

Ece Eden *Editor*

# Evidence-Based Caries Prevention



 Springer

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*This book is dedicated to my dear son,  
Canberk Koparal.*

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## Foreword

Dentistry is continually evolving and improving. The treatment and management of dental caries is a major part of dentistry, with paediatric dentists and general dentists dealing with the results of the disease in numerous patients daily. Further, the prosthodontist has to deal with the aftermath of severe caries, sometimes necessitating major reconstruction in the mouth. If we are truly interested in the best interests of the patient and if we are to be part of the health-care team that promotes wellness, we have to think differently than traditional dentistry. Traditional dentistry “treats” the symptoms of the disease by physical intervention and surgery, but it does not treat the disease. We now have definitive evidence that placing restorations takes care of the physical integrity of the tooth involved, but it does not reduce the caries challenge nor the levels of cariogenic bacteria in the rest of the mouth. About 70 % of high risk patients who have restorative work done come back with new lesions within 2 years, unless chemical therapeutic intervention is added to the mix.

Our challenge for the future is to provide a wellness programme for our patients that identifies their risk of caries initiation or progression and deals with the disease in conjunction with minimally invasive restorative dentistry where necessary. The two go hand in hand as a minimal intervention package. This book addresses the components of this approach and sets the stage for a new era of dentistry that will provide much better oral health and general health for our patients. Each of the components is dealt with in a progressive fashion with each chapter building on the previous one.

We have been able to develop and apply a system of caries management by risk assessment over the last 15 years that provides reductions in new caries of over 40 % in high caries risk patients. This number may be as high as 80 % in private practice where compliance can be much better monitored and promoted. In my experience dental practitioners who adopt caries management by risk assessment as part of minimal intervention dentistry have success with patients, build their practice and are very happy with what they have achieved for the benefit of the patients.

The best approach is to have a basic understanding of the caries process, to adopt a proven caries risk assessment methodology, to provide chemical intervention therapy depending on the level of risk, to provide minimally invasive restorative care and to have a recall and prevention protocol depending also on the level of risk. The simple concept of the caries balance underpins all of this approach. Essentially, the guiding principle is to decrease the pathological factors and increase the protective

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factors. In simple terms this means providing antibacterial therapy where the bacterial challenge is high, including dietary modification, and providing remineralisation therapy at all levels with the intensity depending on the level of pathological factors. Hyposalivation provides particular challenges and requires additional therapy and intensive care. There is now ample evidence for all aspects of this approach.

I challenge all who read this book to practice caries management by risk assessment, which includes caries prevention and intervention, as the basis of wellness for your patients. Practice evidence-based dentistry.

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## Preface

Evidence-based dentistry may be defined as dentistry that uses scientific evidence to guide decision-making, which means that, after having identified a disease (in this case the disease is ‘dental caries’), this approach advocates searching for cogent evidence before attempting to treat the disease. This book has ten chapters, which deal with various aspects of caries prevention. The chapters (and the foreword) have been written by 14 distinguished authors from around the world. Together, we have tried to gather as much evidence as possible from systematic reviews and meta-analysis and sometimes have had to conclude that further research is needed for certain topics. We know that research and technological advancements constantly renew our knowledge and skills, which in turn lead to the revision and adaptation of treatments.

Dental caries has become a disease of socio-economically less advantaged groups in developed and developing nations, and prevention may have lost its popularity for privileged populations. However, the life expectancy at birth of today’s youth is anticipated to increase all around the world, particularly in developing countries in Asia and Africa. These countries will experience a massive increase in oral health problems along with increase in life expectancy. For this reason it is essential that the need for further search for the most appropriate management of the disease be recognised and that immediate efforts to stop inequalities in oral health care are made.

The authors of this book have gone to great lengths to gather up-to-date and useful information regarding dental caries and in Chap. 1 have tried to clarify the terminology and definition of the disease and its epidemiology. The detection of carious lesions, their activity and risk assessment, and caries assessment methods and devices are discussed in Chaps. 2 and 3. Remineralisation of carious lesions and the level of effectiveness of antimicrobials are dealt with in Chaps. 4 and 5. Chapter 6 discusses the effect of diet on preventing carious lesion development. Minimal invasive procedures such as sealant and resin infiltration applications are the topics of Chaps. 7 and 8, while the evidence and appropriateness of non-operative caries preventive measures are presented in Chap. 9. Chapter 10 presents example cases in which various clinical conditions that may be seen in everyday dental practice are treated.

The primary aim of the book is to discuss the evidence on caries prevention and to help clinicians use this knowledge in their routine work. I hope that the



expertise presented in the chapters in this book will guide practitioners in their endeavours to manage dental caries through carious lesion preventive measures in daily practice.

This book is for all practitioners who consider themselves dental healers rather than dental repairers. I hope you will enjoy the book as you read it.

Ece Eden  
Izmir, Turkey

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## Acknowledgements

I would like to thank all the authors for dedicating their valuable time and providing their expert knowledge to create this book. In particular, I would like to thank my dear friend, Jo Frencken, for his immense support, advice and help in constructing the book. My sincere thanks go to Pınar Yanyalı Doğu and Susan van Tonder for their skilful editing of the English text and to Ali Cemal Demir, our kind librarian, for his unceasing support. It was a pleasure to work with all of them.

I was honoured to be asked to edit a textbook by a very well-known publisher in dentistry. I thank Sushil Kumar Sharma, Wilma McHugh and Antonia von Saint Paul from Springer for their kind interest and patience in producing the book.

This book could not have been written without the support of many people. My patients, whom I treated with joy; my students, such as Ecem Ergin and Elif Kuru, who helped and inspired me; and my many colleagues, sometimes knowingly and sometimes unaware of their help, improved the content. My thanks also go to Gözde Tanyan for the illustrations and editing of the photographs.

I would like to thank my parents, who made me who I am, my father with his presence and my mom, who must be watching from up above. I would also like to thank my dear friends, Meltem Ağduk, Elif Yurdun, Güniz Tanker, Zekiye Ayraç and Rabia Çolak, who have always believed in me.

This book is dedicated to my son, Canberk Koparal, who once asked me whether ‘coffee teeth’ would erupt after ‘milk teeth’ exfoliated. I thank him for challenging me with such interesting and thought-provoking questions. This project could not have been possible without his love and support.

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## Abstract

In focusing on dental caries and caries epidemiology, this chapter introduces and discusses up-to-date terminology for improved communication on dental caries among dentists around the world. It introduces the Caries Assessment Spectrum and Treatment (CAST) instrument as a suitable and validated comprehensive epidemiological carious lesion assessment instrument. Lastly, it presents carious lesion prevalence and severity figures for various age groups in a time-lag perspective.

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## 1.1 Dental Caries

The term ‘dental caries’ is used frequently in cariology, but its use when written and spoken has created misunderstandings, and the term has been misinterpreted within oral health communities throughout the world. For some, ‘dental caries’ refers to the name of a disease, while for others dental caries relates to lesions and cavities. ‘I can still see some caries over there’ and ‘do remove the caries in the distal-buccal corner of the cavity’ are examples of how dentists communicate to others about the disease. When dentists use the term in spoken language, the persons spoken to can immediately react if they do not understand the term as used in a specific context. But when the term is used in print, it may cause confusion and perhaps misunderstanding, leading to possible incorrect decisions or procedures and knowledge uptake.

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What is meant by ‘caries’ in the utterance: ‘I can still see some caries over there’? Is the speaker referring to a carious lesion in the enamel or to the one in the dentine, or is the lesion present in both structures? We can only guess. And what is understood by ‘caries’ in the instruction: ‘do remove the caries in the distal-buccal corner of the cavity’? Does it refer to soft demineralised carious tissue or remineralised hard dentine, or perhaps something else? We do not know. These examples are among many that suggest that, as in any discipline-specific terminology, the language of cariology should be unambiguous. It should be able to describe the palette of situations that occur as part of the caries process in such a way that dentists in all corners of the world interpret the related descriptions in the same way.

### 1.1.1 Terminology

We consider ‘dental caries’ as the name of a disease only. The signs of dental caries lead to the name ‘cariou lesion’. When confined to the enamel, a cariou lesion is called an ‘enamel cariou lesion’, and, in the same way, a ‘dentine cariou lesion’ is the name given to a cariou lesion that involves the dentine. If a cariou lesion is cavitated, the correct name is either ‘cavitated dentine cariou lesion’ or ‘cavitated enamel cariou lesion’, depending on the extent of the cavitation.

How do we interpret ‘caries prevalence’? As dental caries is defined by loss of tooth minerals as a result of acid produced from fermenting sugars by microorganisms in a biofilm, the smallest loss of invisible superficial mineral tooth structure would make a particular tooth surface cariou. But we cannot include these invisible cariou lesions in the calculation of a prevalence score. What we can do is to include enamel cariou lesions in the prevalence calculation. Currently, the prevalence of dental caries by definition is restricted to the inclusion of only (cavitated) dentine cariou lesions in the prevalence calculation [1]. However, inclusion of enamel cariou lesions in the cariou lesions prevalence score started decades ago [2, 3] and was re-advocated during the last decade [4, 5]. Re-advocation occurred as a result of the decrease in number and prevalence of cavitated dentine cariou lesions in many population groups in many Western countries from the mid-1970s [6].

Knowing whether enamel cariou lesions are included in the caries prevalence figure is important. Not mentioning this information can cause a remarkably different outcome as has been reported for a child population in the 6–7-year age group. In this study, the prevalence of dentine cariou lesions in the primary dentition with and without the inclusion of enamel cariou lesions, assessed according to ICDAS II, was 95.6% and 67.2%, respectively. The figures for the prevalence of dentine cariou lesions in the permanent dentition of these children were 62.7% (with) and 10.9% (without) inclusion of enamel cariou lesions [7]. This example shows that dental professionals should be precise when reporting cariou lesion-related data. Thus, ‘caries prevalence’ should be replaced by ‘(cavitated) dentine cariou lesion prevalence’ or ‘(cavitated) enamel and dentine cariou lesion prevalence’, depending on how the prevalence was calculated.

### 1.1.2 International Caries Consensus Collaboration (ICCC)

An attempt to improve the nomenclature regarding managing dental caries and carious tissue removal was made by the International Caries Consensus Collaboration (ICCC), a group of 21 cariology experts from 12 countries. They met in Leuven, Belgium, in February 2015 with the goal of reaching consensus on recommendations for managing carious lesions and the terminology around this management, based on current best scientific evidence, through discussion and then consultation. The recommendations regarding the terminology are presented in an open access research article by Innes et al. [8] and those regarding carious tissue removal in an article by Schwendicke et al. [9]. The reader is invited to read these articles.

In this book, authors have defined dental caries in different ways. It is not the intention to add another definition here. However, it is worth mentioning that, in contrast to its description in some textbooks and research articles, dental caries is no longer considered an infectious disease. The definition used these days considers dental caries more of a behavioural, biological disease that is associated with the individual's social context [1].

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## 1.2 Caries Epidemiology

### 1.2.1 Detection and Assessment Criteria

Knowing who will keep their teeth healthy and who will develop carious lesions is very important for individuals and for societies. Factors and models that predict carious lesion development in individuals are presented in Chap. 3. An important aspect of the prediction of carious lesion development is the detection of the lesions and the assessment of the severity of the identified lesions. A number of carious lesion detection and assessment indices and devices are presented in Chap. 2. That chapter also discusses the suitability of these indices and devices for use in the dental clinic in terms of validity and reproducibility only. While perhaps suitable for use in the dental clinic, not all presented indices and devices appear suitable for use in epidemiological surveys. A number of these indices involve electronic devices that have no proven validity, are not field proof and are, therefore, unsuitable for use under field circumstances. Caries epidemiology, therefore, relies predominantly on visual/tactile indices for detecting and assessing carious lesion-related conditions in a field setting [10].











Although many such visual/tactile indices had been introduced since the mid-1950s [11], the International Caries Detection and Assessment system (ICDAS) Collaboration Group developed and promoted the ICDAS two-digit index as a new classification system in caries epidemiology [4]. Within a short time, ICDAS was upgraded to ICDAS II, followed a couple of years later by ICDAS II-PUFA and lately by the International Caries Classification and Management System (ICCMS). A recent analysis of the validity of the widely used ICDAS II revealed the absence of face and content validity, meagre evidence for construct validity related to



primary teeth only and no evidence for construct validity related to permanent teeth [12]. Individual researchers have altered the index over the years because of difficulties encountered when using it in the field [13–15]. Another difficulty in using ICDAS relates to reporting results. Initially the DMFT/S unit was used to report results, followed by the DMFT/S + PUFA modification. A year or so later, the unit of expression was changed again. This time, the ICDAS Collaboration Group suggested the use of DMFT/S<sub>ICDAS/LA</sub> where ‘LA’ stands for ‘lesion activity’. With the introduction of the ICCMS in 2013 [16], the ICDAS II index is split into a care index (former 1<sup>st</sup> digit) and a carious lesion index (former 2<sup>nd</sup> digit), with various options to merge the 7 caries-related codes for reporting results. Because of these inconsistencies, many researchers and dentists have found the scoring confusing and have stopped using ICDAS. ICDAS leaves a legacy of being a good idea but one that was launched too fast without proper research into the scientific requirements for introducing a new detection and assessment instrument for use in caries epidemiological surveys.

In 1999 Nyvad et al. published a set of criteria for assessing both carious lesions and carious lesion activity [17]. A decade later, the pulp/ulceration/fistulae/abscess (pufa/PUFA) index was introduced for assessing the pathological consequences of the caries process [18]. However, together with the World Health Organization [19] criterion, which basically assesses whether a carious lesion is cavitated into dentine or not, the three visual/tactile indices described cannot be used in caries epidemiology on their own. For example, the pufa/PUFA index needs to be complemented by an index that assesses enamel and dentine carious lesions without pulpal involvement; the Nyvad criteria need the addition of a ‘missing teeth due to caries’ category, while the WHO criterion requires an index that includes enamel carious lesions. A newly developed assessment instrument for use in caries epidemiological surveys termed the ‘Caries Assessment Spectrum and Treatment’ (CAST) instrument [5] overcomes the disadvantages of the visual/tactile indices described above. It was introduced for the assessment of dental caries-related conditions and treatment in epidemiological surveys. It permits registration of sound teeth, sealants, restorations, enamel and dentine carious lesions, advanced stages of carious lesions into the pulp and tooth-surrounding tissues, and teeth lost from dental caries (Fig. 1.1). The assessment is performed visually, with the naked eye, and does not use compressed air for drying tooth surfaces. CAST consists of 10 codes that are ordered hierarchically. This implies that a restoration (code 2) is less severe than an enamel carious lesion (code 3) and that a dentine carious lesion (code 5) is less severe than a tooth with an abscess caused by the caries process (code 7).

The research shows that the CAST instrument has face, content and external validity for use in children and adults [20, 21]. It has a high level of reproducibility [22], and the CAST codes can be converted to dmf/DMF counts so that results can be compared with those obtained from using the WHO caries criterion [23]. The CAST instrument still needs to be tested in populations of different ages and backgrounds than those studied so far. The CAST instrument’s quality research was performed in Brazil. Meanwhile, CAST has been used or is in use in epidemiological surveys in Poland, Peru, India, Russia, Surinam and Turkey as far as I know.

Characteristic	Code	Description	Example
Sound	0	Sound No visible evidence of a distinct carious lesion is present	
Sealant	1	Pits and/or fissures are at least partially covered with a sealant material	
Restoration	2	A cavity has been restored with an (in)direct restorative material	
Enamel	3	Distinct visual change in enamel only. A clear caries-related discolouration is visible, with or without localised enamel breakdown	
Dentine	4	Internal caries-related discolouration in dentine. The discoloured dentine is visible through enamel, which may or may not exhibit a visible localised breakdown	
	5	Distinct cavitation into dentine. The pulp chamber is intact	
Pulp	6	Involvement of the pulp chamber. Distinct cavitation reaching the pulp chamber or only root fragments are present	
Abscess/Fistula	7	A pus-containing swelling or a pus-releasing sinus tract related to a tooth with pulpal involvement	
Lost	8	The tooth has been removed because of dental caries	
Other	9	Does not correspond to any of the other descriptions	

**Fig. 1.1** The validated CAST characteristics, codes and descriptions [24]

### 1.2.2 Reporting Data from Caries Epidemiological Surveys

Currently the prevalence of carious lesions is, by definition, calculated on the presence of caries codes that represent stages of the lesions into dentine, restored lesions and missing teeth due to dental caries (D<sub>3</sub>MFT). If required, the code for enamel carious lesion(s) can be included in the prevalence calculation, but this has to be clearly stated (D<sub>1</sub>MFT or D<sub>2</sub>MFT). This definition covers not only actual disease but also past disease (restored and missing teeth). The advantage of considering teeth restored and teeth lost from dental caries not diseased anymore is that dental caries prevalence is calculated on the bases of the actual presence of the disease in the individual and in the society. This reasoning was a cornerstone of the development of CAST. It holds the advantage that monitoring disease interventions can yield lower prevalence rates, which shows the effectiveness of the intervention. This is not possible with the currently calculated prevalence, which is based on the dmf/DMF count [1].

In principle, studies that use CAST do not report the results in dmf/DMF counts. There is no problem in doing so, however, if studies that have used dmf/DMF counts need to be compared with those that have used CAST, as shown for the 7–11-year-old age group [23]. The extent of the caries-related conditions, measured using CAST, is presented as frequency distributions per caries code or for maximum CAST code per tooth or mouth, depending on the aim of the survey. The severity of caries-related conditions within an individual or group after using CAST is calculated according to a mathematical formula. This formula is being tested and for this reason is not presented here. Readers wishing to know more about how to apply CAST and how to report data are referred to the CAST manual [24] that will appear on the CAST website, at present under construction.

### 1.2.3 Prevalence and Severity of Dentine Carious Lesions over Time

Factors that influence the caries process have been identified (Chap. 9). The two major factors that impede the process are controlling intake and frequency of fermentable sugars, in particular free sugars, and removing the biofilm from the tooth surfaces with fluoride-containing toothpaste daily. The effect of these two measures has made a large contribution to the decline in the prevalence and severity of carious lesions in children in many countries, particularly in North America, West and North Europe and Australasia [1]. Obviously, other carious lesion-controlling measures have contributed to this decline, but they are more country specific and relate to factors such as dental insurance and social context. The decline is revealed in a large number of surveys carried out in The Netherlands between 1980 and 2010 [25]. These surveys reveal that the average prevalence of dentine carious lesions decreased from 60 to 50 % among 5-year olds and from 80 to 20 % among 12-year-old children from 1980 to 2010. The average mean dmfs-score was reduced from 4.5 in 1980 to 2.5 in 2010 for 5-year olds and the average

mean DMFS-score from 4.8 in 1980 to 0.8 in 2010 for 12-year olds. The same pattern is seen in children from the countries mentioned above and from some other regions in the world [1]. This laudable result shows that healthy teeth can be kept free from the occurrence of visible carious lesions among the younger section of the population. But can this low level of dental caries be maintained into and through adulthood? A longitudinal research study from New Zealand has given us an answer. A large cohort was followed from age 5 to 38. The most remarkable finding was that the annual increment in carious lesions remained the same over the 32-year period [26], which showed that dental caries is an age-related and life-long disease that requires daily attention and maintenance. The study began in 1972/73 at a time when carious cavities were prevalent in adults and children. It would be interesting to see longitudinal data on the progression of dentine carious lesions in societies that have established measures to keep healthy teeth healthy over a long period.

#### 1.2.4 Prevalence and Extent of Carious Lesions in Infants

It is technically and behaviourally possible to keep healthy primary teeth healthy. Unfortunately, this is not the reality in many world communities. (Severe) early childhood caries ((S-)ECC) is prevalent in many countries with large populations in deprivation. Epidemiological surveys from Brazil [27], Canada [28], Vietnam [29], China [30], Switzerland [31] and Thailand [32] show alarming results. High prevalence figures for S-ECC in 3-year olds of 38 % (Canada) and 44.1 % (Thailand) have been reported, while the prevalence of ECC was 24.8 % in Switzerland and 74.4 % in Vietnam among 1–6-year olds. The mean dmft-score for 1–6-year olds was 3.6 in China and 3.9 in Canada. These figures show that something is drastically wrong in many world communities despite some improvements achieved over the last three decades in other countries and communities [1].

The dental fraternity should take these findings seriously and look for appropriate actions to curb the preventable disease ‘dental caries’ from occurring in the very young. Experience has shown that the dental profession alone is unable to turn the tide in many communities. It is, therefore, applaudable that one of the conclusions of the 2014 Early Childhood Caries Conference states that: ‘ECC oral healthcare should be integrated within medical care settings’ [33]. Indeed, mother-and-child healthcare programmes may be best placed for educating parents and/or caregivers of babies/infants in good oral health behaviour in harmony with the provision of general health information and care. An example is the integrated programme performed in health centres in Lima, Peru, which uses oral health advice and information cards [34]. This programme considers nurses the main oral health guiding and monitoring medical personnel. They have been educated in the development and progression of dental caries and in prevention and promotion of oral health programmes in babies and infants, in addition to recognising early signs of carious lesions. When these signs are recognised in a baby or infant, the mother is advised to

visit the dentist who holds an office in the same health centre. The information cards help the nurses in advising and educating parents/caregivers on appropriate hygiene and diet actions.

(S-)ECC is culturally, socially and economically related. Therefore, tailor-made preventive measures should be discussed with and communicated to the members in these communities in ways that have been found to be effective and attainable locally. Appropriate preventive measures for this group are presented in Chap. 9.

### **1.2.5 Carious Lesion-Susceptible Teeth and Surfaces in Child Populations**

The fluoride studies from the 1950s to the 1980s showed that the largest reduction in the extent and severity of carious lesions in children took place in smooth surfaces, followed by approximal surfaces. Fluoride was less effective in occlusal surfaces. Other researchers have also reported this hierarchy in carious lesion susceptibility [3, 35, 36]. On the basis of data from 20,000 5–16-year-old schoolchildren in the USA, it was established that the predominant susceptible tooth sites in low dentine carious lesion individuals (DMFS < 5) were pits and fissures (95%). The proportion of approximal surfaces and smooth surfaces increased with an increase in mean DMFS score in this age group. In high dentine carious lesion individuals (DMFS > 25), the proportion of dentine carious lesions was about 20% for smooth surfaces, 30% for approximal surfaces and 50% for pits and fissures [36].

Is there also a hierarchy in dentine carious lesions by tooth type? On the basis of the findings of the same USA study, it could be concluded that occlusal surfaces of first molars and buccal pits of lower first molars were the most carious lesion-susceptible type of tooth surface. If all the first molars are affected, then a high probability exists that the second molars will be affected. The occlusal surfaces of the second molars and the buccal surfaces of the second lower molars are the second most susceptible sites for dentine carious lesion development in children with a low DMFS count. Smooth surfaces on the lower anterior region are least susceptible [37].

All in all, pits and fissures in occlusal and pits in buccal tooth surfaces appear to be the most vulnerable sites for dentine carious lesions in the permanent teeth of children and adolescents. In children at high-carries risk, these sites may need extra protection to keep them healthy.

### **1.2.6 Prevalence and Extent of Carious Lesions in the Elderly**

One of the consequences of individuals having maintained healthy teeth over the decades is the presence of a large number of natural teeth in people of old age. This pattern started in many industrialised countries with the introduction of individual and collective preventive oral care programmes in the 1970s.

Fewer epidemiological surveys have been carried out among the elderly than among children and adolescents. In 2003 Swedish 80+-year olds had on average

18.4 teeth, up from 13.7 in 1993. In the 60–70-year-old group, the mean number of natural teeth was 18.6 in 1993 and 23.3 in 2003 [38]. This pattern has also been reported in the UK, Canada and Australia [39].

The prevalence of root carious lesions in 60+-year olds in Japan was 39% in 2006, with poor oral hygiene and low salivary flow rate being potential risk factors [40]. More recently, in South Brazil, approximately 36% of dentate individuals had carious lesions and/or restorations that affected, on average, 5.0 teeth [41]. In an older age group of over 80-year-old Swedish elders, untreated coronal dentine carious lesions were present for between 36% and 56% of the subjects, while between 54% and 75% had untreated root carious lesions [42].

The fact that people are getting older with more natural teeth than in previous times increases the risk for carious lesion development, both in crown and root surfaces, because of an increase in the number of teeth. Ways of assisting the elderly in keeping their teeth healthy are presented in Chap. 9.

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### 1.3 Final Remarks

Dental caries is a behavioural disease that is mostly preventable through diet (free sugar) control and daily biofilm removal with a toothbrush and fluoride-containing toothpaste. To communicate about the disease unambiguously among dentists throughout the world, terminology related to carious lesions and carious tissue removal methods needs to be kept up to date on the basis of evidence. Two recently published open access papers report about these topics after consensus was reached by 21 cariologists from 12 nations.

Generally speaking, dental caries is an age-related disease that occurs in many countries and communities with various levels of disease prevalence, extent and severity. Despite marked decline in children and adolescents in some countries, (S-) ECC is particularly prevalent in many more. Co-operation with medically trained personnel is suggested as the way forward for curbing the disease in babies and infants.

The only validated comprehensive epidemiological visual/tactile instrument for detection and assessment of carious lesion (-related) conditions is the CAST instrument. Launched recently, its use is increasing but not widespread yet. Reporting results uses frequency distributions and a unique formula. CAST codes can be converted into DMF-components for comparison with results of surveys conducted in the past.

Pits and fissures in occlusal first molars and pits in buccal mandible first molars are the tooth types and sites that are the most susceptible to dentine carious lesions. In children at high risk for carious lesions, these tooth types and sites need to be monitored well. Additional evidence-based carious lesion preventable measures, other than sugar-free diet and biofilm control, need to target these vulnerable sites as part of a comprehensive maintenance scheme in children and adolescents at risk.

**Acknowledgements** I would like to thank all colleagues who, through their research, have assisted me in writing this Chapter.

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# Detection and Diagnosis of Carious Lesions

# 2

Hervé Tassery and David J. Manton

## Abstract

The early detection and diagnosis of the carious lesion are a primary consideration of the minimal intervention dentistry (MID) concept. Detection is the identification of a demineralized lesion, while diagnosis is an iterative process using further information from the patient to identify the lesion as carious in nature. Traditionally, visual, tactile, and radiographic methods are used to detect carious lesion; however, recently tactile detection, especially using a sharp explorer, is not recommended apart from the delicate detection of enamel surface integrity/roughness. Use of simple devices such as loupes for magnification of the clean surface can improve detection validity. Advances in technology have led to many different devices being released onto the market to assist in the detection and quantification of carious lesions. Fluorescence of the tooth structure using different wavelengths of light can provide information about lesion extent and area and whether dentine is involved, and several commercial systems are available such as DIAGNOdent®, QLF®, and Soprocure/Soprolife®. It is important to follow standardized procedures with all of these detection techniques, as the validity and reproducibility of the results are highly dependent on this. Electrical

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impedance and photothermal radiometry have been promoted as alternatives to light-based technologies; however, evidence for these devices is still somewhat limited. All of these traditional and new detection methods will be discussed in the context of minimal intervention dentistry and their potential value to the clinician and patient.

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## 2.1 Introduction

Dental caries still remains a relatively ubiquitous disease with a large global burden of care which is predicated to increase in ensuing years [1]. Primary prevention of carious lesion development is an initial important aim; however, due to current dietary habits and poor oral hygiene, this appears unlikely in a large proportion of the global population. Early detection and quantification of carious lesions therefore become important as it allows targeting of those individuals at risk of developing dental carious lesions and preventive efforts to be undertaken, such as dietary modification, placement of fissure sealants, fluoride application, remineralization, etc. There is a need for detection methods to be valid, leading to accurate diagnosis of the carious lesion (obviously also involving caries risk assessment) and maximizing the outcome for appropriate and timely minimally invasive care, MID, for the individual. Diagnosis and detection are interrelated, but not the same process. Detection of a carious lesion is the observation (and hopefully objective quantification) of the lesion, whereas diagnosis is the synthesis of the signs and symptoms of the disease including the assessment of the caries risk factors of the individual in toto.




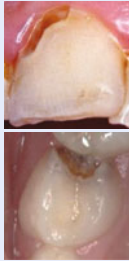
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## 2.2 Visual Carious Lesion Detection

Visual detection of the carious lesion is the mainstay of clinical practice – it is relatively sensitive and specific and is cheap. A limitation of visual detection is its subjectivity; however, this can be improved by the use of indices such as International Caries Detection and Assessment System (ICDAS)/International Caries Classification and Management System (ICCMS) and Caries Assessment Spectrum and Treatment (CAST) [2–4]. The development of the ICDAS and CAST for the quantification of carious lesions has provided a validated method for assessing and quantifying lesions, and the recent addition of an associated management system, the ICCMS, provides evidence-based management options for the various stages of the carious lesion, allowing for individual circumstances. Traditionally, visual caries detection has led to a dichotomous decision – the individual either has a lesion or doesn't. This creates a number of clinical options depending where the diagnostic threshold is placed – i.e., at the lesion primarily limited to enamel level (ICADS 1 and 2), the surface characteristic change level (ICDAS 3 and 4), or the frank cavitation level (ICDAS 5 and 6). Traditionally the threshold was placed at frank

cavitation (such as for the WHO index-D-d) – and the outcome was invasive operative care unless the lesion was at the extent where the tooth required extraction. By moving the diagnostic threshold “to the left,” less invasive treatment options are made available, such as placement of sealants and remineralization of lesions (Table 2.1).

**Table 2.1** Definition of ICCMS™ caries categories (merged codes) (Courtesy ICDAS Foundation)

Definition of ICCMS™ caries merged categories		
<i>Caries categories</i>	<i>Sound surfaces</i> (ICDAS code 0)	 <p><i>Sound tooth surfaces</i> show no evidence of visible caries (no or questionable change in enamel translucency) when viewed clean and after prolonged air drying (5 s)<sup>8-9</sup> <i>(Surfaces with developmental defects such as enamel hypomineralization (including fluorosis), tooth wear (attrition, abrasion, and erosion), and extrinsic or intrinsic stains will be recorded as sound)</i></p>
	<i>Initial stage caries</i> (ICDAS codes 1 and 2)	 <p><i>The first or distinct visual changes in enamel</i> seen as a carious opacity or visible discoloration (white spot lesion and/or brown carious discoloration) not consistent with clinical appearance of sound enamel (ICDAS code 1 or 2) and which show no evidence of surface breakdown or underlying dentine shadowing</p>
	<i>Moderate stage caries</i> (ICDAS codes 3 and 4)	 <p>A white or brown spot lesion with <i>localized enamel breakdown</i>, without visible dentine exposure (ICDAS code 3), or an <i>underlying dentine shadow</i> (ICDAS code 4), which obviously originated on the surface being evaluated <i>(To confirm enamel breakdown, a WHO/CPI/PSR ball-end probe can be used gently across the tooth area – a limited discontinuity is detected if the ball drops into the enamel micro-cavity/ discontinuity)</i></p>
	<i>Extensive stage caries</i> (ICDAS codes 5 and 6)	 <p>A <i>distinct cavity</i> in opaque or discolored enamel with <i>visible dentine</i> (ICDAS code 5 or 6) <i>(A WHO/CPI/PSR probe can confirm the cavity extends into dentine)</i></p>

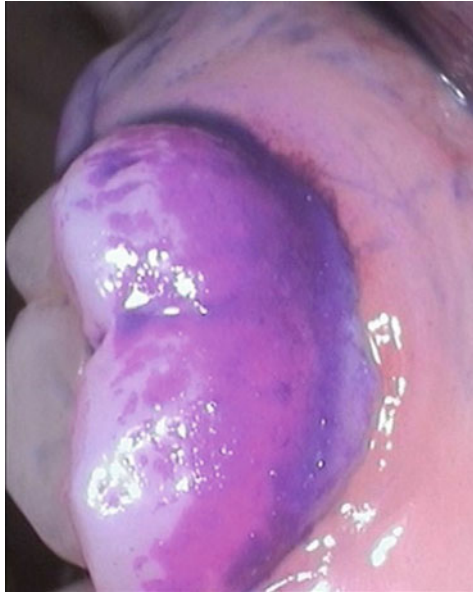
Magnification assists in the determination of the integrity of the enamel surface, especially in pits and fissures where the use of an explorer is contraindicated due to potential damage to the enamel surface integrity (Fig. 2.1) [5]. Many clinicians promote the use of clinical microscopes for both diagnostic and operative procedures, providing even greater magnification than loupes and providing the ability to determine the status of surfaces in fine detail (Fig. 2.2). The ability to detect small areas of surface cavitation would be very difficult without magnification, and the use of an operating microscope potentially improves posture of the clinician. Cleaning of the tooth surface is an essential part of diagnosis for two reasons – carious lesions develop under mature biofilm, so its presence leads the clinician to sites of risk, and to identify and quantify a lesion accurately, it must be cleaned first and

**Fig. 2.1** SEM image of damaged pit after probing (Courtesy of Prof Dr Jan Kühnisch, Munich, Germany)



**Fig. 2.2** Magnified image of initially demineralized fissures (ICDAS initial stage, Soprolife® daylight) 15 s air polishing with Pearl® powder





**Fig. 2.3** Tri Plaque disclosing of the second upper molar (Soprolife® picture daylight mode)

then dried. So plaque disclosing should be done before lesion detection, and an example of GC Tri Plaque (GC Corp, Tokyo, Japan) is illustrated in Fig. 2.3.

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### 2.3 Tactile Carious Lesion Detection

The use of aggressive tactile methods for lesion detection, such as a sharp explorer (probe), has been taught in dental schools for more than a century. Sufficient evidence exists indicating that this method should be discontinued when detecting carious lesions in pits and fissures and on smooth surfaces, as damage can occur to enamel, especially when fragile demineralized lesions are probed or excessive force is used. This can lead to fracture of the enamel surface layer predisposing this area to further lesion development and subsequent need for restorative care [5] (Fig. 2.1). The use of the sharp probe also does not provide any additional diagnostic information to visual and/or radiographic methods [6–8]. The sharp probe should be replaced with a blunt or ball-ended probe that can be used to detect surface integrity. This is done by drawing the probe gently over the enamel surface to detect any changes in surface roughness, with increased roughness indicating initial breakdown of the surface layer. The probe should not be forced into a pit or fissure. The continued use of probing for lesion detection persists among current clinicians; therefore education related to appropriate methodologies is still required [9].

The clinician should determine the activity of the lesion that will be the “driver” of clinical decisions regarding the lesion and oral health as it relates to dental caries in general (see also Chap. 3).

## 2.4 Radiographic Carious Lesion Detection

Clinical dentists have used radiography, in conjunction with visual detection, effectively for nearly 100 years. One of the major limitations of radiography is associated with the detection of carious lesions in the pits and fissures, especially when the lesion is in the early stages of dentinal involvement [10]. This is due to a limited ability to distinguish initial changes in mineral content due to the confounding effects of the superimposed adjacent tissues.










As the ability of current radiographic methods to detect early lesion (especially those where demineralization is limited to the enamel) is limited, radiography should be used in conjunction with other detection methods, such as direct visual and transillumination.

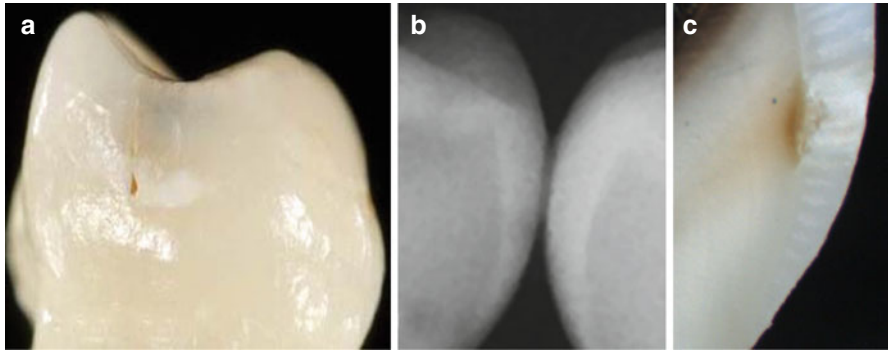
Clinician experience and training may influence the validity of “reading” radiographs, with more experienced clinicians having lower sensitivity and higher specificity for detecting carious lesions when compared to undergraduate students [11]. The use of an index such as the ICDAS radiographic index illustrated in Table 2.2 may help standardize coding and improve longitudinal monitoring of lesion regression/progression as it also considers the clinical appearance of the lesion, allowing clinicians to synthesize combined diagnostic information.

The validity of lower-dose digital radiography in detecting carious lesions is similar to that of film-based methods, and with the ability to store and back up images easily and also transfer the digital images, digital radiography is the most appropriate technology for today’s clinician.

The use of digital manipulation, such as subtraction radiographic methods, increases the accuracy and reproducibility compared to visual assessment of the

**Table 2.2** Definition of ICDAS radiographic codes (Courtesy ICDAS Foundation)

ICCMS™ combined categories (C)		Radiographic categories (R)				
	<i>RO</i> 	<i>RA<sub>1-2</sub></i> 	<i>RA<sub>3</sub></i> 	<i>RB</i> 	<i>RC</i> 	
<i>C<sub>Sound</sub></i> 	Sound <sub>CR</sub>	Initial <sub>CR</sub>	Initial <sub>CR</sub>	Moderate <sub>CR</sub>	Extensive <sub>CR</sub>	
<i>C<sub>Initial</sub></i> 	Initial <sub>CR</sub>	Initial <sub>CR</sub>	Initial <sub>CR</sub> <i>OR</i> moderate <sub>CR</sub>	Moderate <sub>CR</sub>	Extensive <sub>CR</sub>	
<i>C<sub>Moderate</sub></i> 	Moderate <sub>CR</sub>	Moderate <sub>CR</sub>	Moderate <sub>CR</sub>	Moderate <sub>CR</sub> <i>OR</i> extensive <sub>CR</sub>	Extensive <sub>CR</sub>	
<i>C<sub>Extensive</sub></i> 	Extensive <sub>CR</sub>	Extensive <sub>CR</sub>	Extensive <sub>CR</sub>	Extensive <sub>CR</sub>	Extensive <sub>CR</sub>	



**Fig. 2.4** (a) Visual, (b) radiographic, and (c) histological images of an approximal lesion (Courtesy of Dr Narisha Chawla, Melbourne, Australia)

images – this is especially relevant since high-resolution monitors are available at reasonable cost. Computer-assisted analysis using bitewing radiography can improve accuracy, especially when lesions are deeper than halfway through the enamel. Computer manipulation also allows magnification and software enhancement of images to suit various clinical needs.

The *in vitro* visual, radiographic, and histological images of an approximal lesion are illustrated in Fig. 2.4.

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## 2.5 New Detection Methods

The limitations of traditional methods such as poor reproducibility, reliance on experience to be accurate, and associated subjectivity can be supplemented with electronic methods of quantification which allow longitudinal monitoring of the progression/regression characteristics of the carious lesion. Many of these systems have been introduced to the marketplace over the past decade or so; however, there is still a need for further research to validate many of the systems which quantify the lesion numerically, since technique sensitivity and confounding factors such as clinician experience and ability to correctly interpret the results can affect validity.

### 2.5.1 Detection Based on Light Transmission

The physical principle is light, but with application in caries diagnosis, due to the specific wavelength (Table 2.3) used during the illumination, it can be divided into different categories:

- Laser fluorescence with the DIAGNOdent®/DIAGNOpen® and Canary System®
- LED fluorescence with the cameras VistaProof®, Soprolife®, and Soprocure®
- Quantitative light-induced fluorescence (QLF®)
- Transillumination with the camera DIAGNOcam®

**Table 2.3** Wavelength of the different diagnostic devices

Devices	Wavelength
DIAGNOdent/DIAGNOOpen®	655 nm
VistaProof®/Cam®	405 nm
Soprolife®	450 nm
Soprocure®	450 nm/480 nm
DIAGNOcam®	780 nm
Canary System®	655 nm (pulse laser)
QLF	290–405 nm

The light of the different devices (according to their specific wavelength) penetrates the tooth and is scattered or absorbed internally, depending on the crystalline and organic structure of the tooth and extrinsic molecules (e.g., foods, bacteria products, others). Different processes can be described and seen in Fig. 2.5 as direct or diffuse transmission, reflection, scattering, absorption, and backscattering [12].

Scattering: The direction of a photon is changed without loss of energy.

Absorption: Photon energy is converted into heat.

Backscatter: Photons leave through the same surface they entered.

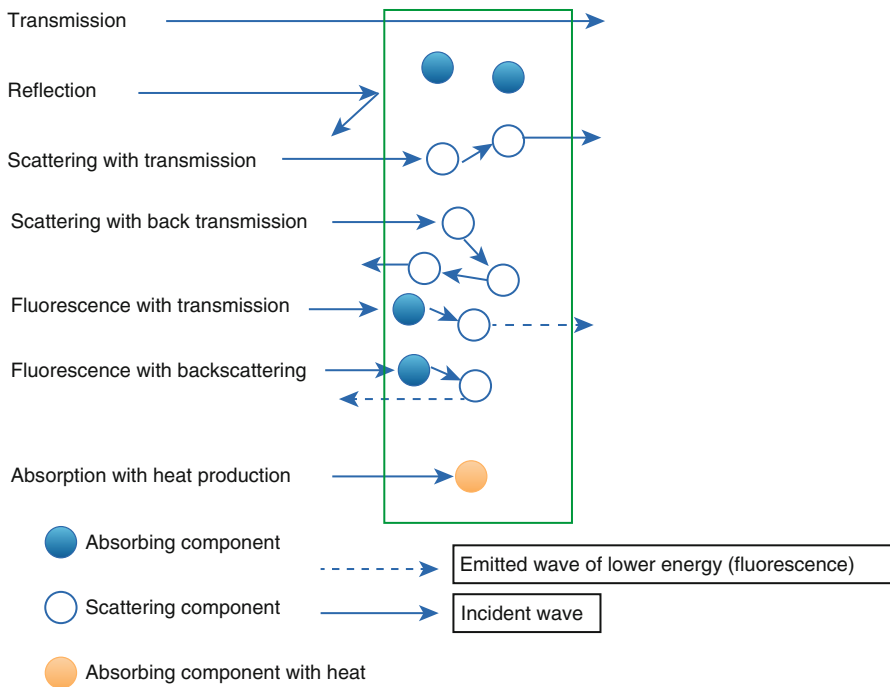
Diffuse transmission: Similar phenomena then backscattering with an irregular surface.

In enamel fluorescence is linked to the presence of fluorescent debris derived from food, bacteria, and the caries process; in dentine that is related to so-called autofluorescence which is observed during the caries process, and to explain the underlying phenomena, various hypotheses were suggested: modification of the collagen fibers, which are non-centrosymmetric structures with fluorescent properties, and/or accumulation of specific caries by-products and bacterial biofilm formation with specific endogenous porphyrins [13, 14]. The best-known by-products of the dental caries process are the so-called Maillard reaction products. The Maillard reaction (browning discoloration) is a nonenzymatic glycation of carbohydrate and protein, forming a brownish polymer of cross-linked proteins and advanced glycation end products (AGEs) [15–17]. Dentine collagen fibers are likely to link with AGEs over the long term, rendering dentine collagen more resistant to proteolytic breakdown and modifying its intrinsic fluorescence. Previous studies also focused on porphyrin and its derivatives in an attempt to link the autofluorescence signal of enamel and dentine caries with porphyrin sediment [18]. Primary teeth show more scattering giving a whiter or bluer appearance than permanent teeth dependant on the LED wavelength. The refractive index ratio between the scattering photon and its environment consisting of water and fluorescent molecules coming from bacteria and caries processes seems to be the key.

## 2.5.2 Transillumination Devices

Based on light transmitted through the tooth and combined with a CCD camera, these devices are more suitable for proximal caries detection.





**Fig. 2.5** Physical principles of light transmission

### 2.5.2.1 Fiber-Optic Transillumination

Fiber-optic transillumination (FOTI<sup>®</sup>, DIFOTI, Electro-Optical Sciences, Irvington, NY), and the more recent digitized DIFOTI<sup>®</sup> technique, uses light transmission through the tooth. The images can be stored and reexamined later.

FOTI was proposed as a valid, efficient alternative to bitewing radiographs in clinical trials of caries-prophylactic agents with a sensitivity of 0.73 and specificity of 0.99 [19]. In a study comparing unaided clinical diagnosis, bitewing radiographs, and FOTI, authors showed that the reproducibility of all three methods was acceptable with kappa values exceeding 0.6. All specificity values exceeded 0.95, but statistically significant differences were seen between sensitivities for clinical (0.38) and bitewing (0.59) diagnosis and between clinical and FOTI (0.67) examination, but not between bitewing and FOTI. It is concluded that the validity of FOTI is at least as high as that of bitewing radiology, and both are superior to unaided clinical diagnosis [20].

