# Erosive tooth wear: Diagnosis, risk factors and prevention

## ADRIAN LUSSI, DDS, MS, ELMAR HELLWIG, DDS, DOMENICK ZERO, DDS, MS, & THOMAS JAEGGI, DDS

**ABSTRACT:** *Purpose:* To provide an overview on diagnosis, risk factors and prevention of erosive tooth wear, which is becoming an increasingly important factor when considering the long term health of the dentition. *Results:* Awareness of dental erosion by the public is still not widespread due to the cryptic nature of this slowly progressing condition. Smooth silky-glazed appearance with the absence of perikymata and intact enamel along the gingival margin, with cupping and grooving on occlusal surfaces are some typical signs of enamel erosion. In later stages, it is sometimes difficult to distinguish between the influences of erosion, attrition or abrasion during a clinical examination. Biological, behavioral and chemical factors all come into play, which over time, may either wear away the tooth surface, or potentially protect it. In order to assess the risk factors, patient should record their dietary intake for a distinct period of time. Based on these analyses, an individually tailored preventive program may be suggested to patients. It may comprise dietary advice, optimization of fluoride regimes, stimulation of salivary flow rate, use of buffering medicaments and particular motivation for non-destructive tooth brushing habits. The frequent use of fluoride gel and fluoride mouthrinse in addition to fluoride toothpaste offers the opportunity to minimize abrasion of tooth substance. (*Am J Dent* 2006;19:319-325).

**CLINICAL SIGNIFICANCE:** Since erosion, attrition and abrasion often occur simultaneously, all causative components must be taken into consideration when planning preventive strategies.

⊠: Prof. Dr. Adrian Lussi, Department of Preventive, Restorative and Pediatric Dentistry, School of Dental Medicine, Freiburgstrasse 7, CH - 3010 Bern, Switzerland. E-⊠: adrian.lussi@zmk.unibe.ch

## Introduction

There is some evidence that the presence of dental erosion is growing steadily. Hence, erosive tooth wear is becoming increasingly significant in the management of the long-term health of the dentition. As lifestyles have changed through the decades, the total amount and frequency of consumption of acidic foods and drinks have also changed. Soft drink consumption in the USA increased by 300% in 20 years,<sup>1</sup> and serving sizes increased from 185 g (6.6 oz) in the 1950s to 340 g (12 oz) in the 1960s and 570 to g (20 oz) in the late 1990s. Between 56% and 85% of children at school in the USA (circa 1995), consumed at least one soft drink daily, with the highest amounts ingested by adolescent males. Of this group, 20% consumed four or more servings daily.<sup>2</sup> Particularly, studies in children and adults have shown that patients with more than four dietary acid intakes per day is associated with the presence and the progression of erosion when other risk factors (such as holding the drink in the mouth) are present.<sup>3,4</sup>

National dental surveys are not routinely undertaken and when conducted have seldom included measures of tooth wear, specifically erosion. Erosion was first included in the U.K. childrens' dental health survey in 1993 and is repeated periodically. The prevalence of erosion was shown to have increased from the time of the children's dental health survey in 1993 to the study of 4 to 18 year-olds in 1996/1997.<sup>5</sup> There was a trend towards a higher prevalence of erosion in children aged between 3.5 and 4.5 years; and in those who consumed carbonated drinks on most days, compared with toddlers consuming these drinks less often. In another U.K. study, 1308 children were examined at the age of 12 years and 2 years later. In ths study, 4.9% of the subjects at baseline and 13.1% 2 years later had deep enamel or dentin lesions. Twelve percent of erosion–free children at 12 years developed the condition over the subsequent 2 years. New or more advanced lesions were seen in 27% of the children over the study period.<sup>6</sup>

Awareness of dental erosion by the public is still not widespread, and the diagnosis of erosion by dentists and the differentiation from abrasion, attrition and abfraction may be difficult. In a survey in England, 34% of children surveyed were aware of tooth erosion but only 8% could recall their dentist mentioning the condition.<sup>7</sup> What is considered an acceptable amount of wear is dependent on the anticipated lifespan of the teeth, which is different for primary teeth compared to permanent teeth. However, erosive damage to the permanent teeth occurring in childhood may compromise the growing child's dentition for their entire lifetime and may require repeated and increasingly expensive restorations.<sup>8</sup>

Therefore, it is important that diagnosis of the tooth wear process in children and adults is made early and adequate preventive measures are undertaken. Early intervention can only be initiated when the risk factors as well as the biological and behavioral modifying factors are taken into account.

