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DEVELOPMENTAL DISORDERS OF THE TEETH

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МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
БЕЛОРУССКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ
1-я КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ

Л. А. Казеко, Е. Л. Колб

НАРУШЕНИЯ РАЗВИТИЯ ЗУБОВ

DEVELOPMENTAL DISORDERS OF THE TEETH

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для студентов учреждений высшего образования, обучающихся
на английском языке по специальности 1-79 01 07 «Стоматология»

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There are many acquired and inherited developmental abnormalities that alter the size, shape and number of teeth. Individually, they are rare but collectively they form a body of knowledge with which all dentists should be familiar.

Developmental defects in dental hard tissue result from the action of harmful agents or other factors that affect the normal course of tooth development stages, such as:

1) initiation and proliferation;
2) morphodifferentiation;
3) histodifferentiation;
4) mineralization and maturation;
5) tooth eruption.

In accordance with the disturbance of tooth development stage, there are various manifestations of teeth abnormalities dentists can see in the patient's mouth. Thus all congenital malformations of the teeth may be divided into the following groups:

– abnormalities in number and eruption (*defective initiation*);

– abnormalities in the size of the teeth (*defective morpho- and histodifferentiation*);

– abnormalities in the shape or form of the teeth (*defective morpho- and histodifferentiation*);

– abnormalities in the tooth structure (*defective calcification*).

ICD-10 CLASSIFICATION (WHO, 2014)

K00 Disorders of tooth development and eruption
Excl.: embedded and impacted teeth (K01.-)
K00.0 Anodontia
Hypodontia
Oligodontia
K00.1 Supernumerary teeth
Distomolar
Fourth molar
Mesiodens
Paramolar
Supplementary teeth
K00.2 Abnormalities of size and form of teeth
Concrescence of teeth
Fusion of teeth
Gemination of teeth
Dens: evaginatus
in dente
invaginatus
Enamel pearls
Macrodontia

Microdontia
Peg-shaped [conical] teeth
Taurodontism
Tuberculum paramolare

Excl.: tuberculum Carabelli, which is regarded as a normal variation and should not be coded

K00.3 Mottled teeth

Dental fluorosis
Mottling of enamel
Nonfluoride enamel opacities

Excl.: deposits [accretions] on teeth (K03.6)

K00.4 Disturbances in tooth formation

Aplasia and hypoplasia of cementum
Dilaceration of tooth
Enamel hypoplasia (neonatal) (postnatal) (prenatal)
Regional odontodysplasia
Turner tooth

Excl.: Hutchinson teeth and mulberry molars in congenital syphilis (A50.5)
mottled teeth (K00.3)

K00.5 Hereditary disturbances in tooth structure, not elsewhere classified

Amelogenesis imperfecta
Dentinogenesis imperfecta
Odontogenesis imperfecta
Dentinal dysplasia
Shell teeth

DISORDERS OF TOOTH DEVELOPMENT AND ERUPTION

Anodontia — congenital absence of the teeth; it may involve all (total anodontia) or only some of the teeth (partial anodontia, hypodontia), and both the deciduous and the permanent dentition, or only teeth of the permanent dentition (fig. 1).

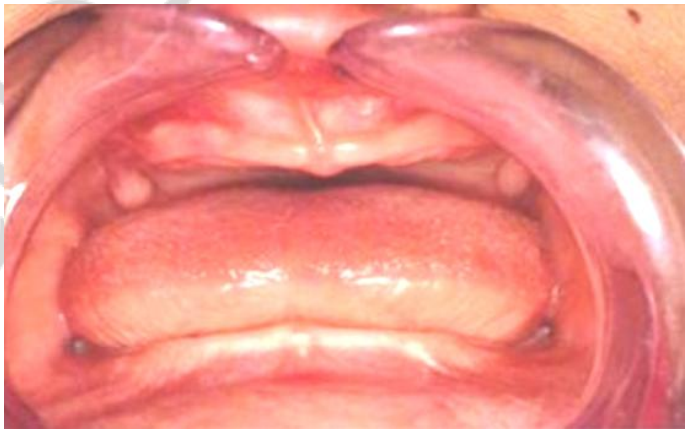


Fig. 1. Total anodontia of permanent teeth as a result of agenesis of teeth; the patient suffered from an ectodermal dysplasia syndrome

Hypodontia (**Oligodontia**) — a condition of having fewer than normal complement of teeth, either congenital or acquired, lack of development of one or more teeth (fig. 2). The affected teeth are usually the third molars and the maxillary lateral incisors. Oligodontia refers to the agenesis of numerous teeth. Anodontia or hypodontia is often associated with a syndrome known as *ectodermal dysplasia*.

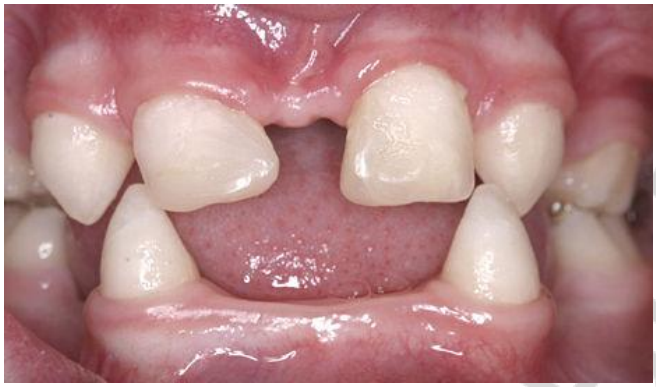


Fig. 2. Hypodontia, absence of the lower incisors

Pseudoanodontia is the clinical presentation of having no teeth when teeth have either been removed or obscured from view by hyperplastic gingiva.

SUPERNUMERARY TEETH

Supernumerary teeth (hyperdontia) are additional number of teeth, over and above the usual number for the dentition. Supernumerary teeth occur as isolated events but are also found in *Gardner's syndrome, cleidocranial dysostosis syndrome*, and in cases of *cleft palate (or cleft lip)*.

Supernumerary teeth that occur in the molar area are called «paramolar teeth»; and, more specifically, those that erupt distally to the third molar are called «distodens» or «distomolar» teeth (fig. 3). Also, a supernumerary tooth that erupts ectopically either buccally or lingually to the normal arch is sometimes referred to as «peridens».

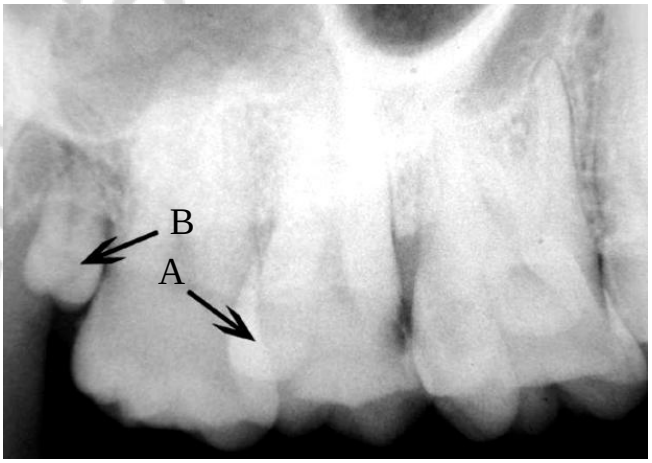


Fig. 3. A — paramolar is a supernumerary tooth in the molar region; B — distodens or distomolar is a supernumerary tooth that is distal to the third molar

The order of frequency of supernumerary teeth is: the mesiodens, maxillary distomolar (4th molar), maxillary paramolar (buccal to first molar), mandibular premolar, and maxillary lateral incisors.

