Dental Erosion and Its Clinical Management

Bennett T. Amaechi
Editor

Springer

Dental Erosion and Its Clinical Management

Bennett T. Amaechi
Editor

Dental Erosion and Its Clinical Management

Springer

Editor
Bennett T. Amaechi
Comprehensive Dentistry
University of Texas Health Science Center
San Antonio
Texas
USA

ISBN 978-3-319-13992-0 ISBN 978-3-319-13993-7 (eBook)
DOI 10.1007/978-3-319-13993-7

Library of Congress Control Number: 2015950482

Springer Cham Heidelberg New York Dordrecht London

Printed on acid-free paper

Springer International Publishing AG Switzerland is part of Springer Science+Business Media (www.springer.com)

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

1 Dental Erosion: Prevalence, Incidence and Distribution 3
D.H.J. Jager

2 The Dental Erosion Process 13
R.P. Shellis

3 Causes of Dental Erosion: Intrinsic Factors 35
Taís Scaramucci, Joana C. Carvalho, Anderson T. Hara, and Domenick T. Zero

4 Causes of Dental Erosion: Extrinsic Factors 69
Anderson T. Hara, Joana C. Carvalho, and Domenick T. Zero

Part II Clinical Practice

5 Diagnosis of Dental Erosion 99
Carolina Ganss and Nadine Schlueter

6 Assessment and Monitoring of Dental Erosion 111
Bennett T. Amaechi

7 Dental Erosive Wear Risk Assessment 121
Alix Young, Kjersti Refsholt Stenhagen, Aida Mulic, and Bennett T. Amaechi

8 Prevention and Control of Dental Erosion: Patient Self-Care 133
Marília Afonso Rabelo Buzalaf, Cristiane de Almeida Baldini Cardoso, Ana Carolina Magalhães, and Bennett T. Amaechi

9 Prevention and Control of Dental Erosion: Professional Clinic Care 151
John A. Kaidonis, Poppy M. Anastassiadis, Dimitra Lekkas, Sarbin Ranjitkar, Bennett T. Amaechi, and Grant C. Townsend

10 Prevention and Control of Dental Erosion: Dietary Management 169
Georgiana S. Gross and Bennett T. Amaechi

11 Prevention and Control of Dental Erosion: Psychological Management 181
Gracemary Leung and Bennett T. Amaechi

12 Prevention and Control of Dental Erosion: Gastroesophageal Reflux Disease Management 203
Akit Patel, Bennett T. Amaechi, and Charles Brady III

13 Restoration of the Worn Dentition 225
Paul A. King

14 Noncarious Cervical Lesions: Prevalence, Etiology, and Management. 249
Karen B. Troendle and Kevin M. Gureckis

15 Dentin Hypersensitivity: Prevalence, Etiology, Pathogenesis, and Management. 275
Cor van Loveren, Patrick R. Schmidlin, Luc C. Martens, and Bennett T. Amaechi

16 Recall, Maintenance Care Cycle, and Outcomes Assessment. 303
Bennett T. Amaechi

Index. 313

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.

D.H.J. Jager, DMD, PhD, Mpros RCSed
Department of Maxillofacial Surgery, Oral Pathology and
Center for Special Care Dentistry (SBT), VU University Medical Center,
Free University, Amsterdam and Stichting Bijzondere Tandheelkunde (SBT),
P.O. Box 7057, 1007 MB,
Amsterdam, The Netherlands
e-mail: d.jager@vumc.nl

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_1

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

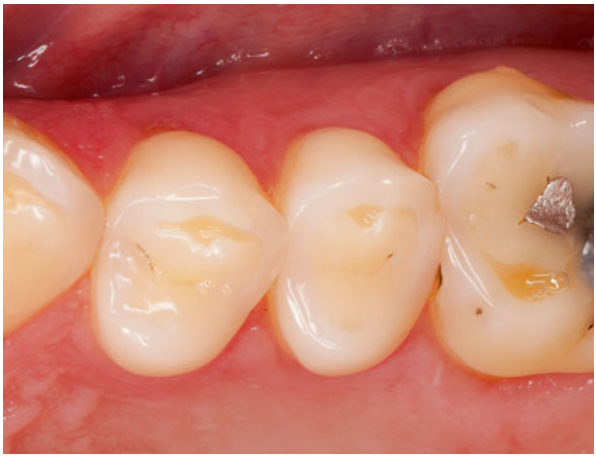


Fig. 1.1 Typical signs of erosion: a smooth silky glazed appearance, change in colour, cupping and grooving on occlusal surfaces

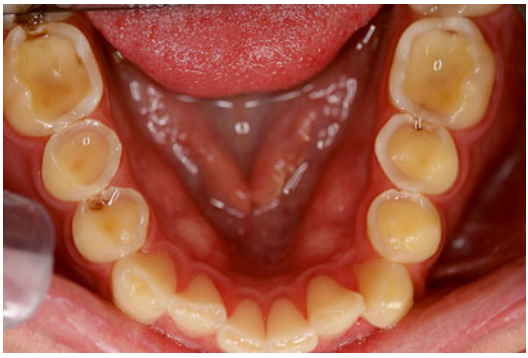


Fig. 1.2 Advanced stage of dental erosion

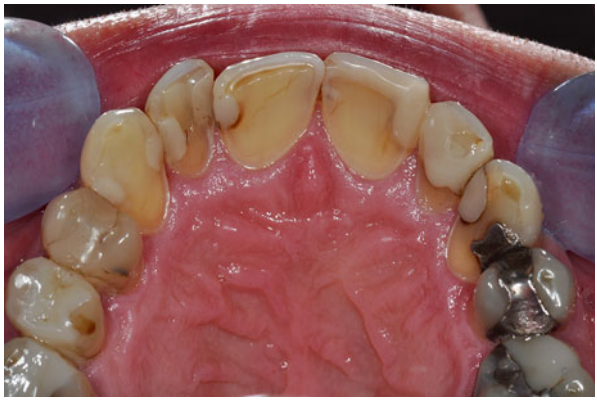


Fig. 1.3 Palatal dental erosion related to gastric reflux

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].

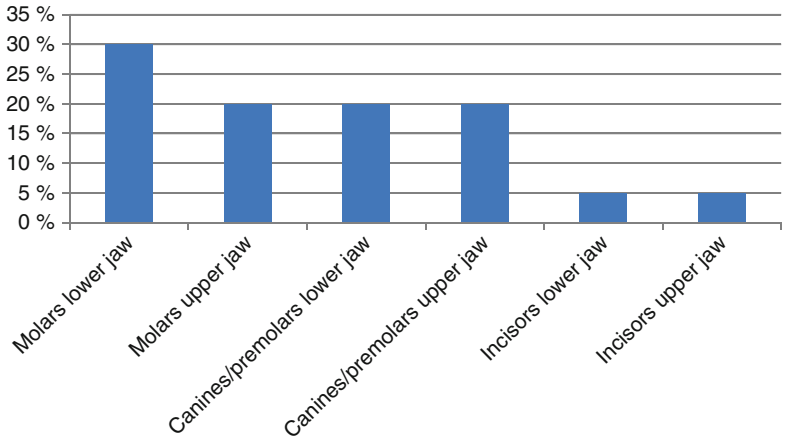


Fig. 1.4 Distribution of occlusal erosion lesions (Modified from Lussi et al. [32])

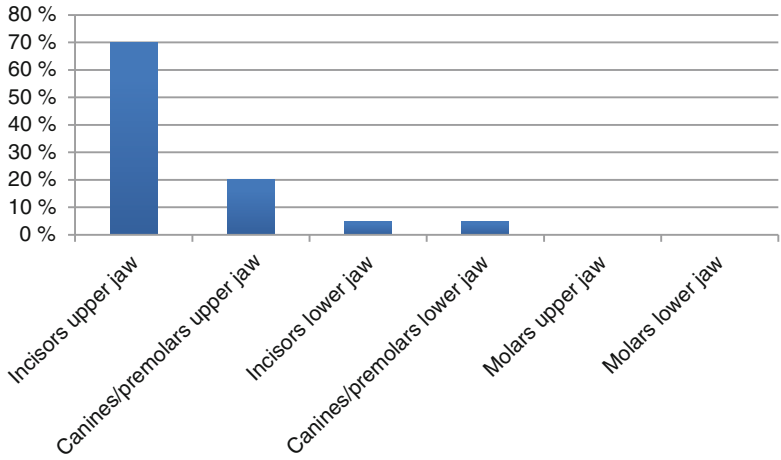


Fig. 1.5 Distribution of lingually located erosion lesions (Modified from Lussi et al. [32])

References

1. Ganss C, Lussi A. Diagnosis of erosive tooth wear. Monogr Oral Sci. 2006;20:32–43.
2. Lussi A, Jaeggi T, Schaffner M. Prevention and minimally invasive treatment of erosions. Oral Health Prev Dent. 2004;2 Suppl 1:321–5.
3. ten Cate JM, Imfeld T. Dental erosion, summary. Eur J Oral Sci. 1996;104(2 (Pt 2)):241–4.
4. Dugmore CR, Rock WP. A multifactorial analysis of factors associated with dental erosion. Br Dent J. 2004;196:283–6.
5. Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. West J Med. 2000;173:378–83. Erratum in: West J Med 2001; 175:235.
6. Smith BG, Knight JK. A comparison of patterns of tooth wear with aetiological factors. Br Dent J. 1984;157:16–9.
7. Lussi A. Erosive tooth wear – a multifactorial condition of growing concern and increasing knowledge. Monogr Oral Sci. 2006;20:1–8.
8. Bardsley PF. The evolution of tooth wear indices. Clin Oral Investig. 2008;12 Suppl 1:S15–9.
9. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008;12 Suppl 1:S65–8.
10. Jaeggi T, Lussi A. Prevalence, incidence and distribution of erosion. Monogr Oral Sci. 2006;20:44–65.
11. El Aidi H, Bronkhorst EM, Truin GJ. A longitudinal study of tooth erosion in adolescents. J Dent Res. 2008;87:731–5.
12. Lanigan LT, Bartlett DW. Tooth wear with an erosive component in a Mediaeval Iceland population. Arch Oral Biol. 2013;58:1450–6.
13. 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.
14. Truin GJ, van Rijkom HM, Mulder J, van't Hof MA. Caries trends 1996–2002 among 6- and 12-year-old children and erosive wear prevalence among 12-year-old children in The Hague. Caries Res. 2005;39:2–8.
15. Bartlett DW, Lussi A, West NX, Bouchard P, Sanz M, Bourgeois D. Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young European adults. J Dent. 2013;41:1007–13.

16. McGuire J, Szabo A, Jackson S, Bradley TG, Okunseri C. Erosive tooth wear among children in the United States: relationship to race/ethnicity and obesity. Int J Paediatr Dent. 2009;19: 91–8.
17. Okunseri C, Okunseri E, Gonzalez C, Visotcky A, Szabo A. Erosive tooth wear and consumption of beverages among children in the United States. Caries Res. 2011;45:130–5.
18. Mungia R, Zarzabal LA, Dang SC, Baez M, Stookey GK, Brown JP. Epidemiologic survey of erosive tooth wear in San Antonio, Texas. Tex Dent J. 2009;126:1097–109.
19. Wang P, Lin HC, Chen JH, Liang HY. The prevalence of dental erosion and associated risk factors in 12-13-year-old school children in Southern China. BMC Public Health. 2010;10:478.
20. Luo Y, Zeng XJ, Du MQ, Bedi R. The prevalence of dental erosion in preschool children in China. J Dent. 2005;33:115–21.
21. Pace F, Pallotta S, Tonini M, Vakil N, Bianchi Porro G. Systematic review: gastro-oesophageal reflux disease and dental lesions. Aliment Pharmacol Ther. 2008;27:1179–86.
22. Böhmer CJ, Klinkenberg-Knol EC, Niezen-de Boer MC, Meuwissen PR, Meuwissen SG. Dental erosions and gastro-oesophageal reflux disease in institutionalized intellectually disabled individuals. Oral Dis. 1997;3:272–5.
23. Myklebust S, Espelid I, Svalestad S, Tveit AB. Dental health behavior, gastroesophageal disorders and dietary habits among Norwegian recruits in 1990 and 1999. Acta Odontol Scand. 2003;61:100–4.
24. Jensdottir T, Arnadottir IB, Thorsdottir I, Bardow A, Gudmundsson K, Theodors A, Holbrook WP. Relationship between dental erosion, soft drink consumption, and gastroesophageal reflux among Icelanders. Clin Oral Investig. 2004;8:91–6.
25. Edeer D, Martin CW. Occupational dental erosion. Richmond: WorksafeBC Evidence- Based Practice Group; 2010. Available at: http://worksafebc.com/health_care_providers/Assets/PDF/occupational_dental_erosion.pdf.
26. Wiegand A, Attin T. Occupational dental erosion from exposure to acids: a review. Occup Med. 2007;57:169–76.
27. Piccininni PM, Fasel R. Sports dentistry and the olympic games. J Calif Dent Assoc. 2005; 33:471–83.
28. Yang XJ, Schamach P, Dai JP, Zhen XZ, Yi B, Liu H, Hu M, Clough T, Li Y, Ma CM. Dental service in 2008 Summer Olympic Games. Br J Sports Med. 2011;45:270–4.
29. Needleman I, Ashley P, Petrie A, Fortune F, Turner W, Jones J, Niggli J, Engebretsen L, Budgett R, Donos N, Clough T, Porter S. Oral health and impact on performance of athletes participating in the London 2012 Olympic Games: a cross-sectional study. Br J Sports Med. 2013;47:1054–8.
30. Mandel L. Dental erosion due to wine consumption. J Am Dent Assoc. 2005;136:71–5.
31. Chikte UM, Naidoo S, Kolze TJ, Grobler SR. Patterns of tooth surface loss among winemakers. SADJ. 2005;60:370–4.
32. Lussi A, Schaffner M, Hotz P, Suter P. Dental erosion in a population of Swiss adults. Community Dent Oral Epidemiol. 1991;19:286–90.

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.

R.P. Shellis
Department of Preventive, Restorative and Paediatric Dentistry,
School of Dental Medicine, University of Bern, Bern, Switzerland

Klinik für Zahnerhaltung, Universität Bern, Freiburgstrasse, Bern, Switzerland
e-mail: peter.shellis@btinternet.com

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_2

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

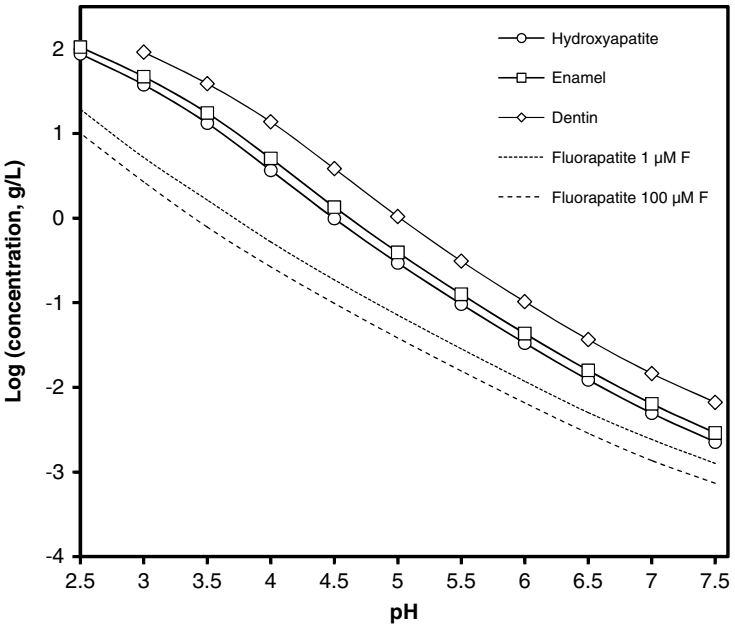


Fig. 2.1 Solubility diagram for solids relevant to erosion. *Lines* represent equilibrium solubilities over the pH range 2.5–7.5. Solubilities for fluorapatite are given for the lowest and highest fluoride concentrations in the products studied by Lussi et al. [7]

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,

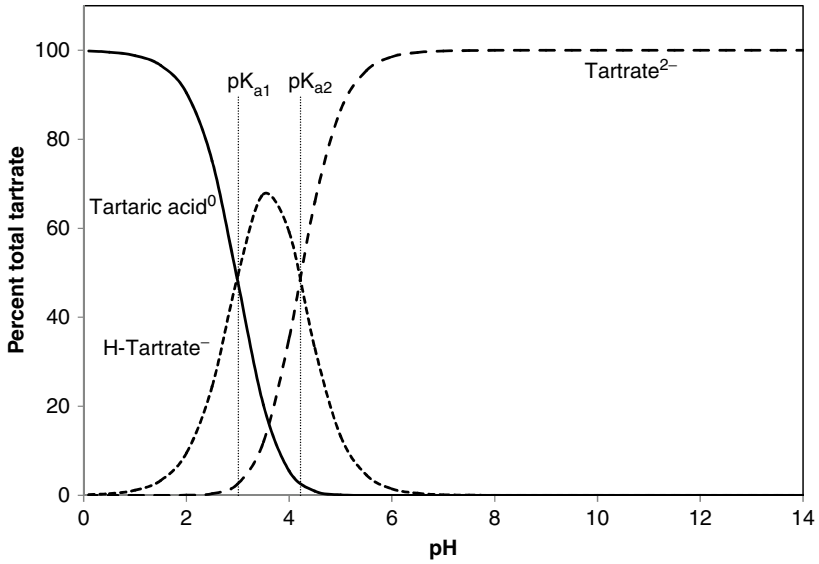


Fig. 2.2 Diagram illustrating ionisation of tartaric acid. As pH rises, tartaric acid dissociates into HTartrate$^-$ ions and this in turn into tartrate^{2-} ions

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.

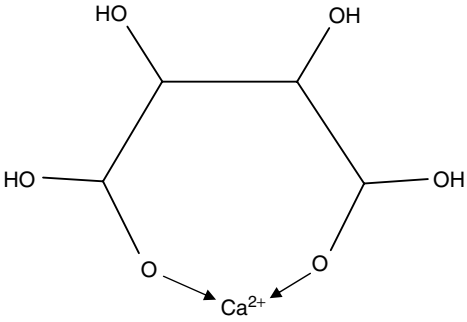


Fig. 2.3 An example of chelation: a schematic diagram of the tartrate-calcium complex. The *arrows* symbolise coordinate bonds, formed by sharing of electrons from the COO^- groups with the calcium ion

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].

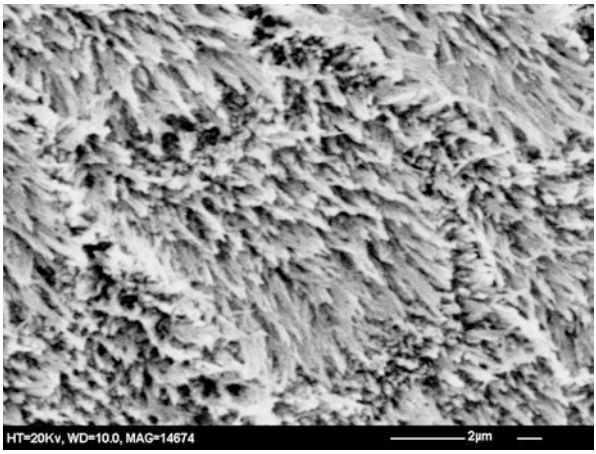


Fig. 2.4 Scanning electron micrograph of the surface of a specimen of enamel which has been exposed to 0.3 % citric acid (pH 3.2) for 20 min. Etching of the surface revealing the profiles of the prisms. At the outer surface, the crystals are more completely demineralised than those deeper in the softened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

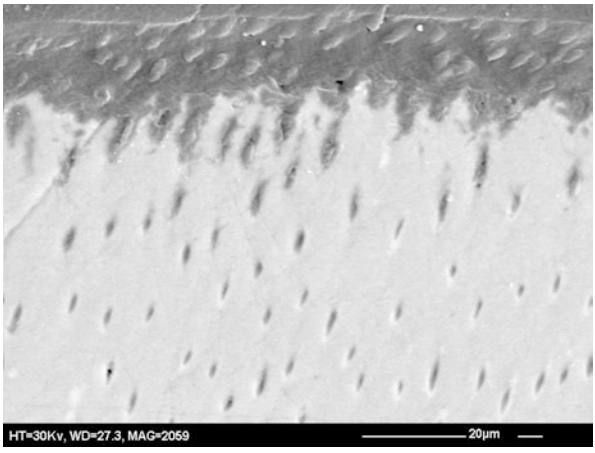


Fig. 2.5 Cross section of dentin exposed to 0.3 % citric acid, pH 3.2, for 20 min. Surface was polished using a graded series of diamond paste then viewed in a scanning electron microscope with a backscattered electron detector. In this mode, contrast from flat surfaces is due to variations in average 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

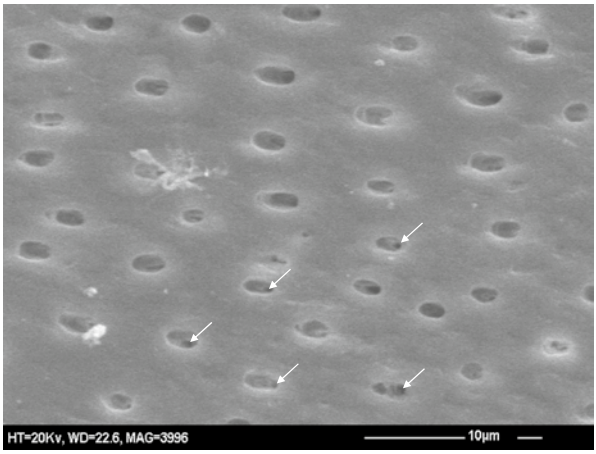


Fig. 2.6 Polished surface of dentin exposed to 0.3 % citric acid solution, pH 3.2, for 20 min. Note the enlarged openings of the tubules and the absence of peritubular dentin. Tubule openings visible beneath the surface (*arrows*) are small because the peritubular dentin is intact at this level

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.

References

1. Kaidonis JA. Tooth wear: the view of the anthropologist. Clin Oral Invest. 2008;12(suppl): 54–8.
2. Bartlett DW, Smith BGN. Definition, classification, and clinical assessment of attrition, erosion and abrasion of enamel and dentine. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. p. 87–92.
3. Johansson AK, Omar R, Carlsson GE, Johansson A. Dental erosion and its growing importance in clinical practice: from past to present. Int J Dent. 2012; Article ID 632907:17.
4. Shellis RP. Transport processes in enamel and dentine. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. p. 19–27.
5. Verbeeck RMH. Minerals in human enamel and dentine. In: Driessens FCM, Wöltgens JHM, editors. Tooth development and caries. Boca Raton: CRC Press; 1986. p. 95–152.
6. Shellis RP, Featherstone JDB, Lussi A. Understanding the chemistry of dental erosion. In: Lussi A, editor. Dental erosion (Monogr Oral Sci 20). 2nd ed. Basel: Karger; 2014.
7. Lussi A, Megert B, Shellis RP, Wang X. Analysis of the erosive effect of different dietary substances and medications. Brit J Nutr. 2012;30:1–11.
8. Stumm W. Chemistry of the solid-water interface. New York: Wiley; 1992.
9. Lussi A, Schlueter N, Rakhmatullina E, Ganss C. Dental erosion – an overview with emphasis on chemical and histopathological aspects. Caries Res. 2011;45 Suppl 1:2–12.
10. Millward A, Shaw L, Harrington E, Smith AJ. Continuous monitoring of salivary flow rate and pH at the surface of the dentition following consumption of acidic beverages. Caries Res. 1997;31:44–9.

11. Shellis RP, Barbour ME, Jones SB, Addy M. Effects of pH and acid concentration on erosive dissolution of enamel, dentine and compressed hydroxyapatite. Eur J Oral Sci. 2010;118: 475–82.
12. Ganss C, Schlueter N, Hardt M, von Hinckeldey J, Klimek J. Effects of toothbrushing on eroded dentine. Eur J Oral Sci. 2007;115:390–6.
13. West NX, Davies M, Amaechi BT. In vitro and in situ erosion models for evaluating tooth substance loss. Caries Res. 2011;45 Suppl 1:43–52.
14. Jensdottir T, Nauntofte B, Buchwald C, Bardow A. Effects of sucking acidic candy on whole-mouth saliva composition. Caries Res. 2005;39:468–74.
15. Mahoney E, Beattie J, Swain M, Kilpatrick N. Preliminary in vitro assessment of erosive potential using the ultra-micro-indentation system. Caries Res. 2003;37:218–24.
16. Lussi A, Jäggi T, Schärer S. The influence of different factors on in vitro enamel erosion. Caries Res. 1993;27:387–93.
17. Lussi A, Jaeggi T, Jaeggi-Schärer S. Prediction of the erosive potential of some beverages. Caries Res. 1995;29:349–54.
18. Barbour ME, Parker DM, Allen GC, Jandt KD. Enamel dissolution in citric acid as a function of calcium and phosphate concentrations and degree of saturation with respect to hydroxyapatite. Eur J Oral Sci. 2003;111:421–33.
19. Davis WB, Winter PJ. The effect of abrasion on enamel and dentine after exposure to dietary acid. Br Dent J. 1980;148:253–6.
20. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res. 1999;33:81–7.
21. Shellis RP, Barbour ME, Jesani A, Lussi A. Effects of buffering properties and undissociated acid concentration on dissolution of dental enamel, in relation to pH and acid type. Caries Res. 2013;47:601–11.
22. Gray JA. Kinetics of enamel dissolution during formation of incipient caries-like lesions. Arch Oral Biol. 1961;11:397–421.
23. Featherstone JDB, Rodgers BE. Effect of acetic, lactic and other organic acids on the formation of artificial carious lesions. Caries Res. 1981;15:377–85.
24. Wong L, Cutress TW, Duncan JF. The influence of incorporated and adsorbed fluoride on the dissolution of powdered and pelletized hydroxyapatite in fluoridated and non-fluoridated acid buffers. J Dent Res. 1987;66:1735–41.
25. Arends J, Christoffersen J. Nature and role of loosely bound fluoride in dental caries. J Dent Res. 1990;69:601–5.
26. Hughes JA, West NX, Parker DM, Newcombe RG, Addy M. Development and evaluation of a low erosive blackcurrant juice drink in vitro and in situ 3. Final drink and concentrate, formulae comparisons in situ and overview of the concept. J Dent. 1999;27:345–50.
27. Amaechi BT, Higham SM, Edgar WM. Factors influencing the development of dental erosion in vitro: enamel type, temperature and exposure time. J Oral Rehabil. 1999;26:624–30.
28. Barbour ME, Finke M, Parker DM, Hughes JA, Allen GC, Addy M. The relationship between enamel softening and erosion caused by soft drinks at a range of temperatures. J Dent. 2006;34:207–13.
29. Shellis RP, Finke M, Eisenburger M, Parker DM, Addy M. Relationship between enamel erosion and flow rate. Eur J Oral Sci. 2005;113:232–8.
30. Wiegand A, Stock A, Attin R, Werner C, Attin T. Impact of the acid flow rate on dentin erosion. J Dent. 2007;35:21–7.
31. Järvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991;70: 942–7.
32. Sánchez GA, Fernandez De Preliasco MV. Salivary pH changes during soft drinks consumption in children. Int J Paediatr Dent. 2003;13:251–7.
33. Zwier N, Huysmans MCDNJM, Jager DHJ, Ruben J, Bronkhorst EM. Saliva parameters and erosive wear in adolescents. Caries Res. 2013;47:548–52.
34. Hannig M. Ultrastructural investigation of pellicle morphogenesis at two different intraoral sites during a 24-h period. Clin Oral Invest. 1999;3:88–95.

35. Lie T. Scanning and transmission electron microscope study of pellicle morphogenesis. Scand J Dent Res. 1977;85:217–31.
36. Sønju Clasen AB, Hannig M, Skjørland K, Sønju T. Analytical and ultrastructural studies of pellicle on primary teeth. Acta Odontol Scand. 1997;55:339–43.
37. Carlén A, Börjesson A-C, Nikdel K, Olsson J. Composition of pellicles formed in vivo on tooth surfaces in different parts of the dentition, and in vitro on hydroxyapatite. Caries Res. 1998;32:447–55.
38. Siqueira WL, Custodio W, McDonald EE. New insights into the composition and functions of the acquired enamel pellicle. J Dent Res. 2012;91:1110–8.
39. Slomiany BL, Murty VLN, Zdebska E, Slomiany A, Gwodzinski K, Mandel ID. Tooth surface-pellicle lipids in the protection of dental enamel against lactic acid diffusion in man. Arch Oral Biol. 1986;31:187–91.
40. Wiegand A, Bliggenstorfer S, Magalhaes AC, Sener B, Attin T. Impact of the in situ formed salivary pellicle on enamel and dentine erosion induced by different acids. Acta Odont Scand. 2008;66:225–30.
41. Zahradnik RT, Moreno EC, Burke EJ. Effect of salivary pellicle on enamel subsurface demineralization in vitro. J Dent Res. 1976;55:664–70.
42. Wetton S, Hughes J, Newcombe RG, Addy M. The effect of saliva derived from different individuals on the erosion of enamel and dentine. A study in vitro. Caries Res. 2007;41:423–6.
43. Hannig M, Fiebiger M, Güntzer M, Döbert A, Zimehl R, Nekrashevych Y. Protective effect of the in situ formed short-term salivary pellicle. Arch Oral Biol. 2004;49:903–10.
44. Wetton S, Hughes J, West N, Addy M. Exposure time of enamel and dentine to saliva for protection against erosion: a study in vitro. Caries Res. 2006;40:213–7.
45. Hannig M, Hess NJ, Hoth-Hannig W, De Vrese M. Influence of salivary pellicle formation time on enamel demineralization – an in situ pilot study. Clin Oral Investig. 2003;7:158–61.
46. Addy M, Hunter ML. Can tooth brushing damage your health? Effects on oral and dental tissues. Int Dent J. 2003;53 Suppl 3:177–86.
47. Gregg T, Mace S, West NX, Addy M. A study in vitro of the abrasive effect of the tongue on enamel and dentine softened by acid erosion. Caries Res. 2004;38:557–60.
48. Vieira A, Overweg E, Ruben JL, Huysmans MC. Toothbrush abrasion, simulated tongue friction and attrition of eroded bovine enamel in vitro. J Dent. 2006;34:336–42.
49. Amaechi BT, Higham SM, Edgar WM. Influence of abrasion in clinical manifestation of human dental erosion. J Oral Rehabil. 2003;30:407–13.
50. Wiegand A, Köwing L, Attin T. Impact of brushing force on abrasion of acid-softened and sound enamel. Arch Oral Biol. 2007;52:1043–7.
51. Voronets J, Lussi A. Thickness of softened human enamel removed by toothbrush abrasion: an in vitro study. Clin Oral Invest. 2010;14:251–6.
52. Wiegand A, Begic M, Attin T. In vitro evaluation of abrasion of eroded enamel by different manual, power and sonic toothbrushes. Caries Res. 2006;40:60–5.
53. 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;49:931–5.
54. Schlueter N, Glatzki J, Klimek J, Ganss C. Erosive-abrasive tissue loss in dentine under simulated bulimic conditions. Arch Oral Biol. 2012;57:1176–82.
55. Bader JD, McClure F, Scurria MS, Shugars DA, Heymann HO. Case-control study of non-carious cervical lesions. Community Dent Oral Epidemiol. 1996;24:286–91.
56. Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion and abrasion. J Dent Res. 2006;85:306–12.
57. Milosevic A, Lennon MA, Fear SC. Risk factors associated with tooth wear in teenagers: a case control study. Community Dent Health. 1997;14:143–7.
58. Al-Dlaigan YH, Shaw L, Smith AJ. Dental erosion in a group of British 14-year-old school children. Part II: Influence of dietary intake. Br Dent J. 2001;190:258–61.

59. Dugmore CR, Rock WP. A multifactorial analysis of factors associated with dental erosion. Br Dent J. 2004;196:283–6.
60. Ganss C, Schlechtriemen M, Klimek J. Dental erosions in subjects living on a raw food diet. Caries Res. 1999;33:74–80.
61. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Res. 2000;34:182–7.
62. Zero D. Behavioral factors. In: Lussi A, editor. Dental erosion. (Monogr Oral Sci 20). Basel: Karger; 2006. p. 100–5.
63. Rytömaa I, Järvinen V, Kanerva R, Heinonen OP. Bulimia and tooth erosion. Acta Odont Scand. 1998;56:36–40.
64. Wiegand A, Müller J, Werner C, Attin T. Prevalence of erosive tooth wear and associated risk factors in 2-7-year-old German kindergarten children. Oral Dis. 2006;12:117–24.
65. Jaeggi T, Lussi A. Toothbrush abrasion of erosively altered enamel after intraoral exposure to saliva: an in situ study. Caries Res. 1999;33:455–61.
66. Attin T, Knöfel S, Buchalla W, Tütüncü R. In situ evaluation of different remineralization periods to decrease brushing abrasion of demineralized enamel. Caries Res. 2001;35:216–22.
67. Amaechi BT, Higham SM. *In vitro* remineralization of eroded enamel lesions by saliva. J Dent. 2001;29:371–6.
68. Hara AT, Turssi CP, Teixeira ECN, Serra MC, Cury JA. Abrasive wear on eroded root dentine after different periods of exposure to saliva *in situ*. Eur J Oral Sci. 2003;111:423–7.
69. Attin T, Siegel S, Buchalla W, Lennon ÀM, Hannig C, Becker K. Brushing abrasion of softened and remineralised dentin: an in situ study. Caries Res. 2004;38:62–6.
70. Ganss C, Schlueter N, Friedrich D, Klimek J. Efficacy of waiting periods and topical fluoride treatment on toothbrush abrasion of eroded enamel in situ. Caries Res. 2007;41:146–51.
71. Attin T, Zirkel C, Hellwig E. Brushing abrasion of eroded dentin after application of sodium fluoride solutions. Caries Res. 1998;32:344–50.
72. Ganss C, Klimek J, Schäffer U, Spall T. Effectiveness of two fluoridation measures on erosion progression in human enamel and dentine in vitro. Caries Res. 2001;35:325–30.

Causes of Dental Erosion: Intrinsic Factors

3

Taís Scaramucci, Joana C. Carvalho, Anderson T. Hara, and Domenick T. Zero

Abstract

The consequences of the severe wear caused by intrinsic erosion, in addition to the high cost and complexity of the treatment to restore severely worn dentition, support the importance of early diagnosis and implementation of preventive/therapeutic measures. Dental professionals must be aware of the signs and symptoms of the disease, as well as all the conditions that may lead to the presence of gastric juice in the mouth, so they can make the referral to the appropriate medical specialist. In this chapter, the main clinical conditions associated with intrinsic erosion are presented and its clinical signs and symptoms discussed from a clinical perspective.

3.1 Introduction

Dental erosion can develop due to the prolonged and frequent contact of endogenous acids with the tooth surfaces. The gastric juice produced by the stomach is the main source of endogenous acid and presents high erosive potential [1–3]. The

T. Scaramucci, DDS, PhD (✉)
Department of Restorative Dentistry, School of Dentistry, University of São Paulo, Av. Prof. Lineu Prestes 2227, São Paulo, SP 05508-000, Brazil
e-mail: tais.sca@usp.br

J.C. Carvalho, DDS, PhD
Faculty of Medicine and Dentistry, School of Dentistry, Catholic University of Louvain, Louvain, Belgium
e-mail: Joana.Carvalho@uclouvain.be

A.T. Hara, DDS, MS, PhD • D.T. Zero, DDS, MS
Department of Cariology, Operative Dentistry and Public Health, Oral Health Research Institute, Indiana University School of Dentistry, Indianapolis, IN, USA
e-mail: ahara@iu.edu; dzero@iu.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_3

introduction of gastric juice into the oral cavity may be the result of a wide range of medical and psychological disorders, such as gastroesophageal reflux and bulimia. Erosive wear of the palatal surfaces of the maxillary anterior teeth is a common clinical sign. As it progresses, the condition becomes more widespread [4], and in some cases, complete destruction of the dentition is observed, supporting the importance of early diagnosis and appropriate intervention, including referral to the appropriate medical specialist [5, 6]. Because in some cases such disorders may not have a clear symptomatology or are associated with behaviors not socially acceptable, the dentist may be the first professional to diagnose it by detecting signs of erosive wear in their patients, as well as other changes in soft oral tissues, such as parotid gland enlargement, in the case of bulimia [5, 7].

3.2 Etiology

The main conditions associated with the repeated presence of gastric juice in the mouth are recurrent vomiting disorders and reflux or regurgitation [2, 4, 8]. Each of these conditions will be discussed under separate headings; however, it must be considered that in many cases multiple disorders may be operational in the same individual, as well as acids of extrinsic origin [5]. The gastric juice produced by the stomach is composed of hydrochloric acid (0.04–0.084 M), electrolytes, organic acids, carbohydrates, nitrogen substances, proteins, vitamins, and pepsin [9]. Although gastric juice was shown to have a pH in the range of 1.6–2.9 [1, 3], these values tend to increase when gastric juice reaches the oral cavity, due to the diluting and buffering actions of saliva [10]. Some studies have used hydrochloric acid to simulate intrinsic erosion [11–13]. It was observed that, after rinsing the mouth with 10 mM hydrochloric acid (pH = 2), the intraoral pH of healthy subjects did not return to baseline levels even after 30 min [14]. As the neutralization of erosive acids by saliva plays a major role in erosion development [15], the more severe pattern of erosion observed in patients with reflux or eating disorders can be a reflection of this prolonged time at low pH [1]. In addition, previous studies have shown that gastric juice has greater erosive potential than some common extrinsic sources of acid, such as carbonated cola drink [1] and orange juice [3]. Gastric juice also has proteolytic enzymes, such as pepsin and trypsin, which can partially degrade the collagen matrix of dentin, decreasing its resistance against the mechanical forces of toothbrushing, thereby increasing dentin erosive wear [16, 17].

3.2.1 Recurrent Vomiting Disorders

Vomiting is the expulsion of the stomach contents through the mouth, due to gastrointestinal motor activity [18]. There are many causes of vomiting [8]. While some disorders involve only transient vomiting, such as motion sickness, others involve chronic vomiting [5]. Several epidemiological studies have associated frequent vomiting with dental erosion [19–26]. The causes of recurrent vomiting can be

didactically separated under eating disorders, medical conditions, cyclic vomiting syndrome, side effect of drugs, psychogenic vomiting syndrome, chronic alcoholism, and pregnancy-induced vomiting.

3.2.1.1 Eating Disorders

An eating disorder can be defined as an unusual eating behavior with insufficient or excessive food intake, which is associated with distress about weight or body shape. It can appear in combination with compensatory behavior, to the detriment of the person's health [27]. A systematic review showed that patients with eating disorders have 12.4 more risk for dental erosion [28]. Among the eating disorders, bulimia nervosa is the condition most closely related to dental erosion. Bulimia nervosa is characterized by recurrent episodes of binge eating followed by compensatory behavior, such as vomiting. Another eating disorder that may be related to dental erosion is anorexia nervosa. Anorexia nervosa is associated with an inadequate intake of nutrition and inability to maintain a minimum healthy weight. It is not rare that these syndromes appear overlapped, that is, patients with anorexia also exhibit bulimic behavior and vice versa. In other situations, the patient presents symptoms that resemble anorexia or bulimia, but it does meet the diagnostic criteria for any of these diseases and it is, therefore, diagnosed as having eating disorder not otherwise specified [27, 29].

Both, bulimia and anorexia, are considered as psychopathologies, with a multifactorial and complex etiology, involving physical, emotional, familial, and social issues [30, 31]. They appear most frequently in young women from Western societies, with bulimia having higher prevalence than anorexia [30, 32]. As the eating disorders can take on different forms, the nature of the oral manifestations may be also different [5]. Table 3.1 shows epidemiological studies associating eating disorders with dental erosion.

Anorexia nervosa usually starts in adolescence, with uncontrolled diet restriction. In some cases, the disorder has a short duration and does not require intervention [30], but in other cases, the process becomes chronic, with a rate of full recovery found to be less than 50 % [50]. It presents a crude mortality rate amounted to 5 % [50], which is the highest among psychiatric diseases [51]. The main physical features of anorexia are related to the consequences of malnutrition, which can affect all organs in the body. Within the common signs and symptoms are increased sensitivity to cold, dry skin, low blood pressure, and low serum levels of sex hormones [52].

While there are many studies relating eating disorder patients with chronic vomiting and the presence of dental erosion, only few reports have described whether erosion is also common in patients with the restrictive type of anorexia, with no vomiting episodes. It was previously identified that anorexic patients have a predisposition toward eating "slimming foods," especially raw citrus fruits, such as orange, lemon, and grapefruit, and their juices. In these patients, dental erosion was commonly seen in the labial surfaces of the incisors, which is the first surface that the acid gets in contact during ingestion [19]. Another report observed severe lingual and moderate buccal erosion in almost all anorexic patients with recurrent vomiting; however, erosion lesions were uncommon in anorexic non-vomiting

Table 3.1 Sample of studies showing association between eating disorders and dental erosive wear

Study	Population	Erosion assessment	Type of eating disorder	Findings
Hurst et al. [19]	17 patients (aged 13–34 years old) with anorexia nervosa from a hospital in London, United Kingdom	Not informed	Anorexia nervosa with and without vomiting episodes or regurgitation habits	Dental erosion was significantly more common in the vomiting and regurgitating population than in non-vomiters ($p<0.04$)
Hellstrom [33]	39 patients with anorexia nervosa, which were divided into a vomiting group (mean age 26.2 ± 1.2) and non-vomiting group (mean age of 24.5 ± 1.2) from an institute in Sweden	Eccles and Jenkins index	Anorexia nervosa	The prevalence of erosion was of 85 % in the vomiting group and of 25 % in the non-vomiting group. Erosion was significantly associated with vomiting
Roberts and Li [34]	47 women (aged 17–36 years old) from the National Institute of Dental Research in the United States	Not informed	30 patients with bulimia nervosa and 17 with anorexia nervosa	6 (35 %) of the patients with anorexia nervosa and 10 (33 %) of the patients with bulimia nervosa showed lingual erosion of the maxillary anterior teeth
Milosevic and Slade [35]	58 eating disorder patients from a clinic and from a dental hospital (mean age ranging from 23.6 to 25 years old) and 50 healthy controls (mean age 23.1) members of a university in Liverpool, United Kingdom	Smith and Knight index (TWI)	33 vomiting bulimics, 7 non-vomiting bulimics, and 18 anorexics	Tooth wear was significantly associated with eating disorders. The prevalence of tooth wear among the controls was 6 %, among the patients with anorexia 33 %, in bulimia without self-induced vomiting 28 %, and in bulimia with self-induced vomiting 42 %
Jones and Cleaton-Jones [36]	11 bulimic women (mean age 29.8 ± 8.4) and 22 controls (mean age 28.9 ± 9) from a private dental office in Johannesburg, South Africa	Index proposed by the authors	Bulimia nervosa	The prevalence of erosion was 69 % in the bulimic group against 7 % in the control group. The bulimic had significantly higher susceptibility for erosion ($p<0.001$). The bulimic groups also showed higher frequency of erosive lesions

Howat et al. [37]	8 patients with bulimia nervosa (mean age 24.6) and 10 controls (mean age 22.2) from a university in the United States	Not informed	Bulimia nervosa	No significant difference was found in the prevalence of erosion between the two groups
Jarvinen et al. [20]	106 patients with erosion (aged 13–73 years old) and 100 controls (17–83 years old) form Helsinki, Finland	Eccles and Jenkins index	_	7 subjects with erosion reported having anorexia nervosa against none of the controls. Vomiting weekly or more frequently was significantly associated with the presence of dental erosion (odds ratio = 31)
Robb et al. [38]	122 patients with eating disorders from psychiatric institutions and 122 controls in London, England	Smith and Knight (TWI) index	Bulimia nervosa (39 patients) and anorexia nervosa (with and without vomiting: 29 and 54 patients, respectively)	All the experimental groups showed significantly higher tooth wear than the controls ($p < 0.005$)
Rytomaa et al. [21]	35 bulimic women (mean age of 25.3 ± 6.8) from a university hospital and 105 controls (mean age of 25.7 ± 7) from a university dental clinic and colleges in Helsinki, Finland	Eccles and Jenkins index	Bulimia nervosa. 25 of the patients vomit daily, 8 vomit weekly, and 2 never	22 (63 %) bulimics had dental erosion against 12 (11 %) subjects of the controls. Dental erosion was mostly observed in the buccal, lingual, and occlusal/incisal surfaces
Ohrn et al. [39]	81 patients with eating disorders (aged 17–47 years old) and 52 controls (aged 19–41 years old) from a hospital in Stockholm, Sweden	Eccles index modified by Lussi	3 subjects were diagnosed with anorexia nervosa, 7 with anorexia and bulimia, 46 with bulimia nervosa, and 25 with an eating disorder not otherwise specified	Erosive tooth wear grade 2 (with dentin involvement) was observed in 45 patients with eating disorders, while tooth wear grade 1 (confined to the enamel) was observed in 77. When compared to the control, subjects with eating disorders showed significantly more erosion. Erosive tooth wear was also significantly correlated with the years of binge/eating habit

(continued)

Table 3.1 (continued)

Study	Population	Erosion assessment	Type of eating disorder	Findings
Ohrn and Angmar-Mansson [40]	35 women (aged 20–48 years old) with eating disorders from a clinic in Stockholm, Sweden	Eccles index modified by Lussi	Follow-up of patients from the study of Ohrn et al. 1999	The authors found that the tooth wear progressed in 16 of the subjects, and no changes were observed in 19 subjects. Subjects with progressive tooth wear showed lower stimulated flow rates when compared to subjects with no erosion development
Montecchi et al. [41]	76 women and 4 males (aged 9–18 years old) with anorexia nervosa from a hospital in Rome, Italy	Not informed	Anorexia nervosa from the restrictive or binge/purging type	Erosion was found in 20 patients, mainly in those engaged in vomiting
Willumsen and Graugaard [42]	197 women (aged 16–69 years old) with eating disorders from an eating disorder group in Norway	Questionnaire	13.7 % suffered from anorexia nervosa, 8.9 % from bulimia nervosa, 26.7 % suffered from binge eating, and 50.7 % belonged to the mixed group (where more than one diagnosis was made)	The presence of self-induced vomiting episodes was reported by 56.1 % of women. 29.9 % of the women believed that they had dental erosion, but the percentage of women ever diagnosed with erosion was only 19.7 %. Significantly more women with self-induced vomiting reported having dental erosion (45.3 %) than those without vomiting 9.2 %
Linfante-Oliva et al. [43]	17 patients with eating disorders (aged 13–32 years old) of a hospital in Albacete, Spain	Not informed	41 % anorexics and 59 % bulimics	Erosion affected 20 % of the anorexic patients and more than 90 % of the bulimics
Dynesen et al. [44]	20 bulimic women (mean age 23.8 ± 4 years old) and 20 healthy matching controls (mean age 23.1 ± 2 years old) recruited at a psychiatric private practice and from the University of Copenhagen, Copenhagen, Denmark	Modified Larsen index	Bulimia nervosa	The dental erosion score was significantly higher in the bulimic group when compared to the control ($p = 0.019$). In the bulimic group, there was a significant relation between the duration of the eating disorder in years and dental erosion score ($p = 0.007$)

Shaughnessy et al. [45]	23 women with anorexia nervosa (aged 15–30 years old) from a children's hospital in Boston, United States	Not informed	Anorexia nervosa	Dental erosion was not observed in any of the participants. 26 % of the subjects reported history of binge eating and purging activity
Emodi-Perlman et al. [46]	women with chronic eating disorders (aged 18–35 years old) and 48 healthy matching controls (aged 18–36) from an eating disorder center and a school of dental medicine, respectively, in Tel Aviv, Israel	Johansson et al. 1993	Of the 79 women, 43 had self-reported vomiting and 36 non-vomiting patients	Women with eating disorders had significantly more erosive lesions ($p<0.001$) than the controls. There was no significant difference in the degree of erosion between vomiting and non-vomiting groups
Ximenes et al. [47]	650 adolescents (aged 12–16 years old) from public schools in Recife, Brazil	Not informed		215 adolescents (33.1 %) were considered to be at risk for eating disorders. Dental erosion showed a significant association to symptoms of eating disorders ($p<0.001$)
Johansson et al. [22]	54 patients (aged 10–50 years old) with eating disorders and 54 healthy matching controls from an eating disorder clinic in Orebro, Sweden	Ordinal scale, which assigned a score to each degree of wear of maxillary incisors and canines	28 % of the patients had anorexia nervosa, 14 % anorexia nervosa, and 58 % an eating disorder not otherwise specified	25 (44 %) patients with eating disorders reported vomiting habits. Patients with eating disorders had 8.5 more risk for erosion. Of these, the presence of vomiting/binge eating behavior showed 5.5 more erosion
Hermont et al. [48]	1,203 female patients (aged 15–18 years old) randomly selected from public and private schools in Belo Horizonte, Brazil. Of these, 72 participants (6 %) showed a score of 20 or more in an adapted version of the Bulimic Investigatory Test indicating high chance for bulimia nervosa	O'Sullivan tooth erosion index	Bulimia nervosa	45 % of the patients in the bulimia nervosa group showed erosion against only 8.8 % in the control group ($p<0.001$)

(continued)

Table 3.1 (continued)

Study	Population	Erosion assessment	Type of eating disorder	Findings
Uhlen et al. [26]	66 patients (aged 20–48 years old) with eating disorders and self-induced vomiting from an eating disorder clinic in Oslo, Norway	VEDE (Visual Erosion Dental Examination)	Eating disorder patients with self-reported vomiting behavior only	46 (69.7 %) of the individuals presented dental erosion. Dentin lesions appeared most frequently on occlusal (58 %) and palatal surfaces of the front teeth (19 %). Individuals with the longest duration of self-induced vomiting (>10 years) showed 71.7 % and 40.4 % of dentin and enamel lesions, respectively
Conviser et al. [49]	201 women diagnosed with bulimia nervosa from different eating disorder treatment centers in the United States (mean age 27.4 years old)	Self-reported	Bulimia nervosa	The presence of erosion was reported by 63.8 % of the patients. 83 % of the patients reported rinsing their mouth with water or mouthrinse after purging, and 32.5 % reported brushing their teeth immediately after purging.

patients [33]. On the contrary, in a sample of anorexic and bulimic patients, the percentage of erosion was found in 35 % and 33 % of the patients, respectively [34]. Other investigations did not observe any sign of dental erosion in 23 young women with anorexia nervosa. It should be pointed out that in this study, 26 % of the participants had history of binge eating/vomiting activity [45]. A recent systematic review showed that patients with eating disorders who have a self-induced vomiting habit showed 19.6 times more risk for erosion than patients without vomiting habits [28]. Therefore, it is clear that the evidence of an association between dental erosion and anorexia is stronger for patients with self-induced vomiting habits. However, it is worth mentioning that both, anorexic and bulimic, patients may also have hyposalivation, as a result of the drugs used in their treatment (antidepressants, neuroleptics, and tranquilizers) or due to the electrolyte imbalance caused by the excessive use of diuretics and laxatives [33, 41, 44, 53]. The reduced salivary flow can significantly increase the risk for dental erosion development [20, 54–56], as detailed in Chap. 2.

Bulimia nervosa involves repeated episodes of binge eating and compensatory behavior, such as fasting, laxative misuse, or vomiting, in addition to diet restriction [57]. The binge episodes are usually accompanied by a sense of loss of control [58]. To characterize bulimia, binge eating and compensatory behaviors must occur at least twice per week for a period of 3 months [59], but the frequency of vomiting has been reported to be as high as 20 times a day [60]. On the contrary of anorexia, the body weight in bulimic patients may be normal and more prone to fluctuations from fluid shifts [61]. In bulimia, the physical features are usually a consequence of the purging methods adopted by the patients to remove the ingested food [52]. Among these, cardiac complications; gastrointestinal complications secondary to binge eating, purging, or vomiting; impaired renal function; esophagitis; parotid swelling; and electrolyte disturbances, as hypokalemia and hypophosphatemia [61, 62], can be cited. The profile of bulimic patients is usually more introspective and depressive than the anorexics [52].

Dental complications in bulimic patients are well documented, especially when the preferred purging method is vomiting. Regarding general oral health, the most common reported signs and symptoms are parotid enlargement, decreased salivary secretion, xerostomia, and dental erosion [57]. Parotid gland swelling is a painless condition that has been reported to occur in 29 % of bulimic patients [63]. Its pathogenesis has not been completely elucidated yet. It was suggested that cholinergic stimulus, because of frequent vomiting, activates the salivary glands in a chronic manner, causing acinar hypertrophy and, thus, gland enlargement [64, 65]. It was also suggested that repeated vomiting episodes can cause a neuropathy involving sympathetic nerve impairment, which results in an enlargement of acinar cells due to zymogen granule engorgement [60, 66]. Regarding salivary secretion, it has been found that bulimic patients had lower stimulated salivary flow rates and lower bicarbonate concentrations than healthy controls, but no difference was observed in the mean pH values [67]. Other studies have shown that bulimic patients had lower unstimulated salivary flow rates, with up to 55 % of them showing signs of hyposalivation (unstimulated salivary flow rate $\leq$0.2 ml/min) when compared to matched controls [21, 44]. It has also been observed that enzymatic activity (such as

proteases, collagenases, and pepsin in resting saliva and proteases in stimulated saliva) is different in bulimic patients with erosion and the controls. It was hypothesized that this may be a contributing factor for the development of erosion, possibly due to the action of these enzymes in the degradation of the dentin's organic matrix and in the weakening of the protective effect of the salivary pellicle [68]. The subjective complaint of xerostomia may be related to an actual reduced salivary flow, or it can be the result of salivary compositional changes [69]. Blazer et al. reported that 77 % of the bulimic patients participating in their study had xerostomia complaint [69].

Although bulimia is often correlated to the presence of dental erosion, it is important to note that not all bulimic individuals exhibit erosion. The occurrence and severity of dental erosion result from its multifactorial nature. In bulimic patients, erosion seems to be associated with the duration of the disease, the frequency of vomiting, and the amount of saliva [21]. A mean frequency of 2.5 vomiting episodes a day, during 7 years, can predispose the manifestation of dental erosion [68]. It was found that individuals who vomited once per week or more had a risk of dental erosion 31 times greater than individuals who vomited less frequently [20]. Other reports showed that patients with eating disorders accompanied by vomiting behavior had 5.5 more dental erosions than patients with eating disorders without this behavior [22]. Dental erosion was observed in 63 % of bulimic patients and in only 11 % of the controls in a study performed in Finland. In this same study, it was also observed that the erosive lesions in the bulimics presented higher degree of severity [21]. These findings were corroborated by a more recent study, which observed erosion in 70 % of the bulimic patients with self-induced vomiting. In the group with the longest duration of vomiting (more than 10 years), the severity of the process was greater, with 71.7 % of lesions reaching the dentin [26].

Rumination is a syndrome consisting of effortless regurgitation of recently ingested food into the mouth, which is then remasticated and reswallowed or sometimes expectorated [70, 71]. Along with bulimia and anorexia, rumination is also classified as a feeding and eating disorder [72]. Regurgitation in the case of rumination is not accompanied by heartburn, abdominal pain, or nausea [73]. This disorder can be found in infants and in mentally disabled individuals, but it was also reported to occur in adolescents, adults with normal intelligence, and bulimic patients [73–75]. Among the complications of rumination are weight loss, malnutrition, halitosis, and dental erosion [4, 73, 74]. There are only few case reports associating rumination with its dental consequences in the dental literature. According to Moazzez and Bartlett, the pattern of dental erosion in these patients is similar to other conditions where stomach acid is regurgitated. It affects the palatal surfaces of the upper incisors, and with progression, other surfaces also become involved [4].

Due to the social stigma associated with eating disorders, it is often difficult for the patients to disclose the disease [76]. Considering that dental practitioners usually examine patients on a regular basis, they may play an important role in the early identification of the disease [77]. In this context, comprehensive knowledge about the oral manifestations of eating disorders is essential for dental professionals.

3.2.1.2 Medical Conditions

The main medical conditions associated with vomiting include gastrointestinal disorders (peptic ulcer, chronic gastritis, and gastric motility problems), metabolic and endocrine disorders (diabetes mellitus and hyperthyroidism), and neurological and central nervous system disorders (migraine headaches and intracranial neoplasms) [5, 8].

3.2.1.3 Cyclic Vomiting Syndrome

Cyclic vomiting syndrome is characterized by recurrent attacks of nausea and vomiting that may last for periods of a few days to several months, which is separated by symptom-free periods [78]. Its reported prevalence was about 2 % in the pediatric population [79], but it can affect all ages, including young and middle-aged adults [80, 81]. This disorder has an uncertain etiology, but it has been related to a dysfunction of the central neural pathways and neuroendocrine mediators involved in the brain-gut pathways of nausea and vomiting [80, 82]. Most attacks are regarded to occur without any predictable preceding event [83]. Among the common triggering factors are stress, emotional excitement, and infections [80]. Milosevic reported a typical pattern of palatal erosion in a 23-year-old female with an 18-year history of cyclic vomiting [84].

3.2.1.4 Side Effect of Drugs

The side effects of drugs are one of the most common causes of vomiting. A wide range of drugs have been associated with central emetic side effects and secondary effects due to gastric irritation [8]. Among the drugs with central emetic effects are opiate analgesics [85] and chemotherapeutics agents [86]. Other drugs can induce vomiting secondary to gastric irritation, such as aspirin, diuretics, and alcohol [5].

3.2.1.5 Psychogenic Vomiting Syndrome

Psychogenic vomiting syndrome affects mostly young women, and it involves recurrent vomiting, which may be caused by an underlying emotional disturbance [87]. It can be diagnosed by several clinical features associated with vomiting, including chronic or intermittent nature over a period of years, occurrence during or after meals, no relationship with nausea and oftentimes self-induced, of little concern to the patient, no changes in appetite, and may subside after hospitalization [5].

3.2.1.6 Chronic Alcoholism

Alcoholism is a neurobehavioral disorder characterized by compulsive seeking of alcohol, excessive and uncontrolled alcohol intake, and a negative emotional state when alcohol is unavailable [59]. The secretive nature of this affliction and denial of the problem may impose difficulties in clinical diagnosis [5]. Lifetime rates of alcohol abuse and dependence were reported to be 11.8 % and 13.2 %, respectively, in an American population, with men having more risk than women [88]. Alcoholism can result in a series of dental implications, such as high caries incidence due to neglected oral hygiene, dental attrition due to alcohol-stimulated bruxism, and oral cancer [89]. Dental erosion due to alcohol abuse can be caused by both intrinsic factors (vomiting and regurgitation) and extrinsic factors [5], depending on the

erosive potential of the alcoholic drink that is ingested. For example, wines and alcopops have a low pH [90, 91] and may be highly erosive to the teeth. The association between chronic alcoholism and dental erosion was reported in case reports [92–94], case-control [95], and cross-sectional [96] studies. Robb and Smith reported erosive tooth wear in the palatal surfaces of the upper anterior teeth in 40 % of alcoholic patients [95]. Erosion was observed in 49.1 % of 1064 teeth from patients with alcoholism. Erosive lesions were most commonly found at the palatal surfaces of the anterior teeth [96]. It was also reported that excessive alcohol intake can lead to chronic problems, as gastroesophageal reflux [8, 92, 97], which may further predispose to the occurrence of dental erosion.

3.2.1.7 Pregnancy-Induced Vomiting

Pregnancy-induced vomiting can affect up to 52 % of women in early pregnancy [98]. Due to the transient nature of this condition, it is generally not considered to be a major risk factor for dental erosion [8]. However, in some cases, vomiting can be prolonged or it can occur during multiple pregnancies [5]. Hyperemesis gravidarum, on the other hand, is a severe and intractable form of nausea that was reported to affect approximately 1.5 % of pregnant women [99]. The consequences of the hyperemesis are dehydration and imbalances of fluid and electrolyte, disturbances on nutritional intake and metabolism, and physical and psychological debilitation, which often involve hospital care [100]. As a secondary effect of dehydration, hyposalivation may also occur, increasing the risk of dental erosion [5]. Evans and Briggs reported a clinical case of a 29-year-old patient with palatal erosion who had severe and prolonged vomiting during two pregnancies [101]. The evidence linking dental erosion with pregnancy-induced vomiting is mostly anecdotal. Well-designed studies are needed to support a possible cause-effect relationship between erosion and pregnancy-induced vomiting in contemporary populations.

3.2.2 Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) may also be an important risk factor for dental erosion. GERD has been defined as “a condition that develops a reflux of gastric contents (into the esophagus or beyond: larynx, oral cavity or lung) and which causes troublesome symptoms and/or complications” [102, 103]. GERD is the most common gastrointestinal disease, affecting 10–20 % of individuals in the Western world and 5 % in Asia [104]. The lower esophageal sphincter is localized at the gastroesophageal junction, and its main function is ensuring that all the acids secreted by the gastric mucosa remain in the stomach and do not flow up onto the esophagus. This sphincter prevents the gastric juice and the ingested food reflux from getting into the esophagus causing damage to the esophageal squamous mucosa or possible aspiration into the upper airways [105, 106]. A relaxation of the lower esophageal sphincter is a physiological process that occurs during venting of air from the stomach and during swallowing. Relaxations that are not related to swallowing are known as transient lower esophageal sphincter relaxations, which

are responsible for the majority of the reflux episodes [106–108]. There are some factors that can predispose lower esophageal sphincter relaxations, such as caffeine, fat, smoking, some drugs, and gastric distention, which will increase the chances for reflux [108]. The presence of hiatus hernia appears to increase the magnitude of the reflux during transient relaxations [108]. This type of reflux mainly occurs after meals during the day [106]. The abnormal transient relaxations, however, seem to be less determinant in cases of severe reflux disease, which is more related to a hypotensive lower esophageal sphincter that allows high-pressure gradients across the diaphragm [106, 108]. This type of reflux is more associated to nocturnal reflux episodes [106]. According to Banks [108], nocturnal reflux causes more damage due to the depressed salivation during sleep, since the saliva plays a role in neutralizing acid in the esophagus. The acid in the esophagus is also cleared by esophageal peristalsis, and impairment of this function can lead to prolonged acid exposure as a result of the reflux episodes [108]. Other risk factors for gastric reflux include conditions that cause an increase in intra-abdominal pressure, such as obesity and pregnancy [109]. It is important to note that the reflux of gastric contents into the esophagus occurs several times a day, but usually they are an asymptomatic condition; however, in some patients, the reflux leads to unpleasant symptoms, such as heartburn and regurgitation, which is then characterized as GERD [102, 110]. Reflux can be differentiated from vomiting because it occurs without nausea, retching, or abdominal contractions [5].

Studies have shown that the refluxate is a heterogeneous mixture of gastric acid; small amounts of undigested food particles and pepsin, as well as bile acids; and trypsin when there is accompanying duodenogastric reflux [111]. GERD can result in several medical conditions affecting the esophagus, such as esophagitis, stricture, the development of columnar metaplasia in place of the normal squamous epithelium (Barrett's esophagus), and adenocarcinoma. Laryngitis, chronic cough, and dental erosion are some of the most common extraesophageal consequences [103]. However, not all affected individuals will have the classical signs and symptoms of GERD. The potential for tooth erosion is variable and depends on the composition and pH of the refluxate; the frequency and the form it reaches the mouth (either through regurgitation or belching acidic vapors); the flow rate, buffer capacity, and clearance action of saliva; and brushing after the regurgitation episodes [112]. The diagnosis and management of GERD is discussed in detail in Chap. 12.

3.2.2.1 Association Between GERD and Dental Erosion

Several case reports [113–119], case-control [120–129], and observational clinical studies in adults [20, 130, 131], adolescents [23], and children [132, 133] have demonstrated the relationship between GERD and erosion or GERD and tooth wear (Table 3.2). In some of these case-control studies, dental erosion was associated with a range of 22–47 % of patients with GERD [121, 127, 129]. A systematic review performed in 2008 to evaluate the prevalence of erosion in individuals with GERD and vice versa found a median prevalence of erosion in GERD patients of 24 % and a median prevalence of GERD in adult patients with erosion of 32.5 % [153], despite difficulties in clearly demonstrating the association between GERD

Table 3.2 Sample of studies showing association between GERD and dental erosive wear

Study	Population	Erosion assessment	GERD assessment	Findings
Jarvinen et al. [130]	109 patients with GERD symptoms	Eccles and Jenkins index	Endoscopy	7 patients with dental erosion were found, and they all came from a group of 35 patients with reflux esophagitis (erosion prevalence of 20 %) or duodenal ulcer (erosion prevalence of 13 %). It was concluded that patients with increased output of gastric acid may be more prone to dental erosion
Jarvinen et al. [20]	106 patients with erosion and 100 controls (aged 13–73 years old) from a university dental clinic in Helsinki, Finland	Eccles and Jenkins index	Questionnaire	From the case and controls, respectively, 35 (33 %) and 12 (12 %) patients had gastric disease, as diagnosed by a physician. 72 (68 %) patients from the case and 24 (24 %) from the controls reported symptoms of GERD. Patients with at least one symptom of GERD weekly showed more risk for erosion (odds ratio=7)
Bartlett et al. [120]	36 patients with palatal erosion and 10 controls without palatal erosion (aged 15–74 years old)	Palatal wear	24 h esophageal pH monitoring	23 patients (64 %) with palatal erosion had GERD. GERD was significantly associated with palatal erosion
Bohmer et al. [134]	63 intellectually disabled patients (aged 15–78 years old) from three institutes in the Netherlands	Eccles and Jenkins index	15 h esophageal pH monitoring	29 patients (46 %) had evidence of erosion. GERD was diagnosed in 19 of these patients (65.5 %). GERD was significantly associated with dental erosion

Milosevic et al. 1997 [135]	80 children with palatally and/or occlusally exposed dentin and 22 controls (aged 15 years old) from schools in Liverpool, United Kingdom	Smith and Knight index (TWI)	Questionnaire	Tooth wear was not significantly associated with having stomach upset
O'Sullivan et al. [136]	53 children (aged 2–16 years old) with GERD from a children's hospital in Leeds, United Kingdom	Index proposed by the authors	Esophageal pH monitoring	Only 9 children (17 %) showed a sign of erosion, with only 1 having erosion affecting the dentin. The prevalence of erosion in the GERD group was lower than the National UK survey
Bartlett et al. [137]	210 school children (aged 11–14 years old) in London, United Kingdom	Smith and Knight index (TWI)	Questionnaire	57 % of the children presented tooth wear in more than 10 teeth, but dentin involvement was rare. No significant association was observed between higher maxillary tooth wear and GERD ($p = 0.06$)
Gregory-Head et al. [138]	10 adult patients with GERD and 10 controls (aged 18–69 years old) referred to the Division of Gastroenterology	Smith and Knight index (TWI)	24 h esophageal pH monitoring	GERD patients presented significantly higher TWI scores than the controls without GERD (means of 0.95 and 0.30, respectively). GERD was significantly associated with tooth wear
Dahshan et al. [132]	24 children (aged 2–18 years old) diagnosed with GERD	Aine index	Elective esophagogastroduodenoscopy	20 patients presented erosion (83 %). Of these, 10 had mild erosion, 6 moderate erosion, and 4 severe erosion. Erosion was more frequently found on the posterior teeth

(continued)

Table 3.2 (continued)

Study	Population	Erosion assessment	GERD assessment	Findings
Linnet et al. [139]	52 children with GERD history and 52 controls (aged 17 months–16 years old), recruited at a children's hospital in Brisbane, Australia	Modified Aine index	Medical records	Although there were no significant differences in erosion between the two groups (prevalence of erosion in GERD group = 14 % and in the controls = 10 %), there were significantly more permanent teeth affected by erosion in the GERD group (4 %) than in the controls (0.8 %)
Munoz et al. [121]	181 patients with reflux (mean age of 47.8 years old) and 72 controls (mean age of 44.43 years old) from a hospital in Spain	Eccles and Jenkins index modified by Hattab and Yassin	GERD symptoms for more than 6 months; esophagogastroduodenoscopy and 24 h esophageal pH monitoring	The presence of erosion was significantly higher in the GERD group (47.5 %) when compared to the control (12.5 %)
Nunn et al. [140]	Data were collected from the 1993 UK children's dental health survey (17,061 children) and the dental reports of two National Diet and Nutrition Surveys (NDNS) of children aged 1 1/2 to 4 1/2 years old in 1992/1993 (1,451 children) and 4–18 years in 1996/1997 (1,726 children)	Modified Smith and Knight index (TWI)	Questionnaire	More 4–6-year-old children with reported symptoms of GERD (79 %) had erosion compared with symptom-free children (62 %), $p < 0.05$
Jensdottir et al. [141]	23 young adults who had been diagnose with GERD in the past but, at the moment of the study, were symptom-free and 57 controls (young adults 19–22 years old) from Iceland	Modified Lussi index	24 h esophageal pH monitoring, manometry, and esophagogastroduodenoscopy	No difference was found in the prevalence of dental erosion between the two studied groups (35 % in the GERD group and 40 % in the control)

Milosevic et al. [142]	2,385 children (14 years old) from several districts in West England, United Kingdom	Simplified scoring criteria for tooth wear	Questionnaire	Tooth wear was significantly associated with the presence of stomach upset (odds ratio = 1.45)
Moazzez et al. [143]	104 patients with GERD and 31 controls (aged between 18 and 75) referred to a hospital in London, United Kingdom	Modified Smith and Knight index (TWI)	24 h esophageal pH monitoring and manometry	Patients with GERD symptoms showed higher frequency of lesions affecting the dentin than the controls. The presence of GERD was significantly correlated with palatal tooth wear
Moazzez et al. [122]	31 patients with GERD and 7 controls (mean age of 43.2 and 22.6 years old, respectively) referred to a hospital in London, United Kingdom	Modified Smith and Knight index (TWI)	24 h esophageal pH monitoring and manometry	Palatal tooth wear was observed only in the GERD group (29 % of the patients) and it was significantly correlated with acid reflux at night
Oginni et al. [123]	125 patients diagnosed with GERD and 100 controls (aged 18–72 years old) from Nigeria, Africa	Smith and Knight index (TWI)	Questionnaire	20 patients with GERD had erosion in the maxillary anterior teeth. The prevalence of erosion was significantly higher in GERD patients than in the controls (16 % versus 5 %, respectively)
Ersin et al. [124]	38 GERD patients and 42 controls (mean age of 6 years old)	Eccles and Jenkins index	24 h esophageal pH monitoring	The prevalence of dental erosion was higher in GERD patients (76 %) when compared to the controls (24 %). GERD was significantly associated with erosion
Di Fedi et al. [144]	200 patients with GERD and 100 controls (aged 19–78 years old) from a university hospital in Palermo, Italy	Smith and Knight index (TWI)	24 h esophageal pH monitoring and esophagogastroduodenoscopy	The prevalence of erosion in GERD patients was 9 % against 13 % from the controls. No significant association was found between GERD and tooth wear

(continued)

Table 3.2 (continued)

Study	Population	Erosion assessment	GERD assessment	Findings
Corrêa et al. [125]	50 patients with GERD and 50 controls (aged 17–75 years old) from a university hospital in Botucatu, Brazil	Eccles and Jenkins index	24 h manometry, endoscopy, and esophageal pH monitoring	GERD patients had increased number of erosive lesions (total of 273) than the controls (total of 5). The lesions were more frequently found on the palatal surfaces of the anterior teeth
Holbrook et al. [131]	351 individuals (aged 6–65 years old), of which 36 % reported symptoms of GERD and 21.3 % were confirmed to have GERD by 24 h esophageal monitoring, in Iceland	Modified version of Lussi index	Gastroscopy, esophageal manometry, and 24 h esophageal pH monitoring	Significant associations were found between erosion and diagnosed reflux disease (odds ratio = 2.77)
Wang et al. [145]	88 GERD patients and 36 controls (aged 20–73 years old) referred to hospitals in different regions of China	Modified version of Smith and Knight index (TWI)	Endoscopy, esophageal 24 h double-probe pH monitoring, and esophageal manometry	Erosion was recorded in 43 patients with GERD (48.86 %) and in only 5 of the controls (13.89 %). Subjects with GERD presented higher TWI scores. GERD patients showed erosion in the posterior and anterior areas, whereas the controls showed erosion only in the posterior area
Wang et al. [146]	1,499 children (aged 12–13 years old) from schools in Guangdong, China	Eccles and Jenkins index	Questionnaire	Sign of erosion in at least one tooth was observed in 416 children (27.3 %). No significant association was found between erosion and the presence of GERD patients

Murakami et al. [133]	967 children (aged 3–4 years old) recruited in a health center in Diadema, Brazil	O'Brien index	Questionnaire	The percentage of children with at least one tooth with erosive tooth wear was of 51.6 %. GERD was significantly associated with erosive tooth wear (odds ratio = 1.96)
Vargas-Ferreira et al. [147]	944 schoolchildren (11–14 years old) in Santa Maria, Brazil	O'Sullivan	Questionnaire	The prevalence of erosion was low (7.2 %). The most affected teeth were the maxillary incisors. 105 (11.1 %) children reported GERD symptoms, while 839 (88.9 %) did not. No significant association was found between GERD and tooth wear
Wild et al. [148]	59 children symptomatic with GERD, from whom 45 were positive for GERD according to the esophageal pH test, and 20 controls (aged 9–17 years old) recruited at the Pediatric Gastroenterology Clinic and General Pediatric Clinics at the University of California, San Francisco, United States	Simplified tooth wear index (STWI)	24 h esophageal pH monitoring	The symptomatic and asymptomatic children showed similar percentages regarding having at least one tooth affected by dental erosion (85 % and 70 %, respectively). However, the number of teeth affected by erosion was higher in the symptomatic group. In symptomatic patients, erosion rates were higher on occlusal/incisal surfaces than the controls but were similar on facial and palatal surfaces
Yoshikawa et al. [127]	40 GERD patients and 30 controls (aged 42–79 years old) from Shimane University Hospital, Japan	Modified Smith and Knight Index	Self-reported and endoscopy	The prevalence of dental erosion was 24.3 % (9 patients) in the GERD group, compared to 0 % in the control
Mulic et al. [23]	3,206 adolescents (aged 18 years old) recruited from a public health clinic in Oslo, Norway, of which 198 reported having GERD	Visual Erosion Dental Examination (VEDE) scoring system	Questionnaire	Of all adolescents, 38 % had at least one tooth with dental erosion. The presence of GERD was significantly associated with higher presence of erosive wear (odds ratio = 2)

(continued)

Table 3.2 (continued)

Study	Population	Erosion assessment	GERD assessment	Findings
Correa et al. [126]	30 patients with GERD and 30 controls (aged 17–60 years old) from a university hospital in Botucatu, Brazil	Eccles and Jenkins	Clinical, endoscopic, manometric, and pH-metric findings	The GERD patients presented a significantly greater ($p<0.001$) mean number of teeth with erosion (4.7) than the controls (0.06)
Tantbirojn et al. [149]	12 patients with GERD and 6 controls (aged 20–65 years old) from Minnesota, United States	Replica of impressions scanned by an optical scanner	GERD diagnosis made by a physician or patients that were taking over-the-counter medicine for reflux	GERD patients had greater surface loss over a six-month-period than the controls
Al-Zarea [150]	400 patients (aged 15–65 years old) from several clinics in Al-Jouf area, Saudi Arabia	Ordinal scale proposed by Carlsson et al.	Questionnaire	90 % of the patients showed the erosive type of tooth wear. The presence of GERD symptoms was significantly correlated with tooth wear
Gatou and Mamai-Homata [151]	243 children (aged 5–7 years old) attending primary schools in Piraeus, Greece	Smith and Knight index	Questionnaire	Only 1.6 % of the children were tooth-wear-free. 41.6 % of these children presented moderated tooth wear into the dentin and 4.1 % had severe tooth wear into the dentin. No association was found between tooth wear and exposure to gastric juice
Holbrook et al. [152]	Retrospective analysis of 2 studies: Arnadottir, 2010, and Holbrook, 2009	Scores transformed to BEWE		Intrinsic acid erosion clearly caused an increased BEWE score
Farahamand et al. [128]	54 GERD patients and 58 controls (aged 3–12 years old) from a children's hospital in Tehran, Iran	Aine et al. index	Questionnaire, endoscopy, 24 h esophageal pH monitoring	59 of the GERD children had erosion (98.1 %) against only 11 children (19 %) from the control group. A significant association between GERD and erosion was found

Bartlett et al. [24]	3187 young adults (aged 18–35 years old) from seven European countries	Basic erosive wear examination (BEWE)	Questionnaire	There was a strong association between tooth wear and having heartburn and reflux symptoms often (odds ratio = 3.21)
Alavi et al. [129]	31 GERD patients and 71 controls from a hospital in Shiraz, Iran	Not named	Questionnaire and endoscopy	22.6 % individuals with positive GERD in endoscopy had dental erosion. GERD was significantly associated with erosion
Hamasha et al. [25]	3812 children (aged 12–14 years old) from 81 schools in 3 different governorates of Jordan	Tooth wear index modified by Millward et al. (1994)	Questionnaire	1229 children showed signs of dental erosion (32.2 %). Students that reported regular bouts of heartburn, indigestion, and acidic taste in the mouth had significantly higher prevalence of dental erosion (74.1 %)

and erosion [154, 155]. A more recent systematic review performed in 2013 confirmed that there is in fact an association between GERD and dental erosion; however, the authors appreciated that the studies used in the review evaluated erosion or tooth wear by different indices, which may complicate comparisons [156]. A parallelism between GERD and erosion in patients with intellectual and developmental disabilities has also been reported in the literature [157, 158]. In a group of institutionalized intellectually disabled individuals identified with dental erosion, 65.5 % were diagnosed with gastroesophageal reflux disease [134]. Abanto et al. found a significant association between patients with cerebral palsy and GERD with erosive tooth wear [158]. Despite these results, it should be mentioned that there are reports in the literature which failed to identify a significant association between GERD and erosion [141, 144, 148].

It should be mentioned that the reflux episodes can also appear as a symptom-free condition [159], the so-called silent reflux. In these cases, the dentist may be the first professional to suspect the presence of GERD by detecting erosive lesions of uncertain etiology in their patients [109].

3.2.2.2 Distribution and Severity of Erosion in GERD Patients

Erosive wear in GERD patients appears to be found more frequently on palatal surfaces of the maxillary anterior teeth and on the molar teeth [121, 131, 156]. It was suggested that during reflux the gastric juice passes over the dorsum of the posterior third of the tongue, reaching the palatal surfaces of the upper molar teeth, and then passes over the buccal surfaces of the lower molars. The lingual surfaces of the lower molars remain protected by the ventral surface of the tongue. The acid also passes over the dorsum of the tongue, reaching the palatal surfaces of the maxillary anterior teeth [131]. Another important clinical feature described is the higher degree of severity of the erosive lesions found in some GERD patients. There was a significant association between molar erosion into the dentin and patients having at least one parameter indicating the presence of GERD (gastroscopy and/or esophageal manometry with 24 h monitoring of esophageal pH) being positive [131]. More than a third of the patients with GERD presented lesions with severe damage, whereas the controls without GERD only showed mild lesions [121]. Tantbirojn et al. observed that patients with GERD presented mean volume loss per tooth significantly higher than the controls during a 6-month follow-up period [149].

3.2.2.3 Saliva Parameters in GERD Patients

The amount and quality of saliva and its relationship with erosion development was also investigated in GERD patients. It was reported that 54 % of GERD patients had low buffer capacity [160]. On the contrary, another investigation did not observe significant differences in buffering capacity, salivary flow rate, or the concentrations of calcium, phosphorus, and fluoride between patients with GERD and healthy matching controls [161]. This latter finding was corroborated by Silva et al., who also did not find any difference in salivary pH, flow, and buffer capacity between subjects with GERD and the controls [162].

3.3 Clinical Signs and Symptoms of Erosion of Intrinsic Origin

Wear of the palatal surfaces of the upper incisors is a very common characteristic of intrinsic erosion, whether it is caused by frequent vomiting or by regurgitation. With lesion progression the lingual surfaces of the premolars and molars become affected, and in more advanced stages, the process extends to the occlusal surfaces of the molars and to the facial surfaces of all teeth [2, 4].

The clinical features of dental erosion related to its intrinsic causes are illustrated below by three clinical cases. Figure 3.1a–f shows the case of a 26-year-old female suffering from anorexia nervosa with self-induced vomiting habits and receiving medical assistance for her condition over a 7-year period. The patient sought dental treatment due to the unpleasant appearance of her maxillary central incisors. At the clinical examination, it was observed that the incisal edges of the maxillary central incisors had their height reduced through a combination of erosion and attrition (Fig. 3.1a). The patient was informed about the causes and consequences of intrinsic erosion, followed by instruction on oral health care including home-based fluoride therapy. At the following appointment, the maxillary central incisors were restored with resin composite and the patient was discharged (Fig. 3.1b). Since then, the patient has been controlled on a regular basis and has reported that the self-induced vomiting continues with alternating periods of infrequent and frequent vomiting episodes. Figure 3.1c–f was taken 2 years after the initial treatment. Since the causal factor of erosion was not eliminated, the erosive challenges have continued. As a consequence, deterioration of the composite resin, particularly on the palatal aspect of tooth 21, and exposure of dentin were observed (Fig. 3.1c). At the buccal aspect of the maxillary front teeth, a thin band of enamel at the gingival margin consistent with eroded enamel was also identified (Fig. 3.1d). Moreover, erosive lesions were readily observed in both maxillary and mandibular occlusal views, being more developed in the maxilla than in the mandible (Fig. 3.1e–f). The palatal aspect of the maxillary premolars and molars were already affected. On the occlusal surfaces, rounding of marginal ridges and cups was observed in addition to cupping characterized by localized exposure of the dentin (Fig. 3.1e). This case exemplifies the difficulty in successfully treating patients with eating disorders for erosion and the limited benefits of restorative procedures.

Figure 3.2a–e shows the case of a 28-year-old female suffering from bulimia. At the time of the consultation, the chief complaints of the patient were tooth hypersensitivity and shortened appearance of the maxillary front teeth. At the clinical examination, advanced erosive lesions were diagnosed in the whole dentition. On the buccal aspect of the maxillary and mandibular teeth, these lesions were characterized by a silk-like appearance of the enamel surface with shortened appearance of the maxillary front teeth (Fig. 3.2a–c). On the palatal aspect of the maxillary teeth, erosive lesions with complete loss of the enamel were readily identified by the presence of a thin band of enamel at the gingival margin and exposure of dentin on both anterior and posterior teeth. On non-restored occlusal surfaces of the premolars and molars, rounding of marginal ridges and cups was observed in addition to the

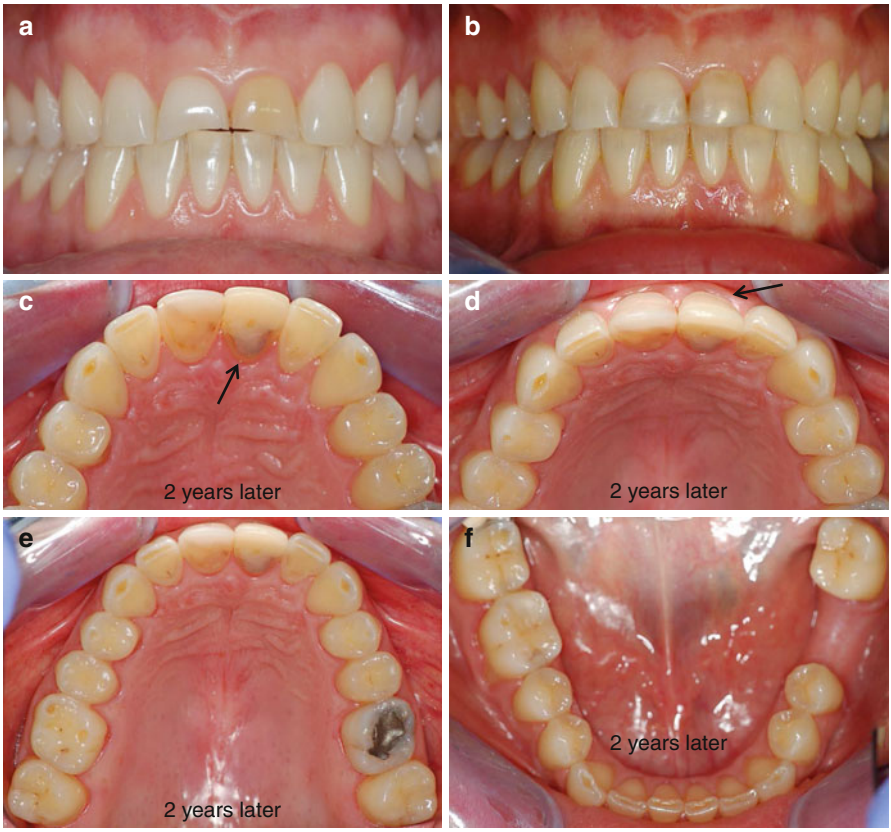


Fig. 3.1 Female, 26 years old, suffering from anorexia with self-induced vomiting habits. Patient sought dental treatment due the esthetic appearance of the front teeth (**a**). Maxillary central incisors were restored with resin composite (**b**). After 2 years deterioration of the composite resin was observed, simultaneously with exposure of dentin on tooth 21 (**c**). The buccal aspect of the maxillary front teeth (**d**) shows a thin band of enamel at the gingival margin (*arrow*). Erosive lesions are more developed in the maxilla (**e**) than in the mandible (**f**)

presence of cupping. Occlusal surfaces of the molar teeth with restored areas appeared at a higher level than the surrounding dental tissue. It is important to note that even in severe cases as the present one, the enamel at proximal surfaces was present, albeit eroded to some extent (Fig. 3.2d–e). The patient was informed about the intrinsic cause and consequences of the erosion process in her mouth, followed by instruction on oral health care.

Figure 3.3a–e presents the case of a 34-year-old male suffering from gastroesophageal reflux disease (GERD). The condition had been recently diagnosed by a gastroenterologist who had referred the patient for oral health examination. The main complaint of the patient was regurgitation. It is worth mentioning that this patient presented with a combination of erosive and caries lesions. Cervical caries

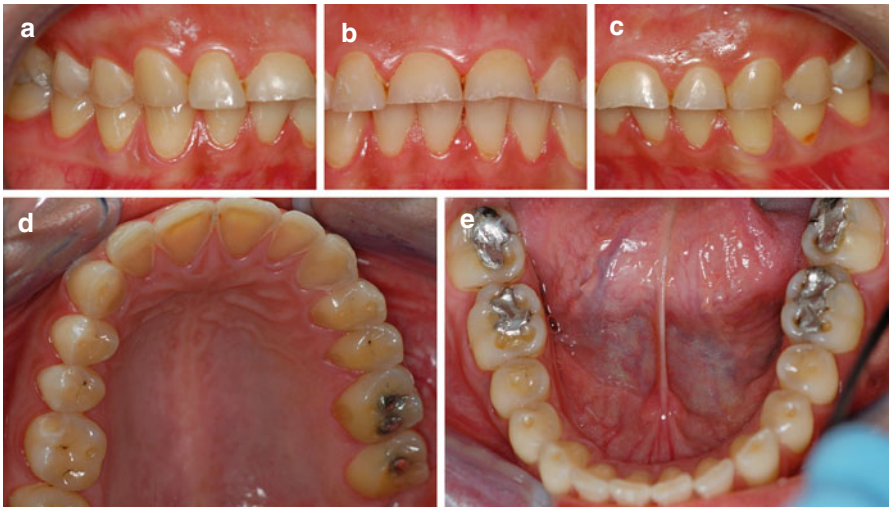


Fig. 3.2 Female, 28 years old, suffering from bulimia and tooth hypersensitivity. Note the presence of advanced erosive wear of the occlusal surfaces of the right posterior teeth (**a**), incisal edges of the anterior teeth (**b**), occlusal surfaces of left posterior teeth (**c**), and the lingual surfaces of the upper (**d**) and lower (**e**) teeth

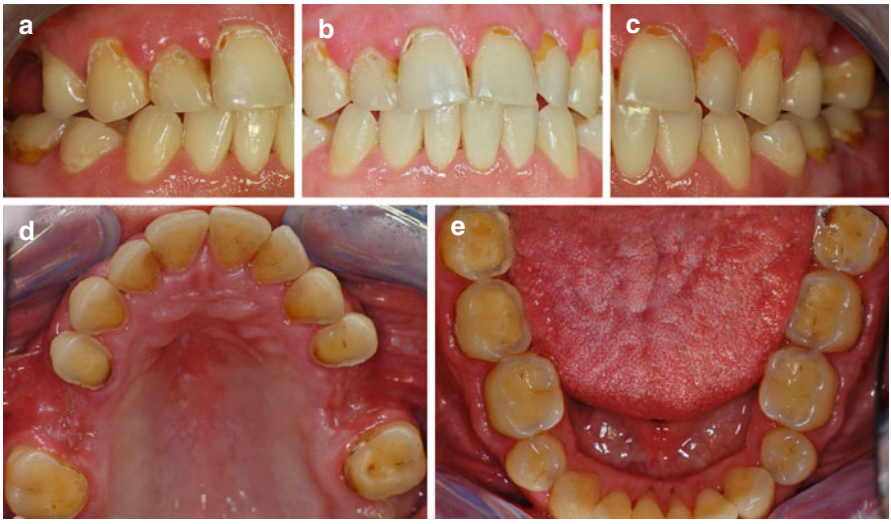


Fig. 3.3 Male, 34 years old, suffering from gastroesophageal reflux disease (GERD) presented with a combination of erosive and caries lesions. Note multiple cervical caries lesions on buccal surfaces of right posterior (**a**), facial surfaces of anterior (**b**), and buccal surfaces of left posterior teeth (**c**). Note also the multiple erosive lesions on palatal surfaces of the maxillary teeth (**d**), and on the occlusal surfaces of both the maxillary (**d**) and mandibular (**e**) teeth

lesions were observed on the buccal aspect of the maxillary anterior teeth, the buccal and palatal aspects of the posterior teeth, and several exposed root surfaces (Fig. 3.3a–e). On the palatal aspect of the maxillary teeth, severe erosive lesions, characterized by almost complete loss of enamel, and sites of exposure of dentin were observed. The same pattern was repeated on the occlusal surfaces of the posterior teeth, both on the maxilla and the mandible. The anatomical profile of the groove-fossa system had been almost completely lost. The dental treatment of a complex case as the present one requires expensive restorative and fixed prosthodontic procedures.

3.4 Prevention and Treatment Strategies for Erosion of Intrinsic Origin

Reduction in acid exposure is the most important preventive strategy against dental erosion, and some of the strategies to achieve this are discussed in Chaps. 10, 11, and 12. When intrinsic acids are the main cause of the pathology, permanent reduction in acid exposure involves referral to the appropriate medical specialist [6, 163]. Meanwhile, preventive measures to counteract the effect of repeated acid exposures can also be implemented. The strategies for prevention and treatment of dental erosion are discussed in detail in Chaps. 8, 9, 10, 11, 12, and 13.

3.5 Summary Statement

Dentists are well positioned to be the first health professionals to detect early signs and symptoms of conditions in their patients that give rise to dental erosion, help assure that the patient is aware of the underlying medical condition, and assist with appropriate medical follow-up. It should be kept in mind that some patients may be secretive about their damaging habits, a situation that is commonly seen in eating disorders and chronic alcoholism. In other cases, the condition can be symptom-free, as occurs in silent acid reflux disease. This highlights the importance of a good professional-patient communication and the need of a comprehensive medical history taking. The damage that the severe wear caused by intrinsic erosion can have on the dentition requires immediate dental assistance, including the implementation of preventive therapies and appropriate restorative treatment.

References

1. Bartlett DW, Coward PY. Comparison of the erosive potential of gastric juice and a carbonated drink in vitro. J Oral Rehabil. 2001;28:1045–7.
2. Bartlett D. Intrinsic causes of erosion. Monogr Oral Sci. 2006;20:119–39.
3. Braga SR, De Faria DL, De Oliveira E, Sobral MA. Morphological and mineral analysis of dental enamel after erosive challenge in gastric juice and orange juice. Microsc Res Tech. 2011;74:1083–7.

4. Moazzez R, Bartlett D. Intrinsic causes of erosion. Monogr Oral Sci. 2014;25:180–96.
5. Zero DT, Lussi A. Etiology of enamel erosion/ – intrinsic and extrinsic factors. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. Clinical advances in restorative dentistry. London: Martin Dunitz; 2000. p. 121–39.
6. Magalhaes AC, Wiegand A, Rios D, Honorio HM, Buzalaf MA. Insights into preventive measures for dental erosion. J Appl Oral Sci. 2009;17:75–86.
7. Burkhart N, Roberts M, Alexander M, Dodds A. Communicating effectively with patients suspected of having bulimia nervosa. J Am Dent Assoc. 2005;136:1130–7.
8. Scheutzel P. Etiology of dental erosion–intrinsic factors. Eur J Oral Sci. 1996;104:178–90.
9. Stefaniak AB, Virji MA, Harvey CJ, Sbarra DC, Day GA, Hoover MD. Influence of artificial gastric juice composition on bioaccessibility of cobalt- and tungsten-containing powders. Int J Hyg Environ Health. 2010;213:107–15.
10. Young A, Tenuta LM. Initial erosion models. Caries Res. 2011;45 Suppl 1:33–42.
11. Hove LH, Holme B, Young A, Tveit AB. The protective effect of TiF4, SnF2 and NaF against erosion-like lesions in situ. Caries Res. 2008;42:68–72.
12. Stenhagen KR, Hove LH, Holme B, Tveit AB. The effect of daily fluoride mouth rinsing on enamel erosive/abrasive wear in situ. Caries Res. 2013;47:2–8.
13. Passos VF, de Vasconcellos AA, Pequeno JH, Rodrigues LK, Santiago SL. Effect of commercial fluoride dentifrices against hydrochloric acid in an erosion-abrasion model. Clin Oral Investig. 2015;19(1):71–6.
14. Lindquist B, Lingstrom P, Fandriks L, Birkhed D. Influence of five neutralizing products on intra-oral ph after rinsing with simulated gastric acid. Eur J Oral Sci. 2011;119:301–4.
15. Barbour ME, Lussi A, Shellis RP. Screening and prediction of erosive potential. Caries Res. 2011;45 Suppl 1:24–32.
16. Schlueter N, Hardt M, Klimek J, Ganss C. Influence of the digestive enzymes trypsin and pepsin in vitro on the progression of erosion in dentine. Arch Oral Biol. 2010;55:294–9.
17. Schlueter N, Glatzki J, Klimek J, Ganss C. Erosive-abrasive tissue loss in dentine under simulated bulimic conditions. Arch Oral Biol. 2012;57:1176–82.
18. Murray KF, Christie DL. Vomiting. Pediatr Rev. 1998;19:337–41.
19. Hurst PS, Lacey LH, Crisp AH. Teeth, vomiting and diet: a study of the dental characteristics of seventeen anorexia nervosa patients. Postgrad Med J. 1977;53:298–305.
20. Jarvinen VK, Rytomaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991;70:942–7.
21. Rytomaa I, Jarvinen V, Kanerva R, Heinonen OP. Bulimia and tooth erosion. Acta Odontol Scand. 1998;56:36–40.
22. Johansson AK, Norring C, Unell L, Johansson A. Eating disorders and oral health: a matched case-control study. Eur J Oral Sci. 2012;120:61–8.
23. Mulic A, Skudutyte-Rysstad R, Tveit AB, Skaare AB. Risk indicators for dental erosive wear among 18-yr-old subjects in Oslo, Norway. Eur J Oral Sci. 2012;120:531–8.
24. Bartlett DW, Lussi A, West NX, Bouchard P, Sanz M, Bourgeois D. Prevalence of tooth wear on buccal and lingual surfaces and possible risk factors in young european adults. J Dent. 2013;41:1007–13.
25. Hamasha AA, Zawaideh FI, Al-Hadithy RT. Risk indicators associated with dental erosion among Jordanian school children aged 12–14 years of age. Int J Paediatr Dent. 2014;24:56–68.
26. Uhlen MM, Tveit AB, Refsholt Stenhagen K, Mulic A. Self-induced vomiting and dental erosion – a clinical study. BMC Oral Health. 2014;14:92.
27. Winkler LA, Christiansen E, Lichtenstein MB, Hansen NB, Bilenberg N, Stoving RK. Quality of life in eating disorders: a meta-analysis. Psychiatry Res. 2014;219:1–9.
28. Hermont AP, Oliveira PA, Martins CC, Paiva SM, Pordeus IA, Auad SM. Tooth erosion and eating disorders: a systematic review and meta-analysis. PLoS One. 2014;9:e111123.
29. Little JW. Eating disorders: dental implications. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;93:138–43.
30. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361:407–16.

