Michel Goldberg
Editor

Understanding Dental Caries

From Pathogenesis to Prevention and Therapy

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ISBN 978-3-319-30550-9 ISBN 978-3-319-30552-3 (eBook)
DOI 10.1007/978-3-319-30552-3

Library of Congress Control Number: 2016941182

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG Switzerland

Preface

Dental and periodontal diseases are two major public health pathologies. Teeth injuries include enamel and dentin carious lesions and periodontal disorders. Carious lesions are the most widespread dental pathologies. They may be limited to simple occlusal fissures or located solely in the proximal aspect, or they expand to complex class II and/or cervical lesions.

In the United States, over 50 % of 5–9-year-old children have at least one cavity and/or one restoration of decayed teeth [(D) and/or filled (F)]. That proportion increases to 78 % among 17-year-old children. The World Health Organization (WHO) estimation of global DMFT [decayed, missing, filled teeth] for 12-year-old children has reported that in the 188 countries included in their database, on a global basis, about 240 millions of teeth are injured in this age group [*Bagramian RA*, *Garcia-Godoy F*, *Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent 2009*; *22*: *3–8*]. These evaluations underline the significance of dental caries and its correlation with dental practice. In contrast, severe periodontitis was limited to only 5–20 % of the adult population.

Younger and older patients are the targets of dental carious decay, well recognized as a major health problem in most industrialized countries, affecting 60–90 % of school-aged children and the vast majority of adults (*Petersen et al*., *The global burden of oral diseases and risks to oral health. Bulletin of the WHO 2005*; *83*: *661–669*). Patients with three DMFT constitute 51 % of the patients at the age of 12, while the other patients displayed higher values. Therefore, most dental practitioners are implicated in their everyday practice by the treatment of dental caries.

Clearly, this implies also that carious lesion is more than likely the most prominent pathology of the mouth, and the importance of carious lesions is fundamental both for patients and for dental practitioners. This underlines also the significance of understanding dental caries, their pathogenesis, prevention, and subsequent therapies.

The present book focuses most exclusively on the carious lesion, going from the initial pathogenesis of the lesion, mild enamel alteration, to deep dentin lesions, which appear as a major pathology with pulpal irreversible incidences. The therapies and prevention of the enamel decay are analyzed in the first part of this book.

After a brief description concentrating on the structure and epidemiology of the diverse forms of enamel alterations, carious lesions are reported. We describe successively enamel softening and analyze the etching pattern of

acidic effects on enamel. Doing so, we moved from the superficial etching to the initial enamel carious lesion. Bacterial films and acidic biofilms of the dental plaque lead to the formation of active and/or inactive lesions. The methods used for an accurate diagnosis of the carious lesion were improved during the past few years, and a specific chapter concludes the first part of the book, by reporting new diagnostic methods.

Another group of chapters is devoted to the carious dentin and to active or inactive lesions, superficial or deep, reaching the dental pulp and/or located exclusively in the cervical region. How the patient brush and eliminate the dental plaque is another topic. Which toothpastes are used, the evolution, and/or the stabilization of the lesion are factors involved in the carious progression. Eventually, non-carious cervical lesions may regenerate, and it is well documented that some cervical pathology remineralize spontaneously. In addition, we note that 8–13 % among adult patients are affected by the increasing problem of dental erosions.

Finally, new trends in resin infiltrations of the initial lesion, minimal invasive therapies aiming to stabilize the carious lesion, and strategies devised to prevent the expansion of the lesion are control and preventive measures restraining the broad field of cariology.

Fluoride is considered to be the main tool of carious prevention. At some high doses, it induces pathologic fluorosis, but if added in minimal quantity in controlled assays, different forms of fluoride prevents the evolution of carious lesions, contributing to remineralization of the initial lesion or to their stabilization. Other preventive therapies have been elaborate, and we focused on the differences that appear between prevention and minimal restorative dentistry.

The different chapters of this book were written as requested by different researchers and clinicians recognized to be the best in their specific domain. We wish sincerely to acknowledge their outstanding contributions, and I wish to thank them warmly for what they did for the dental community.

Paris, France
January 2016

Michel Goldberg

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Part I

The Carious Enamel

Understanding Dental Caries – from Pathogenesis to Prevention and Therapy

1

Nigel Pitts

Abstract

This chapter provides an overview demonstrating the pivotal importance of understanding the caries process in enamel (and then, for a subset of lesions which ever progress, beyond that, into the dentine) if we are to best prevent and control dental caries over the life course in both patients and populations. There is a need also to understand the complexities and opportunities in the detection, assessment and diagnostic steps in order to inform decision-making and effective, personalised care planning. Modern caries care, provided at the right times on the basis of caries risk, should ensure that the disease is controlled and that tooth structure is preserved whenever possible.

1.1 The Carious Enamel

1–1 *Dental caries*: *structure*, *diagnosis*, *which treatment is appropriate*.

1.2 Introduction

This chapter provides an overview demonstrating the pivotal importance of understanding the caries process in enamel (and then, for a subset of lesions which ever progress, beyond that, into the dentine) if we are to best prevent and control dental caries over the life course in both patients and populations. It sets the scene for the detail that follows in subsequent chapters. Aside from appreciating the science around enamel structure which underpins caries prevention and control, there is also a need also to understand the complexities and opportunities in the detection, assessment and diagnostic steps employed by clinicians examining their patients. Rather than just finding "holes to fill", dentists of today need to carefully assess tooth sites and assess both lesion severity and activity. This is in order to inform their decision-making in selecting from an ever-widening choice of preventive and minimally invasive care options. The goal is to achieve effective, personalised and risk-based care planning with a long-term perspective. Modern caries care, provided at the right times on the basis of caries risk, should ensure caries is controlled and

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M. Goldberg (ed.), *Understanding Dental Caries*, DOI 10.1007/978-3-319-30552-3_1

that tooth structure is preserved whenever possible. The era of "automatic" decisions to restore all lesions detected should now be past, but change can be frustratingly slow in many countries and systems.