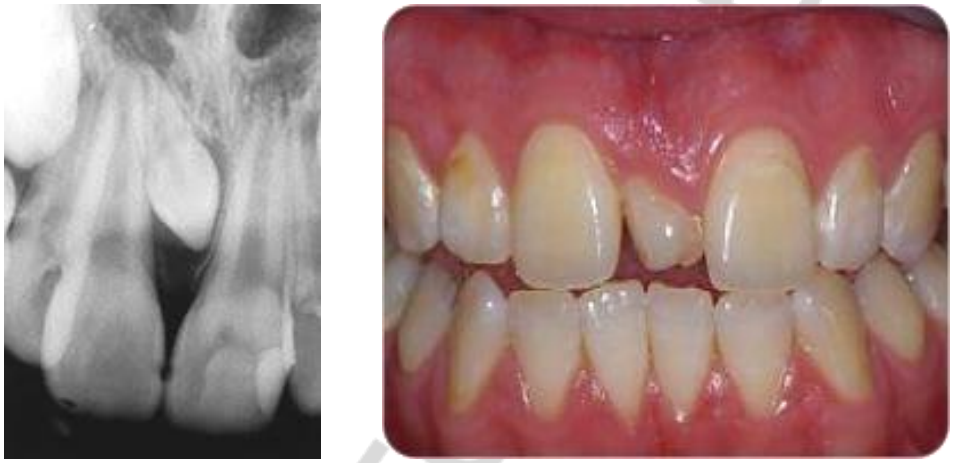
Some clinicians classify additional teeth according to their morphology:

1) supernumerary teeth;

2) supplemental teeth.

Supernumerary teeth are small, malformed extra teeth, for example mesiodens, distomolar and paramolar. Supplemental teeth are extra teeth of normal morphology, for example extra premolars and lateral incisors.

Mesiodens (plural — mesiodentes) is a supernumerary tooth that occurs in the anterior maxilla in the midline region near the maxillary central incisors (fig. 4). There may be one or more mesiodentes. The tooth crown may be cone-shaped with a short root or may resemble the adjacent teeth. It may be erupted or impacted, and occasionally inverted. Mesiodens is the most common supernumerary tooth.



A B

Fig. 4. A — an unerupted cone-shaped mesiodens; B — an erupted mesiodens between the two maxillary central incisors

ALTERATION IN SIZE OF THE TEETH

Macrodontia (megadontia) refers to teeth that are larger than normal (fig. 5). The disorder may affect a single tooth or may be generalized to all teeth as in pituitary *gigantism*. In a condition known as *hemifacial hypertrophy*, teeth on the affected side are abnormally large compared to the unaffected side.

Microdontia refers to teeth that are smaller than normal (fig. 6). Localized microdontia often involves the maxillary lateral incisors or maxillary third molars. The shape of the tooth may be altered as in the case of maxillary lateral incisors which appear as cone-shaped or peg-shaped; hence the term «peg laterals» (fig. 7). Generalized microdontia may occur in a condition known as *pituitary dwarfism*.

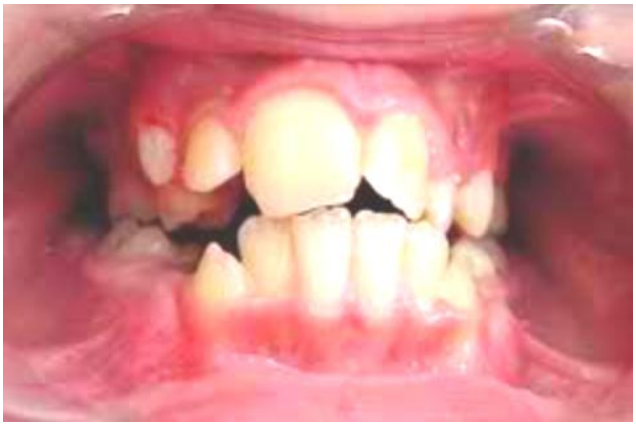


Fig. 5. Macrodont (megadont) right central incisor

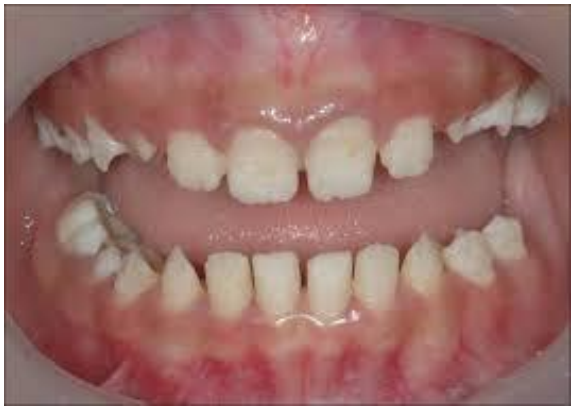


Fig. 6. General microdontia in the patient suffering from pituitary dwarfism

Fig. 7. Maxillary lateral peg-shaped incisor

ALTERATION IN SHAPE OF TEETH

Fusion (Synodontia). Fusion is a developmental union of two or more adjacent tooth germs (fig. 8). Although the exact cause is unknown, it could result from contact of two closely positioned tooth germs which fuse to varying degrees before calcification, or from a physical force causing contact of adjacent tooth buds. The union between the teeth results in an abnormally large tooth, or union of the crowns, or union of the roots only, and may involve the dentin. The root canals may be separate or fused. Clinically, a fusion results in one less tooth in the dental arch unless the fusion occurred with a supernumerary tooth. The involvement of a supernumerary tooth makes it impossible to differentiate fusion from gemination.

Gemination is the incomplete attempt of a tooth germ to divide into two (fig. 9). The resultant tooth has two crowns, or a large crown partially separated, and sharing a single root and root canal. The pulp chamber may be partially divided or may be single and large. The etiology of this condition is unknown. Gemination results in one more tooth in the dental arch. It is not always possible to differentiate between gemination and a case in which there has been fusion between a normal tooth and a supernumerary tooth.

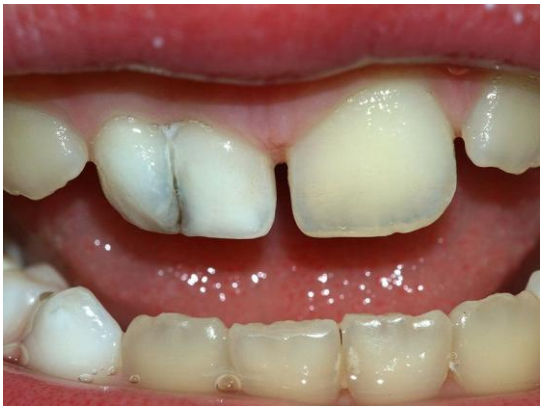


Fig. 8. Fusion of the upper lateral and central deciduous incisors

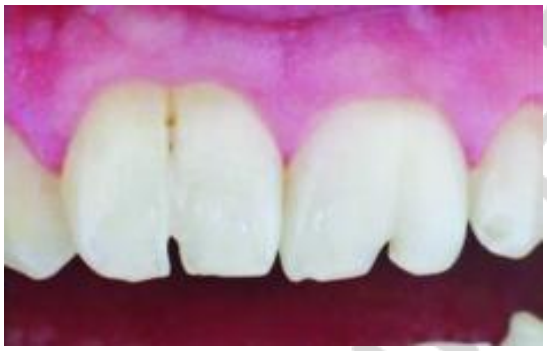


Fig. 9. Gemination of the maxillary central incisors

Concrescence is a form of fusion occurring after root formation has been completed, resulting in teeth united by their cementum. The involved teeth may erupt partially or may completely fail to erupt. Concrescence is most commonly seen in association with the maxillary second and third molars. It can also occur with a supernumerary tooth. On a radiograph, concrescence may be difficult to distinguish from superimposed images of closely positioned teeth unless additional radiographs are taken with changes in x-ray beam angulation. This condition is of no significance, unless one of the involved teeth requires extraction.

