Enamel remineralization: how to explain it to patients

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The term remineralization of initial enamel caries *is frequently used, but mainly at research conferences and in lecture rooms. Clinicians avoid discussing remineralization and white-spot enamel lesion formation with patients because details of the processes are complex. Patients, therefore, incorrectly assume that cavitation occurs right at the onset of caries and that a restoration must be placed to halt further progression of the lesion. Although laboratory and clinical studies have shown that initial white-spot enamel lesions can remineralize, patients have little or no access to this information. The schematic diagrams in this paper explain the diffusion of organic acids from plaque between enamel crystals, the partial loss of mineral from enamel crystals, and subsequent demineralization and remineralization that produces the white-spot lesion. Further, these diagrams may help clinicians explain remineralization as simply as possible to patients who present with white-spot enamel lesions.*
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Introduction

Healthy enamel, although well mineralized at eruption, undergoes continual chemical and physical changes throughout the life of the individual. The chemical changes include enamel maturation, caries, and erosion, while the physical changes may include various types of abrasions, cracks, and fractures. Enamel maturation is the result of the continuous dynamic exchange of ions from pellicle, plaque, and oral fluid into the surface enamel and vice versa. However, caries results from the dynamic interaction of demineralizing and remineralizing phases on enamel covered by an acid-producing plaque. In contrast, enamel erosion occurs when surface enamel crystals are totally etched by dietary or regurgitated acids, without the involvement of plaque bacteria.

The incidence of caries has decreased in several industrialized countries.[1–3] Unquestionably, the reductions are mainly due to administration of fluoride through different vehicles (dentifrices, drinking water, rinses, table salt, tablets, and topical applications).[4] The exact anticarious mechanisms of fluoride are not fully understood, but several well-founded theories have been proposed. They include the ability of fluoride to remineralize enamel and the effect of fluoride on enamel dissolution, bacterial plaque metabolism, surface tension changes of the cell walls of plaque bacteria, and tooth morphology. However, the potential of fluoride to remineralize initial enamel lesions is accepted to be its main mode of action.[5,6]

The purpose of this paper is to summarize the demineralization and remineralization processes using schematic diagrams. The diagrams could also be used to explain remineralization to patients and to stress the advantages of remineralizing initial lesions instead of placing restorations once cavitation has occurred.

Physicochemical nature of enamel crystals

A basic knowledge of enamel crystal structure is needed to grasp the principles of remineralization. Enamel is an impure biologic mineral consisting mainly of several types of apatitic crystals (hydroxylapatite, fluorohydroxylapatite, or fluoroapatite). In addition,

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other crystalline and amorphous forms of calcium and phosphates are also found in enamel (dicalcium diphosphate dihydrate and octacalcium phosphate). Apatite crystals have a lattice containing calcium and phosphate, hydroxyl, fluoride, carbonate, and other ions (Fig 1).[7] The lattice is enveloped by a layer of adsorbed ions and by a hydration layer containing calcium, phosphates, carbonate, bicarbonates, fluoride, hydroxyl, magnesium, and other ions. Stoichiometrically pure hydroxylapatite ($Ca_{10}[PO_4]_6[OH]_2$) has a calcium-to-phosphorus ratio of 1.67 mol/L. However, natural enamel crystals are deficient in calcium and hydroxyl ions, and these vacancies produce calcium-to-phosphorus ratios of less than 1.67 mol/L. Inorganic ions make up 95% by weight of the enamel, while the organic material (1%) is almost equally divided between lipids and proteins.[8] Although the hydration layer constitutes about 4% by weight of enamel, it makes up almost 15% of its volume. It is considered that fluoride exerts its main anticarious effect in this liquid phase.

Demineralization

Based on the diagram of crystals in Fig 1, enamel just prior to eruption may be schematically depicted as shown in Fig 2. However, the enamel surface soon undergoes changes in the oral environment. These changes can be positive, resulting in a better mineralized, more mature enamel. However, negative changes can also occur, particularly when the enamel is covered with plaque (Fig 3).

