DENTAL ANOMALIES: CLASSIFICATION, CAUSES AND TREATMENT

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The close relationship between oral health, systemic and psychological health requires a thorough assessment of oral health as part of health maintenance monitoring. Understanding the normal sequence and patterns of teething is the basis for identifying and treating children with dental malformations and optimizing the condition of their oral cavity. The difference between normal and pathological dental development requires a thorough assessment of the patient, including medical, dental and family history, clinical examination, x-ray evaluation and, possibly, special laboratory tests.

Problems of classification of dental anomalies

In the specialized literature there are a number of works by soviet and russian clinical researchers devoted to the systematization of dental anomalies in classification. Thus, n.i. agapov (1928) in the proposed classification – anomalies of the dental system identified six types of anomalies of individual teeth: shapes, structures of magnitude, numbers, eruption, tooth color; v.y. kurlandsky (1965) identifies anomalies of shape and magnitude with their nosological names: macro, microdentia, spiny, cuboid and etc.; d.a. kalvelis (1964), in the clinical and morphological classification of dental anomalies, supplemented the section anomalies of individual teeth, identified four signs of anomaly differences with a list of up to nine nosological names or conditions. Russian clinicians f.y. khoroshilkina (2005), l.s. persin (2006) in their classifications presented a more complete list of dental anomalies and explanations are given to some.

A critical analysis of the classifications allows us to say that all classifications reflect:

• a simple, habitually consistent list of names of anomalies that determine the diagnosis with different terminology, with little diagnostic information about their signs;

• the methodically unjustified principle of combining types of dental anomalies and anomalies of tooth position: n.i. agapov – in the first class; v.y. kurlandsky – in the first group; f.ya. Khoroshilkina and l.s. persin – in the first section of the classification, with the exception of d.a. kalvelis, who methodically identified dental anomalies separately in the the first section of the classification.

To date, none of the classifications of soviet, russian, and foreign researchers have systematized anomalies of temporary teeth in children. Although their variety and frequency in the structure of the dental anomaly are insignificant, but it is possible to emphasize the statement that the dental anomaly has "rejuvenated". In practice, this issue is not without interest from the point of view of diagnosing signs, determining the diagnosis, understanding the causes and timely implementation of therapeutic and preventive measures.

Icd 10 revision (2003), class xi "diseases of the digestive system" (k00-k93) the block "families" of diseases of the oral cavity, salivary glands and jaws (k00-k14) contains headings with alphanumeric codes (k00.0, k00.1, k00.2, k00.4, k00.6), related to the list of diagnosis or dental condition, where the nosological names of the classification of d.a. kalvelis (1964) are partially preserved, with the exception of the heading (k00.8) tooth color. Categories (k00.2, k00.6) contain multiple explanatory terms that are not accurate enough, imperfect. Of course, the headings require revision, clarification and additions without destroying their codes, since the icd 10 revision has the statistical function of a list of disease diagnoses adapted for use in reporting from different countries and dental anomalies are no exception, which are not fully presented and different terminologies.

Teething abnormalities

Natal and neonatal teeth. The teeth that are in the oral cavity at the time of birth are natal teeth. Those that erupt during the newborn period are neonatal teeth. Most of the "congenital" teeth are the milk incisors of the lower jaw, and not additional or super-complete teeth. Natal teeth can be associated with various syndromes, including chondroectodermal dysplasia (ellis-van creveld syndrome), congenital pachionychia, sotos syndrome and hallerman-schreiff syndrome.

Treatment of "congenital" teeth may include observation, smoothing of the cutting edge (to prevent potential discomfort during breastfeeding and ulceration of the ventral part of the tongue or the floor of the oral cavity [riga-fede disease]) or removal. The removal of "congenital" teeth should only be considered if they cause difficulties in feeding the child or mother. Excessive mobility of the natal teeth creates a risk of aspiration, which, however, is extremely rare.

Teething problems. Complete absence of teething of baby teeth is rare, except for congenital absence of teeth. The non-eruption of one permanent tooth in an otherwise healthy child is more common and has a number of reasons described below.

