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When and How to Intervene in the Caries Process

H Meyer-Lueckel • S Paris

Clinical Relevance

In addition to minimally invasive interventions, noninvasive and microinvasive options should be a focus in modern cariology in order to preserve dental hard tissues to a greater extent, as has been the case in recent decades.

SUMMARY

The decrease in caries prevalence in many industrialized countries and the improved knowledge about the etiology and pathogenesis of caries have shifted the focus of caries therapy over the past decades toward less invasive approaches. Studies on caries progression indicate that it is generally quite slow in most patients today which should lead to a reconsideration of the practice of early invasive intervention. Today noninvasive (eg, fluorides) and microinvasive (occlusal sealing, proximal infiltration) therapeutic options that address etiological factors are gaining importance. The goal of these therapies is to heal or at least to slow down the progress of the disease. Noninvasive treatments are mainly related to controlling pathogenic factors (ie, sugar consumption) and enhancing protective factors (mainly oral hygiene and fluorides). Microinvasive treatments do not rely on the compliance of the patient as much, since these treatments include a resinous material that is applied to serve as a diffusion barrier for acids formed by cariogenic bacteria in the overlying plaque. To establish a minimum intervention treatment strategy for caries, the disease must be diagnosed at an early stage. In addition to assessing caries lesions in single teeth, individual risk factors need to be identified so that the underlying causes related to patients' behavioral patterns that led to the disease can be addressed as well. The patient should be informed about the scientific evidence related to the treatment choices in a participative atmosphere. Decision trees may help to make the range of findings comprehensible and the therapeutic shared decision-making process understandable to the patients.

*Hendrik Meyer-Lueckel, professor, RWTH Aachen University, Department of Operative Dentistry, Periodontology and Preventive Dentistry, Aachen, Germany

Sebastian Paris, professor, Charité – Universitätsmedizin Berlin, Department of Operative and Preventive Dentistry, Berlin, Germany

*Corresponding author: Pauwelsstr. 30, Aachen, 52074, Germany; e-mail: hmeyer-lueckel@ukaachen.de

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INTRODUCTION

Minimum intervention has been proposed as the primary aim of modern caries therapy.[1] To fulfill this strategy, the fundamentals of caries histology and pathogenesis need to be considered. The thought behind the contemporary (caries) model helps us to understand the underlying causes and associated factors involved in the disease process. A feasible way to detect, assess, and document relevant disease stages as well as the individual's caries risk is warranted. Based on the derived diagnoses, several possible treatments at the tooth as well as the

patient level need to be weighed and explained to the patient before shared decision making can become possible.

Nonetheless, interpretation of current knowledge and diagnostic outcomes as well as the success rate of the various treatment options does not seem to be uniform throughout the world. This is reflected by the radiographic stages at which dentists from various countries intervene invasively in proximal caries. In France, a restoration is inserted by almost 90% of the dentists when the radiograph reveals that the caries extends from the outer enamel up to the dentin-enamel junction.[2] In the United States, fewer dentists seem to restore these lesions that early (40%-75%) but this is still much more often than Scandinavian dentists (<10%).[3-5] It is similarly difficult to choose an appropriate therapy for occlusal and cervical carious lesions (root caries).

In fact, only through longitudinal clinical and radiographic monitoring of the caries progression process of a single caries lesion might the progression of the lesion be forecasted reliably. However, this assumes that:

- the dentist and the patient are convinced that caries is a process that can be arrested, at least in its early stages;
- the patient is compliant with regular checkups;
- caries can be objectively detected, assessed, and documented in order to compare different points in time; and
- the diagnostic findings will be transferred when the patient changes dentists, such that no information is lost.

In addition to the biological, diagnostic, and patient-centered (compliance) limitations, the patient's expectations are also relevant when following the principles of minimum intervention. As discussed before, invasive procedures are frequently viewed by many healthcare providers and by patients as well, as the appropriate method by which to manage the caries process. These procedures are consequently honored, be it psychologically (the dentist who drills is a good dentist, because he or she treats the disease actively) and/or financially. "Wait and watch" noninvasive therapy, which is largely based on self-management, is viewed with a certain amount of skepticism, as are microinvasive procedures. Frequently, the patient and even the dentist are afraid of the uncontrollable, rapid progression of caries lesions in the early stages of the process. In addition, dentists fear leaving microorganisms within the tooth after sealing, caries infiltration, or restoration. These considerations often lead the dentist to intervene prematurely with invasive treatment and to extensively excavate the dentin when preparing the cavity.[6]

The current article aims to provide a concept for everyday practice in order to preserve dental hard tissues by three basic treatment options: noninvasive, microinvasive, and invasive therapy for the various sites of caries as long as possible.

PARADIGM SHIFT IN CARIOLOGY

Medical and dental interventions should be determined by the underlying scientific paradigms that guide our understanding of diseases. Since the mid-20th century, the paradigm of the specific plaque hypothesis led to a focus on specific microorganisms that inoculate our biofilms,[7] which was misinterpreted as an infection, although Koch's postulates for infectious disease never applied for caries. Nonetheless, generations of dentists adjusted their preventive and therapeutic efforts with the aim of eradicating (specific) microbes.

