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NON-CARIOUS TOOTH LESIONS DEVELOPING BEFORE ERUPTION

Minsk BSMU 2020

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

Е. А. Мирная, Л. Г. Борисенко, В. Р. Булатова

НЕКАРИОЗНЫЕ БОЛЕЗНИ ЗУБОВ, РАЗВИВАЮЩИЕСЯ ДО ИХ ПРОРЕЗЫВАНИЯ

NON-CARIOUS TOOTH LESIONS DEVELOPING BEFORE ERUPTION

Учебно-методическое пособие

Минск БГМУ 2020

УДК 616.314.14(075.8)-054.6
ББК 56.6я73
М63

Рекомендовано Научно-методическим советом университета в качестве учебно-методического пособия 26.06.2020 г., протокол № 10

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М63 Некариозные болезни зубов развивающиеся до их прорезывания = Non-carious tooth lesions developing before eruption : учебно-методическое пособие / Е. А. Мирная, Л. Г. Борисенко, В. Р. Булатова. – Минск : БГМУ, 2020. – 23 с.

ISBN 978-985-21-0639-9.

Рассматриваются этиология, клиника, лечение и профилактика болезней зубов некариозного происхождения, развивающиеся до их прорезывания. Отражены вопросы диагностики и дифференциальной диагностики некариозных болезней зубов.

Предназначено для студентов 5-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке.

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MOTIVATIONAL CHARACTERISTICS OF THE THEME

Non-carious tooth lesions

Total time: 65–70 minutes.

In International classification of dental diseases (ICD-10, WHO) non-carious lesions of hard tissues developing before tooth eruption are listed in chapter K00 “Disorders of tooth development and eruption”, K01 “Embedded and impacted teeth”.

A number of epidemiological researches have revealed that the prevalence of these diseases has increased significantly and they are wide-spread nowadays. Very similar clinical features of these diseases make difficulties for their differential diagnoses. Making detailed examination is very important to differentiate them from other tooth lesions.

Thus, investigating patients’ life and diseases history, clinical examination, medical conclusions of other specialists (therapist, endocrinologist, gynecologist, geneticist), previous treatment methods and their effectiveness are very important for the objective understanding of these diseases. Additional diagnostic methods allow a dentist to discover the etiology and pathogenesis of the patient’s disease and make up the plan of treatment more rationally.

Seminar task:

1. To study fragments of International WHO classification, including chapters K00, K01.
2. To study etiology, clinic al picture, diagnostics, methods of treatment and prevention of the most widespread diseases given in chapter K00 “Disorders of tooth development and eruption”.
3. To study etiology, clinic al picture, diagnostics, methods of treatment and prevention of the most widespread diseases given in chapter K01 “Embedded and impacted teeth”.
4. To improve knowledge and skills to be able to make up a plan of treatment of tooth hard tissue diseases of non-carious origin.

Requirements for the initial level of knowledge:

1. Classification of non-carious diseases of tooth (ICD-10, WHO).
2. Stages of tooth development.
3. Formation of dentin (dentin genesis).
4. Formation of enamel (enamel genesis).
5. Enamel: anatomy, histology, chemical composition.
6. Dentin: anatomy, histology, chemical composition.
7. Enamel and dentin structure.
8. Clinic al picture, diagnostics of dental caries.

Test questions on the theme of the session:

1. Classification of non-carious tooth diseases. Chapters of International WHO classification: K00 “Disorders of tooth development and eruption”, K01 “Embedded and impacted teeth”.
2. Anodontia. Supernumerary teeth. Abnormalities of size and form of teeth. Short characteristics of diseases of the mentioned groups (K00.0-K00.2).
3. Mottled teeth (K00.3) Characteristics of diseases of the mentioned group. Fluorosis: etiology, pathogenesis, clinic al picture, diagnostics, index diagnostics, treatment and prevention.
4. Disturbances in tooth formation (K00.4) Characteristics of diseases of the mentioned group. Enamel hypoplasia: classification, etiology, clinic, diagnostics, treatment and prevention.
5. Hereditary disturbances in tooth structure, not elsewhere classified (K00.5). Short characteristics of diseases of the mentioned group.
6. Other disorders of tooth development (K00.8). Short characteristics of diseases of the mentioned group.
7. Embedded and impacted teeth (K01). Short characteristics of diseases of the mentioned group.
8. Establishing the diagnosis and administering the treatment measures for non-carious diseases of tooth, developing before eruption.

CLASSIFICATION OF NON-CARIOUS TOOTH LESIONS

Numerous non-carious lesions of tooth were divided by V. K. Parikeev (1968) into two main groups: 1) appearing after eruption; 2) appearing in the period of tissue development. Also it was revealed that developmental anomalies of teeth are not very rare and can occur in various stages of tooth development:

– Dental Lamina formation stage: anodontia (no teeth).

– Initiation and Proliferation (during the formation of tooth bud): partial anodontia, supernumerary, geminated/fused teeth.

