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Abstract

The histopathology of active and arrested human root caries was examined in extracted teeth by different optical methods. Significant differences were observed between the mechanisms operating on the various dental structures. Three different patterns of initial cementum and dentin lesions could be distinguished, depending on the severity of the cariogenic attack, the degree of sclerosis of the peripheral dentin, and the presence of calculus. Advanced lesions were characterized by various patterns of demineralization. In particular, a massive lateral spread of bacteria into intertubular dentin was observed. Consequently, unaffected dentinal areas became continuously undermined. In arrested lesions, either a partial or complete mineralization of the intertubular dentin was apparent. Dentinal tubules were sclerosed passively by re- or precipitation of Ca and PO₄ ions. In contrast, tubules filled with ghosts of bacteria appeared mineralized by fine-granular crystals. Our observations indicate that both the arrestment and the remineralization of active lesions depend on (1) the degree of active sclerosis of dentinal tubules in areas underlying the lesion, (2) the degree of the bacterial infection of the dentin, (3) the degree of progression of the lesions, and (4) the location of the lesions at the various root surfaces. It is suggested that remineralization of active lesions can occur. This supports the concept of non-invasive treatment of root caries lesions without cavitation.
Histopathology of Root Surface Caries

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The histopathology of active and arrested human root caries was examined in extracted teeth by different optical methods. Significant differences were observed between the mechanisms operating on the various dental structures. Three different patterns of initial cementum and dentin lesions could be distinguished, depending on the severity of the cariogenic attack, the degree of sclerosis of the peripheral dentin, and the presence of calculus. Advanced lesions were characterized by various patterns of demineralization. In particular, a massive lateral spread of bacteria into intertubular dentin was observed. Consequently, unaffected dentinal areas became continuously undermined. In arrested lesions, either a partial or complete mineralization of the intertubular dentin was apparent. Dentinal tubules were sclerosed passively by re- or precipitation of Ca and PO4 ions. In contrast, tubules filled with ghosts of bacteria appeared mineralized by fine-granular crystals. Our observations indicate that both the arrestment and the remineralization of active lesions depend on (1) the degree of active sclerosis of dentinal tubules in areas underlying the lesion, (2) the degree of the bacterial infection of the dentin, (3) the degree of progression of the lesions, and (4) the location of the lesions at the various root surfaces. It is suggested that remineralization of active lesions can occur. This supports the concept of non-invasive treatment of root caries lesions without cavitation.


Introduction.

Root caries and its sequelae are of increasing importance, particularly to the geriatric population, and present a difficult treatment problem (Jordan and Sumney, 1973; Nyvad and Fejerskov, 1982, 1987; Billings, 1986; Jones and Boyde, 1987). In contrast to the development of lesions in enamel and coronal dentin, which received much attention and is well understood (Silverstone, 1974; Silverstone and Hicks, 1985; Thylstrup et al., 1983; Thylstrup and Fejerskov, 1986; Adriaens et al., 1988), comparatively little effort has been made to elucidate the histopathology of root surface caries (Nyvad and Fejerskov, 1982, 1987; Frank et al., 1989). Root surfaces become exposed to the oral environment as a result of pathologic changes, mechanical injuries, or periodontal surgery. Caries then frequently develops on the approximal surfaces that become covered by micro-organisms and along the cemento-enamel junction. Early lesions are clinically detectable as multiple, tan to brown discolored areas. These lesions may coalesce, spread over the surface, and eventually may encircle the root and may spread apically too, as the gingiva recedes due to periodontal breakdown. If the root surfaces are still covered by cementum, this tissue may play an important and so-far-neglected role in the development of root surface caries. Studies published hitherto on lesions involving cementum and peripheral dentin (Furseth and Johansen, 1970; Kostlan, 1963) showed that "... the early stages in caries development involve a rather haphazard demineralization of this layer, in particular along bundles of collagenous fibers" (Thylstrup and Fejerskov, 1986). There is, however, no agreement on whether bacteria invade cementum and peripheral dentin. Using histological methods, some workers observed bacterial invasion (Daly et al., 1982; Adriaens et al., 1988; Schüpbach et al., 1989), whereas others using histochemical methods did not (Hughes and Smale, 1986; Moore et al., 1986). Since basic knowledge of the development of initial lesions in cementum and peripheral dentin is a prerequisite for the choice of treatment of root surface caries, this investigation was undertaken to assess the histological features of such lesions.

