Pathogenesis and modifying factors of dental erosion


Dental erosion is caused by acidic solutions which come into contact with the teeth. Because the critical pH of dental enamel is approximately 5.5, any solution with a lower pH value may cause erosion, particularly if the attack is of long duration, and repeated over time. Saliva and salivary pellicle counteract the acid attacks but if the challenge is severe, a total destruction of tooth tissue follows. Ultrastructural studies have shown that erosive lesions are seen in prismatic enamel as characteristic demineralization patterns where either the prism cores or interprismatic areas dissolve, leading to a honeycomb structure. In aprismatic enamel the pattern of dissolution is more irregular and areas with various degrees of mineral loss are seen side by side. In dentin the first area to be affected is the peritubular dentin. With progressing lesions, the dentinal tubules become enlarged but finally disruption is seen also in the intertubular areas. If the erosion process is rapid, increased sensitivity of the teeth is the presenting symptom. However, in cases with slower progression, the patient may remain without symptoms even though the whole dentition may become severely damaged. Regarding the role of causative agents, present data does not allow the ranking of different acids with regard to their potential of causing erosion. Neither is there consensus as to how effective fluorides are in preventing the progression of erosive lesions, or how the chemical and structural factors of tooth tissue in general might modify this pathological process.

Dental erosion is, by definition, a chemical dissolution of the dental hard tissues in a process which does not include bacteria (1). It is caused either by intrinsic or extrinsic acid sources. The intrinsic causes comprise recurrent vomiting as often occurs in patients suffering from anorexia (2), bulimia (3), cytostatic drug treatment, or propulsion of gastric contents into the mouth due to gastroesophageal reflux (4, 5). The extrinsic causes comprise frequent consumption of acidic foodstuffs or drinks (6), use of acidic oral hygiene products (7), and acidic medicines such as effervescent vitamin C or aspirin tablets (8–12). Alcoholics have been reported to be liable for erosion due to both vomiting and consuming of acidic drinks (13). Erosion may be also caused by gaseous acids or other chemicals breathed in the working environment (14, 15). Frequent swallowing of water in improperly chlorinated swimming pools (which leads to the formation of hydrochloric acid) has also been reported to cause erosion (16, 17).

In discussing dental erosion it is anticipated that it is usually caused by acidic solutions which come into contact with the teeth, with one of the main modifying factors being the role of flow of saliva. Many drinks have pH values below 4 and the pH of gastric juice can be below 1, which well explains its erosive capacity. Proteolytic enzymes have also been hypothesized to occasionally cause erosion (15). Erosions which are frequently seen among lactovegetarians are caused by acids derived from low-pH foodstuffs, but it must be emphasized that lactovegetarians also frequently consume coarse fresh foods, more so than controls, which adds to the tooth tissue wear (18). In all these cases mixing with saliva will cause some, but apparently insufficient, diluting and buffering of the acids.

Whatever the cause, the gross pathology encompasses superficial demineralisation of dental hard tissues, primarily by dissolution of the apatite crystals. This may lead to severe or total destruction of the teeth, depending on the strength, in terms of low pH, low calcium, phosphorus, and chelating properties, and frequency of the erosive attacks. In restorative and preventive dentistry, however, deliberate "erosion" by acid etching of
enamel has been successfully used to enhance polymer adhesion to enamel structures (19). Much of our knowledge on enamel dissolution has been derived from studies on caries or from the acid etch technique.

Erosion of enamel

Erosion attacks the enamel surface. Acids also find their way to the pits and pores in enamel and cause enamel prism destruction in subsurface layers (20). Ultrastructural in vitro studies by Meurman & Frank (21, 22) have demonstrated the progression of erosion in prismatic and aprismatic enamel. In these studies, enamel specimens prepared from human or bovine teeth were immersed in chemically defined acidic solutions for various time periods and inspected by scanning electron microscopy. At short immersion times (15 min), malic acid (pH 3.4) appeared less erosive than solutions containing citric acid (pH 2.8) or phosphoric acid (pH 2.6). After longer immersion (60 min), morphological differences between specimens treated with various acids disappeared. It had earlier been shown that malic acid is indeed less erosive than citric acid (23). In vitro, the lower the pH, the more erosive is the solution, but titratable acidity and chelating properties may greatly modify the rate of erosion (see also below) (24). Nevertheless, morphologically, the lesions are very similar. In prismatic human or bovine enamel, the lesion first develops in the prism sheath areas, followed by dissolution of prism cores with prolonged challenge (Fig. 1). Eventually, the interprismatic areas also become affected (21). Depending on the chemical composition of the outer enamel layer (in particular the fluoride content), erosive patterns vary in a similar way as reported for acid etching of enamel (25).