1.3 Structure

One important element of understanding the healthy structure of enamel as well as carious enamel is to appreciate the long-term clinical value of the sound tissue. In terms of coping with a lifetime of thermal, chemical and physical challenges in a hostile wet environment, tooth enamel is still superior in its physical properties to any of the currently available artificial alternatives. This reality underpins the guiding philosophy to protect and preserve as much natural tooth structure and specifically enamel as possible. Even though we have at our disposal the clinical technology to cut enamel very rapidly and efficiently, this should only be done as a last resort (Pitts 2004a, b; Ismail et al 2013).

The following series of chapters describe and illustrate the unique, highly organised structure of the enamel, the hardest mineralised tissue in the human body. This varies from the important superficial aprismatic surface enamel, which matures for a period following eruption into the oral environment, to the deeper bulk of the prismatic enamel – which is comprised of prismatic enamel (carbonated hydroxyapatite) and interprismatic enamel.

Knowledge of the sound structure is important in understanding the differing initial mechanisms of the caries process (producing net sub-surface demineralisation after long alternating periods of de- and remineralisation) as opposed to the direct surface loss of tissue which is associated with erosive tooth wear. In caries, there is a critical interplay between the superficial structure of the enamel and the covering complex biofilm which, when cariogenic, can shift ecologically to produce and retain a low pH at the tooth surface (see Chapter I-4- by Phil Marsh). It is this interplay and the periods of repeated de- and remineralisation that give rise to the macroscopically intact "surface zone" of the initial caries lesion. Preserving this zone has tremendous clinical value in maintaining the potential to arrest and reverse lesions.

1.4 Diagnosis

The evidence-based consensus statements from the ICW-CCT, a major international consensus workshop on caries clinical trials (Pitts and Stamm 2004), clearly differentiated three important and different elements and terms related to caries diagnosis. These were caries: *detection*, *assessment and diagnosis*.

Detection is concerned with the clinical decision as to whether a tissue is classified as sound or carious (and may record degrees of caries severity, which can be staged), *assessment* characterises the behaviour of a lesion (i.e. is this lesion *active* now or is it *arrested*), and finally *diagnosis* represents the human summation by the clinician of all information available (from clinical visual and radiographic assessments as well as from any other special tests which may be employed) to decide the disease status of a particular tooth surface (Pitts and Stamm 2004; Longbottom et al 2009).

The wide variation in the way in which the terminology around caries diagnosis was used in the literature, and specifically the ambiguity associated with many of the different criteria used and reporting for caries clinical trials and research, gave rise after this meeting to the creation in 2002 of a harmonised system built, on integrating the best evidence. This is the "ICDAS", an *I*nternational *C*aries *D*etection and *A*ssessment *S*ystem (www.icdas.org/). This system was developed to facilitate caries epidemiology, research and appropriate clinical management (Pitts 2004a, b; Ismail et al 2007) and has been developed over the years into a number of approved options. The system has also been incorporated into a number of international conventions and recommendations from bodies such as the FDI World Dental Federation (Fisher and Glick 2012) and the American Dental Association (ADA 2015).

The ICDAS criteria were built around the evidence relating the clinical visual appearance of the enamel (and dentine) to the histological extent of the lesion within the tooth tissue. Critically, the very first clinical visual changes in the enamel can only be seen when the tooth is clean and has been dried with compressed air, ideally for 5 s. White spot lesions visible only when dry extend less far into the enamel than those visible when the enamel is wet with saliva, due to changes in the refractive index masking the initial lesion. In the latter case, the lesion may extend across the full thickness of the enamel, and defence reaction changes may be visible in the underlying dentine. It is therefore important to examine teeth which are clean and dry and to use sharp eyes, rather than sharp probes (which have been found many years ago to cause iatrogenic damage whilst not contributing significantly more diagnostic information than visual-alone examination).

Whilst clinical examination is the foundation for assessing caries, for teeth in anatomical contact, there remain fundamental limitations in assessing approximal caries and also in understanding the depth of penetration of some occlusal lesions. For these reasons dental radiography, and particularly the bitewing projection, has become ubiquitous in many countries as part of a routine examination. The use of radiography in addition to clinical visual examination provides the most comprehensive clinical picture of caries status (Pitts and Kidd 1992a; b).

Although there have been some debates and concerns about using ionising radiation too frequently in low caries populations (particularly in Scandinavia), the evidence supporting the use and clinical utility of properly timed radiographs for planning both preventive and operative care remains. The International Caries Detection and Assessment System (ICDAS), which classifies carious lesions, has been developed into a comprehensive International Caries Classification and Management System (ICCMS™). This includes methods for staging of the caries process which combines the findings from both radiographic and visual examinations (Pitts and Ekstrand 2013). This helps dentists manage caries most effectively in their patients when assessed for caries risk. It should be further appreciated that there are a range of other method and technologies which have been developed to act diagnostic aids to clinicians. These are considered in a forthcoming chapter (Chapter I-5- by Neuhaus and Lussi, 2016).

In terms of assessing enamel caries, the *depth of penetration* through the enamel is an important consideration. This information helps determine what kind of treatments and care strategies may be required and are appropriate for each lesion. It also helps in the monitoring of lesion development and, conversely, in establishing the degree of success obtained when seeking to stop further progression of a lesion. The so-called diagnostic threshold (or cut point) at which caries is recognised as disease has dramatic affects at both the individual patient level and at the population health level (Selwitz et al. 2007). There is a need for more clarity and consistency on this issue, both internationally and across dental "silos".