Furthermore, the structural and ultrastructural changes of advanced lesions, their progression toward the root canal, and their lateral spread were investigated. Recent studies by Fejerskov and Nyvad (1986) and Nyvad and Fejerskov (1987) have shown that active root caries lesions can be converted into arrested lesions in response to oral hygiene. These authors suggested that the conversion from an active to an arrested lesion is probably connected to the removal of the outer parts of softened dentin, resulting in the uptake of minerals from oral fluid and their deposition to form a new surface. Our investigations addressed the question whether a conversion of an active to an arrested lesion follows the abovementioned principles. It was shown that remineralization of the demineralized and infected dentin is more complex, involving different remineralization mechanisms in different layers of the lesion which overall depend on the severity of the lesions.

Materials and methods.

The material consisted of 108 human teeth extracted for prosthodontic reasons from 86 patients, aged 50-77 years. Immediately after extraction, the teeth were placed into half-strength Karnovsky's fixative (Karnovsky, 1965) for 48 h at 4°C. Following fixation, the teeth were washed in 0.185 M sodium cacodylate buffer. All carious lesions on the root surfaces were photographed and classified as active or arrested on the basis of clinical criteria proposed by Fejerskov and Nyvad (1986). Active lesions were subdivided into four grades according to the Root Caries Severity Index (Billings, 1986). Subsequently, one 200-µm-thick longitudinal section was cut through the centers of the lesions by use of a diamond wheel on a micro-sectioning machine. These sections were dehydrated in ascending grades of alcohol, air-dried, and photographed with an Olympus Zoom Stereo Microscope in transmitted and reflected light. Contact microradiographs were made with Kodak high-resolution film SO-343 and a Hewlett-Packard Faxitron unit. Radiation at 80 kV and 2 mA was applied for four min.

The remaining right halves of the lesions were decalcified, post-fixed, dehydrated, and embedded in Epon 812 as described previously (Schüpbach et al., 1989). Semithin sections were stained differentially in periodic acid-Schiff (PAS) and methylene blue-Azur II (Schroeder et al., 1980) or in toluidine blue. From selected areas, ultrathin sections were prepared.
These sections were double-contrasted with uranyl acetate and lead citrate and examined in a Phillips 201 transmission electron microscope.

The remaining left parts of the lesions were immersed in liquid nitrogen for 60 min, and the teeth were fractured by use of a sharp scalpel and a hammer. Specimens were post-fixed, dehydrated in ascending grades of alcohol, and finally embedded in Epon 812 as described previously (Schüpbach et al., 1989). Miniature pyramids were prepared with files. Semithin sections were stained with toluidine blue. Some ultrathin sections were double-contrasted with lead and uranyl salts, whereas others remained unstained.

Eight teeth were prepared for scanning electron microscopy. The teeth were fractured as described above. Following initial fixation, the teeth were post-fixed, dehydrated in ascending grades of acetone, and dried in a critical-point apparatus (Balzers, Liechtenstein), with carbon dioxide used as the tertiary fluid (Anderson, 1951). The specimens were sputter-coated with gold and examined in a Cambridge Stereoscan S-180 SEM.

Results.

A total of 108 teeth was examined. Twenty-four teeth had either incipient (grade 1) or shallow (grade 2) lesions. Sixty-six teeth had lesions of the cavitated (grade 3) or the pulpal (grade 4) variety. Finally, 18 teeth had lesions which were classified as arrested. The location of most of the lesions was at the cemento-enamel junction or on the cervical third of the root.

