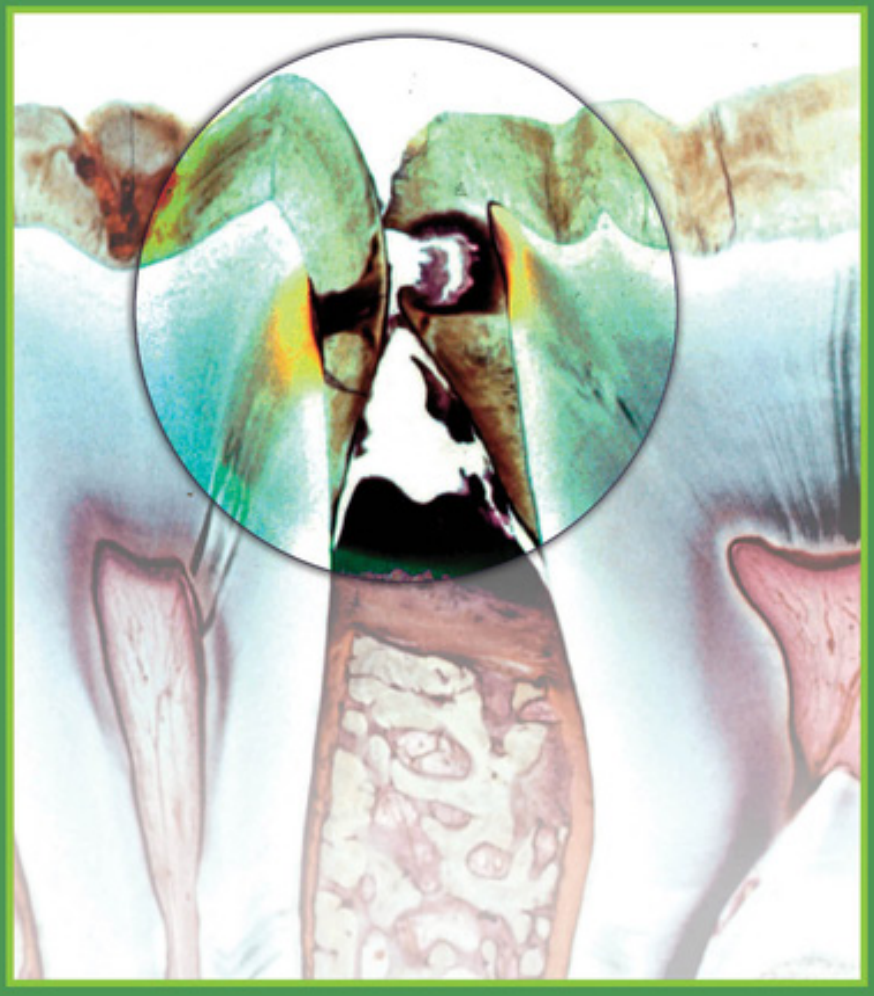
Fourth Edition

Dental Caries

The Disease and Its Clinical Management



Edited by **Ole Fejerskov** and **Bente Nyvad**

WILEY Blackwell

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The Disease and Its Clinical Management

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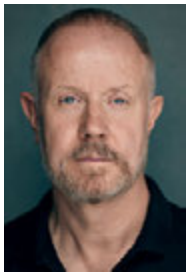

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Preface

In the middle of the previous century, the six heads of the departments of Operative or Restorative Dentistry at all the Scandinavian Dental Schools joined in writing a Nordic Textbook of Cariology (Nordisk Lärobok I Kariologi). The chapters were written in Swedish, Norwegian, or Danish as it was still expected that the dental students would be able to read and understand the different languages of the Nordic countries. Each professor was assigned a part of the subject that they were assumed to be particularly knowledgeable about. In the last 5th edition in 1980, the number of contributors had grown to 15 as the Karolinska Institute in Stockholm included 3 contributors, and 2 came from University of Turku and 2 from Copenhagen. During the entire period the book was edited by Professor Yngve Ericsson from Stockholm. Over the years he gradually took a more firm grip of the editing content and organization of the book, but admitted in the last preface that there were some differences in interpretation of research data and conclusions by the various contributors.

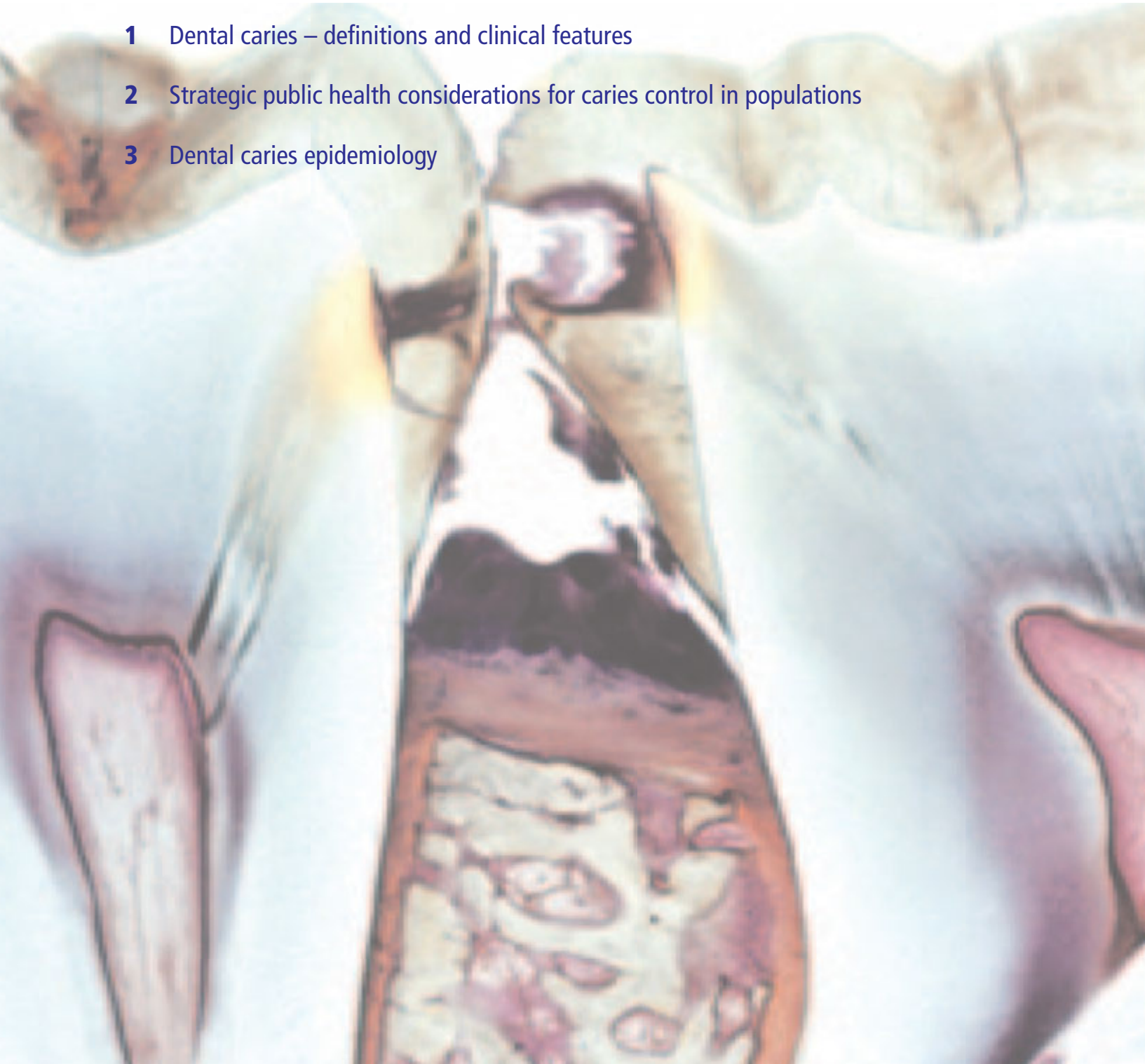
So, when Yngve retired it became evident that two different "schools of thinking" concerning etiology and pathogenesis of dental caries had grown to an extent where it was no longer possible to maintain the principles of a united text combining all departments dealing with dental caries in the four Nordic countries. Thus, one Swedish dental school was insisting that dental caries was a result of *Streptococcus mutans* infection and did not want to join the now 25 other researchers from the different Nordic countries who wrote the first *Textbook of Cariology* edited by Anders Thylstrup from the dental school in Copenhagen and Ole Fejerskov from the Royal Dental College in Aarhus.

Hitherto, cariology was considered a theoretical discipline almost totally separated from the clinical, operative procedures associated with the treatment of the carious cavities and insertion of fillings. This was – and still is – the core of most of the restorative work in the oral cavity – the backbone of dentistry. To bring the message to the dental students that the content of the textbook was indeed highly important for daily clinical decision making we, in the second edition, changed the title to *Textbook of Clinical Cariology*. Professor Thylstrup died all too young, and I decided to bring the future editions even closer to the clinic by joining forces with Professor Edwina Kidd, a well-known English restorative dentist with some experience in cariology as co-editor from 2003. We changed the title to *Dental Caries: The Disease and Its Clinical Management* in order to send the message that this book is the backbone of knowledge necessary for every dentist who wish to conduct up-to-date diagnosis, prevention/control, and treatment of the disease Dental Caries. We are most grateful to Professor Kidd (who decided to retire some years ago) for the immense enthusiasm and inspiration she has been for this book.

Everyone who has followed how this textbook has developed over the years will appreciate that the underlying message has been to reveal what is meant by the concept of "caries control." In international lectures, we have claimed, based on the growing evidence, that "caries cannot be prevented; rather its progression can be controlled." You might say that this is semantics, but we will argue that by making this distinction, dentists can appreciate that no single method or modality can prevent dental caries from occurring. That is true for all populations worldwide. Dental caries is ubiquitous and as old as humankind. The current edition is a thorough update on the basic biological mechanisms behind dental caries and what is presently known about social and commercial determinants of health inequalities as far as dental caries is concerned. Any development and evaluation of community-based interventions to control caries progression – and when necessary perform restorative dental treatment – may have limited success if the interventions are not firmly based on understanding the nature of dental caries.

Ole Fejerskov and Bente Nyvad
Aarhus
January 2024

Part I
Dental caries: What is it and what is the magnitude of the problem?



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Dental caries – definitions and clinical features

Ole Fejerskov and Bente Nyvad

The editors' view on dental caries and introduction to the book

Dental caries is a result of metabolic activities in the microbial deposits (the dental biofilm) covering the tooth surface at any given site. Caries lesions emerge when there is an imbalance in the physiological equilibrium between the tooth mineral and the biofilm fluid. Hence, carious lesions represent the signs and symptoms of multiple de- and remineralization processes accrued over time.

Many symptoms of caries (e.g., cavities) are detected in the late stage of the caries process, where drilling and filling are needed to prevent further breakdown of the tooth. This is symptomatic treatment, referred to as tertiary prevention (Fig. 1.1). Primary prevention corresponds to activities aimed to prevent the earliest signs and symptoms of caries. Secondary prevention refers to any method applied to prevent further progression of already existing disease, such as noncavitated caries lesions. Tertiary prevention covers any attempt to restore/treat pulp involvement including, ultimately, tooth extraction. It is highly important to emphasize that if restorative care is not followed up by proper caries control measures, it merely adds to the vicious cycle of tooth repair and eventual tooth loss (Chapter 15).

It should, however, be realized that what happens in a single tooth or tooth surface cannot be understood without appreciating that it is part of a human being influenced by the environment in which she or he lives (Fig. 1.2).