In contrast, the contemporary paradigm relies on the ecological plaque hypothesis that explains caries as a disturbance in the homeostasis of the oral microflora,[8] which is caused by the selective favoring of (potentially) pathogenic microorganisms by a sugar-rich diet. It is assumed that it is not an exogenous infection with pathogenic species that is responsible for caries. Rather, these species are a part of the physiological (endogenous) flora in healthy humans, and only the qualitative and quantitative changes are pathological.[9] The increased consumption of fermentable carbohydrates favors microorganisms that efficiently metabolize these sugars into organic acids (acidogenic) and also tolerate the resultant low pH (aciduric).

A CURRENT CARIES MODEL

The etiology of caries has been described in various models.[10-12] The well-known Venn diagram by Keyes was based on the three essential etiological factors for caries: "bacteria," "tooth," and "sugar,"[10] but for reasons of simplicity other influencing factors were not shown. The model introduced by Fejerskov and Manji,[11] in contrast, shows caries as a multifactorial disease but elucidates the pathogenesis only to a small extent. Recently, we proposed a pathogenesis model of caries based on the ecological plaque hypothesis (Figure 1).[13] According to present understanding, a sugar-rich diet plays a primary role in the etiology and pathogenesis of caries.[14] A greater

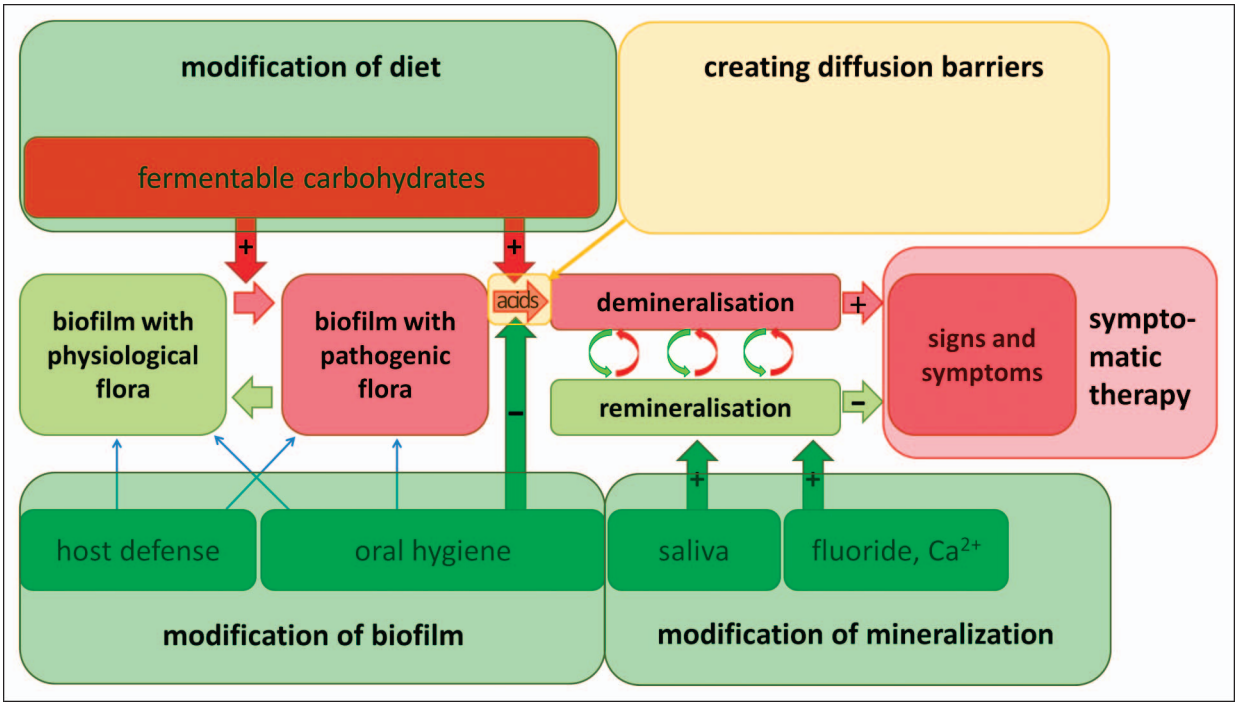


Figure 1. *The pathogenesis of caries (first published in Paris and Meyer-Lueckel[13] and Paris and others[19]). The primary causal pathogenic factor (dark red) for caries is the frequent consumption of fermentable carbohydrates (sugars). This causes an ecological shift in the oral biofilm, with a selection of acidogenic and aciduric species (pathogenic flora). The increased metabolic activity of the biofilm, which is also triggered by sugar consumption, causes the formation of organic acids. This leads to demineralization of dental hard tissues, which consequently results in the characteristic signs and symptoms of caries. On the contrary, there are several protective factors (dark green): the host defense (immune system) and the oral hygiene limit the number of microorganisms. By oral hygiene the biofilm can be completely removed at least locally, and thus acid formation can be avoided. In addition, the components of saliva as well as locally applied fluorides and calcium enhance the remineralization of dental hard tissues, which may alleviate the signs and symptoms of caries. In addition to these (local) direct factors there are several indirect (distant) factors, which only indirectly influence the caries process but may be strongly associated with caries. For simplicity, not all associations are marked. Preventive strategies address the causal factors of the caries process. By creating a diffusion barrier or specifically modifying nutrition, the biofilm, or mineralization, the caries process is altered to inhibit demineralization and promote remineralization. Symptomatic (restorative) therapy is contrastingly restricted to alleviating the clinical signs and symptoms.*

role has been assigned to sugar because caries is a disease of civilizations that consume a greater amount of sugar, which was not the case throughout most of human history.[15] The excessive consumption of fermentable carbohydrates appears to be less physiological or 'normal' than the regular existence of small amounts of potentially cariogenic bacteria in the physiological flora. The frequent consumption of fermentable carbohydrates causes a pathological shift in the oral microflora and promotes acidogenic and aciduric species.[8] Consuming fermentable carbohydrates also causes potentially cariogenic bacteria (eg, *Streptococcus mutans* but also many others) to produce organic acids that demineralize the enamel and dentin. This ultimately causes the characteristic signs and symptoms of caries.