A continual exchange of calcium and phosphates (ionic and bound forms) normally occurs between plaque and the oral fluids (Figs 3 to 5). In addition, fermentable carbohydrates diffuse into plaque, where they are metabolized and produce organic acids of low pK (lactic, formic, pyruvic) and high pK (butyric, propionic, acetic).[9] These acids may be buffered by the various systems available or may diffuse within the plaque or into the oral fluid. However, they may also cross the plaque-enamel interface and partially demineralize the surface and subsurface crystals (Fig 4). Factors affecting the movement of calcium and phosphate ions include their concentrations, diffusion rates, and prevailing pH values.[8]

The organic acids that cross the plaque-enamel interface in either dissociated or undissociated forms can diffuse along the liquid phases between the enamel crystals (Fig 4) or within the crystals themselves.[8,10] Again, dissolution constants, pH values, concentration, and other diffusion factors govern the diffusion rates of the acids into the enamel and the mineral loss from the crystals. The acids desorb or dissolve ions from either the layer of adsorbed ions, the lattice, or both. However, the crystals remain intact because only some of the calcium, phosphate, and other ions originally present in the crystals are lost. Consequently, the affected crystals are termed *partially demineralized* or *hypomineralized* (Figs 4 and 5). On the average, hypomineralized crystals are smaller than their original counterparts.[11] The desorbed hydroxyl ions have the potential to neutralize the acids,[12] probably acting as a self-limiting system.

Demineralization and remineralization

Calcium, phosphate, and other ions dissolved from the crystals can diffuse along the hydration layers or the layers of adsorbed ions. Adjacent crystals, unaffected by the acid, may adsorb these dissolved ions. Such crystals may accumulate a higher mineral content than before the demineralization, are on the average larger than normal crystals,[11] and are termed *hypermineralized* (Fig 4).

The number of acid attacks on the enamel surface increases with a persistent cariogenic challenge. This increases the number of hypomineralized and hypermineralized crystals, and the dissolved ions can diffuse in all directions within and between enamel crystals. These ions can then be adsorbed by hypomineralized crystals and may be termed *remineralized crystals* because of their increased mineral content. The mineral in remineralized crystals may equal or even exceed their original content prior to the partial demineralization (Fig 5). Finally, the dissolved calcium and phosphate ions may also diffuse across the enamel-plaque interface into the plaque.

The role of fluoride

Fluoride is present in the oral fluid (0.02 ppm), in plaque (100 ppm), and in high concentrations in the outer 5 μm of the surface enamel. Several theories have been proposed to explain the role of fluoride in the demineralization and remineralization processes. Fluoride ions can diffuse within the plaque (Fig 3), or they can cross the plaque-enamel interface in either the ionic form or bound as hydrofluoric acid (HF) (Figs 4 and 5). The fluoride in plaque probably decreases the total amount of acid that could cross the plaque-enamel interface. This is because HF is formed

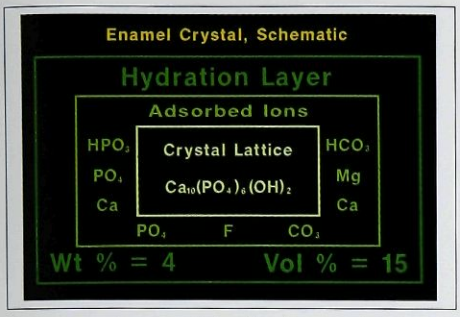

Fig 1 An apatite crystal showing the crystal lattice, the layer of adsorbed ions, and the hydration layer. (Modified from Neumann and Neumann.[7])

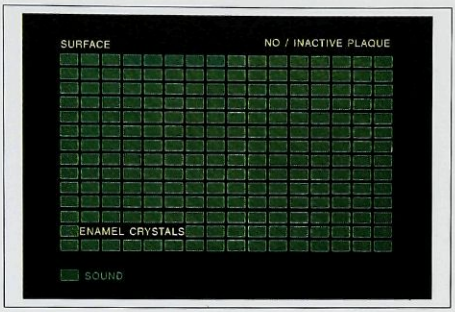

Fig 2 The enamel surface is composed of numerous apatite crystals such as the crystal shown in Fig 1.