Most people around the world do not see a dentist regularly and those who do mostly belong to the economic middle class or are well off and ready to pay for high-tech oral rehabilitation – without necessarily appreciating how dental caries can be controlled by rather simple means. Therefore, the aim of this new edition of *Dental Caries* is to provide an updated knowledge on how to control and manage dental caries in populations and individuals with a specific focus on improved health outcomes.

We start the book by describing the magnitude of the caries problem in different populations (Chapter 3) together with a key chapter (Chapter 2) on strategic considerations for caries control in populations. These chapters are followed by an evaluation of the current caries diagnostic classifications and considerations about the importance of performing a proper caries diagnosis prior to making clinical treatment decisions (Chapters 4 and 5). The subsequent chapters illustrate how the oral environment, saliva (Chapter 8) and the oral microbiome (Chapters 6 and 7) modulate the metabolic processes involved in caries lesion development. Commensal microorganisms are constantly metabolizing in the biofilms of the mouth including dental biofilms on teeth leading to fluctuations in pH. These innumerable pH fluctuations result in demineralization and

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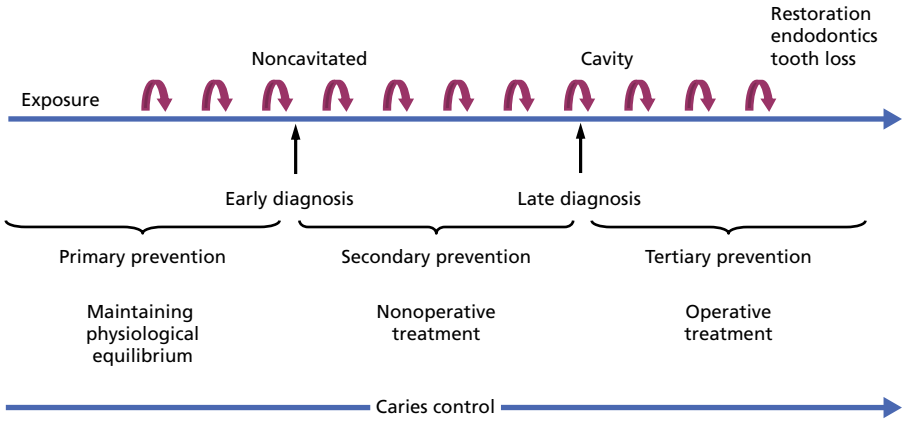


Figure 1.1 Schematic illustration of the caries control concept. Because of continuous exposure to the metabolically active biofilm, disease control must be maintained lifelong. Both nonoperative and operative treatments are part of the caries control concept, but operative treatments should never be the only treatment provided for patients with active caries lesions. See text for a detailed explanation.

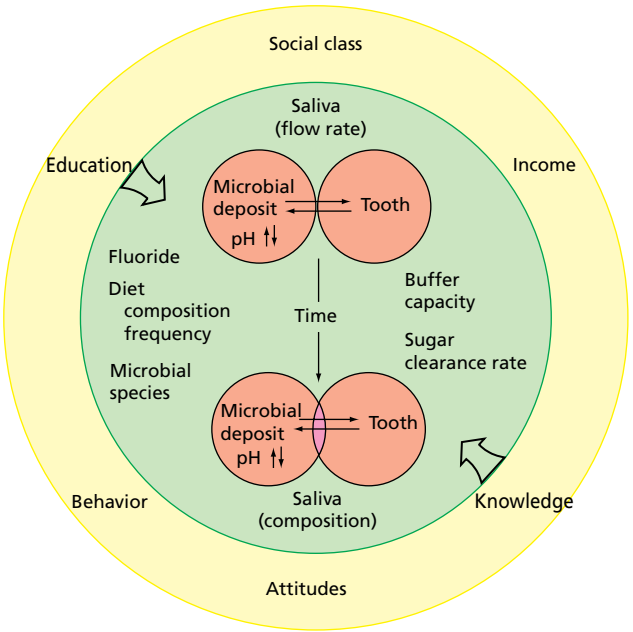


Figure 1.2 Schematic illustration of the determinants of the caries process. Those that act at the tooth surface level are found in the inner circle. In the outer ring are shown determinants that influence these processes at the individual and population levels. Adapted from Fejerskov and Manji 1990 and reproduced with permission of the University of North Carolina School of Dentistry.

"remineralization" of the dental hard tissues (see Chapter 9). These processes are normal and should be considered physiologic for the oral environment. However, depending on the environmental conditions, they may over periods of months or years result in a net loss of minerals from the tooth, that is, caries lesion development and progression (Chapters 9 and 10).

The following chapters provide basic knowledge about the oral environment and the importance of sugar and diets (Chapter 12), oral hygiene (Chapter 13), and fluorides (Chapter 14) in controlling the processes involved in the development of dental caries. These chapters enable the reader to understand, based on scientific evidence, why it is possible *to allow for most people across the world to maintain a natural functional dentition from cradle to grave*. However, it requires that people are informed and not least that public health and governmental authorities of a given society are prepared to integrate oral health into the preventive approaches for general health. A fundamentally important element of health promotion is the recognition that to achieve sustainable and equitable improvements in health, action is needed to create healthier living conditions for all. Hence, caries control cannot be seen out of context with the surrounding society, its culture, and traditions as a whole (see Fig. 1.2).

Dentists might argue that fluoride prevents dental caries. This belief originates from the times when it was first discovered that children living in areas with a higher level of fluoride in the water supplies experience fewer carious cavities than comparable age groups living in low fluoride areas (Chapter 14). However, as is stressed in Chapter 9, increased fluoride levels in the oral environment do not prevent the initiation of disease, but merely slow down the rate of lesion progression.

In Chapter 16, we introduce a clinical decision tree designed for control of dental caries in the individual patient, referred to as "The Dental Traffic Light." This set of rules highlights the principle that only patients with active caries need professional intervention. The basic idea behind this philosophy is that individuals diagnosed with active disease should learn to control their disease activity. The dental personnel plays an important role in guiding the patient in achieving this goal.

In many populations, progression of a carious lesion from the noncavitated stage to the cavity stage is normally a slow process that may last for years. Chapter 18 gives a critical evaluation of the indications for various minimally invasive treatments that may be applied either prior to or after the development of a cavity. The important point is made that restorative treatment should be postponed as far as possible. Removing the signs of lesions by insertion of fillings is merely symptomatic and does not address the causes of the disease. For this reason, restorative treatments should never stand alone; *restorative care without accompanying caries control just adds to the vicious circle of tooth repair and tooth loss*.

This new edition of "Dental Caries" provides the basic knowledge on how the oral environment interacts with the dental hard tissues life-long. There is a physiological balance between the host and the surrounding microbial environment under clinically healthy conditions. However, this balance may be interrupted by consumption of easily fermentable carbohydrates (Chapter 7). Therefore, a detailed understanding of the crucial importance of combatting the extensive misuse of free sugars in modern diets – provoked by commercial interests – is mandatory.

For more than a century, the dental profession has predominantly been focused on restorative care. However, the pandemic experience with the Covid-19 virus (Chapter 20) has necessitated a thorough rethinking of how future restorative procedures can be conducted with due respect to the safety of patients and the staff in the dental office. The evidence presented in this textbook may hopefully facilitate rapid dissemination and implementation of cheap and effective protocols for caries control. By so doing classical operative procedures can be reduced to a minimum while maintaining a low caries incidence to the benefit of populations worldwide. The consequences for the structure of the dental profession in the future in terms of staffing and type of training of future cadres are apparent.

Terminology

Before we describe the clinical features of caries, it may be helpful to introduce some terms that are used in this textbook. Unless the reader is familiar with this terminology, it can be difficult to understand what is written in subsequent chapters.

Caries lesions may be classified in a number of ways. Caries lesions may start on enamel (*enamel caries*) or on the exposed root cementum and dentin (*root caries*). Caries lesions may also be classified according to their *anatomical site*. Remember, there is nothing chemically special about these sites. Therefore, lesions are predominantly found in so-called stagnation areas where dental biofilm is allowed to persist for prolonged periods of time. Thus, lesions may commonly be found in *pits and fissures* or on *smooth surfaces*.

Primary caries is used to differentiate lesions on natural, intact tooth surfaces from those that develop adjacent to a filling, which are commonly referred to as *recurrent or secondary caries*. These two latter terms are synonyms. Recurrent caries is simply a lesion developing at a tooth surface adjacent to a filling. As such, its etiology is similar to primary caries.

Residual caries (as the term implies) is demineralized tissue that has been left behind before a filling is placed.

An important classification is whether a lesion is *cavitated* or *noncavitated*. A cavity is a physical hole in the tooth and it may impinge directly on the management of the lesion.

Caries lesions may also be classified according to their activity (Chapter 4). This is a very important concept and one that impinges directly on management, although it will be evident from the text that the clinical distinction between *active* and *inactive* (arrested) lesions is sometimes difficult.

A lesion considered to be progressing (you anticipate that the lesion would have developed further at a subsequent examination if not interfered with) would be described as an *active caries lesion*. This distinction is based on a judgment of the surface features of the lesion in combination with an assessment of the oral health status of the patient. In contrast to this is a lesion that may have formed years previously and then stopped further progression. Such lesions are referred to as *arrested caries lesions* or *inactive caries* lesions.

You may also meet the terms "*remineralized*" or *chronic lesions* used to signify arrested lesion, but, as you will appreciate later, the term remineralization should be used with caution (Chapter 9). The distinction between active and inactive/arrested lesions may not be totally straightforward. Thus, there will be a continuum of transient changes from active to inactive/arrested and vice versa. A lesion (or occasionally part of a lesion!) may be rapidly progressing, slowly progressing, or not progressing at all. This will be entirely dependent on the metabolic activity in the biofilm covering the site and the environmental challenge. Clinically, if in doubt, the dentist should always react as if he/she is dealing with an active lesion.

Despite the diagnostic difficulties (see Chapters 4 and 5), these distinctions are very important to the clinician because if a lesion is not active, no action is needed to control further progression. If, on the other hand, a lesion is considered active, steps should be taken to influence the metabolic activities and possibly the ecological balance in the biofilm in favor of arrest of further mineral loss.

At this point, it is also sensible to discuss a possible confusion in terminology. The first sign of a carious lesion on enamel that can be detected with the naked eye is often called a *white spot lesion*. This appearance has also been described as an early, *initial,* or *incipient lesion*. These terms are meant to say something about the stage of lesion

development. However, a white spot lesion may have been present for many years in an arrested state and to describe such a lesion as early would be inaccurate. A dictionary definition of incipient is "beginning," an initial stage. In other words, an initial lesion appears as a white, opaque change (a white spot) – but any white spot lesion is not incipient!

Rampant caries is the name given to multiple active carious lesions occurring in the same patient. This frequently involves surfaces of teeth that do not usually experience dental caries. Patients with rampant caries can be classified according to the assumed causality, for example *bottle or nursing caries*, *early childhood caries when observed in young children, and "bakers caries," radiation caries, and drug-induced caries when seen in adults*. Early childhood caries (ECC) is simply caries on teeth of a young child that are not clean, exposed to carbohydrates and located in an area of the mouth where oral clearance is low (Chapter 16).

Hidden caries is a term used to describe lesions in dentin that are overlooked on a visual clinical examination but are large enough and demineralized enough to be detected radiographically. It should be noted that whether a lesion is actually hidden from vision depends on how carefully the area has been cleaned and dried and whether an appropriate clinical examination has been performed.