Protective factors also influence the development of caries. Both the host's defenses and the patient's oral hygiene limit the growth and metabolism of the oral biofilm and hence the production of acids. With its buffering properties and minerals, saliva promotes the remineralization of the enamel. The remineralizing effect of saliva can be supported by the application of fluorides and presumably with calcium compounds.[16]

In addition to these local, direct factors, other behavioral and socioeconomic factors are associated with caries, as revealed by epidemiological investigations.[17] However, these only indirectly influence the caries process through the local factors. With the exception of the consumption of fermentable carbohydrates as a pathogenic factor, all of the other factors that locally influence the caries process are shown as protective factors. However, the minimization or elimination of protective factors can have a significant influence on the caries process. For example, the elimination of saliva's protective function in patients with hyposalivation frequently causes caries to progress extremely quickly, even though other factors are scarcely modified.[18] The

risk factors for caries are accordingly increasing the pathogenic factor of fermentable carbohydrates as well as eliminating or reducing protective factors.

According to our present understanding, caries is a multifactorial disease. If and how fast caries develops depends on the complex interrelationships between the various pathogenic and protective factors. If the protective factors predominate, caries does not develop, or existing caries is arrested or healed. However, if the pathogenic factors predominate, the disease progresses.[12] This dynamic character of the caries process enables the disease to be influenced in every stage. Approaches for preventing caries therefore seek to minimize the pathogenic factors and support the protective factors.

CONSEQUENCES OF THE PARADIGM SHIFT IN CARIOLOGY ON THERAPY

The Traditional View: "Drill & Fill"

The "classic" caries therapy was based mainly on invasive interventions. In accordance with the predominating specific plaque hypothesis, for many years the aim of restorative therapy was the complete excavation of all infected tissue and restoration of the resulting defect. Cavity design followed Black's understanding of cavity form[19] that is frequently expressed by the term "extension for prevention." Accordingly, the goal was to prepare a cavity that offers a sufficient degree of retention for the then-available nonadhesive materials (including cement, amalgam, and gold) and thereby to remove "all" carious tissue. The margins of the restoration were to lie in areas of the tooth that are easily accessible to oral hygiene to prevent the formation of adjacent caries. This meant that all occlusal fissures were included and that the proximal box was extended very widely. This philosophy, summarized by the expression "drill & fill," yielded an invasive treatment strategy that was expensive, possibly painful, and, from an epidemiological perspective, resulted in high DMFT values.[6]

A New Philosophy: "Heal & Seal"

According to the ecological plaque hypothesis, the caries process can be arrested if the factors that promote caries are reduced and/or if counteracting protective factors are performed.[20] As described recently,[6] in most cases it takes several years to even a decade for proximal dentin caries to become detectable on a radiograph.[21] Consequently, there exists a considerable amount of time until the "right" moment for (minimally) invasive caries therapy is reached.

If the tooth surface is easily accessible and the patient is compliant, enamel and dentin caries lesions (root caries) can frequently be arrested by noninvasive measures alone. The specific measures that are chosen depend on the frequency of use and the patient's risk of caries. The probability of arresting a caries lesion solely through noninvasive measures decreases as the extent of the caries and cavitation increases. Correspondingly, a caries lesion tends to progress at a greater rate when it has clinically identifiable cavitation,[22] which offers a favorable milieu for microorganisms. Comparable caries-promoting conditions also exist in deep fissures and grooves as well as in the marginal gaps of restorations.