Lesions in cementum.—Clinically detectable initial lesions appeared as single or multiple, tan to brown discolored areas on exposed root surfaces. Microradiographically, such lesions had three different patterns of demineralization. The first and most predominant pattern was characterized by uniform demineralization of both cementum and underlying dentin (Fig. 1a). Usually, cementum had a higher degree of remaining mineral components than did the affected dentin. The incremental lines of cementum were accentuated (Fig. 1a). The second pattern was observed only in specimens which already showed a microcavity in the outermost peripheral layer of cementum. The demineralization in this pattern consisted of radially oriented radiolucent strips located between the bottom of the microcavity in cementum and the cemento-dentinal junction, and of halo-like areas of increased radiolucency at the endings of the strips in the peripheral dentin (Fig. 1b). The third pattern was characterized by uniform demineralization of cementum and peripheral dentin below a mineralized surface layer (Fig. 1c). The latter was 10-30 μm thick and interrupted over well-defined halo-like areas of advanced demineralization.

Uneedecalcified semithin sections through lesions, microradiographically identified to represent the second pattern, exhibited small clefts traversing the cementum and extending into peripheral dentin (Fig. 1d). Sections stained with toluidine blue revealed small stained areas at sites where the clefts penetrated the peripheral dentin (Fig. 1e). At a later stage, enlarged clefts extending from the surface into cementum, and filled with micro-organisms, were observed (Fig. 1f). Undecalcified ultrathin sections through the surface of cementum revealed irregular small spaces between bundles of demineralized collagen fibrils (Fig. 1g). In general, destruction of cementum occurred step-wise along fractures which seemed to follow the incremental lines of cementum. Then, microcavities filled with micro-organisms were formed, and as a last step, peripheral dentin was exposed (Fig. 1h). Eventually, the cementum borders were subsequently undermined by micro-organisms (Fig. 1h). The exposed peripheral dentin was covered with micro-organisms. There was no evidence of immediate bacterial invasion of peripheral dentin because in our specimens the latter was usually sclerosed, and open dentinal tubules were absent.

Initial lesions in dentin.—In exposed dentin, a step-wise destruction of peripheral dentin was evident in undecalcified semithin sections stained with toluidine blue (Fig. 2a). Areas of progressive dentin destruction could be distinguished from areas of further destruction by the different staining grades. Halo-like areas of advanced demineralization (bright-stained area D1, Fig. 2a) extended into less demineralized areas (darker-stained area D2, Fig. 2a). Beneath the surface to a depth of 10-20 μm, an unstained, mineralized layer was evident. Initial penetration of micro-organisms into peripheral dentin occurred along multiple small clefts which were oriented perpendicular to the surface (Fig. 2b). Undecalcified ultrathin sections revealed that, at this stage, the surrounding dentin was almost completely demineralized (Fig. 2c). The clefts tended to expand laterally and toward the root canal. Often they fused with each other to form microcavities filled with bacteria. As such microcavities enlarged further, the bacteria moved deeper toward the open dentinal tubules and eventually infected them (not shown). Undecalcified ultrathin sections through the exposed surface of peripheral dentin showed a layer of complete demineralization which was demarcated distinctly from the underlying fully demineralized dentin (Figs. 2d,e).

Advanced lesions in dentin.—Microradiographically, advanced lesions showed various areas of both radiolucency and radiodensity, with two patterns predominating. Lesions representing the first pattern had in common that the radiolucent areas observed in initial initial lesions appeared now as enlarged lesions which varied in extent and depth along the exposed root surface (Fig. 3a). Some of the halo-like areas were covered by a radiodense surface layer, while others were not. In addition, radiodense layers were observed bordering the radiolucent areas toward the inner dentin (Fig. 3a). Lesions representing the second pattern had only one radiolucent area, extending saucer-shaped into dentin (Fig. 3b). Occasionally, this area was covered in part by a radiodense surface layer. In some of the specimens, a band-like radiodense layer was observed within the demineralized area (Fig. 3b).