Examples of dental caries

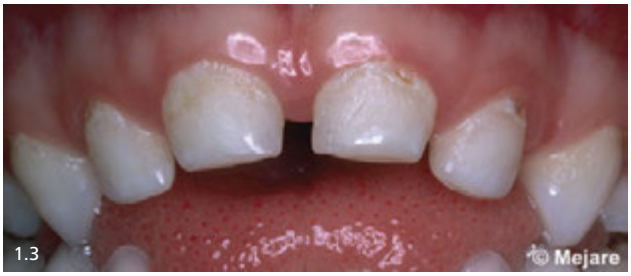
Dental caries is the outcome or symptom of innumerable pH fluctuations in a dental biofilm located on a tooth surface. If the result of this fluctuation in pH over a long time is a net loss of minerals from the tooth enamel, it will gradually result in an increased porosity of the enamel (for details, see Chapter 9), which gives rise to a decrease in enamel translucency. At this stage, we can diagnose a white opaque lesion. It may be discerned with the naked eye when the patient opens the mouth, and if the dentist cleans and dries such a lesion with a blast of air, it becomes more pronounced because salivary moisture is partly removed from within the enamel pores.

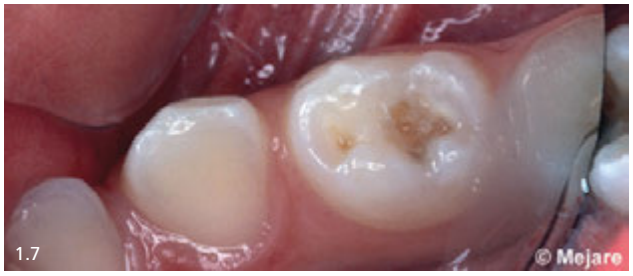
A white spot lesion may become arrested and persist for many years, but because the lesion in enamel is porous, it is to be expected that food stain will sieve into the porosities and hence a white spot lesion may over time change color to brown and even almost black.

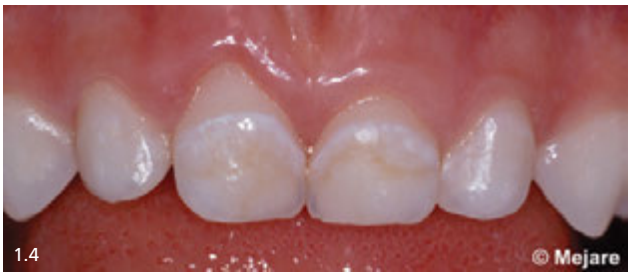
The shape of a lesion reflects the area where the dental biofilm has been allowed to mature and remain for some time. Half a century ago, before the introduction of fluoride toothpaste, it was common to see classical "kidney-shaped" lesions beneath the contact facets on approximal surfaces of molars in children with poor oral hygiene. These lesions often extended as a band of chalky white enamel along the gingival margins onto the buccal and lingual surfaces. In contemporary populations with improved oral hygiene, the extent of such lesions is reduced, but the shape of the lesions still reflects the "shape" of the stagnation area.

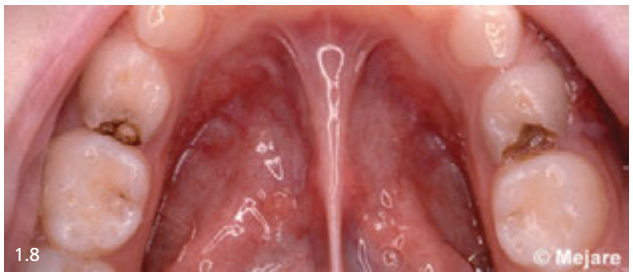
In the following illustrations we shall demonstrate a spectrum of manifestations of caries lesions in children, adults, and elderly. Be aware that you are looking at magnified pictures; in the clinical setting, visual inspection is much more difficult.

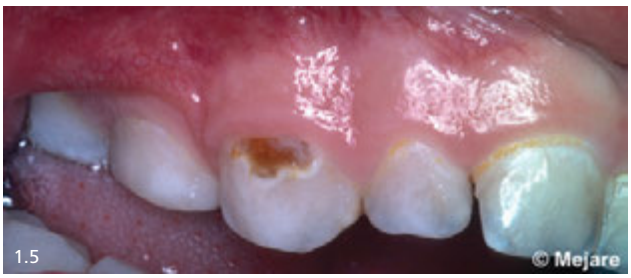
Note: most illustrations are of single teeth! However, in the clinical situation, you should never decide on a treatment by only considering a single tooth. The tooth is part of an oral environment in a patient! Therefore, the choice of treatment and the assessment of the prognosis of the dentition must be based on a comprehensive anamnesis and examination of the entire mouth of the patient (Figs. 1.3–1.61).

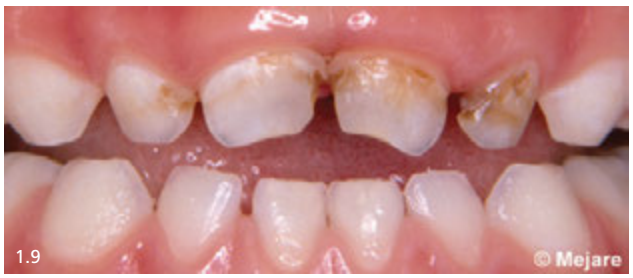


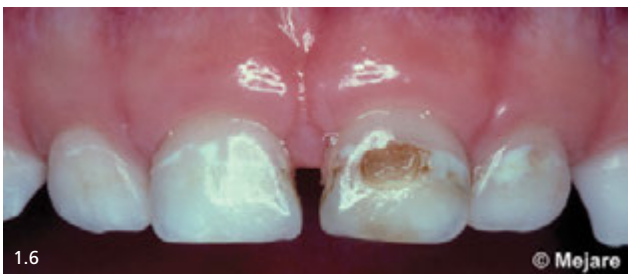


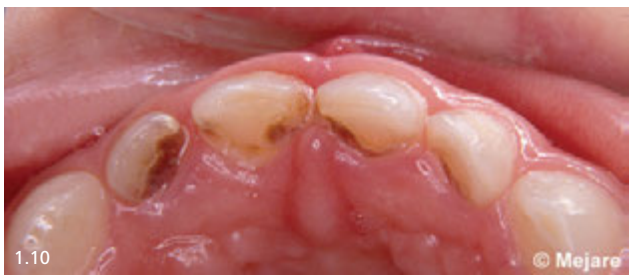




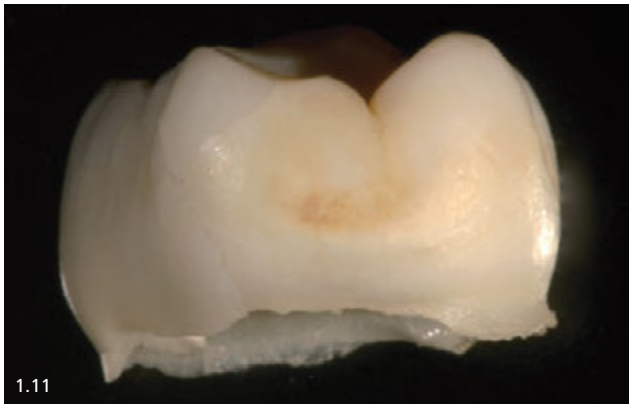


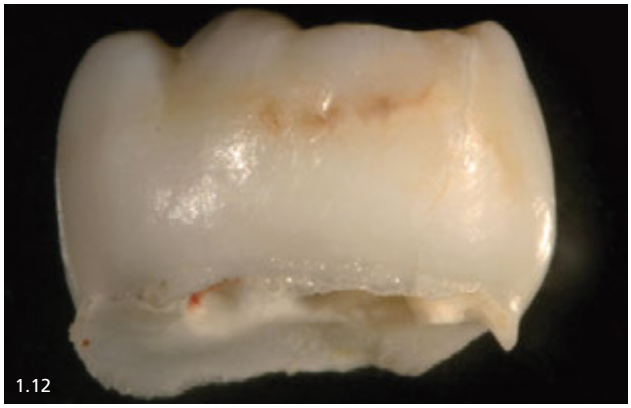




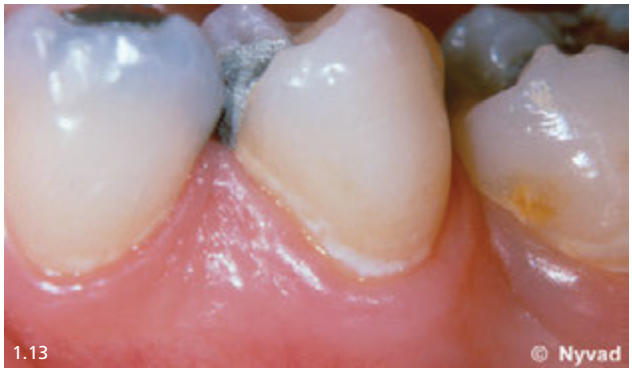


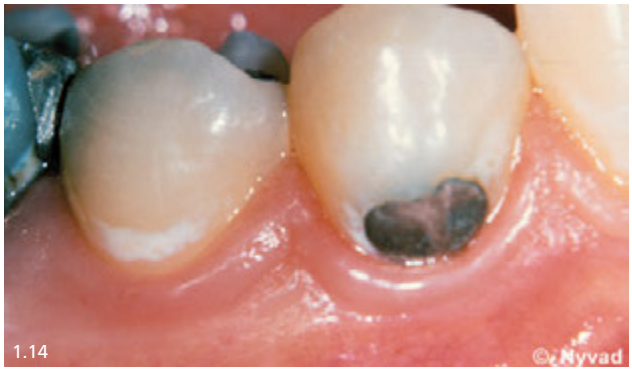
Figures 1.3–1.10 Figure 1.3: A 3-year-old child with thick accumulations of dental plaque along the gingival margin of the buccal surfaces covering active caries lesions, some of which present with distinct cavities. Figure 1.4: Inactive/arrested caries lesions on buccal surfaces of upper central incisor teeth in a 5-year-old child. Note that the shape of the lesions indicates where the gingival margin was located at the time when these lesions developed. The oral hygiene has improved and the surfaces of these noncavitated opaque lesions are now smooth and shiny. Figure 1.5: Upper deciduous canine from a 5-year-old with an active, cavitated lesion along the gingival margin. On probing it would be soft, but there is no reason to probe such a lesion unless you wish to provoke a pain reaction! Figure 1.6: Upper incisors in a 5-year-old child. Several narrow, white opaque inactive caries lesions are located 1–2 mm from the gingival margins. One of the lesions exhibits a large cavity that is hard on probing. This is an example of an inactive, cavitated lesion. Figure 1.7: Deciduous first lower molar in a 2.-year-old child with two cavitated active caries lesions. Note the peripheral white, opaque rim of enamel surrounding the cavities. Figure 1.8: Lower first deciduous molars with active, cavitated lesions in the distal and disto-occlusal surfaces of a 6-year-old child. Figures 1.9 and 1.10: A 2-year-old child with extensive, active, partly cavitated caries lesions encircling the teeth. This is an example of so-called nursing bottle caries – or 'bottle caries.' Figures 1.3–1.10 courtesy of I. Mejare.

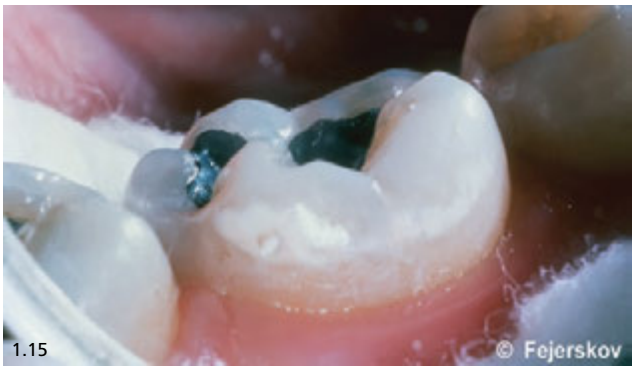


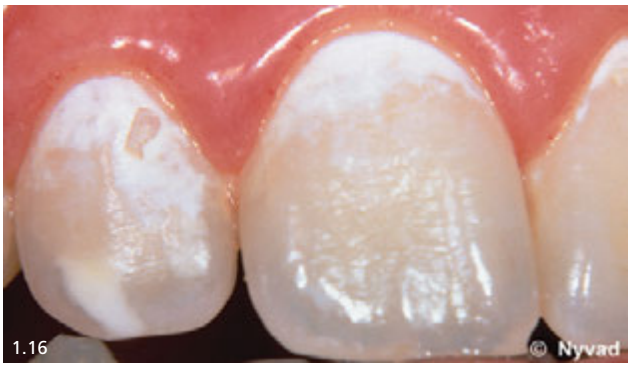


Figures 1.11 and 1.12 Slightly discolored lesions on approximal and buccal surfaces of an exfoliated deciduous molar. Note that the shape of the lesions reflects the areas where dental plaque has been retained above the position of the gingival margin. Note also the opaque kidney-shaped part of the approximal lesion cervically to the brown-stained center of the lesion in Fig. 1.11.