This review provides an overview for the clinical management of erosive tooth wear which includes the early diagnosis and monitoring, identification of risk factors and strategies for its prevention.

## Diagnosis

Diagnosis in the early forms of erosive tooth wear may be easily overlooked, as it is accompanied by few signs and fewer if any symptoms. There is no device available in routine dental practice for the specific detection of dental erosion. Therefore, clinical appearance is the most important feature for dental professionals to diagnose dental erosion. This is of particular importance in the early stage of erosive tooth wear. The appearance of smooth silky-glazed appearance with the absence of perikymata and intact enamel along the gingival



Fig. 1. Severe facial erosive tooth wear. Age of patient: 25 years. Known etiological factors: regurgitation, on average seven acidic intakes per day.



Fig. 3. Severe oral erosive tooth wear. Note the intact enamel along the gingival margin. Age of patient: 28 years. Known etiological factor: vomiting over years.



Fig. 2. Facial erosive tooth wear. Age of patient: 29 years. Note the location of the defect. Known etiological factor: gastroesophageal reflux.



Fig. 4. Severe oral erosion. Note the composite filling rising above the adjacent tooth surface. Age of patient: 25 years. Known etiological factors: regurgitation, on average seven acidic intakes per day (same patient as in Fig. 1).



Fig. 5. Severe oral and occlusal erosive tooth wear. Note the worn cusps and the composite filling rising above the level of the adjacent tooth surface. Age of patient: 25 years. Known etiological factors: regurgitation, on average seven acidic intakes per day (same patient as in Fig. 1).

margin, with cupping and grooving on occlusal surfaces are some typical signs of enamel erosion. It has been hypothesized that the preserved enamel band along the oral and facial gingival margin could be due to some plaque remnants, which could act as a diffusion barrier for acids. This phenomenon could also be due to an acid neutralizing effect of the sulcular fluid.<sup>9</sup> In the more advanced stages, further changes in the morphology can be found. These changes result in further



Fig. 6. Severe occlusal erosive tooth wear. Note the composite and amalgam fillings rising above the level of the adjacent tooth surface. Age of patient: 25 years. Known etiological factors: regurgitation, on average seven acidic intakes per day (same patient as in Fig. 1).

flattening of the surface or develop ing a concavity in enamel, the width of which clearly exceeds its depth. In severe cases, the whole occlusal morphology of the tooth disappears. Figures 1-6 show typical patterns of the erosive tooth wear process. It is difficult to diagnose erosion at an early stage, and it can be very difficult to determine if dentin is exposed or not.<sup>10</sup> It is possible to use disclosing agents to render dentin



Fig. 7. Severe oral and occlusal erosive tooth wear. Note the approximal caries lesion on the canine. Age of patient: 29 years. Known etiological factor: gastroesophageal reflux (same patient as in Fig. 2).

involvement visible. In later stages, it is sometimes difficult to distinguish between the influences of erosion, attrition or abrasion during a clinical examination. Indeed, these conditions may occur simultaneously. Tooth surfaces of patients with active (unstained) erosion have no caries. However, at sites where plaque accumulation is possible (*e.g.* approximal), caries may also occur in patients with erosion (Fig. 7). The most commonly reported areas with wear are occlusal surfaces. These surfaces are also associated with attrition and abrasion, and it can be difficult to separate what is being caused by erosion from what is being caused by other tooth wear factors.<sup>11</sup> Therefore, a modern preventive strategy suggests training of dentists in early detection and monitoring of the process is needed.

Only with these capabilities can dentists comply with their responsibilities for providing adequate care for patients. Often patients themselves do not seek treatment until the condition is at an advanced stage, when the teeth become hypersensitive or when the esthetics are affected. This is particularly true for patients who suffer from anorexia nervosa or bulimia.

#### **Risk factors**

When an acidic solution comes in contact with enamel, it has to diffuse first through the acquired pellicle, and only thereafter can it interact with enamel. The acquired pellicle is an organic film, free of bacteria, covering oral hard and soft tissues. It is composed of mucins, glycoproteins and proteins, including several enzymes.<sup>12</sup> On the surface of enamel, the hydrogen ion component of the acid will start to dissolve the enamel crystal. First, the prism sheath area and then the prism core are dissolved, leaving the well known honeycomb appearance.<sup>13</sup> Fresh, unionized acid will then eventually diffuse<sup>14</sup> into the interprismatic areas of enamel and dissolve further mineral in the region underneath the surface.<sup>15,16</sup> This will lead to an outflow of ions (dissolution) and subsequently to a local pH rise in the tooth substance immediately below and in the liquid surface layer adjacent to the enamel surface.<sup>16</sup> The events in dentin are in principle the same but are even more complex. Due to the high content of organic material, the diffusion of the demineralizing agent deeper into the region and the outward flux of tooth mineral are hindered by the organic dentin matrix.<sup>17</sup> It has been assumed that the organic dentin matrix has a sufficient buffer capacity to retard further deminer-



