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**NON-CARIOUS TOOTH LESIONS  
DEVELOPING  
AFTER ERUPTION**

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

**Е. А. Мирная, Л. Г. Борисенко, Т. Н. Манак**

**БОЛЕЗНИ ЗУБОВ НЕКАРИОЗНОГО  
ПРОИСХОЖДЕНИЯ ПОСЛЕ  
ИХ ПРОРЕЗЫВАНИЯ**

**NON-CARIOUS TOOTH LESIONS  
DEVELOPING AFTER ERUPTION**

Учебно-методическое пособие



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## MOTIVATIONAL CHARACTERISTICS OF THE THEME

**Total time:** 65–70 minutes.

According to the International classification of dental diseases (ICD-DA, WHO1994) non-carious diseases of hard tooth tissues after teeth eruption are listed in chapter K03 “Other diseases of hard tooth tissues”. A huge number of epidemiological researches have revealed that the prevalence of these diseases has increased significantly and it is up to 72.9 % nowadays. Attrition, abrasion, abfraction and erosion are widely-spread. There is a common term for such group of diseases — the tooth surface lost diseases (TSL).

Similar clinical features of these diseases make significant difficulties for their differentiation. On examining patients it is very important not only to identify the nosological forms but to distinguish them from caries lesions. Analyzing patients’ life and diseases history, medical conclusions of other specialists (therapist, endocrinologist, gynecologist, geneticist), previous treatment methods and their effectiveness are very important for the objective understanding of a disease. The usage of additional diagnostic methods allows a dentist to discover the etiology and pathogenesis of patient’s disease and make up the plan of treatment more rationally. The treatment includes methods of teeth color correction, therapy of teeth sensitivity, remineralization, removing risk factors and supporting therapy.

### **The objectives of the seminar:**

1. Methodological — to integrate knowledge about etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention of non-carious tooth lesions developing after eruption.

2. Didactical — to motivate students to understand the importance of diagnosing and choosing the treatment tactics of non-carious tooth lesions developing after eruption.

3. Scientific — to teach students scientifically-based clinical thinking at the time of diagnosis, treatment and prevention of non-carious tooth lesions developing after eruption.

### **Seminar tasks:**

1. To study the fragments of International classification, including chapter K03 “Other diseases of hard tooth tissues”.

2. To study the etiology, clinical manifestations, diagnosis, methods of treatment and prevention of tooth attrition

3. To study the etiology, clinical manifestations, diagnosis, methods of treatment and prevention of tooth abrasion

4. To study the etiology, clinical manifestations, diagnosis, methods of treatment and prevention of tooth erosion.

5. . To study the etiology, clinical manifestations, diagnosis, methods of treatment and prevention of tooth discoloration.

6. To study the etiology, clinical manifestations, diagnosis, methods of treatment and prevention of tooth sensitivity.

### **Requirements for initial level of student's knowledge:**

1. Classification of non-carious tooth lesions developing after eruption (ICD-DA, WHO, 1994; V. K. Patrikeev, 1968)
2. Enamel: anatomy, histology, chemical composition.
3. Dentin: anatomy, histology, chemical composition.
4. Clinical manifestations, diagnosis of dental caries.
5. Dental plaque: structure, properties, its role in the pathology of the oral cavity.
6. Saliva: physical and chemical composition.

### **Test questions:**

1. Classification of non-carious tooth lesions developing after eruption: chapter K03 "Other diseases of hard tooth tissues".
2. Attrition of teeth (K03.0): etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention.
3. Abrasion of teeth (K03.1): etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention.
4. Tooth erosion (K03.2): etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention.
5. Discoloration of teeth (K03.7): etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention.
6. Sensitivity of teeth (K03.8): etiology, pathogenesis, clinical manifestations, diagnosis, treatment and prevention.
7. Treatment methods: vital and non-vital tooth whitening techniques, enamel remineralization, therapy of tooth sensitivity, activities toward the elimination of risk factors.

