Reducing iatrogenic complications in the treatment of cleft lip and palate

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All patients receiving orthodontic treatment with fixed appliances have a significantly higher risk of developing white spot lesions (WSLs), which are considered the most common iatrogenic complication of treatment.
Cleft lip and/or palate (CLP) is a common craniofacial anomaly occurring in approximately one in 800 births. Patients affected by cleft lip and/or palate tend to have poorer oral hygiene and a greater risk of WSLs and caries than non-cleft sibling controls. Patients presenting with a cleft lip and/or palate also have a higher prevalence of molar-incisor hypomineralisation (MIH), which suggests that clinicians involved in the oral health management of cleft patients need to consider all available caries prevention protocols.
Cases presenting with hypomineralised teeth create more difficulty in bonding attachments to affected enamel using an acid etch technique and a composite resin material due to the abnormal prism structure. The bond strength to hypomineralised enamel can be as low as two-thirds that of the bond strength to unaffected enamel, which may not be adequate for routine fixed appliance treatment. Furthermore, the removal of orthodontic brackets from hypomineralised enamel may lead to more severe damage to the affected teeth.
Resin modified glass-ionomer cement (RMGIC), bonded after conditioning the enamel with polyacrylic acid, creates no resin tags as a result of a chemical rather than a mechanical bond. Therefore, there is less enamel loss during bracket debonding when compared with acid-etch preparation and composite resin bonding.
Furthermore, in cases in which the quality of the enamel is deficient causing limits to the shear bond strength of the acid-etched composite resin, the chemical bonding action of RMGIC overcomes the lack of adherence and protects the enamel.
RMGIC has cariostatic properties and clinical evidence supports the routine use of these adhesives for bonding in all fixed appliance cases as a strategy for reducing the incidence of white spot lesions and damage to the enamel during bracket debonding.
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Introduction

Cleft lip and/or palate (CLP) is a common craniofacial anomaly that occurs in approximately one in 800 births.[1] In affected patients, abnormalities such as tooth agenesis and supernumerary teeth, as well as variations in the size, shape and structure of the teeth, occur more frequently than in unaffected populations.[2]

Dental enamel is an ectodermally-derived calcified tissue. Any disruption in the calcification and/or maturation of the developmental processes produces structurally-defective hypomineralised enamel.[3] While the aetiology of enamel defects is unknown, the close chronology of facial development with that of the tooth germs suggests that systemic conditions may effect either event or that both conditions are related.[4] The occurrence of enamel defects is more common in permanent molars and incisors and particularly common in those affected by clefts.[5,6]

Independent of socioeconomic status, patients with a cleft lip and/or palate tend to have poorer oral

hygiene and a greater risk of caries than non-cleft sibling controls.[7] Caries prevalence in cleft patients increases with age as 63% of 4-year-old but only 34% of 12-year-old children are caries free. Developmental defects also increase with age and are highlighted by at least one opacity seen in 56% of 4-year-old and in 100% of 12-year-old patients upon the eruption of the permanent dentition.[8] This suggests that clinicians involved in the oral health management of cleft patients need to consider all available caries prevention protocols.[3]

The treatment of patients affected by CLP presents many challenges in multi-disciplinary management. Orthodontic treatment is often required over many years when commenced in the early mixed dentition to compensate for any midface deficiencies and prepare the dento-alveolar arches for alveolar grafting. Further treatment is usually required in the permanent dentition to detail the occlusion and, depending on possible skeletal imbalance between the maxilla and mandible, orthognathic surgery may be required.

