INCREASING SEALANT RETENTION DURING ORTHOODONTIC TREATMENT WITH GINGIVAL RETRACTION CORD ISOLATION

A Thesis

by

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Submitted to the Office of Graduate and Professional Studies of
Texas A&M University
in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

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May 2018

Major Subject: Oral Biology

ABSTRACT

Introduction: The purpose of this study was to evaluate the effects of gingival retraction cord isolation on the retention of sealants placed on the facial surface of maxillary anterior teeth during orthodontic treatment. **Methods:** An in-vivo randomized split-mouth study, including 32 adolescent orthodontic patients (14 male, 18 female, mean age 14.4 ±1.5 years), was performed. One quadrant had gingival retraction cord isolation prior to sealant placement, while the contralateral control quadrant had no retraction cord placed. Retention of the sealant (Pro Seal, Reliance Orthodontic Products, Itasca, IL) was evaluated with black-light photographs taken prior to, as well as, 3 and 7 months into treatment. The fluorescent sealant was traced digitally using Analyze 12.0 software to quantitatively analyze sealant retention. **Results:** Regardless of whether or not retraction cord was used, greater than 35% of the sealant was lost after 3 months, and over 45% was lost after 7 months of treatment time. The lateral incisors had significantly greater (5-11%) sealant loss than central incisors and canines. The gingival region of teeth had approximately 6-10% greater sealant loss than the overall tooth surface. The use of gingival retraction cord isolation decreased sealant loss by a statistically significant ($p=0.02$) 5-7%. **Conclusions:** Gingival retraction cord had a small positive effect on sealant retention, but the difference was too small to be clinically significant.

DEDICATION

I would like to dedicate this thesis to my wife, Danielle. Thank you for always encouraging me and supporting me through many years of school. I also dedicate this to my parents, Brian and Rebecca Anderson, for teaching me the value of a good education and showing me how to work hard and sacrifice.

ACKNOWLEDGEMENTS

I would like to thank my thesis committee, Dr. Peter Buschang, Dr. Phillip Campbell, Dr. Katie Julien, Dr. Amal Noureldin, and Dr. Frank Higginbottom for your contributions and guidance during this project. I would like to thank the Class of 2019 for allowing me to use their patients for my study. I would also like to thank all of my coresidents in the Class of 2018 for their continued support throughout residency.

CONTRIBUTORS AND FUNDING SOURCES

This work was supported by a thesis committee consisting of Professor Peter Buschang, advisor, and Professors Phillip Campbell and Katie Julien of the Department of Orthodontics, Professor Amal Noureldin of the Department of Public Health Sciences, and Professor Frank Higginbottom of the Department of Restorative Sciences.

The analyses depicted in Chapter III were conducted by Peter Buschang of the Department of Orthodontics.

All other work conducted for the thesis was completed by the student independently.

Funding for the project was provided by the Robert E. Gaylord Endowed Chair in Orthodontics.

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CHAPTER I

INTRODUCTION AND LITERATURE REVIEW

Introduction

White spot lesions are a problem that occurs during orthodontic treatment and remains after fixed appliances are removed. These decalcified enamel lesions not only affect the esthetics of the final orthodontic result, but they negatively affect the health of the dentition. They cause patients and orthodontists to be unhappy with the outcome, sometimes even leading to medico legal action. WSLs are a result of plaque and bacteria accumulating on the enamel surface, which leads to acid by-products from the bacteria, leading to enamel demineralization. The prevalence of WSLs in orthodontic patients ranges from 2 to 96%, depending on the method of assessment, but many studies estimate that at least ¼ of patients will develop at least one WSL during the course of their orthodontic treatment.[1-5]

Since WSLs are caused by plaque and bacteria accumulation, they are therefore preventable with good oral hygiene. However, most orthodontic patients are adolescents with worse oral hygiene practices than adults, and braces tend to increase plaque retention. While good oral hygiene may prevent WSLs, very good compliance is required in order to do so. Therefore, several WSL preventive methods have been attempted that do not require as much compliance, including glass ionomer cements, fluoride-releasing cements, fluoride rinses, fluoride varnish, and resin sealants. This study is focused on resin sealants as a preventive measure against WSLs.