Aprismatic enamel erodes in a highly irregular manner (Fig. 2), and it is probably not as liable to erosive destruction as prismatic enamel. Meurman & Frank (21) observed areas of distinctly eroded enamel adjacent to less affected areas when specimens with aprismatic enamel were subjected to experimental erosion.

The progression of erosion in deciduous and permanent teeth

There are no studies published on the progression of erosion in deciduous teeth. These teeth often have aprismatic enamel on their surface, and their chemical composition differs somewhat from that of permanent teeth. Smith & Shaw (26) have reported on cases of erosion of deciduous teeth caused by baby fruit juices given in a reservoir feeder and used as a comforter at night. Own observations from Saudi-Arabia show large erosive lesions in the deciduous teeth of children who frequently consume acidic beverages in the hot climate. When examined by electron microscopy, these lesions appeared as deep porosities and showed destruction both in the oral and facial surfaces.

The progression rate of erosive lesions has been quantified in an in vitro experiment with bovine enamel (24). Erosion appears to progress from 1.5 to 6 μm/h, and the mineral loss is linear up to 2 h during immersion in acidic beverages. There are no calculations on how rapidly erosion progresses in human enamel. Featherstone & Mellberg (27) have reported that artificial caries lesions in bovine enamel develop three times faster than lesions in human enamel. If the same ratio applies for the acid dissolution of enamel by erosion, then the data by Rytömaa et al. (24) would indicate that unprotected human enamel might erode at 0.5–2 μm/h. However, it must be emphasized that these in vitro situations greatly exaggerate the erosion effects because of lack of protective factors (see below). Savad (16) reported that clinically detectable erosive lesions developed within 4 wk in a patient who was a member of a swimming team and probably got his lesions by repeated exposure to gas chlorinated water. Xhonga et al. (28) measured the rate of erosion in permanent teeth to be 1 μm/d in a 5-month clinical study comprising patients who exhibited various degrees of erosions. Representativeness of this study, however, is questioned because only thirteen subjects were followed up.

Erosion in dentin

Meurman et al. (29) studied experimental erosion in dentin with the same methods as had been used with enamel specimens. Test specimens were prepared from coronal dentin of human third molars, immersed in chemically different acidic solutions from 30 s to 60 min, and analysed by scanning electron microscopy. Unfortunately, no attempt was made to quantify the progression of erosion. The first dentin area to be affected was the peritubular dentin, where destruction and tubular hollowing were observed. With longer immersion times, the lesions progressed to the intertubular areas resulting in rough and porous surfaces. The dentinal tubules became significantly enlarged (Fig. 3). These findings clearly explain why eroded teeth become sensitive to external stimuli, in particular when the lesion progression is rapid. According to Eccles & Jenkins (6), every second patient suffering from dental erosion complains of
painful sensitivity of the teeth. This was confirmed by Järvinen (30) in a case-control study: 58% of her subjects with erosions suffered from hypersensitivity of the teeth in comparison to 26% of patients without erosions. The reason for the symptom was suggested to be the loss of superficial dental tissues, that exposed vital dentin (1). But, if the process is slow, even severe erosive lesions need not cause hypersensitivity symptoms (31, 32). The chemical characteristics of the erosive agent further modify the pathogenesis of the lesion also in dentin, as shown by Rogalla et al. (9) in a study using buffered and unbuffered acetylsalicylic acid to produce erosion.

**Measurement of erosion and etiological factors**

In addition to electron microscopic techniques, erosion has also been studied by methods measuring microhardness, loss of surface contour (by profilometric analysis) and surface porosity (by iodide permeability tests) (23, 24, 33). Finally, tongue surface pH has been measured after consuming test drinks and foodstuffs in order to assess the erosive potential of various products (34). This has lead to a model in which the erosive potential of a product can be predicted from its acidity, pH value and phosphate, calcium and fluoride contents. For example, sour milk products with their high calcium and phosphorus concentrations are not erosive in spite of their low pH (4.4-7) and high lactic acid contents (33, 34). However, these models, like all in vitro models, oversimplify the complex pathogenesis of erosion.

