PULP BIOLOGY AS A PARAMETER FOR CARIOUS DENTIN REMOVAL

Biologia pulpar como parâmetro para remoção de dentina cariada

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Abstract

The present study is a review of the literature which focuses on the development of dental caries in the dentinopulpal complex. Progression of the carious lesion and its consequences to the pulp biology are emphasized since dentinal permeability and endotoxin penetration will bring about various pulpal reactions. The clinical viability of a partial removal of the carious dentin is questioned, given that the histopathologic changes are already present in the pulpal organ.

Keywords: Dentinopulpal complex; Dentin permeability; Endotoxins; Caries.

Resumo

Trata-se de uma revisão de literatura que ressalta os aspectos da biologia do complexo dentino pulpar frente às agressões da cárie dentária. As questões de permeabilidade dentinária e a conseqüente penetração de endotoxinas bacterianas são enfatizadas bem como o substrato protéico originado da própria dentina cariada que favorece o desenvolvimento de bactérias proteolíticas mesmo depois da lesão inativada. Questiona-se a viabilidade clínica da remoção parcial da dentina cariada, uma vez que o tecido pulpar já sofreu alterações em suas bases biológicas.

Palavras-chave: Complexo dentino-pulpar; Permeabilidade dentinária; Endotoxinas; Cárie.

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Introduction

A carious lesion causes degenerative changes in the dental structures; on clinical and microscopic examination, the development can be observed of a process partially represented by bacterial proliferation – with its metabolic products destroying tissues – and partially represented by the reaction of the dentinopulpal complex to the attack of the lesion (1). This denotes the bipolar character of the evolution of tooth decay and its microorganisms in the tooth structure.

Given that both pulp and dentin are tissues derived from the same embryological origin, any damage to or intervention in the outermost layer of the enamel will affect to some degree the pulpal complex (2).

On the one hand, there is a structure where 70% of its weight is mineral and, although fenestrated, its inorganic portion may be re-mineralized. On the other hand, there are bacteria invading the dentin, thus causing problems to the pulp. According to Bergenholtz (3), inflammatory stimuli from the carious process will diffuse to the pulp through the dentinal tubules and then they are considered etiologically significant, for they will have induced a pulpal response. Therefore, as soon as the carious process takes place, structural changes can be observed on the tooth surface, and a response from the pulpal organ follows immediately, either localized or generalized (4).

Removal of the carious tissue and an adequate preparation of the cavity are undoubtedly an important phase in the control of the disease, aiming at preserving the healthy structure with its biological and mechanical functions partially repaired (5).

In its pursuit of sound scientific knowledge, the current literature on this matter reports different viewpoints on whether carious dentin removal must be total or could be partial. Several research works reveal knowledge of the problem's etiology, its evolutive phases, histopathologic aspects, and methods for both prevention and treatment of carious lesions. However, these scientific findings are usually limited to the analysis of lesions present in the enamel and in the dentin, many times leading to premature conclusions due to their short periods of observation, or else because due importance is not given to the fact that the tooth structure is made up of a diversified tissual complex with very particular biological characteristics.

Furthermore, in order to avoid endodontic treatment, several materials and techniques have been described and analysed, in cases where the subsequent absence of symptoms has been interpreted as the result of a successful intervention. However, Langeland (6) reports that the absence of pain, either immediately or in the following period of up to two years, does not mean the pulp is healthy, given that a chronic inflammatory process could be present for years without symptoms.

Therefore, it is the biology of the pulp tissue, due to its own particularities, cellular and vascular content, that should guide the procedures – applicable to all tissues of the dentinopulpal complex.

The present study aims at reviewing the literature that (a) is the basis for what is currently asserted about the response of the pulpal complex to the presence of a carious dentin, and (b) can provide us with a sound scientific basis for clinical procedures concerning removal of the carious dentin.