Dental caries

The formation of dental caries is initiated by the consumption of fermentable carbohydrates by acidogenic bacteria that inhabit the biofilm on teeth. Acids produced by the bacteria dissolve the mineral content of the teeth to eventually create a cavity.[9]

Fixed appliance orthodontic treatment increases the risk of dental caries as the attachments create plaque traps and increase the difficulty of standard, home-based oral hygiene measures. Orthodontic attachments also restrict the ability of the tongue to remove food particles from tooth surfaces, which, coupled with the breakdown of carbohydrates, leads to a prolonged acid challenge to the enamel. As the level of plaque retention increases, colonisation by acid-producing bacteria such as *mutans streptococci* and *lactobacilli* can lead to subsurface demineralisation of the enamel adjacent to the attachments.[10]

The risk factors for the development of caries include poor oral hygiene, a diet of food and drink high in sugar, poor salivary flow and a lack of fluoride exposure.[11] In general, the responsibility has been placed on the patient and or parent/guardian to carry out the necessary oral hygiene and preventive measures. These have routinely involved the use of fluoride-containing toothpaste and mouth rinses, in addition to dietary counselling and effective plaque removal through tooth brushing.[12]

While the maintenance of good oral hygiene is the responsibility of the patient, it is the responsibility of the orthodontist and dental team to collaborate in providing effective oral hygiene instruction and monitoring over the duration of orthodontic treatment.[13] However, the interval between orthodontic visits can vary between four and eight weeks, during which time new carious lesions can develop[14] and remain undetected by the orthodontist. Other measures, such as regular rinses with 0.05% sodium fluoride, the use of fluoridated toothpastes and casein phosphopeptide-amorphous calcium phosphate (CPP-ACP) application, help to reduce enamel demineralisation and promote remineralisation.[15] A limitation of these preventive measures is patient compliance in following instruction and re-instruction.[16] White spot lesions (WSLs) that present following the removal of fixed appliances may require more invasive treatment procedures such as micro-abrasion or whitening.[17]

Due to the scattering of surface light following the removal of fixed appliances, areas of early demineralisation may be seen as chalky (opaque) white markings on the enamel surface, which become more visible under a dry field.[14] More severe areas of demineralisation can lead to more darkly stained patches and/or cavitation (Figure 1). The surface markings are collectively described as white spot

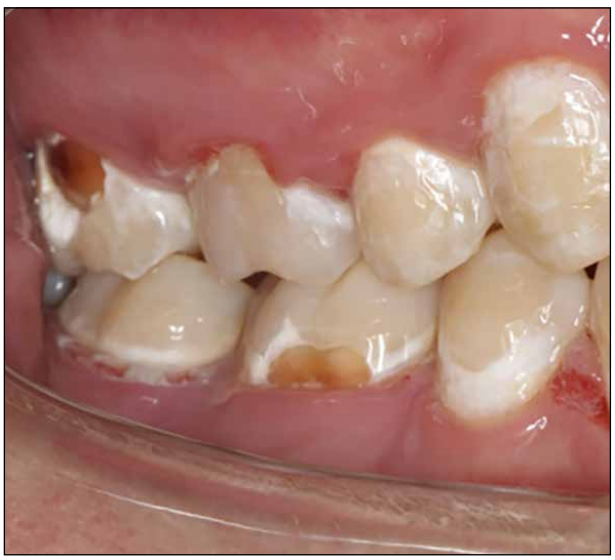

Figure 1. Presentation of white spot lesions (WSLs) at the time of debonding where composite resin adhesive was used. An excellent orthodontic result is compromised with chalky white areas adjacent to gingival margins and cavitation on upper and lower permanent molars.

lesions (WSLs) and can present as halos adjacent to the site of debonded attachments and result in compromised aesthetics.

Patients receiving orthodontic treatment have a significantly higher risk of developing WSLs compared with untreated subjects[16] and WSLs are considered the most common iatrogenic complication of orthodontic treatment[19] with an incidence reported between 50 and 73%.[20]

All members of the dental profession have a responsibility to act in the best interests of their patients and minimise iatrogenic complications during and subsequent to treatment. It is important that all orthodontists are able to diagnose the early carious lesion,[18] especially as more patients are presenting for treatment without referral.