Grobler et al. (35) investigated enamel erosion caused by different fruits while Grenby et al. (36) and Grobler et al. (37) studied soft drinks in this respect. They concluded that the best guide for the erosiveness of a product is its content of titratable acid, but that other factors may greatly modify the erosive potential. These factors include the pH and chemical components of the products, and the dis-

![Fig. 1. In vitro eroded enamel after immersion of the specimen in a cola beverage (pH 2.6) for 15 min at 37°C. The picture shows a characteristic honeycomb structure of demineralized enamel on the affected side where the enamel prism cores have dissolved leaving the interprismatic areas protruding. Bar=10 μm.](image1)

![Fig. 2. In vitro eroded aprismatic enamel. The specimen is from a similar experiment as in Fig. 1. An irregular dissolution pattern can be seen. Bar=10 μm.](image2)

![Fig. 3. In vitro eroded dentin. After 30 min immersion of the specimen in a malic acid containing sport beverage (pH 3.4) a distinct opening and hollowing of the dentinal tubules can be seen on the affected side. C=control. Bar=30 μm.](image3)
association constants of the acids involved. Apple juice, for example, having a pH value 3.4, has been shown to depress intraoral pH longer than a cola beverage with pH 2.3 in an experiment with telemetric interdental plaque pH registration (38). Apple juice has a much higher titratable acidity than cola beverage which explains this finding (38). MEURMAN & FRANK (21) found citric acid to be more erosive than phosphoric acid only in specimens challenged for short periods (15 min). The higher erosiveness of the citric acid, during the early stages of acid dissolution, may be the result of its chelating (calcium binding) properties. When the process continues, the tissue destruction becomes so prominent that no difference can be related to the causative agent. ELSBURY (39) showed that citric acid indeed erodes enamel at a rate more than twice that of hydrochloric or nitric acids of the same concentration. Nevertheless, phosphoric acid, too, is a good chelator and whilst some authors have found citric acid to be more erosive than phosphoric acid (40, 41), other workers have found the opposite (42–44).

Several organic acids contained in beverages and fruits have chelating properties. This is the capacity to bind calcium, which results from the chemical composition of the acids having more than one carboxyl group. Chelation may, in principle, contribute to dental erosion by two mechanisms. The acids could bind strongly to calcium ions in saliva, thus reducing the degree of supersaturation of saliva with respect to the tooth minerals. With a partial loss of the common ion effect of calcium in saliva, the dissolution tendency of enamel in a saliva-organic acid mixture is thus greater than for saliva alone, even apart from pH considerations. Secondly, if the tendency to bind calcium is very large, the chelating forces may enhance direct dissolution of enamel (similar to etching) to meet the saturation levels of the calcium-organic acid complex.

Taking the example of citric acid, which forms soluble calcium citrate complexes in solution with pK values of 3.2, 2.1 and 1.0, respectively, for tri-, di- and monovalent citrate ions (45), it can be calculated that 32 % or 3.1 % of the calcium in saliva is complexed to citrate at a citric acid concentration of 0.1 and 0.01 M, respectively. (Citric acid levels in lemons are reported to be around 0.2 M).

Thus, the chemical structure of the erosive agent, but also the structure and composition of enamel, play a key role in the initiation and progression of erosive lesions. Nevertheless, current data does not allow the ranking of the erosive potential of various acids. Further factors, such as sugar content and carbonation (46), and other ingredients of a drink or foodstuff, such as sorbitol (47), also play a role in this respect.

Protective factors against erosion

Saliva

In the experimental studies cited above, no protective factors (such as salivary pellicle or fluoride) had been taken into account. In vivo, teeth are continuously moistened by saliva which contains a number of protective factors against mineral dissolution. The salivary function was highlighted already in the 1940s (48, 49) and later corroborated by MANNERBERG in 1963 (50). He reported that although the amount of saliva, its pH value, buffering capacity and calcium and phosphorus contents did not differ between cases with erosion and controls, the salivary mucin content did vary (50). Saliva was also discussed in detail at the first international Symposium on Dental Erosion (51). Still, we know very little about the role of saliva in the pathology of dental erosion.

WOLTGENS et al. (52) found significantly lower unstimulated secretion rates in persons suffering from erosion when compared to controls. The same was detected by JÄRVINEN et al. (4) in three erosion patients out of seven who suffered from upper gastrointestinal disorders. Recently, however, MEURMAN et al. (5) found no differences in unstimulated and stimulated salivary flow rates and viscosity values between 117 endoscopically verified reflux disease patients with or without erosion. Nevertheless, low salivary buffering values were more often seen in patients with erosion than in those without. There may be inborn differences in the buffering capacity, as suggested by GUDMUNDSSON et al. (53). These authors found low salivary buffering in most erosion patients, who also suffered from gastroesophageal reflux. Further, salivary buffers may also be exhausted by gastric acid in reflux patients, but it must be emphasized that if the erosive challenge is strong enough, then even normal salivary flow and function cannot protect the teeth (5).