The dentinopulpal complex

Dentin and pulp are different forms of the same tissue. In spite of their being frequently taken as different tissues, they do present embryological origin, histological components, and functions that point to a single tissue. Dentin is made up of inorganic tissue (70% of its weight), organic tissue (20% of its weight), and water (10% of its weight). However, in relation to its volume, dentin is 55% organic tissue – where an infectious process could easily develop. In terms of morphology, dentin is characterized by the presence of countless tubules, filled up by odontoblastic processes, plasmatic liquid, and proteins. This means the dentinal tissue is highly permeable, as shown by Pashley et al. (7), who observed radioactive isotopes diffusing through the dentinal tubules to the pulp chamber, regardless of the presence of pulp tissue. When chemical stimuli were applied to the dentin, the isotopes would reach the pulp quite rapidly – due to the dentin's tubular structure. From the initial developmental stage until it reaches its maturity,

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the dentin suffers alterations in its degree of mineralization, a process which occurs continuously and may be physiological according to the age of the individual or pathological as a response/reaction to a carious process or to a restorative procedure. Such changes in the pattern of dentinal calcification take place at the peritubular level thanks to the continuous odontoblastic activity. As long as there is odontoblastic activity, mineral deposition could eventually obliterate the lumen of the dentinal canaliculi (2).

This mineralization begins at the amelodentinal junction and spreads towards the pulp. The dentinal region with these characteristics of calcification is called physiological sclerotic dentin, and shows areas of partially or totally obstructed canaliculi. A similar process may be stimulated or accelerated by the presence of a carious cavity or else by clinical procedures, in which case it is called reactional sclerotic dentin (8). Unlike the physiological deposition, this does not distribute uniformly throughout the dentin, and is restricted to the damaged canaliculi, thus outlining a clearly visible trail towards the pulp.

Minimal intervention at the upper portions of the dentin is, therefore, immediately perceived by the pulp tissue, and a response, either localized or generalized, starts to develop. Thus, cutting dentinal portions ultimately means cutting live tissue made up of a calcified portion and a cellular portion, the latter represented by the odontoblastic prolongations (4).

The dental pulp is a conjunctive tissue rich in cells, blood vessels and nerves, with little capacity to react and respond to external stimuli, and these reactions and responses usually come from the dentin through the dentinal tubules. The low biological tolerance of the pulpal tissue is attributed to its being surrounded by mineralized tissues, something which hinders vasodilatation as well as exuding processes, both of which are inherent to the inflammatory process.

In active lesions of fast progression, Thoma and Goldman (9) conclude there is not enough time for the pulp to defend itself by forming a secondary dentin, differently from the situation in cases of chronic carious lesion. Hill (10) had already shown that secondary dentins will form in response only to light and moderate stimuli. When the dentinal tubules are affected, one of the first signs of this condition is a change in the odontoblastic layer, that is, one finds a lower number of odontoblasts and the individual form of the cell alters from the normal rectangular shape to a more elongated shape.

Seltzer and Bender (11) have determined that changes in the odontoblastic layers are indicative of a severe inflammatory reaction. They noticed the first alterations in the odontoblastic layers were caused by their exposure during operative procedures. Odontoblastic cell permeability would alter in a matter of minutes after injury, and the presence of metabolic molecules within the cell could be observed. Thus, osmotic pressure increased, causing tumefaction and, in cases of extreme severity, rupture of the cell. The inflammatory process was set off by injury to the odontoblasts, causing vascular changes. The endothelial cells turn more permeable, and the edema widens the spaces between the odontoblasts, putting pressure on the cells and rupturing the dentinopulpal membrane. When the injury is much too severe, firstly leukocytes are observed among the odontoblasts, and then erythrocytes, causing hemorrhage. Odontoblast survival will depend on how distant they are from the site of the injury.

Seltzer et al. (12) have shown that incipient carious lesions do not produce histological changes relevant to the pulp tissue other than an increase in the formation of reparative dentin. As the carious process develops in the dentin, macrophages will appear, as well as lymphocytes, scattered in the pulp coronary portion below the dentinal tubules that have been damaged. In the deeper carious lesions the numbers of macrophages and lymphocytes are even higher, and neutrophile nuclear polymorphs can be observed around the dilated blood vessels. Chronic inflammatory exudate will develop as the carious lesion gets deeper and deeper.