– Histo-differentiation: odontodysplasia.

– Morpho-differentiation: macro/micro size, dens invaginatus, dens evaginatus, Hutchinson’s incisors, talon cusp, taurodontism, dilacerations.

– Apposition–matrix formation: amelogenesis imperfecta, dentinogenesis imperfecta, enamel hypoplasia.

– Calcification–mineralization of the matrix: fluorosis, amelogenesis Imperfecta.

In the International classification (ICD-10, WHO) the diseases of non-caries origin are basically included into three chapters: K00, K01, K03. Thus we can identify two large groups:

1. Diseases appearing in the tooth before eruption:

K00 “Abnormalities of tooth development and eruption”

K01 “Impacted teeth”

2. Diseases appearing after eruption:

K03 “Other diseases of hard tissues of teeth”

Fragments of the International classification of diseases of oral cavity, salivary glands and jaws (ICD-DA, WHO) are presented in Appendix 1.

ANODONTIA (K00.0)

Normally, the human dentition counts 32 permanent teeth whereas the deciduous dentition contains 20. Teeth develop via a series of interactions between odontogenic epithelium and neural crest-derived ectomesenchyme of the early jaw primordia. While considerable information is now available on how these molecules interact to produce an individual tooth, much less is known about the processes that control overall tooth number within the dentition.

However, it has increasingly become clear that both a too high and a too low number can be traced back to a broad area of genetic changes. These numerical aberrations may occur as part of a more general syndrome as well as an isolated feature.

Congenital absence of teeth can be found in either deciduous or permanent dentition. Absence of all teeth is called ***anodontia,*** which is extremely rare. Relatively more frequent is ***hypodontia*** when 2–10 teeth are missing or ***oligodontia*** when it involves more than a half of the total dentition. The teeth that are most often absent are the third molars, followed by the second lower premolars, the lateral upper incisors and the second upper premolars.

Both environmental and genetic factors can cause failure of tooth development. Environmental factors such as irradiation and chemotherapeutic agents can arrest tooth development, but most of the cases are caused by genetic factors. Hypodontia and oligodontia may be inherited as an autosomal dominant trait with incomplete penetration and variable expression, but autosomal recessive, sex-linked and polygenic or multifactorial models of inheritance probably also play a role.

Missing teeth, either hypodontia, oligodontia or anodontia, may occur within the context of a syndrome. Congenital absence of teeth has been reported to be associated with Down syndrome, lateral maxillary incisors, lower central incisors and second premolars being the most commonly missing teeth.

The prototypical syndrome with a decrease in teeth number is ectodermal dysplasia. This is a large and complex group of disorders characterized by various combinations of defects in hair, nails, teeth and sweat glands, either isolated or associated with malformations. Oligodontia is always present and complete anodontia is occasionally seen.

Tooth agenesis may also occur associated with oral clefts. This agenesis may involve teeth in the region of the cleft, but teeth may also be absent in other regions of the mouth pointing to a common underlying genetic cause for both clefting and tooth agenesis.

Tooth agenesis is often associated with different kinds of anomalies in other teeth. These include reduction in tooth dimensions and abnormal morphology, such as shortened roots and taurodontia and enamel hypoplasia. A decreased tooth dimension has also been observed in healthy relatives of patients with severe tooth agenesis. Typical changes in tooth crown morphology include conical or peg-shaped teeth.

SUPERNUMERARY TEETH (K00.1)

Supernumerary teeth, or hyperdontia, refer to the teeth that are formed in addition to the normal number. They can be associated with a syndrome or they can be found in non-syndromic patients. Supernumerary teeth occur as ***mesiodens***, between the maxillary central incisors, or can be located in the premolar-molar region as supernumerary ***paramolars*** or supernumerary ***fourth molar*** or ***distomolar.*** These supernumerary teeth may have a normal morphology or may show little or no resemblance to the other teeth.

A mesiodens usually is peg-shaped with root development at the similar stage or ahead of that of the adjacent teeth. Supernumerary premolars and molars may show more variations in size and form. Clinically they may cause retained or delayed eruption of permanent teeth. In general, a low number of supernumerary teeth are not related with additional abnormalities, whereas a higher number of them is usually are associated with other conditions such as cleft palate and cleft lip or with variable syndromes. Hereditary factors may play a role in both syndrome- and non-syndrome-related hyperdontia.

The prototypical syndrome with supernumerary teeth is cleidocranial dysostosis, an autosomal dominant hereditary disorder. It is characterised by bone deformities among which there are hypoplastic clavicles, short stature and dental abnormalities including variable numbers of supernumerary teeth that occur in almost all of them.

From the other syndromes with hyperdontia, family adenomatous polyposis (FAP) should be mentioned, an autosomal dominant hereditary disorder. It is characterised by gastrointestinal polyps, multiple osteomas and tumours in skin and soft tissues. The jaws are not only involved by showing a high number of supernumerary teeth but also by the presence of osteomas, mostly in the mandible.