Insufficient space. The most common reason for the impossibility of eruption of permanent teeth is the lack of a place for eruption. The lack of space in the dental arch often causes a violation of the eruption (retination) of the third molars, canines of the upper jaw and second premolars of the lower jaw. Insufficient space for eruption can also lead to eruption outside the normal position or ectopic eruptions.

Insufficient space for eruption is treated by selective removal of permanent teeth or orthodontic therapy, depending on the severity and location of the space deficit.

Widely spaced front teeth. The widely spaced central incisors of the upper jaw (diastema) may be the result of a mismatch in the size of the jaws and teeth or the attachment of the labial frenulum to the alveolar ridge between the crowns of the central incisors.

Another reason for widely spaced front teeth is mesiodens, an overcomplicated tooth located between the central incisors of the upper jaw. The frequency of mesiodenses is increased in boys with nancy-horan syndrome. Mesiodenes in baby teeth usually require removal because they prevent the eruption of permanent incisors.

Ankylosis, or the fusion of developing teeth with the underlying bone, can stop the normal process of teething. In a child who is still growing, an ankylosed tooth will overgrow the surrounding teeth, which continue to erupt. Ankylosis should be

suspected if the tooth does not come into contact with the teeth of the opposite arch when the teeth are closed. Ankylosis most often affects the first milk molars and is relatively common in baby teeth. Ankylosed baby teeth usually peel off normally without treatment if they have a permanent successor, however, treatment of ankylosis may be necessary depending on the age of occurrence and severity of the problem.

Clavicular cranial dysplasia is an autosomal dominant disease characterized by hypoplasia or aplasia of the collarbones, a defect in bone formation, low growth, over-complete teeth, a defect in cement formation and abnormal eruption of permanent teeth. It is believed that the violation of teething is caused by an obstacle to the migration of permanent teeth into the oral cavity due to defects in osteoclastic and resorptive activity of the alveolar bone. Clavicular cranial dysplasia is caused by mutations in the gene for transcription factor 2 associated with runt (runx2), a member of the runx family of transcription factors located on chromosome 6p21. The treatment of oral manifestations of clavicular cranial dysplasia includes the removal of supercomplicated teeth and the promotion of eruption of permanent teeth through surgery or orthodontics.

Primary eruption insufficiency is not caused by mechanical obstruction and has a non-syndromic hereditary basis. Mutations in the parathyroid hormone receptor (pthr1) gene are associated with primary teething insufficiency in some families and may affect one or more permanent teeth. Primary eruption insufficiency most often affects the first and second molars.

Super-complete teeth. Although the reported prevalence of overcomplicated teeth varies (usually from 1.2 to 3 percent), they can occur in ≥6 percent of the normal population. Overcomplicated teeth are more common among permanent teeth than among baby teeth. They are more common in boys than in girls.

Overcomplicated teeth are usually considered idiopathic, although there is some evidence of the involvement of molecular factors in the regulation of their development. They have been linked to several genetic disorders and may provide an early clue to diagnosis. Overcomplicated teeth are associated with the following disorders, which are documented or suspected to be related to abnormalities of a single gene:

- clavicular cranial dysplasia
- familial adenomatous polyposis
- trichorhinophalangeal syndrome, type i
- rubinstein-tybee syndrome
- nancy-horan syndrome
- gbbb opitz syndrome
- oculofaciocardiodental syndrome
- autosomal dominant robinov syndrome

