White Spot Lesions During Orthodontic Treatment: Mechanisms and Fluoride Preventive Aspects

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Decalcifications in the form of white spot lesions appear frequently in orthodontic patients as small lines along the bracket periphery and in a few patients as large decalcifications with or without cavitations. Improper oral hygiene around the appliance induces a low resting pH plaque that may reduce the cariostatic properties of fluoride therapy. Optimal oral hygiene and daily use of a fluoride toothpaste and mouth rinse are essential and have a synergistic effect. Recent advances in fluoride research have shown that fluoride agents like stannous fluoride and titanium fluoride deposit reservoirs on the tooth surface that may resist even low pH and severe caries challenges. (Semin Orthod 2008;14:183-193.)

Decalcification or white spot lesion (WSL) development of the enamel surface is by far the most important iatrogenic effect of fixed orthodontic appliance therapy.[1] Individuals with malocclusions often have many retention sites due to the irregularities of their teeth. In addition, bonding attachments to teeth introduces retention sites on surfaces generally not susceptible to caries. Oral hygiene is thus more difficult to carry out and may explain the much stronger relationship between oral hygiene and caries incidence in orthodontic patients than in non-orthodontic individuals.[2]

Clearly, it is the orthodontists' responsibility to be aware of the risk for decalcifications and take precautions to avoid or limit their development. Fluoride is the most important agent to prevent decalcification and lesions from developing and progressing. To be able to use the current fluoride preparations and procedures optimally, the clinician must be familiar with the major aspects of action mechanisms of fluoride and the current preventive principles. The present article presents the most recent research about fluoride and its use during orthodontic treatment with fixed appliances. Fluoride-releasing materials are covered by another author in this issue of *Seminars in Orthodontics*.

This paper is organized into the following five parts:

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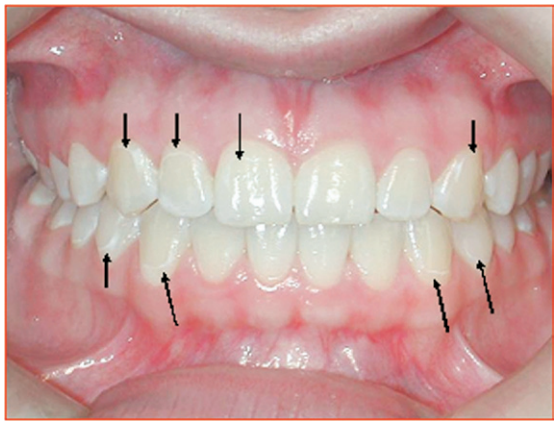

Figure 1. Small white spot lesion along the gingival margin and showing the bracket periphery. (Color version of figure is available online.)

Orthodontic Treatment and White Spot Lesion Development

During Treatment

A number of studies have investigated the relationship between orthodontic treatment with fixed appliances and caries development. Some previous studies showed increased caries frequency with a higher prevalence of fillings in subjects receiving orthodontic treatment, whereas more recent investigations did not confirm such correlation. Several reports have, however, shown an increased incidence of early enamel caries (WSLs) on the labial enamel surface during orthodontic treatment (Fig 1). Changes in light scattering of the decalcified, porous enamel are the reason for the white appearance. These white spot lesions rarely progress into significant cavities and are generally not registered as caries requiring restorative treatment in the DMFT/S (decayed, missing, or filled teeth) indices. Studies where the teeth have been inspected using visual scales (Fig 2) have shown that more than 50% of subjects may experience an increase in the number of WSLs with fixed orthodontic appliances therapy.[3-5] This was recently confirmed in a prospective study from the northern part of Norway. About 50% of the patients receiving orthodontic treatment developed one or more WSLs during treatment and 5.7% of the teeth were affected. This compared with a matched group of non-orthodontic patients in whom 11% developed WSLs on the labial surfaces in the same period of time and 0.4% of the teeth were affected.[6] Even with regular use of a fluoride dentifrice, enamel dissolution may occur around orthodontic attachments, but probably at a lower rate.[7]

Using more advanced detection techniques like quantitative light-induced fluorescence (QLF), Boersma and coworkers[8] observed that 97% of all