The adhesive filling materials and techniques that have been used for decades enable caries to be treated invasively with less destruction of enamel and dentin than is associated with metal and cements. However, the belief that infected dentin needs to be completely removed remains widespread, although it is becoming increasingly doubtful whether complete removal of bacteria is possible or even necessary, especially since radical caries excavation increases the danger of exposing the pulp,[23] with more pulpal complications as a consequence. With adhesive fillings, the substrate supply to microorganisms deep within the cavity is inhibited, the access of other microorganisms remains blocked, and the remaining microorganisms are sealed in. At the same time, this therapeutic measure (again) enables the patient to clean the related tooth surface. The influence of the dental biofilm, the driving force behind the caries process, is thereby reduced.

A similar condition is achieved by sealing plaque-retentive occlusal tooth surfaces that have an elevated risk of caries. In addition to sealing healthy fissures, in particular when the tooth is erupting, it is also recommended to seal initial caries lesions.[24,25] Noncavitated caries lesions on smooth and proximal surfaces can be sealed[26,27] in principle; however, caries infiltration has certain advantages over sealing in this case.[28,29]

FROM DIAGNOSTICS . . .

As argued in our textbook,[30] before therapy is undertaken, a diagnostic process should be followed thoroughly. First, the signs and symptoms characteristic of caries need to be identified (detection).

Table 1: *Therapeutically Relevant Diagnoses of Caries (Taken from Paris and others[19])*

	Inactive caries *(caries non-progressiva)*	**Sound, but at increased risk**	**Active caries** *(caries progressiva)*		
			early *(superficialis)*	**medium** *(media)*	**late** *(profunda)*
Clinical findings	All ICDAS stages , but inactive	0	ICDAS 1-2 (active)	ICDAS 3-4 (active)	ICDAS 5-6 (active)
Radiographic extension	Mainly E0 E1, E2, D1	0	E0, E1, E2, D1	D1, D2	D2, D3
DIAGNOdent values	Mainly <50	<15	0-40	20-99	n/a
(most likely) Therapy	none	noninvasive or microinvasive		minimally invasive	invasive + pulp preservation or endodontics

This is followed by a precise description of the (severity) stage and activity of caries.[31,32] Several methods can be used to detect and describe caries that will produce both confirmatory as well as contradictory information. The findings are then combined to form a diagnosis, which is used to select a therapy.[33,34] The therapeutic decision involves two elements: whether the disease needs to be treated and which therapy is appropriate.[35]

The art of diagnosing consists of combining the various findings into a diagnosis on which to base the ideal therapy for the patient and his or her disease. The different bits of information that have been collected (findings) are weighted, interpreted, evaluated, and then assembled into a coherent picture for a diagnosis. What is frequently a highly complex clinical situation needs to be simplified and explained using a reasonable number of categories (diagnoses). Thresholds need to be defined for the individual categories that delimit the categories from each other. A variety of systems were developed over time for categorizing clinical and x-ray findings.[36] In our view, it is useful to draw a distinction between active and inactive lesions, since only the former require therapy.[37] In addition, a distinction is frequently made between noncavitated and cavitated lesions, since the former can frequently be treated with noninvasive or microinvasive measures (see below), whereas the latter require restorative therapy, at least in tooth surfaces that are not directly accessible, such as occlusal or proximal surfaces.[38]

Table 1 offers a related categorization of three color-coded diagnoses that will be subsequently used when determining various therapeutic options and findings relating to caries. It should be noted that transitional stages in particular (such as ICADAS 3, 4) cannot be strictly assigned to the various categories. Several parameters should be considered when diagnosing (and determining a therapy for) caries. Furthermore, in the late stages it can be helpful to distinguish caries lesions that only require restorative intervention (media) from those that also require pulp-preserving (eg, stepwise excavation) or endodontic treatment or even extraction (profunda) (taken from Paris and others[19]).

. . . TO THERAPY

Aim of Treatment

One major difference between caries and diseases of other tissues and organs is that the hard substance of the tooth cannot be regenerated (enamel), or it can only be actively regenerated by cells to a slight degree (dentin). “Healing” occurs primarily through mineralization processes in which cells do not directly participate. Remineralization can, however, only occur where there are crystal nuclei. Nonetheless, changes in the surface of the lesions, especially those visible on buccal smooth surfaces, which give

Table 2: *Etiological Targets for Various Therapeutic Strategies*

Target	Intervention	Invasiveness
Biofilm	Mechanical: oral hygiene	Noninvasive
	Chemical: antimicrobials	
	Biological: probiotics ?	
Nutrition	Diet modification	
	Sugar substitution	
Mineralization	Provide substances that promote mineralization: fluoride, calcium compounds	
	Stimulate salivation: chewing gum	
Diffusion	Sealants	Microinvasive
	Infiltration	
Signs and symptoms	Restoration	Minimally invasive

the appearance of remineralization, are at least partially due to surface abrasion and not to the resupply of minerals.[39,40] Caries lesions that do not yet manifest cavitation cannot be completely repaired in the sense of restoring the original contours of the tooth. If the dentin and enamel have been destroyed to the extent of cavitation, the caries process can only be arrested at best. The primary aim of therapy in this case is to restore the tooth's shape and function through restorative measures and thereby allow the patient to regularly remove plaque.[41]