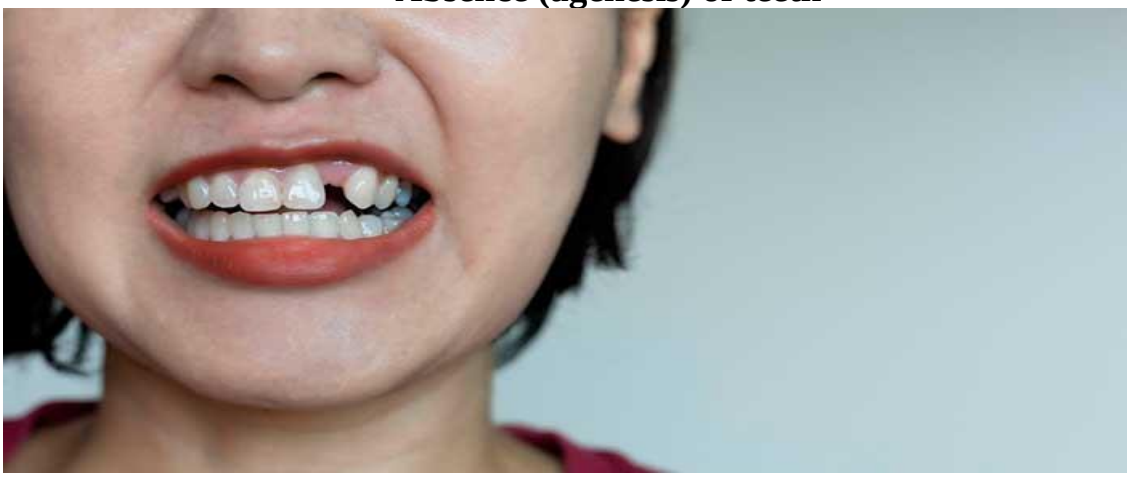
Double (or twinned) teeth occur when two teeth are joined together. Double teeth are found in 3% of children. Most often these are the milk lower central incisors.

Double teeth can occur due to an anomaly of germination (the rudiment of a tooth tries to form a second rudiment, which leads to fused teeth) or the fusion of two teeth. When double teeth result from fusion, the number of teeth decreases. Two adjacent teeth can be connected by enamel, dentin or cement. Cement fusion (i.e. Complete

fusion) is usually associated with an injury or abnormal position of one of the affected teeth.

Children with double teeth should be referred to a dental office to be monitored for complications, including delayed prolapse and permanent malocclusion (for example, hypodontia, aplasia, retention, over-complete teeth, permanent double teeth).

Absence (agenesis) of teeth



An anomaly in the formation of teeth, which leads to their absence. It can be either congenital or acquired.

Congenital tooth loss may be the result of genetic disorders that affect teeth in isolation or as part of a syndrome.

The prevalence of congenital toothlessness depends on the race and type of teeth. Congenital absence of teeth is more common in the white population than in the black population (5 percent versus 1), and in permanent bite than in milk bite. The lateral incisor of the upper jaw and the second premolar are the most frequently missing permanent teeth, and the central incisor of the lower jaw is the most frequently missing baby tooth.

Many children with missing teeth have a family member with a similar history. Several specific genetic mutations have been identified for missing teeth. For example, a missense mutation in the MSX1 gene, which encodes a transcription factor, can cause an autosomal dominant sign of the absence of lateral incisors and third molars.

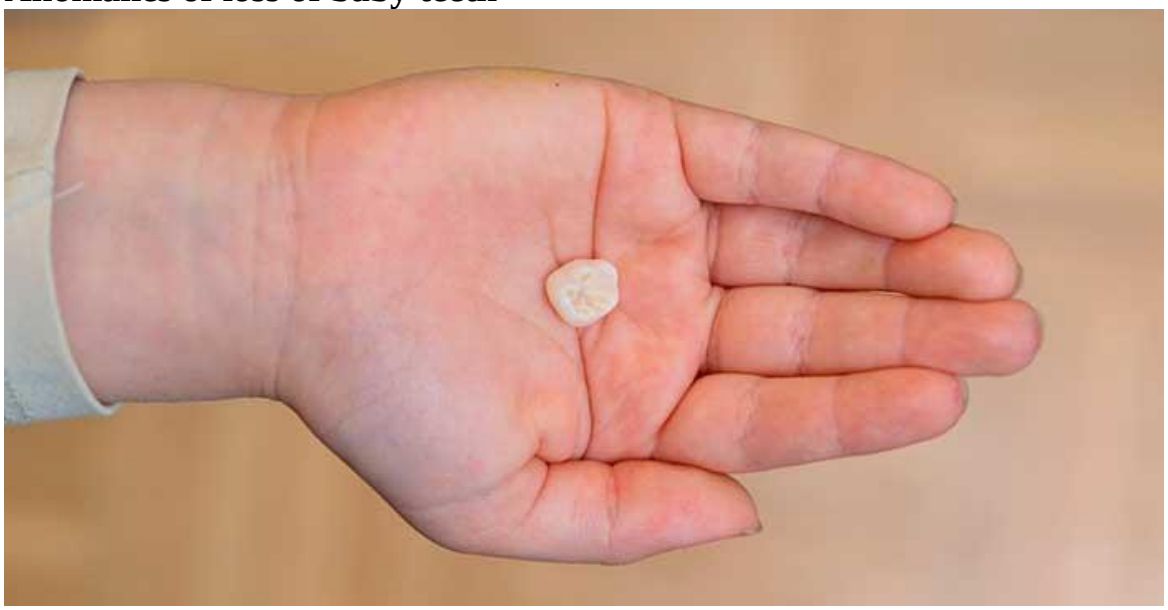
Congenital absence of teeth is a manifestation of numerous genetic syndromes. Some syndromes are associated with the absence of only a few teeth (for example, Down syndrome); other syndromes, such as ectodermal dysplasia, are associated with multiple toothlessness (hypodontia) or complete absence of teeth (anodontia).

