrome, also known as focal dermal hypoplasia, is an X-linked dominant disorder that affects multiple systems and can progress to be fatal. It is inherited genetically. The symptoms of Goltz syndrome can affect multiple systems, primarily involving the skin, bone tissue, eyes, and teeth. The manifestations associated with the syndrome can be of a severity that poses a threat to life.[16]
Hallermann-striff syndrome	Hallermann-Streiff syndrome is a very rare congenital disorder. Its characteristic findings include craniofacial abnormalities, ocular malformations that contribute to a disease-specific facial phenotype, hair thinning, skin atrophy, short stature, skeletal anomalies, and developmental delays. Dental anomalies observed in patients include tooth agenesis, persistent primary teeth, malocclusion, open bite, malformed teeth, severe dental caries, early eruption of primary teeth, supernumerary teeth, and natal-neonatal teeth.[17]

Celiac Disease

Celiac disease is defined as an immune system disorder that causes hypersensitivity to gluten found in grains and grain products, often accompanied by malabsorption. Studies have indicated that children with this condition frequently present with oral manifestations such as enamel hypomineralization, poor oral hygiene, an increased risk of caries, and recurrent aphthous ulcers.[19] Moreover, children with celiac disease have been reported to experience delayed tooth eruption and maturation in both primary and permanent dentitions compared to healthy peers. This delay is thought to be associated with nutritional deficiencies and developmental disturbances related to the disease.[20]

Viral Diseases

Among viral diseases, the Rubella virus, which belongs to the Togaviridae family, is transmitted from the mother to the fetus and may result in congenital rubella syndrome.[21] Infants born with congenital rubella syndrome may present with various conditions and anomalies, including cataracts, cardiac anomalies, sensorineural hearing loss, microcephaly, speech and behavioral disorders, diabetes, bone lesions, thrombocytopenic purpura, hepatitis, and pneumonia.[21] In addition, a study conducted on children who have had this disease reported oral symptoms such as enamel hypoplasia, hypoplastic enamel defects, and delays in the eruption of primary teeth.[22]

Premature Birth

Births occurring before 37 weeks of gestation are termed premature births. Premature infants typically have birth weights that are below the general average. Babies born prematurely or with low birth weight are predisposed to numerous medical issues during the neonatal development period, which can adversely affect the eruption of teeth in both primary and permanent teeth.[23]

Local Factors Affecting the Eruption of Permanent Teeth

Impacted Primary Teeth

The failure of a tooth to take its position in the occlusion when the time for eruption has arrived, often due to a mechanical obstruction, is referred to as tooth impaction. Impacted teeth are commonly found in the permanent dentition, particularly with the third molars (wisdom teeth), while impaction of primary teeth is quite rare.[24] This refers to the failure in the eruption process of teeth and can obstruct the eruption of permanent teeth. The most commonly impacted primary teeth, in order, are the primary second molars,

maxillary and mandibular primary central incisors, primary canines, and primary lateral incisors. The least frequently impacted primary teeth are the primary first molars. In terms of treatment, the primary tooth may be surgically extracted depending on the clinical situation, or managed through periodic monitoring without intervention.[25]

Ectopic Eruption

Ectopic tooth eruption is defined as the abnormal positioning of a tooth during its emergence into the oral cavity. The teeth most commonly affected by ectopic eruption are, in order of frequency: Permanent molars and canines in the maxilla, permanent second premolars and canines in the mandible, and permanent lateral incisors in the maxilla.[26]

Supernumerary Teeth and Odontoma

Supernumerary teeth, including mesiodens and odontomas, are among the primary issues causing tooth eruption disorders in children. If these formations are located around the permanent teeth follicles, they can lead to the permanent tooth remaining impacted and failing to erupt.[1]

Odontomas are typically asymptomatic and are often discovered incidentally during routine radiographic examinations. The primary problems caused by odontomas include dental malformations, retention of primary teeth, delayed eruption of permanent teeth, pain, cortical bone expansion, malposition, cyst formation, displacement of adjacent teeth, and resorption. The most commonly affected teeth due to odontomas are the permanent canines, maxillary central incisors, and third molars. To prevent odontomas from causing eruption issues in neighboring permanent teeth, surgical removal is recommended when one-third of the root development of the adjacent permanent teeth is completed.[27]