Treatment Approaches

The model of caries presented above (Figure 1) describes the various etiological factors influencing the caries process and, hence, the potential risk factors for caries. In principle, all of the etiological factors for caries are factors that should be considered when developing preventive therapies; however, direct influences are particularly suitable for altering the caries process by modifying nutrition, the biofilm, or mineralization (Table 2). This consideration has given rise to the most frequently used preventive measures for individuals. The common element of all causal strategies is that they do not require invasive treatment of the enamel and dentin and are purely noninvasive. Some therapeutic options, such as sealants or infiltration, only slightly modify the enamel and dentin and are therefore considered microinvasive. Contrastingly, restorative measures are almost always associated with the loss of dentin and enamel and are minimally invasive at best. The term "minimal" expresses the fact that in contrast to the classic rules of preparation defined by GV Black ("extension for prevention"), the restoration of carious defects is confined to the severe parts only, if possible.

The Patient

For a proper treatment decision, not only the disease level of the individual teeth (the stage and activity of caries) but also the patient's risk of caries must be assessed. As described recently, "The majority of the factors contributing to the origin or prevention of caries affect the entire oral cavity and not just individual teeth. Correspondingly caries therapy should include both a local therapy of individual teeth and a therapy that addresses the patient. A systematic caries therapy should address the causal risk factors for the individual patient that were identified when determining the risk of caries. Another consideration when choosing a therapy is patient compliance. Many noninvasive therapeutic options need to be regularly used or require an adjustment of the patient's habits. Therapeutic approaches need to be chosen that are most likely to be pursued by the patient."[19]

Limits of Noninvasive Options

To permanently arrest caries progression by using exclusively noninvasive means, the tooth surface needs to be sufficiently accessible to cleaning. This is largely influenced by the surface cavitation level, the caries extension, and the pathological activity of a cariogenic lesion, which in particular depends on the surface quality.

The degree of surface cavitation correlates somehow with the radiographic extension of the lesion. As a consequence, there is a greater probability that deeper proximal lesions (ie, middle third in dentin) on a radiograph will be clinically cavitated, compared with shallow radiographic (enamel) lesions. However, in order to predict the size of the cavitation and thus the probability of the lesion's progression rate with an acceptable degree of precision, more is needed than an awareness and interpretation of the lesion's radiographic extension. A clinical investigation of the surface with a fine probe should be performed, especially in areas difficult to access visually. Therefore, in particular for proximal surfaces it is useful to know the rate of cavitated lesions of each radiographic lesion extension. As observed after tooth separation, approximately 10% and 30% of proximal caries lesions with a radiographic extension into the inner half of the enamel (E2) and approximately 30% into the outer third of the

dentin (D1) are cavitated, respectively,[42,43] which might be seen as clinically relevant cavitation. However, no standards exist for detecting, assessing, and documenting the different sizes of (micro)cavitation of the surface, of which proximal surfaces are rather difficult to access with adjacent teeth.

When a caries lesion is cavitated, it can be assumed that a potentially cariogenic biofilm has become permanently established. Even if the patient regularly flosses, the biofilm will be difficult to remove and caries progression becomes very likely. Here, the pathological activity of a cariogenic biofilm seems to be increased, followed by a higher probability of the caries lesion to progress. With respect to detecting and assessing the cariogenicity of plaque (biofilm), one problem is that the visit to the dentist only offers a snapshot and that no valid measurements are available. Thus, for the most part, the amount of visible plaque is taken as a surrogate. However, an informed patient tends to be more accurate with plaque removal before visiting the dentist. Thus, the plaque level measured in one appointment may only yield a conditionally representative impression. The frequent establishment of proximal plaque correlates with a tendency of in the adjacent gingiva to bleed. Therefore, increased bleeding of neighboring papilla seems to indicate a higher activity of proximal caries, at least in periodontally healthy patients.[44]

Lesion Progression

The stage in the caries process at which exclusively noninvasive options may be recommended by the dentist to manage caries largely depends on knowledge about the probable speed of the caries progression. As reported in the 1980s and 1990s in Sweden, occlusal lesions requiring invasive treatment established before the age of 12 years and leveled off in late adolescence. Up to 12 years of age, proximal lesions in the posterior region were mainly restricted radiographically to the enamel. In the period of adolescence, new proximal enamel and also dentinal lesions became detectable, adding up to five lesions in mean, either extending radiographically into dentin or being filled at the age of 26 years.[22] According to the study, the median time of proximal caries progression from sound status until it reaches the inner enamel radiographically is approximately six years over the ages of 11 to 22 years. The median radiological progression rate of caries lesions from the enamel-dentin junction (EDJ) into the outer third of dentin (D1) was approximately twice as high as the rate of progression within enamel (Figure 2).

