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Tooth Wear- Fundamental Mechanisms And Diagnosis

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Abstract : *Tooth wear is a complex multifactorial phenomenon caused by the interaction of biological, mechanical, chemical and tribological factors. The surface of the hard dental tissues as well as filling materials replacing enamel and dentin are mechanically loaded by antagonists (attrition) or other object in the oral cavity, e.g. toothbrush, toothpaste etc. (abrasion) as well as can be exposed to acidic environment in oral cavity (erosion). These processes lead to the formation of surface defects of hard dental tissues and fillings. The aim of this paper is to summarise modern information about etiology, mechanism and clinical manifestation of tooth wear basic types and their combination as well as to mention the therapeutical and preventive measurements.*

Key words : *abfraction, abrasion, attrition, erosion, tooth wear*

I. Introduction

As wear in a broad sense refers to subject damage by material loss from the surface during the relative motion of two or more materials, or interaction between the environment and material [1]. In dentistry we encounter the term - hard dental tissues wear and restorative materials wear that replace enamel and dentin. Tooth wear is a complex multifactorial phenomenon conditioned by interactions of biological, mechanical, chemical and tribological factors [2, 3]. The physiological and pathological wear of hard dental tissues is distinguished. The physiological loss of hard dental tissues is characteristic for elderly individuals and it is conditioned by natural wear and tear due to the teeth function. Pathological loss of hard dental structures is a condition in which wear has reached such level that the teeth can not fully perform their function or serious aesthetic problems are present [3]. There are two basic mechanisms of hard dental tissues pathological wear. The first is mechanical wear that includes abfraction, attrition and abrasion. Abfraction means microstructural loss of hard dental tissues due to the load concentration with the typical localization in the cervical area of the tooth [4]. Attrition means hard dental tissue contingent loss by physical damage due to direct contact of the teeth without the participation of other substances [3, 4]. Apart from pathological attrition we also distinguish physiological attrition, which is based on the function of the stomatognathic system and the natural mechanism of tissue aging. Unlike attrition, abrasion means the loss of hard dental tissues contingent by physical damage due to mechanical action of substances from the external environment [3, 4]. From a historical perspective the issue of tooth mechanical wear is not new and has been already occurred in the hunter-gatherers era. [5] Modern available studies examined the prevalence of hard dental tissues mechanical wear. This provides that 3 % of individuals in the aged of 20 years have signs of tooth wear, while older individuals (17 % in the age 70) are characterized as people with increasing index [6].

The second type of hard dental tissues wear is conditioned by their chemical dissolution due to the action of acids of exogenous or endogenous origin (erosion or corrosion) [2, 3]. Unlike mechanical wear of hard dental structures, chemical dissolution is a disease of modern time. Due to anthropological research of hunter-gatherers skeletal remains (Australian Aborigines) as well as the skulls of Native Americans, historic and prehistoric European populations, there is no evidence on the occurrence of dental erosion [7]. Increased exposure of hard dental tissues to acids have been occurred with the coming of agriculture, especially in the Middle Ages, when techniques of food processing via fermentation were developed [7]. But in the modern consumer society there was a significant increase in exposure of hard dental tissues to the action of so-called dietary acids. Currently it is possible to observe the increase in the prevalence of erosive defects, particularly among the young generation [3]. According to some studies, the prevalence of erosive lesions in children or adolescents may reach 50–60 % [3, 8].

The aim of this article is to summarize current data about the etiology, mechanism of development and clinical manifestations of the basic types of hard dental tissues wear, including their combination as well as to describe the diagnostic methods and provide an overview of therapeutic and preventive measurements.

II. Basic Types Of Hard Dental Tissues Wear

2.1. Abfraction

The term abfraction was first introduced by McCoy in 1982 and explains the processes in which eccentric occlusal forces cause compression or tension in the enamel and dentin of the cervical area of the tooth and makes it susceptible to damage by erosive and abrasive factors [8, 9]. In literature these lesions are also called "cervical stress lesions" [8]. As flexion results the weakening of the enamel hydroxyapatite in cervical region, it can cause chipping or breaking of enamel prisms and further microfractures of cement or dentin. The abfraction causes are occlusal barriers, premature contacts, bruxismus, teeth-grinding. The wedge shaped defects are advanced form of abfraction. They are localized in the cervical third of the tooth in the cement-enamel junction [10] and have typical shape of the letter V (Fig. 1). The defects have smooth and shiny walls and patients usually do not have significant subjective complaints. The decisive factor for their creation is the effect of transmitting forces over the occlusal surface to the cervical region in combination with the use of improper tooth brushing and inadequate hygienic aids (toothbrush with hard bristles, toothpastes with high RDA) [4]. The degree of abfraction defect is conditioned by the size, duration, direction, frequency and location of inducing forces [8].

