Dentin caries: Progression and clinical management

BUONOCORE MEMORIAL LECTURE 2002

Operative dentistry today focuses very much on cavity design and selecting restorative materials. Less effort has been placed on incorporating what is known about the pattern of caries progression and how it relates to caries removal or excavation.

This presentation updates the progression and clinical management of dentin caries, and how it relates to treating deep caries lesions.

A fundamental issue of restorative treatment is assessing the different conditions of cases, not only from tooth-to-tooth but also from the activity of each caries lesion and the size of the cavity. In addition, restorative treatment is sometimes carried out for prosthetic and cosmetic purposes and involves cutting sound, unaffected dentin. These aspects are outlined in classic textbooks and are based on the principles described by Black [1]. However, each factor and its relative importance has changed over the years. Significant time has been devoted to improving the important technical aspects of performing restorative treatment. Traditionally, these aspects have primarily been related to cavity design, choice of restorative material and the clinical procedures involved. In this context, Dr Michael Buonocore’s contributions must be recognised because the adhesive technique he introduced has become an integral part of modern operative dentistry.

Operative dentistry today also focuses on cavity design and selecting restorative materials. Less effort has been placed on incorporating what is known about the pattern of caries progression and how it relates to caries removal or excavation. Although the reaction pattern of the pulp-dentin organ is quite different in terms of the nature of active (rapid-progressing) and arrested (slow-progressing) lesions, no widespread major distinction has been made regarding the different restorative treatment approaches in these situations.

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**Definition of lesion characteristics**

The dentistry of today has developed into many sub-specialties. As a result, terms used in operative dentistry may have different meanings. Caries is a good example. Researchers involved with cariology would first associate a lesion with specific bacteria and the progressive and dynamic events of mineral loss starting at the surface enamel, whereas the term “caries” used in operative dentistry indicates a cavity in need of restorative treatment. Although the reaction pattern of the pulp-dentin organ is quite different in terms of the nature of active (rapid-progressing) and arrested (slow-progressing) lesions, no widespread major distinction has been made regarding the different restorative treatment approaches in these situations.

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**Enamel lesion without dentin exposure**

A recent textbook has focused on the clinical and histopathological pattern of untreated caries in different stages of lesion progression, in order to update our knowledge of the pulp-dentin organ [2]. It is important to realize that there is a well-defined structural interrelation between dentinal reactions subjacent to the clinically active progressing enamel lesion without clinical exposure to the dentin. Previous interpretation of the first signs of dentin caries focused on the early rapid spread of decay that undermines sound enamel. Then, an independent description of the dentin involvement followed with no strict relation to either the enamel or the pulp [3].

If a thick section taken from an extracted tooth with caries is examined, it is easy to make such an interpretation. However, as soon as the thin undemineralized sections are examined, the first alteration in the dentin resulting from caries can be traced all the way to the pulp and the dentin is hypermineralized [4, 5]. The onset of dentin demineralization and dentin discoloration are not observed until the lesion has reached the dentino-enamel junction, and no lateral spread is noted in relation to enamel lesions without dentin exposure (Figure 1). Quantitative analyses have shown that the extent of the discolor-
ed demineralized dentin typically follows the enamel lesion in contact with the dentino-enamel junction [5, 6]. At this stage of lesion progression, dentin reactions are strictly guided by the cariogenic environment on the enamel surface. From a clinical viewpoint, this means that a non-operative treatment of the enamel lesion, including the removal of cariogenic biomasses at the outer enamel surface, would lead to the arrest of the entire enamel-dentin lesion complex.

It is commonly believed, that early dentin caries spreads along the dentino-enamel junction. This concept is important because the clinical consequence of this early spreading has been to remove the affected dentin as soon as possible, with no specific reference to the status of the lesion on the enamel surface. The presence of discolored dentin has, moreover, led to the conclusion that the treatment was justified, and if done correctly, will prevent further undermining of sound enamel. However, based on an updated view of the structural characteristics of the enamel-dentin lesion, the most effective way to treat the actively progressing, non-cavitated lesion, is by using non-operative treatment principles.