### 2.5.2.2 Fiber-Optic Transillumination with a Camera from KaVo<sup>®</sup>: DIAGNOcam<sup>®</sup>

Fiber-optic transillumination with a camera from KaVo<sup>®</sup>, DIAGNOcam<sup>®</sup> (KaVo Dental, Lake Zurich, IL, USA), a new system developed recently by KaVo, is also

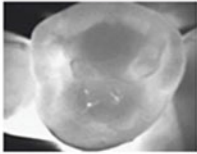

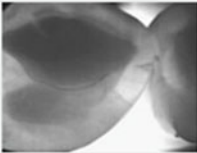

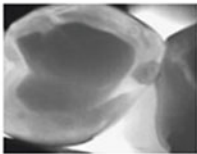

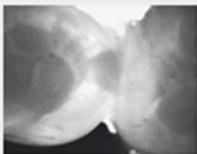

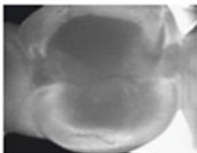

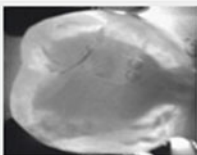

Table 2 Classification of proximal dentin caries lesions based on clinical experiences			
Description	NIR light	Caries extension	Consequences for diagnostics and therapy
0 Sound surfaces			Caries monitoring, no active care advised
1 First visible signs of enamel caries			Caries monitoring, preventive care advised
2 Established caries lesion			Caries monitoring, preventive care advised
3 Established enamel caries with an isolated spot reaching the dentin-enamel junction			Caries monitoring, preventive care advised
4* Dentin caries penetrating the enamel-dentin junction linearly			(Bitewing) Radiography, minimal operative care advised
5* Deep dentin caries lesion			(Bitewing) Radiography, operative care advised

Fig. 2.6 DIAGNOcam® classification [23]

based on simple transillumination. DIAGNOcam® uses a laser diode with 780 nm wavelength (near-infrared=NIR) for transillumination of the tooth. The DIAGNOcam® is placed directly on the tooth; the transilluminating light is positioned either side of the tooth and the picture captured; software allows storage of the picture. Caries scores are based on Fig. 2.6 [21, 22].

Cariou tissue scatters and absorbs more light than surrounding healthy tissue. A camera digitally images the light emerging from the opposite surface. The images are displayed on a monitor and stored. Cariou lesions appear like dark spots. This device is more suitable for proximal diagnosis [24]. Söchtig et al. evaluated

127 proximal lesions and concluded that NIR transillumination is a method that may help to avoid bitewing radiographs for diagnosis of caries in everyday clinical practice [25]. A new classification based on clinical experience and radiographs and comparison was therefore proposed with six scores. But in DIAGNOcam<sup>®</sup> images, the dental pulp is not visible, and the leading indicator seems to be the involvement of the dentinoenamel junction.

### 2.5.3 Laser Light and Fluorescence

The imaging techniques based on the fluorescent response of organic components of teeth have been developed for use in caries detection. The commercially available devices are as follows:

#### 2.5.3.1 Laser-Based Fluorescence System Only

##### The DIAGNOdent<sup>®</sup> (DIAGNOdent 2095, DIAGNOdent 2190, DIAGNOdent, KaVo Dental, Lake Zurich, IL, USA)

The DIAGNOdent<sup>®</sup> (DIAGNOdent 2095, DIAGNOdent 2190, DIAGNOdent, KaVo Dental, Lake Zurich, IL, USA) uses a laser at 655 nm wavelength, creating fluorescence in components such as porphyrins, and the intensity of the emitted fluorescent light is measured [7, 8, 26]. DIAGNOdent<sup>®</sup> values guide clinical decision-making (Table 2.4). After calibration of the instrument on a ceramic disk or adjacent sound enamel, the detection handpiece is placed on the tooth surfaces, and the device provides values that can be recorded. Two crystal probes, one for the occlusal and the other for proximal surfaces, are available.

In clinical situations the DIAGNOdent<sup>®</sup> scores often overestimate the extent of the lesions, with high variability. The main drawback of the DIAGNOdent<sup>®</sup> is the false-positive signal frequency [28]. This may be due to two reasons: the first is due to the persistence of debris in the deepest aspects of the pits and fissures if air polishing was not used, and the second is the size of the crystal probe which is larger than many pits and fissures. Repeated calibrations could be a shortcoming as well. DIAGNOdent<sup>®</sup>, compared to visual inspection, exhibits a sensitivity value that was always higher and a specificity value that was always lower, but the greater number

**Table 2.4** Laser fluorescence scores and clinical recommendations for DIAGNOdent<sup>®</sup> [27]

DIAGNOdent <sup>®</sup> score	Interval 1	Interval 2	Interval 3
<i>Occlusal and pits</i>	0–12	13–24	>25
Histological interpretation	Healthy tissue	Demineralized enamel	Dentine involved
Recommended therapy	Normal prophylactic care	Intensive prophylactic care	Minimally invasive operative care
<i>Proximal area</i>	0–7	8–15	>16
Recommended therapy	Normal prophylactic care	Intensive prophylactic care	Minimally invasive operative care

of false-positive diagnoses suggests that it should not be relied on as a clinician's primary diagnostic method [28–30]. Also increasing the confusion is that cutoff values differ between DIAGNOdent® and DIAGNOpen® [31, 32]. A recent meta-analysis of fluorescence-based methods comparing another fluorescence camera (VistaProof®, Dürr Dental, Germany) versus DIAGNOdent® or DIAGNOpen® confirmed that for these devices there was a trend of better performance in detecting more advanced carious lesions [33].

### **Fluorescence Aided for Caries Excavation (FACE, SIROInspect®, Sirona Dental Systems GmbH, Germany)**

Fluorescence Aided for Caries Excavation (FACE, SIROInspect®, Sirona Dental Systems GmbH, Germany) uses an excitation wavelength around 405 nm. The practitioner needs to use bespoke glasses to see the fluorescence in dentine. Limited information is presently available. The device appears suitable for identifying carious tissue for excavation. No images can be recorded with either system.

### **2.5.3.2 Combination of Camera and Fluorescence Systems**

#### **The QLF®**

The QLF® (QLF, Inspektor Research Systems BV, Amsterdam, the Netherlands), with emission in the wavelength region of 290–450 nm (Fig. 2.7), uses a xenon arc lamp and quantifies the change in transmission from the green fluorescence occurring in the dentine body due to microporosities in enamel as a result of the caries-related demineralization with a probe or a camera [34].

The development of the single-lens reflex (SLR) camera-based QLF-D (QLF-D, Inspektor Research Systems BV, the Netherlands) makes imaging of the whole dentition with a few images possible and incorporates the capture of white-light and fluorescence photos consecutively with the determination of red fluorescence. The SLR camera body (Canon model 450-D or up fitted with a 60 mm macro lens, Canon Inc., Tokyo) has an attached illumination and filtering tube (Biluminator, Inspektor Research Systems BV, Amsterdam) that consists of ring mounted violet-blue LEDs (405 nm) and white LEDs (broad spectrum, 6500 K) with filtering optics. The use of differential filtering techniques results in bright white luminescence of the healthy dentition, while carious lesions appear dark due to decreased fluorescence. The red fluorescence signal from mature plaque, calculus, and carious lesions is electronically enhanced. Quantification of carious lesions determines fluorescence loss ( $\Delta F$ ), lesion area in mm<sup>2</sup>, and level of red fluorescence.

Using a computer algorithm, it was determined that this nondestructive light fluorescence method was a sensitive and valid method for quantification of mineral loss in enamel caries lesions [34, 35]. It has also been demonstrated that this technique was relevant for smooth surface lesions but not yet for approximal lesions. The number of lesions detected on buccal surfaces of post-orthodontic patients by QLF far outnumbered those found by visual examination immediately after removal of fixed appliances [36].

**Fig. 2.7** The Inspektor™ Pro QLF, Inspektor Dental Care BV, the Netherlands



### The Canary®

The Canary® (Quantum Dental Technologies Inc., Ontario, Canada) is a pulse-laser-based system that uses a camera and a combination of heat and light (frequency-domain photothermal radiometry and modulated luminescence, FD-PTR and LUM) putatively examining the crystal structure of teeth and mapping carious lesions [37].

Principles:

- Pulses of laser light in a range of on-off frequencies hit the tooth surface.
- The tooth glows (LUM for luminescence) and releases heat (PTR for photothermal radiometry) at those same frequencies.
- PTR provides a depth profile of tooth properties by varying the frequency of the laser beam.
- The detected LUM and PTR signals combined reflect the tooth's condition.

Canary® scale from 0 to 100: 0–20 healthy/sound tooth structure, 21–70 initial lesions, and 71–100 advanced lesions

By varying the pulse of the laser beam, a depth profile of the tooth can be created to permit detection of decay as deep as 4–5 mm from the tooth surface and as small as 50 microns in size. Simultaneous measurement of the reflected heat and light provides information on the presence and extent of tooth decay below the tooth surface before being detected by dental radiographs. The device is promoted to provide early detection of small occlusal cavities, fissure, smooth surface, and

interproximal carious lesions, therefore being advantageous in comparison to traditional approaches for detection and monitoring early carious lesions [38, 39]. The system is claimed also to detect secondary carious lesions around the visible margins of resin composite [40]. This might reduce the cost barriers to dental services by treating emerging “cavities” before invasive and more expensive treatment is necessary. The handpiece is applied on the tooth: pictures and Canary<sup>®</sup> values are given by the system. Protective glasses are recommended [41–44].

### Soprolife<sup>®</sup> Camera

Soprolife<sup>®</sup> camera (Acteon, La Ciotat, France) [45] (Fig. 2.8). This intraoral camera utilizes two types of LEDs to illuminate tooth surfaces in the visible domain, either in the white light region or in a narrow band (wavelength 450 nm with a bandwidth of 20 nm). The camera is equipped with an image sensor (0.25 inch CCD sensor) consisting of a mosaic of pixels covered with filters of complementary colors. The data collected, relating to the energy received by each pixel, allows retrieval of a tooth image. The camera operates in three modes: For daylight mode four white light LEDs are engaged; for the diagnostic and treatment modes, the light is provided by four blue LEDs (450 nm). A new camera, Soprocure<sup>®</sup> (Fig. 2.8), provides three clinical modes as well: daylight, caries, and periodontal modes. The second mode focuses on enamel and dentine carious lesions, and the latter on periodontal inflammation.

Soprolife<sup>®</sup> and Soprocure<sup>®</sup> putatively reveal AGEs produced from Maillard reactions [46–48].

Soprolife<sup>®</sup> and Soprocure<sup>®</sup> clinical applications are based on the LIFEDT concept (light-induced fluorescence evaluator for diagnosis and treatment; Sect. 2.6) [27]:

- The tooth can be observed in daylight and fluorescence mode with a high level of magnification (x 35).
- Any modification of the reflected light from dentine or enamel in comparison to a healthy area can be detected.
- Clinical decisions are not linked to numerical values, but the system improves visual inspection and informs decision-making.

The Soproiaging<sup>®</sup> software makes it possible to record and compare the pictures. The camera is positioned on the tooth, with magnification and mode (daylight or fluorescence) selected. Pictures are recorded with the specific Soproiaging<sup>®</sup> software. Carious lesion score codes were previously described as follows [49]:



**Fig. 2.8** Camera Soprolife<sup>®</sup> (black) and Soprocure<sup>®</sup> (white)

## Soprolife® Daylight Codes for Coronal Caries

### Code Description

- 0: Sound, no visible change in the fissure.
- 1: Center of the fissure showing whitish, slightly yellowish change in enamel, limited to part or all of the base of the pit and fissure system.
- 2: Whitish changes come up the slopes (walls) toward the cusps; the change is wider than the confines of the fissure, seen in part or all the pit and fissure system; no enamel surface breakdown is visible.
- 3: Fissure enamel is rough and slightly open with beginning slight enamel breakdown; changes are confined to the fissure and do not need to come up the slopes, no visual signs of dentinal involvement.
- 4: Caries process is not confined to the fissure width and presents itself much wider than the fissure; changed area has a “mother-of-pearl” glossy appearance.
- 5: Enamel breakdown with visible open dentine.

## Soprolife® Blue Fluorescence Codes for Coronal Caries

### Code Description

- 0: Sound, no visible change in enamel (rarely a graphite-pencil-colored thin line can be observed) shiny green fissure.
- 1: Tiny, thin red shimmer in the pits and fissure system can come up the slopes, no red dots visible.
- 2: In addition to tiny, thin red shimmer in pits and fissures possibly coming up the slopes, darker red spots confined to the fissure are visible.
- 3: Dark red extended areas confined to the fissures; slight beginning roughness.
- 4: Dark red or orange areas wider than fissures; surface roughness occurs, possibly gray or rough gray zone visible.
- 5: Obvious wide openings with visible dentine.

When dentine is involved from score 3, variations of autofluorescence follow the clinical dentine color guide (Table 2.5).

With improved technologies, it is important to state that the use of high magnification should not lead to excessive operative intervention. Studies confirmed that the Soprolife® camera and other devices were strongly correlated with lesion histology [50, 51], and as a result of a comparison between an unaided visual examination and an operating microscope, the use of magnification improved the restorative treatment decisions regarding posterior occlusal surfaces. The usefulness of

**Table 2.5** Clinical dentine color guide used with the LIFEDT concept and the Soprolife® device

Camera visual inspection	Healthy dentine	Infected dentine	Affected dentine Active process (soft yellow tissue)	Affected dentine Arrested process (brown tissue, quite hard)
Soprolife®	Acid green	Dark gray	Bright red	Dark red
Soprocure®	Gray	Dark gray	Bright red	Dark red

magnification in odontology and more specifically in cariology was also confirmed [52, 53]. Moreover, visual performance decreased with increasing age of the operator, and magnification aids can compensate for visual deficiencies, and the inter- and intra-weighted kappa values showed better results for the fluorescence camera (in daylight and in fluorescence modes) compared to the DIAGNOdent® [54]. When sensitivity and specificity were calculated, the grouping of no lesion/healthy and pre-cavitated lesions together appeared to be the best cutoff point to determine the sensitivity and specificity of each method [55]. Selecting this cutoff point, DIAGNOdent® achieved a sensitivity of 87% with a specificity of 66%. At the same cutoff point, Soprolife® in daylight mode exhibited a slightly higher sensitivity (93%) and a slightly lower specificity (63%). In the blue fluorescence mode, the sensitivity of the Soprolife® was slightly higher, up to 95%, but the specificity decreased to 55%. In a very recent study based on 219 permanent posterior teeth measurements, the authors concluded that Soprolife® is a reproducible and reliable assessment tool. At a specific cutoff point, categorizing non-carious lesions and visual change in enamel, Soprolife® has high sensitivity and specificity [56].

### VistaCam® Camera

VistaCam® camera (Classic, CL, and IX) is an intraoral fluorescence camera (Dürr Dental, Bietigheim-Bissingen, Germany) that illuminates teeth with a violet light (405 nm) and captures the reflected light as a digital image. The reflected light is filtered for light below 495 nm and contains the green-yellow fluorescence of normal teeth with a peak at 510 nm, as well as the red fluorescence of bacterial metabolites with a peak at 680 nm. The software (DBSWIN version 5.3) quantifies the green and red components of the reflected light on a scale from 0 to 3 as a ratio of red to green, showing the areas with a higher than healthy tooth ratio. A new version, the VistaCam® CL-IX with removable head camera, wireless, and a light cure function, has been launched [57, 58]. The camera is placed onto the tooth; pictures are recorded with bespoke software, which then reveals the caries scores (Table 2.6).

The intraoral fluorescence camera VistaProof® (Dürr Dental, Bietigheim-Bissingen, Germany) was developed for caries detection and emits blue light at 405 nm and captures fluorescence images from dental surfaces. The specific software filters and quantifies the fluorescence emitted by the tissue and converts the relationship between green and red fluorescence into numerical values, according to the pixel numbers in each image (Table 2.6). The performance in recent studies was in the range of the DIAGNOdent® [59–63]. In a recent study, the occlusal surfaces of 53 teeth (99 investigation sites) were examined by two

**Table 2.6** VistaProof/Cam® scores and color scales versus the histological scale

0-1	1-1.5	1.5-2	2-2.5	2.5<
Healthy enamel	Initial enamel caries	Deep enamel caries	Dentine caries	Deep dentine caries



examiners with different levels of experience in cariology (one experienced dentist, one final-year dental student) using the VistaProof® [57]. The intra-class correlation coefficients for inter- and intra-examiner reproducibility for the fluorescence-based examinations were 0.76–0.95, and there was a significant correlation between the fluorescence and histological examinations for both examiners ( $r_s = 0.47$  and  $0.55$ ,  $P < 0.01$ ). The VistaProof® has the ability to demonstrate high reproducibility and good diagnostic performance for the detection of occlusal carious lesions at various stages of the disease process. However, the quality of the picture could be improved.

#### 2.5.4 Various Devices

Optical coherence tomography (OCT®, Dental Imaging System, Lantis Laser, Denville, NJ, USA) is a nonionizing imaging technique that can produce cross-sectional images of biological tissues using an infrared light at 1310 nm. Only in vitro studies are available, and often the images are limited to the depth of enamel. The polarized sensitive OCT® (PS-OCT) can be correlated with the degree of demineralization and lesion severity. Monitoring in vivo carious lesion changes could be helpful with this device [64–66]. Combination of near infrared (NIR) radiation imaging and PS-OCT acquired depth-resolved images. Combining this technology with a short-pulsed CO<sub>2</sub> laser ablation system would allow for the selective removal of demineralized carious tissue [67]. Sensitivity, specificity, and advantages of the main different devices are summarized in Table 2.7.

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### 2.6 Clinical Concept When Using Soprolife®: Light-Induced Fluorescence Evaluator for Diagnosis and Treatment (LIFEDT)

This clinical concept is built on five principles:

1. Depending on the diagnostic aids used, your practice will balance between minimally invasive treatment 1 (MIT1) (no drill) and minimally invasive treatment 2 (MIT2) (drill) (Sect. 2.6.1).
2. Carious lesions are minor factors; lesion activity and the presence of cavitation are the main factors.
3. Cleaning the deepest part of the pits and fissures without damaging the enamel is mandatory (Sect. 2.6.2).
4. Fluorescence variations help to evaluate carious lesion activity and excavate the infected dentine tissue.
5. Ad hoc-sized tools have to be smaller than the width of the cavity. If not, sealing is preferable.

**Table 2.7** Sensitivity and specificity of the main different devices [12, 49, 55]

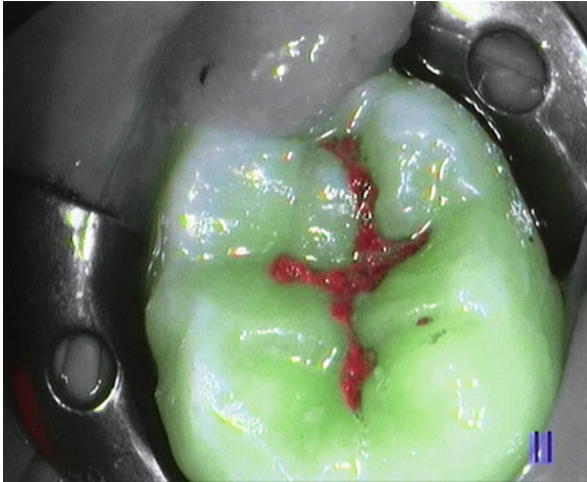
Devices	Sensitivity	Specificity
Electronic caries monitor®	0.65	0.73
Visual inspection	0.6	0.73
Fiber-optic transillumination®	0.73	0.99
Bitewing	0.7	0.80
QLF®	0.5–0.68	0.7–0.9
Spectra® or VistaCam®	0.92	0.37
SIROInspect®	0.94	0.83
DIAGNOdent®	0.87	0.5
Soprolife®	0.93	0.87

### 2.6.1 Clinical Recommendation for the Diagnostic Process

For carious lesion diagnosis, the use of a combination of detection aids is still recommended [27]. Whatever numerical values given by the device, visual inspection remains an essential component in making the final decision: to drill or not to drill [68, 69]. Indeed, it is more on the complexity of the shape, depth, caries activity, and the width of the grooves which governs clinical decision-making, meaning that before the diagnostic step, the pits need to be perfectly clean without being damaged. The general philosophy of the patient-centered approach is of importance too. Indeed, all the techniques described below should be applied within a modern medical approach of determining and managing the patient's caries risk, by applying the CAMBRA system or other similar assessment systems [70–73]. The first threshold of invasive intervention may occur at a lesion with first visible enamel breakdown (ICDAS score 3). The choice between advising preventive care and preventive with operative care, respectively, will be based on this decision and changed depending on the diagnostic devices used [48]. Consequently, preventive and minimally invasive therapies can be divided into two groups: The first (minimally invasive treatment 1 or MIT1) is for treating enamel and enamel-dentine lesions without any preparation (“noninvasive”), provided that there is no gross surface cavitation. The second group (minimally invasive treatment 2 or MIT2) is for treating early enamel-dentine lesions with surface cavitation.

### 2.6.2 Professional Prophylactic Cleaning Steps

This step currently remains one of the most complicated [27, 74]. In fact an ad hoc diagnosis assumed that the deepest part of the groove was perfectly cleaned without damage to the demineralized enamel, providing an overview of an area around 0.1 mm wide in dry conditions. Without clear evidence, we simply limit our purposes to clinical advice. As the crystalline structure is highly unstable and the average width of the pits is around 0.1 mm, the use of sharp probes and burs is strictly forbidden, and cleaning with a rotating brush in combination with prophylactic paste could disturb the values given by the different diagnostic devices. One



**Fig. 2.9** Occlusal picture with Soprolife® camera in fluorescence mode

reasonable clinical proposition is to clean with an air-polishing device using sodium bicarbonate powder as the abrasive (AirNGO®, Kavoprophy®, EMS®, NSK®). The use of slightly harder calcium carbonate powder (Pearl powder®, Acteon Satelec, France; Kavoprophy® prophy powder, KaVo, Germany) is also suitable. Precautions are needed to reduce overflow of the powder (high suction, dental dam). In the case of a high-risk patient with no monitoring possibilities or change in risk unlikely, the decision can be made to seal the groove. Syc® powder, in this particular case (OSSpray®, Abbottstown, USA), selectively removed the demineralized enamel, but a special nozzle is needed due to the extreme hardness of the powder [75]:

- Case 1 shows sandblasting with a calcium carbonate powder using air abrasion where application of a sealant was recommended (Figs. 2.9, 2.10, 2.11, and 2.12).
- Case 2 shows sandblasting with a bioactive glass Syc® powder (Figs. 2.13 and 2.14).

### 2.6.3 The LIFEDT Concept: Treatment of Initial to Moderate Stage of Caries

The challenge is early diagnosis of enamel demineralization, before cavitation occurs, and consequently to manage your clinical decisions depending on the individual's caries risk. Combination of the CAMBRA® approach and LIFEDT could certainly help to better understand this clinical approach (see also Chap. 9). Based on daylight and fluorescence observations coupled with the high magnification, a decision-making diagram is as follows: with a smooth surface, use a polishing cup with prophylaxis paste which removes stains and plaque and polishes the tooth surface [70]. Choose a fine grit, as the crystalline structure is highly unstable, to

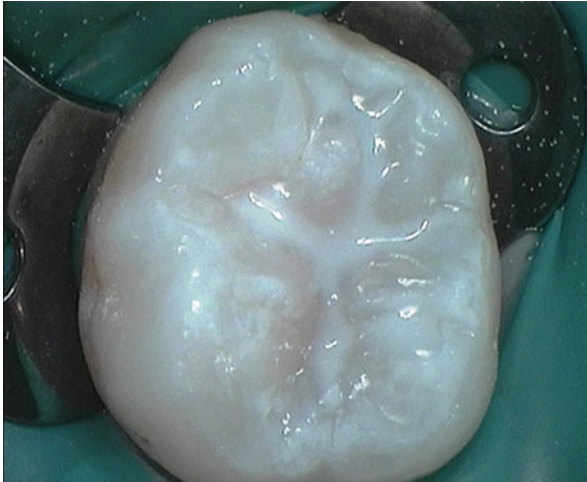


**Fig. 2.10** After 10 s of carbonate calcium air abrasion



**Fig. 2.11** Focus macro-mode in daylight at the end of the cleaning steps

avoid causing any unnecessary abrasion of the tooth's structure and restoration surfaces. Some pastes contain fluoride and should be free of known allergens (e.g., Flairesse<sup>®</sup> prophylaxis paste, DMG, Germany). Furthermore, the LIFEDT system does not address the issue of bacterial load. In the presence of a high-risk oral ecology, any fissured groove and any modification in the natural fluorescence serve as an indication to implement the LIFEDT concept. Until the evidence of surface breakdown and dentinal damage by caries is definitely diagnosed using radiography and other diagnostic aiding cameras, the LIFEDT concept recommends preventive sealing rather than a conventional irreversible mechanistic approach of drilling.



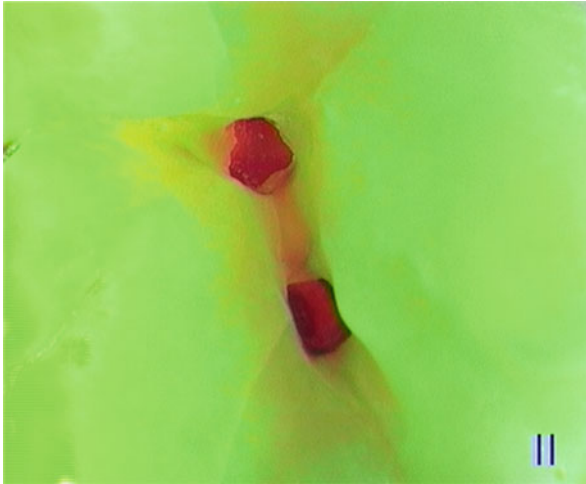
**Fig. 2.12** Sealant application



**Fig. 2.13** Daylight view with SoproLife® camera

#### **2.6.4 The LIFEDT Concept Applied to Occlusal Decay (Case 2: Figs. 2.13 and 2.14)**

Discussions about an appropriate cutoff point to determine operative intervention deserve our attention, but it seems reasonable to intervene restoratively from a score of 4/5 (moderate stage) depending on the width, depth, and cavity shape, as well as the fluorescence information retrieved. The sensitivity and

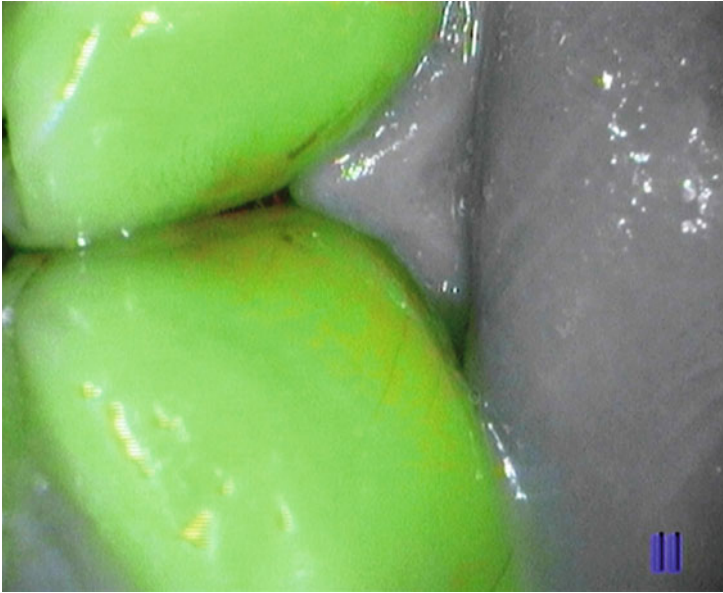


**Fig. 2.14** After 20 s of Sylec® powder air abrasion

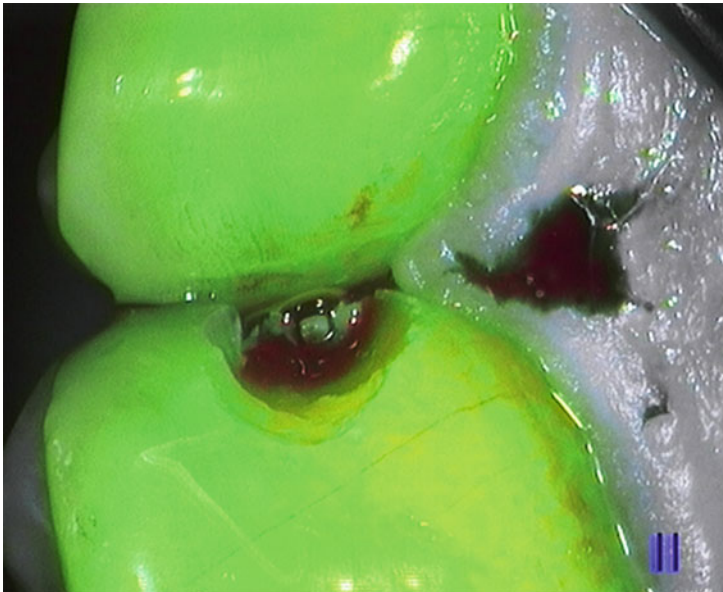
specificity of each device will shift this cutoff point; therefore, the principle is first to gently clean the pits (applicable for all devices) and then observe any modification of the structure and shape of the pits in daylight and fluorescence mode. Systems giving carious lesion scores with no visual inspection can be used complementarily.

### **2.6.5 The LIFEDT Concept Applied to Proximal Caries Diagnosis**

The major difficulty with the proximal tooth surface is associated with the possible presence of a carious lesion in the proximal zones. Even though bitewing radiographs give relatively good information, misdiagnoses are sometimes unforeseeable. In some clinical situations, we propose separating the teeth as far as possible using powerful plastic wedges or orthodontic separators; when using the Soprolife® camera in daylight mode and fluorescence mode, it might be possible in most cases to view cavitation and red shadow revealing the presence of a carious lesion (Figs. 2.15 and 2.16). Transillumination with DIAGNOcam® (Fig. 2.6) could also be helpful. To objectively balance your clinical decision between MIT1 and MIT2 in proximal areas, a combination of radiographs, fluorescence, laser, and magnification could help to achieve better identification. One of the main advantages of the fluorescence image is that before performing a slot preparation, topical information about the point of entry of the carious lesion are given, meaning more or less on the vestibular, palatal, or lingual side (Figs. 2.15 and 2.16).



**Fig. 2.15** Red shadow visible in proximal area with SoproLife® in fluorescence mode (vestibular side)



**Fig. 2.16** Confirmation of the proximal caries in fluorescence mode with SoproLife® camera (vestibular preparation)

## 2.7 Final Remarks

The use of visually based detection methods (including magnification and transillumination) in conjunction with radiography (when needed and digital if possible) is still the mainstay of carious lesion diagnosis. The development of electronically based detection and quantification methods has created the opportunity to use these devices to increase the validity of the diagnostic process in conjunction with the “gold standards.” However, if the instruments are not used in a rigorously standardized manner and if the limitations of the specific instrument are not understood, these can often lead to overdiagnosis and subsequent unnecessary clinically destructive intervention.

Therefore the clinician should rely on the traditional methods of detection as the primary method, with the use of adjunct electronic methods assisting the process. At this point in time, no single electronic instrument has suitable sensitivity and specificity with respect to lesion detection and quantification to be used in isolation.

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# The Assessment of Carious Lesion Activity and Caries Risk

# 3

Soraya C. Leal and Bente Nyvad

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## Abstract

Carious lesion activity assessment and caries risk assessment (CRA) are essential tools that assist professionals in proper caries management. This is to say that the management of carious lesions, the prescription of caries control measures, and the frequency with which these measures are applied vary according to the patient's current caries activity and caries risk profile. Carious lesion activity should be assessed with the Nyvad criteria which have shown prognostic value in clinical trials and which guide the professional to the most appropriate treatment decision. Assessment of caries risk is not always straightforward. Although many attempts have been made to define which factor(s) should be taken into account to better predict new carious lesions, many of the CRA tools currently available have limited validity. When a single factor is used for predicting carious lesions, the patient's past caries experience has been shown to be the most accurate predictor factor. The aim of this chapter is to summarize the evidence about CRA and carious lesion activity assessment and to discuss, through providing a few clinical cases as examples, the importance of these concepts in caries management.

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## 3.1 Carious Lesion Activity Assessment

### 3.1.1 Rationale for Using Carious Lesion Activity Assessment

Every time a patient opens his or her mouth in the dental office, the dentist checks for cavities. Up until the 1990s, cavity formation was the common threshold for diagnosing carious lesions in clinical practice, essentially because filling the teeth was considered the best way to stop further lesion progression. However, following the introduction of fluoride toothpaste worldwide and the accompanying caries decline, it was recommended that caries should also be diagnosed at the non-cavitated level to capture earlier signs of caries [1]. Various diagnostic criteria were developed for this purpose, among which the International Caries Detection and Assessment System (ICDAS) criteria are the most strongly promoted [2]. The ICDAS system is correlated with progressive stages of lesion penetration [3] which is the reason that it may provide information about lesion depth. Unfortunately, for the dentist and the patient, lesion depth does not necessarily reveal anything about the rate of lesion progression. Hence, based on the ICDAS criteria alone, dentists do not learn about the prognosis of lesions.

An alternative classification, the Nyvad criteria (Table 3.1), has circumvented this problem. In addition to cavitated/non-cavitated stages of carious lesions, these criteria score the activity of lesions. The Nyvad criteria were properly validated in longitudinal trials and showed predictive validity for lesion activity, both at site level and at subject level [4, 5]. This indicates that the criteria have prognostic value and are useful for clinicians when identifying lesions and patients in need of treatment. In a high-caries population, it was shown that over 3 years, active non-cavitated lesions had a higher risk of progressing to a cavity than inactive non-cavitated lesions which again had a higher risk of progressing to a cavity than sound surfaces. The Nyvad criteria are so far the only diagnostic criteria that have been validated for lesion activity states in clinical longitudinal studies. The reliability of these criteria is comparable to that of other diagnostic systems that include non-cavitated lesions when tested in both children and adolescents [6, 7].

Table 3.1 provides a detailed description of the criteria associated with the Nyvad individual scores, including scores for secondary caries [6].

Because of the prognostic properties of the Nyvad criteria, this lesion classification immediately guides the practitioner to the most relevant treatment decision (Fig. 3.1). Owing to their poor prognosis, all active lesions should be treated professionally. Active lesions that cannot be cleaned properly by the patient (such as deep cavities) should be treated with a filling, whereas active non-cavitated lesions with or without micro-cavitation may as a first choice be treated with nonoperative interventions. The nonoperative treatment, such as improved oral hygiene with fluoride toothpaste and/or modification of the diet, should reflect the particular risk factors of the patient (see later). In some cases where risk factors (e.g., biofilm, saliva, or diet) cannot be properly controlled, it may also be necessary to apply topical fluorides. In all cases, the effect of the nonoperative interventions on caries activity should be monitored over time and proper reinstruction given when needed. A more

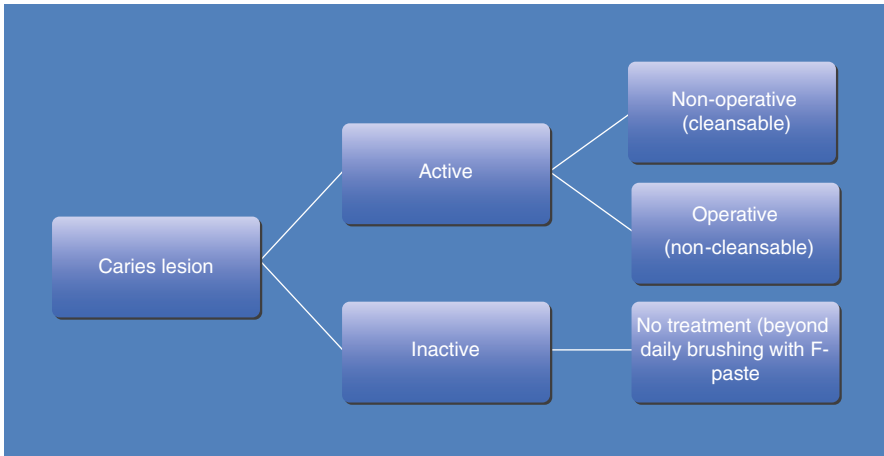
**Table 3.1** Description of the Nyvad criteria for caries lesion activity assessment [6]

Score	Category	Criteria
0	Sound	Normal enamel translucency and texture (slight staining allowed in otherwise sound fissure)
1	Active caries (intact surface)	Surface of enamel is whitish/yellowish <i>opaque</i> with <i>loss of luster</i> ; feels <i>rough</i> when the tip of the probe is moved gently across the surface; generally covered with plaque. No clinically detectable loss of substance. Smooth surface: caries lesion typically located close to gingival margin. Fissure/pit: intact fissure morphology; lesion extending along the walls of the fissure
2	Active caries (surface discontinuity)	Same criteria as score 1. Localized surface defect (micro-cavity) in enamel only. No undermined enamel or softened floor detectable with the explorer
3	Active caries (cavity)	Enamel/dentin cavity easily visible with the naked eye; surface of cavity feels <i>soft</i> or <i>leathery</i> on gentle probing. There may or may not be pulpal involvement
4	Inactive caries (intact surface)	Surface of enamel is whitish, brownish or black. Enamel may be <i>shiny</i> and feels <i>hard</i> and <i>smooth</i> when the tip of the probe is moved gently across the surface. No clinically detectable loss of substance. Smooth surface: caries lesion typically located at some distance from gingival margin. Fissure/pit: intact fissure morphology; lesion extending along the walls of the fissure
5	Inactive caries (surface discontinuity)	Same criteria as score 4. Localized surface defect (micro-cavity) in enamel only. No undermined enamel or softened floor detectable with the explorer
6	Inactive caries (cavity)	Enamel/dentin cavity easily visible with the naked eye; surface of cavity may be <i>shiny</i> and feels <i>hard</i> on probing with gentle pressure. No pulpal involvement
7	Filling (sound surface)	
8	Filling + active caries	Caries lesion may be cavitated or non-cavitated
9	Filling + inactive caries	Caries lesion may be cavitated or non-cavitated

extensive description of the principles of nonoperative caries management is provided in a recent review [8].

### 3.1.2 How to Perform Caries Lesion Activity Assessment

In contrast to other diagnostic systems, the Nyvad criteria use surface-based visual-tactile assessments. This implies that the surface characteristics of the lesions are



**Fig. 3.1** Decision tree for treatment of caries based on lesion activity assessment

recorded without consideration of lesion depth. Two clinical parameters are recorded for each lesion:

- Cavitation stage (presence or absence of a cavity)
- Activity stage (active or inactive, assessed by visual-tactile examination of the surface features of the lesion)

Figure 3.2 illustrates the clinical features of some typical scores of the Nyvad criteria. The illustrations on the left row show increasing stages of severity of active lesions (non-cavitated to micro-cavitation to cavitation), while those on the right row show comparable stages of inactive lesions.

According to the Nyvad classification [6], the caries examination is performed with a sharp metal probe after the tooth is dried with a blast of air. The probe is used to remove biofilm from plaque stagnation areas (with the side of the probe) and to “feel” the surface roughness and hardness of the lesion gently (with the tip of the probe). It is important for the practitioner to apply a loose grip on the probe in order not to damage the surface layer of the lesions. If it is obvious from visual inspection that a lesion is either active or inactive, no probing is needed. In the case of several lesions on the same surface, only the most severe lesion is recorded; an active lesion is considered more severe than an inactive lesion, and a cavitated lesion more severe than a non-cavitated lesion. In some cases, it may be difficult to assess the activity of the lesion properly because the lesions may be at a transitory stage between active and inactive. In such cases, the practitioner should, for prognostic reasons, record the most severe diagnosis which is the active lesion. If “mixed,” lesions contain elements of both active and inactive carious lesions should be scored as active.



**Fig. 3.2** (a) Presents a classic example of an active non-cavitated lesion on the mesial surface of 65. The surface is a whitish opaque color due to active caries processes, and the surface “feels” rough when the tip of a probe is run across the lesion (b) shows a non-cavitated occlusal lesion that has become inactive where the surface of the lesion is shiny and feels smooth on gentle probing (c) shows a micro-cavitated active lesion with sharp demineralized enamel borders while the cavity borders in (d) are rounded. An active carious lesion is shown in (e) a soft area in the mesial fossa is observed which has completely undermined the occlusal surface (f) presents an inactive cavity that has become hard because of long-standing mineral deposition on the surface layer (so-called remineralization)

When diagnosing caries, it may be relevant also to consider the risk of misclassification. For example, not all white or brown spots represent caries. In water-fluoridated areas, it may sometimes be difficult to differentiate between mild cases of dental fluorosis and arrested carious lesions, in particular when lesions are located close to the gingival margin. Demarcation characteristics of



the fluorotic lesions that reflect the perichymatal pattern and the symmetric occurrence on homologous teeth should help the practitioner to arrive at the appropriate diagnosis [9]. Another differential diagnostic problem relates to molar incisor hypomineralization that may erroneously be classified as cavitated carious lesions.

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## 3.2 Caries Risk Assessment

Caries risk assessment (CRA) is defined as the probability of an individual developing new carious lesions during a specific time [10]. Although the definition of CRA seems to be very simple, in practice, it is not. Dental caries is a multifactorial disease that, according to the balance between risk and protective factors that are present in mouth for a certain time, can be controlled in a way that signs of the disease will never be clinically detected; it can progress slowly. It can progress very fast, or it can be arrested. For this reason, emphasis is first given to the factors that positively or negatively impact dental caries development and progression. After this, the instruments that have been developed in the last decades to assist the clinician in performing CRA in a standard way are discussed.

Historically, dental caries has been defined as an infectious and transmissible disease that occurs when three major factors are present: a susceptible tooth surface, fermentable carbohydrates (especially sucrose), and cariogenic bacteria [11]. Nevertheless, the current understanding that the dental community has about the disease is that many other factors are involved in this process, making it dynamic and causing it to last a lifetime.

On the basis of the knowledge acquired through microbiological research in the last years, the concept of dental caries as an infectious disease is considered old-fashioned and questionable [12, 13]. Indeed, for carious lesions to develop, the presence of cariogenic bacteria is mandatory, but the presence of those bacteria in the mouth does not guarantee that a carious lesion will develop. Therefore, in this chapter, dental caries is defined as a complex interaction between cariogenic bacteria within the biofilm and fermentable carbohydrates, which over time mediated by many factors (such as behavior and life style) will lead to an imbalance of the de- and remineralization processes at the tooth-biofilm interface. The net result of these processes may or may not be frank cavitation [14, 15].

Some of the factors involved in the caries process are considered protective (saliva and its components: fluoride, calcium, phosphate), while others are considered pathological factors (bacteria, frequency of ingestion of fermentable carbohydrates, and reduced salivary function) [16]. The dynamic balance between demineralization and remineralization, modulated by these factors, will determine whether a carious lesion will progress or will be arrested [17]. This means that the most important aspect in this equation is the balance among protective and pathological factors. Hence, for example, the regular presence of fluoride in the mouth may not be enough to prevent the development of caries, when other factors, such as high consumption of sugar and lack of proper hygiene, are present.