This literature review will first describe white spot lesions and how they occur. Next, it will look at factors involved in causing WSLs, as well as how often and where they occur. Then, treatment and preventive methods will be reviewed, with a focus on the effectiveness of resin sealants at preventing WSLs. Finally, it will address the retention of sealants and factors that can affect sealant retention

White Spot Lesions

White spot lesions (WSLs) are enamel decalcifications that appear whiter than surrounding enamel due to the way light scatters from the subsurface decalcification. A WSL is the first visible sign of enamel demineralization. Usually the surface appears chalky white and is rougher than normal when checked with an explorer. WSLs are the initial signs of a carious lesion. They begin as a subsurface demineralization with an intact enamel surface, and while they usually do not progress to cavitation, in severe cases the demineralization causes porosity in the enamel and demineralization of the surface layer, causing cavitation.[1]

Even if demineralized enamel can be remineralized, visible white spots are usually irreversible. WSLs begin like any carious lesion, when the cyclic process of demineralization and remineralization of the enamel in the oral cavity is imbalanced in favor of demineralization. As the pH of the oral environment decreases from food or beverages, the diffusion of calcium and phosphate ions out of the enamel surface causes demineralization. This is countered by remineralization from the salivary components or fluoride treatment, but when the balance is offset by long periods of demineralization,

the surface loses too many minerals and the lesion progresses beyond what can be remineralized.[6] The result is a white chalky lesion on the enamel that can only be treated with dental restorative methods.

Etiology

The etiology of WSLs is multifactorial, involving microbial factors, diet, salivary components, salivary flow rate, salivary pH, and oral hygiene.

Microbial factors

The first step in the demineralization process is the formation of an organic pellicle on the tooth surface, which is subsequently colonized by bacteria from the oral cavity. *Strep mutans* and *lactobacilli* are two bacteria that have been strongly associated with the progression of caries.[7] An extracellular protein matrix is then created by the bacteria and the colony grows. The bacteria then produce acidic byproducts at a high rate as well an extracellular polysaccharide that increase the adherence of plaque to the enamel surface.[8] Without physical removal from the tooth surface, these bacterial colonies will continue to produce acidic byproducts, shifting the equilibrium from enamel remineralization to demineralization.[9]

Diet

Diet plays a crucial role in the development of caries and demineralization. Carbohydrates serve as the food source for the cariogenic bacteria previously mentioned.

Therefore, frequent consumption of carbohydrate-rich foods and beverages significantly increase the rate of acid production by the bacteria, creating a low pH in the oral environment. Additionally, it has been shown that the consumption of acidic beverages, such as soft drinks, fruit juices, and some sports drinks causes the pH of plaque to drop below the critical pH of 5.5 for approximately 20 minutes.[10] The saliva serves as a buffering agent against acidic attacks by bringing the pH back to safe levels, but if the carbohydrate consumption is too frequent, the buffer system cannot keep up, and the pH of the oral cavity remains too low for longer periods of time. If the enamel surfaces are exposed to such an acidic environment for too long, demineralization then occurs.

Saliva

Saliva plays several important roles in preventing enamel demineralization. It contains water and electrolytes, including sodium, potassium, calcium, chloride, and inorganic phosphate. It has a vital role in caries prevention by neutralizing acid with its buffering capacity, providing minerals necessary for enamel remineralization, and physically cleansing sugars from tooth surfaces.

The buffering capacity allows saliva to neutralize acid from the oral bacteria. The main buffer is the carbonic acid-bicarbonate system, which maintains a pH range of 6.2-7.4 in healthy adults. Thus the buffer system helps keep the pH above the critical value of 5.5, below which acid causes minerals to be lost from the enamel surface.[8] Additionally, saliva provides those minerals, like calcium and phosphate, which are necessary to keep enamel mineralized. Finally, salivary flow itself cleanses the tooth

surface of plaque and bacteria. Because of all these factors, it makes sense that increased salivary flow rate has been shown in several studies to decrease the rate of demineralization progress, increase the likelihood of enamel repair, decrease caries risk and activity, and increase the ability to buffer the pH drop in fluid following an acid attack.[6,11-13]

Orthodontic Treatment

Orthodontic treatment itself does not cause WSLs to develop. However, it has been shown that orthodontics appliances and composite predispose patients to greater plaque accumulation. One study demonstrated that the overall oral bacterial count in saliva increased after placement of appliances.[14] Another showed that *S. mutans,* specifically, increased after placement of orthodontic appliances.[15] A third study showed via scanning electron microscopy that the increase in bacterial plaque occurs as soon as one week after bonding brackets.[16] This is likely due to the irregular surfaces of the brackets, which allow for greater plaque accumulation and cause greater difficulty in cleaning the tooth surfaces.