## **CLASSIFICATION OF NON-CARIOUS TOOTH LESIONS DEVELOPING AFTER ERUPTION**

V. K. Patrikeev (1968) divided numerous non-carious lesions into two basic groups: 1) dental diseases, appearing in the period of their tissues development before eruption, 2) dental diseases appearing after eruption.

In the International classification (ICD-DA, WHO, 1994) the diseases of non-carious 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 teeth 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, 1994) are presented in Appendix 1.

## TOOTH ATTRITION (K03.0)

**Attrition of a tooth** — a progressive loss of teeth surface — can lead to the impairment of chewing function and other side effects (Appendix 4).

K03.00 Occlusal wear of teeth

K03.01 Approximal wear of teeth

K03.08 Other specified teeth attrition.

K03.09 Unspecified teeth attrition

There are different reasons that cause attrition. They include factors of diet, bite, saliva properties, amount of extirpated teeth and the quality of treatment. It has been proved that abrasive food factors, hyposalivation and xerostomia contribute to the progression of attrition.

The diagnosis should include both basic and additional methods. A well-analyzed history of patient's life and diseases is very important. The progression of the disease is accompanied by dentine sensitivity, changes in the vertical dimension of occlusion and in the temporomandibular joint. Oral cavity examination reveals the loss of teeth surface. Sometimes it is unequal, with greater manifestation on the surfaces prone to occlusion trauma. As a result sharp edges of teeth can injure the oral mucosa.

The following clinical forms of tooth attrition can be identified:

- *horizontal* — loss of tissues in horizontal plane (the most common);
- *vertical* — loss of tissues on approximal surfaces;
- *mixed* — tooth tissues loss to the same extent either vertically or horizontally.

Three levels of attrition can be identified:

- *I* — attrition within the enamel without dentine exposure;
- *II* — complete attrition of chewing cusps/cutting edge within dentine;
- *III* — tissue attrition to the level of pulp chamber which is filled with replacing dentine.

*Anatomic pathology.* Long crystals of hydroxyl apatite with a decreased sharpness of the outline are found in the enamel. Interprismatic spaces are densely filled with crystals. Micro hardness of dentine in attrition zone is increased. Dentine tubules and their branches are completely occluded by crystals of different structure. The increase of width and density of hypermineralized zone of peritubular dentine is observed. Deep pulp impairment can be observed as well. Odontoblasts chaotic location, their vacuolization, pyknosis of nuclei as well as reticular atrophy, moderate sclerosis of vessels and the hyalinization of basic pulp substance can be noticed on the initial stages. As the disease progresses, fibroid pulp regeneration and its local and diffuse petrification can appear.

*The differentiation* from tooth caries, erosion and abrasion must be made. *The treatment* is complex; all possible causative factors should be taken into consideration. For example, if the factor is food or habits, the recommendation about healthy diet and life style will provide the stabilization of process.

## TOOTH ABRASION (K03.1)

**Abrasion of a tooth** is a progressive loss of tooth surface due to overexposure by different items and substances (Appendix 4). The abrasion of teeth is revealed more often than any other TSL diseases. The prevalence and intensity of the disease has the tendency to rise with age. The prevalence in Belarus up to 50.6 % among people aged 45–54.

K03.10 Dentifrice abrasion

K03.11 Habitual abrasion

K03.12 Occupational abrasion

K03.13 Traditional and ritual abrasion

K03.18 Other specified tooth abrasion

K03.19 Unspecified tooth abrasion

Generally the *etiology* and pathogenesis of this disease are still under investigation. Mechanical and chemical theories of origin have been identified as the basic ones. It is considered that abrasion can result from wrong and harmful teeth brushing technique (horizontal movement of brush), usage of high abrasive toothpaste or toothbrush. Also it could be due to bad habits (smoking pipe), occupational traumas by items which accompany professional activity (carpenter, musician). Tooth abrasion was discovered among people of Asia and India, who clean their teeth with wooden sticks and coal. There were ritual abrasions found in some nationalities. Some scientists have determined the role of endocrine, nervous and digestive systems.