31. Ritter AV. Eating disorders and oral health. J Esthet Restor Dent. 2006;18:114.
32. Hoek HW, van Hoeken D. Review of the prevalence and incidence of eating disorders. Int J Eat Disord. 2003;34:383–96.
33. Hellstrom I. Oral complications in anorexia nervosa. Scand J Dent Res. 1977;85:71–86.
34. Roberts MW, Li SH. Oral findings in anorexia nervosa and bulimia nervosa: a study of 47 cases. J Am Dent Assoc. 1987;115:407–10.
35. Milosevic A, Slade PD. The orodental status of anorexics and bulimics. Br Dent J. 1989;167: 66–70.
36. Jones RR, Cleaton-Jones P. Depth and area of dental erosions, and dental caries, in bulimic women. J Dent Res. 1989;68:1275–8.
37. Howat PM, Varner LM, Wampold RL. The effectiveness of a dental/dietitian team in the assessment of bulimic dental health. J Am Diet Assoc. 1990;90:1099–102.
38. Robb ND, Smith BG, Geidrys-Leeper E. The distribution of erosion in the dentitions of patients with eating disorders. Br Dent J. 1995;178:171–5.
39. Ohrn R, Enzell K, Angmar-Mansson B. Oral status of 81 subjects with eating disorders. Eur J Oral Sci. 1999;107:157–63.
40. Ohrn R, Angmar-Mansson B. Oral status of 35 subjects with eating disorders–a 1-year study. Eur J Oral Sci. 2000;108:275–80.
41. Montecchi PP, Custureri V, Polimeni A, Cordaro M, Costa L, Marinucci S, Montecchi F. Oral manifestations in a group of young patients with anorexia nervosa. Eat Weight Disord. 2003; 8:164–7.
42. Willumsen T, Graugaard PK. Dental fear, regularity of dental attendance and subjective evaluation of dental erosion in women with eating disorders. Eur J Oral Sci. 2005;113:297–302.
43. Lifante-Oliva C, Lopez-Jornet P, Camacho-Alonso F, Esteve-Salinas J. Study of oral changes in patients with eating disorders. Int J Dent Hyg. 2008;6:119–22.
44. Dynesen AW, Bardow A, Petersson B, Nielsen LR, Nauntofte B. Salivary changes and dental erosion in bulimia nervosa. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;106:696–707.
45. Shaughnessy BF, Feldman HA, Cleveland R, Sonis A, Brown JN, Gordon CM. Oral health and bone density in adolescents and young women with anorexia nervosa. J Clin Pediatr Dent. 2008;33:87–92.
46. Emodi-Perlman A, Yoffe T, Rosenberg N, Eli I, Alter Z, Winocur E. Prevalence of psychologic, dental, and temporomandibular signs and symptoms among chronic eating disorders patients: a comparative control study. J Orofac Pain. 2008;22:201–8.
47. Ximenes R, Couto G, Sougey E. Eating disorders in adolescents and their repercussions in oral health. Int J Eat Disord. 2010;43:59–64.
48. Hermont AP, Pordeus IA, Paiva SM, Abreu MH, Auad SM. Eating disorder risk behavior and dental implications among adolescents. Int J Eat Disord. 2013;46:677–83.
49. Conviser JH, Fisher SD, Mitchell KB. Oral care behavior after purging in a sample of women with bulimia nervosa. J Am Dent Assoc. 2014;145:352–4.
50. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. Am J Psychiatry. 2002;159:1284–93.
51. Sullivan PF. Mortality in anorexia nervosa. Am J Psychiatry. 1995;152:1073–4.
52. Robb ND, Smith BG. Anorexia and bulimia nervosa (the eating disorders): conditions of interest to the dental practitioner. J Dent. 1996;24:7–16.
53. Paszynska E, Linden RW, Slopien A, Rajewski A. Flow rates and inorganic composition of whole saliva in purging bulimic patients treated with a fluoxetine. World J Biol Psychiatry. 2011;12:282–7.
54. Young W, Khan F, Brandt R, Savage N, Razek AA, Huang Q. Syndromes with salivary dysfunction predispose to tooth wear: case reports of congenital dysfunction of major salivary glands, prader-willi, congenital rubella, and sjogren's syndromes. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92:38–48.
55. Scaramucci T, Borges AB, Lippert F, Frank NE, Hara AT. Sodium fluoride effect on erosion-abrasion under hyposalivatory simulating conditions. Arch Oral Biol. 2013;58: 1457–63.

56. Borges AB, Scaramucci T, Lippert F, Zero DT, Hara AT. Erosion protection by calcium lactate/sodium fluoride rinses under different salivary flows in vitro. Caries Res. 2014;48:193–9.
57. Bretz WA. Oral profiles of bulimic women: diagnosis and management. What is the evidence? J Evid Based Dent Pract. 2002;2:267–72.
58. Birgegard A, Clinton D, Norring C. Diagnostic issues of binge eating in eating disorders. Eur Eat Disord Rev. 2013;21:175–83.
59. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR. 4th ed. Washington, DC: American Psychiatric Association; 2000.
60. Mandel L, Abai S. Diagnosing bulimia nervosa with parotid gland swelling. J Am Dent Assoc. 2004;135:613–6; quiz 655.
61. Campbell K, Peebles R. Eating disorders in children and adolescents: state of the art review. Pediatrics. 2014;134:582–92.
62. Palla B, Litt IF. Medical complications of eating disorders in adolescents. Pediatrics. 1988;81:613–23.
63. Spigset O. Oral symptoms in bulimia nervosa. A survey of 34 cases. Acta Odontol Scand. 1991;49:335–9.
64. Rauch SD, Herzog DB. Parotidectomy for bulimia: a dissenting view. Am J Otolaryngol. 1987;8:376–80.
65. Mandel L, Surattanont F. Bilateral parotid swelling: a review. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2002;93:221–37.
66. Vavrina J, Muller W, Gebbers JO. Enlargement of salivary glands in bulimia. J Laryngol Otol. 1994;108:516–8.
67. Milosevic A, Dawson LJ. Salivary factors in vomiting bulimics with and without pathological tooth wear. Caries Res. 1996;30:361–6.
68. Schlueter N, Ganss C, Potschke S, Klimek J, Hannig C. Enzyme activities in the oral fluids of patients suffering from bulimia: a controlled clinical trial. Caries Res. 2012;46:130–9.
69. Blazer T, Latzer Y, Nagler RM. Salivary and gustatory alterations among bulimia nervosa patients. Eur J Clin Nutr. 2008;62:916–22.
70. Olden KW. Rumination. Curr Treat Options Gastroenterol. 2001;4:351–8.
71. Lee H, Rhee PL, Park EH, Kim JH, Son HJ, Kim JJ, Rhee JC. Clinical outcome of rumination syndrome in adults without psychiatric illness: a prospective study. J Gastroenterol Hepatol. 2007;22:1741–7.
72. Delaney CB, Eddy KT, Hartmann AS, Becker AE, Murray HB, Thomas JJ. Pica and rumination behavior among individuals seeking treatment for eating disorders or obesity. Int J Eat Disord. 2015;48:238–48.
73. Chial HJ, Camilleri M, Williams DE, Litzinger K, Perrault J. Rumination syndrome in children and adolescents: diagnosis, treatment, and prognosis. Pediatrics. 2003;111:158–62.
74. O'Brien MD, Bruce BK, Camilleri M. The rumination syndrome: clinical features rather than manometric diagnosis. Gastroenterology. 1995;108:1024–9.
75. Rajindrajith S, Devanarayana NM, Crispus Perera BJ. Rumination syndrome in children and adolescents: a school survey assessing prevalence and symptomatology. BMC Gastroenterol. 2012;12:163.
76. Valena V, Young WG. Dental erosion patterns from intrinsic acid regurgitation and vomiting. Aust Dent J. 2002;47:106–15.
77. Yagi T, Ueda H, Amitani H, Asakawa A, Miyawaki S, Inui A. The role of ghrelin, salivary secretions, and dental care in eating disorders. Nutrients. 2012;4:967–89.
78. Hejazi RA, McCallum RW. Cyclic vomiting syndrome: treatment options. Exp Brain Res. 2014;232:2549–52.
79. Abu-Arafeh I, Russell G. Cyclical vomiting syndrome in children: a population-based study. J Pediatr Gastroenterol Nutr. 1995;21:454–8.
80. Abell TL, Adams KA, Boles RG, Bousvaros A, Chong SK, Fleisher DR, Hasler WL, Hyman PE, Issenman RM, Li BU, Linder SL, Mayer EA, McCallum RW, Olden K, Parkman HP, Rudolph CD, Tache Y, Tarbell S, Vakil N. Cyclic vomiting syndrome in adults. Neurogastroenterol Motil. 2008;20:269–84.

81. Hejazi RA, Patil H, McCallum RW. Dumping syndrome: establishing criteria for diagnosis and identifying new etiologies. Dig Dis Sci. 2010;55:117–23.
82. Sanger GJ, Andrews PL. Treatment of nausea and vomiting: gaps in our knowledge. Auton Neurosci. 2006;129:3–16.
83. Hejazi RA, McCallum RW. Review article: cyclic vomiting syndrome in adults–rediscovering and redefining an old entity. Aliment Pharmacol Ther. 2011;34:263–73.
84. Milosevic A. Toothwear: aetiology and presentation. Dent Update. 1998;25:6–11.
85. Jitpakdee T, Mandee S. Strategies for preventing side effects of systemic opioid in postoperative pediatric patients. Paediatr Anaesth. 2014;24:561–8.
86. Santana TA, Trufelli DC, de Matos LL, Cruz FM, Del Giglio A. Meta-analysis of adjunctive non-nk1 receptor antagonist medications for the control of acute and delayed chemotherapy-induced nausea and vomiting. Support Care Cancer. 2014.
87. Lee M, Feldman M. Nausea and vomiting. In: Feldman M, Scharschmidt B, Sleisenger M, editors. Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology, diagnosis, management. 6th ed. Philadelphia: Saunders; 1998. p. 117–27.
88. Haberstick BC, Young SE, Zeiger JS, Lessem JM, Hewitt JK, Hopfer CJ. Prevalence and correlates of alcohol and cannabis use disorders in the United States: results from the national longitudinal study of adolescent health. Drug Alcohol Depend. 2014;136:158–61.
89. Friedlander AH, Mills MJ, Gorelick DA. Alcoholism and dental management. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1987;63:42–6.
90. Ablal MA, Kaur JS, Cooper L, Jarad FD, Milosevic A, Higham SM, Preston AJ. The erosive potential of some alcopops using bovine enamel: an in vitro study. J Dent. 2009;37:835–9.
91. Rees J, Hughes J, Innes C. An in vitro assessment of the erosive potential of some white wines. Eur J Prosthodont Restor Dent. 2002;10:37–42.
92. Smith BG, Robb ND. Dental erosion in patients with chronic alcoholism. J Dent. 1989;17:219–21.
93. O'Sullivan EA, Curzon ME. Dental erosion associated with the use of 'alcopop'–a case report. Br Dent J. 1998;184:594–6.
94. Laudenbach JB, Laudenbach JM. Dental erosion related to alcoholism and an eating disorder: a case report. Compend Contin Educ Dent. 2005;26:639–40, 642, 644–9; quiz 650, 669.
95. Robb ND, Smith BG. Prevalence of pathological tooth wear in patients with chronic alcoholism. Br Dent J. 1990;169:367–9.
96. Manarte P, Manso MC, Souza D, Frias-Bulhosa J, Gago S. Dental erosion in alcoholic patients under addiction rehabilitation therapy. Med Oral Patol Oral Cir Bucal. 2009;14:e376–83.
97. Kaltenbach T, Crockett S, Gerson LB. Are lifestyle measures effective in patients with gastroesophageal reflux disease? An evidence-based approach. Arch Intern Med. 2006;166:965–71.
98. Gadsby R, Barnie-Adshead AM, Jagger C. A prospective study of nausea and vomiting during pregnancy. Br J Gen Pract. 1993;43:245–8.
99. Tsang IS, Katz VL, Wells SD. Maternal and fetal outcomes in hyperemesis gravidarum. Int J Gynaecol Obstet. 1996;55:231–5.
100. Ismail SK, Kenny L. Review on hyperemesis gravidarum. Best Pract Res Clin Gastroenterol. 2007;21:755–69.
101. Evans RD, Briggs PF. Tooth-surface loss related to pregnancy-induced vomiting. Prim Dent Care. 1994;1:24–6.
102. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R, Global CG. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol. 2006;101:1900–20; quiz 1943.
103. Kahrilas PJ. Clinical practice. Gastroesophageal reflux disease. N Engl J Med. 2008;359:1700–7.
104. Dent J, El-Serag HB, Wallander MA, Johansson S. Epidemiology of gastro-oesophageal reflux disease: a systematic review. Gut. 2005;54:710–7.

105. Mittal RK, Balaban DH. The esophagogastric junction. N Engl J Med. 1997;336:924–32.
106. Lee YY, McColl KE. Pathophysiology of gastroesophageal reflux disease. Best Pract Res Clin Gastroenterol. 2013;27:339–51.
107. Pandolfino JE, Shi G, Trueworthy B, Kahrilas PJ. Esophagogastric junction opening during relaxation distinguishes nonhernia reflux patients, hernia patients, and normal subjects. Gastroenterology. 2003;125:1018–24.
108. Banks M. The modern investigation and management of gastro-oesophageal reflux disease (gord). Clin Med. 2009;9:600–4.
109. Ranjitkar S, Kaidonis JA, Smales RJ. Gastroesophageal reflux disease and tooth erosion. Int J Dent. 2012;2012:479850.
110. Weijenborg PW, Bredenoord AJ. How reflux causes symptoms: reflux perception in gastroesophageal reflux disease. Best Pract Res Clin Gastroenterol. 2013;27:353–64.
111. Kahrilas P. Gastroesophageal reflux disease and its complications. In: Feldman MSB, Sleisenger M, editors. Sleisenger and fordtran's gastrointestinal and liver disease: pathophysiology, diagnosis, management. Philadelphia: Saunders; 1998. p. 498–517.
112. Ranjitkar S, Smales RJ, Kaidonis JA. Oral manifestations of gastroesophageal reflux disease. J Gastroenterol Hepatol. 2012;27:21–7.
113. Ali DA, Brown RS, Rodriguez LO, Moody EL, Nasr MF. Dental erosion caused by silent gastroesophageal reflux disease. J Am Dent Assoc. 2002;133:734–7; quiz 768–9.
114. Van Roekel NB. Gastroesophageal reflux disease, tooth erosion, and prosthodontic rehabilitation: a clinical report. J Prosthodont. 2003;12:255–9.
115. Cengiz S, Cengiz MI, Sarac YS. Dental erosion caused by gastroesophageal reflux disease: a case report. Cases J. 2009;2:8018.
116. Reston EG, Closs LQ, Busato AL, Broliato GA, Tessarollo FR. Restoration of occlusal vertical dimension in dental erosion caused by gastroesophageal reflux: case report. Oper Dent. 2010;35:125–9.
117. Guo J, Reside G, Cooper LF. Full-mouth rehabilitation of a patient with gastroesophageal reflux disease: a clinical report. J Prosthodont. 2011;20 Suppl 2:S9–13.
118. Petruzzi M, Lucchese A, Campus G, Crincoli V, Lauritano D, Baldoni E. Oral stigmatic lesions of gastroesophageal reflux disease (gerd). Rev Med Chil. 2012;140:915–8.
119. Okida RC, Dos Santos DM, Vechiato Filho AJ, Andreotti AM, de Medeiros RA, Goiato MC. Prosthetic rehabilitation of a patient with gastroesophageal reflux disease: 4-year followup. Case Rep Dent. 2014;2014:270365.
120. Bartlett DW, Evans DF, Anggiansah A, Smith BG. A study of the association between gastro-oesophageal reflux and palatal dental erosion. Br Dent J. 1996;181:125–31.
121. Munoz JV, Herreros B, Sanchiz V, Amoros C, Hernandez V, Pascual I, Mora F, Minguez M, Bagan JV, Benages A. Dental and periodontal lesions in patients with gastro-oesophageal reflux disease. Dig Liver Dis. 2003;35:461–7.
122. Moazzez R, Anggiansah A, Bartlett DW. The association of acidic reflux above the upper oesophageal sphincter with palatal tooth wear. Caries Res. 2005;39:475–8.
123. Oginni AO, Agbakwuru EA, Ndububa DA. The prevalence of dental erosion in nigerian patients with gastro-oesophageal reflux disease. BMC Oral Health. 2005;5:1.
124. Ersin NK, Oncag O, Tumgor G, Aydogdu S, Hilmioglu S. Oral and dental manifestations of gastroesophageal reflux disease in children: a preliminary study. Pediatr Dent. 2006;28:279–84.
125. Correa MC, Lerco MM, Henry MA. Study in oral cavity alterations in patients with gastroesophageal reflux disease. Arq Gastroenterol. 2008;45:132–6.
126. Correa MC, Lerco MM, Cunha Mde L, Henry MA. Salivary parameters and teeth erosions in patients with gastroesophageal reflux disease. Arq Gastroenterol. 2012;49:214–8.
127. Yoshikawa H, Furuta K, Ueno M, Egawa M, Yoshino A, Kondo S, Nariai Y, Ishibashi H, Kinoshita Y, Sekine J. Oral symptoms including dental erosion in gastroesophageal reflux disease are associated with decreased salivary flow volume and swallowing function. J Gastroenterol. 2012;47:412–20.

128. Farahmand F, Sabbaghian M, Ghodousi S, Seddighoraee N, Abbasi M. Gastroesophageal reflux disease and tooth erosion: a cross-sectional observational study. Gut Liver. 2013;7:278–81.
129. Alavi G, Alavi A, Saberfiroozi M, Sarbazi A, Motamedi M, Hamedani S. Dental erosion in patients with gastroesophageal reflux disease (gerd) in a sample of patients referred to the Motahari Clinic, Shiraz, Iran. J Dent (Shiraz). 2014;15:33–8.
130. Jarvinen V, Meurman JH, Hyvarinen H, Rytomaa I, Murtomaa H. Dental erosion and upper gastrointestinal disorders. Oral Surg Oral Med Oral Pathol. 1988;65:298–303.
131. Holbrook WP, Furuholm J, Gudmundsson K, Theodors A, Meurman JH. Gastric reflux is a significant causative factor of tooth erosion. J Dent Res. 2009;88:422–6.
132. Dahshan A, Patel H, Delaney J, Wuerth A, Thomas R, Tolia V. Gastroesophageal reflux disease and dental erosion in children. J Pediatr. 2002;140:474–8.
133. Murakami C, Oliveira LB, Sheiham A, Nahas Pires Correa MS, Haddad AE, Bonecker M. Risk indicators for erosive tooth wear in Brazilian preschool children. Caries Res. 2011;45: 121–9.
134. Bohmer CJ, Klinkenberg-Knol EC, Niezen-de Boer MC, Meuwissen PR, Meuwissen SG. Dental erosions and gastro-oesophageal reflux disease in institutionalized intellectually disabled individuals. Oral Dis. 1997;3:272–5.
135. Milosevic A, Lennon MA, Fear SC. Risk factors associated with tooth wear in teenagers: a case control study. Community Dent Health. 1997;14:143–7.
136. O'Sullivan EA, Curzon ME, Roberts GJ, Milla PJ, Stringer MD. Gastroesophageal reflux in children and its relationship to erosion of primary and permanent teeth. Eur J Oral Sci. 1998;106:765–9.
137. Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J. 1998;184:125–9.
138. Gregory-Head BL, Curtis DA, Kim L, Cello J. Evaluation of dental erosion in patients with gastroesophageal reflux disease. J Prosthet Dent. 2000;83:675–80.
139. Linnett V, Seow WK, Connor F, Shepherd R. Oral health of children with gastro-esophageal reflux disease: a controlled study. Aust Dent J. 2002;47:156–62.
140. 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.
141. Jensdottir T, Arnadottir IB, Thorsdottir I, Bardow A, Gudmundsson K, Theodors A, Holbrook WP. Relationship between dental erosion, soft drink consumption, and gastroesophageal reflux among Icelanders. Clin Oral Investig. 2004;8:91–6.
142. Milosevic A, Bardsley PF, Taylor S. Epidemiological studies of tooth wear and dental erosion in 14-year old children in North West England. Part 2: the association of diet and habits. Br Dent J. 2004;197:479–83; discussion 473; quiz 505.
143. Moazzez R, Bartlett D, Anggiansah A. Dental erosion, gastro-oesophageal reflux disease and saliva: how are they related? J Dent. 2004;32:489–94.
144. Di Fede O, Di Liberto C, Occhipinti G, Vigneri S, Lo Russo L, Fedele S, Lo Muzio L, Campisi G. Oral manifestations in patients with gastro-oesophageal reflux disease: a single-center case-control study. J Oral Pathol Med. 2008;37:336–40.
145. Wang GR, Zhang H, Wang ZG, Jiang GS, Guo CH. Relationship between dental erosion and respiratory symptoms in patients with gastro-oesophageal reflux disease. J Dent. 2010;38:892–8.
146. Wang P, Lin HC, Chen JH, Liang HY. The prevalence of dental erosion and associated risk factors in 12-13-year-old school children in Southern China. BMC Public Health. 2010;10:478.
147. Vargas-Ferreira F, Praetzel JR, Ardenghi TM. Prevalence of tooth erosion and associated factors in 11-14-year-old Brazilian schoolchildren. J Public Health Dent. 2011;71:6–12.
148. Wild YK, Heyman MB, Vittinghoff E, Dalal DH, Wojcicki JM, Clark AL, Rechmann B, Rechmann P. Gastroesophageal reflux is not associated with dental erosion in children. Gastroenterology. 2011;141:1605–11.

149. Tantbirojn D, Pintado MR, Versluis A, Dunn C, Delong R. Quantitative analysis of tooth surface loss associated with gastroesophageal reflux disease: a longitudinal clinical study. J Am Dent Assoc. 2012;143:278–85.
150. Al-Zarea BK. Tooth surface loss and associated risk factors in northern saudi arabia. ISRN Dent. 2012;2012:161565.
151. Gatou T, Mamai-Homata E. Tooth wear in the deciduous dentition of 5-7-year-old children: risk factors. Clin Oral Investig. 2012;16:923–33.
152. Holbrook WP, Arnadottir IB, Hloethversson SO, Arnarsdottir E, Jonsson SH, Saemundsson SR. The basic erosive wear examination (bewe) applied retrospectively to two studies. Clin Oral Investig. 2014;18:1625–9.
153. Pace F, Pallotta S, Tonini M, Vakil N, Bianchi PG. Systematic review: gastro-oesophageal reflux disease and dental lesions. Aliment Pharmacol Ther. 2008;27:1179–86.
154. Milosevic A. Gastro-oesophageal reflux and dental erosion. Evid Based Dent. 2008;9:54.
155. Fan-Hsu J. Evidence linking gastroesophageal reflux disease and dental erosion is not strong. J Am Dent Assoc. 2009;140:1401–2.
156. Marsicano JA, de Moura-Grec PG, Bonato RC, Sales-Peres Mde C, Sales-Peres A, Sales-Peres SH. Gastroesophageal reflux, dental erosion, and halitosis in epidemiological surveys: a systematic review. Eur J Gastroenterol Hepatol. 2013;25:135–41.
157. Bohmer CJ, Klinkenberg-Knol EC, Niezen-de Boer MC, Meuwissen SG. Gastroesophageal reflux disease in intellectually disabled individuals: how often, how serious, how manageable? Am J Gastroenterol. 2000;95:1868–72.
158. Abanto J, Shitsuka C, Murakami C, Ciamponi AL, Raggio DP, Bonecker M. Associated factors to erosive tooth wear and its impact on quality of life in children with cerebral palsy. Spec Care Dentist. 2014;34(6):278–85.
159. Nozu T, Komiyama H. Clinical characteristics of asymptomatic esophagitis. J Gastroenterol. 2008;43:27–31.
160. Filipi K, Halackova Z, Filipi V. Oral health status, salivary factors and microbial analysis in patients with active gastro-oesophageal reflux disease. Int Dent J. 2011;61:231–7.
161. Saksena R, Bartlett DW, Smith BG. The role of saliva in regurgitation erosion. Eur J Prosthodont Restor Dent. 1999;7:121–4.
162. Silva MA, Damante JH, Stipp AC, Tolentino MM, Carlotto PR, Fleury RN. Gastroesophageal reflux disease: new oral findings. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;91:301–10.
163. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33(3):243–52.

Causes of Dental Erosion: Extrinsic Factors

4

Anderson T. Hara, Joana C. Carvalho, and Domenick T. Zero

Abstract

In this chapter, relevant aspects related to the extrinsic causes of dental erosion are addressed, reviewing the existing literature under the following main topics: diet, medications/oral care products, and environmental factors. In addition, lifestyle and behavioral aspects that modulate extrinsic erosion are presented. A wide range of evidence ranging from laboratory tests to clinical trials is available to support the association between extrinsic erosive agents and dental erosion. Relevant dietary factors include a wide range of acidic foods and beverages, although supplementation, especially with calcium compounds, can reduce or eliminate the erosive potential. Medications of acidic nature seem also to pose a risk, as well as some oral care products. Environmental factors have been reported and affect specific target populations, with stronger evidence related to battery and galvanizing workers.

4.1 Introduction

Dental erosion has long been reported [1]; however, it was not until recently that it gained considerable attention of patients, dental practitioners, researchers, and oral care industry. Although observed changes in life expectancy and tooth retention rates have contributed to this scenario, the exposure to extrinsic acids, especially from

A.T. Hara, DDS, MSD, PhD (✉) • D.T. Zero, DDS, MS
Department of Cariology, Operative Dentistry and Dental Public Health,
Oral Health Research Institute, Indiana University School of Dentistry, Indianapolis, IN, USA
e-mail: ahara@iu.edu; dzero@iu.edu

J.C. Carvalho, DDS, PhD
Faculty of Medicine and Dentistry, School of Dentistry, Catholic University of Louvain,
Louvain, Belgium
e-mail: Joana.Carvalho@uclouvain.be

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_4

dietary sources, is mainly responsible for the growing awareness of dental erosion. The literature on extrinsic erosive agents is extensive and encompasses information of varying degrees of relevance, ranging from laboratorial analysis of their erosive *potential* to clinical studies on their erosive *effect*. These two outcomes should be clearly differentiated in order to better understand and apply the existing data. The term erosive potential relates to the innate ability of an agent to cause dental erosion, which may or may not lead to the development of erosive lesions, whereas erosive effect denotes the ability of an acid to cause erosive lesions, considering its properties and characteristics and given particular clinical conditions or experimental setups. These definitions derive from the multitude of factors involved in the development of dental erosion. Although tooth exposure to erosive acids is essential to lead to dental erosion, there are many factors that can modulate their aggressiveness in attacking the dental surfaces. As an illustration, an acidic beverage (pH 3.6) has the potential to cause erosion, as it presents a pH that is below the critical-pH for demineralization of enamel (4.5–5.5); however, this potential may not translate into actual erosive effect leading to the development of erosive lesions, if protective factors such as salivary factors (clearance, buffering capacity, presence of acquired dental pellicle) are in place. Consequently, erosive potential is a term mostly confined to laboratorial studies, while erosive effect applies to more clinically relevant conditions. Ideally, determination of the erosive effect would be preferred; however, most of the available information refers only to the erosive potential of acids.

4.2 Diet

The impact of diet and dietary habits in dental erosion is fairly well documented. The erosive potential of acidic foods and beverages has been described, with most of the laboratory and clinical evidence focusing on the role of acidic beverages on dental erosion. Table 4.1 below shows the type of foods and beverages that have been associated with dental erosion.

4.2.1 Beverages

4.2.1.1 Beverages Consumption Trends

When discussing dental erosion and dietary factors, much of the focus has been given to acidic beverages, especially soft drinks. A survey on the food intake trends by US adolescents showed that the consumption of soft drinks had increased considerably (up to about 300 %) between 1965 and 1996 [3], exposing adolescents to potentially erosive conditions, and increasing their risk of developing dental erosion. This change in dietary pattern was driven in the USA by the increased popularity of cola drinks in the 1980s, fueled by massive marketing strategies imposed by large soft-drink companies. Supporting this trend, the drink serving sizes tripled from the 1950s to late 1990s (6.6–20 oz), when soft drinks consumption seemed to have reached its peak.

Table 4.1 Beverages and foods associated with erosion, and chemical factors affecting their erosive potential

Dietary factors[a]	Chemical factors
Citrus juices and other acidic fruit juices	pH
Acidic (un-)carbonated beverages	Buffering capacity
Acidic sport drinks	Type of acid (pKa values)
Citrus fruits and other acidic fruits and berries	Surface adhesion properties
Salad dressing	Calcium concentration
Vinegar conserves	Phosphate concentration
Wines	Chelating properties
Acidic fruit-flavored candies	
Acidic chewing gums	
Cider	
Acidic herbal teas	
Alcoholic mixed drinks	

[a]Updated from Zero [2]

From the late 1990s to nowadays, soft drinks (specially sugary ones) started to be associated with major US health problems, such as poor diet quality, weight gain, obesity, and, in adults, type 2 diabetes [4]. It has also affected dental health as they have been related to increased risk for dental caries [5] and dental erosion. Effort has been made by governments around the world to limit the availability of soft drinks in schools, which is one of the main channels that encourage greater consumption of soft drinks. More than 30 national and subnational governmental bodies have made efforts to restrict availability, and the soft-drinks industry has also taken some limited voluntary action [6]. Coincidently or not, the overall consumption of beverages has decreased from 54 gal (204 l)/person/year in 1998 to 44 gal (166 l), in 2013 (according to Beverage Digest; http://www.cbsnews.com/news/americans-rekindle-love-for-drinking-water/).

Interestingly, some reports have suggested that water has partially replaced soft drinks, as the consumption of bottled water has increased in the same period [7]. This general trend, however, may not apply to all types of beverages, as the consumption of diet soft drinks, for example, has slightly increased (3.4 and 5.1 %, for US males and females, respectively) from 2000 to 2010 [8]. Since the erosive potential of diet and regular soft drinks has shown to be comparable in laboratorial testing [9], there is no clear advantage in this change of dietary pattern, from the dental erosion perspective. Additionally, we have witnessed a substantial increase in the popularity and availability of other types of beverages, including flavored/enhanced water, sports drinks, energy drinks, and fruit juices, most of them with comparative or higher erosive potential. Therefore, despite the soft-drink consumption having reached a plateau or even declined in the USA, it is safe to assume that the population is still widely exposed to acidic beverages that are potentially erosive.

A general overview of the current beverage consumption worldwide shows great variation among countries (http://a.tiles.mapbox.com/v3/slate.soda.html; source: Euromonitor International, 2011). The increased consumption of acidic beverages

Table 4.2 Clinical evidence linking acidic beverages and dental erosion

Authors	Year	Country	Age	Sample
Significant association				
Lussi et al. [10]	1991	Switzerland	26–30; 46–50	391
Millward et al. [11]	1994	UK	12–14	101
Johansson et al. [12]	1997	Saudi Arabia	19–25	95
O'Sullivan and Curzon [13]	2000	UK	3–16	309
Moazzez et al. [14]	2000	UK	10–16	21
Al-Malik et al. [15]	2001	Saudi Arabia	2–5	987
Harding et al. [16]	2003	Ireland	5	202
Dugmore and Rock [17]	2004	UK	12–14	1149
Luo et al. [18]	2005	China	3–5	1949
El Karim et al. [19]	2007	Sudan	12–14	157
Mungia et al. [20]	2009	USA	12–17	307
Sanhouri et al. [21]	2010	Sudan	12–14	1138
Murakami et al. [22]	2011	Brazil	3–4	967
Okunseri et al. [23]	2011	USA	13–19	1314
Aidi et al. [24]	2011	Netherlands	10–12	572
Huew et al. [25]	2011	Lybia	12	791
Bartlett et al. [26]	2011	UK	18–30	1010
Mulic et al. [27]	2012	Norway	18	1456
Nayak et al. [28]	2012	India	5	1002
Chrysanthakopoulos [29]	2012	Greece	13–16	770
Hamasha et al. [30]	2014	Jordan	12–14	3812
No association				
Bartlett et al. [31]	1998	UK	11–14	210
Williams et al. [32]	1999	UK	14	525
Deery et al. [33]	2000	USA, UK	11–13	129 (USA); 125 (UK)
Milosevic et al. [34]	2004	UK	14	2385
Wiegand et al. [35]	2006	Germany	2–7	463
Gurgel et al. [36]	2011	Brazil	12–16	414
Manaf et al. [37]	2012	Malaysia	19–24	150
Aguiar et al. [38]	2014	Brazil	15–19	675

observed in the USA over the last decades is comparable to other developed countries and may serve as projection for developing countries recently experiencing changes in their dietary habits. Epidemiological studies have shown increase in dental erosion prevalence and some of them have related this fact with the consumption of acidic beverages (Table 4.2). In summary, while beverage consumption has plateaued in some developed countries, there is still an overall trend for increase in soft-drink consumption worldwide, which was projected to grow from just under 83 l/person per year, in 2007 (equivalent to a total of 552 billion liters), to a 95 l/person per year, in 2012 [39].

4.2.1.2 Beverages Involvement in Dental Erosion

Reflecting the high consumption of acidic beverages, the number of clinical studies investigating their relationship with dental erosion has increased in recent years. Although most studies have shown this association, some have failed as can be seen in Table 4.2. The multi-factorial nature of erosion makes it difficult to identify single etiological factors, such as acidic beverages. Other important factors may be the limited

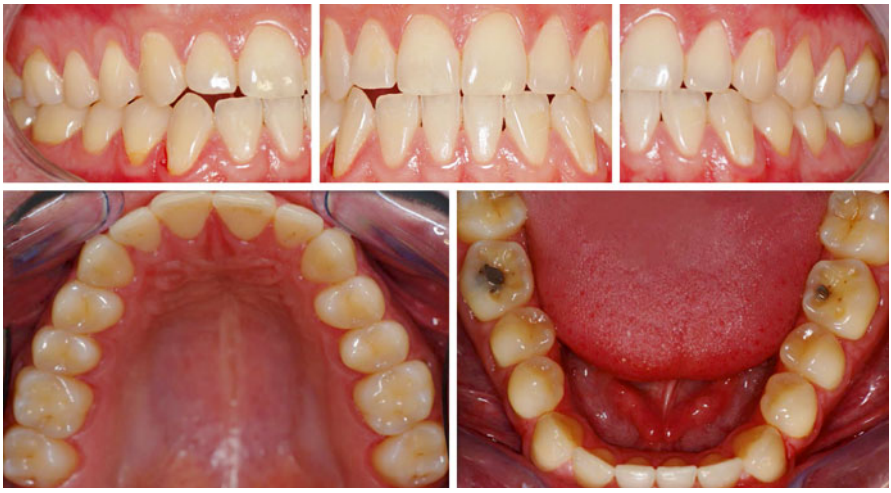


Fig. 4.1 Male 24 years old. Frequent consumption of soft drinks. Early signs of the erosion process may be identified in the whole mouth. Absence of the perikymata pattern and shiny feature of the enamel on facial aspect of maxillary and mandible teeth. Rounding of marginal ridges and cusps on occlusal surfaces of permanent molars. On occlusal surfaces of teeth 36 and 46 observe cupping and restored areas that appear at a higher level than the surrounding dental tissue

relevance of the dietary patterns at the time of collection compared to those when tooth erosion actually occurred [37]. This is confounded by the relatively slow progression of erosive lesions and the difficulty in diagnosing the different stages of lesion development clinically. Nonetheless, an epidemiological study showed that between 56 and 85 % of children at school in the USA 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 [40]. These results are disturbing, since it has been shown that any consumption of carbonated beverages increased the chances of dental erosion by 59 %, while drinking four or more glasses per day resulted in a 252 % increase [17].

A comprehensive evaluation of existing clinical studies has showed that soft drinks were associated with about 2.4-fold risk increase of dental erosion, despite the only moderate number of total studies available and the relatively small number of subjects [41]. Clinical studies in the area provide the most direct evidence that frequent exposure to acidic beverages can result in dental erosion. Based on the clinical data mentioned in Table 4.2, acidic fruits and juices, carbonated and uncarbonated beverages, sports drinks have been associated with causing erosion. Experimental clinical studies involving intraoral pH measurements after drinking or rinsing with acidic beverages (ciders, citric fruit juices, fruit juice drinks, flavored drinks, diet drinks) indicated that most acidic beverages only cause transient lowering of the pH of oral fluids [42]. Unusual or excessive consumption of specific dietary substances such as lemon juice, orange juice, carbonated cola beverage, orange cordial, and fruit-flavored drinks have been implicated based on case reports [42]. Figures 4.1, 4.2, and 4.3 illustrate cases of patients suffering from dental erosion at different degrees of severity. All cases are associated to frequent consumption of carbonated beverages.

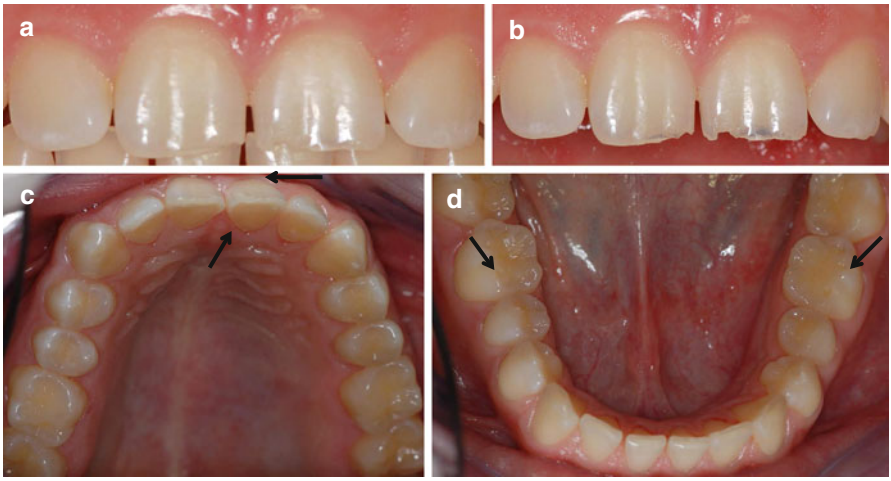


Fig. 4.2 Male 18 years old. Frequent consumption of soft drinks: (**a**) Established erosive lesions on teeth 11 and 21 are masked when the patient occludes. (**b**) Erosive bluish shadow lesions at the incisal edges of teeth 11 and 21. (**c**) Facial and lingual erosion with a thin enamel band at the gingival margin (*arrows*). (**d**) Slight cupping on teeth 36 and 46 (*arrows*)

Of limited clinical relevance, animal models [43–48] and in vitro studies [49, 50] have also been used to evaluate the erosive potential of different foods and beverages. These studies present the ability to simulate specific clinical conditions permitting comparison of large numbers of foods and beverages under well-controlled conditions. Yet, translation of the findings to the clinical reality is confounded by differences in experimental design and methodology among the studies and concerns over their clinical relevance [2]. Despite these limitations, acidic beverages that have been identified in clinical studies as etiologic factors in erosion have been confirmed in animal and in vitro studies.

4.2.1.3 Evaluation of Erosive Potential of Beverages

Although extensive data can be found on beverage consumption, it should be borne in mind that they can greatly differ on their erosive potential. Beverages can be classified into nine broad groups: water (bottled or tap), milk (including flavored), fruit juice (100 %), soda/soft drinks (regular and diet), fruit drinks, sports/energy drinks, coffee, tea, and alcoholic beverages [51]. According to Lussi et al. [39], soft drinks are mainly composed of filtered water, artificial additives, and refined sugar; sports drinks, which are designed to replenish fluids lost during activity, typically contain water, electrolytes, and sugar; and, energy drinks are basically soft drinks that contain some forms of vitamins, caffeine, and other chemicals that boost energy for a very short span. Of interest for dental erosion are those beverages that present lower pH and higher buffering capacity, which includes hundreds of commercially available options. Their erosive potential varies even within each group, depending on brands and even geographic location. For instance, the same beverage formulation

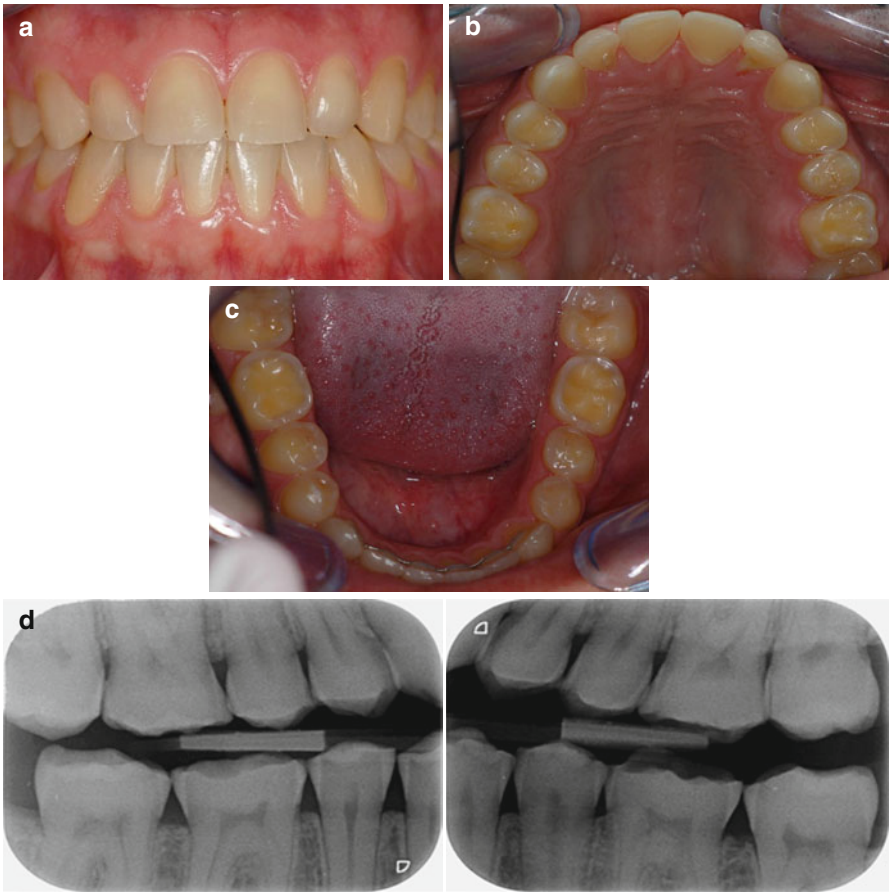


Fig. 4.3 Male 33 years old. Frequent consumption of soft drinks: (**a**) More advanced erosive lesions with dull appearance of front teeth. (**b**) Lingual erosive lesions on maxillary anterior teeth. The groove-fossa-system is no longer identified on occlusal surfaces of posterior teeth. (**c**) Localized exposure of the dentin on occlusal surfaces of permanent first molars. (**d**) Bitewing radiographs show flat eroded occlusal surfaces

was reported to present distinctive erosive potentials because of the different calcium and fluoride contents in the local water supplies used to produce them [9, 52]. Differences in taste or flavor can also affect the erosive potential of drinks and foods, with fruit or other acidic flavorings leading to lower acidity and higher erosive potential [39]. A study comparing different solutions of tartaric, malic, lactic, ascorbic, phosphoric, and citric acids with concentrations to equal their sensorial acidic taste showed that they present different erosive potentials [53]. Some physical characteristics of the beverages, such as increased adhesiveness and displacement, may increase their contact time with the tooth surface [54–56], increasing the erosive potential as well.

Due to the high number of beverages in existence, it is not feasible to conduct clinical test to obtain the erosive effect of all available beverage products, thus one should rely on their erosive potential for the recommendation of preventive measures for dental erosion. The erosive potential of beverages can be determined more easily and faster by laboratorial screening tests [57], which generally consider the pH, buffer capacity (titratable acidity), degree of saturation, calcium and phosphate concentration, and the presence of potential erosion inhibitors. pH has been described as one of the most determinant factor associated with dental erosion [57]; although it is crucial to understand that other properties and characteristics, as well as clinical application conditions including acid volume, frequency, and time of exposure [58, 59] can also affect the erosive potential. As example, calcium-enriched orange juice presents low pH (3.8), but has no erosive potential due to its high calcium content [60]. Titratable acidity, a property that shows how strong the acid is counteracting the protective effects of saliva [61], is also considered an important predictor [62], especially when the solution stays in contact with the tooth surface and is not rapidly cleared by saliva.

Larsen [63] suggested that erosion potential could be calculated based on the degree of saturation with respect to both hydroxyapatite and fluorapatite, by determining the pH, calcium, phosphate, and fluoride content of a beverage. Using a more elaborated approach, Lussi et al. [39, 64] developed an erosion prediction model including some of the beverage properties and characteristics. This model has shown good correlation with the ability of the beverages to soften the surface of enamel. For a more dynamic evaluation of the erosive potential of beverages, a laboratorial method called pH-stat has been used [65]. The pH-stat is based on the reaction between H+ and the test substrate, usually hydroxyapatite (HAp) crystals, and measures the dissolution rate of HAp, at a constant pH determined by the test beverage. Since the dissolution of HAp consumes H+, the rate/volume at which a titrant (acid) is added to maintain the pH of the test beverage is proportional to the rate of HAp dissolution [66], defining its erosive potential. Table 4.3 shows the different erosive potential of acidic beverages, based on the pH-stat test, compared to citric acid (most erosive) and drinking water (not erosive).

These screening tests are valuable providing data on the erosive potential of acidic beverages, in a relatively fast and inexpensive manner. Determination of the erosive effect will depend on many other biological and behavioral variables and can only be achieved in more clinically relevant experimental conditions. Nevertheless, the erosive potential is an important parameter that can be used for patient counseling.

4.2.1.4 Modification of Erosive Potential of Beverages

While patient's behavioral changes targeting the reduction of acidic beverages consumption are probably the most logical recommendation for those suffering from dental erosion, it should be borne in mind that patients may not necessarily comply with those measures. In this scenario, consumption of less erosive alternatives may be considered. Modifications on any of the relevant properties mentioned above (pH, buffer capacity, degree of saturation, calcium and phosphate concentration) may lessen the erosive potential of beverages. The addition of calcium and/or

Table 4.3 Chemical properties of beverages (mean and standard-deviation) determining their erosive potential

Beverage	pH	TA[a]	pH-stat[b]
Citric acid (1%)	2.3 (0.0)	3.8 (0.0)	4.3 (0.2)
Cola-type drink	2.5 (0.0)	0.6 (0.2)	3.5 (0.2)
Orange juice	3.8 (0.0)	2.5 (0.0)	1.2 (0.0)
Energy drink	3.3 (0.0)	2.9 (0.1)	2.0 (0.0)
Fruit drink - lemon	3.2 (0.1)	4.8 (1.3)	0.8 (0.2)
Fruit drink - orange	3.1 (0.0)	3.4 (0.1)	0.5 (0.3)
Fruit drink - grape	2.9 (0.0)	3.0 (0.2)	1.1 (0.5)
Sport drink	3.1 (0.0)	2.2 (0.0)	1.0 (0.2)
Tea - lemon	2.8 (0.0)	1.6 (0.0)	1.0 (0.6)
Drinking water	6.1 (0.4)	0.0 (0.0)	0.2 (0.1)

red: highest, *blue*: lowest
[a]Titratable Acidity. Volume of 0.1 N NaOH (ml), to reach pH7
[b]Volume of 0.1 N HCl (ml) to maintain the beverage pH, during HAp dissolution reaction

phosphate ions to beverages decreases the driving force for dental surface dissolution, as they become saturated with respect to the tooth minerals [67]. Commercially available acidic beverages have been supplemented with calcium in order to compensate for the deficient calcium intake, aiming to prevent some general health conditions, such as osteoporosis. As a positive side effect, these beverages present lower or non-erosive effect. Some studies have shown enamel demineralization reduction or inhibition for Ca-containing drinks when compared to those without Ca [58, 60, 68, 69]. Nowadays, several calcium-containing beverages are available in the market and they have shown reduced erosive potential [59]. Experimental combinations of Ca with other ions (Fe, P, and F) [70, 71] and proteins like ovalbumin [72] have also shown some reduction on erosive potential. Table 4.4 shows some examples of supplemented beverages and how their erosive potential is reduced, as measured by pH-stat.

Higher amounts of calcium seem to provide the best erosive potential reduction. However, it can also change the taste and the stability of the drink [67]. Another potential negative side effect is that excessively higher amounts may exceed the tolerable upper limit (level that may cause adverse health effects) of calcium (60 mmol/day), increasing the risk of kidney stones and of interference in the absorption of other minerals, including zinc, magnesium, and phosphorus [73]. Therefore, lower and effective concentrations are preferred. There is also some speculation on the influence of the type of calcium compound used. For instance, calcium lactate pentahydrate has lactate anions that can contribute to its anti-erosive effects, since it forms stable complexes with calcium [74] that appears to be just strong enough to protect the calcium ions from binding to other more stable complexing compounds present in the juice and thus allowing them to be available to interact with the tooth surface [65].

Table 4.4 Erosive potential of supplemented commercial beverages

Beverage	Supplement	pH	pH-stat[a]
Orange juice	---	3.8 (0.0)	1.2 (0.0)
Orange juice	Calcium	4.1 (0.0)	0.0 (0.0)
Orange juice	HMP[b]	3.8 (0.0)	0.2 (0.0)
Cola-type drink	---	2.5 (0.0)	3.5 (0.2)
Cola-type drink	Calcium	3.9 (0.0)	0.2 (0.0)
Smoothie	Calcium	4.2 (0.0)	0.0 (0.0)

Beverages in *red* show higher erosive potential
[a]Volume of 0.1 N HCl (ml) to maintain the beverage pH, during HAp dissolution reaction
[b]Sodium hexametaphosphate, experimentally added to orange juice

Casein phosphopeptide-stabilized amorphous calcium phosphate (CPP-ACP, Recaldent) has also been explored in laboratorial testing, as beverage additive in sport drinks [75] and citrus-flavored soft drinks (carbonated or not) [76]. The CPP-ACP-modified beverages presented reduced or no erosive potential. The likely mechanism has been described to be related to the increased availability of calcium and phosphate ions at the enamel surface, as well as the formation of CPP-ACP nanocomplexes on the enamel surface, reducing the possible sites for enamel dissolution [76]. Similarly, experimentation with nano-sized hydroxyapatite has been done. Although hydroxyapatite presents undesirable lower solubility than other calcium compounds, the use of nanoparticles seems to allow for higher reactivity and release of calcium and phosphorus [77, 78]. Reduction on the erosive potential of a sport drink has been observed, but pH changes, precipitation and possible changes in taste of the modified drink have also been reported [77], and need to be further investigated.

Contrary to calcium, supplementation with inorganic Pi has not shown to offer relevant reduction in the erosive potential of beverages. According to Lussi et al. [39], at the pH of erosive drinks (approximately 2–4), only a minute fraction of the total Pi is in the form of PO_4^{-3} ion, which is the only important Pi species in the ion activity product of HAP and FAP. As a result, large quantities of Pi would be required to raise the degree of saturation of the acidic solution, in order to see an anti-erosive effect. Foods and beverages are more likely not to present such high levels of Pi.

The literature is contradictory with regard to the erosive potential of acidic drinks and foods containing fluoride. Some laboratorial studies have shown that fluoride addition to beverages, in concentrations excluding toxicologically side effects, is incapable of preventing erosion, as demonstrated testing 18 soft drinks [79]. In drinks with pH above 3, F reduced the in vitro development of erosion by 28 %; while in drinks with pH below 3, erosion was not affected, despite total F concentrations of 20 parts per million and saturation with calcium fluoride [80]. However, some other studies have shown that the erosive capacity of different drinks was

significantly and negatively associated with their F concentration [39, 64, 81]. Methodological differences may have contributed to these conflicting results. It is important to point out, however, that fluorides have been important in erosion prevention, when used in different vehicles (toothpaste, mouthrinses, varnish), at different concentrations, as presented in Chaps. 8 and 9.

Supplementation with food polymers has also been investigated and they have shown ability to reduce erosion due to their possible adsorption to the dental surfaces, leading to the formation of an acid-protective layer. This layer could reduce the exchange of H+ and of calcium and phosphate ions between the hydroxyapatite and the solution [66]. Their protective properties, however, seem to be related to the type of polymer and experimental conditions. Polyphosphates of relatively longer chain length, such as sodium hexametaphosphate, seem to be more capable of reducing dental erosion than others, like tripolyphosphates and pyrophosphate. They also showed enhanced protection against erosion when used in association to calcium [65]. However, its clinical benefit is still unclear, as evidence for protection can be controversial [60, 82], thus requiring further investigation. Similarly, surface-protective effect has been reported to ovalbumin [72]. However, contrasting results were observed for xanthan gum, another food additive [65, 66]. Probably, the protective effect of the gum is minimal and its role as an additive of acid drinks might be more related to the improvement in the acceptability of the calcium-modified drinks than as an anti-erosive agent [65].

Slight modifications in taste, either by the additives themselves or by taste masking agents may make these alternatives less attractive to some individuals. However, in a brief taste evaluation of modified orange juices, only some tasters reported differences between the test juice (with calcium and polyphosphate) and the original, and most of them considered the taste to be acceptable [60]. Dietary supplements as effervescent tablet containing calcium and an acid/base regulating powder were able to reduce the erosive potential of orange juice, without noticeable changes in taste [69]. Although these observations are encouraging, it is still necessary to investigate other combinations of beverages and types and amounts of additives. Despite being a viable and promising alternative, beverage modification should not be seen as the only preventive measure for dental erosion since it is chemically impossible to modify all erosive solutions [83]. Instead, it should be considered as an additional preventive measure for the management of dental erosion.

4.2.2 Other Acidic Foods

In addition to beverages, other acidic foods have also been associated to dental erosion, with some clinical evidence for the consumption of vinegar and vinegar conserves, citric fruits, acidic berries, and other fruits (apple, pears, and plums) [10, 26, 84–86]. Acidic vegetables, a factor not always taken into account in clinical studies, have also shown to be associated to erosion, although their erosive effect may be confounded with the individual's overall preference for acidic foodstuff [87].

4.2.3 Acidic Candies

Acidic candies have been reported to be significant factors on erosion development. Although strong clinical evidence is not available, there are some reports and simulation studies supporting their erosive potential. They contain organic acids such as citric acid and malic acid to develop the characteristic sour flavor [88]. Despite the saliva-protective factors, sucking on sour candies can reduce the salivary pH levels below to the critical value for dental demineralization, therefore posing a risk for erosion of dental surfaces [58, 89]. This has been demonstrated in laboratory [45] and clinical [27, 30, 58, 90] settings. Placing acidified candies immediately next to the tooth surfaces can result in a concentrated solution of the dissolved candy with low pH, which has long been considered as a risk factor for both tooth surface softening and loss [91].

Solid or hard candies, such as lollipops and "jawbreakers" [88, 92], are normally difficult to bite, and therefore usually consumed by sucking or licking. As they dissolve slowly, they can be kept in the mouth for extended periods of time. This allows for prolonged and continuous exposure of the teeth to acids. Jawbreakers are mostly consumed by children with some even competing with each other to keep the candy in the mouth for longest period [88]. The flavor is influenced by the type of acid and concentration used; therefore, it is expected that different flavors may result in different erosive potentials. It has been shown that sour flavors present higher erosive potential compared to original flavors [93]. Similarly, the size of the candy is an important aspect, since it will determine the total length of exposure to the erosive challenge. Another type of acidic candy, the so-called candy spray, is sprayed directly into the mouth creating an immediate sour-fresh taste and tingling feeling on the tongue [89]. It has been reported to present high erosive potential due to low pH and high buffering capacity. Severe dental erosion has been reported to be associated with the extensive consumption of this candy [89].

Acidic candies may pose a high risk for erosion in particular populations. It primarily affects children and adolescents, populations vulnerable to uncontrolled and excessive high consumption behaviors. It has been reported that 70 % of parents were unaware that their children were consuming a type of acidic candy [89]. Acidic candies are also problematic for patients suffering from dry mouth, such as those receiving head and neck irradiation therapy, as they often present low salivary flow rates and buffer capacity [94]. These patients may experience natural dietary changes toward a higher intake of acidic saliva stimulating food, including acidic candies and lozenges [94]. Although some benefits are derived from the salivary flow stimulation, it has been observed that after consumption of acidic hard-boiled candy (with tartaric acid and rhubarb flavor), saliva became significantly more undersaturated with respect to tooth minerals and failed to return to clinically normal values, as observed in healthy individuals [94]. Counseling should therefore be provided for these high-risk populations, regarding the selection and consumption of acidic candies.

Similar to beverages, adding anti-erosion agents, such as calcium, in acidic hard candies has also been considered. Both candies with and without calcium were able

to stimulate saliva secretion, with the former releasing higher amounts of calcium into saliva. This translated into lower ability to demineralize tooth minerals, which indicates reduced erosive potential [95].

4.2.4 Chewing Gum

Chewing gums have been promoted by dental professionals as beneficial for the dentition, because of their ability to increase the flow rate and pH of saliva, potentially clearing and neutralizing acids and promoting enamel remineralization [96]. In addition, gums can also act as vehicles for a sustained delivery of some therapeutic agents for caries prevention [97]. Sugar-free chewing gums are, therefore, considered important for the management of dental caries. However, it has been speculated that frequent use of some of the acidic chewing gums may present potential for dental erosion development, especially on the occlusal surfaces of posterior teeth [98]. In the first minutes after chewing on strawberry-flavored acidic gums, the pH of saliva was observed to be reduced to 3.98, a level lower than the critical for tooth demineralization, while no pH drop was observed for the peppermint-flavored gum [98]. Although this is followed by a rise in pH to normal levels in the subsequent minutes, there is a potential risk that repeated and rapid replacement of gum with a fresh piece, commonly done once the flavor of the gum is lost, may keep the low pH values at tooth surfaces for longer, increasing the risk for dental erosion. For instance, replacement of gum every 4 min was able to cause significant erosive tooth wear on dentin [98]. More clinical evidence is needed to better understand the importance of acidic gums on erosion.

4.3 Medications

The frequent use of acidic medications that come in direct contact with teeth has been identified as an etiologic factor in dental erosion. Generally, acids function as buffering agents contributing to chemical stability, physiological compatibility, drug dispersion, and flavor improvement [99]. Acetylsalicylic acid (aspirin) [100], liquid hydrochloric acid [101], ascorbic acid (vitamin C) [102], iron tonics [103], cocaine [104], acidic oral hygiene products [105] or products with calcium chelators [106], and acidic saliva substitutes [101], salivary flow stimulants [106], and hospital mouth-cleaning aids [107] have been implicated in dental erosion based on case reports and/or laboratory studies. Some of the more relevant findings relating medications to erosion are described as follows.

4.3.1 Analgesic

Many soluble analgesic preparations contain citric acid. Studies have shown that they present erosive potential [108, 109] and case reports suggest that their

excessive use can lead to dental erosion [110, 111]. A laboratory test of six commercial brands of analgesics available in the UK showed that they present different erosive potentials. Although most of them were deemed not to be harmful to enamel, one brand was considered to be potentially erosive even in clinical conditions [109]. This analgesic in particular (Aspro™), presented the highest titratable acidity, the lowest calcium concentration, and no detectable phosphate. Further clinical testing was recommended to better understand and confirm the erosive potential observed. In another study, a buffered (500 mg acetylsalicylic acid, 300 mg calcium carbonate) acetylsalicylic acid chewable tablet showed no changes in the enamel surface structure after laboratory simulation exposure times of 1, 5 and 60 min. In contrast, enamel erosion was observed for an unbuffered acetylsalicylic acid chewable tablet even after 1 min of exposures, which was accentuated after 5 and 60 min [108]. Chewing increases the contact time between the tablet and tooth surfaces, considerably enhancing the risk for erosion. This was observed in a study comparing children with juvenile rheumatoid that were receiving large doses of aspirin in one of two forms, swallowing or chewing, at the time of the study. All children who were chewing the aspirin (25 out of 42) developed severe erosion on their maxillary and mandibular primary molars and first permanent molars. None of the children who swallowed aspirin tablets (17 out of 42) developed any eroded areas [100]. The authors concluded that aspirin was the reason for the erosive lesions. Considering the existing case reports and the reported erosive potential of some analgesics, dentists should advise patients who habitually use soluble/chewable analgesics for chronic conditions to select analgesic options with no or minimal erosive potential.

4.3.2 Vitamins

Reported vitamin consumption showed to be associated with dental erosion progression [87]; however, there was no further discrimination into chewable vitamins and vitamins that are swallowed. The authors suggested that the use of vitamins may represent a lifestyle that increases the chance of erosive wear to progress. There is some evidence associating chewable vitamin C (ascorbic acid) intake with erosion development [86, 112, 113]. This finding was also substantiated by a meta-analysis study investigating dietary factors and dental erosion [41].

4.3.3 Asthmatic Medications

A clinical study has reported higher risk for dental erosion associated with the use of asthmatic medication (cortisol inhaler) [30], although this was not observed in a previous study [17]. This possible link to erosion is primarily attributed to the fact that the majority of these medications present erosive potential due to their acidic nature. In addition, asthmatic medications can decrease the salivary buffering capacity and flow rate [114]. Indirect aspects also should be considered, as the frequent use

of such medications may be followed by the consumption of acidic drinks to compensate for the dry mouth feeling and bitter taste [30]. This is further complicated if medical conditions such as vomiting, heart burn, and gastric problems are involved.

Liquid oral medicines for the treatment of anemia, asthma, bronchitis, and cough are usually prescribed for children to aid compliance and have been shown to have an acidic nature [115, 116]. Nunn et al. [115] assessed the erosive potential (pH and titratable acidity) of eight liquid oral medicines and two effervescent preparations routinely prescribed for long-term use by pediatric patients. Some of the medicines and particularly the effervescent tablets showed erosive potential. Maguire et al. [99] tested the erosive potential of 97 liquid medicines, including sugar-containing and sugar-free formulations. More than half (57 %) of the drugs presented pH values lower than 5.5. Liquids/syrups and effervescent tablets presented the lowest pH values at 5.31 and 4.96, respectively. Special concern was given to effervescent tablets, as they presented substantial higher titratable acidity, indicating their higher potential to keep the low pH for longer. Higher erosive potential was also found in higher strength formulations, fact that can be explained by the higher amounts of acids (mostly citric acid) that are needed to mask and buffer the higher amount of the active ingredient [99]. Similar erosive potential was observed for most of 23 pediatric liquid medicines, consisting of anti-histamines, antitussives, bronchodilators, and mucolytics [117]. Further in vitro testing has verified the erosive potential of liquid medicines in dental enamel [118]. Scatena et al. [116] tested syrups of salbutamol sulfate (anti-asthmatic), ferrous sulfate (iron supplement and/or anti-anemic), and guaifenesin (expectorant). After simulated exposure for up to 28 days, all drugs decreased deciduous enamel surface hardness, indicating their potential to cause erosion. Salbutamol sulfate had the highest erosive potential, probably because of its lower pH and higher titratable acidity.

Health professionals need to be aware of the erosive potential of oral medicines and their consequences to the dental tissues. In addition, other factors such as prolonged and frequent ingestion, bedtime and between meals consumption, and the collateral effect of reduced salivary flow (e.g., tranquilizers, anti-histamines, anti-emetics, and anti-parkinsonian medicaments), should be taken into account as they increase the risk for medication-induced dental erosion.

4.4 Oral Care Products

4.4.1 Low pH Toothpastes and Mouthrinses

Some oral care products such as toothpastes and mouthrinses exhibit low pH, which may be a risk factor for dental erosion. Their acidic nature of these products is intended to enhance the chemical stability of some compounds (including fluoride), but may also enhance the incorporation of fluoride ions in tooth mineral forms, as well as the precipitation of calcium fluoride on the tooth surface [119]. Some studies have indicated calcium fluoride to be a protective factor against dental erosion [120]. The evidence linking oral care products and erosion is limited and not strong.

An EDTA-containing anti-calculus mouthrinse (Calcusan) has led to the development of dental erosion on enamel after 2 h of exposure in vitro [106]. The erosive potential of 11 commercially available mouthrinses was evaluated and reported to be variable, with some showing pH values as low as 3.4. The buffering capacity largely varied as well [105]. Three acidic mouthrinses: acidified sodium chlorite mouthrinse (pH 3.02), essential oil mouthrinse (Listerine®, pH 3.59), and a hexetidine mouthrinse (0.1 %, pH 3.75), were tested in more clinically relevant conditions [121]. All the mouthrinses were shown to cause progressive enamel surface loss over time, similar to that of an orange juice and higher than that of mineral water. The authors recommended that low pH mouthrinses should be used as short- to medium-term adjuncts to oral hygiene and never as prebrushing rinses, as this may exacerbate the abrasive effects of abrasive toothpastes [121]. The erosive potential of Listerine has also been shown in another in vitro study, as it was the only tested product with pH below 5.5. However, the deleterious effect was observed after 14 h of continuous exposure [122], which is an extreme condition of little or no clinical relevance. Frequent and abusive use of low pH oral care products might be considered as potential risk factors for erosion.

In an in vitro evaluation, Lussi and Jaeggi [123] investigated the erosive potential of low-pH toothpaste and rinses and observed that none of the fluoride-containing products caused enamel surface softening. In fact, after 10 and 20 min a significant increase on the enamel surface microhardness values, representing mineral gain, was observed for most of the test products. Only the positive control (orange juice) and a non-fluoridated toothpaste (Weleda Green Toothpaste, Weleda, pH 3.7) resulted in enamel erosion, in both evaluation periods. This finding was attributed to both the presence of citric acid/citrate and absence of fluoride in the toothpaste. The authors speculated that the higher erosion resistance observed for the other toothpastes resulted from the incorporation of fluoride into and/or deposition on the tooth, during treatment with the acidic products. This is substantiated by other studies [124, 125].

4.4.2 Cotton Swabs

Disposable cotton swabs or sponges are often used for cleaning and moistening mouth and teeth in bedridden patients or otherwise disabled persons. In an in vitro analysis they showed to present low pH and high acid content, and therefore high erosive potential. Consequently, their repeated use may lead to the development of erosive lesions. This is particularly relevant, considering that this target population often suffers from reduced salivary flow. Less erosive products should be used as special mouth-cleaning aids [107].

4.4.3 Saliva Substitutes

Saliva substitutes have been recommended for patients suffering from xerostomia. Some of these products such as Biotene® with a pH of 4.15 and Glandosane® with a

pH of 4.08 are considered potentially erosive and have shown to lead to mineral loss in enamel and in dentin. In the same study, other formulations with calcium phosphate and fluoride showed to be not only safe but also helpful in the remineralization of eroded enamel and should, therefore, be recommended [126].

4.4.4 Abrasive Toothpaste

The abrasive level of toothpaste is an important aspect to be considered, as it may lead to dental erosion progression. Highly abrasive toothpastes may facilitate the disruption of the acquired dental pellicle as well as abrade away dental surfaces previously softened by an erosive challenge. This is even more relevant in the presence of exposed root dentin surfaces. Dentin is more susceptible to abrasive and erosive insults and less responsive to remineralization [127, 128]. Although substantial variation is found among different brands, the abrasive level of toothpastes can be roughly estimated according to their types. Data from a standard abrasive test for toothpastes shows that whitening toothpastes would be on the higher abrasive end, while regular or most of the anti-sensitivity toothpastes would be on the medium-lower end. The latter should be recommended for high-risk for erosion patients, especially if exposed dentin surfaces are present.

4.4.5 Toothbrushing

The clinical evidence associating toothbrushing and erosion is conflicting. Prolonged and frequent toothbrushing has been shown to increase the probability of erosive wear [30, 129]. However, a clinical study showed that a significantly higher proportion of adolescents with erosive wear reported brushing less than once per day (68 %) and for less than half a minute (53 %). Also, brushing once per day or less significantly increased the risk of erosive wear compared with brushing twice per day or more [27]. The multi-factorial nature of erosion as well as the cross-sectional nature of the studies makes interpretation of the results very difficult. It is possible that some dietary and/or behavioral aspects may have been overlooked.

4.4.6 Dental Bleaching

Recently, given the increasing attention to esthetic dental procedures, dental bleaching has been investigated for possible detrimental side effects on enamel and dentin. It has been shown that some hydrogen peroxide-based gels may influence enamel surface morphology [130–132] and softening [130, 133] suggesting erosive potential. This is substantiated by the high content of hydrogen peroxide and a low pH value of some bleaching agents. However, these changes are thought to be mostly related to the specific experimental conditions used. Some of the studies did not include artificial or human saliva to simulate clinical conditions. Those simulating

the salivary effects showed no damage to the enamel and dentin surfaces [134–137]. The use of bleaching agents with lower content of hydrogen peroxide and neutral pH has not been reported to be harmful to the tooth structure, and did not increase the susceptibility of enamel to erosion [137] and erosion-abrasion [138] in vitro. When compared to orange juice, the effects of 6 % hydrogen peroxide were found to be insignificant [139].

More clinical evidence is needed to better understand the association between acidic oral care products, highly abrasive toothpastes and erosion, especially considering them in association with other erosive challenges.

4.5 Environmental (Occupational) Factors

The influence of occupation on the risk for dental erosion has been thoroughly reviewed by Zero [2] and Zero and Lussi [42], and they are summarized below.

4.5.1 Industrial Workers

Any industrial processing procedures that expose workers to acidic fumes or aerosols have the potential to cause dental erosion. The incisal edges of anterior teeth are primarily affected, although an increased rate of tooth wear of posterior teeth has also been reported. Sulfuric, nitric, acetic, and hydrochloric acids have all been implicated based on evidence from in vitro studies [140], case reports [141], cross sectional studies [142, 143], and case control studies [144–149]. Occupations involved with galvanizing, electroplating, metal and glass etching, printing, and mouth pipetting of laboratory acids as well as munitions, battery, fertilizer, and chemical manufacturing are all at risk of dental erosion unless appropriate safeguards are taken. Artisans and hobbyists working with inorganic acids are also potentially at risk of dental erosion, especially since these activities are unregulated.

Wiegand and Attin [150] thoroughly reviewed the literature of occupational erosion and concluded that there is sufficient evidence linking higher risk for dental erosion and battery and galvanizing workers. Evidence for other occupational groups described in the following sections, including wine tasting and swimmers, is not as strong and should be further confirmed by additional studies.

4.5.2 Professional Wine Tasters

Professional wine tasting has recently been identified as an occupational risk factor for dental erosion. The pH of wine was reported to range from 2.8 to 3.8 [151]. Ferguson et al. [152] reported that New Zealand wines ranged in pH from 3.3 to 3.8 with tartaric and malic acids accounting for 95 % of the total acids present. Lactic acid may also be present depending on the degree of malto-lactic fermentation. Case reports illustrate erosion as an occupational risk for wine tasters. A 52-year-old

professional wine taster tasting an average of 30 wines a day over 23 years showed extensive palatal erosion [153]. Similar dental findings were observed in another case of a 38-year-old individual working in wine marketing for 10 years, which involved the daily tasting of 20 wines daily on average. Significant dental erosion was observed, in association with a history of sensitivity to heat, cold, as well as wine [154]. A study of 19 Swedish wine tasters who tested usually 20–50 different wines 5 days a week found that 74 % had dental erosion [155]. These authors reported an association between the severity of the damage to the teeth and the number of years as a wine taster. Another clinical study compared the prevalence and severity of dental erosion between 21 wine tasters (exposed) and their spouses (non-exposed), showing significantly more erosion in the exposed population [156].

4.5.3 Competitive Swimmers

Several case reports have associated competitive swimmers using improperly pH-regulated swimming pools with dental erosion [157, 158]. Gas chlorinated swimming pools require daily pH monitoring and adjustment to maintain pool water in the recommended pH range of 7.2 to 8.0. This can also be a problem for recreational swimmers that swim on a regular basis.

4.6 Behavioral/Lifestyle Factors

Despite the clear evidence that most acidic beverages, foods, and medicaments present erosive potential, it is not always clear that it translates clinically into erosive effect, or lead to the development of dental erosion lesions. As previously mentioned, there are many factors that act modulating the development of erosion, including behavioral factors. The multi-factorial nature of dental erosion explains how an otherwise healthy person, who has limited exposure to acids (for example, ingesting a glass of orange juice during meals throughout his/her life) can hardly develop any clinical signs of dental erosion. Specifically, existing protective factors (dental pellicle, saliva buffering and clearing, remineralization) may counteract the erosive attacks (see Chap. 2). However, this healthy balance can be disturbed by some particular behavioral and lifestyle aspects, increasing the ability of the extrinsic acids to attack the dental surfaces causing dental erosion.

Therefore, in addition to the erosive potential of extrinsic erosive agents, a variety of factors, including frequency of acid intake, individual dietary habits (sipping, gulping, frothing, or use of a straw) [159], the flow rate, composition and clearing capability of the saliva, and the presence of the acquired dental pellicle, may influence the progress of tooth erosion [160]. Interestingly, the most evident cases of extrinsic erosion are always associated to behavioral/lifestyle factors.

Case reports have linked abusive or unusual behaviors to erosion. Frequent and excessive consumption of specific dietary items such as citrus fruits, lemon juice, orange juice, fruit squashes, cola-flavored soft drinks, and citrus-flavored drinks

have all been implicated [85, 161, 162]. Although the association between dental erosion and acidic diet have not been clearly found in some studies (Table 4.2), there is strong clinical evidence linking acidic drinks and dental erosion when the consumption is reported to be high among children [13], adolescents [13, 16, 34, 85], and adults [163]. Supporting these findings, no increased risk for dental erosion was found in a population of children and adolescents with generally low daily consumption of fruits [27]. Case-control studies have also shown a considerable higher risk of erosion when citrus foods were eaten more than twice per day [161, 164]. It is been suggested that although the underlying acidic nature of a food or drink is important, the most relevant aspect in the development of erosion is the frequency of consumption [26].

Holding an acid beverage in the mouth before swallowing increases the contact time of an acidic substance with the teeth and thus increases the risk of erosion [13]. This habit reportedly led to the most pronounced drop in the intraoral pH than any other drinking method [165]. Bedtime consumption of acid beverages is also considered as a risk factor especially for children. Ingestion of acidic beverages (lemon and carbonated drinks) at nighttime increases the risk for dental erosion development, especially if done after toothbrushing as this procedure removes the acquired enamel pellicle, a natural organic layer that forms on the dental surface protecting it from erosive acids [30]. Other risk indicators reported to be relevant to the development of dental erosion are the consumption of lemon and sour candies at bed time [17, 32, 67]. Association between the excessive exposure to erosive acids and tooth grinding may further increase the risk for erosion [87].

The use of illegal designer drugs by teenagers at “raves” has been associated with increased risk of erosion due to excess consumption of acidic beverages in combination with the hyposalivatory side effects of the drugs [2].

Healthier lifestyle that includes a diet high in acidic fruits and vegetables may subject teeth to an increased risk of erosion. Frequent dieting with high consumption of citrus fruits and fruit juices as part of a weight-reducing plan may also be a risk factor. Linkosalo and Markkanen [84] compared dental erosion in lactovegetarians to gender- and age-matched controls. Seventy-five percent of the lactovegetarians showed signs of dental erosion and they were mostly associated to the consumption of vinegar and vinegar conserves, citrus fruits, and acidic berries. Strenuous sporting activities and exercise may lead to higher risk of erosion if frequent intake of acidic sport drinks, fruit juices, and other acidic beverages are used for fluid and energy replacement. This problem may be compounded by decreased salivary flow secondary to increased fluid loss associated with strenuous exercise [27, 166], and to possible episodes of gastro-esophageal reflux [167].

Health conscious individuals also tend to have better than average oral hygiene [42]. While good oral hygiene is of proven value in the prevention of periodontal disease and dental caries, frequent tooth brushing with abrasive oral hygiene products may render teeth more susceptible to dental erosion [129]. This is especially true, if brushing is performed right after the erosive attack by acidic beverages [127, 168], not allowing the eroded surfaces to remineralize and regain its physical strength, and using highly abrasive dentifrices [169]. Clinically, erosion is not

generally found under areas covered by plaque, which is most likely due to the ability of plaque to buffer acids and to act as a physical barrier preventing the contact between acids and dental surfaces. There is some evidence that the frequent removal of the pellicle by tooth brushing with dentifrice may render the enamel surface more susceptible to acid erosion.

4.7 Summary and Conclusion Statements

Despite the significant body of literature on the erosive potential of extrinsic factors, it is noted that some of them have not clearly shown an association to erosion, which highlights the need for further investigation with well-controlled clinical trials. The lack of association may result from the multi-factorial nature of this condition that makes difficult the identification of a single causative factor. This complex nature is further complicated by lifestyle and behavioral aspects, which can modify the risk for dental erosion considerably. The adequate knowledge of the extrinsic etiological factors presented allows dental care professionals to apply efficient non-operative and operative measures to manage the erosion process, either by reducing the individual risk of developing new clinically identifiable erosive lesions and/or progression of established lesions.

References

1. Miller WD. Experiments and observations of the wasting of tooth tissue variously designated as erosion, abrasion, chemical abrasion, denudation, etc. Dental Cosmos. 1907;49:1–23.
2. Zero DT. Etiology of dental erosion–extrinsic factors. Eur J Oral Sci. 1996;104:162–77.
3. Calvadini C, Siege-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. Arch Dis Child. 2000;83:18–24.
4. Ogden CL, Kit BK, Carroll MD, Park S. Consumption of sugar drinks in the United States, 2005-2008. NCHS Data Brief. 2011;(71):1–8.
5. Heller KE, Burt BA, Eklund SA. Sugared soda consumption and dental caries in the United States. J Dent Res. 2001;80:1949–53.
6. Hawkes C. The worldwide battle against soft drinks in schools. Am J Prev Med. 2010;38:457–61.
7. Popkin BM. Patterns of beverage use across the lifecycle. Physiol Behav. 2010;100:4–9.
8. Fakhouri TH, Kit BK, Ogden CL. Consumption of diet drinks in the United States, 2009-2010. NCHS Data Brief. 2012;109:1–8.
9. Cochrane NJ, Cai F, Yuan Y, Reynolds EC. Erosive potential of beverages sold in Australian schools. Aust Dent J. 2009;54:238–44.
10. Lussi A, Schaffner M, Hotz P, Suter P. Dental erosion in a population of Swiss adults. Community Dent Oral Epidemiol. 1991;19:286–90.
11. Millward A, Shaw L, Smith AJ, Rippin JW, Harrington E. The distribution and severity of tooth wear and the relationship between erosion and dietary constituents in a group of children. Int J Paediatr Dent. 1994;4:151–7.
12. Johansson AK, Johansson A, Birkhed D, Omar R, Baghdadi S, Khan N, Carlsson GE. Dental erosion associated with soft-drink consumption in young Saudi men. Acta Odontol Scand. 1997;55:390–7.
13. O'Sullivan EA, Curzon ME. A comparison of acidic dietary factors in children with and without dental erosion. ASDC J Dent Child. 2000;67(160):186–92.

14. Moazzez R, Smith BG, Bartlett DW. Oral pH and drinking habit during ingestion of a carbonated drink in a group of adolescents with dental erosion. J Dent. 2000;28:395–7.
15. Al-Malik MI, Holt RD, Bedi R. Clinical and photographic assessment of erosion in 2-5-year-old children in Saudi Arabia. Community Dent Health. 2001;18:232–5.
16. Harding MA, Whelton H, O'Mullane DM, Cronin M. Dental erosion in 5-year-old Irish school children and associated factors: a pilot study. Community Dent Health. 2003;20:165–70.
17. Dugmore CR, Rock WP. The prevalence of tooth erosion in 12-year-old children. Br Dent J. 2004;196:279–82.
18. Luo Y, Zeng XJ, Du MQ, Bedi R. The prevalence of dental erosion in preschool children in China. J Dent. 2005;33(2):115–21.
19. El Karim IA, Sanhouri NM, Hashim NT, Ziada HM. Dental erosion among 12-14 year old school children in Khartoum: a pilot study. Community Dent Health. 2007;24:176–80.
20. Mungia R, Zarzabal LA, Dang SC, Baez M, Stookey GK, Brown JP. Epidemiologic survey of erosive tooth wear in San Antonio, Texas. Tex Dent J. 2009;126:1097–109.
21. Sanhouri NM, Ziada HM, Ahmed GI, Kamis AH. Tooth surface loss, prevalence and associated risk factors among 12-14 years school children in Khartoum State, Sudan. Community Dent Health. 2010;27:206–12.
22. Murakami C, Oliveira LB, Sheiham A, Nahás Pires Corrêa MS, Haddad AE, Bönecker M. Risk indicators for erosive tooth wear in Brazilian preschool children. Caries Res. 2011;45:121–9.
23. Okunseri C, Okunseri E, Gonzalez C, Visotcky A, Szabo A. Erosive tooth wear and consumption of beverages among children in the United States. Caries Res. 2011;45:130–5.
24. Aidi HE, Bronkhorst EM, Huysmans MC, Truin GJ. Factors associated with the incidence of erosive wear in upper incisors and lower first molars: a multifactorial approach. J Dent. 2011;39:558–63.
25. Huew R, Waterhouse PJ, Moynihan PJ, Kometa S, Maguire A. Dental erosion and its association with diet in Libyan schoolchildren. Eur Arch Paediatr Dent. 2011;12:234–40.
26. Bartlett DW, Fares J, Shirodaria S, Chiu K, Ahmad N, Sherriff M. The association of tooth wear, diet and dietary habits in adults aged 18-30 years old. J Dent. 2011;39:811–6.
27. Mulic A, Skudutyte-Rysstad R, Tveit AB, Skaare AB. Risk indicators for dental erosive wear among 18-yr-old subjects in Oslo, Norway. Eur J Oral Sci. 2012;120:531–8.
28. Nayak SS, Ashokkumar BR, Ankola AV, Hebbal MI. Association of erosion with dietary factors among 5-year-old children in India. J Dent Child (Chic). 2012;79:122–9.
29. Chrysanthakopoulos NA. Prevalence of tooth erosion and associated factors in 13-16-year old adolescents in Greece. J Clin Exp Dent. 2012;4:e160–6.
30. Hamasha AA, Zawaideh FI, Al-Hadithy RT. Risk indicators associated with dental erosion among Jordanian school children aged 12-14 years of age. Int J Paediatr Dent. 2014;24:56–68.
31. Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J. 1998;184:125–9.
32. Williams D, Croucher R, Marcenes W, O'Farrell M. The prevalence of dental erosion in the maxillary incisors of 14-year-old schoolchildren living in Tower Hamlets and Hackney, London, UK. Int Dent J. 1999;49:211–6.
33. Deery C, Wagner ML, Longbottom C, Simon R, Nugent ZJ. The prevalence of dental erosion in a United States and a United Kingdom sample of adolescents. Pediatr Dent. 2000;22:505–10.
34. Milosevic A, Young PJ, Lennon MA. The prevalence of tooth wear in 14-year-old school children in Liverpool. Community Dent Health. 1994;11:83–6.
35. Wiegand A, Müller J, Werner C, Attin T. Prevalence of erosive tooth wear and associated risk factors in 2-7-year-old German kindergarten children. Oral Dis. 2006;12:117–24.
36. Gurgel CV, Rios D, de Oliveira TM, Tessarolli V, Carvalho FP, Machado MA. Risk factors for dental erosion in a group of 12- and 16-year-old Brazilian schoolchildren. Int J Paediatr Dent. 2011;21:50–7.

37. Manaf ZA, Lee MT, Ali NH, Samynathan S, Jie YP, Ismail NH, Bibiana Hui Ying Y, Wei Seng Y, Yahya NA. Relationship between food habits and tooth erosion occurrence in Malaysian University students. Malays J Med Sci. 2012;19:56–66.
38. Aguiar YP, dos Santos FG, Moura EF, da Costa FC, Auad SM, de Paiva SM, Cavalcanti AL. Association between dental erosion and diet in Brazilian adolescents aged from 15 to 19: a population-based study. Scientific World Journal. 2014;13:818167.
39. Lussi A, Megert B, Shellis RP, Wang X. Analysis of the erosive effect of different dietary substances and medications. Br J Nutr. 2012;107:252–62.
40. Gleason P, Suitor C. Children's diets in the mid-1990s: dietary intake and its relationship with school meal participation. Alexandria: US Department of Agriculture, Food and Nutrition Service, Office of Analysis, Nutrition and Evaluation; 2001.
41. Li H, Zou Y, Ding G. Dietary factors associated with dental erosion: a meta-analysis. PLoS One. 2012;7, e42626.
42. Zero DT, Lussi A. Etiology of enamel erosion – intrinsic and extrinsic factors; in tooth wear and sensitivity. London: Martin Dunitz Ltd; 2000. p. 121–39.
43. McClure FJ. The destructive action, in vivo, of dilute acids and acid drinks and beverages on the rat's molar teeth. J Nutr. 1943;26:251–9.
44. Miller CD. Enamel erosive properties of fruits and various beverages. J Am Diet Assoc. 1952;28:319–24.
45. Holloway PJ, Mellanby M, Stewart RJC. Fruit drinks and tooth erosion. Br Dent J. 1958;104:305–9.
46. Stephan RM. Effects of different types of human foods on dental health in experimental animals. J Dent Res. 1966;45:1551–61.
47. Sorvari R. Effects of various sport drink modifications on dental caries and erosion in rats with controlled eating and drinking pattern. Proc Finn Dent Soc. 1989;85:13–20.
48. Mistry M, Grenby TH. Erosion by soft drinks of rat molar teeth assessed by digital image analysis. Caries Res. 1993;27:21–5.
49. Young A, Tenuta LM. Initial erosion models. Caries Res. 2011;45 Suppl 1:33–42.
50. West NX, Davies M, Amaechi BT. In vitro and in situ erosion models for evaluating tooth substance loss. Caries Res. 2011;45 Suppl 1:43–52.
51. Drewnowski A, Rehm CD, Constant F. Water and beverage consumption among adults in the United States: cross-sectional study using data from NHANES 2005-2010. BMC Public Health. 2013;13:1068.
52. Heilman JR, Kiritsy MC, Levy SM, Wefel JS. Assessing fluoride levels of carbonated soft drinks. J Am Dent Assoc. 1999;130:1593–9.
53. Beyer M, Reichert J, Bossert J, Sigusch BW, Watts DC, Jandt KD. Acids with an equivalent taste lead to different erosion of human dental enamel. Dent Mater. 2011;27:1017–23.
54. Ireland AJ, McGuinness N, Sherrif M. An investigation into the ability of soft drinks to adhere to enamel. Caries Res. 1995;29:470–6.
55. Aykut-Yetkiner A, Wiegand A, Bollhalder A, Becker K, Attin T. Effect of acidic solution viscosity on enamel erosion. J Dent Res. 2013;92:289–94.
56. Aykut-Yetkiner A, Wiegand A, Ronay V, Attin R, Becker K, Attin T. In vitro evaluation of the erosive potential of viscosity-modified soft acidic drinks on enamel. Clin Oral Investig. 2014;18:769–73.
57. Barbour ME, Lussi A, Shellis RP. Screening and prediction of erosive potential. Caries Res. 2011;45 Suppl 1:24–32.
58. Jensdottir T, Bardow A, Holbrook P. Properties and modification of soft drinks in relation to their erosive potential in vitro. J Dent. 2005;33:569–75.
59. Hara AT, Zero DT. Analysis of the erosive potential of calcium-containing acidic beverages. Eur J Oral Sci. 2008;116:60–5.
60. Scaramucci T, Sobral MA, Eckert GJ, Zero DT, Hara AT. In situ evaluation of the erosive potential of orange juice modified by food additives. Caries Res. 2012;46:55–61.
61. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. Caries Res. 2004;38:34–44.

62. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci. 1996;104:199–206.
63. Larsen MJ. Dissolution of enamel. Scand J Dent Res. 1973;81:518–22.
64. Lussi A, Jäeggi T, Schärer S. The influence of different factors on in vitro enamel erosion. Caries Res. 1993;27:387–93.
65. Scaramucci T, Hara AT, Zero DT, Ferreira SS, Aoki IV, Sobral MA. In vitro evaluation of the erosive potential of orange juice modified by food additives in enamel and dentine. J Dent. 2011;39:841–8.
66. Barbour ME, Shellis RP, Parker DM, Allen GC, Addy M. An investigation of some food-approved polymers as agents to inhibit hydroxyapatite dissolution. Eur J Oral Sci. 2005;113:457–61.
67. Grenby TH. Lessening dental erosive potential by product modification. Eur J Oral Sci. 1996;104:221–8.
68. Hughes JA, West NX, Parker DM, Newcombe RG, Addy M. Development and evaluation of a low erosive blackcurrant juice drink in vitro and in situ. 1. Comparison with orange juice. J Dent. 1999;27:285–9.
69. Wegehaupt F, Günthart N, Sener B, Attin T. Prevention of erosive/abrasive enamel wear due to orange juice modified with dietary supplements. Oral Dis. 2011;17:508–14.
70. Attin T, Weiss K, Becker K, Buchalla W, Wiegand A. Impact of modified acidic soft drinks on enamel erosion. Oral Dis. 2005;11:7–12.
71. Magalhães AC, Moraes SM, Rios D, Buzalaf MA. Effect of ion supplementation of a commercial soft drink on tooth enamel erosion. Food Addit Contam Part A Chem Anal Control Expo Risk Assess. 2009;26:152–6.
72. Hemingway CA, Shellis RP, Parker DM, Addy M, Barbour ME. Inhibition of hydroxyapatite dissolution by ovalbumin as a function of pH, calcium concentration, protein concentration and acid type. Caries Res. 2008;42:348–53.
73. Straub DA. Calcium supplementation in clinical practice: a review of forms, doses, and indications. Nutr Clin Pract. 2007;22:286–96.
74. Shellis RP, Featherstone JD, Lussi A. Understanding the chemistry of dental erosion. Monogr Oral Sci. 2014;25:163–79.
75. Ramalingam L, Messer LB, Reynolds EC. Adding casein phosphopeptide-amorphous calcium phosphate to sports drinks to eliminate in vitro erosion. Pediatr Dent. 2005;27:61–7.
76. Manton DJ, Cai F, Yuan Y, Walker GD, Cochrane NJ, Reynolds C, Brearley-Messer LJ, Reynolds EC. Effect of casein phosphopeptide-amorphous calcium phosphate added to acidic beverages on enamel erosion in vitro. Aust Dent J. 2010;55:275–9.
77. Min JH, Kwon HK, Kim BI. The addition of nano-sized hydroxyapatite to a sports drink to inhibit dental erosion: in vitro study using bovine enamel. J Dent. 2011;39:629–35.
78. Min JH, Kwon HK, Kim BI. Prevention of dental erosion of a sports drink by nano-sized hydroxyapatite in situ study. Int J Paediatr Dent. 2015;25:61–9.
79. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res. 1999;33:81–7.
80. Larsen MJ, Richards A. Fluoride is unable to reduce dental erosion from soft drinks. Caries Res. 2002;36:75–80.
81. Lussi A, Jaeggi T, Jaeggi-Schärer S. Prediction of the erosive potential of some beverages. Caries Res. 1995;29:349–54.
82. Hooper SM, Newcombe RG, Faller R, Eversole S, Addy M, West NX. The protective effects of toothpaste against erosion by orange juice: studies in situ and in vitro. J Dent. 2007;35:476–81.
83. Lussi A. Dental erosion–novel remineralizing agents in prevention or repair. Adv Dent Res. 2009;21:13–6.
84. Linkosalo E, Markkanen H. Dental erosion in relation to lactovegetarian diet. Scand J Dent Res. 1985;93:436–41.
85. Al-Dlaigan YH, Shaw L, Smith A. Dental erosion in a group of British 14-year-old school children. Part II: Influence of dietary intake. Br Dent J. 2001;190:258–61.

86. Ratnayake N, Ekanayake L. Prevalence and distribution of tooth wear among Sri Lankan adolescents. Oral Health Prev Dent. 2010;8:331–7.
87. El Aidi H, Bronkhorst EM, Huysmans MC, Truin GJ. Multifactorial analysis of factors associated with the incidence and progression of erosive tooth wear. Caries Res. 2011;45: 303–12.
88. Brand HS, Gambon DL, Van Dop LF, Van Liere LE, Veerman EC. The erosive potential of jawbreakers, a type of hard candy. Int J Dent Hyg. 2010;8:308–12.
89. Gambon DL, Brand HS, Nieuw Amerongen AV. The erosive potential of candy sprays. Br Dent J. 2009;206, E20; discussion 530–1.
90. Lussi A, Portmann P, Burhop B. Erosion on abraded dental hard tissues by acid lozenges: an in situ study. Clin Oral Invest. 1997;1:191–4.
91. West ES, Judy FR. Destruction of tooth enamel by acidified candies. J Dent Res. 1938;17: 499–504.
92. Brand HS, Gambon DL, Paap A, Bulthuis MS, Veerman EC, Amerongen AV. The erosive potential of lollipops. Int Dent J. 2009;59:358–62.
93. Wagoner SN, Marshall TA, Qian F, Wefel JS. In vitro enamel erosion associated with commercially available original-flavor and sour versions of candies. J Am Dent Assoc. 2009;140: 906–13.
94. Jensdottir T, Nauntofte B, Buchwald C, Hansen HS, Bardow A. Effects of sucking acidic candies on saliva in unilaterally irradiated pharyngeal cancer patients. Oral Oncol. 2006;42:317–22.
95. Jensdottir T, Nauntofte B, Buchwald C, Bardow A. Effects of calcium on the erosive potential of acidic candies in saliva. Caries Res. 2007;41:68–73.
96. Dodds MW, Chidichimo D, Haas MS. Delivery of active agents from chewing gum for improved remineralization. Adv Dent Res. 2012;24:58–62.
97. Ly KA, Milgrom P, Rothen M. The potential of dental-protective chewing gum in oral health interventions. J Am Dent Assoc. 2008;139:553–63.
98. Paice EM, Vowles RW, West NX, Hooper SM. The erosive effects of saliva following chewing gum on enamel and dentine: an ex vivo study. Br Dent J. 2011;210, E3.
99. Maguire A, Baqir W, Nunn JH. Are sugars-free medicines more erosive than sugars-containing medicines? An in vitro study of paediatric medicines with prolonged oral clearance used regularly and long-term by children. Int J Paediatr Dent. 2007;17:231–8.
100. Sullivan RE, Kramer WS. Iatrogenic erosion of teeth. J Dent Child. 1983;50:192–6.
101. Smith BGN. Toothwear: aetiology and diagnosis. Dent Update. 1989;16:204–12.
102. Giunta JL. Dental erosion resulting from chewable vitamin C tablets. J Am Dent Assoc. 1983;107:253–6.
103. James PMC, Parfitt GJ. Local effects of certain medicaments on the teeth. Br Med J. 1953;2:1252–3.
104. Kapila YL, Kashani H. Cocaine-associated rapid gingival recession and dental erosion. A case report. J Periodontol. 1997;68:485–8.
105. Bhatti SA, Walsh TF, Douglas WI. Ethanol and pH levels of proprietary mouthrinses. Community Dent Health. 1994;11:71–4.
106. Rytömaa I, Meurman JH, Franssila S, Torkko H. Oral hygiene products may cause dental erosion. Proc Finn Dent Soc. 1989;85:161–6.
107. Meurman JH, Sorvari R, Pelttari A, Rytömaa I, Franssila S, Kroon L. Hospital mouth-cleaning aids may cause dental erosion. Spec Care Dentist. 1996;16:247–50.
108. Rogalla K, Finger W, Hannig M. Influence of buffered and unbuffered acetylsalicylic acid on dental enamel and dentine in human teeth: an in vitro pilot study. Methods Find Exp Clin Pharmacol. 1992;14:339–46.
109. McNally LM, Barbour ME, O'Sullivan DJ, Jagger DC. An in vitro investigation of the effect of some analgesics on human enamel. J Oral Rehabil. 2006;33:529–32.
110. McCracken M, O'Neal SJ. Dental erosion and aspirin headache powders: a clinical report. J Prosthodont. 2000;9:95–8.
111. Grace EG, Sarlani E, Kaplan S. Tooth erosion caused by chewing aspirin. J Am Dent Assoc. 2004;135:911–4.