Young children with suspected missing teeth or abnormal teething should be referred for a full dental examination. The diagnosis of congenital absence of teeth is confirmed by the absence of a complete dentition on dental radiographs. Genetic testing may then be suggested to determine if the molecular basis of the child's condition is known and to help establish the risk of recurrence, the risk of potentially related health problems. For example, mutations of the axis 2 inhibitor [AXIN2] are associated with missing teeth and the risk of colorectal cancer.

The treatment of missing teeth varies and may require several therapeutic phases. Treatment of hypodontia or anodontia involves the use of dentures or dental implants to replace missing teeth and improve oral function and facial aesthetics. Children who are missing several front teeth should get their dentures before they go to school. The optimal age for treatment is assessed individually and is determined by the amount of treatment needed and the child's ability to cooperate during the procedures and maintain the devices after installation. Dental implantation procedures are usually postponed until the child reaches late adolescence or early adulthood.

Acquired toothlessness can occur as a result of local or generalized environmental influences (for example, trauma to a baby tooth, radiation to the head and neck, chemotherapy).

Anomalies of loss of baby teeth

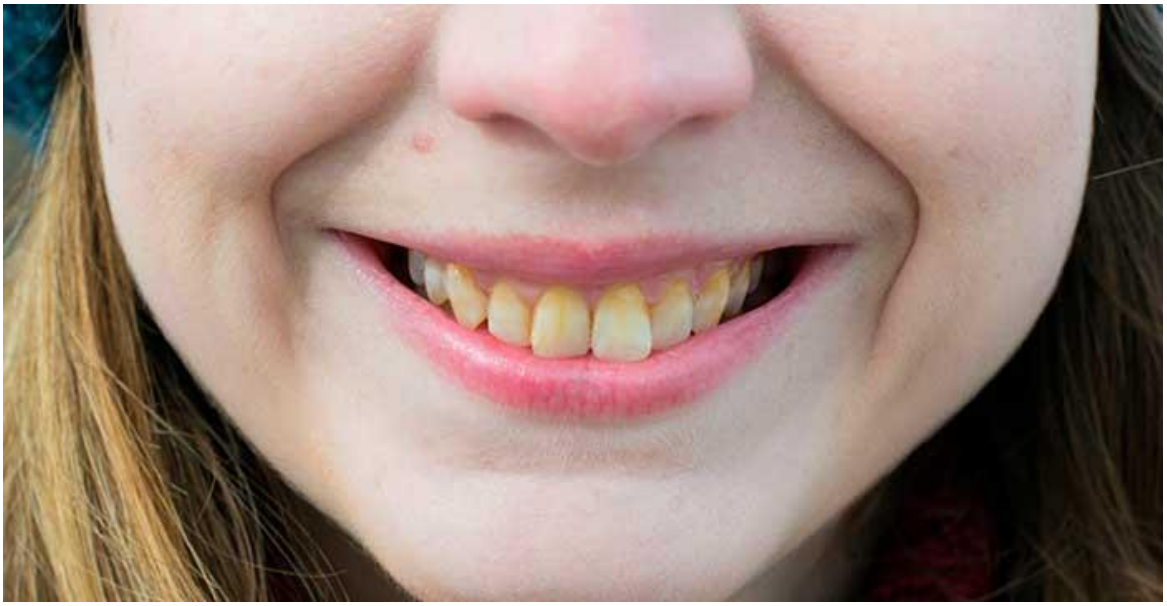


Premature loss of baby teeth may be caused by local factors or systemic health problems. The eruption of permanent teeth can cause the loss of adjacent baby teeth. For example, two milk incisors may fall out at the same time when one large permanent incisor begins to erupt. The loss of several baby teeth, "giving way" to one permanent one, often indicates a mismatch in the size of the dental arch and usually portends crowding in a permanent bite.

In addition, premature detachment of baby teeth may be associated with systemic conditions such as hypophosphatasia, Langerhans histiocytosis and cyclic neutropenia. The absence of root resorption in a fallen baby tooth is a constant clinical sign and an important key to the diagnosis of systemic conditions associated with premature loss of baby teeth.

Children with premature loss of baby teeth should be examined to rule out serious systemic diseases and should be referred to a pediatric dentist for the prompt initiation of appropriate dental therapy. The diagnosis of hypophosphatasia is crucial because enzyme replacement therapy is available.