ABNORMALITIES OF SIZE AND FORM OF TEETH (K00.2)

Teeth may show an abnormal form due to malformations occurring during development or acquired after eruption into the oral cavity. The microscopic structure of the dental tissues usually is normal.

In case of ***fusion***, two adjacent teeth are connected with each other by either enamel and dentine both or, rarely, by enamel alone. The fusion may be complete over the full length of the teeth or only partial. The condition is due to the merging of two adjacent tooth germs. If the tooth germs touch each other after disappearing of the investing dental follicle, odontogenic soft tissues may unite to form a common matrix that subsequently mineralizes as usual.

The number of teeth will be diminished by one in this condition, two normal teeth being replaced by one of abnormal appearance. The dentition can still have a normal teeth number when there is fusion of a tooth with a supernumerary counterpart. Occurrence of a separate dentin stalk projecting from the root surface and capped with enamel and having a common pulp chamber with the main tooth probably represents this type of fusion.

Gemination occurs when there is partial development of two teeth from one single tooth germ. An abnormally formed tooth with usually one root canal is the outcome of this developmental fault. When the division is complete, the condition is called twinning. Then, the normal number of teeth is increased by one, the superfluous tooth usually being the mirror image of its adjacent counterpart.

As already mentioned, the distinction between gemination and fusion is made by counting the number of teeth in the dentition. In case of fusion, their number is decreased by one as two teeth are replaced by one single abnormally formed fusion product. In case of gemination, the number is normal but one of the teeth being replaced by an abnormally formed one.

Concrescence is the connection of two or more teeth by root cementum alone after the tooth crowns have been formed. The teeth themselves are usually normal in size and form. As the uniting tissue is cementum, an increased amount of cementum covering the roots (hypercementosisis) usually also present.

Dens invaginatus, also called dens in dente, is a condition in which the crown surface of the tooth invaginates deeply into the crown and quite often also into the root part. In this way, a deep groove is created, lined by dentin with a thin of enamel layer. Externally, the involved tooth may only show a tiny pit on its outer surface. In the interior of the tooth, this pit dilates to form a large cavity.

Through this pit, bacteria from the oral environment have free access to the inner part of the tooth, and therefore, this form anomaly makes these teeth very vulnerable for carious decay. The outer opening of the invagination may also be located laterally.

In ***dens evaginatus***, an enamel-covered tubercle projects from the occlusal surface of the affected tooth. This evagination consists not only of enamel but also of dentin and pulp tissue. It mostly occurs in molar and premolar teeth. Occasionally, an accessory cusp is seen at the lingual surface of the lower incisor and called a talon cusp.

In ***taurodontism***, the pulp chamber shows an increased vertical dimension, thus extending far into the root area of the involved tooth. As a consequence, in multirooted teeth, the splitting of the root into its several extensions, the so-called furcation, occurs at a level much more apically than it is in case of normal teeth. Also the root bifurcation or trifurcation may be very shallow or even absent.

Enamel pearls are deposits of enamel in an abnormal position, mostly the outer root surface although they can also be found embedded in the dentin. Their size may vary from a pinpoint to several millimeters. Sometimes, the enamel pearl has the form of a cap covering a dome-shaped dentin core. If this type of enamel pearl has a considerable size, its distinction from a supernumerary fused tooth becomes debatable.

MOTTLED TEETH (K00.3)

Enamel opacities are white opaque spots in smooth-surfaced enamel. Probably they are due to transient hypomineralisation of the enamel matrix. Most often, they are seen on the incisor teeth. Incidentally, they may also be brown and mottled. In that case, distinction from local enamel hypoplasia may become equivocal. The prevalence of this condition is rather high. Probably, enamel opacities are the result of short intervals of disturbed deposition of enamel matrix. Subsequent normal formation of enamel matrix will then bury the abnormal area.

Dental Fluorosis is a permanent hypomineralisation of enamel, in its mildest form characterised as small white spots. Fluorosis generally appears as a horizontal striated pattern across a tooth. More severe forms range between white opaque areas to darkly stained and pitted enamel. An overdose of fluoride interferes both with function of ameloblasts and proper calcification of the enamel matrix. It is important to diagnose the condition and differentiate between dental fluorosis and other enamel disturbances. Molars and bicuspids are most frequently affected, followed by upper incisors. The mandibular incisors are usually least affected. Fluorosis tends to be bilaterally symmetrical. Defects may appear as fine white or frosted lines or patches near the incisal edges or cusp tips.

Dental fluorosis is generalized within the dentition and over the entire tooth surface which makes it easy to distinguish fluoride-induced enamel changes from other enamel defects (nonfluoride origin) which may be symmetrically

distributed in the oral cavity. Due to excessive fluoride intake, enamel loses its lustre. In its mild form, dental fluorosis is characterized by white, opaque areas on the tooth surface and in severe form, it is manifestated as yellowish brown to black stains and severe pitting of the teeth. This discoloration may be in the form of spots or horizontal streaks.