Caries *activity* information is prized highly by both clinicians and researchers, but despite this there are evidence gaps and surprisingly few systems currently available to help dentists with this type of caries assessment task (ICDAS website; Pitts 2011). New systems, such as using bioluminescence in order to identify increases in free calcium associated with actively demineralising lesions, show promise for future clinical practice.

1.5 Which Treatment Is Appropriate (When)

The importance (and for many years the neglect of) considering the decision-making process in treatment planning has come to the fore over the last decade. Rather than simple no-drill vs. drill (binary decisions), clinicians today have to consider a wide range of factors at the population, patient, tooth and service levels.

The context for making treatment decisions in caries care is also changing. Factors which are operating in the background include international agreements to phase down the use of dental

amalgam on environmental grounds and to phase up prevention (United Nations Environment Programme 2013), as well as the widespread desire for preventive interventions to link oral and systemic health improvements with the so-called NCDs. A current example is a systematic re-examination of evidence around the role of sugar in caries and the desire to link caries improvements to those in diabetes, obesity and heart disease (Moynihan and Skelly 2014). This has led to new WHO Guidelines on Sugar Intake for Adults and Children (World Health Organisation 2015).

A further influence on decision-making is an increasing awareness of the need to try to reduce health inequalities in caries (Pitts et al. 2011a, b) and, therefore, to consider caries risk by population subgroups. These various threads have led to a renewed interest in international public health advocacy for caries prevention and control, through groupings such as the Alliance for a Cavity-Free Future (www.allianceforacavity-freefuture.org/), and also a parallel push for social movements in caries prevention which links with public/private collaborations (Bonecker et al. 2012).

There has been a focus in recent years on achieving consensus on exactly what a graduating dentist should know about caries. The elements across pathogenesis, prevention and therapy are key parts of this knowledge and skill set. Europe has led the way with a core cariology curriculum which was built by research and education organisations working together to build consensus (Schulte et al. 2011). This process also generated specific guidance on caries risk assessment, diagnosis and the synthesis of all information into a care plan (Pitts et al. 2011a, b) which established that nonoperative and surgical treatments should be deemed to have equal "value", but the surgical treatment should only be used as a last resort. This European resource material has been debated, fine-tuned and localised in a range of territories including Colombia (Martignon et al. 2014), Malaysia and most recently the USA (Fontana et al. 2016).

Since 2010 the ICDAS Foundation (a registered charity) has been working internationally to build the International Caries Classification and Management System, *ICCMS™*. This is a health outcomes focused system that aims to maintain health and preserve tooth structure, by using a simple form of the ICDAS caries severity and activity *classification* model in order to derive an appropriate, personalised, preventively based, risk-adjusted and tooth-preserving *management* plan for each patient (Pitts NB et al. on behalf of the participating authors of the International Caries Classification and Management System (ICCMS™) Implementation Workshop, 2013). An international consensus workshop held at Kings College London in 2013 laid the groundwork for the incremental development of this guide for practitioners and educators. The guide is available in a number of formats and languages, including a short quick reference guide, from the ICDAS website ((www.icdas.org/). The relationships between ICDAS, ICCMS and the more recent Global Collaboratory for Caries Management – GCCM are shown in Fig. 1.1.

The best evidence for deciding which treatment is appropriate and when has been assembled by a group of 75 international academics and practitioners in a cyclical format with four main elements which together lead to beneficial patient outcomes (see Fig. 1.2). These elements are:

1. HISTORY – which provides a patient-level caries risk assessment
2. CLASSIFICATION – caries staging and activity assessment
3. DECISION-MAKING – both synthesis and diagnoses
4. MANAGEMENT – personalised caries prevention, control and tooth-preserving operative care

Leading to OUTCOMES assessed in the domains of health maintenance, disease control, patient-centred quality metrics and wider impacts on systems and society.

Important features of the ICCMS™ are that it is risk based. For some years there have been groups advocating the CAMBRA philosophy (Caries Management by Risk Assessment), and