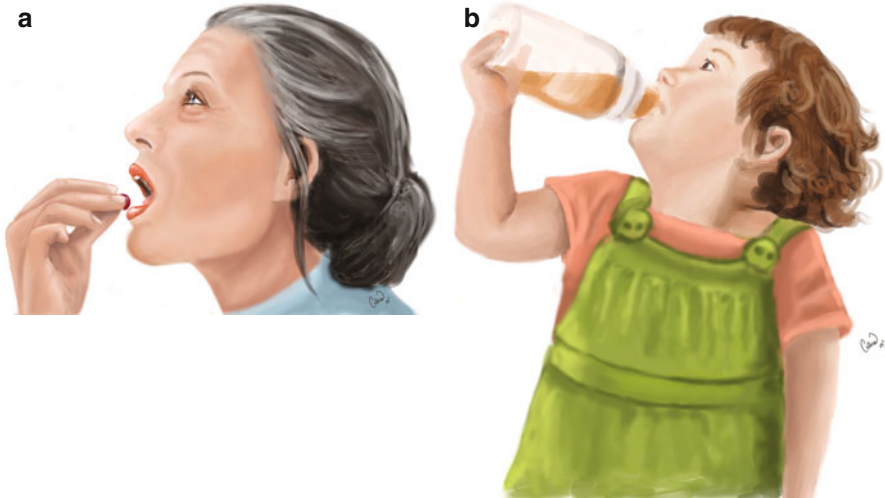
**Table 3.2** Most frequent factors investigated in relation to the development of dental caries

<p><b>Saliva and microbiology</b></p> <ul style="list-style-type: none"> <li>• Salivary flow</li> <li>• Buffer capacity</li> <li>• Mutans Streptococci count</li> <li>• Lactobacilli count</li> </ul> <p><b>Sociodemographic</b></p> <ul style="list-style-type: none"> <li>• Age</li> <li>• Ethnicity</li> <li>• Socio-economic status</li> </ul> <p><b>Behavioral</b></p> <ul style="list-style-type: none"> <li>• Diet</li> <li>• Fluoride exposure</li> <li>• Frequency of dental visits</li> <li>• Toothbrushing</li> </ul>	<p><b>Clinical characteristics</b></p> <ul style="list-style-type: none"> <li>• Oral hygiene               <ul style="list-style-type: none"> <li>- Visible plaque</li> <li>- Gingival bleeding</li> </ul> </li> <li>• Caries experience</li> <li>• Proximal enamel lesions</li> <li>• Orthodontic apparatus</li> <li>• Enamel development defects</li> <li>• Tooth morphology</li> <li>• General health</li> <li>• Use of medication</li> </ul>
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Many attempts to identify factors that can influence the caries balance resulted in the identification of caries *risk factors* and caries *risk indicators*. On the basis of literature [18–21], we have identified some indicators and risk factors that have been investigated in relation to the development of dental caries (Table 3.2). Risk factors are defined as those factors that, when present, directly increase the chance of a disease occurring, as confirmed by longitudinal studies [22], while risk indicators are factors that are detected in cross-sectional studies and are considered putative or potential risk factors [23].

It is believed that many clinicians worldwide collect information in regard to the factors shown in Table 3.2 to establish their treatment plan. However, whether they do this in a standardized manner and/or whether they use this information to set up their patients' recall intervals are questionable. According to two studies that aimed to assess the use of CRA in children and adults patients, the majority of the dentists interviewed stated that they usually perform CRA. However, less than 14% of the dentists that treat children reported that they use a special form on which to record the information retrieved [24]. In regard to dentists that treat adults, a weak correlation was observed between the CRA performed and the use of individualized preventive regimens [25]. A consensus existed regarding the factors that are taken into account when performing CRA by the two groups of dentists. The factors rated as most important were current oral hygiene level, decreased salivary flow, and the presence of active carious lesions [24, 25].

Clinicians that intend to guide their patients in controlling the carious process and/or to predict whether the disease will occur in the near future need to check whether *factors* that modulate the disease development and/or progression are present. This is the foundation of CRA.



**Fig. 3.3** Examples of an elderly person and a child being exposed to caries risk factors that are particularly dominant in their specific age group: (a) medication and (b) bottle with sugar content (Illustrated by Gökçe Taniyan)

### 3.2.1 Caries Risk Assessment: Not Everyone Is Exposed to the Same Risk Factors

Caries prediction, as the reader might have noticed already, is not an easy task considering the multifactorial etiology of the disease. To make matters even more complicated, the clinician should be aware that patients are usually exposed to different caries risk factors and indicators during their lives. This means that caries risk can vary over time, depending on age, individual behavior, and life style [15]. To better understand this concept, we use the extremes of a person's lifecycle, childhood and old age (Fig. 3.3), as examples.

The probability of a child presenting salivary problems, such as a reduced salivary flow rate, is very low, except in cases of rare diseases such as parotitis epidemica [26]. On the other hand, changes in salivary function seem to increase with the intake of certain types of medication which are frequently prescribed to elderly people [27]. This makes salivary secretion rate a much more frequent caries risk factor for the elderly than for young children.

Fatalistic beliefs of parents about oral health measured by asking them whether they agreed with the statement “most children eventually develop dental cavities,” have been found to be a strong caries predictor [21]. Thus, when children are targeted, not taking into account their caregivers' opinion might lead to an underestimation of the children's caries risk. When all this information is combined, it is possible to conclude that some factors, such as the frequency of sugar intake, will have a great influence on the disease development independently of the patient's age, while other factors, such as salivary secretion rate, may be more important in older people.

### 3.2.2 Caries Risk Assessment Tools

A literature search on PUBMED using the key words “caries risk assessment” indicated that one of the first studies that aimed to identify individuals at high risk of caries through a prediction model was performed in the late 1980s [28]. Since then, many different tools have been proposed that could assist the clinician in performing CRA in a standardized manner.

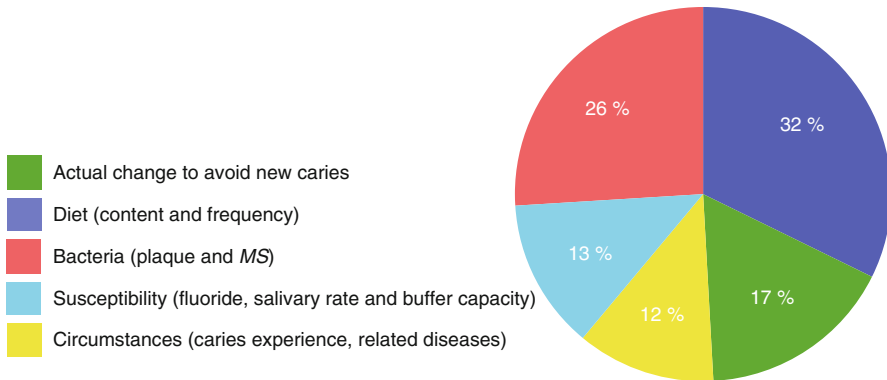
Each CRA tool is usually developed considering risk indicators and factors identified in cross-sectional and longitudinal studies, but each tool has its own rational way of presenting results. Among the many CRA tools presented in the literature, in this chapter emphasis is given to those that are presented in Table 3.3.

**CARIOGRAM** This is a software program developed in Sweden that can be freely downloaded from the Internet [29]. In this program, ten different caries risk factors are considered: caries experience, related diseases, diet content, diet frequency, amount of plaque, mutans streptococci (MS), fluoride program, saliva secretion, buffer capacity, and the clinical judgment of the dentist. The results are presented graphically to the patient, where caries prediction is provided by the “chance of avoiding dental caries.” Moreover, the patient can check his or her caries risk profile by identifying which caries risk factors are more predominant (Fig. 3.4).

When analyzing the hypothetical case presented in Fig. 3.4, the following assumptions can be made: this patient has a high chance of developing new carious lesions in the near future. Among all the factors included in the prediction model, diet is the one that contributes most to his or her caries profile. Thus, if the patient intends to change this prospect, he or she needs to make a drastic change on his or her diet. This program aids the clinician in that the diagram can provide guidance in selecting the most appropriate preventive measures and establishing recall intervals that are appropriate for the patient’s needs.

**Table 3.3** Characteristics of CARIOGRAM, CAT, CAMBRA, and NUS-CRA

CRA tool	Method of assessment	How results are presented	Target group
CARIOGRAM [29]	Computer calculation	Chance to avoid new carious lesions	All age groups
CAT (Caries Risk Assessment Tool) [30]	Manual check list	Classifying individuals in low-, moderate-, and high-caries groups	Preschool children (0–5 years old) School children and adolescents (≥6 years old)
CAMBRA (Caries Management by Risk Assessment) [31]	Manual check list	Classifying individuals in low, moderate, high, and extreme risk caries groups	Preschool children (0–5 years old) Age 6 through adulthood
NUS-CRA (National University of Singapore Caries Risk Assessment) [20]	Computer calculation	Chance of having new carious lesions	Preschool children



**Fig. 3.4** Graph representation of a patient's risk profile and the chance of avoiding new carious lesions by CARIOGRAM

**CAT** The Caries Risk assessment Tool (CAT) is recommended by the American Academy of Pediatric Dentistry (AAPD) [29, 30] and can be downloaded from the AAPD website. Two different forms are proposed according to the child's age: 0 to 5 years old or  $\geq 6$  years old. Both forms are divided into three sections that consider biological, protective, and clinical findings. The main difference between them concerns to questions that are age-related, such as the use of a bottle containing natural or added sugar in bed, which is included in the form for babies, but not in the form for older children. After completing the form, patients are categorized as low, moderate, or high caries risk. Although the instrument indicates a number of factors that should be considered for CRA, it allows the clinician, via his/her clinical judgment, to select just one factor in determining the overall risk.

**CAMBRA** A caries management by risk assessment approach, called CAMBRA, was initially proposed in 2003 by a group of stakeholders enrolled in education, research, industry, government agencies, and private practice in the United States [31]. The CAMBRA philosophy advocates that the clinician should assess the etiologic and protective factors involved in the caries process to be able to establish the patient's risk for future disease and, finally, to develop an individualized evidence-based caries management plan [32]. For doing that, two different CAMBRA forms were developed which considered the patients' age. The forms differ regarding risk factors, disease indicators, and protective factors. However, both forms consider caries experience – carious lesions and/or new restorations within the last 3 years – as the strongest future caries predictor. In addition to the CRA forms, the CAMBRA also provides clinical guidelines according to patients' caries risk profile and age [33, 34]. For example, if an adult is classified as presenting moderate risk to caries, the guideline proposes recall visits every 4–6 months. If the same patient were classified as low-risk, the recall intervals suggested would then be every 6–12 months.

*NUS-CRA* This model was developed specifically for preschool children based on the investigation of multiple risks, indicators, and protective factors [20]. The National University of Singapore Caries Risk Assessment (*NUS-CRA*) is very comprehensive with regard to sociodemographic factors, as it includes the child's age, ethnicity, and family socioeconomic status, but less discriminatory with regard to clinical features when compared to other CRA tools. While enamel carious lesions are considered an important factor for both *CAT* and *CAMBRA* and enamel defects for *CAT*, *NUS-CRA*, similar to the *CARIOGRAM*, just takes into account oral hygiene, past caries, and systemic health as clinical factors.

### 3.2.3 Validity of the Caries Risk Assessment Tools

The usefulness of structured approaches for performing caries risk assessment for education and teaching is unquestionable since these approaches contribute to a much clearer understanding of the dynamic factors involved in the caries process, especially for dental students [17]. However, the important question that arises is whether the use of CRA tools is able to improve the professional clinical judgment, since the literature shows that experienced dentists can estimate with certain accuracy patients' caries risk [19, 35]. This suggests that before a practitioner decides to adopt one of the CRA tools described above, they should discuss the validity of the tool.

The validity of a CRA tool is usually measured in terms of its ability to correctly classify the high-risk (sensitivity, *Se*) and the low-risk (specificity, *Sp*) patients correctly. An ideal situation would be one in which the sum of *Se* and *Sp* achieves at least 160% with a fair balance for both parameters [36, 37], resulting from longitudinal studies.

Another aspect that should be taken into account when analyzing the applicability of a CRA tool is the population in terms of which the validity study was performed. Not only is age important, but some cultural aspects are also important in that they might influence people's life styles.

Two recently published systematic reviews [38, 39] aimed to assess the validity of different CRA tools. One of these studies was dedicated to preschool children and schoolchildren/adolescents [38]. The other, a study by Tellez et al [39], concluded that limited evidence exists regarding the validity of existing CRA tools. Among the models that were investigated, *CARIOGRAM* presented moderate to good performance in classifying elderly people into caries risk groups. For preschool and schoolchildren/adolescents, the quality of evidence was also limited. One issue raised by the authors of that study is that the CRA models included in their systematic review had seldom been validated for different populations [38].

An interesting study that compared the validity of *CARIOGRAM*, *CAT*, *CAMBRA*, and *NUS-CRA* in predicting dental caries in preschool children was carried out in Hong Kong [40]. The same group of patients was assessed using the four CRA tools at baseline and after 12 months. Considering the target of 160% (*Se*+*Sp*), the *NUS-CRA* program was the only one that achieved the stated goal and was considered useful in identifying preschool children at risk for developing new

carious lesions. Nevertheless, longitudinal studies conducted in independent populations are needed to confirm the accuracy of the model.

Interestingly, when only one risk factor is taken into account, consensus is found in the literature that baseline caries experience has moderate to good accuracy. This also leaves the often-advertised bacterial/salivary tests as unreliable for caries prediction. If so, the clinician should question whether the use of CRA is indeed relevant. In that regard, it is important to emphasize that the lack of evidence does not mean that the models are not useful. Some methods may not be particularly effective in predicting future disease, owing to the multifactorial concept of dental caries, but they may still be very helpful in assisting the professional in the management process. By identifying different indicators or risk factors that play an important role in a patient's risk profile and sharing this knowledge with the patient, the professional will empower and motivate the patient to control those factors and, as a consequence, to control the disease. Moreover, without properly identifying indicators and risk factors, it will be very difficult for the professional to establish a preventive regime that best suits the individual patient (does every patient need to receive professional topical fluoride, for example?) or to plan individualized recall intervals (does every patient need to see a dentist every 6 months?).

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### 3.3 Clinical Cases

In this section, we illustrate the practical implications of the link between carious lesion activity assessment, caries risk assessment, and caries management.

Figure 3.5 shows a 5-year-old child with numerous active non-cavitated caries lesions along the gingival margin. The lesions are white and chalky after drying with compressed air, but even without drying, it is possible to see that several of the lesions have developed a micro-cavity (52, 82, 83, and 72). Because all these lesions are likely to progress to a more severe stage if left untreated and because they are easy to clean with the help of a parent, they should be treated nonoperatively. The patient has also developed an arrested cavity in the upper primary canine. This lesion may not progress if the patient continues to brush. Hence, there is no need for a filling in this tooth unless there is a cosmetic concern. The approximal cavity on 84 may not be possible to clean and should be restored. Needless to say, it is mandatory that the risk factors of this child must be identified and modified to avoid further lesion progression. Caries control in children cannot be successful without a caretaker's interest and active interaction in the nonoperative treatment.

Figure 3.6 shows a young teenager with an erupting second molar in the lower jaw. There is a whitish opaque active non-cavitated lesion in the occlusal surface. Although this lesion is difficult to clean, it may be worthwhile focusing on improved biofilm removal during the remaining eruption period to arrest the lesion [41]. Daily supervised cleaning with fluoride toothpaste may be as effective in controlling occlusal carious lesions as the application of a sealant [42]. Indeed, the patient has previously managed to convert an active lesion in the occlusal surface of the first molar into an inactive lesion. The watchful reader may also have noted a small

**Fig. 3.5** Right quadrant of a 5-year-old child showing active non-cavitated and cavitated lesions



**Fig. 3.6** An erupting second molar that already presents an active non-cavitated lesion at the occlusal surface



active non-cavitated lesion on the mesial surface of the first molar which became visible after exfoliation of the second primary molar. This teenager should therefore be checked for additional caries risk factors to optimize the treatment plan.

Figures 3.7a–c present the dentition of an adolescent male of 15 years old who had been under orthodontic treatment for 4 years. He showed evidence of poor oral hygiene and a high ingestion of sugar. The orthodontist referred him to a clinician after braces were removed. During the clinical examination, active cavitated lesions were observed on the buccal surfaces of the 36 and 43 and 44 and 45, along with numerous active non-cavitated lesions on many other teeth. The patient reported temporary pain from 35, 36, 44, and 45 when eating. The treatment of this young person should focus on restoring the ecological balance in the mouth. As a first step, cavities should be filled to relieve pain and to allow tooth brushing. A conservative restorative approach may be recommended to avoid excessive removal of partly demineralized enamel which may further weaken the teeth. The nonoperative treatment plan for the patient is behavioral change. Tooth brushing quality and habits using fluoridated toothpaste should be improved. Ameliorating sugar consumption patterns should be identified and non-cariogenic alternatives introduced. Such





**Fig. 3.7** Dentition of an adolescent after orthodontic treatment: (a) frontal view, (b) right side, and (c) left side

behavioral changes are not easy to perform, and the patient may need close guidance over a long time to achieve this goal. The dentist may support these processes by delivering topical fluorides to lesions during the active stages. However, it should be appreciated that caries is not a fluoride-deficient disease – fluoride treatments cannot stop the caries processes without proper biofilm removal being implemented at the same time [43]. The sad aspect of this case is that the ecological catastrophe on the dentition might have been avoided had the patient received proper instructions about caries control prior to and during the orthodontic treatment.

### 3.4 Final Remarks

The diagnosis of dental caries should not be performed exclusively on the basis of the presence or absence of a cavity. To determine whether the carious lesion, cavitated or not, is active or inactive is of prime importance, since carious lesion activity assessment is essential for the treatment decision. Because of the predictive validity of the Nyvad criteria, such caries diagnostic criteria are recommended for application in daily practice.

Regarding CRA, the literature shows that when risk factors are analyzed separately, past caries experience is the most important predictor for future caries independently of the patient's age. Considering the CRA tools, the predictive value of the models currently available still needs to be validated in different populations

with different cultural backgrounds and caries status. Nevertheless, assessment of caries risk factors is considered an important guide that can assist the practitioner in establishing individualized caries control programs for their patients.

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# Remineralisation and Biomimetics: Remineralisation Agents and Fluoride Therapy

# 4

David J. Manton and Eric C. Reynolds

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## Abstract

The demineralisation and remineralisation cycle needs to be controlled in order to prevent the decomposition of dental tissues. The use of fluoridated products, especially the consumption of fluoridated water and use of fluoridated toothpaste, provides significant reduction in caries risk in a cost-effective manner. Fluoride has also been used as a caries-preventive agent in varnishes and gels; for decades anti-fluoride propaganda questioned the public and the dental sector about its safety. Therefore, it is important to discuss the pros and cons of fluoride therapy using scientific evidence. More recently, a number of products containing calcium and phosphate have been released onto the market – these products use a variety of technologies and have varying levels of evidenced-based justification. The mechanisms of action of fluoride- and calcium-based technologies will be discussed in detail.

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## 4.1 Introduction

Dental enamel is comprised of 95 % mineral by weight and 86 % by volume, with the remainder being water, proteins and other organic materials. The dental caries

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process involves periods of net loss and gain of mineral, and when these periods favour demineralisation, the early white spot lesion appears. Whilst a surface layer remains on the enamel, remineralisation is possible, even with detectable mineral loss within the dentine.

Remineralisation of mineralised tooth structure requires both bioavailable calcium and phosphate to occur. In the healthy person, this is provided by free and protein-stabilised ions in the saliva. The statherins and proline-rich proteins are the major protein groups involved. Therefore, the amount of remineralisation possible is limited by the amount of calcium and phosphate in the saliva, unless an extrinsic source is introduced into the oral environment.

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## 4.2 The Healing of Demineralised Dental Enamel

Dental enamel is the most highly mineralised and hardest tissue in the human body [1]. These characteristics allow effective mastication and resistance to attrition and abrasion inherent in everyday life [1]. Enamel is composed primarily of impure calcium phosphate salts, namely, hydroxyapatite, fluorapatite and carbonated apatite [2]. The distribution of the different anions and cations throughout the crystal matrix affects the physical, diffusion and dissolution characteristics of the apatite.

The demineralisation of enamel due to the caries process occurs after a cariogenic polymicrobial biofilm metabolises fermentable carbohydrates (especially sucrose, glucose and fructose) and excretes a by-product, namely, lactic acid. The ecological plaque hypothesis proposed by Marsh in 1994 [3] is still valid today notwithstanding the recent increase in our understanding of the polymicrobial nature of the supragingival plaque biofilm due to the major advances in microbiomics. This hypothesis explains how frequent consumption of fermentable carbohydrate causes a transition from a commensal plaque to a polymicrobial biofilm dominated by aciduric and acidogenic species including streptococci, lactobacilli, bifidobacteria and *Actinomyces* [3–5]. The lactic acid, if in great-enough concentration, leads to undersaturation of the plaque fluid at the enamel surface and in the enamel fluid surrounding the enamel crystals with respect to enamel mineral, leading to dissolution of the enamel. As saliva can buffer the acid produced and also provide bioavailable calcium and phosphate ions during periods of no fermentable carbohydrate, a period of remineralisation can occur. Hence, there tends to be a regular cycling of demineralisation and remineralisation, and it is only when the balance of this cycling leans towards the demineralisation side that net mineral loss occurs and a carious lesion develops [6].

Once erupted, the teeth are bathed in saliva and gingival crevicular fluid (to a lesser extent). Saliva consists mainly of water, proteins and glycoproteins, mineral ions and enzymes and has roles in the promotion of remineralisation and prevention of demineralisation, lubrication, digestion, taste, assistance with bolus formation and antiviral, antifungal and antibacterial functions. Healthy saliva is supersaturated with regard to tooth mineral and therefore provides a protective environment for the teeth [7]. The supersaturation of saliva with regard to  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  phases is

maintained by the presence of stabilising proteinaceous macromolecules such as statherin and proline-rich proteins.

Saliva is excreted at different rates (0.3–0.4 ml/min unstimulated; 1.0–3.0 ml/min stimulated). Stimulated saliva has increased  $\text{Ca}^{2+}$  (0.5–2.8 mmol/l unstimulated; 0.2–4.7 mmol/l stimulated) and  $\text{PO}_4^{3-}$  (2–22 mmol/l unstimulated; 1.5–25 mmol/l stimulated) concentrations [7]. Decreased salivary flow or quality slows the recovery of plaque pH and saturation with respect to tooth mineral after acid exposure and therefore increases caries risk.

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## 4.3 Fluoride

Fluoride is the primary intervention for the prevention of dental caries. The fluoridation of public water supplies has been rated as one of the most significant public health measures in the last century; however, there are limitations to this method of fluoride delivery, as there is a reduction in the progression of caries but little reduction in incidence [8].

The fluoride ion acts mainly by driving remineralisation due to solubility product differences between fluorapatite and hydroxyapatite and by decreasing enamel solubility in two ways: (1) the fluoride ion is more stable in the crystal lattice than the hydroxide ion, and (2) it interacts closely with the calcium ions on the crystal surface to form  $\text{CaF}_2$ , helping to retain both calcium and fluoride ions to promote remineralisation. At the pH levels commonly found in actively fermenting dental plaque, the plaque fluid may be undersaturated with respect to enamel apatite but supersaturated with respect to fluorapatite, leading to the preferential deposition of the less soluble fluorapatite [ $\text{Ca}_{10}(\text{PO}_4)_6\text{F}_2$ ] to remineralise the lost enamel apatite [9].

The effectiveness of fluoride to remineralise enamel and obtain net mineral gain is limited by the levels of bioavailable  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  [10]. Therefore, as previously stated, if the periods of demineralisation outweigh those of remineralisation, the salivary  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  reservoir is depleted, and net loss of enamel mineral can occur, the so-called caries balance [6].

Another limitation of fluoride is persistence in the mouth. The use of a source of calcium around the time of fluoride exposure increases the formation of  $\text{CaF}_2$  and calcium-fluoride-like mineral, therefore increasing fluoride persistence [11].

### 4.3.1 Delivery Methods for Fluoride

#### 4.3.1.1 Water

The effectiveness of water fluoridation in preventing caries is indisputable. A recent report from the UK indicated that the presence of F in the water nearly halved hospital admissions due to dental caries [12]. Twenty-six studies evaluating the effect of water fluoridation and dental caries on children ranging 5–14-year-olds found significantly greater mean difference where the range of mean change in decayed, missing and filled primary/permanent teeth was 0.5–4.4 (2.25) teeth, and the

proportion of caries-free children was 5–64% (14.6%; interquartile range 5.05–22.1%) [13].

In most instances, fluoride is added at a concentration of 0.7–1.2 ppm, with recent recommendations in the USA suggesting 0.7 ppm as the concentration for water fluoridation [14]. The effect of fluoridated water in preventing dental caries is mostly attributed to topical low dosage providing inhibition of demineralisation and enhancing remineralisation. Community water fluoridation was named by the Centers for Disease Control and Prevention (CDC) as one of the great public health initiatives of the twentieth century.

#### **4.3.1.2 Fluoridated Milk, Sugar and Salt**

There is limited evidence relating to the fluoridation of milk, sugar and salt [15, 16]. Systematic reviews highlight the lack of high-quality evidence, especially for salt and sugar fluoridation. The dietary recommendations in lowering the amount of consumed salt and sugar for overall health possess a controversy as well.

The evidence for fluoridated milk and its effect on caries experience are somewhat stronger, with research in schoolchildren showing substantial reductions in caries increment; however, further high-quality randomised controlled trials (RCTs) are required to substantiate existing evidence [16].

#### **4.3.1.3 Supplements: Tablets, Drops, Lozenges and Chewing Gum**

As with many types of fluoride administration, the evidence related to the use of tablets, drops, lozenges and chewing gum and their effect on caries experience is limited, especially by the quality of existing studies [17]. Many of these supplement studies were conducted before the widespread use of fluoridated toothpastes, and so the reported decreases in caries increment need to be considered in the context of different fluoride exposure and the resultant decrease in the overall efficacy of the supplement [17]. The issue of fluorosis and ingestion of fluoridated products during tooth development must also be considered. Dental caries is not a disease of fluoride deficiency but a behaviourally driven destruction of dental hard tissues (enamel, dentine and cementum). Scientific evidence shows the effectiveness of topical use of fluoride and with the known side effects of systemic intake; the necessity of systemic use of fluoride loses its importance in caries prevention.

#### **4.3.1.4 Toothpaste**

The use of fluoridated toothpaste should be encouraged in all individuals at risk of developing caries. Fluoridated toothpaste is a cost-effective and easy-to-comply-with method of preventing the development of carious lesions, with efficacy increasing as fluoride concentration increases [18]. Little difference has been determined between fluoride types in toothpaste, i.e. between sodium and stannous fluoride and sodium monofluorophosphate; however, this may not be due to a true lack of difference but difficulties in establishing a suitable randomised clinical trial, such as the estimated very large sample size required, associated costs and the reliability of the caries detection method used. There has been some doubt cast on the efficacy of

'children's strength' 500 ppm toothpaste in preventing caries in children at risk [19]. Recently, the American Dental Association published guidelines indicating children at risk should use a small amount of adult-strength toothpaste, as the evidence suggests that this is an effective method of caries prevention, albeit increasing the risk of mild fluorosis in those below 2 years [20]. Increasing evidence suggests that low or 'children's strength' toothpastes are not as effective in preventing caries when compared to adult strength, and therefore if a child is at increased risk, a smear of adult-strength paste should be recommended. Discussion with the parents about the importance of spitting out the paste residue and limiting swallowing to minimise the risk of fluorosis development is important. Prescription of 1.1 % fluoride (5000 ppm)-containing toothpaste has been shown to be effective, promoting remineralisation of root caries in adults [21].

#### **4.3.1.5 Mouth Rinses and Gels**

The evidence for the effect of the use of fluoride gels and mouth rinses on caries experience suggests that there is a preventive effect from their use in the permanent dentition [22, 23]. Evidence of their effectiveness in the primary dentition is limited, and risk of fluorosis from accidental ingestion must also be considered in young children due to their inability to expectorate effectively [22]. Daily (0.05 % NaF – 226 ppm) or weekly (0.2 % NaF – 904 ppm) use of mouth rinses are usually accepted as a supplementary method to the use of fluoridated toothpaste, and the evidence of comparative and additive effects is limited due to the heterogeneity of the existing evidence base [23]. It is reported that a patient is more likely to use a fluoridated toothpaste more regularly than a mouth rinse, so clinicians may want to consider this fact when they are establishing a preventive regimen [23].

Gels (acidulated phosphate fluoride (APF) containing 1.23 % F – 12,300 ppm or neutral 2 % NaF 9040 ppm) can be professionally applied in practice with suction for 4 min and with frequency of application (biannually or every 3 months) relating to the caries risk of the patient older than 6 years of age [22, 24, 25].

#### **4.3.1.6 Varnish**

Fluoride varnish provides a method of spot application of a high-concentration fluoride to areas of risk. The varnish comprises several components that include an alcoholic rosin or resin base, with flavouring and often sodium fluoride (5 %, 22,600 ppm F) that sets rapidly with contamination of saliva. The use of fluoride varnish containing 22,600 ppm F in children has a great preventive effect on caries. There is a dose/frequency response for varnish applied once per year compared with twice per year, with increasing application frequency up to four times per year decreasing caries risk [24, 26]. Recently, varnishes containing bioavailable calcium and phosphate have been released which have a potential to increase the effectiveness of the varnish by providing ions to increase remineralisation; however, limited clinical trial evidence exists currently to indicate superiority over 'traditional varnishes' [27, 28]. The application of varnish has a minimal effect on plasma fluoride concentration when compared with fluoride gels and therefore provides a safe method of 'spot application' in children younger than 6 years of age as well [24].



### 4.3.2 The Limiters of Fluoride Efficacy: Calcium and Phosphate

The intrinsic sources of  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  are from saliva, dissolved tooth structure and, to a lesser degree, gingival crevicular fluid, and therefore to gain net remineralisation, the action of fluoride is limited by the amount of salivary  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  that is available [29]. Increased concentrations of  $\text{Ca}^{2+}$  also retain fluoride by calcium bridging. Therefore, the supplementation of bioavailable calcium and phosphate into the oral cavity, especially in the biofilm, has the potential to significantly decrease enamel demineralisation and increase remineralisation. The main limitation of adding calcium to the oral environment is the low solubility of most calcium salts, meaning that ionic calcium readily forms compounds in the mouth, limiting its bioavailability.

### 4.3.3 Fluoride Safety

Fluoride with respect to caries prevention has been studied for over 70 years, and this body of evidence indicates fluoride use is a safe, cost-effective and beneficial adjunct to caries prevention methods [30]. Fluoride has been linked to many systemic disorders over the years such as osteoporosis, bone cancer and decreased intelligence amongst others. Rigorous review of the evidence supporting these claims has, however, found no sound evidence to indicate that fluoride is anything but safe [13].

Enamel fluorosis is the outcome of the effect of high concentrations of fluoride on enamel formation. The fluorotic enamel lesion is diffuse and hypomineralised in nature, following the developmental lines of the enamel [31]. It is distinct from demarcated hypomineralised lesions such as those in molar incisor hypomineralisation. In some more severe cases, there can be enamel pitting and breakdown; however, this is posteruptive in nature and not hypoplasia [32]. The pathogenesis of fluorosis relates to the retention of poorly or undegraded proteins from amelogenesis in the post-secretory phase and the effect that these retained proteins has on enamel mineral content and appearance [31]. The effect relates to the direct influence that fluoride has on the mineralisation process and calcium regulation enzymes. There appears to be a spectrum of presentation, with the possibility of an 'acute' dose-related fluoride exposure effect as well as a chronic lower concentration effect, so specific susceptibility times are difficult to identify [31]. There is a significant dose-response relationship where the prevalence of fluorosis at a water-fluoride concentration of 1.0 ppm was 48 %, of which 12 % of cases represented an aesthetic concern [13].

Fluoride ingestion should be limited in children during amelogenesis of the permanent dentition especially 2–4 years of age for anterior incisors to levels which minimise the formation of fluorotic enamel; therefore, topical methods of application are favoured [33]. However, in high-risk cases where other caries risk factors cannot be changed readily, sometimes the risks of enamel fluorosis are outweighed by the preventive effect of adult concentration-fluoridated products, and a smear

layer of adult toothpaste may be used after the discussion of the ‘pros and cons’ with the caregivers of the child [34].

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## 4.4 Biomimetic Remineralisation

Biomimetic mineralisation and remineralisation includes the methods which mimic nature with respect to (re)mineralisation [35]. This consists of technologies that involve proteins stabilising calcium and phosphate ions and controlling the mineralisation process. There are two main commercialised technologies that fit into this classification – casein phosphopeptide amorphous calcium phosphate (CPP-ACP; Recaldent™; Recaldent Pty Ltd, Singapore) and Curodont™ (Credentis AG, Windisch, Switzerland); however, other technologies involving calcium and/or phosphate will also be discussed.

### 4.4.1 Casein Phosphopeptide: Amorphous Calcium Phosphate

CPP-ACP has anticariogenic activity reported in in vitro, animal and human in situ and in vivo experiments and RCTs [36]. The CPPs have the ability to stabilise  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  in metastable solution. Through the action of multiple phosphorylated serine residues, the CPPs bind to form nanoclusters of calcium and phosphate ions preventing their growth to the critical size required for nucleation and subsequent crystallisation [36].

The complexes bind into the biofilm and buffer  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  activities in the biofilm fluid at the tooth surface, establishing an environment supersaturated with respect to calcium and phosphate phases including tooth mineral, inhibiting demineralisation and driving remineralisation down a concentration gradient. The CPPs also bind to the cell wall of *Streptococcus mutans*, with the CPP bond being twice the strength of that of calcium and therefore providing a source of calcium and phosphate in the biofilm fluid and also slowing the diffusion and subsequent loss of calcium [36]. The adherence of *S. mutans* and *S. sobrinus* within the biofilm and onto hydroxyapatite is also reduced significantly by CPP-ACP, decreasing the proportion of the mutans streptococci [37].

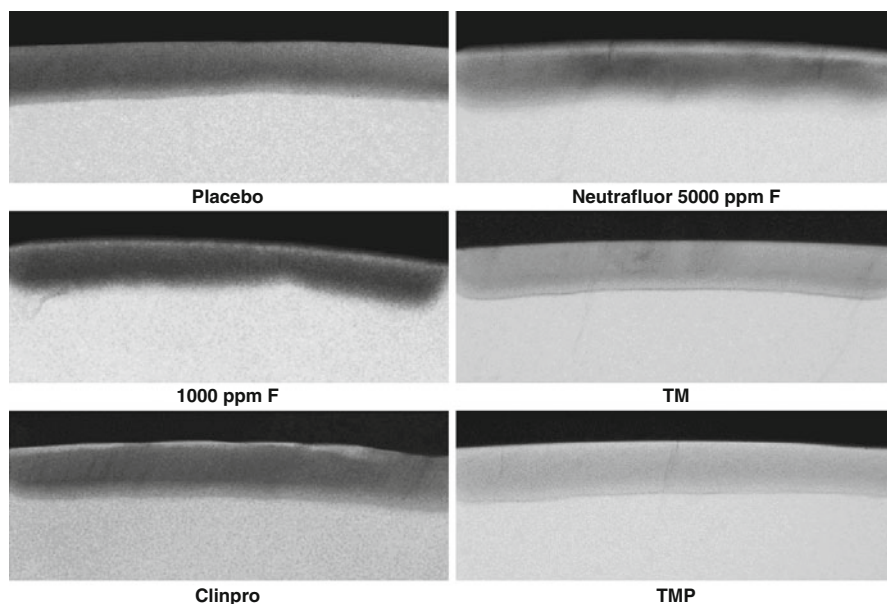
A number of studies have reported the efficacy of CPP-ACP in the in vitro inhibition of enamel and dentine demineralisation and also the promotion of in vitro remineralisation of enamel subsurface lesions (ESL). Similar results have been gained in situ, with CPP-ACP driving remineralisation of ESL with delivery by chewing gum, mouth rinse, crème or lozenge [36].

When CPP-ACP is delivered in combination with fluoride (CPP-ACFP), the potentiating effect of fluoride on enamel remineralisation is significantly increased [29, 38]. Interestingly, the fluoride is not concentrated in the outer ‘hypermineralised’ surface layer as normally reported but spread throughout the depth of CPP-ACP/F-remineralised ESLs [36, 38]. The proposed mechanism is that the CPP stabilises the  $\text{Ca}^{2+}$ ,  $\text{PO}_4^{3-}$  and  $\text{F}^-$  ions at the enamel surface in the correct molar ratio

as electroneutral nanoclusters allowing deep diffusion into the ESL down a concentration gradient subsequently forming fluorapatite. Remineralisation of ESLs is greatest when CPP-ACP and CPP-ACFP are delivered at pH 5.5 which can be obtained when CPP-ACP/F comes in contact with an acidic plaque [36, 38]. The deep remineralisation obtained after CPP-ACP/F exposure four times per day for 14 days is illustrated in Fig. 4.1.

Another feature of stabilised calcium and phosphate ions is the reduction in the reactivity of fluoride at the surface of the white spot lesion. As can be seen in Fig. 4.1, the Tooth Mousse (TM; GC Corp, Tokyo, Japan) and Tooth Mousse Plus (TMP; GC Corp, Tokyo, Japan) lesions have deep remineralisation without obvious relatively hypermineralised surface layer, whereas the unstabilised products (Clinpro, Neutrafluor 5000 and 1000 ppm NaF) all exhibit significant fluoride concentration-related surface layer development, subsequently limiting further ionic diffusion into the body of the lesion and therefore inhibiting lesion return to translucence. This occurs due to the reactivity of fluoride ion in the presence of calcium and phosphate, instantly precipitating at the enamel surface. With stabilisation via CPP, the electroneutral CPP-ACFP nanoclusters can diffuse into the lesion; the  $\text{Ca}^{2+}$  and  $\text{PO}_4^{3-}$  dissociate to maintain ionic equilibrium and then deposit into voids in the demineralised enamel crystals using these demineralised crystals as their template for anisotropic crystal growth.

The dentinal tissue also has the ability to be remineralised by CPP-ACP/F; however, further evidence is required to support this initial research [39].



**Fig. 4.1** Microradiographs of control and test subsurface lesions of enamel illustrating remineralisation of enamel subsurface lesions after 14 days exposure in situ

#### 4.4.2 Self-Assembling Peptide P11-4

Recently, the self-assembling peptide P11-4 (Curodont) has been manufactured to mimic the structure of proteins involved in controlling mineral precipitation in amelogenesis. This peptide is claimed to form a low-viscosity solution that should penetrate the porosity of a white spot lesion and then change structure with decreased pH to create a site for nucleation and precipitation of tooth mineral [40–43]. It is difficult to understand the rationale for an additional mineralisation template in an uncavitated early carious lesion with the large number of demineralised apatite crystals to act as nucleation sites for remineralisation. The rate-limiting factor for remineralisation is the diffusion of calcium, phosphate and fluoride ions into the lesion, not their deposition into crystal voids. Furthermore, several authors have suggested that residual template protein of hypomineralised enamel can actually inhibit mineralisation by blocking precipitation sites and decreasing ionic diffusion into the lesion, with some authors indicating a similar situation in carious lesions [44–46]. Nevertheless, some initial clinical research has demonstrated improvement in appearance of white spot lesions after application of the product; however, more robust clinical research with proper controls is needed to quantify mineral changes in these lesions and to relate them specifically to the presence of the self-assembling peptides.

#### 4.4.3 Arginine

Arginine has been added to dentifrice in addition to fluoride as an adjunct to prevention and remineralisation of carious lesions, especially when delivered with calcium-containing paste [47]. Some research indicates a benefit with the addition of arginine; however, the mechanism of action is still a little unclear, with an increase in plaque pH, the likely mechanism via base production by arginolytic bacteria [48–50]. In a recent commentary in the *British Dental Journal*, a challenge to the validity of the results of research into toothpastes containing arginine was made, so until this issue is resolved, current results must be interpreted with caution [51].

#### 4.4.4 Non-peptide-Based Remineralising Technologies

The lack of a stabilising agent to prevent precipitation of the calcium phosphate salts after saturation concentrations of the ions are reached in the plaque or at the tooth surface is a major disadvantage of these non-peptide technologies. This therefore limits the availability of ionic calcium and phosphate for non-stabilised products at the tooth surface due to their rapid precipitation, subsequently limiting remineralisation potential, for which there is little evidence [52]. Due to this limitation, these technologies are primarily limited to surface precipitation, especially in the presence of fluoride. This mechanism, however, has been reported to decrease dentinal sensitivity.

#### **4.4.4.1 Amorphous Calcium Phosphate (ACP®; American Dental Association Foundation, Chicago, IL, USA)**

Amorphous calcium phosphate (ACP®; American Dental Association Foundation, Chicago, IL, USA) – this product is based on the mixing of two solutions, one containing phosphate ions ( $K_4P_2O_7$ ) and the other calcium ions ( $CaNO_3$ ), after which there is precipitation of calcium and phosphate salts, including hydroxyapatite on the surface of the tooth [53]. There is evidence of efficacy in the remineralisation of root caries clinically after a year, but the remineralising effect when added to bleaching agents was uncertain in an in vitro study [54, 55].

#### **4.4.4.2 Novamin® (GSK, Middlesex, UK)**

Novamin® (GSK, Middlesex, UK) is a bioactive glass calcium sodium phosphosilicate comprising 45 %  $SiO_2$ , 24.5 %  $Na_2O$ , 24.5 %  $CaO$  and 6 %  $P_2O_5$ . The calcium and phosphate ions are released from the bioactive glass on contact with water in saliva and a precipitate layer of calcium phosphate; most likely carbonated hydroxyapatite is formed on the seeding surface once saturation is reached [53]. The product forms a precipitate layer on the tooth surface, making it effective as a desensitising agent [56]. There is limited clinical evidence of its efficacy with respect to remineralisation of enamel lesions.

#### **4.4.4.3 Tricalcium Phosphate (TCP®; 3MEspe, MN, USA)**

Tricalcium phosphate (TCP®; 3MEspe, MN, USA) is based on poorly soluble tricalcium phosphate that has been milled to small particles and then coated with sodium laurel sulphate to isolate the calcium phosphate from fluoride in any oral care product to which it is added. The amount of TCP added to the oral care products is usually very small and defined as a ‘catalytic amount’. The published evidence for remineralisation is limited; however, some recovery in enamel surface hardness has been reported after acidic challenge in vitro, most likely relating to surface precipitation [57]. A recent in situ study has not confirmed release of bioavailable calcium phosphate or remineralisation of enamel subsurface lesions with this technology [58].

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## **4.5 Suggestions for Clinical and Home Use**

The use of fluoridated products as the mainstay of caries prevention should be predicated on caries risk, lesion characteristics, access to fluoridated reticulated water and compliance. For both children and adults, fluoridated toothpaste is a cost-effective and efficient product. The concentration of fluoride in the toothpaste used should reflect caries risk – in low-risk children, a ‘children’s’ strength (500 ppm F) product is suitable. Children at higher risk would benefit from higher adult concentrations, with the amount used limited to a smear across the brush for infants to reduce ingestion. Adults at low to moderate risk should use products with 1000–1450 ppm F, with those at higher risk using 5000 ppm F toothpaste (if available). Teeth should be brushed twice per day without vigorous post-brushing rinsing. The

use of the fluoridated product should be with caution in young children (3 years of age and younger) as most of the product is swallowed, so only used in situations where the benefits outweigh the risks and after discussion with parents.

Fluoridated mouth rinses provide some additional preventive fraction; however, this decreases when used in conjunction with fluoridated toothpaste. Rinsing should take place between brushing times to increase biofilm and salivary fluoride concentration to maximise the preventive effect. Concentration of mouth rinse (220 ppm–900 ppm) should reflect caries risk. Mouth rinses should not be used in children who cannot expectorate effectively as swallowing will increase risk of fluorosis.

Fluoride gels may be used in the clinical setting and have slightly lower preventive efficacy than varnishes [59, 60]. However, gels require the use of a tray and access to suction to limit ingestion, therefore limiting the contexts where they can be used cost-effectively.

Fluoride varnishes are an effective product for ‘spot application’ to at-risk sites due to the high F concentration (22,600 ppm F) and persistence and may be used for young children. Frequency of application should reflect risk, with four times per year being most effective in high-risk individuals.

The use of complimentary products such as those containing calcium and phosphate, like fluoridated products, should relate to caries risk. The evidence base for the clinical efficacy with respect to caries remineralisation for the active ingredients should also be taken into account, and when this is done, the use of CPP-ACP is the only product that can be recommended at present. The product most easily accessed commercially is Tooth Mousse®/MI Paste® (GC Corp, Tokyo, Japan), with fluoridated products also available (Tooth Mousse® Plus/MI Paste® Plus; 900 ppm F); the commercial names of similar products vary depending on the country of sale. These products are recommended for manual application once per day – usually after brushing before bed – increasing the oral persistence due to decreased nocturnal salivary flow/oral clearance.

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## 4.6 Final Remarks

In conclusion, the prevention of caries is the primary aim for our patients. The assessment of the caries risk of the patient and how this relates to the use of remineralisation products should relate to product evidence base regarding efficacy and their likelihood of use by the patient, normally relating to ease of use, frequency of use, taste and expense. Fluoride is still the first product to consider; however, its efficacy is limited with respect to the availability of the enamel ‘building blocks’, bioavailable calcium and phosphate ions. The addition of bioavailable calcium and phosphate enables the remineralisation process to be enhanced, benefitting those who are at increased caries risk or who require more rapid remineralisation of the enamel.