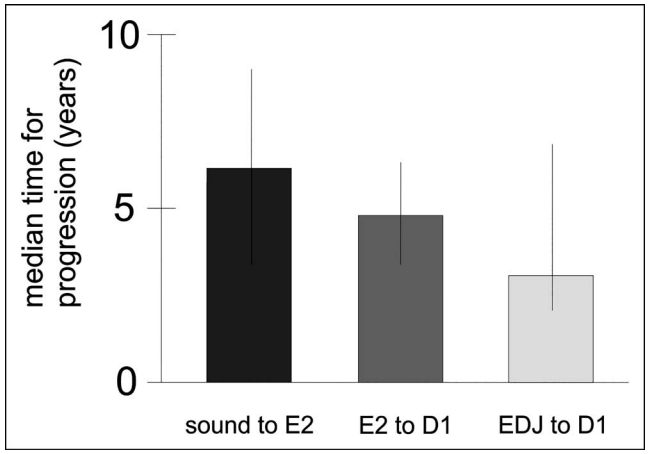


Figure 2. *For an originally sound proximal surface it took about six years in median to establish a caries lesions extending radiographically into the inner half of enamel, as studied in a Swedish cohort in the early 1990s. For a lesion extending into the inner half of the enamel and those up to the enamel-dentin junction it took only five and three years, respectively, to progress into the outer third of the dentin.*[45]

About 10% of the proximal lesions (most presumably in only very few children) progressed within one year from sound to E2 and EDJ to D1, respectively.[45] In conclusion, in most of the patients caries progression is rather low. Occlusal lesions mainly develop shortly after tooth eruption in permanent molars. For proximal lesions, extending radiographically at maximum close to the EDJ noninvasive treatment accompanied by regular monitoring should be favored.

Microinvasive Therapy

In contrast to noninvasive interventions, in the case of microinvasive therapies (ie, fissure sealing and caries infiltration) dental hard tissues are modified in such a way that diffusion barriers are created (eg, with resins). Sealing of occlusal aspects of sound and carious permanent molars is known to be effective in particular in high caries risk fissure and groove systems that can barely be cleaned.[26,46-48] The resin barrier placed onto the surface acts as a diffusion barrier for acids produced in the overlying biofilm; thus, demineralization of the enamel is hampered.

Caries infiltration was introduced as a microinvasive treatment in 2009. After erosion of the surface using 15% hydrochloric acid gel, a low viscous resin, so-called infiltrant is applied onto the caries lesion and penetrates into the lesion pores driven by capillary forces. After three minutes, excess resin is removed from the surface, and the resin inside the lesion pores is light-cured. In this way, the resinous barrier is established inside the lesion, and no

additional sealing, which is in particular difficult to accomplish in proximal areas, is necessary.[49,50] Clinical studies revealed a relative risk reduction of 65%-90% after at maximum three years of followup with regard to the infiltration technique compared with self-applied noninvasive interventions alone. The proximal lesions included were noncavitated and extended radiographically from the inner enamel to the outer third of dentin.[30,51,52] After three years of observation, 46% of the control lesions and 4% of the infiltrated caries lesions had progressed in one of these studies.[52] At present, there are no clinical studies available showing significant clinical effect on postinfiltrated caries progression inhibition for other surfaces. Up until now, fissure caries has not been able to be treated more efficaciously with existing infiltrants compared to sealing alone.[53] In addition to caries inhibition, infiltration of the enamel results in a masking effect of originally whitish caries lesions being used, in particular, for visible vestibular surfaces.[54-56]

Limits to Microinvasive Therapies

To avoid overtreatment, only those caries lesions that are expected to progress and that have not arrested with the use of noninvasive measures, which, for many lesions, cannot be decided at the first dental visit of a patient, should be sealed or infiltrated. However, proximal caries in children (primary molars; four to 10 years of age), adolescents and adults (14 to 35 years of age) manifest a relatively high progression rate,[57] so the danger of overtreatment in this age group is relatively low.[58] For occlusal surfaces of permanent molars, sealing shortly after eruption seems to be most effective.[48] But even established noncavitated occlusal caries lesions can be arrested by sealing if noninvasive treatment alone does not seem to be efficacious enough.[47]

Nonetheless, what is feared far more by many dentists is sealing and infiltrating lesions that are "too deep." This might primarily be related to the concern that the next (invasive treatment–oriented) dentist might argue with the patient that the former (monitoring-oriented) dentist had overlooked a lesion and had not treated it invasively enough. Secondly, this might be due to the misbelief that carious dentin underneath a noncavitated enamel lesion always needs to be removed, since it is infected. In addition, most dentists are experienced in preparing a cavity for a rather shallow proximal caries lesion (ie, extending radiographically up to the enamel dentin junction, but not into dentin), as they were taught that the soft and stained carious dentin histologically extended much deeper (eg, into the middle third of dentin). However, the histological status of the dentin *per se* is no indication for a restoration. As described above, the lesion surface status is the primary factor in determining the therapy. Moreover, noncavitated enamel caries lesions and also underlying carious dentin only contain a few bacteria[59,60] that do not form cariogenic biofilms because of the minimal size of the cavities. It is therefore generally not considered problematic to seal in or infiltrate these areas. This means that when EDJ or D1 lesions are identified on the radiograph and the extent of the caries on the surface is known, one must judge whether the caries can be arrested by noninvasive treatments alone or by infiltration. Only when one has doubt related to the cavitation status might restorative therapy be the right choice.[58]