112. Hays GL, Bullock Q, Lazzari EP, Puente ES. Salivary pH while dissolving vitamin C-containing tablets. Am J Dent. 1992;5:269–71.
113. Maron FS. Enamel erosion resulting from hydrochloric acid tablets. J Am Dent Assoc. 1996;127:781–4.
114. McDerra EJ, Pollard MA, Curzon ME. The dental status of asthmatic British school children. Pediatr Dent. 1998;20:281–7.
115. Nunn JH, Ng SK, Sharkey I, Coulthard M. The dental implications of chronic use of acidic medicines in medically compromised children. Pharm World Sci. 2001;23:118–9.
116. Scatena C, Galafassi D, Gomes-Silva JM, Borsatto MC, Serra MC. In vitro erosive effect of pediatric medicines on deciduous tooth enamel. Braz Dent J. 2014;25:22–7.
117. Neves BG, Farah A, Lucas E, de Sousa VP, Maia LC. Are paediatric medicines risk factors for dental caries and dental erosion? Community Dent Health. 2010;27:46–51.
118. Valinoti AC, Pierro VS, Da Silva EM, Maia LC. In vitro alterations in dental enamel exposed to acidic medicines. Int J Paediatr Dent. 2011;21:141–50.
119. Hellwig E, Lussi A. Oral hygiene products and acidic medicines. Monogr Oral Sci. 2006;20:112–8.
120. Ganss C, Schlueter N, Klimek J. Retention of KOH-soluble fluoride on enamel and dentine under erosive conditions–A comparison of in vitro and in situ results. Arch Oral Biol. 2007; 52:9–14.
121. Pontefract H, Hughes J, Kemp K, Yates R, Newcombe RG, Addy M. The erosive effects of some mouthrinses on enamel. A study in situ. J Clin Periodontol. 2001;28:319–24.
122. Pretty IA, Edgar WM, Higham SM. The erosive potential of commercially available mouthrinses on enamel as measured by Quantitative Light-induced Fluorescence (QLF). J Dent. 2003;31:313–9.
123. Lussi A, Jaeggi T. The erosive potential of various oral care products compared to foodstuffs and beverages. Schweiz Monatsschr Zahnmed. 2001;111:274–81.
124. Attin T, Deifuss H, Hellwig E. Influence of acidified fluoride gel on abrasion resistance of eroded enamel. Caries Res. 1999;33:135–9.
125. Wiegand A, Wolmershäuser S, Hellwig E, Attin T. Influence of buffering effects of dentifrices and fluoride gels on abrasion on eroded dentine. Arch Oral Biol. 2004;49:259–65.
126. Kielbassa AM, Shohadai SP, Schulte-Mönting J. Effect of saliva substitutes on mineral content of demineralized and sound dental enamel. Support Care Cancer. 2001;9:40–7.
127. Hara AT, Turssi CP, Teixeira EC, Serra MC, Cury JA. Abrasive wear on eroded root dentine after different periods of exposure to saliva in situ. Eur J Oral Sci. 2003;111:423–7.
128. Hara AT, Lippert F, Zero DT. Interplay between experimental dental pellicles and stannous-containing toothpaste on dental erosion-abrasion. Caries Res. 2013;47:325–9.
129. Bardolia P, Burnside G, Ashcroft A, Milosevic A, Goodfellow SA, Rolfe EA, Pine CM. Prevalence and risk indicators of erosion in thirteen- to fourteen-year-olds on the Isle of Man. Caries Res. 2010;44:165–8.
130. Murchison DF, Charlton DG, Moore BK. Carbamide peroxide bleaching: effects on enamel surface hardness and bonding. Oper Dent. 1992;17:181–5.
131. Flaitz CM, Hicks MJ. Effects of carbamide peroxide whitening agents on enamel surfaces and caries-like lesion formation: an SEM and polarized light microscopic in vitro study. ASDC J Dent Child. 1996;63:249–56.
132. Zalkind M, Arwaz JR, Goldman A, Rotstein I. Surface morphology changes in human enamel, dentin and cementum following bleaching: a scanning electron microscopy study. Endod Dent Traumatol. 1996;12:82–8.
133. Rodrígues JA, Basting RT, Serra MC, Rodrígues Júnior AL. Effects of 10% carbamide peroxide bleaching materials on enamel microhardness. Am J Dent. 2001;14:67–71.
134. Basting RT, Rodrigues Jr AL, Serra MC. The effects of seven carbamide peroxide bleaching agents on enamel microhardness over time. J Am Dent Assoc. 2003;134:1335–42.
135. de Freitas PM, Turssi CP, Hara AT, Serra MC. Monitoring of demineralized dentin microhardness throughout and after bleaching. Am J Dent. 2004;17:342–6.

136. Arcari GM, Baratieri LN, Maia HP, De Freitas SF. Influence of the duration of treatment using a 10% carbamide peroxide bleaching gel on dentin surface microhardness: an in situ study. Quintessence Int. 2005;36:15–24.
137. Pretty IA, Edgar WM, Higham SM. The effect of bleaching on enamel susceptibility to acid erosion and demineralisation. Br Dent J. 2005;198:285–90.
138. Engle K, Hara AT, Matis B, Eckert GJ, Zero DT. Erosion and abrasion of enamel and dentin associated with at-home bleaching: an in vitro study. J Am Dent Assoc. 2010;141:546–51.
139. Ren YF, Amin A, Malmstrom H. Effects of tooth whitening and orange juice on surface properties of dental enamel. J Dent. 2009;37:424–31.
140. Elsbury WB. Hydrogen-ion concentration and acid erosion of the teeth. Br Dent J. 1952;93:177–9.
141. Bamise CT, Esan TA, Ajayi JO, Olagundoye O, Oziegbe EO. Dental erosion in a road-side battery technician: case report and a review of the literature. Oral Health Prev Dent. 2008;6:249–53.
142. Petersen PE, Gormsen C. Oral conditions among German battery factory workers. Community Dent Oral Epidemiol. 1991;19:104–6.
143. Chikte UM, Josie-Perez AM, Cohen TL. A rapid epidemiological assessment of dental erosion to assist in settling an industrial dispute. J Dent Assoc S Afr. 1998;53:7–12.
144. ten Brugen Cate HJ. Dental erosion in industry. Br J Ind Med. 1968;25:249–66.
145. Tuominen M, Tuominen R. Dental erosion and associated factors among factory workers exposed to inorganic acid fumes. Proc Finn Dent Soc. 1991;87:359–64.
146. Tuominen M, Tuominen R. Tooth surface loss and associated factors among factory workers in Finland and Tanzania. Community Dent Health. 1992;9:143–50.
147. Arowojolu MO. Erosion of tooth enamel surfaces among battery chargers and automobile mechanics in Ibadan: a comparative study. Afr J Med Med Sci. 2001;30:5–8.
148. Amin WM, Al-Omoush SA, Hattab FN. Oral health status of workers exposed to acid fumes in phosphate and battery industries in Jordan. Int Dent J. 2001;51:169–74.
149. Johansson AK, Johansson A, Stan V, Ohlson CG. Silicone sealers, acetic acid vapours and dental erosion: a work-related risk? Swed Dent J. 2005;29:61–9.
150. Wiegand A, Attin T. Occupational dental erosion from exposure to acids: a review. Occup Med (Lond). 2007;57:169–76.
151. Sorvari R, Rytomaa I. Drinks and dental health. Proc Finn Dent Soc. 1991;87:621–31.
152. Ferguson MM, Dunbar RJ, Smith JA, Wall JG. Enamel erosion related to winemaking. Occup Med (Lond). 1996;46:159–62.
153. Chaudhry SI, Harris JL, Challacombe SJ. Dental erosion in a wine merchant: an occupational hazard? Br Dent J. 1997;182:226–8.
154. Gray A, Ferguson MM, Wall JG. Wine tasting and dental erosion. Case report. Aust Dent J. 1998;43:32–4.
155. Wiktorsson AM, Zimmerman M, Angmar-Mansson B. Erosive tooth wear prevalence and severity in Swedish winetasters. Eur J Oral Sci. 1997;105:544–50.
156. Chikte UM, Naidoo S, Kolze TJ, Grobler SR. Patterns of tooth surface loss among winemakers. SADJ. 2005;60:370–4.
157. Savad EN. Enamel erosion…multiple cases with a common cause(?). J N J Dent Assoc. 1982;53:32–7. 60.
158. Centerwall BS, Armstrong CW, Funkhouser LS, Elzay RP. Erosion of dental enamel among competitive swimmers at a gas-chlorinated swimming pool. Am J Epidemiol. 1986;123:641–7.
159. Zero DT, Lussi A. Erosion–chemical and biological factors of importance to the dental practitioner. Int Dent J. 2005;55:285–90.
160. Hara AT, Ando M, González-Cabezas C, Cury JA, Serra MC, Zero DT. Protective effect of the dental pellicle against erosive challenges in situ. J Dent Res. 2006;85:612–6.
161. Järvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991;70:942–7.

162. Jensdottir T, Arnadottir IB, Thorsdottir I, Bardow A, Gudmundsson K, Theodors A, Holbrook WP. Relationship between dental erosion, soft drink consumption, and gastroesophageal reflux among Icelanders. Clin Oral Investig. 2004;8:91–6.
163. Johansson AK, Lingström P, Birkhed D. Comparison of factors potentially related to the occurrence of dental erosion in high- and low-erosion groups. Eur J Oral Sci. 2002;110: 204–11.
164. Künzel W, Cruz MS, Fischer T. Dental erosion in Cuban children associated with excessive consumption of oranges. Eur J Oral Sci. 2000;108:104–9.
165. 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;112:484–9.
166. Walsh NP, Montague JC, Callow N, Rowlands AV. Saliva flow rate, total protein concentration and osmolality as potential markers of whole body hydration status during progressive acute dehydration in humans. Arch Oral Biol. 2004;49:149–54.
167. Moses FM. The effect of exercise on the gastrointestinal tract. Sports Med. 1990;9:159–72.
168. Attin T, Buchalla W, Putz B. In vitro evaluation of different remineralization periods in improving the resistance of previously eroded bovine dentine against tooth-brushing abrasion. Arch Oral Biol. 2001;46:871–4.
169. González-Cabezas C, Hara AT, Hefferren J, Lippert F. Abrasivity testing of dentifrices – challenges and current state of the art. Monogr Oral Sci. 2013;23:100–7.

Part II

Clinical Practice

Diagnosis of Dental Erosion

5

Carolina Ganss and Nadine Schlueter

Abstract

Dental erosion is an acid-mediated tissue loss without bacterial involvement. It occurs on occlusal and smooth surfaces and is a surface phenomenon. The diagnosis can therefore be easily made by visual inspection. In initial stages, the normal surface texture and lustre of enamel is lost. When the acid impacts continue, convex tooth structures flatten and distinct defects develop. On smooth surfaces, erosive lesions are mainly located coronal to the enamel-cementum junction and are flat, dull, with an intact enamel rim at the gingival margin. On occlusal surfaces, cusps are flattened and cupped; restorations may stand proud of the surface. In advanced stages the occlusal morphology may completely disappear. Erosive lesions must be distinguished from attrition, abrasion and abfraction/wedge-shaped defects. For grading, the Basic Erosive Wear Examination (BEWE) is described which is also a guide to management. However, dental erosion is part of the physiological wear of the functioning dentition throughout lifetime. An adequate diagnosis must therefore differentiate between physiological and pathological stages of the condition integrating not only age, extent of tissue loss and progression rate but also the subjective sphere of the patient including pain, as well as functional and social impairment. The disease-illness-sickness triad is described as a concept for integrating the professional and subjective/social dimension of the condition.

C. Ganss (✉) • N. Schlueter
Department of Conservative and Preventive Dentistry,
Dental Clinic Justus Liebig University Giessen, Giessen, Germany
e-mail: Carolina.Ganss@dentist.med.uni-giessen.de;
Nadine.Schlueter@dentist.med.uni-giessen.de

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_5

5.1 Introduction

Establishing a straightforward and well-founded diagnosis is the prerequisite for successful treatment. In the case of erosive wear, the diagnostic process comprises of distinguishing between different forms of tooth wear, defining the stage of the condition, assessing whether a lesion is actually progressing or not, and estimating whether a given stage of erosive wear is pathological or not. Further, the subjective and social dimension of the condition needs to be considered. This complex diagnostic process is the basis for a modern, patient-centred causal and/or symptomatic treatment.

5.2 The Differential Diagnosis of Dental Erosion

Immersing a human tooth crown into an acidic solution causes an amorphous centripetal loss of mineral without the development of characteristic lesions (Fig. 5.1). In the oral cavity, however, such lesions are normally not found because it is not only the acid acting on the tooth surface. Instead, there is a multitude of chemical and physical impacts occurring as result of the normal or pathological (e.g. tooth clenching or grinding) function of the dentition. Other physical impacts are oral health related (e.g. oral hygiene procedures) or culture mediated (e.g. using teeth as tools, tooth modification, betel chewing). Despite the diversity of impacts, however, loss of tooth substance is not indefinitely variable. Instead, several types of lesions occur which allow for more or less reliable conclusions regarding the main causal factor. Thus, lesions are classified as attrition, abrasion, abfraction (wedge-shaped defect) and erosion [2]. Tooth wear in general is a surface phenomenon and occurs

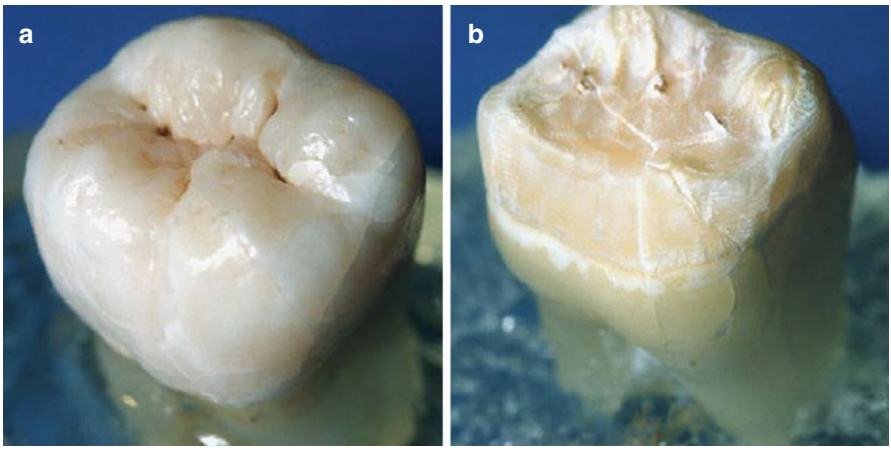


Fig. 5.1 Effect of the continuous exposure of a human third molar to 10 % citric acid. The amorphous, centripetal tissue loss is obvious: (**a**) unaffected tooth, (**b**) tissue loss after 12 h immersion time. In part from Ganss [1]

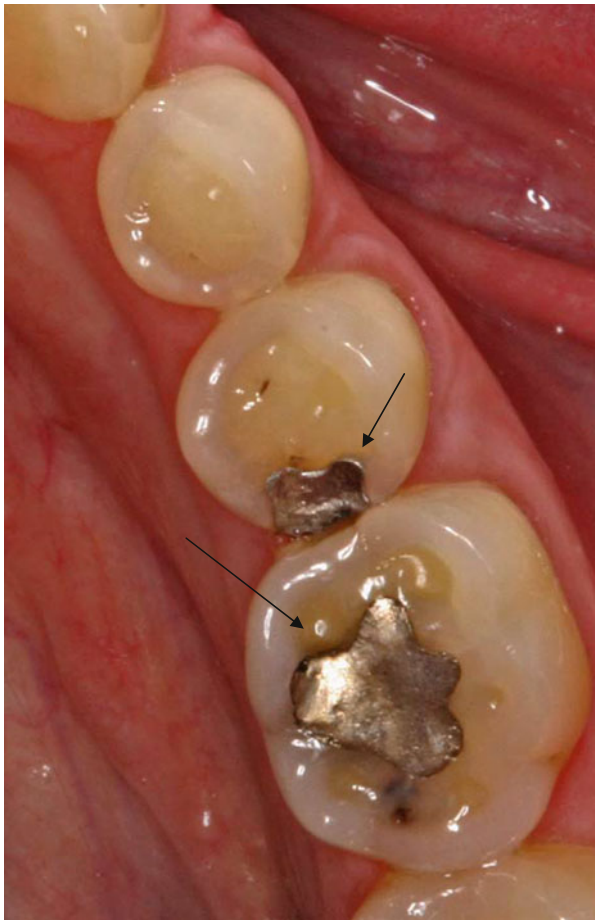


Fig. 5.2 Occlusal erosion. Typical signs are flattening of the occlusal morphology, cupping of the cusps and filling proud of the surface (*arrows*)

on smooth surfaces and occlusal areas easily accessible for the naked eye. The diagnosis is therefore made from visual inspection; further apparative approaches are normally not required.

The diagnostic criteria for dental erosion were derived from early observations in small groups of patients with known acid exposures [3, 4], and are meanwhile established in clinical diagnosis [5] and erosion index systems [6]. This type of tooth wear is acid-mediated with no bacteria involved. In initial stages of lesion development, the tooth surface may appear dull and the normal surface structures of enamel flatten. In later stages, a distinct defect develops. On occlusal surfaces cusps are cupped, the morphology flattens and restorations may stand proud of the surface (Fig. 5.2). Normally, there are no occlusal contacts at the lesion area. Finally, the crown height may be severely reduced. On smooth surfaces, erosive lesions occur coronal from the enamel-cementum junction often leaving an intact cervical rim (Fig. 5.3). The lesions are shallow and can affect the entire tooth surface.

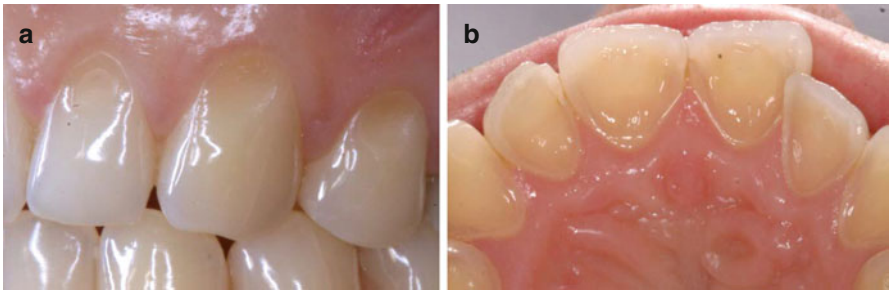


Fig. 5.3 Erosion on smooth surfaces. (**a**) Shallow lesions coronal from the enamel-cementum junctions at the buccal surfaces. Note the intact enamel rim at the gingival margin. (**b**) Palatal erosion: the palatal morphology has disappeared, note also the cervical intact enamel rim

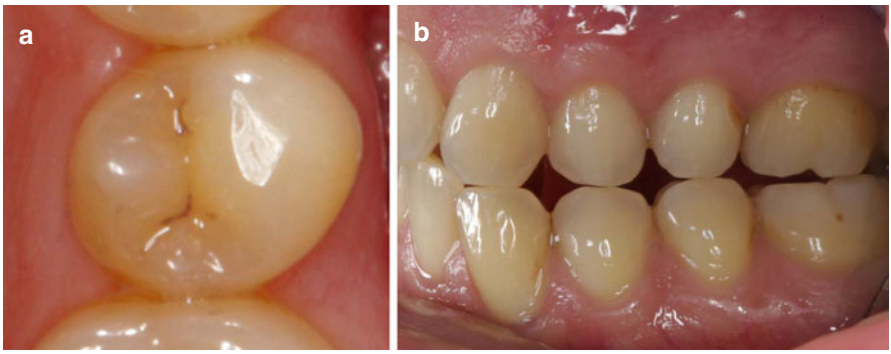


Fig. 5.4 Attrition. (**a**) Typical flat, sharp-bordered tissue loss on the buccal cusp of a second upper premolar. (**b**) The same subject as in (a) – note the attritional defects on opposing teeth

The differential diagnosis of erosive wear is tissue loss predominantly caused by physical forces namely attrition, abrasion and abfraction/wedge-shaped defects (non-carious cervical lesions).

Attrition is the result of two-body wear from tooth-to-tooth contacts. Respective lesions (Fig. 5.4a) can only occur in occluding areas of the dentition, normally on occlusal surfaces. Its typical shape is flat and sharply bordered and its surface appears highly polished. Typically, lesions occur on both opposing tooth surfaces (Fig. 5.4b).

Abrasion occurs as result of three-body-wear, e.g. from toothbrushing with toothpaste or the interaction of opposing teeth with a food bolus (demastication). This form of tooth wear occurs mainly in dentin which is much less wear-resistant than enamel [7]. On smooth surfaces, abrasion thus is mainly located apical to the enamel-cementum junction and occurs on exposed root surfaces (Fig. 5.5). On occlusal surfaces, exposed dentin is hollowed out resulting in cupping of cusps and grooving of incisal edges (Fig. 5.6a). These lesions look very similar to occlusal erosive lesions (Fig. 5.6b). Abrasive lesions are smooth-bordered, and can be of variable shape depending on the causing impact.

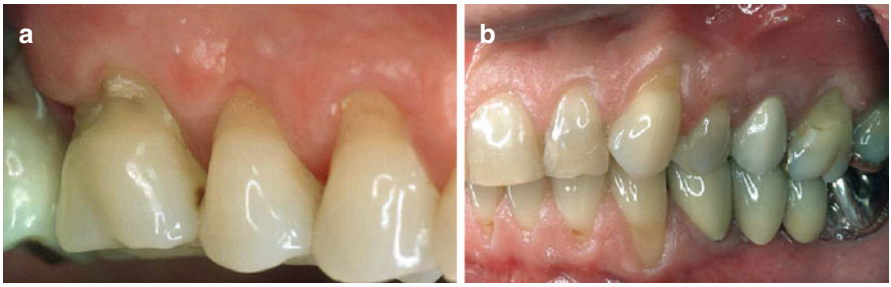


Fig. 5.5 Abrasion on smooth surfaces. Lesions are located apical to the enamel-cementum junction, combined with gingival recession. (**a**) Lesions at teeth 16–14, 65-year-old woman. (**b**) Lesions at 23, 24, 26, 33 and 32, 30-year-old woman. Note that lesions at the first upper molars look very similar and would be regarded as a more severe stage at younger than at older age

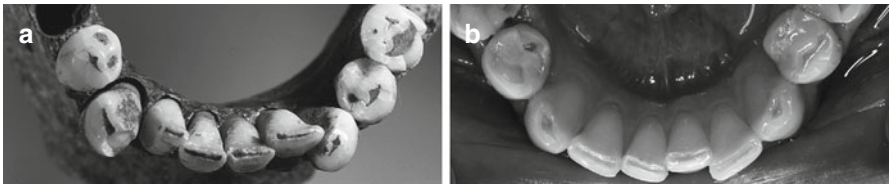


Fig. 5.6 Abrasive lesions on occlusal and incisal surfaces (demastication) in a medieval remain (**a**) resulting from an abrasive diet and in a subject with chronic vomiting (**b**). Note the strikingly similar shape of tissue loss (in part from Ganss [1])

Abfraction/wedge-shaped defects (also called non-carious cervical lesions) are located at the enamel-cementum border. Its coronal part cuts more or less rectangular into the enamel and normally is sharply demarcated, whereas its apical part runs flatly towards the root surface (Fig. 5.7). The aetiology of this type of lesions is still not clarified. It was suggested to be the result of either improper toothbrushing or eccentric functional forces causing flexural stress at the cervical area of the tooth disaggregating the enamel crystals, or an interaction of both [8]. Non-carious cervical lesions and its management is discussed in detail in Chap. 14 of this book.

The differential diagnosis on smooth and occlusal surfaces is illustrated in Figs. 5.8 and 5.9. The most challenging situation is to distinguish between abrasive and erosive lesions on occlusal/incisal surfaces because both types of tissue loss may look very similar (Fig. 5.6a, b). This was shown in a study investigating tooth wear in subjects with substantially different nutrition [9]. Lesions on occlusal surfaces in medieval remains with an abrasive diet and in contemporary subjects with an acidic diet were more severe in the former, but were similar in shape; incisal grooving was a common condition in all subjects and was not related to any form of diet. Strikingly, smooth surfaces were never affected in subjects with abrasive diet, but frequently exhibited shallow lesions coronal to the enamel-cementum junction in subjects with acidic diet. The conclusion from this study is that such shallow smooth surface lesions may be a reliable criterion for distinguishing between abrasive and erosive tooth wear.

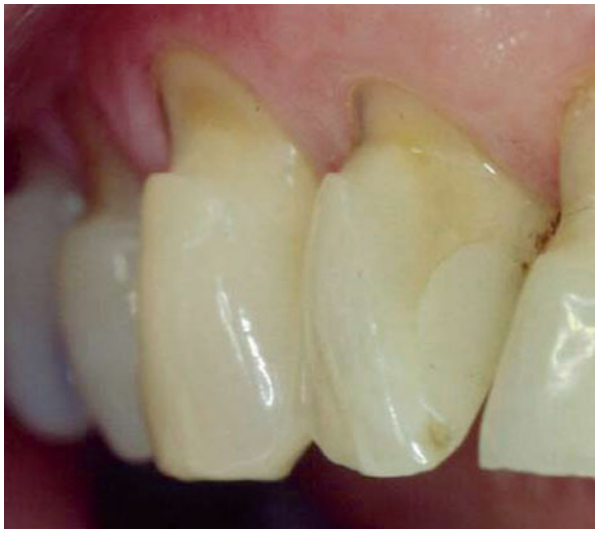


Fig. 5.7 Wedge-shaped defect (abfraction): the lesion is located at the enamel-cementum junction. It cuts more or less rectangular into the enamel at its coronal part and flattens towards the root surface at its apical part

In the individual subject, the clinical differential diagnosis of tooth wear can be substantiated by a thorough anamnesis and by nutrition diaries [5]. The anamnesis should cover the variety of aetiological factors of tooth wear concerning diet, oral hygiene, hobbies, sports and occupation and should address not only current, but also earlier conditions (See Chaps. 3 and 4). Many patients are not aware about the erosive potential of food and drinks, thus nutrition diaries may help identifying acidic food and drinks as well as other nutritional factors contributing to tooth wear. Dietary management in dental erosion is discussed in detail in Chap. 10 of this book.

5.3 Defining the Stage of Dental Erosion and Assessing Progression

At any stage, dental erosion ceases from progression as soon as the causal factor is sufficiently limited. However, when a clinically visible defect has developed, there is no option for restitutio ad integrum and lost dental hard tissue cannot regenerate. Different stages of erosive tissue loss require adapted preventive and therapeutic strategies [10]. Diagnosing early stages is important to initiate appropriate preventive measures to avoid further tissue loss. More advanced tissue loss may require a more complex intervention and in severe cases the lost tissue may need to be restored.

Thus, attempts have been made to score the severity of erosive lesions and to mirror the condition in an index value. Many indices have been proposed either scoring tooth wear as such or dental erosion in particular [6], but so far no index has been generally accepted. In principle, the severity of a given lesion is scored by estimating the percentage of the entire surface affected by the condition and by whether dentin is exposed or not. However, the differentiation between lesions

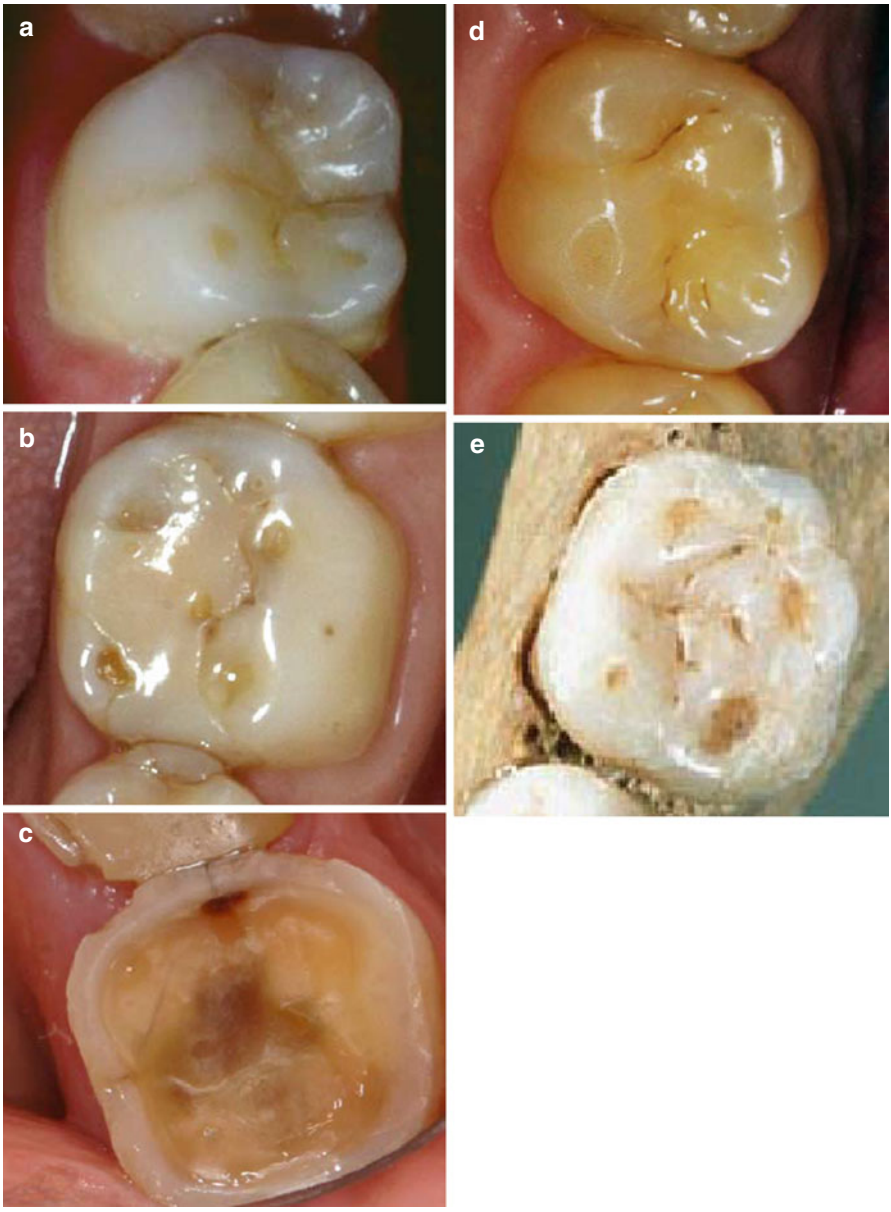


Fig. 5.8 Differential diagnosis of occlusal tooth wear. (**a–c**) Initial, moderate and severe erosive lesions. (**d**) Attrition at the mesio-palatal cusp with the typical flat and sharp-bordered shape. (**e**) Abrasion (demastication) in a medieval remain, note the strikingly similar shape of tissue loss compared to (**b**)

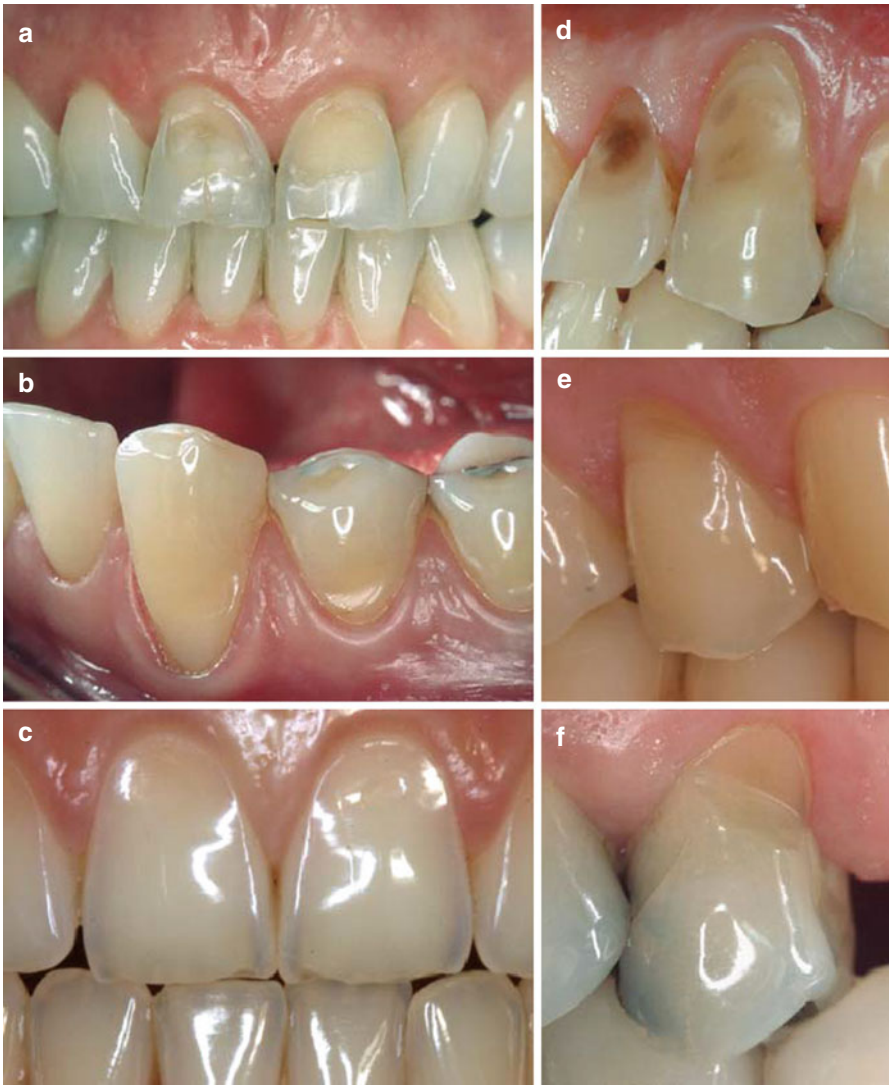


Fig. 5.9 Differential diagnosis of tooth wear on smooth surfaces. (**a**–**c**) Erosive lesions of buccal surfaces mainly located coronal from the enamel-cementum junction with an intact cervical enamel rim. (**d**) Abrasion typically smooth-bordered and located mainly on the root surface. (**e**, **f**) Wedge-shaped defects (abfraction) typically located at the enamel-cementum junction

restricted to enamel and lesions exhibiting exposed dentin is difficult because exposed dentin cannot be reliably diagnosed by clinical means [11]. Further, dentin exposure not necessarily relates to extensive tissue loss. For instance, in cases of cupping dentin is exposed even at early stages (Fig. 5.10). Similar holds true for lesions in the cervical region where the enamel covering is thin.

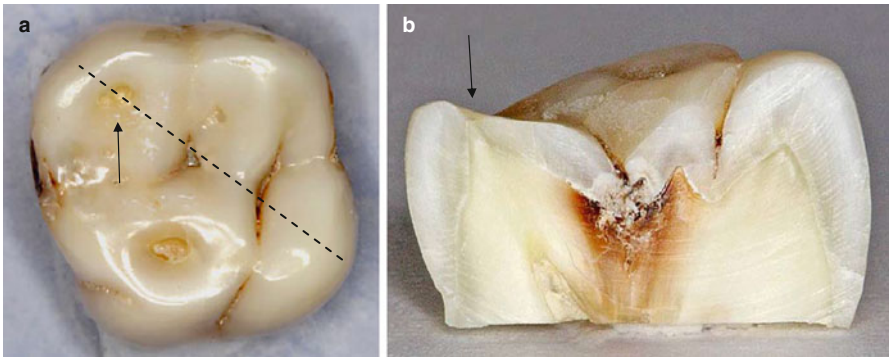


Fig. 5.10 (**a**) First upper molar with cupped cusps and moderate tissue loss. (**b**) Section of the tooth through a line indicated in (a): note the significant overall thickness of the enamel and dentin exposure at the cupped cusp. There is no relation between dentin exposure and tissue loss. In addition, severe dentin caries located at the centre of the tooth crown

Table 5.1 Criteria for grading erosive wear, from Bartlett et al. [10]

Score	
0	No erosive tooth wear
1	Initial loss of surface texture
2	Distinct defect, hard tissue loss <50 % of the surface area
3	Hard tissue loss ≥50 % of the surface area

Most of the indices have been designed – and are mainly used – for epidemiological trials rather than for individual patients in general practice and were not related to treatment needs.

Thus, the Basic Erosive Wear Examination (BEWE) was proposed [10]. The grading of the severity of a lesion is based on its extent on the tooth surface on a four grade level (Table 5.1). The dentition is divided into sextants and the most severely affected surface in a sextant is recorded. The sum of the scores of each sextant constitutes the index value and guides to recommendations for the management of the condition for the practitioner (Table 5.2).

In addition to assessing the stage of a lesion at a given time point, monitoring is important to determine whether a lesion is active and to check whether causal or symptomatic strategies were successfully implemented. A visible sign for inactive lesions is extrinsic staining whereas the absence of staining and dull appearance of the tooth surface may indicate progression. These criteria, however, are subjective and not well suitable for documenting monitoring. A simple and more reliable tool is following the condition with study models (Fig. 5.11) or photographs. The latter would require standardised settings to enable reproducible images. The two-dimensional extension of a given lesion can be quantified by image software tools. For dental practitioners, study models may be more useful as they are easy to obtain and allow for a three-dimensional analysis and visual comparison of lesions at different time points. The numerical quantification of tissue loss on study models, however, is difficult. Several procedures have been suggested [12–16] but all need extensive equipment and are expensive

Table 5.2 Complexity levels as a guide to clinical management, from Bartlett et al. [10]

Complexity level	Cumulative score of all sextants	Management
0	Less than or equal to 2[a]	Routine maintenance and observation Repeat at 3-year intervals
1	Between 3 and 8[a]	Oral hygiene and dietary assessment, and advice, routine maintenance and observation Repeat at 2-year intervals
2	Between 9 and 13[a]	Oral hygiene and dietary assessment, and advice, identify the main aetiological factor(s) for tissue loss and develop strategies to eliminate respective impacts Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces Ideally, avoid the placement of restorations and monitor erosive wear with study casts, photographs, or silicone impressions Repeat at 6–12 month intervals
3	14 and over[a]	Oral hygiene and dietary assessment, and advice, identify the main aetiological factor(s) for tissue loss and develop strategies to eliminate respective impacts Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces Ideally, avoid restorations and monitor tooth wear with study casts, photographs, or silicone impressions Especially in cases of severe progression consider special care that may involve restorations Repeat at 6–12 month intervals

[a]The cut-off values are based on experience and studies of one of the authors (A.L.) and have to be reconsidered

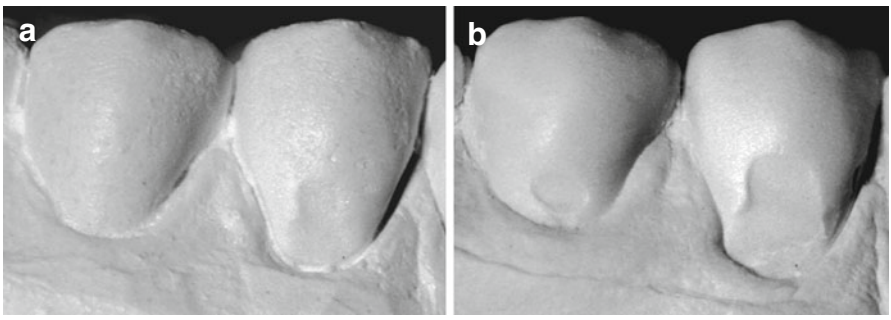


Fig. 5.11 Study models of a young male. (**a**) Minor erosive tissue loss at 44. (**b**) The same subject 4 years later. Note the progression of tissue loss at 44 and the new lesion at 45

and/or time consuming. Similar holds true for direct intra-oral techniques. Ultrasound measurements [17] and optical coherence tomography [18] have been used to measure erosive loss but a major disadvantage of these methods is that they can only quantify tissue loss restricted to enamel. A more promising

approach may be using intra-oral scanning devices for creating digital impressions, but these methods have not been modified for erosion monitoring so far. Monitoring dental erosion is discussed in detail in Chap. 6.

5.4 When Is a Given Stage of Erosive Wear Pathological?

Talking about diagnosis means distinguishing between healthy, physiological stages and pathology (Fig. 5.5a, b), and the decision whether to treat or not. Unlike caries, however, tooth wear in principle is a physiological process occurring from normal functioning of the dentition throughout lifetime. So far, no conclusive concept about dental erosion as an oral disease exists and a debate about the issue is urgently needed [19]. In cases of severe tissue loss threatening the integrity of the tooth or in cases of pulpal involvement or pain, there would be general agreement that this is a pathological condition. In less severe and particularly in initial stages, however, the definition of erosion as an oral disease is much more difficult. A potentially useful definition has been suggested by Smith and Knight [20]:

> Tooth wear can be regarded as pathological if the teeth become so worn that they do not function effectively or seriously mar the appearance before they are lost for other causes or the patient dies. The distinction of acceptable and pathological wear at a given age is based upon the prediction of whether the tooth will survive the rate of wear.

This definition helps in so far as it integrates progression rate and age but lacks precision with respect to matters of function and aesthetics. What has been implicitly included in this definition refers to a multidimensional concept of human ailment. Hofmann and Eriksen [21] outlined a concept integrating the subjective, professional and social dimension of human ailment in a triad of the terms disease, illness and sickness. Disease refers to the professional sphere seeking to classify and explain bodily or mental phenomena for the purpose of treatment or palliation. In this area, dental erosion is diagnosed as tissue loss using defined criteria as described above and would include professional concepts of functioning of the dentition. This concept does not consider the personal, subjective sphere which is addressed by the term illness. It covers the negative experience of the patient like pain, weakness or feeling of incompetence. Function of the dentition is also well situated here for instance with respect to impairment of the ability to chew food, pain from musculature or temporo-mandibular joint or hypersensitive teeth. Finally, there is a social dimension of ailment which is described by the term sickness. It refers to the intersubjective sphere and social activity. Here we would find the place for considering matters of aesthetics arising from (extensive) erosive tissue loss. Whether there is aesthetic impairment would be the patient's judgement in his or her specific social context but would also depend on the overall cultural realm. Just consider that tooth modification is part of the cultural identity of some ethnic entities.

The conclusion from these considerations is that the question whether dental erosion is an oral disease and whether or not to treat is related to the concept of disease. Particularly in the case of dental erosion, a concept beyond the established professional view integrating the various dimensions of human life may help establishing a modern patient-centred treatment concept including the professional definition given here as well as the patient's subjective sphere of ailment in a social and cultural context.

References

1. Ganss C. How valid are current diagnostic criteria for dental erosion? Clin Oral Investig. 2008;12:S41–9.
2. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci. 1996; 104:151–5.
3. Eccles JD, Jenkins WG. Dental erosion and diet. J Dent. 1974;2:153–9.
4. Stafne ECLA, Lovestedt SA. Dissolution of tooth substance by lemon juice, acid beverages and acids from other sources. J Am Dent Assoc. 1947;34:586–92.
5. Lussi A. Dental erosion clinical diagnosis and case history taking. Eur J Oral Sci. 1996;104:191–8.
6. Bardsley PF. The evolution of tooth wear indices. Clin Oral Investig. 2008;12:S15–9.
7. Addy M, Hunter ML. Can tooth brushing damage your health? Effects on oral and dental tissues. Int Dent J. 2003;53(Suppl):177–86.
8. Bartlett DW, Shah P. A critical review of non-carious cervical (wear) lesions and the role of abfraction, erosion, and abrasion. J Dent Res. 2006;85:306–12.
9. Ganss C, Klimek J, Borkowski N. Characteristics of tooth wear in relation to different nutritional patterns including contemporary and medieval subjects. Eur J Oral Sci. 2002;110: 54–60.
10. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008;12:S65–8.
11. Ganss C, Klimek J, Lussi A. Accuracy and consistency of the visual diagnosis of exposed dentine on worn occlusal/incisal surfaces. Caries Res. 2006;40:208–12.
12. Chadwick RG, Mitchell HL. Conduct of an algorithm in quantifying simulated palatal surface tooth erosion. J Oral Rehabil. 2001;28:450–6.
13. Mehl A, Gloger W, Kunzelmann KH, Hickel R. A new optical 3-D device for the detection of wear. J Dent Res. 1997;76:1799–807.
14. Mistry M, Grenby TH. Erosion by soft drinks of rat molar teeth assessed by digital image analysis. Caries Res. 1993;27:21–5.
15. Pintado MR, Anderson GC, DeLong R, Douglas WH. Variation in tooth wear in young adults over a two-year period. J Prosthet Dent. 1997;77:313–20.
16. Sorvari R, Kiviranta I. A semiquantitative method of recording experimental tooth erosion and estimation occlusal wear in the rat. Arch Oral Biol. 1988;33:217–20.
17. Louwerse C, Kjaeldgaard M, Huysmans MC. The reproducibility of ultrasonic enamel thickness measurements: an in vitro study. J Dent. 2004;32:83–9.
18. Wilder-Smith CH, Wilder-smith P, Kaeakami-Wong H, Voronets J, Osann K, Lussi A. Quantification of dental erosions in patients with GERD using optical coherence tomography before and after double-blind, randomized treatment with esomeprazole or placebo. Am J Gastroenterol. 2009;104:2788–95.
19. Ganss C. Definition of erosion and links to tooth wear. Monogr Oral Sci. 2006;20:9–16.
20. Smith BG, Knight JK. An index for measuring the wear of teeth. Br Dent J. 1984;156:435–8.
21. Hofmann BM, Eriksen HM. The concept of disease: ethical challenges and relevance to dentistry and dental education. Eur J Dent Educ. 2001;5:2–8.

Assessment and Monitoring of Dental Erosion

6

Bennett T. Amaechi

Abstract

Once detected and diagnosed, the severity and extent of an erosive tooth wear must be recorded to establish the clinical baseline, so that progression can be detected. Longitudinal monitoring of the erosive process would enable the outcomes of the preventive and therapeutic strategies to be assessed scientifically and quantitatively at the recall and review visits. The following methods described in this chapter are available and useful for clinical assessment and monitoring of erosive tooth wear: silicone matrix system, serial reference casts, clinical photographs, and diagnostic indices such as Basic Erosive Wear Examination and Tooth Wear index.

6.1 Introduction

An accurate assessment and longitudinal monitoring is very important in the clinical management of dental erosion. The severity levels of any detected and diagnosed erosive tooth wear need to be recorded at the initial comprehensive assessment of the patient to establish the clinical baseline and on completion of a particular treatment journey. Thereafter the erosive lesion has to be monitored over time, since a lesion may progress in severity by increasing in size and depth or may remain unchanged (arrested) over time. Besides, longitudinal monitoring of the erosive process would enable the outcomes of the preventive and therapeutic strategies to be assessed scientifically and quantitatively at the recall and review visits. At every

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas, Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_6

recall visit the practitioner should re-assess and monitor the progression status (arrested or progressing) of a previously diagnosed and treated erosive lesion.

The understanding that tooth wear in general is surface phenomenon is not absolutely correct for erosive tooth wear. The erosion process is characterized by initial softening of the enamel surface layer by acid demineralization. If this process is not interrupted, continuous dissolution of the enamel crystals will lead to a permanent loss of tooth substance, creating a crater. Thus an erosive tooth wear consists of a crater of tooth substance loss and a demineralized (softened) layer at the base of the lesion [1, 2] as shown in Fig. 6.1. Thus the diagnosis of dental erosion can be made from visual inspection alone, but the clinical monitoring of erosive tooth wear over time requires a device that is capable of quantifying both the volume of tooth substance loss as well as the mineral loss in the underlying softened tooth tissue. Unfortunately, there is no diagnostic device available at present to accomplish this clinically. However, attempts have been made to develop methods to either objectively and quantitatively measure or subjectively estimate the severity of an erosive tooth wear and monitor its progression status over time without quantifying the mineral loss in the softened layer at the base of the lesion.

Fig. 6.1 A polarized light microscope image of an erosive tooth wear with a crater formed by tooth substance loss (*x*), a layer of softened tooth tissue (*y*) and the sound enamel (*z*). X150

6.2 Objective and Quantitative Monitoring of Erosion

6.2.1 Silicone Matrix

A simple and more reliable tool for monitoring the progression status of an erosive lesion over time is the silicone matrix system described by Shaw et al. [3]. This is an objective and quantitative clinical method of monitoring erosive tooth wear. It is one of the easiest and most useful methods of monitoring tooth wear. This is no difference from the simple method used by dental students to measure and monitor tooth tissue reduction during crown preparation. With this system, a silicone putty impression of the teeth is taken in an "unglued" sectional tray (Fig. 6.2a). The putty is removed from the tray, and it is sliced into sections through the centers of the erosive lesions (Fig. 6.2b). When a section is replaced on tooth surface, it is a perfect fit to the tooth surface (Fig. 6.2c). If the erosive lesion progresses, on the next review visit a gap will become visible between the silicone and the lesion surface (Fig. 6.2d). The depth of the gap can be measured in millimeters (mm) using the calibrated periodontal probe (explorer). An erosive wear can be confirmed arrested when there is no gap after several review visits. Besides being objectively quantitative, the silicone matrix enables slight change in depth and extent of the lesion to be detected and measured, which is its major advantage over the other methods discussed below. Generating quantitative data would also permit quantitative

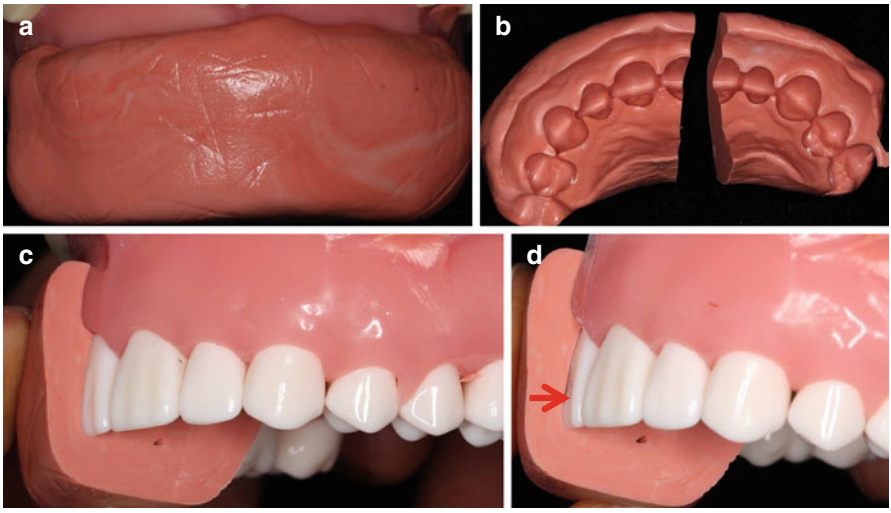


Fig. 6.2 An illustration of the procedure for using silicone matrix to monitor erosive lesion progression status over time. (**a**) Silicone putty impression of the teeth is taken in an "unglued" sectional tray. (**b**) The putty is removed from the tray, and it is sliced into sections through the center of the erosive lesion. (**c**) When a section is replaced on tooth surface, it is a perfect fit to the tooth surface. (**d**) If the erosive lesion progresses, a gap (*red arrowed*) will become visible between the silicone and the lesion surface

comparison of lesions at different time points, and possible statistical comparative analysis of the erosion progression over time.

6.2.2 Other Emerging Quantitative Systems

Following the demonstration of the feasibility of using the ultrasound system to measure enamel thickness [4], measurement of enamel thickness using a high-frequency ultrasonic transducer-based hand-held probe was used to follow the progression of enamel erosion by acid dissolution [5]. However, a study showed that due to measurement variation, thickness changes of less than 0.33 mm cannot be detected reliably [6]. Besides, ultrasound systems can only quantify tissue loss limited to enamel. Wilder-Smith et al. [7] used optical coherence tomography (OCT) to successfully monitor the progression status of erosive tooth wear in patients with GERD; however, just like the ultrasound system, OCT can only measure tissue loss limited to enamel.

6.3 Subjective and Semi-quantitative Monitoring of Erosion

6.3.1 Diagnostic Indices

The diagnostic indices score the severity of erosive lesions to mirror the condition in an index value, hence its semi-quantitative nature. The severity of an erosive lesion is scored by estimating the percentage of the entire surface affected by the condition and by whether dentin is exposed or not. The diagnostic indices are subjective evaluations, and are faced with the difficulty in distinguishing between lesions restricted to enamel and lesions with exposed dentin because exposed dentin cannot be reliably diagnosed by clinical means [8]. Besides, dentin exposure may not necessarily relate to extensive tissue loss, particularly in areas where the enamel covering is thin, such as in cervical regions of the teeth. Unlike the silicone matrix, diagnostic indices also have the problem of inability to detect slight change in lesion severity, particularly if the progression is in depth and not in area of the lesion. The following indices have been proposed [9], but none has been generally accepted.

6.3.1.1 Basic Erosive Wear Examination

Most of the available indices were designed for epidemiological studies rather than for individual patients management in general practice and were not related to treatment needs. This has led to the proposal of a management-based index, the Basic Erosive Wear Examination (BEWE), by Bartlett et al. [10]. With the BEWE, the severity level of a lesion is scored on a four grade level based on its extent on the tooth surface (Table 6.1). The dentition is divided into sextants (Fig. 6.3), and the buccal, occlusal and lingual surfaces of every tooth in each sextant is examined

Table 6.1 Criteria for grading erosive tooth wear by the Basic Erosive Wear Examination [10]

Scores	Erosive wear severity levels
0	No erosive tooth wear
1	Initial loss of surface texture
2[a]	Distinct defect, hard tissue loss <50 % of the surface area
3[a]	Hard tissue loss ≥50 % of the surface area

[a]May involve dentin

Fig. 6.3 An illustration of the division of the dentition into sextants for the Basic Erosive Wear Examination (BEWE)

for erosive wear and awarded a score value between 0 and 3. Then the most severely affected surface in a sextant is recorded. The sum of the scores from the sextants constitutes the index value (BEWE score), and guides the recommendations for the management of the condition by the practitioner (Table 6.2). The BEWE can be used to determine the erosion risk status of an individual patient [11]. Also the BEWE is a useful tool for monitoring the progression status of erosive lesions. However, as stated above, the subjective nature of the BEWE makes it difficult, if not impossible, for slight progression of the erosive lesion to be detected. Furthermore, with the BEWE, there is the possibility of ignoring newly developed erosive lesions.

6.3.1.2 Tooth Wear Index

The Tooth Wear Index (TWI) of Smith and Knight [12] is the most widely used index. TWI records the degree of wear on all visible surfaces of each tooth. Each surface of each tooth is given a score between 0 and 4 according to the criteria detailed in Table 6.3. In case of doubt, a lower score is given. Unlike the BEWE, the scores from all tooth surfaces are recorded, thus facilitating the monitoring of the progression status of each erosive lesion. Also the TWI would enable easy detection of a newly developed erosive lesion. Though a subjective method, the TWI is ideal for detection and longitudinal monitoring of erosive lesion (Fig. 6.4a, b).

Table 6.2 Erosive wear severity levels as scored using BEWE [10]

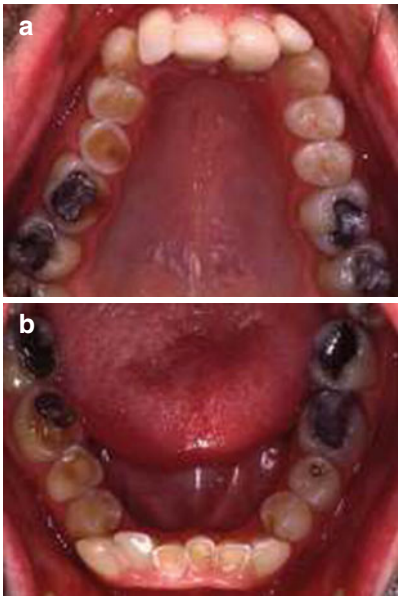
Severity levels	Cumulative score of all sextants	Suggested management
0	Less than or equal to 2[a]	• Routine maintenance and observation • Repeat at 3-year intervals
1	Between 3 and 8[a]	• Oral hygiene and dietary assessment, and advice, routine maintenance and observation • Repeat at 2-year intervals
2	Between 9 and 13[a]	• Oral hygiene and dietary assessment, and advice, identify the main etiological factor(s) for tissue loss and develop strategies to eliminate respective impacts • Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces • Ideally, avoid the placement of restorations and monitor erosive wear with study casts, photographs, or silicone impressions • Repeat at 6–12 month intervals
3	14 and over[a]	• Oral hygiene and dietary assessment, and advice, identify the main etiological factor(s) for tissue loss and develop strategies to eliminate respective impacts • Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces • Ideally, avoid restorations and monitor tooth wear with study casts, photographs, or silicone impressions • Especially in cases of severe progression consider special care that may involve restorations • Repeat at 6–12 month intervals

[a]The cut-off values are based on experience and studies of one of the authors (A.L.) and have to be reconsidered

Table 6.3 Criteria for grading erosive tooth wear by the Tooth Wear Index of Smith and Knight [12]

Score[a]	Surface	Criterion
0	BLOI C	No loss of enamel surface characteristics No change of contour
1	BLOI C	Loss of enamel surface characteristics Minimal loss of contour
2	BLO I C	Enamel loss just exposing dentin <1/3 of the surface Enamel loss just exposing dentin Defect less than 1-mm deep
3	BLO I C	Enamel loss just exposing dentin >1/3 of the surface Enamel loss and substantial dentin loss but no pulp exposure Defect 1–2 mm deep
4	BLO I C	Complete enamel loss, or pulp exposure, or 2° dentin exposure Pulp exposure, or 2° dentin exposure Defect more than 2-mm deep or pulp exposure, or 2° dentin exposure

B buccal or labial, *L* lingual or palatal, *O* occlusal, *I* incisal, *C* cervical
[a]In case of doubt a lower score is given



Upper	8	7	6	5	4	3	2	1		1	2	3	4	5	6	7	8
C	0	0	**4**	**4**	2	0	0	0		0	0	0	0	0	0	0	0
B	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0
O & I	0	**2**	**4**	**4**	**3**	0	0	0		0	0	0	**1**	**1**	0	0	0
L	0	0	**4**	**4**	**2**	0	0	0		0	0	0	0	0	0	0	0

Lower	8	7	6	5	4	3	2	1		1	2	3	4	5	6	7	8
C	0	0	**3**	**3**	**2**	0	0	0		0	0	0	0	0	0	0	0
B	0	0	**4**	**4**	**2**	0	0	**1**		**1**	**1**	0	**1**	**1**	0	0	0
O & I	0	0	0	0	0	0	0	**4**		**4**	**4**	0	0	0	0	0	0
L	0	0	0	0	0	0	0	0		0	0	0	0	0	0	0	0

Fig. 6.4 An illustration of the use of the Tooth Wear Index of Smith and Knight [12] to record and monitor erosive tooth wear. (**a**) Upper and (**b**) Lower dental arches with the charts of the tooth wear indices of their respective teeth

6.3.2 Clinical Photographs

Following the erosion process with clinical photographs is a simple useful, but subjective, method of monitoring erosive tooth wear. However, the dexterity of the photographer and the ambient conditions such as light reflections affects the quality of the product. Hence, photographs would require standardized settings to enable reproducible images. It is a semi-quantitative monitoring technique in that the two-dimensional extension of a given lesion can be quantified using image software tools to detect progression in area and not in depth of the lesion. Many practitioners may be discouraged by the time and effort required for the second step of image analysis to determine progression status, otherwise this can generate a quantitative data to permit quantitative comparison of lesions at different time points, and possible statistical comparative analysis of the erosion progression over time. Although dedicated macro camera systems are the most versatile, and digital intra-oral systems the easiest to use, excellent photographs can be taken with simple cheap equipment available in most camera shops.

6.3.3 Serial Impression Casts

Reference impression casts or study models have been used to monitor erosive tooth wear [13, 14]. They are easy to obtain and allow for a three-dimensional analysis

and visual comparison of lesions at different time points. The practitioner could use the impression casts in four ways to monitor the progression status of erosive lesions. (1) By visual inspection, the original impression cast taken at the initial assessment visit can be macroscopically compared with the patient's teeth at each recall visit. (2) Series of reference impression casts taken at review visits can be compared among each other. (3) If a silicone matrix was originally taken, it can be placed on the latest casts for easier viewing and assessment of lesion progression status. (4) A 3-D digital image of the impression casts can be captured and used to evaluate the area and depth of an erosive lesion by either computer-aided digital image analysis [15, 16] or a reference-free automated 3-D superimposition software [17]. Although sensitive and quantitative, these digital image analysis systems require extensive expensive equipment and are time consuming for a busy practitioner. It is pertinent to caution that impression models are also prone to wear with repeated handling, so they should be cast with die-stone if the practitioner plan to use them for a long term.

References

1. Amaechi BT, Higham SM. Eroded lesion remineralization by saliva as a possible factor in the site-specificity of human dental erosion. Arch Oral Biol. 2001;46:697–703.
2. Amaechi BT, Higham SM. In vitro remineralization of eroded enamel lesions by saliva. J Dent. 2001;29:371–6.
3. Shaw L, Walmsley D, Barclay C, Perryer G, Smith AJ. Tooth wear. Computer assisted learning for general dental practitioners Birmingham. The University of Birmingham; 1999.
4. Huysmans MC, Thijssen JM. Ultrasonic measurement of enamel thickness: a tool for monitoring dental erosion? J Dent. 2000;28(3):187–91.
5. Ślak B, Ambroziak A, Strumban E, Maev RG. Enamel thickness measurement with a high frequency ultrasonic transducer-based hand-held probe for potential application in the dental veneer placing procedure. Acta Bioeng Biomech. 2011;13(1):65–70.
6. Louwerse C, Kjaeldgaard M, Huysmans MC. The reproducibility of ultrasonic enamel thickness measurements: an in vitro study. J Dent. 2004;32:83–9.
7. Wilder-Smith CH, Wilder-smith P, Kaeakami-Wong H, Voronets J, Osann K, Lussi A. Quantification of dental erosions in patients with GERD using optical coherence tomography before and after double-blind, randomized treatment with esomeprazole or placebo. Am J Gastroenterol. 2009;104:2788–95.
8. Ganss C, Klimek J, Lussi A. Accuracy and consistency of the visual diagnosis of exposed dentine on worn occlusal/incisal surfaces. Caries Res. 2006;40:208–12.
9. Bardsley PF. The evolution of tooth wear indices. Clin Oral Investig. 2008;12:S15–9.
10. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008;12:S65–8.
11. Young A, Stenhagen KR, Mulic A, Amaechi BT. Dental erosive wear risk assessment. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 121–32.
12. Smith BGN, Knight JK. An index for measuring the wear of teeth. Br Dent J. 1984; 156:435–8.
13. Wickens JL. Tooth surface loss: prevention and maintenance. Br Dent J. 1999;186:371–6.
14. Chadwick RG, Mitchell HL. Conduct of an algorithm in quantifying simulated palatal surface tooth erosion. J Oral Rehabil. 2001;28:450–6.

15. Mistry M, Grenby TH. Erosion by soft drinks of rat molar teeth assessed by digital image analysis. Caries Res. 1993;27:21–5.
16. Pintado MR, Anderson GC, DeLong R, Douglas WH. Variation in tooth wear in young adults over a two-year period. J Prosthet Dent. 1997;77:313–20.
17. Mehl A, Gloger W, Kunzelmann KH, Hickel R. A new optical 3-D device for the detection of wear. J Dent Res. 1997;76:1799–807.

Dental Erosive Wear Risk Assessment

7

Alix Young, Kjersti Refsholt Stenhagen, Aida Mulic, and Bennett T. Amaechi

Abstract

This chapter presents a newly developed dental erosive wear risk assessment tool designed for use by general dental practitioners, dental educators, and dental students. The tool guides the user through the most common risk factors associated with dental erosive wear, and when combined with the Basic Erosive Wear Examination (BEWE) or other similar scoring system, leads to a final decision on patient risk status. An initial screening section helps the dentist to decide whether it is necessary to perform a more comprehensive dental erosive wear risk assessment. When there is clearly a need for further assessment, all dental erosive wear lesions are then recorded using the index of choice, and the dentist decides how the recorded conditions will be documented. The dentist then uses the *Dental erosive wear risk assessment* (DEWRA) form, where dietary and oral hygiene habits, general health status, and clinical conditions are evaluated using a thorough patient interview. An overall patient risk category is chosen and the section for treatment planning and advice to the patient can then be used. The last part of this chapter provides a brief explanation of the various risk factors that are included in the DEWRA form.

A. Young (✉) • K.R. Stenhagen • A. Mulic
Department of Cariology and Gerodontology, Institute of Clinical Dentistry, Faculty of Dentistry, University of Oslo, Oslo NO-0317, Norway
e-mail: a.y.vik@odont.uio.no; k.r.stenhagen@odont.uio.no; aida.mulic@odont.uio.no

B.T. Amaechi
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_7

7.1 Introduction

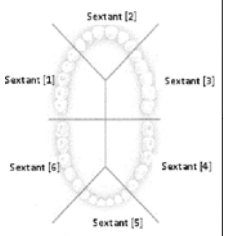
Over the last decades, there has been a growing concern for the increasing prevalence of erosive tooth wear in many populations. Although the overall reduction in the global caries burden in many well-developed societies is undoubtedly partly responsible for the increased focus on dental erosive tooth wear, changes in dietary, social, and oral hygiene habits commonly provide important explanations for a true increased prevalence of this condition.

As is the case for dental caries, restorative treatment options for erosive tooth wear are needed when the condition has progressed so much that there are no other alternatives. It is therefore of great importance to diagnose early stages of erosive tooth wear, in order that appropriate preventive measures can be implemented and the need for extensive restorative treatment be minimized. Although diagnosis of erosive tooth wear requires careful clinical examination, a thorough anamnesis is of even greater importance in patients where the wear is not yet so clinically obvious. Therefore, there is a need for a simple and effective dental erosive wear risk assessment tool that can be performed on patients as part of a routine examination. Dental erosive wear risk may be defined as the probability of an individual developing a certain number of dental erosive wear lesions reaching a given stage of progression during a specified period. Such a risk assessment tool is available for dental caries, and this chapter presents a similar tool designed for the assessment of erosive tooth wear risk.

7.2 Assessment of Erosive Wear Risk Status

The dental erosive wear risk assessment tool presented in this chapter consists of four sections. The first part is the *Screening* section (Table 7.1) that combines relevant questions to the patient, with an overall clinical examination assessing the presence of dental erosive wear lesions by sextants using the Basic Erosive Wear Examination (BEWE). For patients who have signs of dental erosive wear, the second section, *Dental erosive wear recording/monitoring*, is filled out by the clinician following a thorough clinical examination using good lighting and drying of tooth surfaces. The BEWE system can be used or other similar recording system. The dentist also chooses how the dental erosive wear will be documented.

For patients with signs of erosive wear, the third step is then to use the *Dental erosive wear risk assessment* (DEWRA) form (Table 7.2), where the patient's responses to more in-depth history-taking regarding dietary and oral hygiene habits, social, and lifestyle habits, and general health conditions, guide the clinician to choose low, medium, or high risk factor categories. The last part of the DEWRA form uses information from the clinical examination, as well as saliva measurements to choose patient clinical risk categories. The risk level for each risk factor in DEWRA is noted in the right-hand column of the form, and a summary for each patient is written in the open field at the end of the form. An example of a completed DEWRA form is included (Table 7.3).

Screening for erosive tooth wear		
Acid damaged teeth	**Yes**	**No**
Have you previously been informed that you have dental erosion?		
Sensitive teeth	**Yes**	**No**
Do you have any hypersensitive teeth? If yes, how many?		
Basic Erosive Wear Examination (*BEWE*)		
BEWE index criteria: Score 0: No erosive wear Score 1: Initial loss of surface texture Score 2*: Surface loss <50% Score 3*: Surface loss ≥50% (* may involve dentine)	**Sextant diagram** 	
Highest score in sextant: [1]:__ + [2]:__ + [3]:__ + [4]:__ + [5]:__ + [6]:__ = **BEWE score**:_____ (Cumulative score of all sextants: If ≥ 2 a more comprehensive recording should be undertaken for each tooth – See section **Dental erosion recording/monitoring**)		
Other tooth wear types	**Yes**	**No**
Are there other types of tooth wear (abrasion/attrition/NCCL*)? *Non-caries cervical lesion		
If yes: Which type?_____		
Dental erosive wear recording/monitoring		
Scoring of dental erosive wear	**Yes**	**No**
BEWE Index - all tooth surfaces (*see Tooth wear Recording Sheet*)		
Other tooth wear indices (*see Tooth wear Recording Sheet*)		

Recording sheet

Upper	**8**	**7**	**6**	**5**	**4**	**3**	**2**	**1**		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
B																	
O or I																	
P																	

Lower	**8**	**7**	**6**	**5**	**4**	**3**	**2**	**1**		**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
B																	
O or I																	
L																	

Documentation	**Yes**	**No**
Clinical photographs		
Study models		
Silicone matrix		
The **DEWRA form** should be filled out at this stage		

Table 7.1 Screening and recording sheet

Dental erosive wear risk assessment form (DEWRA)				
Patient name: _____	**Birth date:** day/month/year		**Age:**_____	**Dentist initials:** _____
Date: day/month/year	**Low risk (L)**	**Moderate risk (M)**	**High risk (H)**	**Patient Risk (L, M, H)**
Dietary and oral hygiene habits				
Acidic foods (e.g. citrus fruits, apples, sour pickles, salad dressing, sour candies, vit. C fizzy tablets, Chinese candies, Mexican candies, tomato ketchup, sour add-on, etc)	Infrequent consumption. Mainly confined to meal times		Frequent or prolonged between meal consumption.	
Acidic drinks (e.g. fruit juices, soft drinks, squash, flavoured water, sports drinks, energy drinks, herb teas, wine, alcopops, etc)	Infrequent consumption. Mainly confined to meal times. Use of a straw		Frequent between-meal consumption. Rinsing/sipping drinking habit	
Toothbrushing (i.e. toothbrush type, brushing frequency, toothpaste type)	Soft toothbrush and correct brushing technique	Hard toothbrush. High brushing frequency. Incorrect brushing technique. Abrasive toothpaste		
Mouth rinsing (F-mouthrinses)	Regular rinsing with F-mouthrinses	No additional F-exposure than F-toothpaste		
Social and life-style habits				
Alcohol or recreational drugs (e.g. wine, alcopops, narcotics, cocaine, ecstasy, designer drugs)	Occasional user	Regular user		
Occupation/hobbies/exercise	No obvious risk activities	High level of strenuous exercise	Prolonged acidic work environment	
General health conditions				
General medical conditions associated with reduced salivary function (e.g. Sjögrens, rheumatoid arthritis)	No		Yes	
Radiation therapy in head/neck area	No		Yes	
History of vomiting or gastroesophageal reflux disease (GERD)	No	Acidic taste in mouth upon wakening	Yes	
Medications that may affect salivary secretion or oral acid exposure (e.g. hyposalivary medications, sphincter-relaxing medications, acidic medications)	No	Yes		
Clinical conditions				
Dental erosive wear lesions (description of degree of erosive wear)	Erosive wear confined to enamel	Erosive wear - cuppings into dentin	Extensive erosive wear in dentin	
Saliva status (Saliva flow: normal unstimulated = 0.25-0.35 ml/min, hyposalivation <0.1 ml/min; normal stimulated = 1-3 ml/min, hyposalivation <0.7 ml/min Saliva pH: normal stimulated = 7.2-7.5)	Normal saliva secretion levels. Good saliva buffer capacity	Temporary low saliva secretion. Low buffer capacity	Chronic low saliva secretion or hyposalivation. Low buffer capacity	
Comment/summary:			**Overall risk status:** (L, M, H)	

Table 7.2 DEWRA form

Dental erosive wear risk assessment form (DEWRA)				
Patient name: _____X_____	**Birth date:** day/month/year 1 5 1 0 1 9 9 6		Age: 18	**Dentist initials:** _____
Date: day/month/year 1 5 1 0 2 0 1 4	**Low risk (L)**	**Moderate risk (M)**	**High risk (H)**	**Patient Risk (L, M, H)**
Dietary and oral hygiene habits				
Acidic foods (e.g. citrus fruits, apples, sour pickles, salad dressing, sour candies, vit. C fizzy tablets, Chinese candies, Mexican candies, tomato ketchup, sour add-on, etc)	Infrequent consumption. Mainly confined to meal times		Frequent or prolonged between meal consumption.	H
Acidic drinks (e.g. fruit juices, soft drinks, squash, flavoured water, sports drinks, energy drinks, herb teas, wine, alcopops, etc)	Infrequent consumption. Mainly confined to meal times. Use of a straw		Frequent between-meal consumption. Rinsing/sipping drinking habit	H
Toothbrushing (i.e. toothbrush type, brushing frequency, toothpaste type)	Soft toothbrush and correct brushing technique	Hard toothbrush. High brushing frequency. Incorrect brushing technique. Abrasive toothpaste		L
Mouth rinsing (F-mouthrinses)	Regular rinsing with F-mouthrinses	No additional F-exposure than F-toothpaste		M
Social and life-style habits				
Alcohol or recreational drugs (e.g. wine, alcopops, narcotics, cocaine, ecstasy, designer drugs)	Occasional user	Regular user		L
Occupation/hobbies/exercise	No obvious risk activities	High level of strenuous exercise	Prolonged acidic work environment	L
General health conditions				
General medical conditions associated with reduced salivary function (e.g. Sjögrens, rheumatoid arthritis)	No		Yes	L
Radiation therapy in head/neck area	No		Yes	L
History of vomiting or gastroesophageal reflux disease (GERD)	No	Acidic taste in mouth upon wakening	Yes	L
Medications that may affect salivary secretion or oral acid exposure (e.g. hyposalivary medications, sphincter-relaxing medications, acidic medications)	No	Yes		M
Clinical conditions				
Dental erosive wear lesions (description of degree of erosive wear)	Erosive wear confined to enamel	Erosive wear - cuppings into dentin	Extensive erosive wear in dentin	M
Saliva status (Saliva flow: normal unstimulated = 0.25-0.35 ml/min, hyposalivation <0.1 ml/min; normal stimulated = 1-3 ml/min, hyposalivation <0.7 ml/min Saliva pH: normal stimulated = 7.2-7.5)	Normal saliva secretion levels. Good saliva buffer capacity	Temporary low saliva secretion. Low buffer capacity	Chronic low saliva secretion or hyposalivation. Low buffer capacity	M
Comment/summary: Patient X has two 'H' categories which can be modified through good dietary advice, and two 'M' category factors that can also be modified (extra fluoride and salivary stimulation). The overall risk status is therefore recorded as high at this stage. There is a good chance of preventing progression of the erosive wear in the form of cuppings with individualized preventive measures, advice and good patient compliance. Reassessment of risk status after 6 months.			**Overall risk status:** (L, M, H) High	

Table 7.3 Sample of completed DEWRA form

The dentist should now be in a position to categorize the dental erosive wear risk status of the patient following use of the DEWRA form together with a detailed clinical recording of erosive wear lesions using the BEWE or other scoring system. Knowledge of the risk and protective factors as they relate to the individual patient serves both diagnostic and prognostic purposes, and forms the basis for the preventive portion of the treatment plan, while the overall dental erosive wear risk status forms the platform for the integrated, personalized treatment plan for the individual patient. It may be of great use to the dentist in explaining the erosive wear lesions that are present. This is useful because some factors need to be modified in order to reduce risk; exaggerated oral hygiene and frequently consumed acidic drinks or foods are good examples of this. Other factors cannot be modified, and this may affect the preventive management regimen and the prognosis. For instance, a patient with reduced salivary flow due to Sjögrens syndrome is, and will remain, at high risk for dental erosive wear. Furthermore, the dentist might like to make the following prediction: "This patient may develop new erosive wear lesions within the next 12 months unless something is done to prevent it." However, because dental erosive wear is a multifactorial disease, it is probably not surprising that it is difficult to predict accurately. Clinical examination and medical and dental history are the most important sources of information. Thus, a person with no active lesions and no obvious risk factors may safely be designated as currently at low risk. On the other hand, a person with multiple active lesions, and one or more risk factors may be designated as being at moderate or high risk. This means that instead of concentrating on predicting the future, dentists should concentrate on controlling the dental erosive wear lesions their patients have at present. Initial placement of a patient in a high or moderate risk category means that the dentist is responsible for following up the patient's individual preventive treatment plan at recall visits 6–12 months later. Re-assessment of the risk status of the patient needs to be performed at recall visits, and re-adjustment made if necessary [1].

The fourth step involves filling out the *Dental erosive wear preventive treatment plan* (Table 7.4), consisting of a list of the preventive care options from which the clinician can develop a personalized preventive plan for each patient, including also restorative treatment of the erosive wear when necessary. The patient should then receive an individualized letter of advice (*Patient letter*), explaining the results of the risk assessment and the necessary advice for prevention and treatment. An example of such a letter is provided (Table 7.5). As support to the clinician, the final part of this chapter provides a brief explanation of the various risk factors that are included in the DEWRA form with reference to other chapters in this book (Chapters 3 and 4) [2, 5].

Dental erosive wear preventive treatment plan			
Consider the following steps to reduce erosion risk factors and protect eroded surfaces in order to optimize the patient's ongoing oral health *(check the recommended options)*.			
Treatment category	**Details of treatment plan**		**Check here**
Treat underlying medical disorder (*while implementing preventive/control strategies*)	Refer to gastroenterologist for GERD management *(see chapter 12)*		
	Refer to psychiatrist or clinical psychologist for eating disorder management *(see chapter 11)*		
	Refer to physician for other illnesses with vomiting symptoms		
Dental health education/ advice	**Dietary modification**	3-day dietary diary *(see 'Diet Recording Sheet' on page 172-174)*	
		Dietary assessment and analysis *(see page 175-176)*	
		Dietary counseling *(see page 176-179)*	
	Oral hygiene advice/instruction	Non-abrasive toothpaste	
		Soft toothbrush, non-scrubbing method, gentle pressure, brushing duration maximum 2 minutes	
		Avoid toothbrushing immediately after acidic drinks/food – use fluoride mouthrinse to freshen the mouth OR neutralize acid with bicarbonate rinse or antacid tablet/suspension	
	Treat xerostomia/ hyposalivation	Saliva flow stimulation counseling	
		Salivary stimulation products	
		Saliva replacement products	
	Use custom-made poly-ethylene protective 'bite-guards'	Paint inside with antacid suspension	
		Paint inside with fluoride gel	
Protect/treat eroding and/or sensitive tooth surfaces	**Patient-applied products**	Remineralizing/rehardening agent (e.g. toothpastes, mouthrinses, etc) *(see chapter 8)*	
		Desensitizing agents (e.g. toothpastes, mouthrinses, etc) *(see chapter 15)*	
	Dentist-applied products	Remineralizing/rehardening agents (e.g. F-varnish) *(see chapter 8)*	
		Desensitizing agents (e.g. F-varnish, etc.) *(see chapter 15)*	
		Dentin bonding agent	
		Adhesively retained resin composites	
		Fluoridated sealants	
		Porcelain veneers	
Restoration of severely worn surfaces *(see chapter 13)*			
Establish recall visit to review and monitor erosion	Low risk – every 18-24 months		
	Moderate risk – every 6-12 months		
	High risk – every 6 months		
Give a personalized 'Dental erosive wear advice' letter	Summarize personal risk factors *(see Patient letter)*		
	Outline advice in bullet form		

Table 7.4 Preventive treatment plan guide

Dental erosion; information and guidelines - Patient letter (*Example*)

Patient:_____ X _____ **Date:**__15·10·2014_____

Clinical examination:

The examination showed that you have dental erosive wear with loss of enamel on your lower molars· You are considered to be at **moderate** risk of progression of existing erosive lesions and development of new lesions·

Your main contributing erosion risk factors are:

Dietary factors: Orange juice, Coke, citrus fruits and sour candies between meals

Oral hygiene factors:

Medical factors: Anti-depressive medication

Salivary factors: x ml/min; low saliva secretion (induced by your medicines)

You are recommended to:

- Minimize the consumption of: Fruit juice, Coke, sour candies, fruits, in particular between meals
- Use fluoride agents: Fluoride mouth rinse 0·2%, GelKam... 2x/day
- Stimulate saliva flow: Chew sugar free chewing gum after eating/drinking
- Come for a control visit to evaluate the erosive lesions: After 6-12 months

All individuals with dental erosions are advised to:

- Avoid drinking acidic drinks/eating acidic foods between meals
- Avoid sipping acidic drinks over longer periods
- Avoid toothbrushing directly after an acidic impact on the teeth (acidic drinks/food, vomit, reflux)
- Rinse with water directly after an acidic impact on the teeth
- Brush the teeth with a soft toothbrush and low-abrasive toothpaste

Table 7.5 Sample patient letter

7.3 Dental Erosive Wear Risk Factors

7.3.1 Dietary and Oral Hygiene Habits

7.3.1.1 Diet

A thorough dietary analysis is extremely important in patients presenting with dental erosive wear lesions, whether confined only to the enamel or obviously into the dentin. The clinician should not only focus on current dietary habits but ask the patient about previous habits regarding both amount and frequency of intake of acidic dietary foods and drinks [3, 5]. The manner that dietary acids are introduced

into the mouth (e.g., gulping, sipping, use of a straw) and the timing (e.g., with meals versus between meals), frequency and duration of exposure will affect how long the teeth are in contact with the erosive agent, and are therefore of paramount importance. It is also relevant to ask if the patient has a vegetarian diet, since it may often consist of acidic food and also food with high fiber content that can increase erosive tooth wear, in particular raw food diet [4].

7.3.1.2 Oral Hygiene

Exaggerated toothbrushing with abrasive toothpaste can actually contribute to greater erosive wear. In contrast to dental caries, dental plaque is not considered a risk factor for dental erosive wear, rather the presence of plaque along the gingival margins seems to provide protection against non-bacterial acids, as often evidenced by a thin rim of intact enamel in these areas when erosion is present. It is therefore important to gather information about the patient's oral hygiene habits – not only present habits, but previous habits, with regard to toothbrush type (soft, medium, hard), method of brushing (scrubbing technique, other method), frequency of brushing, timing of brushing related to meals (especially acidic foods/drinks) as well as abrasivity and fluoride content of toothpaste [5].

Toothpastes containing sodium or amine fluoride have shown a degree of protection in enamel, but less is known about the effect on dentin [6]. A recent in-situ study has shown that toothbrushing twice daily with fluoridated toothpastes reduces the development of erosive-abrasive lesions in enamel [7]. Various oral hygiene products have been developed in order to reduce dental erosive wear [8]. Toothpastes, rinses, varnishes, and gels that contain high concentrations of acidic and polyvalent fluoride sources would appear to be the most promising at this stage, although novel active agents are being researched.

7.3.2 Social and Lifestyle Habits

7.3.2.1 Alcohol and Recreational Drugs

Typical examples of alcohol and recreational drugs that may be risk factors for dental erosive wear include wine, alcopops, narcotics, cocaine, and ecstasy [5]. An interview with a patient about their use of such products/agents requires tactfulness and considerable care.

7.3.2.2 Occupation/Hobbies/Exercise

Dental erosive wear occurs in persons working in battery, galvanizing, fertilizer, or plating factories despite occupational health and safety measures. Persons working in pharmaceutical industry and as wine tasters may be at increased risk of developing dental erosive wear [5].

Rapid loss of dental hard tissue have been reported in competitive swimmers due to non-buffered chlorinated swimming pool water [9]; however, an additional risk may have been a large consumption of acidic sports drinks. The risk is assumed to be minimal when the pool water is well buffered and pH controlled.

Frequent high intensity physical exercise leads to dehydration and thereby a higher consumption of acidic sport drinks. However, for most athletes sports drinks provide little benefit compared to water. Some sport activities, such as swimming and weight-lifting, may also increase gastroesophageal reflux [10].

7.3.3 Medical History and Medications

Some patients may be at higher risk for developing dental erosive wear due to medical conditions (e.g., Sjögrens syndrome, rheumatoid arthritis), due to the long-term use of medications (e.g., hyposalivary medications, acidic/sphincter-relaxing medications), or due to methods of treatment for these conditions (e.g., radiation therapy). A patient's medical history can change drastically between control appointments and new risk factors may arise requiring the clinician to advise and assist the patient with the potential dental consequences.

Contact of the teeth with the acidic stomach contents due to vomiting or regurgitation is not unusual in some conditions (e.g., reflux disease, eating disorders, chronic alcoholism, and pregnancy), and can also be a side-effect of taking various medications and of certain treatments (e.g., chemotherapy) [2]. If the patient is not aware of such problems, there is still a possibility that reflux is present, since the prevalence of asymptomatic silent reflux is high [11, 12]. The patient should therefore be carefully interviewed (e.g., stomach discomfort, throat-burn, acidic taste in the mouth on waking, etc.). The frequency and duration of the vomiting/reflux, and how long it has lasted will determine the extent of the condition, though palatal lesions are typical. Studies indicate that gastric acids have the potential to induce moderate-to-severe erosion [2, 12].

7.3.4 Clinical Conditions

7.3.4.1 Saliva Status

Saliva is considered to be the most relevant biological factor influencing dental erosion. Saliva is involved in the dilution, clearing, neutralizing, and buffering of acids, plays an important role in the formation of the enamel pellicle, and both reduces demineralization and enhances remineralization of the tooth mineral [13, 14]. A dry mouth can predispose for dental erosive wear, and hyposalivation is one of the main risk factors for erosive tooth wear. The four most common causes of dry mouth are medications (e.g., antidepressants, anti-psychotics, tranquilizers, antihypertensives, and diuretics), Sjögrens syndrome (affects the salivary and lacrimal glands, leading to a dry mouth and dry eyes, and rheumatoid arthritis may indicate the presence of this disorder), eating disorders (may induce hyposalivation if the condition persists over longer periods, and combined with an acidic diet can lead to extensive erosive wear), and radiation therapy in the region of the salivary glands (e.g., for head and neck malignancy).

Table 7.6 Reference values for saliva secretion in adults

Reference values for saliva secretion			
	Saliva flow rate (ml/min)		
Saliva type	Normal	Low	Hyposalivation
Unstimulated whole saliva	0.25–0.3	0.1–0.25	<0.10
Chewing stimulated whole saliva (normal pH 7.2–7.5)	1.00–3.00	0.7–1.0	<0.50–0.70

Important questions to ask the patient include the following: Does your mouth feel dry when you sleep/eat/swallow food? What relieves your dry mouth? Do you always have a glass of water by your bedside at night?

Measurement of salivary flow and of buffer capacity of stimulated saliva is important because a low flow rate and low buffer capacity may help to explain erosive wear. Saliva analyses should to be performed under calibrated conditions so that measurements can be repeated and compared at suitable intervals (see Table 7.6 with reference values for adult patients).

Conclusion

The prevalence of dental erosive wear is assumed to be increasing. Early diagnosis of the condition, as well as identification of individuals with the highest risk, is becoming important. In order to identify these patients, a screening with relevant questions and a thorough overall clinical examination should be performed. When risk patients are identified, the newly developed Dental Erosive Wear Risk Assessment (DEWRA) form is suggested to further investigate the risk factors in more detail, and together with the clinical diagnosis of the condition, the patient risk category may be decided.

It is important to keep in mind that not all dental erosive wear risk factors have the same impact for the patient. Chronic hyposalivation (e.g., Sjögrens syndrome) and unresolved eating disturbances and GERD are factors that place the patient at high risk despite lack of other contributing risk factors.

In order to provide the adequate preventive and, when necessary, restorative treatment for the patients at risk, it is important to relate the findings to each individual, as the condition and treatment need will require different follow-up from one individual to another. In the end, it is important to emphasize that the dental health personnel's advice can only be beneficial to patients if they comply with the suggested measures.

References

1. Amaechi BT. Recall, maintenance care cycle and outcomes assessment. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 303–11.

2. Scaramucci T, Carvalho JC, Hara AT, Zero DT. Causes of dental erosion: intrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 35–67.
3. Gross GS, Amaechi BT. Prevention and control of dental erosion: dietary management. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 169–79.
4. Ganss C, Schlechtriemen M, Klimek J. Dental erosions in subjects living on a raw food diet. Caries Res. 1999;33:74–80.
5. Hara AT, Carvalho JC, Zero DT. Causes of dental erosion: extrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 69–96.
6. Ganss C, Schulze K, Schlueter N. Toothpaste and erosion. In: van Loveren C, editor. Toothpastes. Monogr Oral Sci. 2013;23:88–99.
7. Hara AT, Barlow AP, Eckert GJ, Zero DT. Novel in-situ longitudinal model for the study of dentifrices on dental erosion-abrasion. Eur J Oral Sci. 2014;122:161–7.
8. Buzalaf MAR, Cardoso CAB, Magalhães AC. Prevention and control of dental erosion: patient self-care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 133–50.
9. Dawes C, Boroditsky CL. Rapid and severe tooth erosion from swimming in an improperly chlorinated pool: case report. J Can Dent Assoc. 2008;74:359–61.
10. Clark CS, Kraus BB, Sinclair J, Castell DO. Gastroesophageal reflux induced by exercise in healthy volunteers. JAMA. 1989;261:3599–601.
11. Fass R, Dickman R. Clinical consequences of silent gastroesophageal reflux disease. Curr Gastroenterol Rep Rev. 2006;8:195–201.
12. Patel A, Brady III CE, Amaechi BT. Prevention and control of dental erosion: gastroesophageal reflux disease management. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 203–24.
13. Dodds MW, Johnson DA, Yeh CK. Health benefits of saliva: a review. J Dent. 2005;33:223–33.
14. Hara AT, Ando M, González-Cabezas C, Cury JA, Serra MC, Zero DT. Protective effect of the dental pellicle against erosive challenges in situ. J Dent Res. 2006;85:612–6.

Prevention and Control of Dental Erosion: Patient Self-Care

8

Marília Afonso Rabelo Buzalaf, Cristiane de Almeida Baldini Cardoso, Ana Carolina Magalhães, and Bennett T. Amaechi

Abstract

This chapter presents new insights into the self-applied preventive strategies, comprising of both home-use products and recommended behavioral modifications to prevent dental erosion. It important to emphasize that these products should be delivered with counselling by motivational interview. Dental erosion is a multifactorial condition dependent on the interaction of chemical, biological and behavioral factors. Preventive measures are established according to the causal factors, which may include the dietary intervention, modification of acidic drinks and behavioral changes, in order to reduce the contact between acid and teeth. The modification of the tooth surface, by increasing its resistance against acidic attacks, is one of the most studied strategies. Many available active agents as fluoride, polyvalent metal cations, calcium phosphates in different forms, proteins, protease inhibitors and biopolymers (as chitosan) have been demonstrated to have some protective effect against erosion. Amongst them, the daily application of fluoride, especially those containing titanium or tin (as dentifrice or mouthrinse), has shown the best effect in reducing dental erosion. However, there is need for further clinical trials to better establish the protocols for the use of some of these agents.

M.A.R. Buzalaf (✉) • C. de A.B. Cardoso • A.C. Magalhães
Department of Biological Sciences, Bauru School of Dentistry, University of São Paulo, Al. Octávio Pinheiro Brisolla, 9-75, Bauru, SP 17012-901, Brazil
e-mail: mbuzalaf@fob.usp.br; crisbaldini@usp.br; acm@usp.br

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_8

8.1 Introduction

Dental erosion is a dental lesion caused by exposure to non-bacterial acids, which has received attention from researchers and clinicians by its increasing prevalence and early clinical diagnosis [1, 2]. Dental erosion presents two distinct phases classified as "early erosion" (initial phase), in which there is only softening of the tooth surface and "advance erosion" (advanced phase), with tooth surface loss due to successive erosive attacks with the resultant lesion having a softened surface [2, 3]. The softened layer presents low resistance to chemical challenges and mechanical forces (abrasion and attrition) [4–6]. Attrition is defined as wear due to direct tooth-to-tooth contact, while abrasion occurs due to the presence of particles in movement and contact with the tooth surface as in dentifrice and toothbrush [7].

The aetiology of dental erosion is multifactorial involving chemical, biological and behavioral factors. The multifactorial aetiology helps to explain the difference in erosion susceptibility and activity between people exposed to similar erosive environment [8]. Intrinsic and extrinsic acids are considered the main factors involved in the aetiology of erosion [9]. The extrinsic acids are derived from the diet (acids from food and drinks) and occupation, such as frequent exposure to swimming pool chlorine and sulphuric acid gas from battery industries [10]. The erosive potential of drinks or acidic foods depends on chemical factors such as type of acid, pH, titratable acidity, mineral content, viscosity, clearance and on calcium-chelation properties [9]. Extrinsic acids from the diet are becoming the most important source of erosive attacks due to increasing consumption of acidic drinks [10]. On the other hand, intrinsic acid is originated from the stomach in patients with regurgitation, reflux and psychosomatic disorders [11]. Thus patients with eating disorders are classified as a high-risk group for dental erosion [12].

Salivary properties and tooth position as well as mineralization level forms part of the biological factors. Low salivary flow and buffer capacity may increase the risk for erosion [13, 14]. Furthermore, the formation of the salivary pellicle has shown to reduce the erosion development and progression [15–17]. This has stimulated researchers to investigate the composition of the salivary pellicle (peptides, proteins and fat) and its relationship with erosion susceptibility [18, 19].

Finally, behavioral factors, including frequency and mode of patient exposure to different acids sources, such as bulimic practices and oral hygiene habits are important factors contributing to erosive tooth wear [8, 20].

It is always a challenge to change individual's behavior and lifestyle, hence strategies to increase the tooth resistance to acidic challenge is of utmost importance. Fluoride products, applied via highly concentrated varnishes and gels, have been the major professionally applied methods for protection of teeth against erosion [21]. However, patient's access to professional treatment is limited by its cost and requirement for dental office visit. Therefore, the use of self-applied preventive products might be of higher benefit for patients at high risk for dental erosion.

8.2 Preventive Strategies Based on Lifestyle and Behavioral Modification

The following advice and recommendations should be delivered through motivational interview and counselling in order to ensure success [22]. The counselling should always be repeated at subsequent recall and monitoring visits [23].