Fig. 10. Concrescence of the maxillary second and third molars

Dens in dente (Dens invaginatus, Dilated composite odontome). Dens in dente, also known as dens invaginatus, is produced by an invagination of the calcified layers of a tooth into the body of the tooth. The invagination may be shallow and confined to the crown of the tooth or it may extend all the way to the apex. Therefore, it is sometimes called a tooth within a tooth.

The most commonly used classification of dens invaginatus proposed by Oehlers (1957) is shown in fig. 11. He described the anomaly occurring in three forms:

Type I (a): an enamel-lined minor form occurring within the confines of the crown not extending beyond the amelocemental junction.

Type II (b): an enamel-lined form which invades the rootbut remains confined as a blind sac. It may or may not communicate with the dental pulp.

Type III (c, d): a form which penetrates through the root perforating at the apical area showing a 'second foramen' in the apical or in the periodontal area. There is no immediate communication with the pulp. The invagination may be completely lined by enamel, but frequently cementum will be found lining the invagination.

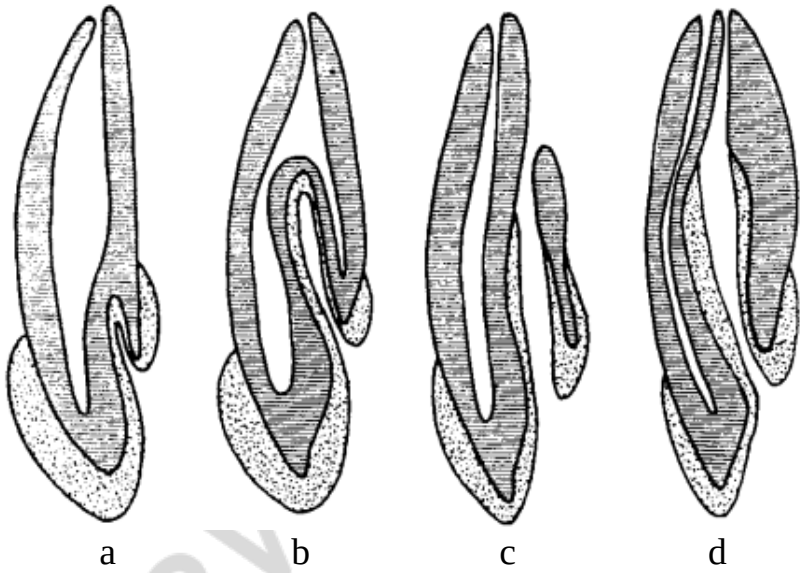


Fig. 11. Classification of invaginated teeth by Oehlers (1957)

In the crown, the invagination often forms an enamel lined cavity projecting into the pulp. The cavity is usually connected to the outside of the tooth through a very narrow constriction which normally opens at the cingulum area. Consequently, the cavity offers conditions favorable for the development and spread of dental caries. The infection can spread to the pulp and later result in periapical infection. Therefore, these openings should be prophylactically restored as soon as possible after eruption. The maxillary lateral incisor is the most frequently affected tooth (fig. 12). Bilateral and symmetric cases are occasionally seen. Dens in dente can also occur in the root portion of a tooth from the invagination of Hertwig's epithelial root sheath. This anomaly is discovered incidentally on radiographic examination.

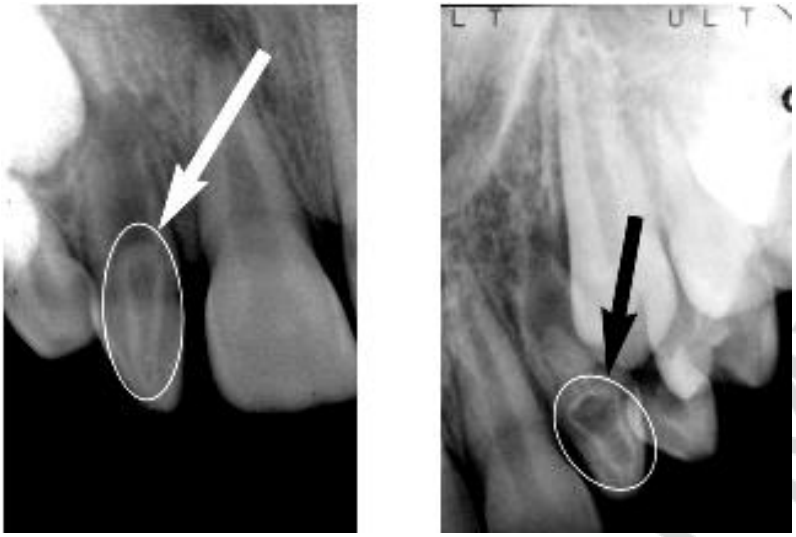


Fig. 12. Dens invaginatus, maxillary lateral incisors

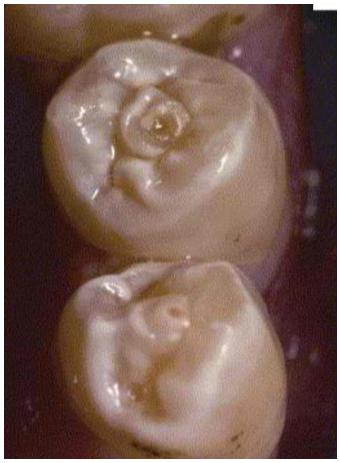


Fig. 13. Dens evaginatus

Dens evaginatus is a developmental condition affecting predominantly premolar teeth. It exclusively occurs in individuals of the Mongoloid race (Asians, Eskimos, Native Americans). The anomalous tubercle or cusp is located in the center of the occlusal surface (fig. 13). The tubercle wears off relatively quickly causing early exposure of the accessory pulp horn that extends into the tubercle. This may result in periapical pathology.

The talon cusp is an accessory cusp located on the lingual surface of maxillary or mandibular teeth (fig. 14). Any tooth may be affected but usually it is a maxillary central or lateral incisor. The cusp arises in the cingulum area and may produce occlusal disharmony. In combination with the normal incisal edge, the talon cusp forms a pattern resembling an eagle's talon.

