Eur J Oral Sci 1996; 104: 199–206
Printed in Denmark . All rights reserved

EUROPEAN JOURNAL OF
ORAL SCIENCES
ISSN 0909-8836

Pathogenesis and modifying factors of dental erosion

J. H. Meurman[1] and J. M. ten Cate[2]
[1]Faculty of Dentistry, University of Kuopio, P.O.B. 1627, FIN-70211 Kuopio, Finland;
[2]Academic Centre for Dentistry Amsterdam, Louwesweg 1, NL-1066 EA Amsterdam, The Netherlands

Meurman JH, ten Cate JM: Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci 1996; 104: 199–206. © Munksgaard, 1996.

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.

Key words: dental; erosion

Prof. Jukka H. Meurman, University of Kuopio, Faculty of Dentistry, P.O.Box 1627, FIN-70211 Kuopio, Finland. Telefax: +358-71162 213. E-mail: Jukka.Meurman@uku.fi

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.

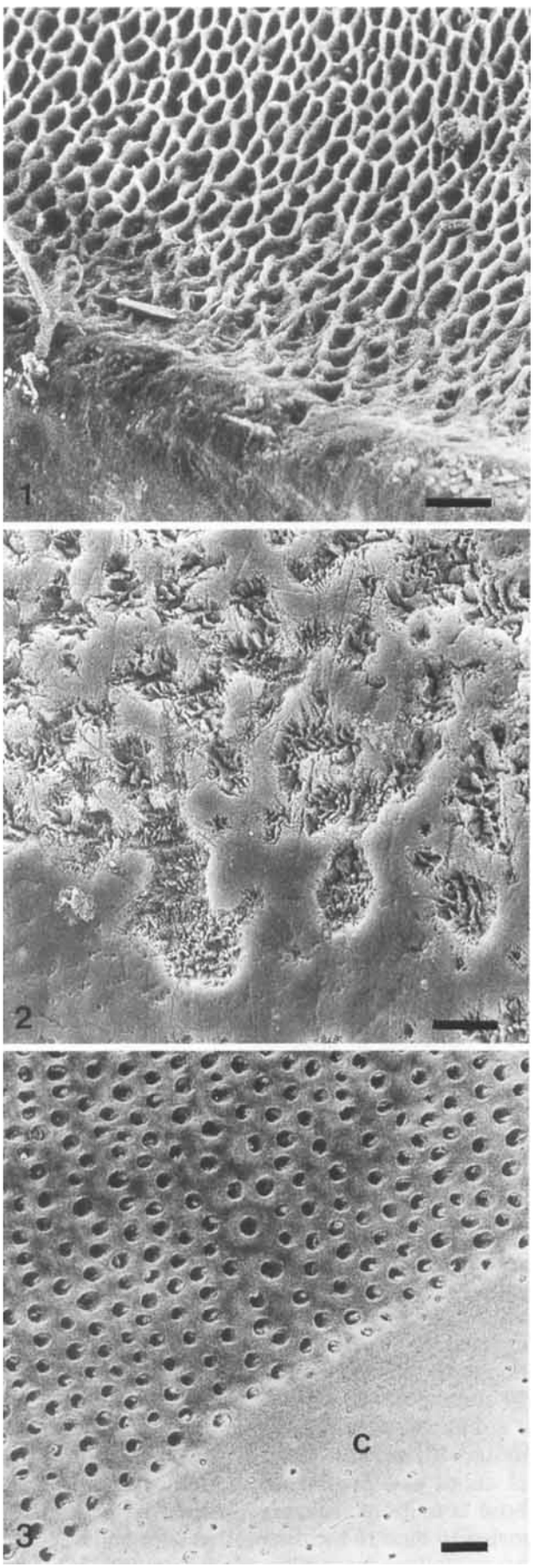
The progression of erosion in deciduous and permanent teeth

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.

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.

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=10 μm.

sociation 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.

WÖLTGENS *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

protective functions of saliva and its various components against erosion should be further studied. In particular the role of salivary mucins needs reconsideration (50). How dentin reacts to an erosive attack *in vivo* is not known either.