Fig. 8. The different factors for the development of erosive tooth wear showing its multifactorial etiology (Lussi  $^{65}$ )

alization and that chemical or mechanical degradation of the dentin matrix promotes demineralization.<sup>18,19</sup>

These erosive processes are halted when no new acids and/or chelating substances are provided. An increase in agitation (*e.g.* when a drink is swished around the mouth) will enhance the dissolution process because the solution on the surface layer adjacent to enamel will be readily renewed.<sup>20</sup> Further, the amount and the composition of an acidic drink in the mouth in relation to the amount and flow of saliva present will modify the process of dissolution.<sup>21</sup> In summary, enamel may, in the initial stage, be softened by acid. When the attack is persistent, the surface is eventually etched away. In both stages a dissolution of tooth substance underneath the surface is possible.

There are many factors which are involved in and interact with erosive tooth wear. Figure 8 shows the different predisposing factors and etiologies of the erosive condition. Biological, behavioral and chemical factors are interacting with the tooth surface, which over time, may either wear it away, or indeed protect it. The interplay of all these factors is crucial and helps explain why some individuals exhibit more erosion than others, even if they are exposed to exactly the same acid challenge in their diets. As known in the carious process,<sup>2</sup> other factors listed in the outer circle will further influence the whole process of erosion development or defense. Comprehensive knowledge of the different risk factors is a prerequisite to initiate adequate preventive measures. Patients with erosive tooth wear are often not aware of, and may not know, the erosive potential of some drinks and foodstuffs. Only when a comprehensive case history is undertaken will the risk factors be revealed. It is advisable to have patients record their complete dietary intake for four consecutive days. The time of day and quantity of all ingested foods and beverages, including dietary supplements, should be recorded. Both weekdays and weekends should be included, as dietary habits during weekends may be considerably different from those during weekdays. This dietary and behavior record should be sent to

#### Etiological factors for erosions checklist

• Take case history (medical and dental)

Diagnose the severity and the site specific distribution

• Record the dietary intake over four days and estimate the erosive potential

• Question the patient for specific factors which they may not be aware of:

*Diet:* Herbal teas, acidic candies, alcohol, sports drinks, effervescent vitamin C tablets, *etc.* 

*Gastric symptoms:* Vomiting, acid taste in the mouth and gastric pain (especially when awakening), stomach ache, any sign of anorexia nervosa.

*Drugs:* Alcohol, tranquillizer, anti-emetics, anti-histamines, lemonade tablets. (Change of acidic or saliva reducing drugs is possible in consultation with patient's physician.)

• Determine the flow rate and buffering capacity of saliva

• Reveal the oral hygiene habits, abrasivity of toothpaste and technique

• Question patient for occupational exposure to acidic environments

• Question patient for X-ray therapy of the head and neck area

• Assess further progression with silicone impressions, study models, and/or photographs

Fig. 9. Checklist used to unveil etiological factors for erosions (in part from from Lussi *et al*,  $^{9.66}$ 

the dentist prior to the next appointment. A thorough knowledge of the erosive potential of drinks and foodstuffs is needed by the dentist, to determine the patient's risk and to bring it into context with the behavioral and biological factors. The different erosive potential of about 50 foodstuffs and beverages can be found in a reference text on dental erosion.<sup>23</sup> This book also shows that yoghurt (pH 4.1) with its high content of calcium and phosphate shows no erosive capacity. The calcium (and phosphate) contents are important as they influence the degree of saturation with respect to enamel or dentin. Indeed, adding of calcium to orange juice, to sports drinks (e.g. Lucozade) or other drinks (e.g. blackcurrant juice drink) has been shown to reduce the erosive effect of the drinks. The drinking method (holding, sipping, gulping, nipping, sucking) may affect tooth-surface pH. It follows that holding or long-sipping of erosive beverages should be avoided, as it causes low pH-values for a prolonged period of time.<sup>24</sup>

Knowing the risk factors and patient symptoms, juxtaposed with the wishes, hopes and possibilities of the individual patient, enables the dentist to initiate adequate preventive and therapeutic measures. Some of these risk factors have been postulated to act as predictors of future dental erosion.<sup>25</sup> When a restoration is inevitable, in all situations, the preparations have to follow the principles of minimally invasive treatment. In no case may early diagnosis of erosive tooth wear be an excuse for a restoration. Instead preventive measures must be initiated.