Another factor in treatment besides the appliances is the composite resin adhesive used to bond the brackets. In the SEM study mentioned previously, the biofilm was actually thicker on the composite surfaces then on the enamel surfaces near the brackets. Other studies also confirm that composite surfaces increase plaque accumulation.[16-18] Hess et al showed recently *in vitro* that the decalcification increased around composite resin, but not around orthodontic brackets themselves.[19] Overall, it is

clear that during orthodontic treatment, patients have a higher risk for plaque accumulation and demineralization.

Prevalence

The prevalence of WSLs varies greatly in the literature, from 2-96%.[1,20-22] However, the highest rates don't take into account comparisons to the control groups. One study showed 84% prevalence, but 72% of patients had previous WSL.[20] Another study reported 96% of patients had WSLs, but the detection method was very sensitive, so the control group also had a high rate of 85%. Gorelick et al, using clinical exam and photographs, reported that 50% of orthodontic patients saw an increase in WSL during treatment, affecting 10% of teeth.[1] Lovrov et al used before and after photographs and found an increase in WSL during treatment of 94.3% of patients, affecting 24.9% of teeth.[2] Chapman also used photographs but looked at the upper eight anterior teeth only, and he reported that 36% of patients developed at least one new WSL.[3] In a recent large-scale study, Julien et al showed that 23% of patients developed at least one WSL during their orthodontic treatment. Another large-scale study by Brown et al utilized a practice-based research network and found that 28% of private practice patients developed a WSL, affecting 12.7% of teeth.[5] Clearly, the development of WSLs during treatment is a significant problem in orthodontics.

Location/Distribution of WSLs

Some of the previous studies noted not only how many WSLs developed, but where those WSLs were located on the teeth. Gorelick et al reported that maxillary lateral incisors and mandibular canines had the highest frequency of WSLs.[1] Ogaard's results agreed with Gorelick, showing maxillary laterals and mandibular canines were most affected.[22] Mizrahi also reported maxillary lateral incisors and mandibular canines being most frequently affected, in addition to mandibular lateral incisors. Mizrahi also noted that the WSLs were most often found in the cervical and middle thirds of crowns.[20,23] Chapman, in evaluating maxillary teeth only, reported that lateral incisors were most frequently affected.[3] The general consensus, then, is that maxillary lateral incisors and mandibular canines develop WSLs the most frequently, with the gingival and middle thirds of the facial surfaces most likely to be affected.

Rate of WSL Development

While long treatment duration has been implicated as a risk factor for WSLs, the process of decalcification can occur quite rapidly during orthodontic treatment. In the study by Ogaard et al, premolars were loosely banded for four weeks prior to being extracted. In only four weeks, some of those premolars had already developed decalcification lesions. In a similar study, O'Reilly and Featherstone bonded brackets to premolars then extracted them four weeks later. Just as in Ogaard's study, enamel decalcification was present within those first four weeks. It is clear that WSLs can begin to develop very quickly in the presence of orthodontic appliances.

Treatment of WSLs

Several methods have been proposed to treat or decrease the appearance of WSLs. While some methods have shown promise, studies show that the ability to remineralize or remove WSLs depends on the severity of the lesions. Small WSLs can be removed with surface abrasion using a hand piece and polishing bur.[24] One study showed that microabrasion with 18% hydrochloric acid and pumice was effective at improving WSLs cosmetically.[25] In another study, a daily regimen of fluoride toothpaste and rinses alone was successful in remineralizing WSLs.[9,26] However, according to Ogaard, highly concentrated fluoride is contraindicated for WSL treatment because it causes hypermineralization of the lesion. While this does prevent further demineralization, it causes the lesion to be stained, remaining permanently on the enamel surface.[27]

Another treatment option for WSLs is the use of a milk protein called Casein Phosphopeptide Amorphous Calcium Phosphate (CPP-ACP). It has shown some success in several studies at remineralizing enamel. Bailey et al in a randomized controlled trial showed that the application of a cream containing CPP-ACP caused 31% more WSLs to regress than a placebo cream.[28] A recent systematic review concluded that CCP-ACP cream does have a long term remineralizing effect on early carious lesions compared to a placebo, but that the effects are similar to that of fluoride containing products.[29]

Knosel et al showed that external bleaching can be successful in camouflaging WSLs.[30] However, bleaching should only be done in patients with good oral hygiene since caries susceptibility increases after bleaching.