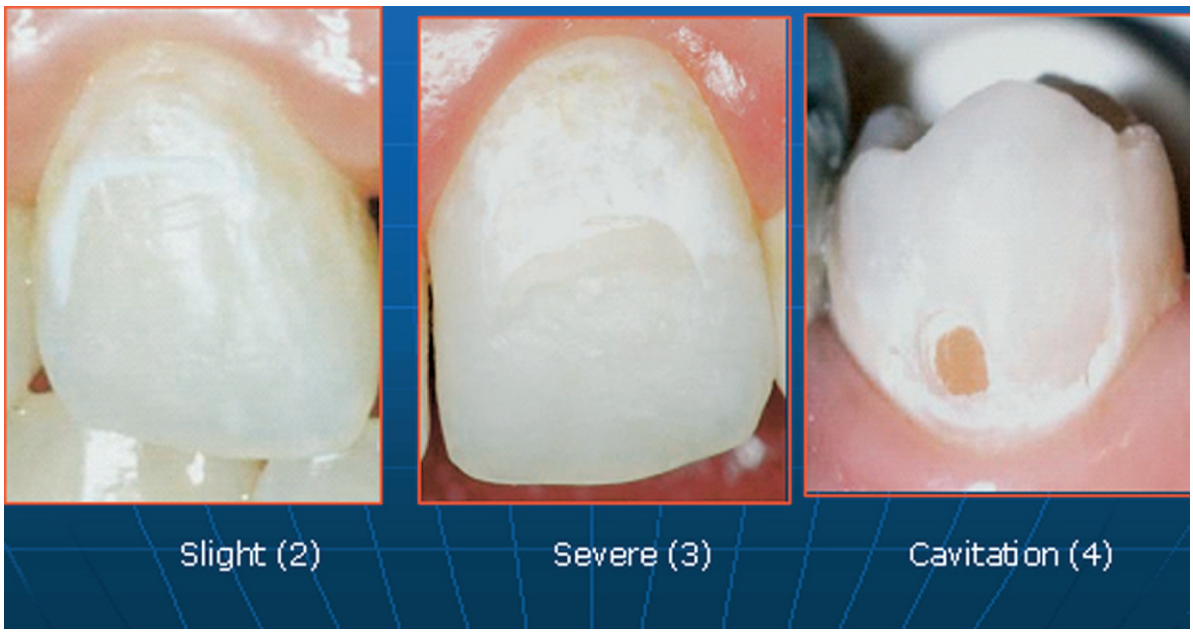

Figure 2. Classification of white spot lesion according to Gorelick and coworkers[3] showing a small lesion (score 2), severe lesion (score 3), and cavitation (score 4). No lesion is recorded as score 1. (Color version of figure is available online.)

subjects and on average 30% of the buccal surfaces in a person were affected. On average, 40% of the surfaces in males and 22% in females showed white spot lesions. This illustrates that surface enamel is changed in almost every single patient receiving fixed orthodontic treatment. The surfaces are therefore at great risk to develop visible WSLs during treatment if the patient does not comply with a caries-preventive program.

White spot lesion development is a very rapid process. Visible WSLs can be induced experimentally in the absence of fluoride within 4 weeks, that is, within the time period between one clinical appointment and the next.[9] Generally, the first molars, upper lateral incisors, and lower canines are the teeth most affected by WSL. In most cases, the lesions are small and restricted to thin bands surrounding the bracket bases or to areas between the brackets and the gingival margin (Fig 1). In some patients, lesion development may be extensive and require rapid debonding unless oral hygiene and fluoride regimens are followed accurately (Figs 3 and 4). Caries lesions may also develop after debonding in association with bonded retainer. If the patients are not aware of the detrimental affects of a loose bond on a retainer, caries may develop rapidly into severe lesions (Fig 5).

Do the Lesions Disappear After Debonding?

The answer is that they disappear to some extent depending on the severity of the lesions. Studies have shown that the prevalence of WSLs on the labial surfaces is higher in previous orthodontic patients than in untreated individuals many years after debonding.[10] Re-examination of 40 individuals who had participated in a randomized, controlled clinical study on the effect of a caries-preventive program[11] 6 years after debonding showed that about 75% of the small WSLs had regressed during that period. Twenty-five percent of the most severe lesions remained visible on the surfaces. The most likely reason for the regression is surface wear from tooth brushing and so forth rather than remineralization.[12]

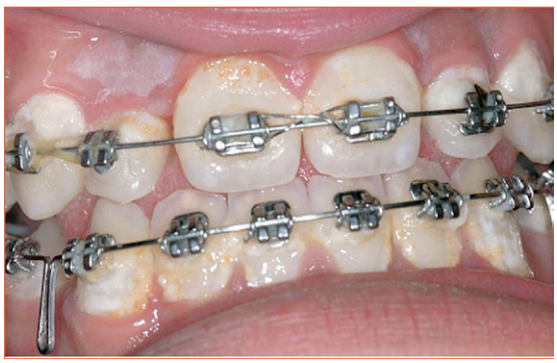

Figure 3. Heavy accumulation of plaque in a noncompliant patient. (Color version of figure is available online.)

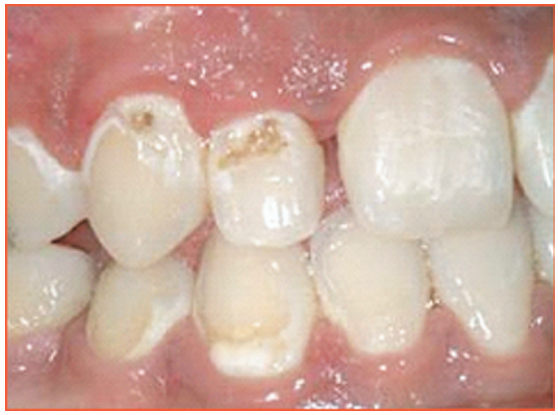

Figure 4. The same patient as in Fig 2 after debonding. Severe white spot lesion and cavitations. (Color version of figure is available online.)

Tooth enamel that is demineralized is porous and may take up stain from food and beverages. A severe case of stained WSL is shown in Fig 6. Poor oral hygiene during orthodontic/orthognathic surgery treatment had resulted in many pronounced WSLs on the labial surfaces. One year posttreatment, the WSLs had taken up stain and were discolored.