Normally, the degree of dental fluorosis depends on the amount of fluoride exposure up to the age of 8 to 10 years, as fluoride stains appear only on the developing teeth while they are being formed in the jawbones and are still under the gums. The effects of dental fluorosis may not be apparent if the teeth are already fully grown prior to the fluoride over exposure. Therefore, the fact that an adult shows no signs of dental fluorosis does not necessarily mean that his or her fluoride intake is within the safety limit.

Dean's Fluorosis Index HT. Dean's fluorosis index was originally given in 1934 and later modified in 1942 and is currently the most universally accepted classification system. An individual's fluorosis score is based on the most severe form of fluorosis found on two or more teeth. Dean's is used to score the amount of dental fluorosis (discoloration) present on teeth. A score is given, based on the two teeth most affected. If the teeth are not equal in appearance, the less affected tooth is the one scored. Indices for scoring dental fluorosis and levels of prevention are presented in Appendix 2–4.

DISTURBANCES IN TOOTH FORMATION (K00.4)

Enamel hypoplasia is a defect that occurs when dental enamel isn't formed completely, usually because of malnutrition or disease. Enamel hypoplasia is identified as a horizontal line, a series of pits or grooves along the outer surface of the tooth. These lines mark points at which the tooth's growth was resumed after it had stopped. Hypoplasia is most common in the permanent teeth and represents episodes of arrested growth in infancy or childhood while these teeth were still developing. Once the enamel is formed, it can no longer be affected. 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.

Environmental and genetic factors that interfere with tooth formation are thought to be responsible for enamel hypoplasia. This includes trauma of the teeth and jaws, infections during pregnancy or infancy, poor prenatal and postnatal nutrition, hypoxia, exposure to toxic chemicals and a variety of hereditary disorders.

Differential diagnoses of this disease is challenging. Some researchers describe enamel hypoplasia as a disturbance in tooth formation leading to macroscopically visible defects on the surface of the enamel. Although this definition also includes the hypoplastic form of amelogenesis imperfecta,

the term enamel hypoplasia in practice is only applied to enamel lesions due to a systemic interference.

This means that alterations may not be universal in that sense that they involve all teeth or the entire tooth crown as in the case of amelogenesis imperfecta. Only the enamel formed during the time span in which the systemic interfering factor was active will be abnormal. Macroscopical inspection of these teeth will show the distinction from amelogenesis imperfect. All teeth show abnormalities, whereas, in enamel hypoplasia only some teeth may be abnormal.

Regional Odontodysplasia is a developmental disturbance consisting of both enamel and dentin abnormalities in several adjacent teeth. Suffix "regional" emphasises usually localised character, but a few cases have been described with involvement of more extensive parts of the dentition, the abnormal teeth being present bilaterally and in both upper and lower jaws. The teeth are abnormally formed and the covering of enamel layer is thin and yellow.

Radiographically, the affected teeth show a ghostlike appearance ("ghost teeth") due to their reduced radiodensity. The pulp chambers are wide and the dentin is greatly reduced in thickness. The enamel is hypoplastic and hypomineralised, and the dentin contains large areas of interglobular dentin. The predentin zone is also very wide. The dental pulp usually contains large and irregular aggregates of mineralised matrix, the so-called denticles. The condition may be accompanied by gingival enlargement.

HEREDITARY DISTURBANCES IN TOOTH STRUCTURE (K00.5)

Amelogenesis imperfecta is a hereditary condition afflicting the tooth **enamel**. It is subdivided into a considerable number of conditions depending on the clinical appearance, the kind of disturbance and the pattern of inheritance that may be autosomal recessive, autosomal dominant or X linked.

Amelogenesis imperfecta has to be discussed to recognize the various subtypes. Teeth are showing either abnormal enamel caps of normal hardness or enamel caps consisting of enamel that is too soft and discoloured. The first is called hypoplastic and the latter hypomineralised or hypomatured.

The ambiguities arising from this phenotypic classification are demonstrated by the combined occurrence of both hypoplastic and hypomineralised amelogenesis imperfecta in a family also having taurodontism. Thus it has been proposed recently that a classification based on the mode of inheritance may be more appropriate.

As amelogenesis is a hereditary condition, it afflicts in principle all teeth or nearly all teeth although there may be variations in expression. Moreover, the defect may be associated with other dental anomalies such as disturbed form, calcification of the dental pulp and delayed eruption of the teeth. Occasionally,

the condition is associated with dental follicular hamartomas and gingival hyperplasia.

Amelogenesis Imperfecta, Hypoplastic A melogenesis imperfect of the hypoplastic type, is the result of a decreased amount of enamel matrix laid down during tooth formation. Therefore, the enamel cap does not acquire its normal thickness. As mineralisation is normal, the hardness of the remaining enamel layer may be normal. However, the reduced thickness of the enamel cap causes an abnormal crown form of the involved teeth. These externally visible abnormalities may vary from an almost absent enamel cap to only a few irregularities in an otherwise normally formed tooth. These irregularities have been classified as rough, pitted and grooved that may run vertically or horizontally.