### Prevention

After the analysis of the dietary report provided by the patient, further questioning using the information listed in Fig. 9 should be undertaken. It has to be kept in mind that acidic candies and herbal teas (such as black currants, loganberries, cranberries, lemons and raspberries) may have an erosive potential and aggravate erosive lesions.<sup>26-29</sup> Possible intrinsic acid exposure should also be taken into account. Based on these analyses, an appropriate preventive program may be suggested

## Patient reccomendations for high risk dental erosion patients

• Reduce acid exposure by reducing the frequency, and contact time of acids (main meals only).

• Do not hold or swish acidic drinks in your mouth. Avoid sipping these drinks.

• Avoid tooth brushing immediately after an erosive challenge (vomiting, acidic diet). Instead, use a fluoride containing mouth rinse, a sodium bicarbonate (baking soda) solution, milk or food such as cheese or sugar-free yoghurt. If none of the above are possible, rinse with water.

• Avoid tooth brushing immediately before an erosive challenge, as the acquired pellicle provides protection against erosion.

• Use a soft toothbrush and low abrasion fluoride containing toothpaste. High abrasive toothpastes may remove the pellicle. Avoid toothpastes with very low pH.

• Gently apply periodically concentrated topical fluoride (slightly acidic formulations are preferable as they form CaF<sub>2</sub> at a higher rate).

• Consider using modified acid beverages with no or reduced erosive potential.

• After acid intake, stimulate saliva flow with chewing gum or lozenges. The use of a non acidic sugar-free lozenge may be more advisable, since gum chewing may have an abrasive effect on softened tooth structure.

• Use chewing gum to reduce postprandial reflux.

• Refer patients or advise them to seek appropriate medical attention (gastroenterologist and/or a psychologist) when intrinsic causes of erosion are involved.

Fig. 10. Recommendations for patients at high risk for dental erosion (modified from  $^{9,21,66}$ )

to patients (Fig. 10). However, the advice has to be made on an individually tailored basis, so not all points listed in Fig. 10 are appropriate for every patient. The aim of this program is to reduce acid exposure by decreasing the frequency of ingestion of potentially harmful drinks and foodstuffs as well as minimizing contact time with the teeth by rapid consumption of them rather than sipping or swishing. In addition, reflux/vomiting should be controlled and the patient's fluoride regime should be optimized. As discussed previously, various processes cause the degradation of tooth substance. Erosion, attrition and abrasion often occur concurrently, though usually one of these factors may be predominant. When giving preventive instructions, all of the causative components must be taken into consideration. Patients suffering from intrinsic erosions, depending on the cause, need further care such as antacids, psychological therapy or even surgical intervention. Adequate preventive measures will often slow down progression of the erosion and reduce the need for immediate restorations. However, assessment of erosion change over time is important; photographs and study casts are simple means of monitoring progression.

Acid-eroded enamel is more susceptible to abrasion and attrition than intact enamel. The thickness of the softened enamel that is removed following different abrasive procedures varies in different investigations, depending on the experimental conditions.<sup>9,30-34</sup> Some of these studies show an approximately 10-fold increase of softened enamel to toothbrush abrasion compared to unsoftened enamel.

In the 1970s, Graubart *et al*<sup>35</sup> showed an *in vitro* protective effect of a 2% sodium fluoride solution on the erosive process. Less wear of softened teeth was produced *in vitro* in the presence of fluoride toothpaste than in the presence of non-