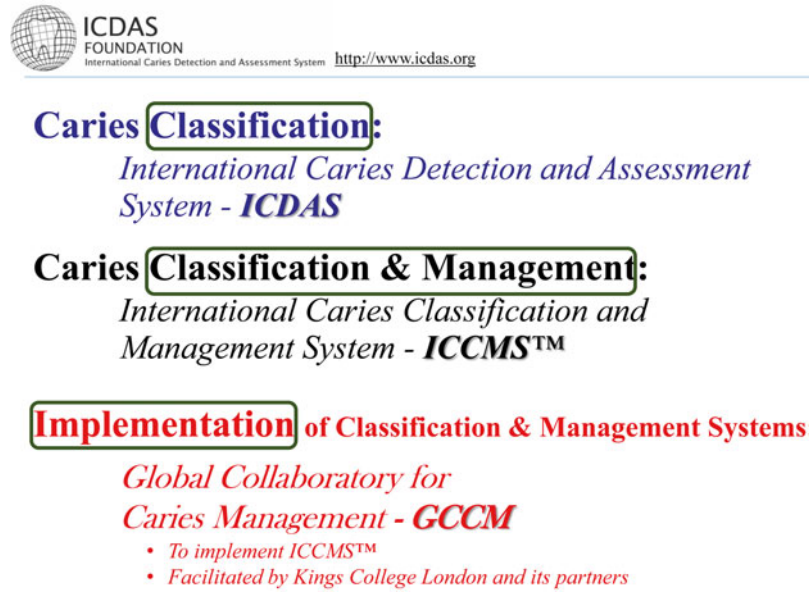

Fig. 1.1 Interrelationships and definitions of *ICDAS* and *ICCMS* and *GCCM*

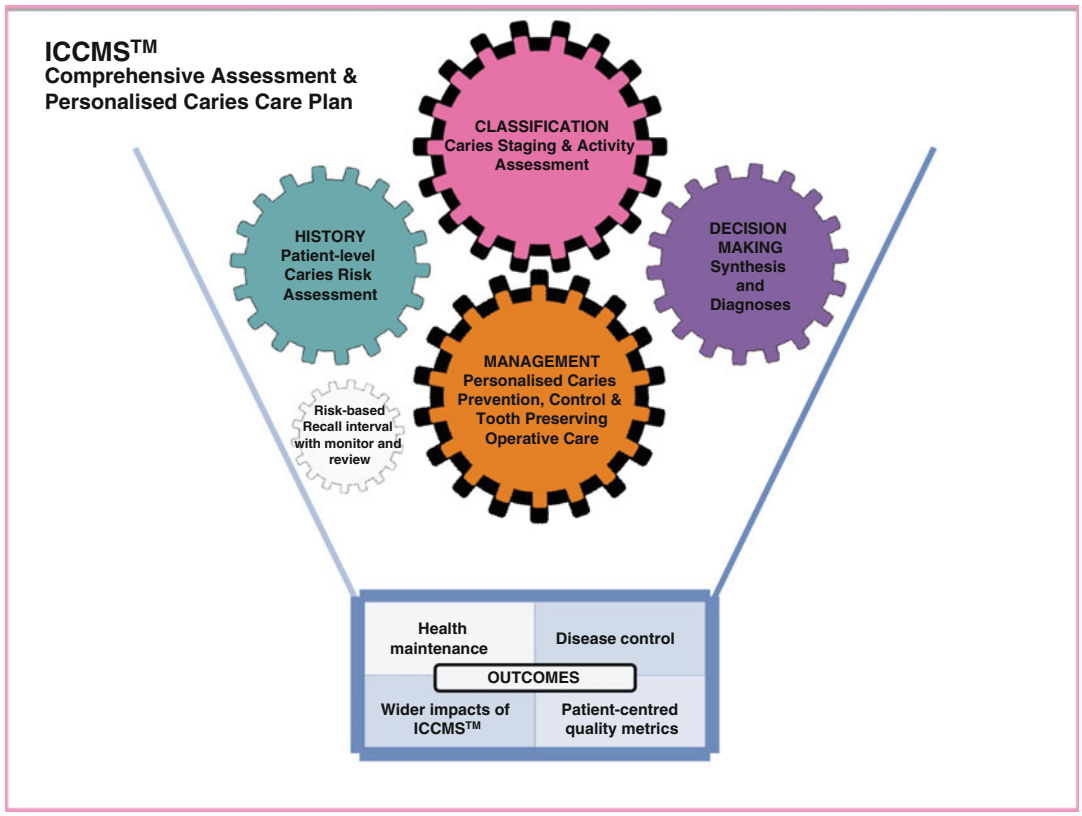

Fig. 1.2 ICCMS™: Overview of the comprehensive assessment and personalised caries care plan

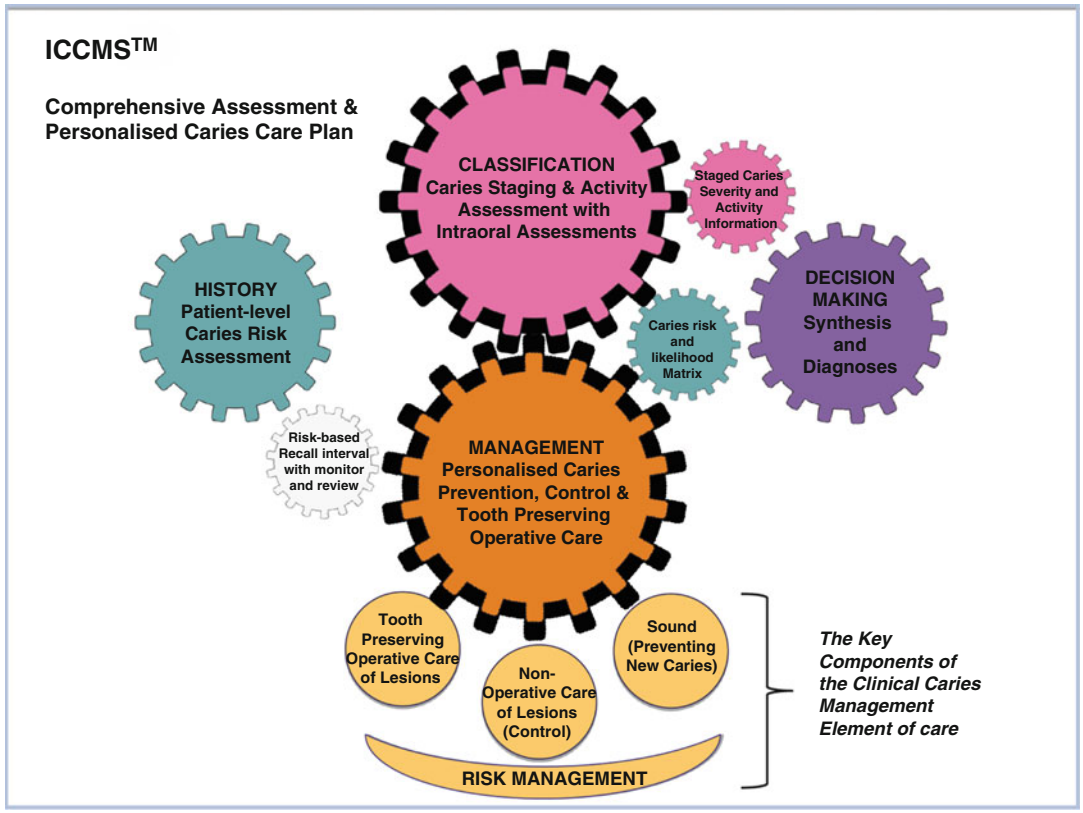

Fig. 1.3 ICCMS™ – Detail showing the key components of the clinical caries management element of care