Mechanisms for White Spot Lesion Development During Orthodontic Treatment

Building Up a Cariogenic Environment

Gwinnett and Ceen[13] showed that fixed orthodontic appliances induce a rapid increase in the volume of dental plaque. Chatterjee and Kleinberg,[14] furthermore, showed that the plaque in orthodontic patients had a resting pH lower than that in nonorthodontic subjects. There is a rapid shift in the bacterial flora of plaque after introducing orthodontic appliances. Scheie and coworkers[15] observed significantly elevated plaque and salivary levels of *S. mutans* after insertion of orthodontic appliances. Both *S. mutans* and lactobacilli are often associated with caries development. They are acidic bacteria and produce organic acids in the

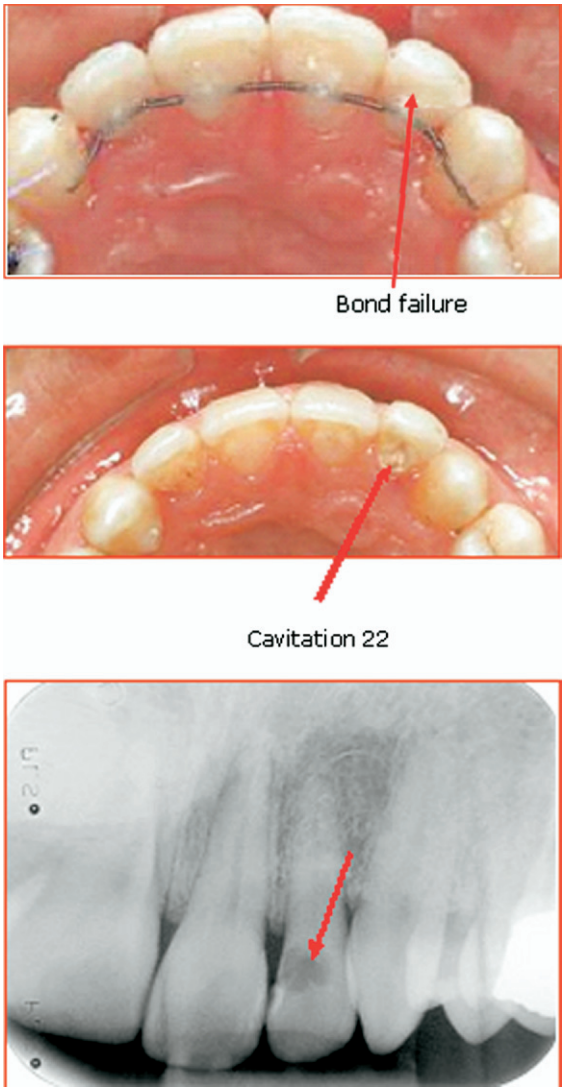

Figure 5. Cavitation after bond failure of a retainer on the upper left lateral incisor. (Color version of figure is available online.)

presence of fermentable carbohydrates. Sucrose plays a particular role in plaque formation inducing the formation of a cariogenic plaque. Lactobacilli are generally associated with progression of caries lesions. High levels of *S. mutans* and lactobacilli in plaque indicate an increased risk of caries. However, the association between caries and bacteria is not a simple one. Research has shown that prediction of caries development based on bacterial counts is uncertain and of minor clinical significance.[16]

Fluoride and the Cariostatic Effect

Fluoride is without doubt the most important caries preventive agent. For many years it was thought that fluoride should be incorporated into the tooth structure to achieve a preventative effect on tooth mineral solubility. Research has shown that this effect is overestimated and that the mechanism is related to fluoride being present in the fluid phase of the caries process. The fluoride ions then execute an inhibiting effect on tooth demineralization and an enhancing effect on remineralization. When topical fluoride is applied, a calcium fluoride-like material (CaF_2) builds up in plaque, on the tooth surface (enamel/dentin), or in incipient lesions. The CaF_2 acts as a reservoir of fluoride ions for release when pH is lowered during a caries attack[17] (Fig 7). The dissolution rate of CaF_2 at different pH is controlled by phosphate and proteins.[1]

Importance of pH on the Fluoride Effect

Arneberg and coworkers[18] studied pH in plaque in orthodontic patients following a sucrose challenge. By using a microtouch electrode, pH changes at different individual sites of the dentition could be monitored rather than using pooled plaque samples. The lowest pH during resting and fermenting conditions was observed in the plaque of the bonded upper incisors. In these sites, pH could fall to as low as 4. The low pH in the upper anterior region is most likely due to the slow salivary clearance at this location allowing a prolonged retention of acids in the plaque. Interestingly, total plaque fluoride levels were also lowest at these sites, suggesting a direct relationship between plaque pH and total plaque fluoride. Accordingly, due to the low pH, any reservoir of fluoride is rapidly lost.

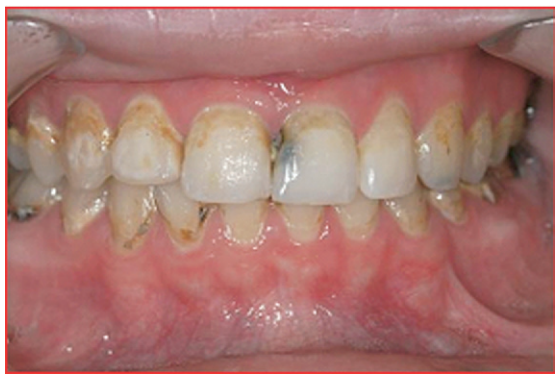

Figure 6. Discoloration of white spot lesion developed during fixed orthodontic treatment 1 year after debonding. (Color version of figure is available online.)

