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Prevention of progression of dental erosion by professional and individual prophylactic measures

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The key elements for the establishment of a preventive program for patients suffering from erosion are described on pathophysiologic grounds. These elements aim 1) to diminish frequency and severity of acid challenge, 2) enhance salivary flow, 3) to enhance acid resistance, remineralization and rehardening by fluoride application, 4) to offer chemical protection by buffering substances, 5) to minimize abrasion, 6) to offer mechanical protection. Recommendations for prophylactic measures are made as a conclusion and summarized in the form of check-lists.

Key words: dental erosion; prevention

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Key elements for the prevention of dental erosion

Erosive lesions are the result of a loss of dental hard tissue chemically etched away from the tooth surface by acid and/or chelation. Along with the irreversible loss of dental hard tissue from the surface, the demineralization leads to a clinically not detectable, but *in vitro* clearly measurable softening of the tooth surface and, consequently, to a much lower resistance against wear. Irrespective of the etiology of erosion, the following elements of prevention must be considered when designing a prophylactic strategy:

1) Measures to diminish frequency and severity of acid challenge
2) Measures to enhance the defense mechanisms of the body, i.e. salivary flow and pellicle formation
3) Measures to enhance acid resistance, remineralization and rehardening of the tooth surface
4) Measures to provide chemical protection
5) Measures to weaken abrasive influences
6) Measures to provide mechanical protection

Diminishing frequency and severity of acid challenge

Obviously the optimal preventive measure would be to remove the source of acid or to prevent it from reaching the teeth. If erosion is of *dietary origin*, the frequency of consumption of acid food should be diminished and such food be restricted to main meals. Acid drinks should be drunk quickly rather than sipped slowly, or they should be consumed through a straw (1). Effervescent vitamin tablets should be substituted for by capsules to be swallowed entirely.

Patients suffering from *chronic regurgitation* of somatic origin should be referred to a physician in order to attempt causal therapy. In very severe cases, confirmation of the suspicion of chronic vomiting, when denied by the patient, can be obtained from a blood probe when analyzing serum electrolytes (sodium, potassium, chloride) and arterial blood gases. The electrolyte concentrations in the analysis of spot urine equally serves this purpose. Both tests easily reveal the metabolic alkalosis that results from regular vomiting (2). Patients having asymptomatic gastroesophageal reflux often have a history of blotted pillous and acid taste when waking up. They should be advised to sleep with head elevation by two or three pillows or the like, and metoclopramide can be prescribed to reduce the frequency and volume of reflux as well as ranitidine to decrease gastric acid production (3). Medical prescriptions should be made by

a physician after confirmation of the anamnestic diagnosis by means of gastroendoscopy. Those patients regurgitating due to psychosomatic etiology should be referred to a psychologist or, preferably, to a psychiatrist experienced in collaboration with the dental profession for causal therapy. Successful alternative treatment in the dental office of patients with nervous vomiting has equally been described (4). Point-stimulating therapy was used, with the calming effect resulting from the action of the meridian, even though therapy was applied to specific acupoints (HoKu and Nei Kuan) (4).

Measures to enhance salivary flow and pellicle formation

Calcium and phosphate as well as an alkaline or neutral environment are prerequisites for remineralization. The state of supersaturation of saliva with respect to calcium and phosphate is maintained by the presence of specific salivary proteins. Proline-rich proteins and a tyrosine-rich protein called statherin are thought to inhibit the precipitation (5, 6). Saliva provides a constant supply of ions to the tooth surfaces and also contains buffers to resist changes in pH. The buffering capacity and bicarbonate content of stimulated saliva is far higher than that of resting saliva. Measures that stimulate salivary flow rate, e.g. the chewing of sugar-free chewing gum consequently are anti-erosive and support remineralization.

Salivary mucins secreted primarily by the submandibular and sublingual glands and present in substantial amounts form a major part of the acquired pellicle. This amorphous proteinaceous membrane adsorbs to and coats the tooth surfaces. The pellicle has been suspected to act as a "permselective membrane", having protective effects against demineralization by microbial acids (7) and against erosive acid challenges (8). Indeed, pellicle formed by 3-d exposure of enamel to salivary mucins *in vitro* gave a 100% protection against demineralization by 1% citric acid (9). Protection from erosion by salivary pellicle *in vitro* has recently again been illustrated using the SEM (10). It may be speculated that pellicle formation is also enhanced by the above measures that stimulate salivary flow.