Clinically, the use of products should relate to the clinical situation. Decreasing caries risk is the first step – therefore, the consideration of diet, oral hygiene efficacy and frequency, use of fluoridated products such as toothpaste (+/- rinse) and

compliance is important. All of these factors should be seen in the context of salivary flow and quality as an overriding determinant of caries risk and the ability to remineralise carious lesions, as saliva provides the intrinsic source of protein-stabilised calcium and phosphate.

In summary, the biomimetic treatment of dental caries relies not only on ‘product use’ but also identification and modification of the caries risk factors of the individual. The clinician should use the evidence base to inform their decision on which products to use in the clinic and for those that the patient will use at home.

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## Abstract

Regularly removing biofilm and controlling risk factors are essential to the individual and the clinician for managing dental caries. As an alternative way of preventing and managing the disease, researchers have developed and investigated antimicrobial agents to counter the bacterial component in the caries process. However, these agents are considered to have limited value and are therefore restricted to special care groups. This chapter summarises the effect of antimicrobial agents such as chlorhexidine, iodine and ozone as well as the antimicrobial effect of natural products on managing carious lesion development.

## 5.1 Introduction

Dental caries is a behavioural disease that is characterised by the demineralisation of the hard dental tissues through acid that results from fermentation of free sugars by certain microorganisms. Early colonisation of the cariogenic microorganisms plays an important role in the development of the disease [1, 2]. Although inherited host risk factors, including salivary composition, enamel structure, taste preferences and immune response of saliva, may vary genetically among individuals [3], most approaches to preventing and controlling the disease rely on behavioural factors such as regular removal of biofilm and reduction in intake and frequency of free sugars.

Traditionally, an important factor in managing dental caries has been the reduction and elimination of a few specific pathologic microorganisms. Oral microorganisms

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become pathologic only when the host consumes large quantities of free sugars at a high frequency per day over a relatively long period. This, together with insufficient removal of biofilm, allows certain microorganisms to flourish, overgrow others and produce acid. The cariogenic microorganisms are harboured in the growing cariogenic biofilm. The acid-producing effect of these pathologic microorganisms can be reduced or eliminated by removing the biofilm through mechanical or electric brushing with a fluoride-containing toothpaste. A non-mechanically or electrically driven reduction in cariogenic microorganisms can be established with antiseptic and antimicrobial agents or antimicrobials. Antimicrobials, in the form of a rinse, gel, paste or varnish, are products that have the potential ability to reduce the number of cariogenic microorganisms for a limited period.

Dental caries is a time-related pathology, and, therefore, in addition to the cariogenic microorganisms, the continuity of the biofilm on the tooth surface, the recolonisation sequence and metabolism of the microorganisms are other active factors that need to be controlled. It is known that the microbiome of a person develops over time and that the dental biofilm is unique for every individual in thickness and content [4]. The microbiome is affected by the presence of glucose and by the characteristics of saliva. Biofilms may be dominated by different kinds of bacteria such as spherical bacteria, which have a compact nature and produce less extracellular space, or sparsely distributed spherical and filamentous bacteria, which have a more open architecture. This difference may be seen even within the same mouth on different locations. This microstructure affects cariogenicity and metabolism as well as the invasion of the active ingredients of the antimicrobials within the biofilm. Not only the content of the biofilm but also the enzymatic reactions such as glucosyltransferase or fructosyltransferase, which enable the bacterial adherence, may be affected by the distinct chemical properties of the active ingredients in the antimicrobial agent. Stratified bacteria are increasingly compacted on the biofilm as it gets older. The antibacterial effect of a chemical is reported to be influenced by the age of the biofilm, with recently formed biofilms more sensitive to microbial agents [5].

High-caries-risk patients, such as children with severe early childhood caries (S-ECC), orthodontic patients and the dentate elderly have high cariogenic microorganism load caused by high biofilm accumulation and low salivary flow rate. This group of patients has an overwhelming amount of *Streptococcus mutans* in an infectious biofilm. Currently, with limited knowledge of individual differences among human microbiota, the aim of using antimicrobials in high-caries-risk patients is to control dental caries by altering the metabolism of the cariogenic biofilm.

Antimicrobials, also named antibacterials, have been used to suppress pathological oral microorganisms and favour the oral health of both soft gingival and hard dental tissues. In general, the antibacterial agent is meant to kill or control the proliferation of the microorganisms and eliminate extensive inflammatory response in gingival tissues. It should promote local immune response in saliva and should not be toxic to the host. Antimicrobial strategy may also be targeted at bacterial adhesion, thus preventing both the adhesion and coaggregation of bacteria to form complex multispecies biofilms as dental plaque [6].

The effect of the antimicrobial agent is closely related to its diffusion capacity within the biofilm and its retention. Its effectiveness is also measured by its ability to reduce bacterial virulence and retard biofilm accumulation [7, 8]. A study conducted by Takenaka et al. in 2008 found that although a contraction of 10 % of the biofilm was measured after using 0.12 % chlorhexidine mouthrinse in vitro, no de-attachment from the tooth surface was observed [7]. More time is needed to reach the centre of the cell cluster as the mean radial dimension of the biofilm enlarges [8]. Biofilm structure does not easily change with rinsing with antimicrobials nor is it detached by the chemical interaction of the active ingredients [8]. Additionally, it is difficult to maintain a stable minimum inhibitory concentration (MIC) of the drug in the oral cavity owing to the dynamics of the oral environment.

In the following sections, the effectiveness of antimicrobials in dental caries management is discussed. Table 5.1 presents commercially available antimicrobials with different concentrations and forms that may be used in caries management.

**Table 5.1** Examples of commercial products with different antimicrobials that are used in caries management

Antimicrobial	Formulation	Concentration	Product
Chlorhexidine gluconate	Mouthrinse	0.05 %	Concool F <sup>a</sup>
		0.12 %	Peridex <sup>b</sup>
		0.2 %	Corsodyl <sup>c</sup>
Chlorhexidine	Gel	0.2 % (+0.2 % NaF)	Cervitec Gel <sup>d</sup>
		0.2 % (+0.32 % NaF)	TePe Gingival Gel <sup>c</sup>
Chlorhexidine	Varnish	1 % (+1 % thymol)	Cervitec Plus <sup>d</sup>
		10 %	Chlorzoin <sup>f</sup>
		10 %	Prevora <sup>g</sup>
		20 %	BioC <sup>h</sup>
		35 %	EC 40 <sup>h</sup>
Iodine (povidone iodine)	Mouthrinse	7 %	Popiyodon Gargle <sup>i</sup>
	Solution gargle	1 %	Betadine <sup>j</sup>
Essential oils + ethanol	Mouthrinse	0.09 % 1,8-cineol	Listerine <sup>k</sup> (Original)
		0.06 % thymol	
		0.05 % methyl salicylate	
		0.04 % 1-menthol	
		27 % Ethanol	

<sup>a</sup>Weltec, Osaka, Japan

<sup>b</sup>3M Espe Dental Products, USA

<sup>c</sup>GlaxoSmithKline Consumer Healthcare, Brentford, TW8 9GS, UK

<sup>d</sup>Ivoclar Vivadent, Schaan, Liechtenstein

<sup>e</sup>TePe, Munhygienprodukter AB, Malmö, Sweden

<sup>f</sup>Knowell, Therapeutic Technologies, Toronto, Canada

<sup>g</sup>CHX Technologies Europe Limited, Hampshire, UK

<sup>h</sup>Biodent, Nijmegen, Netherlands

<sup>i</sup>Yoshida Pharmaceuticals Co., Ltd, Saitama, Japan

<sup>j</sup>Mundipharma Distribution GmbH, Philippines

<sup>k</sup>Johnson & Johnson KK Consumer Company, Morris Plains, NJ, USA

## 5.2 Chlorhexidine

Chlorhexidine is a traditional antiseptic that is accepted as a ‘gold standard’ for use in managing gingivitis [9]. Chlorhexidine is adsorbed onto the cell walls of microorganisms, providing bacteriostatic effect at low concentrations and bactericidal effect at higher concentrations. This biguanide drug has the ability to adsorb on soft and hard tissues in the oral cavity, which provides substantivity [10]. Although chlorhexidine has a proven antibacterial effect on *Streptococcus mutans* in the oral cavity, insufficient evidence is available regarding its preventive effect on coronal carious lesion development [11, 12]. Studies reporting on its antibacterial effect have mostly been carried out in high-caries-risk groups with high levels of *Streptococcus mutans* and *Lactobacilli* on the surface, resulting in a statistically significant suppressing effect [13].

Chlorhexidine is delivered as a mouthrinse, gel or varnish in today’s dentistry. Prescribed chlorhexidine mouthrinses as a plaque- and gingivitis-reducing agent have been proven effective for over 40 years all around the world. The soft oral tissues and tooth surfaces retain the active ingredients in chlorhexidine rinse, providing a long-term antimicrobial effect. Chlorhexidine as a mouthrinse contains different concentrations in different countries (Table 5.1). For example, in Japan only very low concentrations are available owing to allergic reactions reported [14]. In general, 10 ml of 0.2 % chlorhexidine mouthrinse or 15 ml of 0.12 % chlorhexidine-containing rinse twice a day is prescribed. It is important to use antimicrobials for a limited period since, adversely, bacterial resistance may occur. It is reported that long-term antimicrobial mouthrinse may worsen xerostomia in patients taking polypharmacy [15]. A systematic review regarding the effectiveness of chlorhexidine rinse on plaque reduction in gingivitis patients relative to control revealed a significant reduction in plaque and gingivitis levels and a significant increase in tooth staining [9].

Chlorhexidine mouthrinse cannot be used for patients that are younger than six years old because of the high risk of swallowing the liquid. Chlorhexidine gel and varnish may be applied to people of all ages because of good compliance and minimised side effects such as staining.

Chlorhexidine gel may be applied as a paste at home or in trays in the dental clinic. Studies have revealed that intensive application (3–4 daily applications over 2 days or daily application for 10–14 days) may provide reduction in the level of *Streptococcus mutans*, but large individual variations exist regarding its effect. It is, therefore, advised that the levels of these microorganisms are reassessed over the course of the therapy [13].

The antibacterial effect of available chlorhexidine varnish (1 % Cervitec Plus, Ivoclar Vivadent; 10 % Prevora, CHX Technologies; or 40 % EC-40, Biodent BV) may last for 3 months. Different treatment regimes are in use. Regimes that consist of 2–3 applications at the start and 1 month or 3 months’ follow-up of varnish application have been tested and have yielded conflicting results.

Antimicrobial therapy using chlorhexidine may be of interest to clinicians as a strategy for reducing the apparent presence of the high bacterial component in

high-caries-risk patients. These include children with S-ECC, adolescents receiving orthodontic treatment and elderly patients with hyposalivation.

### 5.2.1 Chlorhexidine and Severe Early Childhood Caries

Children with early childhood caries (ECC) harbour high levels of cariogenic microorganisms and a level of *Streptococcus mutans* that exceeds 30% of the cultivable plaque bacteria [16, 17]. This situation occurs as a result of an excessive amount of free sugar consumption and insufficient removal of the biofilm in these babies and infants. As happens with all children, microorganisms are transmitted from the caregiver(s) to the child when the latter is very young [18–20]. It is a misconception, however, that babies acquire ECC solely because of the transmission of high levels of cariogenic microorganisms from their caregiver(s) without considering the positive effect of preventive measures if provided [21]. The link between high levels of cariogenic microorganisms in the mother and in the baby/infant is most probably due to the lack of, or insufficient, good oral health behaviour by the mother and the baby/infant: consumption in various ways of too much free sugar and insufficient oral hygiene practice [22]. The mother/caregiver does not act as a good oral health model.

This adverse situation can be reversed by the caregiver's adoption of good oral health behaviour as shown in a 2015 study conducted in Brazil [23]. In this study high-caries-risk pregnant mothers from low socio-economic areas were invited for comprehensive treatment at the university clinic in Brasilia [23]. After completion of the treatment, each mother was invited to take her baby to the same clinic for monitoring the baby's oral health and providing treatment such as fluoride varnish and instructions about how and when to clean the baby's teeth and which food and drink products are dentally safe. Almost half of the mothers accepted the invitation. Four years after giving birth, all 194 mothers were contacted and invited to visit the university clinic. Children that had attended regularly (2.8 times/year) had a prevalence of dental caries of 9% and a mean dmft score of 0.25, while children that had never attended the clinic had a prevalence of dental caries of 81% and a mean dmft score of 4.12. The odds ratio was 48.

On the basis of two included studies, a recent systematic review reported insufficient evidence for the use of chlorhexidine varnish or gel in children with ECC [24]. However, the antibacterial effect of chlorhexidine may be helpful in suppressing maternal reservoirs of cariogenic microorganisms during the period in which the caregivers change their oral health behaviour.

### 5.2.2 Chlorhexidine and Orthodontic Treatment

Another high-caries-risk group that may benefit from chlorhexidine therapy is the group of people undergoing orthodontic treatment. These people usually have a strong likelihood of accumulating a high level of cariogenic biofilm and, therefore,

are in greater need of preventive measures [25, 26]. In particular, young orthodontic patients with fixed appliances are usually incapable of removing biofilm effectively and need further adjunctive treatment to avoid demineralisation of tooth surfaces.

Although the antimicrobial effect of chlorhexidine on *Streptococcus mutans* has been established in children with fixed appliances [27], evidence of its carious lesion-inhibiting effect has been shown to be weak [28, 29].

### 5.2.3 Chlorhexidine and Root Caries

Studies have reported that chlorhexidine varnish seems to control the development of root carious lesions and reduces their occurrence in the elderly [30, 31]. These results may be explained by the surface characteristics of the root that may retain chlorhexidine better because of the exposure of dentine that provides a reservoir for the surface-bonded antibacterial. Low salivary flow in the elderly patients may also diminish the clearance from the retentive surface on the root, providing a prolonged antibacterial effect.

### 5.2.4 Evidence on the Effectiveness of Chlorhexidine

A recent Cochrane review of the effectiveness of chlorhexidine treatment on children and adolescents [12] and evidence-based clinical recommendations [31] and systematic reviews [13, 32–34] found no advantage in using chlorhexidine over no treatment or a placebo for coronal caries inhibition but showed that chlorhexidine-containing products may diminish the bacterial load of the biofilm [35]. Furthermore, the retentive effect of chlorhexidine varnish within the fissures of permanent molars was thought to prevent carious lesion development in fissures after twice or four times yearly application of the varnish over a period of 2 years [33, 36–40]. However, 1 year after discontinuation of the chlorhexidine varnish, compared to the non-chlorhexidine control group, the carious lesions in a group of children had increased to equal levels [40].

In conclusion, chlorhexidine is a cariogenic microorganism-suppressing antimicrobial and may, therefore, be useful in reducing the bacterial load in high-caries-risk groups such as children with S-ECC, those wearing orthodontic fixed appliances and elderly people with reduced saliva flow in adjunction to biofilm removal. Evidence that supports the effectiveness of chlorhexidine varnish in preventing dental caries and controlling carious lesion development in non-high-caries-risk groups is not shown.

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## 5.3 Ozone

Another antimicrobial agent that is used in oral healthcare is called ozone. Ozone is a triatomic molecule that consists of three oxygen molecules and may be used as a gas or in water or oil in medicine. Ozone gas has a high oxidation potential and

oxidates bacteria, viruses or fungi although it does not affect the healthy human cell negatively. Ozone gas applications are suggested for use as an antimicrobial for cavities, root canals and periodontal pockets as well as in implant surgery. It also stimulates blood circulation and immune response.

### 5.3.1 Ozone Safety

As with many pharmaceutical agents, gaseous ozone is toxic in high concentrations (50 ppm for 60 min). For this reason, safety limits of 0.3 ppm for 15 min (US Occupational Safety and Health Administration – OSHA) and up to 0.1 ppm inhalation without a clear threshold (National Institute for Occupational Safety and Health – NIOSH) should be carefully applied when ozone is used in dentistry [41, 42].

Several ozone generators are available in the dental market. These products have different technologies, each producing different concentrations of ozone gas. The ozone generator HealOzone (KaVo GmbH, Biberach, Germany) was the most well-known ozone generator in dentistry but is no longer available. It delivered relatively high concentrations of ozone (2,100 ppm as 0.052%, v/v in air at a rate of 13.33 ml/s) with a unique evacuation system to avoid inhalation. Other ozone generators such as Prozone (W&H, Germany), Ozi-Cure (Centurion, South Africa), Ozonytron OZ (MIO International Ozonytron GmbH, Germany) and many others in the market produce different concentrations and flow rates of ozone gas from HealOzone. Ozonytron OZ and XP use pure medical oxygen in tanks, and Ozonytron X, XL and XP contain ozone electrodes that produce ozone gas from the atmospheric oxygen on the surface to which it is applied. The ozone concentration produced with the probes in Ozonytron X, XL and XP ranges from 1,000 to 100,000 ppm, whereas Prozone reports producing 140 ppm at 2 l/min. Unfortunately, no scientific evidence is available regarding a standard concentration and application method for ozone in dentistry.

Ozone generators have a safety issue. Evacuation of the ozone gas by using adequate suction for all ozone generators (except HealOzone and Ozonytron OZ) is very important for the safety of the patient and the dental staff. Clinicians should be aware of the potential hazards and it is important to educate the dental staff about the need to adhere to the precautions carefully. A 2007 study in an *in vitro* setting found that the ozone gas concentration measured was above permitted levels in the nasopharynx area of the patient when suction was not used in the Ozi-Cure machine, which did not include a built-in evacuation system [43]. Ozone gas concentration within the mouth for the patient and inhalation for the operator should be carefully controlled.

*In vitro* studies have shown the lethal effect of ozone on cariogenic bacteria. However, several *in vivo* studies using different ozone generators have reported controversial findings. Several studies have concluded that ozone has an antibacterial effect but lack the evidence regarding the elimination of pathologic oral bacteria for the prevention of carious lesions [44, 45]. The known instantaneous lethal effect on oral pathologic bacteria does not last long, and re-colonisation within a week is even more dense [46]. Ozone works better on the biofilm positioned over the dentine surface, and this feature may be used for treating root caries [31, 47].



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### 5.3.2 Evidence for the Effectiveness of Ozone

On the basis of the equivocal evidence regarding its effectiveness, using ozone gas for preventing carious lesions in tooth and root surfaces is not advocated. Knowing that dental caries is not an infectious disease and that the microorganisms in the oral cavity function best in a balanced ecological system, it is clear that short-term killing of microorganisms only disorganises the ecological system but has little effect on preventing carious lesion development in tooth and root surfaces. Furthermore, two systematic reviews have concluded that ozone is not a cost-effective adjunct in the management and treatment of carious lesions [47, 48].

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## 5.4 Iodine

This section describes the effectiveness of iodine in preventing dental carious lesion development. Iodine is a widely used antiseptic in medicine. Its solubility in water is low, and, to improve the delivery of the agent, it is altered by adding polyvinylpyrrolidone (PVP) to form povidone iodine (PI). This alteration increases its solubility and reduces irritation and staining.

Povidone iodine has been tested, particularly for controlling (severe and) early childhood caries. It has been applied as a postsurgical medicament in a small group of children treated restoratively for S-ECC under general anaesthesia [49] and in the clinic [50]. Other studies have tested the effectiveness of PI in combination with fluoride foam [51] and fluoride varnish [52].

Evidence for a carious lesion-controlling effect of PI in children was reported recently [24, 53]. Povidone iodine displayed no beneficial effects on ECC incidence, although some studies have reported significant reductions in salivary mutans streptococci levels [24]. This reduction in streptococci levels was reported to be of temporary duration when evaluated 3 months later [50] and occurred also in the control group of children who had gone through full restorative treatment under general anaesthesia [49]. Besides the Berkowitz et al. study [53], none of the other studies referred to above appear to have informed parents of biofilm and diet control during the study period. Hence, the effect of these two proven carious lesion-preventive measures was not taken up in the analyses of these studies. The quasi-systematic review graded the quality of evidence that anti-caries agents such as povidone iodine prevented or controlled carious lesion incidence in early childhood as very low [24, 52].

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## 5.5 Triclosan

Triclosan (2,4,4'-trichloro-2-hydroxydiphenyl ether) is a bisphenolic and non-cationic agent that has a broad spectrum of antimicrobial potential and antiplaque activity and is effective against gram-positive and gram-negative bacteria [54]. It is used as an antimicrobial added in toothpastes. In an in vitro study, it was found that 100 ppm triclosan in toothpaste had an inhibiting effect on *Streptococcus mutans*

and *Streptococcus sanguinis* but not on *Lactobacillus acidophilus* [55]. In this context, it is important to note that other ingredients within the toothpaste may have a contributory effect on caries prevention. Triclosan is mostly used in toothpastes with a copolymer of polyvinylmethylether/maleic acid (PVM/MA) for enhanced sustainability, in combination with zinc citrate or sodium fluoride [56]. No side effects are reported from long-term use with toothpastes containing triclosan. A Cochrane review [57] revealed a slight reduction in coronal carious lesion increment after 24–36 months of triclosan toothpaste use with a high quality of evidence. However, more studies are needed to show the effect on root carious lesions since only one study with a moderate quality of evidence [58] reported a statistically significant reduction after 3 years.

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## 5.6 Natural Antimicrobials

Evidence reported so far has not shown that chemical antimicrobials are effective in inhibiting carious lesion development but have an effect on *Streptococcus mutans* reduction. In addition, drug resistance and side effects such as extrinsic staining or taste disorders by chemical antimicrobials have led researchers to investigate natural antibacterials as an appealing alternative for oral health. Secondary metabolites of natural products may have anti-caries properties and can be classified into three groups: phenolic compounds with benzene rings, hydrogen and oxygen, terpenoids made up of carbon and hydrogen and alkaloids that contain nitrogen.

Local seeds, plants and spices with known medical characteristics that are used in phytotherapy are mostly antimicrobial as well. Many plant species, and natural animal products such as chitosan and propolis, have been reported to have an antimicrobial effect.

Establishing the safety of natural products apart from normally consumed food for health is crucial, and the US Food and Drug Administration (FDA) assigns substances to the GRAS list, where the acronym ‘GRAS’ is derived from the first initials of the term ‘generally recognised as safe’. Numerous plants are on the GRAS list. In a recent study, the *in vitro* growth-inhibitory effect of 109 GRAS plant species was evaluated, and promising results for using these species as antimicrobials were obtained [59].

Many interesting local plants have been investigated for their antimicrobial and antibiofilm characteristics. Macelignan, which has been isolated from nutmeg, showed an antibiofilm activity *in vitro* [60, 61]. Tea (*Camellia sinensis*), cranberry (*Vaccinium macrocarpon*) and cacao, which all constitute important dietary components all around the world, are reported to display antimicrobial activity against oral pathogens [6, 62, 63]. Green tea has several advantages for oral health, such as an anti-adhesion property for the acquired pellicle, and is considered to be an antimicrobial [64, 65]. Coffee and barley coffee show an antimicrobial effect by inhibiting the adhesion of *Streptococcus mutans* [6, 62, 66].

Propolis alone [67] and in conjunction with chitosan [68, 69] showed a promising antibacterial effect *in vitro*. Twice daily use of mouthrinse that contained

propolis showed an antiplaque effect on six subjects in vivo [70]. Aloe vera is another extract of natural origin used as medicine that showed an in vitro antibacterial effect with propolis [71].

Two meta-analyses of the effect of essential oils revealed clinically relevant benefit of their use in reducing gingivitis and plaque accumulation. Mouthrinses with antimicrobial content are mostly used as an adjunct to mechanical tooth cleaning for gingival health. Daily use of mouthrinse that contained essential oils provided higher percentages of healthy gingival tissues and plaque-free tooth surfaces over mechanical cleaning only where the effect on carious lesion formation was not evaluated [72, 73]. Cinnamon, morrisonii oil, manuka, tea-tree oils and thymol exhibited a growth-inhibitory effect on cariogenic bacteria in vitro where in each case combining the substance with chlorhexidine to lower its concentration was suggested [74].

Commercially available mouthrinse, Listerine, which contains essential oils (EO) and ethanol, has a strong disinfection effect on cariogenic bacteria within the biofilm [5, 14]. On the other hand, its low pH (4.3) has the potential for tooth erosion, and its alcohol content may irritate the soft tissues. For these reasons, it is suggested that short-term usage of Listerine for individualised situations should be implemented in a patient's treatment protocol if necessary [14].

A natural product consumed as a dietary component will not be strong enough to exert the desired antibacterial effect in vivo, and even the obtained extracts cannot be accepted as a treatment vehicle. Using crude natural extracts in caries management presents problems because of their complex nature, which makes them difficult to purify, elucidate their structure and determine their molecular formula. Furthermore, the chemical composition of naturally derived products may vary, depending on the geographic region, season and biological diversity. The natural extract should contain bioactive molecules in proper concentrations. This means that bioactive compounds of natural origin should first be evaluated in vitro for their biological activity, and this is followed by dose-response studies [63].

Possible candidates for new active ingredients of natural origin are available. However, evaluating their reproducibility, proper isolation and safety by performing carefully planned experimental designs is crucial for producing a successful novel product that contains a therapeutically relevant compound with an anti-caries effect from the nature.

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## 5.7 Final Remarks

The systematic reviews cited in this chapter are unable to draw a clear conclusion regarding the effectiveness of antimicrobials from the studies they include. Within the limitations of these studies, we may conclude that, although bacteria act differently in the dental biofilm, some antimicrobials such as chlorhexidine when used for a time provide a tool for the dentist in reducing the plaque accumulation and bacterial content of the oral environment to some extent. Especially in children with (severe) ECC, orthodontic patients, the elderly with root carious lesions and people suffering from hyposalivation, antimicrobial therapy may be an additional tool alongside the removal of biofilm with toothbrush and fluoride toothpaste. In

low- and/or medium-caries-risk patients, chlorhexidine mouthrinse or gel might be a suitable tool for maintaining oral health. Regular recall patients may benefit from chlorhexidine varnish applied by the dentist. Further studies, especially randomised clinical trials, are needed in order to determine which is the most effective antimicrobial for assisting dental caries in vivo.

*Here are some questions to think about before using antimicrobials in the caries management of your patient:*

Why does my patient need an antimicrobial?

(High bacterial load? Xerostomia? High caries risk?)

Will my patient benefit from an antimicrobial alongside the proven methods?

Do I need to use another method in conjunction with the antimicrobial?

If required, which delivery system (solution, rinse, gel or varnish) is more effective for my patient?

Is the antimicrobial applicable for my patient? (In terms of age, habits or lifestyle?)

How long should my patient use the antimicrobial?

How often do I need to see my patient and repeat the treatment?

Is the antimicrobial cost-effective?

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## Abstract

Diet is one of the most important aetiological factors in dental caries. This chapter describes the relationship between sugar consumption and dental caries through history. Cariogenicity of food items and diet as well as factors that influence the cariogenicity are reviewed. The interplay of diet with other preventive measures also receives attention. Prevention through diet with natural sugar alcohol, xylitol, and beneficial bacteria (probiotics) is discussed. Finally, diet counselling for caries prevention for different age groups is summarized and dietary suggestions with health benefits are reported.

## 6.1 Introduction

Dental caries is a multifactorial, behavioural disease where dietary habits are one of the most influential behaviours of the host that affects oral health. In modern societies, besides a healthy diet, children are guided to brush their teeth to maintain regular oral health. It is interesting to look through the history of mankind and observe the situation for children in ancient times as far as diet and oral hygiene are concerned. Historical investigation so far has not provided us with any evidence of toothbrushing or other means of tooth cleaning (Fig. 6.1).

The most common diet of Byzantium in the thirteenth century indicates the consumption of food and drink that was less likely to cause carious lesions (Fig. 6.2). Ancient European populations of the late middle age (eleventh to thirteenth century) from Croatia, England, Italy, Scotland, and Turkey had a low caries prevalence

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**Fig. 6.1** Mandible of a 6-year-old child from seventh-century Byzantium discovered during the metro project in Istanbul with teeth shapes, sizes, and number similar to present-day children with no dental caries



(5.1–13.2%) [1]. Intrinsic sugar was predominant, dental attrition was seen, and despite the promoting effect of diet on oral health, a short life span, diseases, and wars were factors that made dental caries an uncommon consideration in premodern human life. It was Miller [2], in 1890, who first correlated dental caries with diet and concluded that acids produced from starches and sugars by salivary bacteria lead to carious lesions. It is widely accepted that there is an association between the amount and frequency of free sugar intake and carious lesion development [3]. Although the evident threshold of sugar cannot be estimated, dose–response relationship between free sugars and level of caries had been reported [4].

Epidemiological studies showed the relationship between sugar consumption rate and dental caries where the introduction of the sugar industry, and increase in consumption rate, increased the dental caries rate in the Tristan da Cunha islands from 1938 to the 1960s, and the restriction of sugar during World War II and the United Nations sanctions in Iraq showed the opposite. Sequential annual studies conducted by Takeuchi and colleagues in Japan also showed the clear relationship between high sugar intake and increase in cavitated carious lesions over time [4].

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## 6.2 Cariogenic Diet, Cariogenicity of Food, and Dental Caries

Today, it is ethically inappropriate to measure carious lesion progression related to a cariogenic diet. However some dietary intervention studies [5, 6] investigated the effect of consuming sugary foods on special groups of people. The Vipeholm study [5] was the first to show that increased frequency of consumption of sugar between meals was associated with a significant increase in caries progression. This study led the researchers to recognize the importance of eating patterns as well as the content. On the other hand, the Hopewood House study [6] showed that a lactovegetarian diet without sugar provided a healthy dentition in children residing in the

Food Type	Mostly consumed	Never consumed
Meat	<i>Pig**</i> , <i>sheep</i> , <i>goat</i> , <i>chicken</i> , <i>fish</i> , <i>shellfish</i>	beef, cattle(working animals)  turkey†
Dairy products	<i>Cheese*</i> , <i>feta</i> , <i>egg</i> , <i>milk</i> , <i>butter</i>	Other dairyfoods
Sugar/desert	Honey, rose sugar, quince  mamalade, rice pudding, flatcakes (fried in oil)	Sugar cane, other sugar types, sweeteners
Carbohydrates	Bread, paximadion (hard dried bread)**	Biscuits, others
Vegetables	Olive*, cabbage, onion, legume, garlic	Tomato†, potato†, sunflower†
Fruits	Citrus fruits, fig, grape, pomegranate, apple, apricot, hazelnut	Tropical fruits†
Drinks	Wine with warm water**, water vinegar, soft drinks, water (limited amounts)	Tea†, coffee†, carbonated beverages, juices, coke

\* Typical diet of low income families,

\*\* Typical diet of Byzantine army,

*foodstuff*; never consumed in Lenten fare,

†not discovered (discovery of America, 1492)

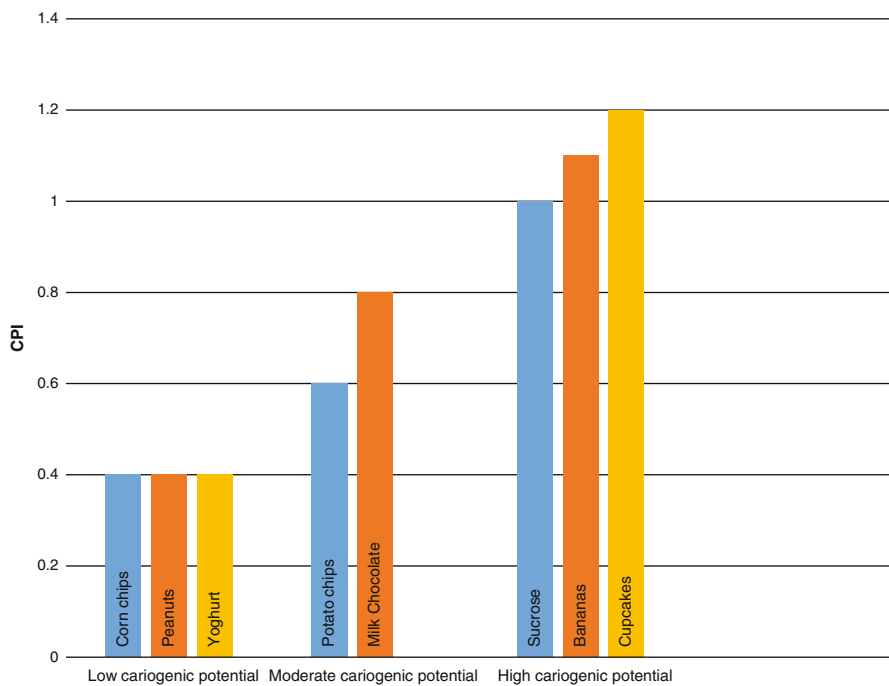
**Fig. 6.2** Typical Byzantine diet in the thirteenth century (Reproduced from Caglar et al. [1])

dormitory, and when they left, environmental change and alteration of the diet caused dental caries as they grow older.

The term ‘sugar’ is used to define sucrose or sometimes any kind of fermentable carbohydrate in the literature. We will refer to ‘sugar’ as fermentable carbohydrate.

There are two major sugar groups: intrinsic (natural) and extrinsic (added) sugars. Intrinsic sugars are those found naturally in the cellular structure of fruits and vegetables. Natural sugars are monosaccharides (glucose, fructose, mannose, galactose) and disaccharides (maltose, sucrose, lactose, trehalose). Extrinsic sugars are free in food. Extrinsic sugars are further divided into milk sugars (lactose) and non-milk sugars (corn syrup, maple syrup, honey, added sugars).

A cariogenic diet is defined as foods and drinks containing fermentable carbohydrates that can cause a decrease in plaque pH below 5.5 [7]. The cariogenicity of a food may be evaluated by animal studies or plaque pH measurement studies in vivo. The acidogenic potential of a food or beverage that can lead to dental caries is called the cariogenic potential index (CPI) [8]. In a rat model, sucrose has a CPI of 1 where >1 means ‘high CPI’ and <1 means ‘low CPI’ (Fig. 6.3). Yogurt has a low CPI of 0.1, while banana, with intrinsic sugar, has a higher CPI of 1.2. When plaque pH is used, tested foods may be ranked according to maximum pH drop, and in an in vivo study conducted in children using micro-touch pH electrodes, the tested snack foods were ranked in ascending order as cheddar cheese < diet coke < milk < banana < potato < potato chips < biscuits < coke < bread < milk chocolate < 10% sucrose solution [9].



**Fig. 6.3** Cariogenic potential index of some snack foods (Data modified from Mundorff et al.) [7]. Authors calculated the ratio of dental caries produced by the test foods to that of sucrose. Sucrose was given a CPI of 1.0

It is important to note that acidogenicity alone cannot define cariogenicity because of the complicating nature of contributing factors on dental caries. Likewise, it is very hard to categorize foods according to cariogenicity because this is affected by complexity of foods in texture and mineral, fibre, and water content.

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### 6.3 Factors That Influence the Cariogenicity of Diet

Early childhood caries is one of the oral conditions that is influenced by the cariogenicity of diet as well as the way the food is consumed. Inappropriate feeding practices such as bottle- and breastfeeding at will are one of the main causes of early childhood caries where the carious lesions are located on tooth surfaces that the cariogenic fluid passes within the mouth. Maxillary incisors exposed to the cariogenic fluid containing sugar the most show severe carious destruction.

Dietary factors have also been associated with root caries in the elderly [10, 11]. The amount of sugary foods, as well as frequency of consumption, was found to be effective in root caries formation in the elderly where gingival recession was mostly seen. Papas et al. [10] reported that caries-free elderly consumed more cheese and fibre- and protein-containing foods and less sugar.

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### 6.4 Interplay of Diet and Other Preventive Measures

Regarding the complex nature of dental caries, other constituting factors interplay with diet. van Loveren and Duggal [12] noted that it would be a mistake to classify a diet as ‘cariogenic’, but prefer to define it as ‘potentially cariogenic’ since other factors may contribute to cariogenicity. The diet has become a lesser factor in caries prevention where oral hygiene and fluoride supplementation are adequate. It should also be highlighted that for the last 70 years, fluoride has been bound into the diet by virtue of its addition to water, milk, and salt [13]. We may think of fluoride rather as a safety belt that protects teeth from hazards. The topical presence of fluoride has a positive effect in caries prevention but by itself will not totally counteract the negative effect of a cariogenic diet.

The retentiveness of a food is very important in its cariogenicity, and oral clearance is influenced by its adsorption onto tooth surfaces, plaque metabolism, saliva content, and flow [8]. The simultaneous mechanism of oral clearance with swallowing will be affected by the amount of saliva. The less the amount of saliva and the longer the food remains in the oral cavity, the higher the caries risk will be. Foods that increase the salivary flow will eventually increase the buffering capacity and neutralize plaque pH, whereas intake of increased alkaline substances containing calcium, phosphate, and casein will enhance remineralization.

## 6.5 Caries Prevention Through Diet

High sugar content, especially the extrinsic sugar in diet, has been found to be effective on carious lesion development. Adolescents consume large amounts of confectionary and sweet foods that contribute to their daily energy intake. Only 30 % of this intake are intrinsic sugars, while 70 % are extrinsic sugars. It is widely accepted that intrinsic sugars, even though calorific, have low cariogenicity. Regarding sugar substitution, it is possible to replace extrinsic sugars with foods containing intrinsic sugar or by sweeteners of low cariogenic potential that are not metabolized by oral bacteria.

The sugar substitutes may be divided into two subgroups: intense sweeteners and sugar substitutes. Intense sweeteners are saccharin, cyclamate, aspartame, and acesulfame-K. Today, intense sweeteners are mostly replaced by sugar substitutes (polyols: sugar alcohols) because of mass production and technological advances. More functional sugar substitutes are derivatives of monosaccharides (sorbitol, xylitol, mannitol, erythritol) and derivatives of disaccharides (isomalt, maltitol, lactitol, palatinit, and lycasin). Generally, sugar substitutes are not used in biscuits or baked products as they cannot be caramelized and turn brown. They are however used in chewing gums, beverages, and medicines.

The sweeteners and health-promoting bacteria are the food substances that are beneficial for teeth and used in the food industry for caries prevention. Therefore xylitol as a sweetener, and bacteriotherapy in relation to probiotics, will be discussed in the following sections.

### 6.5.1 Xylitol as a Sweetener

Xylitol is a natural sugar alcohol commonly used as a low-calorie sweetener [14, 15]. Adequate daily doses of this pentitol might affect the oral ecology where plaque acidogenicity and the proportion of oral mutans streptococci show a decrease [16, 17].

Therapeutic doses of xylitol are reported to be safe for children in terms of prevention from dental caries [18]. Xylitol is accepted as a preventive measure for caries reduction in high-risk populations [19]. The American Academy of Pediatric Dentistry (AAPD) states that daily usage of 6 packs of xylitol chewing gum by mothers will decrease maternal transmission of mutans streptococci [20]. This may also be suggested for children older than 4 years of age. The American Academy of Pediatrics (AAP) does not recommend the use of chewing gum, mints, or hard candy by children less than this age due to the risk of choking [18].

The Turku sugar studies in Finland in 1971–1973 were the first clinical trials reporting the positive effects of xylitol on dental caries [21]. The caries preventive effect of xylitol has been evaluated by clinical and epidemiological studies mostly using chewing gum [22–31]. The Turku sugar studies have shown that the addition of xylitol in the diet results in very much lower caries increments. Xylitol hampers bacterial growth through a fructose-specific phosphotransferase

system and is phosphorylated to xylitol-5-phosphate [32]. Xylitol inhibits further intracellular metabolism of the bacterial cell. Söderling et al. [30] stated that ‘after exposure to xylitol, a shift towards xylitol-resistant mutants streptococci has been shown in saliva and it has been suggested that those strains have a reduced ability to adhere to the tooth surfaces’ [33, 34]. The anticariogenic effect of xylitol-containing gums and candies seem to be based more on saliva stimulation and a lesser extent on antimicrobial effect [34]. The ‘smart habit’ campaigns of the 1970s showed that xylitol reduced dental caries in Finland. However it should be noted that xylitol works hand in hand with a fluoridation programme in the society. Currently Lee et al. [35] regarded xylitol consumption as an effective method of preventing dental caries. However they could not find any additional benefit compared to other prophylactic measures such as oral health education, toothbrushing and fluoridated toothpaste, topical fluoride varnish treatment, and dental sealants.

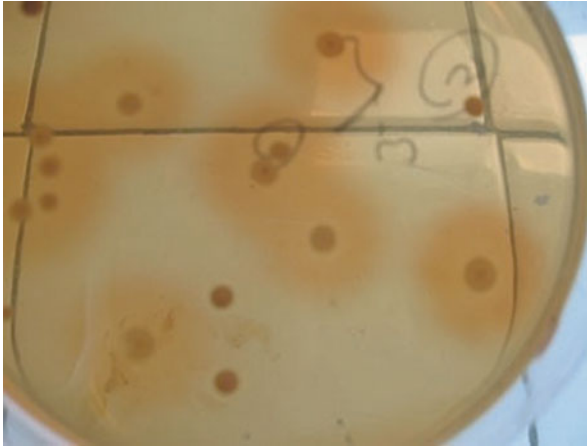
Besides being a dietary component, the use of xylitol as an additive was questioned for its synergistic effect on fluoride. Currently, there are toothpaste formulations with 10% xylitol (dose of 0.1 g/brushing) sold in the USA but have never been tested, and no recommendations are suggested. A recent Cochrane review reported a low quality of evidence on its effect in all available products (gum, lozenges, wipes, toothpaste, etc.) so far [36].

### 6.5.2 Bacteriotherapy and Probiotics

Bacteriotherapy is a replacement therapy where beneficial microorganisms, so-called probiotics, are consumed to keep the immune system sound. Probiotics have been used in daily foods since ancient times, but research currently is interested in the role of these bioactive food compounds in health [37]. These live microbial food supplements are beneficial for the host by influencing the balance between the many species of the commensal flora both in the oral cavity and the rest of the digestive system [38, 39]. Lactobacilli and bifidobacteria are named as the most often investigated and widely used probiotics. Probiotics can be categorized as mono- or multi-strain. A mono-strain may not overcome barriers presented by the host and its endogenous microflora, while multi-strain probiotics have a greater chance of survival of at least one strain [39].

Caglar et al. [40, 41] state that probiotics work in food supplements in one of four basic ways:

- As a culture concentrate added to a beverage or food (such as fruit juice, bio drink)
- Inoculated into probiotic fibres
- Inoculated into a milk-based food (dairy products such as ice cream, milk, milk drink, yogurt, yogurt drink, cheese, kefir)
- As concentrated and dried cells packaged as dietary supplements (nondairy products)



**Fig. 6.4** Reddish brown probiotic zones show reuterin production around the *Lactobacillus reuteri* ATCC 55730

It should be noted that the shelf life of the product is vital in order to maintain the viability of the beneficial bacteria in a stable state [42]. The time period for shelf life is 15 days for a yogurt with decreasing number of viable probiotic cells whereas 2 years for an ice cream that will be kept unaltered [43].

To date, the exact mechanism of probiotic action in the oral cavity is not fully understood. It is accepted rather as a combination of local and systemic immune responses to cariogenic pathogens, as well as non-immunologic defence mechanisms. The principal health-promoting effects are ascribed to enhancement of mucosal immune defence and macrophage activity, production of antimicrobial substances such as reuterin (Fig. 6.4), signalling and resolving adherence mechanism, combating with pathogenic bacteria, and elevations in the numbers of killer cells, T-cells, and interferon in laboratory conditions [44–47].

### 6.5.2.1 Probiotics and Colonization

It is important to understand colonization by oral bacteria and its relation to carious lesion formation in terms of bacteriotherapy. The colonization process by oral microbes is complicated. The major route of early acquisition and colonization by mutans streptococci in humans is a vertical transmission from mother to child [48]. It appears easier to affect or change the levels of caries-associated bacteria at the time of its colonization compared to later in life when the flora is firmly established. It is widely accepted today that early colonization by oral bacteria starts right after birth. Recently, authors developed a probiotic lozenge-administered medical device in the form of a novel pacifier where the infant sucks beneficial probiotic bacteria through a lozenge that totally dissolves into the oral cavity in approximately 12 min providing early colonization (Fig. 6.5) [49].