A recently developed procedure for WSLs is resin infiltration. In this method, a very low-viscosity resin is diffused into the enamel surface to mask the appearance of the lesion, strengthen the enamel and obstruct further demineralization. Kim et al demonstrated that resin infiltration completely masked the appearance of WSLs in 61% of teeth and decreased the appearance of WSLs in another 33% of teeth.[31]

While all of these methods have shown some promise with small WSLs, none of the methods can treat larger or cavitated lesions, and none of the treatment modalities is successful at treating every WSL. Many lesions must remain as unsightly scars on the teeth or be restored with operative and esthetic dentistry, which requires significant time and expenses. Thus, prevention of WSLs is crucial during orthodontic treatment.

Methods of WSL Prevention

Various methods to prevent WSLs have been shown to be successful. Oral hygiene has been shown to be the primary factor in WSL development, and oral hygiene regimens with good patient compliance have been shown to successfully prevent WSLs. Lovrov et al prescribed weekly use of a fluoride gel and saw a decrease in WSLs over 12-18 months.[2] Stratemann et al had patients use a 0.4% stannous fluoride gel and saw a 31% reduction in decalcification.[32] Daily fluoride mouth rinses showed a 25-31% decrease in WSLs.[32,33] Daily use of MI Paste, which contains CPP-ACP showed a 53.5% reduction in WSL severity in one study.[34] However, all of these preventive measures require good patient compliance.

Methods that do not require patient compliance include professional varnish application, glass ionomer cements, and facial surface resin sealants. In a recent study, fluoride varnish application led to a decrease in WSL incidence by 50% when the varnish was applied every 12 weeks.[35] MI Varnish, containing CCP-ACP, has been shown to be comparable and possibly better than fluoride varnish. Pithon et al compared fluoride varnish to MI varnish in an in vitro study and showed that enamel decalcification depth around orthodontic brackets was less in the MI varnish group than the fluoride varnish group.[36] Varnishes can be effective but must be reapplied frequently by a professional.

A recent systematic review showed that glass ionomer cement can reduce the incidence of WSLs by up to 65% but that better studies need to be done. Also, the concern with glass ionomer cement is reduction in bond strength, which doesn't make them a practical option in orthodontic bonding.[37] Facial surface resin sealants are therefore promising because they do not decrease bond strength to a significant degree[38] and they remain on the teeth for a longer period of time than varnish.

Sealants

Filled resin sealants were shown in 2008 to be effective at preventing WSLs in an in-vitro study.[39] 32 extracted teeth were divided into four groups, receiving either no treatment, fluoride varnish, unfilled resin sealant, or filled resin sealant (Pro Seal, Reliance Orthodontic Products, Itasca, IL), then subjected to simulated brushing followed by acidic challenge. The Pro Seal group performed significantly better than all

other groups, decreasing lesion depth by 97% and completely preventing WSL formation in 3/8 teeth.[39] Other studies have shown similarly successful results. An in-vivo split mouth study in 2009 showed that teeth without sealants developed 3.8 times the number of WSLs than teeth that had sealant applied in only the gingival region of anterior teeth.[40] Heinig later showed that covering the entire labial tooth surface before bonding reduced both severity and depth of demineralization.[41]

Further studies sought to improve on the properties of facial surface resin sealants. Filler concentration was evaluated by Van Bebber et al in 2011, and she showed that adding more filler to a saturated resin matrix does not increase the wear resistance. She also suggested that if smaller nanoparticles are added to the matrix rather than same size filler particles, less of the weaker resin matrix would be exposed to environmental factors, thus improving resistance.[42]