8.2.1 Change in Frequency and Method of Beverage Intake

Preventive measures for dental erosion, including chemical, biological and behavioral factors, should be specific according to the patient's history [24, 25]. Clearly, a reduction in acid exposure would be the best preventive strategy to be applied in high-risk patients. However, it is the most difficult approach since it involves lifestyle and behavioral changes. The consumption of potentially erosive foods and beverages, for example, should be limited to main meals only [24, 25]. Some habits, like drinking and holding in the mouth as well as nipping from a bottle, should be avoided, because they prolong the contact time of acid with the tooth, increasing the susceptibility to erosion [8, 24, 26, 27]. The straw, when appropriately used, might be a viable alternative to reduce the contact of the acid with the teeth [28, 29]. The temperature of an acidic drink also influences its erosive potential. Taking the drink ice-cold reduces its erosive effect [30, 31].

8.2.2 Change in Oral Hygiene Method and Materials

The time of toothbrushing after an erosive attack as well as the applied force and type of dentifrice used should be controlled [8]. Conviser et al. [32] reported that 32.5 % of sample of bulimic patients have the habit of brushing their teeth immediately after vomiting and this should be contraindicated because the abrasive forces can increase erosive wear [33, 34, 35]. Thus, an important recommendation is to avoid brushing the teeth immediately after episode of acidic challenge such as vomiting or reflux or intake of acidic beverage. Rather patients should be advised to rinse their mouth with water or, more effectively, to use antacid products or fluoridated mouthrinse immediately after acidic challenge. Although some authors suggested advising individuals at high risk for erosion to wait at least 60 min after acidic challenge before brushing their teeth [4–6, 24], it may be difficult to gain the compliance of the patient with this advice.

The abrasive level of toothpaste is an important aspect to be considered, as it may lead to dental erosion progression, since highly abrasive toothpastes may facilitate the disruption of the acquired dental pellicle as well as abrade away dental surfaces previously softened by an erosive challenge. Although substantial variation is found among different brands, the abrasive level of toothpastes can be roughly estimated according to their types. Data from a standard abrasive test for toothpastes shows that whitening toothpastes would be on the higher abrasive end, while regular or

most of the anti-sensitivity toothpastes would be on the medium-lower end. The latter should be recommended for patients with high risk for erosion, especially if exposed dentin surfaces are present.

Toothbrushes are classified as soft, medium and hard based on the stiffness of the filaments of their bristle. It may be recommended for patients at high risk for erosion soft-bristle toothbrushes, especially if exposed dentin surfaces are present. The effect of toothbrushing abrasion has been shown for sound dental hard tissues [36, 37], and more pronouncedly, for eroded enamel [38]. This effect is mainly influenced by the abrasivity of the dentifrice and, to a lesser extent, by the toothbrush hardness [39, 40]. Toothbrush characteristics such as type, filament stiffness, and filament end-rounding can also modulate the abrasivity of the dentifrice [39, 40]. Hooper et al. [41] showed that abrasion of eroded dentin increased along with the RDA value of the dentifrice, which is in accordance with other in vitro studies showing an increased enamel and dentin abrasion with increasing REA (relative enamel abrasion) or RDA (relative dentin abrasion) values, respectively [39, 40, 42].

8.2.3 Use of Personal Protective Equipment

Personal protective equipment (e.g. respiratory masks for industrial workers or 'bite-guards' for professional swimmers) and adherence to threshold limit values recommended by occupational health legislations are considered an important preventive strategy to decrease occupational exposure to erosive acids [43].

It is pertinent to mention that the bite guards suggested for professional swimmers should have the inside (tooth surface) coated with a small amount of sodium bicarbonate powder or milk of magnesia to neutralize any acidic water pooling in it. The guard should have occlusal coverage only, so that saliva flow to aid remineralization is maintained.

8.2.4 Control of Exposure to Intrinsic Acids

Management of erosive tooth wear in patients suffering from organic or psychosomatic disorders such as gastro-oesophageal reflux disease (GERD) or eating disorder (alcohol abuse, anorexia or bulimia nervosa) requires a multidisciplinary intervention, including general medicine and psychological treatment, in order to decrease the exposure to intrinsic acid [12, 44]. This is discussed in detail in Chaps. 11 and 12 of this book [33, 34].

8.2.5 Saliva Stimulation

Erosion may be associated with biological factors such as low salivary buffering capacity and flow [13, 45]. Changes in saliva flow and quality may be caused by

head and neck radiation, use of some medications, as well as GERD [46, 47]. Considering that saliva has important functions (buffer capacity and remineralizing effect), salivary stimulation through the use of chewing gum, especially those containing remineralizing agents as calcium phosphates [5, 48] and the consumption of cheese or milk after meal [49, 50] can help to reduce the erosion progression. Saliva is also able to promote the formation of the acquired pellicle. The pellicle is a diffusion barrier that reduces the contact between acid and tooth surface [15–17].

8.2.6 Use of Modified Products

The modification of beverages or foods is another preventive strategy to reduce the risk of dental erosion. Reduction of the erosive potential of acidic beverages can be achieved by adding ions (calcium, phosphate and/or fluoride) that make the beverage more saturated in respect to tooth mineral, hydroxyapatite (HA) [51, 52] or by adding polymers (pectin, alginate and gum arabic polymers), which adsorb to the tooth surface to create physical barrier against acid erosion [53, 54]. The addition of calcium or polymer has been shown to reduce the erosive potential of acidic drinks [51–54]. However, future studies should give special emphasis on the consequences of the modification regarding taste and tooth staining, stability of the solution, and systemic effects for the patients.

8.3 Preventive Strategies Based on the Self-Applied Preventive Agents

Commercially available home-use products for prevention and control of erosion are shown in Table 8.1.

8.3.1 Neutralizing Intraoral Acidity

In attempt to raise intra-oral pH, different products have been tested, including antacid tablets, gum arabic lozenges, mineral water, milk and tap water, all used for 2 min immediately after the erosive challenge [55]. All of these products were found to increase intra-oral pH when compared to the negative control (where no treatment was performed); however, the antacid tablet caused the greatest and most rapid increase in intra-oral pH. The use of different antacid suspensions and a bicarbonate solution after erosive challenge with hydrochloric acid also significantly reduced enamel surface loss [56, 57]. Thus, it is advisable to instruct the patients to rinse their mouth with water or, more effectively, to use antacid products immediately after vomiting or reflux episodes.

Table 8.1 Commercially available self-applied products for prevention and control of dental erosion

Generic product	Brand name	Active ingredients	Company
Toothpastes and gels containing polyvalent metal fluorides	Sensodyne *Repair & Protect*™ Sensodyne *Complete Protection*™	SnF_2 0.454 % 0.15 % w/v fluoride ion (pH 5.7)	GlaxoSmithKline (USA)
	Crest *Pro-Health*	SnF_2 0.454 % 0.15% w/v fluoride ion (pH 5.7)	Procter & Gamble (USA)
	GelKam®	970 ppm F^-, 3,030 ppm Sn^{2+} (0.4 % SnF_2) Gel (pH 4.0)	Colgate Oral Pharmaceuticals (USA)
	Erosion Protection®	1,400 ppm F^- (700 ppm F^- from amine fluoride, 700 ppm F^- from NaF), 3,500 ppm Sn^{2+} (0.462 % $SnCl_2$) Chitosan (0.5 %) (pH 4.5)	GABA Int. AG (Switzerland)
	Erosion Protection®	800 ppm Sn^{2+} (0.105 % $SnCl_2$) 500 ppm F^- (125 ppm F^- from Amine F^-, 375 ppm F^- from NaF) (pH 4.5)	GABA Int. AG (Switzerland)
Fluoride containing toothpastes	Sensodyne® *ProNamel*®	0.15 % fluoride ion from sodium fluoride (NaF) Potassium nitrate 5 %	GlaxoSmithKline (USA)
Novamin Technology	Sensodyne *Repair & Protect*™ Sensodyne *Complete Protection*™	Bioactive glass, 15 % calcium sodium phosphosilicate (CSPS), 1,450 ppm F^- as sodium monofluorophosphate (pH 7.0)	GlaxoSmithKline (UK, Canada)
Tricalcium Phosphate Technology	Clinpro™ 5000 toothpaste	Functionalized tricalcium phosphate (TCP), 5,000 ppm $F^-$from NaF Toothpaste (pH 7.0)	3M-ESPE Inc. (USA)
	Clinpro tooth creme	TCP, 850–950 ppm F^- from NaF (pH 7.0)	3M-ESPE Inc. (Asia/ Australia)

Table 8.1 (continued)

Generic product	Brand name	Active ingredients	Company
Recaldent (CPP – ACP) Technology	Tooth Mouse (Asia/Australia) Or MI paste (USA)	Casein phosphopeptide amorphous calcium phosphate (CPP-ACP) Creme (pH 7.0)	GC (USA/Asia/Australia)
	Tooth Mouse plus (Asia/Australia) Or MI paste plus (USA)	Casein phosphopeptide amorphous calcium fluorophosphate (CPP-ACFP), with 900 ppm F^- from NaF Creme (pH 7.0)	GC (USA/Asia/Australia)
Chitosan-containing toothpastes	Chitodent®	Chitosan/chitin 0.5 % (pH 6.0)	Chitodent Vertrieb GmbH (Germany)

8.3.2 Use of Dentifrices Containing Polyvalent Metal Fluorides

Aiming to protect tooth surfaces against acidic challenges, the application of fluoride and polyvalent metal fluoride compounds has been suggested. However, studies have shown that the type of fluoride compound seems to be relevant. Improved enamel protection was observed with dentifrices containing titanium tetrafluoride (TiF_4) and stannous fluoride (SnF_2) when compared to NaF [21, 58–66]. Regardless of the fluoride concentration (1,100 ppm F, 1,450 ppm F or 5,000 ppm F), NaF dentifrices did not show ability to protect the enamel against simulated erosive challenges [65, 67]. The improved protection by SnF_2 and TiF_4 was attributed to the stannous' and titanium's ability to interact with the tooth surfaces forming an acid-resistant film of insoluble compounds, thus increasing the tooth tissue resistance [59, 68–70]. These compounds also demonstrated precipitation of CaF_2-like deposits (CaF_2-globules) that behave as a physical barrier inhibiting the contact of the acid with enamel as well as acts as a fluoride reservoir [71, 72]. The formation of CaF_2 reservoir is known to be increased under acidic conditions compared to neutral conditions [73] and is highly dependent on the concentration of fluoride and frequency of application as well [74]. Therefore, for patients susceptible to erosion, additional measures to the daily use of conventional fluoridated toothpastes should be implemented, with best evidence for acidic formulations containing polyvalent metal fluorides.

For dentin, fluoride/stannous-containing dentifrices (1,100–1,400 ppm F) were able to significantly reduce dentin wear, whereas an amine fluoride (AmF) dentifrice (1,400 ppm F) was not [75]. Recently, Comar et al. [76] compared the effect of dentifrices containing TiF_4, NaF, and SnF_2 on tooth erosion-abrasion and the results showed a significant reduction in enamel and dentin wear (64–70 %) for TiF_4 and SnF_2 compared to placebo in vitro.

8.3.3 Use of High Fluoride Concentration Dentifrices

The efficacy of a NaF dentifrice in erosion prevention seems not to increase along with the fluoride concentration and the reduction of wear seems to be less than 30 % for this fluoride vehicle compared to placebo/control [77, 21]. An in situ study showed that 5,000 ppm F and 1,100 ppm F dentifrices reduced erosive and erosive-abrasive dentin wear by approximately 27.5 % compared to the placebo dentifrice, but their efficacies were not significantly different [78]. Also for enamel wear, no significant differences were found between 1,100 and 5,000 ppm F dentifrices [79]. On the other hand, Ren et al. [80] demonstrated an increase in the protection against enamel erosion (around 55 %), when a dentifrice containing 5,000 ppm (NaF) was compared to a dentifrice containing 1,450 ppm F (NaF) in situ.

On the other hand, low-fluoride dentifrices supplemented with trimetaphosphate (3 %TMP and 500 ppm F) showed inhibition of tooth wear, and, under in vitro conditions, were able to significantly reduce enamel erosion and erosion/abrasion when compared to a 1,100 ppm F dentifrice, not differing from a 5,000 ppm F dentifrice [81].

8.3.4 Use of Paste/Cream Containing Recaldent (CPP-ACP) Technology

Casein phosphopeptide amorphous calcium phosphate (CPP-ACP) incorporated into patient self-applied pastes and cream (GC corporation) has been demonstrated in series of studies to promote the remineralization of enamel and dentin [44, 82–90]. These are commercially available as Tooth Mousse (Asia/Australia) and MI paste (USA) and the fluoride-containing CPP-ACFP (with 900 ppm fluoride) as Tooth Mousse-plus and MI paste-plus. In Recaldent, the calcium and phosphate ions in a soluble amorphous calcium phosphate is stabilized by the protein CPP into nanocomplexes, thus preventing precipitation during storage within the dispensing tube [91]. Following intraoral application, these nanocomplexes bind onto the tooth surfaces and dental pellicle to create a state of supersaturation of calcium and phosphate ions in the oral cavity. When the oral pH drops during an acidic challenge, the calcium is released from the CPP to provide high level of bioavailable calcium and phosphate ions to facilitate remineralization and inhibit demineralization [89]. CPP-ACP products that include fluoride would be the preferred option for erosion. Follow the manufacturer's direction for application. However, there is a need of further studies to compare their erosive protective effect with other products such as polyvalent metal fluorides.

8.3.5 Use of Toothpaste Containing Functionalized Tricalcium Phosphate Technology

This technology is tailored to enhance remineralization by providing a method where calcium, phosphate and fluoride ions are made bioavailable in the oral environment at the same time. In this technology, by milling tricalcium phosphate

(TCP) with organic materials (functionalization), the calcium oxides in TCP become 'protected' by the organic materials, thus allowing the calcium and phosphate ions of the TCP to co-exist with fluoride ions in an aqueous dentifrice base (toothpaste) without premature TCP-fluoride interactions [92]. Once applied in the presence of saliva, calcium compound is activated by saliva that degrades the protective coating, releasing calcium at the tooth surface, resulting in high fluoride and calcium bioavailability on the lesion surface and subsequent diffusion into the lesion to promote remineralization [93, 94]. Products containing functionalized tricalcium phosphate technology are commercially available from 3 M-ESPE Inc., as self-applied toothpastes, Clinpro™ 5000 with 5,000 ppm fluoride (USA) and Clinpro tooth crème with 850–950 ppm fluoride (Asia/Australia). Follow the manufacturer's direction for application. However, there is a need of further studies to compare their erosive protective effect with other products such as polyvalent metal fluorides.

8.3.6 Use of Toothpaste Containing Novamin Technology

Novamin technology is incorporated in numerous products tailored chiefly to relieve dentin hypersensitivity. It is a bio-active glass (calcium sodium phosphosilicate) that binds to the tooth surfaces, and when in contact with body fluid, such as saliva, releases calcium and phosphate ions, enabling the remineralization of tooth tissue, typically forming hydroxycarbonate apatite [95]. The existing Bioactive glass (Novamin™) used in commercial toothpastes such as Sensodyne Repair & Protect and Sensodyne Complete Protection (GlaxoSmithKline, UK) does not contain fluoride, rather sodium monofluorophosphate is added paving way for possible premature formation of CaF_2 [96]. Recent innovation incorporated fluoride, strontium, potassium and zinc within the glass itself, thus enabling the delivery of Ca^{2+}, PO_4^{3-} and F^- ions simultaneously in the appropriate amounts to form fluoroapatite that is more chemically stable against acid attack [97]. The fluoride-containing bioactive glass (F-BG), available in Europe, is engineered to release fluoride over a 12-h period within the oral environment. However, there is a need of further studies to compare their erosive protective effect with other products such as polyvalent metal fluorides.

8.3.7 Use of Polymer-Containing Toothpastes

Recently, some dentifrices containing polymers have been investigated due to their potential to form a protective layer on the tooth surface, strengthening the pellicle [77]. As active ingredients in toothpaste, organic polymers such as casein, ovalbumin, pectin, alginate and arabic gum, and inorganic polymers such as pyrophosphate, tripolyphosphate and polyphosphate have been studied [64]. Some of them are common dentifrice ingredients, such as carboxymethylcellulose, hydroxyethylcellulose and polyethylene glycol.

8.3.8 Use of Chitosan-Containing Toothpastes

Chitosan is a cationic polysaccharide obtained by the deacetylation of chitin and has been used as active ingredient in fluoride-free dentifrice to inhibit erosion and erosion/abrasion [63]. Incorporation of chitosan into dentifrices containing fluoride and tin or Sn significantly increased the anti-erosive/anti-abrasive effect of the dentifrice for both enamel and dentin [64, 98]. Chitosan might be capable of adsorbing to solid structures with negative zeta potentials such as enamel [99], and this adsorbed layer is strikingly persistent under pH cycling conditions and under physical impacts [64]. Furthermore, chitosan seems to be able to enhance the efficacy of the Sn^{+2}containing dentifrice, acting as an anti-erosive/anti-abrasive agent. Immersion in each Sn^{+2}-containing suspension significantly reduced tissue loss. However, after immersion in suspension + brushing, only the treatments with GelKam (3,000 ppm Sn^{+2}, 1,000 ppm F^{-}) and with F/Sn/chitosan (1,400 ppm F, 3,500 ppm Sn and 0.5 % chitosan) significantly reduced loss compared to placebo and F/Sn dentifrices [100].

Table 8.2 summarizes the results of some studies about the effect of dentifrices in the control of tooth wear.

8.3.9 Use of Acidulated Fluoridated Mouthrinses

Mouthrinses have the advantage of simplicity of use and can be formulated to possess a refreshing flavour so as to be used to enhance salivary remineralization after acidic challenges in patients. Fluoride compounds such as polyvalent metal fluorides that might have higher efficacy at lower concentrations to allow daily application have been the focus of new formulations. Wiegand et al. [101] observed that acidic solutions of AmF and SnF_2 with the same fluoride concentration (10,000 ppm F) were similarly more effective than a NaF solution against enamel erosion. Yu et al. [102] reported that a single application of a NaF/$SnCl_2$ solution (500 ppm F and 800 ppm Sn) reduced enamel and dentin erosion up to 6 and 3.5 min, respectively, of constant acid flow, as analysed by calcium released into the acid. In this study, solution of NaF (500 ppm F) alone did not have a significant impact on the progression of enamel and dentin erosion [102]. While titanium tetrafluoride (TiF_4) solution (approximately 9,000 ppm F) at pH 1.2 was able to significantly reduce erosive mineral loss [103] in enamel, dentin erosion was reduced by NaF and TiF_4 solutions of similar fluoride concentration (approximately 9,000 ppm F) and pH 1.2 with no difference between them [104]. TiF_4 has been shown to provide a more acid-resistant layer when compared to SnF_2 solution [58, 60]. This protective layer is related not only to an increased fluoride uptake but also to the formation of new compounds (hydrated hydrogen titanium phosphate and titanium dioxide) [21]. However, the low pH of TiF_4 products does not allow their use as patient-applied, due to the possible cytotoxic effect on fibroblasts [105]. Therefore, this formulation should be improved to allow self-application.

Table 8.2 Summary of the active principle and main results of the dentifrices against erosion and erosion/abrasion

Dentifrice	Substrate	Main results	References
NaF-containing dentifrices	Enamel and dentin	Fluoridated dentifrices have a limited effect (30 % reduction). The efficacy of a NaF dentifrice does not seem to increase along with the F concentration.	[21, 77–79]
Fluoride containing polyvalent metals (titanium and tin) dentifrices	Enamel	These compounds are promising in inhibiting dental erosion effects (around 55 %) rather than erosion associated with abrasion in vitro *and* in vivo.	[21, 62, 66, 69, 76]
Fluoride containing polyvalent metals (titanium and tin) dentifrices	Dentin	SnF_2, SnF_2/NaF and TiF_4/NaF dentifrices with low abrasivity significantly reduced dentin wear (64–79 %) in vitro.	[76]
Low-fluoride dentifrices supplemented with TMP	Enamel	They significantly reduce enamel erosion and erosion/abrasion compared to 1,100 ppm F dentifrice, not differing from a 5,000 ppm F in vitro.	[81]
Dentifrices containing nano-HA	Enamel	Do not seem to be more effective than conventional fluoride dentifrices.	[69]
Dentifrices containing casein phosphopeptide associate with amorphous calcium phosphate (CPP-ACP)	Enamel	They slightly decrease dental erosion (30–35 %) compared to control/placebo in vitro. When applied during brushing, the protective effect was improved (63–79 % reduction).	[44, 84, 86]
Dentifrices containing polymers – chitosan (polysaccharide)	Enamel and dentin	When this polymer is added to dentifrices containing tin and fluoride, it can significantly increase the effectiveness of the product in vitro and in situ.	[64, 98, 100]

The anti-erosion efficacy of low concentrated TiF_4 mouthrinse was evaluated in a two phase study [106]. In the first phase, a commercially available anti-erosion mouthrinse, Erosion Protection® ($SnCl_2$/NaF/AmF), applied twice daily for 1 min on each occasion was compared with low concentrated TiF_4 solutions (500 ppm F^-, pH 2.5) using an in vitro model. In the second phase, combinations of TiF_4 and NaF (pH 4.5) with higher native pH values was compared with TiF_4 (pH 2.5) alone. In both studies, the best anti-erosive effect was still obtained with TiF_4 (pH 2.5) solution (99 % reduction of enamel wear), followed by Erosion Protection® (78 % reduction) and then a specific combination of NaF and TiF_4 solution (41 % reduction). Although combination of NaF + TiF_4 increased the pH, its effect against enamel erosion was reduced compared to TiF_4 alone, as expected, probably due to the reduction of titanium precipitation on enamel [106].

8.3.10 Use of Mouthrinse Containing Polyvalent Metal Fluorides

Promising results were obtained with tin-containing fluoride solutions, able to deposit metal compounds [$Ca(SnF_3)_2$, $SnOHPO_4$, $Sn_3F_3PO_4$], which has been shown to have higher acid resistance than particles of CaF_2 in situ [107–110]. Combination of AmF/NaF/$SnCl_2$ (2,800 ppm Sn^{+2}, 500 ppm F^- solution) was shown to provide 80 % of erosive wear inhibition in vitro. A non-significant difference found among different Sn^{+2} concentrations, ranging from 800 to 2,800 ppm, may mean that a low concentration of tin can be used without loss of efficacy of tin fluoride solution [107]. Concentration of tin in a mouthrinse is of high clinical importance since a higher concentration induces a dull feeling on tooth surface, astringent sensation and tooth discoloration [21]. A fluoride mouthrinse (Erosion Protection®; GABA Int. AG, Switzerland) with low tin concentration (800 ppm Sn^{+2} as $SnCl_2$, 500 ppm F^- as AmF and NaF) is commercially available in Europe and has been shown to reduce substance loss by 67 % in enamel and 47 % in dentin, being significantly more effective than NaF, when applied once daily for 30 s under severe erosive conditions [111] in situ. Interestingly, the anti-erosion effect of $SnCl_2$/AmF/NaF mouthrinse can remain stable on dentin, regardless of the presence or absence of the demineralized organic matrix (DOM), while the effect of NaF mouthrinse is lost when DOM is removed, showing promissing effect of the former from the clinical point of view [111].

Table 8.3 Summary of the active principle and main results of the mouthrinses against erosion and erosion/abrasion

Mouthrinse	Substrate	Main results	References
Fluoride containing polyvalent metals (tin) mouthrinses	Enamel and dentin	AmF/NaF/$SnCl_2$ solution was shown to provide 80 % of inhibition of erosive wear in vitro and also to reduce substance loss by 67 % in enamel and 47 % in dentin at a lower concentration (Erosion Protection®) in situ, being significantly more effective than NaF.	[21, 107–110]
Fluoride containing polyvalent metals (titanium) mouthrinses	Enamel	TiF_4 has been shown to provide a more acid-resistant layer when compared to SnF_2 solution. A low concentrated TiF_4 solution has shown to have the best anti-erosive effect (99 % reduction of enamel wear) when compared to Erosion Protection® (78 % reduction), followed by a specific combination of NaF and TiF_4 solution (41 % reduction) compared to control and NaF only.	[21, 58, 60, 106]
Mouthrinses containing protease inhibitors (chlorhexidine and green tea extract)	Dentin	They reduced dentin erosion (around 30–40 %) when compared with control in situ. Their effect is similar to fluoride.	[113, 114]

8.3.11 Use of Mouthrinse Containing Protease Inhibitors

The erosive process in dentin is different from the one occurring in enamel due to the presence of the DOM that makes ionic diffusion more difficult, thus slowing down the progression of erosion. The DOM is composed mainly of type-I collagen that is susceptible to degradation by proteases, thus allowing the progression of erosion [112]. Based on this, mouthrinses containing protease inhibitors, such as chlorhexidine and green tea extract [113], or even rinses with green tea [114] have been shown to reduce dentin loss (around 30–40 %) in situ when compared with control. The effect of the protease inhibitors was similar to fluoride. Thus mouthrinses containing $SnCl_2$/NaF/AmF, TiF_4/NaF, or protease inhibitors might have potential to benefit patients that are frequently exposed to erosive challenges.

Table 8.3 summarizes the results of some studies on the anti-erosive effect of mouthrinses.

Conclusion

As there are signs that the prevalence of erosion is increasing in several countries, the dentist should have knowledge about its aetiology and be prepared for early diagnosis. Based on this knowledge, preventive measures can be applied appropriately. Therapeutic strategies in high-risk patients should be as conservative as possible, involving multidisciplinary and preventive approaches, including behavioral and lifestyle changes, with a periodic monitoring of treatment success. The modification of the tooth surface to increase its resistance to acidic attacks (e.g. fluoride application) still remains the most useful preventive strategy. The daily application of fluoride, especially those containing tin (dentifrice with chitosan or mouthrinse), is a promising strategy to reduce dental erosion.

References

1. Kreulen CM, Van't Spijker A, Rodriguez JM, Bronkhorst EM, Creugers NH, Bartlett DW. Systematic review of the prevalence of tooth wear in children and adolescents. Caries Res. 2010;44(2):151–9.
2. Huysmans MC, Chew HP, Ellwood RP. Clinical studies of dental erosion and erosive wear. Caries Res. 2011;45 Suppl 1:60–8.
3. Shellis RP, Ganss C, Ren Y, Zero DT, Lussi A. Methodology and models in erosion research: discussion and conclusions. Caries Res. 2011;45 Suppl 1:69–77.
4. Attin T, Knöfel S, Buchalla W, Tutuncu R. In situ evaluation of different remineralization periods to decrease brushing abrasion of demineralized enamel. Caries Res. 2001;35(3):216–22.
5. Rios D, Honório HM, Magalhães AC, Delbem AC, Machado MA, Silva SM, et al. Effect of salivary stimulation on erosion of human and bovine enamel subjected or not to subsequent abrasion: an in situ/ex vivo study. Caries Res. 2006;40(3):218–23.
6. Magalhães AC, Wiegand A, Buzalaf MA. Use of dentifrices to prevent erosive tooth wear: harmful or helpful? Braz Oral Res. 2014;28(Spec no. 1):1–6.

7. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci. 1996;104(2):151–5.
8. Lussi A, Jaeggi T. Erosion – diagnosis and risk factors. Clin Oral Invest. 2008;12 Suppl 1:S5–13.
9. Lussi A, Jaeggi T. Chemical factors. Monogr Oral Sci. 2006;20:77–87.
10. Lussi A. Erosive tooth wear – a multifactorial condition of growing concern and increasing knowledge. Monogr Oral Sci. 2006;20:1–8.
11. Bartlett D. Intrinsic causes of erosion. Monogr Oral Sci. 2006;20:119–39.
12. Hermont AP, Pordeus IA, Paiva SM, Abreu MH, Auad SM. Eating disorder risk behavior and dental implications among adolescents. Int J Eat Disord. 2013;46(7):677–83.
13. O'Sullivan EA, Curzon ME. Salivary factors affecting dental erosion in children. Caries Res. 2000;34(1):82–7.
14. Jensdottir T, Buchwald C, Nauntofte B, Hansen HS, Bardow A. Saliva in relation to dental erosion before and after radiotherapy. Acta Odontol Scand. 2013;71(3–4):1008–13.
15. Hannig M, Fiebiger M, Güntzer M, Döbert A, Zimehl R, Nekrashevych Y. Protective effect of the in situ formed short-term salivary pellicle. Arch Oral Biol. 2004;49(11):903–10.
16. Hara AT, Ando M, Gonzãlez-Cabezas C, Cury JA, Serra MC, Zero DT. Protective effect of the dental pellicle against erosive challenges in situ. J Dent Res. 2006;85(7):612–6.
17. Buzalaf MA, Hannas AR, Kato MT. Saliva and dental erosion. J Appl Oral Sci. 2012;20(5):493–502.
18. Cheaib Z, Lussi A. Impact of acquired enamel pellicle modification on initial dental erosion. Caries Res. 2011;45(2):107–12.
19. Vukosavljevic D, Custodio W, Buzalaf MA, Hara AT, Siqueira WL. Acquired pellicle as a modulator for dental erosion. Arch Oral Biol. 2014;59(6):631–8.
20. El Aidi H, Bronkhorst EM, Huysmans MC, Truin GJ. Multifactorial analysis of factors associated with the incidence and progression of erosive tooth wear. Caries Res. 2011;45(3):303–12.
21. Magalhães AC, Wiegand A, Rios D, Buzalaf MA, Lussi A. Fluoride in dental erosion. Monogr Oral Sci. 2011;22:158–70.
22. Kaidonis JA. Prevention and control of dental erosion: professional clinic care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 151–68.
23. Amaechi BT. Recall, maintenance care cycle, and outcomes assessment. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 303–11.
24. Wang X, Lussi A. Assessment and management of dental erosion. Dent Clin North Am. 2010;54(3):565–78.
25. Ganss C, Lussi A, Schlueter N. Dental erosion as oral disease. Insights in etiological factors and pathomechanisms, and current strategies for prevention and therapy. Am J Dent. 2012;25(6):351–64.
26. Rios D, Magalhães AC, Honório HM, Buzalaf MA, Lauris JR, Machado MA. The prevalence of deciduous tooth wear in six-year-old children and its relationship with potential explanatory factors. Oral Health Prev Dent. 2007;5(3):167–71.
27. Nahás Pires Corrêa MS, Nahás Pires Corrêa F, Nahás Pires Corrêa JP, Murakami C, Mendes FM. Prevalence and associated factors of dental erosion in children and adolescents of a private dental practice. Int J Paediatr Dent. 2011;21(6):451–8.
28. Smith AJ, Shaw L. Comparison of rates of clearance of glucose from various sites following drinking with a glass feeder cup and straw. Med Sci Res. 1993;21:617–9.
29. Amaechi BT, Higham SM, Edgar WM. Factors influencing the development of dental erosion in vitro: enamel type, temperature and exposure time. J Oral Rehabil. 1999;26:624–30.
30. West NX, Hughes JA, Addy M. Erosion of dentine and enamel in vitro by dietary acids: the effect of temperature, acid character, concentration and exposure time. J Oral Rehabil. 2000;27:875–80.
31. Millward A, Shaw L, Harrington E, Smith AJ. Continuous monitoring of salivary flow rate and pH at the surface of the dentition following consumption of acidic beverages. Caries Res. 1997;31:44–9.

32. Conviser JH, Fisher SD, Mitchell KB. Oral care behavior after purging in a sample of women with bulimia nervosa. J Am Dent Assoc. 2014;145:352–4.
33. Leung G, Amaechi BT. Prevention and control of dental erosion: psychological management. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 181–201.
34. Patel A, Brady III CE, Amaechi BT. Prevention and control of dental erosion: gastroesophageal reflux disease management. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 203–24.
35. Amaechi BT, Higham SM, Edgar WM. Influence of abrasion in clinical manifestation of human dental erosion. J Oral Rehabil. 2003;30(4):407–13.
36. Joiner A, Pickles MJ, Lynch S, Cox TF. The measurement of enamel wear by four toothpastes. Int Dent J. 2008;58(1):23–8.
37. Philpotts CJ, Weader E, Joiner A. The measurement in vitro of enamel and dentine wear by toothpastes of different abrasivity. Int Dent J. 2005;55(3 Suppl 1):183–7.
38. Wiegand A, Attin T. Design of erosion/abrasion studies – insights and rational concepts. Caries Res. 2011;45 Suppl 1:53–9.
39. Wiegand A, Kuhn M, Sener B, Roos M, Attin T. Abrasion of eroded dentin caused by toothpaste slurries of different abrasivity and toothbrushes of different filament diameter. J Dent. 2009;37(6):480–4.
40. Wiegand A, Schwerzmann M, Sener B, Magalhães AC, Roos M, Ziebolz D, et al. Impact of toothpaste slurry abrasivity and toothbrush filament stiffness on abrasion of eroded enamel – an in vitro study. Acta Odontol Scand. 2008;66(4):231–5.
41. Hooper S, West NX, Pickles MJ, Joiner A, Newcombe RG, Addy M. Investigation of erosion and abrasion on enamel and dentine: a model in situ using toothpastes of different abrasivity. J Clin Periodontol. 2003;30(9):802–8.
42. Hara AT, Gonzalez-Cabezas C, Creeth J, Parmar M, Eckert GJ, Zero DT. Interplay between fluoride and abrasivity of dentifrices on dental erosion-abrasion. J Dent. 2009;37(10):781–5.
43. Wiegand A, Attin T. Occupational dental erosion from exposure to acids: a review. Occup Med (Lond). 2007;57(3):169–76.
44. Ranjitkar S, Rodriguez JM, Kaidonis JA, Richards LC, Townsend GC, Bartlett DW. The effect of casein phosphopeptide-amorphous calcium phosphate on erosive enamel and dentine wear by toothbrush abrasion. J Dent. 2009;37:250–4.
45. Lussi A, Schaffner M. Progression of and risk factors for dental erosion and wedge-shaped defects over a 6-year period. Caries Res. 2000;34:182–7.
46. Corrêa MC, Lerco MM, da Cunha ML, Henry MA. Salivary parameters and teeth erosions in patients with gastroesophageal reflux disease. Arq Gastroenterol. 2012;49(3):214–8.
47. Yoshikawa H, Furuta K, Ueno M, Egawa M, Yoshino A, Kondo S, et al. Oral symptoms including dental erosion in gastroesophageal reflux disease are associated with decreased salivary flow volume and swallowing function. J Gastroenterol. 2012;47(4):412–20.
48. Prestes L, Souza BM, Comar LP, Salomão PA, Rios D, Magalhães AC. In situ effect of chewing gum containing CPP-ACP on the mineral precipitation of eroded bovine enamel-a surface hardness analysis. J Dent. 2013;41(8):747–51.
49. 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(3):120–2.
50. Magalhães AC, Levy FM, Souza BM, Cardoso CA, Cassiano LP, Pessan JP, Buzalaf MA. Inhibition of tooth erosion by milk containing different fluoride concentrations: an in vitro study. J Dent. 2014;42(4):498–502.
51. Attin T, Weiss K, Becker K, Buchalla W, Wiegand A. Impact of modified acidic soft drinks on enamel erosion. Oral Dis. 2005;11(1):7–12.
52. Magalhães AC, Moraes SM, Rios D, Buzalaf MA. Effect of ion supplementation of a commercial soft drink on tooth enamel erosion. Food Addit Contam Part A Chem Anal Control Expo Risk Assess. 2009;26(2):152–6.

53. Beyer M, Reichert J, Heurich E, Jandt KD, Sigusch BW. Pectin, alginate and gum arabic polymers reduce citric acid erosion effects on human enamel. Dent Mater. 2010;26(9):831–9.
54. Beyer M, Reichert J, Sigusch BW, Watts DC, Jandt KD. Morphology and structure of polymer layers protecting dental enamel against erosion. Dent Mater. 2012;28(10):1089–97.
55. Lindquist B, Lingstrom P, Fandriks L, Birkhed D. Influence of five neutralizing products on intra-oral ph after rinsing with simulated gastric acid. Eur J Oral Sci. 2011;119:301–4.
56. Messias DC, Turssi CP, Hara AT, Serra MC. Sodium bicarbonate solution as an anti-erosive agent against simulated endogenous erosion. Eur J Oral Sci. 2010;118:385–8.
57. Turssi CP, Vianna LM, Hara AT, do Amaral FL, Franca FM, Basting RT. Counteractive effect of antacid suspensions on intrinsic dental erosion. Eur J Oral Sci. 2012;120:349–52.
58. Hove L, Holme B, Ogaard B, Willumsen T, Tveit AB. The protective effect of TiF_4, SnF_2 and NaF on erosion of enamel by hydrochloric acid in vitro measured by white light interferometry. Caries Res. 2006;40(5):440–3.
59. Hove LH, Holme B, Young A, Tveit AB. The erosion-inhibiting effect of TiF_4, SnF_2 and NaF solutions on pellicle-covered enamel in vitro. Acta Odont Scand. 2007;65:259–64.
60. Hove LH, Holme B, Young A, Tveit AB. The protective effect of TiF_4, SnF_2 and NaF against erosion-like lesions in situ. Caries Res. 2008;42:68–72.
61. Stenhagen KR, Hove LH, Holme B, Tveit AB. The effect of daily fluoride mouth rinsing on enamel erosive/abrasive wear in situ. Caries Res. 2013;47:2–8.
62. Young A, Thrane PS, Saxegaard E, Jonski G, Rolla G. Effect of stannous fluoride toothpaste on erosion-like lesions: an in vivo study. Eur J Oral Sci. 2006;114(3):180–3.
63. Ganss C, Lussi A, Grunau O, Klimek J, Schlueter N. Conventional and anti-erosion fluoride toothpastes. Effect on enamel erosion and erosion-abrasion. Caries Res. 2011;45(6):581–9.
64. Ganss C, von Hinckeldey J, Tolle A, Schulze K, Klimek J, Schlueter N. Efficacy of the stannous ion and a biopolymer in toothpastes on enamel erosion/abrasion. J Dent. 2012;40(12):1036–43.
65. Huysmans MC, Jager DH, Ruben JL, Unk DE, Klijn CP, Vieira AM. Reduction of erosive wear in situ by stannous fluoride-containing toothpaste. Caries Res. 2011;45(6):518–23.
66. Faller RV, Eversole SL, Tzeghai GE. Enamel protection: a comparison of marketed dentifrice performance against dental erosion. Am J Dent. 2011;24(4):205–10.
67. Messias DC, Maeda FA, Turssi CP, Serra MC. Effect of dentifrices against hydrochloric acid-induced erosion. Oral Health Prev Dent. 2011;9:269–73.
68. Babcock FD, King JC, Jordan TH. The reaction of stannous fluoride and hydroxyapatite. J Dent Res. 1978;57:933–8.
69. Ganss C, Hardt M, Lussi A, Cocks AK, Klimek J, Schlueter N. Mechanism of action of tin-containing fluoride solutions as anti-erosive agents in dentine – an in vitro tin-uptake, tissue loss, and scanning electron microscopy study. Eur J Oral Sci. 2010;118:376–84.
70. Mundorff SA, Little MF, Bibby BG. Enamel dissolution. II. Action of titanium tetrafluoride. J Dent Res. 1972;51:1567–71.
71. 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(6):561–6.
72. 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(11):931–5.
73. ten Cate JM. Review on fluoride, with special emphasis on calcium fluoride mechanisms in caries prevention. Eur J Oral Sci. 1997;105(5 Pt 2):461–5.
74. Saxegaard E, Rolla G. Fluoride acquisition on and in human enamel during topical application in vitro. Scand J Dent Res. 1988;96(6):523–35.
75. Passos VF, de Vasconcellos AA, Pequeno JH, Rodrigues LK, Santiago SL. Effect of commercial fluoride dentifrices against hydrochloric acid in an erosion-abrasion model. Clin Oral Invest. 2015;19(1):71–6.
76. Comar LP, Gomes MF, Ito N, Salomao PA, Grizzo LT, Magalhães AC. Effect of NaF, SnF(2), and TiF(4) toothpastes on bovine enamel and dentin erosion-abrasion in vitro. Int J Dent. 2012;2012:134350.

77. Ganss C, Schulze K, Schlueter N. Toothpaste and erosion. Monogr Oral Sci. 2013; 23:88–99.
78. Magalhães AC, Rios D, Moino AL, Wiegand A, Attin T, Buzalaf MA. Effect of different concentrations of fluoride in dentifrices on dentin erosion subjected or not to abrasion in situ/ex vivo. Caries Res. 2008;42(2):112–6.
79. Rios D, Magalhães AC, Polo RO, Wiegand A, Attin T, Buzalaf MA. The efficacy of a highly concentrated fluoride dentifrice on bovine enamel subjected to erosion and abrasion. J Am Dent Assoc. 2008;139(12):1652–6.
80. Ren YF, Liu X, Fadel N, Malmstrom H, Barnes V, Xu T. Preventive effects of dentifrice containing 5000ppm fluoride against dental erosion in situ. J Dent. 2011;39(10):672–8.
81. Moretto MJ, Magalhães AC, Sassaki KT, Delbem AC, Martinhon CC. Effect of different fluoride concentrations of experimental dentifrices on enamel erosion and abrasion. Caries Res. 2010;44(2):135–40.
82. Wang CP, Huang SB, Liu Y, Li JY, Yu HY. The CPP-ACP relieved enamel erosion from a carbonated soft beverage: an in vitro AFM and XRD study. Arch Oral Biol. 2014;59(3):277–82.
83. Carvalho FG, Brasil VL, Silva Filho TJ, Carlo HL, Santos RL, Lima BA. Protective effect of calcium nanophosphate and CPP-ACP agents on enamel erosion. Braz Oral Res. 2013;27(6):463–70.
84. Srinivasan N, Kavitha M, Loganathan SC. Comparison of the remineralization potential of CPP–ACP and CPP–ACP with 900 ppm fluoride on eroded human enamel: an in situ study. Arch Oral Biol. 2010;55(7):541–4.
85. Panich M, Poolthong S. The effect of casein phosphopeptide-amorphous calcium phosphate and a cola soft drink on in vitro enamel hardness. J Am Dent Assoc. 2009;140:455–60.
86. Wang X, Megert B, Hellwig E, Neuhaus KW, Lussi A. Preventing erosion with novel agents. J Dent. 2011;39:163–70.
87. Lennon AM, Pfeffer M, Buchalla W, Becker K, Lennon S, Attin T. Effect of a casein/calcium phosphate-containing tooth cream and fluoride on enamel erosion in vitro. Caries Res. 2006;40:154–7.
88. Wegehaupt FJ, Attin T. The role of fluoride and casein phosphopeptide/amorphous calcium phosphate in the prevention of erosive/abrasive wear in an in vitro model using hydrochloric acid. Caries Res. 2010;44:358–63.
89. Cochrane NJ, Cai F, Huq NL, Burrow MF, Reynolds EC. New approaches to enhanced remineralization of tooth enamel. J Dent Res. 2010;89:1187–97.
90. Cochrane NJ, Reynolds EC. Calcium phosphopeptides — mechanisms of action and evidence for clinical efficacy. Adv Dent Res. 2012;24:41–7.
91. Cross KJ, Huq NL, Palamara JE, Perich JW, Reynolds EC. Physicochemical characterization of casein phosphopeptide-amorphous calcium phosphate nanocomplexes. J Biol Chem. 2005;280:15362–9.
92. Karlinsey RL, Mackey AC, Walker ER, Frederick KE. Preparation, characterization, and in vitro efficacy of an acid-modified β-TCP material for dental hard-tissue remineralization. Acta Biomater. 2010;6:969–78.
93. Karlinsey RL, Pfarrer AM. Fluoride plus functionalized β-TCP: a promising combination for robust remineralization. Adv Dent Res. 2012;24:48–52.
94. Amaechi BT, Karthikeyan R, Mensinkai PK, Najibfard K, Mackey AC, Karlinsey RL. Remineralization of eroded enamel by a NaF rinse containing a novel calcium phosphate agent in an in situ model: a pilot study. Clin Cosmet Investig Dent. 2010;2:93–100.
95. Wang ZJ, Sa Y, Sauro S, Chen H, Xing WZ, Ma X, et al. Effect of desensitising toothpastes on dentinal tubule occlusion: a dentine permeability measurement and SEM in vitro study. J Dent. 2010;38(5):400–10.
96. Lynch E, Brauer DS, Karpukhina N, Gillam DG, Hill RG. Multi-component bioactive glasses of varying fluoride content for treating dentin hypersensitivity. Dental Mater. 2012;28:168–78.

97. Mneimne M, Hill RG, Bushby AJ, Brauer DS. High phosphate content significantly increases apatite formation of fluoride-containing bioactive glasses. Acta Biomater. 2011;7:1827–34.
98. Ganss C, Klimek J, Schlueter N. Erosion/abrasion-preventing potential of NaF and F/Sn/chitosan toothpastes in dentine and impact of the organic matrix. Caries Res. 2014;48(2):163–9.
99. Young A, Smistad G, Karlsen J, Rolla G, Rykke M. Zeta potentials of human enamel and hydroxyapatite as measured by the coulter delsa 440. Adv Dent Res. 1997;11(4):560–5.
100. Schlueter N, Klimek J, Ganss C. Effect of a chitosan additive to a Sn^{2+}-containing toothpaste on its anti-erosive/anti-abrasive efficacy – a controlled randomised in situ trial. Clin Oral Invest. 2014;18(1):107–15.
101. Wiegand A, Bichsel D, Magalhaes AC, Becker K, Attin T. Effect of sodium, amine and stannous fluoride at the same concentration and different pH on in vitro erosion. J Dent. 2009;37:591–5.
102. Yu H, Wegehaupt FJ, Zaruba M, Becker K, Roos M, Attin T, Wiegand A. Erosion-inhibiting potential of a stannous chloride-containing fluoride solution under acid flow conditions in vitro. Arch Oral Biol. 2010;55:702–5.
103. Wiegand A, Waldheim E, Sener B, Magalhaes AC, Attin T. Comparison of the effects of TiF_4 and NaF solutions at pH 1.2 and 3.5 on enamel erosion in vitro. Caries Res. 2009;43:269–77.
104. Wiegand A, Magalhaes AC, Sener B, Waldheim E, Attin T. TiF(4) and NaF at pH 1.2 but not at pH 3.5 are able to reduce dentin erosion. Arch Oral Biol. 2009;54:790–5.
105. Sen BH, Kazemi RB, Spangberg LS. Morphologic effects on L929 fibroblasts of titanium tetrafluoride application. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1998;86(3):341–6.
106. Souza BM, Lima LL, Comar LP, Buzalaf MA, Magalhães AC. Effect of experimental mouthrinses containing the combination of NaF and TiF_4 on enamel erosive wear in vitro. Arch Oral Biol. 2014;59(6):621–4.
107. Schlueter N, Klimek J, Ganss C. In vitro efficacy of experimental tin- and fluoride-containing mouth rinses as anti-erosive agents in enamel. J Dent. 2009;37(12):944–8.
108. Schlueter N, Klimek J, Ganss C. Efficacy of an experimental tin-F-containing solution in erosive tissue loss in enamel and dentine in situ. Caries Res. 2009;43(6):415–21.
109. Schlueter N, Klimek J, Ganss C. Effect of stannous and fluoride concentration in a mouth rinse on erosive tissue loss in enamel in vitro. Arch Oral Biol. 2009;54(5):432–6.
110. Ganss C, Lussi A, Sommer N, Klimek J, Schlueter N. Efficacy of fluoride compounds and stannous chloride as erosion inhibitors in dentine. Caries Res. 2010;44(3):248–52.
111. Ganss C, Neutard L, von Hinckeldey J, Klimek J, Schlueter N. Efficacy of a tin/fluoride rinse: A randomized in situ trial on erosion. J Dent Res. 2010;89(11):1214–8.
112. Buzalaf MA, Kato MT, Hannas AR. The role of matrix metalloproteinases in dental erosion. Adv Dent Res. 2012;24(2):72–6.
113. Magalhães AC, Wiegand A, Rios D, Hannas A, Attin T, Buzalaf MA. Chlorhexidine and green tea extract reduce dentin erosion and abrasion in situ. J Dent. 2009;37(12):994–8.
114. Kato MT, Magalhães AC, Rios D, Hannas AR, Attin T, Buzalaf MA. Protective effect of green tea on dentin erosion and abrasion. J Appl Oral Sci. 2009;17(6):560–4.

Prevention and Control of Dental Erosion: Professional Clinic Care

9

John A. Kaidonis, Poppy M. Anastassiadis, Dimitra Lekkas, Sarbin Ranjitkar, Bennett T. Amaechi, and Grant C. Townsend

Abstract

There are basic principles that should be followed for the prevention and control of dental erosion. They rely on diminishing or eliminating the acid challenge, overcoming any hypersensitivity, healing the remaining damaged tooth surface by remineralization and protecting the tooth from any subsequent acid attack. Opinions vary as to how this should be achieved and the plethora of products that are available, each with their specific formulations and different concentrations, often causes confusion among clinicians. Although examples of specific products will be mentioned in this chapter, the emphasis will be on general methods with reference to generic products in order to guide management. Although this chapter focuses only on professionally applied products and oral health education, they should only form part of an overall preventive program that includes home preventive measures for which the patient is responsible. Each management plan needs to follow a preventive philosophy that is tailor-made for the patient.

J.A. Kaidonis, BDS, BSc Dent, PhD (✉) • P.M. Anastassiadis • D. Lekkas
S. Ranjitkar • G.C. Townsend, BDS, BSc Dent, PhD, DDSc
Faculty of Health Sciences, School of Dentistry, The University of Adelaide, Adelaide, SA 5005, Australia
e-mail: john.kaidonis@adelaide.edu.au

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_9

9.1 Introduction

The effects of acids of non-bacterial origin on dental tissues are well-documented and there has been extensive research attempting to explain the different parameters that affect the management of dental erosion [1–3]. However, to date, longitudinal clinical studies are lacking and much of our clinical management is based on inferences resulting from past research on dental caries, in vitro and in situ erosion studies and from clinical experience.

The most essential component in preventing erosion is the elimination or reduction of the acid challenge. In addition, prevention should aim at the remineralization of softened enamel by using fluoride formulations while in the presence of saliva. The remineralizing effects of fluoride can further be enhanced by utilizing techniques and products that provide a longer 'contact time' of fluoride within the oral environment, allowing for a greater volume of saliva to deliver the maximum concentrations of calcium and phosphate ions required for remineralization. If the saliva is compromised, then the remineralizing approach should include reintroducing the calcium and phosphate ions required for repair.

Most often, a remineralized, fluoride-enriched tooth surface offers adequate protection from a mild acid challenge, provided that a remineralizing environment is maintained. However, in uncontrolled severe erosive cases where the frequency of acid exposure is high and the pH is often below the critical pH of fluorapatite, any remineralizing efforts become ineffective. The damage continues, often becoming extensive with associated dentin exposure and hypersensitivity. In such cases, there is a strong argument for the use of prophylactic surface coatings (barriers) to prevent acid contacting tooth surfaces, especially if they themselves have remineralizing capabilities. This is crucial in preventing damage from subsequent acid attack but it must be understood by the patient that these approaches only provide 'temporary' protection and need to be replaced as required. Protective tooth surface coatings are not considered to be 'permanent restoratives' that actually replace missing tooth structure.

The professionally applied approaches discussed below are not presented in any order of preference but simply summarize techniques with some examples of associated products available to the clinician. All techniques have their place in prevention, and their selection is based on the patient case. This chapter does not compare the effectiveness of products but lists possible approaches to preventive erosion management that are available to clinicians and should be used in conjunction with home preventive measures for which the patient is responsible. Each management plan needs to follow a preventive philosophy that is tailor-made for the patient [4].

9.2 Physico-chemical Dynamics of Erosion That Influence Preventive Management

There is enough documented evidence attesting to the protective nature of the acquired tooth pellicle (and associated biofilm) against acid [5, 6]. The pellicle/biofilm is a perm-selective membrane that acts as a physical barrier to acids and the

saliva acts as an excellent buffer both outside [7] and within the structure. However, frequent acid exposure can gradually remove the biofilm in different areas of the mouth [8], exposing the tooth surface to demineralisation and producing the various erosive patterns and gradients of severity seen clinically. In addition, the inevitable mechanical effects of abrasion from mastication and from oral hygiene routines (e.g. toothbrush) add to and complicate the clinical appearance.

More specifically, the duration of acid acting on an exposed tooth surface after biofilm is removed may vary from momentary to prolonged exposure, reflecting the interplay between acid type, frequency of exposure, acid flow rate, saliva's buffering capacity, oral clearance and pellicle formation. Factors such as surface tension and contact angles between liquids explain why many dietary acids readily displace saliva irrespective of its quality and, if the acid challenge is severe, saliva alone has little or no immediate effect [9]. Acids are unsaturated solutions with respect to hydroxyapatite and cause rapid demineralization once in contact with the tooth surface. This is an 'open system' where demineralization products are lost through swallowing and without the potential for them to be reused. It is after an acid attack when the liquid is cleared, and this can take between 2 and 5 min [10], that any remineralizing effects of saliva (and other remineralizing products when applied) come into play. The remineralizing effects of saliva are limited and can take a number of hours when compared to remineralizing products, making the frequency of acid episodes a major factor in repair success. In enamel, remineralization repairs what researchers consider to be the 'softened enamel' (a few microns thick) that consists of the damaged ends of the enamel rods. However, this remineralization process will not 'rebuild' what is lost. That is, the original length of the enamel rod will not grow back (Fig. 9.1).

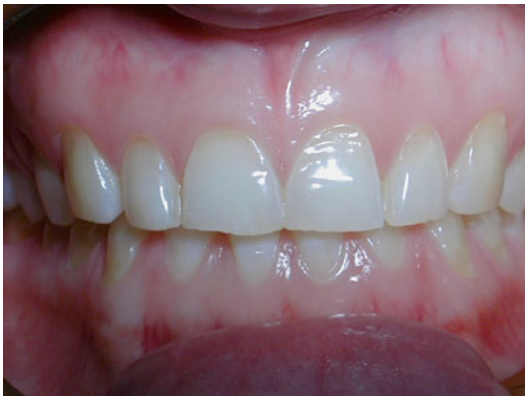


Fig. 9.1 Very early stages of uncontrolled erosion in a 22-year-old patient. Teeth affected include the labial cervical third of 13, 12, 11, 21, 22 and 23. Remineralizing procedures can repair softened enamel but the 'dished-out' appearance will remain (i.e. the enamel rods will not grow back). Prophylactic coverings over the affected surfaces can protect the teeth until management of the acid source is achieved

In cases where the saliva is compromised (e.g. Sjogren's syndrome), automatic repair from saliva after the acid challenge will be ineffective. Therefore, in such cases, the clinical preventive approach should aim to provide the appropriate concentrations of ions (e.g. calcium, phosphate) in the oral environment, replacing what is lost from saliva.

There is now a strong argument to also consider 'fostering' and utilizing the pellicle/biofilm wherever possible in the management of erosion [5]. Apart from providing a physical barrier to acid, biofilm also provides a 'closed system' where ions from saliva and from professionally applied products provide supersaturated conditions for remineralization.

At the other extreme, the main problem with uncontrolled, very active erosion is that the many acids (in particular soft drinks and gastric acid) have a pH well below the critical pH of hydroxyapatite and fluorapatite. Irrespective of how well enamel is repaired by remineralizing products, the next acid episode will bring about gross dissolution. Although in vitro studies have repeatedly shown that the remineralizing effects of saliva and indeed remineralizing products with fluoride can repair a softened surface and make it more resistant to further acid attack, it is difficult to translate this in vitro research to the clinical situation. In reality, the extent of clinical protection provided by remineralization in uncontrolled cases is at best minimal. In these cases, the total mineral loss is more than the total mineral gain, making management of these cases difficult.

Based on clinical experience, uncontrolled active erosive cases cannot be contained by remineralizing products alone, even in the presence of good quality saliva. The preventive management in these cases requires protective surface coatings to be placed over the affected areas to prevent acid from reaching the tooth [11]. In addition, prophylactic barriers that also have the potential to remineralize would, by inference, be more beneficial. Either way, these patients must be made aware that this approach requires regular maintenance due to the breakdown of these barriers over time. Until most prophylactic barriers are 'purpose-made' by manufacturers to endure mechanical and erosive environments at least as well as most restorations, the majority should currently be considered as 'sacrificial' and transient tooth tissue prophylactic agents.

Finally, prophylactic coverings placed on eroded areas have the added advantage of immediately eliminating hypersensitivity by instantly obturating patent dentinal tubules. This is an added advantage when the patient's chief complaint is pain.

9.3 Remineralization Methods for Softened Tooth Surfaces

The professionally applied products discussed below are not presented in any order of preference nor is the effectiveness of the products or techniques compared. They are listed as possible approaches to remineralization that are available to clinicians and should be used in conjunction with home preventive measures.

Traditionally, fluorides in the form of gels and foams have been used and are still being used for the remineralization of softened enamel and for slowing the

demineralization process. However, fluoride is only effective when other elements (e.g. calcium and phosphate ions) also co-exist in the correct proportions to produce effective remineralization. Therefore, fluoride applied professionally in high concentrations on newly eroded surfaces will remineralize by utilizing the existing calcium and phosphate in saliva, after which the excess fluoride is expectorated or ingested and therefore wasted. In order to overcome this, a number of approaches can be considered, either individually or in combination.

Extending the physical presence of fluoride can be achieved if it is applied in the presence of biofilm. Here the fluoride is 'held' within the biofilm and when the pH drops during a subsequent acid challenge, it is allowed to combine with calcium and phosphate from saliva. Normally, calcium ions in saliva are complexed with statherins and proline-rich proteins and will not become available unless there is a drop in pH during an acid attack. This can occur in milder erosive cases where some biofilm/pellicle is most likely to form between episodes of acid challenge. This also supports the premise that a prophylaxis should not be done before preventive measures.

The use of fluoride varnishes can also extend the physical presence of fluoride by allowing more time for the accumulation and increasing concentration of calcium and phosphate ions from saliva to utilize the excess fluoride. Although this method provides more effective remineralization, excess fluoride is still wasted. In addition, excess fluoride can be utilized when applied in high concentrations if comparable high concentrations of bio-available calcium and phosphate are also added to the oral environment, as with the various calcium-phosphate technologies available today.

Opinions vary as to which fluoride concentrations are ideal for erosion. Inferences are often derived from past research relating to caries indicating that low but sustained concentrations of fluoride seem to provide resistance to demineralization and more effective remineralization [12, 13]. However, more recent evidence indicates that for erosion, higher concentrations seem more effective especially if polyvalent fluoride formulations (e.g. stannous fluorides, silver fluoride) are used. In particular, an increase of metal ion to fluoride ratio (e.g. stannous to fluoride ratio) seems more effective [14]. Finally, the effective relevance of a 'one-off' professional application alone is limited; however, when coupled with continuous home applications, the positive effects become long term and more effective.

9.3.1 Professionally Applied Fluorides

9.3.1.1 Gels and Foams

Professionally applied fluorides are usually high concentration *neutral Fluorides* (e.g. 2 % NaF) that can be easily dispensed in disposable trays as either gels or foams (Table 9.1). Although foams require less material than gels to supply the same amount of fluoride, much of the fluoride benefit from these high concentrations is limited by the concentration and availability of calcium and phosphate.

Traditional prophylaxis of teeth before application of these products is not recommended and application of fluoride should occur after the biofilm is allowed to

Table 9.1 Examples of products that can be used professionally for the prevention and control of dental erosion

Generic product	Name	Details	Company
Neutral fluoride	–	–	–
–	Denti-Care Pro-Foam Denti-Care Pro-Gel	2 % neutral sodium fluoride foam 2 % neutral sodium fluoride gel	Medicare (US), (CAN)
–	Nupro Neutral Fluoride Gel	2 % neutral sodium fluoride	Dentsply (AUS)
–	Neutra Foam	2 % neutral sodium fluoride	Oral B (US)
–	Neutracare Gel	1.1 % sodium fluoride	Oral B (US)
APF	Nupro Acidulated Phosphate Fluoride Foam	1.23 % APF	Dentsply
–	Floam	1.23 % APF	Germiphene
–	Ultra Control Foam	1.23 % APF	Waterpik (UK) (EUR)
–	One minute topical Fluoride Foam	1.23 % APF	Laclede (US), (AUS)
Stannous fluorides	–	–	–
–	Pro Health Toothpastes	1450 ppm F (stabilized stannous fluoride 1100 ppm, 350 ppm sodium fluoride and sodium hexametaphosphate)	Oral B (AUS)
–	Crest Pro Health Toothpastes	0.454 % stannous fluoride	Crest (US)
–	Gel Kam	1000 ppm stannous fluoride	Colgate (AUS)
–	Fluorigard Gel Toothpaste	1000 ppm stannous fluoride	Colgate (AUS)
Fluoride varnishes	–	–	–
–	Duraphat	Sodium fluoride varnish 22, 600 ppm fluoride	Colgate (AUS)
–	Cavity Shield	5 % sodium fluoride varnish	3 M (US)
–	Profluorid Varnish	5 % sodium fluoride varnish	Voco (UK), (USA), (AUS)
–	DuraShield Varnish	5 % sodium fluoride varnish	Sultan Healthcare (EUR), (CAN), (US)
–	Nupro	5 % sodium fluoride varnish	Dentsply International
CPP-ACP	Tooth Mousse	Creme	GC (AUS), (EUR)
–	Tooth Mousse Plus (containing fluoride)	Creme	GC (AUS), (EUR)
–	MI Paste	Creme	GC (US)
–	MI Paste Plus (containing fluoride)	Creme	GC (US)

Table 9.1 (continued)

Generic product	Name	Details	Company
–	MI Varnish	5 % sodium fluoride varnish with recaldent	GC (US)
TCP	Clinpro White Varnish	5 % sodium fluoride with Tri-Calcium Phosphate	3 M ESPE (AUS), (EUR)
–	Vanish Varnish	5 % sodium fluoride with tri-calcium phosphate	3 M ESPE (US)
–	Clinpro Tooth Crème (toothpaste)	950 ppm fluoride with TCP	3 M ESPE (AUS), (EUR)
ACP	–	–	–
–	Enamel Pro Varnish	5 % sodium fluoride with ACP	Premier (US)

form. Late afternoon appointments, or preferably asking the patient not to brush on the day of the appointment, have benefits. However, asking patients not to brush should occur with selected, dentally aware patients otherwise the wrong message will be sent about oral hygiene and oral health.

Directions for fluoride gels and foams:

- Prophylaxis is not recommended.
- Close-fitting trays should be used (these can be constructed by thermoforming machines that heat and mold silicone blanks onto patient's casts). The amount of fluoride dispensed should only cover the tray surface rather than filling the trays completely. Each tray should not exceed 2 ml in total.
- The duration of the application should be about 4 min with the excess expectorated for about 30 s after the trays are removed. A salivary ejector should be used during the procedure.
- Finally, the patient is advised not to eat or drink for 30 min after application.

Operators must be aware not to exceed the 'probable toxic dose' measure of 5 mg/kg for fluoride of body weight per day, and age recommendations must be adhered to. High concentration gels are not recommended for children younger than 10 years of age because of the potential of ingestion.

Acidulated Phosphate Fluoride (APF) gels are also high concentration fluorides (1.23 % fluoride) that are recommended by some clinicians (Table 9.1). In vitro studies have shown that APF provides more protection than high concentration NaF gels in both endogenous erosion and particularly in wine tasters erosion where the acidic challenge is extreme [11, 15]. For example, the use of APF with professional wine tasters has shown a high degree of protection especially when biofilm is allowed to establish for at least 24 hours before a wine tasting event. Professional application of APF the day before the tasting and supplemented periodically by patient-applied home preventive care is very effective. In addition, acidified fluoride gels over erosion lesions seem to show higher abrasion resistance than neutral fluorides [16].

Although the pH of APF is about 3.0–3.5 and may not seem appropriate for erosive cases, there are some benefits when saliva is compromised. Here, the low pH of the APF demineralizes the tooth surface, allowing calcium ions from the tooth to be used with the phosphate and fluoride ions in the APF for remineralization. Again, close-fitting-trays are recommended and post-application instructions are similar to those described above. However, the low pH of the product is contra-indicated in patients who have teeth restored with glass ionomer cements and with indirect ceramic restorations.

Directions for APF:

- Prophylaxis of the teeth is not recommended.
- The gel should be wiped into 'thermo-formed' close-fitting trays (as described above) and applied for 1 min.
- The excess is expectorated for 30 s after the trays are removed. A salivary ejector should be used during the procedure.
- Do not eat or drink for 30 min after application.

In vitro studies have consistently shown that bivalent metal-ion fluorides, such as *Stannous Fluoride (SnF2)* (Table 9.1), can protect tooth surfaces from acid attack. Although it has been attributed to the remineralization effects of fluoride, in reality these products provide their effect by the deposition of stannous fluorophosphate (and stannous oxide) precipitate on the tooth surface that is highly insoluble to acid. This occurs when stannous attaches to free phosphate sites on the enamel surface. This precipitate is also effective in obturating patent dentinal tubules, thereby eliminating hypersensitivity.

Where the stannous products are presented as toothpastes (e.g. ProHealth products), they can be used professionally as prophylaxis pastes, and where they are available as gels (e.g. Gel-Kam) they can be used professionally (similar to the gels described above) in closely fitting trays to ensure complete coverage (even though most gels are applied with a finger or toothbrush during home application).

9.3.1.2 Fluoride Varnishes

Extending the physical presence of fluoride can also occur when the fluoride is used as a varnish (Table 9.1). Varnishes are high-concentration fluorides (e.g. 5 %) that provide more effective remineralization because of superior 'contact time', allowing more calcium and phosphate from saliva to be utilized due to a longer period of salivary flow. Unused or excess fluoride is still discarded by swallowing. There are differences of opinion about the use of fluoride varnish. Professionally directed spot application onto very active and affected areas, supplemented with home fluoride use, seems a common approach, while other operators recommend complete coverage. Although the positive effects of these fluorides are based on research with high caries risk patients, evidence on their effects on erosion is slowly becoming clearer. Within an erosive environment, varnishes on their own are effective in erosive wear (i.e. combination of acid and mechanical action) [17]; however their long-term effect seems uncertain and should be supplemented with a home preventive program.

Directions for varnish application:

- Prophylaxis of the teeth is not recommended.
- Varnish is applied manually with a finger or brush after the mouth is dried, using gauze, cotton rolls and air. It sets when in contact with saliva.
- The patient is advised not to eat or drink for 30 min after application and should not brush until the following day.
- There are no current reliable clinical data indicating the number of required applications per year for erosion.

Although the fluoride content of varnishes is traditionally high, the amount ingested when compared to high concentration gels and foams is comparatively low because of the slow fluoride release, making it preferable for younger children below 10 years of age.

9.3.2 Professionally Applied Calcium-Containing Technologies

Products with all the components required for the remineralization of teeth have definite benefits; however, the biggest obstacle that had to be overcome by manufacturers was to keep highly reactive calcium, phosphate and fluoride ions apart and prevent them from precipitating during storage, yet make them bio-available when applied in the oral environment. The general approach taken by manufacturers mimics saliva where statherins and proline-rich proteins naturally stabilize calcium, preventing precipitation, yet allowing the ions to become bio-available when the pH drops in the oral environment. There are a number of different technologies that allow this to happen and the main ones include Recaldent (CPP-ACP) technology, Tri-calcium phosphate (TCP) technology and other types of amorphous calcium phosphate (ACP) technologies.

9.3.2.1 Recaldent (CPP-ACP) Technology

One group of products that include calcium and phosphate for remineralization are those with the Recaldent technology (i.e. CPP-ACP: casein phosphopeptide – amorphous calcium phosphate). Here the calcium is stabilized by the protein CPP, preventing precipitation during storage within the dispensing tube. After application, when the oral pH drops during an acid challenge, the calcium is released from the CPP providing supersaturated conditions and therefore remineralization [18]. Fluoride can also be included in the right proportions to the Recaldent to provide more effective remineralization without fluoride wastage. CPP-ACP has the ability to bind to tooth, tooth pellicle (biofilm), bacteria and to soft tissues, and they bind better if held in situ for longer. There is a lot of clinical evidence supporting the remineralizing efficacy of recaldent™ [19]. CPP-ACP products that include fluoride would be the preferred option for erosion. These products are safe and can be used with patients who are lactose intolerant. However, they are not recommended with patients

who have milk protein allergy. Products containing CPP-ACP come as patient-applied pastes as well as professionally applied varnish (Table 9.1). This chapter discusses only the professionally applied varnish (MI Varnish, GC America; GC Australia).

MI Varnish (GC America; GC Australia) is a 5 % sodium fluoride varnish incorporating Recaldent (CPP-ACP) (Table 9.1). Here the calcium, phosphate and fluoride are released slowly due to the greater contact time provided by the very nature of the varnish. It can be used in spot application, particularly over eroded areas or throughout the mouth and supplemented with Tooth Mousse Plus (GC Australia) or MI Plus (GC America) for home application.

Directions for MI Varnish application:

- Prophylaxis is not recommended (keep any biofilm that is on the teeth).
- Do not dry the teeth
- Apply the varnish directly to the tooth surfaces with a brush (or by tooth brushing).
- The patient is asked not to eat or drink for 30 min after application.

9.3.2.2 Tri-Calcium Phosphate (TCP) Technology

This technology also provides a method where calcium, phosphate and fluoride are all included in the oral environment for more effective remineralization. In order to achieve this, tri-calcium-phosphate is bio-actively isolated from fluoride to prevent premature reaction and instant precipitation during storage within the dispensing tube [20]. Once applied in the presence of saliva, the TCP and fluoride become bioavailable for remineralisation. Evidence for the benefits of TCP is mounting. Placebo-controlled clinical studies have demonstrated that relative to fluoride alone the combination of fluoride plus functionalized TCP can improve remineralization of eroded enamel [21, 22].

The products that include this technology and that can be used for professional application are 5 % sodium fluoride varnishes with TCP (Table 9.1).

Directions for applying the varnish:

- Prophylaxis is not recommended (keep any biofilm that is on the teeth).
- Do not dry the teeth.
- Apply the MI varnish directly to the tooth surfaces with a brush.
- The patient is asked not to eat or drink for 30 min after application.

9.3.2.3 Other Calcium Technologies

There are numerous technologies that are now competing in the market, specific for the management of hypersensitivity and for remineralisation. Some include ACP technology in the form of a varnish (Table 9.1) that is applied as directed with varnishes previously; however, the majority of these products are aimed at regular home use and are not detailed in this chapter.

9.4 Tooth Surface Protection (Prophylactic Coatings)

9.4.1 Surface Protective Coatings with Remineralizing Potential

Currently, glass ionomer cements (GIC) and resin-modified glass ionomer cements (RMGIC) are available that were specifically designed as 'tooth crown and surface sealants' for caries protection but in more recent times have been used as protective coverings against erosion (Table 9.2). In order to maximize the remineralizing potential of these GICs and RMGICs, calcium and phosphate have been added by manufacturers using CPP-ACP, TCP and ACP technologies described previously in this chapter (Table 9.2).

All these products have the ability to obturate patent dentinal tubules, thereby providing instant relief to patients with hypersensitivity, providing remineralization of the affected surface and providing a temporary barrier to subsequent acid challenge. Purely by their nature, they are not resilient against acids; however as

Table 9.2 Examples of products that can be used as prophylactic surface coatings in uncontrolled erosion

Generic product	Name	Details	Company
GIC	Fuji VII	Surface protection	GC (AUS)
–	Fuji Triage	Surface protection	GC (US) (EUR)
RMGIC (varnish)	Clinpro XT	Surface protection	3mESPE
GIC (with CPP-ACP)	Fuji VII EP	Surface protection	GC (AUS)
RMGIC (with TCP)	Clinpro White Varnish	Surface protection	3 M ESPE
GIC (with ACP)	Riva Protect	Surface protection	SDI (AUS)
Nano-filled light cured self-adhesive	G-Coat Plus	Surface and GIC protector	GC (AUS)
Single step tooth adhesive	G-Aenial Bond	Surface protection (add surface resin coating on top)	GC (AUS)
Single step tooth adhesive	Scotchbond Universal	Surface protection (add surface resin coating on top)	3 M ESPE
Resin	*Seal & Protect*™	Surface protection	Dentsply, Weybridge, UK
Resin	*Optibond Solo*™	Surface protection	Kerr Corporation, USA
Resin	*Fissure sealant*	Surface protection	
Highly filled resin	*Pro Seal*	Surface protection	Reliance Inc., USA
Highly filled resin	*BisCover LV*	Surface protection	BISCO, Schaumberg, Ill
Highly filled resin	*Opal Seal*	Surface protection	Ultradent, Salt Lake city, Utah, USA
Highly filled resin	*SeLECT*™ *Defense surface sealant*	Surface protection	Element34 Technology, TX, USA

sacrificial coverings they have their place in dentistry. These products generally have a low viscosity allowing them to flow easily on surfaces and they can all be re-applied as necessary.

In addition, these products are moisture tolerant making them ideal in areas where moisture controls is difficult. The longevity of these materials will depend on the degree of mechanical force acting on the teeth (e.g. masticatory forces or the toothbrush), but the expectation should be that they can be easily re-applied as necessary. The ion-exchange zone (chemical bond) occurring at the tooth-GIC interface is a far more acid resistant surface when compared to either the tooth or the material alone. Even if the GIC is removed over time, a far more acid-resistant surface remains, adding some protection.

The added advantage in the use of these products is the fluoride release they provide to their immediate environment and their potential to act as fluoride reservoirs where they can be recharged with the addition of fluoride. Recent randomized clinical trials confirm that fluoride-releasing sealants provide benefits to adjacent teeth [23] from a caries perspective. By inference, we would expect such benefits with respect to erosion; however independent studies will be required in the future to confirm this.

Although manufacturers' instructions should strictly direct their application, the steps below summarize their application.

Directions:

- Prophylaxis is required to remove any pellicle (biofilm) that may inhibit chemical adhesion.
- The tooth surface should be conditioned (as per manufacturer's instructions) to further clean and prepare the surface for ion exchange. The conditioner should be washed and the tooth surface dried but not desiccated.
- Mix and dispense the material onto a pad and then apply to the tooth surface thinly but evenly with a microbrush.
- The materials are light cured. Some rely on heat from the curing light to speed up curing (e.g. Fuji VII, Fuji VII EP) while others based on RMGIC rely on photo-initiators to cure the product.
- Placement of a hard covering over the cured surface will protect the product and add to the longevity of the material by providing a more durable surface barrier that resists wear (e.g. G-coat Plus: nano-filled, light cured coating: GC International) (Fig. 9.2). It should be remembered however that although such resilient coatings have benefits as far as longevity is concerned, these barriers may also prevent or limit fluoride release to the adjacent teeth and restrict some fluoride benefits. If this is an issue, then these GICs and RMGICs need only be protected from the egress and ingress of water while setting and during early maturation (2–7 days), following the manufacturer recommendations.

Although the above products are specifically made to thinly cover and protect all tooth surfaces, conventional RMGICs can also be used as a last resort if the dedicated products are not available. They are applied as per the manufacturer's instructions but their problem lies in their relative higher viscosity. To overcome this, once

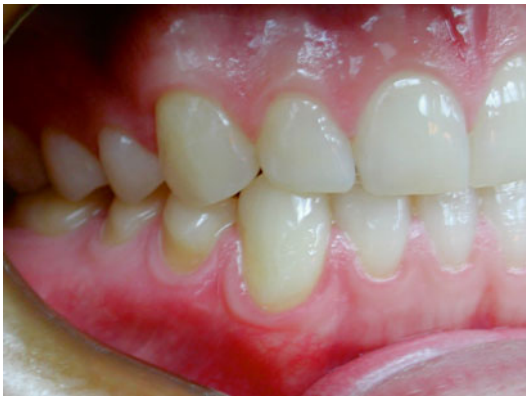


Fig. 9.2 Very active erosion with hypersensitivity on the buccal aspect of 44 and 45. There is enough room for a thin layer of GIC or RMGIC surface covering, topped with G-Coat plus to add to the longevity of the initial coating

the material is mixed and placed on a mixing slab, the material should be spread with a brush dipped in unfilled resin to aid in the process. In this way the material can be applied in a very thin layer.

9.4.2 Surface Protective Coatings Without Remineralizing Potential

There are many coatings available that do not have the ability to remineralize teeth, but when applied directly onto eroded tooth surfaces, they can form a barrier that is able to resist an acid challenge, obturate open dentinal tubules and immediately eliminate hypersensitivity. These are mainly resins that may contain some filler content and are similar to fissure sealants or typical adhesives. They are not the preferred option when compared to coverings that have remineralizing capabilities; however, they do have their place in dentistry, providing minimal protection anywhere from 3 to 9 months depending on their thickness and adhesive capability [24]. When placed in non-stressed areas (e.g. cervical areas and root surfaces), their longevity is improved; however, in dentin-scooped occlusal areas, the longevity of these products can be improved if kept out of occlusion. Some of these materials and associated applications can be referred to as 'clinician-generated' applications that are not originally designed for the purpose of the material's manufacture, yet do provide some protection when alternatives are not available.

In such situations, there are two basic questions that need to be considered by the clinician in the selection of the material. Is the material to be placed over enamel or dentin and how does the material bond to these different dental tissues? Based on these two questions, one must be careful in deciding whether light cured (unfilled) resin sealants should be used in these situations. On their own, they do not bond to dentin unless a primer is included in the technique (e.g. 'self-etch' system) and where enamel is involved, etching is recommended. Opinions vary as to why one would etch an already demineralized surface? In addition, it is not known if an

already eroded enamel surface can provide any micro-mechanical adhesion, especially where pellicle has covered the surface? Furthermore, products containing HEMA (2-hydroxyethyl methacrylate) may be contraindicated if used over dentin. The plasticization of the product from dentinal tubule moisture could markedly reduce the longevity of the product and the HEMA can be detrimental to the pulp.

In addition, although there are only a few studies that give insight to the longevity of these materials as protective coverings against acids [25, 26], it must be remembered that they are not restorative approaches, but are short-term applications that can last for a number of months and then be re-applied as necessary (Table 9.2). Few of these surface protective coverings are discussed below.

G-Coat Plus (GC International) is a nano-filled self-adhesive coating that is light-cured over glass ionomer cements and resin composites in order to make these materials more durable (Fig. 9.2). By thinly laminating the surface, the material adds fracture toughness, wear resistance, acid resistance and longevity to restorations from 6 months and up to 2 years. On its own, G-Coat Plus can be used over eroded dentin. It bonds to dentin (as it contains phosphoric ester monomer), overcoming hypersensitivity and providing protection against the next acid challenge. However, in order to bond to enamel, the surface needs mild etching with orthophosphoric acid before the G-Coat plus is applied and this again can be challenged. In order to overcome this, one-step adhesives (Table 9.2) can be used on both the enamel and dentin prior to the placement of a more durable coating such as G-Coat Plus. Directions for the use of many of these products will vary, so it is up to the clinician to follow the manufacturer's instructions.

With the objective to form a more resistant coating on the dental surfaces, an in vivo study evaluated the effectiveness of a dentin bonding agent marked as a desensitizing product, *Seal & Protect*™ (Dentsply, Weybridge, UK). The study showed that the application of Seal & Protect™ in subjects with palatal wear (reaching dentin) on anterior teeth could offer some protection against erosion, although it was limited to a period of 3 months [27]. In a study comparing the effect of fluoridated mouthrinse applied daily and that of Seal & Protect™ against erosive and abrasive challenges on dentin, more protection was observed with the bonding agent than the fluoridated mouthrinse [28].

An in situ study compared the effectiveness of another bonding agent, *Optibond Solo*™ (Kerr Corporation, USA) with that of Seal & Protect™ (Dentsply, Weybridge, UK). It was observed that both were able to protect the dentin surface against a vigorous erosion regimen, with Seal & Protect™ exhibiting the best effect [29].

In attempt to increase the survival rate of coating, a more durable material, a *fissure sealant*, was also tested regarding their ability to protect dentin wear for longer periods. An in vivo study showed greater wear for the untreated control teeth when compared to the sealed teeth up to a period of 9 months, indicating that fissure sealant may provide longer dentin wear protection than bonding agent [30].

There are other resin sealants that are more commonly used for prevention of demineralization around orthodontic brackets, protection of exposed root surfaces and other surfaces at risk of demineralization. Studies have reported from 73 to 100 % prevented fraction when used around orthodontic brackets [31–36]. Some of the commercially available resin sealant products are *Pro Seal* (Reliance Inc., USA), *BisCover*

LV (BISCO, Schaumberg, Ill.), *Opal Seal* (Ultradent, Salt Lake city, Utah, USA), and *SeLECT™ Defense surface sealant* (Element34 Technology, TX, USA). Pro Seal, SeLECT Defense and Opal Seal are impregnated with fluorescein®, which enables its presence to be detected by a UV light source (sold with Opal Seal). These materials provide a physical barrier to demineralization by an acidic challenge. In addition, further to providing a physical barrier, SeLECT™ Defense sealant (Element34 Technology, TX, USA) also has antimicrobial action against cariogenic plaque biofilms, and as such inhibit the adherence of bacteria and growth of dental plaque [37].

9.4.3 Surface Protection Using 'Bite-Guards'

'Bite-guards' should be fabricated for the following group of individuals to be worn during exposure to an acidic challenge. The inside (tooth surface) of the guard should be coated with a small amount of sodium bicarbonate powder or milk of magnesia to neutralize any acidic substance pooling inside. The guard should have occlusal coverage only, so that saliva flow can aid and maintain remineralization.

- Patients suffering from gastroesophageal reflux disease (GERD) should wear it while sleeping.
- When possible, patients suffering from eating disorders should wear it while vomiting or purging.
- Professional swimmers in ill-chlorinated swimming pools should wear it while swimming.
- Workers facing occupational exposure to erosive acid should wear it at work.

9.5 Oral Health Education to Prevent and Control Erosion

As described in Chap. 3 [38] of this book, one of the factors that can predispose individuals to the risk of dental erosion is their lifestyle. The conventional oral health education approach focuses on offering information and advice, which improve the patient's knowledge but may not translate into sustained changes in behaviour. Information and advice delivered through motivational interviewing has been demonstrated to empower people to adopt and sustain healthy behaviour [39–41]. Motivational interviewing is a patient-centred method for enhancing intrinsic motivation to change by exploring and resolving ambivalence. Individuals assess their own behaviours, present arguments for change and decide what behaviour to focus on while the counsellor helps to create an acceptable resolution that triggers change. Factors that might influence the patient's compliance such as the desire and willingness to change, the ability/skill to carry out the involved tasks, the financial costs to the patient, cultural issues and other personal factors must be considered and discussed with the patient. Counselling should be individualized and directed to risk factors predisposing that patient to dental erosion [1]. Some of the key points to consider as a guide are detailed in Chap. 8 of this book [42].

Conclusion

The use of professionally applied products for the prevention and control of dental erosion should be a part of an overall preventive management program that includes home management by the patient. Patient communication is essential, relying on patient understanding about the reason for their problem, the limitations of the program and what will happen if a preventive program is not put into practice.

Current direction includes the utilization of the pellicle/biofilm in the preventive process and the use of remineralizing agents with a full complement of raw products that have a maximum contact time when applied in the oral environment. However, most essential of all is to identify and eliminate or reduce the erosive challenge wherever possible. If this is not possible, prophylactive surface coverings are an essential step in maintaining tooth structure. This provides time for the erosive agent to be identified, and with the patient's help, eliminated. Opinion varies on the step-by-step use of these agents and it needs to be acknowledged that evidence from true independent double-blind crossover clinical trials does not exist for the majority of these products in an erosive environment.

Finally, although monitoring patient progress in not part of this chapter, it is an essential ingredient to good preventive management. Monitoring is discussed in detail in Chaps. 6 and 16 [43, 44].

Disclosure Statement The authors have no affiliation with any company or product referred to in this chapter. The authors have no conflict of interest to declare.

References

1. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33(3):243–52.
2. Yip K, Smales RJ, Kaidonis JA. Tooth erosion: prevention and treatment. New Delhi: Jaypee Bros; 2006. p. 1–118.
3. Lussi A, Ganss C. Erosive tooth wear: from diagnosis to therapy. In: Lussi A, Ganss C, editors. Monographs in oral science. Basel: Karger; 2014. p. 1–284.
4. Kaidonis JA. Oral diagnosis and treatment planning: part 4. Non-carious tooth surface loss and assessment of risk. Br Dent J. 2012;213(4):155–61.
5. Cheung A, Zid Z, Hunt D, McIntyre J. The potential for dental plaque to protect against erosion using an *in vivo-in vitro* model – a pilot study. Aust Dent J. 2005;50(4):228–34.
6. Hara AT, Ando M, González-Cabezas C, Cury JA, Serra MC, Zero DT. Protective effect of the dental pellicle against erosive challenges in situ. J Dent Res. 2006;85(7):612–6.
7. Hara AT, Zero DT. The potential of saliva in protecting against dental erosion. In: Lussi A, Ganss C, editors. Erosive tooth wear: from diagnosis to therapy. Basel: Karger; 2014. p. 197–205.
8. Hannig M, Hannig C. The pellicle and erosion. In: Lussi A, Ganss C, editors. Erosive tooth wear: from diagnosis to therapy. Basel: Karger; 2014. p. 206–14.
9. Meurman JH, Toskala J, Nuutinen P, Klemetti E. Oral and dental manifestations in gastroesophageal reflux disease. Oral Surg Oral Med Oral Pathol. 1994;78(5):583–9.
10. Millward A, Shaw L, Harrington E, Smith AJ. Continuous monitoring of salivary flow rate and pH at the surface of the dentition following consumption of acidic beverages. Caries Res. 1997;31(1):44–9.

11. Jones L, Lekkas D, Hunt D, McIntyre J, Rafir W. Studies on dental erosion: an *in vivo-in vitro* model of endogenous dental erosion – its application to testing protection by fluoride gel application. Aust Dent J. 2002;47(4):304–8.
12. Featherstone JD, Glena R, Shariati M, Shields CP. Dependence of in vitro demineralization of apatite and remineralization of dental enamel on fluoride concentration. J Dent Res. 1990;69(Spec No):620–5; discussion 634–6.
13. Imfeld T. Prevention of progression of dental erosion by professional and individual prophylactic measures. Eur J Oral Sci. 1996;104:215–20.
14. Lussi A, Ganss C. The role of fluoride in erosion therapy. Erosive tooth wear: from diagnosis to therapy. In: Monographs in oral science. 2nd ed. Basel: Karger; 2014. p. 230–43.
15. Mok TB, McIntyre J, Hunt D. Dental erosion: *In vitro* model of wine assessor's erosion. Aust Dent J. 2001;46(4):263–8.
16. Attin T, Deifuss H, Hellwig E. Influence of acidified fluoride gel on abrasion resistance of eroded enamel. Caries Res. 1999;33(2):135–9. Karger Publishers.
17. Vieira AR, Jager DH, Ruben JL, Huysmans MC. Inhibition of erosive wear by fluoride varnish. Caries Res. 2007;41:61–7.
18. Cochrane NJ, Cai F, Huq NL, Burrow MF, Reynolds EC. New approaches to enhanced remineralization of tooth enamel. J Dent Res. 2010;89:1187–97.
19. Cochrane NJ, Reynolds EC. Calcium phosphopeptides — mechanisms of action and evidence for clinical efficacy. Adv Dent Res. 2012;24:41–7.
20. Karlinsey RL, Mackey AC, Walker ER, Frederick KE. Preparation, characterization, and in vitro efficacy of an acid-modified β-TCP material for dental hard-tissue remineralization. Acta Biomater. 2010;6:969–78.
21. Karlinsey RL, Pfarrer AM. Fluoride plus functionalized β-TCP: a promising combination for robust remineralization. Adv Dent Res. 2012;24:48–52.
22. Amaechi BT, Karthikeyan R, Mensinkai PK, Narjibfard K, Mackey AC, Karlinsey RL. Remineralization of eroded enamel by a NaF rinse containing a novel calcium phosphate agent in an in situ model: a pilot study. Clin Cosmet Invest Dent. 2010;2:93–100.
23. Cagetti MG, Carta G, Cocco F, Sale S, Congiu G, Mura A, et al. Effect of fluoridated sealants on adjacent tooth surfaces: a 30-mo randomised clinical trial. Clin Suppl J Dent Res. 2014;93(7):59S–65S.
24. Buzalaf MA, Magalhaes AC, Wiegand A. Alternatives to fluoride in the prevention and treatment of dental erosion. In: Lussi A, Gunss C, editors. Erosive tooth wear: from diagnosis to therapy. 2nd ed. Basel: Karger; 2014. p. 244–52.
25. Wegehaupt FJ, Tauböck TT, Attin T. Durability of the anti-erosive effects of surface sealants under erosive abrasive conditions. Acta Odontol Scand. 2013;71:1188–94.
26. Wegehaupt FJ, Tauböck TT, Sener B, Attin T. Long-term protective effect of surface sealants against erosive wear by intrinsic and extrinsic acids. J Dent. 2012;40(5):416–22.
27. Sundaram G, Wilson R, Watson TF, Bartlett D. Clinical measurement of palatal tooth wear following coating by a resin sealing system. Oper Dent. 2007;32:539–43.
28. Sundaram G, Wilson R, Watson TF, Bartlett DW. Effect of resin coating on dentine compared to repeated topical applications of fluoride mouthwash after an abrasion and erosion wear regime. J Dent. 2007;35:814–8.
29. 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;196:351–4; discussion 339.
30. Bartlett D, Sundaram G, Moazzez R. Trial of protective effect of fissure sealants, in vivo, on the palatal surfaces of anterior teeth, in patients suffering from erosion. J Dent. 2011;39:26–9.
31. Bergstrand F, Twetman S. A review on prevention and treatment of post-orthodontic white spot lesions – evidence-based methods and emerging technologies. Open Dent J. 2011;5:158–62.
32. Heymann GC, Grauer D. A contemporary review of white spot lesions in orthodontics. J Esthet Restor Dent. 2013;25(2):85–95. doi:10.1111/jerd.12013. Epub 2013 Feb 19. Review.

33. Derks A, Katsaros C, Frencken JE, Van't Hof MA, Kuijpers-Jagtman AM. Caries-inhibiting effect of preventive measures during orthodontic treatment with fixed appliances. a systematic review. Caries Res. 2004;38:413–20.
34. Banks PA, Richmond S. Enamel sealants: a clinical evaluation of their value during fixed appliance therapy. Eur J Orthod. 1994;16:19–25.
35. Heinig N, Hartmann A. Efficacy of a sealant : study on the efficacy of a sealant (Light Bond) in preventing decalcification during multibracket therapy. J Orofac Orthop. 2008;69:154–67.
36. Wenderoth CJ, Weinstein M, Borislow AJ. Effectiveness of a fluoride-releasing sealant in reducing decalcification during orthodontic treatment. Am J Orthod Dentofacial Orthop. 1999;116:629–34.
37. Tran P, Hamood A, Mosley T, Gray T, Jarvis C, Webster D, Amaechi B, Enos T, Reid T. Organo-selenium-containing dental sealant inhibits bacterial biofilm. J Dent Res. 2013;92(5):461–6.
38. Scaramucci T, Carvalho JC, Hara AT, Zero DT. Causes of dental erosion: intrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 35–67.
39. Gao X, Lo ECM, Kot SCC, Chan KCW. Motivational interviewing in improving oral health: a systematic review of randomized controlled trials. J Periodontol. 2014;85(3):426–37.
40. Resnicow K, McMaster F. Motivational Interviewing moving from why to how with autonomy support. Int J Behav Nutr Phys Act. 2012;9:19.
41. Lundahl B, Burke BL. The effectiveness and Applicability of motivational interviewing: a practice-friendly review of four meta-analyses. J Clin Psychol. 2009;65:1232–45. In session.
42. Buzalaf MA, Cardoso CA, Magalhães AC, Amaechi BT. Prevention and control of dental erosion: patient self-care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 133–50.
43. Amaechi BT. Assessment and monitoring of dental erosion. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 111–9.
44. Amaechi BT. Recall, maintenance care cycle, and outcomes assessment. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 303–11.

Prevention and Control of Dental Erosion: Dietary Management

10

Georgiana S. Gross and Bennett T. Amaechi

Abstract

This chapter deals with dietary management to prevent the development of dental erosion in an individual. It discussed the advice that a dental practitioner or health professional should provide to the patient following the confirmation of dietary factors as the cause of the patient's erosive tooth wear. Examples of patient assessment forms such as a Food Frequency form and a 3-day dietary recall form are provided. A method of analysis of food consumption was addressed. A case study is included along with recommendations for changing the dietary components to address the issues. Patient education material is provided to determine extrinsic sources of acids i.e., food, drink, and supplements. Diet counseling is given after a thorough assessment of the diet and is tailored to the individual, emphasizing the positive aspects. Acidic foods and drinks should be taken only at mealtimes. Finish the meal with something to neutralize acid, like cheese or milk. As salivary flow is almost nil at night, advise the patient to avoid acidic food and drink especially before going to bed. Advise that toothbrushing should be done either before a meal or be delayed at 60 min.

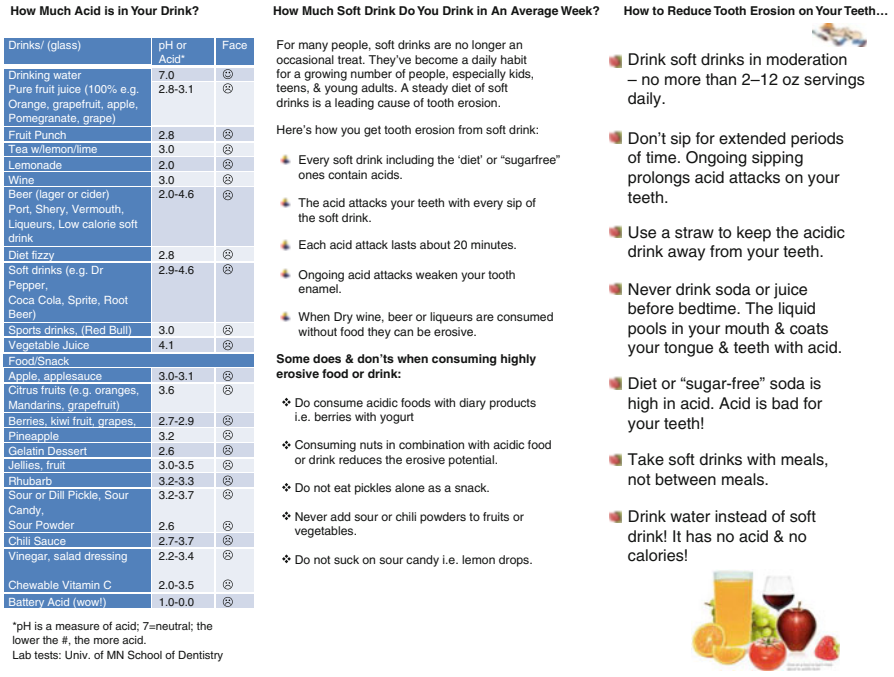
G.S. Gross, MPH, RD, LD (✉) • B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry,
University of Texas Health Science Center at San Antonio,
7703 Floyd Curl Drive, San Antonio,
TX 78229-3900, USA
e-mail: grossg@uthscsa.edu; amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_10

10.1 Introduction

Dental erosion is an acid-associated dental hard tissue disorder. Acids that could cause dental erosion can originate from gastric, dietary, or environmental sources [1, 2]. Based on this fact, diet and dietary factors have been identified as one of the major predictors of an individual's susceptibility to dental erosion, and this is discussed in detail in Chap. 4 of this book [2]. Categories of foods associated with dental erosion and their associated pH are shown in Fig. 10.1 [3].