In the early years of life, there is a space in the ecological niche with sound and pathologic bacteria surviving together (Fig. 6.6). Inviting a guest such as the probiotic



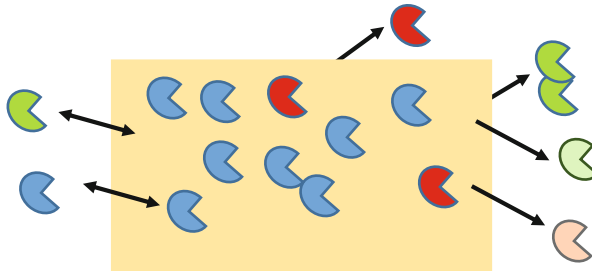
**Fig. 6.5** Probiotic lozenge-administered medical device in the form of a novel pacifier



bacteria needs a pathologic one to leave the room. This process is reversible; however, multi-strains have a better chance in the room. They might be temporary colonizers (guest for a while and leave) or diminish. The same mechanism works for the pathologic bacteria where they leave and survive or diminish. This mechanism somehow works in saliva and biofilm. However there is lack of data regarding how probiotics work in active dentine caries lesions.

In this mechanism, the probiotic crusade might be temporary or permanent. Temporary colonization generally works in adults whereas permanent colonization works in infancy and childhood. Therefore, probiotics must be consumed every day for sustainability in adults. Infants and children have a different way of dealing with the probiotic challenge. Their oral cavities are more open to probiotic competition where newly acquired caries-related microorganisms would lose this battle against supplemented probiotic bacteria. Novel control strategies could control the microbial activities using biofilm and dental plaque to prevent colonization of selected organisms while supporting growth of other selected ones [49, 50].

The possibility of permanent colonization was questioned in a Finnish study [51]. It was reported that although they had withdrawn the use of *L. rhamnosus* (LGG) products, one of their subjects who had received LGG milk during childhood for 1 year was found LGG positive in her saliva up to 5 months later. [51]. The question arose as to whether there was 'permanent colonization of probiotics in childhood'. In another study, the potential role of *L. reuteri* ATCC 55730 on



**Fig. 6.6** Sound (*blue*) and pathologic (*red*) bacteria survive together in an ecological niche (*yellow rectangle*), and inviting a guest such as the probiotic (*green*) bacteria needs a pathologic one (*red*) to leave the room. This process is reversible; however, multi-strains (*double green*) have a better chance in the room. They might be temporary colonizers (guest for a while and leave) or diminish (*faded green*). The same mechanism works for the pathologic bacteria where they leave and survive (*red*) or diminish (*pink*)

infant oral health in relation to early colonization in infancy has been validated in vivo and *L. reuteri* promised to be a good ‘first colonization strain’ and ‘good survivor colonizer’ [52].

### 6.5.2.2 Probiotics and Effects on Oral Health

High counts of mutans streptococci may be one of those influential factors of carious lesion formation. Recent probiotic studies on oral health generally focused on its relationship to mutans streptococci where diminishing mutans streptococci is believed to stimulate healthy bacteria and make saliva/plaque less virulent [50, 53]. Actually, a general decrease in levels of salivary *Streptococcus mutans* was noted in particular studies where probiotic strains such as *Lactobacillus rhamnosus* GG [54, 55], *L. acidophilus* and *L. casei* [56], *L. rhamnosus* LC705 [57], *L. reuteri* [41, 58, 59], *Bifidobacterium* DN-173 010 [49], *Bifidobacterium lactis* Bb-12 [42], or a lactobacilli mix [60, 61] had been administered. However, Stecken-Blicks et al. [62] and Toivianen et al. [63] did not find any alterations within salivary mutans streptococci levels by the probiotic intervention. To date there are some controversial reports that probiotic usage in infancy may reduce caries prevalence in children [64, 65]. A current meta-analysis [66] states that there is insufficient evidence that probiotics can prevent caries, but they can reduce the mutans streptococci counts. Therefore, regarding probiotics’ effect on dental caries, there is still lack of data because of a newborn subject’s nature, and long-term longitudinal studies are further needed to show the relation between probiotics and *Streptococcus mutans* and its further effect on dental caries.

At present it is difficult to advise patients and their children whether to use probiotics or not. All one can say is that there is substantial evidence that probiotics have a negative effect on the presence of the harmful *Streptococcus mutans* bacteria. This can only be a good thing.

## 6.6 Diet Counselling

The availability of processed food in the global market has changed the way people eat. Cultural and social factors influence diet, and a cross-sectional view of the world shows that many industrialized countries are overweight. On the other hand, it has been stated that in developing countries, before people attain a level of prosperity to ensure adequate nutrition, they consume in ways that almost guarantees to make them less healthy [67].

Diet is structured by individual preferences, likes and dislikes, availability, and food habits within the family. Regarding children and adolescents, their parents would also be questioned about their own preferences. It is important to know if it is a 'sugar-oriented house' or not. Growing up in a sweet-dependent family makes the child wish for more sweet foods all their lives. It is also wise to ask parents if they drink any beverages (like coffee) during the day – how often and with how much added sugar. Sometimes 5 cups of coffee a day with two sugars add up to 10 sugar cubes, a halo effect, that the practitioner never sees but cariogenic activity prolongs. Most of the patients claim that they can't understand how carious lesions proceed even though they are brushing. Is there a genetic predisposition in their families? Most people claim that they brush their teeth but are not informed about these halo sugars where their dental tissues are under acid attack all day long. One may observe a child drinking milk with added sugar in the same way his/her parents do with their beverages.

Diet counselling by diet history records may help the practitioner to understand the dietary component on patients' dental caries risk. Recording a cariogenic diet history is also the cornerstone for the dental practitioner's recall programme. It should be noted that diet changes all through life from infants to toddlers, pre-schoolers, schoolchildren, and adolescents, whether you are in Greenland or India. Regarding the present situation, the recall programme must start from the very beginning, even right from the fetus. Therefore, it is wise to inform parents during pregnancy about healthy food and a regular diet (see Chap. 9). In biannual recalls, age-related diet advice should be followed up with specially developed forms or advanced messaging, like sending photos using new technologies, though this may not be possible for all levels of society or patient ability.

Parents should record all food consumption during a specific period of time, normally 3 consecutive days (including a weekend): a '3-day dietary form to check!' Practitioners may think of this as impractical. However, it is encouraging to see most parents reporting back to the dental team in various ways: faxes, emails, and handwritten and 'WhatsApp' messages with pictures and advice given according to the habits of the individual being more effective and applicable (Fig. 6.7). In this manner, the practitioner will be able to inform patients about caries-promoting foods and beverages and on how to organize their consumption frequencies and limitations. Koch et al. [68] recently stated that inquiries into the dietary habits of patients are a necessary basis for advice concerning future changes in diet to prevent dental caries.



**Fig. 6.7** Advanced technologies may motivate young patients regarding dietary habits

The practitioner should also explore the ingredients of food and beverages sold in supermarkets and other outlets. This should be accepted as a dentist's social and medical task. The importance of food labelling all over the world has increased dramatically. All manufactured food packages have their ingredients and nutritional values clearly displayed. It is important to know how to read labels and interpret them correctly (added sugars, fat, etc.). Dentists should guide their patients about the content of processed foods which in turn lets patients know that the food they are eating has a lot of sugar or not.

It is obvious that it is futile to inform a child or adolescent about a healthy diet if the parents are sugar and carbohydrate addicted. Being a successful dentist for a dental patient is like running a long-distance marathon. Supporting attempts such as sugar substitution and bacteriotherapy to affect the oral ecology via diet may be used. It is important to never give up with patients' dietary habits. There will always be new sources of food that will especially challenge youngsters' dietary habits, and several motives and new products may be used in giving advice.

The United Nations' attempt to combat obesity provides an opportunity to reduce free sugar use that will also provide oral health benefits. At the individual level, people can limit energy intake from total fats and sugars and consume more fruits and vegetables, as well as legumes, whole grains, and nuts [69]. The WHO strongly recommends reduced intake of free sugars all through the life span of an individual. Secondly, intake of sugars should be reduced to 10% of total energy intake both for children and adults. This will affect the body mass index of the person and avoid obesity as well. This will only be possible when the public and stakeholders support the individuals by providing information and easily accessible, affordable, and available choices for healthier dietary sources.

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## 6.7 Dietary Guidelines and Practical Suggestions

### 6.7.1 Infants (0–1-Year-Olds) and Toddlers (1–3-Year-Olds)

- Expectant mothers should be educated about oral health behaviours for the newborns.
- Ideal and adequate food for the first 6 months of an infant's life is breast milk [70]. Infants should start receiving complementary foods after 6 months in conjunction to breastfeeding.
- Two years of breastfeeding is promoted by the WHO. Breast milk contains 7% lactose [71, 72]. It has been stated that solely breastfeeding does not contribute to early childhood caries [73, 74]. However, prolonged and night breastfeeding should be avoided because of the aetiologic role of milk sugars in early childhood caries [75, 76]. This advice is not based on findings from controlled interventions.
- The American Academy of Pediatrics [77] states that after 6 months of age, the use of a spoon or glass should be guided.
- Bottle feeding should be avoided after age 1.

- All tooth surfaces of erupted teeth should be cleaned after every feeding as much as possible.
- Frequent food and drink consumption through the day and sleep periods should be avoided for toddlers.
- Toddlers should not consume mashed or grounded food but should chew properly.

## 6.7.2 Childhood

It is important to use appropriate age-related advice while discussing a patient's diet. From infancy to adolescence, a child's eyes look around and perceive details of their surroundings. Furthermore, children have a short-term memory of a particular moment therefore giving too much information will make them confused. Please use age-appropriate language for the patient. Give 'take home messages' for children. A sticker is acceptable for a 5-year-old as a motivation, while for a teenager, a cell phone message is more age appropriate. Always use the word 'I' instead of 'you' (Fig. 6.8). This is the best way to gain sympathy and also empathy [78].

Here are some basic dietary suggestions for schoolchildren:

- A balanced diet is necessary for overall health and oral health [79].
- Guide the child to three main meals (breakfast, lunch, and dinner) and two snacks, one in the morning and the other in the afternoon.
- Refined carbohydrates should be limited to meal times.
- Two intermediate intakes among main meals could be nuts, cheese, and unsweetened yogurt.
- Suggest drinking tea and coffee or other hot beverages without added sugar.
- Advise to drink water. It stimulates saliva and replaces sweet drinks. Children forget to drink water if they are not reminded. Therefore, guide children to consume water.
- Sucrose-containing carbonated beverages and sweet snacking must be avoided. It should be noted that increasing the frequency of sugar intake increases the odds of developing carious lesions, while lowering sugar intake can reduce it [80–83].

*Gjergj, you did not brush your teeth well!! Gjergj, Let me see..*

*Did I understood correctly that you had not enough time to brush?*

*Marin, you won't consume chocolates! Marin, I believe that eating chocolates*

*may hurt your teeth...let's talk about how we'll eat them?*

**Fig. 6.8** Dialogue with the child. Red: negative reinforcement. Green: positive reinforcement

- School canteens and vending machines should serve only healthy food for schoolchildren. If possible speak to the school authorities about this.
- Dried fruits may contain sugars but are a very good source of fibre and micronutrients. Suggest eating them as part of a meal rather than a snack.
- Ice cream and frozen milk products with refined sugars may be replaced by homemade fruit smoothies containing fresh fruit and milk with no added sugar.
- Children of the 1970s grew up with one TV channel and less refined sugar advertising. However, now there is a generation under attack from multimedia exposure and GRAS (generally recognized as safe)-defined extrinsic sugars [84]. Inform patients to consume less extrinsic sugar.
- Encourage parents and adolescents to read the food labelling of manufactured confectionary and buy less.
- Inform patients about the caries and obesity risk due to high fat and sugar content in cakes, biscuits, cookies, donuts, and sugared snacks. Suggest consuming less.
- Advise patients to use nonsugar sweeteners, in particular xylitol, in foods and drinks.
- Inform families and medical staff about the advantages of sugar-free medicines. Medicines for children need to be highly sweetened to make them easier to administer [85, 86]. Damage to children's teeth, especially in children with systemic diseases, has resulted in the widespread availability of sugar-free alternatives for most paediatric medications.
- Encourage patients to use sugar-free chewing gum, when this is acceptable. Chewing gum can be used as a delivery vehicle for fluoride, minerals, alkalizing agents, and chlorhexidine. Chewing gums containing xylitol and sorbitol have anticaries properties through salivary stimulation.

### 6.7.3 Adults and the Elderly

In addition to the suggestions reported above, it is important to note that the number of teeth present affects the nutritional status of adults and the elderly [87]. An inadequate dentition together with a lowered salivary flow due to medications reduces the amount of fibre and chewable food consumption. The elderly prefer to consume overcooked vegetables or over-prepared fruits (cooked or mashed) or high-energy liquid food supplements sweetened with sucrose *ad libitum* for providing sufficient nutrition that leads to carious lesions [88, 89]. It is important to advise adults and the elderly about reducing the consumption of such foods and supplements.

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## 6.8 Final Remarks

Diet is closely related to oral health and the well-being of a person. The sequence of examples from history shows the adverse effect of processed food and added sugar on dental caries development. The frequent consumption of sugar and a cariogenic diet in the Vipeholm study, and restriction of sugar in the Hopewood study, form the

foundation of our knowledge in the sugar–caries relationship. Extrinsic sugars affect the cariogenicity of food. Studies show that at-will bottle- or breastfeeding practices for infants, frequent consumption of sugar-containing snacks, and avoidance of oral hygiene practices cause carious lesions to develop.

Sweeteners lower the overall sugar load of a diet, and especially the sugar alcohol, xylitol, is accepted as a preventive measure for caries reduction in high-risk populations. The most preferred xylitol vehicle has been chewing gum, but this needs a high degree of compliance. Probiotics administered mostly through dairy products should be used to provide local and systemic immune responses to cariogenic pathogens and prevent decay.

Overall, diet counselling according to the habits, needs, and preferences of the patient is necessary for managing dental caries. The proposed dietary suggestions, together with their compliance, will help improve the oral health of all age groups. The attempts of several organizations such as the United Nations and WHO on better overall health also include practices that will certainly affect oral health positively all over the world.

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## Abstract

Research over the last five or so decades has provided ample knowledge regarding risk and protective factors that influence the caries process. One of the protective factors concerns turning potentially vulnerable tooth surfaces into non-vulnerable ones. This procedure is known as sealing pits and fissures systems. The seal changes a rough tooth surface into a smooth one from which the biofilm can be removed relatively easy.

This chapter presents and discusses clinical aspects of sealants. Outcomes from evidence-based studies have been used as much as possible. Aspects that have been given attention include: indications for sealing pits and fissures, (dis)advantages of common materials used for sealing, description of sealant application step by step, their effectiveness over time and to what extent sealants manage micro-cavities.

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## 7.1 Introduction

In essence, dental caries is a cavitated dentine carious lesion preventable disease. The term ‘cavitated dentine carious lesion’ is used here as it more precisely reflects the situation described formerly with the term ‘caries’. The determining factor in the analysis of whether a person or a tooth was considered to have caries was previously always the dentine cavity as carious lesions in enamel were considered sound.

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The statement that dental caries is preventable can be made with full confidence, as millions of young children, adolescents and adults show no evidence of a restoration or an unrestored tooth cavity. This observation is supported by results from epidemiological studies that have shown a decrease in the prevalence of cavitated dentine carious lesions in many countries since the 1980s and, at the same time, remarkable decrease in dmf/DMF counts. The question arises: what are these people likely to have done to achieve this result? They have most probably exercised proper biofilm control with a toothbrush and fluoride-containing toothpaste regularly (daily), controlled their intake of sugar-containing food items and drinks and have visited a dentist for a check-up consultation on a regular basis. Van Loveren and Duggal [1] clearly explain the interplay among sufficient oral hygiene practice, sugary diet control and sufficient saliva production in a 2001 study.

This reality is not applicable to all people. For various reasons, people in different parts of the world cannot or do not perform the three major cavitated dentine carious lesion-related preventive actions on a regular basis so as to avoid the occurrence of tooth cavities. Under such circumstances, cavitated dentine carious lesion-promoting factors become important.

One of these factors, which relates to the focus of this chapter, concerns the morphology of tooth surfaces, in particular those that contain a pits and fissures system. Pits and fissures in people's teeth vary in form, width and depth. This variety is found across systems and within the same system. If the morphological structure of the pits and fissures is narrow and deep, it is usually difficult to remove the biofilm from the deeper parts of the system. Depending on the regular availability of sufficient fermentable sugars and the absence of sufficient carious lesion-protective factors, the (de)remineralisation cycle will move out of balance and move towards the demineralisation site. If this process continues over time, the effect of demineralisation is seen in the appearance of a carious lesion(s) in the enamel. These lesions should not go unnoticed as healthy teeth-maintaining actions can be taken to stop the demineralisation process from progressing and from further destroying the enamel. If this situation is seen in children who are categorised as high-caries risk, additional actions need to be taken (for caries risk assessment, see Chap. 3).

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## 7.2 Protective Measures for Keeping Pits and Fissures Healthy

A number of measures have been proposed over the years as having a carious lesion-preventive effect on pits and fissures. These include biofilm removal through toothbrush and fluoride-containing toothpaste, fluoride gel, fluoride varnish, silver diamine fluoride (SDF) and chlorhexidine application, casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) paste and ozone [2]. The protective action of toothbrushing with fluoride-containing toothpaste and of CPP-ACP acts on the removal of biofilm mechanically in conjunction with remineralisation of enamel. The protective action of fluoride gel and fluoride varnish also acts on the remineralisation of enamel, while SDF, chlorhexidine varnish and ozone act on

reducing the number of micro-organisms in the biofilm of a pits and fissures system and on the occlusal surface in general. Following investigations, there appears to be no or insufficient evidence that application of SDF, chlorhexidine varnish and ozone is effective in controlling carious lesion progression in enamel [2], although the effectiveness of fluoride gel and fluoride varnish application has been established [2, 3]. Studies on the effectiveness of CPP-ACP appear to have conflicting results [2], while studies on toothbrushing with fluoride-containing toothpaste and those that focus on erupting molars (Nexo Programme) show these methods to be effective, provided that the brushing is performed regularly and under good guidance [4].

All these healthy teeth-maintaining options presented above rely for their effectiveness on the cooperation of a person with their dental practitioner. In other words, a person has to visit his/her dental practitioner to receive any of these treatments and needs to do this regularly if the person's caries risk is moderate to high. A commonly used healthy tooth-maintaining measure that has not been mentioned yet is the dental sealant; this measure is the focus of this chapter.

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### 7.3 Indications for Placing a Sealant in General

In contrast to sealing any deep pits and fissures, a sealant should be placed only on pits and fissures that show signs of carious lesion activity or are at risk of developing carious lesions in the near future. As sealants are usually placed on (just) erupted (pre)molars, the level of caries risk of the child in the primary dentition is a good predictor for carious lesion development in pits and fissures of permanent molars [5]. This individual caries risk cut-off point between low- and high-caries risk is in some studies expressed as  $dmft \geq 2$  [6, 7]. But being a high-caries risk individual is not a strong enough reason for using a sealant, according to cost-effective principles. The caries risk at the tooth surface level should also be established. Pits and fissures morphology (medium and deep), in combination with or without signs of carious lesion activity (presence of biofilm, roughness and/or whitish colour of the surface), is a factor that determines the state of carious lesions in pits and fissures (see Chap. 3).

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### 7.4 Reasons for Placing a Sealant

The fact that a child has (pre)molars with high-caries risk pits and fissures shows that she/he has been unable to adhere to sufficient proper biofilm control in combination with a healthy, low-sugary diet. It means that these teeth need immediate protection.

For various reasons, large differences exist in biofilm removal and control of sugar consumption among population groups across the world. Some parents start controlling the biofilm in their child from the moment the primary teeth erupt, and others do this only when the first permanent teeth erupt. The former may have been better educated than the latter on oral health. The chance that pits and fissures are at

a stage of high-caries risk is assumed to be higher in children that have been educated in biofilm removal at a later age than those that started at an early age. This assumption suggests that these high-caries risk older children are in the process of being educated in applying biofilm control. Placing a quality sealant will provide time for educating the child on good oral health behaviour without the fear of the onset and/or progression of carious lesions during this process. It also means that sealants should not necessarily be considered permanent, such as is the case for restorations.

This rationale has implications for the decision about whether or not to reseal. If the re-exposed tooth surface, after disappearance of sealant material, is seen to be 'free' from biofilm and if there are no signs of caries activity in other teeth in the mouth, resealing should not be considered necessary. The child and parents/guardians have shown that they can master the biofilm removal process and should be commended for having achieved this. A sealant is therefore considered a temporary measure that can be expected to last for some 5 years. But it has a major advantage over the other healthy teeth-maintaining measures. It is applied only once, and if it is applied well, using quality sealant materials, the dental operator does not need to reapply material, as is necessary for all the operator-related healthy teeth-maintaining measures discussed.

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## 7.5 Choice of Sealant Material

The principal reason for placing a sealant is to allow easy biofilm removal from pits and fissures systems. In fact, a sealant is meant to alter a morphologically uneven 'mountainous' surface, in which biofilm can be trapped, into a smooth surface. And from a smooth surface, biofilm can be easily and simply removed.

Following the development of the adhesive materials, which are resin- and glass-ionomer-based, used for sealing purposes, there is no longer a need to carry out pits and fissures enameloplasty for retaining restorative material. This invasive treatment was part of the GV Black concept of managing carious lesion progression, which relied on the knowledge and opportunities available a century or more ago. Nevertheless, enameloplasty is still practised by many practitioners and perhaps is still taught at dental schools in many countries. This practice violates the principles of modern-day caries management, exemplified by the Minimal Intervention Dentistry concept, which advocates keeping healthy tooth tissue in place and allowing remineralisation of demineralised carious tissue to take effect. Not only is the enameloplasty superfluous but also the use of the drill holds the possibility of making children anxious about dental treatment in the future, and that should be avoided.

The two types of materials that have been used for sealing pits and fissures are resin- and glass-ionomer-based. These include the auto-cured and light-cured resins, compomers, resin-modified glass-ionomers and the low-, medium- and high-viscosity glass-ionomers [8]. The auto-cured resin, compomers, and the low- and medium-viscosity glass-ionomers are considered inferior sealant materials



compared to the light-cured resin, resin-modified and high-viscosity glass-ionomers (HVGIC). The choice of material is dependent on the clinical situation, the country's economic situation, and whether, for example, encapsulated or only hand-mixed glass-ionomer is available, the experience of the dental practitioner in handling the materials and the effectiveness of the type of sealants over time. Mind you, going for cheap materials also often means going for inferior health outcomes, as has been shown regarding glass-ionomer [9]. The dental operator has to use his or her conscience in case a choice between superior and inferior material can reasonably be made. In low-resourced countries and in certain healthcare systems, usually in public services, only one resin- or one glass-ionomer-based sealant material is available. The dental operator has to communicate to the people who are responsible for purchasing medical and dental materials that only quality materials should be purchased if the budget allows for it in order to achieve a high standard of care.

Despite no evidence being found that resin-modified glass-ionomer sealants differ significantly from resin-based sealants [10], this glass-ionomer-based material is not discussed further in this chapter neither will sealants placed with compomer. In the remainder of the chapter, auto-cured high-viscosity glass-ionomer and resin sealants form the focus of discussion as they are not hybrid or modified materials.

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## 7.6 Effectiveness of Sealants

In the literature, effectiveness is often expressed as the survival of fully and partially retained sealant material and as the survival of a cavitated dentine carious lesion-free tooth surface.

It is known that sealants deteriorate over time. The rate of sealant disappearance varies from brand to brand but is usually higher within the group of glass-ionomer than for resin-based materials [11]. Among the glass-ionomers, retention of the high-viscosity type, particularly when applied under finger pressure as part of the Atraumatic Restorative Treatment (ART) approach, is on average higher than for the medium-viscosity type [12]. Encapsulated high-viscosity glass-ionomers show higher mechanical strength values than the hand-mixed version [13].

Despite the early exposure of parts of pits and fissures to the oral environment following the use of glass-ionomer-based sealants, the failure rate, expressed in the development of a cavitated dentine carious lesion, is not higher in glass-ionomer-based than in resin-based sealants [8, 14–16]. This phenomenon led Frencken and Holmgren [17] in 1999 to state that sealant retention should be considered only a surrogate endpoint of sealant effectiveness, the true endpoint being the prevention of the occurrence of a cavitated dentine carious lesion. This statement has been investigated since then. A systematic review conducted in 2013 showed that loss of sealant retention appears to be an invalid predictor of clinical outcome [18] and should not even be considered a surrogate endpoint [19]. Mind you, this statement can only be made because the sealant materials that are currently available on the market fail rather sooner than later. If, in future,

sealant materials become available that have a much longer rate of retention than the current ones, this conclusion will need to be reassessed. But, in general, less attention should be given to the level of retention of sealant material as it is currently considered a temporary measure that assists the child in a period in his or her life in which the practice of biofilm removal through toothbrushing and fluoride-containing toothpaste should be mastered fully.

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## 7.7 Sealing Partly and Just Erupted Molars

The indication for placing a high-viscosity glass-ionomer sealant is not different from that for placing a resin-based sealant. However, glass-ionomers are more hydrophilic than resin-based materials. It is therefore logical to assume that a glass-ionomer- rather than a resin-based material should be used in sealing carious lesion-prone pits and fissures that cannot be kept absolutely moisture-free, such as in partly and just erupting molars and in children with behaviour problems. This does not mean that glass-ionomer sealants can be placed under ‘wet’ conditions. However, placing a resin-based sealant under moist to wet conditions is contraindicated [20].

A recent study compared resin-based sealants and sealants of glass-ionomer, placed using finger pressure as part of the ART approach, in erupting molars [21]. Using a low-viscosity glass-ionomer sealant material in erupting molar teeth, the study showed no differences between the caries-preventive effect of the glass-ionomer- and the resin-based sealants after 2 years. A different outcome was reported for a pilot study that had investigated the cavitated dentine carious lesion-preventive effect of an HVGIC ART sealant in newly erupted and erupting first molars [22]. This study showed that initially un-erupted first molars had a 2.1 times higher chance than sealed newly erupted and erupting first molars of developing cavitated dentine lesions, in a high-caries risk population of 6- to 7-year olds after 5 years. These few studies suggest that sealing partly and just erupted teeth is best undertaken using a glass-ionomer, preferably a high-viscosity type, for reasons of material characteristics and treatment protocol. Glass-ionomers do not require rubber dam for isolation, as it is impossible to place on partly and just erupted molars.

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## 7.8 The High-Viscosity Glass-Ionomer ART Sealant

The ART approach was developed in an attempt to manage dental caries in care-deprived communities [23]. Unlike what the name expresses, the ART approach consists not only of restoring tooth cavities (ART restorations) but also of sealing carious lesion-prone pits and fissures (ART sealants). ART sealants are defined by the fact that a high-viscosity glass-ionomer is placed over carious lesion-prone pits and fissures under finger pressure. Hand instruments, such as an excavator and an applier/carver, are used for adjusting the bite and removing

**Table 7.1** Step-by-step description of the placement of an ART sealant [31]

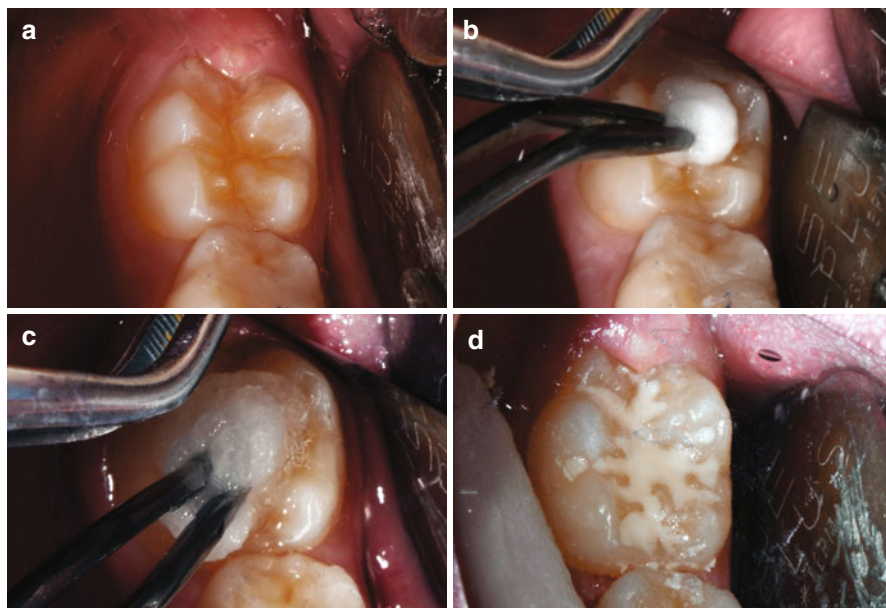
1. Isolate the tooth with cotton wool rolls. Keep the treatment area free from saliva
2. Gently remove plaque and food debris from the deepest parts of the pits and fissures with an explorer
3. Wash the pits and fissures, using wet cotton wool pellets
4. Apply enamel conditioner into the pits and fissures according to the manufacturer's instructions. Condition for the specified time
5. Immediately wash the pits and fissures, using wet cotton wool pellets to clean off the conditioner. Wash 2–3 times
6. Dry the pits and fissures with cotton wool pellets. Do not use the three-way syringe. The enamel surface should not be desiccated
7. Mix the glass-ionomer and apply it in all pits and fissures with the round end of the ART applicator/carver instrument or shake the encapsulated glass-ionomer in a suitable mixing machine and extrude the mixture into all pits and fissures
8. Rub a small amount of petroleum jelly on the gloved index finger
9. Press the glass-ionomer mixture into the pits and fissures with the index finger (press-finger technique). Then, remove finger sideways after 10–15 s
10. Remove visible excess of mixture with the carver or a large excavator
11. Check the bite, using the articulation paper, and adjust until comfortable
12. Remove the petroleum jelly top surface with the carver or a large excavator when the mixture is partly set
13. Apply a new layer of petroleum jelly
14. Remove the cotton wool rolls
15. Ask the patient not to eat for at least 1 h

excess material. In applying this approach, sealants can be placed independent of the need for rotary instruments and thus electricity and running water. The protocol for application of an ART sealant is presented in Table 7.1 and presented in Fig. 7.1.

## 7.9 The Resin-Based Sealant

Resin-based materials have a long tradition of use in sealing pits and fissures and consist of auto- and light-cured materials. Coloured and transparent materials are available, and both materials have their advantages and disadvantages. If a dentist is concerned about detecting sealant loss early, he or she might wish to use a coloured sealant material.

Controversy exists regarding whether a resin sealant needs to be applied under rubber dam or under cotton wool roll isolation. If the latter is used, the dentist has to ensure that the tooth surface will not be contaminated with saliva and will be kept moisture-free after the etch gel is washed away. Four-handed dentistry might then be a necessity. Etching and light-curing time differ from one product to the other, and it is therefore important to read the 'Directions for Use' of the product for finding the right instructions. When using light-cured resin sealant material, the dentist has to realise that the material can shrink up to 4% and that unpolymerised sealant,



**Fig. 7.1** ART sealant step by step on a newly erupted tooth with a pit and fissure system in a caries-active child that required a sealant protection using a high-viscosity glass-ionomer (Ketac Molar Easymix, 3M ESPE, Seefeld, Germany). (a) Remove debris from the pits and fissures with a sharp probe and wash surface with a wet cotton wool pellet. (b) Condition the occlusal surface and pits and fissures with a cotton wool pellet, dipped in 20% polyacrylic acid for 10 s. (c) Wash the occlusal surface and pits and fissures with a wet cotton wool pellet and dry the occlusal surface and pits and fissures with a dry cotton wool pellet (do not use air spray) (d) Press the glass-ionomer mixture into the pits and fissures with the index finger removing the finger after 10–15 s, check the bite and remove excess glass-ionomer material with hand instruments. Apply a layer of petroleum jelly over the ART sealant and ask the patient not to eat for at least 1 h (Courtesy of Prof. Dr. E. Eden, İzmir, Turkey)

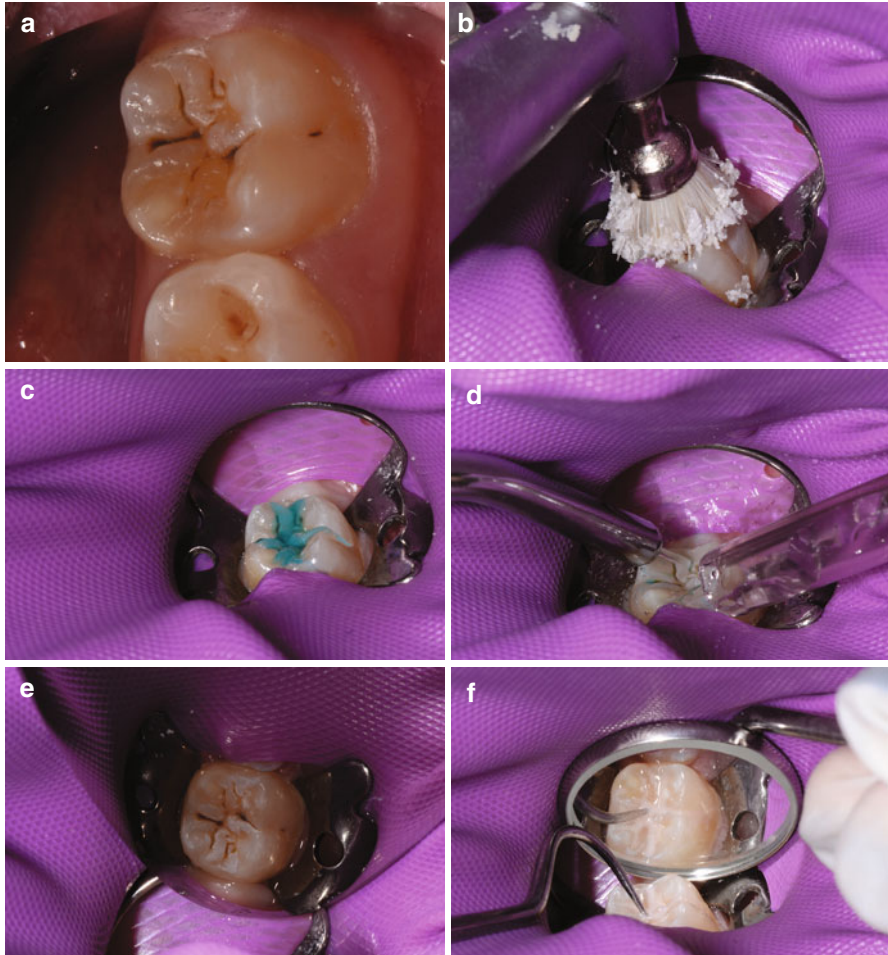
containing Bisphenol A (BPA) and/or BPA-(dimethacrylate DMA), is left at the surface layer. This layer can easily be removed when adjusting the bite or through whipping the surface with a cotton pledge. The placement of a resin composite sealant step by step is presented in Fig. 7.2.

## 7.10 Cavitated Dentine Carious Lesion Prevention with Glass-Ionomer- and Resin-Based Sealants

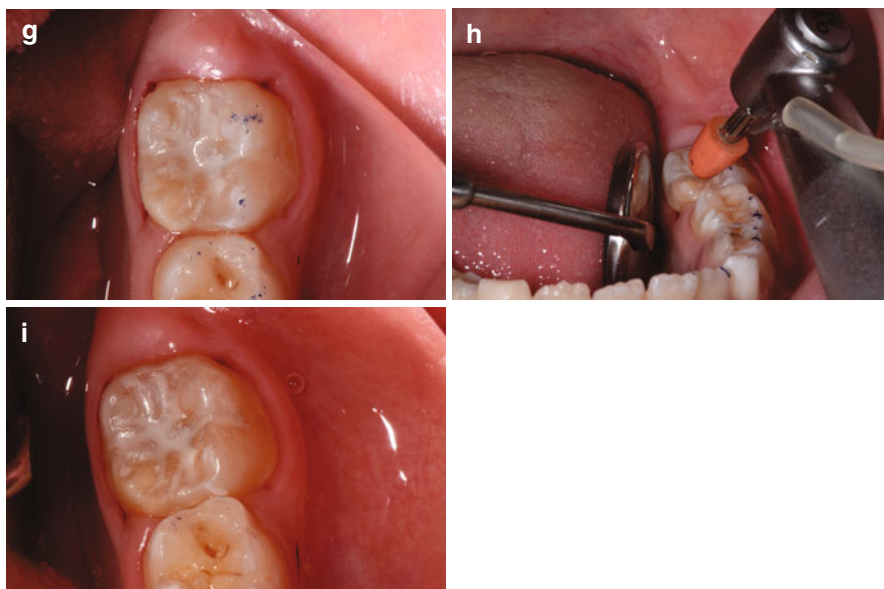
Already in the mid-1990s, Simonsen [24] concluded his critical review by stating that evidence concerning the cavitated dentine carious lesion-preventive effect of low- and medium-viscosity glass-ionomer- and resin-based sealants was equivocal. This conclusion matched the outcome of a systematic review [8] that included twice as many publications as that of Simonsen [24]. Two published systematic reviews and meta-analyses, which had included high-viscosity glass-ionomer ART sealants, showed the same conclusion as the two previously mentioned reviews: there is no

evidence that one sealant material has a higher cavitated dentine carious lesion-preventive effect than the other [14, 15].

As most long-term comparisons between the effectiveness of glass-ionomer-based materials and resin composite sealants over the last decade have used high-viscosity glass-ionomers applied according to the ART concept (using finger pressure), it is of interest to analyse the outcomes of these studies. An attempt to do this is presented in Table 7.2. A total of five studies could be retrieved. These studies ranged from 2 to 5 years in length; were carried out between 2006 and 2015 in



**Fig. 7.2** Resin composite sealant (Grandio Seal, VOCO, Cuxhaven, Germany) step-by-step (a) needs assessment, (b) cleaning pits and fissures with pumice and spray with water, and (c) etch surface with phosphoric acid for 30 s. (d) Rinse and dry pits and fissures with water air spray, (e) check etched surfaces, and (f) apply sealant material and run probe through the material to eliminate air bubbles and light cure the material for 20 s. (g) Adjust bite, and (h) remove top layer with a polishing bur. (i) Final check, patient be instructed to maintain good plaque and diet control (Courtesy of Prof. Dr. Ece Eden, İzmir, Turkey)



**Fig. 7.2** (continued)

**Table 7.2** Comparison of ART/HVGIC and resin composite sealants' effectiveness in preventing cavitated dentine carious lesion development in occlusal surfaces by years of study

Author	Year	ART/HVGIC		Resin composite		
		<i>N</i>	%	<i>N</i>	%	<i>P</i> value
Beiruti et al. [16]	5	139	94.1	115	78.8	0.003
Barja-Fidalgo et al. [50]	5	21	87.0	28	80.0	0.27
Zhang et al. [26]	4	239	97.3	297	96.4	0.31
Liu et al. [27]	2	179	92.7	178	96.1	0.17
Hilgert et al. [25]	3	69	90.2	169	91.4	0.59

*N* number of occlusal sealants, *ART/HVGIC* atraumatic restorative treatment/high-viscosity glass-ionomer cement

Brazil, China and Syria; and used the hand-mixed high-viscosity glass-ionomers Fuji IX (GC, Tokyo, Japan), Ketac Molar (3M ESPE, Seefeld, Germany) and Ketac Molar Easymix (3M ESPE, Seefeld, Germany) and the light-cured resin sealants Clinpro (3M ESPE, Seefeld, Germany), Delton (3M, St Pauls, USA), Fluoroshield (Dentsply, York, USA) and Helioseal (Ivoclar, Schaan, Liechtenstein).

Of the five studies, one showed a significantly higher survival rate for cavitated dentine carious lesion-free occlusal surfaces for ART/HVGIC than for resin composite sealants [16], while no difference was obtained for the four remaining studies. Three studies used the same ART carious lesion assessment criteria [16, 25, 26], while four studies sealed only high-carries risk occlusal surfaces in the first permanent molars [16, 25–27]. Given, in addition to the methodological differences, the different brands



**Fig. 7.3** (a) ART sealant using Ketac Molar (3M ESPE, Seefeld, Germany) after 4 years (Courtesy of Dr. C.J. Holmgren, France). (b) Resin composite sealant in 46 using smartseal & locF (Detax, Ettlingen, Germany) in an 11-year-old after 3 years (Courtesy of Prof. Dr. Ece Eden, İzmir, Turkey)

of materials used and the different operators, it is remarkable that sealants produced through the ART procedure using high-viscosity glass-ionomers show a performance that is similar to or significantly higher than resin composite sealants, which were considered to be the reference sealant [28]. A clinical picture of a 4-year-old ART sealant and a 3-year-old resin composite sealant is presented in Fig. 7.3.

### 7.10.1 Possible Extra Reason for the Effectiveness of Glass-Ionomer Sealants

The findings of these systematic reviews and meta-analysis may be a surprise to many, particularly as resin-based sealant materials are retained longer than the glass-ionomer sealant materials and should, therefore, automatically have a high dentine carious lesion-preventive effect. More than two decades ago, Mejäre and Mjör [29] and Torppa-Saarinen and Seppä [30] discovered that in situations where the low-viscosity glass-ionomer sealant material used had completely disappeared clinically, remnants were observed in the deeper parts of the pits and fissures systems. They described the remnants as being most probably glass-ionomer. They further suggested that the relative absence of dentine carious lesions in relation to the high level of clinically completely disappeared glass-ionomer sealants might, among other reasons, be explained by the presence of those remnants. The remnants may allow the removal of plaque from a lower-than-normal depth of the pit and

fissure system and thus better control possible demineralisation. This possible explanation for the low level of dentine carious lesions observed in pits and fissures previously sealed with glass-ionomers received further support from a case study [31]. In this case study, four long-term (8 to 13-years-old) HVGIC/ART sealants were subjected to scanning electron microscopy (SEM) investigations. In all these sealed teeth, remnants were observed on places that had been clinically assessed as not containing glass-ionomer material. These remnants were assumed to be of glass-ionomer. Further proof for glass-ionomer remnants being left behind in the deeper sections of pits and fissures came from an *in vitro* study that showed remnants in all sampled teeth sealed with a glass-ionomer [32]. Further investigations into this subject are required.

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### 7.11 Can Sealants Be Used to Seal (Micro-)Cavities?

If the ‘seal is the deal’ (implying that if an enamel/dentine carious lesion can be sealed off from being constantly covered by biofilm that might become cariogenic, by sealing over the lesion with a sealant material), then the progression of the carious lesion will come to a stop [33]. For sealing non-cavitated enamel carious lesions, this practice has become acceptable [34], irrespective of whether the pits and fissures contain micro-organisms at the start [35]. But studies have been performed in which not only non-cavitated enamel/dentine carious lesions have been sealed over but, more surprisingly, also cavitated dentine carious lesions. This practice started already in the early 1980s when Mertz-Fairhurst et al. [36] sealed over micro-cavities with a resin-based sealant material, followed in the late 1990s by Frencken et al. [37], who sealed over micro-cavities in dentine, which the CPITN probe could not penetrate, with a medium-viscosity glass-ionomer. After 3 years, it was found that 12.7% of these micro-cavities had progressed. Current evidence seems to indicate that sealing over micro-cavities in pits and fissures of permanent molars avoids invasive treatment with hand or rotary instrumentation to a large extent. This conclusion makes the question of when to intervene invasively very relevant, particularly in the context of minimal intervention dentistry [2, 38]. It goes without saying that the practice of sealing over micro-cavities requires high-quality sealant materials and further research.

Some researchers go even further and seal frank cavities in dentine. The most well-known study is that of Mertz-Fairhurst et al. [39], in which a resin composite restorative material was placed in the opening of 85 cavities in permanent teeth and sealed over with a resin fissure material. After 10 years, it was found that one of the sealed composite resin restorations had failed because of dentine carious lesion development and 12 such restorations for mechanical reasons. The 10-year survival rate was no different from conventional amalgam restorations but was lower than that of adjusted amalgam restorations sealed over with a resin fissure material. This study used a restorative composite to seal cavity openings, but it is unclear what can



be expected when only a fissure sealant material that is mechanically weak is used for sealing the openings of cavities.

Such an investigation was performed in primary teeth over 18 months [40] and in permanent teeth over 2–3 years [41]. Compared to conventional restorative treatment, the survival rate of sealed over cavities with a resin fissure sealant material was significantly lower in both studies. The results of these studies show that not only should one attempt to stop carious progression in cavities non-invasively but that this practice is limited by the strength of the sealing material used. According to an *in vitro* study, sealing small cavitated dentine carious lesions in pits and fissures showed a higher level of micro-leakage and insufficient penetration of resin sealant material into the cavity, compared to the sealing of sound pits and fissures [42]. Evidence so far indicates that sealing over obvious dentine cavities with a (resin) fissure material is not indicated.