Figure 1 Abfraction (wedge-shaped defects in cervical part of upper teeth)

2.2. Attrition

Attrition is defined as the loss of hard dental tissues contingent by physical damage due to relative mechanical abrasion of teeth in (para)functional contact areas without the participation of other substances (two body wear) [2, 3]. Mostly it occurs on the antagonist teeth in the incisal edges of the anterior teeth and the occlusal surfaces of posterior teeth, where there are defects in the shape of smooth shiny facets bounded by a sharp edge (Fig. 2). Further progression of defects leads to reduction of cusps height and flattening of the occlusal surface that can provide a reduction of tooth crown [8]. Attrition can also occur on the buccal and oral surfaces, particularly at the malocclusion, as well as on the proximal surfaces - in areas of contact points, where during the life due to wear processes gradual loss of hard dental tissues as well as flattening of contact points occurs (physiological attrition) [4]. Attrition can be reinforced by abrasive food components [11]. Attrition rate is also affected by parafunctions (e.g. bruxismus), TMJ function as well as load distribution in the residual teeth (pathological attrition) [4]. In the advanced stage the bite height reduction, severe morphological and functional changes of stomatognathic system as well as dentin hypersensitivity can be occured.

Figure 2 Attrition on upper and lower antagonist teeth

2.3. Abrasion

Abrasion is the loss of hard dental tissues contingent by physical damage due to mechanical action of substances or objects from the external environment (e.g. food, toothbrushes, toothpaste etc.) (three body wear) [3]. Both localization and appearance of abrasive defects are conditioned by the type of causing object or substance.

Causing factors of abrasion are:

• abrasive effect of food

• traumatizing technique of oral hygiene (toothbrushing using traumatic technique with a predominance of horizontal movements using high pressure, using a toothbrush with hard bristles, inconsiderate use of floss or toothpicks, abrasive effect of toothpastes).

• some bad habits- biting of nails, tacks, clips, threads, as well as holding of different objects in the teeth (pencils, pens, pipes) that can be related to some professions (musicians playing wind instruments, dressmakers, glass blowers, etc.) [2 3, 8]. The most typical localization of abrasive defects in this case is the upper central incisor, where the notch on the incisal edge can be seen.

• iatrogenic factors like nonarticulated fillings, crowns, dentures clips

• peculiarities of the environment (mines, quarries, deserts), where the sand particles and other substances can get into the oral cavity

According to the depth of abrasive defect we can distinguish [9]:

• enamel abrasion

• enamel and dentin abrasion

• abrasion of whole dental crown with the risk of the dental pulp exposing

Abrasive defects are localized on the incisal edges of the anterior teeth and the occlusal surfaces of the posterior teeth as well as in the cervical area of the teeth (Fig. 3). Clinical manifestations are similar to attrition. The term abrasion can also include the fatigue wear. This is a process where one object exerts a force on the surface of second one with simultaneous linear movement. This creates a compression zone in front of the tip of the first object in the direction of its movement and due to plastic deformation the tension zone will be formed behind it. In cyclic repetition subsurface cracks are formed that may subsequently occur on the surface like a material loss. Additionally, loose particles can also act as third wear component [12, 13].