An important step towards management of dental erosion in a patient is determination of the dental erosion risk status of the patient. Through the process of risk assessment, the diet and the dietary factors predisposing the patient to the risk of dental erosion will be identified. So in this chapter we should first discuss the tools and procedures that can be used during the risk assessment exercise to enable identification of the dietary factors causing the patient's dental erosion. Then we should discuss the dietary advice to prevent development of new erosion lesions or progression of existing erosion lesions in a patient.



How Much Acid is in Your Drink?

Drinks/ (glass)	pH or Acid*	Face
Drinking water	7.0	☺
Pure fruit juice (100% e.g. Orange, grapefruit, apple, Pomegranate, grape)	2.8-3.1	☹
Fruit Punch	2.8	☹
Tea w/lemon/lime	3.0	☹
Lemonade	2.0	☹
Wine	3.0	☹
Beer (lager or cider) Port, Shery, Vermouth, Liqueurs, Low calorie soft drink	2.0-4.6	☹
Diet fizzy	2.8	☹
Soft drinks (e.g. Dr Pepper, Coca Cola, Sprite, Root Beer)	2.9-4.6	☹
Sports drinks, (Red Bull)	3.0	☹
Vegetable Juice	4.1	☹
Food/Snack		
Apple, applesauce	3.0-3.1	☹
Citrus fruits (e.g. oranges, Mandarins, grapefruit)	3.6	☹
Berries, kiwi fruit, grapes,	2.7-2.9	☹
Pineapple	3.2	☹
Gelatin Dessert	2.6	☹
Jellies, fruit	3.0-3.5	☹
Rhubarb	3.2-3.3	☹
Sour or Dill Pickle, Sour Candy, Sour Powder	3.2-3.7 2.6	☹ ☹
Chili Sauce	2.7-3.7	☹
Vinegar, salad dressing	2.2-3.4	☹
Chewable Vitamin C	2.0-3.5	☹
Battery Acid (wow!)	1.0-0.0	☹

*pH is a measure of acid; 7=neutral; the lower the #, the more acid.
Lab tests: Univ. of MN School of Dentistry

How Much Soft Drink Do You Drink in An Average Week?

For many people, soft drinks are no longer an occasional treat. They've become a daily habit for a growing number of people, especially kids, teens, & young adults. A steady diet of soft drinks is a leading cause of tooth erosion.

Here's how you get tooth erosion from soft drink:

- Every soft drink including the 'diet' or "sugarfree" ones contain acids.
- The acid attacks your teeth with every sip of the soft drink.
- Each acid attack lasts about 20 minutes.
- Ongoing acid attacks weaken your tooth enamel.
- When Dry wine, beer or liqueurs are consumed without food they can be erosive.

Some does & don'ts when consuming highly erosive food or drink:

- Do consume acidic foods with diary products i.e. berries with yogurt
- Consuming nuts in combination with acidic food or drink reduces the erosive potential.
- Do not eat pickles alone as a snack.
- Never add sour or chili powders to fruits or vegetables.
- Do not suck on sour candy i.e. lemon drops.

How to Reduce Tooth Erosion on Your Teeth…

- Drink soft drinks in moderation – no more than 2–12 oz servings daily.
- Don't sip for extended periods of time. Ongoing sipping prolongs acid attacks on your teeth.
- Use a straw to keep the acidic drink away from your teeth.
- Never drink soda or juice before bedtime. The liquid pools in your mouth & coats your tongue & teeth with acid.
- Diet or "sugar-free" soda is high in acid. Acid is bad for your teeth!
- Take soft drinks with meals, not between meals.
- Drink water instead of soft drink! It has no acid & no calories!

Fig. 10.1 List of foods and drinks with erosive potential and recommendations for prevention of erosion [3]

10.2 Identification of Dietary Factors Associated with a Patient's Dental Erosion

10.2.1 Food History

Taking a food history is vital in determining the source of the dietary factors predisposing the individual to the risk of dental erosion, including the type and frequency of intake of the food and drink. This is very important as the preventive advice can only be effective if it is personalized and directed towards the information obtained from this history. To be precise in capturing accurate information, a clear instruction, as shown in Table 10.1, should accompany the

Table 10.1 Typical food recall instructions

Do not change your eating habits while keeping your food/drink record
Record what you really eat and drink
Fill in the information *NOW* instead of relying on your memory
Keep a 3-day food record (including 1 weekend day)
Write down *EVERYTHING* (no matter how small)! Keep your form with you all day long and write down everything you eat and drink. A piece of candy, a handful of pretzels, a bottle of soda, or a small donut may not seem like much, but it's important that your dietitian knows about all foods and drinks that you have during these 3 typical days
Do it now! Don't depend on your memory at the end of the day. Record what you're eating as you go
Be specific. Make sure you include "extras," such as gravy on your meat, cheese on your sandwich, or vegetables, butter, and salad dressings
Estimate amounts. If you have a bowl of cereal, measure out or estimate the actual amount (rather than writing "bowl" of cereal). If you eat at a restaurant, just estimate as closely as possible
Be specific in how the foods were prepared (baked, broiled, fried etc.)
Include the brand name of the product used
If eating away from home, include the name of the place or restaurant
Be specific, for example, include salad dressings, gravy, and or sauces
Estimate amounts (hand and fingers can be used as measuring devices)
Write legibly, but don't worry about your spelling!
Your food record will help the clinical team understand which foods and how much you are actually eating right now. In other words, we need your help understanding your diet, in terms of what is *TYPICAL* and what is *TRUE*. It is very important to follow the guidelines below in order to help us. We really appreciate your help!
Don't change your eating habits while keeping your food record (this is the *TYPICAL* part)
Record what you really eat (this is the *TRUE* part)
You have been given enough copies of the food record form to record 3 days of dietary intake (including 1 weekend day). There are extra sheets so you will have plenty of room to write everything down
Please call or email one of your dietitians if you have *any* questions
Be sure to bring your completed 3-day food record with you to your next clinical appointment

Table 10.2 Three-day food record

How to Complete Your Three-Day Food Record Write down everything you eat and drink for three days. Choose any two weekdays and one weekend day. It's very important that we are able to understand exactly what and how much you ate on these days. Thank you very much for your help!						
Food Group	DAY 1		DAY 2		DAY 3	
BREAKFAST	Food Item	Serving Size	Food Item	Serving Size	Food Item	Serving Size
Grains						
Vegetables						
Fruit						
Milk Products						
Meat & Alt						
Other Foods						
Drinks						
LUNCH	Food Item	Serving Size	Food Item	Serving Size	Food Item	Serving Size
Grains						
Vegetables						
Fruit						
Milk Products						
Meat & Alt						
Other Foods						
Drinks						
DINNER	Food Item	Serving Size	Food Item	Serving Size	Food Item	Serving Size
Grains						
Vegetables						
Fruit						
Milk Products						
Meat & Alt						
Other Foods						
Drinks						
SNACKS	Food Item	Serving Size	Food Item	Serving Size	Food Item	Serving Size
Grains						
Vegetable						
Fruit						
Milk Products						
Meat & Alt						
Other Food/Drink						

forms for collecting 3-day dietary history. A typical food history should include week and weekend days, and this can be achieved using either a Dietary Food Record (Table 10.2) or a Food Frequency Questionnaire (Table 10.3) described below.

Table 10.3 Food Frequency Questionnaire

PLEASE PUT A TICK (✓) ON EVERY LINE									
FOODS & AMOUNTS	AVERAGE USE LAST YEAR								
DRINKS/(glass)	Never or less than once/month	1-3 per month	Once a week	2-4 per week	5-6 per week	Once a day	2-3 per day	4-5 per day	6+ per day
Pure fruit juice (100%) e.g. orange, grapefruit, apple, pomegranate, grape									
Fruit Punch									
Tea w/lemon/lime									
Lemonade									
Wine									
Beer, lager or cider (half pint)									
Port, sherry, vermouth, liqueurs									
Low calorie or diet fizzy soft drinks									
Fizzy soft drinks, e.g. Coca Cola, lemonade ,Sprite									
Sports drinks, "Red Bull"									
Vegetable Juice									
FOOD/SNACKS									
Apple, applesauce									
Citrus fruits e.g. oranges, mandarins, grapefruit									
Berries, kiwi fruit, Grapes, pineapple									
Gelatin dessert									
Rhubarb									
Sour or Dill pickle									
Sour candy									
Chinese candy									
Lucas									
Chili sauce									
Vinegar, salad dressing									
Chewable vitamin C									
	Never or less than once/month	1-3 per month	Once a week	2-4 per week	5-6 per week	Once a day	2-3 per day	4-5 per day	6+ per day
Please check that you have a tick (✓) on EVERY line									

10.2.1.1 Dietary Food Record

The Food Record (Table 10.2) is a method of collecting dietary information in diary format over a 3-day period, typically 2 weekdays and 1 weekend selection. It is well known that diet changes from the structure of weekdays to variability of weekend activities. Thus collecting a 3-day record gives a picture of food choice habits. Food information, including the quantities, should be recorded immediately or as soon as practicable. The food quantity can be estimated using the method shown in either Fig. 10.2 or 10.3. Some problems occur when patients do not fill in the diary as they go but instead depend on recall from the previous day. A major advantage of the

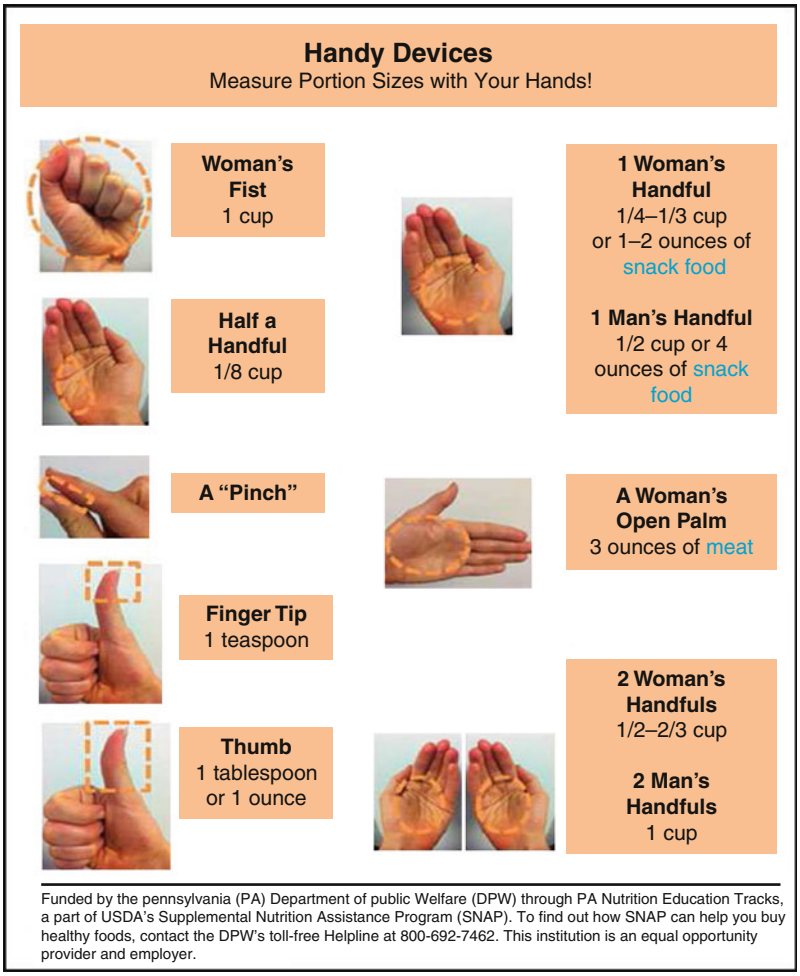


Fig. 10.2 Method for estimating "portion sizes" with your hands (Adapted from http://www.panen.org/EatTogetherPA/measure_terms)

3-day food record is that the patient's personal daily eating patterns are revealed. Disadvantage will always be the inaccuracy or misrepresentation of the recorded intake. One way to collect the information from the patient is to have it mailed to the dental office before the next visit. In this way the dentist may review and determine the source of the problem prior to the appointment.

10.2.1.2 Food Frequency Questionnaire

The Food Frequency Questionnaire (Table 10.3) is a checklist by which a patient identifies the particular food and drink that cause harm. This method of gathering dietary information requires the patient to take the record home and return by mail. The purpose in compiling this information is to reveal the type and amount consumed

Fig. 10.3 Method "size it right" for estimating the quantity of food taken (Adapted from http://authoritynutrition.com/contact/)

over time. The practitioner may interpret the data collected and address the erosive potential of the diet selections. An advantage of this type of data collection is that the interpreter may determine the patient's frequency of consumption of the offending food or drink. The disadvantage of this method of collecting dietary information is that a patient may not be accurate in his or her estimation of quantities. However, as stated above, the food quantity can be estimated using the method shown in either Fig. 10.2 or 10.3. It is important to stress the importance of accuracy as that would determine the type of dietary advice to be delivered by the dental professional.

10.2.2 Analysis of Dietary Record Data

Analysis of either the Dietary Food Record or the Food Frequency Questionnaire can be done during the normal course of a dental visit. The dentist should review the data to elicit the quantity, frequency, and times of consumption of the foods that

possess the factors associated with dental erosion, such as low pH, high titratable acidity, type of acid, etc. Chapter 4 of this book has the details on this [2]. This information will guide the dentist's advice to the patient and determine the program for behavioral modification. It is not necessary for the dentist to go into the detail of nutrient analysis; however, if the dentist is interested in nutrient content of the foods and drinks, there are different versions of nutrient analysis software approved by the USDA [4]. Only USDA-approved nutrient analysis software may be used to conduct nutrient analyses for patient advice and behavior modification program.

Case Study

Nora Gonzales is an active 20-year-old female who is attending college and shares a dormitory room with her roommate. Her dental history is unremarkable as she did not need dental treatment prior to leaving home. Because she finds college life stressful and does not always have time to eat regular meals, she sips on soda and sports drinks frequently during each day. She also has a habit of sucking on lemons with Lucas to help curb her appetite. Nora has noticed that her teeth are sensitive and appear to look different. She has presented to the dental office with early onset of enamel erosion. The dental practitioner begins with the interview process by asking Nora to fill out a Food Frequency Questionnaire and Food Record at home and mail it to the dental office. The practitioner has ruled out gastric symptoms and saliva-reducing drugs as the cause and determines that her problem stems from dietary habits. Finally, a plan is designed to address her specific diet recommendations. Health education material (Fig. 10.1) was provided to help her make an informed decision on the particular foods and drinks that should be eliminated or reduced.

10.3 Dietary Recommendations to Prevent Erosion

Upon review of the diet analysis and dietary habits of the patient, recommendations, tailored to the particular need of the patient, should be given. The following dietary advice is recommended to prevent the development of dental erosion.

1. Reduce the intake of highly acidic foods and drinks, and if possible limit their intake to mealtimes [5–8]. Such foods and drinks as:
   - Carbonated soft drinks to include diet and sports drinks
   - Fresh citrus fruit juices and fruit juice drinks
   - Wine, cider, and spirits consumed with mixers
   - Some herbal teas (citrus and berry types)
   - Fresh citrus fruits (if consumed in large quantities)
   - Vinegar, sauces, ketchup, pickles, and chilies
   - Acidic sweets such as fruit drops
   - Chewable vitamin C tablets
   - Acidic candy that is hard or sticky, instead use sugar-free gum
   - Powdered drinks with a tangy, fizzy, or acidic flavor

2. Reduce erosive impact of food and drinks by observing the following [9]:
   - Drink acidic drinks quickly and use a straw in order to reduce the contact of the drink with your teeth.
   - Do not swish drink around or hold them in your mouth for long periods.
   - After consuming food or drink that is high in acid content, rinse with water to dilute the acid, and wait an hour before brushing your teeth.
   - Or finish the meal with something to neutralize acid, like cheese or milk.
   - As salivary flow is almost nil at night, avoid acidic food and drink especially before going to bed.
   - Chew sugar-free gum to produce more saliva so your teeth can remineralize.
   - Brush with a soft toothbrush and be sure your toothpaste contains a high amount of fluoride.

10.4 Counseling Technique to Promote Behavioral Change in Health Care

Modifying the patient's behavior is the key to the success of treatment. To be effective and successful, recommendations for behavior modification in all patients should be delivered through motivational interviewing. Effective patient education and counseling increases patient understanding of information. This information enhances the patient's ability to integrate information into daily life and promotes a partnership approach to behavior change. Information and advice delivered through motivational interviewing has been demonstrated to empower people to adopt and sustain healthy behavior [10–12]. This method involves the 5 A's process developed by Glasgow et al. [13]. The 5 A's as related to changing dietary factors causing erosion are described thus (Fig. 10.4):

1. *Assess*: Current behavior pattern using assessment tools provided (Tables 10.1, 10.2 and 10.3).
2. *Advise*: Provide patient specific recommendations for behavior modification and health promotion such as shown in Fig. 10.1.
3. *Agree*: Use shared decision-making strategies that include collaborative goal setting. Incorporate patient's suggestions as to what foods to use as substitutions for the acidic ones. Utilize alternatives to your patient's current intake such as non-erosive food and drink snack substitutions (Table 10.4).
4. *Assist*: Offer strategies that include action planning and address any barriers to change. Help the patient identify barriers to making these changes by negotiating dietary changes.
5. *Arrange*: Review action plan and progress during next appointment to reinforce compliance. Follow-up goals should be planned together with your patient and reviewed at that appointment.

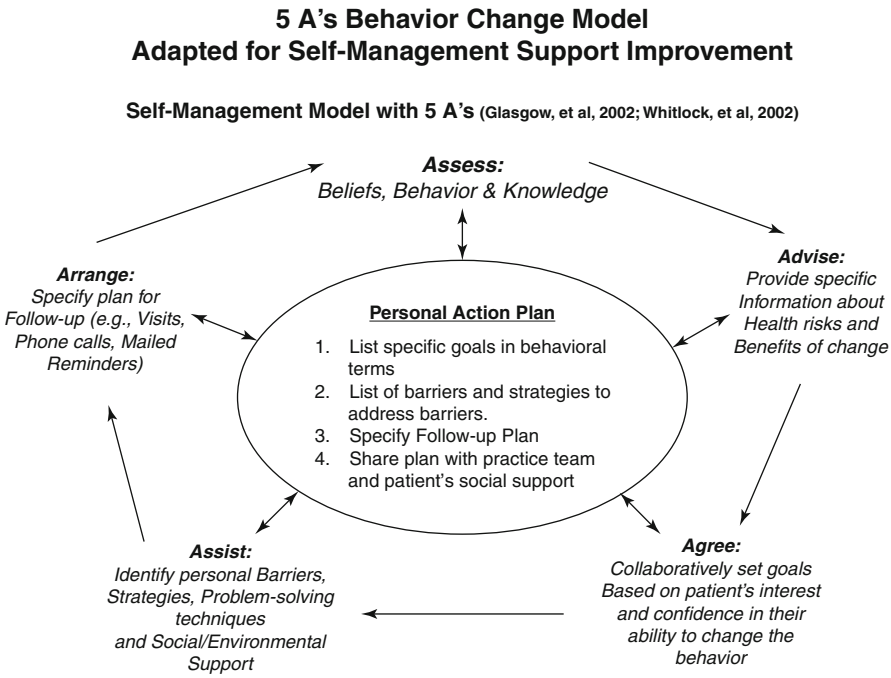


Fig. 10.4 The 5 A's behavior modification model (Reprinted from Glasgow et al. [13] by permission of Lawrence Erlbaum Associates, Inc.)

Table 10.4 Non-erosive food and drink snack substitutions

Substitute regular citrus fruit juice with calcium fortified citrus fruit juice
Between meals
Eat dairy products as snack foods, i.e., low-fat cheese, yogurt, milk, and ice cream
Coffee and tea without sugar may be consumed
Drink water instead of soda or sports drinks
After consuming an acidic food or drinks, rinse with water
Some non-acidic fruits are bananas, pears, dates, figs, melon, papaya, and lychee
High-fiber vegetables help with saliva production which neutralizes acids
Raw nuts provide calcium and phosphates which will strengthen the tooth surface

10.5 Summary on Diet Counseling

Counseling is given after a thorough assessment of the diet, and is tailored to the individual, emphasizing the positive aspects. Acidic foods and drinks should be taken only at mealtimes. Finish the meal with something to neutralize acid, like

cheese or milk. As salivary flow is almost nil at night, advise the patient to avoid acidic food and drink especially before going to bed. Advise that toothbrushing should be done either before a meal, or be delayed for 60 min.

References

1. Sc aramucci T, Carvalho JC, Hara AT, Zero DT. Causes of dental erosion: intrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 35–67.
2. Hara AT, Carvalho JC, Zero DT. Causes of dental erosion: extrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 69–96.
3. U.S. Food and Drug Administration. Approximate pH of foods and food products. 2008. Web. May 2014. Http://www.cfsan.fda.gov/~comm/lacf-phs.html.
4. USDA-approved nutrient analysis software. http://healthymeals.nal.usda.gov/menu-planning/nutrient-analysis-software-approved-usda/nutrient-analysis-software-approved-usda.
5. Moynihan P, Peterson PE. Diet, nutrition and the prevention of dental diseases. Public Health Nutr. 2004;7(1A):201–26.
6. Moynihan PJ. The role of diet and nutrition in the etiology and prevention of oral diseases. Bull World Health Organ. 2005;83:694–9.
7. Auad S, Moynihan P. Diet and dental erosion. Quintessence Int. 2007;38:130–3.
8. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33:243–52.
9. Academic of General Dentistry factsheet. http://www.agd.org/2/practicemanagement/factsheet/.
10. Gao X, Lo ECM, Kot SCC, Chan KCW. Motivational interviewing in improving oral health: a systematic review of randomized controlled trials. J Periodontol. 2014;85(3):426–37.
11. Resnicow K, McMaster F. Motivational Interviewing moving from why to how with autonomy support. Int J Behav Nutr Phys Act. 2012;9:19.
12. Lundahl B, Burke BL. The effectiveness and applicability of motivational interviewing: a practice-friendly review of four meta-analyses. J Clin Psychol. 2009;65:1232–45. In session.
13. Glasgow RE, Funnell MM, Bonomi AE, Davis C, Beckham V, Wagner EH. Self-management aspects of the improving chronic illness care breakthrough series: implementation with diabetes and heart failure teams. Ann Behav Med. 2002;24:80–7.

Prevention and Control of Dental Erosion: Psychological Management

11

Gracemary Leung and Bennett T. Amaechi

Abstract

Dentists often come across patients with psychological difficulties that underlie their dental problems, and dealing with these may be an integral part of their treatment. This chapter explains how anxiety, fear, depression, obsessive-compulsive problems, and eating disorders may prevent these patients from seeking dental treatment or reluctant to learn how to improve on their dental health. It is important to emphasize that knowledge and skills involved in the psychological assessment and management can play an important part in the prevention of dental erosion. Psychoeducation leaflets are recommended to help them improve their future dental health. Dental schools can incorporate relevant psychological training in their core curriculum.

11.1 Introduction

Although not immediately obvious, the dentists' task of managing oral diseases is often made more difficult by subtle psychological factors. What psychological difficulties are there in addition to the readily observed fear or anxiety related to visiting dental clinic? An answer may be seen by considering dental erosion. It is now

G. Leung, PhD, MSc, MBA, CPsychol, AFBPsS (✉)
Faculty of Social Sciences, The University of HK, Pok Fu Lam, Hong Kong

Focus PEC CO. Ltd., Sheung wan, Hong Kong
e-mail: leunggmk@hku.hk

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_11

understood that dental caries, caused by acid of bacterial origin, is not the only acid-dependent dental disorders. Dental erosion, a dental hard tissue disorder, is now known to be caused by acids of nonbacterial origin. Acids that cause dental erosion are of intrinsic (gastric) and extrinsic (dietary and environmental) origin; thus, the etiology of dental erosion is multifactorial, involving chemical, biological, and behavioral factors [1, 2]. Relevant to the present discussion is the fact that an individual's personality trait or psychopathology predisposes him/her to prefer foods or drinks with acidic content or exhibit psychological illness associated with vomiting or regurgitation of gastric acid into the oral environment and in contact with the teeth.

Many individuals with eating disorders have dental problems. Why may eating disorders lead to dental problems? How can a dentist tell that a patient is suffering from eating disorders? This brief introduction suffices to show that dental treatment may implicate psychopathology.

11.2 Psychology and Dental Problems

Psychology may be linked to tooth erosion directly or indirectly. Consider first how psychological factors affect dental health directly.

11.2.1 Direct Linkage

A common psychological factor is stress. It affects the operation of some body organs and the endocrine system.

11.2.1.1 Psychological Stress

The brain influences our immune system, as witnessed by the fact that we are more prone to catching colds, viruses, and infections when we are under stress. By the same token, dental problems may also erupt more readily when an individual is under stress. We tend to feel more anxious and entertain negative thoughts when we cannot cope with life stresses. For example, if one is anxious, one may conclude that one must have offended one's friend who does not come to dinner as expected. Consequently, our coping skills become less effective. We become more lethargic towards life and daily chores. Our immune system also mimics these behavioral symptoms; it becomes less effective.

Hans Selye [3] described the difficulties and strains experienced by organisms as they struggled to cope with, as well as adapt to, changing environmental conditions.

Both good and bad stresses would tax an individual's problem-solving potentials and coping skills.

When confronted with a threat or danger (be it real or perceived), the body undergoes a series of biological changes. The stress response starts in the hypothalamus that stimulates the sympathetic nervous system (SNS). The SNS, in turn, causes the adrenal medulla to secrete adrenalin and noradrenalin. While high levels of cortisol levels are beneficial in the short term, they are harmful in the long term.

Negative emotional states can impair the functions of the immune and the cardiovascular systems, leaving the persons more prone to infections and diseases. As the effects of stressful experiences are cumulative, prolong exposure to stresses sensitizes the individual's immune system. The individual thus becomes more reactive to subsequent stresses. There is hence a perpetuating sequence of increasing sensitivity (to stress) and weakening of the immune system.

11.2.1.2 Anxiety

Anxiety is a condition in which an individual experiences severe fear (of various objects or life events) that the individual has panic attacks. Some may suffer from anxiety without any specific instigating stimulus or event (called this "general anxiety"). Symptoms may be ill-defined discomfort or malaise. The individual cannot sit still or cannot eat or does not sleep well. These individuals may use alcohol or medication or chemical substances to calm themselves.

In any case, the individual's autonomic nervous system anticipates a "fight" or a "flight" reaction in which there is stomach churning or nausea. In short, the body produces more adrenalin. For some, visiting the dentist is one of those fear-inducing events. The person with anxiety tends to conjure up specific fearful images (i.e., injection fear, cleanliness of instruments, infections) which may set off a panic attack, after which she/he may learn to avoid attending dental checkups.

11.2.1.3 Obsessive-Compulsive Disorders

People suffering from obsessive-compulsive problems often have an anxiety predisposition. They can be very obsessive about how they look, particularly, how their teeth feature in their general appearance. Fearing that people may laugh at them because their teeth do not look perfect, they may coerce their dentists to make their teeth perfect even though there is no defect at all. In extreme cases, they may ask the dentist to remove the whole set of their healthy teeth and to choose to have dentures instead. However, more often than not, problems with their teeth are the result of their having excess of acidic drinks or sweets. At the same time, their unhealthy dietary preference is due to the underlying feeling of being depressed.

An example of obsessive-compulsive disorder is the case with Marianne, 40 years old. She has been suffering from an obsessive-compulsive problem. Her worry is that she/he might catch HIV through contamination and might die because of HIV. Her daily thoughts and routines center around ways to avoid going out to any public places (including hospitals, doctors, and dentists). She avoids any injection or any surgical investigations. At the same time, she/he is obese because she/he eats a lot, drinks a large quantity of sodas, and does not do any exercise. She has been told that she/he may have diabetes. As her/his personal and dental hygiene are poor, it is not surprising that she/he has dental problem (probably tooth erosion). Yet, she/he would not visit any dentist for fear of contamination.

11.2.1.4 Depression

Depression is a common problem encountered by individuals who feel that their lives are on the whole pretty negative. Some of them may have suicidal ideation.

The individual's chronic negative mind-set and emotive states affect the biological mechanisms which tend to "shut down," resulting in a less effective capability for dealing with infections or diseases.

The physical symptoms of depression include poor sleep, low motivation, less stamina, and poor appetite. Instead of having main meals, the sufferers tend to turn to eating snacks and soft drinks for a quick surge of energy. The individual thus has insufficient healthy and balanced nutrients. At the same time, the individual might turn to alcohol for relief.

People who are depressed also tend to ignore personal hygiene. They pay no, or less, attention to dental care or hygiene. They consequently have more oral infections. The regurgitation or vomits through chronic alcoholism or excessive stomach acids promote tooth erosion more readily.

An example of depression is Leonard, aged 55. He became depressed 10 months ago when his business went downhill, and he declared bankrupt. His mind kept searching and concentrated more on his past failures. He was not able to look at his own achievements. He became more moody and argued a lot with his wife and children. He did not want to get out of bed in the morning, and he had no motivation to do anything. Even though he used to enjoy food, he gave up eating after the onset of depression. Instead, he drank whisky and beers in order to "numb" his mind and he used alcohol to help him sleep. Leonard lost a lot of weight after 6 months. His health deteriorated as he neglected his hygiene more and more. He suffered stomach pains and vomited frequently as a result of excessive drinking. Excessive stomach acids damaged his teeth. When he was taken to hospital for treatment of his depression, his dental problem became an issue.

11.2.2 Indirect Linkage

Adverse past experiences can create distorted patterns of thinking. The resultant anxiety or misguided beliefs lead to avoidance and other maladaptive behavior.

11.2.3 Avoidance of Dental Visits

Like adults, children experience dental problems or have to have their teeth cleaned. What the child's experience is like depends on how the child (a) is taught about dental hygiene and (b) is prepared for the child's first visit to the dentist. For example, does the stranger in a white coat appear as a threat? Is sitting on a lovely swiveling dental chair a fun thing to do? Would the word "dentist" conjure up a negative picture involving needles and pain?

The process of psychological conditioning plays an important part in inducing fear in children. There are two types of dental fear, namely, subjective fear and objective fear.

Any child who has never been to a dentist may be fearful of dental clinic as a result of having been told by someone he/she trusts that going to the dentist is

painful. This type of fear may be characterized as "subjective" because it is based on someone else's subjective opinion. The second type is objective fear. For example, some children learn to be fearful of dental visit after the first visit when they have toothache. This is an example of fear induced by a bad memory of, or an unpleasant experience of, visiting the dentist.

Adolescents or adults with psychiatric difficulties, or psychological problems (such as depression, eating disorders, and anxiety), are more likely to resist visits to the necessary dental clinic because of their either subjective or objective fears. As anxiety or fear is a self-taught cognitive learning process, there is a need to change the patient's beliefs and attitudes in order to achieve appropriate dental care.

11.2.3.1 Classical Conditioning

Just one visit to the dental surgery (e.g., for tooth extraction) may be sufficient to instill fear in a child. The child remembers the pain of extraction, but not the reason why (viz., the toothache that requires dental attention). The dental clinic is the setting in which the child experiences pain. The mere mention or the sight or the memory of the clinic is sufficient to evoke the memory of pain. This is an example of classical conditioning (Pavlovian), much like the capability of the bell to elicit a dog's salivation when the bell has preceded the presentation of food pellets. The word "dentist" is like the bell, where painful process of tooth extraction plays the role of food pellets. The fear reaction is analogous to salivation.

Children who had a history of tooth extractions were three and half times more likely to be anxious than children who had no extraction history. Extraction under general anesthesia is quite traumatic for young children.

11.2.3.2 Operant Conditioning

While the fear of going to the dentist is the result of classical conditioning, avoiding the dentist is established by operant conditioning [4]. A behavior may be strengthened if it is followed by a reward. This phenomenon is called "positive reinforcement." By the same token, any behavior that removes a negative stimulus (e.g., pain) is also strengthened, a phenomenon called "negative reinforcement."

Having something else to do (instead of going to the dentist) is the negative reinforcer because it removes the anxiety or experience of pain. An individual can cancel a dental appointment by scheduling a "more important" appointment. This is an example of avoidance behavior. Such a psychological feat can maintain dental fear forever.

11.2.4 Overcoming Fear of Dental Visit

Some ways that may be helpful in overcoming the fear of visiting the dentist are the following:

- Find a dentist whom you can trust.
- Build up a therapeutic relationship.
- Ask the dentist questions about dental health or treatment.

- Feel comfortable to talk with your dentist.
- Be convinced that dental treatment is important to your health.
- Be optimistic that dental treatment will be successful.
- Learn to relax.
- Find a distraction (music) or imagine other pleasant pictures during dental treatment.
- Request sedative medication if everything else fails.
- Be conscientious and positive in looking after the teeth with regular checkups.

11.3 Eating-Related Maladaptive Behavior

Eating disorders (anorexia nervosa, bulimia nervosa) are recognized as a difficult-to-deal-with health concern. Such is the case because eating disorders often occur simultaneously with other forms of psychopathology. Consequently they tend to be missed because it is difficult to spot them [5]. Mortality rate for sufferers of eating disorders has been estimated to be 4.5 % [6] due primarily to medical complications. They tend to be resistant to treatment, either psychiatric or psychological. Be that as it may, over a third of the patients suffering from eating disorder have been diagnosed by dentists when these patients are treated for tooth erosion [7].

Anorexic and bulimic patients tend to adopt the chronic habits of purging or induced vomiting as a way of evacuating what they have eaten in order to avoid putting on weight. Stomach acids plus the high acidic content of foods would erode the surfaces of the enamel of their teeth. The front of their tooth is worn down and becomes thin. The next common complaints are discoloring of the tooth and heighten tooth sensitivity. Putting their fingers down the throat (not an uncommon behavior) causes soft palate damage.

Patients who purge tend to experience dry mouth as a result of the use of laxatives and diuretics. With recurrent vomiting, their bodies are poorly nourished with diminished minerals, vitamins, and proteins, which are needed to keep dental tissues healthy and clean; as a result bad breath is another common problem. Over an extended period of time, the anorexic and bulimic patients may also develop osteoporosis due to calcium deficiency. This medical problem leads to the shrinking of their jawbones.

Finally, a frequent binge and purge cycle can cause an enlargement of the salivary gland. Enlarged glands can be painful and are often visible to others; this causes further embarrassment to the persons with eating disorders.

11.3.1 What Are Eating Disorders?

Eating disorders are complex syndromes encompassing physical, psychological, and social features. There have been attempts to produce subcategories of this disorder so as to provide better understanding. The most significant split was between (a) sufferers who maintain a low body weight (anorexia nervosa) mainly by

restricting food intake and (b) individuals who resorted to vomiting [8]. Many of this latter group also show binge eating behavior. Another group resembled the latter bingeing and vomiting group but maintained normal body weight, emerged as bulimia nervosa [9, 10] in the third revision of the Diagnostic Statistical Manual (DSM-III) of the American Psychiatric Association [11].

11.3.1.1 Anorexia Nervosa

The term "anorexia nervosa" refers to a potentially life-threatening eating disorder characterized by an intense fear of gaining weight, a distorted body image, and amenorrhea [12]. Currently the *Diagnostic Statistical Manual* (5th edition) (DSM-V) sets out the Criteria for Anorexia Nervosa [13]. That the person has an intense fear of gaining weight or becoming fat, despite being underweight, thus undertakes persistent methods to stop any weight gain and refuses to maintain normal body weight of which she/he has no insight that it is a problem. There are two subcategories of anorexia nervosa: (1) restrictive type – the individual has no binge eating or purging behavior and (2) binge eating or purging type – the person engages in binge eating and purging behavior (i.e., self-induced vomiting, misuse of laxatives, diuretics, enemas) during the current episode.

Dentists may find it helpful to know the prevalence rate for as well as demographic features of anorexia nervosa.

(i) Females make up 90–95 % of cases.
(ii) Peak age of onset is between 14 and 18 years of age.
(iii) Often it escalates from dieting to anorexia.
(iv) Often during a stressful event (e.g., parental divorce, moving, or experiencing failure), dieting can escalate into anorexia.
(v) The motivation to become anorexic is often the fear of growing up.

11.3.1.2 Bulimia Nervosa

The term "bulimia nervosa" refers to a disorder marked by frequent eating binges followed by forced vomiting or other extreme compensatory behaviors to avoid gaining weight. The criteria for this disorder in DSM-V [13] suggest that the person feels she/he is not in control, consumed a definitely large amount of food within 2 h period, more than most people can eat, with inappropriate compensatory behavior, at least once a week for 3 months. There are also two subcategories of bulimia nervosa: (i) purging type – the person regularly engages in self-induced vomiting or misuse of laxatives, diuretics, or enemas and (ii) non-purging type – the person has used inappropriate compensatory behaviors such as fasting or exercise, but not self-induced vomiting.

The prevalence rate for, as well as demographic features of, bulimia nervosa is as follows:

(i) Women make up 90–95 % of cases.
(ii) Usually begins in adolescents or adulthood (between 15 and 21).
(iii) Weight usually remains within a normal range.

(iv) May have between 2 and 40 binges per week, though number is usually close to 10.
(v) Usually sweet, high calorie, typically soft food and drinks.
(vi) The binge eating starts usually and is preceded by feelings of overwhelming tension and followed by self-blame, shame, guilt, and depression.

It is estimated that about 1 in 250 females and 1 in 2000 males suffer from anorexia nervosa in terms of the UK Guidelines on Eating Disorders (2006). The phenomenon is seen generally in adolescence or young adulthood. About five times that number will suffer from bulimia nervosa. There are other atypical eating disorders, apart from anorexia nervosa and bulimia nervosa.

11.3.1.3 Other (Less Well-Known) Types of Eating Disorders

Relative to anorexia nervosa and bulimia nervosa, far less is known about *binge eating disorder* [14]. Apart from binge eating, the systematic profile of *binge eating disorder* overlaps little with other eating disorders. It presents itself much later after 20 and is noticeable in their 30s or 40s. It is difficult to define clinically what is bingeing or having a tendency to overeat with no dietary restraint. The sex ratio of *binge eating disorder* is more even. Many people with binge eating disorder are obese. Self-induced vomiting and laxative misuse are not present. Depressive features and dissatisfaction with shape are common, and the overevaluation of the weight and shape is less marked than in bulimia nervosa.

Some eating behaviors are so atypical that they do not fit in with the diagnostic criteria of anorexia or bulimia nervosa, but their weight is kept in the low normal range, and they do excessive exercise and kept an extreme dietary restraint. Many people with *atypical eating disorders* have suffered from anorexia nervosa or bulimia nervosa in the past.

11.3.1.4 Why Are There Eating Disorders?

Sensitivity to one's body image leads to dissatisfaction with one's body image. An instigating factor may be some negative comments on how the adolescent look. This distorted thinking is further perpetuated by fashion and fad that glorifies thinness. Contemporary societies seem to emphasize on "small is beautiful," as witnessed by TV advertising on various means of reducing one's weight. These TV advertisements introduce new diet drinks, giving people the idea that having diet drinks is the norm.

Anxious young persons with a perfectionistic personality are readily influenced by fashion and fads. One of my young patients, aged 10, watches obsessively every evening one TV program on weight loss. He is already choosing his mini-diet sheet. Eating disorders may be triggered by an emotional turmoil due to life crises (e.g., losing a boy friend). In order to "improve" their body image, individuals thus motivated would select a host of questionable diets. They may adopt excessive behaviors (e.g., taking up excessive exercise or using laxatives) in order to make themselves "acceptable." These problems stem from early days of their lives.

11.3.1.5 Effects of Eating Disorders on Self and Others

Family members of patients with eating disorders are always at a loss as to how to help the patients, or cope with the problems of being confronted with the phenomenon. One particular issue is that a prolonged period of anorexia nervosa leads to acute physical complications. Severe weight drop (i.e., under BMI 20) could result in mortality. Long-term effects on anorexia nervosa patients include depression, avoidance of relationships with others, poor academic or occupational functioning, infertility, and interpersonal problems, particularly with the parents.

11.3.1.6 Prognosis for Eating Disorders

Dealing with eating disorders is extremely difficult, let alone assessing the success rate of treatment. To begin with, many patients simply deny the problem and resist treatment. When they do receive treatment, they are reluctant to disclose information necessary for the treatment. Furthermore, there are insufficient services with experienced staff in the health sectors in some part of the countries. Unfortunately, a number of those with eating disorders are stigmatized by inexperienced clinical staff, and they are on occasion frightened of being trapped in treatment rather than helped by it.

Be that as it may, there are a few studies with a lengthy follow-up period of the course and outcome of bulimia nervosa in the community. With the most effective treatments, about 50 % of bulimia nervosa patients can be asymptomatic from 2 to 10 years after initial diagnosis. Twenty percent are likely to continue with the full form of bulimia nervosa, while 30 % may have remissions or relapses. Many people with bulimia nervosa are not receiving any form of help [15].

One 10-year follow-up study of 50 people with bulimia nervosa found that 52 % had fully recovered and only nine percent continued to experience symptoms of bulimia nervosa [16]. A larger study of 222 followed up for a mean of 11 years revealed that 11 % still met the criteria for bulimia nervosa whereas 70 % were in full or partial remission [17]. However, many will remain chronic or relapsed, maintained by their overvalued belief in the importance of appearance and thinness [18].

11.4 Effects of Eating Disorders on Dental Health

Eating disorder has effects on both soft and hard oral tissues, which can signal, to the dentist, the existence of eating disorder not revealed by the patient at the medical history taking. The most common effects on oral soft tissues are (i) dry mouth due to the use of laxatives and diuretics and (ii) enlargement of the parotid salivary glands in response to the repeated stimulus for an increased salivary flow rate due to frequent binge and purge cycle. Dental erosion (erosive tooth wear) is the most common effect on oral hard tissues. Erosive tooth wear as an effect of eating disorders is detailed in Chap. 3 of this book [1], and its prevalence is discussed in detail in Chap. 1 of this book [19]. Dental erosion is the wear of dental hard tissue by acids of dietary or gastric origin. As discussed above, eating disorder may be associated with bingeing on acidic foods and/or drinks followed by vomiting and/or purging. These

characteristics are associated with frequent bathing of the teeth with gastric or dietary acids over an extended period of time, with consequent wearing away of the dental hard tissue through acid demineralization, initially affecting the enamel (Fig. 5.3b), and with progression to advanced stage, dentin is exposed (Figs. 11a, b). Exposure of dentin will result to hypersensitivity in response to external stimuli of cold, hot, tactile, or osmotic nature. Acid of gastric juice, due to vomiting and/or purging, causes wear of the palatal surfaces of upper incisors (Fig. 12.4), and with lesion progressing, the lingual surfaces of premolars and molars become affected, and in more advanced stages, the process extends to the occlusal surfaces of molars and to the facial surfaces of all teeth [2, 4, 19]. This characteristic distribution is illustrated in Figs. 1.4 and 1.5 in this book. Erosion due to dietary acid has no specific distribution pattern but depends on factors such as method of application (Fig. 5.9). However, dental erosion due to eating disorder cannot be managed without dealing with the underlying psychological problem.

11.5 Training Health Professionals to Manage Eating Disorder Patients

It takes time and efforts to uncover eating disorder. However, primary healthcare professional often does not have sufficient knowledge or contact time with people with eating disorders. They are often reluctant to disclose their problems for fear of stigmatization. At the same time, research has found that individuals willing to talk about their difficulties are more likely to seek treatment. Professionals proficient in establishing rapports and who are being approachable and empathetic are essential for helping eating disorders patients. For these reasons, it helps to consider psychological assessment and management.

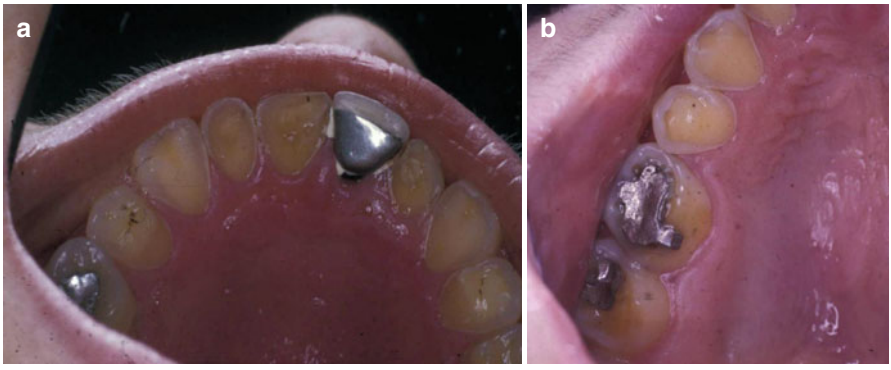


Fig. 11.1 Palatal surfaces of the upper (**a**) and lower (**b**) arches with advanced erosive wear exposing the dentin.

When confronted with a serious case of tooth erosion, the dentist needs to have the following information about the patient:

(a) How often does the patient have dental visits?
(b) Is the patient apprehensive of dental visits?
(c) What is the patient's psychological state of well-being ?
(d) What is the cause of the patient's dental erosion?

11.5.1 Rapport Building as Management

To build the necessary patient-dentist rapport, the dentist must show empathy with their patients. Specific rapport building techniques involve both verbal and nonverbal behaviors.

11.5.1.1 Verbal Behavior

(a) Speak softly to the patient.
(b) Ask relevant background questions.
(c) Give positive feedback for answers.
(d) Use gentle prompts to help the patient focus on the dental issues or to prevent the patient from digressing.
(e) Give indirect advice, rather than using condescending directives.
(f) For example, say "I am hearing what you are saying. I will try to understand what you are going through …" instead of saying "I understand." A skilful dentist is able to combine different interviewing styles within an encounter. Soliciting the dental history and psychological state of mind of a patient should be like a flow of a natural conversation.

11.5.1.2 Nonverbal Behavior

(a) Adopt an open mind.
(b) Focus on the patient as a person.
(c) Maintain a supportive, nonjudgmental attitude and demeanor towards the patient.
(d) Encourage the patient to be autonomous.
(e) Encourage the patient to make choices.
(f) Foster a sense of collaborative relationship in the treatment.

The aforementioned objectives may be achieved by (a) holding warm eye contact with the patient, (b) maintaining relaxed facial muscles, (c) smiling, and (d) occasional nodding to indicate that you are listening or empathizing with the patient's difficulties.

11.5.1.3 Engagement

The dentist must also consider the patient's feelings about, as well as the motivation to rectify, the dental erosion problems. This consideration is particularly important for patients with eating disorders. A "pushy" dentist would provoke resistance. In particular, the eating disordered patients may have mixed feelings and ambivalence in disclosing or seeking help. A strategy opens to the dentist is make explicit a dilemma in the patient's mind. The dilemma being whether (a) to live a healthy life/good teeth and cope with body image or (b) to self-inflict harm as a result of restraining her/his diets. At the same time, the dentist suggests a means to resolve the dilemma.

Every patient has his or her own pace of processing information. DiClementi and Prochaska [20] put forward the *Stages of Change* model (see below). The model is a very effective way of conceptualizing a patient's motivation. The model is, moreover, particularly appropriate for dental patients with addictive behaviors (e.g., substance abuse, excessive gambling, excessive intake of alcohol, obsessive rituals, eating problems).

11.5.2 Motivation and Stages of Change

Suppose that the dentist is confronted with a patient (Patient *A*) with eating disorders. What difficulties may the dentist encounter? What can the dentist do? Answers to these questions have to be sought by examining the patient's psychological processes as follows.

11.5.2.1 Precontemplation Stage

Patient *A*, in the first, *precontemplation*, stage has no insight, but denial of any problems. Patient *A* would be resistant to any treatment by a dentist because she/he does not realize (a) that there is any underlying psychological problem and (b) how dental health may be affected by psychological difficulties.

11.5.2.2 Contemplation of Psychological Issue

In the second, *contemplation*, stage, Patient *A* has a feeling of ambivalence about seeking help. The second issue is the onset of severe pain due to dentin hypersensitivity. A knowledgeable and friendly dentist may prompt Patient *A* to explore the possible causal link between (a) maladaptive behaviors (like eating disorders or addictive behaviors) and (b) serious dental problems. Patient *A* begins to consider how the tooth erosion problem can be helped by dealing with the underlying causes of his or her maladaptive behavior.

11.5.2.3 Preparation for Psychological Treatment

Patient *A* is now prepared to "face the challenge," and Patient *A* becomes more receptive to the dentist's suggestions. *Preparation* is the stage in which patient is convinced of the necessity of receiving both dental and psychological treatments. The success of preparation depends on the support and reassurance offered by the dentist (as well as cognate health professionals).

11.5.2.4 Action

Action is a stage when the patient follows a prescribed plan of change in attitude and cognition, in addition to (a) changing diet, refraining from sweet and acidic foods, (b) observing dental hygiene, and (c) having regular dental checkup.

The next step for those with eating disorders and alcoholism is (i) to discuss the problems and (ii) to set targets for change with full commitment from the patient to be referred to the clinical psychologist or psychiatrist. These patients need constant support and empathy from other people in addition to a lot of courage from themselves.

11.5.2.5 Maintenance

Maintenance is a completion stage in which the patient assumes a lifestyle incompatible with eating disorders or addictive behaviors. The new lifestyle is conducive to dental health. The challenges for the patient are how to (a) eliminate the psychological hang-ups that cause the problems in the first place and (b) minimize the danger of relapse. The dentist remains the source of support and praise.

11.5.3 Soliciting Clinical Information

Suspecting Patient *A*'s dental problems may be caused by some underlying psychological difficulties, and the dentist may wish to first identify some symptoms in general terms by asking the following respective sets of questions.

11.5.3.1 Identify General Symptoms of Depression

Whether or not a patient is having some general symptom of depression may be ascertained with the questions tabulated in Table 11.1. If they answer "yes" to more than seven questions, it is appropriate to suggest that the patient may have some depressive symptoms which may contribute to the dental problem. After getting a fuller history, the dentist may suggest referrals to her/his doctor.

11.5.3.2 Psychological Issue Questions for Eating Problems

Whether or not a patient is having some eating disorders may be ascertained with the questions tabulated in Table 11.2. At the end of the assessment, the dentist is in a better position to identify and draw an approximate profile of the patient's eating problem behaviors and elicit some of the full story.

11.5.3.3 General Screening Questionnaires for Eating Disorders

Further tools are available for refining the dentist's understanding of a patient with eating disorders in primary care setting. Several simple screening questionnaires have been developed and evaluated over the years. These include SCOFF [21], rapid screen for college girls [22], Edinburgh's Bulimic Investigatory Test (BITE) and the Binge Eating Scale (BES) [23], the eating disorders not otherwise specified (EDS-5) [24], and Eating Disorder Screen for Primary Care (ESP) [25, 26].

The SCOFF questionnaire [21, 27, 28] was developed and validated in the United Kingdom. The name is an acronym that comes from the questions (Sick = vomit;

Table 11.1 Symptoms suggestive of underlying psychological issues

	Symptoms	Yes	No
1	Do you find it difficult to get up in the morning?		
2	Do you feel you can't cope?		
3	Do you feel you can't face the day?		
4	Do you feel hopeless and helpless?		
5	Do you feel tired all the time?		
6	Has you lost your appetite every day?		
7	Can you fall asleep easily?		
8	Do you sleep too much?		
9	Do you find visiting a dentist or a doctor a chore? Why?		
10	Do you feel fearful?		
11	Do you notice that you have elevated heart beat with no reasons? When did that happen?		
12	Do you find it difficult to breathe? Specify the circumstances		
13	Are you on medication or receiving help for some psychological problems? Elaborate on the answer		

Table 11.2 Symptoms suggestive of eating disorders

	Aspects relevant to eating disorders	Symptom	Yes	No
1	Issues of weight	Do you feel overweight?		
		What is your actual weight?		
		What is your ideal weight?		
2	Body image	Do you dislike your own body image?		
3	Issues of eating	Are you restraining your eating?		
		What would happen if you did not control your eating?		
		What is the pattern of restraint?		
		What foods/drinks do you prefer?		
		Do you avoid certain foods? Why?		
		How do you feel if you do not control your eating?		
4	Issue of overeating	How do you know that you have eaten too much?		
5	Means of dealing with overeating	What do you do if you feel you have eaten too much?		
		Do you make yourself vomit?		
		Have you vomited blood?		
		Do you wash out "excess foods" by drinking copious fluids?		
6	Awareness	Do you think you are suffering from an eating disorder?		
		Have you told anyone about your difficulties?		

Control, One stone off =14 lb, Fat, Food). It consists of five questions designed to clarify suspicion that an eating disorder might exist rather than to make a diagnosis. The questions can be delivered either verbally or in written form. There is one study validating the use of the SCOFF in adult women of a general practice population.

Further research is needed to evaluate the SCOFF questions before they can be recommended for use in primary care.

The Eating Attitudes Test, EAT [29], is probably the most widely used screening tool in epidemiological studies. In addition there are a number of other pencil and paper measures to assess eating disorder psychopathology (e.g., the Eating Disorder Inventory, EDI [30]). However, these tests take a long time to administer, and the results have to be interpreted by specialists. Such instruments may be well suited for evaluating treatment progress in patients with eating disorders, but may not perform well in screening for eating disorders in community samples due to symptom denial and low prevalence [31, 32].

11.5.3.4 General Screening Questionnaires for Depression

Apart from eating disorders, dental problems may also be caused by depression. It would be helpful to the dentists if they have access to tools measuring depression. A commonly used questionnaire is the Beck Depression Inventory II [33], which is easy to use. The symptoms of depression being assessed are appetite, sleep, sex interests, concentration, memory, motivation, and daily hobbies. Other items in the inventory look at self-esteem, confidence, problem-solving abilities, guilt, and whether there is suicidal ideation. There is a cutoff score in determining whether the respondent is normal or mildly depressed or moderately depressed or severely depressed. Once the person scores above mild depression, the individual should receive psychological or medical therapy.

11.5.3.5 General Screening Questionnaires for Anxiety

As may be seen, anxiety plays an important role in eating disorders and depression. Dental professionals may feel the need to be able to measure their patients' anxiety. A commonly used anxiety questionnaire is by Speilberger's [34], the State Trait Anxiety Inventory. One part of the questionnaire looks at an individual's personality trait of anxiety, that is, whether or not the individual would get anxious readily. It also measures the severity of the individual's anxiety. The second part of this test is used to investigate the individual's current state of anxiety (viz., how severe is the current situation). The scores are used to classify individuals into mild, moderate, and severe anxiety categories. Both medication and psychological therapies are appropriate and beneficial when an individual is in a severe state of anxiety.

11.5.4 Establish Antecedent, Feeling, Consequent Behavior, and Maintenance

If a patient shows symptoms of depression or anxiety or eating disorders, the dentist may choose to listen and talk with the patient about the problems bothering her/his during each dental consultation. Try to solicit information as to establish a link between an *antecedent* (condition that happens beforehand), *feelings* (causing how do they feel), the *consequent* maladaptive behaviors (e.g., drinking, or not eating, or vomiting), and *maintenance* (how the behavior is being maintained). Two examples of such a link may be found in Tables 11.3 and 11.4.

Mary's behavior is an example of how thoughts, be they ill-founded, distorted, or otherwise, may trigger sad feelings which, in turn, leads an individual to maladaptive behaviors (like bingeing).

11.5.5 What to Do with the Information

Information collected in Tables 11.1 and 11.2 is used to suggest a cause-effect link between psychology and maladaptive behavior that leads to dental problem. The dentist may use the causal link thus established to discuss with the patient the relationship between psychological issues and dental problems (e.g., tooth erosion). It becomes easier for the dentist to elaborate on how the patient might feel if the problem becomes more serious. The discussion would then lead to the efficacy of dealing with the underlying psychological problems as a means to prevent further dental deterioration. In the course of discussion, the dentist may be the first to spot the eating disorder and persuade the patient to accept referrals to her/his local clinical psychology service.

11.6 Simple Counseling Skills Using Cognitive-Behavioral Therapy (CBT) Techniques

Cognitive-behavioral therapy is the evidence-based therapy working with people who have mental health difficulties (e.g., depression, anxiety, eating disorders, and the like). Aaron Beck is the instigator of the cognitive theory that dysfunctional beliefs underpin most of the mental health problems. The emphasis of this type of therapy is an active collaboration and experimentation that both patient and therapist contribute. The therapeutic objective is to explore how to change the

Table 11.3 An example of the antecedent, feeling, behavior, consequence, and maintenance

Causal component	Nature of component
Antecedent	After eating a chocolate bar, Susan had a bloated stomach
How do you feel?	She felt being fat, ugly, and stupid
What do you do then?	She put her finger down her throat to purge
Maintaining factor	She felt better in the stomach

Table 11.4 Second example of the link between antecedent, feeling, behavior, consequence, and maintenance

Antecedent	Mary suspected that people were criticizing her
Feeling	She felt depressed and miserable
Behavior	She could not help from bingeing on crisps and pops before going to bed
Maintaining factor	She refused to meet people or go out

patient's dysfunctional beliefs without arguing with the patient or exposing the absurdity of the patient's beliefs. It is about encouraging the patient to collect evidence which may or may not support the patient's beliefs. The patient will then be able to evaluate his or her ideas in light of the hard empirical evidence.

To begin with, CBT encourages the anorexic patient to reveal her/his negative, self-defeating thoughts such as, "People are staring at me because I am so fat." Such revelation facilitates the discussion of how negative thoughts or beliefs affect the patient's moods and behavior.

The use of CBT for bulimia nervosa was pioneered by Christopher Fairburn [35] and was evaluated by his team and others [36]. His model proposes a vicious cycle, linking (a) low self-esteem, (b) misperception of body size, (c) strict dieting that ultimately breaks down, (d) bingeing, and (e) compensating behavior in vomiting.

Waller et al. [37] have developed a simplified and condensed version of CBT for use in primary care. It is administered in eight 20-min sessions. It includes the educational and behavioral components but not the cognitive restructuring part of CBT. Four general practitioners and a nurse went for two introductory training workshops. They were provided with a simple treatment manual. In a pilot study [37], 11 women with bulimia nervosa were treated, 6 improved substantially, 1 was more concerned with losing weight than overcoming the eating problem, 2 did not commit for treatment, and 2 had a comorbidity of personality disorder. Even though this is encouraging result, therapists do require more training.

However, there are a number of self-help programs and books [38–40] based on CBT for bulimia nervosa or a nonspecialist therapist guided self-help. The findings were that while those given guided self-help fared better, individuals not receiving the guidance did not.

11.7 Oral Health Education

Mistaken health beliefs could lead to unnecessary dental problems. For the general public, it is important to raise their awareness about prevention, regular checkup, and timely treatment. More needs to be done for patients with problem of depression or eating disorders. Although the general responsibilities of the patient and that of the dentist in the management of dental erosion have been discussed by Amaechi and Higham [41] and also in Chaps. 8 and 9 of this book [42, 43], eating disorder patients, in particular, should take the following precautions [41].

– When possible, "bite guards" should be worn while vomiting or purging. The inside (tooth surface) of the guard should be coated with a small amount of sodium bicarbonate powder or milk of magnesia, to neutralize any gastric acid pooling in it.

– Toothbrushing Instruction:
  (a) Use a medium nylon brush.
  (b) Do not use abrasive "whitening" toothpastes.
  (c) Brush with the Bass technique to avoid horizontal strokes.
  (d) Avoid toothbrushing immediately after each episode of vomiting or purging or bingeing on acidic food or drink; rather patients should use any of the following to freshen their mouth and wait for at least 60 min before toothbrushing:
    • Fluoride mouthwash to enhance rapid remineralization of the softened tooth surface.
    • Fluoride tablets and fluoride lozenge, which have been demonstrated as effective remineralizing agent.
    • Sugar-free lozenges or chewing gum to increase saliva flow to facilitate rapid remineralization of the softened tooth tissue, neutralize the acidity, and provide alkaline environment necessary for remineralization. Buffering capacity and bicarbonate content of stimulated saliva is higher than that of unstimulated saliva. It is also speculated that saliva stimulation would enhance the formation of acquired salivary pellicle, which has been shown to protect teeth against erosive attack.
    • Dairy products (e.g., fresh milk) have been shown to reharden softened tooth surface.
    • Sugar-free antacid tablets or a pinch of sodium bicarbonate (or baking soda) dissolved in some water may be used to neutralize the acidic oral fluid.
– Use high fluoride concentration toothpaste as well as fluoride mouthrinse for their routine daily oral hygiene practice.
– Visit their dentist regularly for professional clinical care.

These information should be provided to them with a handout on oral health (see Table 11.5) in order to remind them.

11.8 De-stigmatization of Mental Health Problems

A survey revealed that stigmatizing attitudes towards mental health problems came from distorted beliefs that mental health problems are not curable, self-inflicted, and a danger to others and that it is difficult to communicate with mental health patients. In view of the aforementioned erroneous beliefs about mental health, the Royal College of Psychiatrists [44] organized an anti-stigma campaign. Gowers and Shore [45] therefore argued for more improved training of health service staff and greater public education. They suggested that stigma can be reduced by more therapeutic encountering with mental health patients, particularly individuals suffering from eating disorders.

Table 11.5 Health education leaflets on tooth erosion

Item	Domain	Specific issue
1	*Education*	
	Diet (see Table 10.4 and Fig. 10.1)	Avoid drinking acidic juices or choose juices fortified with calcium Choose drinks with calcium (like milk) or phosphate bases
	Attitudes	Is your ideal body weight reasonable? Is there a real difference between your perceived and actual weights?
	Healthy eating	Need to maintain a minimum body mass index (BMI)
2	*Prevention and protection*	
	Patient self-care (see Chap. 8 this book)	
	Dentist-applied clinical care (see Chap. 9 this book)	
3	*Professional help*	Ask medical practitioners and pharmacologists about the side effects of prescribed medication All professionals should advise their patients to keep an eye on the process of tooth erosion and seek constant dental checkups
		Ask medical practitioners and pharmacologists about the acidity of medication

11.9 Recall, Review, and Monitor

It is important that dentists treating patients with tooth erosion take note that they seek confirmation from their medical practitioners whether their dental patients are on psychotropic medications or receiving psychological outpatient treatments and that they would persuade and refer patient with psychological issues to the specialist. It is important that the patients are followed up, monitored, and reviewed so that relapse can be spotted timely.

11.10 Summary and Conclusion

This chapter briefly outlines that tooth erosion can happen because of underlying psychological problems, namely, depression, eating disorders, and anxiety. Often dentists and general practitioners are the ones who discovered these conditions. When the dentists adopt therapeutic alliance in assessments, these patients can be approached in ways that (a) enhance the patients' insights about the cause of the dental problems, (b) make more efficacious prevention strategies, and (c) render possible further referral for psychological treatment. Confronted with dental erosions and treatment, dentists can be more aware of their role in some mental health issues. There is a growing awareness in the dental profession of the need to incorporate the behavioral sciences in the core element of their curriculum.

References

1. Scaramucci T, Carvalho JC, Hara AT, Zero DT. Causes of dental erosion: intrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 33–5.
2. Hara AT, Carvalho JC, Zero DT. Causes of dental erosion: extrinsic factors. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 69–96.
3. Selye H. Stress in health and disease. Reading: Butterworth; 1976. p. 33–35.
4. Leung GM, Smales R, Yip K. Psychological behaviors and tooth erosion: Ch 3. In: Yip KHK, Smales RJ, Kaidonis JA, editors. Tooth erosion, prevention and treatment. New Delhi: Jaypee Brothers Medical Publishers (P) Ltd; 2006.
5. Hudson JI. The prevalence and correlates of eating disorders in the National Comorbidity Survey replication. Biol Psychiatry. 2007;61(3):348–58.
6. Crow SJ, Peterson CB, Swanson SA, Raymond NC, Specker S, Eckert ED, Mitchell JE. Increased mortality in bulimia nervosa and other eating disorders. Am J Psychiatry. 2009;166:1342–6.
7. Foster M. Dental problems associated with disordered eating. Toronto: Natural ED Information Centre. 2008. www.medic.ca.
8. Beumont PJ, George CG, Smart DE. Dieters' and "vomiter and purgers" in Anorexia Nervosa. Psychol Med. 1976;6:617–22.
9. Vanderycken W. Emergence of Bulimia Nervosa as a separate diagnostic entity: review of literature from 1960–79. Int J Eat Disord. 1994;16:105–16.
10. Russell GFM. Bulimia Nervosa: an ominous variant of Anorexia Nervosa? Psychol Med. 1999;9:429–48.
11. Dsm III. APA diagnostic statistical manual of mental disorders, 3rd version. Washington, DC: American Psychiatric Association; 1980.
12. Misra M, Aggarwal A, Miller KK, Almazan C, Worley M, Soyka LA, Herzog DA, Klibanski A. Effects of anorexia nervosa on clinical, hematologic, biochemical, and bone density parameters in community-dwelling adolescent girls. Pediatrics. 2004;114(6):1574–83.
13. Dsm V. APA diagnostic statistical manual of mental disorders, 5th version. Washington, DC: American Psychiatric Association; 2013.
14. Fairburn CG, Harrison PJ. Eating disorders. Lancet. 2003;361(9355):407–16.
15. Hsu LK. Outcome of bulimia nervosa. In: Brownell KD, Fairburn CG, editors. Eating disorders and obesity: a comprehensive handbook. New York/London: Guildford Press; 1995. p. 238–46.
16. Collings S, King M. Ten year follow-up of 50 patients with Bulimia Nervosa. Br J Psychiatry. 1994;164:80–7.
17. Keel PK, Mitchell JE, Miller KB, Davis TL, Crow SJ. Long-term outcome of bulimia nervosa. Arch Gen Psychiatry. 1999;56(1):63–9.
18. Fairburn CG, Cooper Z, Doll H, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiatry. 2000;57:659–65.
19. Jager DHJ. Dental erosion: prevalence, incidence and distribution. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015.
20. Diclemente C, Prochaska JO. Towards a comprehensive transtheoretical model of change. In: Miller W, Heather N, editors. Treating addictive behaviors. New York: Plenum Press; 1998. p. 3–24.
21. Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. Br Med J. 1999;319:1467–8.
22. Anstine D, Grinenko D. Rapid screening for disordered eating in college-aged females in the primary care setting. J Adolesc Health. 2000;26(5):338–42.
23. Ricca V, Mannucci E, Moretti S, Di Bernardo M, Zucchi T, Cabras P, Rotella CM. Screening for binge eating disorder in obese out-patients. Compr Psychiatry. 2000;41:111–5.

24. Rosenvinge JH, Perry JA, Bjørgum L, Bergersen TD, Silvera DH, Holte A. A new instrument measuring disturbed eating patterns in community populations: development and initial validation of a 5-item scale (EDS-5). Eur Eat Dis Rev. 2001;9:123–32.
25. Cotton MA, Ball C, Robinson P. Four simple questions can help screen for eating disorders. J Gen Intern Med. 2003;18(1):53–6.
26. Freund KM, Graham SM, Lesky LG, Moskovitz MA. Detection of bulimia nervosa in a primary care setting. J Gen Intern Med. 1993;8(5):236–42.
27. Luck AJ, Morgan JF, Reid F, O'Brien A, Brunton J, Price C, Perry L, Lacey JH. The SCOFF questionnaire and clinical interview for eating disorders in general practice: comparative study. Br Med J. 2002;325(7367):755–6.
28. Perry L, Morgan J, Reid F, Brunton J, O'Brien A, Luck A, Lacey H. Screening for symptoms of eating disorders: reliability of the SCOFF screening tool with written compared to oral delivery. Int J Eat Disord. 2002;32(4):466–72.
29. Garner DM, Garfinkel. The eating attitudes test: an index of the symptoms of anorexia nervosa. Psychol Med. 1979;9(2):273–9.
30. Garner DM, Olmsted MP, Polivy J. Development and validation of a multidimensional eating disorders inventory for anorexia nervosa and bulimia. International Journal of Eating Disorders. 1983;2:15–34.
31. Carter J, Moss RA. Screening for anorexia and bulimia nervosa in a college population: problems and limitations. Addict Behav. 1984;9(4):417–9.
32. Williams P, Hand D, Tarnopolsky A. The problem of screening for uncommon disorders-a comment on the Eating Attitudes Test. Psychol Med. 1982;12(2):431–4.
33. Beck AT, Steer RA. Beck depression inventory II manual. San Antonio: The Psychological Corp.; 1984.
34. Spielberger CD, Gorsuch RL. State trait anxiety inventory (STAI). Palo Alto: Consulting Psychologist Press; 1968.
35. Fairburn CG. A Cognitive behavioral approach to the management of bulimia. Psychol Med. 1981;11:707–11.
36. Wilson GT, Fairburn CG, Agras WS. Cognitive behavior therapy for bulimia nervosa. In: Garner DM, Garfinkel PE, editors. Handbook of treatment for eating disorders. New York: Guildford Press; 1999.
37. Waller D, Fairburn CG, McPherson A, Kay R, Lee A, Nowell T. Treating bulimia nervosa in primary care: A pilot study. Int J Eat Disord. 1996;19(1):99–103.
38. Cooper PJ. Bulimia nervosa and binge eating. London: Robinson; 1995.
39. Fairburn CG. Overcoming binge eating. NY: Guildford Press; 1995.
40. Freeman C. Overcoming anorexia nervosa. A self-help guide using cognitive behavioral techniques. London: Constable & Robinson Ltd; 2002.
41. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33:243–52.
42. Buzalaf MA, Cardoso CA, Magalhães AC, Amaechi BT. Prevention and control of dental erosion: patient self-care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015.
43. Kaidonis JA. Prevention and control of dental erosion: professional clinic care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 151–68.
44. Cowan L, Hart D. The Royal College of Psychiatrists College report CR168 (2014). Junior MARSIPAN: management of really sick patients under 18 with anorexia nervosa. 1998. http://www.rcpsych.ac.uk.
45. Gowers SG, Shore A. The stigma of eating disorders. Int J Clin Pract. 1999;53(5):386–8.

Prevention and Control of Dental Erosion: Gastroesophageal Reflux Disease Management

12

Akit Patel, Bennett T. Amaechi, and Charles Brady III

Abstract

Approximately 10–20 % of the general population suffers from gastroesophageal reflux disease or GERD. GERD can manifest as esophageal and extraesophageal symptoms. GERD may damage the dental tissues, causing disorders such as dental erosion. According to studies, 24 % of patients with GERD have dental erosion, and 32 % of adults and 17 % of children with dental erosion have GERD. However, not all affected persons will have classic symptoms of GERD. Dentists may be the first persons to diagnose GERD in these "silent refluxers," particularly when observing unexplained tooth erosion. The cause of GERD is multifactorial, but the basic cause is incompetent antireflux barriers at the gastroesophageal junction. However, other causes have also been attributed to GERD which include decreased saliva production, diet, eating habits, medications, and obesity. Typical manifestations of GERD are heartburn, regurgitation, and dysphagia. Other symptoms have also been associated with GERD, and if any "alarm symptoms" are present, then further evaluation is required. The location of the erosive tooth wear in the dentition is specific to each etiologic factor. Refluxed acid first damages the palatal surface of the upper incisors then the other surfaces of the maxillary teeth. In chronic GERD, the labial or buccal surfaces are affected then the occlusal surfaces of maxillary and mandibular teeth. The diagnosis of GERD can be made clinically, physiologically, anatomically, or functionally depending on the testing modality. The mainstay management

A. Patel, MD (✉) • C. BradyIII , MD
Department of Medicine, Gastroenterology, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, Office 5. 247S, San Antonio, TX 78229, USA
e-mail: Patela13@uthscsa.edu; bradyce@uthscsa.edu

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_12

strategy for GERD includes lifestyle modifications, dentist's teeth protection care, and medical therapy such as antacids, histamine-2 receptor blockers, and proton pump inhibitors.

12.1 Introduction

Gastroesophageal reflux disease, or GERD, is a very common disease, with an estimated prevalence of up to 10–20% of the general population. The consequences of gastroesophageal reflux are not only localized to the esophagus, but extraesophageal involvement is frequently seen as well. GERD may involve damage to the oral tissues, and dental erosions may occur as a result [1–3].

Dental erosion is defined as the loss of tooth structure through a chemical process of dissolution of dental hard tissue due to acidification of the oral cavity, which is not associated with bacterial activity. The etiology of dental erosions is multifactorial. The causes may consist of extrinsic factors like acidic foods, beverages, or medications. However, there can also be intrinsic factors, the most common being GERD with regurgitation of gastric acid into the oral cavity [3].

12.2 Epidemiology

A global definition and classification of GERD has been developed by the Montreal consensus group. GERD has been defined as "a condition that develops when the reflux of stomach contents causes troublesome symptoms and/or complications," and its manifestations have been subclassified into esophageal and extraesophageal syndromes [4]. One of the known extraesophageal manifestations of GERD is dental erosion.

A recent systematic review involving 17 observational and case-control studies of GERD and dental erosion found a strong association between the two conditions [5]. The median prevalence of dental erosion in GERD patients was 24 %, and the median prevalence of GERD in adults and in children with dental erosions was 32.5 and 17 %, respectively. Therefore, from their observations of tooth erosion, dental practitioners may be the first persons to diagnose the possibility of GERD, particularly in the case of "silent refluxers" (i.e., patients with asymptomatic GERD). This diagnosis is important, as GERD has increased in prevalence in many countries and may have severe health effects if not adequately treated [6, 7]. Consequently, dental practitioners should be more aware of the various manifestations of GERD in both children and adults (Table 12.1).

The prevalence of GERD in the United States appears to be rising. It accounts for at least nine million physician office visits in the United States each year, and annual costs for managing GERD are estimated to be greater than $9 million [8]. In Western populations, 25 % of the people report having heartburn at least once a month, 12 % at least once a week, and 5 % describe having symptoms daily (Table 12.2). There appears to be no gender predominance of heartburn symptoms; men and women are

Table 12.1 Prevalence of gastroesophageal reflux disease (GERD) and dental erosion

Gastroesophageal reflux disease, or GERD, is a common disease
10–20 % of the general population suffers from GERD
GERD affects adults and children
GERD can cause dental erosion
24 % of patients with GERD have dental erosion
32 % of adults with dental erosion have GERD
17 % of children with dental erosion have GERD
Patients with GERD are not only more likely to have dental erosion but also more likely to have severe erosion

Table 12.2 Frequency of heartburn in Western populations

Symptoms once a month	25 %
Symptoms once a week	12 %
Symptoms daily	5 %

affected equally. The relationship of age and reflux is unclear. One study has suggested an association between advancing age and fewer reflux symptoms; however, there is a higher presence of more severe esophagitis, or inflammation of the esophagus due to acid reflux [9].

Studies have shown that up to 60 % of patients with dental erosions had pathological levels of acid reflux when assessed by ambulatory esophageal pH monitoring [3]. Dental erosions as a manifestation of GERD have not only been seen in adults but also in children. A recent study of 249 children and adult, of whom 91 had molar erosion and/or symptoms of acid reflux and had undergone endoscopy, esophageal manometry, and 24-h esophageal pH monitoring, found a significant association between diagnosed GERD and dental erosion [10]. Studies have shown that children diagnosed with GERD according to esophageal pH monitoring had more dental erosions compared to healthy children. A study of 38 children with a diagnosis of GERD according to esophageal pH recordings was evaluated for the prevalence of dental erosions. Children with confirmed GERD had more severe dental erosions compared to children with no GERD [11]. Another study evaluated 52 children with GERD and 52 healthy siblings. In this study, the severity of dental erosion was greater in the study group; 43 % of the affected teeth had grade 3 erosions and 9 % in the control group [12]. In a study in the United States, dental erosion was measured using Aine index in 24 children diagnosed with GERD. Twenty children had dental erosion, ten with mild erosion (grade 1), six with moderate (grade 2), and four with severe erosion (grade 3) [13]. The presence of dental erosions, especially in posterior primary teeth in children, is associated with GERD [14].

12.3 Etiology

Gastroesophageal reflux is the retrograde flow or reflux of gastric contents other than air into or through the esophagus. GERD refers to reflux that produces frequent symptoms or results in damage to the esophageal mucosa or contiguous organs of

the upper respiratory tract and occasionally the lower respiratory tract and oral cavity.

The pathogenesis of gastroesophageal reflux is complicated and multifactorial. The antireflux barriers of the gastroesophageal junction are anatomically and physiologically complex and vulnerable to a number of potential mechanisms of reflux. The basic cause of GERD is incompetent antireflux barriers at the gastroesophageal junction, which normally prevents backflow of gastric acid into the esophagus. Normally, a ring of muscle tissue called the lower esophageal sphincter (LES), which is located in the lower portion of the esophagus where it joins the stomach (gastroesophageal junction), prevents reflux (or backing up) of acid from the stomach. Normally, this sphincter relaxes during swallowing to allow food to pass and then tightens to prevent flow in the opposite direction (i.e., reflux). With GERD, however, the sphincter relaxes between swallows or is weakened, allowing stomach contents and corrosive acid to reflux up the esophagus and damage the lining of the esophagus. The two main patterns of LES dysfunction are (1) hypotensive LES and (2) pathologic transient lower esophageal sphincter relaxations (TLESRs). Anatomic disruption of the gastroesophageal junction is commonly associated with a hiatal hernia, which is a partial herniation of the stomach through the diaphragm and into the thorax. Hiatal hernias contribute to the pathogenesis of reflux disease by impairing LES function [15, 16].

Gastric factors can play a significant role in producing GERD. Gastric factors that promote GERD include poor acid clearance from the esophagus; diminished salivary flow; increased gastric acid production; increased gastric volume after meals; increase gastric pressure due to obesity, recumbency, or lying down after meals; and delayed gastric emptying or gastroparesis. Increased gastric distention can cause an increase in transient LES relaxation and the volume of reflux,

Table 12.3 Etiologic factors of gastroesophageal reflux disease (GERD)

Motility disorders	Damaging factors	Resistance factors	Others
Transient lower esophageal sphincter relaxations (TLESRs)	Increased gastric acid production	Reduced saliva and bicarbonate production	Hiatal hernia
Weak lower esophageal sphincter (LES)	Reflux of bile and pancreatic juice from small intestines	Diminished mucosal blood flow	Diet
Weak esophageal peristalsis or motility		Decreased protective mucus	Eating habits
Delayed gastric emptying or gastroparesis			Increased intra-abdominal pressure
Scleroderma and CREST syndrome[a]			Obesity
			Medications
			Obstructive sleep apnea

[a]CREST syndrome: calcinosis cutis, Raynaud's phenomenon, esophageal dysfunction, sclerodactyly, and telangiectasia

particularly in GERD patients with large hiatal hernias. Delayed gastric emptying may be present in approximately 15% of patients with GERD and is frequently underdiagnosed [17]. There is a well-known association between body mass index (BMI) and reflux symptoms. Evidence has shown that inappropriate relaxation of the LES can be exacerbated by obesity [18, 19]. Even moderate weight gain among people with normal weight is thought to cause or exacerbate reflux symptoms [20] (Table 12.3, Fig. 12.1).

Other factors that decrease LES pressure and contribute to GERD are medications, lifestyle behaviors, and the ingestion of certain foods. Certain medications can exacerbate GERD by lowering LES pressure, like calcium channel blockers and nitrates; others can cause esophagitis, or damage to the lining of the esophagus, by direct mucosal injury, like nonsteroidal anti-inflammatory drugs. Behaviors like binge eating; bulimia, which is binge eating followed by regurgitation of food in order to lose weight; and recumbency after eating all create gastric distension and weaken the LES leading to acid reflux. Certain foods like fatty foods, peppermint, chocolate, caffeinated beverages, and alcohol can all decrease LES pressure and contribute to GERD. Smoking tobacco products can also lead to GERD by decreasing LES pressure. The contribution of these various factors varies from patient to patient [9] (Table 12.4, Fig. 12.1).

12.4 Pathogenesis

Esophageal clearance is important to prevent gastric content from reaching the oral cavity and causing dental erosion. Esophageal clearance is achieved by two mechanisms: esophageal peristalsis, or sequential esophageal contractions causing propulsion of food to stomach, and the buffering action of saliva, which neutralizes the acid. Peristalsis empties the esophagus of its contents, and this process is followed by the

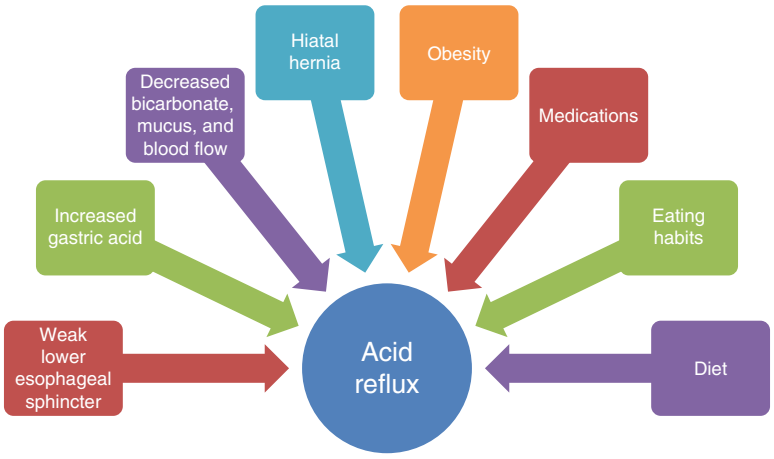


Fig. 12.1 Factors contributing to gastroesophageal reflux disease (GERD)

Table 12.4 Factors that precipitate or exacerbate GERD symptoms

Medications	Foods	Behaviors or conditions that increase intra-abdominal pressure
α-Adrenergic antagonists (e.g., doxazosin, prazosin, tamsulosin, terazosin)	Alcohol	Binge eating
Anticholinergics (e.g., atropine, benztropine, buproprion, dextromethorphan, ipratropium, oxybutynin, tolterodine, tiotropium)	Caffeine	Bulimia (binge eating followed by regurgitation in order to lose weight)
β-Adrenergic agonists (e.g., albuterol, formoterol)	Carbonated beverages	Eating large-volume meals
Calcium channel blockers (e.g., amlodipine, diltiazem, nifedipine, verapamil)	Chocolate	Eating prior to recumbency (lying down)
Diazepam	Citrus fruits	Pregnancy
Estrogens	Fatty foods	Sleep apnea (pause in breathing during sleep)
Narcotics	Peppermint	Smoking
Nitrates (e.g., isosorbide mononitrate or dinitrate, nitroglycerin)	Spicy foods	Weight gain
Nonsteroidal anti-inflammatory drugs (NSAIDs) (e.g., aspirin, diclofenac, ibuprofen, naproxen, meloxicam, piroxicam)	Tomato-based products	
Progesterone	Vinegar	
Theophylline		
Tricyclic antidepressants (e.g., amitriptyline, desipramine, imipramine, nortriptyline)		

GERD gastroesophageal reflux disease

neutralization of the acid of the esophageal lumen by the saliva [21]. Saliva has a buffering and protective role against the demineralizing effects of acids in the oral cavity. The saliva in patients with GERD has a greater buffering capacity compared to patients without GERD. This difference is due to modifications in the saliva composition resulting from an increased concentration of inorganic phosphates [22].

The two possible mechanisms by which acid reflux damages extraesophageal tissue may include (1) direct damage from mucosal contact (reflux theory) and (2) vagal nerve-mediated reflex from distal esophageal acid exposure (reflex theory) [23]. In either case, the refluxed acid can damage extraesophageal tissues including the oral tissues, particularly its hard and soft tissues. In these cases, the quality and amount of saliva play an important role in hard and soft oral tissues changes [24]. The hydroxyapatite crystals constituting the dental inorganic material may be dissolved by acid having a pH under the critical pH (5.5) for dental enamel dissolution [25]. The gastric reflux has a pH less than 2.0, so it can erode dental tissues [26].

The surfaces of the teeth during active endogenous acid erosion are largely devoid of protective biofilm and saliva due to gastric acid and also possible proteolytic pepsin. The raw products resulting from hard tooth tissue demineralization are lost and are not available to be reused when the oral pH increases back to neutral levels [27]. The chemical action causes rapid dissolution of exposed tooth surfaces that is distinctly different from the subsurface dissolution seen with plaque acids [28]. Under magnification, the eroded tooth surfaces will show damage to the ends of the enamel rods, which will only remineralize after the endogenous acid has been cleared from the oral cavity and after salivary pellicle has been reestablished on the tooth surfaces.

The addition of remineralizing ions to the eroded surfaces will only result in the repair of the ends of the enamel rods as the "gross" surface damage is irreversible. Even when fluorapatite is present in high concentrations, the remineralized surfaces provide little or no extra protection to further sustained demineralization as the endogenous acid has a pH well below 4.5, which is the approximate critical pH for fluorapatite dissolution [29]. These findings are supported by observations that fluoride-based and casein-based (amorphous calcium phosphate stabilized by casein phosphopeptide) remineralizing agents provide some protection against erosion at pH 3.0 [30–33], but not at a highly erosive environment of pH less than 2 [34–36].

12.5 Clinical Manifestations

Typical manifestations of GERD are heartburn, regurgitation, and dysphagia (difficulty swallowing). Other symptoms associated with GERD include water brash (regurgitation of excessive saliva), a globus sensation (lump in the throat), odynophagia (pain with swallowing), and nausea (Table 12.5). Heartburn, or pyrosis, is defined as a retrosternal burning discomfort located in the midepigastric area that may radiate up toward the neck or throat and typically occurs in the postprandial (post-meal) period [37]. Patients who present with typical symptoms with a minimum frequency of twice a week for 4–8 weeks or more should be considered as having GERD. At initial presentation, it is important to consider a patient's age and the presence of "alarm signs" (Table 12.6). Alarm signs should be investigated in every patient with GERD symptoms as they may indicate complications from GERD, including esophageal stricture (i.e., scar tissue resulting in narrowing of the esophageal lumen), Barrett's esophagus (i.e., a precancerous condition with changes in the cells lining the esophagus), or malignancy (i.e., esophageal or gastric cancer). The presence of any alarm signs necessitates the evaluation of GERD symptoms with further testing such as an upper endoscopy and/or imaging [9].

Table 12.5 Typical GERD symptoms

Heartburn
Regurgitation
Dysphagia (i.e., difficulty swallowing)
Water brash (i.e., regurgitation of excessive saliva)
Globus (i.e., sensation of lump in the throat)
Odynophagia (i.e., pain with swallowing)
Nausea

GERD gastroesophageal reflux disease

Atypical manifestations of GERD refer to symptoms that are extraesophageal, including pulmonary, ear, nose, and throat manifestations, as well as noncardiac chest pain. Studies have shown that GERD can be found in 30–80% of adults with asthma. A new, more frequently recognized atypical manifestation of GERD is otitis media in children [38]. It is recommended that in patients who present with atypical respiratory, or ear, nose, or throat (ENT) symptoms, GERD needs to be ruled out (Table 12.7).

Patients with gastroparesis (i.e., delayed gastric emptying) and GERD may present with concomitant nausea, vomiting, or early satiety (i.e., sensation of feeling full early after eating a small amount). Gastroparesis should be suspected in patients with acute or subacute onset of GERD, particularly after an episode of viral respiratory infection or gastroenteritis [9].

Table 12.6 "Alarm signs" that necessitate further evaluation of GERD

Dysphagia (i.e., difficulty swallowing)
Odynophagia (i.e., pain with swallowing)
Weight loss
Gastrointestinal bleeding (i.e., vomiting blood, blood in stools, or black stools)
Anemia
Advanced age (>50 years old)
Chest pain
Family history of upper GI tract cancer

GERD gastroesophageal reflux disease

Table 12.7 Extraesophageal manifestations of GERD

Otitis media
Chronic sinusitis
Dental erosions
Aphthous ulcers
Halitosis
Pharyngitis
Laryngitis
Hoarseness of voice
Subglottic stenosis
Laryngospasm
Postnasal drip
Frequent throat clearing
Globus
Tracheobronchitis
Chronic cough
Asthma
Aspiration pneumonia
Pulmonary fibrosis
Chronic bronchitis
Bronchiectasis
Noncardiac chest pain
Sleep apnea

GERD gastroesophageal reflux disease

12.5.1 Oral Manifestation

With respect to dental erosions, the location of the dental erosive zones is specific to each etiologic factor. The diagnostic criteria for dental erosions are loss of tooth structure of non-carious etiology, outside the areas of contact or occlusal guidance, and a glossy, smooth, rounded shape. In addition, negative gap areas at the edges of amalgam or composite fillings can also be seen [39]. If lesions occur due to extrinsic factors, then they are more frequently situated on the vestibular areas of the teeth. When the etiology is intrinsic in nature (gastric acids), the areas of erosion have characteristic distribution. Refluxed acid first attacks the palatal surface of the upper incisors (Figs. 12.2 and 12.3). The palatal surfaces of the maxillary teeth are also affected early due to the fact that they are not protected by the major salivary glands (Fig. 12.4). In the early stages, the tongue protects the lower teeth, but in the later stages, if the exposure to acid continues, erosion of the posterior mandibular teeth occurs, starting with the lingual, then occlusal, and buccal surfaces (Fig. 12.5). The labial or the buccal surfaces are affected by erosion only if acid reflux persists for an extended period of time. In chronic GERD, the occlusal surfaces of maxillary and mandibular teeth are affected. In the early stages, lesions are difficult to identify during normal examination, but advanced erosions are easily noticeable and have characteristic appearances (Table 12.8) [3, 40].

12.6 Diagnosis and Workup

The diagnosis of GERD may be:

- *Clinical* (presentation with typical symptoms, such as heartburn)
- *Physiologic* (evidence of abnormal esophageal pH levels in the distal esophagus)
- *Anatomic* (evidence of esophagitis on endoscopy)
- *Functional* (clinical response to antacid medications)

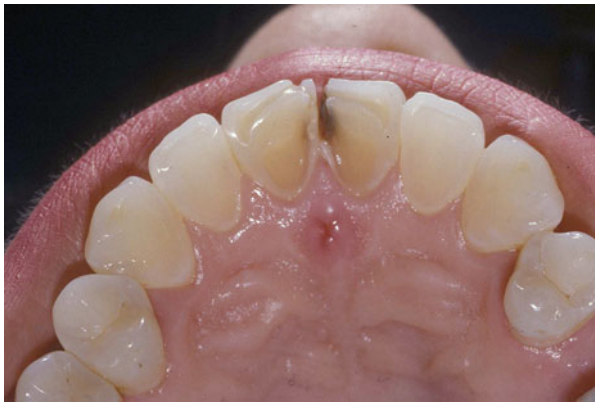


Fig. 12.2 Dental erosion on the palatal surface of upper incisors (Courtesy of Dr. Bennett T. Amaechi)

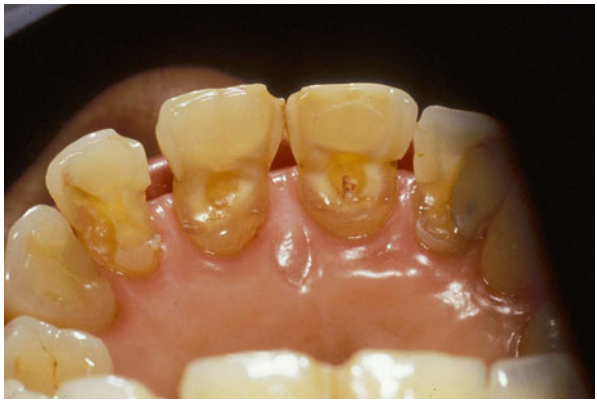


Fig. 12.3 Severe dental erosions on the palatal surface of upper incisors (Courtesy of Dr. Bennett T. Amaechi)

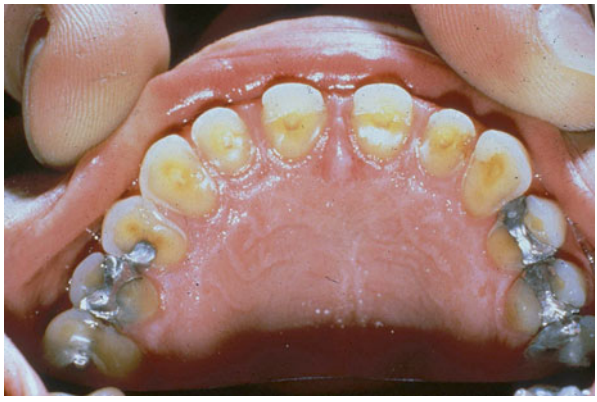


Fig. 12.4 Severe dental erosions on the palatal surface of upper incisors and maxillary premolars (Courtesy of Dr. Bennett T. Amaechi)

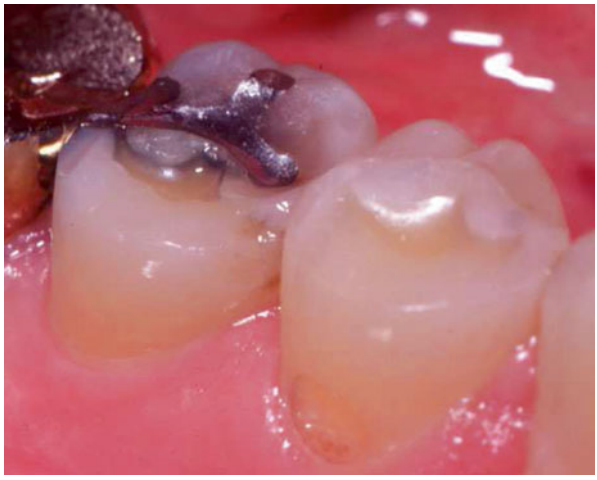


Fig. 12.5 Early stage erosion on lingual surface of mandibular molar and premolar (Courtesy of Dr. Bennett T. Amaechi)

Table 12.8 Pattern of dental erosion due to gastroesophageal reflux disease (GERD)

Refluxed acid first damages the palatal surface of the upper incisors then the palatal surfaces of the maxillary teeth
In the early stages, the tongue protects the lower teeth, but in the later stages, erosion of the posterior mandibular teeth occurs, starting with the lingual, then occlusal, and buccal surfaces
The labial or the buccal surfaces are affected by erosion only if acid reflux persists for an extended period of time
In chronic GERD, the occlusal surfaces of maxillary and mandibular teeth are affected

However, the correlation between these diagnostic approaches is relatively poor. For example, many asymptomatic patients have esophagitis, abnormal pH values, or even Barrett's esophagus.

There are several different methods for diagnosing GERD. Classic GERD can also be diagnosed by taking a thorough symptom history and confirmed by a complete response to medical therapy (a "PPI [proton pump inhibitor] test"). Acid-suppressive medications like proton pump inhibitors (PPIs) can be used not only as therapy for GERD but also as a diagnostic test. A meta-analysis that assessed the accuracy of normal to high-dose PPI for 1–4 weeks in the diagnosis of GERD found a sensitivity of 78 % and a specificity of 54 % when ambulatory esophageal pH was used as a gold standard. In the absence of serious symptoms and signs, PPIs administered over 1–4 weeks are a cost-effective initial diagnostic test and treatment therapy for GERD [41]. Patients who have no symptom response to PPI therapy are unlikely to have GERD [9].

In addition to a simple PPI test, several different questionnaires have been developed to facilitate the diagnosis of GERD, but many of them lack proper validation or lack the simplicity required to be an integrated part of routine care [42–46]. Recently, the Gastroesophageal Reflux Disease Questionnaire (GerdQ) has been developed as a tool to improve and standardize symptom-based diagnosis and evaluation of treatment response in patients with GERD. Studies have shown that family practitioners and gastroenterologists who use the GerdQ have moderate and similar accuracy for diagnosing GERD, approximately 70 % [47]. In this patient symptom-centered questionnaire, a total score of 0–2 points indicates a 0 % likelihood of GERD, a score of 3–7 points indicates a 50 % likelihood of GERD, a score of 8–10 points indicates a 79 % likelihood of GERD, and a score of 11–18 indicates an 89 % likelihood of GERD (Fig. 12.6).