MEURMAN & FRANK (22) showed by electron microscopy how efficient saliva is in protecting the enamel surface against erosion. They used clarified saliva to form pellicles onto enamel specimens and reported a distinct protective function, even though the resulting salivary films were not coherent and did not cover the specimens totally (that is at a microscopic level). The protective mechanisms presumably parallel those of pellicle in experimental caries and apatite dissolution studies. Under those conditions, salivary proteins have been reported to bind to the dissolution sites and to cover the crystallites by specific adsorption (54, 55). The
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neutral pH with respect to apatite. The rate at which apatite precipitates depends, amongst others, on factors like calcium binding in saliva and diffusion inhibition by salivary pellicle.

With decreasing pH, saliva ‘crosses’ the saturation line at a point known as the critical pH. For enamel this has been calculated to be around 5.5, the precise value depending on the saliva (or rather plaque fluid) and enamel composition. This value is also influenced by the degree of impurities in the enamel mineral, mainly carbonate and magnesium (59). Fluoride is a complicating factor as this may give rise to the formation of a fluoridated (or fluor)apatite, which has a significantly lower solubility. For fluorapatite, at the prevailing fluoride levels, a critical pH can be calculated at around 4.5 (Fig. 4). In the case of caries, fluoride incorporation in enamel is particularly effective in prevention, as it results in a lowering of the critical pH, which reduces the time period when the plaque is undersaturated after eating fermentable carbohydrates.

When enamel is immersed in an acid solution at lower pH (i.e. 1–4), the situation is different because both hydroxyl- and fluorapatite are undersaturated (see Fig. 4). Then, neither fluoridation of the enamel, nor fluoride present in the ambient solutions, affect the degree of undersaturation of the solution with respect to the tissue minerals.

In summary, saliva and its components protect the dentition against erosion by various mechanisms:

- increased salivary flow helps to dilute acids in the mouth, which also leads to their rapid removal by swallowing,
- salivary buffers partly neutralize the acids in the oral fluid,
- the calcium and phosphate levels in the saliva act as common ions to the minerals in enamel and dentin, resulting in a slower dissolution rate of mineral,
- salivary mineral ions may also reprecipitate during a remineralization of the erosive lesions,
- salivary mucins and other organic components form a pellicle on the tooth surface, which inhibits or slows down mineral loss during acid dissolution,
- saliva production is increased by the acid induced stimulation of the salivary glands.

**Enamel chemistry**

Enamel is composed of a carbonated calcium hydroxyapatite. The solubility of this mineral is pH dependent, as both phosphate and hydroxyl-ions bind protons. With saliva containing calcium and phosphate ions, a state of supersaturation exists at neutral pH with respect to apatite. The rate at which apatite precipitates depends, amongst others, on factors like calcium binding in saliva and diffusion inhibition by salivary pellicle.

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preventing erosion is small as compared to its effects on caries (see below).

Fluoride

Although the effect of fluoride on erosion process can be theoretically discussed, as above, the effectiveness of fluoride against erosion has not been thoroughly investigated. SPENCER & ELLIS (61) found 50 ppm sodium fluoride to reduce erosion up by 60% in rats. HOLLOWAY et al. (41) found some protection in rats by 2 ppm sodium fluoride. XHONGA & SOGGNAES (62) evaluated the effect of topical fluorides at substantial concentrations on erosion progression in vivo and found only minimal protection against erosion by three fluoride treatments, namely potassium fluoride (60,000 ppm), sodium silicofluoride (9,400 ppm), and a fluoride-containing fissure sealant (100,000 ppm). Later rat experiments with fluoridated acidic drinks (15 ppm) have shown protection against erosion (63), while in the in vitro study by RYTMÄÄ et al. (24) no effect of 2 ppm fluoride could be seen on erosion in bovine enamel. SORVARI et al. (64) studied the effect of concentrated fluoride varnish (22,600 ppm) and rinses (12,000 ppm) on enamel erosion. Both treatment modes inhibited erosion caused by acidic beverages in human third molars. In the dental office, topical fluoride treatments are commonly recommended to patients with erosions, although scientific evidence for such a practice is virtually lacking. There are, nevertheless, intuitive reasons to assume that it is good clinical practice, but further work is indeed needed to confirm the efficacy of topical fluoride treatments in patients with erosion (see also the paper by ISMIELED in this issue).