fluoride toothpaste with an otherwise identical formulation.<sup>36</sup> In recent years, more studies using different fluoride formulations, e.g. sodium fluoride, acidulated phosphate fluoride, stannous fluoride, amine fluoride or titanium tetrafluoride showed a protective effect in vitro.<sup>37-40</sup> Stannous fluoride showed better protection than sodium fluoride when teeth were immersed in 0.1 M HCl with a pH of 2.2. However, when the pH was further lowered to 1.2 (which is lower than the acid-content of the stomach) there was no protection.<sup>40</sup> Titanium fluoride was found to be a more effective pre-treatment agent against citric acid erosion when compared with sodium fluoride.<sup>38</sup> It appears that in vitro, highly concentrated fluoride gels demonstrated the best protection against further erosion/abrasion. Attin et  $al^{41}$ showed in vitro that a slightly acidic gel (pH 4.5) had a higher abrasion resistance compared to gels which are either unfluoridated or neutral. In this study samples were alternately stored in Sprite light (a citric-acid containing soft drink) (5 minutes) and in artificial saliva (1 minute) four times.<sup>4</sup> Unfortunately, there are only a few case reports or in situ studies dealing with the protective effect of fluoride. Lussi et  $al^{42}$  compared *in situ* the impact of different fluoride procedures on the prevention of toothbrush abrasion prior to and after 3 minutes of enamel softening. After exposing the tooth slabs to the oral milieu for 60 minutes, the samples were brushed for 15 seconds with medium abrasive toothpaste and the substance loss was measured. The least amount of toothbrush abrasion occurred when using a slightly acidic sodium/amine fluoride gel for 1 minute before softening the enamel, when compared to all of the other experimental groups. There was, however, only partial protection. This could be as a result of some incorporation of material into and/or deposition of material on the enamel surface, most probably as a  $CaF_2$ -like material,<sup>37</sup> which will lead to less softening than without this layer. This CaF<sub>2</sub>-layer is considered stable at a neutral pH, but may dissolve as the pH drops during erosive attacks. This deposited CaF<sub>2</sub>-like layer may be dissolved readily by most acidic beverages.43,44

Jensen *et al*<sup>45</sup> published a case report investigating the importance of fluoride in maintaining the dentition. In this study, teeth were obtained from a deceased anorexic and bulimic patient. Just prior to death, saliva analyses and enamel biopsies were made, before and after a 3-week regimen of daily rinsing with 0.05% NaF. After 4 years of daily regurgitation, there was an almost normal thickness of the enamel present on the palatal surfaces of the anterior teeth, with normal hardness measurements 10 µm away from the outer surface. SEM micrographs showed an irregular topography, with crystalline deposits rich in calcium, phosphate and fluoride. The tooth surfaces exposed to gastric juice were more reactive to uptake of additional fluoride when given as a daily rinse, than the more protected surfaces. This case report suggests that frequent daily use of fluoride products can minimize the erosive effect of gastric contents on tooth enamel. Ganss et al<sup>46</sup> showed in an in situ model that treatments with fluoridated toothpaste in combination with F solution and slightly acidic F gel could significantly reduce tooth erosion by 50% and 90% on enamel and 10% and 55% on dentin, respectively. Lussi et al<sup>47</sup> studied in situ the effect of fluoride rinsing on the prevention of toothbrush abrasion of softened enamel. A single rinse with sodium/amine fluoride solutions before or after erosion had no significant effect on subsequent abrasion by tooth brushing for 30 seconds. From the above it is clear that further *in situ* studies are needed. However, it must be noted that although essentially most of toothpastes globally are fluoridated, the incidence of dental erosion appears to be on the increase. Thus, the effectiveness of fluoride in concentrations usual for toothpastes against dental erosion appears to be limited to specific models and may be dependent on the fluoride concentrations. The compliance for beneficial but demanding regimes as mentioned above<sup>46</sup> requires patients that are very motivated and dedicated. It has to be determined whether such strict regimes are feasible in daily practice.

Exposure to saliva has been shown to be effective for rehardening eroded enamel. The mechanism for this is thought to be that once the erosive agent is neutralized or cleared from the tooth surface, the deposition of salivary calcium and phosphate may lead to rehardening of the acid softened enamel.<sup>48,49</sup> Enamel specimens eroded by citric acid for 2 hours were immersed in artificial saliva and showed partial rehardening after 1 to 4 hours. These specimens remineralized for 6 to 24 hours and demonstrated complete rehardening.<sup>50</sup> Saliva is an important biological factor in the prevention of erosion. It has been speculated that saliva stimulation will enhance the formation of the acquired salivary pellicle. It is known that the pellicle forms rapidly and has some protective effect against erosion.<sup>51-54</sup> Procedures that remove or reduce the thickness of the salivary pellicle may compromise its protective properties and therefore accelerate the erosion process. Procedures such as toothbrushing with abrasive dentifrice products, professional cleaning with prophylaxis paste, and tooth whitening will all remove or weaken the pellicle and may render teeth more susceptible to erosion.<sup>21</sup> Acidic beverages may interfere with the pellicle formation and thus further modify the protective barrier.55

As previously discussed, various processes may cause degradation of the tooth substance. When giving preventive instructions, all of the possible causative components must be taken into consideration. Other behaviors that either stimulate salivary flow (such as chewing gum), or directly help neutralize acids (such as rinsing with sodium bicarbonate), may counter the destructive effects of dietary acids.<sup>25,56</sup> There is the possibility that chewing gum may have an abrasive effect on softened tooth structure. Thus, the gentle use of non-acidic, saliva-stimulating lozenges may be preferable to chewing gum.<sup>21</sup> However, chewing gum after a meal helps to reduce postprandial esophageal acid exposure.<sup>57,58</sup> It has also been suggested that chewing gum might be a treatment option for some patients with symptomatic reflux.<sup>59,60</sup>