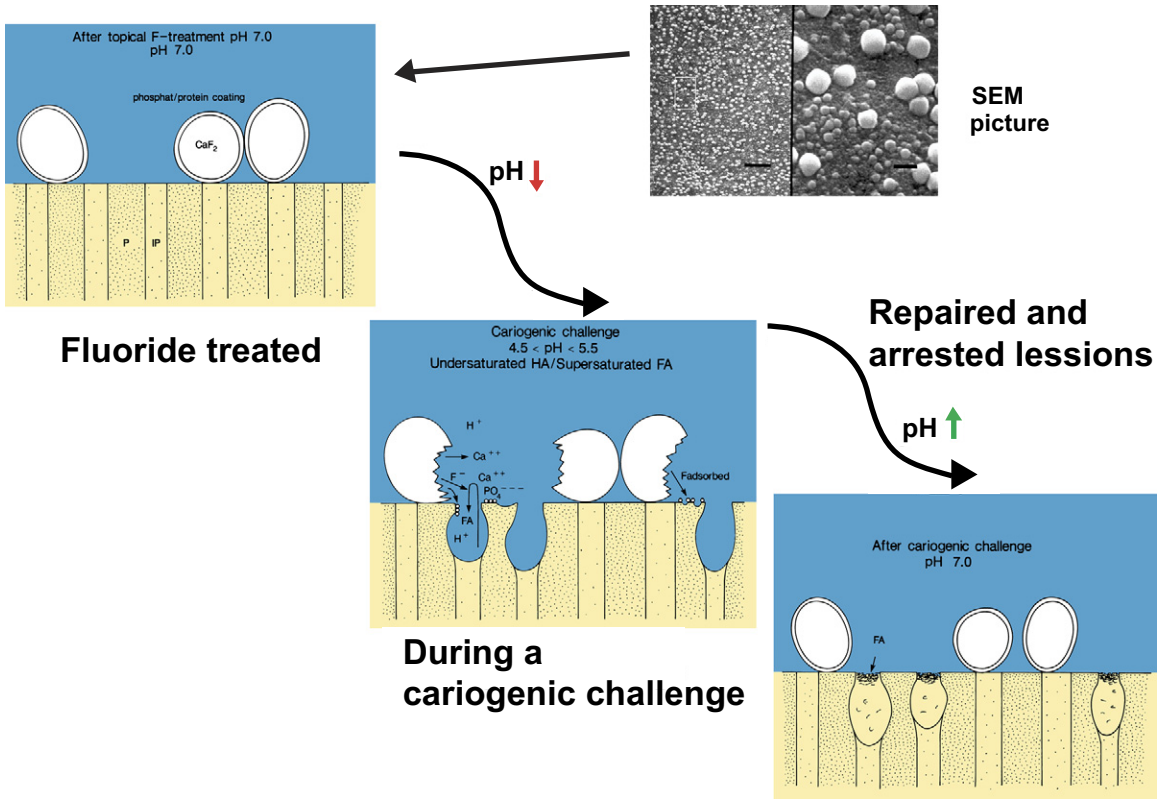

Figure 7. Illustration of how calcium fluoride acts as a pH-controlled reservoir of fluoride to be released during an acid attack. See text. (Color version of figure is available online.)

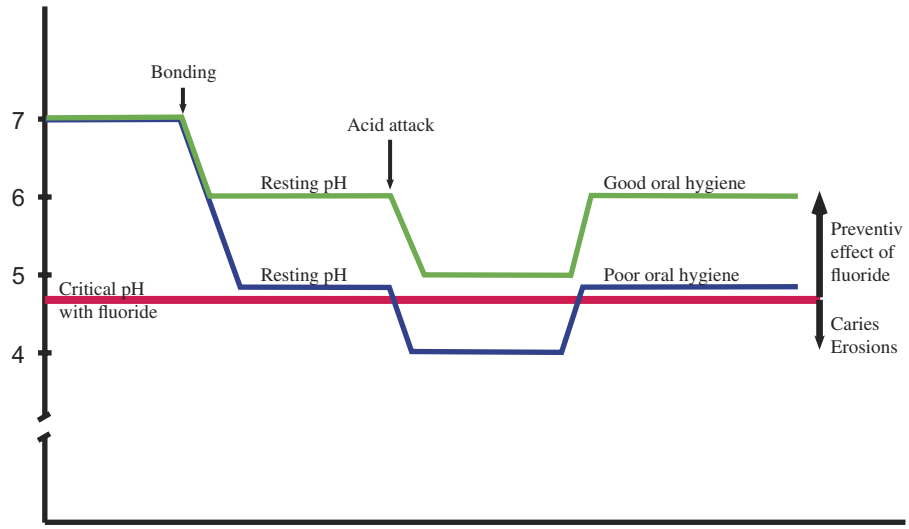

Figure 8. The Stephan curves in orthodontic patients with good or bad oral hygiene. After bonding, resting pH is lowered. An acid attack lowers the pH in the patient with bad oral hygiene below the critical pH of enamel. Assuming fluoride is frequently used, the critical pH of enamel is around 4.5 compared with 5.5 in the absence of fluoride. In the patient with good oral hygiene, fluoride is able to prevent lesions to develop. (Color version of figure is available online.)