*Oral cavity examination* reveals the loss of teeth surface as V-shaped defect. The depth of this defect is more than the width. It is usually located in cervical part of tooth crown. The walls of the defect are smooth, bright, and hard. It isn't colored by a staining agent.

*Anatomic Pathology*: high density enamel is revealed. Interprismatic spaces with the loss of sharpness of hydroxyl apatite crystals borders are significantly narrowed. The obliteration of dentine tubules in the ground substance of dentine is noticed. On the surface dentine tubules practically are not identified. Peritubular dentine has more precise structure distant from the defect places but its high density is defined as well. Atrophic changes of pulp are noticed.

*The differentiation* from dental caries, erosion and abrasion of teeth should be carried out. *The treatment* is complex and depends on the defect depth. Sometimes fillings or prosthetic restoration are needed. *Prevention* includes motivation to create healthy daily habits closely related to dental health; advice on methods of oral hygiene should be given.

## TOOTH EROSION (K03.2)

**Erosion of a tooth (K03.2)** is a progressive loss of tooth enamel and dentine that results from their dissolution by acid and further mechanical removal (Appendix 4).

K03.20 Occupational erosion

K03.21 Erosion due to persistent regurgitation and vomiting

K03.22 Erosion due to diet (food)

K03.23 Erosion due to drugs and medicaments

K03.24 Idiopathic erosion

K03.28 Other unspecified teeth erosion

The prevalence of teeth erosion in the Republic of Belarus varies from 2.4 % at the age of 25–34 years till 5.7 % at the age of 45–54 years (N. A. Uhdina, 2001).

*Etiological* factors of erosion are the influence of acid and attrition. Acid appears in oral cavity by different ways: due to industrial acids fumes entering, excessive consumption of sour fruit juices, sport drinks, lemonade, yogurt and other foods containing acid (ascorbic, acetic). It is proved that eating citrus fruits more than twice a day increases the risk of erosion 30–40 times as much. It is noticed that among professional wine tasters with long work experience the prevalence of teeth erosion is very high. Incorrect application of different medical preparations also can cause erosions. Frequent vomiting due to bulimia, pregnancy, or alcoholism leads to erosive changes on the palatine surface of teeth. Normally acid can be neutralized by saliva. However hyposalivation and xerostomia cause the increase of intensity of teeth erosion.

*Oral cavity examination* reveals the loss of a thin layer of tooth enamel at the early stage of erosion. Then any mechanical factors contribute to the attrition of tooth surface. Erosion is developing as an oval or round enamel defect. It is located transversally on the most bulging part of vestibular surface of tooth crown. Erosion floor is bright, smooth and dense. The process of deepening and widening of erosion leads to the loss of the whole enamel of vestibular tooth surface and a part of dentine. The eroded tooth surface usually has the natural tooth tissue color. However in case of bad oral hygiene the erosion surface is colored by food pigments becoming yellow and light-brown.

Two variants of erosion course can be identified: active and chronic. However, generally enamel and dentine erosion has a chronic course. There are 3 stages of erosion according to the depth of lesion: stage I or initial — the impairment of enamel surface, stage II or moderate — the impairment of the whole layer of enamel up to enamel-dentine junction, stage III or deep — surface dentine layers are involved in the erosion.

*Anatomic pathology.* In places of erosion the enamel and dentine have ultrastructure which is typical for demineralization. Striae of Retzius are clearly marked. A transparent zone appears in dentine. The zone of interglobular dentine is not expressed; dentine tubules are a bit obliterated. The tooth cavity is partially or completely filled with tertiary dentine.

