Dental Erosion and Its Clinical Management

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Preface

Dental erosion, otherwise known as erosive tooth wear, is the loss of tooth tissue through dissolution by acid of intrinsic (gastric) and extrinsic (dietary or environmental) sources. There has been a growing concern for the increasing global prevalence of this dental disorder in all age groups. Changes in dietary, social, and oral hygiene habits commonly provide important explanations for a true increased prevalence of this condition. Continued destruction of the dental hard tissue in an uncontrolled erosive tooth wear may lead to severe tissue loss with exposure of the dentin, hypersensitivity, unpleasant appearance, and reduced masticatory function. Thus it is now generally agreed that prevention of further tooth wear should form the basis of any ongoing lifelong dental management.

Today, the etiology and pathogenesis, the factors that modify erosion manifestation, agents, and methods that can be used to control and prevent erosion, and the different parameters that may affect the management of dental erosion, have been established through numerous researches. It is now time for these information to be distilled into an accessible, practical, and clinically focused format to be used by dental practitioners, educators, and students as well as other health professionals for evidence-based clinical management of dental erosion and establishment of preventive programs to control the prevalence of this disorder.

The aim of this 16-chapter book is to present the dental practitioners, other health care professionals, and students with evidence-based clinical guidelines for the management of erosive tooth wear. The book is in two sections: the science section informs the reader of the causes and pathogenesis as well as the prevalence of tooth wear due to acid erosion, while the clinical practice section details the various treatment and preventive strategies for dental erosion management. Also included in the book are topics on the etiology, prevalence, and management of dentin hypersensitivity, and the restoration of worn dentition and noncarious cervical lesions. The book was concluded with a chapter on the maintenance care cycle (recall visits) as well as outcomes measures.

Chapter 7 presents the Dental Erosive Wear Risk Assessment (DEWRA) form, the first ever tool for assessment of an individual's risk of developing dental erosion, with guide for a personalized management of the individual's erosive tooth wear. Chapters 8 and 9, respectively, present the patient's and the practitioner's responsibilities in the management of the patient's erosive tooth wear. These information may be repeated in a summary format in Chaps. 10, 11, and 12, which detail the

management of patients with dental erosion as a complication of their medical problems such as gastroesophageal reflux disease and eating disorders. All chapters end with a comprehensive list of references that provided the scientific evidence in support of the recommended clinical management strategies, thus enabling the reader to consult the original articles for more details.

The efforts and enthusiasm of our international experts from around the globe who contributed the various chapters in this book as well as the professional skills of our publishers are highly appreciated.

San Antonio, TX Bennett T. Amaechi

Contents

Part I

Science

Dental Erosion: Prevalence, Incidence and Distribution

1

D.H.J. Jager

Abstract

Dental erosion is one of the most common dental diseases and it is a growing problem. Numerous epidemiological studies have investigated the prevalence of dental erosion. For these studies different cross sections of the population are investigated. Large differences were found between countries, geographic locations and age groups. Most prevalence data is available from European studies and it is estimated that 29 % of the adults is affected by tooth wear. Next to geographical differences there are large differences per age group and the highest prevalence (11–100 %) was found in children between 9 and 17 years old. Studies with adults aged 18–88 years showed prevalence between 4 and 83 %. There is evidence that the prevalence of erosion is growing steadily, especially in the older age group. Furthermore, it is suggested that gastro-oesophageal reflux disease (GERD) is an important aggravating factor of dental erosion. Erosive wear is most common on, but not limited to, occlusal and palatal surfaces of the teeth. The occlusal erosions are often found on first mandibular molars. Lingually located lesions are most common on the palatal surfaces of the maxillary anterior teeth, and are often linked to intrinsic erosion.

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1.1 Introduction

The two most common dental diseases in children and adolescents, dental caries and erosion both, have strong dietary components in their causation. In many cases dental erosion can be related to dietary intakes, conditions causing dehydration and gastro-oesophageal reflux. The early signs of erosive wear appear as a smooth silky-shining glazed surface (Fig. 1.1). Initial lesions are located coronal from the enamel-cementum junction with an intact border of enamel along the gingival margin [1] which could be the result of plaque remnants acting as a diffusion barrier for acids or as a result of an acid-neutralizing effect of the sulcular fluid [2]. In more advanced stages of erosive wear, changes in the original tooth morphology occur (Fig. 1.2). On smooth surfaces the convex areas flatten or concavities become apparent.

Dental erosion may be caused by extrinsic and intrinsic factors. Probably the most investigated extrinsic cause of dental erosion is excessive consumption of acidic beverages [3, 4]. The consumption of acidic beverages has risen during the last decades. In the United States, a 300 % increase in soft drink consumption has

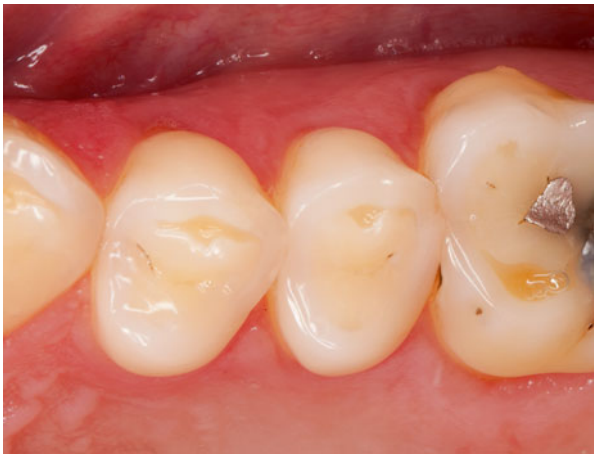

Fig. 1.1 Typical signs of erosion: a smooth silky glazed appearance, change in colour, cupping and grooving on occlusal surfaces

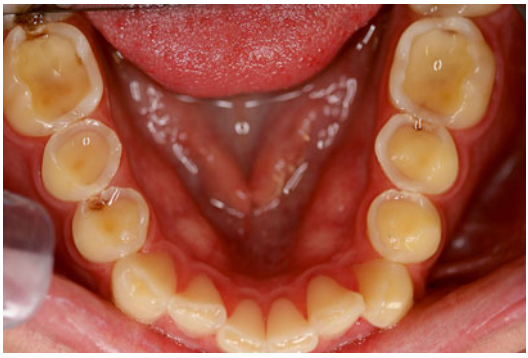

Fig. 1.2 Advanced stage of dental erosion

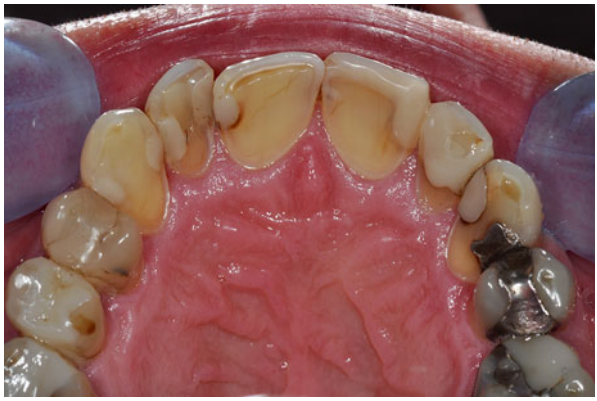

Fig. 1.3 Palatal dental erosion related to gastric reflux

been reported between 1980 and 2000 [5]. Recently, Mexico surpassed the United States as the biggest consumer of soft drinks in the world: 136 L/year in Mexico versus 118 L/year in the United States. Next to problems such as obesity and diabetes, this overconsumption of high glucose and acidic drinks can cause an increase in caries and dental erosion prevalence.

The intrinsic cause of dental erosion is contact of teeth with gastric acid during vomiting or reflux. Vomiting and reflux are rather frequently observed in conditions such as anorexia nervosa, bulimia, gastrointestinal disorders, alcoholism and pregnancy [6]. A typical clinical sign pointing towards erosion caused by gastric juice is palatal dental erosion (Fig. 1.3). Based on only a few reports, it appears that gastric acids are equally likely to induce moderate to severe erosion as dietary acids [7]. Based on the increase in soft drink consumption and the frequently observed reflux symptoms a rise in the incidence of dental erosion can be expected.