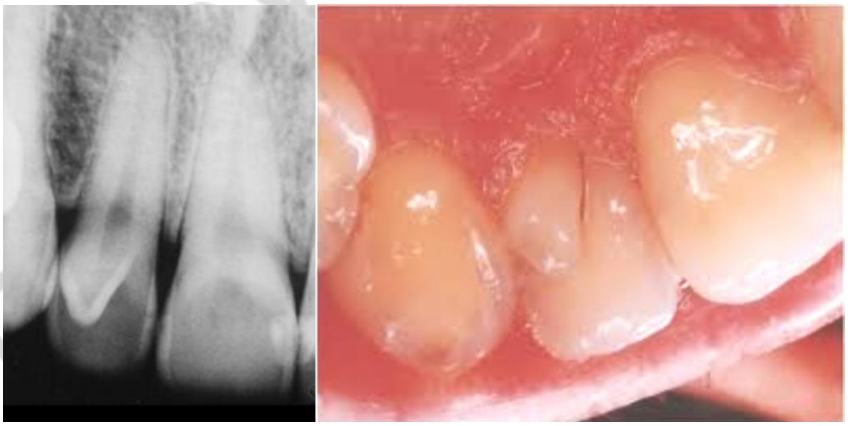


Fig. 14. Talon cusp

Taurodontism. Taurodont teeth have crowns of normal size and shape but have large rectangular bodies and pulp chambers which are dramatically increased

in their apico-occlusal heights. The apically displaced furcations result in extremely short roots and pulp canals. This developmental anomaly almost always involves a molar tooth (fig. 15). In an individual, single or multiple teeth may be affected either unilaterally or bilaterally. Taurodontism is reported to be prevalent in Eskimos and in Middle Eastern populations. The condition has sometimes been seen in association with *amelogenesis imperfecta*, *tricho-dento-osseous syndrome*, and *Klinefelter's syndrome*. This anomaly is not recognizable clinically but on a radiograph, the rectangular pulp chamber is seen in an elongated tooth body with shortened roots and root canals.

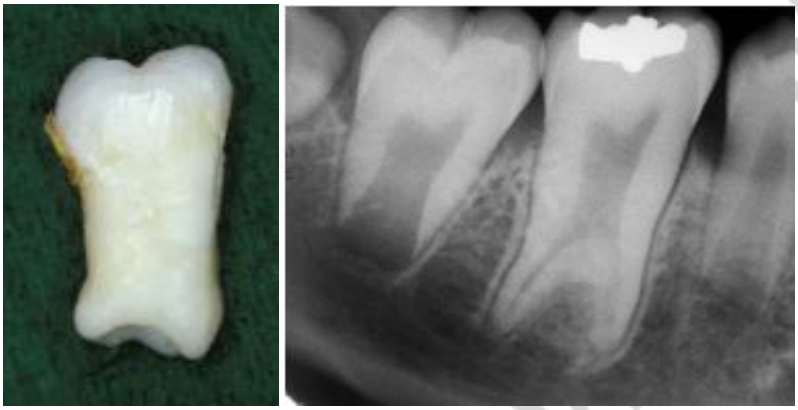


Fig. 15. Taurodont

Enamel pearl (Enameloma). Enamel pearl, also known as enameloma, is an ectopic mass of enamel which can occur anywhere on the roots of teeth but is usually found at the furcation area of roots (fig. 16). The maxillary molars are more frequently affected than the mandibular ones. An enamel pearl does not produce any symptom, and when explored with a dental explorer it may be mistaken for calculus. On a radiograph, the enamel pearl appears as a well-defined round radiopacity.

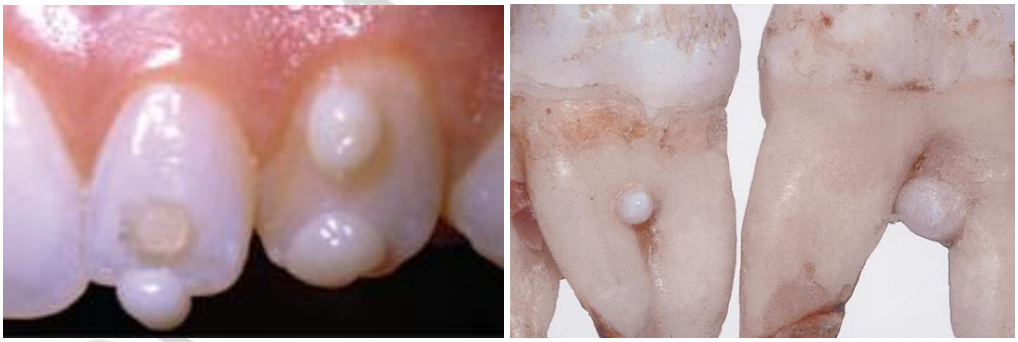


Fig. 16. Enamel pearls

MOTTLED TEETH

Dental fluorosis, a specific disturbance in tooth formation and an esthetic condition, is defined as a chronic fluoride-induced condition in which enamel development is disrupted and the enamel is hypomineralised. Simply put, dental fluorosis is a condition in which an excess of fluoride is incorporated in

the developing tooth enamel. The severity of dental fluorosis depends on when and for how long the over exposure to fluoride occurs, the individual response, weight, degree of physical activity, nutritional factors and bone growth. However, the most important risk factor for fluorosis is the total amount of fluoride consumed from all sources during the critical period of tooth development.

Points to remember:

1. Fluorides are toxic for all hard tissue forming cells.
2. Fluoride combines to form calcium fluoroapatite instead of calcium hydroxyapatite.
3. The disease is endemic in areas where the concentration of fluorides in the communal water supply is 1.5 ppm or more (Moldova, Ukraine, Central Asian countries).
4. There is individual variation in the effect of fluorides, not all individuals of the same community show the same degree of mottling, this may be related to some dietary and drinking habits.
5. The severity of the lesions increases with the increase in the fluoride levels.
6. At intermediate levels (2–6 ppm) the matrix is normal in quantity and structure and the defect is mainly in calcification.
7. At higher levels (more than 6 ppm) defective matrix formation starts (the enamel is pitted).
8. Deciduous teeth are rarely affected because excess fluorides are taken up by the maternal skeleton.
9. At higher levels (more than 8 ppm) deciduous teeth begin to be affected; sever mottling sclerosis of the skeleton may develop.
10. At exceptionally very high levels, rickets and osteoporosis may develop.
11. About 25 countries all over the world are affected by fluorosis.

Sources of fluorosis are:

1) hydrofluorosis (water-born);
2) drug-induced;
3) food-born;
4) industrial dust & fumes of fluoride.

Food items rich in fluoride: rock salts, canned fish and meat, packed food items like fruit juice, black tea (tea without milk), tea with lemon, green tea.

Clinically, mild enamel fluorosis is seen as diffuse white spots or white opaque lines or striations or a white parchment-like appearance of the tooth surface that run horizontally across the enamel. These may be invisible to the individuals and clinicians but often can be seen after the enamel has been dried. The opacities may coalesce to form white patches. In the moderate or more severe forms, the enamel may become discolored and/or pitted due to uptake of extrinsic stains mainly from the diet. At high concentrations of fluoride, discrete or confluent pitting of the enamel surface is seen, accompanied by extrinsic stain.

Fluorosis is symmetrically distributed, but the severity varies among the different types of teeth. Teeth that develop and mineralize later in life such as premolars, have a higher prevalence of fluorosis, and are more severely affected. The primary dentition and lower incisors are affected rarely.

It is necessary to measure dental fluorosis for surveillance purposes, research purposes and for treatment decisions. The instrument employed in dental fluorosis measurement are indices and imaging techniques.

Dean‘s Index (DI) (1934)

It was first described in 1934 and was later modified in 1942. The index was developed to gain an understanding of the relationship between fluoride concentrations in drinking waters and mottled enamel. It was designed to reflect the clinically visible features of dental fluorosis in a population and approximate the actual biologic effects of fluoride on developing dental enamel. It emphasizes the esthetic aspect of dental fluorosis. It became the most universally acceptable classification system for dental fluorosis found on two or more teeth. If two teeth are not equally affected, the less affected will be scored. This index categorizes dental fluorosis on a six point ordinal scale as ***normal, questionable, very mild, mild, moderate, and severe,*** as shown below.