The spreading pattern of caries following enamel breakdown has led to designs of micro-cavities, for example tunnel preparation or internal occlusal fossa preparation [7], whereby early dentin exposure is treated by a retrograde approach. In addition, the introduction of dental operating microscopes represent a challenging improvement in relation to the clinical assessment of early caries, but the observations made must be correlated to the knowledge of lesion activity and the spreading pattern of caries. Taken together, the field of micro-procedures and micro-cavities also represents a potential risk of introducing clinical procedures at early stages of lesion progress, where further progression could have been avoided because the enamel lesion was already inactive or because it could be arrested by preventive means.

**Examination of the pulpal response to non-cavitated caries**

Through the use of thin, undemineralized tooth sections, it is possible to simultaneously examine the events taking place in the hard and soft tissue. As also indicated by Brännström & Lind [8], cellular changes in the odontoblastic and subodontoblastic regions have recently been shown to take place subjacent to subsurface enamel lesions (Figures 2 and 3) confirmed by the use of thin undemineralized tooth sections [9]. It was also noted that the cellular reactions along the pulp-dentin interface from rapid- and slow-progressing enamel lesions were different. In clinical terms, this means that removing cariogenic biomasses during non-operative treatment of enamel lesions is not only reflected at the enamel tooth surface, but also along the pulp-dentin interface. In short, the clinical concept of different discolorations of demineralized dentin (Figures 4 and 5) characterizing different lesion activities can be biologically confirmed at the cellular level, thus demonstrating the reversible nature of the early pulpal response [9].

**Lesions involving dentin**

Considerable debate has been devoted to the degree of bacterial infection in relation to the surface status of the caries lesion [10, 11]. In non-cavitated enamel lesions, the level of bacterial invasion is very low, if at all present. Even though the enamel is demineralized, the spaces in-between the crystals are too small for micro-organisms to penetrate (Figure 6), and

**FIGURE 2.** Macroscopical view of a freshly extracted third molar. Two cutting lines in the occlusal surface indicated the following hard tissue specimen, involving rapidly progressing enamel lesions, still with cariogenic plaque. (Original magnification X 2.5.)

**FIGURE 3.** Reduced odontoblastic layer and reduced predentin width subjacent to the active occlusal lesions shown in Fig. 2. (Toluidine blue-pyronin stain; original magnification X 2.5.)
Dentin caries

the subjacent discolored and demineralized dentin, in general, is without the presence of micro-organisms in precavitated lesions. As soon as the demineralized enamel layer crumbles, particularly from forces of mastication and any stress toward the lesion area, a growing accumulation of micro-organisms will take place (Figure 7). Eventually, the dentin becomes infected by micro-organisms, and the bacterial spreading is markedly changed. The bacterial growth conditions change from being a bacterial plaque at the external tooth surface, to being protected deep within the lesion environment by the broken down enamel, which can be observed along the dentino-enamel junction (Figure 8). This process has classically been described by Black [1] as "backway decay of enamel". Clinically, this spreading pattern can be traced through the sound undermined enamel, as a shift in enamel translucency. Looking for these lesion characteristics, one is seldom surprised that the extent the final preparation will reach deep into the dentin (Figure 9).

The pulpal response to dentin exposed by caries

Deep dentinal lesion progress is often presented as the point of no return for the specific pulp involved. The signs and symptoms of irreversible pulpitis followed by apical pathosis enter the discussion. In deep lesions, however, large variations and changes within the lesion environment may be detected. Closed and open ecosystems have been suggested for these phenomena [12] related to the clinical features of the cavity. The open lesion environment represents a lesion where the undermined enamel has been broken down, changing the growth condition for the cariogenic biomasses. As Figure 10 illustrates and with focus on caries activity, different rates of progression can be present within one tooth. The occlusal and central part of the vital

Figure 4. Ground section through an active occlusal lesion with yellowish discoloration of the demineralized dentin. (Reflected light; original magnification X 2.5.)

Figure 5. Ground section through an arrested lesion with a dark brownish discoloration of the demineralized dentin. (Reflected light; original magnification X 2.5.)

Figure 6. Detail of enamel lesion covered with cariogenic plaque. The enamel rods are clearly visible in the 15 µm thin undemineralized tooth section. No bacteria are penetrating the demineralized rod structure. (Transmitted light; original magnification X 50.)