Studies indicate that symptom-based questionnaires have been demonstrated to be useful research tools, but they are either of insufficient validity or are too complicated to use to be useful in clinical practice. They may however have potential in directing the appropriate diagnostic workup and for tailoring of subsequent medical treatment. Therefore, its value in the evaluation of patients with possible GERD in clinical practice may be in aiding primary care physicians in identifying patients with low likelihood of GERD who may benefit more from further testing and in providing the typical GERD patient with better treatment and follow-up [48–50]. However, at this time, current guidelines do not recommend routine use of symptom questionnaires for the diagnosis or management of GERD.

When you think of symptoms you have had in the past 7 days, how did you experience the following:				
Answer the questions by marking one square in each row and sum the total number of points.				
	Number of days			
	0	1	2–3	4–7
1. How often did you have burning feeling behind your breastbone (heartburn)?	☐ [0]	☐ [1]	☐ [2]	☐ [3]
2. How often did you have stomach contents (liquid or food) moving upwards to your throat or mouth (regurgitation)?	☐ [0]	☐ [1]	☐ [2]	☐ [3]
3. How often did you have a pain in the middle of the upper stomach?	☐ [3]	☐ [2]	☐ [1]	☐ [0]
4. How often did you have nausea?	☐ [3]	☐ [2]	☐ [1]	☐ [0]
5. How often did you have difficulty getting a good night's sleep because of your heartburn and/or regurgitation?	☐ [0]	☐ [1]	☐ [2]	☐ [3]
6. How often did you take additional medication for your heartburn and/or regurgitation in other than what the physician told you to take? (i.e. Zantac, Pepcid, Prevacid, Prilosec, etc)?	☐ [0]	☐ [1]	☐ [2]	☐ [3]
Total score: 0 – 2 points = 0 % likelihood of GERD 3 – 7 points = 50 % likelihood of GERD 8 – 10 points = 79 % likelihood of GERD 11 – 18 points = 89 % likelihood of GERD				

Fig. 12.6 GerdQ (gastroesophageal reflux disease questionnaire)

In general, diagnostic testing is reserved for patients who fail to respond to a trial of adequate medical therapy, like histamine-2 receptor blocker (H_2RB) or PPI, or for patients who have alarm symptoms with GERD. Available tests include upper GI series, upper endoscopy (esophagogastroduodenoscopy [EGD]), 24-h esophageal pH study, wireless capsule pH study, and esophageal impedance testing [8, 9] (Table 12.9, Fig. 12.7).

Radiographic imaging is of limited use in the diagnosis of GERD due to poor sensitivity in milder GERD, but they can detect moderate to severe esophageal reflux, hiatal hernias, or complications from GERD such as esophageal strictures or tumors. The studies that are most commonly used are the barium swallow, which only examines the esophagus, and the upper GI series, which examines the esophagus, stomach, and upper small intestines. The primary utility of radiographic imaging in GERD is to rule out other diseases in patients in whom there is a low clinical suspicion. Compared with endoscopy, radiographic imaging is noninvasive, widely available, and relatively inexpensive. However, they are less sensitive and specific than upper endoscopy and require operator skill [9, 51, 52] (Table 12.9).

Upper endoscopy (esophagogastroduodenoscopy [EGD]), in addition to excluding other disease such as tumors and peptic ulcer disease, can detect and grade the severity of reflux-induced esophagitis. Upper endoscopy is highly specific at 90–95 % for GERD but has limited sensitivity at 50 %. An EGD also allows for the evaluation of any complications of GERD including esophageal strictures or Barrett's esophagus. It is therefore the test of choice in patients with alarm signs [9, 51–54] (Table 12.9).

Table 12.9 Summary of diagnostic tests for GERD

Test	Description	What does it measure?
PPI (proton pump inhibitor) test	Empiric PPI therapy 1–4 weeks	Positive response suggests GERD is a possible cause of symptoms
Symptom questionnaire (i.e., GerdQ)	Symptom-based questionnaire	Not widely used or recommended Not sensitive to detect asymptomatic GERD
Radiographic imaging (barium esophagram, upper GI series)	X-ray imaging with contrast of the esophagus (esophagram) or esophagus, stomach, and upper small intestines (upper GI series)	Can reveal anatomical findings such as strictures, hiatal hernia, or tumors Can also detect gastroesophageal reflux but cannot differentiate between physiologic and pathologic reflux
Endoscopy (esophagogastroduodenoscopy [EGD])	A thin flexible tube with a camera and light is passed into the esophagus after sedation has been given	Test of choice in patients with alarm signs Can detect strictures, esophagitis, Barrett's esophagus, and tumors Allows for biopsies
Esophageal pH monitoring	A probe is placed into the esophagus for 24 h or a wireless capsule is placed endoscopically	Measures esophageal pH for 24 h Quantifies reflux Determines pathologic reflux Allows correlation between reflux and symptoms
Esophageal impedance	A catheter is placed into the esophagus	Detects changes in the resistance of electrical current in the esophagus Records esophageal pH Differentiate both antegrade and retrograde transit of liquid and gas Detects acid and nonacid reflux
Esophageal manometry	A catheter is placed into the esophagus	Measures pressures within the esophagus and esophageal sphincters Does not measure reflux

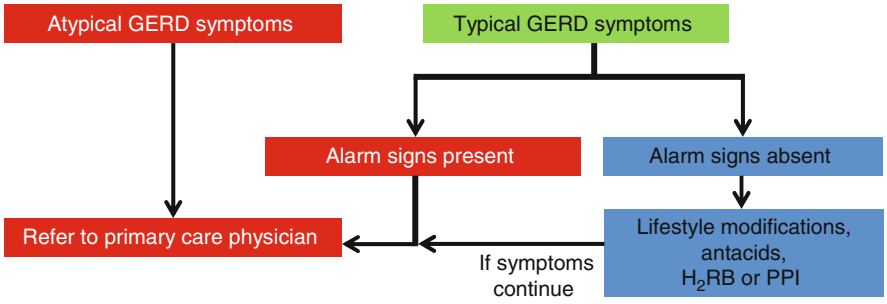


Fig. 12.7 Proposed management algorithm for GERD (gastroesophageal reflux disease) for dental practitioners. *H2RB* histamine-2 receptor blocker, *PPI* proton pump inhibitor

Ambulatory esophageal pH monitoring utilizes a probe that can record the lower esophageal pH for 24 h. The test is done with a pH probe that is passed transnasally to just above the lower esophageal sphincter (LES). The data is collected by a battery-powered device carried by the patient, who also records when he/she is eating and when symptoms are being experienced. The technique allows for correlation of symptoms with reflux episodes. This pH probe monitoring is a sensitive test to detect the presence of esophageal acid. This study is most useful in patients with typical or atypical GERD symptoms who do not respond to therapy. However, this study is not widely available, can be uncomfortable for the patients, and is expensive. The newer Bravo wireless capsule pH testing is more comfortable, but is still expensive as an upper endoscopy is usually needed for placement. This pH probe is attached to the esophageal wall above the LES and lasts for 48 h. The main disadvantages include the capsule becoming dislodged prematurely, esophageal bleeding, or in some rare cases esophageal perforation [9, 51–54] (Table 12.9).

Esophageal impedance testing detects changes in the resistance of electrical current on a catheter placed within the esophagus. In addition to recording esophageal pH, this catheter can differentiate both antegrade and retrograde transit of liquid and gas. Thus, acid and nonacid reflux can be detected, and this test has greater sensitivity than pH testing alone for the detection of GERD. This test is useful for those patients with suspected GERD but with negative esophageal pH testing [9, 51–54] (Table 12.9).

12.7 Management of Dental Erosion Due to GERD

The immediate goal in the treatment of dental erosion resulting from GERD is formulation of the correct differential diagnosis and prompt referral to a primary care physician or gastroenterologist. However, it is important to institute strategies to prevent and control erosion while referring to the above specialists.

12.7.1 Management in Medical Office

It is generally agreed that the overall management of GERD should focus on reducing acid reflux and regurgitation with the use of acid-suppressive medications initially and antireflux surgery if required subsequently [55]. Management of GERD is typically a combination of lifestyle modifications and medication therapy.

12.7.1.1 Patient Lifestyle Modifications to Reduce Acid Reflux

Lifestyle modifications involve changing eating habits, abstaining from smoking or drinking alcohol, and/or weight loss. There are two main categories of lifestyle modifications. One includes modifying the patient's eating habits by avoiding foods and drinks that contribute or aggravate GERD symptoms, avoiding eating fatty or spicy foods, and abstaining from smoking or drinking alcohol or caffeine (Tables 12.4 and 12.10). The other main lifestyle modification is mechanical and includes

Table 12.10 Patient lifestyle modifications that should be encouraged in all patients for non-medication treatment for GERD

Elevating head of bed at least 4–6 inches
Avoid eating food and drinking liquids 3 h prior to bedtime
Eat smaller but more frequent meals
Avoid fatty and spicy foods
Avoid any aggravating foods or drinks
Abstain from smoking and drinking alcohol and caffeine
Reduce weight
Avoid tight-fitting clothes

GERD gastroesophageal reflux disease

decreasing intra-abdominal pressures, such as eating smaller meals, and changing the angle of the gastroesophageal junction by trying to avoid eating 3 h prior to bedtime and elevating the head of the bed [56, 57] (Table 12.10). Lifestyle modification alone may produce remission in 20–30 % of patients with GERD symptoms, but only a few patients are compliant with the restrictions. However, to be effective and successful, recommendations for lifestyle modification in all patients with GERD should be delivered through motivational interviewing. Information and advice delivered through motivational interviewing has been demonstrated to empower people to adopt and sustain healthy behavior [58–60].

12.7.1.2 Medication Therapy to Reduce Acid Reflux

The mainstay medical therapy for GERD includes antacids, histamine-2 receptor blockers (H_2RB or H_2 blockers), and proton pump inhibitors (PPI). Over-the-counter antacids provide symptom relief by neutralizing refluxed gastric acid thereby increasing esophageal pH. Antacids alone may produce remission in 20–30 % of patients with GERD symptoms. Antacids are inexpensive, readily available, convenient, and effective. Short-term use of antacids for occasional symptoms is safe [9, 56, 57] (Table 12.11).

Histamine-2 receptor antagonists or blockers (H_2RB or H_2 blockers) inhibit the secretion of gastric acid competitively by blocking the H_2 receptors located on the gastric parietal cells. H_2RB have a low side-effect profile and are generally well tolerated. H_2RB not only prevent GERD symptoms when taken before meals but also relieve postprandial symptoms in 15–40 min when taken postprandially. H_2RB should be given twice daily to be most effective in patients with frequent GERD. H_2RB are appropriate for patients with mild to moderate GERD and have 50–75 % effectiveness in healing reflux esophagitis [9, 56, 57] (Table 12.11).

Proton pump inhibitors (PPIs) are effective in controlling GERD symptoms that are refractory to antacids and H_2 blockers. PPIs act by blocking the hydrogen-potassium ATPase on the apical surface of the parietal cell. PPIs are more effective than H_2RB because they act on the final common pathway of acid secretion rather than one of the three receptors (histamine, acetylcholine, and gastrin). The timing of PPI use is important as these drugs block only activated proton pumps. They are most effective when taken in the fasting state 30 min before meals, typically before

Table 12.11 Medical therapy for GERD

Therapy	Agent	Dosage	Healing rate (%)[a]
Acid-neutralizing agents	Sodium bicarbonate (Alka-Seltzer)	4 g (2 tabs) PO q 4 h (follow recommendations on container as dosing varies)	20–30
	Calcium carbonate (Maalox, Mylanta, Tums, Rolaids)	1–3 g PO q 6 h (follow recommendations on container as dosing varies)	
	Magnesium hydroxide (milk of magnesia, Maalox, Mylanta, Phillips)	400–1200 mg PO q 6 h (follow recommendations on container as dosing varies)	
	Aluminum hydroxide (Maalox, Pepto-Bismol, Mylanta)	320–1280 mg PO q 6 h (follow recommendations on container as dosing varies)	
Histamine-2 receptor blockers	Cimetidine (Tagamet)	800 mg PO qhs or 400 mg PO BID-QID	50
	Ranitidine (Zantac)	300 mg PO qhs or 150–300 mg PO BID	
	Famotidine (Pepcid)	20–40 mg PO BID	
	Nizatidine (Axid)	150 mg PO BID	
Proton pump inhibitor	Esomeprazole (Nexium)	20–40 mg qday or BID	>80
	Lansoprazole (Prevacid)	15–30 mg qday or BID	
	Omeprazole (Prilosec)	20–40 mg qday or BID	
	Pantoprazole (Protonix)	20–40 mg qday or BID	
	Rabeprazole (Aciphex)	20–40 mg qday or BID	
Surgery	Nissen fundoplication		>80

GERD gastroesophageal reflux disease, *PO* per os or mouth, *qday* every day, *q 4 h* every 4 h, q 6 h every 6 h, *qhs* at bedtime, *BID* 2 times a day, *QID* 4 times a day
[a]Healing rates for erosive reflux esophagitis

breakfast. Patients are instructed to wait 30–40 min then eat breakfast. The PPIs attach to the activated pump, blocking parietal cell acid secretion. PPIs are indicated as initial therapy in patients with moderate to severe GERD and in patients with complications from GERD such as bleeding and strictures. PPI therapy was significantly better than H_2RB therapy in the treatment of GERD and esophagitis at 4–8 weeks. Independent comparisons of the currently available PPIs have consistently shown first-generation PPIs (except for esomeprazole) to be essentially equal in esophagitis healing and gastric pH profiles. A comparison of five available PPIs in controlling 24-h gastric pH showed a statistically significant advantage for esomeprazole on day 5 of therapy [61]. PPIs also have low side-effect profile with 3% of patients getting headaches or diarrhea, and rare side effects include hepatitis and interstitial nephritis and osteoporosis with chronic use [51, 56, 62] (Table 12.11).

Once patients with GERD symptoms have responded to lifestyle modifications and medical therapy and achieved symptom remission, most should have a trial of medication withdrawal. Symptom relief should be sustained for 2–3 months before attempting to withdraw medications. PPI therapy can be tapered to an every-other-day regimen or a reduced dose or switched to a H_2RB. Most patients

who are treated with H_2RB are on twice-daily regimen, and the initial tapering should be to a once-a-day regimen. If a patient tolerates a taper for 2–4 weeks without an increase in symptoms, the dosage can be further reduced or possibly discontinued. The goal of long-term treatment is to step down management to the lowest level of medical therapy that controls symptoms or consider surgery. However, if a patient experiences recurrent symptoms, the medication regimen should be increased until symptom resolution is achieved. If long-term PPI therapy is abruptly withdrawn (not tapered), many patients will experience symptoms that are worse than their initial presenting complaints. This phenomenon of "rebound acid hypersecretion" occurs when parietal cells secrete elevated amounts of acid after a prolonged blockade. A prolonged tapering of PPI therapy helps improve these symptoms [9, 51, 56, 63, 64].

Therapy with PPI not only has efficacy in GERD, but studies have also shown PPI therapy suppresses tooth erosion in patient with GERD. A randomized control trial revealed short-term suppression of active tooth erosion following the treatment of medically confirmed GERD with a PPI. Optical coherence tomography was used to quantify the extent of enamel demineralization at multiple specific sites in specific visibly eroded teeth both before and after 3 weeks of esomeprazole therapy. In this double-blinded study, there was significantly less enamel thickness lost in the 14 adults taking esomeprazole (mean = 7.20 μm) than in the 15 adults taking a placebo (mean = 15.25 μm). Evidence of a mild remineralization of eroded teeth in the esomeprazole patients was shown by a decreased optical reflectance at a depth of 25 μm. Because nocturnal acid control may be inadequate with PPIs, some erosion from GERD may have continued during sleep. Most of the patients had mildly symptomatic GERD, as they presented with a primary complaint of dental erosion [65].

For patients with gastroparesis, or delayed gastric emptying, the majority of patients will achieve symptomatic resolution with H_2RB or PPI, although some will need treatment with prokinetic agents. Prokinetic agents may offer some symptomatic relief and reduce gastric volume; however, they have severe potential neurologic side effects, which limit their use and should only be prescribed by an experienced physician. Dietary changes such as low-fat, frequent, small meals may also be helpful in controlling symptoms [51, 56].

12.7.1.3 Surgical Therapy to Reduce Acid Reflux

While most patients with GERD can be successfully managed with lifestyle modifications and medical therapy, some patients can have persistent symptoms that ultimately require surgical intervention. The goal of antireflux surgery is to narrow the lower esophageal luminal diameter to prevent the reflux of gastric contents. The most widely performed procedure is the Nissen fundoplication. Several studies have reported a symptomatic response of 80–90 % in patients undergoing a Nissen fundoplication. However, it has been noted that 62 % of patients again require medication therapy 10 years after antireflux surgery. Potential complications from this procedure include dysphagia, chest pain, gas-bloat syndrome, and vagal nerve injuries that can lead to gastroparesis and diarrhea. The prevalence of these complications ranges between 5 and 20 % [51, 56, 66, 67].

12.7.2 Management in Dental Office

12.7.2.1 Patient Self-Care

Although the general responsibilities of the patient in prevention and control of his/her erosive tooth wear have been discussed in detail in Chap. 8 of this book [68], patients with GERD, in particular, should take the following precautions [69], which should be provided to them with a handout on oral health.

A. "Bite guards" should be worn while sleeping. The inside (tooth surface) of the guard should be coated with a small amount of sodium bicarbonate powder or milk of magnesia, to neutralize any refluxed acid pooling in it. The guard should have occlusal coverage only, so that saliva flow to aid remineralization is maintained.
B. Tooth brushing instruction:
   (a) Use a medium nylon brush.
   (b) Do not use abrasive "whitening" toothpastes.
   (c) Brush with the Bass technique to avoid horizontal strokes.
   (d) Avoid tooth brushing immediately when you wake up in the morning because the refluxed acid has softened the tooth surfaces, which may wear away with tooth brushing. Thus patients should use any of the following strategies to freshen their mouths and also wait for at least 60 minutes before tooth brushing.
   - Fluoride mouthwash to enhance rapid remineralization of the softened tooth surface.
   - Fluoride tablets and fluoride lozenge, which have been demonstrated as effective remineralizing agent.
   - Sugar-free lozenges or chewing gum to increase saliva flow to facilitate rapid remineralization of the softened tooth tissue, neutralize the acidity, and provide alkaline environment necessary for remineralization. Buffering capacity and bicarbonate content of stimulated saliva is higher than that of unstimulated saliva. It is also speculated that saliva stimulation would enhance the formation of acquired salivary pellicle, which has been shown to protect teeth against erosive attack.
   - Dairy products (e.g., fresh milk) have been shown to reharden softened tooth surface.
   - Sugar-free antacid tablets or a pinch of sodium bicarbonate (or baking soda) dissolved in some water may be used to neutralize the acidic oral fluid.
C. Use high-fluoride concentration toothpaste as well as fluoride mouthrinse for their daily routine oral hygiene practice.
D. Visit their dentist regularly for professional clinical care.

12.7.2.2 Dentist's Clinical Care

The general responsibilities of the dental practitioner in prevention and control of dental erosion in a patient have been discussed in detail in Chap. 9 of this book [70] and include the following:

- Professionally applied remineralization methods for softened tooth surfaces
- Surface protection coatings with or without remineralizing potential
- Motivational interviewing for behavioral change
- Referral to a physician
- Recall, review, and monitoring

In general, in a dental practice, the management of a patient suffering from dental erosion due to gastroesophageal reflux disease should be based on the following:

1. The dentist should advice the patient on self-care strategies to reduce acid reflux and tooth erosion, which should consist of the above discussed (a) patient life-style modifications to reduce acid reflux and (b) patient self-care to prevent and control dental erosion.
2. The dentist's clinical care to protect the teeth against erosive attack, including motivational interviewing for behavioral change.
3. The Dentist should then refer the patient to a physician for management of GERD, which may consist of medication therapy and if necessary, surgical therapy, to reduce acid reflux.

Conclusion

Gastroesophageal reflux disease, or GERD, is an increasingly common and potentially serious condition, with various esophageal and extraesophageal adverse health effects, of which dental practitioners should be aware. Clinicians should also be aware of the predisposing risk factors for GERD and its classical esophageal and extraesophageal symptoms and signs. However, not all affected persons will have the classic symptoms of gastric reflux. Dental practitioners may be the first persons to diagnose the possibility of GERD in these "silent refluxers," particularly when observing unexplained instances of tooth erosion. Numerous studies in adults and children have shown a clear relationship between GERD and dental erosion. Dental practitioners should consider the presence of this pathology when they observe significant loss of dental tissue. Identification is required in order to treat and eliminate etiological factors. The high prevalence of dental erosion in GERD that has been reported justifies giving GERD-related dental damage more attention. Management of dental erosions due to GERD should include proper diagnosis and referral for workup, lifestyle modifications, and acid-suppressive medications, in addition to the proper dental checkup and appropriate dental therapy.

References

1. Bartlett DW, Evans DF, Smith BGN, et al. The relationship gastro-oesophageal reflux disease and dental erosions. J Oral Rehabil. 1996;23:289–97.
2. Mair LH. Wear in the mouth: the tribological dimension. In: Addy M, Edgar WM, Embery G, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. p. 181–8.

3. Picos A, Chisnoiu A, Dumitrascu DL, et al. Dental erosion in patients with gastroesophageal reflux disease. Adv Clin Exp Med. 2013;22:303–7.
4. Vakil N, van Zanten SV, Kahrilas P, et al. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol. 2006;101:1900–43.
5. Pace F, Pallotta S, Tonini M, et al. Systematic review: gastro-oesophageal reflux disease and dental lesions. Aliment Pharmacol Ther. 2008;27:1179–86.
6. Wong BC-Y, Wong WM, Smales R, et al. Gastroesophageal reflux disease and tooth erosion. In: Yip KHK, Smales RJ, Kaidonis JA, editors. Tooth erosion: prevention and treatment. New Delhi: Jaypee Brothers Medical; 2006. p. 47–53.
7. Pisegna J, Holtmann G, Howden CW, et al. Review article: oesophageal complications and consequences of persistent gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2004;20:47–56.
8. Sandler RS, Everhart JE, Donowitz M, et al. The Burden of selected digestive diseases in the United States. Gastroenterology. 2002;122:1500–11.
9. Saltzman JR. Gastroesophageal reflux disease. In: Greenberger N, Blumberg R, Burakoff R, editors. Current diagnosis & treatment: gastroenterology, hepatology, and endoscopy. 2nd ed. New York: McGraw-Hill; 2012. p. 147–55.
10. Holbrook WP, Furuholm J, Gudmundsson K, et al. Gastric reflux is a significant causative factor of tooth erosion. J Dent Res. 2009;88:422–6.
11. Ersin NK, Onçağ O, Tümgör G, et al. Oral and dental manifestations of gastroesophageal reflux disease in children: a preliminary study. Pediatr Dent. 2006;28:279–84.
12. Linnett V, Seow WK, Connor F, et al. Oral health of children with gastro-esophageal reflux disease: a controlled study. Aust Dent J. 2002;47:156–62.
13. Dahshan A, Patel H, Delaney J, et al. Gastroesophageal reflux disease and dental erosion in children. J Pediatr. 2002;140:474–8.
14. Farahmand F, Sabbaghian M, Ghodousi S, et al. Gastroesophageal reflux disease and tooth erosion: a cross-sectional observation study. Gut Liver. 2013;7:278–81.
15. Jones MP, Sloan SS, Rabine JC, et al. Hiatal hernia size is the dominant determinant of esophagitis presence and severity in gastroesophageal reflux disease. Am J Gastroenterol. 2001;96:1711–7.
16. Dodds WJ, Dent J, Hogan WJ. Mechanisms of gastroesophageal reflux in patients with reflux esophagitis. N Engl J Med. 1982;307:1547–52.
17. Horowitz M, Su YG, Rayner CK, et al. Gastroparesis: prevalence, clinical significance and treatment. Can J Gastroenterol. 2001;15:805–13.
18. El-Serag HB, Ergun GA, Pandolfino J, et al. Obesity increases oesophageal acid exposure. Gut. 2007;56:749–55.
19. Jacobson BC, Somers SC, Fuchs CS, et al. Body-mass index and symptoms of gastroesophageal reflux in women. N Engl J Med. 2006;354:2340–8.
20. Moayyedi P, Axon AT. Review article: Gastroesophageal reflux disease: the extent of the problem. Aliment Pharmacol Ther. 2005;22:11–9.
21. Helm JF. Role of saliva in esophageal function and disease. Dysphagia. 1989;4:76–84.
22. Bouchoucha M, Callais F, Renard P, et al. Relationship between acid neutralization capacity of saliva and gastro-esophageal reflux. Arch Physiol Biochem. 1997;105:19–26.
23. Groen JN, Smout AJ. Supra-oesophageal manifestations of gastro-oesophageal reflux disease. Eur J Gastroenterol Hepatol. 2003;15:1339–50.
24. Filipi K, Halackova Z, Filipi V, et al. Oral health status, salivary factors and microbial analysis in patients with active gastro-oesophageal reflux disease. Int Dent J. 2011;61:231–7.
25. Gudmundsson K, Kristleifsson G, Theodors A, et al. Tooth erosion, gastroesophageal reflux, and salivary buffer capacity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1995;79:185–9.
26. Lazarchik DA, Filler SJ. Dental erosion: predominant oral lesion in gastroesophageal reflux disease. Am J Gastroenterol. 2000;95:33–8.

27. Busscher HJ, Goedhart W, Ruben J, et al. Wettability of dental enamel by soft drinks as compared to saliva and enamel demineralization. In: Addy M, Embery G, Edgar WM, Orchardson R, editors. Tooth wear and sensitivity. London: Martin Dunitz; 2000. p. 197–200.
28. Imfeld T. Prevention of progression of dental erosion by professional and individual prophylactic measures. Eur J Oral Sci. 1996;104:215–20.
29. Jones L, Lekkas D, Hunt D, et al. Studies on dental erosion: an in vivo-in vitro model of endogenous dental erosion—its application to testing protection by fluoride gel application. Aust Dent J. 2002;47:304–8.
30. Rees J, Loyn T, Chadwick B. Pronamel and tooth mousse: an initial assessment of erosion prevention in vitro. J Dent. 2007;35:355–7.
31. Ranjitkar S, Narayana T, Kaidonis JA, et al. The effect of casein phosphopeptide-amorphous calcium phosphate on erosive dentine wear. Aust Dent J. 2009;54:101–7.
32. Ramalingam L, Messer LB, Reynolds EC. Adding casein phosphopeptide-amorphous calcium phosphate to sports drinks to eliminate in vitro erosion. Pediatr Dent. 2005;27:61–7.
33. Ganss C, Klimek J, Brune V, et al. Effects of two fluoridation measures on erosion progression in human enamel and dentine in situ. Caries Res. 2004;38:561–6.
34. Willumsen T, Øgaard B, Hansen BF, et al. Effects from pretreatment of stannous fluoride versus sodium fluoride on enamel exposed to 0.1M or 0.01M hydrochloric acid. Acta Odontol Scand. 2004;62:278–81.
35. Ranjitkar S. Biology of tooth wear: preventive strategies. Ph.D. thesis. Adelaide: The University of Adelaide; 2009.
36. Ranjitkar S, Kaidonis JA, Smales RJ. Gastroesophageal reflux disease and tooth erosion. Int J Dent. 2012. Article ID 479850, 10 pages.
37. Rabine JC, Nostrant TT. Oral manifestations of gastrointestinal diseases. In: Yamada T, Alpers DH, Kalloo AN, et al., editors. Atlas of gastroenterology. 4th ed. Oxford: Blackwell Publishing; 2009. p. 839–45.
38. Tasker A, Dettmar PW, Panetti M, et al. Is gastric reflux a cause of otitis media with effusion in children? Laryngoscope. 2002;112:1930–4.
39. Lussi A, Jaeggi T, Scharer S, et al. The influence of different factors on in vitro enamel erosion. Caries Res. 1993;27:387–93.
40. Smith BGN, Knight JK, et al. An index for measuring the wear of teeth. Br Dent J. 1984;156:435–8.
41. Fass R, Ofman JJ, Gralnek IM, et al. Clinical and economic assessment of the omeprazole test in patients with symptoms suggestive of gastroesophageal reflux disease. Arch Intern Med. 1999;159:2161–8.
42. Jones R, Coyne K, Wiklund I. The gastro-oesophageal reflux disease impact scale: a patient management tool for primary care. Aliment Pharmacol Ther. 2007;25:1451–9.
43. Manterola C, Muñoz S, Grande L, et al. Initial validation of a questionnaire for detecting gastroesophageal reflux disease in epidemiological settings. J Clin Epidemiol. 2002;55:1041–5.
44. Rubin G, Uebel P, Brimo-Hayek A, et al. Validation of a brief symptom questionnaire (ReQuest in Practice) for patients with gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2008;27:846–51.
45. Ofman JJ, Shaw M, Sadik K, et al. Identifying patients with gastro-esophageal reflux disease: validation of a practical screening tool. Dig Dis Sci. 2002;47:1863–9.
46. Shaw MJ, Talley NJ, Beebe TJ, et al. Initial validation of a diagnostic questionnaire for gastroesophageal reflux disease. Am J Gastroenterol. 2001;96:52–7.
47. Vakil N, Dent J, Jones R, et al. The accuracy and predictors of a clinical diagnosis of GERD by family practitioners and gastroenterologists. Gastroenterology. 2008;134:A598.
48. Bai Y, Du Y, Zou D, et al. Gastroesophageal reflux disease questionnaire (GerdQ) in real-work practice. J Gastroenterol Hepatol. 2013;28:626–31.
49. Jonasson C, Moum B, Bang C, et al. Randomised clinical trial: a comparison between a GerdQ-based algorithm and an endoscopy-based approach for the diagnosis and initial treatment of GERD. Aliment Pharmacol Ther. 2012;35:1290–300.

50. Jonasson C, Wernersson B, Hoff DA, et al. Validation of the GerdQ questionnaire for the diagnosis of gastro-oesophageal reflux disease. Aliment Pharmacol Ther. 2013;37:564–72.
51. DeVault KR, Castell DO. American College of Gastroenterology. Updated guidelines for the diagnosis and treatment of gastroesophageal reflux disease. Am J Gastroenterol. 2005;100:190–200.
52. Hirano I, Richter JE. Practice Parameters Committee of the American College of Gastroenterology. ACG practice guidelines: esophageal reflux testing. Am J Gastroenterol. 2007;102:668–85.
53. Lichtenstein DR, Cash BD, et al. Standards of Practice Committee: role of endoscopy in the management of GERD. Gastrointest Endosc. 2007;66:219–24.
54. Pritchett JM, Aslam M, Slaughter JC, et al. Efficacy of esophageal impedance/pH monitoring in patients with refractory gastroesophageal reflux disease, on and off therapy. Clin Gastroenterol Hepatol. 2009;7:743–8.
55. Vaezi MF, Richter JE. Duodenogastroesophageal reflux and methods to monitor nonacidic reflux. Am J Med. 2001;111:160S–8.
56. Kahrilas PJ, Shaheen NJ, Vaezi MF. American Gastroenterological Association Institute, Clinical Practice and Quality Measurement Committee. American Gastroenterological Association Institute technical review on the management of gastroesophageal reflux disease. Gastroenterology. 2008;135:1392–413.
57. Kaltenbach T, Crockett S, Gerson LB. Are lifestyle measures effective in patients with gastroesophageal reflux disease? An evidence-based approach. Arch Intern Med. 2006;166:965–71.
58. Gao X, Lo ECM, Kot SCC, Chan KCM. Motivational interviewing in improving oral health: a systematic review of randomized controlled trials. J Periodontol. 2014;85(3):426–3.
59. Resnicow K, McMaster F. Motivational Interviewing moving from why to how with autonomy support. Int J Behav Nutr Phys Act. 2012;9:19.
60. Lundahl B, Burke BL. The effectiveness and applicability of motivational interviewing: a practice-friendly review of four meta-analyses. J Clin Psychol. 2009;65:1232–45. In session.
61. Klok RM, Postma MJ, van Hou BA, et al. Meta-analysis: comparing the efficacy of proton pump inhibitors in short-term use. Aliment Pharmacol Ther. 2003;17:1237–45.
62. Yang YX, Metz DC. Safety of proton pump inhibitor exposure. Gastroenterology. 2010;139:1115–27.
63. Inadomi JM, Jamal R, Murata GH, et al. Step-down management of gastroesophageal reflux disease. Gastroenterology. 2001;121:1095–100.
64. Inadomi JM, McIntyre L, Bernard L, et al. Step-down from multiple-to single-dose proton pump inhibitors (PPIs): a prospective study of patients with heartburn or acid regurgitation completely relieved with PPIs. Am J Gastroenterol. 2003;98:1940–4.
65. Wilder-Smith CH, Wilder-Smith P, Kawakami-Wong H, et al. Quantification of dental erosions in patients with GERD using optical coherence tomography before and after double-blind, randomized treatment with esomeprazole or placebo. Am J Gastroenterol. 2009;104:2788–95.
66. Catarci M, Gentileschi P, Papi C, et al. Evidence-based appraisal of anti-reflux fundoplication. Am J Surg. 2004;239:325–37.
67. Finlayson SR, Laycock WS, Birkmeyer JD. National trends in utilization and outcomes of anti-reflux surgery. Surg Endosc. 2003;17:864–7.
68. Buzalaf MA, Cardoso CA, Magalhães AC, Amaechi BT. Prevention and control of dental erosion: patient self-care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 133–50.
69. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33:243–52.
70. Kaidonis JA. Prevention and control of dental erosion: professional clinic care. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 151–68.

Restoration of the Worn Dentition

13

Paul A. King

Abstract

Prevention of excessive tooth wear should form the basis of any ongoing lifelong dental management. In some situations, however, it may also become necessary to consider interventive restorative treatment in order to protect vulnerable tooth surfaces and re-establish satisfactory appearance and function. A wide range of treatment options currently exist when considering the restoration of the worn dentition ranging from the more conventional fixed and removable prosthodontic approach to some of the newer, less invasive and minimal preparation adhesive techniques. This chapter illustrates and discusses the treatment options now available to the dental surgeon when restoring the worn dentition. While a number of the minimal preparation adhesive methods would appear to offer significant advantages over more traditional measures, only time and careful clinical evaluation will dictate if these become the accepted treatment patterns of tomorrow.

13.1 Introduction

The effects of tooth wear can present in a variety of forms and severity (Fig. 13.1) depending on the aetiological factors responsible. Although to some extent the severity of tooth wear is age dependent [1], an increasing number of both adult and younger patients are experiencing tooth wear, usually erosive in nature [2–4] (Fig. 13.2). It is generally agreed that prevention of further tooth wear should form the basis of any ongoing lifelong dental management [5, 6]. However, in some

P.A. King, BDS, MSc, FDSRCS
Restorative Dentistry, University of Bristol Dental Hospital and School,
Lower Maudlin Street, Bristol BS1 2LY, UK
e-mail: paulkinginbath@btinternet.com

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_13

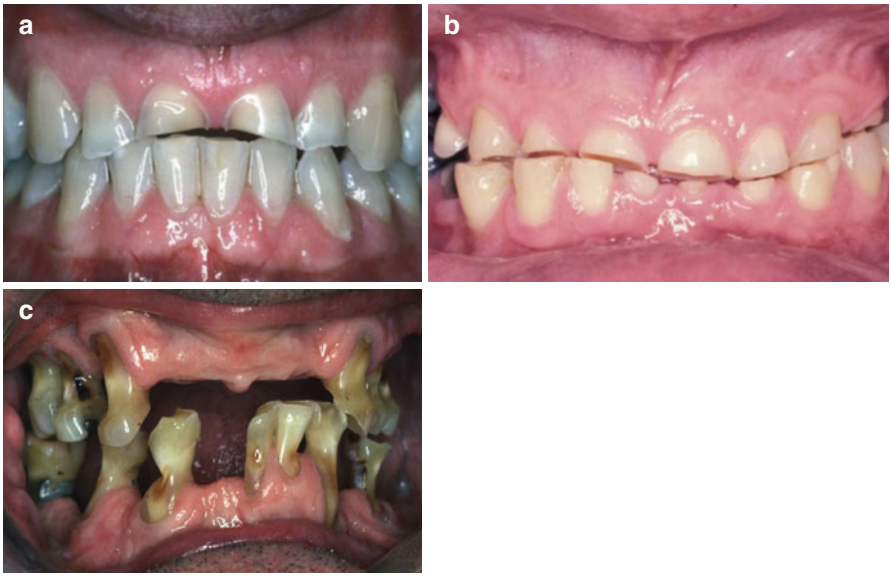


Fig. 13.1 (**a**–**c**) Patients (middle to late-aged) presenting with moderate/severe tooth wear. (**a**) Anterior tooth wear mainly erosion/abrasion. (**b**) Tooth wear mainly attrition. (**c**) Generalised tooth wear multifactorial

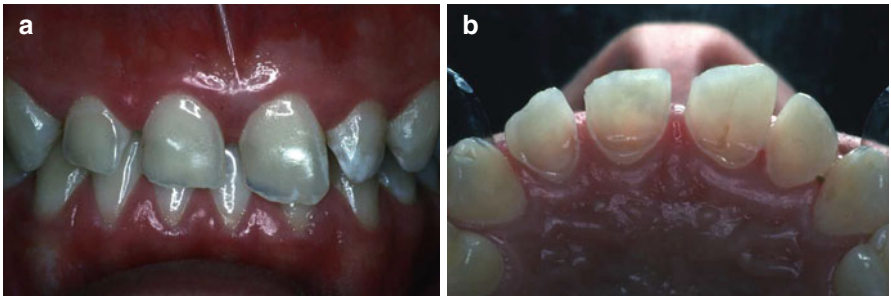


Fig. 13.2 (**a**, **b**) Young patient (age 17 years) exhibiting severe localised tooth wear mainly erosive in nature. (**a**) Labial view. (**b**) Palatal view

situations, it may also become necessary to consider interventive restorative treatment [7, 8]. The clearest indications are where:

- The patient feels that the appearance of the teeth is unacceptable.
- Normal function is disrupted due to the loss of tooth tissue and/or ongoing pulp sensitivity which does not respond to conservative measures.
- There is progressive tooth wear which may result in pulp necrosis and/or teeth becoming difficult to restore.

A broad range of restorative treatment options are possible with today's materials and techniques and usually range between:

- Conventional fixed restorations
- Removable onlay/overlay prostheses
- Minimal preparation adhesive restorations

This chapter will illustrate and discuss some of the conventional methods used to restore the worn dentition as well as the newer, less invasive and minimal preparation adhesive techniques now available to the dental surgeon.

13.2 Conventional Fixed Restorations

Conventional fixed restorations, usually in the form of porcelain-fused-to-metal (PFM) and all-metal crowns, have been used for many years for restoring worn and broken down teeth. The clinical performance of conventional crown and fixed bridge restorations have been well documented, and in general tolerable survival times have been demonstrated [9–12]. Unfortunately, studies have not assessed risk factors specifically associated with the various forms of tooth wear, although there is general agreement that restorations in patients exhibiting distinct parafunctional clenching and grinding habits have a higher risk of failure compared with other types of tooth wear.

Tooth preparation for conventional crown restorations is an invasive procedure, especially if porcelain is required to satisfy aesthetic demands, and as a consequence may further compromise an already damaged dentition [13]. Pulp necrosis, tooth fracture, loss of cementation and marginal caries can all occur following the placement of crown restorations, which will often complicate any subsequent repair or replacement, resulting in possible premature tooth loss [14, 15]. This is particularly the case if crowns are prescribed for the younger patient. Notwithstanding some of the risk factors described, these type of restorations are extensively used and offer an effective way of restoring worn and missing teeth, allowing a degree of versatility with regard to appearance and occlusal form. Additionally, one of the potential advantages of this treatment approach is the ability to use provisional crowns for a period of time, thus providing the opportunity for clinician and patient to assess appearance and function and make any modifications as necessary.

Often complicating the restoration of worn teeth is the need to recreate inter-occlusal space lost as a result of compensatory eruption of opposing teeth during the process of tooth wear [16]. This is often the case with anterior teeth and if not addressed will result in a compromised finish (Fig. 13.3). In these circumstances, there are a number of well-established conventional restorative techniques available to overcome the difficulties of reduced crown height and lack of inter-occlusal space [17]. The main options either individually or in combination are:

- Opposing tooth reduction
- Elective endodontic treatment and post retention
- Occlusal adjustment (retruded arc of mandibular closure)
- Periodontal surgical crown lengthening
- Localized orthodontic tooth movement (Conventional Fixed Appliance or 'Dahl' appliance)
- Overall increase in occlusal vertical dimension

For an adequate restoration of good appearance and durable function, the role of opposing tooth reduction, elective pulp extirpation and post retention, occlusal adjustment and periodontal surgical crown lengthening alone may be of limited help and can compromise remaining tooth structure and periodontal support. Periodontal surgical crown lengthening is capable of dramatically improving available coronal tooth structure for adequate crown preparation (Fig. 13.4) and is often required when restoring more severe forms of tooth wear. However, it is an invasive procedure with postoperative sensitivity and can create a number of subsequent restorative difficulties, such as interproximal spacing and placement of crown margins on root surfaces [18, 19].

One of the more satisfactory and conservative ways of recreating space, particularly in situations of localized anterior tooth wear, is by orthodontic tooth movement. A number of techniques can be used to achieve this [20], with a fixed or

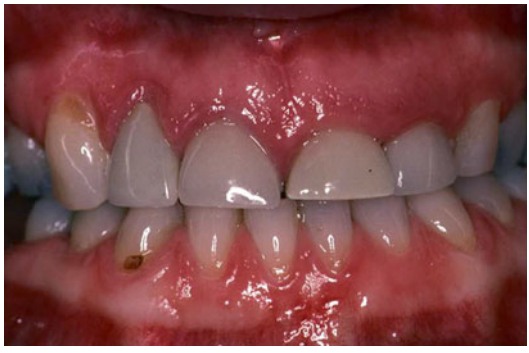


Fig. 13.3 Anterior crowns constructed to conform to the existing worn teeth without recreation of lost inter-incisal space resulting in poor aesthetics and retention form

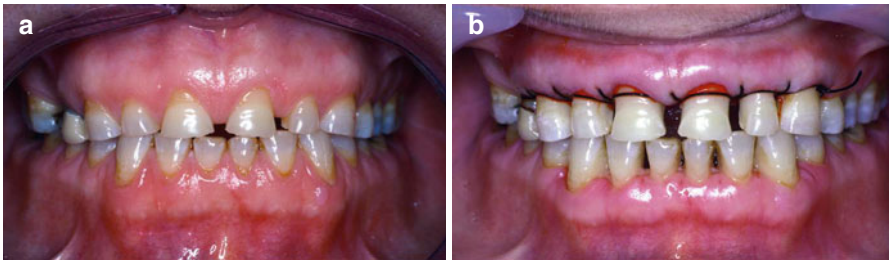


Fig. 13.4 (**a**, **b**) Periodontal surgical crown lengthening on worn upper anterior teeth prior to the construction of conventional crown restorations. (**a**) Before surgery. (**b**) Immediately after surgery

removable metal-based anterior bite plane being an established method or more often today the use of direct composite resin techniques. The so-called Dahl appliance, described after the author, achieves space recreation by a combination of anterior tooth intrusion and posterior tooth extrusion [21, 22]. This localized orthodontic treatment provides the opportunity to maximize the appearance and function of the subsequent crowns and preserves tooth tissue (Fig. 13.5).

In certain situations where there is generalized tooth wear and sufficient indications to consider crown restorations for the posterior teeth, a full mouth crown reconstruction at an overall increase in occlusal vertical dimension will usually provide adequate space for anterior restorations (Fig. 13.6). This situation will avoid the need for an anterior orthodontic appliance as well as preserving valuable incisal

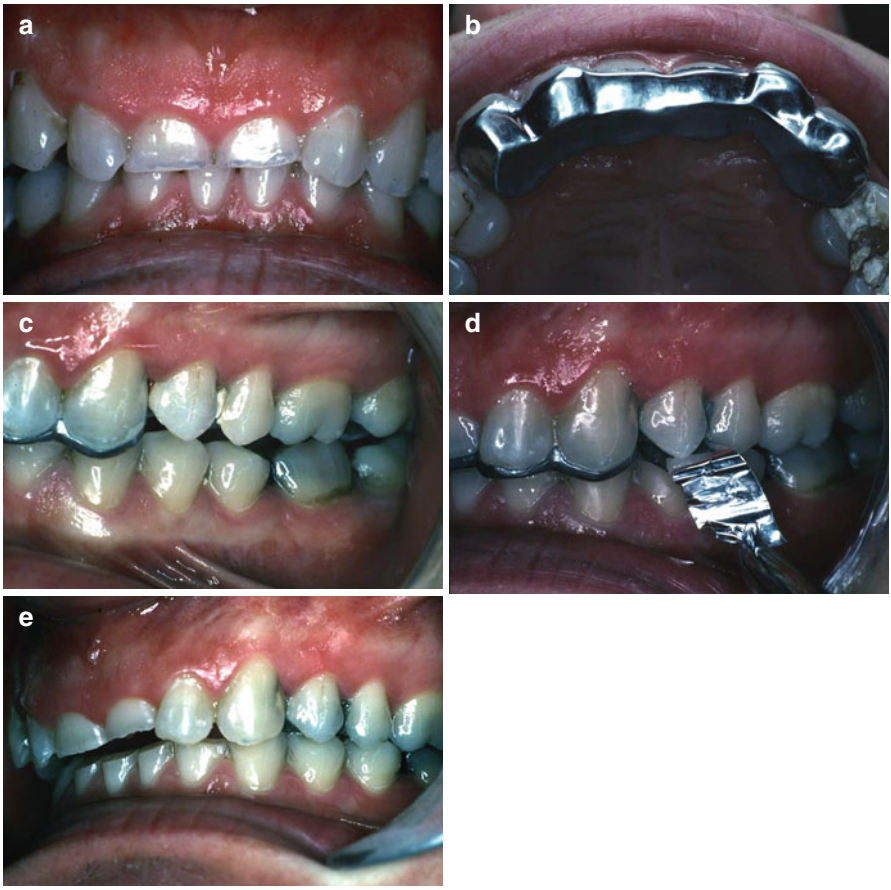


Fig. 13.5 (**a**–**e**) The use of an orthodontic 'Dahl appliance' to recreate lost inter-incisal space prior to the restoration of worn anterior teeth. (**a**) Localised anterior tooth wear. (**b**) 'Dahl appliance' cemented in place. (**c**) Initial space in posterior quadrants. (**d**) Regained posterior tooth contacts after 6 months. (**e**) Inter-incisal space recreated following the removal of the 'Dahl appliance' prior to anterior restorations

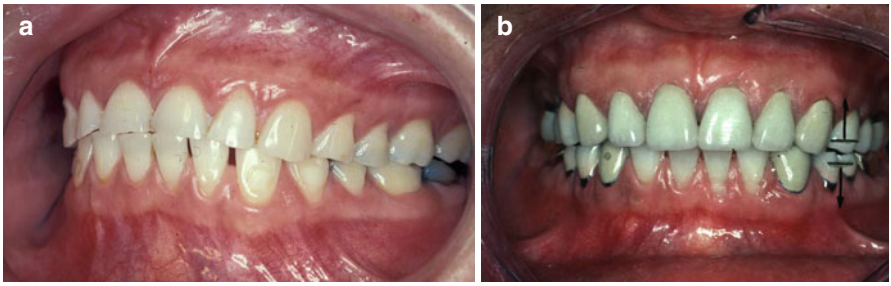


Fig. 13.6 (**a**, **b**) Generalised tooth wear restored with conventional crown restorations in the anterior and posterior segments at an overall increase in occlusal vertical dimension. (**a**) Before restoration. (**b**) After restoration

and occlusal tooth tissue. Although excellent and predictable results can be achieved by this method, the process is relatively complex and requires a number of carefully planned stages, appropriate operator skills and knowledge, time and good technical laboratory support [23, 24]. As a consequence, this form of treatment may not be accessible for many patients.

It is possible to achieve an increase in occlusal vertical dimension with the use of a removable posterior onlay prosthesis in combination with anterior fixed crown restorations. This approach relies heavily on patient compliance and may result in an unpredictable outcome for the anterior restorations due to adverse occlusal loads if the removable prosthesis is not used periodically. If a removable prosthesis is being considered to provide posterior occlusal support in this situation, then it may be more sensible and predictable to restore the worn anterior teeth with the same removable prosthesis.

13.3 Removable Onlay/Overlay Prostheses

The use of a removable onlay/overlay prosthesis can be a valuable means of rehabilitating patients with moderate/severe tooth wear, particularly when there are also missing strategic teeth to be replaced. This form of treatment has been advocated by a number of clinicians for patients with more severe forms of tooth wear [25–29]. This approach provides a relatively simple, non-invasive and cost-effective way to achieve improvements in appearance and function of the dentition.

The construction of a provisional acrylic resin removable prosthesis is recommended initially, allowing the opportunity to carry out modifications to the shape, position and occlusal relationship of the prosthetic teeth and soft tissues, as well as assessing the patient's tolerance of a removable prosthesis (Fig. 13.7). It is advisable to avoid any significant tooth preparation at this stage, but if this treatment approach is to be continued in the longer term then subsequent prostheses, often incorporating a cobalt-chromium framework, will usually require some tooth preparation in order to optimize appearance, fit and retention. Space demands are usually greatest in the

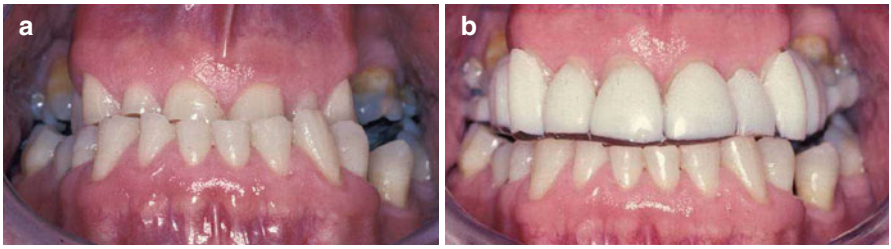


Fig. 13.7 (**a**, **b**) Moderate/severe tooth wear with an unfavourable occlusal relationship initially restored with a provisional onlay/overlay removable prosthesis to assess appearance and function. (**a**) Before restoration. (**b**) After restoration with removable prosthesis

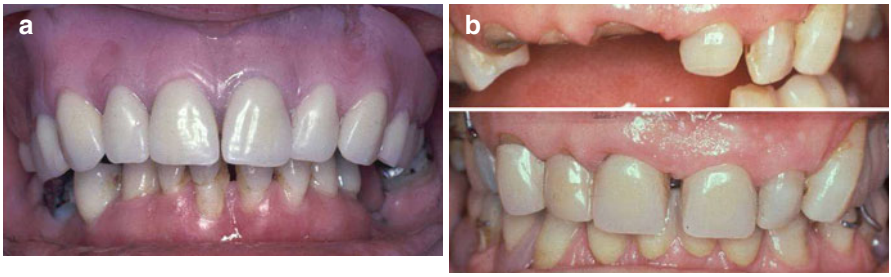


Fig. 13.8 (**a**, **b**) Examples of overlay removable prostheses: (**a**) Full labial flange, (**b**) gingival-fitting anterior tooth facings

anterior region, both in the vertical and labio-lingual dimensions, and will be influenced by the amount of sound tooth structure remaining and changes to the occlusal vertical dimension. Unless modified by tooth reduction or extraction, the available space will determine whether or not an anterior labial flange can be used, or alternatively, gingival fitting and/or butt-fitting tooth facings (Fig. 13.8). The final decision may to some extent depend on the patient's aesthetic demands and desire to avoid or limit any necessary tooth reduction.

There are some well-established advantages in retaining teeth as overdenture abutments, such as maintenance of alveolar bone and support, improved sensory feedback, masticatory performance and reduced psychological trauma of tooth extraction [28]. Some disadvantages also exist, particularly if patients are unable to establish and maintain adequate oral and denture plaque control. In this situation, the abutment teeth will be at an increased risk of primary dental disease (Fig. 13.9), although the daily application of a non-acidulated fluoride gel may reduce the risk of root surface caries [30].

Maintenance demands are relatively high for this form of prosthesis [31], with material wear and fracture being common, particularly in patients exhibiting parafunctional clenching/grinding habits (Fig. 13.10). The use of extensive metal frameworks, including incisal and occlusal coverage (Fig. 13.11), may reduce the regularity of repair but increase the clinical and technical complexity when repair or

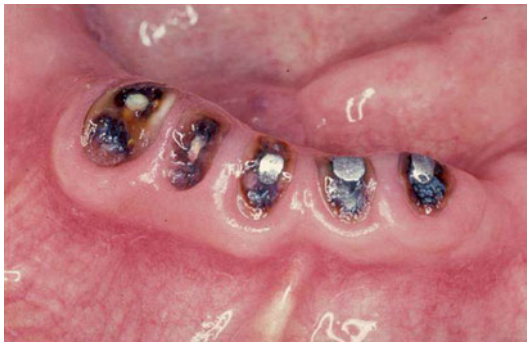


Fig. 13.9 Root surface caries affecting grossly worn teeth restored with an overlay removable prosthesis

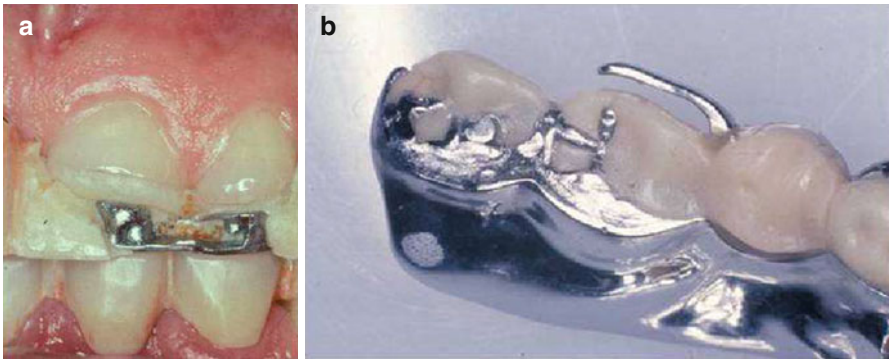


Fig. 13.10 (**a**, **b**) Fracture and wear of acrylic resin facings on removable prostheses often demonstrated in patients with parafunctional clenching/grinding habits. (**a**) Anterior tooth facings. (**b**) Posterior onlay components

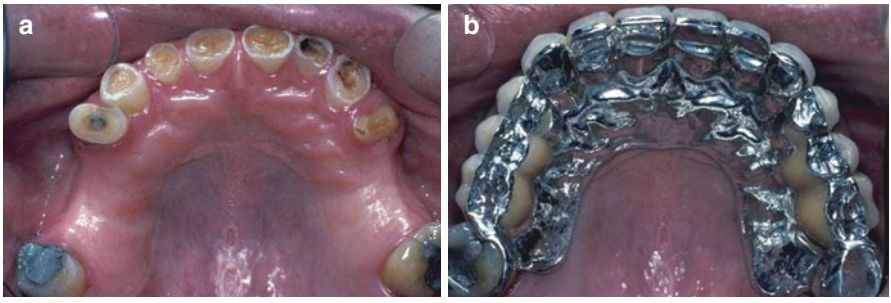


Fig. 13.11 (**a**, **b**) The use of a metal framework incorporating incisal and occlusal coverage used to strengthen removable onlay/overlay prostheses for patients demonstrating significant parafunctional clenching/grinding habits. (**a**) Before restoration. (**b**) After restoration with removable onlay/overlay prosthesis

replacement eventually becomes necessary. This complexity may be further exacerbated if copings and precision attachments have been utilized on abutment teeth to increase retention and stability of the prosthesis [28].

While for certain situations this form of treatment offers a satisfactory way of restoring the worn dentition, for many patients difficulties arise in adapting both functionally and psychologically to a removable prosthesis and this approach is often seen as a last resort.

13.4 Minimal Preparation Adhesive Restorations

Since the introduction to the dental profession of the acid-etch technique using phosphoric acid by Buonocore in 1955 [32], and an early form of Bis-GMA-based composite resin by Bowen in the early 1960s [33], there has been significant progression in the development of adhesive materials and techniques within dentistry. With the additional development of glass ionomer cements, dentin bonding agents, silane primers and composite resin luting cements, it is now possible to produce acceptable adhesive bonds between enamel and dentin tooth tissue with a variety of materials such as composite resins, ceramics and metal alloys suitable for use in situations where restoration of the worn dentition is necessary [34–42].

The final section highlights the range of adhesive materials available to the dental surgeon and discuss how they may be applied when restoring the dentition of patients with moderate to severe tooth wear. The following areas are discussed:

- Cervical tooth wear
- Anterior tooth wear
  - Palatal tooth wear
  - Incisal/palatal tooth wear
  - Labial/incisal/palatal tooth wear
- Posterior and generalised tooth wear

13.4.1 Cervical Tooth Wear

Cervical tooth wear lesions are common and present in a variety of forms depending on the type and severity of the causative factors. Not all lesions require restoration, but if aesthetics, sensitivity, or prevention of further tooth wear dictates then some form of adhesive restoration will usually be most suitable.

A plethora of tooth-coloured restorative materials are now available. Materials can either be composite resin or glass ionomer-based, or a combination of both; either in a layered technique with the individual materials or with formulated resin-modified glass-ionomer cements. The choice of materials can be bewildering, with new materials and techniques seemingly introduced to the market on a monthly basis [38, 43–45].

There are a number of approaches to bonding restorations to cervical tooth tissue. For lesions with margins that are still confined to enamel, the use of a microfine or polishable densified composite resin, in conjunction with acid-etched enamel, will usually produce good aesthetic and durable results. Unfortunately, most

cervical lesion margins are not confined to enamel and usually involve root cementum and dentin. In this situation, some form of dentin bonding agent is required, in combination with a composite resin or a self-adhesive composite resin formulation. Alternatively, a glass-ionomer cement restoration with inherent bonding properties to both dentin and enamel may be considered.

In situations where aesthetics are paramount, then a polishable composite resin combined with some form of adhesive bonding agent is often the material of choice. However, despite ongoing improvements, questions remain as to the longer term durability of dentin bonding agents, which may result in micro-leakage and characteristic marginal discolouration of the restoration [46].

Where lesions are not as visually prominent and involve more of the root surface, often partly below the gingival margin, then a glass-ionomer material may prove to be more durable. The dynamic bond of glass-ionomer cements to both dentin and enamel through an ionic exchange provides the opportunity for continual repair of the adhesive bond at the tooth and cement interface. There is also the possible additional benefit of fluoride ion release from the glass-ionomer cement reducing the possibility of marginal caries in susceptible individuals [47].

Although much improved over recent years, the colour properties of conventional glass-ionomer cements are not ideal. However, in deeper cervical lesions, it is possible to consider a layered technique combining the adhesive properties of the glass-ionomer cement with the superior colour properties of a polishable composite resin. This can be carried out in one visit or preferably following a minimum set time of 24 h; the superficial portion of the glass-ionomer cement restoration can be reduced and a layer of composite resin added (Fig. 13.12). The new generation of light-activated resin-modified glass-ionomer materials attempt to combine some of the better properties of composite resin and conventional glass-ionomer cements. Certainly the command set, improved colour and easier finishing of some of these materials allow the opportunity to provide very acceptable conservative restorations for cervical tooth wear lesions [48, 49].

Only longer term observation and assessment will determine how durable the variety of materials and techniques available for non-carious cervical lesions will ultimately prove to be [50].

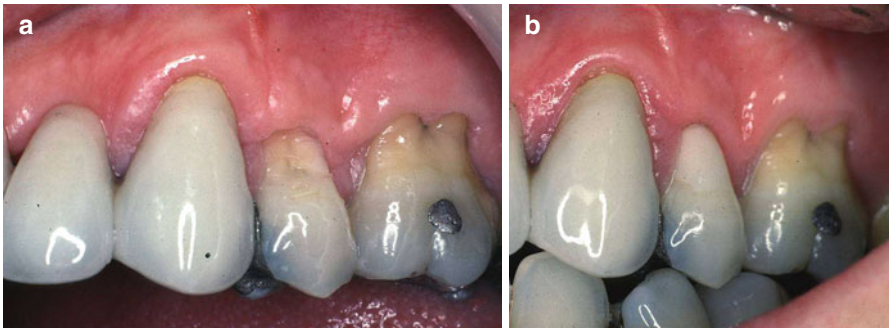


Fig. 13.12 (**a**, **b**) Restoration of a cervical abrasion/erosion lesion using a layered glass-ionomer and direct composite resin technique. (**a**) Before restoration. (**b**) After restoration

13.4.2 Anterior Tooth Wear

Although tooth wear can generally affect the whole dentition, it is often localized to the anterior teeth and the maxillary anterior teeth in particular.

13.4.2.1 Palatal Tooth Wear

This pattern of tooth wear is usually characteristic of acid erosion, possibly combined with a degree of attrition, and is the type more commonly seen in the younger age groups. Often, the labial and incisal surfaces are relatively intact and the main indications for restorative treatment are to offer some resistance to further palatal tooth wear which will reduce the risk of significant enamel fractures to the weakened incisal edges and pulp tissue inflammation. The use of resin-bonded palatal metal alloy veneers is an acceptable method to manage this form of tooth wear [51, 52] and has been shown to be a relatively durable technique [39, 53]. Either heat-treated gold alloys or nickel-chromium alloys, as used in resin-bonded bridge frameworks, are currently the cast metal alloys of choice. The decision as to which of the two materials to use is based on the improved bond strength of resin to nickel-chromium alloys versus the easier working properties and wear characteristics of the gold alloys.

Tooth preparation is minimal, usually restricted to smoothing the incisal and palatal peripheral enamel margins. Laboratory fabrication of the metal alloy veneers is either directly on a refractory working cast or by a wax/resin 'lift-off' technique. When restoring worn anterior teeth, creation of inter-occlusal space is usually required in order to accommodate the thickness of the restoring material. As the tooth structure is already compromised, avoiding further tooth reduction to create space is obviously advantageous. Orthodontic tooth movement using an anterior 'Dahl' appliance has been described as a method of achieving inter-occlusal space. Although this is a predictable method, there are some disadvantages, not least the increased treatment time and extra laboratory procedures. An alternative approach, based on similar principles to the Dahl appliance, is to deliberately design and construct the palatal veneers in such a manner that they are cemented initially high in occlusion. Expected tooth movement is enhanced if a positive cingulum contact can be achieved with the occluding lower incisor teeth in an attempt to direct orthodontic forces along the long axis of the contacting teeth. This method would appear to contradict traditional occlusal teaching but to date has proved to work well in these particular circumstances where tooth preparation is minimal [54].

Luting cements are usually resin-based and used in combination with the manufacturer's dentin bonding agent where appropriate. The use of opaque resin-based cement will overcome any potential greying of the incisal third caused by the underlying palatal metal veneer. Rubber dam isolation is used when necessary and occasionally gingival retraction cord in situations where there has been excessive tooth wear in the cervical region. By including metal coverage of the palatal veneers onto the incisal edges, location during cementation is made somewhat easier and this design will also offer some increased resistance to shearing loads. An example of the use of resin-bonded metal alloy palatal veneers is illustrated in Fig. 13.13

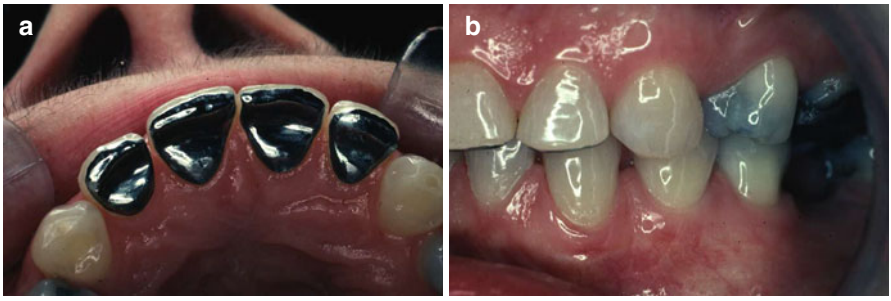


Fig. 13.13 (**a**, **b**) The use of nickel/chromium alloy resin-bonded palatal veneers used to restore localised palatal tooth wear for maxillary incisor teeth. (**a**) Palatal view of veneers. (**b**) Labial view demonstrating re-establishment of posterior occlusal contacts

13.4.2.2 Incisal/Palatal Tooth Wear

Although the use of metal alloy palatal veneers is an excellent conservative method of managing localized anterior tooth wear, it is not possible to improve the appearance of lost incisal and labial tooth tissue. In these circumstances, it is feasible to build up the incisal portion of the tooth with direct acid-etch retained composite resin and then construct a resin-bonded metal alloy palatal veneer to cover both the palatal tooth tissue and composite resin [55, 56]. However, potential difficulties arise with this technique, in that it is difficult to know where to finish the composite resin build-up palatally and the adhesive bond of the metal alloy palatal veneer will be somewhat compromised because of the reduction in available tooth enamel.

An alternative and very conservative approach is to restore both the incisal and palatal tooth surfaces with direct acid-etch retained composite resin at an increase in occlusal vertical dimension to accommodate the thickness of the restorative material [57–59]. This particular technique is relatively straightforward and avoids the need for extensive laboratory support; although the longer term durability can be unpredictable, particularly when restoring lower anterior teeth. However, the technique provides the opportunity for undemanding repair or replacement and allows the possibility of more involved and complex procedures in the form of conventional crowns to be considered at a later date once inter-occlusal space has been gained. Median survival figures of 5–7 years have been reported with biological complications being rare thereby preserving the original presenting tooth structure for further restoration [60]. An example of the technique is shown in Fig. 13.14. This particular clinical approach is rapidly becoming the first treatment of choice when restoring the worn dentition. A stepwise demonstration of the clinical technique is further illustrated in Fig. 13.15.

Alternatives to using direct composite resins are available in the form of indirect densified composite resins, with the potential advantages being the improved physical properties and better control regarding occlusal and interproximal contouring [61]. Modified porcelain laminate veneer restorations of both the incisal and palatal worn tissue have also been described by a number of clinicians [62, 63]. There are, however, potential difficulties with both these techniques in that it is often very difficult to disguise the junction between the incisal porcelain or indirect composite

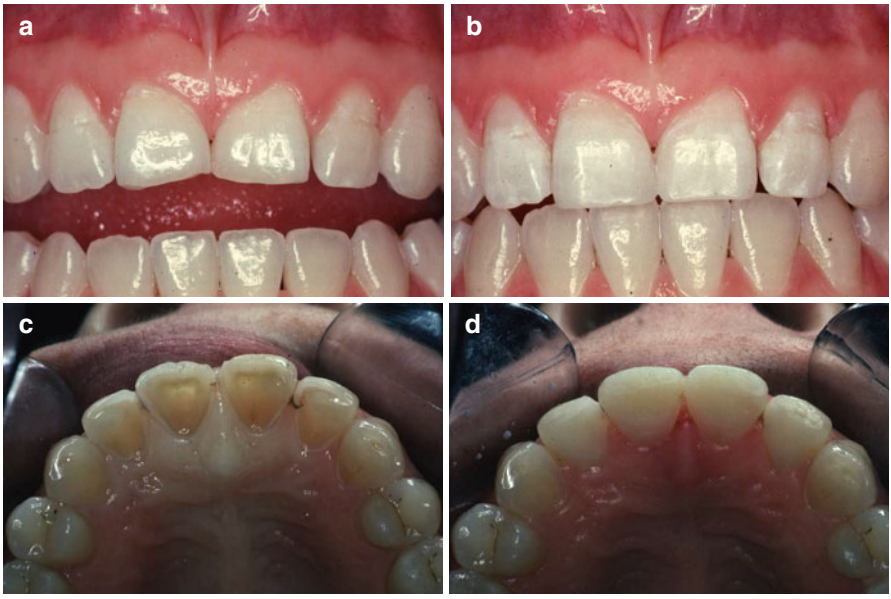


Fig. 13.14 (**a**–**d**) Restoration of incisal and palatal aspects of worn upper anterior teeth with direct composite resin restorations. (**a**) Labial view before and (**b**) after restoration. (**c**) Palatal view before and (**d**) after restoration

resin with the remaining tooth structure on the labial aspect of the tooth. When using porcelain, it is also necessary to create greater inter-occlusal space to provide sufficient bulk of material to reduce the risk of material fracture. Theoretically, both these indirect techniques should be more durable than using direct composite resin, although to date there is very little scientific clinical evidence to confirm this assumption [64]. Figure 13.16 illustrates the use of modified porcelain laminate and gold alloy veneers to restore worn upper anterior teeth.

13.4.2.3 Labial/Incisal/Palatal Tooth Wear

A number of adhesive approaches have been recommended for the restoration of teeth with lost tooth structure on all three major surfaces. These have included the use of a labial porcelain laminate veneer in conjunction with a metal alloy veneer for the palatal surface [65, 66], a resin-bonded minimal ceramic crown [67] or an adhesive metal-ceramic crown restoration [68]. All these techniques are relatively complex and would normally require some inter-occlusal space creation prior to completion of the restoration. In these circumstances, consideration should be given to the provision of a more conventional full coverage crown. In many situations, however, it is possible to restore all tooth surfaces with direct composite resin at an increased occlusal vertical dimension in an attempt to initially recreate lost inter-occlusal space [69, 70]. This method is often more acceptable to the patient compared with the use of a metal-based 'Dahl' appliance, and has the additional advantage of not committing the patient to conventional crown restorations. Once completed, however, a decision can then be taken either to continue with ongoing

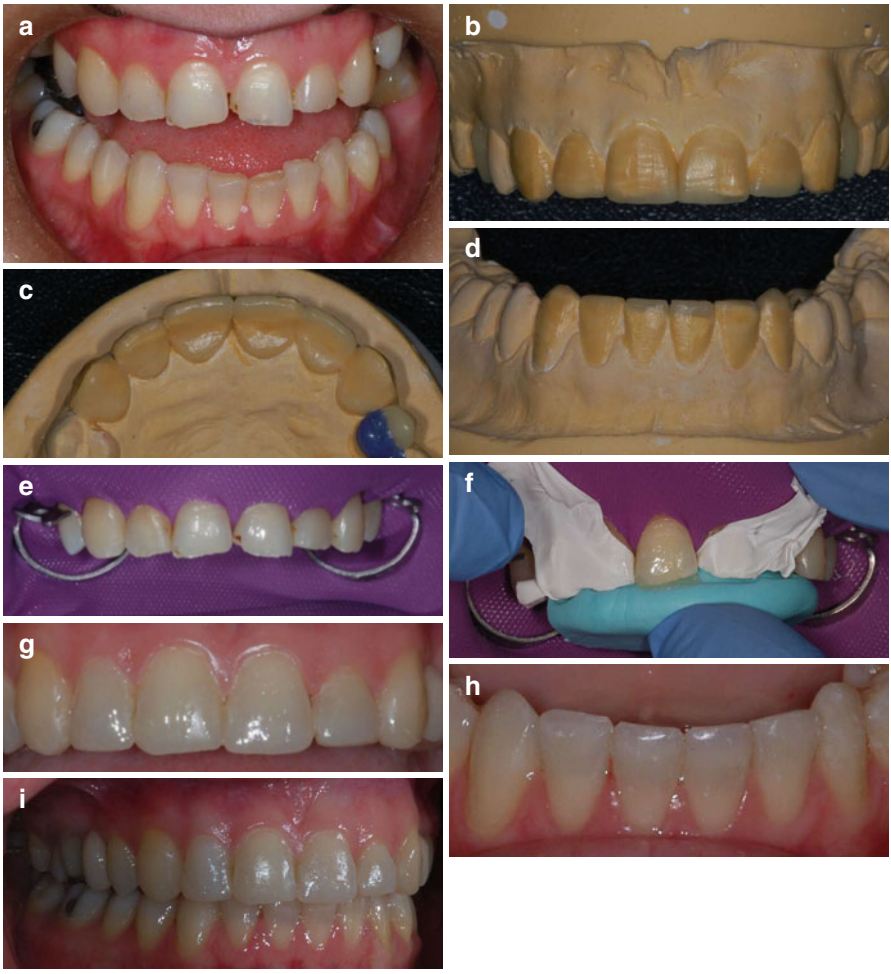


Fig. 13.15 (**a**–**i**) Restoration of worn upper and lower anterior teeth with direct composite resin restorations. (**a**) Before restoration. (**b**, **c**, **d**) Diagnostic wax-up on stone casts of planned restorations. (**e**) Rubber dam isolation of teeth prior to adhesive restorations. (**f**) Use of silicone putty index and interproximal tape to aid restoration. (**g**) Post restoration upper anterior teeth. (**h**) Post restoration lower anterior teeth. (**i**) Restorations 6 months following placement demonstrating re-establishment of posterior occlusal contacts (Illustrations courtesy of Dr Tanya Cerajewska)

maintenance of the composite resin restorations or alternatively to consider proceeding to conventional crowns conforming to the newly established occlusion. This conservative approach is advantageous for patients with particularly compromised dentitions where more involved restorative options are not always appropriate due to patient compliance and cost (Figs. 13.17, 13.18 and 13.19). Repair and/or replacement of aging composite resin restorations is relatively straightforward and continues to avoid any significant tooth preparation compared with that of conventional crown restorations (Fig. 13.20).

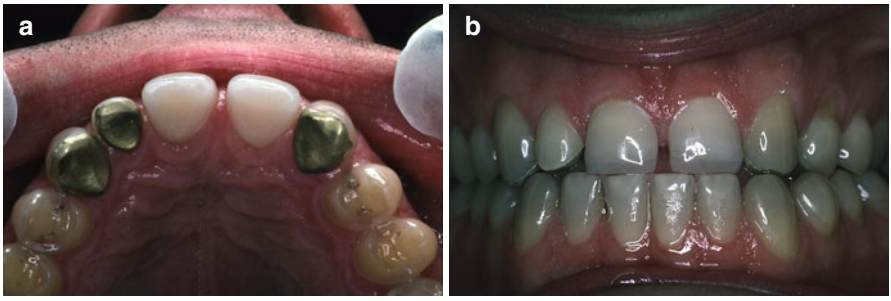


Fig. 13.16 (**a**, **b**) Resin-bonded porcelain laminate veneers used to restore the incisal and palatal aspects of maxillary central incisor teeth, with resin-bonded gold alloy palatal veneers used for the remaining worn anterior teeth. (**a**) Labial view after treatment. (**b**) Palatal view after treatment

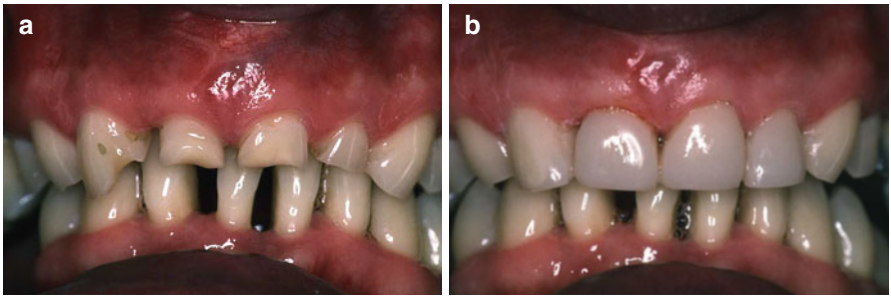


Fig. 13.17 (**a**, **b**) Direct acid-etch retained composite resin restorations used to restore the labial, incisal, and palatal aspects of compromised and worn maxillary incisor teeth at an initial increase in occlusal vertical dimension. (**a**) Before restoration. (**b**) After restoration

In some situations where remaining tooth structure is minimal, for example advanced wear of anterior mandibular teeth, it may be advantageous to consider a degree of localised crown lengthening surgery in an attempt to capture all remaining tooth enamel (Fig. 13.21). If for any reason surgical crown lengthening is not available, then occasionally there may be value in considering indirect splinted composite resin restorations to aid retention and durability. It is essential however to carefully monitor for any 'quiet' debond and ensure the patient can manage effective plaque control in the interproximal regions (Fig. 13.22). The presence of a 'gingival enamel ring' is often considered key to maximising the restoration bond and reducing early failure particularly in the parafunctional patient [59].

13.4.3 Posterior and Generalised Tooth Wear

Tooth wear affecting posterior teeth in isolation is rare and is usually part of a generalized condition affecting the whole dentition. Occasionally the pattern of tooth wear is such that individual posterior teeth may require restoring, and it is possible in these situations to consider some of the adhesive materials and

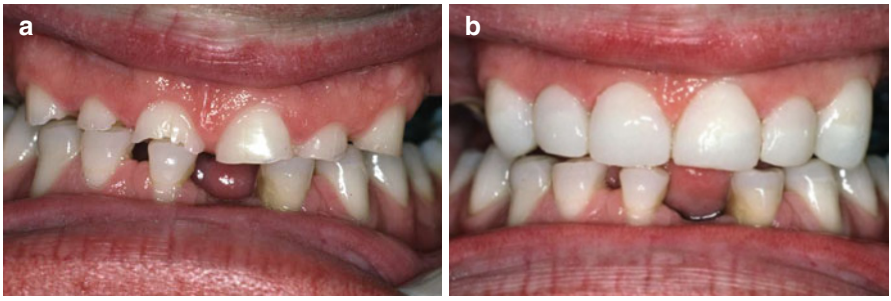


Fig. 13.18 (**a**, **b**) Direct acid-etch retained composite resin restorations used to restore extensively worn maxillary anterior teeth at an increase in occlusal vertical dimension. (**a**) Before restoration. (**b**) After restoration

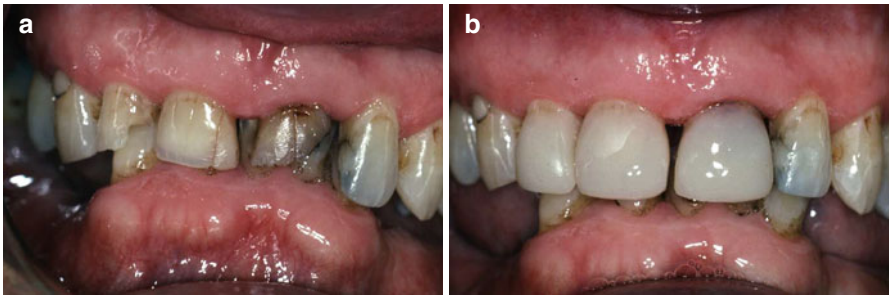


Fig. 13.19 (**a**, **b**) Direct acid-etch retained composite resin restorations used to restore compromised and worn maxillary incisor teeth at an increase in occlusal vertical dimension accepting the lack of ideal posterior occlusal support. (**a**) Before restoration. (**b**) After restoration

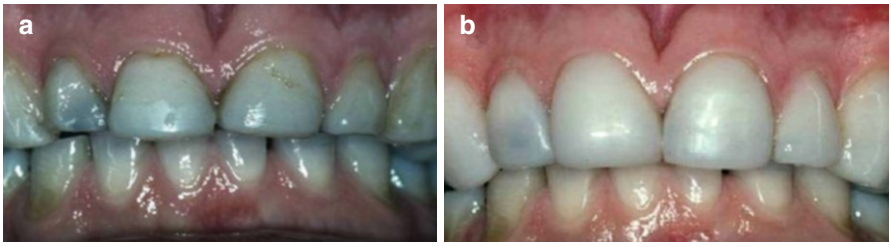


Fig. 13.20 (**a**, **b**) Previously treated worn upper anterior teeth with direct acid-etch retained composite restorations maintained and repaired with a new application of direct composite resin restorations at 8 years. (**a**) Before re-restoration. (**b**) After new restorations

techniques described earlier. If aesthetics is not paramount, then the use of a resin-bonded heat-treated gold alloy restoration can be advantageous [71]. Alternatively, if aesthetics dictate then a resin-bonded ceramic or indirect composite resin onlay can be considered. These techniques are helpful where retention and resistance

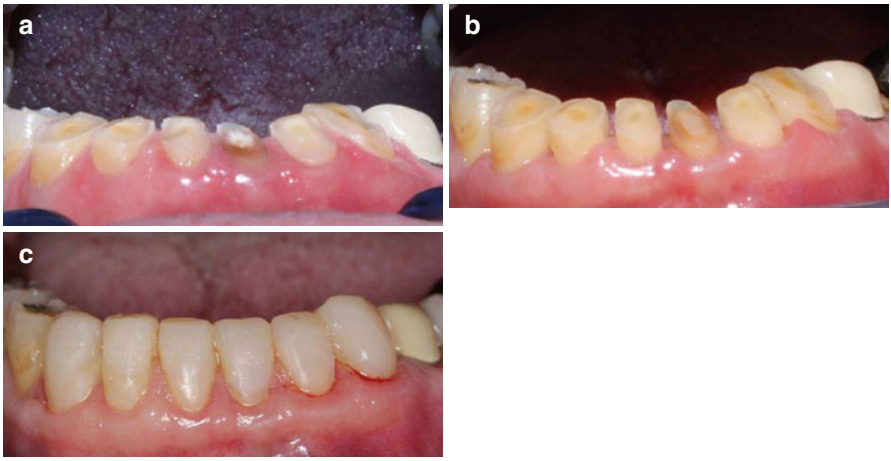


Fig. 13.21 (**a**–**c**) Direct acid-etch retained composite resin restorations used to restore severely worn lower anterior teeth following periodontal surgical crown lengthening to expose available tooth structure. (**a**) Before surgery. (**b**) After periodontal surgical crown lengthening. (**c**) Immediately following placement of restorations(Illustrations courtesy of Dr James Ban)

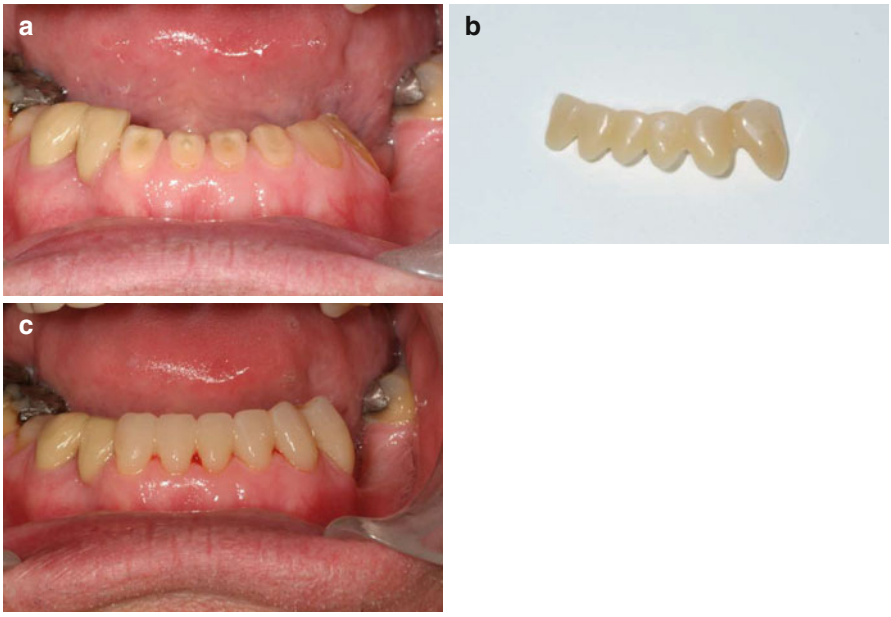


Fig. 13.22 (**a**–**c**) Indirect linked acid-etch retained composite resin restorations used to restore severely worn lower anterior teeth. (**a**) Before restoration. (**b**) Laboratory constructed linked composite resin restoration. (**c**) Immediately following placement of restorations

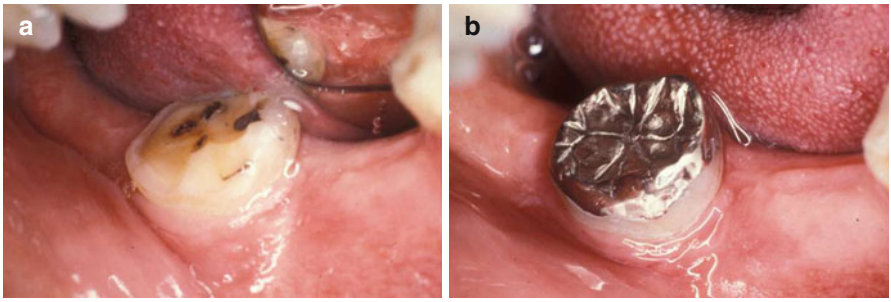


Fig. 13.23 (**a**, **b**) Minimal preparation resin-bonded gold alloy onlay used to restore worn posterior tooth. (**a**) Before restoration. (**b**) After restoration

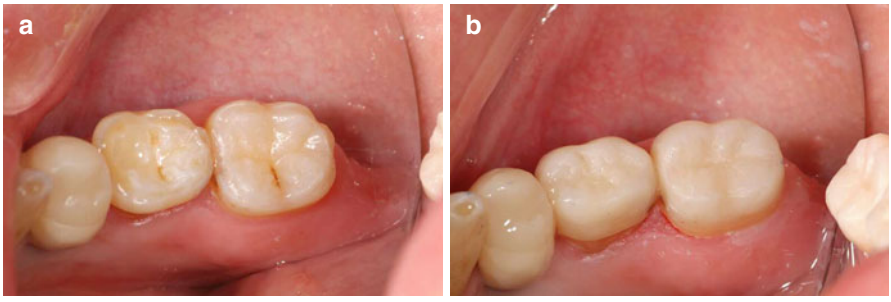


Fig. 13.24 (**a**, **b**) Minimal preparation resin-bonded indirect composite resin onlay restorations used to restore worn posterior teeth. (**a**) Before restoration. (**b**) After restoration

form for conventional crowns are particularly compromised, and there is a desire to avoid adjunctive treatments such as periodontal surgical crown lengthening (Figs. 13.23 and 13.24).

In situations of generalized tooth wear where there are indications to consider a full mouth reconstruction of the dentition, then the use of adhesive onlay restorations in the posterior quadrants can be of value in certain circumstances [72]. Restoring posterior quadrants with adhesive onlays is a conservative method, although on occasions it is not always possible to create sufficient inter-occlusal space by opening the vertical dimension alone, particularly if opposing occluding surfaces in the molar regions need to be restored. In these circumstances, some occlusal tooth reduction may also be necessary. Where space is at a premium, the selection of a gold alloy as opposed to porcelain will be advantageous. Because of the normal arc of mandibular closure, there will often be more space available in the premolar regions, allowing the opportunity to use more aesthetic tooth-coloured restorations. The space created by restoring the posterior quadrants, at an overall increase in occlusal vertical dimension, will provide the opportunity to successfully restore the worn anterior teeth either by conventional or adhesive methods (Fig. 13.25).

In selected cases, it is possible to consider a full mouth reconstruction of the worn dentition using resin-bonded ceramic or indirect composite resin restorations.

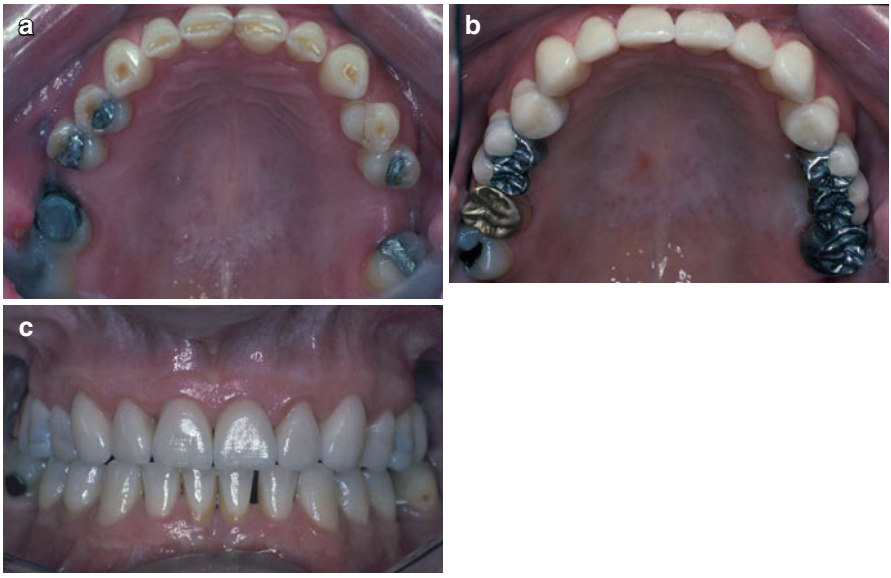


Fig. 13.25 (**a**–**c**) Reconstruction of a worn maxillary dentition using modified resin-bonded laminate porcelain veneers for the anterior teeth, and resin-bonded bridges and onlays for the posterior teeth at an overall increase in occlusal vertical dimension. (**a**) Occlusal view before restoration. (**b**) Occlusal view after restoration. (**c**) Labial view after restoration

However, the longer term durability, particularly of the posterior onlay restorations, still remains unpredictable and characteristically small fracture lines can appear in time which may eventually result in a catastrophic failure [73]. This is more likely to be the outcome for patients who exhibit parafunctional clenching/grinding habits (Fig. 13.26). The use of a full coverage occlusal splint to be worn chiefly at night may offer some protection to vulnerable restorations.

An occasional alternative to laboratory-manufactured restorations for full mouth rehabilitation is to consider a technique [74, 75] using direct acid-etch retained composite resin materials at an increase in occlusal vertical dimension. This will of course involve greater clinic time and the need ideally to restore multiple teeth in one session in order to control the increase in occlusal vertical dimension. The longer term durability is likely to be inferior to indirect materials and techniques, but if appropriate, would have the advantage of being easier to repair and maintain on a piecemeal basis.

Ongoing development of clinical techniques and materials nevertheless has dramatically advanced treatment options by means of minimal preparation adhesive restorations for the rehabilitation of eroded and worn dentitions. The three-step technique described by Vailati and Belser [76, 77] is a striking example of what can be achieved with careful planning, material selection and expert clinical execution.

New techniques bring with them new difficulties and challenges, and the use of adhesive onlay restorations in managing the worn dentition is no exception to this

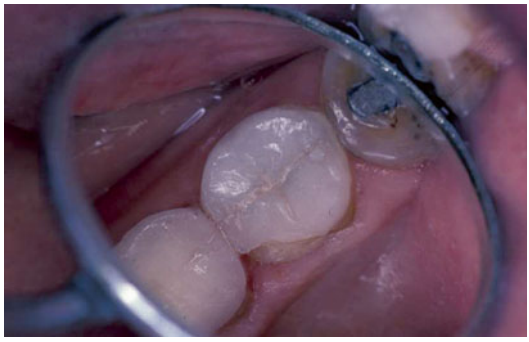


Fig. 13.26 Failure through fracture of minimal preparation resin-bonded indirect ceramic onlay restoration used to restore worn posterior tooth

fact. Temporization following tooth preparation can be problematic. Procedures involving complete resin bonding of the temporary restoration to the underlying tooth tissue may compromise the subsequent adhesive bond for the final restoration. There is also a risk of damage to the tooth preparation during the removal of the interim resin lute. Conversely, avoiding this approach by using a less adhesive material or technique may result in the early loss of any temporary restorations, with the possible consequences of unplanned tooth movement. Although not ideal, laboratory-manufactured acrylic or composite resin quadrant splinted temporary restorations, cemented with a composite resin lute to spot-etched enamel, have proved to be reasonably reliable in these circumstances.

Checking the occlusal relationship at the try-in stage can also be a challenge due to the relative lack of retention of the restorations before cementation. It is therefore critical that accurate jaw records are secured and transferred to the working casts on at least a semi-adjustable articulator so that any occlusal form manufactured in the laboratory will be close to the clinical situation. Time and attention to detail at this stage will always be productive, and will usually reduce the need for any major adjustment to the restorations following cementation. Removal of all the residual composite resin lute following the final cementation of the restorations can also be demanding and time-consuming, particularly in the proximal regions.

Despite some of these present limitations, as ceramic and composite resin technology improves and durable, less abrasive, castable materials become available, it may become more realistic to consider resin-bonded ceramic and composite resin restorations as routine when managing patients with posterior and generalized tooth wear.

13.5 Conclusions

A wide range of treatment options exist when considering the restoration of the worn dentition, ranging from the more conventional fixed and removable prosthodontic approach to some of the newer, less invasive and minimal preparation adhesive techniques now available to the dental surgeon. While a number of the minimal preparation adhesive methods would appear to offer significant advantages over more traditional measures, only time and careful clinical evaluation will dictate whether these become the accepted routine treatment patterns of tomorrow.

References

1. Steele JG, Walls AW, Ayatollahi SM, Murray JJ. Major clinical findings from a dental survey of elderly people in three different English communities. Br Dent J. 1996;180:17–23.
2. Nunn J, Shaw L, Smith A. Tooth wear – dental erosion. Br Dent J. 1996;180:349–52.
3. Nunn J, Gordon P, Morris A, Pine C, Walker A. Dental erosion – changing prevalence? a review of British national childrens' surveys. Int J Paediatr Dent. 2003;13:98–105.
4. Van't Spijker A, Kreulen C, Bartlett D. Prevalence of toothwear in adults. Int J Prosthodont. 2009;22:35–42.
5. Lussi A, Hellwig G, Zero D, Jaeggi T. Erosive tooth wear: diagnosis, risk factors and prevalence. Am J Dent. 2006;19:319–25.
6. Mehta S, Banerji S, Millar B, Suarez-Feito J. Current concepts on the management of tooth wear: part 1. Assessment, treatment planning and strategies for the prevention and the passive management of tooth wear. Br Dent J. 2012;212:17–27.
7. Williams DR. A rationale for the management of advanced tooth wear (ATW). J Oral Rehabil. 1987;14:77–89.
8. Smith BG, Bartlett DW, Robb ND. The prevalence, etiology and management of tooth wear in the United Kingdom. J Prosthet Dent. 1997;78:367–72.
9. Schwartz NL, Whitsett LD, Berry TG, Stewart JL. Unserviceable crowns and fixed partial dentures: lifespan and causes for loss of serviceability. J Am Dent Assoc. 1970;81:139–401.
10. Walton IN, Gardner FM, Agar JR. A survey of crown and fixed partial denture failures, Length of service and reasons for replacement. J Prosthet Dent. 1986;56:416–21.
11. Tan K, Pjetursson BE, Lang NP. A systematic review of the survival and complication rates of fixed partial dentures (FPDs) after an observation period of at least 5 years. Clin Oral Implants Res. 2004;15:654–66.
12. Fyffe HE. Provision of crowns in Scotland – a ten year longitudinal study. Community Dent Health. 1992;9:159–64.
13. Edlehoff D, Sorenssen J. Tooth structure removal associated with various preparations designs for anterior teeth. J Prosthet Dent. 2002;87:503–9.
14. Eckerbom M, Magnusson T, Martinsson T. Reasons for and incidence of tooth mortality in a Swedish population. Endod Dent Traumatol. 1992;8:230–4.
15. Jackson CR, Skidmore AE, Rice RT. Pulpal evaluation of teeth restored with fixed prostheses. J Prosthet Dent. 1992;67:323–5.
16. Berry DC, Poole DFG. Attrition: possible mechanisms of compensation. J Oral Rehabil. 1976;3:201–6.
17. Ricketts NJ, Smith BGN. Minor axial tooth movement in preparation for fixed prostheses. Eur J Prosthodont Restor Dent. 1993;1:145–9.
18. Briggs P, Bishop K. Fixed restorations in the treatment of tooth wear. Eur J Prosthodont Restor Dent. 1997;5:175–80.
19. Talbot T, Briggs P, Gibson M. Crown lengthening: a clinical review. Dent Update. 1993;20:301–6.
20. Ricketts NJ, Smith BGN. Clinical techniques for producing and monitoring minor axial tooth movement. Eur J Prosthodont Restor Dent. 1994;2:5–9.
21. Dahl BL, Krogstad O. The effect of a partial bite raising splint on the occlusal face height. An X-ray cephalometric study in human adults. Acta Odontol Scand. 1982;40:17–24.
22. Dahl BL, Krogstad O, Karlsen K. An alternative treatment in cases with advanced attrition. J Oral Rehabil. 1975;2:209–14.
23. Ibbetson RJ, Setchell DJ. Treatment of the worn dentition 1. Dent Update. 1989;16:247–53.
24. Ibbetson RJ, Setchell DJ. Treatment of the worn dentition 2. Dent Update. 1989;16:300–7.
25. Brown KE. Reconstruction considerations for severe dental attrition. J Prosthet Dent. 1980;44:384–8.
26. Licht WS, Leveton EE. Overdentures for treatment of severe attrition. J Prosthet Dent. 1980;43:497–500.
27. Graser GN. Removable partial overdentures for special patients. Dent Clin North Am. 1990;34:741–57.