Normal: the enamel represents the usual transluscent semi-vitriform type of structure, surface is smooth, glassy, pale, creamy white transluscent.

Questionable: the enamel discloses slight aberrations from the translucency of normal enamel ranging from a few white flecks or occasional white spots.

Very mild: small opaque paper white area scattered irregularly over the tooth covering less than 25 % of tooth surface. Bicuspids/second molars not showing more than 1–2 mm of white opacity at the tip of cusps summit are also frequently involved in this classification.

Mild: opaque white area in the enamel of the tooth covering less than 50 % of the tooth surface (fig. 17).

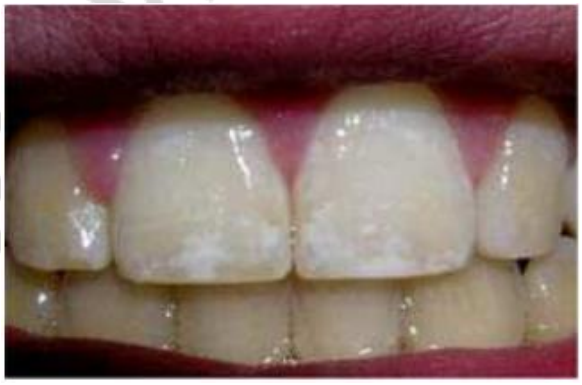


Fig. 17. Mild fluorosis

Moderate: all enamel tooth surfaces are affected, and surfaces subject to attrition show marked wear. Brown stain may be present (fig. 18).

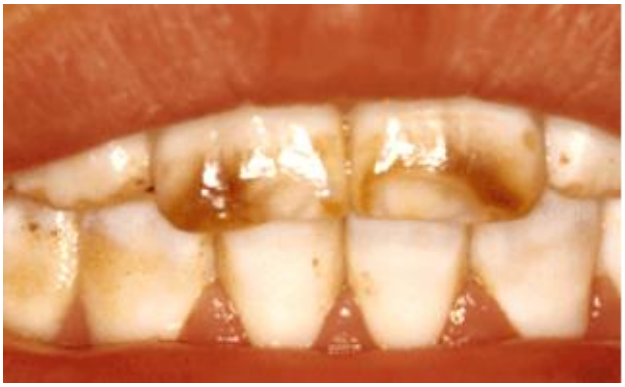


Fig. 18. Moderate fluorosis

Severe: all enamel surfaces are affected and hypoplastic brown stains are widespread and teeth often present as corroded appearance. The major diagnostic sign of this classification is the discrete or confluent pitting (fig. 19).

Fig. 19. Severe fluorosis

Dean's index has remained popular because of its simplicity.

The Thylstrup and Fejerskov Index (TFI)

This TFI was proposed by Thylstrup and Fejerskov (1978) with the aim of overcoming the shortcomings of the Dean's index. Like the Dean's index, TFI is a tooth based scoring system that produces a maximum of 28 scores per subject. It is a 10 point classification scale with numeric values from 0–9. This original index (with 10 categories involving description of all tooth surfaces) of fluorosis attempts to correlate clinical appearance with pathological changes in tissue. Therefore, it is a useful tool when evaluating dental fluorosis severity in epidemiological studies. The index was later modified to be based solely on examination of facial tooth surfaces

The TFI Score Criteria

0 — Normal translucency of enamel remains after wiping and drying of the surface.

1 — Narrow opaque/white lines running across the tooth surface, slight snow capping of cusps or incisal edges may also be seen.

2 — *Smooth surfaces*: more pronounced lines of opacity that follow the perikymata; occassional confluence of adjacent lines; *occlusal surfaces*: scattered areas of opacity less than 2 mm in diameter and pronounced opacity of cuspal ridges; snow-capping is common.

3 — *Smooth surfaces*: merging and irregular cloudy areas of opacity; accentuated drawing of perikymata is often visible between opacities; *occlusal surfaces*: confluent areas of marked opacity; worn areas appear almost normal but usually circumscribed by a rim of opaque enamel.

4 — *Smooth surfaces*: the entire surface exhibits marked opacity, or appears chalky white; parts of the surface exposed to attrition appear less affected; o*cclusal surfaces*: the entire surface exhibits marked opacity; attrition is often pronounced shortly after eruption.

5 — *Smooth surfaces and occlusal surfaces*: the entire surface displays marked opacity; focal loss of the outermost enamel (pits) is less than 2 mm in diameter.

6 — *Smooth surfaces*: pits are regularly arranged in horizontal bands less than 2 mm in vertical height; o*cclusal surfaces*: confluent areas less than 2 mm in diameter exhibit loss of the enamel marked attrition.

7 — *Smooth surfaces*: loss of the outermost enamel in irregular areas involving less than half of the entire surface; *occlusal surfaces*: changes in the morphology caused by merging pits and marked attrition.

8 — *Smooth and occlusal surfaces*: loss of the outermost enamel involving more than half of the surface.

9 — *Smooth and occlusal surfaces*: loss of the main part of the enamel with change in anatomic appearance of the surface.

Cervical rim of almost unaffected enamel is often noted.

The sensitivity of TFI comes from its 9 stages reflecting the histopathology and fluoride content in the enamel. It is sensitive, easy to understand, reliable and the most outstanding for evaluating the severity of fluorosis. TFI had an excellent reproducibility despite its extended scale, was suitable to categorize mild forms of dental fluorosis with ease, due to drying of teeth before scoring and had a clear description and discrimination of the categories in the lower end of the index, whereas DI lacked accuracy to discriminate within the low fluoride scores. TFI also facilitated discrimination of severe cases of dental fluorosis that were categorized in one score by DI.

The WHO classification developed by the Danish scientist Møller is used for endemic research in foreign countries as well as in Belarus (table 1). According to this classification there are five degrees of fluorosis severity. Determination of the degree of disease is based not only on the fluorosis elements morphology (stripes, spots, defects, pits of enamel), but also on the areas of enamel destructions.

Table 1

Classification of dental fluorosis, Møller

Code	The severity of the disease	Clinical manifestations
I	**Very mild**	Strips or white patches, slightly different from the normal color of enamel. In case of doubt teeth are considered to be healthy
II	**Mild**	Clearly defined stripes and white spots, which occupy less than a quarter of the surface of the tooth crown
III	**Moderate**	Strips and white patches, covering not more than half of the surface of the tooth crown
IV	**Moderate severity**	There are mostly brown strips and spots on the surface of the teeth
V	**Severe**	On a brown background are identified foci of enamel destruction in the form of pits, erosion, splits etc.

Summarizing the clinical manifestations, we can say that dental fluorosis is basically a deviation from the normal color of the enamel in the form of a milky white or brown color clouding. This may lead to esthetic discomfort; the patients complain of having brown teeth. In severe cases, when the degradation of the tooth surface in the form of dimples, grooves, splits and increased abrasion of the teeth are seen, the patients may complain not only of teeth discoloration but also of the teeth destruction.