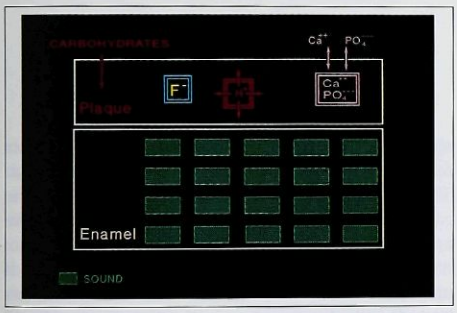

Fig 3 Surface enamel apatite crystals at the enamel-plaque interface before any (H^+) acids have crossed the interface. A continuous exchange of calcium and phosphates occurs between the oral fluid and the plaque, depending upon pH, diffusion coefficients, and concentrations. Fluoride is also present in the plaque. Fermentable carbohydrates diffuse into the plaque from which acids are formed that can diffuse in all directions.

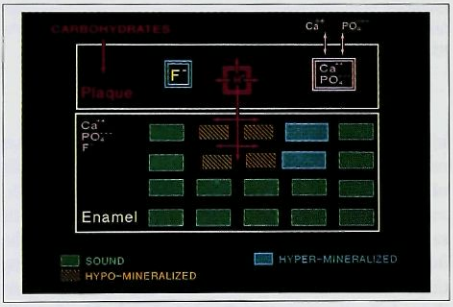

Fig 4 Acids crossing the enamel-plaque interface. Sound enamel crystals have been partially demineralized (hypomineralized) by the acids. They are smaller than the sound crystals but are not totally dissolved. The surface remains intact but more porous. Sound adjacent crystals have adsorbed the dissolved calcium and phosphate ions and have become larger (hypermineralized). Fluoride ions in the enamel fluid phase facilitate the adsorption.

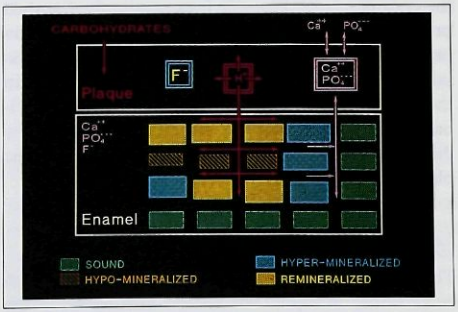

Fig 5 The progressive diffusion of acids across the intact enamel-plaque interface. Subsurface crystals have been partially demineralized (hypomineralized), while other sound crystals have become hypermineralized by adsorbing the dissolved calcium and phosphate ions. Previously hypomineralized crystals have also adsorbed dissolved mineral and become remineralized. Dissolved calcium and phosphate may also diffuse into the plaque. From there the mineral could diffuse either into the oral fluid or back into the enamel.

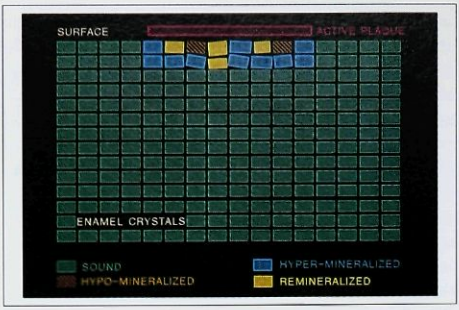

Fig 6 A small initial enamel lesion covered with plaque, which is actively producing acids. The relative sizes of the sound, hypomineralized, hypermineralized, and remineralized apatite crystals and their changed spatial orientation to each other are shown. The chemical and physical changes of the crystals in the initial lesion reflect light differently, producing the clinically visible, opaque "white spot."