Mucosal Barrier

The mucosal barrier is one of the etiological factors that can lead to delayed eruption of permanent teeth. Delays in the degradation of the mucosa may occur as a result of the inability of the dental follicle to integrate with the mucosa during the eruption process, thereby obstructing the eruption of the tooth.[28]

Ankylosis

The etiology of ankylosis is not fully understood; however, it is believed that genetic factors, metabolic conditions, congenital absence of premolars, dental traumas, avulsion, and luxation injuries may contribute to its development. Ankylosis can lead to various complications, including the cessation of alveolar process growth, resulting in open bite formation, an unesthetic smile, infraocclusion of the ankylosed tooth, tilting of adjacent teeth, elongation of antagonist teeth, and malocclusion. Conditions associated with ankylosis include cleidocranial dysplasia, ectodermal dysplasia, osteopetrosis, hypopituitarism, hypothyroidism, Fanconi syndrome, Down syndrome, epidermolysis bullosa, and deficiencies of vitamins A and D.[29]

Dental Trauma

The close relationship between the apex of the primary tooth and the germ of the permanent teeth can lead to eruption problems in the permanent teeth following trauma to the primary teeth. The severity of the trauma is dependent on the patient's age, the developmental stage of the permanent teeth germs, the type and size of the trauma, and the extent of root resorption. According to research, the injuries that most adversely affect the permanent teeth germs are reported to be intrusive and avulsive injuries. Problems observed in permanent teeth as a result of trauma to primary teeth include changes in enamel color, enamel hypoplasia, coronal laceration, root lacerations, odontoma-like malformations, and eruption problems.[30]

Regional Odontodysplasia

Regional odontodysplasia is defined as a rare developmental anomaly characterized by hypoplasia and hypocalcification of dental hard tissues, including enamel and dentin. Evidence indicates that it may affect both primary and permanent teeth, with a predilection for the maxilla, particularly the left segment.[31]

Lack of Space

Lack of space is an important etiological factor for dental misalignment and the impaction of teeth. Research has indicated that a reduced arch length delays the eruption of maxillary permanent second molars, although it does not have any effect on tooth development. Moreover, it has been emphasized that crowding not only leads to delayed eruption but also often results in ectopic eruptions.[28]

Early Loss of Primary Teeth

The primary causes of early loss of primary teeth are reported to be dental caries and trauma. Table 2 presents the systemic diseases that can lead to early loss of primary teeth.[32] Early loss of primary teeth can result in elongation of antagonist teeth, migration and inclination of adjacent teeth, impaction of permanent teeth, or eruption problems (either delayed or premature), leading to a shift in the midline.[33]

Table 2. Systemic diseases that cause early loss of primary teeth[32]

Systemic disease	Causes of early loss of primary teeth
Papillon–Lefevre syndrome	Periodontitis due to insufficient cortisol production
Erythromelalgia	Periodontitis associated with the accumulation of Langerhans cells
Congenital adrenal hyperplasia	Periodontitis related to metabolic absorption disorders
Langerhans cell histiocytosis	Periodontitis due to accelerated cellular aging
Short bowel syndrome	Periodontitis resulting from abnormalities in osteoblast and osteoid functions
Dyskeratosis congenita	Periodontitis due to osteoclastic degeneration in the jaws
Hajdu–Cheney syndrome	Insufficient development of acellular cementum
Cherubism	Insufficient development of acellular cementum
Hypophosphatasia	Decreased production and release of neutrophils
Coffin–Lowry syndrome	Phagocytic dysfunction
Cyclic neutropenia	Phagocytic dysfunction
Leukocyte adhesion deficiency type I	Reduced leukocyte chemotaxis and phagocytic dysfunction
Down syndrome	Deficiency of the catalase enzyme
Chediak–Higashi syndrome	Periodontitis due to insufficient cortisol production
Acatalasemia	Periodontitis associated with the accumulation of Langerhans cells

Table 3. Syndromes associated with delayed eruption and disturbances[34]