*The differentiation* of erosions located on the vestibular surface of upper frontal teeth from excessive attrition and abrasion presents no difficulty. The localization and shape of the defect as well patient's history data are taken into consideration. It should be noted that the erosion of posterior teeth is limited



by buccal or occlusal surface and it has a concave shape. Damaged surfaces of antagonists affected by abrasion adjust each other precisely. The differentiation of erosion from multiple caries can present some difficulties, especially if the abrasion process in erosion is slowing down and there are a lot of demineralized enamel zones around eroded parts. But in case of multiple caries the pathological process develops deep inside, and in case of erosion it extends peripherally (Appendix 2). Sometimes the erosion of enamel is called necrosis. But this kind pathology doesn't exist in the International classification.

*The elimination of causative factors* plays the main role in *the treatment of erosion*. Brushing teeth must not be stopped even if there is an evident sensitivity of teeth. The remineralization measures are carried out. Filling or prosthetic treatment can be done.

*Prevention:* to exclude professional hazards, to stabilize systemic diseases (under the supervision of a general practitioner), to exclude the excessive consumption of sour fruit juices, soda, lemonade, yogurts and other food products containing acids (ascorbic, acetic).

### **TOOTH DISCOLORATION AFTER ERUPTION (K03.7)**

Chapter K03.7 includes diseases which are accompanied by extrinsic, intrinsic and internalized staining of teeth (Appendix 4).

K03.70 Metallic staining.

K03.71 Discoloration due to pulp bleeding.

K03.72 Discoloration due to chewing tobacco and herbs.

K03.78 Other changes of tooth color.

The reasons of tooth discoloration are variable (Appendix 3). The researchers have defined extrinsic, intrinsic and internalized staining of teeth. Extrinsic staining results from local influence of a range of factors on tooth tissues and in this case chromogens are located on the tooth surface. Extrinsic staining is classified according to its origin. At the same time it can be divided into two categories: direct and indirect.

Direct external staining results when chromogens penetrate pellicle/dental plaque. Pellicle like a sponge can absorb and hold liquids and stains. It is established that polyphenol combinations are responsible for the color of spots. Direct-staining chromogens are found in many food products (tea, coffee, spices, soy sauce, mustard, tomatoes, blueberries, red wine, chocolate) in smoking and chewing tobacco and in a number of medicines.

Yellow-brown/black staining of teeth, mostly in cervical area of lingual tooth surface, appears when tobacco is smoked. Clearly marked rings of stain around the necks of teeth, immediately at the gingival edge, appear on the teeth of marijuana smokers. While chewing tobacco, the stain penetrates the micro cracks of enamel causing a significant staining.

Green/orange teeth discoloration can appear in children who have poor oral hygiene in combination with chromogens of microorganisms present in the mouth.

There is an opinion that the reason of green color of plaque is chlorophyll, released by bacteria.

Indirect external staining appears due to chemical reactions on the tooth surface. Indirect staining in dentistry is connected with antiseptics and salt of polyvalent metals, which are either colorless or give a color as a result of chemical reactions. The mechanism of staining by metal salt was actively discussed. The results of laboratory and clinical investigations have determined that discoloration can be due the precipitation of chromogen anions such as polyphenols from diet or medicines on the teeth surface.

Examples: black discoloration of teeth in patients who take iron-containing medicine or work in iron foundry (staining by metals as manganese, iron, nickel). Bromide and iodine fumes produce a yellowish discoloration on cervical areas of teeth. Green discoloration of teeth is observed after using a mouthwash containing copper salts; potassium permanganate produces purple-black discoloration. The long-lasting application of chlorhexidine, hexetidine, chloride cetylpyridinium causes brown-black staining seen around labial and lingual surfaces of frontal teeth after 7–10 days of application.