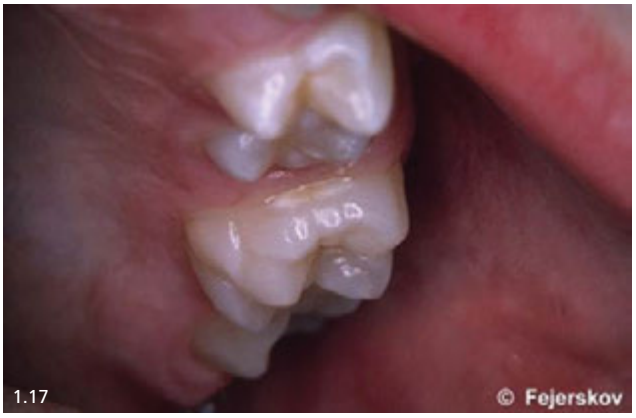


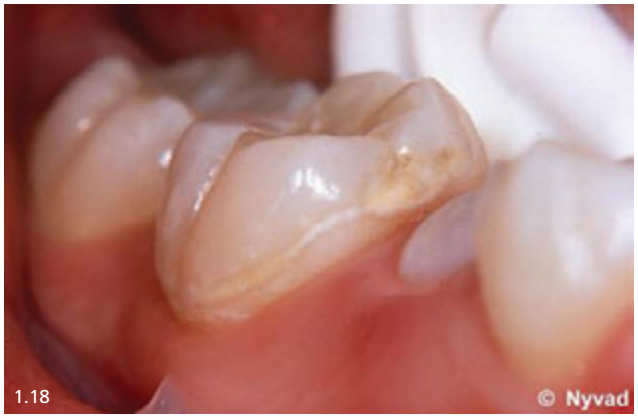


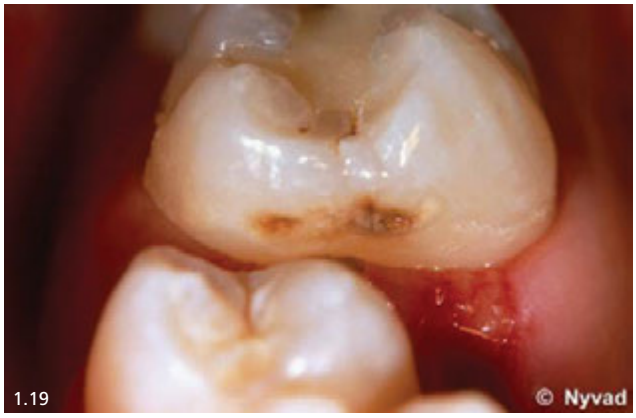


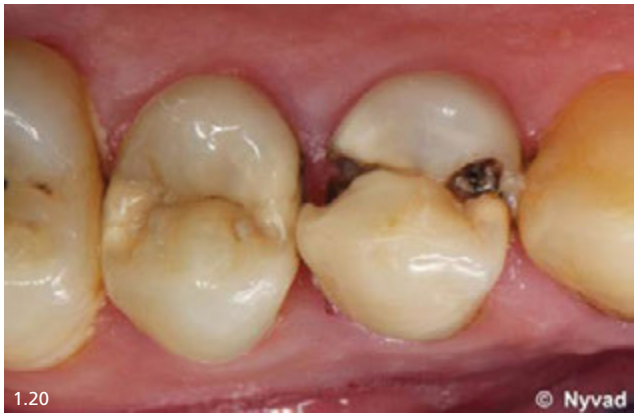


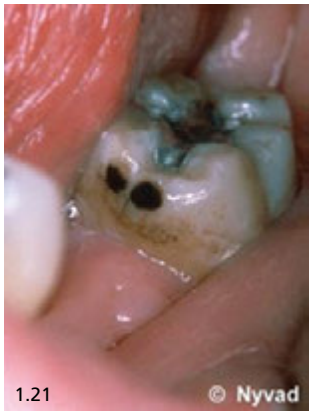
Figures 1.13–1.16 Figure 1.13: Active, noncavitated carious lesion on lower second premolar. The shape is typical, as it follows the curvature of the marginal gingiva and corresponds to where a narrow band of dental plaque has been located in a stagnant area. The surface is dull and chalky. It is called a "white spot lesion," although it extends from the approximal amalgam filling all along the gingival margin. On the mesio-buccal surface of the lower first molar another noncavitated lesion has taken up brown stain. Note also the very thin lesion on the buccal surface of the first premolar along the gingival margin. Figure 1.14: Active, noncavitated carious lesion at lower second premolar with a typical banana-shape of the white, opaque lesion with the cervical border following the shape of the slightly inflamed marginal gingiva. A 1 mm rim of normal enamel between the lesion and gingiva indicates that the gingivitis, with swelling of the tissue, has been reduced as a result of attempts to control the oral hygiene. Note also the remains of a white opaque lesion on the lower first premolar along the mesial and distal margin of the amalgam filling. On the lower first molar a band of partly discolored, noncavitated lesion extends from an amalgam filling. This could be classified as secondary caries (recurrent caries), but is obviously the remains of a primary lesion. Figure 1.15: Arrested/inactive, noncavitated ("white spot") lesion on the lower first molar. The lesion exhibits a localized circular surface defect. The position of this lesion corresponds to where the marginal gingiva would have been at some stage during eruption of this tooth 30 years earlier. When viewing the lesion from different angles it is apparent that the surface is shiny and smooth, although the tip of a probe will clearly detect the defect (which is also hard). Figure 1.16: Extensive active, white, opaque and chalky buccal lesions which are noncavitated on the upper central incisors. A large superficial defect is seen on the upper right lateral incisor. Notice the obvious difference between the chalky, dull appearance of the carious lesion along gingiva and the creamy appearance of the white, opaque hypomineralized lesion of developmental origin (impaired enamel maturation) on the incisal third of this tooth. If a probe tip is moved gently across the surface, an obvious difference in surface texture is felt between the smooth (and shiny) surface of the developmental defect and the chalky texture of the carious lesion.

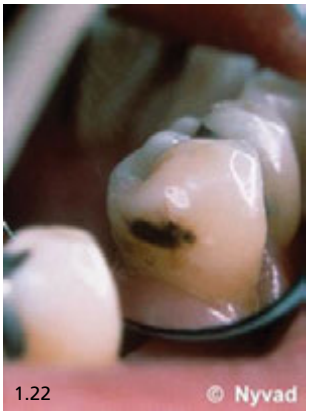


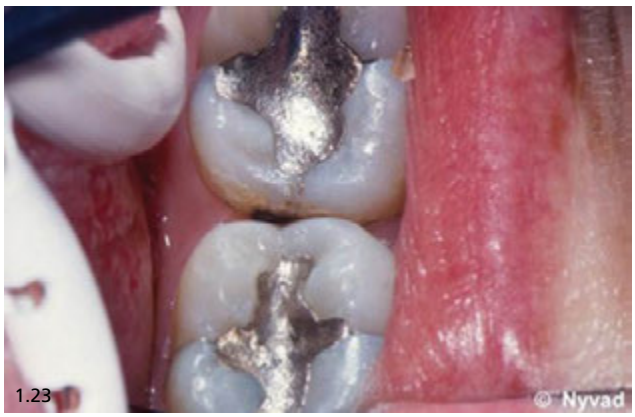




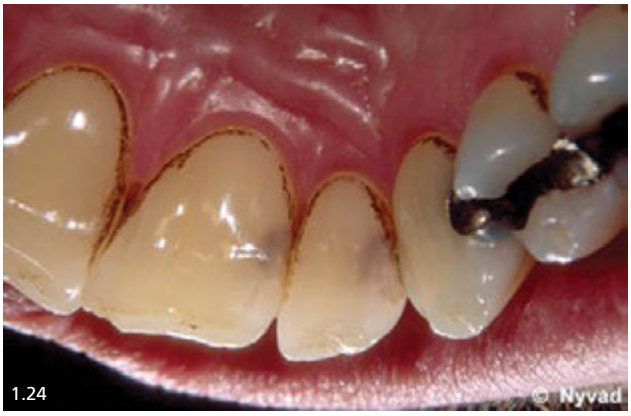


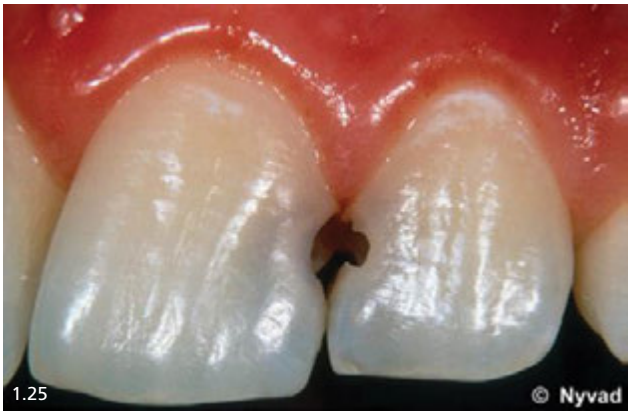


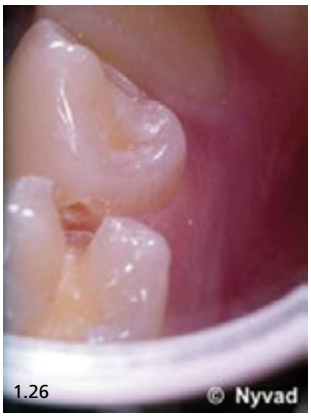


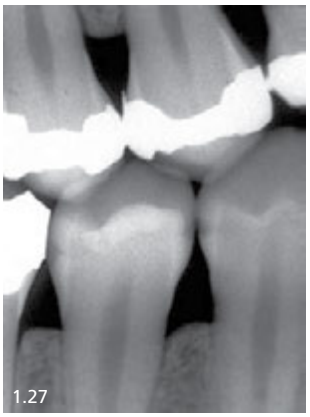


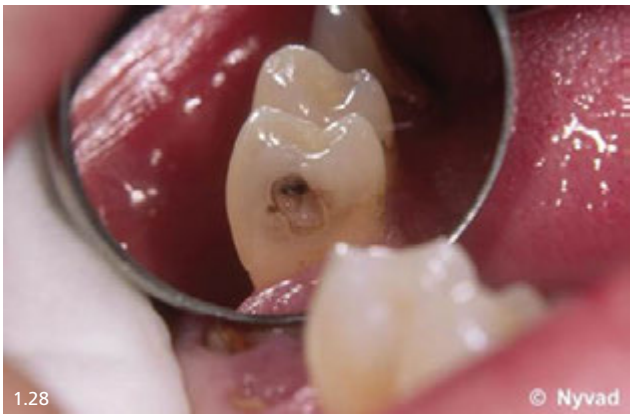
Figures 1.17–1.23 Figure 1.17 and 1.18: Active, noncavitated "early white spot" lesions on mesial surfaces of upper and lower first molars are easily observed following shedding of primary teeth. The shape of each lesion indicates the stagnant areas where the biofilm (dental plaque) remained undisturbed. In the most demineralized areas in the center of the lesions, the porous enamel has taken up stain. The lesion in Fig. 1.17 was treated nonoperatively and has remained as an inactive, noncavitated lesion for almost 35 years! Figure 1.19: Active, discolored lesion on first molar with small cavity containing microbial deposits (dental plaque). Figure 1.20: Different stages of active, cavitated lesions in upper premolars. Note that undermined enamel in the second premolar is reflected by a yellow–whitish translucency of the enamel. Figures 1.21–1.23: Approximal lesions may be difficult to detect by direct visual inspection (Fig. 1.23), but inactive, severely discolored lesions can easily be diagnosed once the neighboring tooth is extracted (Figs. 1.21 and 1.22).