Figure 7. Detail of dentino-enamel junction subjacent an active, progressing enamel cavitated lesion. The enamel layer has crumbled down to the dentin, ending with real dentin exposure. Presence of bacteria in the dentin. (Transmitted light; original magnification X 50.)
tooth is actually without heavy plaque accumulation, because chewing and other functions prevent it. The corresponding dentin also appears to have signs of slowly arrested dentin caries shiny with brown discoloration. In contrast, the peripheral parts are still protected by undermined enamel and heavy accumulations of cariogenic biomasses. This marked change within lesion environment can also be reflected within the pulp as different types of tertiary dentin \[13\]. In "closed", active and rapidly progressing lesions, the initial tertiary dentin is laid down without dentinal tubules, also defined as fibrodentin \[14\] or interface dentin \[15\], whereas in slow-progressing lesions the extra-dentinal matrix laid down is tubular, resembling primary dentin. These observations indicate that deep dentin affected by caries is not unconditionally related to an irreversible pulp pathology, as traditionally described in textbooks that advocate pulp invasive treatment procedures \[16, 17\].

**Treatment of deep caries**

**– the history of different concepts**

The philosophy of restorative treatment of deep caries can be traced back to two thoughts "... it will often be a question whether or not the pulp will be exposed when all decayed dentin overlaying it is removed". "It is better to expose the pulp of a tooth than to leave it covered only with softened dentine" \[1\]. In contrast, Tomes \[18\] wrote: "It is better that a layer of discolored dentine should be allowed to remain for the protection of the pulp rather than run the risk of sacrificing the tooth". Today, eventhough maintenance of pulp vitality in deep lesions has been widely discussed, the subject has been dominated by this contradictory and controversial information \[19\].

Different methods for preventing exposure and damage to the pulp have been advanced. The first is the indirect pulp-capping procedure employed particularly in the primary dentition \[20\] and in mixed dentitions \[21–23\]. The second method is the two-stage excavation procedure \[24–26\] or stepwise excavation \[27\], which, more recently, has been applied even in the permanent dentition \[28, 29\]. The main difference is that the indirect pulp-capping procedure almost completely removes the affected dentin, leaving a thin layer of residual de-mineralized dentin and re-entry is not made; that is, it is a one-step procedure, while the stepwise excavation procedure involves re-entry at varying intervals.
Treatment of deep caries lesions
based on an understanding of caries pathology

Recently, a less invasive first excavation procedure
in the stepwise approach was introduced (Figure 11). It aims at further reducing the risk of pulp
exposures during the first excavation and promo-
ting physiological reactions in the pulp-dentin or-
gan but with particular focus on the overall change
in lesion activity [29–31]. This first excavation pro-
cedure is not, as originally attempted, to reach the
pulp as closely as possible in order to stimulate the
formation of extra-dentinal tissue per se. Instead,
it is to promote arresting the lesion by changing
the cariogenic environment.

Clinical changes before and after such a modified
less invasive procedure [29] have shown that the
caries arrestment can be assessed easily. A harder,
more brownish demineralized dentin (Figures 11 b
and c) will be found following the temporary seal-
ing of the lesion, in accordance with Miller’s [32]
description of active and arrested dentinal lesions.

This approach also allows reparative dentinogene-
sis to occur [33]. Dentin permeability will be re-
duced during the treatment and will affect the long-
term effectiveness of the permanent restoration.

Why perform the final excavation?

When using a less invasive first excavation ap-
proach, bacterial counts of dentin samples have
been shown to decrease in a similar way during a
treatment interval [29], as seen in studies where
the excavation was performed to the residual level,
which is much closer to the pulp [20, 22]. How-
ever, since completely sterile conditions are not
created, no long-term data supports avoiding the
final excavation into deep lesions. Clinical obser-
vation of dentin changes during the treatment
provides, the clinician with relevant information
about the change in lesion progress. The final ex-
cavation is facilitated, because it is more con-
venient to excavate in harder and darker carious
tissue close to the pulp (Figure 11 c) than in soft