Amelogenesis Imperfecta, Hypomineralised I n the hypomineralised type of amelogenesis imperfecta, the enamel initially develops a normal thickness, but the matrix is not mineralized in a normal way. Therefore, the teeth erupt with an initially normal appearance of their crowns, but the poor mineralized enamel is soft and, therefore, is easily worn away in the mechanically demanding oral environment, thus exposing the underlying dentin. The enamel is light yellow-brown to orange and becomes brown to black after eruption because of stains from food or beverages. Characteristically, remnants of enamel remain present in mechanically privileged niches, for example, the cervical part of the tooth crown. Due to its low content of minerals, this defective enamel will not dissolve completely during decalcification, and therefore, it may be present in paraffin sections made from these teeth.

Amelogenesis Imperfecta, Hypomatured A melogenesis of the hypomatured type is difficult to discern from the hypomineralised type as it also shows the presence of enamel that is softer than it should be and that shows discoloration.

Structural abnormalities of ***dentin*** mostly have a hereditary background with an autosomal dominant way of transmission. In general, there is a failure of normal dentin formation after the initial deposition of a small amount of mantle dentin. As a result of this, involved teeth may show either abnormally short or entirely absent roots, whereas the pulp chambers may obliterate partly or entirely due to the formation of abnormal dentin masses.

Two main groups are recognised: ***dentinogenesis imperfecta and dentina dysplasia.*** Three different types of dentinogenesis imperfecta are recognised: type I that forms part of the spectrum of abnormalities that is shown by the generalised skeletal disorder osteogenesis imperfecta and types II and III that both occur isolated.

Tooth crowns show an amber discoloration and have an ovoid shape, due to cervical constriction. Roots are short and thin. The pulp chambers are initially larger than normal but, with course of time, become almost obliterated by

ongoing abnormal dentin formation. As the covering of enamel tends to fracture from the underlying weakened dentin layer, the impression of coexisting amelogenesis imperfecta may arise.

Dentinogenesis imperfecta type I and type II are clinically similar and discernable only by the already mentioned association of type I with osteogenesis imperfect. Type III, which is limited by a specific racial subpopulation, differs from type I and II in not showing a time-dependent obliteration of the pulp chamber, teeth remaining extremely thin and therefore known as shell teeth.

Dentin dysplasia is subdivided into two types. In type I, both deciduous and permanent teeth have a normal shape, but radiographically the roots are shown to be abnormally short or even absent, while pulp as well as root canals are obliterated by dentin masses. Dentin dysplasia type II may represent a variant of dentinogenesis imperfecta.

The features seen in the deciduous dentition resemble those observed in this latter condition, whereas the permanent teeth are of normal shape and form but may show a deformed root canal and pulp stones. For a precise diagnostic classification, dental radiographs, either intraoral X-rays or panoramic radiographs, are the most useful tool as in this way, both tooth contour and features of the root and the pulp cavity are clearly shown.

Due to overlapping phenotypes for all above-mentioned clinical categories of heritable dentin defects, their categorization has been extensively discussed in the past, and currently it is proposed that these disorders are reflecting a variable expression of a common genetic disorder rather than different entities.

For the histopathologist, the heritable dentin defects are recognizable by the presence of a small rim of normal dentin covering either an unusually wide pulp space in shell teeth or encasing a pulp chamber filled with irregular masses of dentin-containing haphazardly running dentin tubuli in the other categories. This abnormal dentin should not be confused with the tertiary dentin formed locally in the dental pulp as a response to some external irritant.

In ***odontogenesis imperfecta,*** both enamel and dentin exhibit pathologic changes in all teeth. The enamel is hypoplastic and the dentin shows changes similar to those seen in dentinogenesis imperfecta.

OTHER DISORDERS OF TOOTH DEVELOPMENT (K008)

This group includes: ***colour changes during tooth formation and intrinsic staining of teeth NOS*** (not otherwise specified). It includes ***tetracycline discoloration (K0083)***, which has to be rare, but still is not uncommon where tetracycline has been given to children or pregnant women.

Binding with calcium, tetracycline is deposited together with it in any tissue undergoing mineralization. After its incorporation during mineralization, it can be demonstrated in teeth and bones in ultraviolet light, showing up as

florescent yellow bands. Grossly, tetracycline causes a greyish-black discoloration of the tooth crown (Appendix 4).

When making ground sections of these teeth, the tetracycline bands can be observed under UV light illumination both in dentin and in enamel, each band indicating a time point of tetracycline administration. After decalcification, the tetracycline has been lost together with the calcium, and therefore, in decalcified paraffin sections, this fluorescence is not present anymore.