Molar incisor hypomineralisation (MIH)

MIH is a congenital anomaly that affects the permanent molars and to a lesser extent the permanent incisors. MIH is a developmental deficit in the mineralisation of tooth enamel that produces a reduced concentration of calcium and phosphate ions of up to 19% when compared with normal enamel.[21-23] Affected enamel is of normal thickness but the mineralisation process is incomplete, resulting in well demarcated, yellowish-brown opacities and hypersensitive teeth.[24] MIH affects an average of 16% of children worldwide[25] and exhibits variations between geographic distribution and age bracket, but is continuously rising in prevalence across the world.

Patients with cleft lip and/or palate have a higher occurrence of MIH and are potentially at a greater risk of caries. Caries prevalence in cleft patients increases with age as 37% of 4-year-old and 66% of 12-year-old children present with carious lesions. Developmental defects also increase with age, with at least one opacity evident in 56% of 4-year-old and 100% of 12-year-old children with the eruption of the permanent dentition.[8]

A lower enamel fracture resistance can lead to enamel breakdown, which increases the risk of plaque accumulation and rapid caries progression.[6,26] Within a sample, 75% of the bilateral and 39% of the unilateral CLP patients had moderate to severe enamel defects with MIH scores significantly greater in the bilateral CLP subjects.[3] Furthermore, the occurrence of MIH is greater in those affected by CLP, which puts these patients at a higher risk of developing caries.

MIH should not be confused with fluorosed teeth, which can also appear discoloured. In fluorosis-affected teeth, the opacities are diffuse[27] and the enamel is hypermineralised, which confers resistance to acid attack and caries.[28] Hypomineralised enamel is more porous and has a lower mechanical resistance, rendering the affected tooth more susceptible to acid attack and rapidly developing tooth decay.[3] Patients with developmental hypomineralised enamel have an increased likelihood of presenting with untreated severe carious lesions.[29]

Orthodontic treatment using fixed appliances

Orthodontic treatment using fixed appliances involves the placement of brackets and tubes on the enamel surfaces as attachments for the fastening of active components such as wires or elastics to apply forces that initiate tooth movement. A critical factor in the success of orthodontic treatment is the reliability of the bond between the attachment and the enamel surface.

The standard procedure for the bonding of attachments is via acid etching of the enamel surface as initially described by Buonocore.[30] The tooth surfaces are etched using 35–37% phosphoric acid to open spaces between the enamel rods, allowing the resin to penetrate and form a mechanical bond.

In cases presenting with hypomineralised teeth, it is more difficult to bond attachments to the affected enamel using the acid etch technique and composite resin due to the abnormal prism structure. The bond strength to hypomineralised enamel can be as low as two-thirds that of the bond strength to normal enamel,[31] which may not be adequate for routine fixed appliance orthodontic treatment. Furthermore, the removal of orthodontic brackets from hypomineralised enamel may lead to more severe surface damage.

Glass-ionomer cements (GIC)

Diedrich, in 1981,[32] stated "for the future, it may be desirable to develop orthodontic adhesives that make the acid pre-treatment of enamel unnecessary". While the standard bonding agent is still composite resin with a mechanical bond to the enamel surface, there is mounting evidence from both *in vitro* and *in vivo* studies to support the routine use of resin modified

glass-ionomer cements (RMGIC) for bonding attachments.[33-36]

In vitro studies have demonstrated that the mean shear bond strength of RMGIC is in a range of 6.0–9.56 MPa with composite resin in a range of 18.46–20 MPa.[33,37] While the bond strength of composite resin is considerably greater than RMGIC, Reynolds, in a review of direct bonding in orthodontics, demonstrated that a bond strength of at least 5.9 MPa is clinically acceptable.[38]

There is a higher incidence of compromised enamel in cleft lip and/or palate patients, which limits the effectiveness of composite resin as a bonding agent due to the porosity of the enamel. Alternatively, the mechanism for adherence of GICs is a chemical bond via an ion exchange reaction between the setting cement and the enamel surface. This interactive zone is a feature of glass-ionomer cements and contributes to the durability of the adhesive bond and the resistance to micro-leakage.[39]