Carious lesion and pulp biology: microbiota and dentinal permeability

The carious process is characterized by a pathological imbalance in the oral microbiota that is linked to the inherent features of the host structure (defense, salivary immunoglobulin), as well as to the substratum, that allow for bacterial growth and proliferation (13).

In the initial carious lesion, the fermentable carbohydrates, such as saccharose, nourish and aggregate bacteria for the formation

of the biofilm. Aciduric and acidogenic microorganisms adhere to the tooth, causing an imbalance in the re-mineralization/demineralization process.

In the dentinal carious lesion, a selection of bacteria takes place – as the lesion advances, bacteria are more and more proteolytic. Moreover, the substratum the bacteria need to make the infectious process endure is now obtained from the organic part of the dentin and not just from the oral medium (14). Inside the dentinal tubules, besides the odontoblastic process and nervous fibrils, there is a considerable quantity of collagen (types I and V), specially in the innermost dentin, which can be used as substratum. Proteoglycan, tenascin, fibronectin, serum albumin, alpha 2HS, and transferrin may also be found composing a complex matrix in the form of hydrogel (15).

Histopathologically, four different zones may be found in a carious lesion, which are, from pulp to surface: translucid or sclerotic zone, demineralized zone, zone of penetration or invasion, zone of destruction (16). These zones, however, are not representative of the dynamic form which is characteristic of the carious process, for the bacterial presence is not restricted to the zone of invasion only, as shown by Lundy and Stanley (17), who prepared cavities in human teeth exposed to saliva and measured the bacterial invasion in the dentinal tubules as the postoperative period progressed. The authors observed that the rate of bacterial invasion was approximately 1.6 μm/day during the first 25 days, and 14 μm/day for 120 days. Bacterial depth went as far as 3.0 mm after 210 days. Furthermore, Nagaoka et al. (18), with similar methodology, observed that maximum depth of penetration was 2.1 mm for non-vital teeth in 150 days, and in vital teeth, 18.9% of the dentinal tubules evaluated had been invaded by bacteria during that 150-day period.

Mertz-Fairhurst et al. (19) analyzed the evolution of carious lesions in bilateral permanent first molars in children. Their control group was made up of lesions which were kept open, while their test group was made up of sealed cavities. The authors observed that the sealed lesions did not develop as fast, and their evolution towards the pulp was reduced – due to their having less substrata from the oral medium. The sealing of the cavity itself causes an impact on the microbiota, reducing the bacterial production of acids.

However, the carious lesion does not stop developing, for there are bacteria resistant to dental procedures, and there may be a proteolytic substratum present in the deep dentin.

Substratum for the microorganisms

The availability of proteic substrata originated in the carious dentin itself results in a selection of protein-degrading microorganisms. Larmas (20) traced *in vitro* the enzymatic activity in both healthy and carious teeth through the dentin. Most enzymes showed high proteolytic activity in the deep dentin of teeth with carious lesions, proving there is substratum for proteolytic bacteria in the dentin, even after the carious lesion has been inactivated.

According to this author, the levels of hydrolysis of proteins per milligram were at least ten times higher in the carious than in the healthy dentins. These findings do not exclude the possibility that the enzymes in carious dentins in mature teeth could be of endogenous origin. The natural substratum of dental tissues for this type of enzymes comes from lipidic and glycolipidic membranes of the odontoblastic processes.

It is also possible that bacteria interact with proteins of the host structure, thus facilitating their invasion of the tubules. The literature has already described how some bacteria adhere to the collagen. Some features of *streptococcus mutans*, *lactobacilli*, and *enterococcus faecalis* facilitate their adhering to collagenic fibrillae present in the carious dentin.

Love (21) carried out an *in vitro* study where three specimens of bacteria were cultivated: *streptococcus mutans*, *streptococcus gordonii*, and *enterococcus faecalis*. In human blood plasma, the cultures grew. In non-carious extracted teeth, the invasion of dentinal tubules by bacteria was analyzed, as well as the behavior of the different bacteria when exposed to collagen or blood plasma.