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## 7.12 Latest Development Regarding Sealant Materials

It is known that BPA derivatives are released from dental resins [43–45]. These substances have recently been linked to a number of biological disorders [46–48]. This has led the World Dental Federation (FDI) to issue a policy statement on BPA, discouraging its use in the manufacturing of dental materials and highlighting the importance and need for greater awareness of caries prevention, thereby reducing the need for dental restorative materials [49]. As the future of dental materials that contain BPAs is uncertain, the dental profession can assure the public that it is able to manage carious lesions through the use of sealants because of the availability of a non-BPA-containing (ART) high-viscosity glass-ionomer sealant that has a high level of effectiveness.

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## 7.13 Final Remarks

Fissure sealants should be placed in high-caries risk pits and fissures in high-caries risk children. Resin-based sealant materials appear to be retained for longer than glass-ionomer-based sealant materials. But systematic reviews have shown that the cavitated dentine carious lesion-preventive effect of glass-ionomer-based and of resin-based materials is comparable. In comparison to resin composite sealants, high-viscosity glass-ionomer ART sealants have a high ability to prevent cavitated dentine carious lesion development. Sealant materials are increasingly being tested for sealing micro-cavitated dentine carious lesions. Sealants are a reliable measure for maintaining pits and fissures that are free of cavitated dentine carious lesions, provided that skilled operators apply quality materials.

A recent published study questioned the need for placing sealants in high-caries risk occlusal surfaces of first permanent molars in high-caries risk 6- to 7-year olds. Compared to supervised toothbrushing with a fluoride-containing toothpaste during school days (200 days/year), the rate of cavitated dentine carious lesion-free molars

in children brushing (96.6%) was not significantly different from those of comparable pits and fissures sealed with a resin composite (91.4%) and HVGIC/ART (90.2%) sealants after 3 years [25]. Dental caries is a behavioural disease, and its prevention is behaviourally related!

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# Resin Infiltration: A Microinvasive Treatment for Carious and Hypomineralised Enamel Lesions

# 8

Leandro A. Hilgert and Soraya C. Leal

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## Abstract

The rationale for using resin infiltration is based on the success achieved in obliterating the diffusion pathways of demineralised enamel with a low-viscosity resin. After treating enamel surface with an acid, and rinsing and drying, this resin is infiltrated into the lesion's porous enamel, filling the voids and arresting carious lesion progression. This microinvasive technique is indicated for treating non-cavitated proximal lesions in both primary and permanent teeth. Also, resin infiltration may be used aesthetically to improve the appearance of white-spot lesions and other hypomineralised lesions in anterior teeth, since successfully infiltrated lesions clinically look similar to sound enamel. This chapter discusses the principles of the resin infiltration technique on the basis of the available scientific evidence and presents clinical applications of this treatment option.

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## 8.1 Introduction

Enamel carious lesions in their initial stages are characterised by a subsurface demineralisation that is notably porous under an apparently intact enamel surface that is in contact with the cariogenic biofilm [1]. Clinically, such lesions appear as white spots, since refraction indices of water (1.33) and air (1.0), which now occupy the spaces between the hydroxyapatite crystals, are inferior to sound enamel (1.62) [2–4]. This means that the demineralised enamel has a whitish appearance when

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wet, which is even more evident after air-drying. Early enamel lesions, independent of location, are not cavitated. A cavitation only occurs after a usually slow and long development of the subsurface lesion when the enamel surface loses support and collapses.

The progression of early enamel carious lesions may be halted by nonoperative protocols that change the ecological factors that led to the lesion development. Oral health-promotion measures and increased access to fluorides are the most common methods used to obtain an oral ecology that is compatible with demineralisation/remineralisation cycles shifted towards health [5]. Initial active white-spot lesions, which usually present a rough and dull surface, can become smooth, glossy and inactive as a result of remineralisation and daily abrasion. Some lesions during this process may incorporate pigments and become brownish. Other lesions, usually the very shallow ones, eventually may 'disappear'. However, many lesions still present a visible whitish appearance even after inactivation. This occurs because the subsurface enamel (lesion body) is not fully remineralised and porosities are still occupied by water or air with their low refractive indices. For this reason, despite a successful treatment of the disease, nonoperative measures may not always bring back the aesthetics of sound enamel, leaving 'scars' of previously existing carious lesions [6, 7].

The control of early carious lesion progression (inactivation) by nonoperative protocols relies strongly on the cooperation of the patient. This compliance with treatment is much more easily obtained for smooth surface lesions than for approximal ones. Approximal early carious lesions are more difficult to diagnose and, even when patients have access to fluorides and receive oral hygiene and dietary instructions, are difficult to control. Therefore, they may progress and become cavitated [8].

The resin infiltration technique relies on providing penetration of the porous demineralised enamel with a low-viscosity resin for caries management. An effective isolation of the operative field is mandatory for performing the resin infiltration. Under isolation, an acid is applied to the affected enamel with the aim of eroding the thin surface layer to provide access to the porous subsurface (lesion body). Then, after rinsing and drying (with air and alcohol), a low-viscosity resin (with a high penetration coefficient) is infiltrated by capillary action into the lesion, where the resin is polymerised [9–11].

Resin infiltration recovers part of the structural integrity of the lesion body and occludes its porosities with an acid-resistant material, blocking the diffusion pathways of the biofilm-derived acids and halting lesion progression. Additionally, in buccal surfaces, an aesthetic benefit is derived from the refractive index of the infiltrant, which is close to the enamel's refractive index. Consequently, when water and air are replaced by resin in the lesion body, the appearance of the infiltrated lesion becomes similar to sound enamel.

The aims of the present chapter are to discuss principles, indications and evidences of the effectiveness of resin infiltration in approximal and buccal surfaces and to present clinical cases that illustrate such microinvasive treatment.

## 8.2 Approximal Surface Carious Lesions and Resin Infiltration

The prevalence of approximal carious lesions is high, particularly in deciduous molars and permanent posterior teeth of young adults [12]. These approximal lesions when diagnosed early – before a cavitation occurs – may be treated with non-invasive measures that aim to enhance remineralisation and arrest the progression of the lesion, such as oral hygiene instructions, dietetic counselling and an individualised fluoride regime [11, 13, 14]. Unfortunately, it is known that some approximal carious lesions ultimately will progress, mainly the more advanced lesions and those in patients that do not adhere to the ‘behavioural’ recommendations of the non-invasive therapy [14]. In cases in which caries progression is observed to a cavitation level, an operatory intervention is required. Cavity preparation and restorative procedures in approximal surfaces are laborious procedures and, in some cases, difficulties in accessing the lesions lead to invasive preparations that sacrifice sound dental structure. When a primary carious lesion is treated by means of a restoration (particularly in young patients), it may begin a cycle of successive restorative procedures also known as ‘tooth death spiral’ [15]. This suggests that alternatives for arresting carious lesion progression that are more effective than using only traditional secondary prevention measures, and that could prevent the need for restoration, may fill a serious gap in the current philosophy of Minimal Intervention Dentistry [16].

In occlusal surfaces, fissure sealants have been shown to be effective in preventing the development and progression of carious lesions in high-caries-risk patients (see Chap. 7) [17, 18]. Sealing approximal surfaces using fissure-sealing material is a possible treatment that has been tested with positive results [19–21]. However, the protocol for sealing such surfaces usually requires at least two clinical sessions: a first one to install elastic bands that promote a slight separation between teeth and a second one, a few days later, in which the sealing procedure is performed using a composite resin sealant that is applied to the approximal surface, which covers the lesion.

In contrast, the resin infiltration technique can be performed in just one session, since previous tooth separation is rarely required. A plastic wedge is used to create a minimal interproximal space where a special applicator tip is inserted. The applicator tip allows the operator to control which of the approximal surfaces should receive the product. The first step of the resin infiltration protocol involves etching the lesion surface with a hydrochloric acid to erode the lesion surface layer (approximately 30–40  $\mu\text{m}$ ) [22] and to promote access to the porous lesion body. After rinsing and drying with alcohol and air, a very low-viscosity resin is applied. The resin then infiltrates the porous enamel by capillarity, after which the material is light cured as shown in Case Report 1 (see Sect. 8.2.1).

Several *in vitro* studies were carried out prior to the establishment of the resin infiltration protocol. Type of acid and conditioning time [22] and resin composition and duration of infiltrant application [23, 24] were some of the elements evaluated. These studies, in addition to *in situ* studies, were essential to attest the ability of

resin infiltration to arrest the progression of carious lesions [25, 26]. Moreover, *in vitro* studies have shown that infiltrated carious lesions present increased mechanical resistance when compared to those that have not been treated by resin infiltration [27, 28]. This increased resistance may result in less enamel breakdown and, consequently, a lower number of cavitations. An *ex vivo* study in which primary molars were infiltrated before exfoliation has shown that the pattern and depth of infiltration obtained in the laboratory are compatible with those achieved under clinical conditions [29].

Currently, only a few medium-term randomised clinical trials that evaluated the efficacy of resin infiltration in arresting caries in approximal surfaces are available [8, 14, 21, 30]. In approximal surfaces of primary teeth, using a split-mouth experimental design, fluoride varnish (control) and resin infiltration (test) were compared in considering lesion progression for 1 year. Clinically, 67% of the control group lesions progressed versus 31% of the test group. Radiographically, 62% of the control group lesions progressed, while only 23% of the infiltrated lesions showed signs of progression [8].

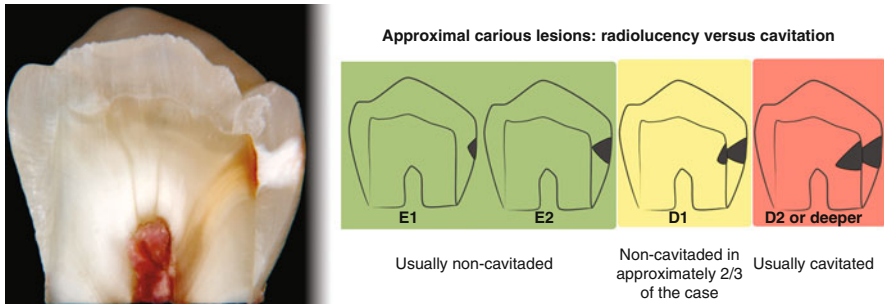
For the permanent dentition, a split-mouth experimental design was also used to evaluate approximal carious lesion progression. Resin infiltration was compared to nonoperative measures (instructions on oral hygiene, access to fluoride and dietary counselling). After a 3-year follow-up period, using digital subtraction radiography, 42% of the lesions treated with nonoperative measures only (instructions on flossing, tooth brushing and dietary advice) were found to have progressed, while only 4% of the infiltrated lesions showed signs of progression [30].

Another randomised clinical trial compared the effect of resin infiltration, approximal sealing and a placebo treatment on the control of carious lesions presenting radiolucency near to the dentin-enamel junction or already at the outer third of dentine. After 3 years, the efficacy (absence of lesion progression by digital subtraction radiography) of resin infiltration was found to be 68%, approximal sealing 60% and the placebo treatment 30% [21].

However, it is important to emphasise that cavitated lesions should not be infiltrated, since the infiltrant cannot adequately fill existing enamel cavities [31]. This makes a careful clinical and radiographic analysis essential for a correct diagnosis, which will lead to a correct indication of resin infiltration.

Some studies [32–34] demonstrate that when the radiolucency of an approximal lesion is confined to the outer half of enamel (E1 lesion), a cavitation is rarely observed. E2 lesions (inner half of enamel) are cavitated in approximately 8–11% of the cases and D1 lesions (outer third of dentine) in 22–44% of the cases. When the radiolucency extends to the half of the dentine depth (D2 lesion) or inner third of dentine (D3 lesion), a cavitation is usually present [32–34]. This means that resin infiltration is indicated for treating the E1, E2 and D1 lesions in which a cavitation is usually not present. Where doubt regarding the presence of a cavity exists, it is recommended that the teeth are separated using an elastic band in a way that the surface can be clinically assessed. A schematic illustration of caries progression in approximal surfaces observed in bitewing radiographs and the usual rate of cavitation occurrence is presented in Fig. 8.1.





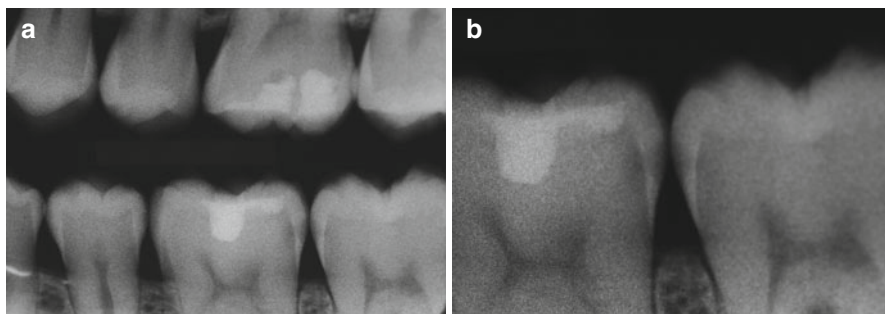
**Fig. 8.1** Relationship between stage of radiolucency of approximal lesions in bitewing radiographs and presence of cavitation

The success of the resin infiltration technique can only be observed by the absence of clinical or radiographic signs of carious lesion progression over time. However, the clinician should be aware that the resin infiltrant is radiolucent, which means that when the analysis is performed by comparing the baseline and the follow-up radiographs, the material cannot be detected visually. For this reason, it is important that the patient is informed about the procedure to which he or she was submitted. To facilitate this process, it is worthwhile for the clinician to provide the patient with written information about the tooth that was treated and to keep a good record about it. Otherwise, an uninformed dentist may execute an invasive treatment on a carious lesion that has been successfully arrested.

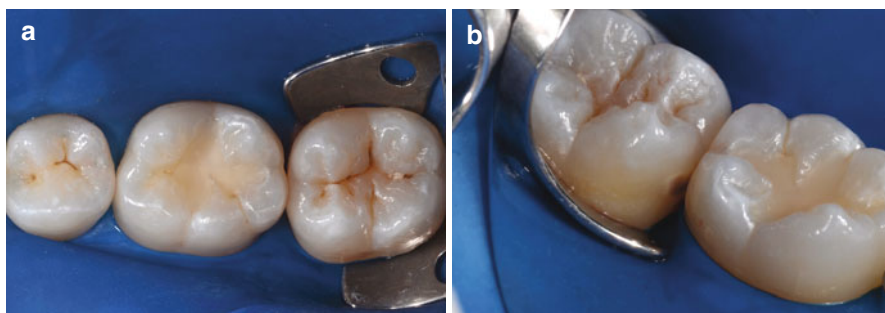
A 2014 study evaluated the cost-effectiveness of treatment options for approximal carious lesions [35], including non-invasive measures, resin infiltration (microinvasive) and invasive treatments (restorations) using settings of the German healthcare system as a basis. This study observed that, when compared to invasive therapy, both non- and microinvasive treatments decreased lifetime costs associated with the treatment of approximal posterior E2 or D1 lesions. Microinvasive treatment was shown to be more effective but was also more expensive than non-invasive measures. The greatest cost-effectiveness of resin infiltration was achieved when treating D1 lesions in permanent teeth of young patients.

### 8.2.1 Case Report 1

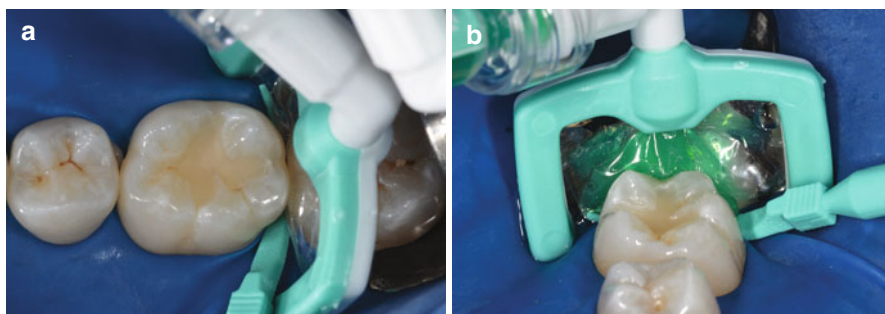
The first case report of the present chapter describes a young adult patient that presented D1 lesions on the distal surface of tooth 36 and mesial surface of tooth 37 (Fig. 8.2). Both lesions were judged to be non-cavitated under clinical examination. Resin infiltration was performed with the aim of arresting carious lesion progression and avoiding cavitation and the consequent need for invasive interventions. The clinical steps presented and described in Figs. 8.3, 8.4, 8.5 and 8.6 were performed in one session. Clinical and radiographical follow-up was performed after 18 months, and no sign of lesion progression was observed (Fig. 8.7).



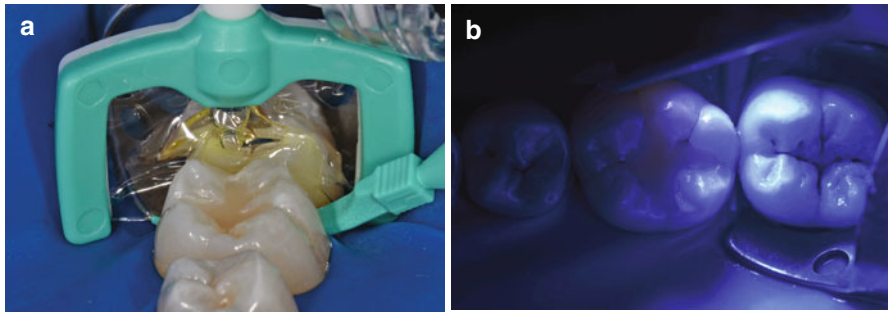
**Fig. 8.2** (a) Bitewing radiograph showing D1 lesions on the distal surface of tooth 36 and mesial surface of tooth 37. (b) Closer radiographic view of clinically non-cavitated lesions on 36 and 37



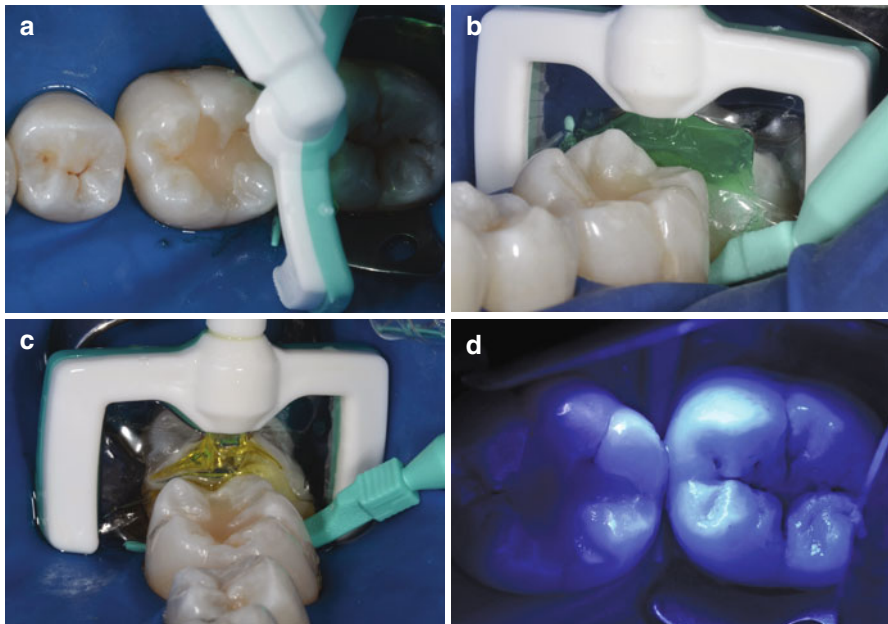
**Fig. 8.3** (a) The resin infiltration technique requires an adequate isolation of the operative field. In this case a rubber dam was used. (b) It is possible to see the lesion on 37. The absence of cavitation was observed by flossing and gentle probing. In some cases, the diagnosis can be confirmed by tooth separation and direct observation



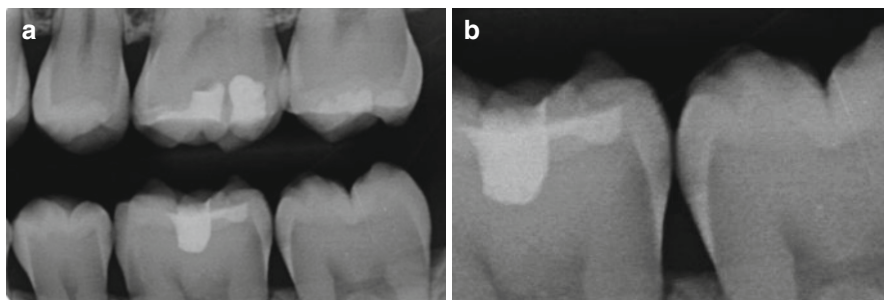
**Fig. 8.4** (a) Slight tooth separation was obtained using a plastic wedge and the tip of the applicator positioned in the interproximal space. The green side of the tip (a double foil perforated on the green side only) should be in contact with the surface to be infiltrated. (b) This figure shows the hydrochloric acid (Icon Etch, DMG, Hamburg, Germany) application (2 min), followed by rinsing, air-drying, alcohol (Icon Dry, DMG, Hamburg, Germany) (30 s) and air-drying



**Fig. 8.5** (a) After the etching and drying procedures, a new applicator tip is used to apply the low-viscosity resin (Icon Infiltrant, DMG, Hamburg, Germany). The resin is allowed to penetrate the lesion (for 3 min), excess is removed by flossing and the resin is light cured (40 s). (b) A new application of the infiltrant is performed (1 min), excess is removed and the area is light cured once more



**Fig. 8.6** (a) The mesial surface of tooth 37 was also infiltrated (observe the direction of the green side of applicator tip). (b) In this clinical case, for didactic reasons, the etching and drying were executed separately for the distal and mesial surfaces. In the daily clinic, to speed up the procedure, it is possible to etch and dry adjacent surfaces at once. (c) Infiltrant was applied and excess removed. (d) Light curing was performed



**Fig. 8.7** (a) 18-month follow-up. Clinically and radiographically there are no signs of lesion progression. (b) Closer view (compare this image to Fig. 8.2b)

### 8.3 Resin Infiltration as an Aesthetic Treatment on Buccal Surfaces

White-spot lesions in buccal surfaces of anterior teeth represent a relevant aesthetic problem for many patients. These whitish lesions, when caused by the caries process, are usually observed in adolescents and young adults and are frequently associated with the use of fixed orthodontic appliances [36].

Several treatment options are possible for white-spot lesions in buccal surfaces, such as remineralisation (induced by improved oral hygiene, fluorides and changes in dietary habits), microabrasion, restorations and, more recently, resin infiltration [2, 37]. The non-invasive remineralisation measures are, doubtless, the most indicated for arresting the carious process and inactivating the lesion. In buccal surfaces, the removal of the orthodontic brackets is usually enough to allow better hygiene standards and to arrest the lesion progression. In the case of shallow white-spot lesions, the visual aspect of sound enamel might be obtained by a combination of remineralisation and abrasion, owing to access to saliva and the effect of tooth brushing with fluoride toothpaste.

However, in deeper white-spot lesions, the remineralisation/abrasion process usually does not involve the entire carious lesion. As a consequence, the subsurface lesion body remains porous and filled with water or air that has a lower refractive index than sound enamel. In these cases, the carious lesion is clinically judged as inactive, since the enamel is now shiny and smooth, but the white spot is still visible and may remain visible for many years. Moreover, during the remineralisation process, pigments may be incorporated into the enamel and result in brownish-stained areas [7]. In both situations, it is accepted that the carious lesion is controlled, while the aesthetic appearance of the enamel may not be satisfactory. If so, different approaches might be proposed according to the severity of the lesion to the patient, as follows:

- Resin infiltration: as for the approximal lesions, the technique principle is based on filling the subsurface porous enamel of the lesion body with a resin infiltrant that has a refractive index at 1.52 that is closer to that of sound enamel (1.62)

than are the indices of water (1.33) and air (1.00). In this way, the colour difference between infiltrated white-spot lesions and sound enamel is reduced, and, in most cases, it becomes imperceptible or, at least, more aesthetically acceptable. It is considered a microinvasive procedure since it requires the erosion of the enamel surface layer (approximately 30–40  $\mu\text{m}$ ) [22] to create access to the lesion body.

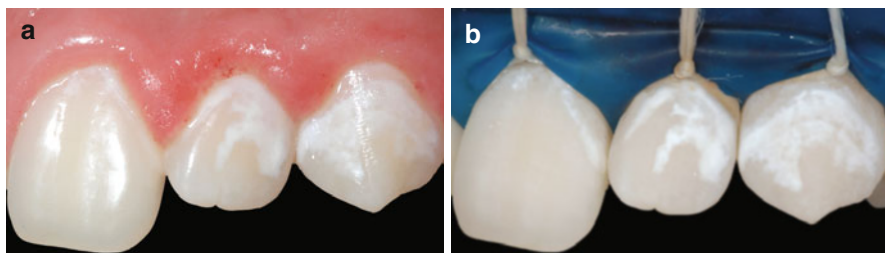
- **Microabrasion:** in this technique, an association of an acid with abrasive agents produces a chemical-mechanical wear of the enamel surface, removing the superficial (affected) enamel. The depth of enamel wear depends on the microabrasion product and on the operator (pressure of application and number of applications), but it is estimated to be on average around 200  $\mu\text{m}$  [38, 39]. Microabrasion is an effective treatment for lesions that are not much deeper than the depth of worn enamel.
- **Cavity preparation:** this can be defined as the removal of the affected enamel using rotary instruments and restoration with composite resins. This method, also called ‘macroabrasion’, is usually indicated only for those lesions in which less invasive therapies were not effective in recovering the aesthetics of the tooth.

The ability of resin infiltration to mask carious white-spot lesions has been evaluated *in vitro*, and the results suggest that it is an effective treatment [4]. A clinical study [2] tested the efficacy of resin infiltration in masking post-orthodontic carious white-spot lesions and in those whitish lesions caused by developmental defects (such as fluorosis and molar-incisor hypomineralisation). In developmental defects, a complete masking occurred in 25 % of the cases; partial masking was achieved in 35 %, and 40 % showed no relevant aesthetic improvement. In infiltrated post-orthodontic carious white-spot lesions, a complete masking was observed in 61 % of the cases, a partial masking in 33 % and in only 6 % was no improvement shown.

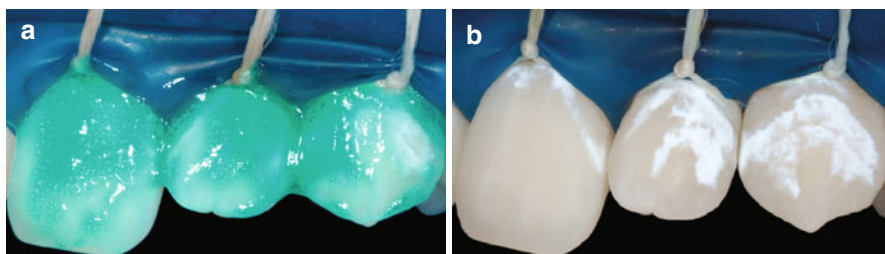
A clinical study [40] that was conducted in a university orthodontics clinic setting and that used a split-mouth experimental design compared resin infiltration (test) and non-invasive measures (control) regarding colour difference between the treated carious white-spot lesions and the adjacent sound enamel. After the 6-month follow-up, resin infiltration showed significant superior performance in reducing the colour difference and, in this way, improving aesthetic appearance. The patients were followed-up for an additional 6 months and, 1 year after treatment, the infiltrated lesions were evaluated as colour stable, with no adverse events or side effects reported [41]. Also, several case reports have been published that highlight the efficacy of the resin infiltration technique in providing aesthetic treatment of post-orthodontic carious white-spot lesions and developmental defects such as fluorosis-affected and hypoplastic enamel [3, 42].

### 8.3.1 Case Report 2

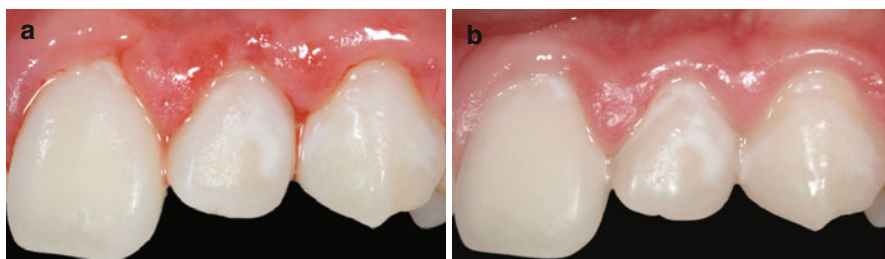
This second case report shows the effect of the use of resin infiltration on carious white-spot lesions in buccal surfaces of anterior teeth in an adolescent patient.



**Fig. 8.8** (a) Carious white-spot lesions on the buccal surfaces of anterior teeth. (b) A rubber dam isolation was used to protect soft tissues and retract gingiva



**Fig. 8.9** (a) The hydrochloric acid was applied for 2 min and rinsed off and the tooth surface air-dried. (b) After alcohol application and air-drying, it was possible to observe the characteristic chalky appearance of a white-spot lesion that had been etched and dried



**Fig. 8.10** (a) After infiltrant application and light curing, the surface was polished and the rubber dam removed. The photograph shows the clinical aspect immediately after resin infiltration. (b) After 10 months

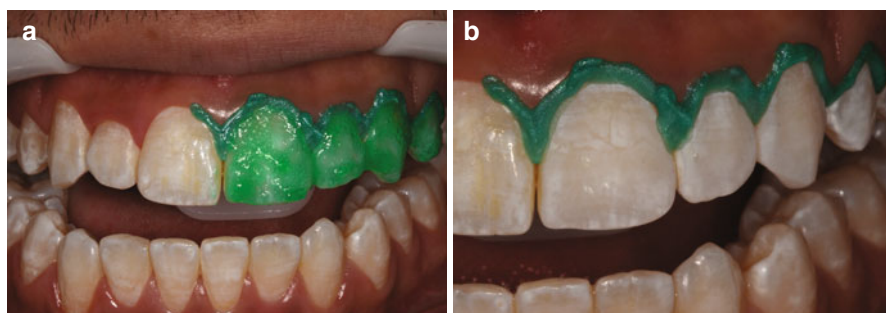
Figures 8.8, 8.9 and 8.10 present the pre- and post-infiltration images, up to a 10-month follow-up.

### 8.3.2 Case Report 3

Case Report 3 presents the case of a young adult who required aesthetic treatment of a mild to moderate fluorosis. Resin infiltration was performed with the aim of



**Fig. 8.11** Initial situation showing a mild to moderate fluorosis for which the patient required an aesthetic treatment. (a) Frontal view. (b) Right-side view

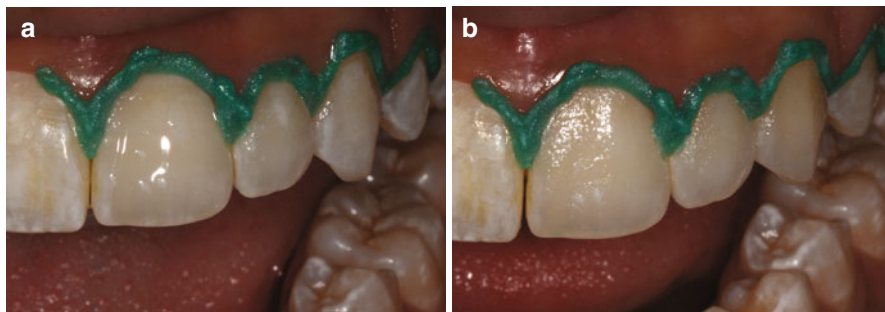


**Fig. 8.12** (a) Acid etching was performed for 2 min. (b) The acid was rinsed off and the tooth surface air-dried

reducing the whitish appearance of the fluorotic enamel and modifying the visual aspect of the buccal surface towards a more harmonic and uniform colour. Figure 8.11 shows the preoperative state; Figs. 8.12, 8.13, 8.14, 8.15 and 8.16 the clinical steps of the resin infiltration (the authors decided first to perform the left side and later the right side for the didactic purpose of allowing a visual comparison of the immediate results); and Figs. 8.17 and 8.18 the immediate and 18-month follow-up results of the treatment.

### 8.3.3 Case Report 4

The fourth and last case report of this chapter shows a 7-year-old patient presenting molar-incisor hypomineralisation (MIH). The patient and his mother requested aesthetic treatment for tooth 21, which presented a significant white-yellow opacity (Figs. 8.19 and 8.20). Resin infiltration alone was not effective in this case (Fig. 8.21), and a cavity preparation was required to create space for a restoration using composites (Fig. 8.22). After removal of the superficial enamel, to avoid deeper preparation, resin infiltration was used to reinforce and to create colour



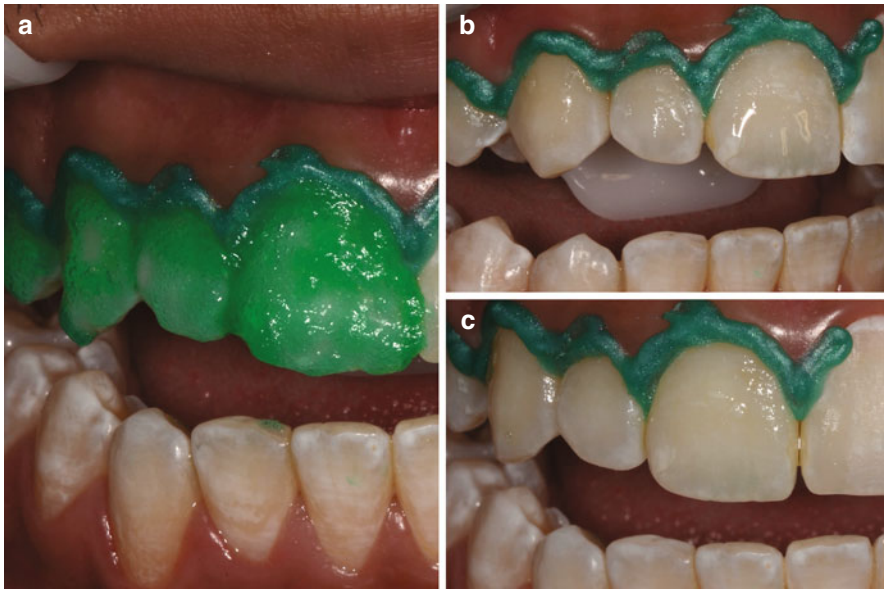
**Fig. 8.13** (a) Alcohol was applied and, before its evaporation, it was possible to see that it had penetrated the lesion pores and improved appearance. This ‘test’ is interesting for previewing the results of infiltration. If after acid etching, during alcohol application, the visual aspect is not improved, repetition of acid etching is recommended, to allow better access to the porous enamel. (b) This photograph shows the infiltrated enamel, just after the infiltrant was light cured

**Fig. 8.14** This image shows the immediate result after resin infiltration of the buccal surfaces of teeth 21, 22, 23 and 24. A polishing procedure with abrasive disks and/or rubber cups is still required

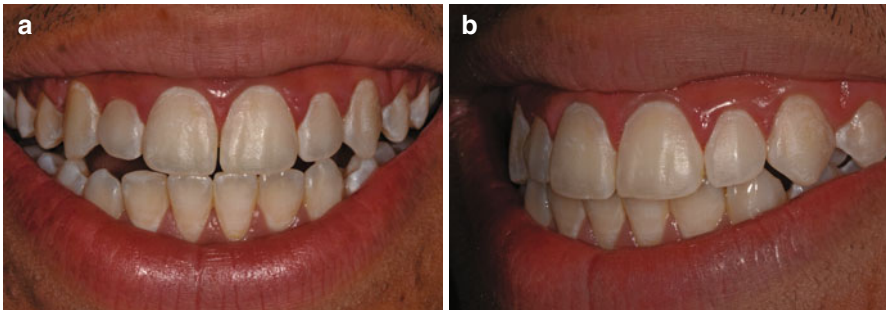


**Fig. 8.15** The immediate result after resin infiltration of teeth 21–24 from angles that allow visual comparison with the nontreated homologous teeth. Once again, the decision to perform one side before the other was didactic. In a normal clinical routine, all teeth could be treated at once to increase speed and productivity. (a) Frontal view. (b) Left-side view





**Fig. 8.16** The resin infiltration protocol was performed on teeth 11, 12, 13 and 14. (a) Acid etching. (b) Drying. (c) Resin infiltration

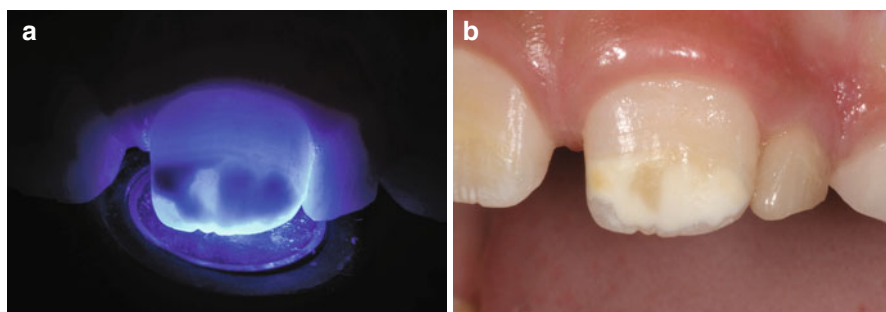


**Fig. 8.17** Immediate result of resin infiltration in mild to moderate fluorosis with a satisfactory aesthetic improvement. (a) Frontal view. (b) Left-side view

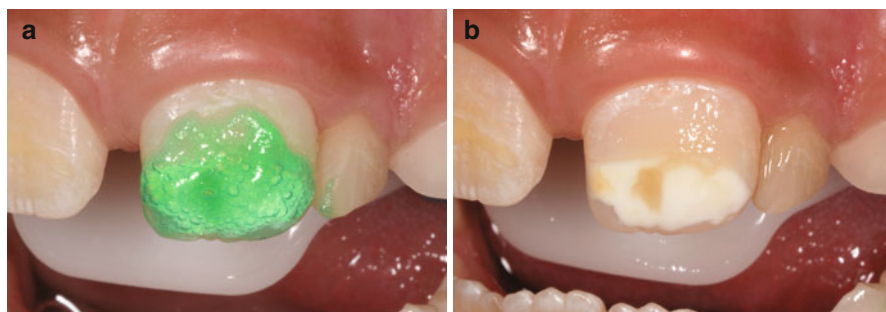
**Fig. 8.18** 18-month follow-up of the case, showing very good stability of treatment



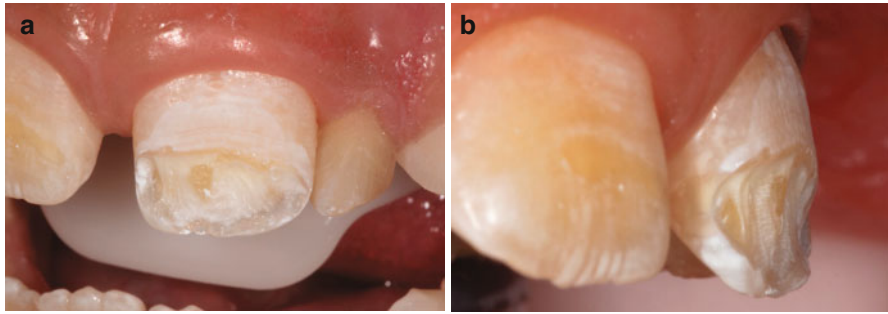
**Fig. 8.19** Initial situation, showing an MIH-affected buccal surface on tooth 21, which caused aesthetic concern for the patient and his mother



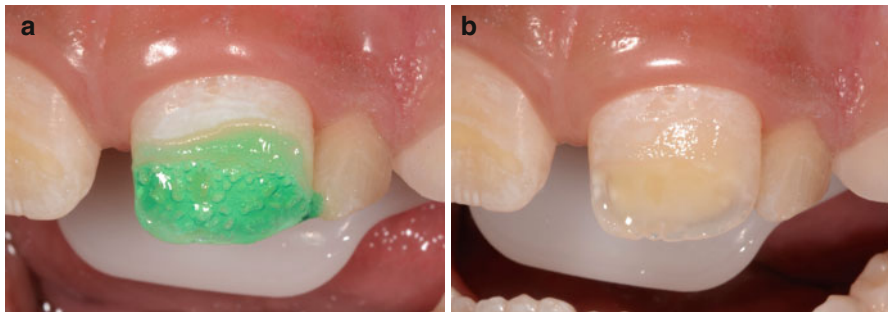
**Fig. 8.20** (a) Transillumination may help to determine depth of the lesion where examination suggests that it is not very shallow, since light transmittance is significantly reduced. (b) Clinical close up



**Fig. 8.21** (a) The resin infiltration technique was tested. (b) After acid etching, during alcohol application, no visual change was observed. This response suggests that the procedure will not succeed, requiring a more invasive treatment



**Fig. 8.22** (a) The surface of the affected enamel was removed with rotary instruments to create space for a composite restoration. (b) Approximately 0.5 mm of enamel was worn, and the substrate still showed a whitish appearance. However, the operator wished to avoid exposing dentine



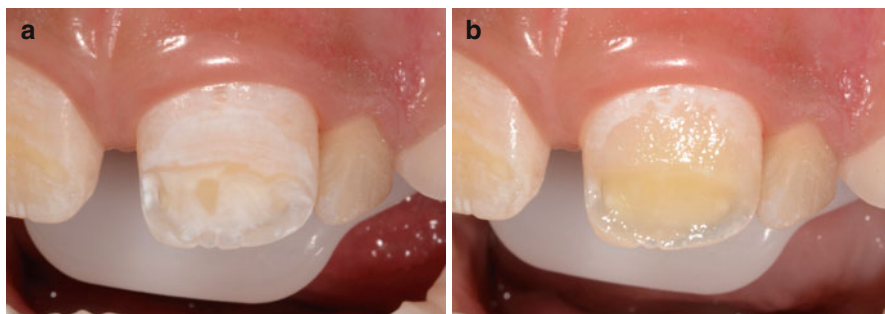
**Fig. 8.23** (a) Resin infiltration was performed on the worn enamel. (b) After acid etching, during alcohol application, a very good visual improvement was observed, suggesting a good prognosis for resin infiltration

homogeneity of the remaining enamel (Figs. 8.23, 8.24 and 8.25). A composite resin restoration was executed on the resin-infiltrated preparation (Fig. 8.26). Post-operative images show the satisfactory aesthetic outcome that was achieved (Figs. 8.27 and 8.28).

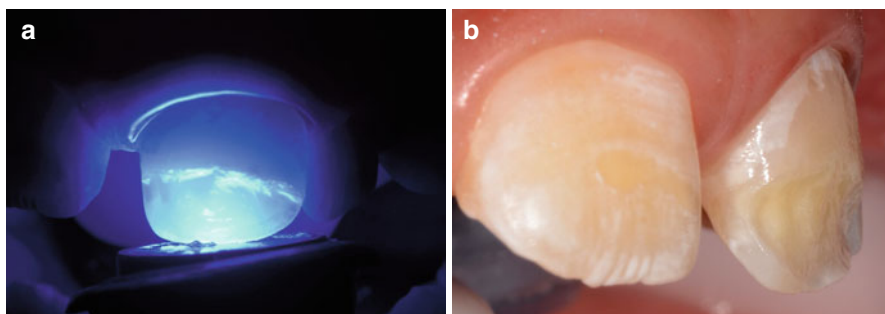
## 8.4 Final Remarks

The resin infiltration technique has been thoroughly studied in the last years. Results are promising and suggest efficacy in arresting approximal carious lesions and in improving the visual appearance of white-spot lesions in buccal surfaces.

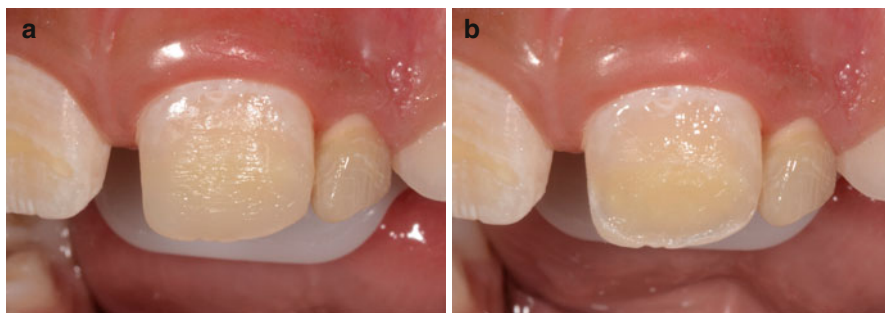
In approximal lesions, the technique is indicated only in non-cavitated cases. Enamel cavities cannot be successfully ‘restored’ using the infiltrant. This implies that a careful clinical and radiographic examination is required so that a correct diagnosis can be established.