In carious lesions it has been documented that a redeposition of calcium and phosphate occurs from saliva (56). This precipitation takes place in the subsurface regions of white spot lesions, in a chemical form similar to the original tooth mineral hydroxyapatite. Whether the same phenomenon may be expected to occur in etched surfaces, is yet to be established. A remineralization of etched enamel has been reported (57). Later, it has been questioned whether the disappearance of the etched prism structure was not merely the result of the deposition of an organic pellicle layer. In the process of erosion the conditions make the surface a less likely substrate for remineralization: the pH being lower than inside the lesion, the lack of an (eligible) crystallite surface for mineral precipitation, and the rapid wear of a rough enamel surface, which adds to the loss of precipitation substrate.

Erosion, however, does not only lead to an *ad integrum* loss of enamel but also to a slight subsurface mineral loss. This layer, showing small histological changes but with the gross enamel structure being essentially intact, has been shown to be remineralizable (58).

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.

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

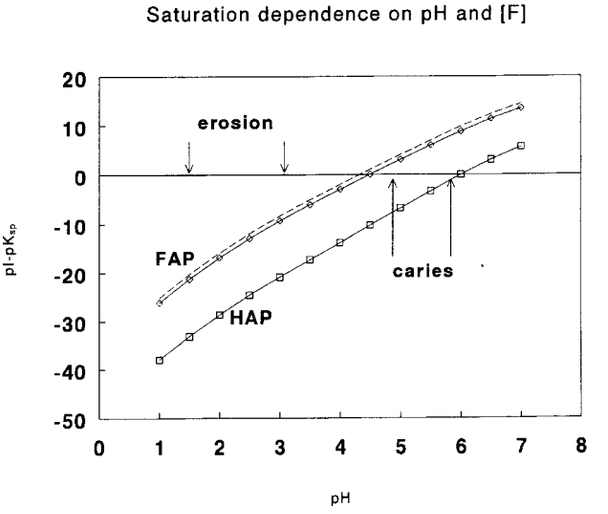


Fig. 4. Degrees of saturation versus pH with respect to hydroxyapatite (HAP) and fluorapatite (FAP), calculated for salivary concentrations of 10^{-3} mol/l calcium, 10^{-2} mol/l total phosphate, 10^{-4} mol/l (2ppm) fluoride and μ=0.06. Increasing the fluoride concentration in solution to 10^{-3} mol/l (20 ppm) has a very small effect on the degree of saturation with respect to FAP. Positive values denote supersaturated solutions. Caries occurs at pH values at which FAP can be formed, which stabilizes the surface layer characteristic for initial lesions. The low pH of erosive fluids renders them undersaturated with respect to FAP as well; even when the fluoride concentration is increased no surface layer is formed.

(60). Thus, the effect to be expected of fluoride in 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 RYTÖMAA *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 IMFELD 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 LUSSI *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. Cariogenic 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 chemicals in the environment. Erosion affects dental enamel causing dissolution of the apatite crystals. Depending on the histological structure of enamel, erosion proceeds either by dissolving enamel prisms or by chemically wearing off the aprismatic surfaces. Once in dentin, the process continues via dentinal tubules and eventually involves the pulp tissue. A characteristic increase in tooth sensitivity to any outer stimuli is seen as a main symptom of erosion. However, if the process is slow, an increased sensitivity may be delayed, presumably because of accumulation of tertiary dentin in the pulp. Depending on the severity and longevity of erosion, the process may lead to a total destruction of the dentition. Finally, erosion is rarely a factor operating alone in causing tooth wear.