As with glass ionomer cements, a concern with using resin sealants is bracket bond strength. Lowder investigated this question using adhesive-sealant combinations versus adhesive-only controls and found that, while there was a slight decrease in bond strength, it remained well above the clinically acceptable level and is therefore not a concern.[38]

Sealant Retention

It is clear from previous studies that filled resin sealants are effective in reducing enamel decalcification, but their effectiveness is dependent on their retention to the enamel surface throughout the duration of treatment. The dental literature has reported

on the retention of pit and fissure sealants on occlusal surfaces many times, and the average retention is 81-95% over one year and 77-92% over two years.[43-48] Boksman et al and Handelman et al showed that 75-84% of the sealants were left after 2 years.[43,45] Feigal et al quantified the loss at about 5-10% loss per year.[44] Li et al showed that the greatest sealant loss was greatest during the first 6 months after sealing.[46] However, those sealants have the benefit of mechanical retention from the occlusal anatomy of molars and premolars. There is little research regarding retention of smooth surface sealants such as those used in orthodontics. In Van Bebber's in vitro study with a filled resin orthodontic sealant, Pro Seal retention on extracted teeth over a simulated two years of brushing ranged from 90-97%.[42] Chau compared in vivo sealant retention when using conventional 37% phosphoric acid etch with a self-etching primer prior to application of Opal Seal orthodontic sealant (Ultradent, South Jordan, UT). The traditional acid etch method showed significantly less sealant loss, but still had an average sealant loss of about 25-50% of the initial sealant surface area.[49] This rate of sealant loss is much greater than Van Bebber's in vitro sealant loss rates of 90-97%. This is likely due to the fact that in vitro simulations cannot completely replicate the oral environment. During in vitro application of sealants, isolation from blood, saliva, and gingival crevicular fluid are not significant factors like they are in vivo. Chau's study found that by far the majority of the sealant loss took place by the first time point, 4 months after initial sealant application. It could have occurred much earlier, since sealants were not checked previous to that time. Sealant loss occurring not long after the initial application indicates that contamination during the bonding process could have

contributed to the loss of sealant. Additionally, edge loss rather than non-edge loss could be explained by moisture contamination at the edges of the sealant, particularly in the gingival regions, which showed greater sealant loss than mesial and distal edges, only slightly less than incisal edges. This all leads to the conclusion that improved isolation of the teeth during sealant application could increase the retention of the sealant over time.

Previous sealant retention studies have used cheek retractors for isolation, but no isolation was used near the gingival margin. Several options are potentially available to isolate the gums from the teeth, including light-cured dental dams, gels, traditional rubber dams, and gingival retraction cord. Light cured dams and gels both isolate well, but they infringe upon the enamel surface, hindering the application of a sealant to the entire facial surface. A rubber dam or gingival retraction cord are both effective at isolating individual teeth, and in a recent randomized clinical trial, it was shown that retention of restorations was similar when using either a rubber dam or gingival retraction cord for isolation.[50] Therefore, it is proposed that the use of gingival retraction cord for placing sealants will improve tooth isolation and therefore increase sealant retention over time.

CHAPTER II

MATERIALS AND METHODS

Thirty-two patients starting orthodontic treatment at Texas A&M University College of Dentistry were selected based on the following criteria: healthy with no significant medical problems, under 18 years of age, and having a treatment plan that included bonded orthodontic brackets on the maxillary anterior teeth. Teeth in anterior crossbite or teeth inaccessible for sealant application were excluded. Before treatment, written informed consent to participate in the study was obtained from each patient; the study was approved by the Institutional Review Board of Texas A&M University College of Dentistry (IRB2016-0534).

The study utilized a split mouth design, randomly assigning either the upper right or upper left anterior teeth-- central incisors, lateral incisors, and canines-- to either the experimental or control sides. A random number generator in Excel was used to randomly assign the sides of the mouth. Gingival retraction cord was placed on the experimental side prior to applying the sealant; the contralateral teeth did not have retraction cord placed prior to applying the sealant.

Each patient had their upper anterior teeth sealed. Unerupted or missing teeth were not included in the study. There was a total of 182 teeth evaluated. One operator placed the gingival retraction cord and applied the sealant on all of the teeth. The teeth were first isolated with cheek retractors, cleaned with a slurry of non-fluoridated flour of pumice and water with a hand-held prophy brush, rinsed, and then air dried.