GICs are acid-base cements comprised of fluoro-aluminosilicate glass powder and polyalkenoic acid mixed with water. The tooth is prepared for bonding by surface conditioning using a 10% aqueous solution of polyacrylic acid for 10 seconds, then rinsed off with water, leaving the surface moist. This serves to remove the tooth's smear layer and reduce the surface tension to facilitate the flow of adhesive across the enamel. Water promotes the acid-base reaction as contact with the acid dissolves the surface of the glass particles to free aluminium ions and calcium ions from the tooth surface.[40] The ions cross link with the polyalkenoic acid chains to form a matrix and solidify the mix. This two phase setting reaction commences at the start of mixing with cross linking of calcium ions with the acid,[41] during which time the matrix is sensitive to excess water or desiccation. As cross linking with aluminium ions occurs, strength and physical properties increase.

Bond strength develops to approximately 80% of the final strength within 15 minutes and continues to increase for several days. Failures in RMGIC bonding are cohesive in nature within the cement rather than at the interface. Therefore, reported bond values are a measure of tensile strength rather than adhesive strength and suggest that the quoted values in the literature are understated and not a true measure of the bond strength of these materials.[42]

The resin glass-ionomer cements are modified by the inclusion of a resin monomer such as hydroxyethyl methacrylate (HEMA). Light cured resin modified GICs have three setting reactions. Once the cement is mixed and placed on the tooth surface, irradiation with light initiates the polymerisation of the HEMA in conjunction with the acid/base reaction of the glass ionomer. Any remaining HEMA not irradiated by light will continue to set by chemical polymerisation.[43] The depth of cure with irradiated RMGIC is significantly higher than auto-curing RMGIC such that light cured RMGICs are stronger materials with greater adhesion than auto-cured RMGICs.

A major advantage of RMGIC is the ability to act as a reservoir of fluoride in the oral cavity and to provide a mechanical barrier that protects the tooth surface against bacteria.[41] RMGICs are able to take up fluoride from external sources such as toothpaste or fluoride gels then slowly release ions to reduce the acidic nature of any surrounding biofilm.[44] RMGICs have a therapeutic effect against caries[45] through the leaching of fluoride into adjacent enamel in a manner similar to conventional glass-ionomer cements in the prevention of white spot lesions.[15,36] The cariostatic effect of RMGICs has also been shown *in vivo,* adjacent to orthodontic attachments bonded with RMGIC.[46] This is a particular advantage in communities without fluoridated water supplies and where only bottled (non-fluoridated) drinking water is available.

Previous authors have suggested alternatives to the conditioning of the enamel surface using acid etching and/or sodium hypochlorite as methods of increasing shear bond strength. *In vitro* studies have shown an increase in material shear bond strength when compared with conditioning by polyacrylic acid.[47] However, this is contra-indicated for all glass-ionomer cements.[48] These adhesives particularly bond to the mineral phase of the tooth material via chemical bonding between carboxylic acid groups of the polymer and calcium ions in the enamel.[49] This is a significant concern relative to the hypomineralised enamel in cleft lip and palate cases due to the already porous surface.

The use of RMGICs has increased value in developing countries. Bonding with composite resin requires more elaborate equipment to maintain a dry etched enamel surface. Alternatively, the hydrophilic nature of RMGIC material requires a moist field to enhance the setting through the acid-base reaction. The dismissal

of RMGIC as an orthodontic bonding agent would appear to neglect the value of the anticariogenic and hydrophilic properties that are not characteristic of the resin adhesives.[48]