EMBEDDED AND IMPACTED TEETH (K01)

Tooth eruption comprises the movement of teeth through the soft tissues of the jaw and the overlying mucosa into the oral cavity. The involved biological processes are not yet entirely elucidated, but the importance of the dental follicle and its role in initiating eruption by regulating alveolar bone resorption and alveolar bone formation has been firmly established. Teeth may erupt too early, too late, not in the proper position or not at all. In the latter event, one speaks of impaction.

According to definition of ICD-10, WHO classification, ***embedded tooth*** is a tooth that has failed to erupt without obstruction by another tooth. An ***impacted tooth*** is a tooth that has failed to erupt because of obstruction by another tooth.

Teeth may erupt at the wrong place or not at all if local conditions interfere with the eruption pathway. Usually, these are diseases of the jaw bone or the overlying soft tissues.

Also, odontogenic tumours arising around the crown area of developing teeth may hinder proper eruption. Sometimes the failure of a tooth to erupt will be the first sign of such a jaw tumour. Eruption of a tooth in an aberrant position more often is due to the jaw lesions lying adjacent to the developing roots than to obstacles in the eruption pathway.

ESTABLISHING THE DIAGNOSIS AND ADMINISTERING THE TREATMENT MEASURES

The diagnosis of the patient's symptoms and other significant disease processes sometimes leads to a self-evident conclusion. The diagnosis is usually established by:

– Reviewing the patient's history and examination data.

– Listing those items that may suggest the possibility of a significant health problem.

– Grouping items into primary and secondary, acute and chronic, high priority versus low priority, etc.

– Categorizing the disease on the basis of this grouping.

For an effective treatment it is essential that the diagnosis should be written down into the patient's case history. Detailed history and examination data are important for accurate understanding of the disease. The main diagnostic methods include at least: complaints, history, visual characteristic of the defect, density and sensitivity during probing, localization on the tooth, number of teeth involved in the process, percussion, palpation, drying.

A definite diagnosis cannot be always made, despite possessing full examination and history data. In such cases, a descriptive term may be used for describing the probable health problem, termed as the "provisional diagnosis". The clinician should always keep in mind the differential diagnosis. The 'provisional diagnosis' indicates to list those items that indicate an abnormality or suggest the possibility of significant health problems. Additional diagnostic methods can include: index evaluation, thermometry, enamel staining, X-ray, assessment of biophysical and biochemical properties of oral fluid, luminescent stomatoscopy, referral to consultation with other specialists, including endocrinologist, geneticist.

Dental indices provide a quantitative method for measuring, scoring, and analyzing dental conditions in individuals and groups. An index describes the status of individuals or groups in respect to the condition being measured. It is an objective mathematical description of a disease or condition based on carefully determined criteria under specified circumstances.

In private practice, index scores are used to educate, motivate, and evaluate the patient. By comparing scores from the initial exam with a follow-up exams, the patient can measure the effects of personal daily care. Dental indices can be simple, measuring only the presence or absence of a condition, or they can be cumulative, measuring all evidence of a condition, past and present. Indices used in assessing dental fluorosis are presented in the Appendix 3–4.

All the records, clinical findings, the provisional diagnosis and investigations are clubbed together to frame the final diagnosis on which treatment is planned. The final diagnosis is first made on the bases of chief complaints of the patient and then other problems are considered. Patients must be informed of their diagnosis and the results of the various examinations and tests performed. Also, the patient should be informed of the nature, significance and treatment of the health problem that has been clearly diagnosed.

Treatment measures include the methods of discoloration correction, restorative dental care, veneers, crowns, elimination of risk factors and supporting treatment. Whitening of teeth can be reached by the removal of stained dental plaque and improvement of oral hygiene. At the same time, modern dentistry offers a wide range of methods which are able to correct tooth color: bleaching, microabrasia, esthetic restoration. Tooth bleaching is a chemical process in which free radicals of hydrogen dioxide lead to the whitening of hard tooth tissues. The up-to-date systems of bleaching are

based on application of hydrogen dioxide and carbamide peroxide agents in combination with activating factors. Nowadays tooth bleaching is classified as follows: 1) vital, external, internal, combined; 2) office, home, mixed; 3) controlled, uncontrolled; 4) “waiting room” bleach technique.

Dental restoration can help to solve esthetic problems in case of low efficiency of bleaching techniques. The choice of filling material depends on the type of defect and its depth. Good results are achieved in the treatment of non-carious lesions with compomers. In deeper and extensive defects involving dentine glass ionomer cements providing binding with tooth tissue can be used. The use of compomers and composites with up-to-date bond-systems also is recommended.