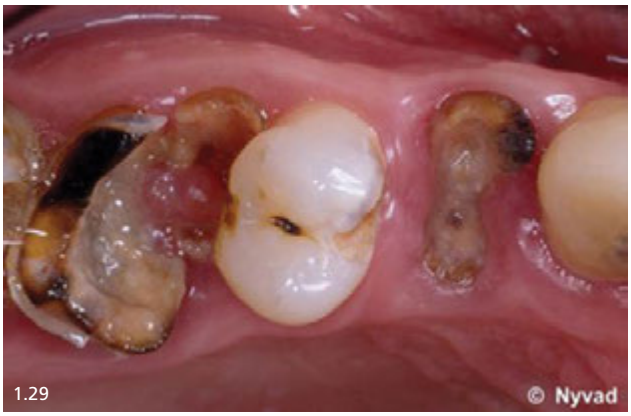


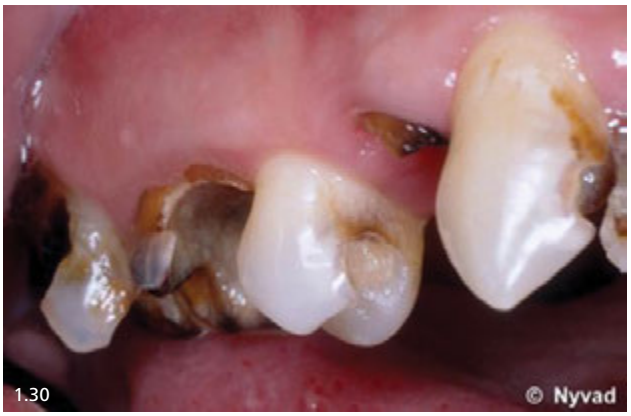




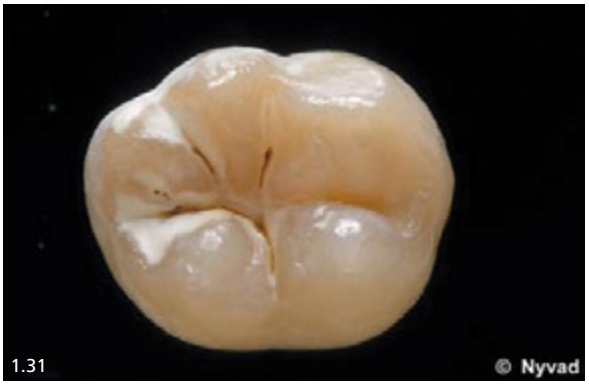


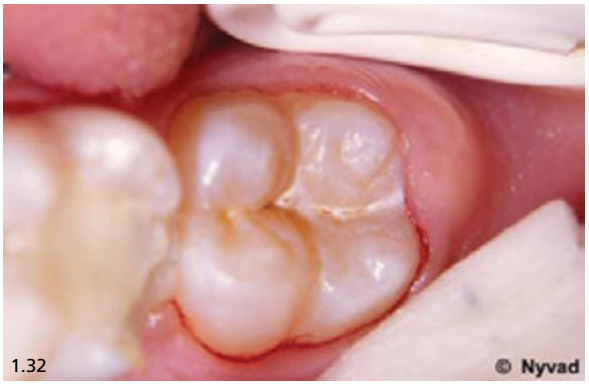
Figures 1.24–1.28 Figures 1.24 and 1.25: In incisors, approximal lesions are easily discerned either directly or by reflected light, as shown in the distal surfaces of the incisors (Fig. 1.24). The cervical black rim of discoloration is a result of cigarette smoking and can be removed by polishing. Figures 1.26 and 1.27: In the premolar and molar regions it is much more difficult to see approximal lesions by direct inspection, even with careful training and experience. In this example the cavity in the first premolar came as a surprise, considering the relatively shallow enamel lesion recorded on the bitewing radiograph – a so-called iatrogenic damage when the dentist was drilling in the neighboring tooth. Figure 1.28: Even extensive active, cavitated lesions can remain difficult to detect until the adjacent tooth is lost. Such lesions may, however, reveal themselves by a bluish or yellowish discoloration of the undermined occlusal enamel ridge – compare with Fig. 1.20.

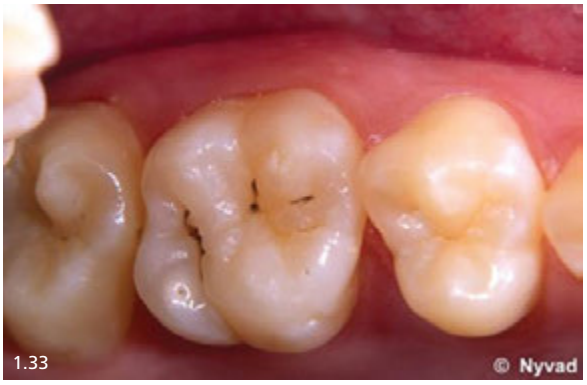


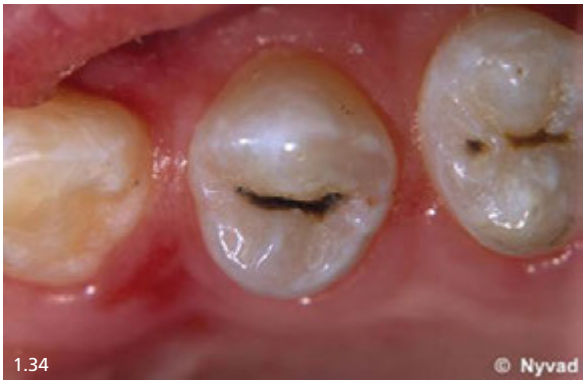


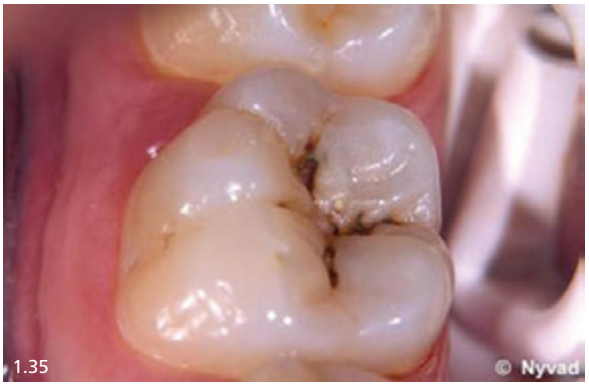
Figures 1.29 and 1.30 Dental caries is a local destructive lesion that, if not controlled or treated operatively, will continue to progress until the entire crown is destroyed and the lesions penetrate further into the root dentin.