References

1. Pindborg JJ. *Pathology of the dental hard tissues.* Copenhagen, Munksgaard 1970, 294–325.
2. Hellström I. Anorexia nervosa – odontologiska problem. *Swed Dent J* 1974; **67**: 253–269.
3. Trygstad O. Bulimi – et liv uten kontroll. *Nord Med* 1986; **101**: 72–77.
4. Järvinen V, Meurman JH, Hyvärinen H, Rytömaa I. Dental erosion and upper gastrointestinal disorders. *Oral Surg Oral Med Oral Pathol* 1988; **65**: 298–303.
5. Meurman J, Toskala J, Nuutinen P, Klemetti E. Oral and dental manifestations in gastroesophageal reflux disease. *Oral Surg Oral Med Oral Pathol* 1994; **78**: 583–589.
6. Eccles JD, Jenkins WG. Dental erosion and diet. *J Dent* 1974; **2**: 153–156.
7. Rytömaa I, Meurman JH, Franssila S, Torkko H. Oral hygiene products may cause dental erosion. *Proc Finn Dent Soc* 1989; **85**: 161–166.
8. Meurman JH, Murtomaa H. Effect of effervescent vitamin C preparations on bovine teeth and on some clinical and salivary parameters in man. *Scand J Dent Res* 1986; **94**: 491–499.
9. Rogalla K, Finger W, Hannig M. Influence of buffered and unbuffered acetylsalicylic acid on dental enamel and dentine in human teeth: An *in vitro* pilot study. *Meth Find Exp Clin Pharmacol* 1992; **14**: 339–346.
10. Hannig M, Rogalla K, Albers H-K. Scanning electron microscopy study of the erosive action of acetylsalicylic acid on human dental enamel in comparison with other acidic agents. *Drug Invest* 1992; **4**: 484–491.
11. Hannig M, Albers H-K. Die erosive Wirkung von Acetylsalicylsäure auf Zahnschmelz und Dentin *in vitro*. *Dtsch Zahnärztl Z* 1993; **48**: 293–297.
12. Hannig M. Mikromorphologische Veränderungen an der Schmelzoberfläche durch verschiedene Säuren. *ZWR* 1993; **102**: 604–611.
13. Smith BGN, Robb ND. Dental erosion in patients with chronic alcoholism. *J Dent* 1989; **17**: 219–221.
14. Bruggen Cate JH ten. Dental erosion in industry. *Br J Ind Med* 1968; **25**: 249–266.
15. Westergaard J, Moe D, Pallesen U, Holmen L. Exaggerated abrasion/erosion of human dental enamel surfaces: a case report. *Scand J Dent Res* 1993; **101**: 265–269.
16. Savad EN. Enamel erosion... Multiple cases with a common cause(?) *J New Jersey Dent Assoc* 1982; **53**: 35–37.
17. Centers for disease control. *MMWR Weekly Reports* 1993; **32**: 361.
18. Linkosalo E, Markkanen S. Dental erosions in relation to lactovegetarian diet. *Scand J Dent Res* 1989; **93**: 436–441.
19. Buonocore MG. A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. *J Dent Res* 1955; **46**: 37–45.
20. Thylstrup A, Fejerskov O. Clinical and pathological features in dental caries. Enamel surface structure. In: Thylstrup A, Fejerskov O. (eds.). *Textbook of clinical cariology.* Munksgaard, Copenhagen 1994, 2nd ed., 112–115.
21. Meurman JH, Frank RM. Progression and surface ultrastructure of *in vitro* caused erosive lesions in human and bovine enamel. *Caries Res* 1991; **25**: 81–87.
22. Meurman JH, Frank RM. Scanning electron microscopic study of the effect of salivary pellicle on enamel erosion. *Caries Res* 1991; **25**: 1–6.
23. Meurman JH, Härkönen M, Näveri H, Koskinen J, Torkko H, Rytömaa I, Järvinen V, Turunen R. Experimental sports drinks with minimal dental erosion effect. *Scand J Dent Res* 1990; **98**: 120–128.
24. Rytömaa I, Meurman JH, Koskinen J, Laakso T, Gharazi L, Turunen R. *In vitro* erosion of bovine enamel