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Appendix 1

Fragment of International Statistical Classification of Diseases and Related Health Problems 10 Revision (ICD-10)-WHO Version for 2019

Chapter XI Diseases of the digestive system (K00-K93)
Diseases of oral cavity, salivary glands and jaws (K00-K14)

K00 Disorders of tooth development and eruption
Excl.: embedded and impacted teeth

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
- Fusion
- Gemination

of teeth

Dens:
- evaginatus
- in dente
- invaginatus

Enamel pearls
Macrodontia
Microdontia
Peg-shaped (conical) teeth
Taurodontism
Tuberculum paramolare

Excl.: tuberculin the 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

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, mottled teeth

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

- Amelogenesis
- Dentinogenesis | imperfecta
- Odontogenesis

Dentinal dysplasia
Shell teeth

K00.8 Other disorders of tooth development

Colour changes during tooth formation
Intrinsic staining of teeth NOS

K01 Embedded and impacted teeth

Excl.: embedded and impacted teeth with abnormal position of such teeth or adjacent teeth

K01.0 Embedded teeth

K01.1 Impacted teeth

Appendix 2

LEVELS OF PREVENTION FOR DENTAL FLUOROSIS

Primary prevention	Secondary prevention	Tertiary prevention
Specific guidelines on the use and appropriate dose levels of fluoride supplements, and use of fluoride tooth paste for young children. In high fluoride areas: • Provide an alternate supply of drinking water • Employ defluoridation techniques at the community or individual level	Improve the nutritional status, especially of expecting mothers, newborns and children up to the age of 12 years. Treat other causes of fluoride toxicity such as kidney and thyroid diseases, etc.	Treat the discolored/ disfigured dentition by appropriate aesthetic treatment such as bleaching, microabrasion, laminates, veneers, crowns

Appendix 3

THE DEAN`S FLUOROSIS INDEX

The Dean fluorosis index is simple and easy to use, natural light is enough for performing examination, no need to completely dry tooth surfaces. It is widely used in epidemiological studies. Index is determined by formula: $F_{ci} = (n \cdot w) / N$, where N is the total number of teeth tested; n is the number of persons found to have fluorosis; w is the established score.

Scores and criteria for dean's fluorosis index

Score	Criteria
Normal (0)	The enamel represents the usually translucent semivitriform type of structure. The surface is smooth, glossy, and usually a pale creamy white color
Questionable (0.5)	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilized when a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of "normal" is not justified
Very mild (1)	Small, opaque, paper white area scattered irregularly over the tooth but not involving as much as approximately 25 percent of the tooth surface. Frequently included in this classification are teeth showing no more than 1 to 2 mm of white opacity at the tip of the summit of the cusps of the bicuspids or second molars
Mild (2)	The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50 percent of the tooth
Moderate (3)	All enamel surfaces of the teeth are affected, and surfaces subject to attrition show marked wear. Brown stain is frequently a disfiguring feature
Severe (4)	All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be altered. The major diagnostic sign of this classification is the discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded appearance

Appendix 4

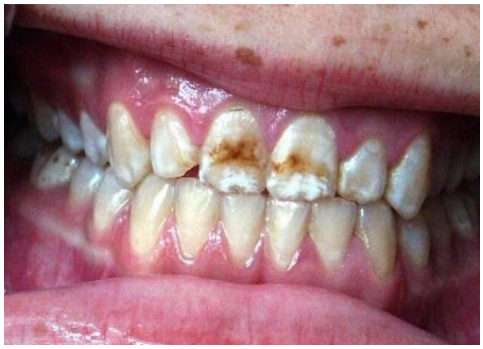
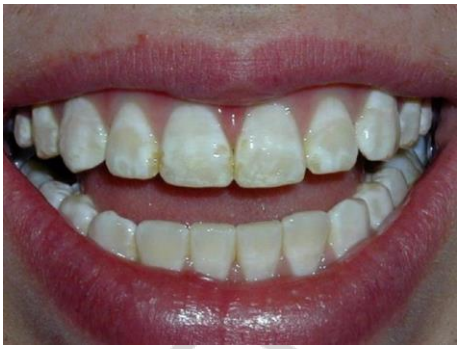
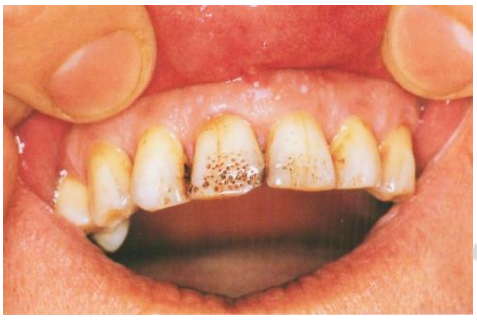
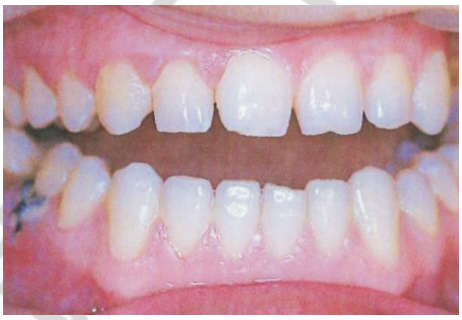
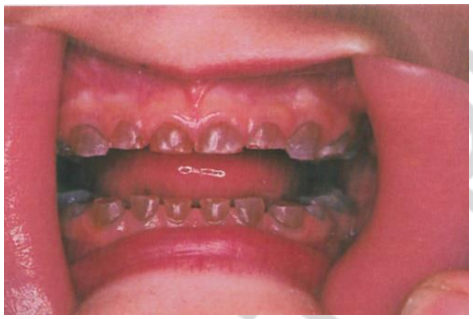
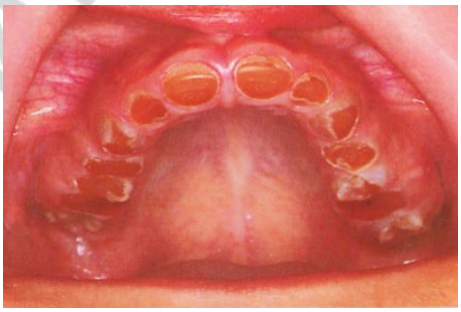
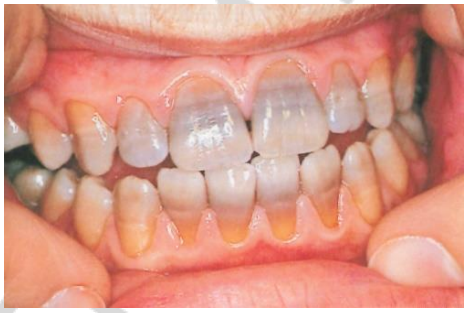
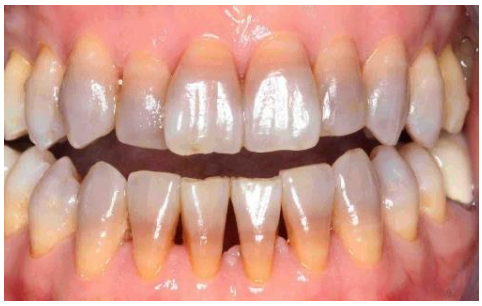
TOOTH SURFACE INDEX OF FLUOROSIS (TSIF)