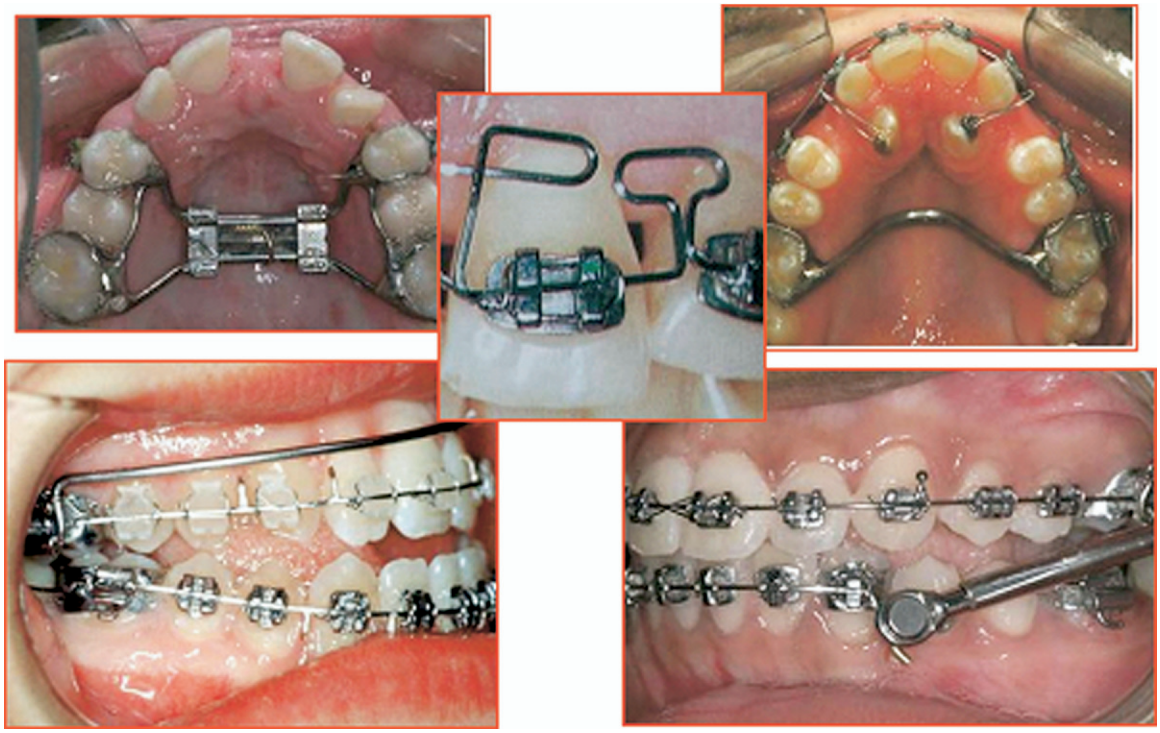

Figure 9. Complicated designed appliances make oral hygiene difficult to carry out. (Color version of figure is available online.)

The limit of the fluoride effect is reached when pH drops so low that even the solubility product of pure fluorapatite is exceeded. This pH is presumably below 4.5. It has been shown that bacteria associated with caries (*S. mutans* and lactobacilli) may lower the pH in dental plaque below 4.5. At this low pH the liquid phase of the plaque will be undersaturated with respect to both hydroxyapatite and fluorapatite and no redeposition of lost mineral (remineralization) will occur.[19] This is most important for the clinical situation. In old, acidic plaque more fluoride will not necessarily give further protection against lesion progression due to the low pH. This loss of fluoride and a limited cariostatic effect of fluoride in the low pH in plaque is perhaps one explanation why WSLs develop frequently on bonded upper incisors. Under such conditions, a dose response to fluoride may not be apparent; for example, more fluoride may not give a better clinical effect.[20]

The preventive effect of fluoride can be illustrated using the Stephan curve (Fig 8). After bonding, the resting pH is lowered slightly but good oral hygiene may keep the pH above the critical point of 4.5 and fluoride prevents lesions by increasing remineralization and inhibiting demineralization. With poor oral hygiene, plaque builds up around the appliance and the resting pH may reach the limit of the fluoride effect at pH 4.5. During an acid attack, caries and even erosions develop.

Important Clinical Aspects to Notice

Patient cooperation with optimal oral hygiene and fluoride prevention is crucial. However, there are several types of appliance design that may enhance the risk of plaque aggregation despite good oral hygiene efforts by the patient. Complicated appliance designs with loops, auxiliary archwires, springs, coils, and some Class II correctors (Fig 9) create areas that are almost impossible to clean with normal skills and equipment. Also excess bonding material around the attachment base creates pockets where the bacteria can multiply (Fig 10). Steel ligatures or self-ligating brackets are preferable to elastic ligatures[21] (Fig 11).