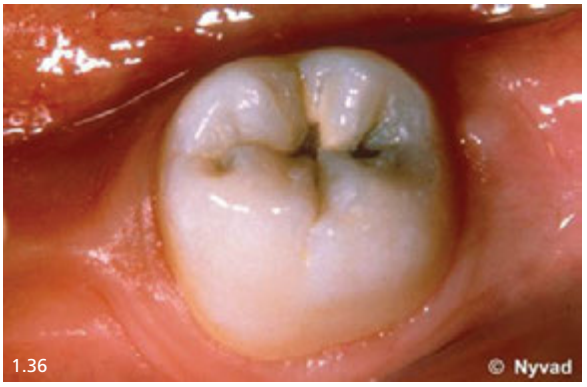


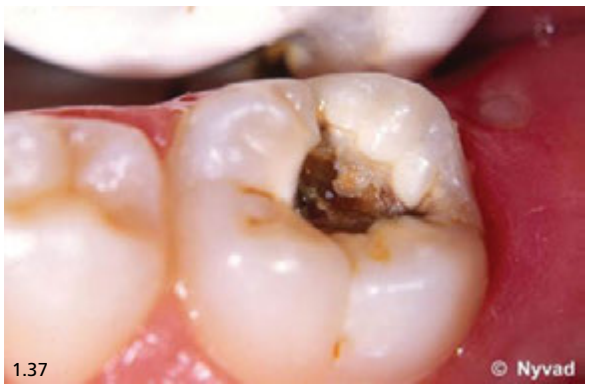


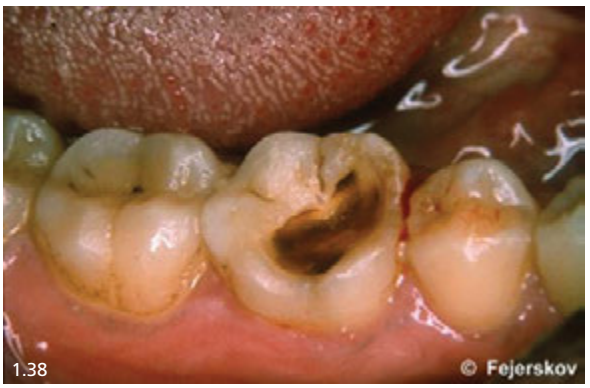




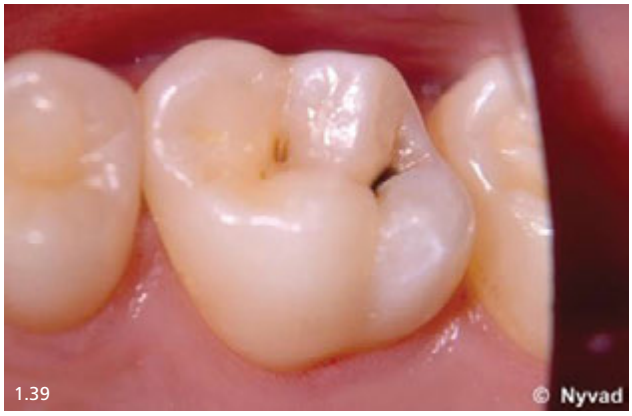


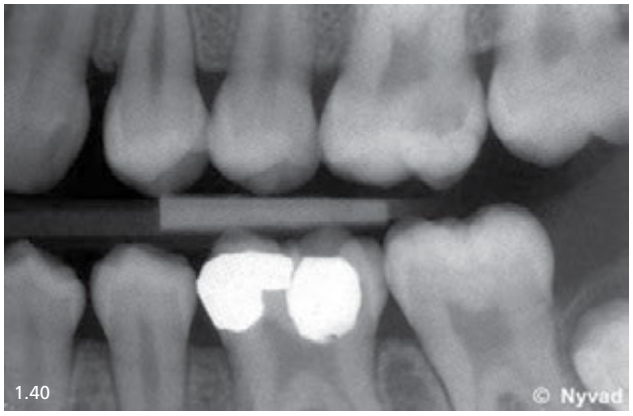


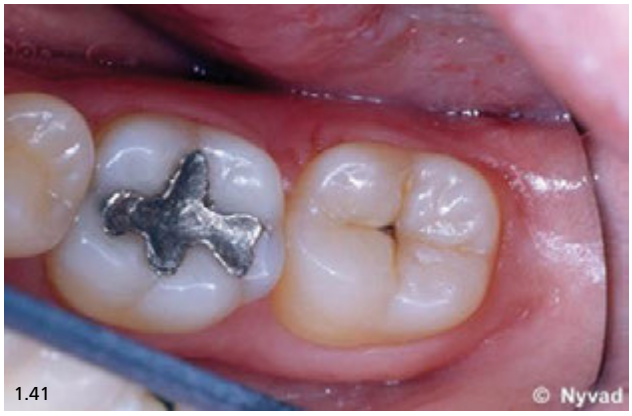


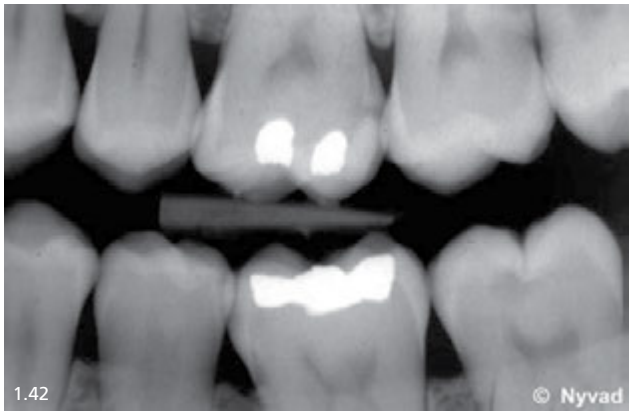


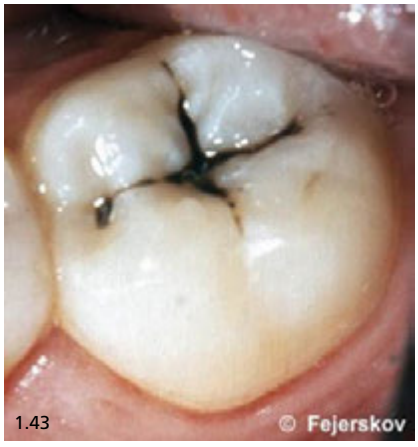
Figures 1.31–1.38 Figure 1.31: Parts of the irregular occlusal surface in molars represent plaque stagnation areas and hence predispose to lesion development. Active, noncavitated lesions appear as chalky white, opaque lesions along the groove, fossa, pits and fissure systems. Figure 1.32: In the clinic the plaque must be removed gently from the occlusal surface either with a brush or explorer as otherwise this active, noncavitated lesion might not be seen. Figures 1.33 and 1.34: Arrested, noncavitated lesions often present as darkly stained pits and fissures. In Fig. 1.34, the cloudy, opaque areas in the premolars with a shiny enamel surface on cusps and enamel ridges represent dental fluorosis. Figures 1.35 and 1.36: Active carious lesions with small and large cavities. Note in Fig. 1.36 how the enamel appears bluish along the fissures as a result of the undermining nature of the occlusal caries lesions. When opened with a bur the occlusal surface is likely to show substantial destruction of the dental tissues. Figure 1.37: Active carious lesion with large cavity extending deep into dentin. Figure 1.38: Arrested occlusal caries lesion. The partly undermined enamel margins have been fractured and abraded away by mastication, and the dental plaque in the dentin cavity has been removed because the surface is in functional occlusion. The dark-brown dentin is hard and painless.

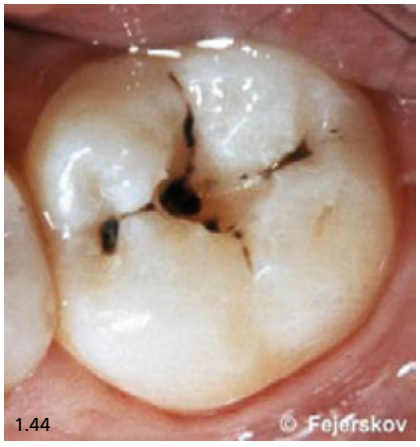


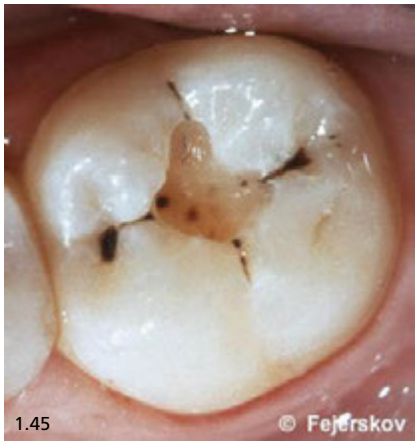




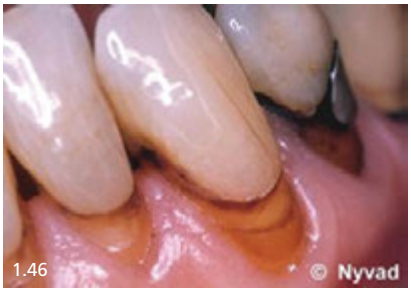


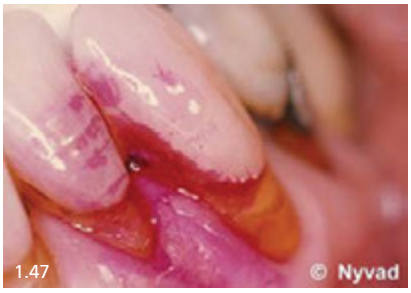


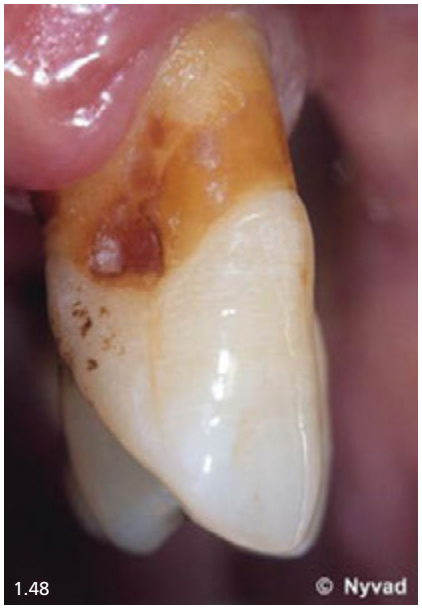


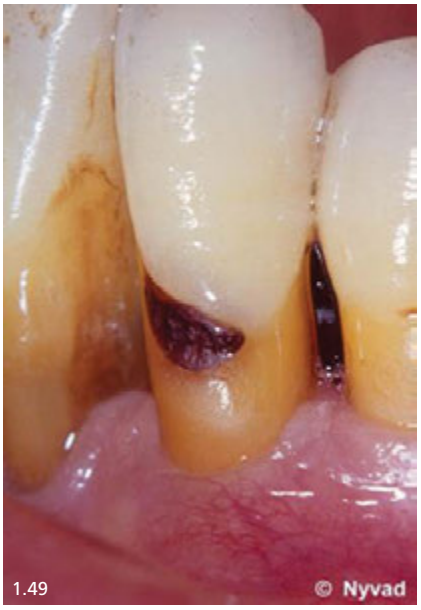


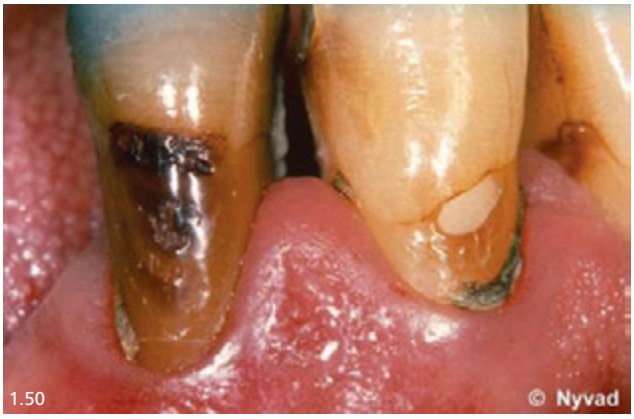
Figures 1.39–1.45 The figures demonstrate lesions that clinicians had misdiagnosed as an arrested lesion and sound. The lesions might be easy to miss unless the tooth surface is absolutely well illuminated and dry. The radiographs in both cases demonstrate extensive radiolucent areas in the occlusal dentin indicative of rather deep carious lesions (Figs 1.40 and 1.42). The bluish appearance of the disto-lingual cusp in Fig. 1.39 should make the clinician aware of a possible undermining larger lesion. Likewise, there is an obvious cavity in the central fossa in Fig. 1.41. These cases represent examples of so-called hidden caries because the dentist had overlooked the clinical signs of lesions and the patient had not complained of any symptoms. The fact that these patients had otherwise very few fillings, and no other signs of active or arrested carious lesions despite being 18–20 years old, probably led the dentists to perform a more superficial dental examination. Figures 1.43–1.45: Example of an inactive occlusal lesion that the dentist assumed to be in need of operative treatment. The lesion in both enamel and dentin was hard on probing and in fact did not extend far into the dentin.