**Fig. 8.24** (a) After thorough drying, (b) the resin infiltrant was applied

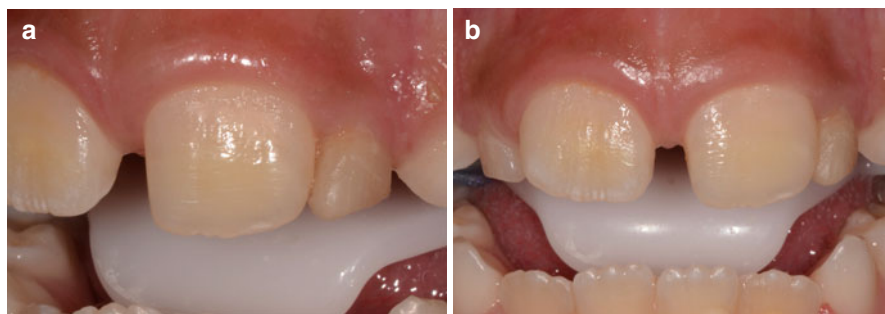


**Fig. 8.25** (a) The infiltrant was light cured. (b) The substrate, now with a homogenous colour, was ready to receive the restorative composite resin layers



**Fig. 8.26** (a) Body and (b) enamel shades of composite resins (Filtek Supreme Ultra, 3 M ESPE, USA) were used to restore the tooth

The infiltrated approximal lesions remain radiolucent after treatment. The absence of radiopacity is considered a limitation of the technique, since a uniformed dentist (judging only the radiography) could perform an invasive treatment on an infiltrated and arrested lesion. On the other hand, when the patient understands the benefits of a microinvasive treatment that aims to arrest a lesion at a stage in which it does not require a restoration and that treatment success will be measured by



**Fig. 8.27** After a week, the patient returned and final pictures, with rehydrated teeth, were taken. (a) Close view. (b) Colour harmony was obtained

**Fig. 8.28** Despite being invasive, cavity preparation associated to resin infiltration and composite resin restoration was effective in improving the aesthetics of the tooth to the patient's requirements



periodic follow-ups that show no lesion progression, the lack of radiopacity does not seem to be a real problem.

In post-orthodontic white-spot lesions on buccal surfaces, resin infiltration does not seem to be necessary for controlling the carious process and arresting lesion progression. This is because carious lesion inactivation is usually achieved when the orthodontic appliance is removed and oral hygiene is improved. However, the aesthetic improvement that follows resin infiltration in these cases is considerable. It may also be the least invasive among the effective procedures for enhancing smile attractiveness after a long-lasting orthodontic treatment that caused carious white-spot lesions. Since resin infiltration is easier in the more porous 'active' carious lesions, it should be performed as soon as possible after brackets are debonded.

Resin infiltration is a preventive and therapeutic technique that fills a serious gap between non-invasive and invasive treatments of approximal carious lesions and white-spot lesions on buccal surfaces. It is an important addition to the efforts of the current generation of dentists, who aim to control caries disease and promote aesthetics at the least possible biological cost.

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# Caries Prevention Through Life Course Approach

# 9

Francisco Ramos-Gomez and Cor van Loveren

## Abstract

Recent epidemiological analyses showed that caries risk continues throughout all age groups. Moreover, it seems that there are trajectories of oral health. Individuals seem to enter such a trajectory at an early age and it shows to be difficult to escape to another trajectory with better health perspectives. Therefore it is important to start caries prevention even before birth in order to ensure that children start in a favorable trajectory. Also health depends on social, environmental, and economic determinants which are conceptually summarized in the life course theory. For oral health, this implies that primary health-care providers integrate oral health into their routine examinations with oral health screenings, preventive education, and prophylactic fluoride applications. The main message for each age group is the twice daily toothbrushing with a fluoridated toothpaste individually supplemented with other oral hygiene techniques. If the oral hygiene technique fails, improvement can be achieved through composing self-care management goals. This method stimulates patients to formulate self-management goals, action and coping planning, and control. Patient's adherence to his own goals is strengthened by the oral care provider who alerts the patient (and parents, caregivers, or voluntary aids) continuously about their own decisive

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influence on the control of caries in his own mouth. This requires tailored recall intervals for monitoring, motivation, and stimulation. The self-management goals are the take-home message towards creating a stronger and healthier dental atmosphere at home and are a vital facet to preventing caries for all age groups.

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## 9.1 Introduction

Traditionally caries-preventive dentistry focuses on children and adolescents. Reasons for this relate to the organization of the dental care but also to an obsolete paradigm that all risk sites developed caries before adulthood and that sites that developed no caries by that time were non-risk sites thereafter. In adulthood, oral health was believed mainly to be threatened by periodontal disease. This paradigm is not valid today. Following a cohort from the age of 5 till the age of 38, Broadbent et al. [1] found that the population was divided in three subpopulations in a society where in principal all preventive treatments are available to all. Approximately 40% of the population did not develop caries significantly, approximately 45% developed a moderate amount of caries, and approximately 15% of the population developed high numbers of caries lesions. In all three groups, caries incidence was constant over the time span and did not differ between the period where caries prevention was covered by insurance or when the people were charged for it. Already at a young age, it was clear to which subpopulation a person belonged and hardly anyone passed from one to another subpopulation (see also Chap. 1). It is imperative that these and other observations are taken seriously by the dental profession and that strategies are employed to deliver preventive care tailored to the needs but also to the resources of patients. A more profound conclusion on oral health care was formulated by Fejerskov et al. [2]: “the whole ethos and philosophy of dentistry are too focused on a downstream, patient-centered, curative and rehabilitative approach to oral diseases that grants a primary role to the fully trained dentist for population’s oral disease control and prevention. A system focused primarily on treatment of disease will never be effective in controlling chronic diseases.” The FDI has concluded that current approaches to deal with oral diseases are “not economically sustainable nor socially desirable or ethically responsible” and asserts that “the time is now right for developing a new model for oral health care, which considers oral health as an integral part of general health and addresses the needs and demands of the public and the right of each individual to good oral health” [3].

These comments of Fejerskov et al. [2] and FDI [3] may also have repercussions to preventive measures that require professional performance. In spite of being effective in clinical trials, preventive measures may not be effective in populations as they do not reduce caries risk or do not specifically increase the awareness of being responsible for your own dental health nor increase the (believe in) self-efficacy of patients. In this, the studies of Hausen et al. [4, 5] are very illustrative. An intensive program consisting of all known measures added to a basic program (Table 9.1) was not effective in high-risk children [4]. The same program showed to be effective (prevented fraction 44.3%) when the components of the intensive

**Table 9.1** The preventive programs used in the study of Hausen et al. [4]

Basic program (BP)	Intensive program (IP)
Duraphat 1x/year	Basic program
Sealants in deep fissures	+
Principles of good oral hygiene and diet were mentioned	Additional Duraphat 1x/y
F-toothpaste 2x/day	All fissures sealed
No after-brush rinse	Intensive oral hygiene and dietary counseling.
	Xylitol gum after meal
	Dental floss 3x/week
	CHX-gel 2x/y when indicated
	F-lozenges 4x/day

preventive program were individually aimed at identifying and eliminating factors that had led to the presence of active caries. The program included counseling sessions with emphasis on enhancing use of the children's own resources in everyday life [5]. These studies show that telling the patients what to do is not sufficient. Instead, health-care providers should coach patients and parents about the factors that lead to and protect against dental disease and assist them in selecting self-management goals to improve their own and their children's oral health [6]. Another example making clear that a techno-medical solution may not be sufficient to prevent caries development is a 3-year longitudinal study that analyzed the caries-preventive effect of sealants in adolescents in the setting of the German national health system [7]. This study showed that sealants on occlusal surfaces of first permanent molars were only protective in individuals with low or moderate caries activity. Adolescents with high baseline DMFS values had an even higher risk of caries increment with an increasing number of sealants compared to adolescents with fewer sealants. This indicated the need for other measures to reduce caries activity in high-risk adolescents [7].

Both the studies of Hausen et al. [4, 5] and the German study of Heyduck et al. [7] also evoke the statements by Fejerskov et al. [2] and the FDI [3] and raise issues of economical sustainability, social desirability, and ethical responsibility. Stacking professionally performed preventive measures will raise the costs of prevention. Before advising so, the cost-effectiveness should be clear. Assuming that the first choice of preventive treatment is the most evidence-based one, the added measure will always have a lower degree of evidence. Therefore, the first question to answer is why a certain program with a high level of evidence is not working: is the proposed measure not powerful enough or are compliance and self-care management too low? Subsequent action should be directed by the answer to these questions.

Another suggestion from the quotes of Fejerskov et al. [2] and FDI [3] is that dentistry should involve more in upstream prevention. For upstream prevention, dentistry has to leave the office and get involved in or even generate sociopolitical initiatives to improve health. Examples are ensuring the availability of effective and affordable fluoride toothpastes or participation in healthy settings, e.g., healthy city or healthy school initiatives. A setting for health is a place or social context in which people engage in daily activities in which environmental, organizational, and personal factors interact to affect health and well-being [8]. Upstream strategies for

effective prevention often adopt a shared common risk factor approach [9]. Oral health is also determined by diet, hygiene, smoking, alcohol use, stress, and trauma. As these causes are common to a number of other chronic diseases, adopting a collaborative approach is rational. A very elegant example of a healthy setting program with a common risk approach (sanitation) is the “Fit for School” program in the Philippines ([10]; <https://www.youtube.com/watch?v=0yXn6RCwN2A#t=36>).

The cornerstone of the program is the use of school structures for the implementation of preventive health strategies. “Fit for School” consists of simple, evidence-based interventions like hand washing with soap, tooth brushing with fluoride toothpaste, and other high-impact interventions such as biannual deworming as a routine school activity for all children visiting public elementary schools. Started in the Philippines, the program has been successfully rolled-out in Indonesia, Laos, and Cambodia. Also in high- and middle-income countries, there is a need for dentistry to participate in healthy setting programs, while nobility obliges to take initiatives for healthy settings for children that do not yet and for elderly that do not visit the dental office anymore.

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## 9.2 Use of Life Course Theory (LCT) in Caries Prevention

The life course theory (LCT) is a conceptual framework in health that seeks to encompass patterns for health and disease amongst populations in the context of all possible influences [11]. It is deeply rooted in the social, environmental, and economic determinants of population health with a focus on early or upstream causes. Fine and Kotelchuck also incorporate the concept of health trajectories or pathways based on patterns, viewed as a life course, or an “integrated continuum of exposures, experiences, and interactions” [11]. LCT places particular emphasis on the prenatal period, early childhood, and young adulthood as critical periods: times of “early programming.” For instance, the overall health, environmental exposures, and experiences of the mother prior to conception or delivery can result in the disease or susceptibility of the child.

LCT is community (or “place”) focused, since social, economic, and environmental patterns are closely linked to community and neighborhood settings [11].

Public health is a logical home for LCT since the mission of public health includes improving and protecting the health of the population, eliminating health disparities and promoting health equity across population groups, and building healthy communities. Some branches of public health, including Maternal and Child Health (MCH), have been leaders in addressing social and environmental factors that affect health, a focus in keeping with LCT [11].

Four life course concepts are identified – timeline, timing, environment, and equity [11]:

1. Timeline: Today’s experiences and exposures influence tomorrow’s health.
2. Timing: Health trajectories are particularly affected during critical or sensitive periods.

3. Environment: The broader community environment – biologic, physical, and social – strongly affects the capacity to be healthy.
4. Equity: While genetic make-up offers both protective and risk factors for disease conditions, inequality in health reflects more than genetics and personal choices. Marked and persistent differences in health across populations and communities cannot be explained solely in terms of genetic make-up or individual choices but rather reflect the impact of broader societal and environmental conditions over time. LCT tells us disparities in the life circumstances of population groups within our society lead to disparities in health across these same groups.

For “Timeline,” LCT holds that health develops over a lifetime, with health improving or diminishing based in part on exposures to risk and protective factors. Planning should integrate health services and systems across life span and generations, to maximize protective factors. This includes a greater focus on health promotion from the youngest ages forward and on developing services that provide routine, early identification of health risks, and early intervention to minimize the impact of risks [11]. It also recognizes the need of elderly once the abilities to perform good self-care are diminishing.

Regarding “Timing,” LCT points to the importance of the earliest experiences and exposures and of critical periods throughout life, in shaping the health of individuals and populations. Thus, “strategic planning should incorporate a focus on assuring the availability of services and supports during critical or sensitive periods throughout the lifespan” [11] (see Chap. 10).

Regarding “Environment,” LCT recognizes that physical, social, and economic environments play an important role in shaping health and disease patterns across populations and communities. LCT suggests that planning should include strategies that link women, children, and families to other service systems that can address environmental factors such as employment services, housing, and family support programs. At the community and state level, planning should include a focus on promoting integrated, multi-sector service systems and assuring that those systems are easily accessed [11].

Regarding “Equity,” thus, LCT speaks to the importance of focusing on health equity from the perspective of population and place and tells us broad population-level and system-level changes are needed. This means going beyond tracking disparities, to identify and address root causes of disparities at the population level [11].

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### 9.3 Interprofessional Education and Practice

Many children do not visit the dental office regularly at a young age. They will more likely visit well baby clinics, nurse practitioners, physician assistants (PAs), or urgent care family doctors than they are to visit a dentist. Children may visit these facilities at least ten times within the first 3 years of their lives, if caregivers follow the medical periodicity schedule as recommended by many health authorities [12, 13]. The primary health-care providers can integrate oral health into their routine examinations

with oral health screenings, preventive education, and prophylactic fluoride applications. For these reasons, “front-line clinicians, PAs, often provide a first line defense in oral health for their patients” [14]. The FDI World Dental Federation’s General Assembly agrees that providers of children’s health-care services should all be trained in pediatric oral health care [15]. In a 2009 study by Lewis et al. [16], it was, however, determined that “only 50% of pediatricians receive oral health training during their residency and report a lack of training as a barrier to incorporating oral health in their practices.” In the past decade, the emphasis on interprofessional education (IPE) has been a focus for US and European universities. Students go through a comprehensive oral health curriculum that covers core oral health competencies. These core competencies include Part 1, wherein basics of oral anatomy and physiology, etiology of dental caries, basic caries identification methods, and prevention of dental caries are taught. Part 2 incorporates hands-on training, performing oral health exams and allowing future medical providers to become fully comfortable to “put their hands into a patient’s mouth”. Finally, Part 3 consists of IPE group case reflection and team-based decision making by the medical providers and/or health-care professionals themselves. It is the hope of IPE to reach a much larger patient population and help prevent dental caries, identify oral health problems early, and emphasize establishment of dental homes. Besides the USA and Europe, other countries can easily adapt this model in order to shed light on the importance of the interprofessional collaboration and provide a new learning environment for health-care professionals worldwide.

Another strategy is to deliver oral health services via nontraditional providers. Lay health-care workers, such as promotoras, farm health workers, community dental health coordinators, outreach workers, health services managers, and family, are being trained in dental health screening [17]. Similarly, nontraditional providers in other countries can adapt the same role, so that they too can provide dental health screenings. Workers conduct caries risk assessment and provide oral health-related anticipatory guidance and preventive health education. Since most of these workers belong to the communities in which they serve, they are particularly equipped to provide culturally competent care.

While dental hygienists, assistants, or other providers deliver (hands-on) care in underserved communities telehealth projects, such as the Virtual Dental Home Project, created by the Arthur A. Dugoni School of Dentistry at the University of the Pacific in California, they use technology to enable dentists to view patients’ dental records and documentation remotely [18]. The goal is to expand such programs to schools and nursing homes. Similarly, mobile dental programs can be used to serve the homeless, migrant workers, and individuals in inner city or rural communities, while the integration of oral health-care programs into school-based health centers provides a way to reach at-risk school-aged children [19].

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## 9.4 Behavior Management

Dental health professionals are mindful of the relationship between psychosocial determinants of health and their patient’s dental status but still tend to employ

approaches to health promotion and patient education that solely involve traditional knowledge transmission and advice-giving. Such an approach ignores the knowledge on motivational and volitional factors when it comes to adapt preventive behavior. These factors are described in the health action process approach (HAPA; [20]), an open framework of various motivational and volitional constructs that are assumed to explain and predict individual changes in health behaviors. HAPA suggests that the adoption, initiation, and maintenance of health behaviors should be conceived of as a structured process including a motivation phase and a volition phase. The former describes the intention formation, while the latter refers to planning and action (initiative, maintenance, recovery). The model emphasizes the particular role of perceived self-efficacy at different stages of health behavior change. In the transtheoretical model (TTM; [21]), six stages of behavior change are defined:

- Precontemplation (not being aware that a certain behavior leads to disease)
- Contemplation (being aware and weighing pros and cons of a behavioral change)
- Preparation (small changes are made, prone to relapse)
- Action (behavioral change is effectuated)
- Maintenance (action sustained for at least 6 months and working to prevent relapse)
- Termination (individuals have zero temptation and they are sure they will not return to their old unhealthy habit)

A technique for the actual interaction with the patients, the counseling conversations, is known as motivational interviewing. Motivational interviewing is nonjudgmental, nonconfrontational, and non-adversarial. In order for a therapist to be successful at motivational interviewing, four basic interaction skills should first be established: the ability to ask open-ended questions, the ability to provide affirmations, the capacity for reflective listening, and the ability to periodically provide summary statements to the client.

Two separate reviews by Gao et al. [22] and Cascaes et al. [23] examined a total of 26 randomized controlled trials in order to assess the effectiveness of motivational interviewing (MI) on oral health-related clinical and behavioral outcomes. The effectiveness of motivational interviewing was measured in comparison to giving conventional education (CE). The design and delivery of the motivational interviewing intervention differed across studies, ranging from one to seven MI sessions, lasting between 5 and 90 min, being delivered by different health-care professionals (with and without previous MI experience), administered on adults, adolescents, and parents with young children. Follow-up times, after the intervention was delivered, ranged from 1 month to 2 years. In terms of outcomes, a variety of target behaviors and oral health outcomes were assessed using a number of clinical and self-report measures. Reporting on studies investigating clinical and behavioral outcome measures, there was some evidence of positive MI effect for reducing dental caries in children through changing parental behavior [24]. This study appeared in both reviews and was rated as having good quality.

## 9.5 Preventive Programs

Prevention is traditionally divided in primary, secondary, and tertiary prevention. In primary prevention, the goal is to protect healthy people from developing a disease or experiencing an injury. Secondary prevention happens after an illness or serious risk factors have already been diagnosed. The goal is to halt or slow the progress of disease in its earliest stages. Tertiary prevention focuses on preventing further deterioration and maximizing quality of life. In cariology, these categories are translated in keeping a sound surface with no signs of risk sound (primary prevention), reducing risk factors (secondary prevention), preventing (non)cavitated lesions to progress (secondary prevention), reducing and treatment of cavitated lesions, and avoiding recurrence of the disease (tertiary prevention).

For all 3 types of prevention, fluoride toothpaste is the most significant preventive measure irrespective of the presence of water fluoridation. Cleaning of teeth and gums is a widely accepted cultural norm, which makes the use of fluoridated toothpaste as prime preventive measure an easy choice. The brushing technique should warrant that all reachable surfaces are thoroughly cleaned. For teeth in eruption, the cross toothbrushing technique is advised. The efficacy of fluoride toothpaste, however, depends on daily tooth-cleaning which requires a significant compliance. For young children, the job should be done by the parents, and at least till the age of 10, parental supervision is a prerequisite. The preventive strategy of the dental team should constantly emphasize compliance, which should not be ransomed by other preventive measures. Besides the use of fluoride toothpaste, the program for primary prevention also comprises adequate plaque removal and a sensible dietary advice. When conscientiously performed, this basic program may be sufficient to remain caries free.

There are specific circumstances that the program for primary prevention cannot be effectively exercised, e.g., by handicapped persons and elderly with reduced tooth-cleaning capabilities. Ways should be found to assist them. For handicapped and elderly, the oral hygiene procedure has to be supported by family or professional care.

When secondary and tertiary prevention is needed, the first question to be answered is why the program for primary prevention was not effective and advice should be given for correctly following the program for primary prevention. Additionally, a safety net might be given by additional daily fluoride exposures, twice-yearly professionally applied fluoride treatments, or fissure or approximal sealants (Chaps. 4 and 7). When cavitated lesions present, one might choose for a non-restorative therapy [25–27]. If chosen for this therapy, it is advised to treat the exposed dentine surface with fluoride varnish, silver diamine fluoride (SDF; [28]), or a lining material. When deciding to utilize the fluoride opportunities, one should also formulate when the treatments can be stopped. The ultimate goal should always be to inactivate caries where after the program for primary prevention would be sufficient.

If it turned out that there was caries activity while the program for primary prevention was correctly followed, other reasons for the caries activity should be diagnosed; one of these may be reduced salivary flow.

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## 9.6 Perinatal and Infant Oral Health

The oral disease status of the mother during pregnancy has been a possible contributor to negative birth outcomes such as gestational diabetes, preeclampsia, spontaneous preterm birth, low birth weight, or even fetal loss [29–32]. Life course theory challenges us to look further downstream and determine how the child's health and development trajectory, and perhaps increased susceptibility to oral diseases later in life, is affected by exposure to the negative conditions resulting from the mother's periodontal infection.

It is critical to determine ways to minimize the negative impact on maternal and fetal responses to maternal periodontitis or gingivitis [33]. Dental screening at the *first* prenatal visit is recommended, to increase the likelihood of early intervention [34]. Oral health roles for dentists, obstetricians, and allied health-care workers have been identified to underscore the need for counseling pregnant women as early as possible about their responsibility for their child's dental health [35–37]. Importance of good oral health of both mother and their children has to be consistently outlined including breastfeeding, oral hygiene, and dental care utilization practices.

Most Pediatric Dentistry Associations currently recommend that children receive their first dental visit within the first year of life. Moreover, the FDI World Dental Federation [15], which serves as the principal representative body for more than one million dentists worldwide, in over 130 countries worldwide, agree that health-care providers should engage in behavioral change in preventive dental care for the infant, beginning with the eruption of the first tooth. This allows for early dental intervention, to determine a risk status based on parental information. Anticipatory guidance for children's dental health is an important part of preventive care and may be the most effective way to prevent problems that traditional infectious-disease surgical models have failed to address, such as early childhood caries (ECC) [11]. Fluoride varnish efficacy in children provides additional rationale for an early dental visit, especially for children in high caries risk groups [29].

The model of anticipatory care guidance is valuable because its emphasis is preventive rather than restorative care. A comprehensive perinatal and infant oral care program utilizes (1) risk assessments at regularly scheduled dental visits, (2) counseling sessions with parents during regular dental visits or additional visits scheduled if child is at risk, (3) preventive treatment, and (4) outreach and incentives to reinforce attendance [30]. Facilitating access to early and regular dental care is a crucial part of any effective intervention strategy, and intervention techniques should be tailored to the community being served.

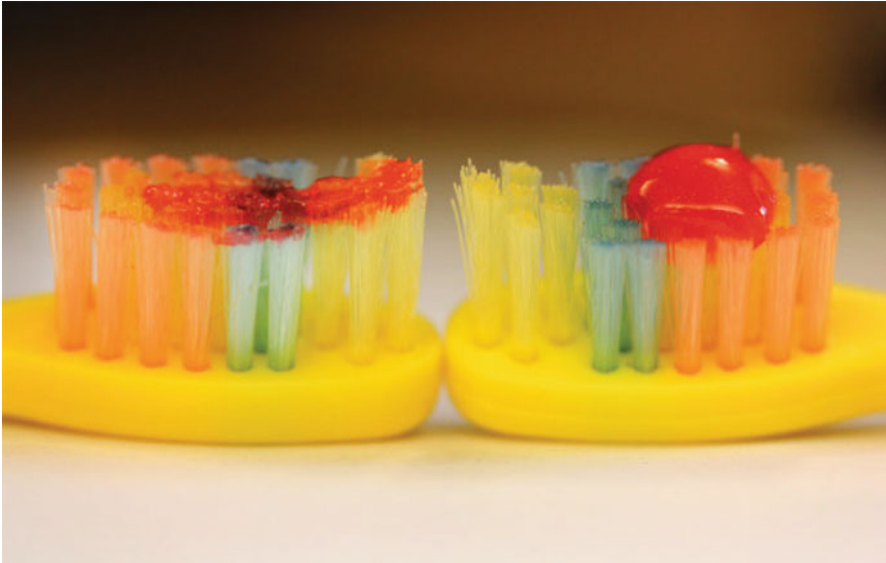


## 9.7 Infant Oral Health

Infancy is generally a time of relative health, with great parental investment in preserving that health. During the first six months, breastfeeding is the most appropriate form of food for the infant. Therefore, it must be highly encouraged. Further, infant mouths are free of cariogenic bacteria, but this bacteria will be transmitted from caregivers for instance by a high bacterial load pre-chew food, “clean” a pacifier in their mouth, or allow babies to stick their fingers in someone else’s mouth, which is especially important when the caregiver is at high caries risk and has high cariogenic bacteria. Wiping gums and then brushing teeth after eruption can set an infant onto a healthy trajectory. If it fails, early childhood caries (ECC) might occur. ECC is demineralization of the enamel with different degrees of cavitation in the primary dentition [38]. Predominantly, mutans streptococci and lactobacilli, which metabolize simple sugars to produce acid that demineralizes enamel, results in cavities in young children [39]. Despite its high prevalence, ECC is highly treatable and preventable, with early intervention or preventive care. The American Dental Association [40] and the American Academy of Pediatrics [41] both recommend that infant be seen by a dentist by age 1, or within 6 months of the eruption of the first tooth.

Traditionally dentists tend to make restorations when carious lesions are observed. If they do so, they ignore the difference between active and inactive lesions. They do not treat the disease but the appearance of the disease. An active carious lesion is one from which, over a specified period of time, there is net mineral loss, i.e., the lesion is progressing. Clinical observations to be taken into consideration for assessing carious lesion activity include visual appearance, tactile feeling, and potential for plaque accumulation (See Chap. 3). The best way to be sure of activity is to follow a lesion over time (photographs are useful). However, it is not recommended to “watch” things get worse. In cases of doubt, a chairside decision can be made based on the criteria mentioned in Chap. 3, plus the overall risk of the patient, so that if a lesion is likely active, a protocol can be started to help arrest it before it turns into a cavity.

The key to good oral health in children is to begin early with preventive care and risk assessment. If available, consumption of limited amounts of fluoridated drinking water during infancy can help lay the foundation for healthy teeth. Once teeth have erupted, brushing with fluoridated toothpaste twice per day helps prevent ECC. Worldwide, there are two strategies; either to use a smear of 1000 ppm fluoride toothpaste or to use 500 ppm pea-size fluoride toothpaste without this restriction (Fig. 9.1). For high caries risk, 500 ppm is not found effective so only 1000 ppm is suggested. At the child’s first dental visit, the oral health-care provider should complete a caries risk assessment such as Caries Management by Risk Assessment (Fig. 9.2) (CAMBRA; [42]) or any other described caries risk assessment model to determine risk level (see Chap. 3) and help the child’s caregiver set self-management goals. After detecting the causes of caries through biological predisposing risk factors, protective factors, and clinical disease indicators throughout the clinical examination of the child, the next crucial step involves self-management goals. Dentist works synergistically with the parent and/or caregiver through the use of encouragement and motivational interviewing, to select two to three realistic goals with the parents to adapt at home, before their next recare visit. Such goals include brushing with fluoride toothpaste at least two times a day, not drinking



**Fig. 9.1** If fluoridated toothpaste with 1000 ppm fluoride is used, it is advised to use a smear of toothpaste before the 3rd birthday and with a pea-size amount thereafter till the age of 6 with no specific restriction thereafter

soda, committing to regular dental visits for the child and the family, weaning the child off the bottle, drinking tap water, etc. The habit of familiarizing parents with self-management goals helps prevent childhood caries from developing, persisting, and worsening. The self-management goals are the caregiver's take-home message towards creating a stronger and healthier dental atmosphere at home and are a vital facet to preventing early childhood caries (Fig. 9.3).

There are several obstacles to obtaining early, preventive dental care for children. A large proportion of pediatricians do not incorporate oral health evaluations or counseling into their practices. Even when (pediatric) dentists are available, many do not treat children or do not choose for a preventive strategy, a fact that prevents many at-risk children from obtaining care. One of the biggest issues is the “drill and fill” nature of many dental practices, where surgical interventions are the norm, and preventive care is deemed financially impractical, as insurance reimbursement rates are much higher for repairs after oral disease has already taken hold than for early and regular case management aimed at preventing ECC.

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## 9.8 Mechanical Plaque Removal

### 9.8.1 Brushing

Mechanical plaque removal with a fluoridated toothpaste is one of the main components for caries prevention and control. For infants, it is recommended that the caregiver wipe the infant's gums with a clean damp cloth at least daily after feedings and

**CARIES RISK ASSESSMENT FORM FOR AGES 0 TO 5 YRS OLD**

Patient Name: \_\_\_\_\_ I.D.# \_\_\_\_\_ Age: \_\_\_\_\_

Date: \_\_\_\_\_ Assessment Date: \_\_\_\_\_

NOTE: Any one YES in Column 1 signifies likely "High Risk" and an indication for bacteria tests	YES = CIRCLE			Comments:
	1	2	3	
<b>1. Risk Factors (Biological Predisposing Factors)</b>				
(a) Mother/caregiver has active dental decay in past year	YES			
(b) Bottle with fluid other than water, plain milk and/or formula		YES		Type(s):
(c) Continual bottle use		YES		
(d) Child sleeps with a bottle, or nurses on demand		YES		
(e) Frequent (> 3 times/day) between-meal snacks of sugars/cooked starch/sugared beverages		YES		# times/day: Type(s):
(f) Saliva-Reducing factors are present, including: 1. medications (e.g., asthma [albuterol] or hyperactivity) 2. medical (cancer treatment) or genetic factors		YES		
(g) Child has Special Health Care Needs		YES		
(h) Parent and/or caregiver has low SES (Socio-economic status) and/or low health literacy, WIC/Early Head Start		YES		
<b>2. Protective Factors</b>				
(a) Child lives in a fluoridated community (note zip code)			YES	Zip Code:
(b) Takes fluoride supplements			YES	
(c) Child drinks fluoridated water (e.g., tap water)			YES	
(d) Teeth brushed with fluoride toothpaste (pea size) at least 2x daily			YES	# times/day:
(e) Fluoride varnish in last 6 months			YES	
(f) Mother/caregiver understands use of xylitol gum/lozenges			YES	
(g) Child is given xylitol (recommended wipes, spray, gel)			YES	
<b>3. Disease Indicators - Clinical Examination of Child</b>				
(a) Obvious white spots, decalcifications, or decay present on the child's teeth	YES			
(b) Existing restorations	YES			
(c) Plaque is obvious on the teeth and/or gums bleed easily		YES		
(d) Visually inadequate saliva flow		YES		
(e) New remineralization since last visit (List teeth):			YES	Teeth:
<b>Child's Overall Caries Risk (circle): HIGH MODERATE LOW</b>				
Child: Bacteria/Saliva Test Results: MS: LB: Flow Rate: ml/min: Date:				
Caregiver: Bacteria/Saliva Test Results: MS: LB: Flow Rate: ml/min: Date:				

**Self-management goals:**

- \_\_\_\_\_
- \_\_\_\_\_












**Since Last Visit:**

- New Cavitation:** Y / N
- New White Spot Lesions:** Y / N
- Dental Pain:** Y / N
- Referral Needed on:** \_\_\_\_\_

Fig. 9.2 CAMBRA – Caries Risk Assessment Form for age 0–5 years [42]

especially after nighttime feedings. This helps the infant get comfortable with someone working in his/her mouth. Once the teeth have erupted, usually around 6 months of age, it is recommend that the caregiver brush the infant’s teeth with a soft-bristled toothbrush with a small head twice a day (after breakfast and before bed) or wipe with a piece of gauze. While caregivers are brushing/cleaning the child’s teeth, caregivers should lift the lip and inspect the oral cavity for spots on teeth or unusual appearances of the gums.

Children may be able to start “brushing” their own teeth at around 18 months. Initially, the child may only play with the toothbrush. It is recommended that

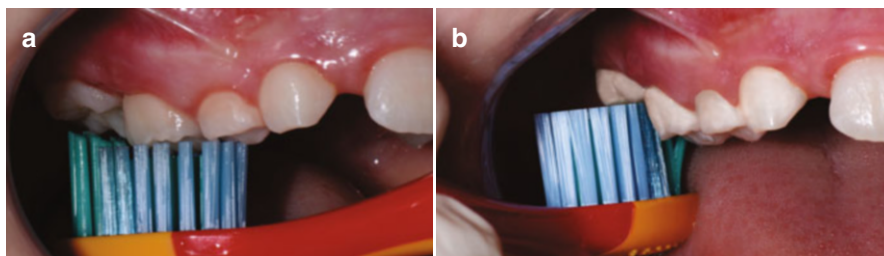
Patient Name _____			DOB _____
 <i>Regular dental visits for child</i>	 <i>Family receives dental treatment</i>	 <i>Healthy snacks</i>	 <i>Brush with fluoride toothpaste at least 2 times daily</i>
 <i>No soda</i>	 <i>Less or no juice</i>	 <i>Wean off bottle (no bottles for sleeping)</i>	 <i>Only water or milk in sippy cups</i>
 <i>Drink tap water</i>	 <i>Less or no junk food and candy</i>	 <i>Use xylitol spray, gel or dissolving tablets</i>	<p><i>Important: the last thing that touches your child's teeth before bedtime is the toothbrush with fluoride toothpaste.</i></p>
Self-management goals 1) _____ 2) _____			
On a scale of 1–10, how confident are you that you can accomplish the goals? 1 2 3 4 5 6 7 8 9 10			
Signature _____			Date _____
Practitioner signature _____			Date _____

**Fig. 9.3** Self-management goals for parent/caregiver

Caregivers make the toothbrushing experience a joyful experience by using songs and musical timers while brushing.

Caregivers must supervise the child’s brushing at least until age of 10. Children lack the fine motor control needed for adequate toothbrushing and therefore cannot clean their teeth without parental help. By age 7 or 8, the child acquires the appropriate manual dexterity to brush himself/herself, yet it is still recommended that caregivers provide supervision. For children with special health-care needs, it is strongly recommended that parents continue to brush as long as it seems necessary and obtain special oral health equipment (e.g., adapting a toothbrush or interdental cleaners) to brush the teeth.

During the eruption of the permanent molars, the occlusal surfaces may not be touched by the toothbrush when the brushing is in mesial-distal direction. Transversal



**Fig. 9.4** (a) With the normal alignment of the toothbrush, the occlusal surface of the newly erupting molars may be missed; (b) it is advised to place the brush transversal to the arch to brush the erupting permanent molars

(buccolingual) placement of the brush will ensure that the occlusal surfaces are properly cleaned (Fig. 9.4).

## 9.8.2 Electrical Toothbrushes

Manual or electric/powered toothbrushes are both viable options for plaque removal for children and adults. Both types can effectively remove plaque. At the minimum, the electric toothbrush bristles should be free of sharp or jagged edges and endpoints, the handle material should be manufacture tested for durability under normal use, the bristle should not fall out with normal use, and it is safe for use in children and adults.

A 2014 Cochrane review of electric versus manual toothbrushes concluded that there is moderate quality evidence that powered toothbrushes provide a statistically significant benefit compared with manual toothbrushes with regard to the reduction of plaque in both the short-term as well as long-term. A variety of different electric toothbrushes were included in this review (side to side, counter oscillation, rotation oscillation, circular, ultra-sonic, ionic, etc.) and it was concluded that the rotation oscillation brushes demonstrated a statistically significant reduction in plaque and gingivitis [43]. Caregivers need to make the personal choice if they want to use a manual toothbrush for their child or prefer an electric toothbrush. Especially for children, elderly, and the handicapped, who have issues of adequate dexterity, an electric toothbrush might be the better option.

## 9.8.3 Interdental Cleaning

Toothbrushing alone, either manually or electrically, may not clean the interproximal areas sufficiently. Many patients will develop gingivitis or periodontitis in these areas of which the prevalence is increasing with age. Then, additional interproximal cleaning is necessary. The techniques for interproximal cleaning are complicated and the patients or the caregivers have to be carefully instructed to be able to make the cleaning effective. Many studies do not show an additional benefit of interproximal cleaning which is generally believed to be caused by insufficient techniques.

These results undermine the value and credibility of interproximal cleaning and may give the patients a false sense of being protected. There is a myriad of interdental cleaners available such as interdental brushes and tips and plastic and wooden picks. Interdental cleaning products should be chosen according to comfort, cost, and ease of use. Often times, interdental cleaners with small handles are easier to use than manual flossing. Interdental cleaning is also advised to use frequently in areas of food impaction.

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## 9.9 Tailored Recall Intervals

To establish proper oral hygiene practices, experiments suggest that individual tailored recall intervals are very helpful. There are various models which all base the recall interval on an estimate of the caries risk of the patients [44–47]. One of the first successful models was developed in Nexø, a small community of 9000 inhabitants on the island of Bornholm in Denmark. The oral care providers implemented a special nonoperative caries treatment program (NOCTP) for children in 1987 [44]. The treatment program is based on three principles dosed at individually assessed recalls: (1) education of parents, children, and adolescents in understanding dental caries as a localized disease; (2) intensive training in home-based plaque control; and (3) early professional nonoperative intervention (2% NaF). Education of parents starts when the child is 8-month-old and called to the clinic for the first time. The parents are trained in home-based plaque control. The professional nonoperative treatment comprised plaque removal by means of toothbrush or rubber cup and dental floss and surface drying for visual examination for indications of caries progression. In case of progression further education and training in plaque removal and topical application of fluoride are indicated. For the mixed and permanent dentition, the caries diagnosis is supported by X-ray photography if required. During the eruption of the first and second molar, special emphasis is given to brushing the occlusal surfaces by placing the brush transversal. There is a simple scheme to set the time between the recall visits based on diagnosis and compliance (Table 9.2).

The program was successful [44, 48] and has successfully been copied in other settings such as the Odder Municipal Dental Service in Denmark [2], Moscow [49], and the Netherlands [50]. The interesting starting point of the program is to use as less resources as possible. This has resulted in only the use of the measures with the highest level of evidence. Vermaire et al. [50] started the program when the children were 6 years of age in order to prevent caries development in the first permanent molars. Although all experiments with the NOCTP program were carried in children, the principal can be applied at all ages of patients.

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## 9.10 Elderly

At later age, oral health after being stable for a long period of time may become at risk again. There are two major causes which either alone or in combination may be detrimental: a severe reduction of the salivary flow and reduction of

**Table 9.2** Overview of the system used in the Nexø Project to determine the individual recall interval [44]

Criteria		
Cooperation	Inadequate	2 points
	Good	1 point
Caries progression within the dentition	Yes	2 points
	No	1 point
Stage of eruption of permanent first/second molars	Partly erupted	2 points
	Full occlusion	1 point
Occlusal surfaces of permanent first/second molars	Caries progression	2 points
	Caries free or arrested lesions	1 point

dexterity or awareness to perform adequate oral hygiene which might result in dependence for care.

Many people become at risk for a reduction of salivary flow as beside being a physiological phenomenon it is related to the use of multiple medication, systemic diseases, and radiation therapy for cancer treatment in the area of the salivary glands. The dentist might be the first to diagnose reduced salivary flow when stable oral health suddenly decreases. Plaque may be present at areas which used to be properly cleaned. Caries may rapidly develop at sites that were caries free before or at sites that would normally not develop caries. The gums and mucosa may have a dry appearance. When seeing (older) patients, the dental professionals should be alert for these signs of reduced salivary flow. Meticulous oral hygiene is necessarily supported by daily additional topical fluoride applications. Exercises can be given to stimulate salivary flow or saliva stimulants can be described.

When dexterity diminishes the daily hygiene practices, the toothbrush can be adjusted to compensate any deficiency. It might be an option to start to brush electrically. The oral hygiene routine may be supplemented by using an oral rinse. When people are dependent, care should be delivered by voluntary or professional aids.

## 9.11 Final Remarks

Caries prevention is a lifelong commitment. Careful, diligent daily use of fluoride toothpaste (irrespective) and exposure to water fluoridation, the use of fluoride varnishes, and the use of pit and fissure and approximal sealants are evidence-based caries-preventive measures. Other technologies are far less evidenced based and would not logically yet fit in preventive protocols [51]. Dietary advice takes a special place (Chap. 6). It is clear and without dispute that the intake of sugars and fermentable carbohydrates is essential for caries to develop. Frequency of intake seems to be a more relevant determinant than the total amount. However there is virtually no data on which frequency of intake is permissible when the teeth are twice a day carefully brushed with fluoride toothpaste. Some advices permit a total of seven times a day but this number seems to be convenience-based and not evidence-based.

Ecological studies reveal that a large part of the population benefit sufficiently from the use fluoride toothpaste and water fluoridation while others do not. The reason for this is unanswered but it is argumentative that this results more from improper use of the products (improper compliance to the protocol) than from insufficient quality of them. It is also unanswered whether this problem is solved by adding products that will need proper use and compliance as well or by increasing the compliance to the original, more simple and more evidence-based protocol. To achieve increased compliance to the protocol, patients and dental professionals should work together to formulate self-management goals and plan a coping action to accomplish these goals at home. The key question then is whether increasing compliance to self-management goals is executable in the dental office. Is the dental professional willing and equipped to do so? The answer to this latter question is pivotal. If the dental professional is willing and equipped to effectively support and incentivize patients through self-management goals, then an individualized practical disease management strategy can be chosen. However, if the dental professional is unable to increase compliance to self-management goals in the dental office, then the traditional strategies need to be employed focusing on provider selected individualized preventive measures. The latter approach risks that the patient feels erroneously being protected neglecting his self-management. To increase compliance, patients should live in healthy environment and settings.

**Conflict of Interest** The authors have no conflict of interest to declare.

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Ece Eden and Hande Şar Sancaklı

## Abstract

This chapter will present sample cases from different life phases starting from pregnancy to senescence, as well as various complicated conditions such as systemic disorders or cancer treatment, where various regimes of prevention are suggested and discussed.

## 10.1 Introduction

It is accepted that the biofilm over the tooth surface always possesses the potential of developing dental caries. Preventing this disease is only a matter of maintaining the balance between prophylactic and pathologic components within the mouth. This is a paradigm shifting innovation from mere prevention to managing dental caries through prevention. Though, it is important to note that this new paradigm should be fused with traditional dental care.

There are several differences among countries in traditional dental care protocols on account of the content of the curriculum. Although many clinicians attend meetings or follow new literature, clinical practice is mostly based on knowledge and clinical experiences obtained through the years spent in education and profession, rather than new innovations in the science of dentistry. Additionally, factors such as

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social background and workplace of the dentist (e.g. private clinic, public health care centre or dental hospital), availability of products (in the clinic or in the dental market) and time devoted for each patient play important roles in the treatment modalities preferred. Focusing on prevention is difficult when patient load is high, time is scarce and there is no demand from the patient. However, quality of life and long lifespan are the new concerns of today's dentistry all around the world, and the contemporary trend is treating the patient and fulfilling the needs of the individual for proper oral health, rather than just eradicating the decay by drilling and filling.

The decision-making process for each patient is unique and should appraise scientific evidence and must be applied as much as possible in conjunction with the above-mentioned barriers. Therefore, the ability to determine the appropriate treatment for the patient in addition to planning a prevention regime to obtain life-long oral health needs to be the target of a genuine dentist.

Development of good oral health starts early in pregnancy and continues all through life. Every life phase has its own needs, deficiencies and expectations. This chapter will try to find answers to certain questions, illustrated with cases within the life stages. Strategies suggested should be considered as examples, and the reader should know after reading the book so far that there is not only one remedy or a magical tool to solve the problem.

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## **10.2 Phase 1: Pregnancy**

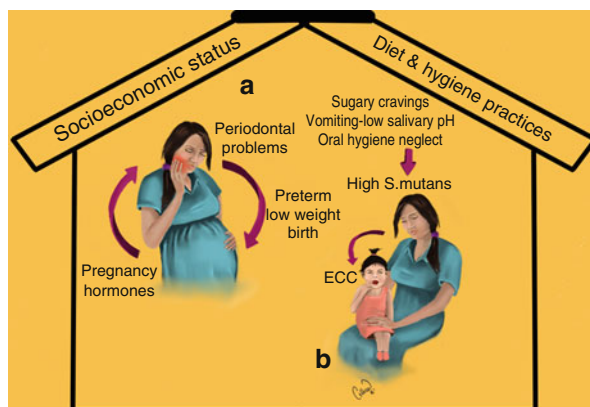
Pregnancy is a very important phase in a woman's life. From the health perspective, it is the only stage in human life where both the mother and the infant are affected at the same time. As such, we will look at this phase from two different perspectives.