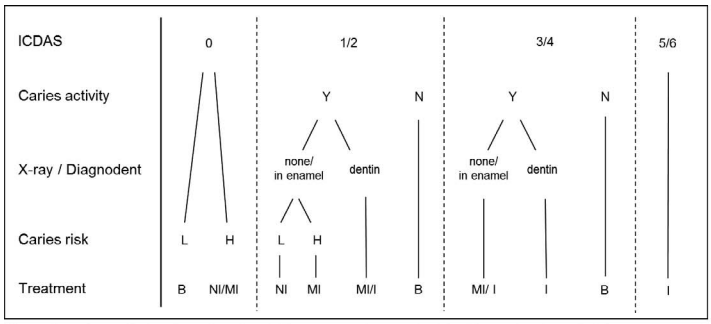
(Minimally) Invasive Therapy

If a proximal or an occlusal lesion is cavitated either into enamel only or into dentin, restorative measures seem to be the best choice. For these, the risks of treating noninvasively or microinvasively alone (ie, caries progression is likely or the tooth gets painful) become greater than the anticipated benefits (ie, protection of sound dental hard tissues, lower costs, and less treatment stress).[61]

In addition to the mechanical, functional, and esthetic aspects, the primary aims of a restoration are to stop disease progression[6,38] and to restore the tooth's ability to be cleaned by the patient by means of plastic reconstruction.[62] Through this process, the heavily diseased parts are removed, but parts being affected only by the caries process are inevitably sealed in, as is the case with microinvasive treatments for noncavitated lesions. Nonetheless, it needs to be remembered that all restorations age over time and will be replaced from time to time by presumably larger restorations.[63] The other articles in this special issue will focus on the material aspects, different techniques, and also repair of restorations.

DECISION TREES AND CHOICE OF THERAPY

To relate the three possible therapies for coronal primary caries (noninvasive, microinvasive, and minimally invasive) to practice, decision trees that can also be employed in the quality management of clinical settings might be helpful in a shared decision-making process.[6] We give two examples, including the most important diagnostic criteria for occlusal and proximal lesions without a prior restoration (Figures 3 and 4). In addition to these decision trees, the most frequent findings and



Y = yes, N = no, L = low, H = high, B = basic prophylaxis, NI = noninvasive (e. g. cross-brushing), MI = microinvasive (sealing), I = invasive (restoration)

Explanation of findings for the decision trees		
Criterion	**Severity**	**Explanation**
Diagnodent caries evaluation (occlusal)	none/ enamel	value <30
	dentin	value =30
General risk of caries	low	value <33%*
	high	value =33%*
Local risk of caries	low	no anatomical predisposition
	high	anatomical predisposition

*according to caries risk assessment

Figure 3. *Decision tree for caries of occlusal surfaces without a restoration (modified from[6]).*

treatment options ("golden rules") are also described. As said previously, beyond the therapies, on the tooth level the dentist or patient should also pursue patient-related noninvasive interventions according to the caries risk.

Occlusal Surfaces

Whereas it is frequently difficult to diagnose caries in fissures and grooves, the therapy of caries in these locations is relatively easy, since the contour of the cusps and fissures is generally readily accessible. Noninvasive methods, such as controlling the biofilm by mechanical (cross-brushing during eruption) or chemical (chlorhexidine varnish) means or local fluoridation, are used for healthy fissures with an elevated risk of caries and for fissures with early stages of caries. For more severe findings, wither sealing or minimally invasive resin restorations are indicated (Figure 3).[19,38,64]

The following general rules can be applied to this tooth surface:[6]

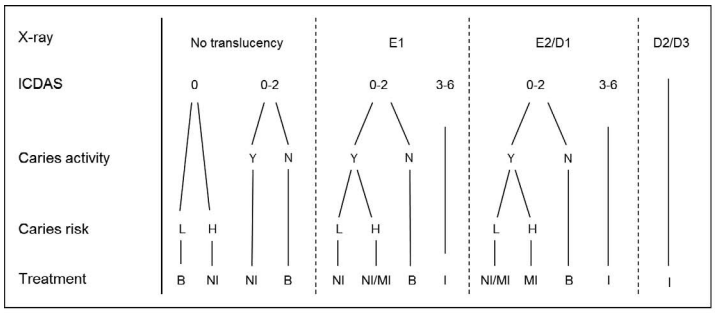
- If the caries is inactive, it should only be monitored and basic prophylaxis reinforced;
- Surfaces categorized as ICDAS 0 and active caries of stages ICDAS 1-2 and occasionally 3 should be treated noninvasively or sealed if there is a higher risk of caries;
- Active caries of stages ICDAS 4-6 should be filled in most cases. If the lesions are very deep, consider removing the caries in stepwise excavation technique or incompletely.