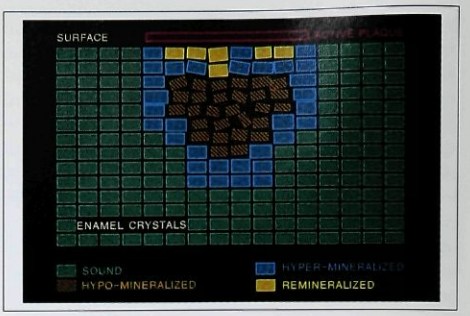

Fig 7 A more advanced initial enamel lesion that is covered with active plaque. The body of the lesion, consisting mainly of hypomineralized crystals, and the hypermineralized layer are seen, although the surface layer is intact.

in plaque when the pH of the plaque drops and is readily transported across the walls and into the cells of plaque bacteria.[13]

In addition, fluoride already present in the crystals reduces the rate of acid demineralization because the ions must first be desorbed from the crystal during the demineralization process. It is thus possible that fluoride in the crystal will minimize the demineralization at any pH reached during a drop in pH.[14] Fluoride ions in the liquid phase (hydration layer and layer of adsorbed ions) also encourage the remineralization of partially demineralized crystals using mineral derived either from other crystals or from the plaque. The inclusion of fluoride into enamel crystals at the expense other ions, such as carbonate, increases the crystallinity.[14,15] Fluoride ions previously desorbed or dissolved from the surface enamel crystals may also be taken up by other sound or hypomineralized crystals and expedite the subsequent uptake of mineral in those crystals, thus increasing their crystallinity. Finally, fluoride may also be lost from the enamel by diffusing across the enamel-plaque interface into the plaque (Figs 4 and 5).

Lesion progression or regression

The early enamel lesion represented in Fig 5 can also be shown as in Fig 6. The enamel directly below the plaque contains sound, hypomineralized, hypermineralized, and remineralized crystals, while the surface remains intact and without cavitation at this stage. The enamel surface remains without cavitation because it is remineralized by mineral from the plaque and oral fluid or by mineral dissolved from the surface or subsurface crystals. However, the intact surface is more porous than that of sound enamel. Some authors have described micropores in the "intact outer layer" and these may represent localized areas in which some crystals have been totally dissolved.[16]

Enamel caries, however, is a continuous dynamic process characterized by phases of acid production alternating with phases of virtually no or minimal acid production in the plaque on the enamel. Minerals in the plaque or oral fluid diffuse through the intact surface layer and remineralize the partially demineralized crystals during the phase of minimal or no acid production.[11] If demineralization exceeds remineralization, the number of partially demineralized crystals continually increases, forming the body of the cone-shaped initial enamel lesion (Fig 7).

The body of the lesion is surrounded mainly by sound and hypermineralized crystals which have adsorbed the mineral lost from crystals in the body of the lesion. Figure 8 is a polarized light photomicrograph of an initial enamel carious lesion prepared from an extracted tooth. The body of the lesion and the hypermineralized layer correspond to the orange and blue, respectively, seen in Fig 7. Cavitation of the

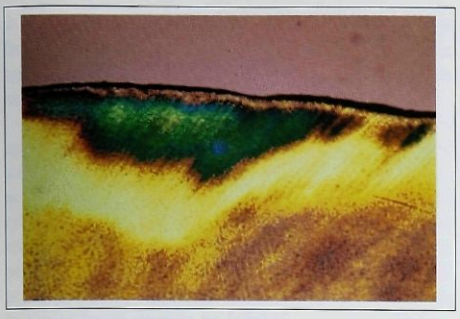

Fig 8 Polarizing light photomicrograph of an initial carious lesion in a tooth extracted for orthodontic reasons. (Original magnification ×60.) The body of the lesion *(green)* is surrounded by a hypermineralized layer *(orange and yellow)*, while the surface layer is intact (compare with Fig 7).