Figure 3 Advanced abrasion on upper teeth

2.4. Erosion (corrosion)

Unlike the above-described types of wear that are conditioned by physical causes the erosion is the chemical damage of tooth conditioned by loss of hard dental tissues due to the action of acids on the tooth surface without plaque and microorganisms [2, 3, 14]. The acids causing the loss of hard dental structures can have exogenous or endogenous origin. Exogenous acids are contained in foodstuffs and drinks, sport and energy drinks, certain medications, environment and employment. Endogenous acids are gastric acids that are getting in contact with the oral cavity during an episode of reflux, vomiting or regurgitation. In the oral cavity, these acids dissociate into hydronium cation and anion of acid residue. Hydronium cation reacts with phosphate and carbonate anion of hard dental tissues that leads to its gradual softening in the repeated contact with the tooth surface. Furthermore, chelating agents (anions of some organic acids) may form the complex compounds with calcium of hard dental tissues hydroxyapatite.

Erosive potential of foodstuffs and beverages is conditioned by their chemical properties (pH, buffering capacity, concentration and power of contained acids, saturation by mineral components), but also the physical properties (temperature, ability to adhere to the tooth surface) [3, 11].

Erosive defects according to stages are divided into the initial, advanced and severe [3]. The initial erosive lesions affect only the enamel surface layer and appear as beginning demineralization. There is a loss of

surface relief of enamel, vanish of perikymata. The enamel is smooth and dull. Advanced erosion is characterized by extensive loss of enamel. In the cervical region the loss may extend over the dentin. In posterior teeth the reduction in the cusps height, pitted depressions on the occlusal surface as well as protruding polishing shiny amalgam filling can be observed (Fig. 4). Dental erosion in severe stages is characterized by large enamel defects with exposure of dentin. Erosive defects with exposed dentin are usually accompanied with dentin hypersensitivity. Advanced and severe erosive defects are often aesthetic problem for the patient. In the case of bite height loss a number of symptoms of stomatognathic system disfunctions as well as pain of chewing muscles or temporomandibular joint disorders can be often occured.

Figure 4 Erosive tooth wear on palatal and occlusal surfaces of upper teeth

2.5. Multifactorial character of tooth wear

The above-described mechanisms of hard dental tissues wear is very rarely act in isolation. Mostly combined lesions occur. Multifactorial character of wear includes three basic mechanisms such as friction, chemical influences and the load due to compression, flexion and tension. Various types of hard dental structures wear are the result of isolated or combined action of causing factors.

Erosion and abfraction combination is characterized by a loss of hard dental tissues during synergistic effect of erosive chemical substances at places of load concentration.

Erosion and abfraction combination is described as a loss of hard dental tissues when erosive substances act on places with attrition wear [4]. These defects can be observed in patients with gastroesophageal reflux disease or frequent vomiting (bulimia mentalis or anorexia nervosa) [3].

Combined erosive-abrasive lesions are the most common type of hard dental tissues noncarious lesions. They are formed by the synergistic action of erosive agents and abrasive factors. It is proven that the enamel disturbed by action of acids has greater susceptibility to abrasion and attrition compared to intact enamel [15]. This condition can occur during the teeth brushing, their enamel has been demineralised due to erosive attack. In this case the erosive-abrasive additional effect is observed. It can be characterized by almost 50% increase influence of abrasive factors with the simultaneous action of acidic substances [16]. The in vitro studies revealed that the action of erosive agents reduces the hardness of hard dental tissues in proportion to the time over which acid acts on the tooth. Attin et al. (1997) in their experiment established that at 15 min exposure carbonated beverage with pH 2.9 Vickers hardness of the bovine enamel samples was reduced from 311 to 215 MPa [17]. The simultaneous action of abrasive factors (effect of a toothbrush with a specified force and number of movements and experimental toothpaste) showed significant loss of hard dental tissues (up to 2 μm) compared with the samples that before brushing were not exposed to erosive substances (in this case the loss of hard dental tissues after exposure to abrasive factors was just 0.17 μm). Jaeggi and Lussi also found that eroded enamel loss after tooth brushing is 10 times larger than the intact enamel one [15]. Similarly, the abrasive-erosive additional effect impacts on restorative materials.