Nowadays, the **differential diagnosis** between fluorosis and non-fluoride-induced opacities needs to establish differences between symmetrical and asymmetrical opaque defects. These criteria imply that all symmetrically distributed opaque conditions of enamel are fluorosis. Diagnostic difficulties occur mostly with mild forms of fluorosis, or when a mix of fluorotic and non-fluorotic conditions is evident. Diagnosis of fluorosis is flawlessly assisted by three additional features:

1) systemic failure (all teeth have white and/or brown spots on its surfaces);
2) low intensity of caries;
3) the patient was or is in an area of high fluoride content in drinking water.

Mild fluorosis is also similar to an initial caries. The difference is that the fluorosis white spots are mainly localized closer to the cutting edge and occlusal surface of the tooth, while carious white spots are mainly localized in the cervical area. White spots on the fluorosis enamel are not stained and remain shiny after drying.

Controlling the fluoride intake is the best preventive measure for dental fluorosis, however, when this is already installed and causing esthetic problems to the patient, some treatment techniques are described in the literature and will depend on the severity of the condition. Bleaching and enamel microabrasion techniques are conservative, and provide highly satisfactory results, without excessive wear of sound dentin. Composite resin and resin-modified glass ionomer are also used for treating discolored areas. Composite restorations can be associated either with microabrasion or with esthetic veneers; the use of prosthetic crowns might be needed.

DISTURBANCES IN TOOTH FORMATION

Enamel hypoplasia is a defect that occurs as a result of a disturbance in the formation of the organic enamel matrix. Enamel hypoplasia is a defect in tooth enamel that results in a smaller quantity of enamel than normal. The defect can be a small pit or dent in the tooth or can be so widespread that the entire tooth is small and/or misshaped. This type of defect may cause tooth sensitivity, may be unsightly or may be more susceptible to dental cavities. Some genetic disorders cause all the teeth to have enamel hypoplasia. EH can occur on any tooth or on multiple teeth. It can appear white, yellow or brownish in color with a rough or pitted surface. In some cases, the quality of the enamel is affected as well as the quantity.

Types and etiology of the enamel hypoplasia:

1. *Hereditary*: enamel is partly or wholly missing. An example is *amelogenesis imperfecta*.

2. *Systemic (environmental).* Factors that may contribute to enamel hypoplasia during tooth development include severe nutritional deficiency, particularly rickets; fever-producing diseases, such as measles, chickenpox, and scarlet fever; congenital syphilis; hypoparathyroidism; birth injury; prematurity; Rh hemolytic disease; fluorosis; or idiopathy.

3. *Local*: a single tooth can be affected; trauma or periapical inflammation above a primary tooth can injure the adjacent developing permanent tooth *(Turner tooth)* (fig. 20).

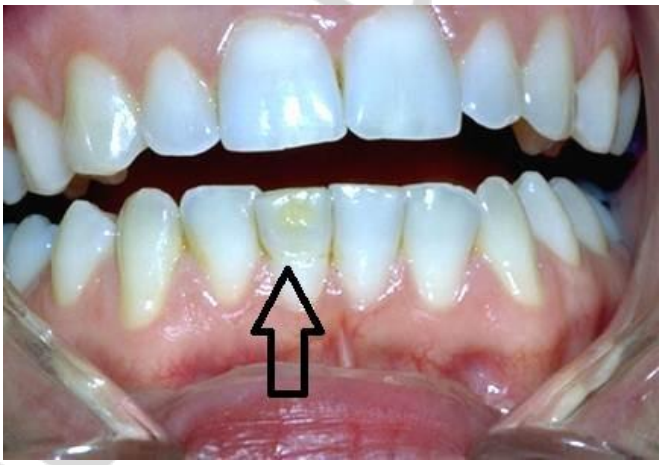


Fig. 20. Turner tooth

Systemic hypoplasia is also called *«chronologic hypoplasia»* because the lesions are found in areas of those teeth where the enamel was formed during the systemic disturbance.

a) single narrow zone (smooth or pitted): disturbance lasted a short period of time (fig. 21);

b) multiple: disturbance to the ameloblast occurred over a period of time, or several times.

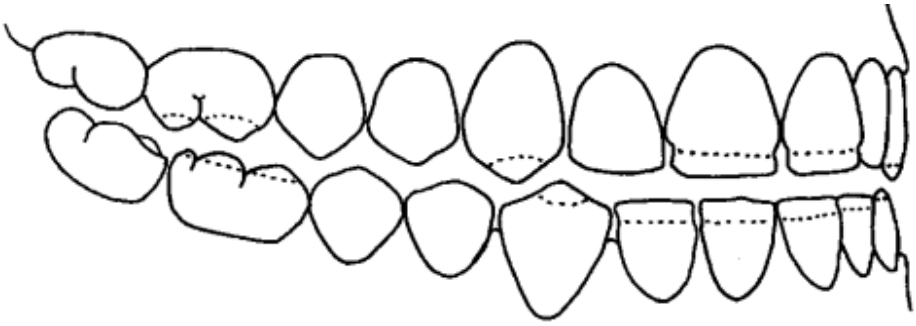


Fig. 21. Enamel Hypoplasia. Chronologic hypoplasia, usually in the form of grooves or pits, appears in the enamel at a level corresponding to the stage of the teeth development. For this patient, the disturbance in enamel development occurred at approximately 10 months of age

Teeth most frequently affected: first molars, incisors, canines, because disturbances generally occur during the first year when those teeth are mineralizing (fig. 22).

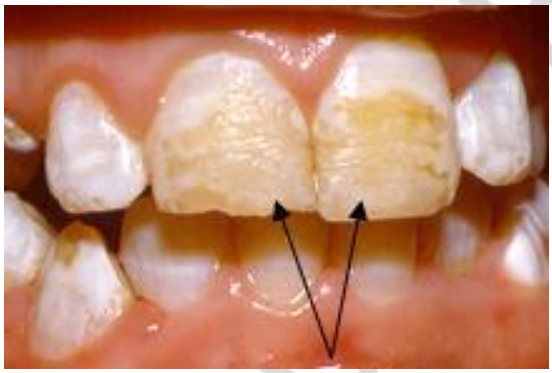


Fig. 22. Enamel hypoplasia

Developmental defects of enamel index (DDE Index)

This was proposed in 1982 by Commission on Oral Health, Research and Epidemiology arising from a lack of a well-defined and internationally accepted classification of enamel defects (table 2). The index was designed to promote the use of standard terminology, simplicity and to provide an effective system for recording enamel defects in large studies. The first version was, however, complicated and difficult to analyze, thus, Clarkson and O'Mullane suggested a modified and simplified version of the index that has now been widely adopted.