Fluoride Prevention During Orthodontic Treatment

It is logical to differentiate between prevention of caries lesion development during orthodontic

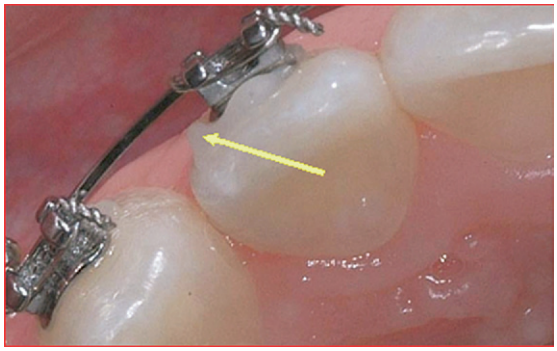

Figure 10. Excess bonding material retains plaque. (Color version of figure is available online.)

treatment and treatment of lesions present on labial surfaces at debonding. Since fluoride is the most important agent in caries prevention, most emphasis will be given to different forms of fluorides.

Current Fluoride Agents and Programs

Fluoride toothpaste is the basis for all caries prevention. Most toothpastes contain sodium fluoride, monofluorophosphate, stannous fluoride, or amine fluoride. The fluoride concentrations may vary, but the maximum concentration allowed in the European community is 0.15%. A dose-response effect to fluoride in toothpastes has been demonstrated, and fluoride concentrations below 0.1% should not to be recommended for orthodontic patients.

The cariostatic potential of fluoride toothpastes is most likely greater than generally shown in clinical studies. This is due to the effect of fluoride on remineralization as a function of time. Furthermore, the cariostatic effect will improve significantly if oral hygiene is also improved, as discussed above. Good oral hygiene is thus more important in orthodontic patients treated with fixed appliances than in nontreated individuals.[2,22]

O'Reilly and Featherstone[7] found that toothpastes were unable to stop lesions from developing in the average orthodontic patient. Orthodontic patients are therefore requested to use a fluoride mouth rinse (0.05% NaF) daily in addition to fluoride toothpaste. Studies using experimental orthodontic caries models and clinical studies have shown a good cariostatic effect of fluoride mouth rinses.[1,23]

Fluoride mouth rinsing is dependent on patient cooperation. Geiger and coworkers[24] showed that less than 15% of the orthodontic patients rinsed daily with fluoride as requested. Since fixed orthodontic appliances introduce a high cariogenic challenge, there is a need for more continuous fluoride supplementation independent of patient

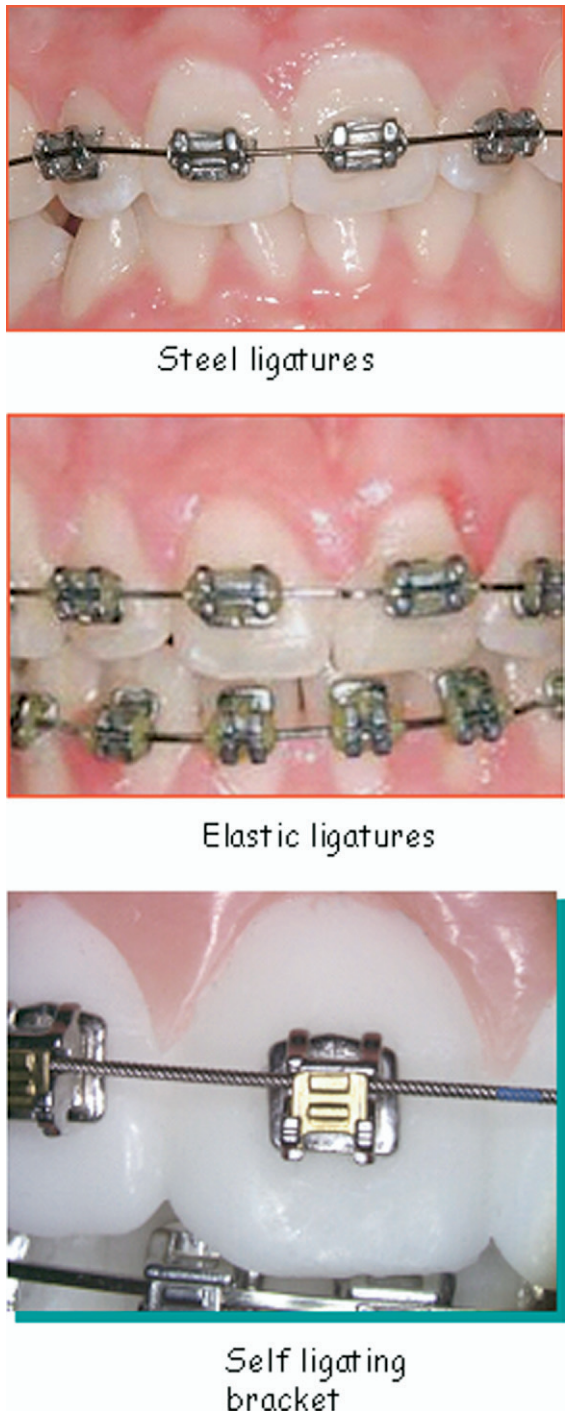

Figure 11. Steel ligature, elastic ligatures, and self-ligating brackets. (Color version of figure is available online.)

cooperation. Therefore, some topical fluoride in the forms of varnishes, solutions, or gels may be recommended. There is no distinct difference in the caries preventive effect of these concentrated fluoride agents. Thus, the choice of method depends on cost, convenience, patient acceptance, and safety. The use of fluoride varnishes has proven to be a feasible and safe method of fluoride application. With fluoride varnishes, the amounts of fluoride exposure can be better controlled and less chair time is required compared with conventional solutions and gels. No dose-response effect to concentrated fluoride agents is apparent and the benefit of frequent application is not clearly established.[25] If caries activity remains high despite regular use of toothpaste and topical fluoride applications, additional methods of caries prevention with the aim of reducing the challenge (improved oral hygiene, antimicrobials, agents depositing acid-resistant coatings [see below]) should be applied instead of increasing the level of fluoride exposure.