III. Compensated and protective mechanisms during the loss of hard dental tissues

During the hard dental tissues wear several protective mechanisms are present. It is primarily the formation of reparative dentin, which may lead to reduction or complete obliteration of the pulp chamber and root canals. Saliva is generally an important protective factor in the oral cavity. Its protection mechanisms include dilution, purification, buffering capacity, and neutralization of the acids causing erosion. Saliva also reduces the demineralization and promotes the worn hard dental tissues remineralization by containing of calcium, phosphate and fluoride ions [3]. The protective effect of saliva is manifested against the penetration of acids into the oral cavity by increasing of saliva production in response to external stimuli, such as the appearance and smell of food [3]. Important role in the dental erosion prevention play saliva mineral components. They are carbonic acid, hydrogen carbonate, dihydrogen phosphate, hydrogen phosphate, calcium and fluoride. Buffering capacity of saliva has the ability to neutralize the low pH in the oral cavity resulting after acidic substances consumption. Besides the saliva composition its amount is also very important. Inadequate amount of saliva (hyposalivation) and xerostomia is a major risk factor for both noncarious and carious defects.

Dental pellicle is another factor that protects the tooth structure against wear. This is a protein layer which is rapidly formed on the tooth surface after the cleaning with a toothbrush or chemical means as well as after a professional cleaning. Pellicle acts as a lubricant between the antagonist teeth and thus protects their surface against abrasion and attrition [3]. Dental pellicle also protects the tooth surface against erosion due phosphoprotein and mucins content as well as properties of diffusion barrier preventing direct contact between acids and the tooth surface and reducing the dissolution of hard dental tissues hydroxyapatite. The presence of salivary enzyme carbonic anhydrase VI prevents dental erosion by accelerating of hydrogen ions neutralization on the tooth surface. Moreover pellicle serves as a reservoir of electrolytes participating in the process of worn hard tissues remineralization [3].

IV. Examinattion of the patient with hard dental tissues wear

The key to successful treatment of patients with wear of hard dental tissues is a careful examination, determination of etiological factors and a thorough understanding of the basic principles of occlusion as well as physical and chemical properties of the currently available materials and techniques of treatment [8].

The first step of hard dental tissues wear correct diagnosis is a thorough history, aiming to identify the possible causes of the defects. The dentist must be very aware of all possible etiological factors and ask them for patients. The present general disease can be the direct cause of tooth wear or influences on its formation through therapeutic agents used. Anatomical defects of the esophagus, gastroesophageal reflux disease, gastroesophageal cancer, conditions with frequent vomiting (eating disorders, rumination, diabetes mellitus, peptic ulcer, uremia, etc.) can cause erosive defects of hard dental tissues. Vomiting like one of the dental erosion factor can be also observed in pregnant women (especially in the first trimester), in patients receiving chemotherapy or cytostatic therapy, in chronic alcoholism or severe chronic stress. Autoimmune diseases (especially Sjögren's syndrome), diabetes, diseases of salivary glands, chronic renal failure, infectious diseases (HIV, AIDS, hepatitis C), radiotherapy of the head and neck, regular use of certain medicines (psychotropic drugs, anticholinergics/antispasmodics, anorexiants, antihistamines, antihypertensives, diuretics, medicines for smoking quitting, etc.) can lead to hyposalivation or xerostomia, that disrupted self cleaning and remineralization ability of the oral cavity. These patients show an increased incidence of dental caries or erosion. Other remedies, such as iron preparations and preparations containing hydrochloric acid, an asthma medication, aspirin, vitamin C (ascorbic acid), various food supplements in the form of soluble tablets have a low pH and therefore induce an acidic environment in the oral cavity, which on repeated action may lead to dental erosion [3].

Social history is also very important, because it can detect other possible causes of hard dental tissues loss, such as exposure to erosive (workers of chemical and metallurgical factories, professional swimmers, wine tasters) or abrasive factors in employment and the environment (residents of mines, quarries, deserts, dressmakers, musicians playing wind instruments, glass blowers, etc.). Certain habits (smoking, frequent alcohol consumption, dietary habits, regular sports) must also be thoroughly investigated. Improper dietary habits are one of the most common factors of erosive lesions. For their detection the patient’s daily diet should be described. It should include the frequency and amount of consumed foodstuffs and beverages, time and manner of their consumption (retention in the mouth, quick swallowing or thorough chewing, drinking through straws etc.) for at least three consecutive days [8]. Frequent use of alcohol may be associated with frequent vomiting that decreases pH in the oral cavity, so it can cause erosive-abrasive damage to the hard dental tissues. Regular sports are related to dehydration during exercise as well as the consumption of sport or energy drink with an acidic pH. Their regular consumption can therefore lead to erosive lesions.