This index was developed in 1984 by Horowitz H. S., Driscoll W. S., Meyers R. J. and used by researches in the National Institute of Dental Research. It is probably more sensitive than Dean's index specially for the mildest forms of fluorosis. Each tooth surface is seen and scored on a 0–7 scale, whereas Dean's index applies only to two worst teeth in the mouth. Selection of a teeth are assessed.

Surface of anterior teeth: Separate score is given for every intact labial or lingual surface of anterior teeth. Posterior teeth: a separate score, is given, for every buccal, occlusal and lingual surface.

Scoring Criteria Clinical Criteria and Scoring System for the Tooth Surface Index of Fluorosis

Score	Criteria
0	Enamel shows no evidence of fluorosis
1	Enamel shows definite evidence of fluorosis, namely areas with parchment-white color that total less than 1/3rd of the visible enamel surface. This category includes fluorosis confined only to incisal edges of anterior teeth and cusp tips of posterior teeth ("snow capping")
2	Parchment-white fluorosis totals at least 1/3rd of the visible surface but less than 2/3rd
3	Parchment- white fluorosis totals at least 2/3rd of the visible surface
4	Enamel shows staining in conjunction with any of the preceding levels of fluorosis. Staining is defined as an area of definite discoloration that may range from light to very dark brown
5	Discrete pitting of enamel exists, unaccompanied by evidence of staining of intact enamel. A pit is defined as a definite physical defect in the enamel surface with a rough floor that is surrounded by a wall of intact enamel. The pitted area is usually stained or differs in color from the surrounding enamel
6	Both discrete pitting and staining of the intact enamel exists
7	Confluent pitting of the enamel surface exists. Large areas of enamel may be missing and the anatomy of the tooth may be altered. Darkbrown stain is usually present

Dental fluorosis		
Enamel hypoplasia		
Dentinogenesis imperfect		
Tetracycline staining		

CONTENTS

Учебное издание

Мирная Елена Андреевна
Борисенко Людмила Григорьевна
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НЕКАРИОЗНЫЕ БОЛЕЗНИ ЗУБОВ, РАЗВИВАЮЩИЕСЯ ДО ИХ ПРОРЕЗЫВАНИЯ

NON-CARIOUS TOOTH LESIONS DEVELOPING BEFORE ERUPTION

Учебно-методическое пособие

На английском языке

Ответственная за выпуск Т. Н. Манак
Переводчик Е. А. Мирная
Компьютерная вёрстка Н. М. Федорцовой

Подписано в печать 15.09.20. Формат 60×84/16. Бумага писчая «Xerox office».
Ризография. Гарнитура «Times».
Усл. печ. л. 1,39. Уч.-изд. л. 1,12. Тираж 80 экз. Заказ 431.

Издатель и полиграфическое исполнение: учреждение образования
«Белорусский государственный медицинский университет».
Свидетельство о государственной регистрации издателя, изготовителя,
распространителя печатных изданий № 1/187 от 18.02.2014.
Ул. Ленинградская, 6, 220006, Минск.

ISBN 978-985-21-0639-9