For individuals who are at high risk for erosive tooth wear and those with active erosion, it is suggested that tooth brushing should be postponed after consumption of erosive foodstuffs or beverages, in order to minimize the risk for potential tooth loss. Another possibility is to gently apply fluoride prior to the erosive attack, *e.g.*, application before bedtime when regurgitation is the problem. This has to be achieved carefully so that the protecting pellicle is not disturbed. For subjects prone to caries, the risk of enhancing the progression of carious lesions by postponing tooth brushing may be too great because of the rapid decrease in plaque pH after ingestion of sugarcontaining foods or beverages. A measure that can be beneficial for both erosion and caries is rinsing with fluoride solution, thereby enhancing remineralization and stimulating salivary secretion. Adhesive systems may protect enamel and dentin from acid attack and brushing abrasion for a limited period of time.<sup>61-63</sup> There is clearly a need for improvement and development of substances with a high protective capacity against the erosive/abrasive insult.

This overview has described the importance of correct diagnosis and prevention of erosive tooth wear. A more detailed analysis can be found in a recently published book.<sup>64</sup>

Dr. Lussi is Professor and Head, and Dr. Jaeggi is Assistant Professor, Department of Preventive, Restorative and Pediatric Dentistry, School of Dental Medicine, University of Bern, Bern, Switzerland. Dr. Zero is Professor and Head, Department of Preventive and Community Dentistry, Indiana University, School of Dentistry, Indianapolis, Indiana, USA. Dr. Hellwig is Professor and Head, Department of Operative Dentistry and Periodontology, University Clinic of Dentistry, Freiburg, Germany.

#### References

- Calvadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. Archs Dis Child 2000;83:18-24.
- Gleason P, Suitor C. Children's diets in the mid-1990s: Dietary intake and its relationship with school meal participation. Alexandria, VA, USA:US Department of Agriculture, Food and Nutrition Service, Office of Analysis, Nutrition and Evaluation, 2001.
- 3. O'Sullivan EA, Curzon MEJ. A comparison of acidic dietary factors in children with and without dental erosion. *J Dent Child* 2000;67:186-192.
- Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. *Caries Res* 2000;34;182-187
- Nunn JH, Gordon PH, Morris AJ, Pine CM, Walker A. Dental erosion changing prevalence? A review of British National childrens' surveys. *Int J Paediatr Dent* 2003;13:98-105.
- Dugmore CR, Rock WP. The progression of tooth erosion in a cohort of adolescents of mixed ethnicity. *Int J Paediatr Dent* 2003;13:295-303.
- Dugmore CR, Rock WP. Awareness of tooth erosion in 12 year-old children and primary care dental practitioners. *Community Dent Health* 2003;20:223-227.
- Lussi A, Jaeggi T. Dental erosion in children. In Lussi A. Dental erosion: From diagnosis to therapy. Basel: Karger 2006; 140-151.
- Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. *Caries Res* 2004;38 (Suppl 1):34-44.
- Ganss C, Klimek J, Lussi A. Accuracy and consistency of the visual diagnosis of exposed dentine on worn occlusal/incisal surfaces. *Caries Res* 2006;40:208-212.
- 11. Bartlett D. The implication of laboratory research on tooth wear and erosion. *Oral Dis* 2005;11:3-6.
- 12. Hannig C, Hannig M, Attin T. Enzymes in the acquired enamel pellicle. *Eur J Oral Sci* 2005;113:2-13.
- Meurman JH, Frank RM. Scanning electron microscopic study of the effect of salivary pellicle on enamel erosion. *Caries Res* 1991;25:1-6.
- Featherstone JDB, Rodgers BE. Effect of acetic, lactic and other organic acids on the formation of artificial carious lesions. *Caries Res* 1981;15:377-385.
- Eisenburger M, Hughes J, West NX, Shellis RP, Addy M. The use of ultrasonication to study remineralisation of eroded enamel. *Caries Res* 2001;35:61-66.
- Lussi A, Hellwig E. Erosive potential of oral care products. *Caries Res* 2001;35:52-56.
- Hara AT, Ando M, Cury JA, Serra MC, Gonzalez-Cabezas C, Zero DT. Influence of the organic matrix on root dentine erosion by citric acid. *Caries Res* 2005;39:134-138.
- Ganss C, Klimek J, Starck C. Quantitative analysis of the impact of the organic matrix on the fluoride effect on erosion progression in human dentine using longitudinal microradiography. *Arch Oral Biol* 2004;49:931-935.
- Kleter GA, Damen JJ, Everts V, Niehof J, Ten Cate JM. The influence of the organic matrix on demineralization of bovine root dentin *in vitro*. J Dent Res 1994;73:1523-1539.
- Shellis RP, Finke M, Eisenburger M, Parker DM, Addy M. Relationship between enamel erosion and liquid flow rate. *Eur J Oral Sci* 2005;113:232-238.
- 21. Zero D, Lussi A. Erosion. Chemical and biological factors of importance to