Clinical Protocol

On the experimental side, size #00 knitted cotton gingival retraction cord (Ultrapak, Ultradent, South Jordan, UT) was placed in the gingival sulcus of the central incisors, lateral incisors, and canines (Figure 1). Size #00 cord was used because it could be inserted in the gingival sulcus without patient discomfort. The cord was first soaked in water to allow for easier placement. Excess water was then squeezed out of the cord, and the cord was placed next to the gingival margin with cotton pliers. Using a bracket-positioning tool (ODG-88002, Dentsply GAC, Bohemia, NY), the cord was pressed into the sulcus until it was submerged just below the gingival margin.

The teeth were air dried before placing 37% phosphoric acid etchant gel (Reliance Orthodontic Products, Itasca, IL) on the entire facial surface of all six teeth. After 20 seconds, the teeth were rinsed completely with water, and then thoroughly dried with an oil-free and moisture-free syringe until the enamel surfaced appeared frosty white. All teeth (both experimental and control) then had a thin layer of 18% filled Pro Seal orthodontic sealant (Reliance Orthodontic Products) applied to the entire facial surface according to the manufacturer's instructions. The sealants were light cured for 12 seconds each using an Ortholux Luminous Curing Light. (3M Unitek, Monrovia, CA) After curing the sealant, the retraction cord on the experimental side was gently pulled out of the gingival sulcus. Full coverage of the facial surfaces was verified using a hand-held black light. Orthodontic brackets were then bonded on the sealed teeth by other operators using Transbond XT adhesive. A variety of orthodontic brackets were placed by different orthodontic residents.

Records

After bracket placement, initial intraoral photographs of each tooth were taken by a blinded investigator with a Canon T3i digital camera, using a 100 mm macro lens in a dark room with a hand-held black light to visualize the sealant (Figure 2). The photographs were taken in Shutter Priority Mode with exposure time of 1/160 second and ISO 1600. Progress intraoral photographs were taken by the same blinded investigator at subsequent orthodontic visits, approximately 2.8 (T1) and 7.3 (T2) months after initial bonding. If a bracket debonded during the study, no more photos were taken of that tooth.

Analysis of the photographs was performed by a single blinded investigator using the Analyze 12.0 Region of Interest software (Biomedical Imaging Resource, Mayo Clinic). A photo of each tooth was uploaded to the program and displayed on a 21-inch LCD monitor. Using the mouse cursor, the entire enamel surface was traced, excluding the bracket and archwire. The tooth surface area above the archwire was labeled as the gingival tooth surface area; the area below the archwire was labeled as the incisal tooth surface area. The fluorescent sealant on each tooth was then outlined using the mouse cursor (Figure 3). By comparing the sealant surface area and the total tooth surface area, the percentage of sealant remaining on the enamel surface was calculated.

SPSS version 23 (SPSS Inc., Chicago, IL) was used to analyze the data (at a significance level of $p<0.05$). The skewness and kurtosis statistics showed that the distributions were normal. Generalized linear models were used to estimate sealant loss. Multivariate tests were used to determine if there were differences in sealant loss

between teeth, as well as differences in sealant loss between the control and experimental sides. These were followed up with pairwise comparisons to evaluate differences between teeth and between the two sides.

CHAPTER III

RESULTS

During the study, due to scheduling conflicts, one patient was unable to have T1 photos, and 2 patients were unable to have T2 photos. Some teeth that had their entire gingival tooth surfaces covered by hyperplastic gingiva were not evaluated. A total of 25 patients had complete data for both time points. T1 and T2 records were taken approximately 2.8 ±1.2 months and 7.3 ±1.6 months, respectively, after initial bonding.

Replicate analyses of 12 teeth showed no statistically significant systematic measurement errors. Method error was 0.72% for total sealant measurements and 0.90% for gingival sealant measurements. The intraclass correlation coefficient was 0.996.