in terms of planning appropriate recall strategies for patients, the UK National Health Service's National Institute for Clinical Excellence (NICE) convened a guideline development group to look at the evidence in this area. They recommended that for dental recall, the interval between routine dental examinations should be personalised and risk based (NICE 2004). For caries management this means monitoring and review is required.

Figure 1.3 shows the key components of the clinical caries management element of care. These are all underpinned by risk management strategies and comprise (a) preventing new caries for sound surfaces; (b) nonoperative care of lesions, that is, caries control using remineralisation strategies including fluorides and newer methods; and (c) as a last resort – tooth-preserving operative care of lesions that cannot be arrested or reversed or are to extensive when a patient is first seen.

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Enamel Softening (Dental Erosion)

2

Michel Goldberg

Abstract

For many years, most of the published reports of enamel softening dealt with consequences of acid or chelator etching. It occurs without bacterial involvement. The 2–5 nm thick outer enamel surface layer is mainly concerned. Limited crystallite dissolution is due to abrasion, attrition, abfraction, or erosion. More than likely, enamel dissolution is due to acidic demineralization. At early stages, it is a reversible process. Preventive strategies include dietary counseling, stimulation of salivary flow, optimization of fluoride regimens, modification of erosive beverages, and adequate oral hygiene measures.

2.1 Introductory Remarks

For many years, most of the published reports were dealing with three well-identified enamel-induced pathologies, which have been specifically studied and compared. They included three groups of major studies studying *enamel softening* getting worse and becoming a more severe *acid or chelator etching* and ultimately being converted into *carious lesions*. The etiopathologic complexity of the early enamel lesions was increased, whereas a parallel decreased of carious lesions was noted.

Dental enamel is the most highly mineralized structure of the skeleton. The percentage of inorganic components of enamel is greater than in bone and dentin. It is the actual target of specifically defined aggressions directed toward the enamel structure. Due to the presence of an outer aprismatic layer, dental erosions, attritions, abfractions, and abrasion became the most prominent lesions during the past decades. Such phenomena are obviously in continuity with enamel softening. After an acid attack, or following the action of chelators, the presence of a prismatic enamel layer is determinant for an etching pattern, beneath the dental plaque, which is responsible for the progression of carious lesions within enamel. The cascade of enamel decays leads from one type of superficial alteration to more severe deterioration.

The comparison between the three enamel alterations provides a better understanding of the carious process, the specific topic of this book.

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M. Goldberg (ed.), *Understanding Dental Caries*, DOI 10.1007/978-3-319-30552-3_2

Hence, analysis of enamel softening opens gates on enamel etching and shed lights on the early stages of the enamel carious lesion.

2.2 Dental Erosion

Tooth wear or dental erosive lesion involves multifactorial causes. There is an interplay between chemical, biological, and behavioral factors. Erosion as a pathology entity results from the *chemical loss of dental hard tissue by an acid, without bacterial involvement*. Attrition (physical wear through tooth-tooth contact) and abrasion (physical wear produced by the interaction between a tooth surface and another material) are two other well-recognized factors. Dental erosion is exclusively a surface phenomenon, whereas in contrast, the initial carious lesion implicates both surface and subsurface modifications.

Erosion is due to structural feature of the tooth, physiological properties of saliva, and intrinsic and extrinsic acidic sources (Lussi et al. 2011). The critical pH below which enamel dissolves is about 5.5. Erosion starts by initial softening of the enamel surface, followed by a loss of volume with a softened layer persisting at the surface of the remaining tissue. Exposure to acids combined with insufficient salivary flow results in enhanced dissolution. Enamel tends to dissolve more slowly than dentin.

During the initial stage of dental erosion, the loss of tooth hard tissue affects up to 80 % of the adults and ~ 50 % of the children (more recent evaluations indicate a prevalence of erosion as high as 68 %). Enamel dissolution is due to acidic demineralization. At these early stages, it is a reversible process. Soft drink consumption of acidic food and drinks is correlated with the decrease of milk consumption, and in addition, this points at calcium deficiency.

2.3 Crystallite Dissolution

Hydrogen ions or chelating agents begin their deleterious effects by dissolving enamel crystal, either in the center of crystallites (central dissolution affecting a screw-like structure, due to the presence of defective Burger vectors) or at the lateral border and eventually at the edge of the crystallite. This outer screw dislocation occurs at the edge of the crystallite. The prism sheath (organic extracellular matrix) and the prism core are dissolved, leaving apparent a honeycomb structure mostly identifiable after acid etching. Differences in the behavioral, biological, and chemical factors may contribute to explain why some individuals display more erosion than others.

If the acidic impact persists (e.g., longer periods of interaction and/or increased concentrations), further dissolution of the enamel occurs. The dissolution becomes irreversible and leads to a severe alteration associated with a reduction of enamel thickness.