Amelogenesis imperfecta	Goltz syndrome	Osteogenesis imperfecta
Apert syndrome	Hallermann-striff syndrome	Osteopetrosis
Carpenter syndrome	Hereditary gingival fibromatosis	Otodental syndrome
Cherubism	Hyper IgE syndrome (Buckley syndrome)	Parry-Romberg syndrome (PRS)
Dentin dysplasia	Incontinentia Pigmenti (Bloch-Sulzberger syndrome)	Progeria (Hutchinson-Gilford syndrome)
Down syndrome	Cleidocranial Dysplasia	Rothmund-Thomson syndrome
Ectodermal dysplasia	Ellis-van Creveld syndrome	SHORT syndrome
GAPO syndrome	Mucolipidosis Type II (I-Cell) disease	Singleton–Merten syndrome
Gaucher syndrome	Mucopolysaccharidosis	Von Recklinghausen disease (Neurofibromatosis Type I)

Systemic Factors Affecting the Eruption of Permanent Teeth

Genetics

Certain genetic disorders have been reported to affect tooth eruption as amelogenesis imperfecta, osteogenesis imperfecta, cleidocranial dysplasia, etc. Table 3 presents a list of genetic conditions that are associated with delayed eruption and related disturbances.[34]

Gender

Research on tooth eruption has indicated that permanent teeth generally erupt earlier in girls than in boys, with an average difference in eruption timing ranging from 4 to 6 months. Teeth that exhibit significant differences in eruption timing based on gender include the maxillary permanent lateral incisors and canines, as well as the mandibular permanent canines. It is believed that this difference arises because girls typically begin pubertal development earlier than boys, leading to the earlier eruption of permanent teeth. In addition, variations in eruption order can also be observed based on gender. In girls, the maxillary permanent canines are expected to erupt before the second premolars, while the mandibular second premolars are anticipated to erupt before the permanent second molars. Conversely, in boys, the order of eruption is reported to be the reverse.[34]

Vitamin and Mineral Deficiency

The deficiency or excess of vitamins and minerals significantly impacts the development of oral and dental structures. In particular, deficiencies in calcium, phosphorus, and Vitamins A, B, C, and D can lead to hypomineralization, delayed eruption of teeth, gingival bleeding, disrupted alveolar bone patterns, and periodontal diseases. Among the oral effects, the literature contains the most studies on Vitamin D.

Vitamin D is essential for the absorption of elements such as calcium, magnesium, and phosphorus from the intestines, which are necessary for the calcification of bones and teeth. Long-term Vitamin D deficiency can lead to delayed tooth eruption associated with hypoparathyroidism, while hyperparathyroidism, which occurs in the opposite situation, increases the rate of the calcification process.[35]

Nutrition

Childhood obesity is becoming one of the significant health issues worldwide, with an increasing prevalence. Obesity refers to an abnormal or excessive accumulation of body fat that poses a risk to health. A study has indicated that obesity affects the eruption of permanent teeth, noting that obese children aged 10–11 years tend to have a greater number of erupted permanent teeth compared to their non-obese counterparts.[36] In addition, because obese children tend to enter puberty earlier, they are likely to erupt their teeth approximately 1.2–1.5 years earlier.[34]

Human Immunodeficiency Virus (HIV) Infection

HIV is one of the factors that can affect tooth eruption in children. Children with HIV infection commonly experience periodontal problems; however, delayed tooth eruption has also been reported in these individuals.[1] Research on the relationship between HIV infection and delayed tooth eruption has indicated that children infected with HIV may face health and nutritional problems due to their low socioeconomic status. Consequently, these issues can contribute to delays in tooth eruption among these children.[6]

Anemia

The most common oral findings in patients with anemia include pallor of the gums, tongue, mucosa, and lips. Specifically, in individuals with sickle cell anemia, literature has noted issues related to dental eruption, with observed oral findings including mucosal pallor, jaundice, especially on the soft palate and floor of the mouth due to hemolysis, delayed eruption of teeth, enamel hypoplasia, symptomatic pulp necrosis, pulp stones, trabecular changes in the bone, and osteomyelitis. In addition, it has been reported that these patients are predisposed to dental infections. Therefore, it is crucial for dental practitioners to implement preventive measures for these individuals.[37]

Cerebral Palsy

Cerebral palsy is a condition that arises from brain lesions that can occur during a child's development, leading to perceptual or sensory issues and resulting in motor dysfunction. Patients with cerebral palsy often struggle with maintaining oral hygiene, making them more susceptible to oral problems. The oral issues commonly observed in these patients include musculoskeletal anomalies, early dental eruption, mouth breathing, and increased overjet and overbite due to inadequate lip closure.[38]