Undecalcified ultrathin sections through the radiolucent areas of lesions of both patterns revealed an almost complete demineralization of the intertubular dentin (Figs. 3c,d). The dentinal tubules and their side-branches were occupied by micro-organisms. The peritubular dentin of such tubules was absent (Fig. 3d). Sections through the radiodense surface layer showed accumulations of small needle-like crystals (Fig. 3c, inset). A massive spread of micro-organisms was observed.
The invasion of dentin occurred initially along the dentinal tubules toward the root canal and then progressed by a lateral spread to the partially demineralized intertubular dentin. Micro-organisms invading the latter were oriented parallel to the collagen fibers. By their lateral spreading, the micro-organisms frequently reached adjacent dentinal tubules containing unidentified structures. Subsequently, the micro-organisms eventually engulfed such structures (Fig. 3g). Sections through the radiodense band bordering the demineralized areas toward the inner dentin (Fig. 3d) showed that in this area the dentinal tubules were partially or totally occluded by mineral deposits (Fig. 3h).

Arrested lesions in dentin. — Eighteen lesions were classified as arrested on the basis of their dark brown to black discolored surface. Based on the histological assessment of these lesions, 12 lesions were definitively arrested, while six lesions belonged to an intermediate group showing areas of both active and arrested cavities.

Contact microradiographs of mixed lesions showed, in areas of arrested cavities, a mineralized surface layer and underneath this layer, a mineralization front advancing to demineralized dentin (Fig. 4a). Truly arrested lesions were almost completely mineralized. In dentin underlying such lesions, a distinct zone of tubular sclerosis was evident. In scanning electron micrographs of the zone of tubular sclerosis, the fillings of such tubules appeared as solid rods embedded in partially demineralized dentin (Fig. 4b). Undecalcified ultrathin sections through the rods showed that the tubular lumen was completely occluded by fine granular crystals.

Undecalcified ultrathin sections through the surface area of both mixed and arrested lesions revealed that the intertubular dentin was mineralized up to the surface. The surface was densely covered by micro-organisms (Fig. 4d). The dentinal tubules near the surface were packed with ghosts of micro-organisms (Fig. 4d). At higher magnification, these tubules showed accumulations of fine granular crystals, deposited primarily between the remaining of bacterial walls and, in more advanced stages of mineralization, inside such ghost cells as well (Fig. 4e). Sections through dentinal tubules near the surface not occupied by micro-organisms showed deposition of large irregular crystals (Fig. 4f). Sections through the mineralizing front of mixed lesions exhibited diffuse accumulations of crystals in the intertubular dentin and usually an almost crystal-free gap around the dentinal tubules (Fig. 4g). The deposited crystals were irregular in shape and dimension (Fig. 4h).

Discussion.

In our material, originating from a rural region in Switzerland, the exposed root surfaces were usually covered by cementum, indicating that they had not been subjected to repeated root scaling. In addition, the material originated from individuals representing the age group 50 to 80 years. The teeth from such individuals showed sclerotic age changes of the dentin, visible in undecalcified longitudinal sections as an increased transparency starting at the root apex and progressing coronally along the periphery of the roots with advancing age (Nalbandian et al., 1960). Recently, we reported that in such sclerosed portions of the peripheral root dentin, the dentinal tubules were completely obliterated (Schüpbach et al., 1989), and it was suggested that the progression of caries following destruction of cementum is slow because open dentinal tubules were absent in the exposed dentin and, until its destruction, the sclerosed peripheral dentin acts as a barrier to the cariogenic challenge.