The aetiology and predisposing factors of dental erosion are discussed in more detail in Chaps. 3 and 4.

1.2 Measuring Dental Erosion for Epidemiological Studies

Numerous epidemiological studies have investigated the prevalence of dental erosion. For these studies different cross sections of the population are investigated. A problem with comparing these studies is that different methods and indices are used to score the prevalence. Since the last decades, multiple indices have been developed for diagnosing, grading and monitoring dental hard tissue loss [8]. These indices have been designed to identify increasing severity and are usually numerical. Some record lesions irrespective of aetiology (tooth wear indices), others record wear on an aetiological basis (e.g. erosion indices). A widely used tooth wear index is the Smith and Knight Tooth Wear Index (TWI) [6]. The most recently developed index is the Basic Erosive Wear Examination (BEWE) index. With this index the dentition is divided in sextants; and the buccal, occlusal and lingual surfaces of every tooth in each sextant is examined for tooth wear and awarded a score value

between 0 and 3. '0' means no erosive wear, '1' is initial loss of surface texture, '2' is a distinct defect with hard tissue loss <50 % of the surface area and '3' is hard tissue loss >50 % of the total surface area. For each sextant, the highest score is recorded, and when all the sextants have been assessed, the sum of the scores is calculated [9].

The assessment of dental erosion is discussed in more detail in Chap. 6.

1.3 Prevalence and Incidence of Erosion

Erosion prevalence studies have been performed in developed and developing countries, and large differences were found between countries, geographic locations and age groups. Most epidemiological studies are performed in Europe and much less information is available for the United States and Asia. Overall it is found that erosive wear is a common condition. Multiple studies show that primary and permanent teeth can both be affected.

1.3.1 Prevalence by Age

A review of numerous studies on the prevalence of dental erosion found large differences per age group [10]. In the younger age groups (2–9 years) prevalence is between 6 and 50 %. The highest prevalence (11–100 %) was found in children between 9 and 17 years old. Studies with adults aged 18–88 years showed prevalence between 4 and 83 % [10]. Data on the risk for certain age groups to develop erosion (incidence) is less widely available in contrast with prevalence data. There is some evidence that the prevalence of erosion is growing steadily, especially in the older age group [11].

1.3.2 Prevalence by Geographical Region

In this section the findings of a selection of prevalence and incidence studies are discussed by geographic regions.

1.3.2.1 Europe

Dental erosion is generally thought of as a modern phenomenon, but recent archaeological investigation showed that the condition, to some degree, has always been present in the population. Skeletal material from a mediaeval farm population in Iceland was used to study the degree of wear using the Smith and Knight Tooth Wear Index. It was found that in this group dentin was exposed on 1464 surfaces (31 %) and the appearance was characteristic of both chemical and physical wear [12].

Probably the largest reservoir of data on prevalence and incidence of erosion and tooth wear is available in the United Kingdom. Many regional and nationwide studies have been performed. One cross-sectional study using the UK children's dental

health survey and the dental report of the National Diet and Nutrition Surveys (NDNS) reported that dental erosion increases between different age cohorts of young people over time between 1993 and 1997. The data from the NDNS was compared to the children's dental health survey conducted 3 years earlier and it was found that the prevalence of erosion in both primary and permanent incisors was increased. For example, amongst 4–6-year-olds, an increase from 18 % in 1993 to 38 % in 1996 of labial surfaces of primary incisors affected by erosion was found. In general, the increase in prevalence is the case for incisors as well as molar teeth. Weak associations were found between erosive wear and diet, symptoms of gastro-oesophageal reflux and socio-demographic variables such as geographical region and socioeconomic status [13].

A study investigated the prevalence and incidence of erosive wear in the Netherlands, and found that 24 % of the 12-year-old children demonstrated erosive wear [14]. Another Dutch study showed even higher figures; in 2008 a prevalence of 32.2 % was found in subjects aged between 10 and 13 years. Even more striking was the observation in the latter study that 24 % of the children that were free of erosion at baseline developed erosion over the subsequent 1.5 years [11].

A Europe wide study investigating the prevalence of tooth wear including erosion was published in 2013. The BEWE score was used to assess the prevalence of tooth wear on buccal/facial and lingual/palatal tooth surfaces in a sample of young European adults, aged 18–35 years. The BEWE score was 0 for 1368 patients (42.9 %), 1 for 883 (27.7 %), 2 for 831 (26.1 %) and 3 for 105 (3.3 %). There were large differences between different countries with the highest levels of tooth wear observed in the United Kingdom. Associations were found between tooth wear and acid reflux, repeated vomiting, residence in rural areas, electric tooth brushing and snoring. As 29 % of this adult sample had signs of tooth wear, it was concluded that tooth wear is a common problem in Europe [15].

1.3.2.2 The United States

Until now, relatively little is known about the prevalence of erosive wear in the United States. Only one nationwide study on the prevalence of erosion is available [16]. In this study, 45.9 % of the children aged 13–18 years showed erosive wear on at least 1 tooth. The study also reported that, although not significant, 'overweight' (obese) children had increased odds of having erosive wear and those at 'risk for overweight' had lower odds compared to 'healthy weight' children. Another study on the prevalence of dental erosion and its relationship with soft drink consumption in the United States was published in 2011 [17]. Examiners used the modified Smith and Knight Tooth Wear Index to measure erosive wear and information about soft drink consumption was collected. Prevalence of erosive wear was highest in children aged 18–19 years (56 %), males (49 %), and lowest in Blacks (31 %). Children with erosive wear had significantly higher odds of being frequent consumers of apple juice after adjusting for age, gender and race/ethnicity. An association was found between erosive wear and frequent intake of apple juice.

An earlier study focused on the prevalence of erosive wear in children aged 12–17 years in the southwest region of San Antonio, Texas. A convenience sample

of 307 children aged 12–17 years showed an overall prevalence of 5.5 % measured with TWI. All affected children showed erosive tooth wear low in severity and confined to the enamel with no exposed dentin. An association with soda drink consumption was found [18].

1.3.2.3 China

As in many developing countries the lifestyle of the Chinese people is changing significantly. Dental erosion has begun to receive more attention but data about the prevalence of dental erosion in China is still scarce. One study investigated the prevalence of dental erosion in 12–13-year-old children. At least one tooth surface with signs of erosion was found in 27.3 % of the children. The loss of enamel contour was present in 54.6 % of the tooth surfaces with erosion. Furthermore, an association was found with the consumption of carbonated drink once a week or more and also with social economic background [19].

Using the TWI, a total of 5.7 % of preschool children (3–5 years) in Guangxi and Hubei provinces of China showed erosive wear on their maxillary incisors. Of the children affected by erosion, 4.9 % was scored as being confined to enamel and 17 % as erosion extending into dentin. Same as in the previous study a positive association between erosion and social class was found [20].

1.3.3 Prevalence in Relation to Gastro-oesophageal Reflux Disease

In recent years, gastro-oesophageal reflux disease (GERD) has been described as an important aggravating factor of dental erosion. Dental erosion is now considered a co-morbid syndrome with an established epidemiological association with GERD [21]. In a review paper, the prevalence of dental erosion in GERD patients and the prevalence of GERD in erosion patients were investigated. It was found that the median prevalence of dental erosion in GERD patients was 24 % with range 21–83 %. Adult patients with dental erosion had a median GERD prevalence of 32.5 % with range 14–87 % [21]. Other studies also showed large differences in prevalence rate. According to Böhmer et al. [22], 65.5 % of intellectually disabled and institutionalized patients with GERD also presented dental erosion. In contrast with these numbers, two Scandinavian studies using military personnel found no correlation or association between the prevalence of erosion and GERD [23, 24].

1.3.4 Work-Related Prevalence

It could be expected that people who are exposed to acid in their working environment develop dental erosion. Examples of such workplaces are occupations in mineral, battery, chemical, tin, dyestuff, fertilizer and also metal industries [25]. However, Wiegand and Attin [26] concluded, from their review on occupational

dental erosion, that occupational acid exposure might increase the risk of dental erosion for only workers in the battery and galvanizing industry.