Radiotherapy and Chemotherapy

Cancer is one of the most common causes of death in childhood, following accidents. In children undergoing oncological treatment, the side effects of chemotherapy are generally shorter in duration and temporary, whereas the effects of radiotherapy tend to be more long-lasting and permanent. These treatments can lead to changes in the quantity and composition of saliva. In addition, they can result in anomalies related to tooth size (often microdontia), number (commonly agenesis), crown-root structure, crown-root relationship, and hard tissue calcification. Delays in tooth eruption and an increased risk of caries and periodontal diseases may also be observed. The most serious complications in dental development occur as a result of radiation exposure during the formative and differentiation stages of tooth development. Complications seen in primary teeth can lead to severe malocclusions and deficiencies in facial development.[39]

Drug Use

The use of long-term chemotherapy agents, medications that inhibit the prostaglandin pathway (such as aspirin, acetaminophen, ibuprofen, indomethacin, and clodronate), as well as bisphosphonate-containing drugs, can reduce osteoclastic activity in periodontal tissues. This reduction may slow the rate of tooth eruption and lead to delays in the eruption process.[40]

Socioeconomic Factors

Some studies have found that children from higher socioeconomic backgrounds tend to experience earlier eruption of teeth compared to those from lower socioeconomic backgrounds. It is suggested that this phenomenon may be attributed to the healthier and better nutritional status of children with higher socioeconomic status, which triggers earlier tooth eruption.[34]

Evaluation of the Literature and Clinical Considerations

A comparative analysis of the literature suggests that systemic factors – particularly hormonal and nutritional disorders – have stronger and more consistent effects on eruption timing than local mechanical factors. While endocrine disorders

such as hypothyroidism and hypopituitarism are commonly associated with delayed eruption, nutritional excesses or deficiencies (e.g., vitamin D imbalance, obesity) may accelerate or decelerate eruption depending on the context. However, the available evidence remains heterogeneous due to differences in study design, population characteristics, and diagnostic criteria. These inconsistencies highlight the need for standardized assessment protocols and longitudinal clinical research to clarify the relative impact of systemic and local influences on tooth eruption.

In clinical practice, early recognition of deviations in tooth eruption patterns is essential for timely diagnosis and management. Pediatric dentists should evaluate whether an eruption disturbance has a local or systemic origin and act accordingly – implementing interceptive measures for local causes and initiating multidisciplinary consultation when systemic involvement is suspected. Regular monitoring, preventive guidance, and early intervention help minimize complications such as malocclusion and ensure proper development of the dentition.

Conclusion

Tooth eruption is an important biological process influenced by numerous mechanisms and factors. The eruption process is influenced by two major groups of factors: systemic and local factors. These factors can either slow down the eruption process, leading to delays, or accelerate it, resulting in early tooth eruption. Changes observed during this process may indicate the presence of a disease, syndrome, genetic condition, or environmental factor, all of which fall within the scope of pediatric dentistry. The pediatric dentist should identify the underlying cause of the eruption problem through clinical and radiological examinations and evaluations, subsequently developing a treatment plan tailored to this factor. Local factors can typically be managed through treatments implemented in dental clinics. However, since the effects of systemic factors extend beyond the oral cavity and may also cause other systemic manifestations, their management may require a multidisciplinary approach involving collaboration among physicians from various specialties, including a pediatric dentist. Therefore, pediatric dentists must possess a thorough understanding of both local and systemic factors that may be associated with eruption problems and should be able to provide appropriate referrals as necessary.

Despite advances in understanding the biological and clinical aspects of tooth eruption, many mechanisms underlying eruption disturbances remain unclear. Future research should focus on identifying molecular and genetic pathways that regulate eruption timing and tooth movement, as well as the interaction between systemic diseases and dental development. Moreover, longitudinal and population-based studies are needed to establish standardized diagnostic criteria and evidence-based clinical guidelines for managing eruption disturbances in pediatric patients.