Dental history is also really important. It focuses on the presence of various parafunctions in stomatognathic system (bruxism or bruxomania), previous dental treatment quality, orthodontic braces presence and etc. Finally, it is oral hygiene implementation, and in particular toothbrushing method and frequency, toothpaste and toothbrush type used, interdental hygiene, mouthwashes and means for local fluoridation, regular use of sugar-free chewing gum etc.

A thorough history is followed by the careful extraoral and intraoral examination of the patient. An extraoral examination is primarily focused on the status and function of the temporomandibular joint (pain, crepitation, click, deviation of the mandible during the mouth opening or closing, maximal opening of the mouth) and the adjacent muscles (hypertrophy, tone and tenderness of the chewing muscles), presence of enlarged parotid gland that is typical for patients with frequent vomiting, signs of chronic alcoholism (red, swollen face, spider nevus on the skin, typical alcohol foetor ex ore, as well as the detection of face vertical proportions consisting of assessment of freeway space, rest vertical dimension and occlusal vertical dimension [8]. The measurement can be performed using special calipers, by the evaluation of certain sounds pronunciation (especially sibilants), an analysis of face soft tissue contours as well as using techniques of muscle electrical stimulation and others [8].

Intraoral examination should include a careful assessment of soft tissues status (possible presence of buccal mucosa keratosis, tongue changes or symptoms of xerostomia), oral hygiene level determining as well as basic examination of periodontal tissues. Consequently a thorough examination of teeth must be provided. It includes the record of present and missing teeth, tooth decay, reconstructions, fractures, abrasive or erosive lesions and the number of affected teeth (localized, generalized wear), localization and stage of these defects (incipient lesions in enamel, advanced lesions with dentin exposure, serious lesions with significant loss of hard dental tissues), that can be evaluated using different indices.

In addition, it is necessary to conduct a comprehensive examination of the patient's occlusion involving the examination of tooth position in the dental arch, noting the possible crowding, rotation, inclination, wandering, spacing, supernumerary teeth, looseness, bite type etc. [8]. It is also very important to determine a stable central occlusion and central jaws relation as well as contact points of teeth in maximum intercuspation position. The teeth contacts during lateral excursion (canine guidance) and protrusive movement of the lower jaw, as well as the description of all occlusal interference on the "working" and "non-working" side must be carefully examined and recorded.

The additional examination of patients with hard dental tissues wear includes radiographic examination focusing primarily on determination of the distance between the remaining hard dental tissues and dental pulp, anatomy of the pulp chamber, anatomy and morphology of the root system, the condition of the bone tissue, the quality of the previous endodontic treatment, state of periapical tissues, signs of tooth decay, extension or breach of lamina dura. These tests also include the testing of dental pulp vitality, saliva testing (determination of the amount of unstimulated and stimulated saliva, saliva pH and buffering capacity), making of situational intraoral photos, study models as well as diagnostic wax mock-ups that can be also used to monitor lesions [3, 8, 18].

V. Management of the patient with hard dental tissues wear

The treatment plan for patients with hard dental tissues wear is based on the same principles as for other diseases of the oral cavity. Above all, it is necessary to relieve the patient of acute problems associated with the pathological condition. These measures include the polishing of sharp cusp or incisal edges of worn teeth, application of desensitizing agent on the surface of exposed dentin or making temporary reconstruction using GIC, as well as the extirpation of dental pulp in case of its baring due to extensive loss of hard dental tissues or sometimes tooth extraction or TMJ changes therapy resulting from parafunctions [8].

The next step is the prevention of further defects formation and progression of existing lesions. It should be noted that the prevention of tooth wear is not the same as prevention of tooth decay. Tooth decay occurs in the majority of people in the world, while tooth wear attacks individual patients, so it can not be regarded as a social problem yet. Even though the prevalence of tooth wear increases, it is very difficult to predict which individuals will exactly affect, so primary prevention is intricately achievable.