Anomalies of tooth enamel



More than 100 known genetic causes of enamel defects in humans have been identified. There are also more than 100 known environmental conditions associated with enamel defects. Enamel formation is strictly regulated at the molecular level. Environmental impacts on enamel production that may affect the secretion or processing of the matrix include exposure to injury, infection, lead, mercury, and fluoride. Defects in tooth enamel are described in 25-80 percent of the general population.

Fluorosis. Fluoride is widely used for the prevention of caries, but excessive consumption of fluoride (more than 0.05 mg /kg per day) at an age associated with dental development can cause hypomineralization of tooth enamel or fluorosis. Hypomineralization makes teeth more susceptible to wear and breakage. Mild fluorosis is indicated by the appearance of white spots or lace on the enamel, and severe fluorosis is indicated by a change in color to brown. The mechanism by which excessive consumption of fluoride causes fluorosis is not fully understood and is probably complicated. Fluorosis can be prevented by using appropriate fluoride supplements and limiting excessive consumption of fluoride, including fluoride toothpaste.

Hypomineralization of incisor molars. The prevalence of this dental anomaly depends on the geographical factor. It is characterized by abnormal mineralization of the enamel of the first permanent molars, but it can also affect permanent incisors. The milk molars are affected in some cases. Enamel abnormalities increase the risk of caries and tooth decay. The severity of the disease varies greatly: from slightly discolored enamel in certain areas (usually yellow-brown) to the destruction of tooth enamel during eruption. This can cause extreme sensitivity to thermal and chemical stimuli. The etiology is not clear, but children who are more likely to suffer from childhood diseases have an increased prevalence, and there may be a genetic component.

Hereditary enamel abnormalities. Hereditary conditions that cause enamel abnormalities may be part of the syndrome or isolated from the enamel. Enamel abnormalities associated with syndromic conditions vary significantly depending on the molecular defect and the role of the gene in tooth formation.