If, on the one hand, *streptococcus mutans* and *lactobacilli* adhere to and subsequently invade the dentinal tubules (partially inhibited by the human blood plasma), on the other hand *enterococcus faecalis* was seen to grow in the presence of human blood plasma. In the dentin, the presence of plasma prevented the invasion of both *streptococcus gordonii* and *streptococcus*

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mutans, while it did not prevent the invasion of *enterococcus faecalis* (22). The latter, a highly virulent microorganism, is all but unaffected by the presence of human blood plasma, therefore preserving its potential for invasion and keeping viable by means of adhering to collagen.

Enterococcus faecalis has been cited in the literature as a possible cause for unsuccessful endodontic treatments. Sundqvist et al. (23) carried out a microbiological analysis in teeth that had been submitted to unsuccessful endodontic treatments and concluded that *enterococcus faecalis* was linked to a significant number of such cases. The author says this is due to (a) the high resistance of this microorganism to medication used during treatment and (b) its ability to survive in the canals as a simple organism, needing no other bacteria for support.

This finding was corroborated by Fabricius et al. (24) in studies with monkeys, they isolated several bacteria indigenous to these animals and provoked periapical infections with those microorganisms. The *enterococcus faecalis* survived as pure culture in all the canals analyzed.

In relation to the difficulty medication have in acting on *enterococcus faecalis*, Han et al. (25) carried out experiments in root canals infected by this microorganism. The authors verified the difficulty in eliminating the *enterococcus faecalis* with calcium hydroxide in teeth not previously submitted to acid attack.

Given that the dentinal tubules contain a significant quantity of non-mineralized elements, *enterococcus faecalis*, once there, becomes an extremely virulent microorganism. Invasion of the tubules is linked to cellular adhesion to collagen and to the oral *streptococcus*-induced morphological growth of the collagen. These functions are mediated by the antigen I/II of the proteins of the cellular wall. Some of these antigens are involved in the invasion

of other species which do not have this remarkable invasive capacity. However, Love (21) has shown that the *enterococcus faecalis* adheres to the collagen as well, and keeps on invading the dentinal tubules in the presence of human blood plasma. Besides, the capacity these three bacteria (*streptococcus mutans*, *streptococcus gordonii*, and *enterococcus faecalis*) have of growing and keeping viable in the presence of human blood plasma for a long time has come to prove that bacteria involved

in the processes of carious lesion and endodontic disease are capable of obtaining nutrition from the tissual fluids.

Dentinal permeability

The tubular composition makes the dentin a structural element where substances are diffused due to its extension. Such characteristic is of paramount importance to the dentinal adherence of restorative materials. On the other hand, the same dentinal tubules will function as canals for bacterial penetration and endotoxin invasion to the pulp.

When analyzing the diffusion of niacin and the growth of *l. arabinosus* in extracted teeth both with and without preparation *in vitro*, Brown et al. (26) observed that in teeth with exposed dentin the diffusion of niacin was nine times higher than in non-prepared teeth, and there would be a significant growth of bacteria depending on the concentration of niacin. This study showed the action of both a growth factor and a microorganism. In the natural process of a carious lesion, several essential metabolites and a large number of bacteria are present, which could produce similar results to those of that 1962 study.

Pashley et al. (27) compared *in vitro* the dentinal permeability in carious lesions and in sound teeth, finding it is comparatively lower in the former. Nonetheless, when the smear layer was removed, the permeability of carious teeth increased significantly.

With a study in dogs, Pashley et al. (28) prepared cavities in first molars of the animals and observed the changes in their dentinal permeability in terms of the preparations. They verified a decrease lower than 20% from the normal permeability within six hours of follow-up. However, this decrease took place only in teeth with an intact wholesome pulp.