The hard dental tissues wear further progression basic preventive measures include:

1. *Strengthening of the enamel structure by application of remineralization means.* This is the regular use of fluoride contained for example in toothpastes, mouthwashes, means for office use (gels, paints, foams), in chewing gums or drink. Their beneficial effect on enamel has been demonstrated in numerous in vitro and in vivo studies [3]. Fluorides promote remineralisation processes in the oral cavity, which makes the enamel more resistant to the influence of adverse factors, particularly acids. Moreover fluoride means are also used in the treatment of dentin hypersensitivity which may accompany advanced stage of hard dental tissues wear. A similar effect has the means containing CPP-ACP (casein phosphopeptide amorphous calcium phosphate-RECALDENT) [3]. It is contained in oral hygiene means as well as in dairy beverages Meiji Milk de RECALDENT or chewing gum Trident. They have one contraindication for their application: the allergic reaction to milk protein.

   Other substance that is beneficial for enamel is xylitol. It is a natural sweetener occurring in fruits, vegetables as well as in the wood of some trees (xylem) [19]. His cariostatic and anti-cariogenic effect is already known for several decades [20]. Xylitol is able to form a complex compounds with calcium ions that can subsequently penetrate into the enamel and prevent loss of calcium and phosphate ions from hard dental tissues hydroxyapatite during demineralization by reduction of the ions diffusion coefficient. Thus, xylitol plays important role in the remineralization of hard dental tissues [21]. Besides this xylitol increases the buffering capacity of saliva, which helps to increase the pH in the oral cavity, and promotes remineralization processes [20]. Xylitol is available in the form of pellets, powder, chewing gum or nasal spray. It is added into sweets including chocolate. It is also part of some mouthwashes, toothpastes and gels [20].
2. *Modification of dietary habits*, as the reduction of the amount and frequency of acidic beverages and foodstuffs consumption as well as the time shortening for acid exposure. It is recommended to drink beverages quickly using straws, to consume cold drink and immediately after consumption of acidic foodstuff or drink to rinse the mouth with water, milk or mouthwash with fluoride [22, 23, 24]. Adverse

effects of acidic foodstuffs and drinks can be reduced by enriching with the mineral components that strengthen hard dental tissues (calcium, phosphates, fluorides). It can be, for example, fruit juices and carbonated soft drinks containing calcium. An important role in the prevention of erosive tooth wear belongs to so-called functional foods, which are generally referred as a safe and beneficial to oral health. These foodstuffs can be labeled as "tooth friendly products" and they have on their package the symbol "Happy Tooth". The functional foodstuffs are mainly dairy foodstuffs with high content of protein and calcium, especially cottage cheese and hard cheese, which is recommended like final foodstuff after the main meal [3]. The chemical substances contained in these foodstuffs help to neutralize the acidic environment formed after the meal. They are effective both in prophylaxis of dental erosion and caries. Another functional component that is beneficial for oral health is already mentioned casein phosphopeptide amorphous calcium phosphate as well as xylitol. Another substances like glycomacropeptide (GMP) or caseinmacropeptid (CMP) containing in some beverages, wafers, biscuits, chocolate, pudding, peanut butter, cereals or gelatin promote remineralization of hard dental tissues (increase of the calcium, iron and zinc absorption) and also reduce plaque formation, thereby protect the tooth surface against dental caries and erosion. Furthermore, other functional components, such as calcium sodium phosphosilicate, polyphosphates, iron, copper, xanthan gum, carboxymethylcellulose reduce the demineralization of hard dental tissues and protect them against erosive damage.