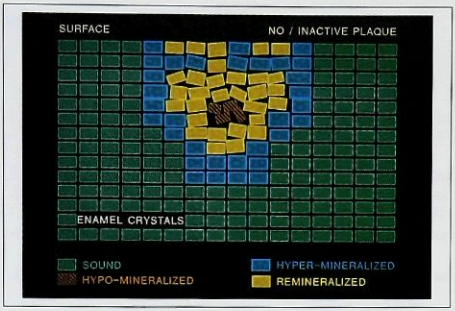

Fig 9 A remineralized initial enamel lesion. The enamel surface is either plaque-free or covered with an inactive plaque that is producing minimal amounts of acid. The previously hypomineralized crystals in the body of the lesion have now been remineralized by mineral from the oral fluid. The volume of the body of the lesion has thus decreased and the carious process has been arrested.

enamel surface probably occurs because at some stage the continually enlarging body of the lesion becomes so undermined that it cannot support the crystals constituting the intact outer surface.

In contrast, the remineralizing phases will exceed the demineralizing phases if the acid production by the plaque is low or if there is no plaque. Calcium, phosphates, and other ions of the oral fluid can continue to diffuse across the intact surface enamel into the body of the lesion. Fluoride ions are preferentially taken up by partially demineralized enamel compared to sound enamel.[17] The hypomineralized crystals in the body of the lesion become remineralized or hypermineralized by adsorbing the calcium, phosphates, and fluoride ions. Consequently, the volume of the lesion's body decreases and its intact outer layer increases in width[11] (Fig 9). The presence of fluoride, particularly in the liquid phase of the crystals, expedites the redeposition of calcium and phosphates into the partially demineralized crystals.

Discussion and clinical implications

A better understanding of the initial enamel carious lesion, or white spot, is the clue to its professional treatment. Compared to translucent sound enamel, white spots are opaque because their crystals have changed physical properties and a different chemical make up. These changes are produced by the alternating demineralizing and remineralizing phases. The orientation of the ions in hypomineralized, remineralized, and hypermineralized crystals differs from that of their sound counterparts. The spatial configuration of the crystals reflects light off the enamel surface differently in an initial carious lesion than it does off the sound surface.

Further, hypomineralized crystals are smaller, while remineralized and hypermineralized crystals are larger, than their normal counterparts. Other crystalline forms of calcium and phosphorus formed during the remineralizing phases are also present in white-spot lesions. Hypermineralized and remineralized crystals have higher concentrations of calcium, phosphorus, and fluoride ions, but less magnesium and carbonates than do sound enamel.[18] These physical and chemical changes account for the opaque white appearance of the initial enamel lesions irrespective of whether they are actively progressing or already remineralized.

Nondestructive diagnosis of initial enamel lesions

Smooth surface and proximal enamel caries can progress to dentinal caries within 1 to 3 years, depending on the cariogenic challenge and the host's defense. Initial enamel caries is diagnosed optically, radiographically, with a probe, or with fiber-optics. Proximal and smooth surface enamel that is diagnosed as

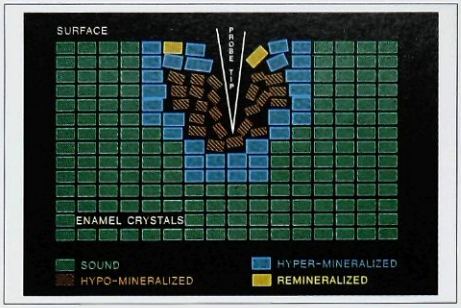

Fig 10 An active initial enamel lesion. The previously intact enamel surface has been accidentally disrupted by the probe tip during the dental examination. The disruption has produced an opening to the oral environment.