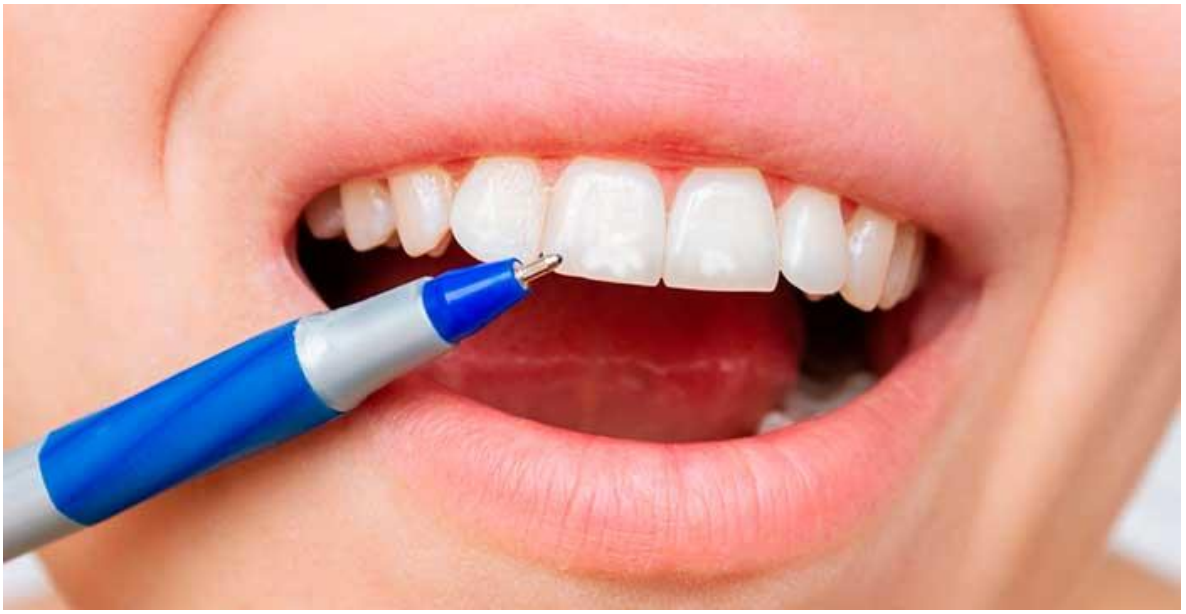
Borderline epidermolysis bullosa is an autosomal recessive disease characterized by variable severity of skin fragility and blistering, as well as generalized enamel hypoplasia of varying severity. The molecular defects that cause borderline epidermolysis bullosa are associated with genes that produce proteins necessary to maintain the integrity between the dermis and epidermis and important for the normal functioning of ameloblasts.

Amelogenesis imperfecta is a group of hereditary diseases with effects limited by tooth enamel, they include four clinically and genetically distinct types and 14 subtypes. Mutations in numerous genes (> 16) are currently associated with various types of imperfect amelogenesis.

Mutations in different genes and allelic mutations of the same gene lead to different enamel phenotypes, ranging from normal-colored hypoplastic enamel to brown hypomineralized enamel, which is erased from teeth when they erupt into the oral cavity. Affected teeth can be extremely sensitive to thermal and chemical stimuli.

The treatment of enamel abnormalities depends on the diagnosis and the specific phenotype. For example, hypoplastic enamel, which is well mineralized, can often be treated with bonding procedures to protect and improve the appearance of teeth. On the other hand, teeth with highly hypomineralized enamel are usually treated with restorations that cover the entire crown (for example, stainless steel or plastic crowns). Infants who have conditions that are associated with enamel abnormalities should be examined by a dentist to assess the need for early intervention before their first birthday.

Dental anomalies



Although environmental factors can influence the development of dentin, genetic factors have the main influence. Dentin malformations affecting the shape and function of teeth occur in syndromic and non-syndromic hereditary conditions.

Imperfect dentinogenesis and dentin dysplasia are classified according to clinical, radiological and histopathological signs.

Imperfect dentinogenesis is classified according to its association with or without other conditions:

• Type I – associated with osteogenesis imperfecta and type I collagen defects; characterized by reduced pulp chambers.

• Type II – not associated with osteogenesis imperfecta; characterized by reduced pulp chambers.

• Type III – often associated with the Brandywine tri-racial isolate (a genetically isolated tri-racial population from southern Maryland); characterized by large pulp chambers in young teeth. This form is allelic to type II DI and is actually a phenotypic variant of type II DI, rather than a separate unit.

In all types, teeth have a variable opalescent shade from blue-gray to yellow-brown, caused by abnormally colored dentin shining through translucent enamel.

Type II is an autosomal dominant disease and is caused by mutations in the dentin sialophosphoprotein (DSPP) gene. Interestingly, the dental phenotypes of type I and type II are very similar. This is not surprising, since type I is associated with type I collagen defects, and type II is associated with DSPP mutations, and type I collagen and DSPP interact during dentin development and mineralization.