3. *Adjustment of oral hygienic habits.* It includes first of all the recommendations do not brush the teeth immediately after exposure to acids and instead of it to rinse the mouth with water, milk or mouthwash with fluoride and purify the tongue from acid residues. Patients with hard dental tissues wear should use the toothbrush with soft bristles, mouthwashes and toothpastes with low abrasiveness containing special components strengthening hard dental tissues (ions of fluoride, polyvalent metal, chitosan, etc.) as well as toothbrushing technique that is friendly to the teeth. It is advisable to provide daily topical application of fluoride at home dental care, as well as 2–4 times a year at professional care in the dental office. It is also important to support the salivary secretion using chewing gum without sugar with fluoride or carbamide [8], as well to increase its saturation by calcium and phosphate. In the case of xerostomia it is recommended to use special means (e.g. GC Dry Mouth, Xerodent, artificial saliva solutions, etc.). It is also important to get rid of bad habits that cause damaging of hard dental tissues (nails or thread biting, holding of a pen, pipes, pencils in the mouth, etc.).
4. *Desensitisation therapy*- use of home or office application of so-called desensetizers that reduce the dentin hypersensitivity occurring in the advanced stages of hard dental tissues wear due to exposing dentinal tubules. These means according to their mechanism of action are divided into physical (cause the mechanical closure of dentinal tubules entrances) and chemical (cause the precipitation and coagulation of the dentinal tubules). They include the potassium, fluoride, stannous, oxalate ions, arginine, hydroxyapatite, CPP-ACP and calcium sodium phosphosilicate contained in pastes and gels for home and office use [3, 18]. Other methods of dentin hypersensitivity treatment include the application of dentin adhesives and sealant that seal the dentinal tubules entrances and provide some level of hard dental tissues loss protection. They can also contain fluoride ions and antimicrobial substances and reduce the accumulation of plaque on the tooth surface. The disadvantage of this method is the necessity of repeated application of the means.
5. *Making of individual splints.* In the case that the patient with hard dental tissues wear suffers from bruxism, as prevention of further loss of enamel and dentin, it is recommended to use the acrylic splint. Regular wearing of the splint will help to eliminate the contact of teeth and returns the normal function of the muscles and TMJ. Furthermore, for the patients with frequent reflux or vomiting splint may provide the protection against the action of stomach acid. It is necessary to draw the attention of these patients to the need for removal and rinsing of the splint immediately after an episode of reflux or vomiting, as this may cause the detention of acid residues under the splint [8]. The splint can be also used as a reservoir for the application of fluoride or desensitisation means.

It is also important to stabilize other pathologies in oral cavity: treatment of tooth decay, periodontal diseases, pathologies of the oral mucosa, extraction of teeth with unfavorable prognosis. The next step is making final reconstruction and comprehensive rehabilitation of the oral cavity. Filling therapy is generally indicated in the case of hard dental tissues loss to 2 mm in the vertical direction [3]. For larger defects the prosthetic treatment is recommended. Fot the treatment of advanced erosive defects on palatal surfaces of the upper teeth caused by reflux or vomiting metallic veneers can be used [18]. In advanced stages of wear the issue of the bite increasing must be kept in mind. It is very important to plan the treatment correctly. Before reconstruction using the crowns and prosthetic rehabilitation it is sometimes necessary to provide endodontic or periodontal treatment, surgical extension of clinical crown, implementation and fixation of root pins or orthodontic repositioning of the teeth. In the case of extensive involvement of antagonist teeth it is necessary to take into account the possible wear of the materials that were used for reconstruction of the opposite jaw teeth [18].

The final phase of the measurements is the monitoring of lesions progression maintenance of therapy state achieved. The clinical situation photos acquired with a certain frequency (usually with 6–12 months) are commonly used for the above-stated purposes as well as study models (made of stone type plaster using the PVS impression) or silicone sectional indices are also carried out. A more accurate method of monitoring of tooth wear progression is the examinatio of tooth surface changes using special computer software, whose practical use due to the financial cost does not have a significant expansion.

VI. Conclusion

Defects of hard dental tissues contingent by physical or chemical wear are frequently occurring condition. Reconstruction of advanced stages is very demanding and requires a comprehensive, interdisciplinary approach. If these defects are diagnosed at an incipient stage and their etiologic factors are determined and eliminated, their progression can be stopped only by preventive or minimal invasive measures. In the treatment and prevention of defects is important to provide a regular monitoring of existing lesions and the stabilization of other pathologies, such as periodontal diseases, xerostomia or dentin hypersensitivity.