Enhancing acid resistance, remineralization and rehardening

The processes involved in remineralization of carious lesions have been studied extensively. Calcium and phosphate as well as traces of fluoride must be present in remineralizing solutions. The rehardening of enamel surfaces *in vivo* is not the result of a remineralization by regrowth of etched and partly dissolved apatite crystals in the sense of a *restitutio ad integrum*, but rather the result of the precipitation of various calcium phosphates which are poorly soluble in acid, such as brushite ($CaHPO_4$) or dicalcium phosphate ($Ca_3[PO_4]_2$) into the porous disorganized enamel matrix (11, 12). In connection with erosion, the term "repair" might therefore be more appropriate than the term "remineralization". The most important factors in the repair of softened enamel are the saliva (see above) and fluoride. The effect of fluoride on the progression of dental erosion has not been investigated as thoroughly as the action of fluoride involved in the remineralization of early carious lesions. Several animal experiments have, however, shown that the addition of low fluoride concentrations to erosive acidic beverages or the topical application of higher fluoride concentrations reduced the erosive process. The admixture of 20 ppm F^- to the drinking water clearly reduced erosion in rodents (13–15). SPENCER & ELLIS (16) achieved a 60% reduction of erosion in rats by the addition of 50 ppm F^- to grapefruit juice. HOLLOWAY and co-workers (17) reported that even 2 ppm F^- provided some protection. It has been speculated that fluoride uptake from fluoride-containing fruit drinks (2 ppm) is more substantial than from fluoridated water (2 ppm) because of the action of fruit acids and citrate on the enamel surface (18). In an *in vitro* model using bovine teeth, RYTÖMAA and co-workers (19) could, however, not confirm a protective effect of 2 ppm F^- in saliva. Sodium monofluorophosphate (40 ppm F^-) also significantly reduced erosion in rat molars (20). More recently, the admixture of 15 ppm F^- to acidic sports drinks was found to reduce erosion in rat teeth, reducing both size and severity of the lesions (21). As etching of enamel increases the surface-reactive area (22), and topically applied fluoride has been shown to accumulate in demineralized lesions (23), the use of topical fluoride seems indicated to provide protection against erosive challenges.

The question whether high or low concentrations of topically applied fluoride are more effective has been studied with respect to the remineralization of carious lesions. In that case there is consensus within a majority of researchers and clinicians that low fluoride concentrations perform better (24, 25). It is believed that high fluoride concentrations reduce the ion exchange activity of surface enamel and promote the formation of a poorly permeable remineralized surface layer, blocking enamel pores and hindering the remineralization of the underlying lesion. The effectiveness in the repair of erosion of highly concentrated topical fluorides might thus also be questioned. Indeed, there is a great reduction of enamel solubility, as the work of GRAUBART *et al.* (26) has shown: a 24

h immersion in 1:4 thinned grapefruit juice removed a 22 µm layer of enamel from premolars. A 4 min pretreatment with 2% NaF reduced the thickness of the dissolved layer to 9.5 µm. Such a reduction in solubility must, however, be preceded by an acid etching (27). CHOW & BROWN (28) showed that acid etching of enamel leads to a transformation of apatite to brushite as an intermediate product. This in turn is readily recrystallized into fluorapatite when exposed to low concentrations of fluoride (<1000 ppm F^-), especially in the presence of a low F/Ca ratio (29). Remineralization by this mechanism occurs at a much faster rate than by solid state diffusion and the exchange of F^- ions for OH^- ions. In the presence of higher fluoride concentrations, however, brushite reacts instead to form calcium fluoride (CaF_2) (30).

Initial carious lesions (white spots) and erosive lesions differ in pathology, and in these two conditions fluoride is applied for different purposes. In the case of white spots, the progression of caries is initially delayed by mechanical plaque removal, which, in effect, is equivalent to the withdrawal of cariogenic acids, and fluoride is then used to arrest the process and to remineralize the deep subsurface lesions. For this goal, frequent applications of low concentrations of fluoride presumably perform best. In the case of erosion, however, fluoride is applied primarily to stop the progression by reducing the acid solubility of the surface, and there is no deep subsurface lesion in need of remineralization but only a thin enamel surface layer in need of rehardening. With erosion, the application of high concentrations of fluoride consequently appears to be better suited to the purpose. Indeed, topical treatment of human enamel with NaF solution (1.2 % F^-) for 48 h inhibited enamel softening and protected it from erosion during subsequent exposure *in vitro* to an acid cola beverage (31).