Systematic Reviews of the Cariostatic Effect of Fluorides in Orthodontics

Several systematic reviews[26-28] on caries prevention in orthodontic patients have recently been published. It is not surprising that these reviews are of minor practical importance for clinicians. If the clinicians were to only rely on outcomes from systematic reviews or so-called evidence-based orthodontics, orthodontic treatment options would be very limited.[29,30] The reason is that the inclusion criteria for clinical studies in these reviews are very strict and most of the published literature, especially studies related to mechanisms, fails to be evaluated. Under the Cochrane databases only a few subjects related to orthodontic treatment are covered, including WSLs and orthodontics. The general conclusions from these systematic reviews are that it is not possible to recommend which topical preparations or schedules provide the greatest decrease in decalcification. However, Benson and coworkers[26] recommend daily fluoride mouth rinsing (0.05% NaF) in their review. This was also recommended by Zachrisson[31] more than 30 years ago.

Recent Advances in Fluoride Prevention

No evaluation of the level of oral hygiene and its impact on the fluoride effect on WSL development is given in any of the systematic reviews. This is despite the knowledge that the major challenge in orthodontic patients is, as already mentioned, the limitation of the fluoride effect in the low pH of plaque adjacent to orthodontic appliances. Several attempts have therefore been performed to enhance the cariostatic potential of current fluoride agents and procedures for orthodontic purposes. The amount of CaF_2 on enamel can be substantially increased by lower pH of the fluoride solution. A continuous layer of small particles of CaF_2 is formed that protects the enamel against acid to a greater extent, since CaF_2 is less soluble than fluorapatite.[32] An acidic fluoride solution (pH $\approx$ 2.0) can be easily made from a regular neutral sodium fluoride solution without adding phosphate.

Titanium tetrafluoride solutions inhibit lesion development in association with fixed orthodontic appliances markedly more efficiently than conventional preparations. The cariostatic mechanism of titanium tetrafluoride is probably due to the retentive, titanium rich, glaze-like surface coating formed on treated enamel surfaces. At low pH, titanium binds with an oxygen atom of a phosphate group that is densely distributed on enamel surfaces. Following the application of aqueous solutions of titanium tetrafluoride, -Ti-O-Ti-O- chains are formed on the tooth surface and covalently bound titanium covers the tooth surface. A strong complex is thus formed between the titanium compounds and the hydroxyapatite. This surface coating has been found to resist challenges even under extreme alkaline and acidic conditions[33] (Fig 12).

Stannous fluoride has a plaque-inhibiting effect in addition to the anticaries effect. The stannous ion rather than the fluoride ion is responsible for the plaque-inhibiting effect. Stannous ions interfere with the adsorption of plaque bacteria to enamel by being bound to the phosphate polymer lipoteichoic acid present on the surface of Gram-positive bacteria. Stannous fluoride also interferes with the acidogenicity of plaque. It is possible that tin bound to the surfaces of the bacteria also blocks the passage of sucrose into the cell and inhibits acid formation. Stannous fluoride may therefore offer beneficial effects not only against caries, but also against plaque-induced gingival diseases during orthodontic treatment.[34]

The advantages of stannous fluoride have been rediscovered recently due to its protective effect of the enamel surface against strong acids.

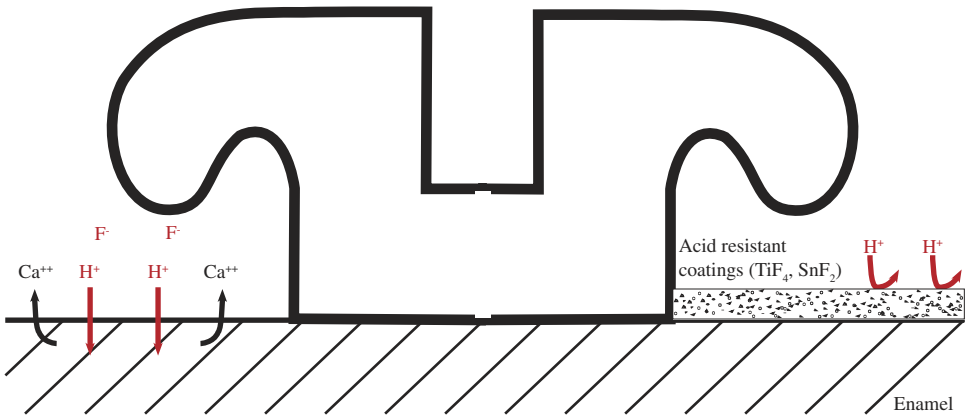

Figure 12. Acid resistant coating deposited from titanium fluoride or stannous fluoride protect the enamel surface against severe acid challenges (H^+ ions under the right bracket wing). Conventional fluoride preparations have a reduced cariostatic effect in plaque with low pH (under the left bracket wing). Ca^{2+} loss illustrates the caries process. (Color version of figure is available online.)