The three described patterns of demineralization of cementum and peripheral dentin were discussed extensively in a previous paper (Schüpbach et al., 1989). Therefore, only major considerations are compiled in the following. We suggest that the second pattern of cementum demineralization, characterized by small areas of demineralization and by tiny clefts traversing cementum and extending into peripheral dentin, is the result of a very local, weak cariogenic challenge by bacterial plaque initially present on the intact cementum layer. These clefts were not considered to be artefacts produced during extraction or during specimen preparation, because micro-organisms were frequently present (Fig. 1f). Furthermore, demineralized areas were seen at the sites where such clefts penetrated the peripheral dentin (Fig. 1e). Demineralization most likely progresses along loci minorae resistentiae, as we designate the hypomineralized areas between traversing Sharpey’s fiber bundles or along the un- or hypomineralized cores of the fiber bundles (Selvig, 1965; Furseth and Johansen, 1970). Clefts in cementum had been previously observed by Furseth and Johansen (1968), who interpreted these spaces as “micro-channels” allowing an exchange of fluid from the surface to the underlying dentin. The first types of initial lesions (pattern 1, Fig. 1a) prevailed in our material. This type is characterized by a uniform demineralization of both cementum and the underlying dentin. It seems to be the consequence of a more vigorous cariogenic challenge. The third type of initial lesion (Fig. 1c) developed apparently only on calculus-covered cementum, as demonstrated recently (Schüpbach et al., 1989). We suggest that these halo-like lesions are the result of a moderate cariogenic attack affecting a relatively narrow area after the dissolution of the calculus layer.

Our findings indicate that the penetration of micro-organisms into cementum starts primarily along small clefts and then progresses into deeper layers of cementum (Fig. 1f). By the step-wise destruction of cementum along the incremental lines, resulting in microcavities, micro-organisms finally cover the exposed peripheral dentin. In our specimens, there was no evidence of a direct vertical continuation of bacterial penetration into the exposed sclerosed and tubule-free peripheral dentin. This is in contrast to the findings of Adriaens et al. (1988), who described bacterial invasion in cementum and peripheral dentin of human teeth affected by periodontal destruction. These authors observed the presence of micro-organisms in lacunar defects in cementum. These lacunae—the result of dentinoclast activity—extended into the peripheral dentin, and in some of their specimens micro-organisms invaded the open dentinal tubules. However, their paper does not disclose from which age group the teeth were obtained, and it cannot be excluded that the peripheral dentin in their specimens was not sclerosed, and probably the peripheral dentin had been removed by repeated scaling.

Exposed dentin was covered by a compact layer of micro-organisms. The acids produced by this layer led to widespread
HUMAN ROOT CARIES

(a) [Image of a sectional view with labeled areas D1, D2, and D3, measuring 50 μm.]
(b) [Image of a different sectional view with labeled area D, measuring 20 μm.]
(c) [Image with labeled areas M and D, measuring 1 μm.]
(d) [Image with labeled areas M, D, and D*, measuring 10 μm.]
(e) [Image with labeled area D*, measuring 1 μm.]
Fig. 3 — Advanced stages of carious lesions in dentin. (a,b) Microradiographs demonstrating the multifocal demineralization of dentin and the occurrence of mineralized layers at the surface (arrowheads) and in deeper parts of the lesion (arrows). (c,d) Undecalcified ultrathin section through carious dentin showing a mineralized surface layer (arrow in c and inset) and infected dentinal tubules and side-branches (d). Note the demineralized intertubular dentin and absence of peritubular dentin. (e) Decalcified semithin section and (f) corresponding ultrathin section revealing lateral spreading of micro-organisms into intertubular dentin (arrows). (g) Decalcified ultrathin section through a dentinal tubule occupied with micro-organisms surrounding unidentified structures (arrow). (h) Undecalcified ultrathin section through a dentinal tubule partially occluded by needle-like crystals. [Figs. 3 a, b, d-h from Schüpbach et al. (1990): Caries Res 24:145-158.]
demineralization within both the peripheral tubule-free and the underlying tubular dentin (Fig. 1a). Ions from the dissolved mineral diffusing outward were then in part re-precipitated to form a new mineralized layer located 10 μm below the dentinal surface, consisting most probably of hydroxyapatite crystals (Schüpbach et al., 1989). The bacterial plaque covering exposed dentin also affected the collagenous matrix of intertubular dentin, most likely because extracellular bacterial enzymes diffused into peripheral dentin. This was evident in stained, undemineralized semithin sections, which showed halo-like, pale, stained areas of advanced demineralization extending into intensively stained areas, while the mineralized surface layer remained unstained (Fig. 2a). This could only be the result of a complete masking of the collagenous matrix in the mineralized surface layer, while in the pale, stained areas the organic matrix was partially destroyed. We conclude that the dissolution of apatite precedes the destruction of the collagenous matrix of the intertubular dentin. This is in accordance with recently published results of Frank et al. (1989).