Measures to provide chemical protection

The buffering of acids which are capable of etching the tooth surfaces is an important measure to diminish the acid challenge and thereby stop the progression of erosion. The buffer capacity of saliva is consequently of utmost importance. Frequently repeated erosive acid challenges, however, overwhelm the salivary capacity, and the introduction of additional buffering substances into the mouth becomes necessary. Citric and lactic acids erode rat molars to a similar extent at a pH of 2.6 (15). When buffered to a pH above 3.5, their erosive action is only very weak (32). Although phosphoric acid has been shown to be very erosive at pH 2.5, at pH 4.5, both phosphoric and lactic acid produced hardly any erosion on rat molars (15). Because erosion not only depends on acid but also on chelation, soluble calcium salts and calcium phosphates must have an anti-erosive effect. HARTLES & WAGG (33) showed in animal experiments that the erosive activity of malic acid containing water could be reduced by the admixture of NaH_2PO_4, $CaCl_2$ and Na_2CO_3. *In vitro* enamel dissolution by fruit juice was considerably weakened by phosphate added as brushite and calcium acetate. At a pH of 4, enamel could not be eroded in the presence of 300 ppm phosphorus (as phosphate) and 600 ppm calcium. In man, the addition of 2.5% dicalcium phosphate to citric acid-containing tablets appeared to protect against erosion (34). In addition, according to REUSSNER *et al.* (20), the admixture of brushite (0.15%, 0.3%) to fruit juices protected rat molars better than did sodium diphosphate (NaH_2PO_4), sodium trimetaphosphate and sodium hexametaphosphate respectively. MCDONALD & STOOKEY (35) were less negative with respect to sodium phosphate. They found that sodium diphosphate stopped the erosion by acid beverages and fruit juices equally well on human enamel *in vitro* and in rodents. It can be concluded that every neutralizing procedure is useful for the prevention of erosion, given it is employed immediately following the (extrinsic or intrinsic) acid challenge.

Acid neutralization may be attempted by *buffering components of the diet.* Thus, patients were advised to hold some milk in the mouth for a short time after fruit consumption or vomiting (36). Milk (37) and also cheese (38) have been shown to reharden presoftened enamel specimens worn *in situ.* Attempts have also been made to improve salivary alkalinization by adding various neutralizing agents to chewing gum formulations, thus combining the mechanical stimulation of flow with the chemical neutralization effected by the additives. An early example for this approach is a Norwegian patent (No. 46152) from 1929 for a chewing gum containing magnesia oxide. Modern analogues contain dicalcium mono- or orthophosphate, sodium carbonate, sodium bicarbonate, diammonium phosphate and similar compounds. Another approach to enhance intraoral acid neutralization was the idea to incorporate urea (carbamide) into a chewing gum. By the enzyme urease, some intraoral bacteria readily hydrolyze both naturally occurring salivary urea and added urea into carbondioxide (CO_2) and ammonia (NH_3). The latter leads to an alkalinization of oral fluid and plaque. The neutralizing effect of chewing gums containing phosphates and carbonates or urea has recently been illustrated (39). Whether an overuse of chewing gum represents a risk of demastication of acid

etched occlusal tooth surfaces has not been investigated to date.

Acid neutralization can equally be achieved by the sucking of sugarfree *antacid tablets*. Rinsing with a pinch of sodium carbonate (Na_2CO_3) or, more easily available in the household, with baking powder ($NaHCO_3$) dissolved in some water has been suggested for patients suffering from erosion (36). A much more convenient galenic form that meets with better patient compliance, namely the sucking of sugarfree antacid tablets, sold over the counter, was proposed by IMFELD in 1984 (40). *In vivo* telemetric recordings of the pH of oral fluid showed the good neutralizing efficiency of sugarfree antacid tablets following regurgitation in anorectic patients (40, unpublished own data). The buffering capacity of antacid preparations from the Finnish market was also shown *in vitro* (41).

Another possibility to introduce buffering agents into the oral cavity is the use of *bicarbonate-containing toothpastes*. Alkaline toothpastes or gels, applied with the finger tip are often recommended before going to bed to protect against erosion from reflux during sleep. The buffering effect of a toothpaste containing 67% of baking soda has been illustrated *in vivo* (42). Many toothpaste manufacturers have produced bicarbonate-containing toothpastes that are advertised for their specific neutralizing effect.

Measures to minimize abrasive influences

Erosive lesions are often exacerbated by mechanical abrasion of the etched tooth surface. SCHWEIZER *et al.* (36) have shown that it is possible to substantially abrade enamel, previously etched by orange juice, with a toothbrush even without toothpaste. In combination with toothpaste, 2–4 μm of softened enamel could be removed *in vitro*. Only very low-abrasive toothpastes should therefore be recommended for patients suffering from erosion. The process of abrasion resulting from toothbrushing and toothpaste is not yet fully understood. Many factors, alone and in combination, seem to be involved: number, thickness and arrangement of the bristles, strength and direction of force, speed and duration of brushing, as well as the abrasive, binding agent, humectant etc. of the toothpaste. HARTE & MANLEY (43, 44) investigated, among other factors, the influence of bristle hardness, the type of abrasive and its concentration, and the suspension medium of dentifrices. They found that using glycerin as a suspension medium reduced abrasion about 88% in comparison to saliva or carboxymethylcellulose. Unfortunately, glycerin is not widely used in dentifrices because of its relatively high price. In 1970, the American Dental Association's Council on Dental Therapeutics made recommendations for the testing of dentifrice abrasivity. Relative dentin abrasion (RDA) and relative enamel abrasion (REA) are today measured, and some producers declare the respective values on the label. A review on RDA / REA has recently been published (45).