### **10.2.1 Does Pregnancy Cause Tooth Loss?**

In many communities, there is a belief that the infant causes weakening of the expectant mother's teeth through calcium depletion in pregnancy. However, dental enamel does not have a circulating bloodstream from which calcium could be removed, and, as reported previously, dental caries is the disease of the oral biofilm on the tooth surface and not an elementary deficiency disease of the enamel. Thus, we need to find the reason behind this myth through scientific perspective and see what really happens in pregnancy.

Studies in dental literature have mostly focused on oral health in relation to hormonal changes during pregnancy and evaluated periodontal problems and dental caries. Several studies have strived to show the relationship between periodontal health in pregnancy and its effect on birth. Recent meta-analysis has revealed that periodontitis in pregnant women has an influence on adverse pregnancy outcomes such as preterm low birth weight [1], but the authors report

**Fig. 10.1** (a) Relationship among pregnancy and periodontal health and birth outcomes. (b) Sugary cravings, vomiting and oral hygiene neglect may cause dental caries in the mother, and vertical transmission of *Streptococcus mutans* from the mother and similar socio-economic status with diet and hygiene practices predispose ECC (Illustration by Gökçe Taniyan)



that such evidence needs further studies. Oxidative stress markers in relation to periodontal health in pregnancy defined the relation between preterm birth and periodontitis [2–6].

On the other hand, increased risk for dental caries during pregnancy is attributed to sugary in-between meal cravings, increased acidic saliva due to vomiting and neglect of oral hygiene practices [7–10]. Systematic review of intervention studies during pregnancy revealed the need for rigorously designed oral health guidelines for improving oral health of pregnant women [11].

Optimum oral care during pregnancy will improve quality of life of the expectant mother, provide a proper gestation period and lead the newborn to grow to the optimum healthy birth weight and further sustainable oral health. Intraoral evaluation of the mother, providing treatment before pregnancy, teaching proper oral hygiene and diet counselling will help having healthy oral structures throughout gestation without losing any teeth (Fig. 10.1a).

### 10.2.2 Can an Expectant Mother Ensure to Have Healthy Dentition in Her Baby?

The health of the mother is closely related with the health of the child, which also includes oral health. Recent meta-analysis and systematic review revealed that there is a transmission of *Streptococcus mutans* from the mother to the child [12]. Early colonization by vertical transmission from the mother should be avoided with preventive strategies in pregnancy to diminish dental caries risk in children [13, 14]. Therefore, it is important to educate pregnant women about oral hygiene and diet wherever possible (see Chap. 9). This knowledge and practice will change the environmental factors predisposing early childhood caries (ECC) for the newborn and help maintain good oral health for mothers and their babies (Fig. 10.1b).

**Fig. 10.2** Healthy dentition at the age of 3



An expectant mother with low socio-economic status attending a programme on caries prevention received intraoral examination and oral health education and was provided with a toothbrush and a paste containing 1450 ppm fluoride. The most outstanding behaviour of the expectant mothers attending the programme was avoidance of oral care and fear for having dental examination.

The education programme included a one-to-one tooth brushing session and small group education about diet, periodontal health and vertical transmission. Saliva-sharing activities such as using the same cup or other utensils were defined clearly.

She did not have any caries, so no treatment was needed. She was not brushing her teeth. She improved her oral hygiene with the help of the provided toothbrush and paste. First dental examination of the baby was carried out when he was 1 year old and continued biannually. Figure 10.2 shows healthy dentition at the age of 3.

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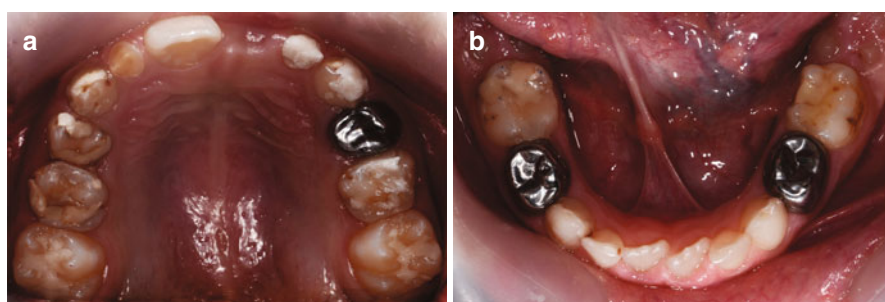
## 10.3 Phase 2: Childhood

Childhood is the age span from birth to adolescence lasting around 12–13 years. This is the transition state of dentition where primary teeth erupt, exfoliate and are replaced with permanent teeth. Here are two questions to answer.

### 10.3.1 How Can We Prevent Early Childhood Caries?

Early childhood caries (ECC) is defined as the presence of one or more caries (d), missing (m) or filled (f) tooth surface in a child of 71 months old or younger (Fig. 10.3). Any sign of caries before age 3 and dmfs score more than the number obtained by the age of the child plus one is called severe early childhood caries (S-ECC) [15]. Avoidance of early vertical *Streptococcus mutans* colonization from the caregiver and removal of plaque after feeding and disuse of nursing bottle during sleep after eruption of primary teeth will help to prevent early childhood caries. A smear layer of toothpaste containing 1000 ppm fluoride may be used for high caries risk children before age 3. Casein phosphopeptide and amorphous calcium

**Fig. 10.3** Early childhood caries (ECC)



**Fig. 10.4** (a) Maxillary and (b) mandibular intraoral view of S-ECC at the age of 7 who was treated since he was 4

phosphate (CPP-ACP)-containing pastes (Tooth Mousse, GC Dental, Leuven, Belgium) can be used safely without the risk of fluorosis (see Chap. 4).

### 10.3.2 How Can We Treat Severe Early Childhood Caries?

Figure 10.4 shows a 7-year-old boy who has been treated for S-ECC since he was 4. No new carious lesions developed and no tooth was extracted. Glass ionomer restorations (Ketac Molar Easymix, 3M ESPE, Seefeld, Germany) and stainless steel crowns (3M ESPE, USA) are preferred for the restorative treatment. The patient was seen at 3-month intervals and chlorhexidine varnish (Cervitec Plus, Ivoclar Vivadent, Schaan, Liechtenstein) in combination with fluoride varnish (Duraphat, Colgate, New York, USA) was applied in the first year. Fluoride varnish application continued, and the maxillary first permanent molars were sealed with glass ionomer ART sealants (Ketac Molar Easymix, 3M ESPE, Seefeld, Germany) soon as they erupt. CPP-ACP-containing paste before going to bed was prescribed. He is brushing his teeth twice a day with 1450 ppm fluoridated toothpaste and has been receiving diet counselling together with his mother (see Chap. 9).

## 10.4 Phase 3: Adolescence

Adolescence is the transitional period from puberty to adulthood in the teen years where physical and sexual maturation is observed. The child needs to develop identity and gain skills for social and economic independence, as well as responsibility for his/her well-being and health. Several oral health issues arise during this period. With changes in hormones in adolescence and as a result of oral hygiene neglect, periodontal problems may be elevated. Adolescents may develop eating disorders such as anorexia nervosa or bulimia, which would cause an acidic oral environment. Erupting permanent dentition increases total number of caries susceptible tooth surfaces, and usually orthodontic treatment is carried out during adolescence for malocclusions. The adolescent's newly developing social independence affects his/her diet and hygiene practices, and fixed orthodontic appliances contribute to dental caries risk [16].

### 10.4.1 How to Deal with Enamel Carious Lesions in Orthodontic Patients?

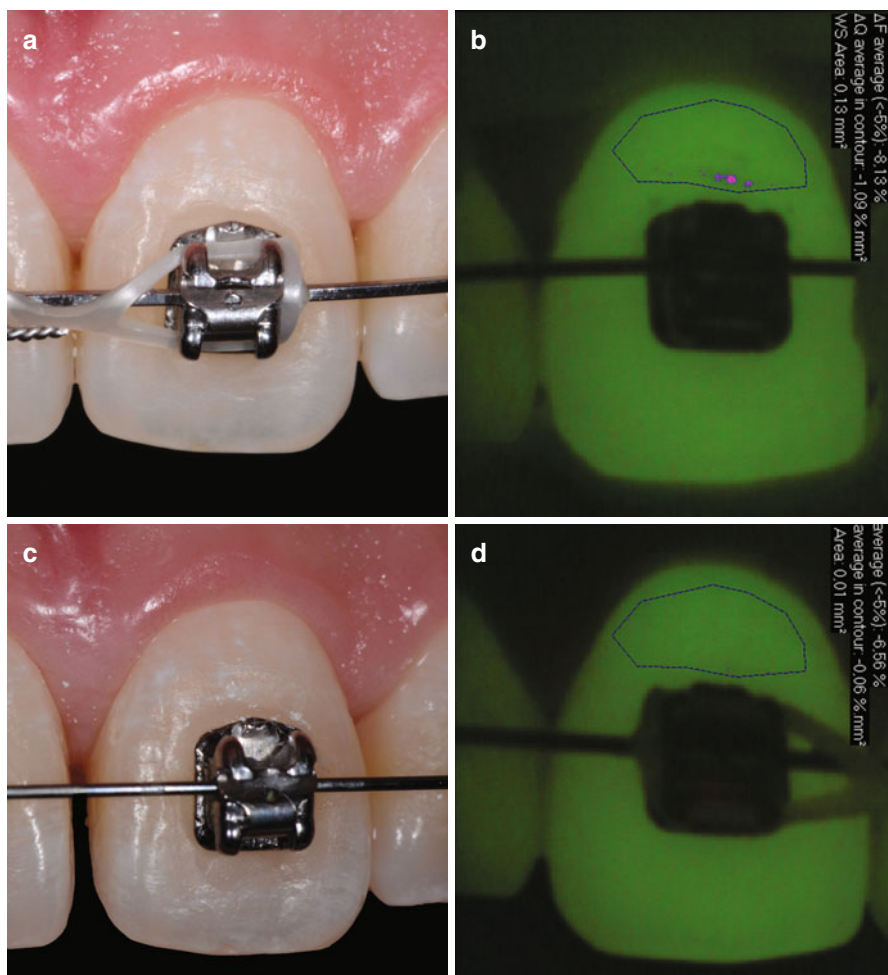
Orthodontic patients, usually during their adolescence, do not like to take responsibility for their health and usually resist authority. Intraorally, they have a high cariogenic bacteria load in their saliva and in dental plaque around fixed appliances. The high load bacteria accumulated plaque most probably causes demineralization – enamel carious lesions also called white spot lesions around brackets close to gingival margin [17, 18]. Therefore, it is important to provide oral health education for these teenagers including diet counselling and one-to-one tooth brushing practices in a manner that can be accepted and applied by them regularly.

The relationship between behaviour and consequences should be addressed. They need interdental brushes, dental floss and single-tufted brushes for proper plaque removal in combination with a manual or electric powered brush. If preferred, water-pick or flushes may also be used. 1450 ppm fluoridated toothpaste must be used twice a day. Mouth rinses containing antimicrobials may be prescribed from time to time in order to control cariogenic bacteria (see Chap. 5).

Remineralization agents (see Chap. 4) such as fluoride varnish (Fig. 10.5) or self-assembling peptide P11-4 (Fig. 10.6) may be used to treat the enamel carious lesions and avoid further destruction. For deeper lesions and better aesthetics, resin infiltration may be used (see Chap. 8).

Orthodontic patients in their adolescence may also develop carious lesions in the posterior regions of their dentition. It is advantageous to use resin infiltration for early proximal carious lesions for young patients instead of drilling and further destroying proximal contact that can lead to consecutive postoperative problems. Proximal carious lesions of a 15-year-old male patient were treated with resin infiltration (Fig. 10.7). He was also asked to use interdental brushes with a gel containing chlorhexidine and fluoride (TePe Gingival Gel; Malmö, Sweden) every night and brushed twice daily with 1450 ppm fluoride toothpaste. Three-month interval follow-ups for 2 years revealed no requirement for further restorations (Fig. 10.8).

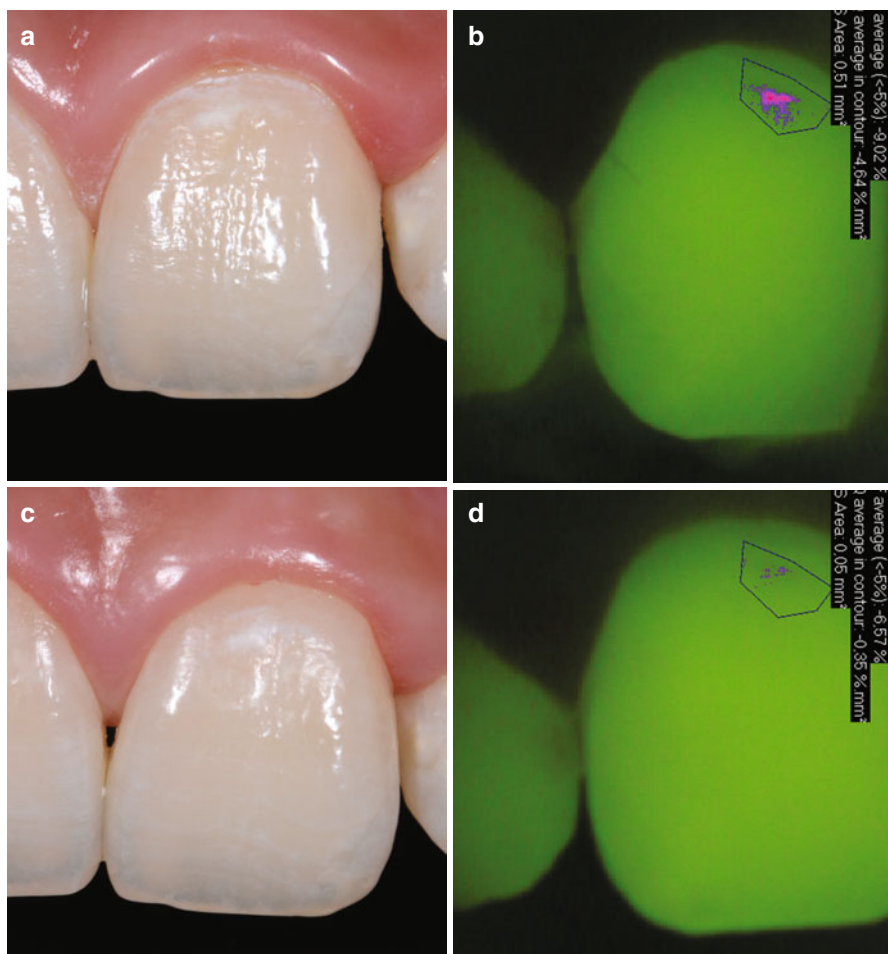




**Fig. 10.5** Remineralization of enamel carious lesion on 21 by fluoride varnish (Clinpro White Varnish, 3M ESPE, St. Paul, USA) in an adolescent with fixed orthodontic appliances. (a) Baseline photo. (b) QLF (Inspektor™ Pro, Inspektor Dental Care BV, Netherlands) measurement at baseline ( $\Delta F = -8.13$ ). (c) Intraoral view 6 months later. (d) QLF measurement 6 months later ( $\Delta F = -6.56$ ) (Courtesy of Dr. Ecem Ergin, İzmir, Turkey)

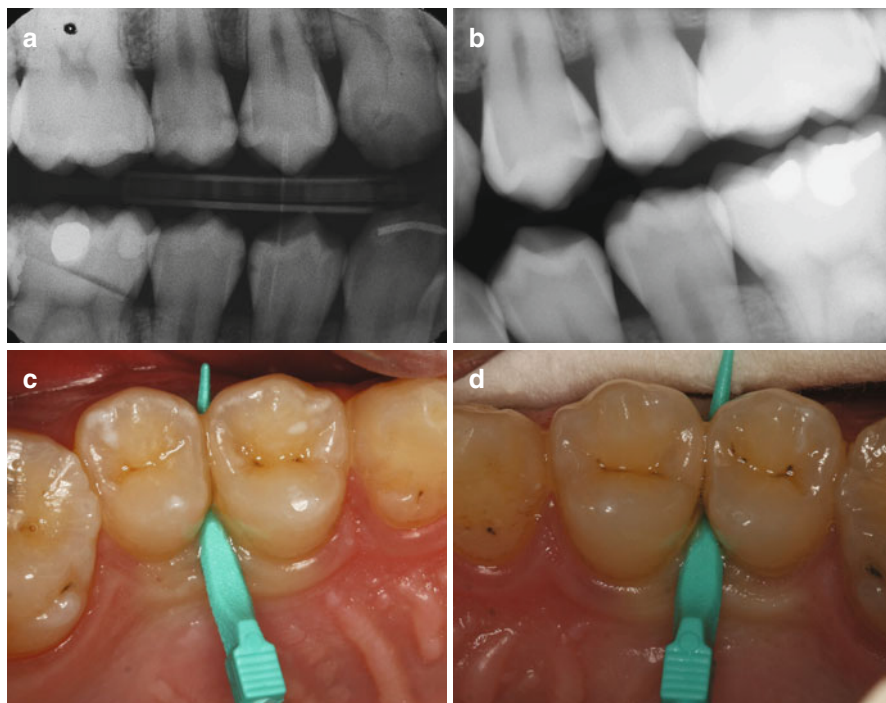
#### 10.4.2 Early Caries and Their Consequences: Can We Treat Them at Home?

A 17-year-old female had a complaint of aesthetic disturbance due to the whiteness located at the cervical third of the mandibular incisors (Fig. 10.9a). Currently, her oral health is good with a low caries risk status. Through her dental history, she mentioned demotivated oral care habits due to vomiting reflex while brushing besides an



**Fig. 10.6** Remineralization of enamel carious lesion on 21 with self-assembling peptide P11-4, Curodont Repair (Credentis AG, Windisch, Switzerland). (a) Baseline photo. (b) QLF (Inspektor™ Pro, Inspektor Dental Care BV, Netherlands) measurement at baseline  $\Delta F = -9.02$ . (c) Intraoral view 3 months later. (d) QLF measurement 3 months later ( $\Delta F = -6.57$ ) (Courtesy of Dr. Ecem Ergin, İzmir, Turkey)

un-oriented time period during her first years in the boarding school. Regarding the visual inspection, the lesion surface seemed to be limitedly porous, and the demineralized area was in a consistent curve with the gingival edge, which may help identify these lesions as late-term white spot lesions occurred during eruption phase but currently are rather chronic with showing smooth surfaces. Casein

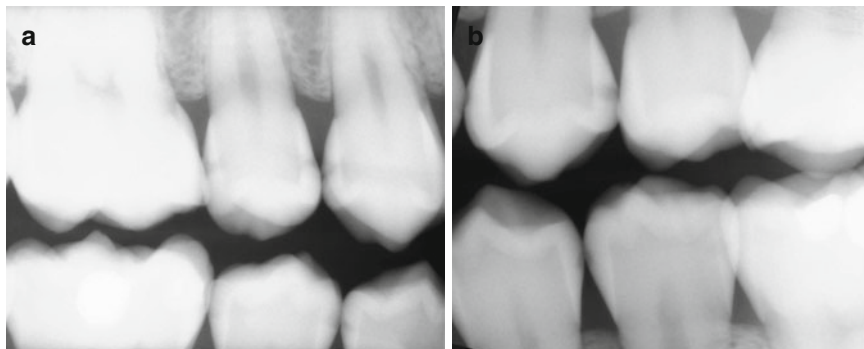


**Fig. 10.7** Bitewing radiographs showing (a) E2 caries for 14 distal and E1 caries for 15 mesial and (b) D1 caries for 24 distal and E2 caries for 25 mesial. (c) Clinical views of 14–15 and (d) 24–25. (Courtesy of Dr. Hande Şar Sancaklı, İstanbul, Turkey)

phosphopeptide-amorphous calcium phosphate-containing crème (Tooth Mousse, GC Dental, Leuven, Belgium) was prescribed for 8 weeks to apply once a day and especially during the night. At the end of the 2-month period, the lesions were reduced and were only visible in a smaller area. Even though the white spot lesion surfaces diagnosed at the baseline were not so acute for an ideal remineralization procedure, the lesions improved to become aesthetically quite acceptable (Fig. 10.9b).

## 10.5 Phase 4: Adulthood

Adulthood is the stage of full physical and intellectual maturity. The adult should take responsibility and control of his/her own life without relying on others, and this includes his/her health.



**Fig. 10.8** 24-month recall bitewing radiographs (a) 14–15 and (b) 24–25

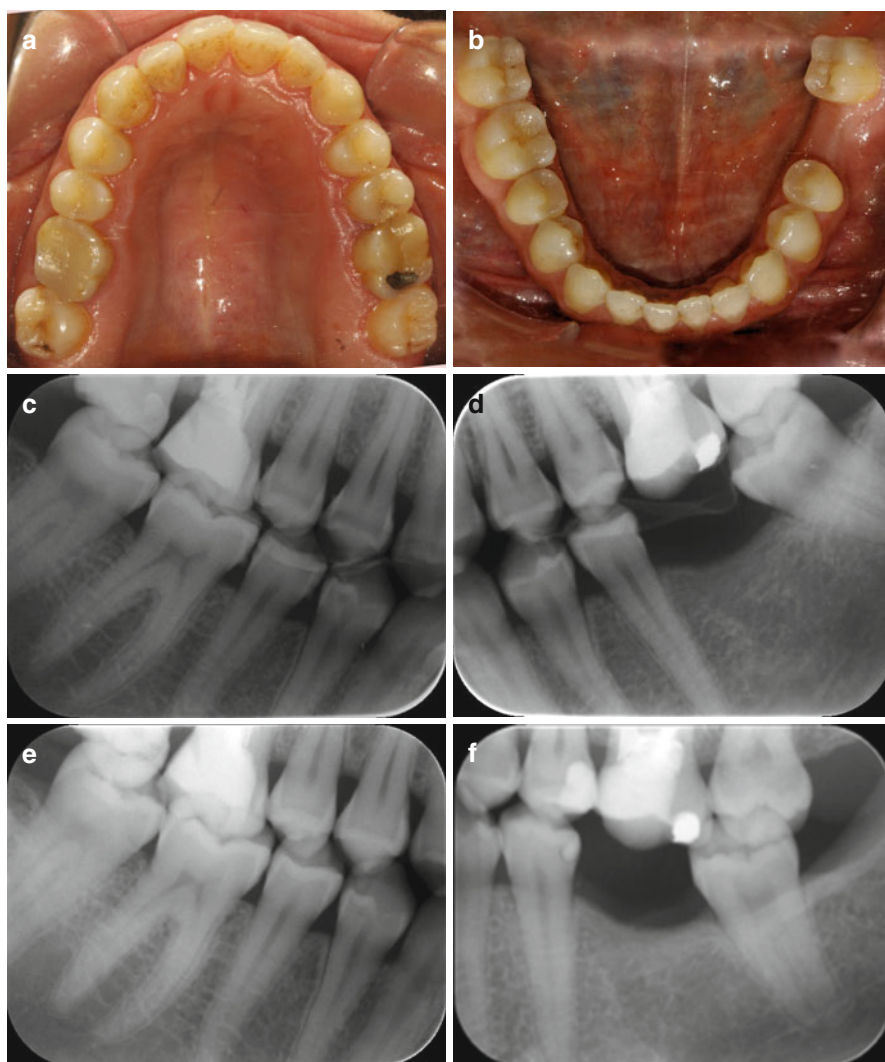
**Fig. 10.9** (a) Enamel carious lesions in 43–42–41–31–32. (b) Remineralization after 8-week application of CPP-ACP crème (Tooth Mousse, GC Dental, Leuven, Belgium) *arrows* indicating diminished/remineralized enamel lesions (Courtesy of Assoc. Prof. Dr. Hande Şar Sancaklı, İstanbul, Turkey)



### 10.5.1 Do Some Patients Have Weak Teeth?

A 22-year-old female dentistry student stated that she was unlucky and had weak teeth. She believed that there was nothing to be done for her teeth besides accepting that she needs to have treatment and eventually will lose them.

A detailed patient history revealed that she had lost 36 when she was 15 years old. She had root canal treatments for 16 and 26 at the age of 18. Intraoral and radiographic evaluation revealed that she had new proximal lesions on her second premolars. Early destruction of three first molars of the patient suggested a definition for ‘weak teeth’ as molar incisor hypoplasia (MIH) (Fig. 10.10a–d). MIH is defined as demarcated defects of enamel affecting one or more first permanent molars, with or without involvement of the incisor teeth [19]. This may explain carious destruction resulting in endodontic treatment and extraction of first molars for her, but present proximal lesions show the need for further management.



**Fig. 10.10** Caries management of a 22-year-old dentistry student with proximal caries. (a) Maxillary intraoral view. (b) Mandibular intraoral view. (c, d) Baseline bitewing radiographs. (e, f) 1-year follow-up bitewing radiographs

She was brushing her teeth regularly twice a day with toothpaste containing 1450 ppm fluoride. She was not flossing.

Seven-day diet record showed that she had a high sugar score and frequent snacks containing sugary products. She was consuming fruit soda three to four times a day between meals. Her meal times were not regular.

She had proximal cavitated carious lesion on 35, and, since 36 was missing, a small cavity only on the distal surface of the tooth was prepared and a composite

resin restoration (Filtek Ultimate, Universal, 3M ESPE, Seefeld, Germany) was applied. There was distal proximal dentine carious lesion without any extension to occlusal surface with sensitivity on 25. A glass ionomer restoration (Ketac Molar Easymix, 3M ESPE, Seefeld, Germany) was performed on 25. Professional tooth cleaning was performed and Listerine mouthwash with fluoride was prescribed. Fluoride varnish (Duraphat, Colgate, New York, USA) was applied every 3 months in the first year. She improved her tooth brushing and began flossing.

She started to consume less soda and restricted it to meal times. She reported her diet history during the summer holidays through Whatsapp. She needed some more time to reduce her sugar score. She still needs counselling for her diet during exam periods and on vacations. A high compliance was possible with her since it was possible to see her frequently (Fig. 10.10e, f).

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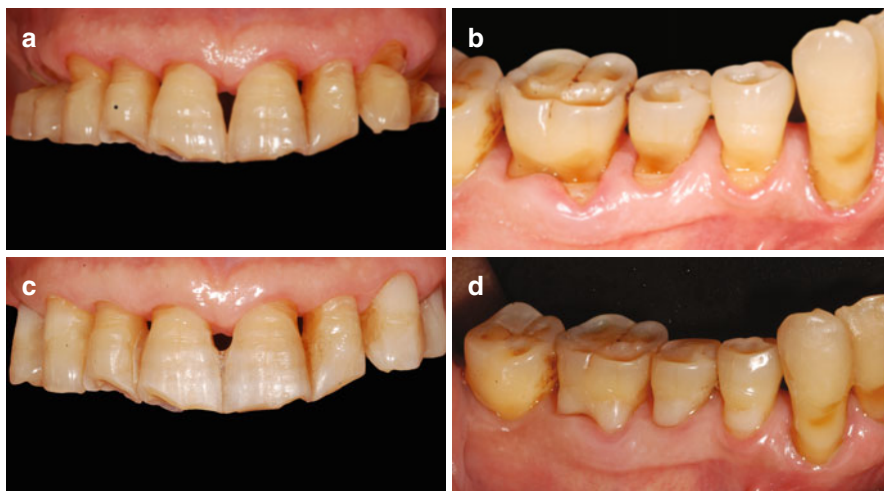
## 10.6 Phase 5: Senescence (Old Age)

Biologically, ageing diminishes the metabolism of the organism through time, which gradually decreases physical and mental capacity and risks overall health. There is no general consensus on the calendar age as the threshold of old age, which is closely related with living conditions, but retirement age for the population usually marks the beginning. Old age is also characterized by health disorders and higher medication needs [20].

### 10.6.1 Do Teeth Go Rotten as Patients Get Old?

A significant proportion of the world's population is living longer – especially in developed countries. Older adult population over 65 years of age with 21 natural teeth and more is accepted as having functional dentition and needs further care to avoid loss [21]. Dentate elderly usually suffer from periodontal problems, which cause gingival recession exposing the root surface [22–24] and hyposalivation end up with root caries. Figure 10.11 shows an 81-year-old dentate elderly who had a heart operation 25 years ago. He is taking medication for diabetes and high blood pressure. For this case, it is important to maintain good oral hygiene in conjunction with proper antimicrobial treatment for periodontitis (see Chap. 5). He has 24 teeth, and he is using chlorhexidine gel (Cervitec Gel, Ivoclar Vivadent, Schaan, Liechtenstein) in addition to fluoridated toothpaste from time to time. He is brushing his teeth regularly and uses interdental brushes and toothpicks for interproximal areas.

His inactive root carious lesions were treated by self-curing resin material, and splint was applied to avoid mobility. The planned preventive regime included chlorhexidine varnish (Cervitec Plus, Ivoclar Vivadent, Schaan, Liechtenstein) at 3-month intervals for suppressing cariogenic bacteria in combination with fluoride varnish (Duraphat, Colgate, New York, USA). Probiotics are added to healthy diet suggestions as well as chewing gum containing xylitol to increase the salivary flow and relieve xerostomia (see Chap. 6).



**Fig. 10.11** An 81-year-old dentate elderly with periodontitis and root caries. (a) Maxillary dentition. (b) Right mandibular dentition. (c, d) Intraoral views after treatment (Bondfill SB and Superbond C&B, Sun Medical Co., Ltd., Moriyama City, Shiga, Japan) (Courtesy of Assoc. Prof. Dr. Nejat Nizam, İzmir, Turkey)

## 10.7 Phase 6: Extraordinaries in Life: Life Phases May Be Different for Some

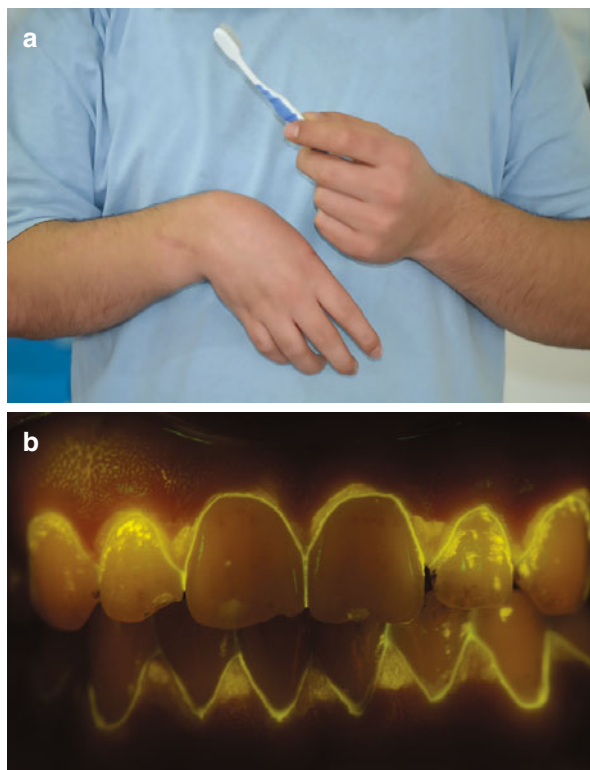
Natural course of life may be affected by innate deficiencies or changing health conditions. A person with any kind of disability will face various challenges in life, and a normal person may become ill and illness and treatment may affect natural flow. Therefore, disabilities, chronic systemic diseases, medication and especially cancers and their post-treatment processes play a vital role on the individual oral health risk status.

### 10.7.1 Do Disabilities Have an Effect on Oral Health?

Systematic review [25] reported that mentally disabled individuals have poorer oral hygiene and increased prevalence of periodontal problems than the general population, whereas either same or lower caries rates. The most outstanding finding is the high level of untreated decay. Extraction was the treatment of choice when treatment was given. The caregiver must carry out daily hygiene procedures, and oral health training should be targeted to the caregiver to change the attitude and help overcome any obstacles. Physical impairment such as blindness or deafness or conditions defined as needing special care also affect oral health and quality of life of the individual [26].

A developmental deficiency on the right hand with movement restriction in a 25-year-old male affected his oral hygiene practices. The need to use incisors to

**Fig. 10.12 (a)** Developmental deficiency on right hand and restriction of movement. **(b)** Plaque accumulation seen by yellow fluorescence (Plaque Test; Ivoclar Vivadent AG, Liechtenstein) and incisal edge fractures on 12, 11 and 21



support actions done by the left hand like changing clothes or opening packages caused fracture of the incisal edges of 12, 11 and 21 (Fig. 10.12). The incisors were treated with composite restorations (Filtek Ultimate Universal, 3M ESPE, Seefeld, Germany) and need to be repeated from time to time. Electrical toothbrush is suggested for an easier plaque removal with 1450 ppm fluoridated toothpaste.

### 10.7.2 Will Cancer Treatment Ruin the Teeth?

Cancer and cancer therapies cause oral complications that have an impact on quality of life. Acute oral complications are mucositis, infection, saliva and neurosensory changes, whereas rampant caries, soft tissue and bone necrosis are the long-term hazards. Alkali oral rinses containing salt and baking soda should be preferred for mucositis. Oral rinses without alcohol and antimicrobials are used to relieve pain caused by secondary infections. Cryotherapy (using rounded ice chips in the mouth) during infusion of the chemotherapeutic agent helps reduce mucositis. Consequently, oral pain limits food intake and oral hygiene practices [27, 28].

In order to overcome the problems due to hyposalivation, physical stimulation of salivary glands by xylitol chewing gum or sucking sugar-free lozenges may be



suggested. Salivary substitutes, available as rinses, gels, sprays and lozenges, may be prescribed. Diet counselling should include avoidance of spicy, crunchy, hot, acidic food and beverages. Fluoride and remineralization agents should be administered with frequent dental appointments in order to control oral health [27–31]. Basic oral care for haematology–oncology patients addresses pain control, maintains oral function, prevents infections, manages oral complications and provides good quality of life [32].

A male patient (15 years old) had been diagnosed with left cranium floor soft tissue sarcoma at the age of 4. He has been treated with chemotherapy for 18 months and received radiotherapy. Rampant caries was treated by glass ionomer restorations when he was 6 years old. Restriction in opening the mouth makes treatment problematic. A posterior composite restoration (Filtek Ultimate Universal, 3M ESPE, Seefeld, Germany) was performed on 36, and glass ionomer ART sealants (Ketac Molar Easymix, 3M ESPE, Seefeld, Germany) for the remaining first molars were applied when he was 8 years old. Fluoride varnish was applied at 3-month intervals up to last 2 years. He moved to another city and could not be followed for a while. In his recent visit, he stated that he is brushing with 1450 ppm fluoridated toothpaste twice a day and flossing regularly but he had several problems with the left ear due to erosion of the bone so he has been hospitalized several times and changed school within last 2 years that highly affected his diet and hygiene practices. The intraoral evaluation revealed an inactive proximal carious lesion on 21 (Fig. 10.13). It was possible to restore the lesion with composite resin. Fluoride varnish (Duraphat, Colgate, New York, USA) was applied and 3-month recalls were scheduled.

A 62-year-old male who was diagnosed with a multiple myeloma 2 years ago received chemotherapy prior to double haematopoietic stem cell transplantations in a 2-year duration. He is presently under routine recall at every 2 months without any specific on-going treatment. As stated in the basic oral care recommendations, the treatment plan was designed by the identified risk status of the patient according to the characteristics of his saliva and bacterial load [33]. Extraoral diagnosis showed that the occlusal vertical dimension has decreased due to the

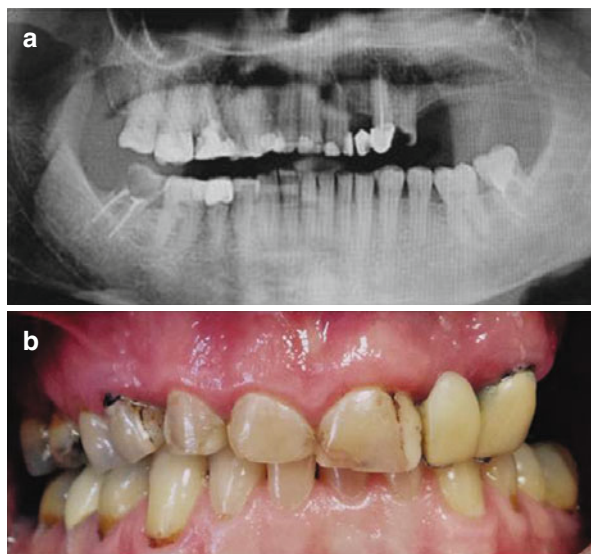


**Fig. 10.13** A 15-year-old patient with mouth opening restriction due to radiotherapy that he received at the age of 4

pattern of occlusion and attrition. Before providing further complicated treatments of the case with prosthesis, the primary step was the need for prophylactic interventions. Regarding chemotherapy and immune-suppression as a complementary part of the transplantation procedure, he has suffered from hyposalivation and immune deficiency against infections. Root caries, cervical lesions and caries lesions on the incisal edges were diagnosed (ICDAS3-5) as shown in Fig. 10.14. He mentioned severe pain in right maxillary segment, which was related to the carious premolars and molars.

Emergent treatments were planned, and direct cervical composite restorations (Filtek Z550, 3M ESPE, Seefeld, Germany) of vital teeth were performed primarily on the maxillary arch. Regarding his low saliva buffering capacity and high caries risk status, lower premolar cervical lesions and caries on the incisal tips of the canines were restored with resin-modified glass ionomer (RMGI, GC Fuji II LC; GC, Dental, Leuven, Belgium) material, followed by application of high concentration of sodium fluoride varnish (Clinpro White Varnish, 3M ESPE, St. Paul, USA) (Fig. 10.15). He was then prescribed 1450 ppm fluoride-containing toothpaste in addition to chlorhexidine gel to use for every 2 weeks in a 3-month period and a lubricant mouth gel (GC Dry Mouth Gel; GC Dental, Leuven, Belgium) to moisturize the oral mucosa when needed. Dental diet recommendations were in accordance with the regulated low-carbohydrate-containing diet that was suggested by his dietician and physician for controlling the progress of multiple myeloma. He was then referred for his fixed prosthodontic restoration replacement, and a 3-month recall was asked to promote his oral health in a preventive-based approach.

**Fig. 10.14** A 62-year-old male chemotherapy patient. (a) Orthopantomogram. (b) Clinical examination showing impaired marginal fittings of the existing restorations as well as carious lesions specific to hyposalivation (Courtesy of Assoc. Prof. Dr. Hande Şar Sancaklı, İstanbul, Turkey)



**Fig. 10.15** Maxillary teeth were restored by composite resin and mandibular cervical lesions restored with RMGI (Courtesy of Assoc. Prof. Dr. Hande Şar Sancaklı, İstanbul, Turkey)



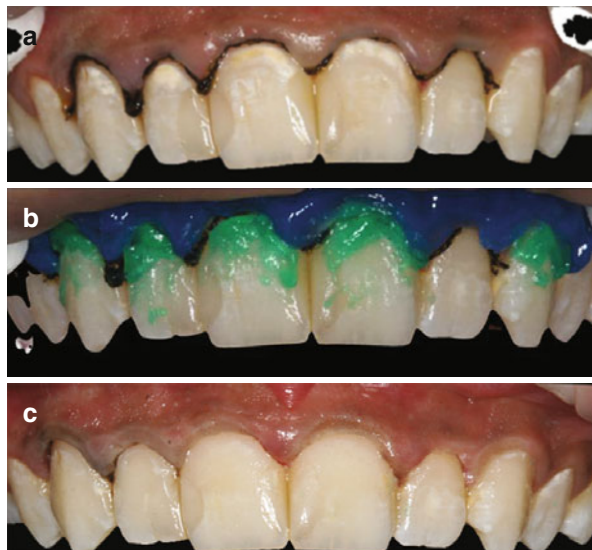
### 10.7.3 Do Systemic Conditions Affect Oral Health?

As the lifespan of mankind is getting longer, systemic diseases threaten most lives. Non-communicable diseases (NCD) like cardiovascular disease, cancer and diabetes are the leading diseases associated and furthermore the main reason of the human deaths worldwide. Oral diseases are recently acknowledged to share the common risk factors with the mentioned systemic diseases, thus highlighting a significant link between oral health and general well-being [34]. It is inevitable to declare that optimal oral health maintenance have a great impact on general health of an individual.

Diabetes mellitus is the most prominent NCD related to oral disease in means of sharing common risk factors as behavioural, dietary and physiopathological determinants. Diabetes mellitus is a chronic metabolic disorder with systemic complications such as retinopathy, neuropathy, micro- and macrovascular alterations and oral complications. Due to the microvascular changes and neuropathic complications, diabetic patients are more often at an increased risk of oral diseases such as gingivitis, periodontal disease and dental caries which are frequently associated with poor glycemic control [35]. Regarding oral complications of diabetes, especially periodontal disease, which is accepted as ‘the sixth chronic complication of diabetes’, is evidently shown to be related to the presence of cytokines, dentobacterial plaque, haemoglobin A1c levels, duration of diabetes and body mass index parameters in both diabetic children and adults [36, 37]. Thus, it is impossible to disseminate the knowledge on diabetes and oral health management to raise the lifelong health quality of the diabetic patient.

Hyperglycaemia in diabetic children, adolescents and adults has also been associated with decreased salivary secretion and high salivary glucose frequently leading to dental caries [38]. It means that dental caries can be defined as another oral complication of diabetes which is not directly related to physiopathology but intersects with the diabetes complications and immune system alterations such as the change in the saliva sugar/glucose level, decrease in salivary flow and unstable salivary pH. All these determinants increase caries susceptibility during diabetes and mostly become dominant in the hyperglycaemia condition. On the other hand, studies report inconsistent results about caries prevalence and relate it to strict nutritional regimen,

**Fig. 10.16** (a) Enamel carious lesions of vestibular surfaces and proximal cavitated carious lesions in patient with uncontrolled type 1 diabetes before being hospitalized. (b) Isolation of the gingival margins and hydrochloric acid application for resin infiltration (Icon, DMG, Hamburg, Germany). (c) Resin infiltration on 13, 12, 11, 21 and 23 cervical lesions and RMGI restorations on 12 mesial, 11 mesial and distal, 21 mesial and 22 mesial and vestibule carious lesions



followed by type 1 diabetes children [39, 40]. Since the above-mentioned predisposing factors are severely associated with failure of the blood glucose control of the diabetic patient, disregarding the type of diabetes, prophylactic measures are highly needed [35].

It is essential to design individual ‘oral coaching programmes’ including caries and periodontal disease prevention strategies regarding the individual needs and demands of each patient, oral motivation trainings and dietary consulting for oral health in controlled diabetic patients. Besides oral health motivation and behavioural intervention focusing on a common risk factor approach, regular control of blood sugar is also ‘a must have’ priority to promote a healthy dentition and oral cavity in diabetes.

An advanced case at the age of 16 with type 1 severe diabetes unfortunately in an unregulated status was appointed for dental consultation while he was hospitalized. After periodontal treatment, which is an emerging treatment step to regulate the required blood glucose control, restorative treatment planning was designed in a preventive-based approach regarding his high caries risk status. Acute demineralized enamel carious lesions were obviously seen on the cervical vestibular surface of the maxillary incisors beside the proximal carious lesions that certainly need restorative treatment (Fig. 10.16a). After isolating the gingival crevices, the enamel carious lesions were infiltrated with resin infiltrant, and the remaining proximal carious lesions were restored with resin-modified glass ionomer (RMGI) restorative material regarding the fluoride-releasing characteristics that may withstand against hyposalivation and acidic challenges (Fig. 10.16b, c). The restorations were restored primarily with such material until his systemic conditions get stable. Fluoride varnish containing tricalciumphosphate (TCP; Clinpro White Varnish, 3M ESPE, St. Paul, USA) was applied all over the teeth surfaces, and an additional

CPP-ACP crème (Tooth Mousse, GC Dental, Leuven, Belgium) was prescribed for daily use in order to protect against further demineralization until the recall sessions planned at every 3 months.

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## 10.8 Final Remarks

Several life phases with specific determinants and needs are present through the lifespan of an individual. All these phases reveal various oral conditions due to behaviours, changes and the status of the organism. Undoubtedly, oral changes within all these life stages lie in dental history of the patient. Thus, a dentist should accompany as a ‘dental coach’ during all these phase transitions of an individual to address the oral health disturbances and required needs in a preventive-based approach. Under all these circumstances, we should take into account that as dentists we all have a great responsibility and a broadened role to strongly defend oral health as a fundamental human right and an integrated part of general health and well-being [41].

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