28. Basker RM, et al. Overdentures in general dental practice. 3rd ed. London: British Dental Association Press; 1993.
29. Watson RM. The role of removable prostheses and implants in the restoration of the worn dentition. Eur J Prosthodont Restor Dent. 1997;5:181–6.
30. Toolson LB, Taylor TD. A 10-year report of a longitudinal recall of overdenture patients. J Prosthet Dent. 1989;62:179–81.
31. Ettinger RL, Jakobsen J. Denture treatment needs of an overdenture population. Int J Prosthodont. 1997;10:355–65.
32. Buonocore MG. A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. J Dent Res. 1955;34:849–53.
33. Bowen RL. Properties of a silica-reinforced polymer for dental restorations. J Am Dent Assoc. 1963;66:57–64.
34. McLean JW, Wilson AD. The clinical development of the glass-ionomer cements. Formulations and properties. Aust Dent J. 1977;22:31–6.
35. Calamia JR. Etched porcelain veneers: the current state of the art. Quintessence Int. 1985;16:12–5.
36. Banks RG. Conservative posterior ceramic restorations: a literature review. J Prosthet Dent. 1990;63:619–26.
37. Duke ES. Adhesion and its application with restorative materials. Dent Clin North Am. 1993;37:329–39.
38. Willems G, Lambrechts P, Braem M, Vanherle G. Composite resins in the 21st century. Quintessence Int. 1993;24:641–58.
39. Nohl FSA, King PA, Harley KE, Ibbetson RJ. Retrospective survey of resin-retained cast metal palatal veneers for the treatment of anterior palatal tooth wear. Quintessence Int. 1997;28:7–14.
40. King P. The use of adhesive restorations in the management of localised anterior tooth wear. Prim Dent Care. 1999;6:65–8.
41. Mehta S, Banerji S, Millar B, Suarez-Feito J. Current concepts on the management of tooth wear: part 2. Active restorative care 1: the management of localised tooth wear. Br Dent J. 2012;212:73–82.
42. Mehta S, Banerji S, Millar B, Suarez-Feito J. Current concepts on the management of tooth wear: part 4. An overview of the restorative techniques and dental materials commonly applied for the management of tooth wear. Br Dent J. 2012;212:169–77.
43. Watson TF, Bartlett DW. Adhesive systems: composites, dentine bonding agents and glass ionomers. Br Dent J. 1994;176:227–31.
44. Van Meerbeek B, De Munck J, Yoshida Y, Inoue S, Vargas M, Vijay P, et al. Buonocore memorial lecture: adhesion to enamel and dentin: current status and future challenges. Oper Dent. 2003;28:215–35.
45. Chee B, Rickman L, Satterthwaite JD. Adhesives for the restoration of non-carious cervical lesions: a systematic review. J Dent. 2012;40:443–52.
46. Van Dijken JW, Sunnegardh-Gronberg K, Lindberg A. Clinical long-term retention of etch-and-rinse and self-etch adhesive systems in non-carious cervical lesions. A 13 years evaluation. Dent Mater. 2007;23:1101–7.
47. Matis BA, Cochran M, Carlson T. Longevity of glass-ionomer restorative materials: results of a 10-year evaluation. Quintessence Int. 1996;27:373–82.
48. Gladys S, Van Meerbeek B, Lambrechts P, Vanherle G. Marginal adaptation and retention of a glass-ionomer, resin-modified glass-ionomers and a polyacid-modified resin composite in cervical Class-V lesions. Dent Mater. 1998;14:294–306.
49. Sidhu SK. Clinical evaluations of resin-modified glass-ionomer restorations. Dent Mater. 2010;26:7–12.
50. Peumans M, De Munck J, Mine A, Van Meerbeek B. Clinical effectiveness of contemporary adhesives for the restoration of non-carious cervical lesions. A systematic review. Dent Mater. 2014;30:1089–103.
51. Darbar UR. Treatment of palatal erosive wear by using oxidised gold veneers: a case report. Quintessence Int. 1994;25:195–7.

52. Hussey DL, Irwin CR, Kime DL. Treatment of anterior tooth wear with gold palatal veneers. Br Dent J. 1994;176:422–5.
53. Chana H, Kelleher M, Briggs P, Hooper R. Clinical evaluation of resin-bonded gold alloy veneers. J Prosthet Dent. 2000;83:294–300.
54. Poyser N, Porter R, Briggs P, Chana H, Kelleher M. The Dahl concept: past, present and future. Br Dent J. 2005;198:669–76.
55. Foreman PC. Resin-bonded acid-etched onlays in two cases of gross attrition. Dent Update. 1988;15:150–3.
56. Creugers HC, Kayser AF. The use of adhesive metal partial crowns to restore attrition defects: a case report. Quintessence Int. 1992;23:245–8.
57. Darbar UR, Hemmings KW. Treatment of localised anterior tooth wear with composite restorations at an increased occlusal vertical dimension. Dent Update. 1997;24:72–5.
58. Mizrahi B. A technique for simple and aesthetic treatment of anterior tooth wear. Dent Update. 2004;31:109–14.
59. Poyser N, Porter R, Briggs P, Kelleher M. Demolition experts: management of the parafunctional patient: 2. Restorative management strategies. Dent Update. 2007;34:262–8.
60. Gulamali AB, Hemmings KW, Tredwin CJ, Petrie A. Survival analysis of composite Dahl restorations provided to manage localised anterior tooth wear (ten year follow-up). Br Dent J. 2011;211(4), E9.
61. Briggs P, Bishop K, Kelleher M. The use of indirect composite for the management of extensive erosion. Eur J Prosthodont Restor Dent. 1994;3:51–4.
62. McLundie AC. Localised palatal tooth surface loss and its treatment with porcelain laminates. Restorative Dent. 1991;7:43–4.
63. Milosevic A. The use of porcelain veneers to restore palatal tooth loss. Restorative Dent. 1990;6:15–8.
64. Smales R, Berekally T. Long term survival of direct and indirect restorations placed for the treatment of advanced tooth wear. Eur J Prosthodont Restor Dent. 2007;15:2–6.
65. Cheung SP, Dimmer A. Management of the worn dentition: a further use for the resin-bonded cast metal restoration. Restorative Dent. 1988;4:76–8.
66. Bishop K, Bell M, Briggs P, Kelleher M. Restoration of a worn dentition using a double-veneer technique. Br Dent J. 1996;180:26–9.
67. Holmes R, Dan Sneed W. Treatment of severe chemomechanical erosion using castable ceramic restorations and a new dentin/enamel bonding system: a case report. Quintessence Int. 1990;21:863–7.
68. Bishop K, Priestley D, Deans R, Joshi R. The use of adhesive metal-ceramic restorations as an alternative to conventional crown and bridge materials. Br Dent J. 1997;182:101–16.
69. Magne P, Magne M, Bleser U. Adhesive restorations, centric relation and the Dahl principle: minimally invasive approaches to localized anterior tooth erosion. Eur J Esthet Dent. 2007;2:260–73.
70. Nixon P, Gahan M, Chan F. Techniques for restoring worn anterior teeth with direct composite resin. Dent Update. 2008;35:551–8.
71. Crawford PJM, Aboush YEY. The use of adhesively retained gold onlays in the management of dental erosion in a child: a 4-year case report. Br Dent J. 1993;175:414–6.
72. Rawlinson A, Winstanley RB. The management of severe dental erosion using posterior occlusal porcelain veneers and an anterior overdenture. Restorative Dent. 1988;4:10–6.
73. Bartlett D, Sundaram G. An up to 3-year randomised clinical study comparing indirect and direct resin composite used to restore worn posterior teeth. Int J Prosthodont. 2006;19:613–7.
74. Roessler DM. Amalgam and composite resin as an alternative to crown and bridgework in the rehabilitation of an extensively debilitated dentition. Case report. Aust Dent J. 1990;36:1–4.
75. Schmidlin P, Filli T, Imfeld C, Tepper S, Attin T. Three-year evaluation of posterior vertical bite reconstruction using direct resin composite – a case series. Oper Dent. 2009;34:102–8.
76. Vailati F, Belser UC. Full-mouth adhesive rehabilitation of a severely eroded dentition: the three-step technique. Part 1. Eur J Esthet Dent. 2008;3:30–44.
77. Vailati F, Belser UC. Full-mouth adhesive rehabilitation of a severely eroded dentition: the three-step technique. Part 2. Eur J Esthet Dent. 2008;3:128–46.

Noncarious Cervical Lesions: Prevalence, Etiology, and Management

14

Karen B. Troendle and Kevin M. Gureckis

Abstract

In this chapter, current theoretical information and evidence-based clinical management strategies for noncarious cervical lesions are presented. Topics addressed include the prevalence, etiology, clinical presentation, and noninvasive and invasive management of lesions created by the loss of hard tissue from the cervical areas of teeth through processes unrelated to caries lesion formation. Treatment modalities outlined range from noninvasive to invasive and include dentin desensitization and/or periodontal management as well as restorative therapy using different restorative materials.

14.1 Prevalence of Noncarious Lesions

"Noncarious cervical lesions" (NCCLs) are lesions that involve the loss of hard tissue from the cervical areas of teeth through processes unrelated to the dissolution of tooth structure caused by bacterial activity associated with dental caries. Commonly, their shape is like a wedge with the apex pointing inward, but they can appear in various shapes. These lesions can range from shallow depressions that involve enamel only to deep wedge-like lesions that extend far enough into the tooth that they can compromise tooth vitality (Fig. 14.1).

Although evidence of occlusal attrition and abrasion exists among the dentition of hunter-gather populations from thousands of years ago, the prevalence of noncarious cervical lesions has not been observed in remains of these populations. Noncarious cervical lesions should therefore be viewed as pathology unique to modern man [1].

K.B. Troendle, DDS, MPH (✉) • K.M. Gureckis, DMD
Department of Comprehensive Dentistry, University of Texas Health Science Center,
7703 Floyd Curl Drive, San Antonio, TX 78229, USA
e-mail: troendle@uthscsa.edu; gureckis@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_14

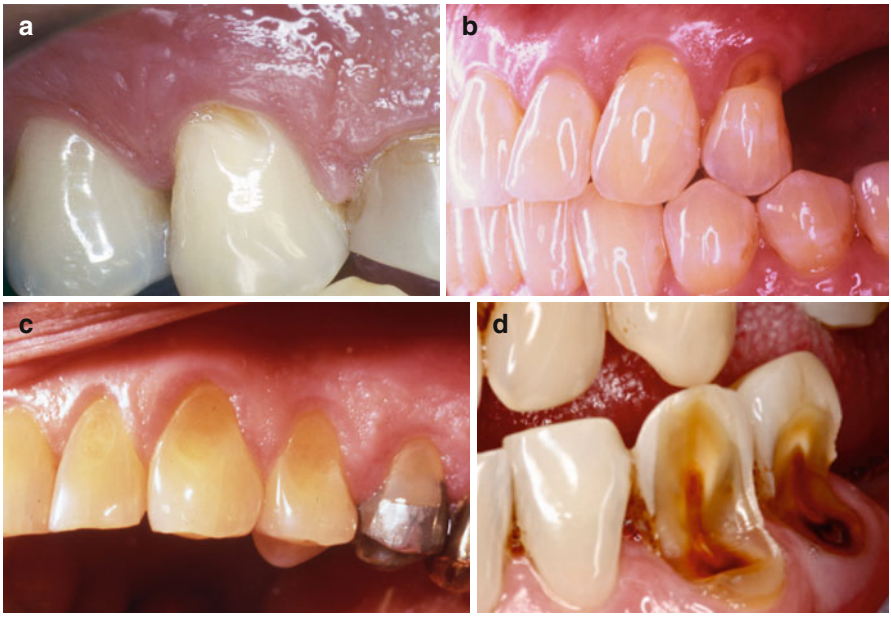


Fig. 14.1 Noncarious cervical lesions can range from shallow depressions that involve enamel only (**a**), enamel and radicular tooth structure (**b, c**), or radicular tooth structure only. They vary in shape based on the combination of etiologic factors present. Lesions can progress to a size that can compromise tooth vitality (**d**)

Today, these lesions can be found in both children and adults. Prevalence studies have resulted in conflicting results. Shulman and Robinson recorded prevalence as low as 2 % [2], whereas Bergstrom and Eliasson [3] recorded findings of 90 %. In a review of 15 studies carried out between 1941 and 1991, Levitch et al. [4] reported the prevalence data for these lesions ranging between 5 and 85 %. The wide range can be explained in several ways. Some studies had small sample sizes and used very different age groups, and different studies classified lesions in different ways. When you add in the number of variables to consider within a population or within individuals of a population over time, it is not surprising that the prevalence data is so confusing.

Just as the prevalence of NCCLs is unclear, so is the exact picture of their intraoral distribution. Rees et al. [5] and Sognnaes et al. [6] reported that the lesions are most prevalent on the labial surface of maxillary incisors; whereas, Radentz et al. [7] reported maxillary first molars to be the most commonly affected teeth, and Zipkin and McLure [8] found the maxillary first premolars to be the most involved tooth. This variability in reported distribution patterns is possibly due to the confusing terminologies and variable diagnoses that were employed in different studies at different times. However, despite all of the flaws and contradictions of the early prevalence studies, some facts clearly emerge: the older the study population was, the greater the numbers of lesions per individual were found, and the larger the lesions were. Also, these lesions are almost exclusively found on the facial or buccal aspects of teeth, seldom on the lingual and rarely on the proximal surfaces.

14.2 Etiology of Noncarious Lesions

A number of theories regarding the etiological mechanism of tooth wear in the absence of caries have developed over time; however, the entire etiology of noncarious cervical lesions is still controversial. In 1907, Miller [9–11] conducted an exhaustive investigation of noncarious tooth surface lesions and was the first to associate these lesions, which he termed "wastings" with mechanical and chemical factors. NCCLs were thus first classified according to their supposed origin: abrasion or erosion. Abrasion was generally ascribed to either the toothbrush and or dentifrice pathologically wearing away tooth structure by mechanical or frictional forces; however, other factors that exert a repetitive and sometimes excessive force on teeth such as toothpicks, floss, removable appliances, and parafunctional habits can also contribute to cervical tooth wear (Fig. 14.2). Erosion has been defined as loss of tooth structure produced by chemical dissolution from acids other than those produced by bacteria [12]. In the strictest definition of the word, erosion is not a chemical mechanism but rather a physical mechanism by which wear is caused by friction from the movement of liquids. Therefore, a paradigm shift has been suggested to supplant the term "erosion" with the more accurate term "biocorrosion" defined as the chemical, biochemical, or electrochemical action that causes the molecular degradation of tooth substance [13]. Biocorrosion as a term takes into consideration more than just chemical exogenous and biochemical endogenous acids, it also encompasses proteolytic agents and the piezoelectric effects observed on dentin.

Another relatively new term to the discussion of NCCLs is that of "abfraction" introduced by Grippo in 1991 [14], and amended in 2004 [15]. Abfraction describes the microfracture of tooth substance in areas of stress concentration. The term abfraction has been abused and misused by the dental community as a generic designation for all NCCLs, whereas the etiology of these lesions is generally believed to be multifactorial. In order to appropriately address the causative factors surrounding noncarious cervical lesions in an individual, the clinician must appreciate the unique interplay among those factors for that individual. The three major potential mechanisms in lesion development consist of abrasion/friction, abfraction/stress, and biocorrosion. Failure to appropriately prevent and treat noncarious cervical

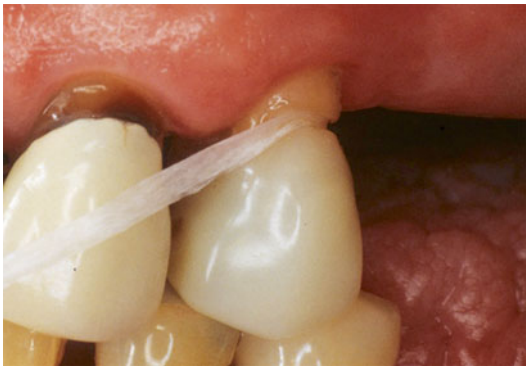


Fig. 14.2 Noncarious cervical lesions can result from aggressive or improperly implemented oral hygiene techniques. In this instance, the repetitive sawing action of the floss wore a notch in the root surface of the tooth (Photo courtesy of Dr. Stephan J Haney)

lesions can result in their progression, the potential for tooth sensitivity, and, in severe cases, endodontic therapy or even tooth loss.

It is critical for a clinician to perform a thorough medical and dental history and to consider the complex interaction of the contributory factors and modifying factors before completing the diagnosis and initiating treatment. The following section will discuss these factors in more depth.

14.2.1 Biocorrosion

Biocorrosion of teeth can occur as a result of exogenous chemical and endogenous biochemical acids, as well as by proteolytic enzymes, and piezoelectric effects on dentin. The fact that tooth enamel and dentin can be dissolved by acid is a well-established fact, regardless of whether the acid is produced as a by-product of oral bacteria as is in the case of dental caries, ingested in the form of acid containing food or beverage, or intrinsically produced as in gastric reflux or vomiting or purging. Enamel is mainly composed of inorganic hydroxyapatite, with only a small percentage of organic matrix material, and, thus, it is readily disintegrated by acid. A pH below 5.5 has been shown to dissolve enamel [16]. Dentin contains less hydroxyapatite than enamel and more organic matrix material. Thus, the surface layer of the dentin hydroxyapatite can be demineralized by acid leaving the organic matrix, which is not water soluble but which is subject to attack by proteolytic enzymes (proteases) that can be produced by the microorganisms in plaque or come from gingival crevicular fluid. One of the reasons that gastroesophageal reflux disease or habitual regurgitation is so devastating to teeth is the combined exposure to the extremely acidic gastric juice and the proteolytic enzymes from the stomach (pepsin) and pancreas (trypsin).

It is interesting to note, when considering the effect that acids have on demineralizing tooth structure, that pH does not tell the whole story. Different acids have different corrosive potentials. Therefore, the pH of a substance alone is not totally predictive of its potential to cause biocorrosion. For acidic drinks, it was found that not only the pH value but also the type of acid, the amount of titratable acid (buffer capacity), and possibly chelating properties are factors in determining the progression of biocorrosion [17]. In vitro studies show that citric acid and phosphoric acid both produce more tissue loss than maleic acid [18]. The citrate ion may be particularly destructive because of its binding or chelating action on calcium. Larsen and Nyvad found that the addition of calcium and phosphate to acidic drinks reduced their ability to dissolve enamel [19]. Lussi et al. demonstrated the same protective effect of calcium and phosphate using the example of yogurt, which has a pH of close to 4 but has no corrosive effects on tooth structure because of its high calcium and phosphate content [17]. Additionally, the oral cavity has several protective mechanisms against biocorrosion, the most prominent being saliva. Salivary flow is increased by acid-induced stimulation of the glands. Clearance of acids from the oral cavity is, to a large extent, dependent on the saliva flow rate and the saliva buffering capacity. Low saliva flow rate and poor buffering capacity allow prolonged retention of extrinsic and intrinsic acids in the mouth, which will accelerate the biocorrosive

process. Therefore, the quantity and the quality of a patient's saliva is an important modifying factor to the progression of NCCLs and should be a part of patient evaluation. The pellicle on the tooth's surface, which represents a diffusion barrier to the acids, also aids in preventing acids from directly dissolving the tooth mineral.

14.2.2 Abrasion

The loss of tooth structure in the cervical area of the tooth as a result of mechanical rubbing with some object is known as abrasion. This may be due to aggressive toothbrushing or flossing techniques, stiff brushes, the use of abrasive dentifrices, or even repetitive habits.

Normally, enamel is very resistant to wear, and abrasion is low especially as compared to dentin or cementum. However, the abrasion resistance of both enamel and dentin is weakened after exposure to acid. There is evidence that the loss of tooth structure from abrasion is accelerated when acid demineralization of enamel and dentin occurs prior to or during mechanical abrasion of those surfaces [20]. Fortunately, demineralized tooth structure can be remineralized if the weakened tooth structure is not removed as during brushing or other mechanical rubbing before it can be remineralized through exposure to saliva for a long enough period of time. In fact, when teeth are exposed to saliva, delaying brushing for as little as 1 h after an acid challenge can increase their resistance to abrasion [21]. The advice for patients to brush immediately after every meal needs to be re-evaluated and modified. Additionally, patients exhibiting NCCLs should be advised to use only a small amount of a minimally abrasive fluoride containing dentifrice with light force when brushing or skip the dentifrice all together and use a fluoride rinse instead.

Combining occlusal loading (as in abfraction) with abrasion does not seem to have the same additive effect resulting in tooth loss that combining abrasion with exposure to acid has. Litonjua et al. found that without any acids present, occlusal loading had no effect in creating NCCLs in recently extracted teeth subjected to toothpaste slurry and tooth brushing at the cervical margins. Brushing with the toothpaste slurry caused similar cervical wear patterns regardless of whether or not the tooth was loaded [22].

14.2.3 Abfraction

The theory that occlusal forces are an etiologic factor in the formation of NCCLs is a relatively new concept, and one that has definitely been the focus of much attention and controversy. The term "abfraction" which means "to break away" has been proposed for this phenomenon. Abfraction represents the mechanical flexure theory where tooth bending and flexing during function and parafunction create flexural stress in the cervical area of the tooth resulting in microfractures of the crystalline structure of the enamel and dentin in that area. The theory suggests that the lesion would continue to enlarge as the bending and flexing is repeated finally resulting in

chipping away of the hard tissue. Tensile stress resulting from oblique occlusal forces rather than compressive forces is considered to be the principal factor responsible for the disruption of the bonds between the hydroxyapatite crystals and the separation of the enamel from the dentin. While there is general agreement that occlusal loading can concentrate stress in cervical areas and that dentin and enamel have different tensile strengths, the confusion comes with the conflicting data from studies describing the association between occlusal wear and NCCLs. The degree of association varies from 15 % [23] to 95 % [24, 25]. Variables that may contribute to the disparity of results range from differences in the study design such as populations studied, exclusion criteria applied, and direction of the force being applied, to variations in the teeth being evaluated. Differences in support provided by the bony socket, gross morphology of the tooth, the presence or absence of restorations, and the microscopic structure of the tooth are all confounding variables that could influence the results of the study. In their review of the literature regarding noncarious cervical lesion, Pecie et al. [26] concluded that recent literature reveals an important number of clinical investigations showing a strong correlation between bruxism, parafunctions, and NCCLs. They went on to conclude that the literature supports a constant implication of occlusal stress, although rather in association with other factors than alone.

14.2.4 Stress Corrosion

In the engineering field, stress corrosion describes the concept that the presence of acidic substances in combination with stresses can cause more damage than either one alone [27]. Grippo and Masi [28] tested this theory as it applies to the formation of NCCLs. Their in vitro study, combining the application of citric acid and tensile stress to teeth, found that the addition of tensile stresses increased enamel loss by 20 %. Palamara et al. [29] also found that enamel dissolution increased significantly when teeth were subjected to cyclic tensile loads while immersed in 1 % lactic acid (pH 4.5). So, unlike combining occlusal loading with abrasion, which had no additive effect, loading teeth in an acidic environment did make teeth more susceptible to cervical tissue loss. In fact, Whitehead et al. [30] found that axial loading of extracted premolar teeth in the presence of an acidic solution resulted in lesions similar on a macroscopic and microscopic level to the NCCLs found in vivo. This phenomenon may in part explain the lack of NCCLs in anthropological samples that demonstrated evidence of heavy wear. Modern diets expose teeth to numerous acid challenges, particularly acidic fluids. This frequent acidic exposure may allow the impact of occlusal forces on cervical tooth structure to fully manifest.

14.2.5 Piezoelectric Effects

Piezoelectricity may be defined as “the acquisition of a surface electrical charge on opposing faces when under load.” Enamel does not possess any piezoelectric effects but dentin displays some piezoelectric properties because of its collagen content.

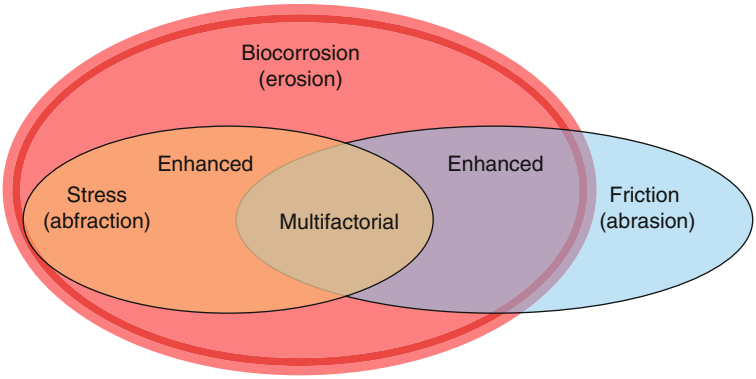


Fig. 14.3 Three conditions that are considered predisposing factors to NCCL formation are biocorrosion (erosion), abrasion, and occlusal stresses. The contribution to NCCL formation of both abrasion and occlusal stresses is enhanced in the presence of an acidic environment; whereas, when occlusal stresses and abrasion are combined outside of an acidic environment, there does not seem to be the same additive effect. And while biocorrosion or abrasion may at times seem to be the major single contributing factor in some cases of NCCLS, occlusal stresses when implicated are usually thought of as being in association with other factors rather than alone. The etiology of NCCLs is multifactorial [42]

Grippo and Masi [28] have reported that a patient with a severe bruxing habit was able to generate a surface charge of 0.4 V, which they suggest is sufficient to cause enamel demineralization [28]. Cyclic changes in surface charge could also attract and repel charged erosive agents, such as simple organic acids, and contribute to cervical tooth loss by that mechanism. Unlike some of the other possible etiologic factors contributing to noncarious lesions, the piezoelectric effect has not been examined in any great detail.

In summary, while older studies would point out an absence of conclusive evidence to support one etiology exclusively over another, more recent studies recognize the multifactorial nature of the etiology of noncarious cervical lesions. An important consideration when evaluating the etiological mechanism of NCCL formation is the enhanced effect of contributing factors when they are combined. As an example, abrasion following a recent exposure of the teeth to an acidic challenge is greater than it would have been prior to the tooth's exposure to acid, and the combined action of occlusal stresses and an acid environment is more harmful than either factor acting alone (Fig. 14.3).

14.3 Clinical Presentation of Noncarious Cervical Lesions

Noncarious cervical lesions may present on coronal tooth structure, a combination of coronal and radicular structure, or radicular structure only. Some authors have proposed that the morphological characteristics of noncarious cervical lesions are determined by their specific etiology [31].

Shallow defects on smooth surfaces coronal to the CEJ are considered to be a pathognomonic sign of biocorrosive tooth loss. U-shaped or disk-shaped broad shallow lesions with poorly defined margins and adjacent smooth enamel are consider to be erosive/biocorrosive lesions resulting from extrinsic acid sources such as those consumed through diet, medication, or recreational drug use. Extrinsic biocorrosive agents generally result in lesions on the facial surfaces of anterior teeth. On the other hand, intrinsic forms of biocorrosive lesions, caused by the reflux of gastric contents, are generally located on the lingual and incisal surfaces of maxillary anterior teeth.

Abrasive lesions generally exhibit sharply defined margins and a hard surface that may display traces of scratching.

Abfraction lesions are characterized as being irregular and may be wedge-shaped or saucer-shaped with sharp internal angles. However, the fact that the etiology of NCCLs is multifactorial makes differentiation between causative factors difficult. Acidity, abrasion, and tooth flexure each play a role to a greater or lesser degree from patient to patient and tooth to tooth, and so shape cannot be considered totally predictive of etiology.

14.4 Management of Noncarious Cervical Lesions

The decision on how or when to treat NCCLs varies widely among practitioners. In 2003, the Academy of Operative Dentistry published recommendations for the treatment of noncarious cervical lesions that should still serve as guidelines today [32]. The decision to restore these lesions depends upon the following factors:

1. Inability to eliminate or greatly reduce the rate of lesion progression through elimination of etiologic factors.
2. Lesion is esthetically unacceptable to the patient.
3. Significant sensitivity of exposed dentin to cold liquids, food, and air that cannot be handled more conservatively.
4. Depth of the lesion threatens the strength of the tooth and integrity of the coronal-radicular unit.

Once all of the contributing factors for noncarious cervical lesion formation have been identified, the first therapeutic measures to be considered should be directed at preventing new lesions and halting the progression of existing lesions. Some of the treatment options for NCCLs, depending upon their severity, include dentin desensitization, restoration, periodontal surgery, or some combination of the three.

14.4.1 Preventive Measures

If acid plays a significant role, sources should be identified and eliminated as much as possible. Dietary counseling should address such issues as the frequency of intake of acidic foods, as well as the buffering capacity of certain foods. When

drinking acidic beverages, the use of a straw followed shortly afterward by rinsing the mouth or drinking milk or eating cheese should be encouraged. Patients with conditions such as gastric reflux or bulimia should be counseled to seek medical attention to attempt to elicit and treat the underlying causes. If elimination of the acid source is not feasible, the damage caused by the acid should be mitigated as soon as possible through buffering. The use of antacid lozenges, alkaline mouth rinses, or sugar free gum to stimulate salivary flow all help buffer acidity. Enhancing resistance against acid attack through the use of fluoride toothpaste, rinses, gels, and varnishes should also be considered as a part of a preventive treatment plan.

Identifying potential sources of abrasion to teeth should be investigated. Patients should be educated not only on proper oral hygiene techniques and types of preventive aids, but also on how and when to consider brushing. When teeth have had time to remineralize after an acid challenge, the potential for mineral loss can be minimized.

As discussed earlier, abfraction as a primary factor in causing NCCLs continues to be challenged [33]. Though laboratory studies have evaluated the role of occlusal forces in the formation of NCCLs, and have duplicated the cervical area on the tooth where stress seems concentrated [34], there is no consensus on treatment strategies. A review of the literature by Wood et al. [35] in 2008 found that there was no evidence to support occlusal adjustment as a being helpful in terms of slowing down lesion formation or improving the retention of restorations when placed to restore NCCLs [35]. A systematic review of 286 articles on this topic, by Senna et al. in 2012, found that only 28 articles met the criteria for review, and that much of the literature that was published were review articles that were merely reiterating literature that was both quantitatively and qualitatively weak in terms of types of study design. With that in mind, they reported that in the available literature, a causal relationship between NCCLs and occlusion had not yet been demonstrated by prospective studies, and that cross-sectional studies only very lightly support that contention [36]. They encouraged researchers to plan further studies to seek a causal relationship, taking into consideration the necessity of eliminating bias.

Therefore, trying to stop the initiation of new NCCLs or the progression of existing lesions by adjusting the occlusal forces on the tooth is not currently supported by the literature. Clinicians that try to do so must be cognizant that inappropriate occlusal adjustments may increase the risk for other occlusal problems and that they should limit their adjustments to altering inclined cuspal inclines, reducing heavy contacts, and removing interferences as these help in reducing laterally directed stresses that are, in theory at least, the most damaging. Though controversial and not evidence based, the use of occlusal splints is another conservative option to try and mitigate the damage from those forces [37].

14.4.2 Dentin Desensitization

Dentin hypersensitivity has been referred to as one of the most painful and least successfully treated chronic dental conditions of teeth [38]. Exposed dentin in the cervical area may become hypersensitive, whether it is the result of loss of significant

tooth structure or not. Exposure to normal thermal, evaporative, tactile, osmotic, or chemical stimuli can all result in exaggerated painful responses for patients with dentin hypersensitivity. It is important to distinguish dentinal sensitivity pain, which is short in duration from pain of longer duration, which may be the result of pulpal inflammation. The most widely accepted explanation of dentinal sensitivity is the hydrodynamic theory [39]. The basis of this theory is that changes in direction of fluid movement within open dentinal tubules are perceived as pain by mechanoreceptors near the pulp. Under normal conditions, dentin is not exposed to the oral environment because it is covered either by cementum or enamel. In the case on NCCLs, that protective layer is removed and dentin becomes exposed. However, not all exposed dentin becomes sensitive. For pain to be experienced, the dentin tubules must be open at the surface. Studies have demonstrated that sensitive dentin contains as many as eight times as many open or patent tubules per unit area than nonsensitive dentin. Tubule diameters in sensitive teeth were also twice as wide in diameter [40].

Traditionally, the therapy for management of dentin hypersensitivity is primarily aimed at either occluding the dentinal tubules, creating coagulates inside the tubules to stop or minimize fluid movement within them, or by interfering with the transmission of pain signal at the synapse (typically with potassium nitrate). Diverse agents or formulations have shown various degrees of effectiveness in reducing the symptoms of dentin hypersensitivity, with some being applied professionally and others being applied as at-home treatments. A randomized clinical effectiveness study published in 2013 evaluated the comparative effectiveness of three treatments for hypersensitive NCCLs over 6 months. Potassium nitrate dentifrices, placement of a resin composite restoration, and placement of a sealant (DBA) were compared. The sealant and restoration proved equally effective in reducing hypersensitivity, while the dentifrice reduced hypersensitivity, but over time. The study recommended long-term studies of 3–5 years to determine the most effective treatment modality [41].

An in-depth discussion on dentin hypersensitivity can be found in Chap. 15 of this book.

14.4.3 Restoration of NCCLs

When desensitization of the tooth is not enough or there are esthetic or structural integrity issues that need to be considered for the tooth, a restoration may become necessary. Whether or not a restoration is truly necessary should be weighed heavily against the slowly progressing nature of the lesion and the high capacity of the patient's self defense mechanisms for sclerotic dentin formation since restoration failure could lead to a cycle of rerestoration that would require restoration replacement numerous times throughout the patient's life. This is especially important in light of the fact these restorations can be some of the least predictable or durable restorations that are placed (Fig. 14.4).

The challenges presented by material weaknesses as well as the lesions' cervical location and the inherent presence of variable bonding substrates, the restorative material's elastic modulus, polymerization shrinkage, and lack of resistance to wear

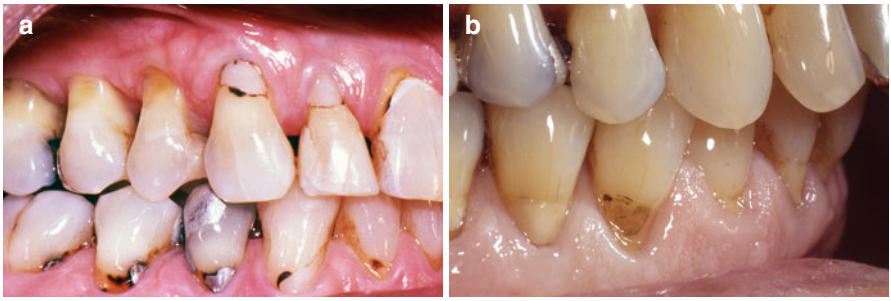


Fig. 14.4 NCCL restorations are prone to recurrent caries (**a**) and dislodgment (**b**)

and erosion can all compromise clinical longevity. Additional contributing factors to failure include the fact that cervical lesions are not prepared to include any macromechanical retention. In addition, they often present a bonding surface that is mostly in dentin, and often involving sclerotic dentin in an area of the tooth that is difficult to isolate. Therefore, restoration failure is likely due to the combined effect of insufficient material properties, the continued presence of the etiological factors that caused the lesion initially, and the specific biological environment in the cervical area [42].

14.4.3.1 Selection of Restorative Material

Materials available for the restoration of NCCLs fall into categories that are either macromechanically retained to the tooth, or bonded. Macromechanically retained materials include gold foil and amalgam. In the past, preparation of an NCCL for either gold foil or amalgam required that additional tooth structure be removed to ensure the retention and resistance form dictated by the limitations of those materials. And even though both materials were predictable and durable, they have fallen out of favor because they lack the esthetics currently possible with bonded tooth colored restorations.

Therefore, bonded tooth colored materials are most often selected when restoration of an NCCL is required. As a category, bonded restorations can be further subdivided by their mechanism of attachment to the tooth, either micromechanical and/or chemical.

Micromechanical retention for bonded restorations basically involves an exchange mechanism wherein minerals removed from the tooth's surface through etching are replaced by resin monomers that, once set, are micromechanically interlocked in the porosities and/or collagen fibrils exposed on the tooth surface. For resin composites, the resin adhesive provides the interface between the tooth and restorative resin, micromechanically locking into the tooth surface on one side and chemically reacting to the more viscous restorative resin composite on the other.

In order for resin bonding to work, three things need to happen: the tooth needs to be etched to create a retentive surface, the hydrophylic dentin needs to be modified or "primed" to accept the hydrophobic adhesive resin, and the adhesive resin needs to effectively fill the retentive areas and adequately cure. Over the years, different formulations of resin adhesives have been advanced to simplify the rather

technique-sensitive and time-consuming process of etching, priming, and applying the adhesive bonding agent. Products were initially classified by generation, but have since been reclassified by how many steps are involved and whether or not the etchant is rinsed away. In "total etch" or "etch and rinse" (ER) systems, the entire preparation is first etched with 30–40% phosphoric acid, which is then rinsed off. Timing and agitation of the etchant as well as the moisture level of the tooth after rinsing off the etchant were critical to success, and so efforts were made to try and minimize the potential for error. Adhesives were developed that eliminated the need to rinse and dry the tooth following etching. In the "self-etch" (SE) systems, the etching process is combined with additional steps eliminating some of the technique sensitivity associated with etch and rinse adhesives (Fig. 14.5).

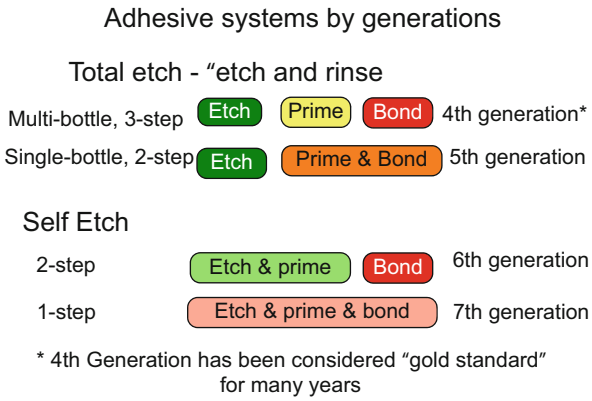


Fig. 14.5 Resin bonding systems all need to create a retentive surface, prime the hydrophilic dentin to accept a hydrophobic resin adhesive, and include a fluid resin that penetrates the micro-mechanical retentive areas created within the tooth's surface before curing. Various materials have been developed to accomplish these steps in different combinations

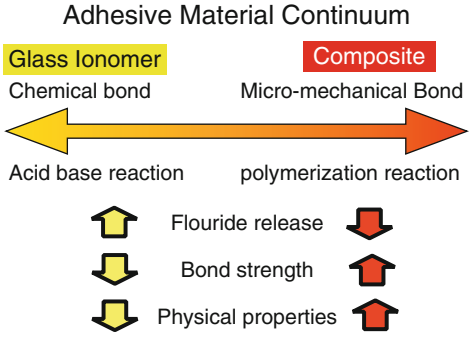


Fig. 14.6 Resin composite, which sets by polymerization and is micromechanically retained to the tooth, is at one end of the adhesive material continuum; glass ionomer, which is an acid-base reaction and is chemically as well as micromechanically retained to the tooth is at the other. Materials that blend some of the features of each include resin-modified glass ionomers, giomers, and compomers, and fall within a continuum between glass ionomer and resin composite. Depending upon where they lie within the continuum, their properties, etc., can be anticipated

A second category of bonded restorations includes those that chemically bond to tooth structure. Glass ionomers are still considered the only materials that are self-adhering to tooth tissue. A weak polyalkenoic acid pretreatment cleans the tooth surface and removes the smear layer and exposes collagen fibrils up to about 0.5–1 μm in depth. This allows the glass ionomer to establish a micromechanical bond. However, in addition, there is a chemical bond obtained by ionic interaction of the carboxyl groups of the polyalkenoic acid with the calcium of the tooth hydroxyapatite that remains attached to the collagen fibrils. So, at one end of the spectrum of bonded restorations there is resin composite, which sets by polymerization and is micromechanically retained to the tooth and on the other end there is glass ionomer, which is an acid base reaction and is chemically as well as micromechanically retained to the tooth. Each class of materials has advantages and disadvantages, and so manufacturers have come up with modified materials that blend some of the features of each. These materials include resin-modified glass ionomers, giomers, and compomers. These materials can be considered as falling within a continuum between glass ionomer and resin composite. Depending upon where they lie within the continuum, their properties, etc., can be anticipated (Fig. 14.6).

Peumans et al. [43] reviewed the literature from 1998 to 2004, describing clinical trials that tested the clinical effectiveness of adhesives in noncarious class-V lesions to determine the restoration retention rates of various adhesives. The adhesives were divided into five classes: three- and two-step etch and rinse (ER) adhesives, two-and one-step self-etch (SE) adhesives, and glass ionomers. Unfortunately, in the compilation of data, the glass-ionomer studies consisted mostly of resin-modified glass ionomers, and over half of the two-step self-etch adhesives tested were applied including additional selective etching of the enamel with phosphoric acid; making the data more difficult to interpret.

In evaluating the effectiveness of each adhesive class, general trends seem to emerge showing that glass ionomers for the most part demonstrate a more effective and durable bond followed by 3-step (ER) adhesive systems and the 2-step (SE) adhesive and with the 1-step SE showing the poorest results. However, the wide range of variability among the individual adhesives within each category shown in Table 14.1 illustrates that adhesive classification alone is not enough to make the best clinical choice of adhesive strategy for NCCL lesions. Additional systematic reviews of adhesive retention rates for NCCLs [42, 44] have provided additional evidence that clinical results vary significantly with the product employed within the adhesive classification.

Other material considerations in addition to retention include wear characteristics, elastic modulus, and esthetics. Since restoration failures are found more frequently in bruxers and areas with high stress concentrations, the theory has been proposed that a microfill or flowable resin composite may be preferable to a hybrid resin composite, if tooth flexure has been identified as an etiologic factor. The hypothesis being that an intermediate layer of flowable composite, which has a lower modulus of elasticity, could provide somewhat of an "elastic wall" that would absorb the stress created by polymerization shrinkage of the overlying layer of resin composite. It was also hypothesized that materials with a lower elastic modulus would be more likely to flex with the tooth when it is deformed under loading. However, this theory has not been proven clinically. In his review of materials for

Table 14.1 Average annual failure rates of adhesive systems

Adhesive system	Average annual % failure rate	Annual failure rate range within the category (%)
Glass ionomer	1.9 ± 1.8 %	0–7.6
3-step etch and rinse	4.8 ± 4.2 %	0–16
2-step etch and rinse	6.2 ± 5.5 %	0–19.5
2-step self-etch	4.7 ± 5.0 %	0–19.3
1-step self-etch	8.1 ± 11.3 %	0–48

M Peumans et al. in 2005 reviewed restoration-retention rates in NCCLs for different adhesives reported in peer-reviewed papers and abstracts from January 1998 to May 2004. Adhesives were categorized into one of five adhesive categories. The retention rates were depicted as a function of time in graphs for each of the categories and an annual failure rate percentage was calculated. The wide range of retention rates between products within a category demonstrates that product choice may be more important than adhesive classification

the restoration of NCCLs, Pecie et al. [42] generally found inconsistent and product-dependent results when composite materials were compared suggesting that presently, there is a lack of significant difference between flowable, microfilled, and hybrid composites. The review goes on to describe the advantages and disadvantages of alternative materials for use in NCCLs.

Compomers exhibit some fluoride release and have been shown to have retention rates comparable to composite resin. However, they are inferior in terms of other critical features such as mechanical properties, marginal integrity, color, and surface texture.

Conventional glass ionomers release fluoride over extended periods of time and have higher retention rates than certain resin adhesive systems; however, they have poor esthetics and low abrasion resistance, which limit their usefulness.

Giomers are hybrids of glass-ionomer and resin composite. They release fluoride and have surface characteristics that are better than glass-ionomer and resin-modified glass ionomer, and have esthetics close to resin composite. However, there are few clinical studies that have investigated marginal seal or long-term retention.

The choice of restorative material must be determined before any preparation is begun since the preparation must accommodate the material, in light of their physical properties. The most popular restorative material selections include resin composite, resin-modified glass-ionomer cement, or a combination of both using what is known as the “sandwich” technique.

The choice of using resin composite for restoring NCCLs is based on its reliability, conservation of structure, and excellent esthetics. The downside of resin composite, as with most bonded restorations, is that it requires meticulous isolation for proper dentin bonding.

The use of resin-modified glass-ionomer cement (RMGIC) is supported by its biocompatibility, adhesion to calcified substrates like sclerotic dentin, a modulus of elasticity comparable to dentin, and the ability for fluoride release and recharge [45]. The disadvantages include the difficulty in handling the restorative material due to its stickiness; solubility in an acidic environment; retention rates; strain-softening, which can weaken the bond; and an overall brittleness, which requires

that the tooth preparation have a marginal configuration similar to amalgam, since thin margins of RMGIC are weak [37].

The combination of a resin-modified glass ionomer and resin composite as either an open or close sandwich technique may be indicated for deep NCCLs, where the RMGIC is basically used as a dentin replacement, which is subsequently veneered with resin composite to replace missing enamel. This combination provides esthetics and abrasion resistance. Whether this is an open or closed sandwich technique depends on whether the RMGIC substructure is totally or partially veneered with resin composite.

From this discussion it is obvious that selecting a reliable and durable adhesive system and restorative material is not only very important, it is also very challenging. Regardless of which bonded restorative material is chosen, the design and surface treatment of the NCCL cavity preparation must be accomplished based upon the specific restorative material selected to maximize success. The technique employed for bonding, restorative material placement, and subsequent finishing and polishing must follow attention to detail. Also, the continued maintenance of these restorative materials must be ensured.

14.4.3.2 Isolation

When considering isolation of an NCCL lesion for restoration, there are challenges with moisture control from gingival fluid and blood contamination, as well as impaired access to subgingival margins. Isolation options include such techniques as the use of a rubber dam or the use of retraction cord alone or in combination with devices such as the *Isodry* dental isolation system, saliva ejectors, absorbent pads, etc.

The tissues around NCCLs that develop at the level of the gingival margin are found to display less inflammation than gingival tissue approximating active caries lesions. In these situations, retraction cord isolation technique is often a predictable choice. The use of retraction cord sizes #00 or #000 that have not been impregnated with a hemostatic agent can be used. The strands are cut just long enough to extend from the mesial proximal, through the facial sulcus to the distal proximal area. It can be gently tucked into the sulcus with an interproximal carver (IPC) or retraction cord packer. With increased sulcular depth, an additional cord can be placed to ensure the tissue is retracted away from the gingival margin of the preparation (Fig. 14.7).

When the lesion is subgingival to a point that retraction cord will not create adequate access to the gingival margin, the best isolation can be anticipated with the use of the rubber dam with a retracting clamp such as a #212SA. A number of modifications to routine rubber dam application need to be implemented when using a retracting clamp. The hole punched for the tooth being restored is placed slightly to the facial of the typical position so that the jaws of the clamp can push the tissue and rubber dam gingivally below the most gingival extent of the lesion more easily. Due to the force the clamp is required to exert on the tissue and rubber dam to displace it, the bows of the clamp must be stabilized with modeling compound, so that the clamp remains stable and does not dislodge or unnecessarily disrupt the cementum

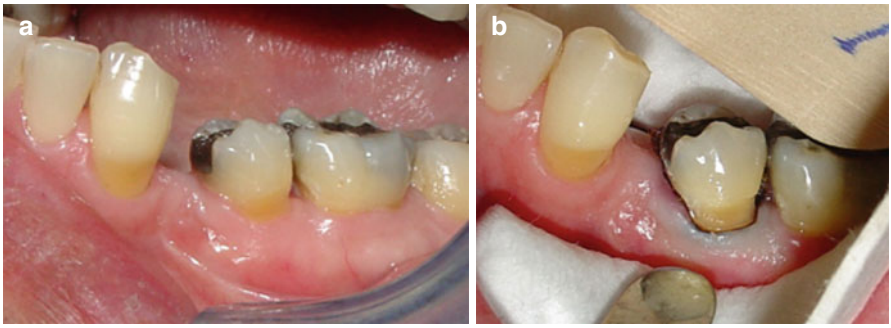


Fig. 14.7 The gingival margin of tooth 20 is at the level of the free gingival margin. (**a**) Retraction cord sizes #00 or #000 can be used to help expose the gingival preparation margin and control sulcular fluid. The strands are cut just long enough to extend from the mesial proximal, through the facial sulcus to the distal proximal area (**b**)

surface. Interestingly, a meta-analysis of clinical performance of cervical lesions reviewed 50 clinical studies and found that the type of isolation (rubber dam versus retraction cord) did not have a statistically significant influence on clinical success [46] Therefore, the specific isolation technique employed is less important than maintaining a well-controlled operative field.

If additional tissue release is needed for isolation and the lesion does not extend beyond the facial line angles of the tooth, a miniflap can be performed before rubber dam placement (Fig. 14.8). Two small vertical incisions are placed within the attached gingiva, and the small segment of tissue is gently reflected and stabilized gingivally with the #212SA clamp after the rubber dam is secured. After the procedure, the flap is repositioned and held in place with dampened gauze for a short period of time to allow for hemostasis. Sutures are not necessary. The soft tissue will heal uneventfully due to controlled reflection and retraction.

If greater access is required, a full thickness mucogingival flap can be reflected and stabilized under rubber dam isolation with a stabilized clamp at the site of the lesion (Fig. 14.9).

14.4.3.3 Tooth Preparation

With NCCLs there is little need for preparation when using resin composite, since the dentin bonding system will provide retention and the material does not require a specific marginal configuration. Beveling the enamel cavosurface margin for composite resin has been recommended to expose the ends of the enamel rods for a better etching pattern [47]. However, coronal enamel beveling has not been shown to increase clinical performance [46]. It can, however, help to esthetically blend the restoration with the adjacent enamel surfaces. The gingival margin that is on cementum or dentin is not beveled.

The placement of retentive features such as grooves in the preparation is controversial, because it may create what has been termed a “mud flap” phenomenon. Here the restoration is held in the preparation by the retention grooves while the

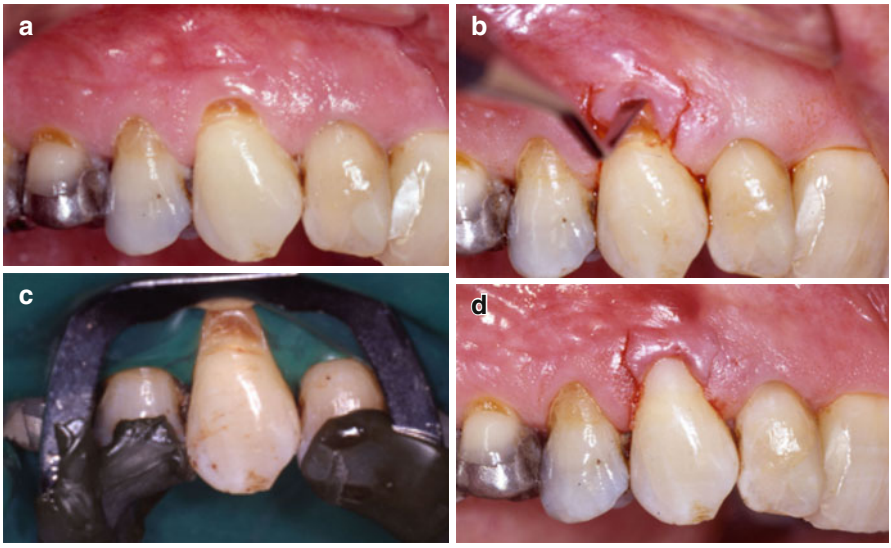


Fig. 14.8 The cervical lesion on tooth #6 (**a**) extends subgingival. A miniflap (**b**) is performed prior to rubber dam application and #212SA stabilization with modeling compound (**c**) for appropriate lesion access. (**d**) The final restoration with the miniflap replaced. There is no need for sutures

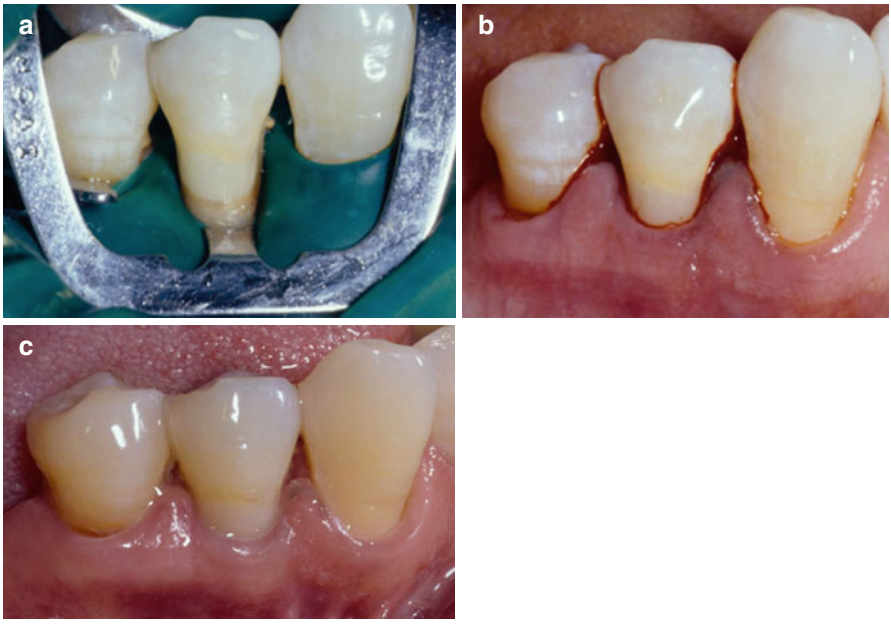


Fig. 14.9 Tooth #28 with radicular NCCL restored with full thickness flap reflection and RD isolation (**a**). Flap is re-positioned (**b**) and follow-up photo shows excellent adaptation and gingival health without clinical attachment loss (**c**)

bond and seal of the restoration to tooth may have failed. The chance for continued leakage and subsequent recurrent caries lesion formation can ensue, rather than a catastrophic total loss of the restoration, which, in essence, alerts the patient and clinician that the bond has failed.

When restoring an NCCL with a resin-modified glass ionomer, the preparation is somewhat like one for amalgam, but without retentive features. The preparation margins must be close to 90° since this material is very brittle and requires bulk for strength, so enamel beveling is contraindicated.

Regardless of the material chosen for bonding, the quality of the dentin available for bonding is very important (Fig. 14.10). With sound dentin, there is no benefit to roughening it with a bur when using etch and rinse or self-etch dentin bonding agents. With sclerotic dentin, there is a special bonding challenge due to hypermineralized surfaces. Tay and Pashley reported that hypermineralized surfaces can serve as a barrier to diffusion of bonding agents interfering with uniform hybridization. Though there is no predictable retention improvement when roughening or removing the outer dentin surface of sclerotic dentin with a diamond bur, resin bonds that extend to peripheral sound dentin can create bond strengths high enough for successful retention [48]. Improving the efficacy of bonding by increasing the etching time and number of applications of primer have produced equivocal results.

14.4.3.4 Insertion Techniques

The challenges with the insertion techniques for resin composites are partly due to the disparity between the bonding capacity of enamel and dentin margins, and polymerization shrinkage of the restorative material through configuration

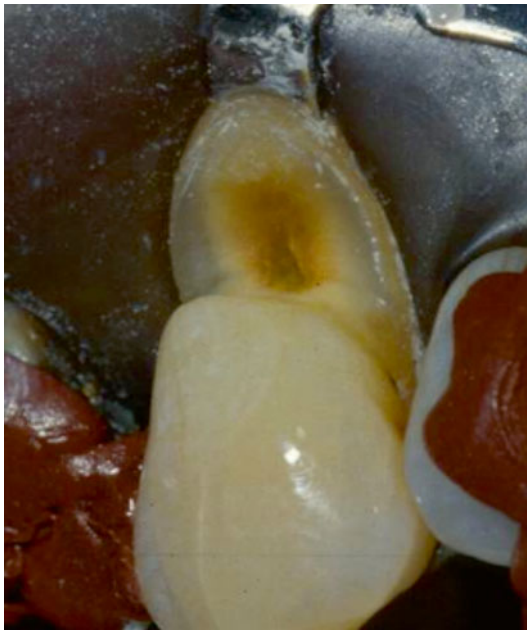


Fig. 14.10 This well-isolated NCCL demonstrates a variable dentin substrate with sound peripheral dentin, an intact coronal enamel margin, increased depth of the lesion, and deep margin locations that must be addressed

(C-factor) [49] effects. C-factor is the numerical ratio of bonded to unbonded or free surfaces. The more bonded surfaces a preparation has, the greater the C-factor, which means greater contraction stresses on the adhesive bond. So, a Class I occlusal restoration would generate much greater stress on its bonds than a Class 5 NCCL. However, to minimize the effect of polymerization shrinkage and to improve marginal adaptation, incremental placement of resin composite in no more than 2 mm is recommended for moderate to large preparations. There are options to the sequence of incremental placement. The first increment can be placed occlusally where the enamel margin is, to reduce the gingival margin gap formation with subsequent increments. Or the gingival margin increment can be placed first where there is no enamel to reduce the gingival margin gap when bonding to enamel. Incremental placement with the last one at the enamel margin can reduce the stresses on dentin margins.

If restoring the NCCL with a RMGIC, the bonding surfaces are treated with conditioning agent, which removes the smear layer. It is agitated for 10 s if it is a 20 % concentration, or 20 s if it is a 10% solution. The "rule of 200" is a simple application guideline to use, where the concentration of the conditioning agent multiplied by the number of application seconds should equal 200. After rinsing and gently drying with air (without desiccating), the RMGIC, if in a capsule, is activated and triturated. Encapsulated RMGIC has been shown to provide improved physical properties over hand-mixed equivalents [50]. A clear cervical matrix can be used to keep the material, which tends to pull away from the preparation surface, compressed within the preparation margins during light curing.

If there are concerns about the esthetics, abrasion resistance, and size of the lesion when using a RMGIC, the "sandwich technique" is another restorative option. The RMGIC is placed as a dentin substitute and light cured first, followed by a thin resin composite overlay as the enamel substitute. When the sandwich technique is employed, the tooth must be appropriately prepared for each material. In other words, first the tooth is conditioned; the RMGIC is placed and cured. It is then cut back with a bur to allow room for the resin composite overlay. Then the adhesive bonding system is applied according to manufacturer's directions to the remaining exposed tooth structure and exposed RMGIC. Finally, the resin composite overlay is placed, sculpted, and cured (Fig. 14.11).

Sculpting prior to polymerization can reduce stresses from finishing and polishing techniques by reducing the amount that has to be removed for proper contours. The finishing techniques can involve the use of a 12 or 12-B blade for gingival trimming. It is advisable to avoid rotary instrumentation at the gingival margin so the cementum and dentin margins are not inadvertently abraded. Fine diamond and multifluted carbide burs can trim axial and proximal contours. Sequential aluminum oxide polishing discs can provide a smooth surface. Finally, a low viscosity surface sealant should be applied to fill any contraction gaps formed around the periphery of the restoration and seal any of the microscopic defects created by finishing and polishing on the surface of the restoration (Fig. 14.12).

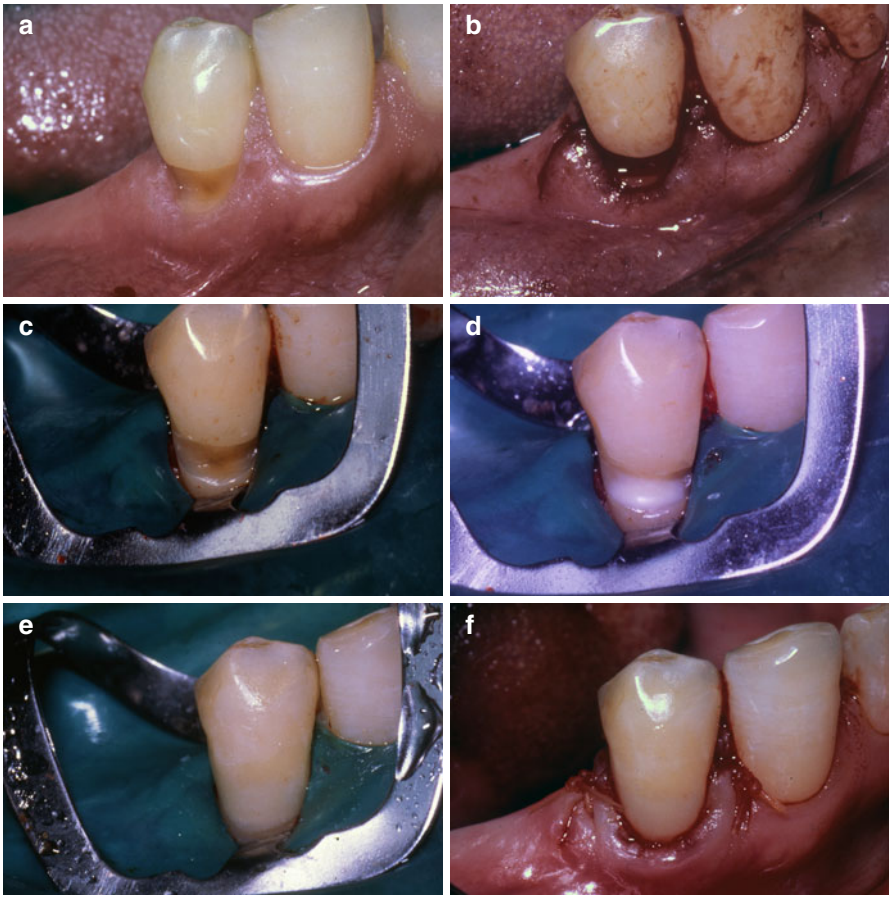


Fig. 14.11 The NCCL on 28 extended subgingival (**a**). Due to the extent of the lesion onto the distal surface of the tooth, a full thickness flap was reflected (**b**) and the margins of the lesion were exposed with a 212SA retracting clamp and rubber dam. (**c**) A closed sandwich technique was used to restore the tooth. RMGIC was applied as a first layer (**d**), which was then prepared for and covered by a veneer of resin composite. The flap was repositioned and sutures placed

14.4.4 Combined Periodontal and Restorative Intervention

There are times when the clinician must not only select the most appropriate restorative material, but must also decide whether treatment should be confined to a restorative procedure, or should be a combination periodontal-restorative procedure for maximum success. Because gingival recession is often associated with NCCLs, soft tissue root coverage can improve esthetics from gingival recession and cover the radicular portion of the NCCL to decrease root sensitivity and prevent further recession by improving the amount of attached gingiva [51]. Sometimes soft tissue root coverage can be combined with a restorative procedure when it is necessary to recreate the anatomic position of the CEJ, which may have been lost. The loss or

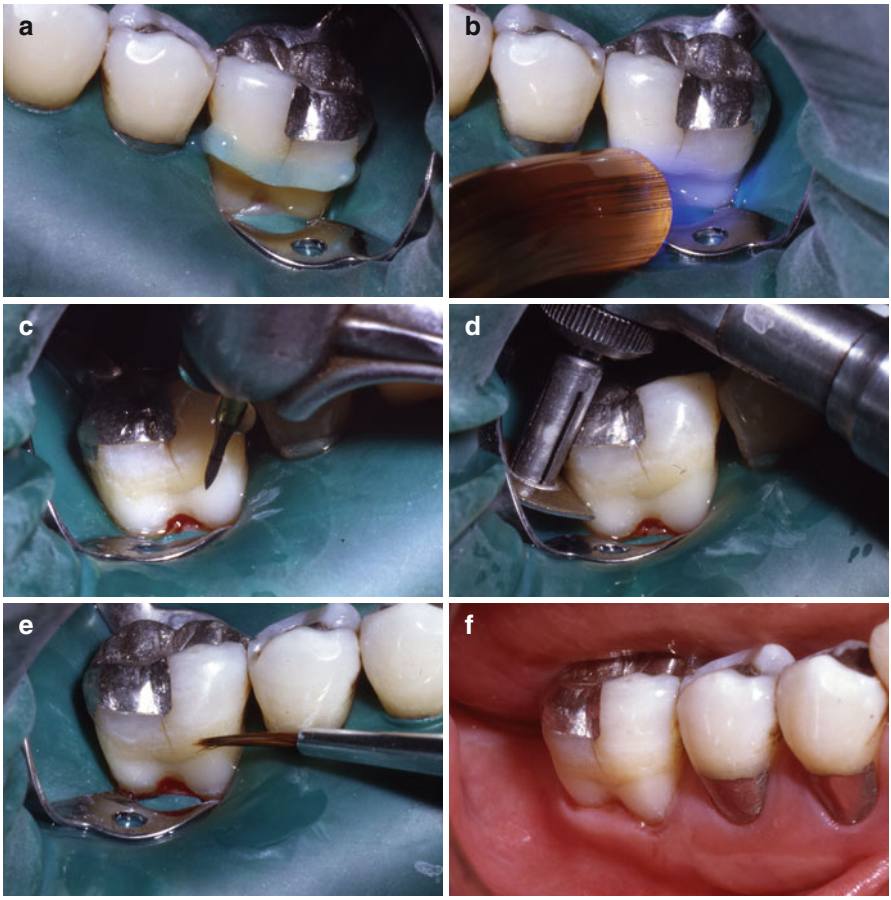


Fig. 14.12 Tooth surface preparation is accomplished according to manufacturer's directions. (**a**) The adhesive agent and each increment are thoroughly cured. (**b**) Fine diamond and multifluted carbide burs can trim axial and proximal contours. (**c**) Sequential aluminum oxide polishing discs can provide a smooth surface. (**d**) Finally, a low viscosity surface sealant should be applied to fill any contraction gaps formed around the periphery of the restoration and seal any of the microscopic defects created by finishing and polishing on the surface of the restoration. (**e**) Final restoration (f)

partial loss of a definitive CEJ in cases of abrasion has been reported to be as high as 70 % [52]. The goal of this interdisciplinary approach is to restore the missing coronal portion of the tooth to recreate the original anatomic position of the CEJ. This is followed by repositioning the soft tissue in esthetic harmony with adjacent teeth, which may not have recession and/or loss of tooth structure, and to cover exposed root surfaces.

Several authors have suggested techniques to predict the potential success of this interdisciplinary approach. In 1985, Miller [53] classified gingival recessions into four categories. In Miller Class I and II gingival recessions, there is no loss of interproximal periodontal attachment and bone and complete root coverage can be

achieved. In Miller Class III, the interdental periodontal support loss is mild to moderate, and partial root coverage can be accomplished. In Miller Class IV, the interproximal periodontal attachment loss is so severe that no root coverage is feasible.

However valuable, the Miller classification system only serves as a cursory examination for assessment of the success of root coverage procedures. It does not take into account some of the subtle components that might have an effect on the success of root coverage procedures in the presence of NCCLs. These components include, but are not limited to, the etiology of the lesion, its location, and depth, and the presence or absence of an identifiable CEJ. In 2011, Allen and Winter took these additional factors into consideration and created guidelines to help clinicians determine whether a graft or restoration could suffice as a stand-alone treatment, or whether a combination of the two would be more successful [54].

Zucchelli et al. [52] proposed a method for determining how much a case would benefit from surgical intervention, since complete root coverage is not always achieved. An estimate of the maximum root coverage (MRC) that can be expected is determined. The MRC is the most coronal level of soft tissue advancement achievable with surgical procedures, such as coronally advanced flaps and/or connective tissue grafts. It is determined by measuring the ideal papilla height, i.e., the distance from the interproximal contact measured apically to a line extrapolated horizontally from the location of the CEJ at the line angle. That length determined as the ideal papilla height is used to measure apically from the actual papilla to determine the maximum coronal tissue height that can be expected during root coverage procedures in that area. This is measured for both papillae, and a curved line connecting these measurement points give an estimate of the MRC (Fig. 14.13). This serves as a guideline for the necessary apical extent of the preparation and final restoration. The restoration can be completed prior to surgery to help with achieving proper emergence contour and marginal integrity.

The relationship of the MRC to the CEJ can provide the clinician with information on whether the root coverage can be accomplished with just soft tissue, or if a restoration should be placed first. This approach hinges on whether or not the CEJ has been disrupted by the NCCL formation.

The success of this combination restorative/surgical approach compared to just a coronally advanced flap procedure has been examined with Miller Class I cases by Santamaria et al. as a 2-year RCT [55, 56]. They found both procedures provided similar soft tissue coverage of roots after 6 months. Dentinal sensitivity was significantly reduced by the combined procedure.

Conclusions

Noncarious cervical lesions are complicated in their etiology, diagnosis, and treatment. A successful diagnosis and treatment plan requires a thorough patient history and careful clinical observation. Different approaches to treatment may be necessary for individual patients presenting with similar lesions based upon the most likely combination of contributing and modifying factors confronting that particular patient. Due to the threat of increased failure rate for NCCL

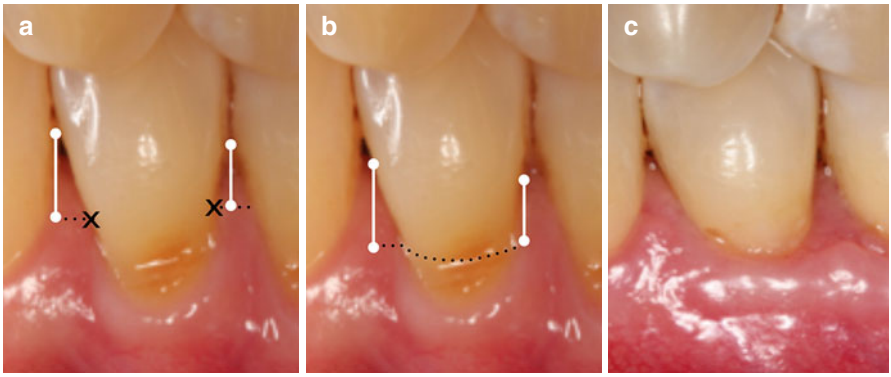


Fig. 14.13 The patient presented with a symptomatic NCCL on tooth #21. The maximum root coverage (MRC) was estimated by first measuring the ideal papilla height on each side of the tooth from just apical to the contact area to a level even with the location where the gingival tissue crosses the CEJ. (**a**) That distance was then extrapolated on each side starting at the actual papilla height directed apically. (**b**) A conservative Class V resin composite restoration was placed by the restorative dentist to recreate the original CEJs at the predetermined maximum root coverage (MRC) level. Surgical intervention with a connective tissue graft and flap was performed in concert with the periodontist. The final photo (**c**) reveals the gingival contour and level of attachment of this interdisciplinary treatment approach. The patient's initial dentin hypersentivity symptoms have ceased and the tissue level has stabilized

restorations over time, clinicians should weigh the advantages and disadvantages of treatment options seriously before choosing to restore. When a restoration is indicated, the material of choice should be based on the highest level of current evidence. Consideration of all these important issues can increase the likelihood for clinical success in identifying and managing noncarious cervical lesions.

References

1. Kaidonis JA. Tooth wear: the view of the anthropologist. Clin Oral Investig. 2008;12 Suppl 1:21–6.
2. Shulman EH, Rjobinson HG. Salivary citrate content and erosion of teeth. J Dent Res. 1948;27:541–4.
3. Bergstrom J, Eliasson S. Cervical abrasion in relation to tooth brushing and periodontal health. Scand J Dent Res. 1988;96:405–11.
4. Levitch LC, Bader JD, Shugars DA, Heymann HO. Noncarious cervical lesions. J Dent. 1994;22:195–207.
5. Rees JS, Hammadeh M, Jagger DC. Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. Eur J Oral Sci. 2003;111:149–54.
6. Sognnaes RF, Wolcott RB, Xhonga FA. Dental erosion. J Am Dent Assoc. 1972;84:571–6.
7. Radentz WH, Baarnes GP, Cutwright DE. A survey of factors possibly associated with cervical abrasion of tooth surfaces. J Periodontol. 1978;47:210–3.
8. Zipkin I, McLure FJ. Salivary Citrate and dental erosion. J Dent Res. 1949;28:613–26.
9. Miller WD. Experiments and observations the wasting of tooth tissue variously. Designated as erosion, abrasion, chemical abrasion, denudation, etc. Dent Cosmos. 1907;49:1–13.

10. Miller WD. Experiments and observations the wasting of tooth tissue variously. Designated as erosion, abrasion, chemical abrasion, denudation, etc. Dent Cosmos. 1907;49:109–24.
11. Miller WD. Experiments and observations the wasting of tooth tissue variously. Designated as erosion, abrasion, chemical abrasion, denudation, etc. Dent Cosmos. 1907;49:225–47.
12. Yip HK, Smales RJ, Kaidonis JA. Management of tooth tissue loss from erosion. Quintessence Int. 2002;33:516–20.
13. Grippo JO, Simring M, Coleman TA. Abfraction, abrasion, biocorrosion, and the enigma of noncarious cervical lesions: a 20-year perspective. J Esthet Restor Dent. 2012;24:10–23.
14. Grippo JO. Abfraction: a new classification of hard tissue lesions of teeth. J Esthet Dent. 1991;3:14–8.
15. Grippo JO, Simring M, Schreiner S. Attrition, abrasion, corrosion and abfraction revisited. J Am Dent Assoc. 2004;135:1109–18.
16. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci. 1996;104:199–206.
17. Lussi A, Kohler N, Zero D, Schaffner M, Megert B. A comparison of the erosive potential of different beverages in primary and permanent teeth using an in vitro model. Eur J Oral Sci. 2000;108:110–4.
18. ten Gate JM, Imfield T. Dental erosion, summary. Eur J Oral Sci. 1996;104:241–4.
19. Larsen MJ, Nyvad B. Enamel erosion by some soft drinks and orange juices relative to their pH, buffering effect and contents of calcium phosphate. Caries Res. 1999;33:81–7.
20. Attin T, Koidl U, Buchalla W, Schaller HG, Kielbassa AM, Hellwig E. Correlation of microhardness and wear in differently eroded bovine dental enamel. Arch Oral Biol. 1997;42:243–50.
21. 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.
22. Litonjua LA, Bush PJ, Andreana S, Tjobias TS, Cohen RE. Effects of occlusal load on cervical lesions. J Oral Rehabil. 2004;31:225–32.
23. Piotrowski BT, Gillette WB, Hancock EB. Examining the prevalence and characteristics of abfraction like cervical lesions in a population of US veterans. J Am Dent Assoc. 2001;32:1694–701.
24. Mayhew RB, Jessee SA, Martin RE. Association of occlusal, periodontal, and dietary factors with the presence of non-carious cervical dental lesions. Am J Dent. 1998;11:29–32.
25. Miller N, Penaud J, Ambrosini P, Bisso-Boutelliez C, Briancon S. Analysis of etiologic factors and periodontal conditions involved with 309 abfractions. J Clin Periodontol. 2003;30:828–32.
26. Pecie R, Krejci I, Garcia-Godoy F, Bortolotto T. Noncarious cervical lesions – a clinical concept based on the literature review. Part 1: prevention. Am J Dent. 2011;24:49–56.
27. Rees JS, Jagger DC. Abfraction lesions: myth or reality? J Esthet Restor Dent. 2003;15: 263–71.
28. Grippo JO, Masi JV. Role of biodental engineering factors (BEF) in the etiology of root caries. J Esthet Dent. 1991;3:71–6.
29. Palamara D, Palamara JE, Tyas MJ, Pintado M, Messer HH. Effect of stress on acid dissolution of enamel. Dent Mater. 2001;17:109–15.
30. Whitehead SA, Wilson NH, Watts DC. Development of noncarious cervical notch lesion in vitro. J Esthet Dent. 1999;11:332–7.
31. Lussi A. Dental erosion: clinical diagnosis and case history taking. Eur J Oral Sci. 1996;104:191–8.
32. Academy of Operative Dentistry. Recommendations for clinical practice: non-carious cervical lesions. Oper Dent. 2003;28:109–13.
33. Litonjua L, et al. Noncarious cervical lesions and abfractions: a re-evaluation. J Am Dent Assoc. 2003;134(7):845–50.
34. Kuroe T, Hidemi I, Caputo AA, Konuma M. Biomechanics of cervical tooth structure lesions and their restoration. Quintessence Int. 2000;31:267–74.
35. Wood I, Jawas Z, Paisley C, Brunton P. Non-carious cervical tooth surface loss: a literature review. J Dent. 2008;36(10):759–66.

36. Senna P, Del Bel Cury A, Rosing C. Non-carious cervical lesions and occlusion: a systematic review of clinical studies. J Oral Rehabil. 2012;39:450–62.
37. Perez Cdos R, et al. Restoration of noncarious cervical lesions: when, why, and how. Int J Dent. 2012; Article ID 68758;1–8.
38. Silverman G, Berman E, Hanna CB, et al. Assessing the efficacy of three dentifrices in the treatment of dentinal hypersensitivity. J Am Dent Assoc. 1996;127(2):191–201.
39. Brännström M, Johnson G, Linden LA. Fluid flow and pain response in the dentin produced by hydrostatic pressure. Odontol Revy. 1969;20:15–6.
40. Absi EG, Addy M, Adams D. Dentine hypersensitivity. A study of the patency of dentinal tubules in sensitive and non-sensitive cervical dentine. J Clin Periodontol. 1987;14:280–4.
41. Veitz-Keenan A, et al. Treatments for hypersensitive noncarious cervical lesions. J Am Dent Assoc. 2013;144:495–506.
42. Pecie R, Krejci I, Garcia-Godoy F, Bortolotto T. Noncarious cervical lesions (NCCL) – a clinical concept based on the literature review. Part 2: restoration. Am J Dent. 2011;24:183–92.
43. Peumans M, Kanumilli P, De Munck J, Van Landuyt K, Lambrechts P, Van Meerbeek B. Clinical effectiveness of contemporary adhesives: a systematic review of current clinical trials. Dent Mater. 2005;21:864–81.
44. Chee B, Rickman LJ, Satterthwaite JD. Adhesives for the restoration of non-carious cervical lesions: a systematic review. J Dent. 2012;40:183–92.
45. Burrow MF, Tyas MJ. Clinical evaluation of three adhesive systems for the restoration of non-carious cervical lesions. Oper Dent. 2007;32:11–5.
46. Heintze SD, Ruffieux C, Rousson V. Clinical performance of cervical restorations- a meta – analysis. Dent Mater. 2010;26:993–1000.
47. Hilton TJ, Ferracane JL. Cavity preparation factors and microleakage of Class III composite restorations filled at intraoral temperatures. Am J Dent. 1998;11:123–30.
48. Tay FR, Pashley DH. Resin bonding to cervical sclerotic dentin: a review. J Dent. 2004;32(3):173–96.
49. Feilzer AJ, De Gee AJ, Davidson CL. Setting stress in composite resin in relation to configuration of the restoratives. J Dent Res. 1987;66:1636.
50. Dowling AH, Fleming JP. Are encapsulated anterior glass-ionomer restoratives better than their hand-mixed equivalents? J Dent. 2009;37:133–40.
51. Camargo PM, Lagos RA, Lekovic V, Wolinsky LE. Soft tissue root coverage as treatment for cervical abrasion and caries. Gen Dent. 2001;49(3):299–304.
52. Zucchelli G, Testori T, De Sanctis M. Clinical and anatomical factors limiting treatment outcomes of gingival recession: a New method to predetermine the line of root coverage. J Periodontol. 2006;77:714–21.
53. Miller Jr PD. A classification of marginal tissue recession. Int J Periodontics Restorative Dent. 1985;5(2):8–13.
54. Allen EP, Winter RR. Interdisciplinary treatment of cervical lesions. Compendium. 2011;32(Special Issue 5):16–20.
55. Santamaria MP, Suaid FF, Casati MZ, Nociti FH, Sallum AW, Sallum EA. Coronally positioned flap plus resin-modified glass ionomer restoration for the treatment of gingival recession associated with non-carious cervical lesions: a randomized controlled clinical trial. J Periodontol. 2008;79(4):621–8.
56. Santamaria MP, da Silva Feitosa D, Nociti FH, Casati MZ, Sallum AW, Sallum EA. Cervical restoration and the amount of soft tissue coverage achieved by coronally advanced flap: A 2-year follow-up randomized-controlled clinical trail. J Periodontol. 2009;36(5):434–42.

Dentin Hypersensitivity: Prevalence, Etiology, Pathogenesis, and Management

15

Cor van Loveren, Patrick R. Schmidlin, Luc C. Martens, and Bennett T. Amaechi

Abstract

Dentin hypersensitivity is simply defined as a short sharply painful reaction of the exposed and innervated pulp-dentin complex in response to stimuli being typically thermal, evaporative, tactile, osmotic, or chemical and which reaction cannot be attributed to any dental defect or pathology. To be hypersensitive, dentin must be exposed and the exposed tubules must be open and patent to both the oral cavity and the pulp. Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or by neglected oral hygiene. Exposure of dentin by the loss of the protecting enamel is mainly caused by erosion, abrasion, and abfraction or a combination thereof. Clinical examination for dentin hypersensitivity would include a pain provocation test by a tactile stimulus, an evaporative air stimulus, or a cold stimulus. A number of other dental conditions can give rise to pain symptoms, which may mimic those of dentin

C. van Loveren, DDS PhD (✉)
Department of Preventive Dentistry, Academic Center for Dentistry Amsterdam (ACTA), University of Amsterdam and VU University Amsterdam, Gustav Mahlerlaan 3004, Amsterdam 1081 LA, The Netherlands
e-mail: c.van.loveren@acta.nl

P.R. Schmidlin
Clinic of Preventive Dentistry, Periodontology and Cariology, Centre of Dental and Oral Medicine, University of Zurich, Plattenstrasse 11, Zurich 8032, Switzerland
e-mail: patrick.schmidlin@zzm.uzh.ch

L.C. Martens, DDS, MSc, PhD
Department Paediatric Dentistry & Special Care Dentistry, Dental School of Ghent, De Pintelaan 185 (P8), Ghent B-9000, Belgium
e-mail: Luc.Martens@UGent.be

B.T. Amaechi, BDS, MSc, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_15

hypersensitivity. Therefore, careful examination is necessary to exclude the conditions, which need different treatment options. When the patients do suffer from dentin hypersensitivity, there is broad range of treatment options comprising home-use and professional approaches. It is advised to start with the less invasive home-use therapies and only expand to professional in-office treatments when the home-use treatments are not effective. When decided to continue with in-office treatments, again one should start with the least invasive ones. The working mechanisms fall under two basic categories being nerve desensitization (potassium salts and guanethidine) and occlusion of exposed dental tubules (chemically: strontium, fluoride, stannous, oxalate, calcium phospho silicate, arginine calcium carbonate, nano-hydroxyapatite, and glutaraldehyde; mechanically: pumice paste, glassionomers, dentin bondings, and resins; laser therapy). Regenerative mucogingival therapy also remains an alternative, where hard and soft tissue conditions allow.

15.1 Prevalence

Dentin hypersensitivity (DHS) is simply defined as a short sharply painful reaction of the exposed and innervated pulp-dentin complex in response to stimuli being typically thermal, evaporative, tactile, osmotic, or chemical. An important part of the definition is that the reaction cannot be attributed to any dental defect or pathology [1].

Studies report a wide range of prevalence rates varying form 3 up to 98 %, which can be explained in part by different evaluation methods and different patient populations, but, generally, patients display higher degrees immediately after periodontal treatment [2, 3]. In a large European study in which over 3000 18–35 years old patients from general dental practices in France, Spain, Italy, United Kingdom, Finland, Latvia, and Estonia were enrolled, the self-reported prevalence of dentin hypersensitivity was approximately 27 %, while 42 % of the patients reported pain upon cold air stimulation of exposed dentin surfaces [4]. There was a significant heterogeneity regarding this prevalence data between the countries. The differences in prevalence between self-reported sensitivity and clinical elicited sensitivity may reflect that patients develop coping strategies to avoid incitement of the pain.

The exposure of dentin of the root or crown is essential. For root dentin exposure, it is important to acknowledge that mainly localized attachment loss due to anatomically predisposing factors or periodontal disease is probably the most widespread and relevant factor leading to root surface denudation and subsequent dentin hypersensitivity. Several predisposing factors of gingival recessions have been identified, e.g., dehiscency or fenestration of the alveolar bone and soft tissue morphotypes, but triggering pathological, therapeutic, or iatrogenic factors are also crucial for its development [5, 6]. At the (mainly cervical aspects of) crowns, loss of the protecting enamel is considered an alternative pathway of dentin exposure and is mainly caused by erosion, abrasion, and abfraction or a combination thereof [5]. These

processes may also increase the patency of tubules depending on the specific etiologic factor. West and coworkers [4] found significant associations between the elicited dentin hypersensitivity and erosive tooth wear. This study showed a significant association between fresh fruit, isotonic/energy drinks, but less clearly fruit/vegetable juices with the increased dentin hypersensitivity. A significant association was found for dentin hypersensitivity in patients reporting frequent heartburn and gastric reflux and to a lesser extent frequent vomiting. These associations are consistent with increased erosive tooth wear and the impact on dentin hypersensitivity response by removing the dentin smear layer and opening tubules [6].

This clear implication of a topically acidic environment explains why dentin hypersensitivity is discussed in a book on tooth wear.

15.2 Etiology and Mechanisms

The currently accepted hypothesis is the hydrodynamic theory, first suggested by Gysi [7] and later substantiated by Brännström [8] (Fig. 15.1). Dentin hypersensitivity is caused by movement of the dentinal tubule contents which may exert a shear force and a so called "streaming potential" exciting intradental A-type nerve fibers causing a sharp, shooting pain [9]. An increased outward movement may be more painful than an inward flow of the tubule fluid. This explains that cold, which causes an outward stream, generally triggers more dramatic pain than heat, which causes a fluid retreat [10]. The characteristic sharp pain experienced with dentin hypersensitivity can persist as a dull, throbbing ache for variable periods of time. The nerves causing this pain are not excited by the hydrodynamic mechanism. Hypersensitivity may sometimes persist despite of blocking the tubules, which also indicates that some other mechanisms may operate in the nerve activation instead of, or in addition to the hydrodynamic one. Inflammation may sensitize the nerve endings to such an extent that smaller fluid shifts would be sufficient for nerve activation or, for example, thermal stimulation may activate the nerves by a direct effect

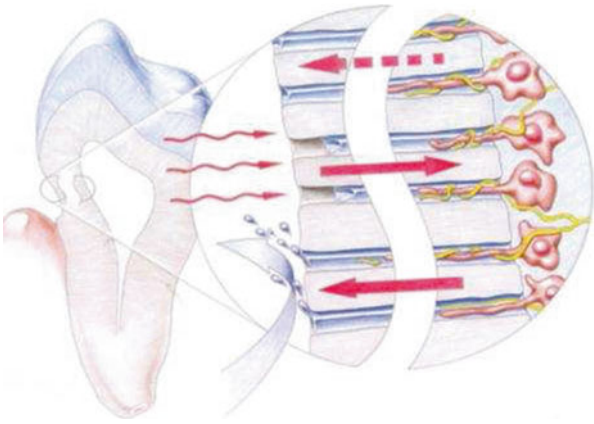


Fig. 15.1 Schematic drawing of the hydrodynamic mechanisms causing dentin hypersensitivity. Excitation at the dentin surface (i.e., root surface) causes an inward or outward flow of the intratubular fluid exciting the pulp nerve fibers (Courtesy of Dr. Luc M Martens)

[11]. In cases of interdental dentin hypersensitivity occurring in periodontally involved teeth, microorganisms invading the root dentin have also been discussed [12]. This condition may be of different etiology but results in similar pain symptoms. This type of dentin hypersensitivity is often referred to as root sensitivity.

Regarding the hydrodynamic mechanism, dentin will only be sensitive if the tubules are patent from the pulp to the oral environment (Fig. 15.2). Sensitive teeth have up to eight times more and up to two times wider tubules at the buccal cervical area as compared to nonsensitive teeth [13]. It has also been shown that smear layers in sensitive dentin are thinner and less calcified as compared to those of nonsensitive dentin [14]. As the patency will change with production and removal of the smear layer, episodic conditions are possible [13]. Spontaneously occurring changes in the exposed dentin, which in many cases seem to block the tubules, may reduce the responses to hydrodynamic stimulation and, thus, have an alleviating effect on dentin sensitivity.

15.2.1 Predisposing Factors

15.2.1.1 Gingival Recession Exposing Dentin

To be hypersensitive, dentin must be exposed and the exposed tubules must be open and patent to both the oral cavity and the pulp [10, 15]. Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or by neglected oral hygiene (Fig. 15.3). The exact mechanism by which too meticulous oral hygiene causes loss of tissue is not very well understood and often implies brushing force and brush bristle characteristics. Several studies have shown the injury potential of sharp nonrounded filament tips on gingival abrasion [16, 17]. Surprisingly there is no information on the role of toothpastes in this process. Such a role could be both physical, through abrasion, and chemical, through cytotoxicity of ingredients such as detergents to the soft tissues [18]. In any case, modifying

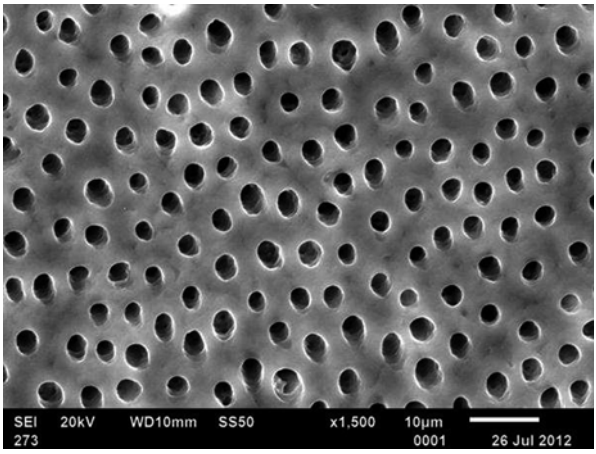


Fig. 15.2 Open tubules are a prerequisite for dentin hypersensitivity in most cases (Courtesy of Dr. Bennett T. Amaechi)

factors like inserting frenula, thin gingival biotypes, a lack of keratinized gingiva, or absence of the buccal bone may be implicated and should be considered. The mechanism by which neglected oral hygiene causes recessions runs through acute and chronic periodontal diseases and nonsurgical and surgical treatments.

15.2.1.2 Loss of Hard Tissue Exposing Dentin

Exposure of dentin by the loss of enamel is often ascribed to abrasion. However, most abrasives are softer than enamel and it must be concluded that toothpaste abrasion alone would play a clinically insignificant role in exposure of dentin [18]. In contrast, acids from intrinsic or extrinsic sources are more harmful for enamel by dissolution and by softening. The softened enamel is subsequently abraded away by mechanical forces. Shear forces of the oral soft tissues may be sufficient to abrade the softened enamel [19], but toothbrushing surely will as will grinding and clenching [20]. So when there is exposure of dentin as a result of loss of enamel, the patient's history should reveal the role of intrinsic or extrinsic acids (Table 15.1).

Since nonsensitive dentin reveals few if any open dentinal tubules at the surface [13], it is assumed that the tubules are covered by a "smear layer," consisting of protein components and calcium phosphate deposits derived from saliva [23] (Fig. 15.4). To initiate dentin hypersensitivity this layer has to be removed, and in vitro and in situ studies implicate erosive wear, as the smear layer is sensitive to acids [24, 25]. When acids have softened the smear layer and dentin, the materials

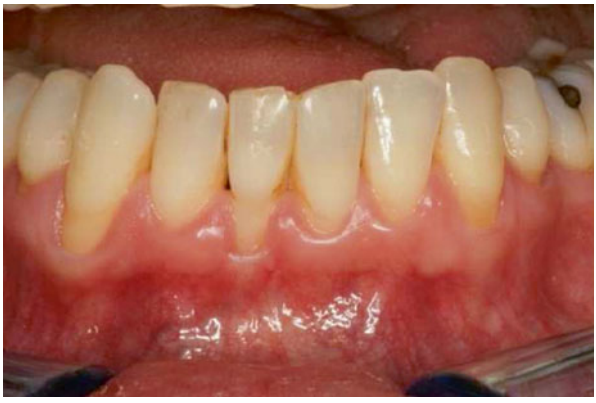


Fig. 15.3 Exposure of dentin through the loss of gingival and periodontal tissue may be caused by either too meticulous or neglected oral hygiene (Courtesy of Dr. Luc M Martens)

Table 15.1 Patient history

Ask patient to describe pain (look for description of pain as short, sharp)
Ask patient to identify pain-inciting stimuli (thermal, tactile, evaporative, osmotic, chemical)
Determine patient's desire for treatment
Probe for lifestyle habits/practices, intrinsic and extrinsic acid (citrus juices and fruits, carbonated drinks, wines, ciders)
Obtain detailed dietary information including dietary intake relevant to medical problems
Probe for gastric acid reflux and excessive vomiting

Canadian Advisory Board on Dentin Hypersensitivity, 2003; Martens, 2013 [21, 22]

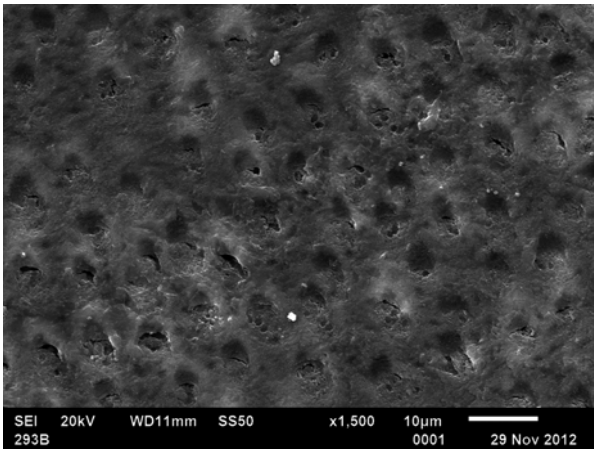


Fig. 15.4 Dentin surface of Fig. 15.2 covered by a smear layer (Courtesy of Dr. B Amaechi)

are more susceptible to physical forces, such as toothbrushing. Clinical data suggest that physical forces alone are not a key factor in removing the smear layer and opening exposed dentin tubules [10]. Also toothpaste will remove the smear layer [25, 26] probably by a combined abrasive and detergent action. Moore and Addy [27] have suggested that certain "mild" surfactants and "gentle" abrasives might have advantages over their more traditional counterparts in toothpastes marketed for the relief of dentin hypersensitivity [27]. However, this hypothesis does not appear to have been clinically validated in well-designed clinical studies [15]. One study showed no difference in desensitizing effect after elicitation using the evaporative method when using four desensitizing toothpastes different in abrasivity with RDA 60, 108, 150, or 210, respectively [28].

Subsequent to tubule exposure, toothpaste may reduce patency by secondary abrasive smearing or deposition of toothpaste constituents onto the dentin surface and into tubules. This makes the role of toothpaste without active ingredients to reduce dentin hypersensitivity inconclusive, even of fluoride containing pastes. Additionally it suggests that when using desensitizing toothpastes application with a fingertip or cotton swab after brushing may be beneficial.