References

[1]. J. Pošta, P. Veselý, and M. Dvořák, Degradace strojních součástí (Prague: ČZU, 2002).
[2]. M.J. Pickles, Tooth wear, in R.M. Duckworth (Ed.), The teeth and their environment. Physical, chemical and biochemical influences (Basel: Karger, 2006) 86-88.
[3]. A. Lussi, C. Ganss, Erosive tooth wear from diagnosis to therapy (Basel: Karger, 2014).
[4]. J.O. Grippo, M. Simring, and S. Schreiner, Attrition, abrasion, corrosion and abfraction revisited. A new perspective on tooth surface lesions. Journal of the American Dental Association, 135(8), 2004, 1109-1118.
[5]. B.G. Smith, and J.K. Knight, An index for measuring the wear of teeth. British Dental Journal, 156(12), 1984, 435-438.
Van't Spijker, C. Kreulen, and D. Bartlett, Prevalence of tooth wear in adults. International Journal of Prosthodontics, 22(1), 2009, 35-42.
[6]. Kaidonis, Tooth wear: the view of the anthropologist. Clinical Oral Investigations, 12(Suppl 1), 2008, 21-26.
[7]. S.B. Mehta, S. Banerji, B. J. Millar, and J.-M. Suarez-Feito, Current concepts on the management of tooth wear: part 1. Assessment, treatment planning and strategies for the prevention and the passive management of tooth wear. British Dental Journal, 212(1), 2012, 17-27.
[8]. D.W. Bartlett, and P. Shah, A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. Journal of Dental Research, 85(4), 2006, 306-312.
Lussi, and M. Schaffner, Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Research, 34(2), 2000, 182-187.
[9]. M.E. Barbour, M. Finke, D.M. Parker, J.A. Hughes, G.C. Allen, and M. Addy, The relationship between enamel softening and erosion caused by soft drinks at a range of temperatures. Journal of Dentistry, 34(3), 2006, 207-213.
[10]. L.H. Mair , T.A. Stolarski, R.W. Vowles, and C.H. Lloyd, Wear: mechanisms, manifestations and measurement. Report of a workshop. Journal of Dentistry, 24(1-2), 1996, 141-148.
[11]. K.-H. Zum Gahr, Classification of wear processes, in K.-H. Zum Gahr (Ed.), Microstructure and wear of materials, (Amsterdam: Elsevier, 1987) 80-131.
[12]. J. Morozova, Erozivní defekty tvrdých zubních tkání. Část 1. Praktické zubní lékařství, 59(1), 2011, 4-13.
A. Lussi, T. Jaeggi, and M. Schaffner, Diet and dental erosion. Nutrition,18(9), 2002, 780-781.
[13]. M. Eisenburger, R.P. Shellis, and M. Addy, Comparative study of wear of enamel induced by alternating and simultaneous combinations of abrasion and erosion in vitro. Caries Research, 37(6), 2003, 450-455.
[14]. T. Attin, U. Koidl, W. Buchalla, H. G. Schaller, A. M. Kielbassa, and E. Hellwig, Correlation of microhardness and wear in differently eroded bovine dental enamel. Archives of Oral Biology, 42(3), 1997, 243-250.
[15]. H.K. Yip, R.J. Smales, and J.A. Kaidonis, Tooth erosion prevention and treatment (New Delhi: Jaypee Brothers, 2-nd printing, 2009).
[16]. http://www.xylitol.org/
Maguire, and A.J. Rugg-Gunn, Xylitol and caries prevention- is it a magic bullet? British Dental Journal, 94(8), 2003, 429-436.
[17]. S. Chunmuang, S. Jitpukdeebodintra, Ch. Chuenarrom, and P. Benjakul, Effect of xylitol and fluoride on enamel erosion in vitro. Journal of Oral Science, 49(4), 2007, 293-297.
[18]. R.E. Davis, T.A. Marshall, F. Qian, J.J. Warren, and J.S. Wefel, In vitro protection against dental erosion afforded by commercially available, calcium-fortified 100 percent juices. The Journal of the American Dental Association, 138(12), 2007, 1593-
[19]. J.A. Hughes, K.D. Jandt, N. Baker, D. Parker, R.G. Newcombe, M. Eisenburger, and M. Addy, Further modification to soft drinks to minimise erosion. Caries Research, 36(1), 2002, 70-74.
[20]. N.X. West, J.A. Hughes, D.M. Parker, R.G. Newcombe, and M. Addy, Development and evaluation of a low erosive blackcurrant juice drink in vitro and in situ. 2. Comparison with a conventional blackcurrant juice drink and orange juice. Journal of Dentistry, 27(5), 1999, 341-344.