![Radiograph of P2 inf with deep dentin exposed lesion. A: No evidence of apical pathosis. Pulp is vital. B: A stepwise excavation is decided, and a survey of the cavity following first excavation is shown before sealing and temporary filling. C: After a six months treatment interval and removal of base material (calcium hydroxide) and temporary filling (amalgam) the retained carious dentin shows signs of a slowly progressing lesion. D: The premolar after completed excavation, and final restoration can be performed.](image-url)
yellow demineralized dentin. The dual function of final excavation is therefore to perform clinical control of the tooth reaction and to remove the slow-progressing but still slightly infected discoloring demineralized dentin, before carrying out the permanent and final restoration. The 10-year results by Mertz-Fairhurst & others [34] which make a one-step procedure that arrests dentin lesions by sealants, seems as a break through in terms of controlling a cariogenic environment, but it is also important to note that these results were based on less advanced dentinal lesions being located in the outer half of the dentin. Therefore, outcome studies comparing the indirect pulp capping procedure with a stepwise excavation approach in similar sized deep lesions are needed.

Experience from private practitioners performing stepwise excavation
Experience from a dental practice environment has shown the effectiveness of the stepwise excavation procedure for the management of deep carious lesions, and long term recall (3'/4'/5' years) has shown a high success rate (92 %) with teeth treated by this approach [35]. Although the total group of failed cases was less than 10 %, in half of them insufficient temporary and permanent restorations were noted, underlining the importance of performing a high-quality temporary as well as permanent seals. A two-step excavation procedure will add to the cost of the restorative treatment, including control examinations with regard to pulp sensitivity and vitality because of the possibility of the asymptomatic development of irreversible pulp degeneration over time.

Comparisons with other studies must be done cautiously. However, 5 % of the cases treated by the general practitioners in the above study had pulpal complications during final excavation. In contrast, the traditional and more invasive step-by-step approach [27, 28] presents a higher proportion of pulp complications during the final treatment (~15 %). This could indicate the positive effect of a less invasive first excavation procedure. In addition it probably reduces the frequency of iatrogenic pulp exposures.

Case selection and clinical comments on stepwise excavation
As long as there are no non-invasive tools to assess the histopathological condition of the pulp and for the measurement of the severity of the pulpal inflammation, discussion of reversible or irreversible development of pulpitis will remain controversial in relation to treatment of vital, non-symptomatic deep dentin lesions. On this basis, the only alternative is to depend on patients’ information, the objective evaluation of pulp vitality as well as information obtained from radiographs [36]. Based on data from the Danish dental practice environment [31] the following guidelines are currently suggested in relation to case selection for the stepwise excavation:

- Deep lesions likely to result in pulp exposure if they were treated by a single and terminal excavation. Evaluated by radiographs, the dentinal lesion should involve more than 75 % of the entire dentin thickness (Figure 11).
- No history of subjective pre-treatment symptoms, such as spontaneous pain or provoked pulpal pain; however, mild to moderate pain upon thermal stimulation is accepted.
- Positive pulp vitality tested by an electric pulp tester, thermal stimulation or drilling.
- Pre-treatment radiographs to exclude apical pathosis.

Clinical comments concerning a less invasive stepwise excavation approach include:

- Decide at an early stage in the treatment that it will be a stepwise excavation case.
- Finish the peripheral excavation of the lesion followed by a central excavation that removes the outermost necrotic and infected demineralized dentin (Figure 11 b), so that a provisional restoration can be properly placed.
- Do not attempt to excavate as close to the pulp as possible during first step, thus reducing the risk of pulp exposure.
- Decide which material (usually calcium hydroxide) to cover the remaining carious dentin.
- Decide which provisional restorative material will be used in relation to the length of the treatment interval, ranging between 6 and 8 months (amalgam, glass ionomers and composites may be excellent temporary materials).
- Performing the final excavation often ends up being less invasive than expected due to the altered dentinal changes gained during the treatment interval (Figure 11 d).

Final comments
Change in dentin permeability, including tertiary dentin formation following a stepwise excavation procedure, represents secondary biological reaction that is not only produced for the protection of the pulp, but also as a consequence of a change in the cariogenic environment. Therefore, tissue changes per se cannot permanently serve as a barrier against new, potential cariogenic biomasses to create the reestablishment of low pH-gradients or prevent a re-invasion of bacterial antigens.

A full understanding of the pulp–dentin organ in restorative dentistry also includes knowledge of
its limitations. Control and prevention of further secondary damages to the restored tooth will, besides providing an optimal restoration, include the care of proper oral hygiene procedures for removing cariogenic biomasses that tend to accumulate where it all began – in the area of the restored tooth surface.


References