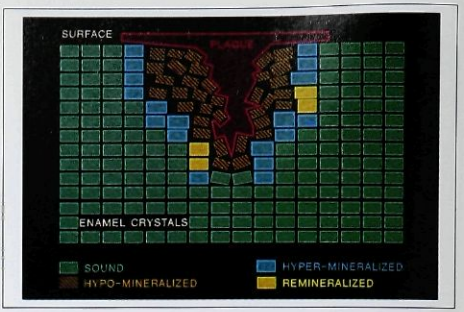

Fig 11 An active initial enamel lesion showing the consequences of the disrupted surface layer. Plaque has extended through the opening into the body of the lesion. Mechanical removal of this plaque is impossible and acids formed in the lesion can more easily reach the enamel-dentinal margin.

caries-free more often than not has microscopic initial enamel caries.[11] The dentinal changes under an initial enamel lesion can extend up to 200 µm into dentin when first visible on bite-wing radiographs. Care must be exercised not to disrupt the integrity of the intact surface enamel, particularly proximally and in fissures, when sharp probes are used during a dental examination. Experimentally produced initial enamel lesions that have been purposely damaged by probes increase in size and width more rapidly than do non-traumatized lesions.[19]

Figures 10 and 11 depict the effect of disrupting the intact outer surface of an initial enamel carious lesion. A probe tip that has accidentally been pushed through the surface into the body of the lesion (Fig 10) mechanically destroys some hypomineralized crystals. This defect fills rapidly with plaque, which is then impossible to remove because it extends into the many inaccessible areas between the damaged crystals (Fig 11). Bacteria in the plaque rapidly multiply in this "protected" environment and the acids produced from fermentable carbohydrates can more readily diffuse towards the enamel-dentinal margin. The possibility of subsequent remineralization is thus markedly reduced, if not totally lost. Such iatrogenic accidents can be avoided by conducting a more careful dental examination and using "a sharp eye and a blunt probe."

Optimizing the remineralizing potential

Initial enamel lesions are ideally treated by reducing the frequency and duration of the cariogenic challenges, optimizing the patient's oral hygiene habits, and by using fluorides. The processes of demineralization and remineralization could be explained as simply as possible to those patients presenting with initial enamel lesions. The fact that cavitation has not yet occurred should be stressed. This alone may motivate patients to maintain good oral hygiene and to adopt a noncariogenic or less-cariogenic diet.

The treatment of white-spot lesions involves both the professional dental team (dentist, hygienist) and the patient. However, active patient involvement is the more important prerequisite for the successful remineralization of diagnosed initial lesions. Equally important is the care clinicians must take to avoid "iatrogenic cavitation" of the initial enamel lesions during the dental examination.

Remineralization of initial enamel caries is encouraged by ensuring the regular availability of low fluoride concentrations in the oral fluid.[5,6] Fluoride should be delivered by the patients themselves and professionally by the dental team. If a basic, passive fluoridation project is available (water or salt fluoridation) patients should still brush with fluoride dentifrices (0.10% to 0.15% F^-) and use fluoride rinses (0.02%

to 0.05% F^-). Other methods of ensuring a constant fluoride supply include fluoride tablets and topical applications by the patient (up to 1.23% F^-) using individually fabricated polyethylene trays.

Topical fluorides (up to 1.23% F^-) should also be applied professionally, and regular recalls should be scheduled according to the initially assessed carious activity. Such recalls will enable clinicians to assess the effectiveness of the selected therapeutic program. The recall frequency and the type of home program depends on the patient's prevailing dental health, carious activity, and compliance and should vary according to the assessed success or failure.