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References

1. Koch G, Kreiborg S, Andreasen JO. Eruption and shedding of teeth. In: Koch G, Poulsen S, Espelid I, Haubek D, editors. Pediatric Dentistry: A Clinical Approach. 3rd ed. Chichester (UK): Wiley Blackwell; 2016. p. 40-54.
2. Jain P, Rathee M. Anatomy, head and neck, tooth eruption. [Updated 2023 Sep 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available at: https://www.ncbi.nlm.nih.gov/books/NBK549878/
3. Dean JA, Turner EG. Eruption of the teeth: local, systemic, and congenital factors that influence the process. In: Dean JA, editor. McDonald and Avery's Dentistry for the Child and Adolescent. 10th ed. St. Louis (MO): Elsevier; 2016. p. 349-74. [CrossRef]
4. Figueiredo N, Meena M, Ajit DD, Khorate M, Satoskar S. EruptionCyst: A Case Report. Ann Essence Dent 2013;5:9-11.
5. Şen-Tunç E, Açikel H, Sönmez I S, Bayrak Ş, Tüloğlu N. Eruption cysts: A series of 66 cases with clinical features. Med Oral Patol Oral Cir Bucal 2017;22(2):e228-e32. [CrossRef]
6. Demirel T, Bodrumlu EH. Eruption Anomalies. J Int Dent Sci 2018;(3):141-6. [in Turkish] [CrossRef]
7. Anusha Durairaj B, Gajula Shivashankarappa P, Muthukrishnan K, Saraswat Y. Natal and neonatal teeth: early diagnosis and management. BMJ Case Rep 2023;16(11):e256249. [CrossRef]
8. Mhaske S, Yuwanati MB, Mhaske A, Ragavendra R, Kamath K, Saawarn S. Natal and neonatal teeth: an overview of the literature. ISRN Pediatr 2013;2013:956269. [CrossRef]
9. Sønstevold T, Johannessen AC, Stuhr L. A rat model of radiation injury in the mandibular area. Radiat Oncol 2015;10:129. [CrossRef]