Miller and Massler (29) verified different levels of dentinal permeability in carious lesions of different characteristics. The carious lesions with a softened consistency and light brown coloration, taken to be active, are more permeable than lesions with a firm consistency and dark brown coloration, taken to be chronic. But one should bear in mind, as discussed previously, that the presence of bacteria between the disorganized dentinal zone

and the healthy dentinal tubules will contribute to a long lasting infectious process, once they thrive on a substratum from the organic portion of the dentin.

Watts and Paterson (30) carried out a study in order to verify bacteria in histological sections in rat teeth. Occlusal cavities were produced in the molars of the animals, and were divided into two experimental groups: cavities with and without pulp exposition. Euthanasia of the animals was performed at different times: immediately, 3, 7, 14, and 28 days. In the group without pulp exposition bacteria could be observed in the dentin in practically all sections analyzed. In those teeth with pulp exposition, 3-day euthanasia, a dense inflammatory infiltrate could be seen, as well as areas of pulp necrosis, and bacteria could be seen in all teeth. The other three groups (7, 14 and 28 day euthanasia) showed extensive areas of necrosis and bacterial invasion could be observed in the canals. Some teeth showed signs of periapical inflammation.

The one dentinal characteristic which helps explain this phenomenon, i.e., the bacterial invasion in deep carious lesions, is the distribution of the dentinal tubules. In the crown zone, close to the pulp, the number of tubules is twice larger than in the zone closer to the enamel. The diameter of the tubules also varies: close to the amelodentinal limit, it is 900 µm, while the deeper it gets into the dentin and the closer to the pulp, this diameter increases up to 2.5 µm.

Penetration of endotoxins

Given that the nature of the dentin is permeable due not only to the number of tubules but due also to their diameter, besides bacterial invasion, the dentinal tissue is susceptible to endotoxin penetration. Soluble irritants and inflammatory stimuli spread from the carious lesion through the dentin, and they may be deemed etiologically important (2).

Nissan et al. (31) carried out an *in vitro* experiment with five human teeth (extracted third molars), preparing cavities in their occlusal and pulpal faces, leaving a 0.5-mm layer of dentin separating the two cavities. Observing the passage of endotoxins through this dentin wall, they verified they passed relatively fast in four out of the five

teeth. It should be noted, though, that the teeth were no longer vital, i.e., there was no hydrostatic pressure from the inside to outside of the pulp, something which most probably reduces endotoxin penetration.

Pulpal reactions

Pulpal response to a carious dentin represents an immunologic phenomenon which is launched by components or substances produced in the carious lesion itself. These components, once they cross the dentin, they have access to the pulp through the dentinal tubules (32).

In an attempt to explain the evolution of the defensive reaction of the pulp to these intense stimuli, Shroff (33) described degrees of supraoptimal stimulation. In the first degree of stimulation, the intensity is discreet enough to create changes only to those portions of the odontoblast closer to the area involved. Metabolic changes then take place, leading to the degeneration of the extremities which were affected by the odontoblastic prolongations, and these areas will undergo an intense deposition of calcium salts. Thus, zones of common reactional sclerotic dentin will develop under either carious lesions of slow progression or other mild irritations. At the pulpal level, the odontoblasts undergo partial atrophy, and the formation of dentin in theses areas is hindered.

The second degree of stimulation represents an injury able to cause degeneration of the odontoblastic prolongation throughout its total extension, whereas at the pulpal level the odontoblastic reorganization sets off the formation of a dentinal barrier. The odontoblastic prolongations, deprived of their connection with the pulp by this barrier of reparative dentin lose their function completely, and leave inside the dentin a zone of empty tubules, or else a zone of tubules with residues of cell prolongations, both identified as a dead tract or zone of infarcted dentinal tissue.

In the third degree of stimulation, trauma is so severe that the odontoblasts eventually deteriorate. The more severe stimuli may lead to complete destruction of the odontoblasts and to an inhibition of the capacity for recovery of the pulpal tissue. Under such conditions, adequate therapeutic measures are called for, entirely

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dependent on an exact diagnosis of the clinical condition of the pulp.