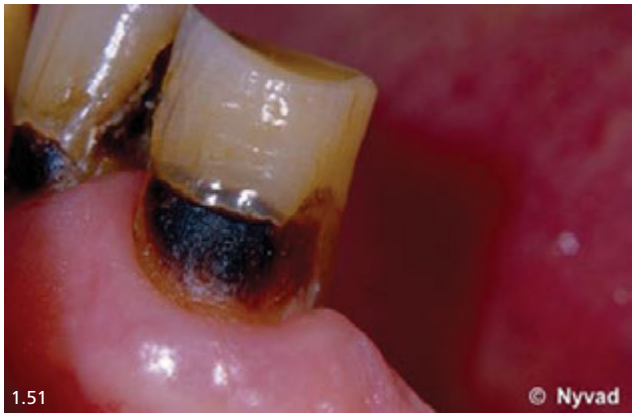




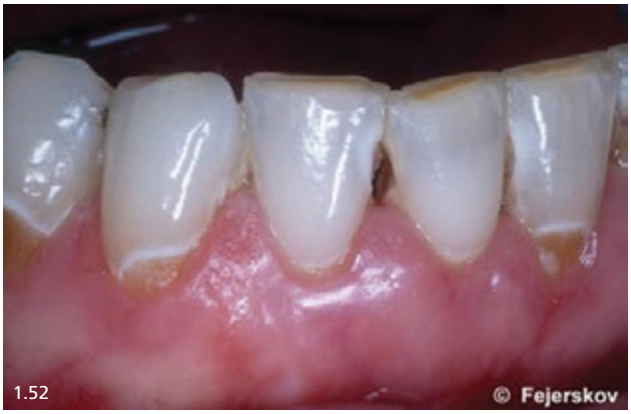


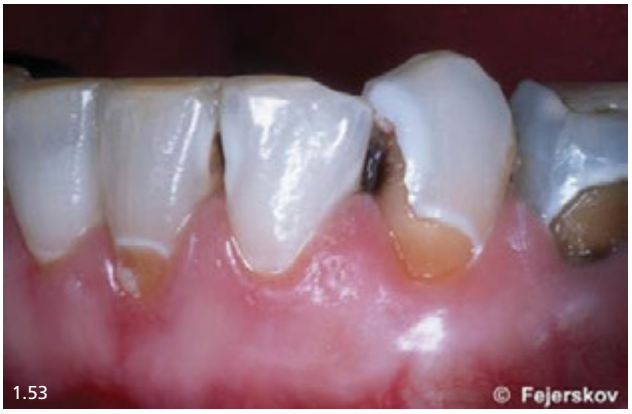


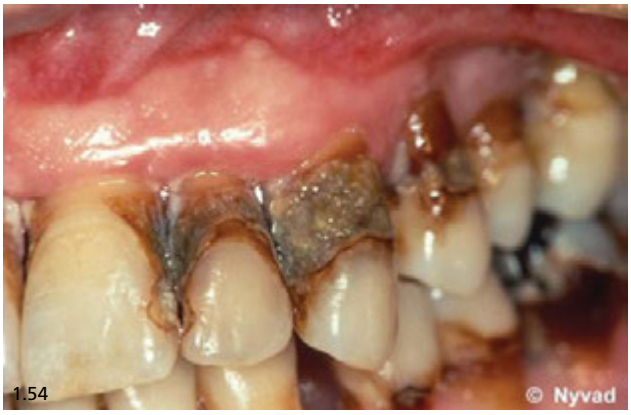


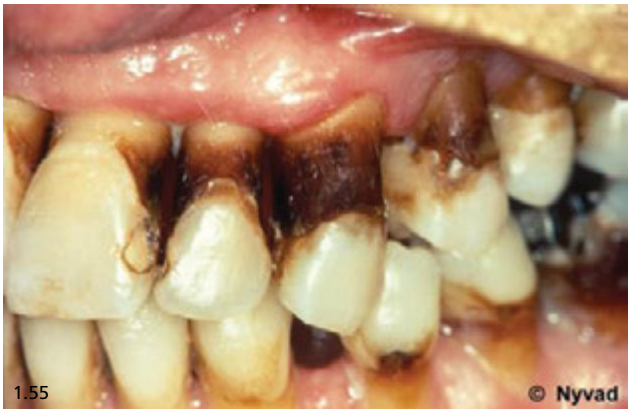


Figures 1.46–1.51 Anywhere on root surfaces where dental plaque accumulates (along the cervical margin at the enamel–cementum junction and along the gingival margin), active root surface lesions may develop with or without distinct cavities. Cavities may be soft (Fig. 1.48) or leathery (Fig. 1.49) and partly filled with microbial deposits. The color of the lesions may vary from yellowish to brownish or black. Figure 1.50: Meticulous oral hygiene can arrest root surface caries lesions and make the root surface appear shiny, although small surface cavities may remain. Arrested root surface lesions feel hard on gentle probing and show a brownish or black discoloration. Figure 1.51: Root surface lesions in the transition stage from active to arrested often exhibit a dull, leathery appearance. Lesion arrest is often a slow process that continues over years. The changes comprise surface abrasion and polishing, as well as mineral uptake (see Chapter 5).

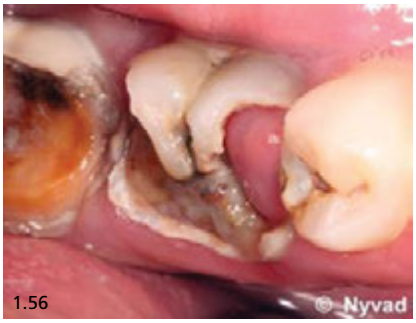


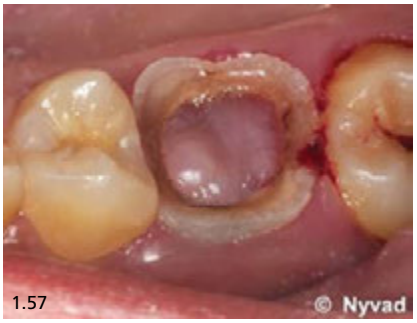


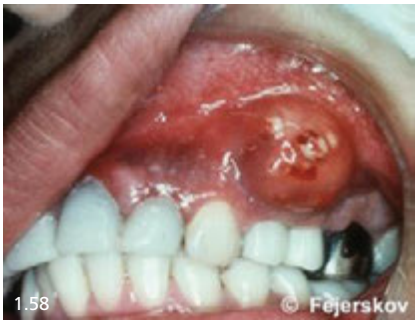


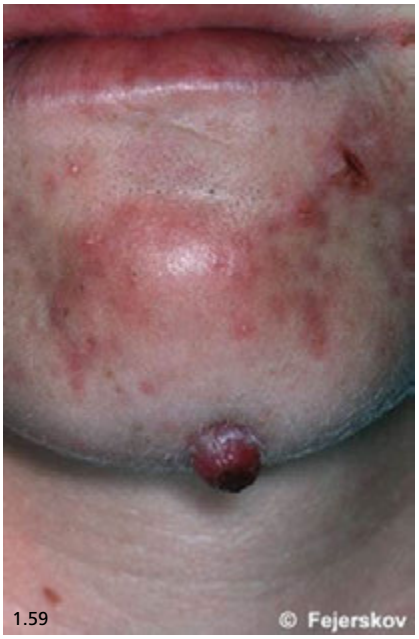


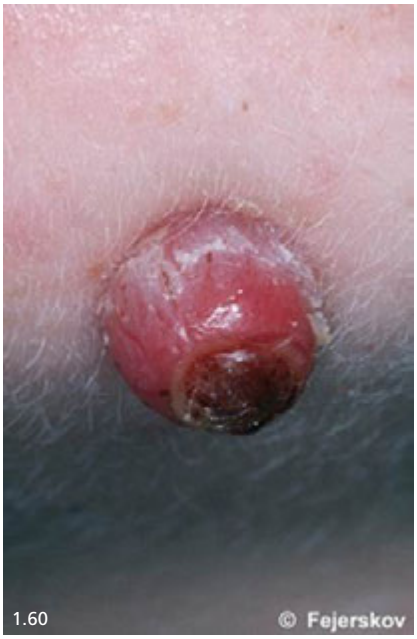
Figures 1.52–1.55 These cases represent a dentist's nightmare! There are extensive active root surface caries lesions. Figs. 1.52 and 1.53 show a patient who has undergone radiation of the head and neck. Although only very small amounts of biofilm can be seen, the lack of saliva results in extensive cervical and approximal active caries lesion. Note how the enamel is undermined along the cavity margins. The patient in Figs. 1.54 and 1.55 had received antidepressants for a long time and presented with heavy soft microbial deposits on all exposed root surfaces. These teeth are very difficult if not impossible to restore. Figure 1.55 shows the patient at 4 months following intensive plaque control with a fluoride toothpaste. The lesions are now mostly arrested. The previously soft surface is leathery to hard, and from a biological point of view restorative dentistry has no role to play. Any restorative treatment would still be difficult, even using contemporary adhesive materials. Restorations might help the patient to improved cosmetics, but they would not contribute to better tooth survival – rather the opposite.

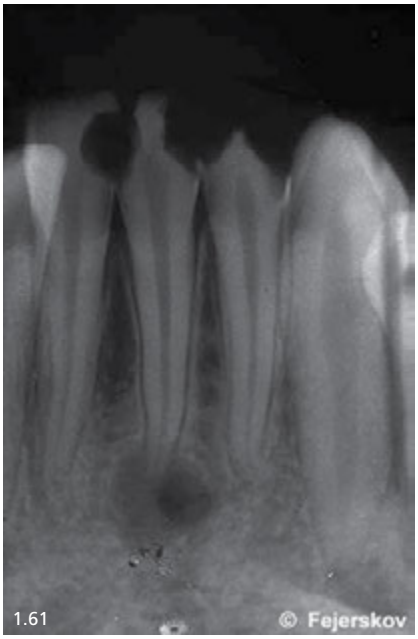












Figures 1.56–1.61 Examples of caries sequelae. The total destruction of the crown of a tooth may result in a local pyogenic granuloma of the gingiva (Fig. 1.56). In Fig. 1.57 the pulpal tissue has survived but is freely exposed to the oral cavity and covered by squamous epithelium (pulpal polyp). Most often, untreated dental caries results in necrosis of the pulp and development of a periapical abscess that may penetrate the bone to the oral cavity (Fig. 1.58) or in rare cases even directly to the surface of the skin (Figs. 1.59–1.61). The abscess from the lower central incisor has penetrated the mandible and pus is emptied regularly through a fistula. As long as the duct of the fistula remains open there is hardly any pain.

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2

Strategic public health considerations for caries control in populations

Anja Heilmann and Richard G. Watt

Introduction

Dental caries is a global public health problem. Despite being preventable, analysis in the 2022 World Health Organization's (WHO) Global Oral Health Status Report shows that more than a third of the world's population now suffers with untreated caries [123]. The condition affects all ages across the lifecourse from early childhood to old age. Globally, it is estimated that 514 million children have untreated caries in their deciduous dentition and over 2 billion young people, adults, and older people have caries in their permanent teeth [123]. The prevalence and severity of dental caries are socially patterned – poorer, less educated, and vulnerable groups disproportionally suffer from the disease. The extraordinarily high prevalence of dental caries and stark inequalities across the globe are a damning indictment of the failure of the dental profession and modern dentistry to effectively prevent and treat this condition. Dental caries is caused by the consumption of free sugars – yet globally, the dental profession has given only limited attention to public health measures to reduce sugar consumption. Instead, dentistry has been trapped in a biomedical treatment paradigm that concentrates all its efforts on restoring carious teeth – this outdated restorative approach will never succeed in tackling the global burden of dental caries especially in low- and middle-income countries where the costs of modern dentistry are beyond the budgets of healthcare systems and the majority of the population [27, 110] and where an acute shortage of clinical dental personnel exists. A radically different approach to tackling this global public health problem is now needed.

Oral health is at a tipping point. At a global level, the public health importance of oral diseases including dental

Dental Caries: The Disease and Its Clinical Management, Fourth Edition. Edited by Ole Fejerskov and Bente Nyvad.

caries is now being recognized by organizations such as the World Health Organization, which has published a Global Oral Health Strategy outlining six strategic objectives to reduce the global burden of oral diseases by 2030 [126]. In this WHO strategic document, the limitations of the current clinical approaches to caries treatment are highlighted and the need for an alternative public health approach is stressed. In this chapter, the authors will present an overview of public health principles and approaches for the prevention and control of dental caries. Rather than focusing efforts on an individual clinical level, public health is concerned with understanding the underlying causes of diseases at a population level and implementing population-level integrated policies and interventions to effectively tackle disease burdens and health inequalities.

The global burden of dental caries

Global prevalence and trends

Untreated caries of the permanent teeth is the most common of all chronic non-communicable diseases worldwide. According to the Global Burden of Disease study, an estimated 2.3 billion people were affected in 2017, with an age-standardized prevalence of around 29%. In addition, caries of deciduous teeth affected more than 500 million children globally [30], corresponding to a global prevalence among children aged 1–9 years of 43% [123].

Between 1990 and 2019, global prevalence has slightly decreased; however, this is masking sharp increases in absolute case numbers in low- and lower-middle-income countries due to population growth [123].

Impact

Dental caries is not a harmless disease. Its sequelae include pain, infection, and tooth loss, which affect fundamental oral functions such as eating, speaking, and smiling, as well as facial appearance. As such, the state of someone's teeth is bound up with their sense of identity, self-esteem, and overall quality of life. Both quantitative and qualitative studies on the link between dental caries and oral health-related quality of life have shown that caries-associated pain can cause functional and psychosocial limitations, such as difficulty eating a varied diet, sleepless nights, and negative impacts on confidence and social activities [1, 32, 52, 133]. In children, dental caries has been linked to poorer school performance and attendance [76], and can even affect growth and development [1, 22, 90].

With an estimated $245 billion (including $161 billion in direct treatment costs and $84 billion in indirect productivity losses), the global economic costs of dental caries and resulting tooth loss are immense [101]. In the UK, tooth extraction under general anesthesia due to dental caries is the main cause of hospitalization among children aged five to nine years [82].