The erosion may be also dietary or result from chronic regurgitation. Soft drinks have an erosive potential. The effects of soft drinks on enamel can be evaluated by measuring the amount of calcium or phosphate ions released by enamel. There was no significant correlation between the % SMHC (percentage of superficial microhardness changes) and the other variables tested for a number of drinks leading to enamel surface softening. The pH seems to have more influence on the erosive potential of these drinks. Increased protective and defensive factors can restore or prevent loss of calcium and phosphate from enamel.

Tooth brushing, citric acid, or orange juice may remove the superficial part of the softened enamel (Figs. 2.1a, 2.2, and 2.4a). The thickness of the softened layer varied between 254 and 323 nm, depending on the acid used and its concentration. In any case, it is obvious that soft drink consumption increases the potential for enamel erosion.

Remineralization agents induced reduced caries and appropriately promote subsurface deposits. Acid neutralization may be obtained by buffering components of the diet. In addition to an early diagnosis of dental erosion, the detection of isolated factors like sports drinks may be implicated in multifactorial erosions. The significance of the mechanisms involved constitutes prerequisites, which are mandatory to initiate preventive and therapeutic measures.

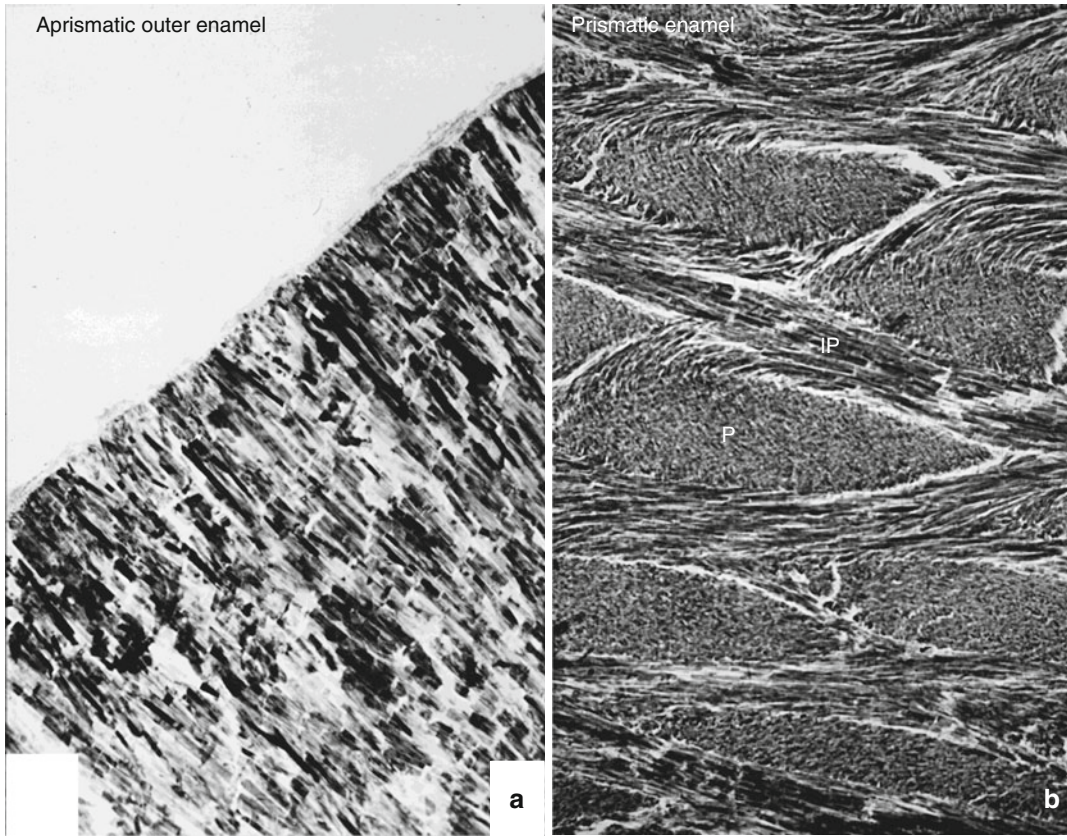

Fig. 2.1 Rat's molar enamel. (**a**) Normal aprismatic outer enamel/hydroxyapatite crystals are parallel, forming a continuous palisade-like structure. (**b**) Beneath the surface, enamel displays a prismatic structure, where prisms (*P*) (or rods) alternate with interprismatic enamel (*IP*) (interrod enamel)

2.3.1 Four Types of Physical and Chemical Dental Erosions Have Been Reported

Imfeld (1996a, b) has defined different types of tissue losses. According to Ganss (2006), each type of alteration bears its own specificity.

1. *Abrasion* is located on the incisal or occlusal surfaces and depends on the abrasiveness of the individual diet. The most abrasive agents are toothpaste, as shown by clinical data and *in vitro* studies. Both patient factors and material factors influence the prevalence of abrasion.
2. *Attrition* results from the action of the antagonistic teeth. It has also been named *demastication*. It is a mixture of abrasion and attrition.
3. *Abfraction* is located mainly in the cemento-enamel region where microfractures occur (also defined as fatigue wear). Wedge-shaped defects are related to abfractions or wedge-shaped non-carious cervical lesions. Analyses that demonstrate theoretical stress concentration at the cervical areas of the teeth have not yet been actually proven. However, there are some strong arguments favoring this possibility. The non-carious stress-induced cervical lesion or abfractions are observed in the buccal surface at the cemento-enamel junction of the teeth, with prevalence ranging in humans from 27 to 85 % (Sarode and Sarode 2013)
4. *Erosion* results from the action of acids or chelators acting on plaque-free tooth surfaces. The loss of tooth structure is due to acid dissolution without involvement of bacteria.