In the absence of open dentinal tubules, initial penetration of micro-organisms into cementum-free dentin occurred along small clefts as shown in Figs. 2 b, c. We are not able to decide whether these clefts represent the continuation of the clefts traversing cementum, or whether these clefts developed only after complete destruction of cementum. In this context, it is of interest that the Sharpey’s fibers seem to be anchored in the peripheral dentin (Furseth, 1974; Schüpbach et al., 1989). The possible importance of these fibers as foci for bacterial invasion and root caries progression has been emphasized above. Taken together, we suggest that the resulting, histologically distinguishable reaction patterns observed during initial stages of root surface caries depend on both the strength of the challenge and the structural features of cementum and peripheral dentin.

Demineralization and invasion of peripheral dentin with micro-organisms seem to be the prerequisite for the further progression of the lesion, which may follow different patterns. Once open dentinal tubules are reached, the micro-organisms invade dentin primarily by following the tubules toward the root canal. Undecalcified ultrathin sections through lesions of this stage revealed that the peritubular dentin was destroyed and the intertubular dentin was almost completely demineralized (Fig. 3d). In addition to this progression of micro-organisms toward the root canal, a lateral spread of micro-organisms was observed which occurred independently from the side-branches of the dentinal tubules (Figs. 3e, f). By this lateral spread, intertubular dentin became heavily infected with micro-organisms which were oriented parallel to collagen fibers (Schüpbach et al., 1990). This could explain the saucer shape of root caries lesions and their tendency to spread laterally over the root surface, which is in contrast to lesions in coronal dentin, where the micro-organisms follow mainly the curvature of the dentinal tubules.

Microradiographs of advanced lesions reveal a further phenomenon which needs to be addressed. Apart from a mineralized surface layer, mineralized bands were observed that either bordered the lesion peripherally toward inner dentin (Fig. 3a) or were located within the lesion (Fig. 3b). Although the origin and the location of mineralized bands bordering the lesions can be explained as the consequence of tubular sclerosis occurring in areas underlying the lesions (Fig. 3h), the location of mineralized bands within the lesions remains unclear. Ultrathin sections through such areas revealed that the intertubular dentin was partially mineralized by the accumulation of large crystals similar to hydroxyapatite. That suggests that these layers either represent further remineralized areas formed by precipitation of outwardly diffusing ions, or are the result of an irregular destruction of the dentin layers leading to a “banded” aspect. However, the question of their location remains to be answered. Interestingly, such bands located beneath the anatomical surface were a common observation in in vitro models of root surface caries (Wefel et al., 1987). These authors showed that the areas between the surface and the bands were nearly devoid of mineral, and they suggested that “...it is possible that an organic matrix which has lost certain components will not act as an effective crystal growth substrate. Likewise, a certain level of mineral may be needed in order to induce crystal growth or remineralization” (Wefel et al., 1987).