Brännström and Ove Lind (34) carried out an experiment with human teeth, analyzing under microscope the behavior of the pulp in relation to carious lesions at their initial stage. Changes in the pulp were observed, adjacent to lesions in the enamel with or without cavitation in 50 out of the 74 teeth examined. In 33 cases with pulpal reactions, the lesions were limited to the enamel. The most frequent pulpal changes were alignment of the odontoblastic layer, hyperchromic line in the dentin, and accumulation of inflammatory cells.

Reeves et al. (35) carried out a histopathologic evaluation of both deciduous and permanent human teeth with carious lesions, correlating bacterial penetration with degree of pulpal inflammation. They concluded those teeth that had their reactional dentinal zone invaded by bacteria presented irreversible pathological consequences.

Kuwabara and Massler (36) had teeth with both chronic and acute carious lesions examined histologically. The authors observed that pulpal reactions under active lesions were more severe than under arrested lesions, most probably because irritants had been blocked by a barrier of calcification. Deep active lesions usually will develop severe pulpal inflammations which may result in pulpal death when left untreated.

Massler (37) took into consideration the characteristics typical of carious lesions, and believed most pulpal reactions were constructive/regenerative lesions rather than degenerative lesions. However, according to Johnson (38), for dentinal sclerosis to develop, it is *sine qua non* the presence of an intact dentinal tubule inside the tubule of the affected area, and sclerosis is more pronounced in chronic than in acute lesions. In acute lesions, the typical aggressiveness of the process destroys the odontoblasts very quickly, leaving the tooth with dead tracts. According to Trowbridge (2), these dead tracts are much more permeable than the dentinal sclerosis, suggesting an accelerated evolution of the lesion.

Lopes et al. (39) state that the clinical and radiological signs of the carious dentin are no parameter for a precise diagnosis, for through them one cannot evaluate whether all of the infected tissue has been removed, and through them one cannot verify how deep the microorganisms have gone – as several bacteriologic and histological studies have shown.

According to Trowbridge (12) pulpal reaction to carious processes is an issue that raises much dispute among the authors. Massler (37) had already pointed out that the intermittency of the carious process, sometimes acute, sometimes chronic, made it difficult both to diagnose and classify the process, which is indeed characterized by this dynamicity.

Discussion/Clinical viability

After bibliographical review of the above-mentioned studies on pulp biology, microbiota and its substratum, dentinal permeability, and the penetration of endotoxins resulting in pulpal reactions, it becomes clear the carious process is dynamic: intermittent, rather than continuous, with acute and chronic phases. Many are the factors which contribute to such dynamicity and corroborate the studies that approach the caries as a multifactorial disease. Therefore, clinical diagnosis and classification of carious lesions is a rather complex task.

According to Massler (37), only the infected dentin should be removed during cavity preparation, whereas the demineralized dentin could be left there permanently. The rationale for this approach says there should be no viable bacteria left in the dentin. However, is has already been shown beyond doubt, by SEM and by anaerobic-culture methods, that the dentin, once contaminated, will always present bacteria. Therefore, an optimum substratum would be being left there, under the restorative work and, in the case of infiltration, a new path opens up for pulp infection.

Moreover, through clinical examination, it is extremely difficult to distinguish the different layers of carious dentin. Clinical evidence points to a complete removal of the caries, and this is also strongly recommended on account of an undisputable practical benefit, which is, only healthy uninfected dentin is left in the cavity. Both medical doctors and doctors of dental surgery agree that contaminated material, once present in the human body, should be removed. Thus, leaving infected material in the tooth during cavity preparation is deemed unethical.

According to Hasslegren and Calev (40), a decision not to remove carious tissue, both intentionally and permanently, constitutes technical error, and this type of procedure could be related to an indirect pulp capping.