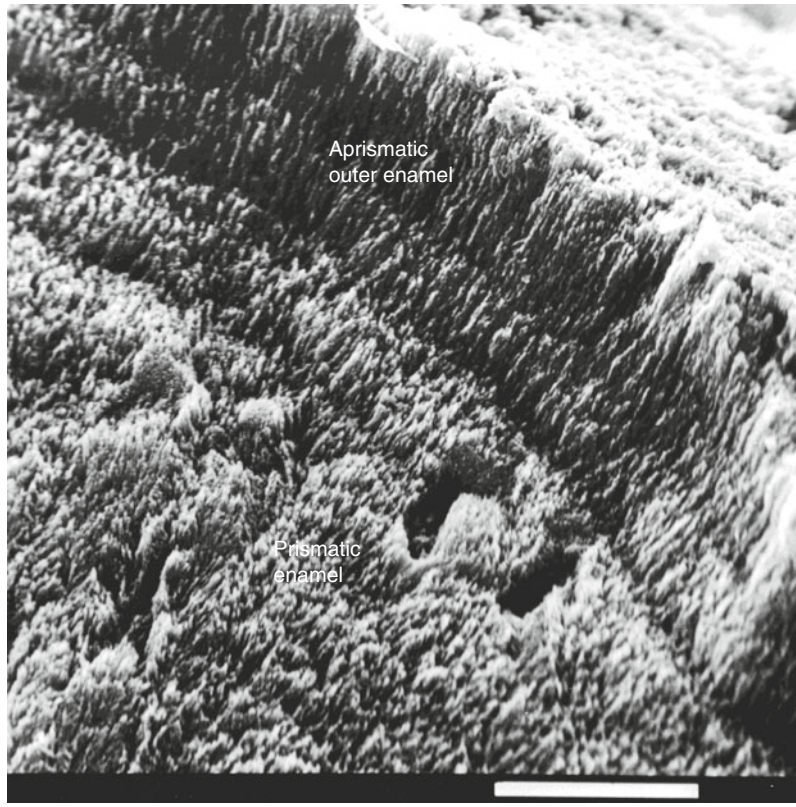

Fig. 2.2 Human enamel. The surface outer enamel layer looks laminated. This is not the case for the prismatic enamel located underneath

Regurgitated gastric acids or extrinsic components (soft drinks, acidic fruits) may act as extrinsic agents. Erosion is related to enamel surface softening. Repeated direct removal of a softened enamel layer favors more rapid demineralization and tissue loss (Figs. 2.3b and 2.4b).

2.3.2 Structure and Chemistry of Dental Erosion

Table 2.1

Structurally, the outer layer of enamel (aprismatic zone) consists of parallel crystals forming a palisade-like structure (Figs. 2.1a and 2.3b). The free ending of the crystals, located beneath a glycoprotein structure, which is controlled by the dental plaque, melts in response to the acidic extracellular gel or displays corroded extremities with enlarged inter-crystallite spaces. With effects that are time dependent and concentration dependent, the 5–15 μm thick aprismatic layer disappears gradually; and consequently the overall enamel thickness is reduced. The 3–5 μm prismatic enamel, which is now exposed by the erosion, displays a scalloped profile with numerous indentations and a drastic calcium and phosphate loss that is important near the new surface. The loss is gradually reduced until it reaches the original enamel level. The inner prismatic enamel is not altered, and both the enamel shape and thickness are undamaged.

The thickness of the softened outermost enamel and dentin layers is estimated to be 2–5 μm. Citric acid erosion caused a mean substance loss of 16.0 μm (SD ± 2.5 μm). Remineralization of re-hardened enamel is apparently similar to the original structure (Feagin et al. 1969).