Intrinsic staining results from systematic influence of chromogens on a human organism. Chromogens are located in dental tissues, usually dentine. Intrinsic discoloration can result from endodontic diseases. Pulpal hemorrhagic products following trauma are the reasons of tooth discoloration. Molecules of hemoglobin are accumulated in the traumatized tooth. Ions of iron combine with oxygen and create ferrous oxide. Further they can combine with sulfur creating dark-grey ferric sulfide.

Internalized staining is known as a separate category. External chromogens penetrate dental hard tissues through the defects in tooth structure. The penetration of an external staining agent into porous tooth structure with inherited developmental defects often discolors the tooth which has already been colored. Some defects caused by functioning and malfunctioning, caries as well as restorative materials defects also can result in tooth discoloration:

- Attrition, abrasion, erosion. Damage of enamel and dentine due to these diseases can result in exposure of dentine to extrinsic chromogens. The exposure of dentine or the loss of enamel gives rise to darker looking teeth as more of the yellow color of dentine is visible.

- Gingival recession. Gingival recession and exposure of dentine makes teeth more sensitive to the external stain internalization.

- Dentinal caries. The progression of the carious lesion is usually associated with changes in color, ranging from the initial white spot lesion to the black arrested lesion that picks up stain from an extrinsic source.

- Defects of restorative materials. It is established that grey-black discoloration seen around old amalgam restorations is caused by the migration of tin into the dentinal tubules.

– Defects of endodontic treatment. Eugenol can give the tooth an orange-yellow stain. Silver points in root canals create grey/black appearance to endodontically treated teeth after corrosion.

It should be noted that tooth color changes with age. The enamel becomes thinner and loses its transparency; vestibular surfaces of frontal teeth become flatter. A combination of thinned non-transparent enamel layer and opaque dentine creates the color of “old-aged teeth”.

*The treatment* of diseases accompanied by teeth discoloration is complex. It includes the correction of oral hygiene, whitening and restoration.

*Prevention:* to exclude bad habits and factors causing teeth discoloration.

### **DENTINE SENSITIVITY (K03.80)**

Dentine sensitivity is disease which is accompanied by an evident painful reaction of teeth after all types of stimuli. They are allocated in separate nosological form and demand an appropriate treatment.

The prevalence of dentine sensitivity is about 89.9–92.8 % among people with non-carious diseases and 25–27 % among patients with periodontal disease. Hydrodynamic theory of dentine sensitivity is considered to be the most common. According to this theory, the movement of fluid in dentine tubules leads to the narrowing and widening of odontoblasts, some of them move into dentine tubules. This process leads to nervous fibers irritation. However, the mechanism remains under investigation.

Dentine sensitivity is classified by localization, origin and clinical course:

1. Localization: local or generalized

2. Origin:

– dentine sensitivity associated with the loss of hard tooth tissues (inside of carious cavity, after tooth preparation for crowns, due to attrition, abrasion or erosion of a tooth);

– dentine sensitivity not connected with the loss of hard tooth tissues (in case of losing the level of epithelial attachment or idiopathic, accompanying general diseases).

3. Clinical course:

– stage I: dental tissues react to temperature (cold, heat) stimuli; electroodontodiagnostic data are 5–8 mkA (physiological rate);

– stage II: dental tissues react to temperature (cold, heat) and chemical (salted, sweet, sour, bitter food) stimuli; electroodontodiagnostic data are 3–5 mkA;

– stage III: dental tissues react to all kinds of stimuli; electroodontodiagnostic data are up to 2.5 mkA.

*The treatment* depends on dentine sensitivity origin. It includes conservative and prosthetic methods: the use of desensitizers, remineralization, fillings and restorations. After tooth preparation for prosthetic crown it is strongly recommended to make temporary crown to prevent dentine sensitivity or to protect teeth applying desensitizers. *The prevention* is directed to the elimination of risk factors.

## TREATMENT OF DENTAL DISEASES OF NON-CARIOUS ORIGIN

Treatment measures include the methods of discoloration correction, treatment of teeth sensitivity, remineralization measures, 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.