Conclusions

If the cariogenic challenge is too frequent or too strong, fluoride alone may not suffice to remineralize initial enamel caries lesions. Plaque removal by practicing good oral hygiene is an important additional adjunct. Patients presenting with white-spot lesions may better grasp what is required from them, and why, if they understand the processes of demineralization and remineralization. Patients generally know what plaque and calculus are, but they rarely discuss remineralization with their hygienist or dentist. Possible reasons for this include the practitioner's poor understanding of the demineralizing and remineralizing processes, insufficient time available to explain these processes to the patients, and lack of simple diagrams to facilitate the explanations. The schematic diagrams in this paper may enable clinicians to overcome these problems in an acceptable time span. The explanations are not necessarily meant for well-motivated dental patients. Patients with white-spot lesions should be told the advantages of remineralizing the lesions as opposed to placing restorations and that restorations are necessary only when definite cavitation is diagnosed.

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References

1. Glass RL: The first international conference on the declining incidence of dental caries. *J Dent Res* 1982;61(special issue):1304–1383.
2. Frank RM, O'Hickey S: *Strategy for Dental Caries Prevention in European Countries According to Their Laws and Regulations.* Oxford, IRL Press Ltd, 1987.
3. Marthaler TM, Steiner M, Menghini G, et al: Kariespraevalenz bei Schuelern im Kanton Zurich, Resultate aus dem Zeitraum 1963 bis 1987. *Schweiz Monatsschr Zahnmed* 1988;98:1309–1315.
4. Ekstrand J, Fejerskov O, Silverstone LM: *Fluoride in Dentistry.* Copenhagen, Munksgaard, 1988, pp 229–276.
5. Mellberg JR: Remineralization. A status report for the *American Journal of Dentistry.* Part I. *Am J Dent* 1988;1:39–43.
6. Mellberg JR: Remineralization. A status report for the *American Journal of Dentistry.* Part II. *Am J Dent* 1988;1:85–89.
7. Neumann WF, Neumann MW: *The Chemical Dynamics of Bone Mineral.* Chicago, University of Chicago Press, 1958, pp 39–100.
8. Featherstone JDB: Diffusion phenomena and enamel caries, in Guggenheim B (ed): *Cariology Today.* Basel, Karger, 1984, pp 259–268.
9. Geddes DAM: Current view of plaque acidogenicity, in Guggenheim B (ed): *Cariology Today.* Basel, Karger, 1984, pp 199–204.
10. Arends JE: Dislocation and dissolution of enamel. Theoretical consideration. *Caries Res* 1973;7:261–268.
11. Silverstone LM: Experimental caries models and their clinical explanations, in Guggenheim B (ed): *Cariology Today.* Basel, Karger, 1984, pp 236–244.
12. ten Cate JM: *Remineralisation of Enamel Lesions. A Study of the Physico-Chemical Mechanism,* thesis. Groningen, 1979.
13. Eisenberg AD, Marquis RE: Enhanced transmembrane proton conductance in *Streptococcus mutans* GS-5 due to ionophores and fluoride. *Antimicrob Agents Chemother* 1981;19:807–812.
14. ten Cate JM: The effect of fluoride on enamel de- and remineralisation in vitro and in vitro, in Guggenheim B (ed): *Cariology Today.* Basel, Karger, 1984, pp 231–236.
15. Arends J, Jongebloed WL, Schuthof J: Crystallite diameters of enamel near the anatomical surface. An investigation of mature, deciduous and non-erupted enamel. *Caries Res* 1983;17:97–105.
16. Haikel Y, Frank RM, Voegel JC: Scanning electron microscopy of the human enamel surface layer of incipient carious lesions. *Caries Res* 1983;17:1–13.
17. Mellberg JR: Penetration of fluorine from sodium monofluorophosphate into artificially produced incipient enamel lesions. *Caries Res* 1980;14:115–120.
18. Robinson C, Weatherell JA, Hallsworth AS: Alterations in the composition of permanent enamel during carious attack, in Leach SA, Edgar WM (eds): *Demineralisation and Remineralisation of the Teeth.* Oxford, IRL Press Ltd, 1983, pp 209–223.
19. van Dorp CS, Exterkate RA, ten Cate JM: The effect of dental probing on subsequent enamel demineralisation. *J Dent Child* 1988;55:343–347. □