Smulson and Sieraki (41) say it is generally accepted that bacteria do not invade the dental pulp if there is healthy dentin between the pulp and the cavity bottom. However, bacterial invasion through the dentinal tubules may occur, and even induced by clinical procedures. Anaerobic methods were used by the authors in order to demonstrate bacterial invasion in teeth with non-exposed pulp. The results of this study show that bacteria can invade the pulp without causing pulp exposure when carious lesions are present. This suggests that bacteria are probably invading the pulp continuously, and some may already have been eliminated by the immune system.

Upon invading the pulp, the bacteria may sensitize the patient. The immune system will be immediately activated when the same species of bacterium invades the pulp again, causing pain. In the case that some of these bacteria survive and multiply, pulp infection is to be expected (42).

Our current knowledge is limited in relation to the humoral immune response of the dental pulp to a carious lesion. Increased levels of immunoglobulin have been detected in cases of pulp tissue inflammation. Specific antibodies for microorganisms of the species *lactobacillus, streptococcus, bacteroides intermedius,* and *eubacterium* have been observed, with a significant variation in their levels, in healthy pulps as well as in cases of irreversible pulpitis (43). The presence of natural antibodies in the normal pulp points to their protective function during the invasion of the carious process. However, the biological meaning of the presence of immunoglobulin in the normal pulp fluid is not yet clear. It seems that the immunoglobulin detected in cases of irreversible pulpitis are most probably derived from tissual fluid, whereas those found in inflamed pulps apparently derive from cell plasma.

Aiming at avoiding endodontic treatment, for it is time-consuming, painstaking and costly, many professionals choose to leave carious tissue in the deeper layers of the cavitary preparation. This technique's rate of success is often and again evaluated by the sheer absence or presence of pain. We cannot fail to mention most pulp inflammations are asymptomatic, and our stand is in accordance with Hasselgren and Calev (40) meaning the procedure above-mentioned is unreliable. Emergencies will often attest to cases of pulp and/or periapical inflammation caused by caries that were left under restorations.

Conclusion

Carious lesions have their initial microbiota composed mainly of *streptococcus mutans.* These microorganisms will form a thick plaque, being aciduric and acidogenic. However, these are not the only bacteria involved in the carious process. It is known that *lactobacilli* and many other bacteria take active part in the development of a carious process.

If, on the one hand, it is possible to turn bacteria such as the *streptococcus mutans* and the *lactobacilli* unviable (44), on the other hand, when it comes to inactivating the carious disease, one has to consider the range of microbial interactions that are present in the process and which will bring or have already brought pernicious consequences to the dental pulp. *Enterococcus faecalis,* highly virulent, can remain viable even when it is confined under a restoration, using for substratum the very dentinal tissue produced by the lipidic and glycolipidic membranes of the odontoblastic processes (45).

One should also bear in mind, in relation to carious lesions, that the activity of the lesion is highly dynamic. In the chronic lesion, with a slower progression, some defense on the part of the tooth is possible, by way of a mineralization of the dentinal tubules and consequent protection of the dentinopulpal complex. However, in the acute lesion, with a higher permeability, bacterial invasion is facilitated, resulting in injury to the pulpal organ.

We do agree with Fejerskov and Kidd (46) when they say well controlled clinical studies are needed, together with microbiological and laboratorial studies, if one aims at clarifying the issues related to partial removal of the carious dentin and its consequences in the pulp tissue.

Clinical procedures involving the pulp should take into consideration the complex range of the various microbial, cellular, and enzymatic interactions that take place in the arterioles and venules. Given the precise and meticulous operative

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skills needed to approach these tissues, confined as they are to inelastic walls and deprived of collateral circulation, the use of conservative methods when removing the carious dentin should be seriously questioned.

This whole range of above-discussed factors makes it all but impossible to analyze in more precise terms an infection of carious dentin and even an irreversible inflammation of the pulp. Consequently, in order to make sure that our therapeutic procedure brings about a favorable prognosis, we had better not leave any carious dentin under restorations.

Evaluating the histopathologic changes that have taken place in the pulpal organ due to the presence of caries is more important than determining whether the bacteria are active or inactive in the carious lesion. It must not be forgotten that the biology of the dental pulp is the one parameter to be followed in this matter.

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