Data on the oral health from athletes participating in the Olympic Games indicate that they have high levels of oral health problems [27, 28]. In a study investigating the oral health status of 302 athletes participating in the London 2012 Olympic Games, the prevalence of dental erosion, scored using the BEWE index, was found to be high (44.6 %) among athletes. This can be attributed to excessive consumption of acidic sport drinks, dehydration and exposure to acidic water in swimming pools. The erosion was equally distributed between the anterior and posterior regions of the dentition: 37.6 % of the anterior teeth and 48 % of the posterior teeth were affected [29].

Wine tasters perform many tastings per day and are therefore highly exposed to organic acids such as tartaric and citric acids in wine [30]. In a study investigating dental erosion in professional wine tasters it was found that there was a higher prevalence and severity of tooth surface loss in winemakers compared to the general public [31]. It is believed that this is exacerbated by the rinsing and swirling involved, which prolongs the period of contact of the acidic wine with the teeth. Erosion associated with wine tasting is usually localized on the maxillary labial and incisal surfaces of teeth.

1.4 Distribution

Erosive wear is most common on, but not limited to, occlusal and palatal surfaces of the teeth. The occlusal erosions are often found on first mandibular molars. Lingually located lesions are most common on the palatal surfaces of the maxillary anterior teeth, and are often linked to intrinsic erosion. Lussi et al. investigated the distribution of erosive lesions in the Swiss population, and an overview of their data is presented in Figs. 1.4 and 1.5 [32, 10].

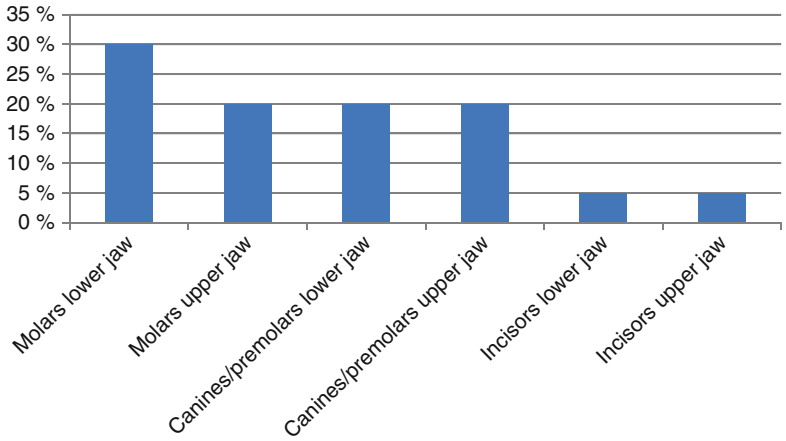

Fig. 1.4 Distribution of occlusal erosion lesions (Modified from Lussi et al. [32])

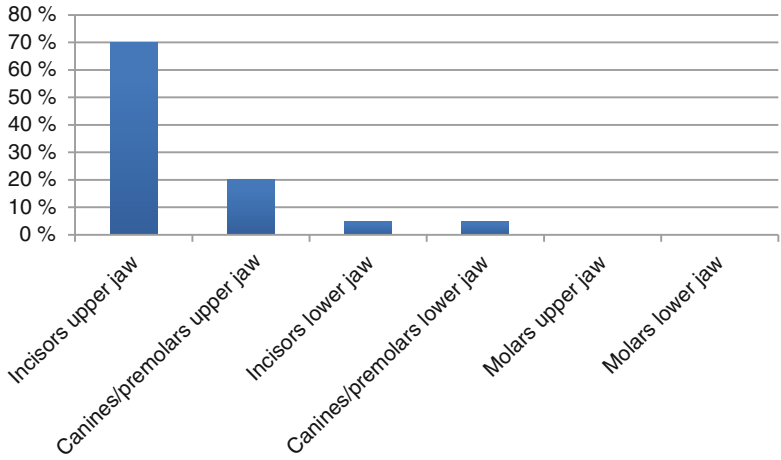

Fig. 1.5 Distribution of lingually located erosion lesions (Modified from Lussi et al. [32])

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The Dental Erosion Process

2

R.P. Shellis

Abstract

Erosive tooth wear is a two-stage process. In the first stage (erosion), acids derived mainly from dietary sources partially demineralise and soften tooth surfaces. In the second stage (wear), the weakened tooth surfaces are worn by intra-oral frictional forces. The microstructure, porosity and mineral solubility of enamel and dentin influence the histological patterns and relative rates of erosion. The erosive potential of acidic products seems to be determined largely by pH and buffering properties, although fluoride and calcium concentrations could also be important. Raised temperature and increased fluid movement accelerate erosion. Eroded surfaces are worn by toothbrushing, attrition and even abrasion by food or the soft tissues. Because the initial erosion affects all exposed tooth surfaces, the clinical appearance of erosive wear is unlike that of purely mechanical wear. Variations in behaviour, such as patterns of toothbrushing or the frequency of drinking erosive beverages, cause wide differences in the degree of erosion experienced by individuals. Saliva ameliorates erosion considerably, by dilution and neutralisation of acids and by formation of salivary pellicle which protects tooth surfaces against demineralisation. However, remineralisation seems to occur too slowly to reverse the erosion process.

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2.1 Introduction

In pre-industrial human populations, in which the diet tends to be coarse and to be contaminated with abrasive particles such as millstone grit, heavy tooth wear is ubiquitous. The loss of tooth material is the result of mechanical wear processes: attrition (caused by direct contact between opposing tooth surfaces) and abrasion (caused by abrasive particles between moving tooth surfaces). The characteristic pattern of mechanical wear consists of flattening of the incisal and occlusal surfaces, with wear of the interproximal surfaces [1]. The evolution of the wear process with time is predictable and varies little between individuals, because all members of the population share the same diet.

In modern Western populations, the diet has become softer and easier to process, so the amount of mechanical wear is typically minimal. When marked wear is seen, it differs in appearance from the pattern described above [2, 3]. Thus, on occlusal surfaces, there may be 'cupping' of cusps, and restorations may stand proud of the surface owing to loss of the adjacent surface. Wear is often seen in the cervical area of root surfaces (non-carious cervical lesions) or on facial and lingual/palatal tooth surfaces: sites which are not subject to mechanical wear from the diet. It is widely recognised that such phenomena involve not only mechanical wear but also *erosive wear*, in which exposure to acid partially demineralises the tooth surface and renders it vulnerable to weak forces that would normally cause negligible wear: erosive wear is thus the result of a combination of chemical attack (erosion) and mechanical wear.

The main focus of this chapter is on the creation of the initial erosive lesion. As this is a reaction of the tooth surface to acidic conditions in the oral environment, erosion is influenced both by the properties of the tooth surface and by the characteristics of the acidic challenge. The interaction between the tooth and acid is in turn modulated by the formation of salivary pellicle and by other aspects of saliva. Finally, the erosive wear experienced by an individual is the cumulative result of their behaviour over time, in such respects as diet and oral hygiene habits.

2.2 Dental Tissues

Both enamel and dentin are composite materials consisting of an organic matrix, mineral and water. The mechanical properties of each tissue are determined by the proportions of the three constituents and by their structural organisation. Two aspects of structure that are important in the development of erosion are, first, the pore structure and, secondly, the size and shape of the mineral crystals. The water within a dental tissue is the medium in which dissolved substances diffuse into the tissue, so the total water content (porosity) and the distribution of pores within the tissue influence the penetration of acid [4]. Crystal morphology is important because, for a given mass of mineral, the smaller the crystal size the larger the surface area of crystals available for attack from acid, so the mineral will tend to dissolve more quickly.

Table 2.1 Composition of enamel and dentin by volume

Constituent	Enamel (vol %)	Dentin (vol %)
Mineral	91.3	48.7
Organic material (protein and lipid)	5.3	29.9
Water	3.4	21.4

2.2.1 Enamel

As Table 2.1 shows, the proportion of mineral in enamel is very high: this is responsible for the exceptional hardness of the tissue. The crystals have roughly hexagonal cross sections and are on average 70 nm wide and 25 nm thick [5]. Their length is much greater than their width (probably > 1000 nm). The largest pores are found at the boundaries of the rods, where there is an abrupt change in crystal orientation, but these constitute only about 0.3 % of the total porosity. The rest of the pores are extremely small and distributed between the crystals making up the rest of the enamel. In the rods, which make up about 75 % of the volume of enamel, the crystals are very closely packed, so that the pores are very small and often inaccessible [4]. In the inter-rod regions, the porosity is slightly greater. From knowledge of crystal orientation in enamel, it can be deduced that most of the pores are orientated at about 70–90° to the tooth surface.