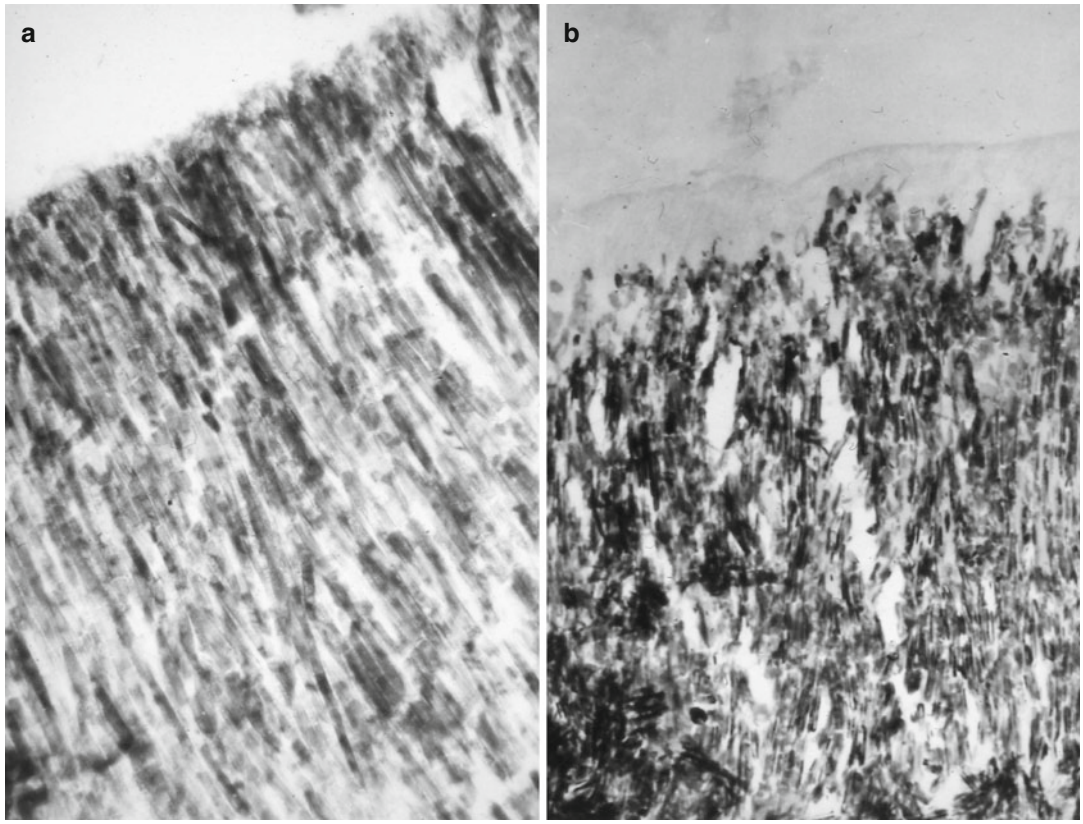

Fig. 2.3 (**a**) The outer enamel layer displays some interruptions or defects after softening. (**b**) The surface of corroded enamel displays structural alterations at the tip of crystallites and, at some locations, between crystallites

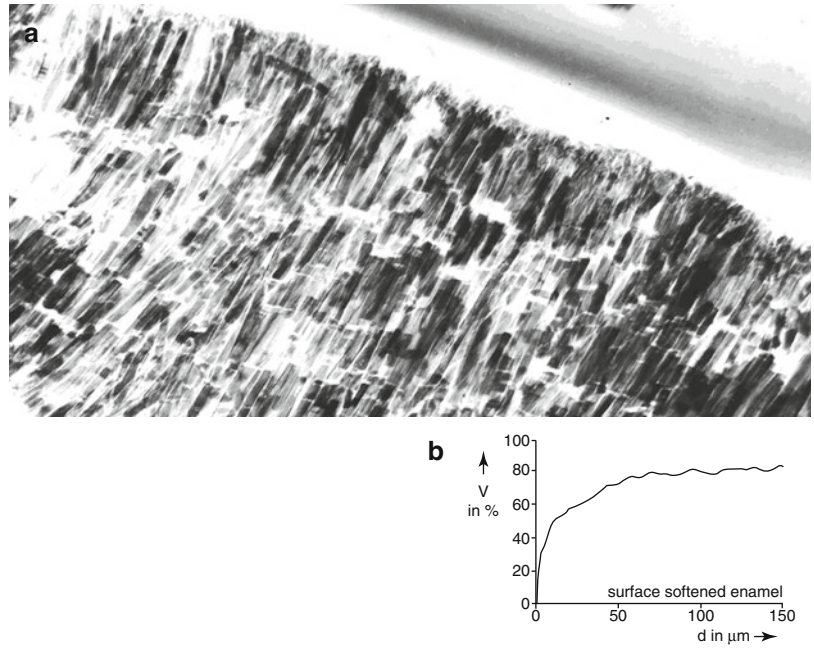

Fig. 2.4 (**a**) At the surface, crystallite endings look irregular with a scalloped profile. (**b**) There is a gradual mineral increase after the mineral loss due to the softening agent in the outer enamel surface. The percentage of mineral reaches a "normal" value at 50 µm under the enamel surface

Table 2.1 Composition percentage in enamel and dentine

Component	Enamel percent by volume	Dentine percent by volume
Carbonated hydroxyapatite	85	47
Water	12	20
Protein and lipid	3	33

See references by: Curzon and Featherstone (1983); De Carvalho Sales-Peres et al. (2007); Featherstone and Lussi (2006); and Lussi and Jaeggi (2008)

The mineral part of teeth is a calcium-deficient carbonated hydroxyapatite. Substitutions in the crystal lattice render the mineral phase more acid soluble. The direct attack by hydrogen ion combines with the carbonate and phosphate release of the crystal surface, leading to direct surface etching. The citrate ion can also form complex with the calcium removed from the crystal surface. The substitutions in the mineral crystal lattice disturb the structure. The carbonate content of "sound" enamel is approximately 3 %, while in dentine it is 5–6 %. Therefore the mineral is even more acid soluble in dentine. Crystals in dentine are smaller than in enamel; consequently the dissolution process needs shorter periods of time in dentine compared with enamel.

Chemical factors explain erosive attack. Addition of calcium and phosphate salts to erosive drinks show protection of surface softening. Biological factors such as saliva, acquired dental pellicle, and tooth structure and positioning are related to dental erosion development. Behavioral factors also play role in erosion. Frequent tooth brushing and good oral hygiene are important in the etiology of dental erosion. Sport and increased gastroesophageal reflux enhance the risk of developing dental erosion.

2.3.3 Intrinsic Causes of Erosion

Gastric juice entering the mouth causes dental erosion. This disease affects reflux (suppress disease), eating disorders, and concern about 65 % of the western population. In such case, the erosion is mostly palatal, due to reflux disease, regurgitation, anorexia, bulimia, and rumination (Eisenburger 2009).

Micro- and nanoindentations, indentation length, and surface hardness measurements, according to Knoop or Vickers, have identified rhomboid indentation of about 30–40 μm length. They have been recorded immediately and are not time dependent. Surface profilometry allows determining by scanning electron microscopy the tendency of the stylus to penetrate the fragile outer layer. Altogether, other methods such as SEM and ESEM, surface hardness measurements, surface profilometry, iodide permeability test, atomic force microscopy, nanoindentations, and ultrasonic measurement of enamel thickness may provide insights on the effects of acid solutions on enamel softening and the subsequent erosion (Fig. 2.4b).

Preventive strategies of patients suffering from erosion include dietary counseling, stimulation of salivary flow, optimization of fluoride regimens, modification of erosive beverages, and adequate oral hygiene measures (Magalhaes et al. 2009).

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