Direct bonding clinical technique

Fjeld et al.[50] compared three different systems available for the bonding of orthodontic attachments: 35% phosphoric acid etch with composite resin, a self-etching bonding system with composite resin and, finally, conditioning with 10% polyacrylic acid and the use of RMGIC. It was noted that the three bonding systems induced different effects on the enamel structure. Phosphoric acid etching produced a rough, etched surface displaying the typical honeycomb pattern. Bonding brackets to this surface resulted in thick resin tags penetrating relatively deeply into the enamel. Less pronounced etching of the surface enamel was obtained by the self-etching primer system, and bonding resulted in smaller and fewer resin tags. The RMGIC bonded after conditioning the enamel with polyacrylic acid showed no resin tags, as a result of the chemical rather than a mechanical bond. The study concluded that the possible higher failure rate of the bonding systems based on conditioning the enamel surfaces with a self-etching primer or a polyacrylic acid might be outweighed by fewer irreversible effects on the enamel structures.

Risks of debonding

Ryf et al.[51] noted that orthodontic treatment with bracket bonding causes irreversible damage to the dental enamel, independent of the protocol used to remove the adhesive remnants.

The bonding and debonding of orthodontic attachments results in a loss of enamel at each stage of the process. The preparation of the enamel surface with a pumice slurry can remove up to 6.9 µm,[52,53] while acid etching using 37% phosphoric acid can remove up to 170 µm.[32] The resin tags filling the etched microclefts generally reached a depth of 80 µm, sometimes extending to about 100 to 170 µm in length. Furthermore, the micro-morphologic findings showed clearly that the direct-bonding technique entails an artificial weakening of the superficial enamel. Clean-up and the removal of the adhesive after debonding can result in a further loss of enamel of up to 149 µm,[54] while fine resin tags will probably remain incorporated in the enamel.[32] The most effective method for the removal of the resin adhesive remnants after bracket debonding was the use of a tungsten carbide multi-laminated, high-rotation bur, followed by the use of a tungsten carbide multi-laminated, low-rotation bur, although this still proved to be inefficient for total removal of the adhesive remnants.[55]

When debonding composite resin from hypomineralised enamel, the bond failure is more often at the resin-enamel interface compared with the attachment resin-interface of normal enamel. This results in greater damage to the hypomineralised enamel when attachments are removed.[56] Similarly, teeth with WSLs are more prone to enamel loss during the debonding of resin bonded brackets.[57]

Ireland et al.[58] compared acid etching and composite resin bonding with polyacrylic conditioner and Fuji Ortho LC (RMGIC). It was shown that the least enamel loss occurred following the use of polyacrylic conditioner and Fuji Ortho LC in which the removal of the adhesive from the tooth surface was carried out using a slow speed tungsten carbide bur rather than an ultrasonic scaler or high speed tungsten carbide bur.

Al Shamsi et al. showed a significant difference in enamel loss between Fuji Ortho LC and composite resin following clean-up. The mean enamel loss in the Fuji Ortho LC group was 40.9 µm while in the composite resin group the mean was 72.7 µm.[59]

Procedures for bonding and debonding with RMGIC

Bonding

1. Clean the enamel surface to be bonded with a rubber cup or brush with a slurry of pumice and water
2. Rinse thoroughly with water
3. Place tongue and cheek retractors as indicated for a clear view
4. Condition the enamel surface for 10 seconds with 10% polyacrylic acid (Figure 2)
5. Rinse thoroughly with oil free water and leave the enamel surface moist
6. Prepare a capsule of Fuji Ortho LC (GC Corporation, Tokyo, Japan) and mix in an automatic mixer
7. Load the mixed capsule into the extrusion gun

and place a small amount of adhesive onto the mesh base of the bracket (Figure 3)

8. Press the bracket firmly onto the enamel surface, wipe off excess adhesive from the margins and fine tune the bracket position to clinical requirements
9. Up to three brackets can be placed with one capsule depending on ambient temperature
10. Light activate each bracket for 60 seconds moving across the bracket margins
11. Place and fasten initial arch wires of low force in either small diameter steel or NiTi once each bracket has been fully light cured to ensure maximum bond strength (Figure 4).