Table 2

Modified DDE Index for use in screening studies (Clarkson and O'Mullane, 1989)

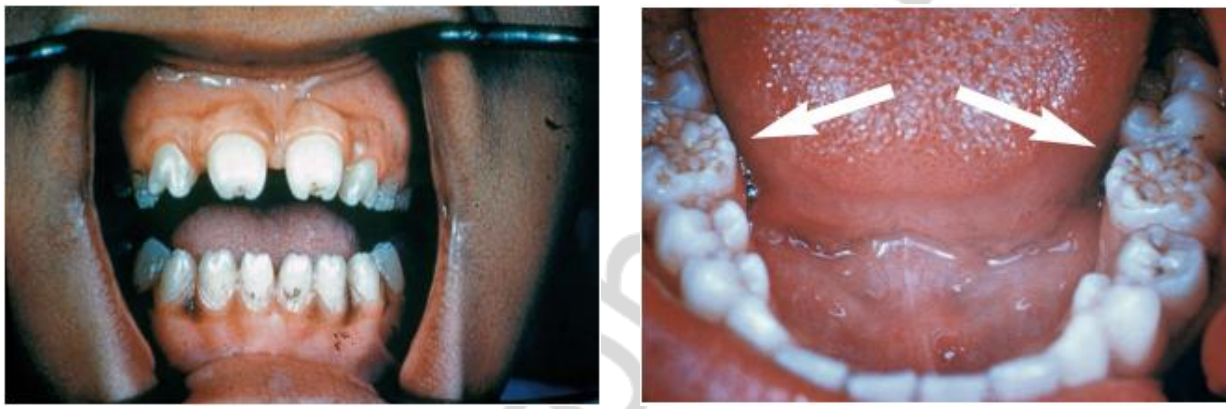
Type of Defect	Code
Normal	0
Demarcated Opacities	1
Diffuse Opacities	2
Hypoplasia	3
Other defects	4
Demarcated & diffuse opacities combined	5
Demarcated opacities & hypoplasia	6
Diffuse opacities & hypoplasia	7
Demarcated & diffuse opacities plus hypoplasia	8

DDE is now the most currently and widely used index to study enamel defects.

Treatment options depend on the severity of the enamel hypoplasia on a particular tooth and the symptoms associated with it. The most conservative treatment consists in bonding a tooth colored material to the tooth to protect it from further wear or sensitivity. In some cases, the nature of the enamel prevents the formation of an acceptable bond. Less conservative treatment options, but often necessary, include the use of stainless steel crowns, permanent cast crowns, or extraction of affected teeth and replacement with a bridge or implant.

Hypoplasia of congenital syphilis

Transmission of syphilis from mother to fetus after the 16th week of pregnancy may alter the development of the tooth germs. The mesiodistal width may be reduced, and incisors are frequently narrowed at the incisal third. «Screwdriver» incisors *(Hutchinson's incisors)* and «Mulberry molars», dental defects seen in congenital syphilis and caused by direct invasion of tooth germs by Treponema pallidum organisms (fig. 23).



A B

Fig. 23. A — «screwdriver teeth»; B — «mulberry molars»

Dilaceration of the tooth. Dilaceration is an abnormal bend in the root of a tooth (fig. 24). Though the exact cause is not known, it is believed to arise as a result of trauma to a developing tooth which alters the angle between the tooth germ and the portion of the tooth already developed. Dilaceration of roots may produce difficulties during extraction or root canal therapy.

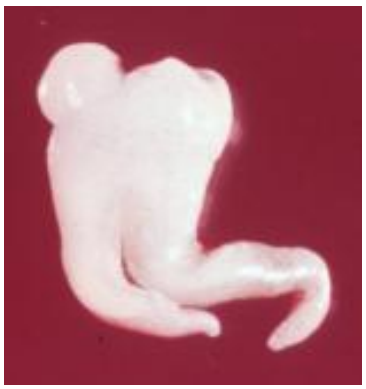


Fig. 24. Dilaceration of the tooth

Regional odontodysplasia is an unusual, non-hereditary anomaly of the dental hard tissues with characteristic clinical, radiographic and histological findings. Clinically, regional odontodysplasia affects the primary and permanent dentition in the maxilla and mandible or both jaws. Radiographically, there is a lack of contrast between the enamel dentin, both of which are less radiopaque than unaffected counterparts. Additionally, enamel and dentin layers are thin, giving the teeth a «ghost-like» appearance (fig. 25). Histologically, areas of hypocalcified enamel are visible and enamel prisms appear irregular in direction. Coronal dentin is fibrous, consisting of clefts and a reduced number of dentinal tubules; radicular dentin is generally more normal in structure and calcification. The regional odontodysplasia etiology is uncertain; numerous factors have been suggested and considered as local trauma, irradiation, hypophosphatasia, hypocalcemia, hyperpyrexia.

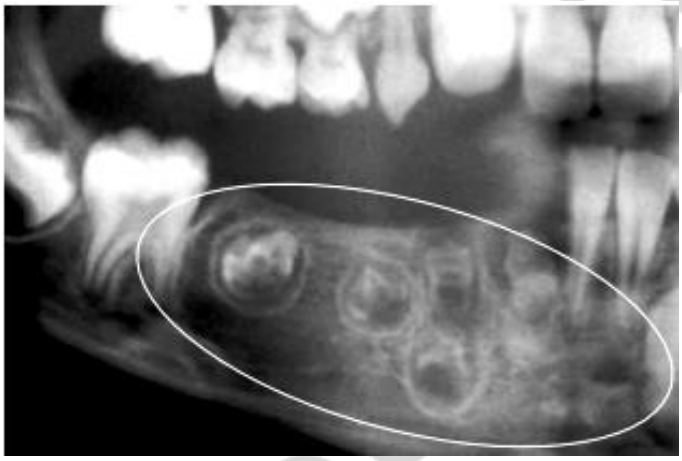


Fig. 25. Regional odontodysplasia

HEREDITARY DISTURBANCES IN TOOTH STRUCTURE

Amelogenesis imperfect. There is a large group of inherited developmental defects in enamel collectively referred to as *amelogenesis imperfecta*. At least 14 phenotypes have been identified and autosomal dominant, recessive and X linked inheritance have been reported. The condition is rare, only about 1 in 14,000 have it. Establishing a pattern of inheritance requires constructing a pedigree (family tree) of several generations, identifying those with and without the condition.

Three general categories of AI are recognized:

1) *Hypoplastic type*: inadequate formation of enamel matrix, both pitting and smooth types exist. Enamel may be reduced in quantity but is of normal hardness (fig. 26);

2) *Hypomaturation type*: a defect in the crystal structure of enamel leads to a mottled enamel with white to brown to yellow colors (fig. 27);

3) *Hypocalcified type*: a defect not in the quantity but in the quality of enamel. It is soft and poorly mineralized; it chips and wears easily (fig. 28).