the dental practitioner. Int Dent J 2005;55:285-290.

- Fejerskov O. Changing paradigms in concepts on dental caries: Consequences for oral health care. *Caries Res* 2004;38:182-191.
- Lussi A, Jaeggi T. Extrinsic causes of erosion. Chemical factors. In: Lussi A. Dental erosion: From diagnosis to therapy Basel: Karger: 2006, 77-87.
- Johansson AK, Lingström P, Imfeld T, Birkhed D. Influence of drinking method on tooth-surface pH in relation to dental erosion. *Eur J Oral Sci* 2004;112:484-489.
- Amaechi BT, Higham SM. Dental erosion: Possible approaches to prevention and control. J Dent 2005;33:243-252.
- Lussi A, Portmann P, Burhop B. Erosion on abraded dental hard tissues by acid lozenges: An in situ study. Clin Oral Investig 1997;1:191-194.
- Behrendt A, Oberste V, Wetzel WE. Fluoride concentration and pH of iced tea products. *Caries Res* 2002;36:405-410.
- Phelan J, Rees J. The erosive potential of some herbal teas. J Dent 2003;31:241-246.
- Jensdottir T, Nauntofte B, Buchwald C, Bardow A. Effects of sucking acidic candy on whole-mouth saliva composition. *Caries Res* 2005;39:468-474.
- Davis WB, Winter PJ. The effect of abrasion on enamel and dentine after exposure to dietary acid. Br Dent J 1980;148:253-256.
- Kelly MP, Smith BGN. The effect of remineralizing solutions on tooth wear in vitro. J Dent Res 1988;16:147-149.
- Jaeggi T, Lussi A. Toothbrush abrasion of erosively altered enamel after intraoral exposure to saliva - An *in situ* study. *Caries Res* 1999;33:455-461.
- Attin T, Buchalla W, Gollner M, Hellwig E. Use of variable remineralization periods to improve the abrasion resistance of previously eroded enamel. *Caries Res* 2000;34:48-52.
- Attin T, Knofel S, Buchalla W, Tutuncu R. *In situ* evaluation of different remineralization periods to decrease brushing abrasion of demineralized enamel. *Caries Res* 2001;35:216-222.
- Graubart J, Gedalia I, Pisanti S. Effects of fluoride pretreatment *in vitro* on human teeth exposed to citrus juice. *J Dent Res* 1972;51:1677.
- Bartlett DW, Smith BG, Wilson RF. Comparison of the effect of fluoride and non-fluoride toothpaste on tooth wear *in vitro* and the influence of enamel fluoride concentration and hardness of enamel. *Br Dent J* 1994;176:346-348.
- Ganss C, Klimek J, Schäfer U, Spall T. Effectiveness of two fluoridation measures on erosion progression in human enamel and dentine *in vitro*. *Caries Res* 2001;35:325-330.
- van Rijkom H, Ruben J, Vieira A, Huysmans MC, Truin G-J, Mulder. Erosion-inhibiting effect of sodium fluoride and titanium tetrafluoride *in vitro*. *Eur J Oral Sci* 2003;111:253-257.
- Hughes JA, West NX, Addy M. The protective effect of fluoride treatments against enamel erosion in vitro. J Oral Rehabil 2004;31:357-363.
- Willumsen T, Øgaard B, Hansen BF, Rølla G. Effects from pretreatment of stannous fluoride versus sodium fluoride on enamel exposed to 0.1 M or 0.01 M hydrochloric acid. *Acta Odontol Scand* 2004;62:278-281.
- Attin T, Deifuss H, Hellwig E. Influence of acidified fluoride gel on abrasion resistance of eroded enamel. *Caries Res* 1999;33:135-139.
- Lussi A, Jaeggi T, Schaffner M. Prevention and minimally invasive treatment of erosions. Oral Health Prev Dent 2004;2:321-325.
- Larsen MJ. Prevention by means of fluoride of enamel erosion as caused by soft drinks and orange juice. *Caries Res* 2001;35:229-234.
- Larsen MJ, Richards A. Fluoride is unable to reduce dental erosion from soft drinks. *Caries Res* 2002;36:75-80.
- Jensen OE, Featherstone JD, Stege P. Chemical and physical oral findings in a case of anorexia nervosa and bulimia. J Oral Pathol 1987;16:399-402.
- Ganss C, Klimek J, Brune V, Schurmann A. Effects of two fluoridation measures on erosion progression in human enamel and dentine *in situ*. *Caries Res* 2004;38:561-566.
- Lussi A, Jaeggi T, Gerber C, Megert B. Effect of amine/sodium fluoride rinsing on toothbrush abrasion of softened enamel *in situ. Caries Res* 2004;38:567-571.
- Gedalia I, Dakuar A, Shapira L, Lewinstein I, Goultschin J, Rahamim E. Enamel softening with Coca-Cola and rehardening with milk or saliva. *Am J Dent* 1991;4:120-122.
- Amaechi BT, Higham SM. Eroded lesions remineralisation by saliva as a possible factor in the site - specificity of human dental erosion. *Arch Oral Biol* 2001;46:697-703.
- Eisenburger M, Addy M, Hughes JA, Shellis RP. Effect of time on the remineralisation of enamel by synthetic saliva after citric acid erosion. *Caries Res* 2001;35:211-215.
- Nieuw Amerongen AV, Oderkerk CH, Driessen AA. Role of mucins from human whole saliva in the protection of tooth enamel against demineralization *in vitro*. *Caries Res* 1987;21:297-309.
- 52. Meurman JH, Frank RM. Scanning electron microscopic study of the effect