2.2.2 Dentin

Dentin differs radically from enamel in that about 30 % of the tissue is made up of organic matrix (Table 2.1), of which about 90 % is the fibrous, insoluble protein collagen. The remainder consists of a variety of proteins and carbohydrates and a small amount of lipid. The mineral crystals are platelike in form rather than ribbon-like as in enamel and are also much smaller and thinner: approximately 30 nm wide, 3 nm thick [5].

During dentinogenesis, many of the crystals are deposited within the collagen fibres, so are intimately associated with the sub-fibrils, while the remaining crystals are deposited between the fibres. The proportion of crystals within the fibres is between 25 and 80 % [6].

Of the overall porosity (about 21 vol%: Table 2.1), about 6.5 vol% is associated with the tubules, which run from the enamel-dentin junction to the pulp chamber. As these are wider and more closely packed towards the pulp, they occupy a greater proportion of the inner dentin (22 vol%) than of the outer dentin (1 vol%). Within the intertubular dentin, the average porosity is about 15 vol%. The interfibrillar regions are probably less porous than the intertubular regions, because of the close packing of organic and inorganic material. It is unlikely that the pores of dentin show much directionality because the crystals are very small and platelike.

2.3 Chemistry of Dental Mineral

Dental minerals are forms of a sparingly soluble calcium phosphate known as hydroxyapatite, which in its pure form has the formula $Ca_{10}(PO_4)_6(OH)_2$. An important property of dental mineral is the solubility, which determines whether a given solution will allow dissolution to proceed, and the concept requires a few words of introduction.

When a sparingly soluble solid such as hydroxyapatite is immersed in water, it will begin to dissolve. If there is an excess of solid and a limited volume of water, dissolution will not continue indefinitely but will eventually cease. At this point, the system is in equilibrium and the solution is said to be *saturated*. Analysis of the saturated solution enables the solubility of the solid to be determined. The fundamental thermodynamic solubility is defined in terms of the chemical activity of the dissolved solid and is a constant for a given temperature. In this chapter, a more practical definition of solubility will be used, namely, the *gravimetric solubility*, which is the concentration (mass per unit volume) of dissolved solid in solution.

Solutions in which the concentration of dissolved solid is less than in a saturated solution are *undersaturated* and solutions with a higher concentration are *supersaturated*. Undersaturated solutions can support dissolution of the solid but not precipitation, while supersaturated solutions support precipitation but not dissolution.

In the dental tissues, the mineral contains a number of impurity ions, which take the place of ions in the hydroxyapatite structure. Thus, Ca^{2+} ions can be replaced by Na^+ or Mg^{2+} ions; PO_4^{3-} ions can be replaced by CO_3^{2-} ions and OH^- ions by CO_3^{2-} or F^- ions [5]. In most of these cases, the impurity ion has a different charge or size to the ion it is replacing. This results in misfits in the crystal lattice which disturb the crystal structure and in turn make the mineral chemically less stable: in other words more soluble.

Table 2.2 shows that the major impurities in both dentin and enamel mineral are carbonate, magnesium and sodium. In relation to the calcium and phosphate concentrations (i.e. the total mineral), dentin contains more carbonate and magnesium than enamel and is also much less well crystallised. In Fig. 2.1, the curves represent the equilibrium concentration of relevant solids over a range of pH from neutral to the low values typical of erosive products. The higher the concentration at any particular pH, the greater the solubility. The figure shows that enamel is slightly more soluble than pure hydroxyapatite but dentin is much more soluble. The figure also

Table 2.2 Principal inorganic constituents of dentin and enamel (percent dry weight) [5]

Constituent	Enamel	Dentin
Ca	36.6	26.9
P	17.7	13.2
CO_3	3.2	4.6
Na	0.7	0.6
Mg	0.4	0.8
Cl	0.4	0.06
K	0.04	0.02

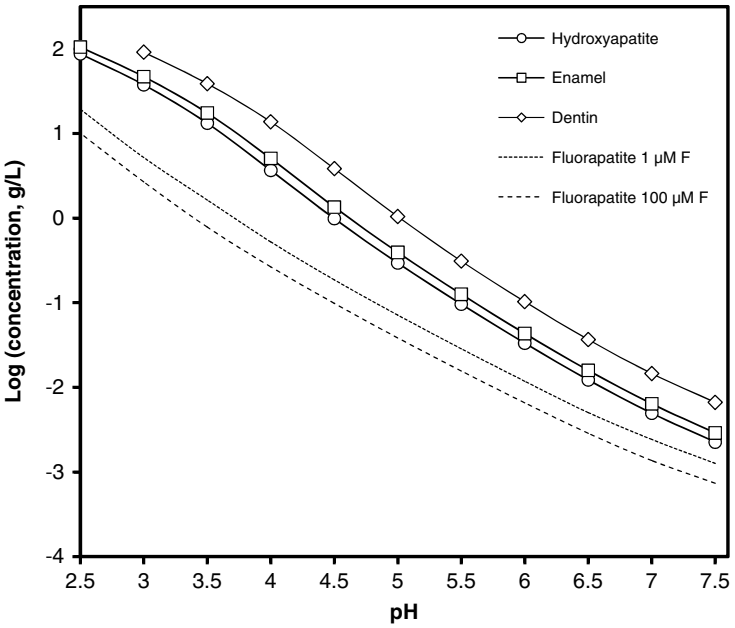

Fig. 2.1 Solubility diagram for solids relevant to erosion. *Lines* represent equilibrium solubilities over the pH range 2.5–7.5. Solubilities for fluorapatite are given for the lowest and highest fluoride concentrations in the products studied by Lussi et al. [7]

shows that the gravimetric solubility of these solids increases as the pH of the solution decreases.

Figure 2.1 also shows solubility of an additional solid, fluorapatite, which is structurally close to hydroxyapatite but in which all the OH^- ions are replaced by F^-. Because the F^- ion has the same charge as the OH^- ion and is slightly smaller, this substitution (unlike those discussed above) results in a more stable crystal structure and hence reduces solubility. The solubility of fluorapatite depends on the fluoride concentration in solution, so its solubilities are given for a range of fluoride concentrations found in representative erosive products [7].

2.4 Acids and Demineralisation

The acids responsible for erosion (Table 2.3) may be *intrinsic* or *extrinsic* in origin. The intrinsic acid is hydrochloric acid, the principal component of gastric juice. This comes into contact with teeth when gastric juice is regurgitated, either as an occasional occurrence or more frequently, as in gastro-oesophageal reflux disorder. Extrinsic acids reach the mouth via two routes. Certain industries, e.g. battery production, are associated with vapours of strong acids such as sulphuric acid, which attack the teeth after inhalation and dissolution in the saliva. Of far greater importance at population level are acidic components of products intended for human

Table 2.3 Acids associated with dental erosion

Acid	Occurrence
Intrinsic acid	
Hydrochloric acid	Gastric juice reflux
Extrinsic acids	
Sulphuric acid, chromic acid	Vapours associated with battery production
Phosphoric acid	Cola
Acetic acid	Vinegar, pickles
Lactic acid	Cheese, yoghurt, wine, fermented cabbage (e.g. sauerkraut)
Malic acid	Apples, grapes, wine
Tartaric acid	Grapes, tamarind, wine
Citric acid	Citrus fruits
Ascorbic acid	Vitamin C supplements

consumption: soft drinks, fruit juices, acidic fruits and vegetables, some alcoholic drinks, some vitamin supplements and medications. The acids found in foods may be metabolic products of fruits or vegetables (malic, tartaric, citric acids) or products of bacterial fermentation (acetic, lactic).