caused by acid drinks and other foodstuffs. *Scand J Dent Res* 1988; **96**: 324–333.
25. Silverstone LM, Saxton CA, Dogon IL, Fejerskov O. Variation in the pattern of acid etching of human dental enamel examined by scanning electron microscopy. *Caries Res* 1975; **9**: 373–387.
26. Smith AJ, Shaw L. Baby fruit juices and tooth erosion. *Br Dent J* 1987; **162**: 65–67
27. Featherstone JDB, Mellberg JR. Relative rate of progress of artificial caries lesions in bovine, ovine and human enamel. *Caries Res* 1981; **15**:109–114.
28. Xhonga FA, Wolcott RB, Sognnaes RF. Dental erosion II. Clinical measurements of dental erosion progress. *J Am Dent Assoc* 1972; **84**: 577–582.
29. Meurman JH, Drysdale T, Frank RM. Experimental erosion of dentin. *Scand J Dent Res* 1991; **99**: 457–462.
30. Järvinen V. *Hammaseroosio ja sen syyt* (Thesis). Helsinki. University Printing House 1990.
31. Lussi A, Schaffner M, Hotz P, Suter P. Dental erosion in a population of Swiss adults. *Community Dent Oral Epidemiol* 1991; **19**: 286–290.
32. Lussi A, Schaffner M, Hotz P, Suter P. Erosion der Zahnhartsubstanz. Epidemiologie, klinisches Erscheinungsbild, Risikofaktoren und Verhaltensregeln. *Schweiz Monatsschr Zahnmed* 1992; **102**: 321–329.
33. Lussi A, Jäggi T, Schärer S. The influence of different factors on in vitro enamel erosion. *Caries Res* 1993; **7**: 387–393.
34. Meurman JH, Rytömaa I, Kari K, Laakso T, Murtomaa H. Salivary pH and glucose after consuming various beverages including sugar-containing drinks. *Caries Res* 1987; **21**: 353–359.
35. Grobler SR, Senekal PJC, Kotze TJVW. The degree of enamel erosion by five different kind of fruits. *Clin Prev Dent* 1989; **11**: 23–28.
36. Grenby TH, Phillips A, Desai T, Mistry M. Laboratory studies of the dental properties of soft drinks. *Br J Nutr* 1989; **62**: 451–464.
37. Grobler SR, Senekal PJ, Laubscher JA. *In vitro* demineralization of enamel by orange juice, apple juice, Pepsi Cola and Diet Pepsi Cola. *Clin Prev Dent* 1990; **12**: 5–9.
38. Imfeld T. Evaluation of the cariogenicity of confectionery by intra-oral wire-telemetry. *Schweiz Mschr Zahnheilk* 1977; **87**: 450.
39. Elsbury WB. Hydrogen-ion concentration and acid erosion of the teeth. *Br Dent J* 1952; **93**: 177–179.
40. Miller CD. Enamel erosive properties of fruits and various beverages. *J Am Diet Assoc* 1952; **28**: 319–324.
41. Holloway PJ, Mellanby M, Steward RJC. Fruit drinks and tooth erosion. *Br Dent J* 1958; **104**: 305–309.
42. McCay OM, Will L. Erosion of molar teeth by acid beverages. *J Nutr* 1949; **39**: 313–324.
43. Stephen RM. Effects of different types of human foods on dental health in experimental animals. *J Dent Res* 1966; **45**:1551–1561.
44. Reussner GH, Coccodrilli G Jr, Thiessen R Jr. Effects of phosphates in acid containing beverages on tooth erosion. *J Dent Res* 1975; **54**: 365–370.
45. Martell AE, Smith RM. Other organic ligands. In: *Critical stability constants*. Plenum Press, New York, 1977: **3**: pp 161
46. Mistry M, Grenby TH. Erosion by soft drinks of rat molar teeth assessed by digital image analysis. *Caries Res* 1993; **27**: 21–25
47. Bibby EH, Mundorff SA. Enamel demineralization by snack foods. *J Dent Res* 1975; **54**: 1461–1470.
48. Zipkin J, McClure FJ. Salivary citrate and dental erosion. *J Dent Res* 1949; **28**: 613–626.