The conversion of active lesions into arrested lesions is a well-known phenomenon. Several authors described structural and microbiological differences between active and arrested lesions, but most of these studies were performed on coronal dentin (Miller and Massler, 1962; Sarnat and Massler, 1965; Levine, 1973, 1974; Parikh et al., 1963). Only Nyvad and Fejerskov (1987) described the occurrence of a significant uptake of minerals from oral fluid into the surface layer of arrested lesions on roots. This observation confirmed results from earlier investigations reporting arrested lesions in coronal dentin (Sarnat and Massler, 1965). However, it remained open to which extent an active lesion can remineralize and what parameters govern such an event. The major characteristics of arrested lesions were a mineralized surface layer and a front of mineralization advancing toward the root canal in areas of demineralized dentin (Fig. 4a). They seem to be the result of two different mechanisms, namely, a precipitation of salivary components and a reprecipitation of dissolved and outward-diffusing calcium ions. The deposited crystals were first scattered and randomly distributed between the dentinal tubules. This was most obvious in areas of advancing remineralization (Figs. 4g, h). Our study confirmed observations that active caries, arrested caries, and remineralization may all be found within the same lesion (Takuma et al., 1975; Duculsi et al., 1979). Hence it is difficult to distinguish clearly between areas of arrested caries undergoing remineralization and areas of active caries showing ongoing demineralization. However, we observed, in the intertubular dentin of arrested lesions, a rather irregular precipitation of crystals which led to a mineralization pattern in arrested lesions that is clearly distinguishable from that of normal sound dentin (Fig. 4f). Furthermore, the crystals of arrested lesions had irregular widths and lengths and resembled hydroxyapatite. This is in accordance with data from Duculsi et al. (1979) which showed that in arrested coronal lesions the crystals of intertubular dentin were hydroxyapatite and much thicker than crystals from sound dentin. The same authors concluded from their results that the remineralization of intertubular dentin occurred by growth of residual crystals in the tissue.

A zone of sclerosis was observed in our material which formed an advanced guard between the lesion and the root canal (Fig. 4b). The degree of tubular sclerosis, which also occurs in dentin underlying active lesions, is the basic condition for a successful arrestment of further progression. By the reduced permeability of dentin, the endogenous diffusion of substrate from the pulp to the bacteria is interrupted. It is generally thought that the crystals deposited in the lumen of dentinal tubules are actively synthesized by the odontoblast processes (Frank and Voegel, 1980). Therefore, the conversion of an active to an arrested lesion should also depend on the integrity of the layer of odontoblasts in the pulp. In fact, we found several teeth with an almost completely occluded root canal and an injured, irregular layer of odontoblasts (Schüpbach et al., 1990). In these specimens, the tubular sclerosis underlying the caries lesions was absent, and micro-organisms were found in the dentinal tubules near the pulp.
A completely different type of mineralization occurred in dentinal tubules near the surface not occupied by bacteria. These tubules showed intratubular deposition of large crystals resembling those described by Vahl et al. (1964) in sclerosed tubules of coronal caries (Fig. 4f). Other dentinal tubules, located nearby, were occluded by small crystals with the characteristics of hydroxyapatite (not shown). In contrast, tubules filled with ghosts of micro-organisms were mineralized by fine granular amorphous crystals (Fig. 4e).

By the abovementioned mechanisms, surface lesions may
remineralize completely before a mineralized surface layer covers the entire surface. In the case of deeper lesions, however, a compact surface layer may form, preventing the influx of ions into the body of the lesion and thus preventing complete remineralization. We therefore suggest that the degree of remineralization is also largely dependent on the severity of an existing lesion. Interestingly, we never observed remineralization in areas of dentin infected by laterally spreading micro-organisms. This indicates that remineralization also depends on the degree of bacterial infection. However, it must be emphasized that advanced lesions could be converted into arrested lesions, when the softened and infected outer dentin was lost and a cavity was formed. In such cases, the remineralization occurred at the floor of the cavity. Finally, all lesions observed in our study showing complete remineralization were located at the lingual or buccal aspects of the teeth and not approximally, indicating that remineralization also depended on the location of an active lesion.

The occurrence of carious lesions in roots seems to depend on a number of factors, of which only some could be assessed in the present study. The history of the examined teeth remains unknown, i.e., the strength of the caries challenge (diet and bacteria) and the time-point and duration of exposure to this challenge. Our description of early root caries in elderly people helps explain why the progression of root caries is so inhibited by cementum and sclerosed portions of peripheral dentin. Optimal oral hygiene, fluoride, and dietary control are beyond any doubt valuable measures to prevent root caries lesions, but our findings indicate that repeated scaling and root planning may reduce the resistance against the caries attack because of the removal of calculus-, cementum-, and tubule-free dentin layers. Furthermore, our study shows that demineralized dentin has, under certain conditions, a great potential to remineralize. The treatment concept of active root surface caries, therefore, should be reconsidered.

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REFERENCES