15.3 Clinical Assessment

Clinical examination for dentin hypersensitivity would include a pain provocation test. However, the patient's perception of dentin hypersensitivity is subjective and clinical evaluation based on any scoring or rating system regarding its severity is challenging. Nevertheless, it is important to detect, rate, and monitor the pain as accurately as possible in order to define the baseline status and to observe any changes in due course and after therapy. Ideally, the latter ends in a status where "no pain" can be attested, but this ideal dichotomous treatment goal is still difficult to achieve.

Provocation tests are most frequently used to simulate pain and to assess the immediate reaction:

1. Tactile stimulus. This is the use of a probe, which is used as a "scratch" test on the exposed dentin, preferably with a standardized pressure. The use of probes would be contraindicated in evaluating treatments that use adhesive restorative materials, or other barrier methods. In such cases, the use of controlled air stimuli, graded cold water, or contact cold probes would be more appropriate [29].
2. An evaporative air stimulus. The Schiff Cold Air Sensitivity Scale is frequently used to assess the subject response to the air blast hypersensitivity [30]. This scale is scored as follows:
   0 = Subject does not respond to air stimulus.
   1 = Subject responds to air stimulus but does not request discontinuation of stimulus.
   2 = Subject responds to air stimulus and requests discontinuation or moves from stimulus.
   3 = Subject responds to air stimulus, considers stimulus to be extremely painful, and requests discontinuation of the stimulus.
   Noteworthy, the teeth on either side of the tooth under investigation should be isolated so that no referred pain is detected.
3. A cold stimulus, which can be graded cold water or contact cold probes.

After this pain induction, either a scoring system such as "Dental Pain Scale (DPS)" rates the pain answer or a visual analogue scale (VAS) can be used to "quantify" the severity in millimeter (mm) (Fig. 15.5).

Often individuals will not respond to all types of stimulus or may respond differently to different stimuli [31–33], so it is recommended that at least two

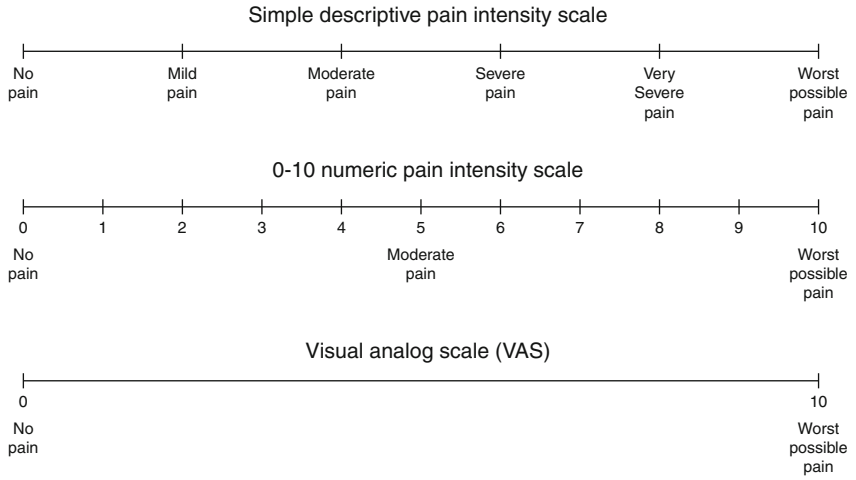


Fig. 15.5 Frequently used scales for pain intensity measurement

hydrodynamic stimuli should be used. The interval between stimulus applications should be of sufficient duration to minimize interactions between stimuli. If multiple stimuli are used to help to achieve a diagnosis, the order of application should be that which causes the least to the most amount of pain [34]. Repeated testing should be avoided as it is not known how long it takes to reach threshold evaluation. In case of a negative provocation test, any dentin hypersensitivity therapy becomes needless.

It is also questionable to what extent therapies should be performed in cases where patients display with no self-reported pain but show the typical signs of dentin hypersensitivity during a routine clinical examination. In fact, prophylactic measures, which protect the exposed surfaces against cariologic and wear challenges, may be considered, but the patient's awareness of nonexisting subjective pathologic conditions should not be stimulated.

15.3.1 Oral Health-Related Quality of Life

Dentin hypersensitivity may disturb the patient during eating, drinking, toothbrushing, and sometimes even breathing. The resulting restrictions on everyday activities can have an important effect on the patient's quality of life [35]. Oral health-related quality of life (OHRQoL) is a relatively new concept in dentistry. It is an aspect of dental health addressing the patient's perception of whether his/her current oral health status has an impact upon his/her actual quality of life [35]. Therefore, OHRQoL may provide a new perspective when looking at a patient, by measuring treatment efficacy in terms of patient satisfaction. There is only little research into the relevance of the various quality of life questionnaires in the treatment of dentin hypersensitivity, yet it may be very valuable to the patient to evaluate the treatment according to these values. Boiko et al. [36] developed, based on in-depth and focus group interviews, a dentin hypersensitivity experience questionnaire (Table 15.2) to capture subjective impacts on patients. The questions can be phrased like "Having the sensations in my teeth takes a lot of the pleasure out of eating and drinking" after which the patients can indicate to what extent he agrees or disagrees.

15.4 Differential Diagnosis

A number of other dental conditions can give rise to pain symptoms, which may mimic those of dentin hypersensitivity. Therefore, careful examination is necessary to exclude the following conditions, which need a variety of different treatment options [6, 22, 37, 38]:

- Cracked tooth syndrome
- Incorrect placement of dentin adhesives in restorative dentistry, leading to nanoleakage
- Fractured restorations and incorrectly placed dentin pins

- Inappropriate application of various medicaments during cavity floor preparation
- Lack of care while contouring restorations so the tooth is left in traumatic occlusion
- Pulpal response to caries and recent restorative treatment
- Palatogingival groove and other enamel invaginations and defects
- Chipped/fractured teeth causing exposed dentin
- Tooth bleaching
- Acute periodontal infections (e.g., necrotizing gingivitis/periodontitis or abscesses)

Table 15.2 The items of the dentin hypersensitivity experience questionnaire developed by Boiko et al. [36] to determine the impact of dentin hypersensitivity on a patient's quality of life

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Restrictions	Pleasure out of eating					
	Cannot finish meal					
	Longer to finish meal					
	Problems with eating ice-cream					
Adaptation	Modification of eating					
	Careful when breathing					
	Warming food/drinks					
	Cooling food/drink					
	Cutting fruit					
	Putting a scarf over mouth					
	Avoiding cold drinks/ foods					
	Avoiding hot drinks/ foods					
	Avoiding contact with certain teeth					
	Change toothbrushing habits					
	Biting in small pieces					
	Avoiding other food					
Social	Longer than others to finish					
	Choose food with others					
	Hide the way of eating					
	Unable to take part in conversations					
	Painful at the dentist					

(continued)

Table 15.2 (continued)

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Emotions	Frustrated not finding a cure					
	Anxious of eating contributes					
	Irritating sensations					
	Annoyed with myself for contributing					
	Guilty for contributing					
	Annoying sensations					
	Embarrassing sensations					
	Anxious because of sensations					
Identity	Difficult to accept					
	Different from others					
	Makes me feel old					
	Makes me feel damaged					
	Makes me feel unhealthy					

15.5 Preventive Strategies

Prevention is always better than cure. Thus, primary prevention represents the first line of defense against dentin exposure, i.e., the formation of gingival recession and dental hard tissue deterioration. Careful oral hygiene instructions and dietary advices are crucial. When dentin is already exposed, patients should be instructed in order to minimize the risk of opening the tubules and, thus, increasing the patency. Suggestions for patients and the dental professionals to avoid aggravating behavior or iatrogenic damage developed by Martens [22] are given in Table 15.3.

15.6 Treatment Strategies

When the patients do suffer from dentin hypersensitivity, there is broad range of treatment options comprising home-use and professional approaches. It is advised to start with the less invasive home-use therapies and only expand to professional in-office treatments when the home-use treatments are not effective. When decided to continue with in-office treatments, again one should start with the least invasive ones.

Table 15.3 Suggestions for patients and the dental professionals to avoid aggravating behavior or iatrogenic damage developed by Martens [22] based on Chu et al. [39] and Drisko [40]

Suggestions for patients
Limit dietary acids
Use soft-medium toothbrush and adequate brushing technique
Use additional topical fluorides
Avoid picking, scratching at the gingival margins
Avoid excessive flossing or improper use of toothpicks
Suggestions for dental professionals
Avoid overinstrumentation of the root surfaces during scaling
Avoid excessive polishing of exposed dentin during stain removal
Avoid burning the gingival tissues during in-office bleaching
Advise patients to be careful during home-bleaching
Avoid harmful instruments and materials

Home-use products have several benefits including ease of use, convenience of self-application, and easier access but may require several weeks before taking effect. In-office treatments are generally more invasive and more effective under specific conditions and can provide instant relief, e.g., an adhesive sealing or restoration.

A remaining aspect is the placebo effect, which is an important and potentially beneficial side effect when dealing with pain and its treatment and management. Using arthritis of the knee as an example in the medical field, it has been impressively shown that sham endoscopic interventions lead to the same reduction of pain and symptoms as conventional treatment modalities [41]. In addition, prescription of differently colored pills resulted in significant differences in pain reduction [42]. Whereas a red placebo tablet, for instance, showed comparable pain relief as the best antirheumatic test pill used, the blue equivalent showed the least effect. Thus, improved psychological cotherapeutic strategies may one day become an important auxiliary aspect in dentin hypersensitivity management, especially when it comes to changing patients' expectations of treatment outcomes and confidence. The psychological training of dental professionals still has some room for development.

15.6.1 At-Home Therapy

For home use, both toothpaste and mouthrinses are available. There are a few studies on chewing gum but the results are not very reliable [43, 44]. The working mechanisms fall under two basic categories, being nerve desensitization and occlusion of exposed dental tubules (Table 15.4).

15.6.1.1 Nerve Desensitization

Potassium salts and, to a lesser extent, strontium and calcium [45] are agents that may have a direct desensitizing action on the nerves located at the pulpal side of the

Table 15.4 Active ingredients in products for at-home therapy

						Calcium/hydroxyapatite carriers		
Active ingredient	Potassium	Strontium	Fluoride	Stannous salts	Oxalates	Calcium phospho silicate (Novamin)	Arginine calcium carbonate (Pro-arginin)	Nanohydroxyapatite
Mechanism of action	Nerve Desensibilization	Precipitative occlusion of dentin tubules						
Compatible with Fluoride	Yes	SrCl not SrAc yes	Yes	Yes	Yes	MonoFluoro Phosphate (MFP)	MFP Sodium Fluoride	Yes Caveat: some pastes do not contain F

tubules. Therefore, the ions must be able to pass through the dentinal tubules against the dentin fluid flow and build up a sufficiently high concentration to desensitize the nerves at the interface of the inner dentin surface and the pulpal chamber. A concentration of 8 mM might be necessary needing a lag time of several weeks before pain relief is experienced. Once at the nerve site, potassium alters the cell's electrical potential, resulting in depolarization, making the cell less responsive to stimuli. When people stop using the product, the potassium will diffuse away, and sensitivity reestablishes. Strontium as other divalent cation may operate by a different mechanism from potassium, such that the membrane of the nerve cell is stabilized but the potential of the cell remains unchanged [45].

Mainly potassium nitrate (5 %), citrate (5.5 %), and chloride (3.75 %) have been formulated into toothpastes as each of the salts provides 2 % potassium, which is needed for relief. In the United States of America, desensitizing toothpastes typically contain 5 % potassium nitrate, to meet FDA regulations. Many manufacturers have a potassium-based desensitizing product, suggesting it being (or having been) the "golden standard." A recent Cochrane review included six studies in a meta-analysis, which showed a statistically significant effect of potassium nitrate toothpastes on air blast and tactile sensitivity tests at 6–8 weeks follow-up, respectively. The subjective reports of the patients on dentin hypersensitivity, in contrast, failed to show a significant effect at the respective time points (Table 15.5) [46].

15.6.1.2 Tubule Occlusion

As mentioned previously, tubules must be patent in order to allow for fluid movements. Blocking or occluding these patent tubules, therefore, seems a simple and conceptually effective way of decreasing sensitivity. There are several mechanisms by which products for home use can occlude exposed dentinal tubules. Mechanical formation of a natural smear layer by burnishing dentin induces tubule occlusion. Topically applied compounds, which form insoluble materials that precipitate in the tubules and on the surface, are also effective (Fig. 15.6). Such compounds include abrasive particles, strontium, stannous, arginine calcium carbonate, oxalate, or bioactive glasses.

Addy and Mostafa [47] examined in vitro three artificial silica abrasive based toothpastes, two with strontium acetate alone or combined with fluoride and one without. The study showed that these formulations coated the dentin surface and occluded the tubules. The analysis revealed that the occluding agent was the artificial silica, which was not water or acid labile. A parallel clinical study also showed all three artificial silica formulations to be effective in the treatment of dentin hypersensitivity [48]. Many of today's toothpaste contain similar artificial silica abrasives, but are not as effective in reducing dentin hypersensitivity. One explanation may lay in the use of sodium lauryl sulphate (SLS) as detergent in most tooth pastes, which would compete with silica for the adsorption to dentin. The experimental toothpaste in the study of Addy and Mostafa [47] did not contain SLS. It is important to note, however, that the dentin hypersensitivity benefits attributed to the presence of silica abrasive in these strontium toothpastes have not been reproduced in other studies, which included silica-based toothpastes [49, 50].

Table 15.5 Results of a systemic review and meta-analysis on the effect of potassium-containing toothpastes on dentin hypersensitivity

Outcome and comparison	No. of studies	No. of participants	Effects size (95 % CI) (std mean difference[a])
Tactile	*5*		*1.19 (0.79, 1.59)*
Potassium nitrate no F Versus No potassium nitrate no F	1	110	0.72 (0.33, 1.11)
Potassium nitrate plus F Versus No potassium nitrate plus F	4	246	1.34 (0.97, 1.71)
Air blast	*6*	*392*	*−1.25 (−1.65, −0.85)*
Potassium nitrate no F Versus No potassium nitrate no F	2	146	−1.18 (−1.88, −0.48)
Potassium nitrate plus F Versus No potassium nitrate plus F	4	246	−1.30 (−1.88, −0.72)
Subjective	*3*	*206*	*−0.67 (−1.44, 0.10)*
Potassium nitrate no F Versus No potassium nitrate no F	2	146	−1.01 (−1,53, −0.49)
Potassium nitrate plus F Versus No potassium nitrate plus F	1	60	0.10 (−0.41, 0.60)

From Poulsen et al. 2006 [46]

[a]The standardized mean difference is used as a summary statistic in meta-analysis when the studies all assess the same outcome but measure it in a variety of ways (i.e., the use of different scales). In this circumstance, it is necessary to standardize the results of the studies to a uniform scale before they can be combined. The standardized mean difference expresses the size of the intervention effect in each study relative to the variability observed in that study

Strontium chloride was introduced more than 50 years ago. Today, most products contain strontium acetate due to its improved clinical efficacy and its compatibility with fluoride and potassium nitrate. But there are still strontium chloride toothpastes on the market that do not contain fluoride. Several mechanisms are hypothesized by which strontium would reduce dentin hypersensitivity: (1) precipitation of particles on the tooth surface, (2) incorporation in the dentin matrix making it less soluble, and (3) stabilization of the membrane of dental nerves [45]. There is very little scientific evidence to support any of these mechanisms, but the first one has been proposed to be the most likely one [51]. A recent review on clinical studies found insufficient data for making any absolute conclusions about the efficacy of strontium treatment due to the diversity of testing methods used in the studies [15].

Stannous salt solutions precipitate onto dentin and may block tubules. The deposits are water and acid resistant and may even provide a protective effect against acid erosion [52]. Clinical studies reported efficacy of stannous fluoride gel or

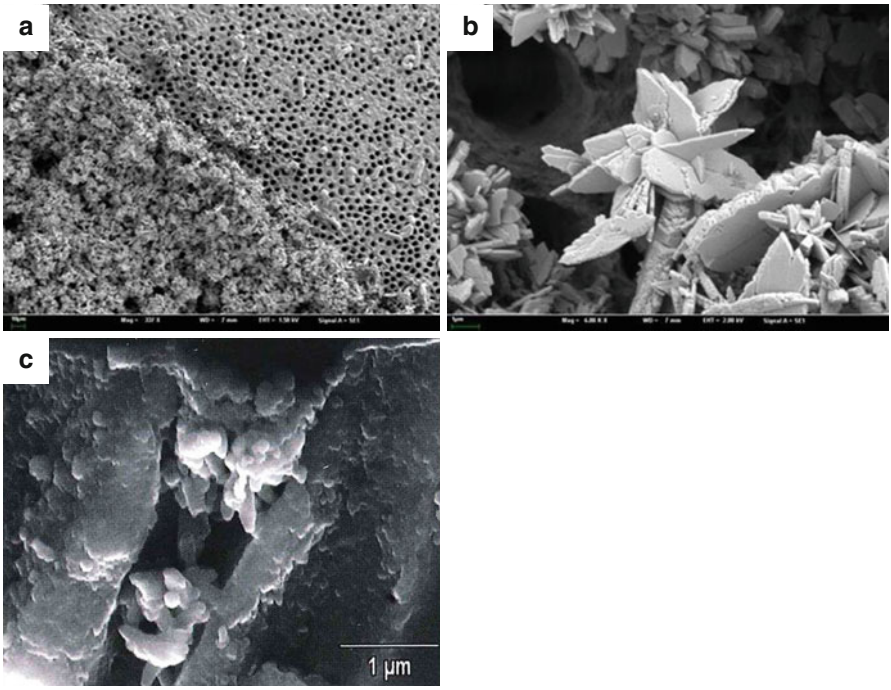


Fig. 15.6 SEM image of dentin treated with a precipitating agent: (**a**) shows treated (*bottom left*) and untreated (*top right*) areas. However, despite clear evidence of crystallite deposition, uncovered dentin areas and tubule entrances can be seen (**b**). A plug precipitated in the orifice of a dental tubule (**c**). (Courtesy of Dr. L.M. Martens)

solutions in the treatment of dentin hypersensitivity [53–55]. More recently, randomized controlled trials have reported that hexametaphosphate stabilized stannous fluoride toothpaste provided some immediate relief, as well as after 4 and 8 weeks [56–59].

Recently, a mouthrinse containing 1.4 % potassium oxalate has also been introduced. Soluble oxalate salts have been shown to occlude tubules by reacting with naturally occurring calcium ions in the oral fluids to precipitate as insoluble calcium oxalate crystals [60]. This precipitate blocks fluid flow in the dentinal tubules, leading to decreased hypersensitivity. The precipitates of oxalates are relatively resistant to dissolution in acidic environments, increasing their durability [61].

Bioactive glass consists of specific proportions of SiO_2, Na_2O, and P_2O_5 (calcium sodium phosphosilicate). Bioglass in solution or toothpaste interacts on the dentin surface and forms a hydroxyapatite-like silica deposit over the dentin and in the tubules [62]. This tubule blocking deposit appears water and acid insoluble and mechanically resistant. A number of randomized controlled trials extending up to 8 weeks showed significant benefits for the CSPS product in the treatment of dentin hypersensitivity [63–66].

Arginine, an amino acid naturally present in saliva, works in conjunction with calcium carbonate and phosphate to create a plug in dentinal tubules that prevents fluid flow [15]. The hypothesized mechanism of action suggests that the positively charged arginine is attracted to negatively charged dentin. The alkaline pH promotes deposition of calcium, phosphate, arginine, and carbonate on the dentin surface and inside the dentin tubules [67]. Several studies have shown 8 % arginine toothpaste and 0.8 % arginine mouth washes to be effective against dentin hypersensitivity [68–71].

Nanohydroxyapatite (nHAP) in dentifrice promotes deposition of precipitate layer over and within the dentin tubules by acting as a calcium and phosphate reservoir, helping to maintain a topical state of supersaturation of these ions with respect to tooth minerals, and thus causing deposition on the surface of tooth tissue. In a double-blind randomized clinical trial comparing the efficacy in reducing DHS of a dentifrice formulation containing 15 % nHAP without fluoride, with fluoride dentifrice, and a placebo, a significant reduction of cold air sensitivity and tactile sensitivity were observed for the nHAP group at 2 and 4 weeks compared to baseline and the two comparison groups [72].

At-home product comparison reveals that the products should be considered equally effective and can be recommended for use [18]. The various modes of actions and different solubility of the various precipitates that form suggest that when one product does not give sufficient relief, it might be worthwhile to try another product. After an evaluation period of 4–6 weeks (for potassium salts maybe 8 weeks), another product may be tried before proceeding to the in-office treatments.

With regard to laboratory studies, which are frequently used to show occluding effects on dentin, one should critically amend that most of these studies were performed without the simulation of dentin fluid dynamics, i.e., a liquid outflow. Therefore, precipitation phenomena should not be overestimated. In addition, brushing and acid challenges are also not performed in most studies, which may additionally impair the long-term stability of any claimed layer formation.

15.6.2 In-office Treatment

In the dental office, comparable compounds are available as mentioned before, but, as said, in more powerful compositions. Products containing the following agents can be used:

- Dentin bonding agents
- Composite resins
- Fluoride varnishes
- NaF ionthophoresis
- Glutaraldehyde-based agents
- Remineralization promoting cements
- Laser therapy

Dentin bonding agents and composite resin materials exhibit long-term or permanent effects. These materials can effectively seal dentinal tubules by forming a hybrid layer, block tubules by forming tags, and create a covering layer [73].

Some primers contain glutaraldehyde, which can lead per se to protein coagulation within the dentinal tubules, while the adhesive resin materials form an occluding barrier on a more hydrophobic surface. Such materials have shown good results in dentin hypersensitivity management in clinical trials [29].

When using cements, there may be benefit from using the remineralization promoting cements, which are cements containing calcium and phosphates [74, 75], and recently a calcium silicate paste, derived from Portland cement, was shown to be effective in the occlusion of tubules in in vitro experiments [76].

Sgolastra et al. [77] systematically reviewed the literature on lasers for the treatment of dentin hypersensitivity. They identified several theories by which lasers may be effective. For low-intensity lasers (e.g., Gallium-Aluminum-Arsenide (GaAlAs)), the irradiation may have a photo-bio-modulating effect on cellular activity, increasing the deposition of tertiary dentin by odontoblastic cells [78]. Middle-output-power lasers (e.g., Erbium: Yttrium Aluminium Garnet (Er:YAG), Neodymium: Yttrium Aluminium Garnet (Nd:YAG), and Erbium, Chromium: Yttrium, Scandium, Gallium, Garnet (Er,Cr:YSGG)) may reduce or obliterate the dentinal tubules [79]. For Er:YAG and Er,Cr:YSGG, the efficacy in reducing dentin hypersensitivity is thought to be related to the thermo-mechanical ablation mechanism and to the high absorption of their wavelengths by water [80]. These effects may lead to the evaporation of the superficial layer of dentinal fluid, reducing the flow within the dentinal tubules. Due to exposure to Nd:YAG laser, dentin may be fused, solidifying into a glazed, nonporous surface [81]. Nd:YAG irradiation can also directly act at the nerve level by blocking C and Aβ fibers [82]. Sgolastra et al. [77] concluded that Er:YAG, Nd:YAG, and GaAlAs lasers appear to be efficacious in reducing dentin hypersensitivity. However, given the high heterogeneity of the included studies, future randomized controlled clinical trials are needed to confirm these results.

Recently Lin et al. [83] evaluated in-office treatments for dentin hypersensitivity in a systematic review with a network meta-analysis (Table 15.6). In this meta-analysis, articles were chosen that used evaporative air test to elicit dentin hypersensitivity. Forty studies were included. The standardized mean difference (the mean difference in each study divided by that study's standard deviation) between placebo and physical occlusion was 2.57 [95 % CI: 0.94–4.24], placebo versus chemical occlusion was 2.33 (95 % CI: 1.04–3.65), placebo versus nerve desensitization was 1.72 (95 % CI: 0.52–4.00), placebo versus laser therapy was 2.81 (95 % CI: 1.24–4.41), and placebo versus combined treatment was 3.47 (95 % CI: 5.99–0.96). The comparisons between the five active treatments showed no significant differences. Therefore, it was concluded that most active treatment options have a positive effect and show significantly better treatment outcomes than placebo treatment.

Table 15.6 Grouping of the included articles for network meta-analysis (Lin et al. [83])

Group	Group I Placebo	Group II Physical occlusion of dentinal tubules	Group III Chemical occlusion of dentinal tubules	Group IV Nerve desensitization	Group V Photobiomodulating action	Group VI Combined treatment
Treatment option	No treatment Water Not specified placebo desensitizing toothpaste	Pumice paste Sodium bicarbonate Hydroxyapatites Bioglasses Glass ionomers Dentin bonding agents Resins	Fluorides Oxalates Glutaraldehyde-based agents Calcium compounds Arginine bicarbonate calcium carbonate	Potassium nitrates Guanethidine	Laser therapy	Any combination of Groups II–V

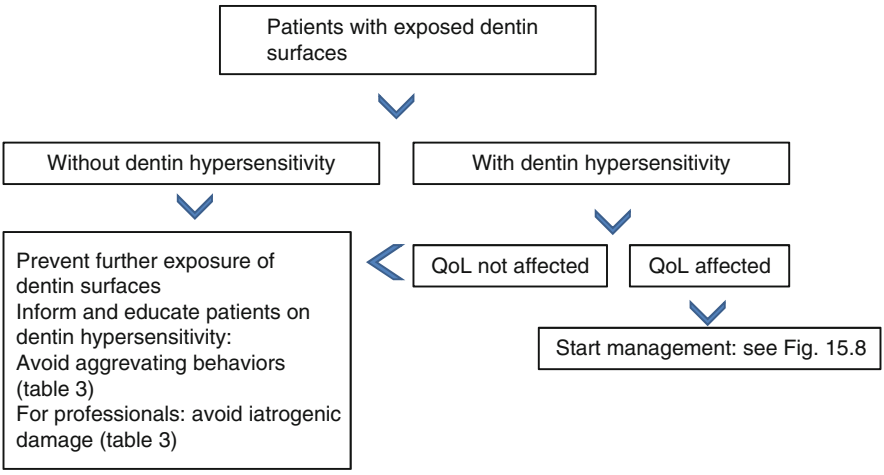


Fig. 15.7 Treatment decision tree for patients with exposed dentin surfaces (Adapted from Martens [22])

15.7 The Decision to Treat

Most of the patients who experience DHS wait to mention until the next recall visit and most of them do not specifically seek treatment for this problem, most likely because they do not view it as a significant dental health problem [35]. However, it is clearly shown that DHS can significantly be related to substantially impaired oral health–related quality of life [36, 84]. If the patient presents with exposed cervical dentin (ECD) combined with a complaint of DHS, one has to point out if this pain sensation affects the patient's quality of life (Qol). In this respect, the patient can be questioned as indicated in Table 15.2.

Figure 15.7 represents a flow diagram which can be followed for patients with exposed dentin surfaces [22]. If the patient has no dentin hypersensitivity, no treatment is required. However, a preventive strategy might be envisaged. The latter avoids further exposure of dentin surfaces and includes patient information and education, avoidance of aggravating behaviors that could induce dentin hypersensitivity, and, for the professionals, avoidance of iatrogenic damage (Table 15.3). This prevention program has also to be started in patients with dentin hypersensitivity without complaints of Qol. In addition, a desensitizing fluoride containing toothpaste may be advised. If the Qol is affected in patients with exposed dentin surfaces with dentin hypersensitivity, the treatment decision tree in Fig. 15.8 can be followed. A complete patient history especially focused on nutritional habits, oral hygiene habits, and the promoted diagnosis by exclusion has to be performed (Table 15.1). If there is no consistency between history and examination, causes other than dentin hypersensitivity must be identified and treated accordingly. If

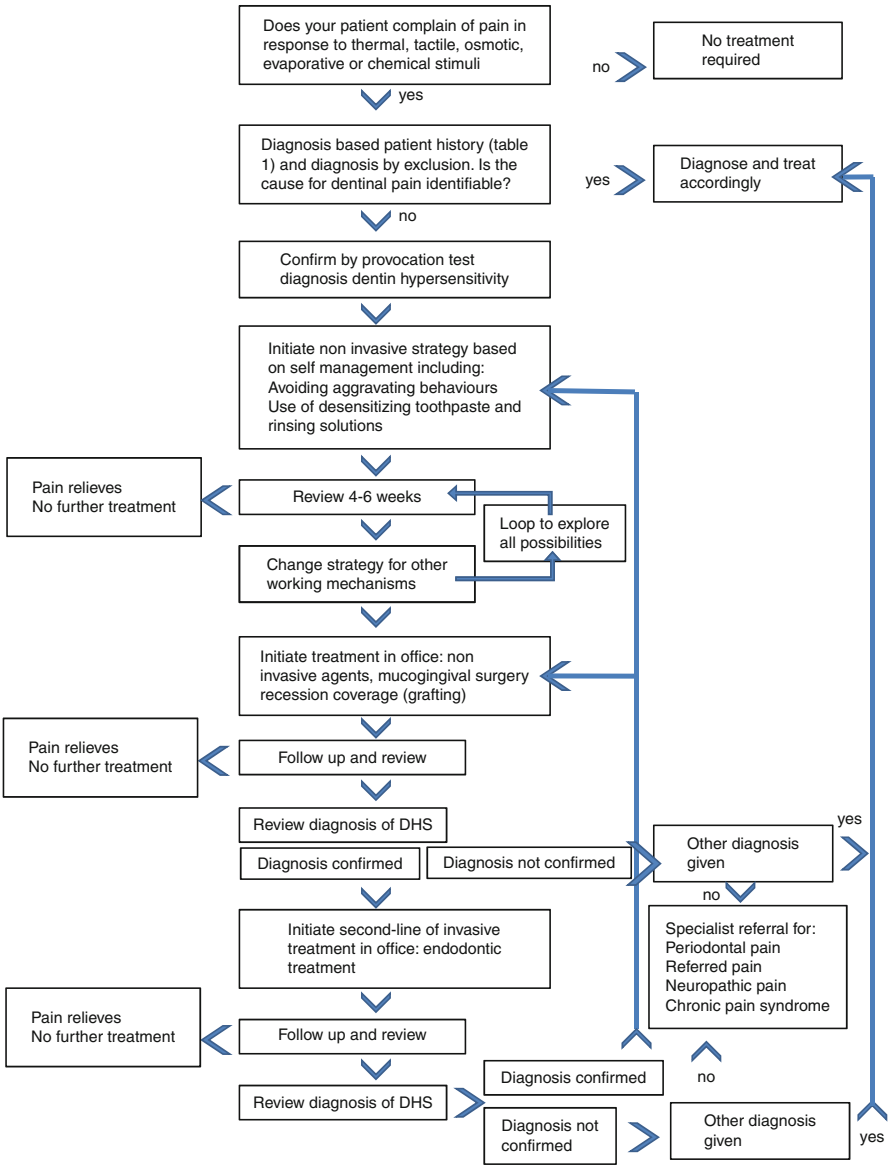


Fig. 15.8 Flow chart of the treatment strategy for dentin hypersensitivity (Adapted from Martens [22], Orchardson and Gillam [43] and the Special topic nr 6 on sensitive teeth by Colgate and Adelaide University (Adelaide University) [85])

consistency is present, management of dentin hypersensitivity must be initiated. The latter should be focused on suggestions for patients as well as for professionals (Table 15.3). Regarding the patients, dietary counseling and nonharmful oral hygiene habits are very important. This can be supported by the daily use of desensitizing toothpastes. If necessary, a less traumatic brushing method may also be introduced. While at-home treatment can be the first choice for generalized dentin hypersensitivity, when localized to one or two teeth or when immediate relief is required, practitioners may elect to use an in-office method as the first choice of treatment for dentin hypersensitivity [43]. Regarding the professionals, nonharmful professional dental care must be carried out. This must result in a well-considered choice and use of instruments and additional tools performing restorative dentistry. If, during follow-up typically 4–6 weeks, symptoms are relieved or disappeared, improving the patient's Qol, no further treatment is required. Regarding desensitizing toothpastes, two treatment approaches are well known: occluding dentinal tubules (plugging) or blocking the neural transmission to the pulp. For the occlusion of the tubules, various strategies are aimed at by the various pastes. Therefore, it is suggested if one product with a certain working mechanisms is not sufficiently effective to try a product based on another working mechanism.

If symptoms are confirmed, no pain relief present, or a further decrease of the patients' Qol is present, professional in-office treatments for DHS must be initiated. It is recommended to start with less invasive procedures first such as the use of topical fluorides and dentin bonding agents or laser therapy, which were presented in the Table 15.6. Still, all procedures can – or even should – be accompanied by the use of desensitizing toothpastes twice a day, i.e., concomitant at-home therapy as individually suggested. If treatment is carried out successfully, one should maintain and review the therapy on a regularly basis at given recall appointments.

The methods described above are indicated especially in cases with limited amounts of dental hard tissue loss, i.e., no classical abrasive or erosive defect characteristics. In cases where a class V restoration is indicated, an adhesive filling is a valid option (Fig. 15.9). Regenerative mucogingival therapy also remains an alternative, where hard and soft tissue conditions allow [86] (Fig. 15.10). A suggested strategy for dentin hypersensitivity management, taking morphological aspects into consideration, is depicted in Fig. 15.11.

If after all these treatment procedures still no pain relief can be achieved, one should start an advanced diagnosis based on exclusion before deciding to proceed to endodontic therapy, which really represents the last option of an actually failing therapy. If the diagnosis is not confirmed but revaluated, the patient should be treated accordingly. If the diagnosis is not confirmed and no other diagnosis can be given, the patient should be referred to a specialist to examine for acute periodontal infections, referred pain, neuropathic pain, or chronic pain syndrome.

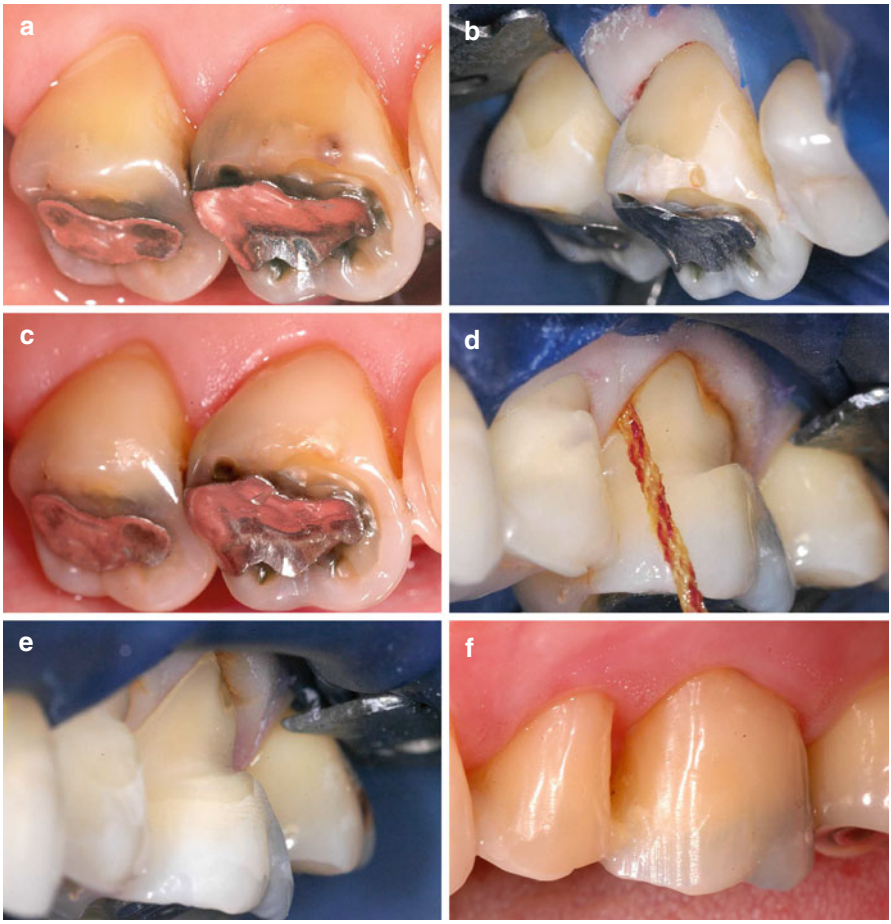


Fig. 15.9 Patient suffering from severe dentin hypersensitivity in the second quadrant (teeth 25, 26, and 27 with Schiff scores 2 and 3, respectively) and mixed defects (erosion and abrasion) at the palatal (**a**–**c**) and buccal (**d**–**f**) cervical aspects, which were treated with adhesively placed fillings. (**a**) Palatal aspect before treatment, (**b**) isolation with glued rubbed dam and retraction cords after etching, (**c**) restorations after 6 months, (**d**) buccal aspect, placement of a cord after rubber dam placement, (**e**) situation after cavity finishing and etching with phosphoric acid, and (**f**) restorations after 6 months. The pain was completely removed. Only one single aspect at tooth 25 buccally still displayed a Schiff 1 score after 6 months (Courtesy of Dr. P.R. Schmidlin)

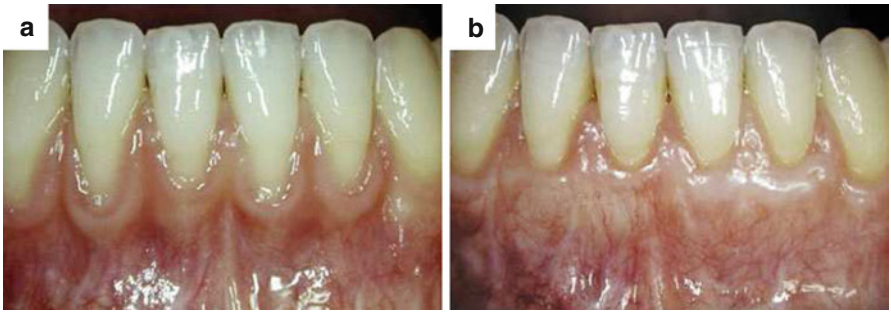


Fig. 15.10 Recession coverage using a connective tissue graft before it can be indicated, especially if the tooth substance loss is limited and the soft tissue morphology is adequate for a mucogingival approach (panel (**a**) before treatment and (**b**) 1 year after mucogingival surgery using a coronally advanced flap and connective tissue graft)

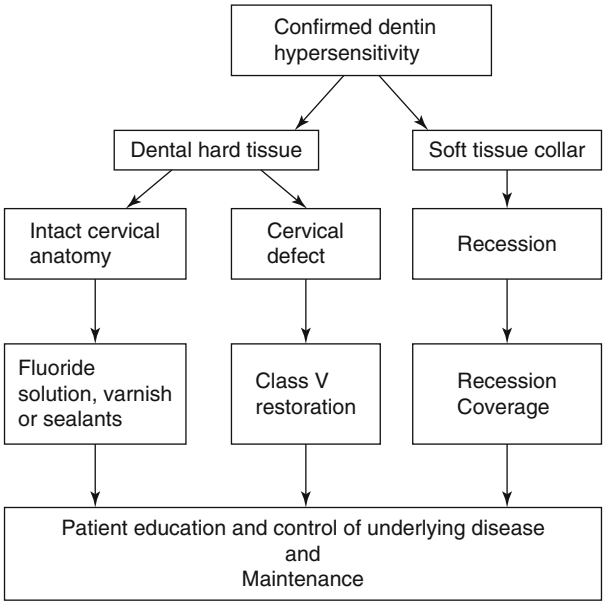


Fig. 15.11 Flow-chart of the decision-making process based on the underlying defect. Depending on the dental hard tissue damage and the morphology of the surrounding soft tissues, an adequate therapy can be initiated (Modified from Schmidlin and Sahrmann [87])

Conclusion

Dentin hypersensitivity is a problematic clinical entity that may become an increasing clinical problem for dentists to treat as a consequence of patients retaining their teeth throughout life and improved oral hygiene practices. For that, it is strongly recommended to screen routinely all dentate patients for exposed dentin surfaces and dentin hypersensitivity. In this respect, underdiagnosis of the condition will be avoided and the preventive management can be initiated early. Active management of dentin hypersensitivity usually will begin with at-home therapy of which brushing with desensitizing toothpastes is the most important. Complete management will usually involve a combination of at-home and in-office therapies.

References

1. Dowell P, Addy M. Dentine hypersensitivity – a review, aetiology, symptoms and theories of pain production. J Clin Periodontol. 1983;10(4):341–50.
2. Chabanski MB, Gillam DG. Aetiology, prevalence and clinical features of cervical dentine sensitivity. J Oral Rehabil. 1977;24(1):15–9.
3. Addy M. Etiology and clinical implications of dentine hypersensitivity. Dent Clin North Am. 1990;34(3):503–14.
4. West NX, Sanz M, Lussi A, Bartlett D, Bouchard P, Bourgeois D. Prevalence of dentine hypersensitivity and the study of associated factors; a European population-based cross-sectional study. J Dent. 2013;41(10):841–51.
5. West NX, Lussi A, Seong J, Hellwig E. Dentin hypersensitivity: pain mechanisms and aetiology of exposed cervical dentin. Clin Oral Investig. 2012;17 Suppl 1:S9–19.
6. West NX, Hughes J, Addy M. Erosion of dentine and enamel in vitro by dietary acids: the effect of temperature, acid character and concentration. J Oral Rehabil. 2000;27(10):875–80.
7. Gysi A. An attempt to explain the sensitiveness of dentin. Br J Dent Sci. 1900;43:865–8.
8. Brännström M. A hydrodynamic mechanism in the transmission of pain-produced stimuli through the dentine. In: Anderson DJ, editor. Sensory mechanisms in dentine. London: Pergamon Press; 1963. p. 73–9.
9. Vongsavan N, Matthews B. Fluid flow through cat dentine in vivo. Arch Oral Biol. 1992;37(3):175–85.
10. Addy M. Dentine hypersensitivity: new perspectives on an old problem. Int Dent J. 2002;52 Suppl 5:367–75.
11. Närhi M, Kontturi-Närhi V, Hirvonen T, Ngassapa D. Neurophysiological mechanisms of dentin hypersensitivity. Proceedings of the Finnish Dental Society. Suomen Hammaslaakariseuran Toimituksia. 1992;88(Suppl 1):15–22.
12. Adriaens PA, DeBoever JA, Loesche WJ. Bacterial invasion in root, cementum and radicular dentine of periodontally diseased teeth in humans – a reservoir of periodontopathic bacteria. J Periodontol. 1988;59(4):222–30.
13. Absi EG, Addy M, Adams D. Dentine hypersensitivity: a study of the patency of dentinal tubules in sensitive and non sensitive cervical dentine. J Clin Periodontol. 1987;14(5):280–4.
14. Rimondini L, Baroni C, Carrassi A. Ultrastructure of hypersensitive and non-sensitive dentine. A study on replica models. J Clin Periodontol. 1995;22(12):899–902.
15. Cummins D. Recent advances in dentin hypersensitivity: clinically proven treatments for instant and lasting sensitivity relief. Am J Dent. 2010;23(Spec issue A):3A–13.
16. Alexander JF, Saffir AJ, Gold W. The measurement of the effect of toothbrushes on soft tissue abrasion. J Dent Res. 1977;56(7):722–7.

17. Breitenmoser J, Mörmann W, Mühlemann HR. Damaging effects of toothbrush bristle end form on gingiva. J Periodontol. 1979;50(4):212–6.
18. Addy M, West NX. The role of toothpaste in the aetiology and treatment of dentine hypersensitivity. In: Van Loveren C, editor. Toothpastes. Monogr Oral Sci, vol. 23. Basel: Karger; 2013. p. 75–87.
19. Amaechi BT, Higham SM, Edgar WM. Influence of abrasion in clinical manifestation of human dental erosion. J Oral Rehabil. 2003;30(4):407–13.
20. Addy M, Shellis RP. Interaction between attrition, abrasion and erosion in tooth wear. In: Lussi A editor. Dental erosion from diagnosis to therapy. Monogr Oral Sci. Basel Karger. 2006;20: 17–31.
21. Canadian Advisory Board on Dentin Hypersensitivity. Consensus-based recommendations for the diagnosis and management of dentin hypersensitivity. J Can Dent Assoc. 2003;69(4):221–6.
22. Martens LC. A decision tree for the management of exposed cervical dentin(ECD) and dentin hypersensitivity (DHS). Clin Oral Investig. 2013;17 Suppl 1:S77–83.
23. Pashley DH. Smear layer: physiological considerations. Oper Dent. 1984;9 Suppl 3:13–29.
24. Absi EG, Addy M, Adams D. Dentine Hypersensitivity. The effects of toothbrushing and dietary compounds on dentine in vitro: a SEM study. J Oral Rehabil. 1992;19(2):101–10.
25. Banfield N, Addy M. Dentine hypersensitivity: development and evaluation of a model in situ to study tubule patency. J Clin Periodontol. 2004;31(5):325–35.
26. Absi EG, Addy M, Adams D. Dentine hypersensitivity: uptake of toothpastes onto dentine and effects of brushing, washing and dietary acid. J Oral Rehabil. 1995;22(3):175–82.
27. Moore C, Addy M. Wear of dentine in vitro by toothpaste abrasives and detergents alone and combined. J Clin Periodontol. 2005;32(12):1242–6.
28. Curro FA, Hays RD, Stewart B, Masters JG. Clinical effects of increasing toothpaste abrasivity on tooth hypersensitivity. J Dent Res. 2008;87(Spec issue):(Abstr 1024).
29. Holland GR, Närhi MN, Addy M, Gangarosa L, Orchardson R. Guidelines for the design and conduct of clinical trials on dentine hypersensitivity. J Clin Periodontol. 1997;24(11):808–13.
30. Schiff T, Dotson M, Cohen S, De Vizio W, McCool J, Volpe A. Efficacy of a dentifrice containing potassium nitrate, soluble pyrophosphate, PVM/MA copolymer, and sodium fluoride on dentinal hypersensitivity: a twelve week clinical study. J Clin Dent. 1994;5(Spec No):87–92.
31. Närhi MV. The characteristics of intradental sensory units and their responses to stimulation. J Dent Res. 1985;64(Spec No):564–71.
32. Orchardson R, Collins WJ. Thresholds of hypersensitive teeth to 2 forms of controlled stimulation. J Clin Periodontol. 1987;14(2):68–73.
33. Orchardson R, Collins WJ. Clinical features of hypersensitive teeth. Br Dent J. 1987; 162(7):253–6.
34. Gillam DG, Newman HN. Assessment of pain in cervical dentinal sensitivity studies. A review. J Clin Periodontol. 1993;20(6):383–94.
35. Bekes K, Hirsch C. What is known about the influence of dentine hypersensitivity on oral health-related quality of life? Clin Oral Investig. 2013;17 Suppl 1:S45–51.
36. Boiko OV, Baker SR, Gibson BJ, Locker D, Sufi F, Barlow AP, Robinson PG. Construction and validation of the quality of life measure for dentine hypersensitivity (DHEQ). J Clin Periodontol. 2010;37(11):973–80.
37. Dowell P, Addy M, Dummer P. Dentine hypersensitivity: aetiology, differential diagnosis and management. Br Dent J. 1985;158(3):92–6.
38. West NX. Dentine hypersensitivity: preventive and therapeutic approaches to treatment. Periodontol 2000. 2008;48(1):31–41.
39. Chu CH, Lam A, Lo EC. Dentin hypersensitivity and its management. Gen Dent. 2011;59(2):115–22. quiz 123–4.
40. Drisko CH. Dentine hypersensitivity: dental hygiene and periodontal considerations. Int Dent J. 2002;52:385–93.

41. Moseley JB, O'Malley K, Petersen NJ, Menke TJ, Brody BA, Kuykendall DH, Hollingsworth JC, Ashton CM, Wray NP. A controlled trial of arthroscopic surgery for osteoarthritis of the knee. N Engl J Med. 2002;347(2):81–8.
42. Huskisson EC. Simple analgesics for arthritis. Br Med J. 1974;4(5938):196–200.
43. Orchardson R, Gillam DG. Managing dentin hypersensitivity. J Am Dent Assoc. 2006;137(7):990–8.
44. Gillam DG, Seo HS, Bulman JS, Newman HN. Perceptions of dentine hypersensitivity in a general practice population. J Oral Rehabil. 1999;26(9):710–4.
45. Markowitz K, Kim S. The role of selected cations in the desensitization of intradental nerves. Proc Finn Dent Soc. 1992;88 Suppl 1:39–54.
46. Poulsen S, Errboe M, Lescay Mevil Y, Glenny AM. Potassium containing toothpastes for dentine hypersensitivity. Cochrane Database Syst Rev. 2006;19(3), CD001476.
47. Addy M, Mostafa P. Dentine hypersensitivity II. Effects produced by the uptake in vitro of toothpastes onto dentine. J Oral Rehabil. 1989;16(1):35–48.
48. Addy M, Mostafa P, Newcombe RG. Dentine hypersensitivity: a comparison of five toothpastes used during a 6-week period. Br Dent J. 1987;163(2):45–51.
49. Pearce NX, Addy M, Newcombe RG. Dentine hypersensitivity: a clinical trial to compare 2 strontium desensitizing toothpastes with a conventional fluoride toothpaste. J Periodontol. 1994;65(2):113–9.
50. Gillam DG, Newman HN, Bulman JS, Davies EH. Dentifrice abrasivity and cervical dentine hypersensitivity. Results 12 weeks following cessation of 8 weeks' supervised use. J Periodontol. 1992;63(1):7–12.
51. Markowitz K. The original desensitizers: strontium and potassium salts. J Clin Dent. 2009;20(5):145–51.
52. Ganss C, Schulze K, Schlueter N. In: Van Loveren C editor. Toothpastes. Toothpaste and erosion. Monogr Oral Sci. 2013;23:88–99.
53. Blong MA, Volding B, Thrash WJ, Jones DL. Effects of a gel containing 0.4% stannous fluoride on dentinal hypersensitivity. Dent Hyg (Chic). 1985;59(11):489–92.
54. Snyder RA, Beck FM, Horton JE. The efficacy of a 0.4% stannous fluoride gel on root surface hypersensitivity. J Dent Res. 1985;62:201. abstr 237.
55. Thrash WJ, Dodds WJ, Jones DL. The effect of stannous fluoride on dentine hypersensitivity. Int Dent J. 1994;44 Suppl 1:107–18.
56. Schiff T, He T, Sagel L, Baker RJ. Efficacy and safety of a novel stabilized stannous fluoride and sodium hexametaphosphate dentifrice for dental hypersensitivity. J Contemp Dent Pract. 2006;7(2):1–8.
57. Schiff T, Saletta L, Baker RA, Winston JL, He T. Desensitizing effect of a stabilized stannous fluoride/sodium hexametaphosphate dentifrice. Compend Contin Educ Dent. 2005;26(9 Suppl 1):35–40.
58. He T, Barker ML, Qaqish J, Sharma N. Fast onset sensitivity relief of a 0.454% stannous fluoride dentifrice. J Clin Dent. 2011;22(Spec issue 2):46–50.
59. He T, Cheng R, Biesbrock AR, Chang A, Sun L. Rapid desensitizing efficacy of a stannous-containing sodium fluoride dentifrice. J Clin Dent. 2011;22(Spec issue 2):40–5.
60. Pashley DH. Dentin permeability, dentin sensitivity and treatment through tubule occlusion. J Endod. 1986;12(10):465–74.
61. Pashley DH, Galloway SE. The effects of oxalate treatment on the smear layer of ground surfaces of human dentine. Arch Oral Biol. 1985;30(10):731–7.
62. Earl JS, Topping N, Elle J, Langford RM, Greenspan DC. Physical and chemical characterization of dentin surface following treatment with Nova Min technology. J Clin Dent. 2011;22(Spec issue 3):62–7.
63. Du Min Q, Bian Z, Jiang H, Greenspan DC, Burwell AK, Zhong J, Tai BJ. Clinical evaluation of a dentifrice containing calcium sodium phosphosilicate (NovaMin) for the treatment of dentin hypersensitivity. Am J Dent. 2008;21(4):210–4.

64. Pradeep AR, Anuj SJ. Comparison of the clinical efficacy of a dentifrice containing calcium sodium phosphosilicate with a dentifrice containing potassium nitrate and a placebo on dentinal hypersensitivity. J Periodontol. 2010;81(8):1167–73.
65. Litkowski L, Greenspan DC. A clinical study of the effect of calcium sodium phosphosilicate on dentin hypersensitivity- proof of principle. J Clin Dent. 2010;21(Spec issue 3):77–81.
66. Salian S, Thakur S, Kulkarni S, LaTorre G. A randomized controlled clinical study evaluating the efficacy of two desensitizing dentifrices. J Clin Dent. 2010;21(Spec issue 3):82–7.
67. Petrou I, Heu R, Stranick M, Lavender S, Zaidel L, Cummins D, Sullivan RJ, Hsueh C, Gimzewski JK. A breakthrough therapy for dentine hypersensitivity: how dental products containing 8% arginine and calcium carbonate work to deliver effective relief of sensitive teeth. J Clin Dent. 2009;20(Spec issue 1):23–31.
68. Lavender SA, Petrou I, Heu R, Stranick MA, Cummins D, Kilpatrick-Liverman L, Sullivan RJ, Santarpia RP. Mode of action studies on a new desensitizing dentifrice containing 8 % arginine, a high cleaning calcium carbonate system and 1450 ppm fluoride. Am J Dent. 2010;3(Spec issue A):14A–9.
69. Hamlin D, Williams KP, Delgado E, Zhang YP, DeVizio W, Mateo LR. Clinical evaluation of the efficacy of a desensitizing paste containing 8.0 % arginine and calcium carbonate for the in-office relief of dentin hypersensitivity associated with dental prophylaxis. Am J Dent. 2009;22(Spec issue A):16A–20.
70. Elias Boneta AR, Galán Salás RM, Mateo LR, Stewart B, Mello S, Arvanitidou LS, et al. Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a commercial mouthwash containing 2.4% potassium nitrate and 0.022% sodium fluoride and a control mouthwash containing 0.05% sodium fluoride on dentine hypersensitivity: a six-week randomized clinical study. J Dent. 2013;41 Suppl 1:S34–41.
71. Hu D, Stewart B, Mello S, Arvanitidou L, Panagakos F, De Vizio W, et al. Efficacy of a mouthwash containing 0.8% arginine, PVM/MA copolymer, pyrophosphates, and 0.05% sodium fluoride compared to a negative control mouthwash on dentin hypersensitivity reduction. A randomized clinical trial. J Dent. 2013;41 Suppl 1:S26–33.
72. Vano M, Derchi G, Barone A, Covani U. Effectiveness of nano-hydroxyapatite toothpaste in reducing dentin hypersensitivity: a double-blind randomized controlled trial. Quintessence Int. 2014;45(8):703–11.
73. Orchardsen R, Gillam D. Managing dentin hypersensitivity. J Am Dent Assoc. 2006;137(7):990–7.
74. Thanatvarakorn O, Nakashima S, Sadr A, Prasansuttiporn T, Ikeda M, Tagami J. In vitro evaluation of dentinal hydraulic conductance and tubule sealing by a novel calcium-phosphate desensitizer. J Biomed Mater Res B Appl Biomater. 2013;101(2):303–9.
75. Endo H, Kawamoto R, Takahashi F, Takenaka H, Yoshida F, Nojiri K, Takamizawa T, Miyazaki M. Evaluation of a calcium phosphate desensitizer using an ultrasonic device. Dent Mater J. 2013;32(3):456–61.
76. Gandolfi MG, Silvia F, H PD, Gasparotto G, Carlo P. Calcium silicate coating derived from Portland cement as treatment for hypersensitive dentine. J Dent. 2008;36(8):565–78.
77. Sgolastra F, Petrucci A, Severino M, Gatto R, Monaco A. Lasers for the treatment of dentin hypersensitivity: a meta-analysis. J Dent Res. 2013;92(6):492–9.
78. Ladalardo TC, Pinheiro A, Campos RA, Brugnera Júnior A, Zanin F, Albernaz PL, Weckx LL. Laser therapy in the treatment of dentine hypersensitivity. Braz Dent J. 2004;15(2): 144–50.
79. Sgolastra F, Petrucci A, Gatto R, Monaco A. Effectiveness of laser in dentinal hypersensitivity treatment: a systematic review. J Endod. 2011;37(3):297–303.
80. Yilmaz HG, Kurtulmus-Yilmaz S, Cengiz E, Bayindir H, Aykac Y. Clinical evaluation of Er, Cr:YSGG and GaAlAs laser therapy for treating dentine hypersensitivity: a randomized controlled clinical trial. J Dent. 2011;39(3):249–54.

81. Birang R, Poursamimi J, Gutknecht N, Lampert F, Mir M. Comparative evaluation of the effects of Nd:YAG and Er:YAG laser in dentin hypersensitivity treatment. Lasers Med Sci. 2001;22(1):21–4.
82. Orchardson R, Peacock JM, Whitters CJ. Effect of pulsed NdYAG laser irradiation on action potential conduction in isolated mammalian spinal nerves. Lasers Surg Med. 1997;21(2):142–8.
83. Lin P-Y, Cheng Y-W, Chu C-Y, Chien K-L, Lin C-P, Tu Y-K. In-office treatment for dentin hypersensitivity: a systematic review and network meta-analysis. J Clin Periodontol. 2013;40(1):53–64.
84. Bekes K, John MT, Schaller HG, Hirsch C. Oral health-related quality of life in patients seeking care for dentin hypersensitivity. J Oral Rehabil. 2009;36(1):45–51.
85. Adelaide University special topic nr 6. Dentine hypersensitivity—is it an erstwhile problem or a modern day enigma? https://www.adelaide.edu.au/…/hypersensitivity/Sensit.
86. Chambrone L, Pannuti CM, Tu YK, Chambrone LA. Evidence-based periodontal plastic surgery. II. An individual data meta-analysis for evaluating factors in achieving complete root coverage. J Periodontol. 2012;83(4):477–90.
87. Schmidlin PR, Sahrmann P. Current management of dentin hypersensitivity. Clin Oral Investig. 2013;17 Suppl 1:S55–9.

Recall, Maintenance Care Cycle, and Outcomes Assessment

16

Bennett T. Amaechi

Abstract

Failure to review and monitor the patient may result to relapse of condition; therefore, in erosion risk management, it is essential that a maintenance care regime (recalls) matched to each patient's disease levels and erosion risk status is established, to check patient compliance, monitor wear, reinforce advice, and for encouragement to maintain changed behavior. This chapter describes the importance of recall visits, the establishment of recall intervals, and the activities required to be performed at every recall visits, including evaluation of the outcomes of the treatment with regard to disease control, quality of care, health maintenance, and improvement in patient's quality of life. Through recall visits, a cyclic risk-based maintenance care is established for each patient, with the objective to establish and maintain long-term oral health.

16.1 Introduction

A continuing care regime matched to the patient's erosion risk status must be established through review and monitoring visits (conventionally referred to as recalls). The mission of recalls is to establish a cyclic risk-based maintenance care for each patient, which would provide and maintain long-term oral health for the patient through disease control, quality care, health maintenance, and improved quality of life. Thus, in every recall visit the practitioner should;

B.T. Amaechi, BDS, MS, PhD
Department of Comprehensive Dentistry, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229-3900, USA
e-mail: amaechi@uthscsa.edu

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7_16

- Re-assess and monitor the progression status of a previously diagnosed and treated erosion lesion.
- Detect any newly developed lesions
- Assess and monitor patient compliance with previous advice and treatment
- Reassess and reinforce the appropriateness of previous advice
- Encourage maintenance of behavioral changes that will improve and maintain their oral and general health
- Consider altering the treatment regimen for a more favorable outcome

Accomplishment of the above activities in a recall visit would prevent disease, reduce morbidity associated with oral and dental disease, and improve the patient's quality of life.

The management of a patient's risk of dental erosion may fall into the hands of both dental and medical professionals, necessitating the patient to be visiting two clinics for recalls. A patient suffering from dental erosion due to medical conditions such as gastroesophageal reflux disease (GERD) or eating disorder, and referred to a medical professional, would have recalls established by the medical office as well as the dental office that manage the oral effect of the patient's medical condition (dental erosion).

16.2 Determining the Recall Interval

With regard to the erosion risk management by the dental office, unless the patient's only dental problem is erosion, the recall visit to review and monitor the dental erosion may be scheduled to coincide with that of the patient's other oral disease risk management (e.g., caries management). In line with the recommendation of the United Kingdom National Institute of Health and Clinical Excellence (NICE) guidance that the individual patient's disease risk status should inform his/her recall interval [1], Young et al. [2] in Chap. 7 of this book recommend that the duration of the personalized intervals between visits to review and monitor a patient's dental erosion status (recall interval) should be adjusted based upon the erosion severity levels and his/her erosion risk status. The assessment of erosion levels and the patient's risk of erosion should be performed during the initial comprehensive history taking and clinical oral examination. Thus, an important step in determining the recall interval is choosing the tools for assessing and monitoring both the erosion risk status of the patient and the severity levels of the erosive wear lesions over time.

Although there is not yet a quantitative objective clinical method of assessing the erosion severity levels, at the moment this can be accomplished with the silicone matrix [3, 4] described in Chap. 6 of this book, which is an objective method of monitoring the change in size and depth of an erosive wear. This can be used to monitor if the lesion is progressing or has become arrested. The Basic Erosive Wear Examination (BEWE) proposed by Bartlett et al. [5] can also be used. BEWE is a scoring system in which the grading of the severity of a lesion is based on its extent on the tooth surface on a four grade level (Table 16.1). There are other assessment

Table 16.1 Criteria for grading erosive wear, from Bartlett et al. [5]

Score	
0	No erosive tooth wear
1	Initial loss of surface texture
2	Distinct defect, hard tissue loss <50 % of the surface area
3	Hard tissue loss ≥50 % of the surface area

and monitoring methods that can be used [6]; however, the practitioner should ensure that the same assessment method used at the initial assessment should be used for reassessment at subsequent review visits to enable comparison of lesion activity and progression status.

The erosion risk status can be determined with the Dental erosive wear risk assessment (DEWRA) form (Table 16.2) developed by Young et al. [2] and described in detail in Chap. 7 of this book [2]. With DEWRA, the patient's responses to more in-depth history-taking regarding dietary and oral hygiene habits, social and lifestyle habits, general health conditions, together with a detailed clinical recording of erosive wear lesions using the BEWE or other scoring system, guide the practitioner to assign the patient to low, medium, or high risk status categories. The risk status of the patient guides the practitioner's recommendations for the management of the condition, which includes the recall interval for review and monitor. The DEWRA guidance indicates that the frequency range for recall could be as high as once every 6 months for patients at high risk of developing erosive tooth wear, to a low of once every 2 years for those at low risk of developing erosion. Thus the DEWRA recommends that patient with erosive tooth wear should be reviewed and monitored as follows:

- Low risk – every 18–24 months
- Moderate risk – every 6–12 months
- High risk – every 6 months

It is important to mention that the same risk assessment tool used for the initial risk status assessment should be used for reassessment at each subsequent recall visit. The above recommended recall interval accords with that of the BEWE, which depends on the severity level of the lesion. With BEWE, the dentition is divided into sextants and the most severely affected surface in a sextant is recorded. The sum of the scores of the six sextants constitutes the index value, which guides the management of the condition, including the recall interval (Table 16.3).

In general, recommended interval between oral health reviews should be determined specifically for each patient and tailored to meet his or her needs. Besides patient's erosion severity levels and erosion risk status, recall intervals may be influenced by whether it is for review and monitor of overall risk management, for assessing preventive interventions, for monitoring of the progression status of existing erosion lesions, or for reviews of behavioral and lifestyle modification plans. Interval should be set with the clinical judgment and expertise of the dental team. The recall interval should be chosen either on completion of a specific treatment journey or at

Table 16.2 Dental erosive wear risk assessment (DEWRA) form [2]

Dental erosive wear risk assessment form (DEWRA)				
Patient name:	**Birth date:** day/month/year		**Age:**____	**Dentist initials:**
Date: day/month/year	**Low risk (L)**	**Moderate risk (M)**	**High risk (H)**	**Patient Risk (L, M, H)**
Dietary and oral hygiene habits				
Acidic foods (e.g. citrus fruits, apples, sour pickles, salad dressing, sour candies, vit. C fizzy tablets, Chinese candies, Mexican candies, tomato ketchup, sour add-on, etc)	Infrequent consumption. Mainly confined to meal times		Frequent or prolonged between meal consumption.	
Acidic drinks (e.g. fruit juices, soft drinks, squash, flavoured water, sports drinks, energy drinks, herb teas, wine, alcopops, etc)	Infrequent consumption. Mainly confined to meal times. Use of a straw		Frequent between-meal consumption. Rinsing/sipping drinking habit	
Toothbrushing (i.e. toothbrush type, brushing frequency, toothpaste type)	Soft toothbrush and correct brushing technique	Hard toothbrush. High brushing frequency. Incorrect brushing technique. Abrasive toothpaste		
Mouth rinsing (F-mouthrinses)	Regular rinsing with F-mouthrinses	No additional F-exposure than F-toothpaste		
Social and life-style habits				
Alcohol or recreational drugs (e.g. wine, alcopops, narcotics, cocaine, ecstasy, designer drugs)	Occasional user	Regular user		
Occupation/hobbies/exercise	No obvious risk activities	High level of strenuous exercise	Prolonged acidic work environment	
General health conditions				
General medical conditions associated with reduced salivary function (e.g. Sjögrens, rheumatoid arthritis)	No		Yes	
Radiation therapy in head/neck area	No		Yes	
History of vomiting or gastroesophageal reflux disease (GERD)	No	Acidic taste in mouth upon wakening	Yes	
Medications that may affect salivary secretion or oral acid exposure (e.g. hyposalivary medications, sphincter-relaxing medications, acidic medications)	No	Yes		
Clinical conditions				
Dental erosive wear lesions (description of degree of erosive wear)	Erosive wear confined to enamel	Erosive wear - cuppings into dentin	Extensive erosive wear in dentin	
Saliva status (Saliva flow: normal unstimulated = 0.25-0.35 ml/min, hyposalivation <0.1 ml/min; normal stimulated = 1-3 ml/min, hyposalivation <0.7 ml/min Saliva pH: normal stimulated = 7.2-7.5)	Normal saliva secretion levels. Good saliva buffer capacity	Temporary low saliva secretion. Low buffer capacity	Chronic low saliva secretion or hyposalivation. Low buffer capacity	
Comment/summary:			**Overall risk status:** (L, M, H)	

the end of an oral health review if no further treatment is indicated [7]. The dentist should discuss the recommended recall interval with the patient and the patient's agreement or disagreement with it should be sought. This discussion should involve consideration to the patient's ability or desire to visit the dentist at the recommended interval, the financial costs to the patient of having such review visits and any subsequent treatments, and other possible impediments to the patient's compliance to this review interval [8]. It is important to inform the patient that his/her recall interval may change from time to time subject to change in his/her erosion risk status.

Table 16.3 Complexity levels as a guide to clinical management, from Bartlett et al. [5]

Complexity level	Cumulative score of all sextants	Management
0	Less than or equal to 2[a]	• Routine maintenance and observation • Repeat at 3-year intervals
1	Between 3 and 8[a]	• Oral hygiene and dietary assessment, advice, routine maintenance, and observation. • Repeat at 2-year intervals
2	Between 9 and 13[a]	• Oral hygiene and dietary assessment, advice, identify the main aetiological factor(s) for tissue loss, and develop strategies to eliminate respective impacts • Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces • Ideally, avoid the placement of restorations and monitor erosive wear with study casts, photographs, or silicone impressions • Repeat at 6–12 month intervals
3	14 and over[a]	• Oral hygiene and dietary assessment, advice, identify the main aetiological factor(s) for tissue loss, and develop strategies to eliminate respective impacts • Consider fluoridation measures or other strategies to increase the resistance of tooth surfaces • Ideally, avoid restorations and monitor tooth wear with study casts, photographs, or silicone impressions • Especially in cases of severe progression consider special care that may involve restorations • Repeat at 6–12 month intervals

[a]The cut-off values are based on experience and studies of one of the authors (A. L.) and have to be reconsidered

16.3 Activities at the Recall Visit

The activities that the practitioner has to perform at the next oral health review for his/her patient suffering from erosive tooth wear should consist of review, monitor, synthesis, and health outcomes analysis as described below.

16.3.1 Review

At every recall visits, the patient's compliance with the behavior modifications recommended and discussed in regards to the erosion risk management plan should be evaluated. The behavioral modification counseling at each review visit should continue to be offered by motivational interviewing. Motivational interviewing is a patient-centered method for enhancing intrinsic motivation to change by exploring and resolving ambivalence. Individuals assess their own behaviors, present arguments for change, and decide what behavior to focus on while the counselor helps to create an acceptable resolution that triggers change [9–11]. The goals of the

behavior modification should be revisited, considered, and discussed motivationally as necessary. Besides evaluating the status of recommended behavioral changes, patient's perception of value of oral health and treatment choices should be assessed. It may be necessary for the patient to receive an individualized letter of advice, listing the newly designed behavior modification goals for the patient to take home. Future behavior goals may need to be developed and recorded at this stage.

16.3.2 Monitor

Erosive tooth wear may progress in severity by increasing in size and depth or may remain unchanged (arrested) over time. The available methods for monitoring the progression status of previously detected and treated or untreated erosive wear are discussed in detail in Chap. 6 of this book [3]. Clinical assessment of lesions should be performed at every recall visit. Are the lesions progressing or have the lesions become arrested? At the moment, the best monitoring method for answering this question is the Silicone Matrix [3, 4]. The lesion status at each visit should be compared with that recorded at the previous treatment appointments. All teeth/surfaces should be examined and assessed for new lesion development using the same erosive wear assessment method that was used at the initial assessment appointment such as BEWE, Silicone matrix, or any other erosive wear assessment method used. Previous treatments, such as protected eroded surfaces should be evaluated for possible alteration of treatment choice for a more favorable outcome.

16.3.3 Synthesis

At every recall visit, the erosion risk status of the patient should be reassessed using the same assessment tool, DEWRA, which was used at the initial comprehensive assessment appointment. Based on the outcome of the reassessment, the erosion risk level of the patient may be readjusted if necessary. Based on the outcomes of the review and monitor, the goals of the behavioral modification may be modified as necessary. The final activity at the recall visit would be to determine the next recall interval. The recall interval range should be reconsidered and either modified or remain the same, based on the findings of the review and monitoring. The review and monitor should advice the practitioner of the suitability and appropriateness of previously recommended intervals.

16.3.4 Health Outcomes Analysis

Every care plan is focused on achieving health outcomes for the patient. Analysis of the health outcomes should be performed for the first time on

completion of the planned treatments and then at every recall visit. The analysis of the outcomes should facilitate feedback on the success of care to the patient, the practitioner, and a third-party payer. The health outcomes achieved from a particular treatment journey can be learned from the patient's responses to the oral care provided. Dental erosion risk management plan should be designed to achieve health promotion, disease control, and patient empowerment through improvement of patient's quality of life and provision of care of quality that is satisfactory to the patient. Thus, health outcomes evaluation should address questions such as: Is the disease controlled? Is the quality of care satisfactory? Can the patient maintain his or her health? Has the quality of life of the patient improved?

With regard to disease control, active erosion should be arrested and eroded surfaces should be protected from further acid erosion. Severely worn surfaces should be restored to the patient's satisfaction. There should be no new erosive lesion developing in either sound or treated surfaces. For erosion caused by medical conditions such as GERD or eating disorders, the conditions should be under control and the recommended preventive and protective goals met. Evaluation of behavioral modification outcomes should indicate if the dietary goals as well as the review and recall goals have been met. Overall, patient should be capable of maintaining oral health and well-being.

Erosive tooth wear at advanced stage may be associated with dentin hypersensitivity (DHS) and esthetic problem to the patient. Both conditions are known to substantially impair patient's oral health-related quality of life [12, 13]. With regard to this, one has to point out at the initial examination appointment if the DHS pain affects the patient's quality of life (QoL). The QoL can be evaluated with such questions as indicated in Table 16.4. Such OHIP scale should be able to indicate if the patient's QoL has been improved by the treatment and behavioral modification goals.

Table 16.4 The items of the dentin hypersensitivity experience questionnaire developed by Boiko et al. [12] to determine the impact of dentin hypersensitivity on a patient's quality of life

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Restrictions	Pleasure out of eating					
	Cannot finish meal					
	Longer to finish meal					
	Problems with eating ice-cream					

(continued)

Table 16.4 (continued)

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Adaptation	Modification of eating					
	Careful when breathing					
	Warming food/ drinks					
	Cooling food/drink					
	Cutting fruit					
	Putting a scarf over mouth					
	Avoiding cold drinks/foods					
	Avoiding hot drinks/ foods					
	Avoiding contact with certain teeth					
	Change toothbrushing habits					
	Biting in small pieces					
	Avoiding other food					
Social	Longer than other to finish					
	Choose food with others					
	Hide the way of eating					
	Unable to take part in conversation					
	Painful at the dentist					
Emotions	Frustrated not finding a cure					
	Anxious of eating contributes					
	Irritating sensations					
	Annoyed with myself for contributing					
	Guilty for contributing					
	Annoying sensations					
	Embarrassing sensations					
	Anxious because of sensations					

(continued)

Table 16.4 (continued)

		1 disagree strongly	2 agree a little	3 agree	4 agree moderately	5 agree strongly
Identity	Difficult to accept					
	Different from others					
	Makes me feel old					
	Makes me feel damaged					
	Makes me feel unhealthy					

References

1. National Institute for Clinical Excellence (NICE): guide on dental recall: recall interval between routine dental examinations. Clinical guideline 19. London. 2004. www.nice.org.uk/CG019. NICE guideline. Accessed March 2009.
2. Young A, Stenhagen KR, Mulic A, Amaechi BT. Dental erosive wear risk assessment. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 121–32.
3. Amaechi BT. Assessment and monitoring of dental erosion. In: Amaechi BT, editor. Dental erosion and its clinical management. Berlin: Springer; 2015. p. 111–9.
4. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005;33:243–52.
5. Bartlett D, Ganss C, Lussi A. Basic Erosive Wear Examination (BEWE): a new scoring system for scientific and clinical needs. Clin Oral Investig. 2008;12:S65–8.
6. Bardsley PF. The evolution of tooth wear indices. Clin Oral Investig. 2008;12:S15–9.
7. Clarkson JE, Amaechi BT, Ngo H, Bonetti D. Recall, reassessment and monitoring. In: Pitts N, editor. Detection, assessment, diagnosis and monitoring of caries. Basel: Karger; 2009. p. 188–98.
8. Gibson BJ, Drennan J, Hanna S, Freeman R. An exploratory qualitative study examining the social and psychological processes involved in regular dental attendance. J Public Health Dent. 2000;60:5–11.
9. Gao X, Man Lo EC, Kot CCS, Chan WKC. Motivational interviewing in improving oral health: a systematic review of randomized controlled trials. J Periodontol. 2014;85:426–37.
10. Resnicow K, McMaster F. Motivational Interviewing moving from why to how with autonomy support. Int J Behav Nutr Phys Act. 2012;9:19.
11. Lundahl B, Burke BL. The effectiveness and applicability of motivational interviewing: a practice-friendly review of four meta-analyses. J Clin Psychol. 2009;65:1232–45.
12. Boiko OV, Baker SR, Gibson BJ, Locker D, Sufi F, Barlow AP, Robinson PG. Construction and validation of the quality of life measure for dentine hypersensitivity (DHEQ). J Clin Periodontol. 2010;37(11):973–80.
13. Bekes K, John MT, Schaller HG, Hirsch C. Oral health-related quality of life in patients seeking care for dentin hypersensitivity. J Oral Rehabil. 2009;36(1):45–51.

Index

A
Abfraction
  lesions, 100
  mechanical flexure theory, 253
  NCCLs, 251
  noncarious cervical lesions, 103
  wedge-shaped defects, 106
Abrasive toothpastes, 86–88, 129, 135
Acidic candies, 82–83, 176
Acidulated phosphate fluoride (APF) gels
  concentration, 157
  pH, 158
  prevention and control, 156–157
  professional application, 157
Anorexia nervosa
  binge eating, 43
  compensatory behavior, 43
  physical features, 37
  prevalence rate, 187
  psychopathologies, 37
  self-induced vomiting habits, 57, 58
  slimming foods, 37
Anterior tooth wear
  composite resin restorations, 238–241
  Dahl appliance, 237
  mandibular teeth, 239, 241
  metal alloy palatal veneers, 236
  palatal tooth wear, 235–236
  porcelain lamination, 237, 239
  restoration, anterior teeth, 236–238
APF gels. *See* Acidulated phosphate fluoride (APF) gels
Assessment and longitudinal monitoring
  acid demineralization, 112
  BEWE, 114–116
  dexterity, 117
  light microscope, 112
  macro camera systems, 117
  serial impression casts, 117–118
  severity levels, 111
  silicone matrix, 113–114
  TWI, 115–117
  ultrasound system, 114
Asthmatic medications, 84–85

B
Basic erosive wear examination (BEWE)
  dental erosion, 9
  dentition, 5
  and DEWRA, 126
  grading, 107, 114, 115, 304
  lingual/palatal tooth surfaces, 7
  Olympic Games, 9
  prevalence score, in Europe, 7
  recording/monitoring, 122
  severity levels, 115, 116
  sextants, dentition, 114–115
  silicone matrix, 308
  TWI, 5, 115
Behavioral/lifestyle factors
  acidic fruits and vegetables, 90
  dietary items, 89–90
  extrinsic erosion, 89
  oral hygiene, 90–91
  protective factors, 89
  swallowing, 90
Behavior modification program
  beverage intake, 135
  calcium/polymer, 137
  intrinsic acids, 136
  oral hygiene method, 135–136
  personal protective equipment, 136
  saliva stimulation, 136–137
Beverages
  adolescents, 72
  animal models, 76
  buffering capacity, 76
  calcium and phosphate ions, 78–79
  chemical properties, 78, 79

B.T. Amaechi (ed.), *Dental Erosion and Its Clinical Management*,
DOI 10.1007/978-3-319-13993-7

Beverages (*cont.*)
clinical evidence, 73–74
CPP-ACP, 80
dietary substances, 75
etiological factors, 74
fluorides, 80–81
and foods, 72, 73
hydroxyapatite and fluorapatite, 78
modifications, taste, 81
polyphosphates, 81
screening tests, 78
severity, 75, 76
side effects, 79
supplementation, 79, 80
taste/flavor, 77
US health problems, 73
water, 73
BEWE. *See* Basic erosive wear examination (BEWE)
Biocorrosion
dentin, 251
enamel, 252
lesion development, 251
maxillary anterior teeth, 256
noncarious lesions, 251
Bite-guards, 136, 165
Body mass index (BMI), 189, 207
Bulimia nervosa
binge eating, 43
compensatory behavior, 43
enzymatic activity, 43–44
occurrence and severity, 44
prevalence rate, 187–188
psychopathologies, 37
purging methods, 43
signs and symptoms, 37, 43
tooth hypersensitivity, 57–59

C

Calcium, remineralization methods
CPP-ACP technology, 159–160
hypersensitivity, 160
TCP technology, 160
Casein phosphopeptide-stabilized amorphous calcium phosphate (CPP-ACP)
calcium and phosphate ions, 140
enamel surface, 80
laboratorial testing, 80
MI Varnish application, 160
prevention and control, 156
professional application, 159
Recaldent technology, 159–160
remineralization, 140
Chewing gums, 83, 137, 198, 220, 285
Cognitive-behavioral therapy (CBT), 196–197
CPP-ACP. *See* Casein phosphopeptide-stabilized amorphous calcium phosphate (CPP-ACP)
Cyclic vomiting syndrome, 45

D

Dahl appliance, 229
Demineralisation, dental erosion
buffer capacity, 19
chelation, 19–20
hydrochloric and sulphuric acids, 18
hydroxyapatite, 18
intrinsic/extrinsic acids, 17–18
ionic complexes, 19
pH values, 18–19
tartaric acid, 18–19
titratable acidity, 19
Dental erosion process
acidic products, 23, 24
acids and demineralisation, 17–20
advanced stage, 4
calcium and phosphate, 25
chelation, 25
crystal growth, 29
dental tissues, 14–15
early signs, 4
epidemiology, 5–6
erosive wear, 14
extrinsic cause, 4
fluorapatite, 24
intrinsic cause, 5
lesions, occlusion, 9
maxillary anterior teeth, 9
mechanical wear processes, 14
microhardness/profilometry, 22, 23
occlusal surfaces, 14
oral soft tissues/diet, 29
palatal, 5
pH and buffer capacity, 24
physical factors, 25
potential erosion, 23
prevalence (*see* Prevalence, dental erosion)
salivary pellicle, 14
salivary proteins, 29
soft drink consumption, 4–5
tooth surface, 20–22
undersaturation degree, 23
Dental erosive wear risk assessment (DEWRA)
clinical examination, 122
form, 122, 124–126, 305, 306

patient's responses, 305
risk factors, 131
Dental office management
acid reflux, 221
erosion risk management, 304
medication therapy, 221
patient self-care, 220
Dental pain scale (DPS), 281
Dental tissues
crystal morphology, 14
dentine, 15
enamel, 15
mechanical properties, 14
Dentine hypersensitivity (DHS)
arthritis, 285
decision-making process, 295, 297
dental office, 290–292
DPS, 281
ECD, 293
endodontic therapy, 295
erosive tooth wear, 277, 309
gingival recession, 276, 278, 279
hard tissues, 279–280
home therapy, 285, 286
hydrodynamic mechanisms, 277
iatrogenic damage, 293
inflammation, 277, 278
nerve desensitization, 285, 287, 288
network meta-analysis, 292, 295
OHRQoL, 282
open tubules, 278
pain intensity measurement, 281
pain provocation test, 280–281
patients and dental professionals, 284, 285, 295
placebo effect, 285
regenerative mucogingival therapy, 295, 297
root/crown, 276
sensitivity, 276
streaming potential, 277
treatment decision tree, 293, 294
tubules (*see* Tubule occlusion)
DEWRA. *See* Dental erosive wear risk assessment (DEWRA)
DHS. *See* Dentine hypersensitivity (DHS)
Dietary management
behavior modification program, 176
dental erosion, 170, 176
food factors, 171–175
habits, 176
hard tissue disorder, 170
health care, 171–173, 177–178
non-erosive foods, 177, 178
nutrient analysis, 176
risk assessment, 170
Diets
acidic candies, 82–83
acidic foods, 81
anorexia nervosa, 37
behavioural factors, 29
beverages consumption (*see* Beages)
chewing gums, 83
wear risk factors, 128
Differential diagnosis
abfraction, 103, 104
abrasion, 102, 103
amorphous centripetal loss, 100
anamnesis and nutrition diaries, 104
attrition, 102
BEWE, 107
complexity levels, clinical management, 107, 108
enamel-cementum junction, 101, 102
human ailment, 109
occlusal erosion, 101, 103, 105
optical coherence tomography, 108
physical impacts, 100
physiological process, 109
smooth surfaces, 103, 106
tissue loss, 104, 106–108
ultrasound measurements, 108
DPS. *See* Dental Pain Scale (DPS)

E
Ear, nose, throat (ENT) symptoms, 210
Eating attitudes test (EAT), 195
Eating disorders
anorexia (*see* Anorexia nervosa)
behavioural factors, 29
binge eating, 188
bulimia (*see* Bulimia nervosa)
dental health, 189–190
epidemiological studies, 37–42
intrinsic acids, 136
long-term effects, 189
osteoporosis, 186
psychopathology, 186
recurrent vomiting disorders, 36–37
remissions/relapses, 189
rumination, 44
saliva, 130
sensitivity, 188
social stigma, 44
tooth sensitivity, 186
ECD. *See* Exposed cervical dentin (ECD)
EGD. *See* Esophagogastroduodenoscopy (EGD)

Environmental (occupational) Factors
competitive swimmers, 89
industrial workers, 88
professional wine tasters, 88–89
Epidemiology, GERD
acid reflux, 205
children and adults, 204
heartburn symptoms, 204–205
Montreal consensus, 204
pH monitoring, 205
Erosive wear
attrition/abrasion surfaces, 28
behavioural factors, 29
dentine, 28
enamel, 27
noncarious cervical lesions, 28
perikymata, 28
Erosive wear risk status. *See also* Dental erosive wear risk assessment (DEWRA)
alcohol and recreational drugs, 129
BEWE (*see* Basic Erosive Wear Examination (BEWE))
dietary hygiene habits, 128–129
gastric acids, 130
medical and dental history, 126
occupation, hobbies and exercise, 129–130
oral hygiene habits, 129
patient letter, 126, 128
preventive treatment plan guides, 126, 127
protective factors, 126
recording/monitoring, 122
saliva status, 130–131
screening, 122, 123
vomiting/reflux, 130
Esophageal impedance testing, 214, 216
Esophagogastroduodenoscopy (EGD), 214
Exercises, 29, 90, 129–130, 170, 188
Exposed cervical dentin (ECD), 293
Extrinsic factors
analgesics, 83–84
asthmatic medications, 84–85
behavioral/lifestyle, 89–91
dietary habits (*see* Diets)
environmental (occupational), 88–89
vitamins, 84

F

Fluorides, remineralization methods
gels and foams
APF gels, 157–158
directions, 157
SnF2, 158
stannous products, 158
traditional prophylaxis, 155, 157
prevention and control, 156–157
varnishes, 158–159
Fluoride varnishes
MI Varnish, 159
physical presence, 154, 158
prevention and control, 156–157
professional application, 158
TCP, 160
Food, dietary management
eating patterns, 174
Frequency Questionnaire, 172–175, 177
habits, 173
portion sizes, 173–175
quantity, 173
recall instructions, 171, 177
records, 172, 177
risk assessment, 171

G

Gastroesophageal reflux disease (GERD)
acid-suppressive medications, 213
alarm signs, 209, 210
antireflux barriers, 206
asthma, 210
bulimia, 207
dental erosion, 204
epidemiology, 204–205
extraesophageal manifestations, 210
gastric factors, 206
heartburn/pyrosis, 209
hiatal hernia, 206
lower esophageal sphincter, 46–47
mandibular molar, 211, 212
maxillary and mandibular teeth, 58–60
maxillary premolars, 211, 212
nocturnal reflux, 47
obesity, 207
oral cavity, 204
pH testing, 216
radiographic imaging, 214
refluxate, gastric acid, 47
retrograde flow, 205
saliva parameters, 56
signs and symptoms, 47
tooth wear, 47–55
treatment response, 213
upper incisors, 211, 212
viral respiratory infection, 210
Gastroparesis, 206, 210, 219

GERD. *See* Gastroesophageal reflux disease (GERD)
GERD pathogenesis
demineralization, 209
esophageal clearance, 207
fluorapatite dissolution, 209
hard and soft tissues, 208
hydroxyapatite crystals, 208
peristalsis, 207–208
saliva, 207–208
Gerd questionnaire (GerdQ), 213, 214

H
Health professionals, psychology
alcoholism, 193
antecedent, 195
anxiety, 195
consequent behavior, 195–196
dentin hypersensitivity, 192
depression, 193, 195
eating disorders, 193–195
maintenance, 193
maladaptive behavior, 192, 195, 196
precontemplation stage, 192
rapport building techniques, 191–192
stigmatization, 190
treatments, 192
Histamine-2 receptor blocker (H_2RB)
diagnostic testing, 214
gastric acid, 217
medication therapy, 217
PPIs, 217
reflux esophagitis, 217

I
Intrinsic factors
gastric juice, 35–36
GERD (*see* Gastroesophageal reflux disease (GERD))
hydrochloric acid, 36
prevention and treatment, 60
recurrent vomiting disorders (*see* Vomiting disorders)

L
Lower esophageal sphincter (LES)
dysfunction, 206
gastroesophageal junction, 206
muscle tissue, 206
obesity, 207
reflux disease, 47

M
Maximum root coverage (MRC), 270, 271
Medical office management
acid-suppressive medications, 216
lifestyle modifications, 216–217
medication therapy, 217–219
non-medication treatment, 216, 217
surgical therapy, 219
Medication therapy
antacids, 217
cell acid secretion, 218
esomeprazole therapy, 219
H_2RB, 217
optical coherence tomography, 219
PPIs, 217
prokinetic agents, 219
rebound acid hypersecretion, 219
Mouthrinses
acidulation-fluoridation, 142–143
erosion/abrasion, 144, 145
and low pH Toothpastes, 85–86
oral care products, 85–86
polyvalent metal fluorides, 144
protease inhibitors, 145
MRC. *See* Maximum root coage (MRC)

N
Nanohydroxyapatite (nHAP), 290
Noncarious cervical lesions (NCCLs)
abfraction, 251, 253–254
abrasion, 251, 253
biocorrosion, 251–253
buffer acidity, 257
coronal tooth structure, 255
dentin desensitization, 257–258
dietary counseling, 256
flaws and contradictions, 250
gastric contents, 256
maxillary incisors, 250
occlusal attrition and abrasion, 249
oral hygiene techniques, 251, 257
piezoelectric effects, 254–255
potential mechanisms, 251
restoration (*see* Restoration, NCCLs)
shallow depressions, 249–250

O
OHRQoL. *See* Oral health-related quality of life (OHRQoL)
Oral care products
abrasive toothpaste, 87
cotton swabs, 86

Oral care products (*cont.*)
dental bleaching, 87–88
low pH toothpastes, 85–86
mouthrinses, 85–86
saliva substitutes, 86–87
toothbrushing, 87
Oral health education, 165
Oral health-related quality of life (OHRQoL), 282, 293, 309

P

Patient self-care
advance erosion, 134
attrition, 134
behavioural modification, 135–137
bulimic practice, 134
chitosan, 142
CPP-ACP, 140
dental office visit, 134
eating disorders, 134
erosion/abrasion, 142, 143
extrinsic acids, 134
fluoride concentration dentifrices, 139
mouthrinses, 142–145
neutralizing intraoral acidity, 137
novamin technology, 141
organic polymers, 141
polyvalent metal fluorides, 139, 140
salivary pellicle, 134
TCP, 140–141
Piezoelectricity, 251, 254–255
Porcelain-fused-to-metal (PFM), 227
Posterior tooth wear
composite resin technology, 244
dentition, 239
gold alloy restoration, 240
minimal preparation, 242
occlusal tooth reduction, 242
temporization, 244
worn maxillary dentition, 242, 243
PPIs. *See* Proton pump inhibitors (PPIs)
Prevalence, dental erosion
geographic regions
China, 8
Europe, 6–7
United States, 7–8
GERD, 8
oral health problems, 8–9
risk, 6
working environment, 8–9
Preventive management
acid attack, 153
ions concentrations, 154
pellicle/biofilm, 152–153
prophylactic barriers, 154
remineralization repairs, 153
Prophylactic coatings. *See* Tooth surface protection
Proton pump inhibitors (PPIs)
gastric pH, 218
GERD, 213
H_2RB therapy, 218
medical therapy, 214, 217
nocturnal acid control, 219
rebound acid hypersecretion, 219
Psychogenic vomiting syndrome, 45
Psychological management
adolescents/adults, 185
anxiety, 183
CBT techniques, 196–197
depression, 183–184
de-stigmatization, mental health, 198
eating disorders, 186–190
endocrine system, 182
gastric acids, 182
hard tissue disorder, 182
health professionals, 190–196
nonbacterial origin, 182
obsessive-compulsive disorders, 183
operant conditioning, 185
oral health education, 197–199
stress, 182–183
tooth extraction, 185

R

Recaldent technology. *See* Casein phosphopeptide-stabilized amorphous calcium phosphate (CPP-ACP)
Recall visits, dental erosion
behavior modification, 307–308
BEWE, 304
complexity levels, 305, 307
DEWRA, 305, 306
erosion risk management, 304, 308
GERD, 304
grading erosive wear, 304–305
health outcomes analysis, 308–311
motivational interviewing, 307
oral health reviews, 305
risk-based maintenance care, 303
silicone matrix, 308

Remineralization methods
calcium, 159–160
fluorides (*see* Fluorides, remineralization methods)
gels and foams form, 154–155
in saliva, 155
varnishes, usage, 155
Resin-modified glass-ionomer cement (RMGIC)
and GIC, 161
and NCCL, 267
tooth preparation, 263
Restoration, NCCLs
adhesive systems, 261, 262
esthetic harmony, 269
etch and rinse (ER) systems, 260, 261
gingival recession, 268
giomers, 262
glass ionomers and resin composites, 260, 261
gold foil/amalgam, 259
insertion techniques, 266–269
isodry dental, 263
micromechanical retention, 259
Miller classification system, 269–270
MRC, 270, 271
mucogingival flap, 264, 265
polyalkenoic acid, 261
resin adhesives, 259–260
resin bonding systems, 260
retraction cord sizes, 263, 264
RMGIC, 262–263
sandwich technique, 262
sclerotic dentin formation, 258–259
self-etch (SE) systems, 260, 261
tooth preparation, 264, 266
RMGIC. *See* Resin-modified glass-ionomer cement (RMGIC)

S
Saliva
chewing gums, 83
dilution and buffering effects, 26
dry mouth, causes of, 130
enamel, 26–27
enamel pellicle formation, 130
flow, measurement, 131
glycoproteins and lipids, 26
hyposalivation, 46
lactic acid diffusion, 26
liquid erosion, 25
parameters, in GERD patients, 56
pellicle, 26
proteins change, 26
secretion values, in adults, 131
stimulation, 136–137
Sjögrens syndrome, 126, 130
Sodium lauryl sulphate (SLS), 287
Stannous fluoride (SnF_2)
acid attack, protection, 158
amine fluoride (AmF), 142
enamel protection, 139
prevention and control, 156–157
Sympathetic nervous system (SNS), 182

T
TCP. *See* Tricalcium phosphate (TCP)
Tooth surface protection
acid diffuses, 20
bite-guards, 165
chemical bond, 162
dentine, 21–22
enamel, 20–21
fluoride, 162
hypersensitivity, 162, 163
remineralizing potential, 163–165
resins, 163
RMGIC, 161
surface sealants, 161
tubules, 161
Tooth wear index (TWI)
grading erosion, 115, 116
maxillary incisors, 8
soft drink consumption, 7
upper and lower arches, 115, 117
Tricalcium phosphate (TCP)
fluoride interactions, 141
functionalization, 140–141
remineralization, 160
Tubule occlusion
acid erosion, 288
arginine, 290
bioactive glass, 289
dentine fluid dynamics, 290
DHS, 288
nHAP, 290
SEM image, 287, 289
SLS, 287
soluble oxalate salts, 289
stannous fluoride gel, 288, 289
strontium chloride, 288
TWI. *See* Tooth wear index (TWI)

V
Visual analogue scale (VAS), 281
Vomiting disorders
  alcoholism, 45–46
  biocorrosion, 252
  bite guards, 197
  cyclic vomiting syndrome, 45
  drugs, side effect, 45
  eating disorders, 37–44
  medical conditions, 45
  pregnancy inducement, 46
  psychogenic vomiting syndrome, 45

W
Worn dentition
  adhesive materials, 233
  anterior restorations, 229, 230
  cervical tooth wear, 233–234
  Dahl appliance, 229
  dental management, 225
  occlusal vertical dimension, 230
  onlay/overlay prosthesis
    acrylic resin, 230–232
    clenching/grinding habits, 231, 232
    moderate/severe tooth wear, 230, 231
    root surface, 231, 232
    tooth wear, 230
  orthodontic tooth movement, 228, 229
  periodontal surgery, 228
  PFM, 227
  pulp necrosis, 227
  restorative treatment options, 227
  tooth preparation, 227

X
Xerostomia, 43, 44, 86