Inequalities in dental caries

The socially graded distribution of oral diseases is a universal finding in epidemiological studies across countries and age groups, and social inequalities in dental caries are particularly stark. Those who are socially more advantaged, whether in terms of income, education, social class, neighborhood deprivation, or other indicators of socio-economic position, are less likely to have untreated caries and on average have fewer decayed, missing, and filled teeth [89]. Indeed, dental caries among children has been described as a 'canary in the coalmine' – a marker of early life disadvantage and possible precursor for the accumulation of further risks affecting health over the lifecourse [109]. Inequalities also exist between racially minoritized and white people [12], and affect people living with disabilities and vulnerable groups such as refugees, prisoners, and people who are homeless [108]. Despite the overall decline in caries levels in many high-income countries since the introduction of fluoride toothpaste, caries burdens are still high, and social gradients in caries persist. These inequalities are not inevitable; they are socially produced, potentially avoidable, and therefore considered unfair and unacceptable [113]. The causes for the unequal distribution of health and oral health have been described as the social determinants of health and are further explained below.

The role of sugar in the etiology of dental caries

Dental caries is a preventable disease. The main cause of caries is excessive sugar consumption [63, 92]. Factors such as salivary flow and exposure to fluoride are important effect modifiers as they inhibit the caries process; however, their absence does not in itself cause caries – for caries to occur, sugar must be present [70]. From a lifecourse perspective, there is evidence that the timing of the first introduction of sugar to children's diets is important, with earlier introduction linked to an increased caries risk [15, 23].

The WHO classification of sugars distinguishes between intrinsic sugars naturally present in fresh, intact fruits and vegetables; milk sugars (lactose) that are naturally present in milk and milk products; and free sugars (Fig. 2.1). Only free sugars are considered cariogenic. WHO defines free sugars as 'sugars added to foods or beverages by the manufacturer, cook or consumer; as well as sugars naturally present in honey, syrups, fruit juices and fruit juice concentrates' [119]. In 2015, WHO published an evidence-based guideline on the intake of free sugars for adults and children [119]. The guideline was informed by two systematic reviews specifically commissioned for this purpose, which summarized the epidemiological evidence on the role of free sugars in the development of dental caries [63] and unhealthy weight gain [95]. In view of this evidence, the WHO recommends a reduced intake of free sugars throughout the lifecourse (strong recommendation), and for adults and children older

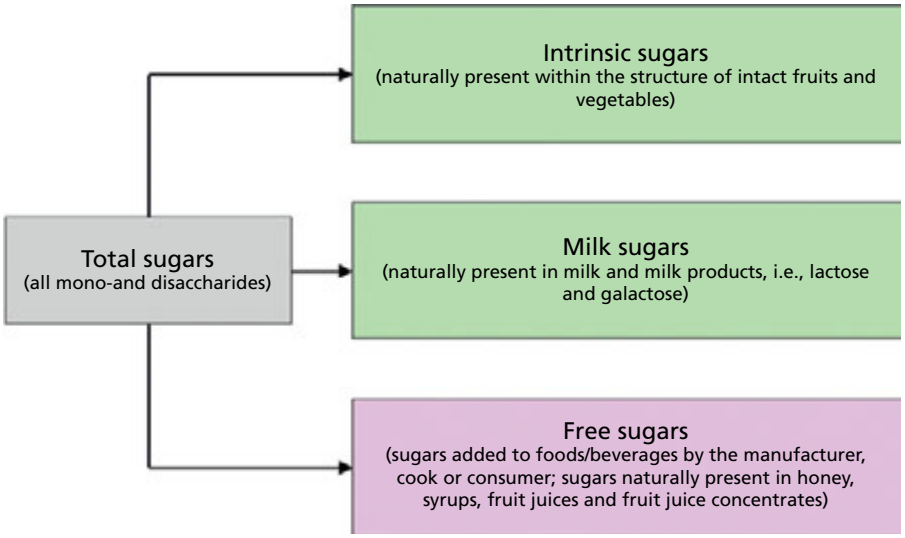

Figure 2.1 WHO classification of sugars [34, 119].

than two years, to restrict intake of free sugars to no more than 10% of total energy (strong recommendation). A further reduction to less than 5% of total energy intake is suggested (conditional recommendation) [119].

It is important to stress that fresh fruits and vegetables are an important part of a healthy diet and should be encouraged by all health professionals including dentists. The intrinsic sugars contained in intact, fresh fruits and vegetables are not a cause of dental caries [62].

Important dietary sources of free sugars

While dietary patterns vary between countries, there are some types of dietary products that are of particular concern for health. A key source of free sugars in the global diet is ultra-processed foods and drinks. These are products that are industrially produced, cheap, convenient, highly palatable, and energy-dense, while often lacking in nutritional value. They contain a large number of ingredients including derivatives and additives, and high amounts of free sugars. Ultra-processed food and drink products include sugar-sweetened beverages (SSB), breakfast cereals, confectionary, and many commercial baby food and drink products [59]. SSBs are the largest source of dietary sugars in many countries [19, 50] and a major public health concern. Apart from their role in the development of dental caries [100], SSBs have also been causally linked to unhealthy weight gain, diabetes, and cardiometabolic diseases [50].

What influences free sugar consumption?

Evolution and the nutrition transition

Millions of years of human evolution has created humans who are 'hard-wired' to crave sweet, fatty, and energy-dense foods. From an evolutionary perspective, this makes sense: for our hunter-gatherer ancestors, these preferences conferred a selective advantage because they increased the chances of survival and therefore reproduction [65]. However, the nutrition transitions undergone by middle- and high-income countries over the second half of the twentieth century, which are now also rapidly occurring in lower-income countries, mean that there has been a shift from traditional diets that were rich in legumes, vegetables, and coarse grains toward so-called 'Western' diets characterized by an abundance of sugary, fatty, and ultra-processed foods that are cheap and highly palatable [71]. These dietary changes have resulted in a mismatch between our inherent preferences and a modern environment in which these preferences leave us susceptible to chronic diseases. This mismatch is the main reason for the sharp rise in the global prevalence of chronic non-communicable diseases seen over the past few decades [21, 71].

Recognizing that preferences for sweetness are driven by evolution, and therefore innate, helps us to understand why they are so powerful. Indeed, most decisions about food are automatic and made subconsciously prompted by environmental cues, whereas for most people, resisting sweet foods and drinks requires conscious effort and discipline [103]. For health professionals aiming to reduce the burden of dental caries and other sugar-related diseases, these considerations highlight the importance of creating supportive environments where healthy options are the default. They also explain why behavior change interventions that rely on health education alone have had only limited success [104, 132].

Social determinants of sugar consumption

Sugar consumption is one of several health-related behaviors often referred to as 'lifestyle choices'. The term 'lifestyle' implies behaviors that are freely chosen. However, choices about sugar consumption or indeed any other health-related

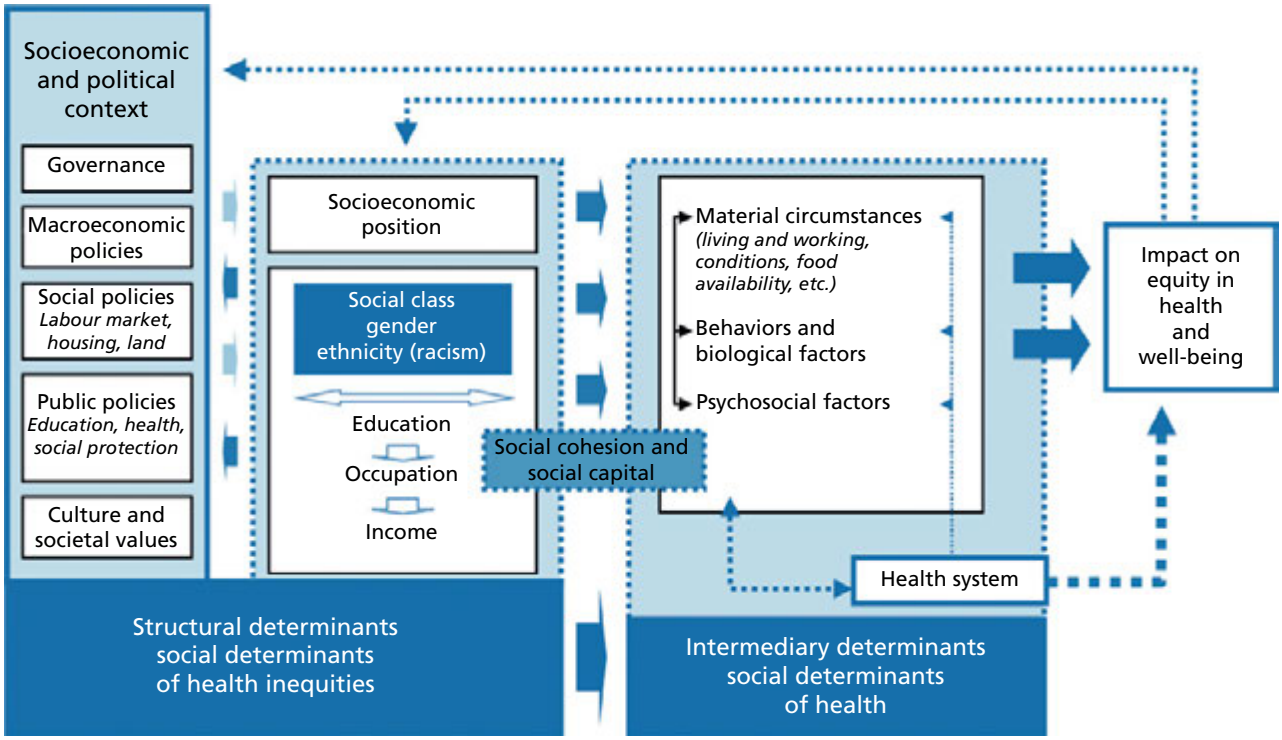

Figure 2.2 WHO conceptual framework for action on the social determinants of health [93]/with permission of WHO.

behavior are not made in a vacuum – they are shaped by a range of environmental factors at different levels, which are largely outside the control of individuals. These factors are known as the social determinants of health and explain the social inequalities in the distribution of health and disease observed in almost all societies [93]. The WHO conceptual framework for action on the social determinants of health (Fig. 2.2) illustrates how structural aspects of the wider society and social stratification influence so-called intermediate determinants, including behaviors that are interlinked with material circumstances and psychosocial factors. These intermediate determinants influence health outcomes and the extent of health inequalities [93].

Applied to sugar consumption, structural determinants include governance and economic policies, which regulate production, trade, marketing, pricing, and local availability of sugary products. At an individual level, choices are shaped by social norms and restricted by affordability and access to healthy, as well as unhealthy options [116]. Psychosocial factors such as stress also play a role in sugar consumption [40].

Sugar as a commercial determinant of oral health

The commercial determinants of health are a more recent concept that has been introduced in recognition of the significant impact that for-profit corporations have on the health of populations in a globalized world. They include activities by the tobacco, alcohol, and food industries, but also industries that threaten human and planetary health by causing pollution and climate change, among others [29]. Commercial determinants of health have been defined as the 'strategies and approaches used by the private sector to promote products and choices that are detrimental to health' [41]. They exert their influence mainly through marketing, lobbying, corporate 'social responsibility' strategies (activities that improve the public image of a brand), and their market penetration and global supply chains [41].

The economically powerful and politically influential global sugar industry is a prime example of how commercial influence adversely affects population health. Sugar is cheap and profit margins are high. The strategies used by the sugar industry to promote and sell their products are similar to those used by manufacturers of other harmful products, such as tobacco and alcohol, and have been described and documented [17, 69]. Table 2.1 provides an overview of common industry tactics with relevant examples. The advertising and marketing of sugary products are relentless and pervasive, shaping norms and wants. Of particular concern is the marketing of high-sugary products aimed at children. A UK study surveyed the sugar content of fruit juices and smoothies specifically marketed toward children and often advertised as part of a healthy diet. The study revealed that more than 40% of products contained a child's entire maximum daily recommended amount of free sugars [14]. There is evidence from a systematic review of randomized trials that exposure to advertising of unhealthy foods and drinks is effective in that it alters children's preferences and increases intake of the advertised products [83]. Apart from direct marketing,