10. Wassner AJ. Pediatric Hypothyroidism: Diagnosis and Treatment. Paediatr Drugs 2017;19(4):291-301. [CrossRef]
11. Reis CLB, Barbosa MCF, Henklein S, Madalena IR, de Lima DC, Oliveira MAHM, Küchler EC, de Oliveira DSB. Nutritional Status is Associated with Permanent Tooth Eruption in a Group of Brazilian School Children. Glob Pediatr Health 2021;8:2333794X211034088. [CrossRef]
12. Chandna S, Bathla M. Oral manifestations of thyroid disorders and its management. Indian J Endocrinol Metab 2011;15(Suppl 2):S113-6. [CrossRef]
13. Shoback DM, Bilezikian JP, Costa AG, Dempster D, Dralle H, Khan AA, et al. Presentation of Hypoparathyroidism: Etiologies and Clinical Features. J Clin Endocrinol Metab 2016;101(6):2300-12. [CrossRef]
14. Mayoral-Trias MA, Llopis-Perez J, Puigdollers Pérez A. Comparative study of dental anomalies assessed with panoramic radiographs of Down syndrome and non-Down syndrome patients. Eur J Paediatr Dent 2016;17(1):65-9.
15. da Silva JD, Tkachenko N, Soares AR. Ellis-van Creveld syndrome. In: Adam MP, Bick SL, Mirzaa GM, Pagon RA, Wallace SE, Amemiya A, editors. GeneReviews®. Seattle (WA): University of Washington, Seattle; 1993-2026. Available at: https://www.ncbi.nlm.nih.gov/books/NBK596643/
16. Wang L, Jin X, Zhao X, Liu D, Hu T, Li W, Jiang L, Dan H, Zeng X, Chen Q. Focal dermal hypoplasia: updates. Oral Dis 2014;20(1):17-24. [CrossRef]
17. Schmidt J, Wollnik B. Hallermann-Streiff syndrome: A missing molecular link for a highly recognizable syndrome. Am J Med Genet C Semin Med Genet 2018;178(4):398-406. [CrossRef]
18. Zhang YF, Xu JW, Yang Y, Huang X, Yu XQ. The association between body lead levels and childhood rickets: A meta-analysis based on Chinese cohort. Medicine (Baltimore) 2019;98(8):e14680. [CrossRef]
19. Elbek-Cubukcu C, Arsoy HA, Ozkaya G. Assessment of oral manifestations in pediatric patients with celiac disease in relation to marsh types. Med Oral Patol Oral Cir Bucal 2023;28(1):e9-e15. [CrossRef]
20. Wieser H, Amato M, Caggiano M, Ciacci C. Dental manifestations and celiac disease-an overview. J Clin Med 2023;12(8):2801. [CrossRef]
21. Mawson AR, Croft AM. Rubella virus infection, the congenital rubella syndrome, and the link to autism. Int J Environ Res Public Health 2019;16(19):3543. [CrossRef]
22. Bhatia SK, Goyal A, Dubey M, Kapur A, Ritwik P. Congenital rubella syndrome: dental manifestations and management in a 5 year old child. J Clin Pediatr Dent 2012;37(1):71-5. [CrossRef]
23. Buhamer SN, Kaklamanos E, Kowash M, Hussein I, Salami A, Al-Halabi M. What is the effect of preterm birth on permanent tooth crown dimensions? A systematic review and meta-analysis. PLoS One 2021;16(11):e0259293. [CrossRef]
24. Ness GM, Blakey GH, Hechler BL. Impacted teeth. In: Miloro M, Ghali GE, Larsen PE, Waite PD, editors. Peterson's Principles of Oral and Maxillofacial Surgery. 4th ed. Cham (CH): Springer; 2022. p. 131-169. [CrossRef]
25. Bulut S, Uzun T, Toptaş O. Mandibular Impacted Primary Molar Tooth: A Case Report. Cumhuriyet Dental Journal 2016;19(2):129-34.
26. Tirali EB, Yalçınkaya Erdemci Z, Çehreli SB. Eruption Anomalies. GÜ Diş Hek Fak Derg 2011;28(3):217-23. [in Turkish]
27. Gündüz K, Büyük C. Odontoma, Adenomatoid Odontogenic Tumor. Türkiye Klinikleri J Oral Maxillofac Radiol-Special Topics 2017;3(1):13-7. [in Turkish]
28. Alshukairi H. Delayed tooth eruption and its pathogenesis in paediatric patient: a review. J Dent Health Oral Disord Ther 2019;10(3):209-12. [CrossRef]
29. Şenışık NE, Akalın Y. Dental ankylosis: treatment alternatives. Journal of Ege University School of Dentistry 2016;37(2):75-87. [in Turkish] [CrossRef]
30. Caeiro-Villasenín L, Serna-Muñoz C, Pérez-Silva A, Vicente-Hernández A, Poza-Pascual A, Ortiz-Ruiz AJ. Developmental dental defects in permanent teeth resulting from trauma in primary dentition: a systematic review. Int J Environ Res Public Health 2022;19(2):754. [CrossRef]
31. Nijakowski K, Woś P, Surdacka A. Regional odontodysplasia: a systematic review of case reports. Int J Environ Res Public Health 2022;19(3):1683. [CrossRef]
32. Spodzieja K, Olczak-Kowalczyk D. Premature loss of deciduous teeth as a symptom of systemic disease: a narrative literature review. Int J Environ Res Public Health 2022;19(6):3386. [CrossRef]
33. Nadelman P, Bedran N, Magno MB, Masterson D, de Castro ACR, Maia LC. Premature loss of primary anterior teeth and its consequences to primary dental arch and speech pattern: A systematic review and meta-analysis. Int J Paediatr Dent 2020;30:687-712. [CrossRef]
34. Almonaitiene R, Balciuniene I, Tutkuviene J. Factors influencing permanent teeth eruption. Part one--general factors. Stomatologija 2010;12(3):67-72.
35. Şahin M, Toptanci İR. The role of vitamin d in eruption; review. HRU International Journal of Dentistry and Oral Research 2022;2(2):111-5. [in Turkish]
36. Mohamedhussein N, Busuttil-Naudi A, Mohammed H, UlHaq A. Association of obesity with the eruption of first and second permanent molars in children: a systematic review. Eur Arch Paediatr Dent 2020;21(1):13-23. [CrossRef]
37. Lopes CMI, Cavalcanti MC, Alves E Luna AC, Marques KMG, Rodrigues MJ, DE Menezes VA. Enamel defects and tooth eruption disturbances in children with sickle cell anemia. Braz Oral Res 2018;32:e87. [CrossRef]
38. Uysal C, Aslan BI. Neurodevelopmental disorders and orthodontics. Acta Odontologica Turcica 2014;31(1):36-42. [in Turkish] [CrossRef]
39. Delilbaşı AE. Oral and dental health in children receiving oncological therapy. Acta Odontologica Turcica 2013;30(1):39-43. [in Turkish]
40. Choukroune C. Tooth eruption disorders associated with systemic and genetic diseases: clinical guide. J Dentofac Anom Orthod 2017;20(4):402. [CrossRef]