*Vital tooth bleaching* — a bleaching agent is applied on the vestibular surface of vital teeth. *External bleaching* — a bleaching substance is applied on the vestibular surface of both vital and devitalized tooth. *Internal bleaching* — a bleaching agent is introduced into a tooth cavity in bleaching devitalized teeth. *Combined bleaching* is a combination of external and internal bleaching.

There are office and home bleaching techniques. A combination of office and home bleaching — *mixed bleaching* — has been proposed. A doctor decides what method is the best in each clinical case, thus providing a *controlled bleaching*.

Nevertheless hydrogen dioxide gels on polyethylene stripes for application on vestibular surface can be easily bought at a chemist's shop. Disposable trays which are filled with 9 % hydrogen dioxide with gingiva protection inside are also available. A range of products of carbamide peroxide of different concentration are sold without doctor's prescription and are used in the same way as bleaching stripes. The listed methods refer to *uncontrolled bleaching*.

Vital, external, internal and combined bleaching can be referred as professional methods. The agents with high concentration of hydrogen dioxide (15–38 %) or carbamide peroxide are applied. To activate bleaching agents, laser, ozone, plasma or ultraviolet light source, hot water or a heated instrument can be used. As dentists use high concentrations of bleaching agents, systems protecting surrounding soft tissues have been designed.

Many patients prefer home bleaching which is more economical and provides as good results as office bleaching does. Low concentrated agents of hydrogen dioxide (3–6 %) or carbamide peroxide (10–12 %) in the form of sticky gel can be used in personal or standard trays. Systems for home bleaching are divided into those that are used according to the doctor's prescription and systems of uncontrolled customer's using.

“Waiting room” bleaching is a quite popular method. At first 35 % carbamide peroxide is activated by heating the syringe under hot running water during several minutes before application. Then gel is put in the tray and it is inserted in

the mouth. The remaining material is removed, and the patient is ushered to the waiting room. After 30–45 minutes the patient is invited back to the consulting room, the trays are taken off; the bleaching gel is removed by vacuum and washed off. The procedure can be repeated twice or three times in one visit. In some cases bleaching is conducted in several visits to achieve a desirable result. “Waiting room” bleaching is recommended to the patients who need a quick effect as well as to those who need a “quick start” before home bleaching.

In a comprehensive dental treatment it is very important to define the place and role of tooth bleaching. It is especially necessary when several specialists take part in the treatment. On the base of numerous investigations some practical recommendations have been suggested:

1. Bleaching is carried out before an esthetical restoration as well as before esthetical prosthetic treatment.

2. As the teeth color after bleaching can be significantly lighter than B1, it is necessary to have lighter composites which are manufactured by companies especially for teeth after bleaching.

3. If a filling has a failure of marginal adhesion, it is necessary to isolate its edges by blocking material for the period of tooth bleaching.

4. In the presence of decay cavity it is necessary to choose one of three ways of preparation for bleaching: 1) to fill in cavities with temporary fillings; 2) to fill in cavities with permanent fillings the color of which should match the predictable result of bleaching; 3) to isolate small decayed cavities by blocking material for the period of teeth bleaching.

5. While bleaching teeth in patient with gingival recession it is necessary to isolate open parts of teeth roost. Such patients are advised to undergo office bleaching procedures.

6. Temperature teeth sensitivity is a common side effect. It is noticed by patients on the third day of home bleaching and can persist for 3–4 hours after tray removal. In this case it is strongly recommended to stop the bleaching and carry out the treatment of sensitivity.

7. The tenderness of gingiva can be caused by pressure exerted by trays during home bleaching and extra amount of bleaching agent. In this case trays are cut and applied again. A patient is instructed to use an optimal amount of bleaching agent.

8. Between any restoration treatment and bleaching procedures is necessary to keep an interval of 1–3 weeks for tooth color stabilization and deoxidizing of hard tissues.