Proximal Surfaces

Given its location below the contact point, proximal caries represents both a diagnostic and therapeutic challenge. Noninvasive methods such as plaque control or local fluoridation are appropriate for healthy tooth surfaces or for surfaces with early forms of caries to prevent or arrest the disease. The control of plaque on proximal surfaces is, however, much more difficult than on other tooth surfaces. The sealing and infiltration of caries are microinvasive measures that can be used to arrest the progress of noncavitated forms of caries. If clinically significant cavitation exists, restorative measures are indicated to restore the ability of the tooth surface to be cleaned. The poor accessibility frequently means that a large amount of enamel and dentin must be removed during the restoration to reach the diseased hard tooth substance (Figure 4).

The following general rules can be applied to this tooth surface:[6]

- In the case of inactive caries of stages ICDAS 1 and 2, basic prophylaxis is sufficient, even given a



Y = yes, N = no, L = low, H = high, B = basic prophylaxis, NI = noninvasive (e.g. fluorides), MI = microinvasive (infiltration), I = invasive (restoration)

Explanation of findings for the decision trees		
Criterion	**Severity**	**Explanation**
Diagnodent caries evaluation (occlusal)	none/ enamel	value <30
	dentin	value =30
General risk of caries	low	value <33%*
	high	value =33%*
Local risk of caries	low	no anatomical predisposition
	high	anatomical predisposition

*according to caries risk assessment

Figure 4. *Decision tree for caries of proximal surfaces without a restoration (modified from Meyer-Lueckel and others[6]).*

radiographic extension into the first third of the dentin;
- Active caries of stages ICDAS 1 and 2 with a radiographic extension of E1-E2 should be treated noninvasively (floss, fluoride), if the risk of caries is low;
- Active caries of stages ICDAS 1 and 2 with a radiographic extension of E2-D1 should be infiltrated, if the risk of caries is increased;
- Active caries of stages ICDAS 3-6 should be filled in most cases. If the lesions are very deep, consider removing the caries in a stepwise caries excavation process or incompletely.

Accessible Smooth Surfaces and Anterior Teeth

Oral and buccal smooth surface caries in anterior teeth is less frequent, since oral hygiene can much more easily be established in this area. Here, caries prevalence has decreased over the last decades compared with during the prefluoride era. Nonetheless, with increased plaque retention, as is the case in patients with fixed orthodontic appliances and patients with little oral hygiene, caries lesions are still occurring.[65-68] These lesions are relatively easy to diagnose, and noninvasive measures such as improved plaque control with fluoride toothpaste plus local fluoridation are effective in arresting these caries lesions.[19,64] Only when oral hygiene is difficult to perform (ie, fixed braces) is there a concern that noninvasive measures alone might not be sufficient to avoid caries lesions.

For anterior teeth, esthetic aspects come into play as well. Although an arrested lesion might be a valuable goal form a cariologist's perspective, the unappealing appearance of the "scar" demands further therapies.[69] Nonetheless, the esthetic rehabilitation is most often accompanied by loss of more (eg, microabrasion, composite, or veneers) or less (caries infiltration) additional dental hard tissue.

Root Caries

Today, root caries is found more frequently in older patients with a periodontal attachment loss and exposed dentinal root surfaces.[70,71] Therapy depends on the accessibility of the lesions for oral hygiene. Buccal lesions might even be arrested in cavitated stages by noninvasive therapies (eg, highly fluoridated toothpaste, chlorhexidine, or sodium fluoride varnish).[72] Nonetheless, brownish discolorations of the dentin yield more esthetic treatments, such as adhesive restorations. For proximally located, cavitated root caries lesions, noninvasive therapies are not supposed to be as effective, since plaque removal cannot be accomplished sufficiently. For invasive procedures similar to proximal enamel caries, relatively large amounts of sound dental hard tissues have to be removed to get access to the carious tissues and for restoration placement.

CONCLUSIONS

Minimum interventional treatment of caries involves local treatments at the tooth level as well as addressing the causative factors of the disease on the patient level. Monitoring of caries is essential to assess caries activity and also to support successful treatment outcomes. In addition to minimally invasive interventions, noninvasive and microinvasive options should be a focus of modern cariology in order to preserve dental hard tissues to a greater extent, as has been the case in recent decades.

Note

Parts of this article have been published in sections of the textbook[6,13,19] *Caries Management—Science and Clinical Practices*.[73] Reprinted by permission.

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Regulatory Statement

The preparation of this manuscript was conducted in accordance with all the provisions of the local human subjects oversight committee guidelines and policies of RWTH Aachen University in Germany.

Conflict of Interest

HML and SP are appointed as inventors of US and European patents for an infiltration technique for dental caries lesions, held by Charité-Universitätsmedizin Berlin, and receive royalties from DMG, the manufacturer of a product for caries infiltration.

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