49. Shulman EH, Robinson HBG. Salivary citrate concentration and erosion of the teeth. *J Dent Res* 1948; **27**: 541–544.
50. Mannerberg F. Saliva factors in case of erosion. *Odontologisk Revy* 1963; **15**: 156–166.
51. Meurman JH. Symposium: dental erosion. *J Dent Res* 1989; **68**: Spec. Iss. S 20.
52. Wöltgens JHM, Vingerling P, de Blieck-Hogervors JMA, Bervoets DJ. Enamel erosion and saliva. *Clin Prev Dent* 1985; **7**: 8–10.
53. Gudmundsson K, Kristleifsson G, Theodors A, Holbrook WP. Tooth erosion, gastrointestinal reflux, and salivary buffer. *Oral Surg Oral Med Oral Pathol* 1995; **79**: 185–189.
54. Zahradnik RT, Moreno EC, Burke EJ. Effect of salivary pellicle on enamel subsurface demineralization in vitro. *J Dent Res* 1977; **55**: 664–670.
55. Voegel JC, Frank RM. Influence of salivary glycoproteins and fluoride on synthetic apatite dissolution. In: Leach SA (ed.) *Dental plaque and surface interactions in the oral cavity*. London, IRL Press, 1980, pp 301–311.
56. Koulourides T, Feagin F, Pigman W. Remineralization of dental enamel by saliva *in vitro*. *Ann N Y Acad Sci* 1965; **131**: 751–757.
57. Albert M, Grenoble DE. An *in-vivo* study of enamel remineralization after acid etching. *J South Carolina Dent Assoc* 1971; **39**: 747–751.
58. Mühlemann HR, Lenz H, Rossinsky K. Electron microscopic appearance of rehardening enamel. *Helv Odont Acta* 1964; **8**: 108–11.
59. Ten Cate JM, Featherstone JBD. Mechanistic aspects of the interactions between fluoride and dental enamel. *Crit Rev Oral Biol Med* 1991; **2**: 283–296.
60. Larsen MJ. Chemical events during tooth dissolution. *J Dent Res* 1990; **69**: 575–580.
61. Spencer IJ, Ellis LN. The effect of fluoride and grapefuit juice on the etching of teeth. *J Nutr* 1950; **42:** 107–15.
62. Xhonga FA, Sognnaes RF. Dental erosion: progress of erosion measured clinically after various fluoride applications. *J Am Dent Assoc* 1973; **87**: 1223–1228.
63. Sorvari R. *Sport drink studies with an animal model. Special reference to dental caries and erosion*. Thesis. Publications of the University of Kuopio. Medicine. *Original reports* 1989; 10.
64. Sorvari R, Meurman JH, Alakuijala P, Frank RM. Effect of fluoride varnish and solution on enamel erosion in vitro. *Caries Res* 1994; **28:** 227–232.
65. Schweitzer-Hirt CM. *Erosion und Abrasion des Schmelzes. Eine experimentelle Studie*. Thesis. University of Zurich, 1978.
66. Smith BGN, Knight JK. A comparison of patterns of tooth wear with etiological factors. *Br Dent J* 1984; **157**:16–19.
67. Lewis JK, Smith BGN. The relationship of erosion and attrition in extensive tooth tissue loss. Case report. *Brit Dent J* 1973; **135**: 400.
68. Sorvari R, Pelttari A, Meurman JH. Surface ultrastructure of rat molar teeth after experimentally induced erosion and attrition. *Caries Res* 1996 (in print).
69. Järvinen V, Rytömaa I, Meurman JH. Location of dental erosion in a referred population. *Caries Res* 1992; **26**: 391–396.
70. Hellström I. Oral complications in anorexia nervosa. *Scand J Dent Res* 1977; **85**: 71–86.
71. Michalek SM, McGhee JR. Oral streptococci with emphasis on *Streptococcus mutans*. In: McGhee JR, Michalek SM, Cassell GH (eds.) *Oral microbiology*. Haper & Row Publishers, Philadelphia, 1982, pp 679–690.