9. During the first 7–10 days after the course of bleaching patients should avoid food products containing colorants and avoid smoking.

10. The teeth of patients with small pulp chamber and discoloration caused by food chromogens are bleached successfully.

11. Bleaching procedure gives good results in teenagers with yellow teeth.

12. White spots due to fluorosis have a tendency not to respond to bleaching, but they become less visible as a result of lightening of surrounding tooth tissue.

It is hard to bleach severe tetracycline discoloration, but insignificant or moderate discoloration can respond to long-lasting bleaching within 3–6 months. In some cases a non-bleached part of tooth is covered with a restoration material.

13. Some researchers informed about the improvement of gingiva condition after bleaching procedure. It is established that bleaching substance is toxic to microbes. At the same time the patients who bleach their teeth pay more attention to the teeth condition, so they improve oral hygiene during the treatment.

14. To preserve the effect of bleaching it is necessary to keep oral hygiene including regular brushing teeth not less than twice a day, daily use of floss, regular visits to a dentist for professional oral hygiene.

15. To consolidate the results whitening tooth pastes are prescribed. The effect of whitening tooth pastes is reached due to:

- abrasive influence of sodium bicarbonate, hydrogenated silicon as well as soap substances helping to improve polishing properties of abrasive materials in tooth pastes;

- chemical dissolving of stained pellicle by proteolytic enzymes (papain);

- lightening of surface pigment (carbamide peroxide);

- masking of stains due to titanium dioxide inclusions;

- antiseptics (triclosan) which slow down tooth deposit.

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.

Remineralization therapy includes the use of fluoride contained varnishes, gels or solutions of sodium fluoride of different concentration (0.05; 0.1; 0.2 %). At the same time, to treat teeth sensitivity desensitizers can be used. They may contain fluorides, potassium nitrate, potassium chloride, strontium chloride, zinc citrate. The mechanism of their activity is based on dentine tubules obturation, blocking fluid movement and the prevention of nerve repolarization.

The obturation of dentine tubules is carried out by binding an active component of desensitizer, e.g. strontium, with protein matrix of dentine so the diameter of tubules will decrease significantly. Potassium chloride and potassium nitrate after their diffusion into dentine tubules inhibit the activity of nerve fibers by the depolarization of their membrane by  $K^+$  ions as repolarization is impossible at their high concentration. Citrate creates complexes with calcium and obturates dentine tubules thus decreasing the flow of fluid. Fluorine ions create insoluble calcium fluoride which is deposited in dentine tubules, decreasing their diameter.

Teeth sensitivity to stimuli can appear while bleaching. Active forms of the problem treatment are the use of fluoride and potassium nitrate gels in trays, worn at night. Another method is to wear trays with potassium nitrate agents and fluoride and potassium nitrate containing gel 2 hours before or after the bleaching

procedure. The effect of using tooth paste for sensitive teeth is seen only after 2–3 weeks.

Passive forms of treatment are: decreasing bleaching gel concentration, decreasing its amount and the time of bleaching. If necessary, trays are cut, less gel is put in, and remains are thoroughly removed. A patient is recommended to apply bleaching material every second or third night. It makes the period of bleaching longer, but provides a comfort regime of the procedure.

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**Fragment of the International Classification of Dental Diseases (ICD-10, WHO)**