Light activation provides a deeper depth of cure than auto-cure GICs and the greater the depth of cure, the stronger is the bond to enamel. Therefore, do not reduce the time spent on this step.

Debonding

Bonded brackets can be removed using debonding pliers similar to resin bonded attachments. However, in contrast to resin adhesives, RMGICs fracture more at the adhesive-bracket interface leaving the bulk of the adhesive on the tooth surface as a reflection of a cohesive fracture. The surface adhesive can be removed using a slow speed tungsten carbide bur in a dry field. This will clearly demarcate the RMGIC from the enamel and reduce the risk of surface damage. High speed suction should be used to evacuate the adhesive dust while cleaning up to reduce ingestion by the patient as well as environmental risks to the operator and chairside assistant. The tooth surface can then be polished with a slurry of pumice and water to produce a smooth clean surface (Figures 5–8).

Conclusion

The placement of orthodontic fixed appliances increases the incidence of white spot lesions and, although this can be controlled with adequate oral hygiene and additional fluoride measures during treatment, compliance can be as low as 15% of patients. Resin modified glass-ionomer cements are clinically acceptable for the direct bonding of orthodontic brackets and, by way of their cariostatic properties, minimise the problems of non-compliance in home care. Furthermore, in those cases in which the quality

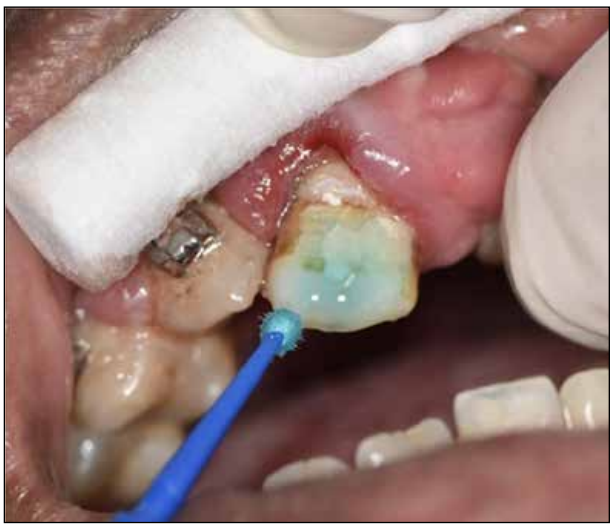

Figure 2. Condition the enamel surface with 10% polyacrylic acid for 10 seconds then rinse off with water leaving the surface moist.

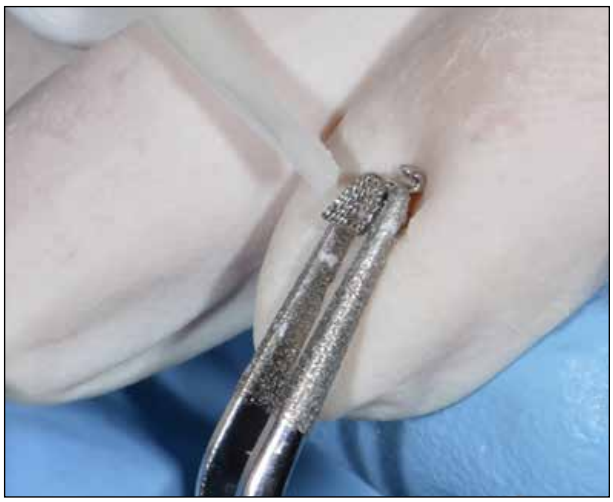

Figure 3. Extrude a small amount of RMGIC adhesive to the mesh base of the bracket.

Figure 4. Place small diameter arch wire in either steel or NiTi after light curing each bracket for a minimum of 60 seconds.

Figure 5. Debond with debonding pliers.