Since defective dentin in children with this condition often cannot support the enamel, the enamel often breaks off from the teeth, making them susceptible to rapid wear and abrasion. Teeth affected by imperfect dentinogenesis can be erased to the gums if left untreated.

Treatment depends on the severity of the discoloration and the tendency to enamel loss. In children who do not have enamel fractures, discoloration of teeth can be treated with bonding; when severe enamel destruction occurs, complete coating of crowns is usually necessary.

Dentin dysplasia. There are two types of dentin dysplasia. Type I dentin dysplasia is inherited in an autosomal dominant type with a frequency of 1:100,000 people. Mutations in the SSUH2 and VPS4B genes are associated with type I dentin dysplasia. It is believed that the classical cascade histopathology of dentin is the result of a cyclic process of premature death of odontoblasts, recruitment of new odontoblasts, dentin deposition and death of odontoblasts. The crowns of the affected teeth look normal, but short blunted roots and pulp obliteration are visible on radiographs, which leads to teeth becoming mobile, being lost prematurely and prone to abscess formation. There are no known treatments, but dental longevity can be increased by minimizing occlusal forces and avoiding orthodontic treatment of malocclusion.

Type II dentin dysplasia is also inherited by autosomal dominant type. Mutations in the DSPP gene are associated with type II dentin dysplasia. Because they are caused by allelic mutations in the DSPP gene, the phenotype of dentin dysplasia type II and imperfect dentinogenesis type II is identical in temporary teeth. They have yellow-brown to blue-gray discoloration and pulp obliteration. The permanent dentition of children with type II dentin dysplasia is usually colored or has minimal discoloration, but has abnormal pulp morphology and may be associated with pulp stones.

The mechanism leading to the different phenotypes of permanent teeth in these two conditions is not completely clear. However, one study, which included a clinical and genetic assessment of 23 members of the genus from four generations, including 10 members with dentin defects, showed that dentin dysplasia type II and type II

imperfect dentinogenesis represent mild and severe forms, respectively, of the same disease.

Treatment of type II dentin dysplasia in baby teeth is the same as in case of imperfect dentinogenesis.

Systemic conditions may be associated with abnormal dentin formation as a result of molecular defects that interfere with the pathways of dentin development. For example, since dentin consists of 60 percent minerals, systemic conditions associated with mineralization defects (for example, hypophosphatasia and vitamin D-resistant rickets) affect the development of dentin. Other systemic conditions with dentin damage include Ehlers-Danlos syndrome, mucopolysaccharidoses, and tumor calcification.

Conclusions about dental anomalies

• In order to distinguish normal dental development from an anomaly, a thorough assessment of the patient is required, including a medical, dental and family history, clinical examination, X-ray evaluation, and possibly special laboratory tests.

• Teething abnormalities include congenital and neonatal teeth, premature teething and non-teething (retination).

• The most common cause of permanent teething abnormality is the lack of a place for teething. Lack of space often causes retention of the third molars, canines of the upper jaw and second premolars of the lower jaw. Insufficient space for eruption is treated by selective removal of permanent teeth or orthodontic therapy.

• Young children with teething abnormalities should be referred for a full dental examination. The congenital absence of teeth is confirmed by radiography of the jaws. Genetic testing can help establish the risk of recurrence and the risk of related health problems. Replacement of missing teeth is possible due to dentures or dental implants, this affects both the functions of the oral cavity and the aesthetics of the face.

Premature loss of baby teeth may be caused by local factors or systemic health problems. The absence of root resorption in a fallen baby tooth is an important sign that there may be some kind of systemic health problem. Children with premature loss of baby teeth should be examined to rule out these very systemic diseases.

• Enamel abnormalities include fluorosis, hypomineralization of incisor molars and hereditary conditions. Hereditary conditions that cause enamel defects may be part of a syndrome (trichodoossal localization syndrome, epidermolysis bullosa) or be isolated from enamel (for example, imperfect amelogenesis). The treatment of enamel abnormalities depends on the diagnosis.

• Dentin abnormalities are primarily genetic, they occur in syndromic and non-syndromic hereditary conditions.

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