- K03.0 Excessive attrition of teeth
  - K03.00 Occlusal wear of teeth
  - K03.01 Approximal wear of teeth
  - K03.08 Other specified teeth attrition
  - K03.09 Unspecified teeth attrition
- K03.1 Abrasion of teeth
  - K03.10 Dentifrice abrasion
  - K03.11 Habitual abrasion
  - K03.12 Occupational abrasion
  - K03.13 Traditional and ritual abrasion
  - K03.18 Other specified teeth abrasion
  - K03.19 Unspecified teeth abrasion
- K03.2 Erosion of teeth
  - K03.20 Occupational erosion
  - K03.21 Erosion due to persistent regurgitation and vomiting
  - K03.22 Erosion due to diet (food)
  - K03.23 Erosion due to drugs and medicaments
  - K03.24 Idiopathic erosion
  - K03.28 Other unspecified teeth erosion
  - K03.29 Unspecified teeth erosion
- K03.3 Pathological resorption of teeth
  - K03.30 External
  - K03.31 Internal (inner granuloma, pink spot)
  - K03.39 Pathological teeth resorption unspecified
- K03.4 Hypercementosis
- K03.5 Ankylosis of teeth
- K03.6 Deposits on teeth
  - K03.60 Pigmentation (black, green, orange).
  - K03.61 Tobacco deposits
  - K03.62 Betel deposits
  - K03.64 Supragingival calculus
  - K03.65 Subgingival calculus
  - K03.66 Tooth debris
  - K03.68 Other tooth debris
- K03.7 Color change of hard tooth tissue after eruption
  - K03.70 Metallic staining.
  - K03.71 Discoloration due to pulp bleeding.
  - K03.72 Discoloration due to chewing tobacco and herbs.
  - K03.78 Other changes of tooth color.
- K03.8 Other specified diseases of hard tooth tissues
  - K03.80 Sensitive dentine
  - K03.81 Irradiated enamel
    - K03.88 Other diseases
  - K03.9 Unspecified diseases of hard tooth tissues








### Diagnosis of Some Dental Diseases

<b>Disease</b>	<b>Defect characteristics</b>	<b>Localization</b>	<b>The number of affected teeth</b>	<b>Sensitivity</b>	<b>Reaction to staining</b>
<b>Erosion</b>	Loss of tissue of oval shape with dense, smooth, shining floor	Usually the vestibular surface; but palatine surface can also be affected	Different, usually several except lower incisors	Yes, typical for active stage	Not stained
<b>Abrasion</b>	V-shape defect with dense smooth walls	Mostly neck of teeth crown	Different, can affect many teeth	Yes / no	Not stained
<b>Caries (chronic)</b>	The shape can be different with dense, uneven, brown floor and uneven edges	In the place of dental plaque accumulation	Mostly single	On probing dentine	Stained
<b>Caries (acute)</b>	The defect of a round shape with soft light floor with festoon edges	In the place of dental plaque accumulation	Mostly single	On probing dentine	Stained
<b>Caries (spot lesion)</b>	Chalky-like with indistinct borders	In the place of dental plaque accumulation	Mostly single	No	Stained

### Tooth Discoloration: Causes, Ways and Colors Produced

Causes and ways of tooth discoloration	Color produced
Extrinsic	
1. Tea, coffee and other foods	Brown to black
2. Cigarettes / tobacco	Yellow / brown to black
3. Dental plaque / poor oral hygiene	Yellow / brown
4. Metal salt and antiseptics (including chlorhexidine bigluconat)	Black and brown
Intrinsic	
1. Metabolic (including genetic erythropoietin porphyria)	Purple / brown
2. Genetic (including the impairment of enamel and dentine development)	Brown / black (in form of stripes and spots)
3. Iatrogenic: – tetracycline; – fluorine (fluorosis)	Yellow, brown, blue, black or grey White, yellow, grey or black
4. Traumatic (blood)	Brown/ reddish (pink spot)
5. Age-depending	Yellow
Internalization	
1. Caries	Orange to brown
2. Restoration	Brown / grey / black / other

<p><b>TOOTH ATTRITION</b></p>		
<p><b>TOOTH ABRASION</b></p>		
<p><b>TOOTH EROSION</b></p>		
<p><b>TOOTH DISCOLO- RATION</b></p>		

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ПОСЛЕ ИХ ПРОРЕЗЫВАНИЯ**

**NON-CARIOUS TOOTH LESIONS DEVELOPING  
AFTER ERUPTION**

Учебно-методическое пособие

На английском языке

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