All of these acids provide hydrogen ions (H^+) which dissolve dental mineral. Taking hydroxyapatite as an example for this process, the reaction is:

$$\begin{aligned} &Ca_{10}\left(PO_4\right)_6\left(OH\right)_2+\left(2+3x+2y+z\right)H^+ \rightarrow 10Ca^{2+}+xH_3PO_4^0 \\ &+yH_2PO_4^-+zHPO_4^2+2H_2O \\ &\left(x+y+z=6\right) \end{aligned} \tag{2.1}$$

Here, the fully dissociated phosphate anion, PO_4^{3-}, is omitted because its concentration is exceedingly low. The proportions of the other forms of phosphate (x, y, z) depend on pH. The reaction for dental mineral is similar but also involves the conversion of carbonate ions to carbon dioxide and water:

$$CO_3^{2-}+2H^+ \rightarrow CO_2\left(\text{gas}\right)+H_2O \tag{2.2}$$

Hydrochloric and sulphuric acids are *strong acids*: i.e. at all pH values they are fully dissociated into H^+ ions and Cl^- or SO_4^{2-} ions. The remaining acids in Table 2.3 are *weak acids*. At low pH, they consist almost entirely of undissociated acid. As the pH increases, the acids dissociate progressively. Each molecule of acid may provide one H^+ (acetic, lactic), two H^+ (malic, tartaric) or three H^+ (citric, phosphoric). The dissociation reactions for tartaric acid are:

$$H_2\text{Tartrate}^0 \rightleftharpoons \text{HTartrate}^- + H^+ \left(pK_a = 3.04\right) \tag{2.3a}$$

$$\text{HTartrate}^- \rightleftharpoons \text{Tartrate}^{2-} + H^+ \left(pK_a = 4.37\right) \tag{2.3b}$$

The pH values at which dissociation occurs is determined by the acid dissociation constant(s), K_a, which are given after the above equations as the negative logarithms (pK_a). The dissociation process is illustrated graphically in Fig. 2.2. Weak acids,

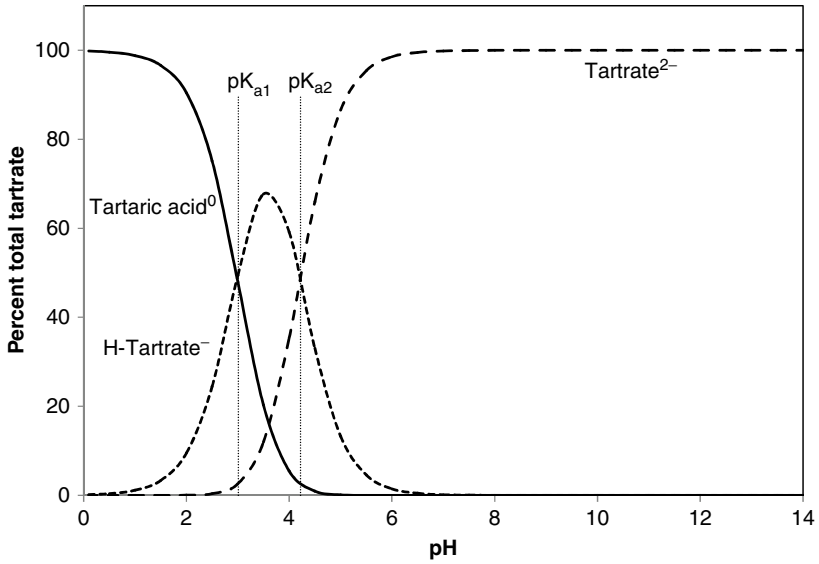

Fig. 2.2 Diagram illustrating ionisation of tartaric acid. As pH rises, tartaric acid dissociates into HTartrate$^-$ ions and this in turn into tartrate^{2-} ions

because they dissociate progressively, act as buffers, so can resist changes in pH. When pH equals a pK_a, the buffering strength is at a maximum. It is considered that there is effective buffering over the pH range $pK_a \pm 1$. Polybasic acids can therefore buffer over a wide pH range: for instance, citric acid, with pK_a of 3.13, 4.74 and 6.42, is a good buffer over the pH range 2.1–7.4.

The strength of buffering is related to the total concentration of acid, and it can be quantified in different ways, using different techniques of titration with a base such as sodium hydroxide. The *buffer capacity* measures the strength of buffering at the pH of the solution. The *titratable acidity* measures the amount of buffering between the pH of the solution and some defined higher pH, usually 5.5 or 7.0. The titratable acidity to pH 5.5 is preferable, mainly because the pH region between 5.5 and 7.0 is of little or no interest to erosion.

A possibly important property of weak acids which form anions with more than one negative charge is that they are capable of forming *ionic complexes* with cations such as Ca^{2+}. Complexes are stabilised by formation of chemical bonds rather than by simple electrostatic attraction. One type of complex is formed by *chelation*, in which formation of coordinate bonds between two or more negative anionic groups and the Ca^{2+} ion results in a ring structure (see Fig. 2.3 for an example). Chelation will remove calcium ions from solution and reduce their concentration, but they could also speed up the process of mineral solution more directly. Chelating anions can bind to Ca^{2+} ions at the surface of the solid, thus weakening bonds holding the Ca^{2+} in place and causing them to be solubilised [6, 8].

The possible roles of buffering and chelation in erosion will be discussed later.

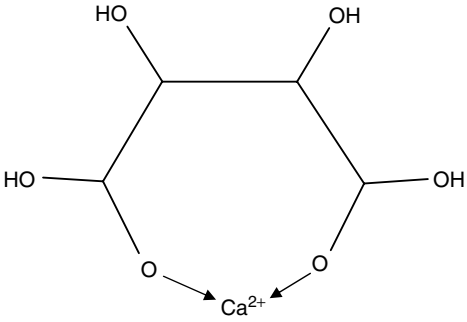

Fig. 2.3 An example of chelation: a schematic diagram of the tartrate-calcium complex. The *arrows* symbolise coordinate bonds, formed by sharing of electrons from the COO^- groups with the calcium ion

2.5 Erosion of the Tooth Surface

When an erosive solution comes into contact with a tooth, the surface starts to dissolve. Simultaneously, acid diffuses into the tissue and begins to demineralise the tissue beneath the surface [9].

2.5.1 Enamel

Acid diffusing into the narrow pores between the crystals results in partial loss of mineral, increased porosity and reduction of mechanical strength of the outer layer of enamel, which is hence referred to as the 'softened layer' [9] (Fig. 2.4). Even after partial demineralisation, the pores within the enamel are still extremely narrow so that the acidic solution can only diffuse inwards for a short distance before becoming saturated with respect to enamel mineral, thus losing its erosive capacity. Consequently, the softened layer produced by an average challenge is no more than a few micrometres thick [9]. The high degree of orientation of the pores in enamel means that there is a gradient of mineral content within the softened layer, content being least at the outer surface and increasing towards the unaffected enamel [9].

Intra-oral measurements show that drinking an erosive beverage causes the pH at tooth surfaces to fall for a few minutes [10]. A single such challenge from acid is unlikely to cause the loss of surface enamel. However, after more prolonged erosion, or after repeated challenges, the outermost enamel eventually becomes completely demineralised, causing a loss of surface profile. Acidic foods could also produce this effect, because the contact time with the teeth would be longer than for drinks and also because the mixing effect of chewing would accelerate demineralisation. However, no measurements of tooth-surface pH during mastication of acidic foods have been made. During prolonged erosion, the overall rate of mineral loss from enamel becomes constant a few minutes after the initial contact [11].