Because freshly etched enamel is easily abraded, patients with erosion are advised not to brush immediately following the consumption of acid food, reflux or regurgitation. There is as yet, however, insufficient scientific data on the appropriate length of time to allow to elapse before brushing acid challenged teeth. Patients should brush gently, using a vertical rather than a cross brushing technique and apply low or non-abrasive toothpaste (often labelled as "sensitive" by manufacturers) by means of a soft or medium type of brush.

Providing mechanical protection

It has long been known that the application of a sealant onto etched intact enamel leads to a marked increase of acid resistance. SCHWEIZER *et al.* (36) have therefore proposed the etching and sealing of early erosive lesions in order to stop further progression by chemical dissolution and mechanical wear. SORVARI *et al.* (31) have recently used a sodium fluoride varnish based on an alcoholic suspension of natural resins. Their experimentation on extracted human molars showed that this product inhibited enamel surface softening during acid exposure. *In vivo*, the product cannot, however, provide mechanical protection as it is only a suspension of natural resins. This is in contrast to a polyurethane-based lacquer containing difluorosilane. Both fluoride varnishes deposit large amounts of fluoride in enamel (46), but the

Table 1

Professional preventive measures performed by the dentist and/or dental hygienist irrespective of the etiology of erosion.

- Patient information and instruction about the cause of the erosion
- Patient advice on appropriate oral hygiene products and techniques
- Professional stain removal when needed to compensate for the low abrasive toothpaste used by the patient
- Application of fluoride lacquer on initial erosive lesions 4 times per yr or more depending on whether the erosion is progressing or stable
- Monitoring of erosion progression every 6 months by comparing the actual with the initial study models (macro level)
- Replacement of occlusal restorations when they are lost because of erosion of the surrounding enamel as a means to prevent the elongation of a single tooth or the loss of vertical dimension when multiple teeth are involved (metal, fine hybrid or microfilled composites)

Table 2

Individual preventive measures performed by the patient irrespective of the etiology of erosion

- Soft or medium type toothbrush
- Low abrasive (RDA <40), fluoride- and bicarbonate-containing toothpaste
- No toothbrushing immediately following an (intrinsic or extrinsic) acid challenge
- Vertical brushing technique
- Low concentrated (0,025–0,05 % F^-), non-acidulated fluoride mouthwash twice per d or more
- pH neutral, highly concentrated (>1% F^-) fluoride gel twice a week
- Sugar-free (safe for teeth) chewing gum, preferably products containing bicarbonate and/or other buffering substances or urea-containing gum, used alternatively with fluoride-containing gum to stimulate salivary flow following an acid challenge

Table 3

Advice to patients suffering from dietary erosion

- Diminish frequency of consumption of acid foods and beverages
- Restrict acid foods to main meals
- Finish meal with neutral food, e.g. cheese, rather than acid food, e.g. fruit salad
- Drink acid beverages quickly or with a straw, do not sip or swish around
- Rinse with water after acid consumption
- Apply preventive measures listed in Table 2

Table 4

Establishment of differential diagnosis of erosion etiology

- Intraoral examination
- Dental anamnesis (case history taking)
- Medical anamnesis
- Dietary anamnesis (food diary, minimally 5 d)
- History of occupation and lifestyle
- Salivary tests (flow rate, buffer capacity)
- Intraoral photographs
- Study models
- Consultation with the patient's physician

lacquer can be expected to provide a double protective action, namely fluoride-dependent decrease of acid solubility and mechanical protection against wear. Fluoride lacquer is therefore recommended as a professional prophylactic measure for initial erosive lesions. Larger lesions warrant the use of composites and direct bonding.

Recommendations

Whereas many prophylactic measures for erosion are independent of the etiology of the lesion (Tables 1 and 2), some of them, and the recommendations to the individual patients may vary according to the etiology (Table 3). It is therefore important to establish a differential diagnosis of the origin of erosion, based on careful clinical inspection and a thorough anamnesis (case history taking).

A clinical registration of the localization, severity (Class I to III) and pathogenetic activity (latent, manifest) is the first step. Intraoral photographs for documentation and study models to monitor progression at the macro level are equally necessary. Salivary tests including resting and stimulated flow rate as well as buffer capacity should be performed. Medical anamnesis must cover chronic medication, reflux, heartburn, acid mouth taste etc. An extensive dietary anamnesis is best performed by asking the patient for a 5-d food diary (3 working days and a weekend). The diary must detail type, amount and preparation of foods as well as the exact time of day, duration and mode of consumption. The diet record should equally include medicines, vitamin supplements etc. The steps towards a differential diagnosis are summarized in Table 4.

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