of salivary pellicle on enamel erosion. Caries Res 1991;25:1-6.

- Hannig M, Balz M. Influence of *in vivo* formed salivary pellicle on enamel erosion. *Caries Res* 1999;33:372-379.
- Hannig M, Fiefiger M, Guntzer M, Dobert A, Zimehl F, Nekrashevych Y. Protective effect of the *in situ* formed short-term salivary pellicle. *Arch Oral Biol* 2004;49:903-910.
- 55. Finke M, Parker DM, Jandt KD. Influence of soft drinks on the thickness and morphology of *in situ* acquired pellicle layer on enamel. *J Colloid Unterface Sci* 2002;251:263-270.
- Amaechi BT, Higham SM, Edgar WM. Influence of abrasion in clinical manifestation of human dental erosion. J Oral Rehabil 2003;30:407-413.
- Avidan B, Sonnenberg A, Schnell TG, Sontag SJ. Walking and chewing reduce postprandial acid reflux. *Aliment Pharmacol Ther* 2001;15:151-155.
- Moazzez R, Bartlett D, Anggiansah A. The effect of chewing sugar-free gum on gastro-esophageal reflux. J Dent Res 2005;84:1062-1065.
- Von Schönfeld J, Hector M, Evans DF, Wingate DL. Oesophageal acid and salivary secretion: Is chewing gum a treatment option for gastrooesopha-

geal reflux? Digestion 1997;58:111-114.

- Smoak BR, Koufman JA. Effects of gum chewing on pharyngeal and esophageal pH. Ann Otol Rhinol Laryngol 2001;110:1117-1119.
- Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The measurement and prevention of erosion. J Dent 2001;29:395-400.
- Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The surface effects of erosion and abrasion on dentine with and without a protective layer. Br Dent J 2004;196:351-354.
- Schneider F, Hellwig E, Attin T. Protection of dentin from acids and abrasion by adhesives. *Deutsche Zahnärztl Z* 2002;57:302-306 (In German).
- 64. Lussi A. Dental erosion: from diagnosis to therapy. Basel: Karger 2006.
- Lussi A. Erosive tooth wear A multifactorial condition of growing concern and increasing knowledge. In: A. Lussi. *Dental erosion: from diagnosis to therapy*. Basel: Karger 2006, pp 1-8.
- Lussi A, Hellwig E. Risk assessment and preventive measures. In: A. Lussi. Dental erosion: from diagnosis to therapy. Basel: Karger 2006; 190-199.

## IS YOUR ADDRESS CHANGING? Please let us know!

*Subscribers Only*: Please attach a current mailing label or photocopy of your label, and complete the form below with your new address. Send to: Subscription Department, American Journal of Dentistry, 318 Indian Trace, #500, Weston, FL 33326, U.S.A.

Attach "old address" label, or photocopy of mailing label here:

	L	 	 i
New address:	As of what date?: _	 	
Name			
Address:			