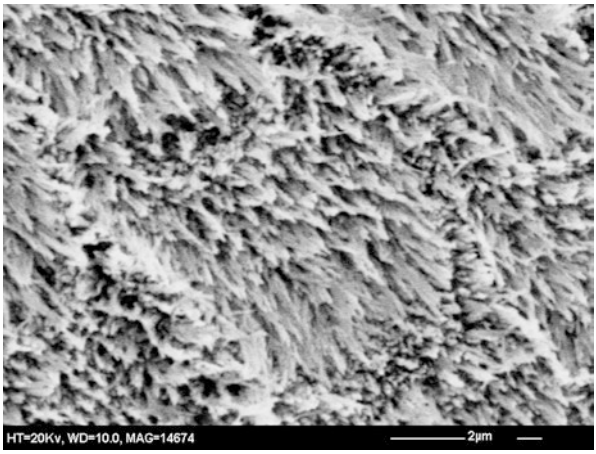

Fig. 2.4 Scanning electron micrograph of the surface of a specimen of enamel which has been exposed to 0.3 % citric acid (pH 3.2) for 20 min. Etching of the surface revealing the profiles of the prisms. At the outer surface, the crystals are more completely demineralised than those deeper in the softened layer

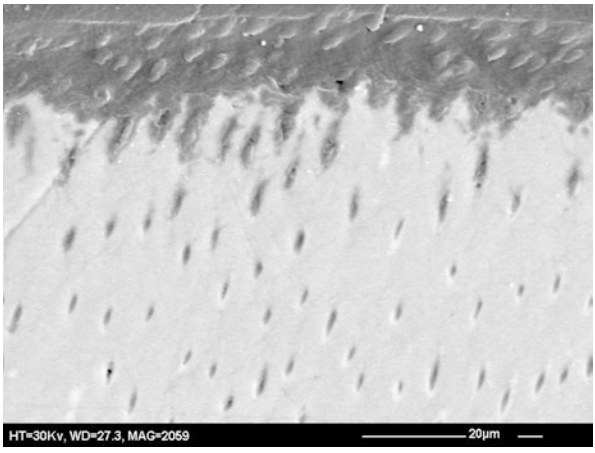

Fig. 2.5 Cross section of dentin exposed to 0.3 % citric acid, pH 3.2, for 20 min. Surface was polished using a graded series of diamond paste then viewed in a scanning electron microscope with a backscattered electron detector. In this mode, contrast from flat surfaces is due to variations in average atomic number, so areas with high mineral content appear lighter than areas with reduced mineral content. At top of field is a layer of demineralised dentin, with obliquely sectioned dentin tubules (as demineralised dentin is relatively soft, it cannot be polished completely flat and displays some topographic contrast). The junction between the dark demineralised and pale sound dentin is sharply defined. Preferential dissolution of peritubular dentin at and just beneath the junction between sound and demineralised dentin is clearly seen

2.5.2 Dentin

The erosion of dentin follows a different pattern [9]. Whereas erosion of enamel eventually causes loss of the surface tissue, erosion of dentin leaves behind a persistent layer of demineralised collagenous matrix [12] (Fig. 2.5). With continuing

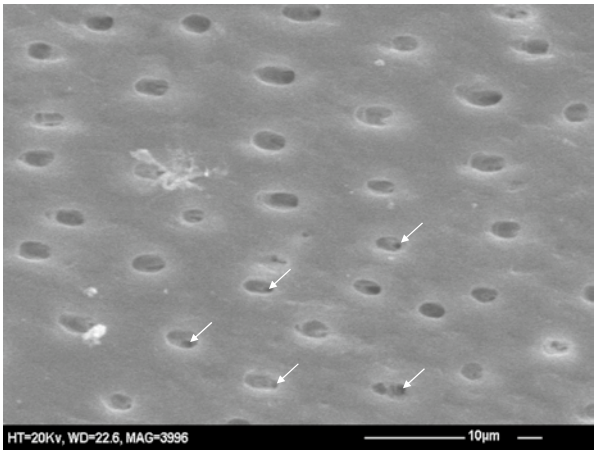

Fig. 2.6 Polished surface of dentin exposed to 0.3 % citric acid solution, pH 3.2, for 20 min. Note the enlarged openings of the tubules and the absence of peritubular dentin. Tubule openings visible beneath the surface (*arrows*) are small because the peritubular dentin is intact at this level

exposure to acid, this layer becomes thicker, which means that inward diffusion of acid and outward diffusion of mineral end products, between the surface and the demineralisation front, are slower: the overall rate of demineralisation therefore slows down as erosion proceeds [11]. Because of their great solubility and small size, the dentin crystals are completely dissolved over a short distance, so that there is only a narrow zone of partially demineralised dentin between the unaffected dentin and the demineralised outer layer. The intrafibrillar domains are demineralised more slowly than the intrafibrillar regions because of diffusion inhibition by the collagen fibrils [6]. Peritubular dentin is attacked first, then the intertubular dentin: a sequence observed both at the surface (Fig. 2.6) and at the interface between sound dentin and the demineralised surface layer (Fig. 2.5).

Dentin mineral is more soluble than enamel mineral, so in theory should be more susceptible to erosion. However, in practice, the relative rate varies with pH. Erosion tends to be faster in enamel at low pH (< pH 3) [11], possibly because a high concentration of H^+ ions promotes dissolution at the outermost enamel surface, whereas loss of acid from dentin is always hindered by the presence of collagen fibres. The variation in relative erosion rates of dentin and enamel with pH probably reflects the relative contributions of dissolution rate of the individual mineral crystals and of the rate of diffusion out of the respective tissue.

2.6 Factors Controlling Erosive Demineralisation

2.6.1 Chemical Factors

The rate of erosion is influenced by a variety of chemical properties of the erosive solution. To understand which factors are important, information from two complementary types of study is required. In both, standardised specimens are exposed to the solution under defined conditions of temperature and stirring rate for a preset

Table 2.4 Statistically significant bivariate and multivariate associations between properties of acidic products and erosion

Tissue	Variable	Reference
Bivariate		
Enamel	Degree of saturation with respect to hydroxyapatite	[7, 14]
Enamel	Degree of saturation with respect to fluorapatite	[7]
Enamel	pH	[14, 15]
Enamel	Buffer capacity	[14]
Enamel	Fluoride concentration	[15]
Enamel	Phosphate concentration	[14, 15]
Dentin	Buffer capacity	[15]
Dentin	Calcium concentration	[15]
Multivariate		
Enamel	pH	[7, 16, 17]
Enamel	Buffer capacity	[7, 16, 17]
Enamel	Fluoride concentration	[7, 16, 17]
Enamel	Calcium concentration	[7]
Enamel	Phosphate concentration	[16, 17]

time, and the extent of erosion is then measured by an appropriate technique, such as microhardness or profilometry. Experiments on *defined solutions* allow chemical variables such as pH or ionic concentrations to be controlled and manipulated as desired and solution variables can be studied over a wide range of values. These studies are free of possible interference from ingredients found in commercial products, so they need to be compared with tests on *erosive potential* of such products. Since both types of study are performed in vitro, neither takes into account the diverse effects of the oral environment, particularly saliva. Of course, in situ experiments can address this problem but, because of the greater variability of any experiments in humans, these require more replicates and are expensive. However, the available studies indicate that, although the rates of erosion in vitro and in situ differ considerably, tests on the same products under the two conditions place their erosive potentials in the same rank order [13]. Thus, in vitro tests seem to give reliable estimates of erosive potential.

In tests of erosive potential, statistical analysis is performed to establish the extent to which erosion is correlated with the chemical properties of the products. Some studies have employed simple bivariate tests, which assess the association between the extent of erosion (the dependent variable) and the individual properties of the test product (the independent variables) in turn. Multivariate tests, in which associations between the dependent and independent variables are tested simultaneously, are more informative because they take into account correlations between the independent variables. The results of tests on erosive potential are summarised in Table 2.4.

The basic requirement for a solid to dissolve in a solution is that the solution is undersaturated (see above). In experiments with defined solutions, the rate of dissolution at first increases with the *degree of undersaturation* (i.e., how far the solution is from equilibrium). Eventually, however, the rate becomes constant, and no

further reduction in the degree of undersaturation has any effect on dissolution [6]. This means, for instance, that the erosion rate in *completely unsaturated* solutions (containing no calcium or phosphate) should be maximal.

In general, erosive potential conforms to these principles, in that most undersaturated products erode enamel, whereas saturated or supersaturated products are not erosive [6]. Two studies have identified significant bivariate associations between erosion and the degree of saturation with respect to hydroxyapatite (Table 2.4). However, a minority of products appear to be undersaturated and yet to be non-erosive [6]. In many cases, it is likely that the product is actually erosive but that the erosivity is too low to be detectable by available methods. In other cases, the absence of erosion is due to the presence of inhibitors of dissolution. These substances adsorb to the sites on the surfaces of mineral crystals at which dissolution occurs. Hence, they block exchange of ions between the mineral solid and the solution [6]. Dissolution also tends to be reduced or even abolished in undersaturated solutions in which the calcium concentration is much higher than the phosphate concentration [18].