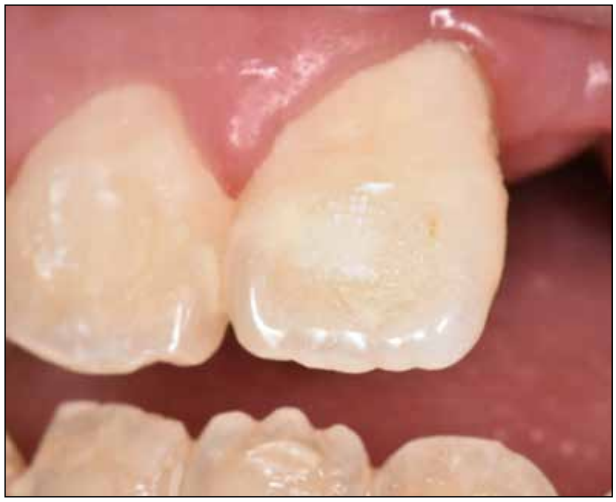

Figure 6. The bulk of adhesive remains on the enamel surface.

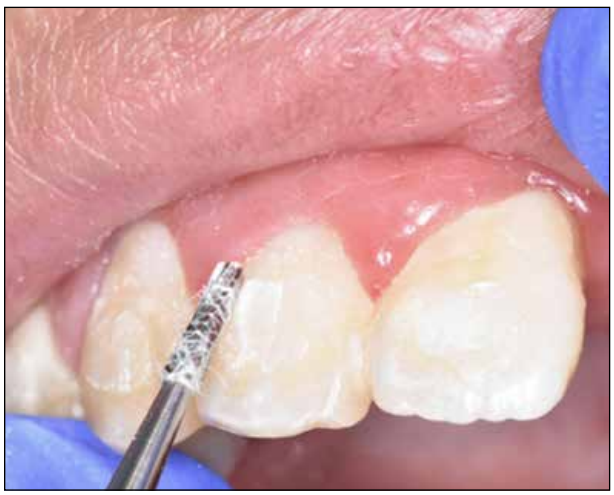

Figure 7. Polish adhesive with a slow speed tungsten carbide bur.

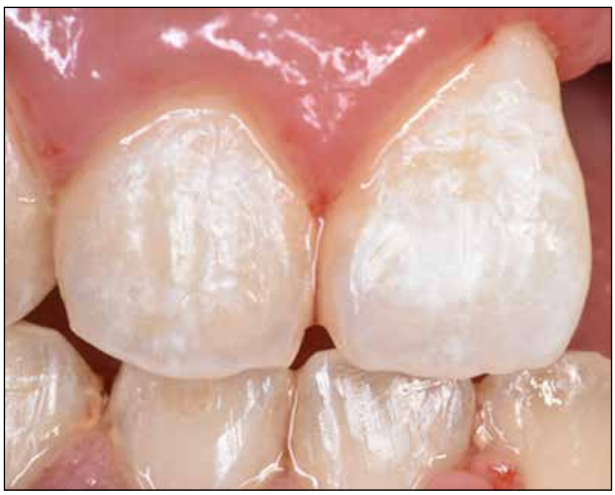

Figure 8. Smooth enamel surfaces after polishing with a pumice slurry.

of the enamel is suspect and limits the shear bond strength of composite resin to acid etched enamel, the chemical bonding action of RMGIC overcomes the limitations and offers enamel protection.

Enamel surface preparation prior to RMGIC bonding requires 10% polyacrylic acid to remove the smear layer and reduce surface tension which enhances the ion exchange reaction. Alternative enamel preparation using phosphoric acid or sodium hypochlorite may increase the shear bond strength to normal enamel but limits the chemical bonding of the RMGIC. There is no need to increase the bond strength beyond that required for clinical performance demonstrated in clinical trials, therefore these alternative enamel preparations are ill-advised.

Clinical evidence supports the routine use of resin modified glass-ionomer cements for enamel bonding in all fixed appliance cases as a strategy for reducing the incidence of white spot lesions and damage during bracket debonding.

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