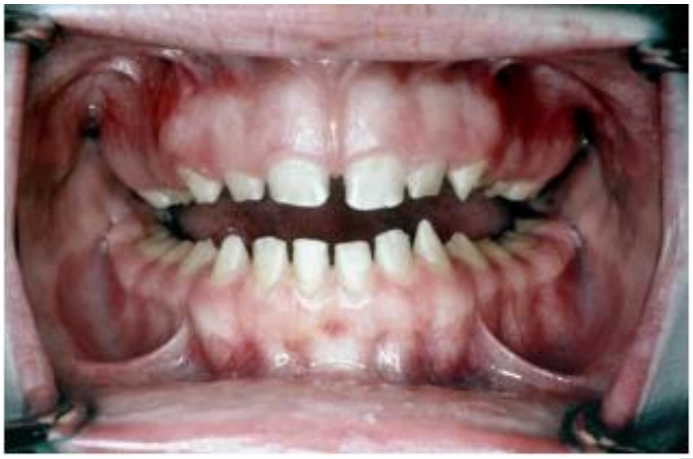


Fig. 26. Hypoplastic amelogenesis imperfecta

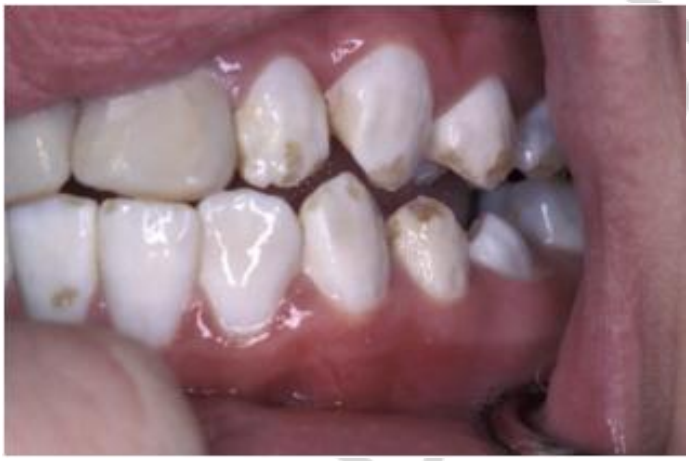


Fig. 27. Amelogenesis imperfecta, hypomaturation type

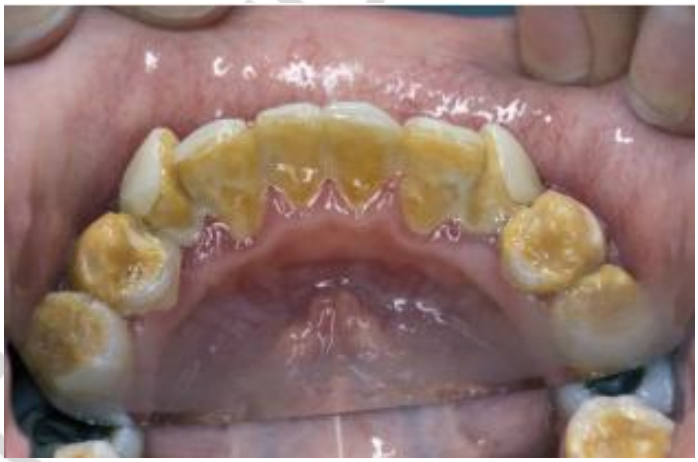


Fig. 28. Hypocalcified amelogenesis imperfecta

Dentinogenesis imperfect. Dentinogenesis imperfecta represents a group of hereditary conditions that are characterized by abnormal dentin formation. These conditions are genetically and clinically heterogenous and can affect only the teeth or can be associated with the condition osteogenesis imperfecta. The dentinogenesis imperfectas and dentin dysplasias were classified using

clinical, radiographic and histopathologic features in 1973 and this nosology remains in use today. Dentinogenesis imperfecta has been subdivided based on:

– its association with osteogenesis imperfecta (Type I);

– or having no association with osteogenesis imperfecta (Type II);

– or being associated with the Brandywine triracial isolate and large pulp chambers (Type III).

In all the three dentinogenesis imperfecta types the teeth have a variable blue-gray to yellow brown discoloration that appears opalescent due to the defective, abnormally colored dentin shining through the translucent enamel (fig. 29). Due to the lack of support of the poorly mineralized underlying dentin, the enamel frequently fractures from the teeth leading to rapid wear and attrition of the teeth. The severity of discoloration and enamel fracturing in all dentinogenesis imperfecta types is highly variable even within the same family. If left untreated, it is not uncommon to see the entire dentinogenesis imperfecta affected dentition worn off to the gingiva.

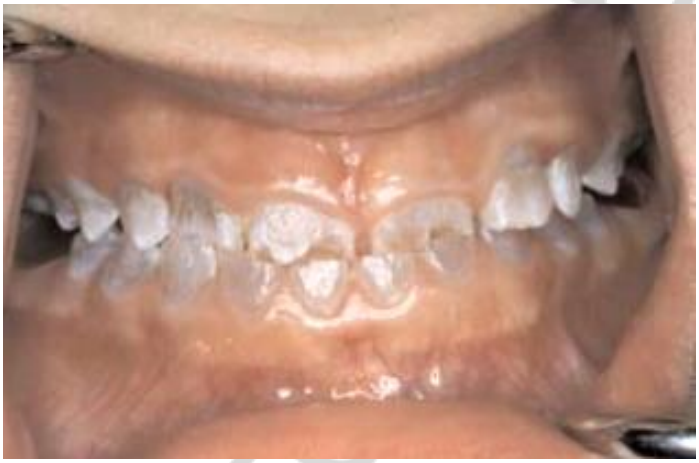


Fig. 29. Dentinogenesis imperfecta

Radiographs show pulpal obliteration in dentinogenesis imperfecta types I and II due to rapid and excessive deposition of dentin. The pulp chambers are large in DI type III.

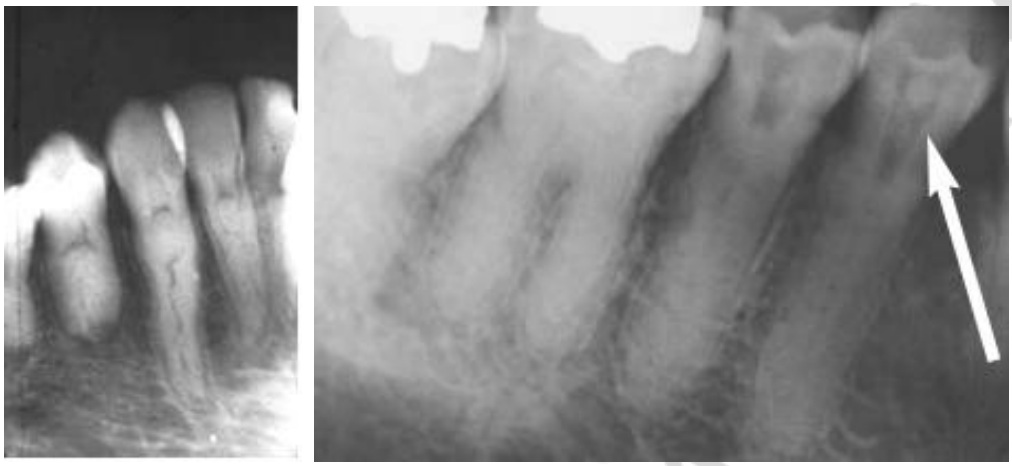
Dentin dysplasia is a rare hereditary disturbance of dentin formation characterized by defective dentin development with clinically normal appearing crowns, severe hypermobility of teeth and spontaneous dental abscesses or cysts. Radiographic analysis shows obliteration of all pulp chambers, short, blunted, and malformed or absent roots and periapical radiolucencies of non carious teeth. Dentin dysplasia is an autosomal dominant hereditary disturbance in dentin formation, which may present with either mobile teeth or pain associated with spontaneous dental abscesses or cysts. It is a rare anomaly of unknown etiology that affects approximately one patient in every 100,000. This condition is rarely encountered in dental practice. In 1972, Witkop classified dentin dysplasia into two types:

1) radicular dentin dysplasia as type I;

2) coronal dentin dysplasia as type II.

In type I, both the deciduous and permanent dentitions are affected. The crowns of the teeth appear clinically normal in morphology but defects in dentin formation and pulp obliteration are present (fig. 30, A). Radiographic examination is important for the identification of dentin dysplasia type I.

Dentin dysplasia type II is characterized by teeth of nearly normal length but as the pulp ascends to the crown, it flares into a flame shape or thistle tube shape (fig. 30, B).



A B

Fig. 30. Dentin dysplasia:
A — type I; B — type II

Shell teeth — rare hereditary disturbance of tooth formation characterized by normal thickness of enamel, extremely thin dentin, and enlarged pulp chamber. The thinning of the dentin may involve the entire tooth or be confined to the root, and is most frequent in deciduous teeth.

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CONTENTS

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2-е издание

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