Most likely the effect is attributed to the stannous ion rather than the fluoride ion, since strong tin complexes are known to form on enamel after stannous fluoride application.[35] This has opened up the possibility to use stannous fluoride in toothpastes and rinses to prevent erosions and decalcification in association with orthodontic treatment. An inhibiting effect of a combined stannous fluoride/amine fluoride toothpaste/mouth rinse against both decalcification and gingival bleeding was demonstrated in a prospective, randomized clinical study in orthodontic patients.[36]

Also fluoride combined with antimicrobials like chlorhexidine (CHX) has shown improved cariostatic effects in an orthodontic caries model compared with conventional fluoride. For long-term clinical use, daily CHX rinsing is not recommended because of frequent discoloration of the teeth and tongue and a metal taste. CHX varnishes for long-term use have been introduced and may reduce the cariogenic challenge sufficiently to improve the fluoride effect on WSL.[11]

Fluoride Prevention After Debonding

Debonding the orthodontic appliances reduces or eliminates the cariogenic environment on the labial surfaces, especially on the maxillary anterior teeth. However, resin remnants may be present in the labial surfaces of teeth after debonding depending on the bonding system used.[37] This indicates a potential risk for plaque to accumulate on the labial surface also after debonding, perhaps in particular on the posterior and mandibular teeth where brackets are bonded close to the gingival margin. Lesions in these locations may thus progress after debonding if plaque is allowed to accumulate. Evaluation of lesions that have developed during appliance therapy in the different sites of the dentition represents a clinical challenge whether or not plaque may still accumulate on the surface.

Fluoride and Remineralization

It is well established that fluoride increases the initial rate of remineralization of early enamel lesions and then slows down the caries process. This is due to the reaction of fluoride with minerals mainly in the surface of the lesion and results in arresting of the lesion.[17] These arrested lesions will persist lifelong, exhibiting a white color as in WSLs, or they may become yellowish or dark brown in color due to exogenous uptake of stains. This is supported by clinical data showing a higher prevalence of WSLs in areas with water fluoridation and in orthodontically treated patients several years after debonding. Whether complete remineralization may occur or not seems to be related to the type of lesion present. Initial surface-softened lesions appear to remineralize quickly in saliva even without fluoride.[38]

Al-Khateeb and coworkers[39] followed seven patients with incipient enamel lesions developed during orthodontic treatment longitudinally for 1 year after debonding. These lesions were most

likely of the subsurface type. A fluoride toothpaste was used daily. No additional topical fluoride was used. The changes in mineral content of the lesions were monitored using quantitative laser fluorescence. During the 1-year period of the study, the fluorescence radiance in the lesions increased and the area of the lesions decreased indicating remineralization. Complete regain of lost minerals was not achieved. Since the minerals are not necessarily deposited in the same way as sound enamel, light scattering from the partly remineralized lesion may not be identical to the sound enamel. Lack of complete remineralization of WSL was recently confirmed in a clinical evaluation using quantitative light-induced fluorescence of enamel surfaces 2-years posttreatment.[40] Perhaps the regression of WSL lesions following debonding as reported by some researchers is mainly due to surface wear rather than redeposition of minerals.[12]

Acid Etching to Enhance Remineralization

It has been suggested that acid etching of WSLs may increase the surface porosity and hence remineralization. Al-Khateeb and coworkers[40] induced WSLs in enamel in vitro and investigated longitudinally the rate of remineralization in etched and nonetched lesions in the presence or absence of fluoride. The rate of remineralization was measured weekly for 12 weeks using a quantitative light-induced fluorescence method. The mineral profile of the remineralized tissue was analyzed with transverse microradiography and the topography of the surface layer studied with scanning electron microscopy. Substantial lesions persisted at the end of the experiments irrespective of the treatments given. The rate of remineralization varied significantly between the groups during the first few weeks of the experiment. Etched enamel exhibited more pronounced lesion reduction than nonetched enamel, especially in the absence of fluoride. Later, the remineralization process slowed down for all groups and at the end of the experiment no significant differences were found for any of the treatments. The etched lesions retained a porous structure of their surface layer even after a long period of remineralization in vitro. It should be noted that in vitro remineralization is known to proceed more rapidly than in vivo remineralization.

Conclusions and Clinical Recommendations

Without doubt, enamel decalcification is a major clinical problem in treatment with fixed orthodontic appliances. It is most important to prevent caries lesions from developing during treatment. Once the lesions are established, in particular larger subsurface lesions, it is extremely difficult, or even impossible, to achieve complete remineralization.

Fluoride will arrest these lesions, but in due time they may discolor. This is an unfavorable development and may detract from the beneficial esthetic effect of the orthodontic treatment altogether. Fluoride is the most potent cariostatic agent available that can prevent the development of lesions. All orthodontic patients must brush daily with fluoride toothpaste. Daily use of a fluoride mouth rinse is also recommended. Optimal oral hygiene around the appliances is essential to obtain the full effect of fluoride, since the cariogenic challenge is much higher and plaque pH lower than in individuals who had not had fixed orthodontic appliances placed. Topical fluoride in the form of solutions, varnishes, or gels should be applied around the brackets on a regular basis. Newer formulations, resulting from more current findings of fluoride mechanisms in caries prevention, or fluoride in combination with antimicrobial agents, may improve the clinical outcomes when compared with the use of currently employed fluoride preparations. Agents depositing acid-resistant coatings on the enamel surface adjacent to the appliance may prevent lesion development almost totally and should be the subject of further research.

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