The complexity of tooth wear pathology

Tooth wear is hardly ever caused by a single factor alone (65, 66). Erosion, attrition and abrasion occur simultaneously, although one of the three may be most prominent from time to time and from case to case. LEWIS & SMITH (67) have pointed out that erosion is readily overlooked, in particular in cases of attrition.

SORVARI et al. (68) have shown that dental hard tissue softened by erosion is more susceptible to attrition. This was tested in animals fed a rough diet, either with or without an acidic drink. The same can be expected to hold true also in human teeth. In clinical cases, however, it is difficult to discriminate between the causes of tooth wear. All the three causative components must be taken into consideration when patients are counselled.

Further, the underlying cause for erosion is not necessarily reflected in any characteristic appearance of the lesions, or specific location in the dentition. It has been a common belief that intrinsic erosion would affect more the palatal surfaces, while extrinsic causes for erosion would preferentially cause labial lesions. This is apparently not so straightforward (see below), unless the reason is obvious; for example, a habit of sucking lemons in a patient who developed erosions in the labial surface of the front teeth. Nevertheless, there may also be site-specific patterns and individual variations of acid clearance in the mouth and, subsequently, erosive lesions develop in an area where retention of acid may be high (32). The most vulnerable sites would then be those less readily bathed in saliva. But there is no consensus on the site specificity of erosive lesions regarding their cause, and more studies are called for to clarify this clinically important aspect.

In their case-control study of 106 patients with dental erosion in Finland, JÄRVINEN et al. (69) did not find any signs and symptoms of the lesions or their location on the teeth, which could be associated with the intrinsic or extrinsic causes. Erosion was in all cases most common in the upper incisors, canines and premolars. The most severe lesions were seen in the palatal surfaces, irrespective of the underlying cause for erosion. In an epidemiologic study on a Swiss cohort by LUSSE et al. (31), erosion was most often found in maxillary anterior teeth. In this study material palatal erosions were scarce, and half of these patients had a history of chronic vomiting.

In clinical work, but also in epidemiological studies, it is not possible to distinguish between the underlying reasons for erosion, because in many cases there are concomitant extrinsic and intrinsic causes (69). The picture is further complicated by great individual variations in salivary flow, distribution, clearance properties and buffering capacity. It seems therefore unrealistic to draw conclusions based only on the lesion location on the teeth. The most severe lesions are commonly found in patients suffering from psychiatric (anorexia or bulimia nervosa; 70) or gastrointestinal disorders (reflux disease, peptic ulcer disease; 4). In these cases, the pathology may encompass the whole dentition (see also the paper by SCHEUTZEL in this issue). This should prompt the clinician to carefully examine the patients and to ask simple questions so as to clarify why the patient’s teeth are affected. A dentist is usually the first to observe the signs in the teeth. He/she may thus play a key role in referring the patient for further medical examination or to persuade him/her to quit the habit of consuming too many acidic drinks or foodstuffs in order to avoid further destruction of the teeth.
Erosion vs. dental caries

Although the final phase of the destructive pathology in erosion and caries is similar, namely dissolution of apatite crystals, these two pathologies rarely occur simultaneously (see also Fig. 4). Caries occurs on plaque-covered sites whereas erosion occurs on plaque-free sites. The concentration of acids that comes into contact with the dental tissues is far greater in erosion than in caries. In erosion, the teeth are practically rinsed with acids, while caries is the disease of a site where acids are formed by plaque microorganisms. Depending on the strength and frequency of erosive challenges, no plaque microorganisms can tolerate the low pH. As mentioned, many acidic foodstuffs and drinks have pH values below 3, and the pH of gastric contents may be below 1. Carious mutans streptococci cease to metabolize at pH values below 4.2 (71). This explains why erosion and caries are usually not seen on the same tooth surfaces. In many erosion patients the whole dentition may otherwise be sound, which further emphasizes the insidious nature of erosion (30). Erosive lesions are indeed often seen in patients who are very conscious of their general health and well-being, an example being the lactovegetarians (18). It should also be stressed, once more, that erosion is primarily a surface phenomenon while caries begins as subsurface demineralization of enamel structure.

Conclusion

The pathogenesis of dental erosion is not completely understood. Most published studies deal mainly with gross morphological changes due to in vitro acidic dissolution of dental hard tissues by erosive agents. Erosion is caused either by frequently consumed acidic foodstuffs and drinks or by propulsion of gastric contents to the mouth, due to vomiting or reflux. Rarely, the cause is consciousness of their general health and well-being, an example being the lactovegetarians (18). It should also be stressed, once more, that erosion is primarily a surface phenomenon while caries begins as subsurface demineralization of enamel structure.

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