The factors that have been most consistently identified as significant factors in erosive potential (against enamel) are pH and buffer capacity (Table 2.4). Laboratory experiments show that erosion of enamel is very rapid at pH of about 2.5 but slows down as the pH increases until at pH 5–5.5 is extremely slow and becomes difficult to measure [19, 20].

Buffering is important because, during erosion of enamel, dissolution of mineral takes place within the near-surface tissue. The dissolution of mineral consumes H^+ ions (see Eqs. 2.1 and 2.2), meaning that the pH will tend to rise. Within the pores where dissolution takes place, the pH can rise quickly because of the very large ratio between the surface area of mineral and the very small volume of liquid within the pores. Thus, mineral would soon stop dissolving, if the solution cannot resist the change in pH, i.e. if it is inadequately buffered. The higher the buffer capacity, the less the pH of the solution within the pores will rise and the faster the overall rate of erosion. Because the sites of mineral dissolution are a short distance away from the solution bathing the tooth surface, it is probably not the buffer capacity of the solution that is directly removed, but a related quantity, the concentration of undissociated acid molecules [21]. The molecules are uncharged, so will diffuse more readily into the tissue, because they are not attracted or repelled by the electric charge on the surfaces of the pores. Once inside the tissue, the molecules of acid can dissociate to provide H^+ ions and in this way buffer the solution against a rise in pH [22, 23].

The limited information available suggests that the rate of dentin erosion is much less responsive to either pH or buffering than is enamel [11]. This is perhaps due to the presence of the surface layer of demineralised collagen.

A factor that appeared as a significant factor in erosion in both bivariate and multivariate models is the fluoride concentration. Figure 2.1 shows that fluorapatite is less soluble than hydroxyapatite, indicating that replacement of all the OH^- ions in hydroxyapatite by F^- ions causes a large decrease in solubility. However, solubility can be reduced without 100 % substitution. The presence of F^- ions in an acid solution surrounding hydroxyapatite crystals reduces the rate of dissolution [24]. Adsorption of F^- ions at the crystal surfaces, ions stabilise surface Ca^{2+} ions and in

effect convert that portion of the crystal surface to fluorapatite. The more of the surface that is modified in this way, the lower the overall solubility of the crystals [25].

The concentrations of calcium and phosphate have been identified as factors for erosive potential in some, but not all, studies. It has been shown that calcium addition to acidic solutions can reduce erosive potential [18, 26]. An effect of phosphate on erosive potential seems unlikely on theoretical grounds, since concentration of the phosphate ion directly involved in mineral solubility $\left(PO_4^{3-}\right)$ is negligible at low pH [7].

In theory, chelation could augment the effect of pH in erosion, as outlined above. At the moment, however, the importance of chelation in erosion remains conjectural and needs to be tested by controlled experiments. In the low pH range typical of erosive products, the polyvalent anions required for chelation make up a minority of the total acid (see Fig. 2.2). Thus, there may not be a high enough concentration of chelators available to make a significant impact [6].

2.6.2 Physical Factors

As well as the chemical factors outlined above, the rate of erosion is strongly influenced by two physical factors: temperature and fluid movement.

2.6.2.1 Temperature

Temperature affects the rate of most chemical reactions and erosion is no exception. Studies show that both early erosion (measured by softening) and later erosion (loss of surface) increase over the range 4–75 °C [27, 28].

2.6.2.2 Fluid Movement

Fluid movement ensures that the reagents participating in a chemical reaction are continually replenished, so that the reaction does not slow down. The layer of liquid at the interface between a dental tissue and an erosive solution is more or less static. Therefore, transport of H^+ ions and acid molecules from the bulk liquid into the tissue, and of dissolved mineral from the tissue to the solution, can only occur by diffusion, which is relatively slow. If the bulk solution is well stirred – i.e. in active movement – the static interfacial layer of liquid becomes thinner and there is an improved supply of H^+ ions and removal of mineral-ion end products. Increased movement of fluid thus speeds up dissolution. Erosion of enamel increases very rapidly at low flow rates and then increases more slowly [29], whereas dentin erosion increases gradually with flow rate [30].

2.7 Erosion In Vivo: The Role of Saliva

In vivo, an erosive challenge is reduced considerably through dilution of the erosive liquid by saliva and through the increase in pH caused by salivary buffers. These effects are enhanced by the stimulation of salivary flow and increased buffer

capacity brought about by ingestion of acidic products. Recordings of pH at tooth surfaces show that the dilution and buffering by saliva effectively limit an erosive challenge to a few minutes [10]. Low salivary flow rate and buffer capacity have been identified as risk factors for erosion in several studies [31–33].

After the dilution and buffering effects, probably the most important role of saliva is the formation of a protective pellicle at exposed tooth surfaces. The salivary pellicle is a thin film composed mainly of protein plus lipid, which is firmly adsorbed to the tooth surface and which has been demonstrated in numerous studies to inhibit demineralisation of the underlying hard tissue by acids. Starting from a bare tooth surface, pellicle is visible even after 1 min of intra-oral exposure as a condensed organic film 10–20 nm thick [34]. On permanent teeth, the pellicle then thickens by accretion of globular structures, about 100–300 nm in diameter, reaches a maximum thickness after 60–90 min [34] then becomes denser in texture [35]. The pellicle formed on deciduous teeth consists only of a thin, condensed film and, over a 24-h period, shows no trace of the globular structure seen in permanent teeth pellicles [36].

Adsorption of proteins to form pellicle is selective, so the composition differs significantly from that of saliva. The principal identifiable proteins in established pellicle appear to be high-molecular-weight mucous glycoprotein, α-amylase, albumin, secretory immunoglobulin A, proline-rich proteins and cystatin SA-1 [37, 38]. After adsorption, salivary proteins change, especially through the action of salivary and bacterial enzymes. Some proteins are partially degraded [38], but there is also evidence for enzyme-mediated cross-linking of pellicle constituents.

Besides proteins and peptides, pellicle contains carbohydrate, which might largely be associated with glycoproteins, and lipids: pellicle formed in vivo for 2 h contains about 23 % glycolipids, free fatty acids and phospholipids [39].

Many studies have shown that demineralisation of dental tissues is reduced (although not prevented) if they are coated with salivary pellicle (for recent review, see [38]). Erosion is reduced in both enamel and dentin, but the protective effect is much greater for enamel [40]. It has been shown that the pellicle reduces diffusion of anions while not affecting diffusion of water [41]. At least in part, this permselectivity might be associated with the lipid component, since removal of lipids reduced the retardation of lactic acid diffusion [39]. Therefore, it is generally assumed that the permselectivity of pellicle underlies the inhibition of demineralisation. It is not known, however, whether pellicle shows any selectivity towards ions of different charge, which could be important for protection against demineralisation. Some pellicle proteins are capable of adsorbing to hydroxyapatite and acting as inhibitors of dissolution and this might also contribute to the protective effect of pellicle [38].

In one in vitro study, a wide variation in the degree of protection was found, and treatment of enamel with saliva from one donor even slightly increased erosion [42]. The extent of variation in protection by pellicle between individuals would therefore be worth further investigation.

The protection by pellicle does not persist indefinitely, because exposure to an acidic challenge removes most of the pellicle, leaving only the thin, dense layer attached to the tooth surface [43]. Therefore, although the pellicle continues to have

some effect, the protection of the tooth surface will be considerably reduced and will be fully restored only after a new pellicle has formed. One in vitro study indicates that the time required for pellicle formation to provide significant protection against dentin is 2 min and against erosion of enamel 1 h [44]. In situ studies suggest that protection of enamel against erosion is established after 3, 60 and 120 min [45] and that pellicle formed for 2 h, 6 h, 12 h or 14 h all provide the same degree of protection [43]. Further work to establish more definitely for how long enamel remains at increased risk of erosion after each erosive episode would obviously be of interest.

A few studies in which erosion by the same product was studied in vitro and in situ suggest that erosion is much faster in vitro than in vivo: perhaps as much as ten times faster [13]. This result is usually interpreted as reflecting the influence of the salivary factors outlined above, but this interpretation is probably to some extent misleading, as it is very unlikely that the in vitro and in situ challenges are identical in all respects. In particular, the flow of solution over the specimen surface will most likely be much lower in vivo than in vitro, and this can have a profound effect, particularly at lower flow rates. Thus, although there is no doubt that saliva and pellicle reduce the severity of an erosive challenge, the effect is not likely to be as large as a factor of ten.

2.8 Erosive Wear

2.8.1 Enamel

In industrialised societies, the main source of abrasion to which the teeth are regularly exposed is toothbrushing with toothpaste. Providing the paste is within ISO guidelines for abrasivity, even this represents a very limited challenge. It has been estimated that wear of enamel from normal toothbrushing amounts to only about 10 µm/year [46].

Erosion amplifies the abrasion of enamel because the softened layer is much more easily removed by abrasion than is uneroded enamel: wear after exposure to erosion and abrasion is greater than after either erosion or abrasion alone [19]. Softened enamel is vulnerable even to brushing without toothpaste and to friction from oral soft tissues such as the tongue [47–49]. The extent of wear produced by toothbrushing with paste is obviously of most clinical importance. Following an erosive challenge similar to that experienced during drinking a beverage, about 0.25–0.5 µm is lost during subsequent toothbrushing [50, 51]. This is about 2.5–5 % of a whole year's wear of sound enamel so represents a great loss of wear resistance. Abrasion removes a variable amount of the more demineralised outer region of the softened layer and leaves behind the more resistant inner region. More of the softened layer is removed by brushing with paste than with a brush alone [50]. The force applied during brushing also affects wear. Thus, wear increases with the force applied to a manual brush, while power brushes and sonic brushes create more wear than manual brushes [50, 52].

2.8.2 Dentin

The layer of demineralised dentin persisting after erosion is a tough material, consisting mostly of cross-linked, fibrous collagen, and seems, from in vitro experiments, to be quite resistant to brushing [12]. It is therefore possible that, for some time after an erosive attack, this layer could provide some mechanical protection to an erosive lesion and could also act as a diffusion barrier, thereby slowing the progression of erosive wear. It also appears that the layer can act as a reservoir for fluoride which can inhibit erosion [53].

Demineralised dentin is exposed not only to abrasive forces but to the action of salivary and endogenous proteolytic enzymes. Although there is some evidence that matrix breakdown is accelerated in the presence of proteases [54], the size of the effect is uncertain [6].

2.8.3 Clinical Manifestations of Erosive Wear

As well as accelerating tooth wear beyond normal levels, erosion alters the clinical appearance of wear. Erosion affects all surfaces of the teeth that are not covered with plaque, which has enough buffering power to counteract the erosive acids. Therefore, wear can be observed on the buccal/labial and lingual/palatal surfaces, which are not affected by wear in a purely abrasive environment. These surfaces typically appear glossy or silky because of the loss of small-scale surface features such as perikymata [2, 3].

On occlusal surfaces, attrition and abrasion produce flat wear surfaces clearly demarcated from adjacent unworn surfaces by well-defined angles. Attrition surfaces are smooth, with parallel, fine scratch marks, whereas abrasion surfaces present scratch marks varying in depth and direction, which reflect the complexity of jaw movements and the variety of materials producing the scratch marks [1]. Attrition or abrasion surfaces on dentin are covered by a smear layer, which closes off the dentinal tubule openings and prevents hypersensitivity [1]. When abrasion has occurred on erosion-softened surfaces, the occlusal wear surfaces tend to have rounded borders and a smooth transition to the adjacent tooth surface [1–3]. This reflects the fact that wear can be produced by weak forces and is not restricted to areas which are subject to occlusal forces. The same vulnerability to friction underlies the ‘cupping’ due to loss of dentin from cuspal areas and the loss of tissue from the occlusal surfaces which causes restorations to stand proud of the surface. Active erosive wear may be associated with dental hypersensitivity, when the dentinal tubules remain patent [1]. It is generally considered that a combination of erosion and abrasion of exposed root surfaces is also responsible for initiation and progression of non-carious cervical lesions [55, 56]. Such lesions are found very rarely, if at all, in populations where mechanical wear is predominant [1].

2.8.4 Behavioural Factors

In most people with erosive wear, the main risk is from extrinsic acids, especially fruit juices and soft drinks (both carbonated and uncarbonated). These products have been identified as risk factors for erosion in a number of epidemiological studies [e.g. 31, 57–59]. A high risk of dental erosion is also associated with consumption of a raw-food diet, which includes a large component of acidic fruit [60], and the intake of acidic foods and drinks was identified as a risk factor for progression of erosion [61].

The method of consuming drinks is likely to influence the erosion experienced by an individual. Retention of drinks in the mouth before swallowing extends contact time with the teeth [62]. Certain erosive products, e.g. some fruit cordials, can be consumed either cold or hot, and in vitro studies clearly suggest that the latter will present a higher risk for erosion [27, 28]. The increase in demineralisation rate brought about by increased fluid movement is of the greatest importance, because in different methods of drinking acidic beverages, the speed of fluid movement varies considerably, and this results in widely varying erosive challenges. Drinking from a cup or glass, or through a straw, with the liquid directed over the surface of the palate and with minimal contact with the tooth surfaces, clearly presents a relatively low challenge. In contrast, drinking the same beverage through a straw with the tip anterior to the incisors, or swishing the drink around the mouth, will maximise flow at the tooth surfaces and hence create a severe erosive challenge [62].

An increased frequency of regurgitation of gastric contents increases the risk of erosion. The most important causes are gastro-oesophageal reflux disorder and eating disorders such as bulimia [63]. Excessive consumption of alcohol is associated with more frequent vomiting. Another suggested cause of increased reflux is an excessive level of exercise [62].

As the main source of tooth abrasion is toothbrushing, it might be expected that the frequency and intensity of brushing would be correlated with the extent of erosion. However, while an association of abrasion-related factors and development of non-carious cervical lesions has been demonstrated in several studies [55], many studies on erosion of coronal surfaces have found no association with toothbrushing [e.g. 57, 59, 64]. Bearing in mind that softened enamel is more vulnerable to mechanical wear than demineralised dentin, these results perhaps reflect the greater work needed to remove eroded dentin.

2.9 Is Erosion Reversible?

Since the softened layer resulting from an erosive challenge is incompletely demineralised, it retains a framework of partly dissolved crystals which could form a substrate for crystal growth. As saliva contains dissolved calcium and phosphate ions, and indeed is supersaturated with respect to hydroxyapatite, it is theoretically

possible that it could support remineralisation of erosive lesions between acid challenges, which would restore the mechanical integrity of the softened layer. A number of studies have explored this possibility and have shown that some reduction of erosive tissue loss occurs after various periods of in situ exposure to saliva of acid-challenged tooth surfaces [65–70]. The results have led to recommendations that toothbrushing should be avoided for about 30–60 min after consumption of erosive products [65, 66, 69]. However, complete restoration of the tooth surface is not achieved in a short time, even after application of fluoride at extremely high levels [71, 72]. It must be remembered that, unlike caries lesions, erosive lesions are not protected from the oral environment by a surface layer so are vulnerable to frictional forces immediately after formation. As even friction from the oral soft tissues [47–49] or the diet is capable of removing softened enamel, it is very unlikely that the limited amount of remineralisation observed in tests in situ suggests would significantly counteract the effect of a series of erosive challenges during the course of a day. The main factors limiting remineralisation seem to be the rather low degree of supersaturation of saliva and the presence of salivary proteins, e.g. statherin, which inhibit crystal growth. Complete loss of the outermost enamel through exposure to a very prolonged or severe erosive challenge cannot be repaired by remineralisation as there no longer exists even a framework for crystal growth.

As reliance cannot be placed on remineralisation of erosive lesions, it is preferable to avoid formation of erosive lesions, by reducing consumption of erosive products or by application of oral healthcare products to reduce the susceptibility of tooth surfaces to erosion.

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