Overall Tooth Surface

Initially (T0), 100% of the teeth's surface areas were covered with sealant. At T1, approximately 59.5% and 64.6% of the sealant remained on the control and experimental sides, respectively. This was not a statistically significant difference, but closely approached the significance level ($p = 0.052$) (Table 1, Figure 4). The central incisors had 62.7%, the lateral incisors had 59.0%, and the canines had 64.4% of sealant remaining on their surfaces (Table 2, Figure 5). The difference between the amounts of sealant remaining on the lateral incisors and canines was significant ($p = 0.006$) (Table 3, Figure 5).

At T2, there was a significant difference ($p = 0.021$) between the control and experimental sides in the amount of sealant remaining on teeth. The control side had 48.7% sealant remaining, while the experimental cord side had 53.9% of the sealant remaining (Table 1, Figure 6). The central incisors had 51.5%, the lateral incisors had 50.2%, and the canines had 52.2% of the sealant remaining (Table 2, Figure 7), with no statistically significant differences between any of the teeth (Table 3).

Gingival Tooth Surface

At T1, the amount of sealant remaining in the gingival half of the control teeth was 52.3%, while the sealant remaining on the experimental side was 58.2%. This difference closely approached a statistically significant level ($p = 0.054$) (Table 1, Figure 8). The lateral incisors had significantly less gingival sealant remaining than central incisors ($p = 0.012$) and canines ($p < 0.001$) (Table 3, Figure 9). Approximately 46.9%, 56.5% and 62.5% of the gingival sealant remained on the lateral incisors, central incisors and canines, respectively (Table 2, Figure 9).

At T2, there was significantly ($p = 0.019$) more gingival sealant remaining on the experimental than control side. The control side had 38.7% of sealant remaining on the gingival surface, while the cord side had 46.3% of sealant remaining (Table 1, Figure 10). Again, the lateral incisors showed significantly greater loss than the central incisors ($p = 0.010$) and canines ($p =0.006$) (Table 3, Figure 11). Central incisors, lateral incisors and canines had 46.3%, 35.2%, and 46.1% gingival sealant remaining, respectively (Table 2, Figure 11).

CHAPTER IV

DISCUSSION

Whether or not retraction cord was used, the amount of sealant loss was great. During the course of the present study, the control teeth lost an average of 40.5% of sealant over 2.8 months and 51.3% over 7.3 months of orthodontic treatment time. This loss of sealant is higher than the 3-16% reported for controls in other in-vitro studies,[42,51] but similar to the 25-50% loss reported in-vivo.[49,52] Lower in-vivo retention rates would be expected because the in-vitro conditions cannot adequately simulate the fluctuations in temperature, acidity, moisture, and masticatory effects, all of which have been shown to decrease resin retention.[53-57]

There appears to be greater sealant loss in the gingival region than the coronal aspect of teeth. After 2.8 months, gingival loss was 7.2% greater than overall loss for the control teeth, and after 7.3 months, sealant loss was 10% greater in the gingival region. A factor that could play an important role in gingival sealant loss is the susceptibility of the enamel surface to etching and bonding. Enamel has two layers, an outer prismless layer and inner prismatic layer.[58] The prismless layer has enamel rods that are oriented parallel to the outer surface, and have been shown to have less porosity and surface roughness after etching than prismatic enamel.[59] The cervical region of teeth has more prismless enamel and is thinner than the middle and incisal regions of the enamel.[60] When sealant is applied to the etched enamel, in the cervical region, the resin tags are shorter and fewer in number than in other regions of the enamel surface.[60] This

difference could explain why a great percent of sealant is lost, especially in the gingival region of teeth.

Chau et al reported that the gingival sealant loss was not significantly greater,[51] but they may not have been able to detect a 7-10% difference due to the less precise nature of their measurements. The digital tracing method used to quantify sealant loss in the current study was reliable and more precise than the methods used in previous sealant retention studies because it used quantitative analysis rather than qualitative analysis. Rather than tracing exactly where the sealant was located, previous investigators divided the tooth surface into quadrants and estimated loss on a 0-4 scale, based on percentages of 0, 25, 50, 75, and 100% loss.[49,52] The analysis performed in this current study was more quantitative, tracing tooth and sealant surface areas, which allowed more precise calculations of the sealant loss that had occurred.

Of the sealant loss that occurred during the study, the majority occurred during the first three months. At T1, 2.8 months after bonding, 40.5% of sealant had been lost overall, while at T2, 7.3 months after bonding, only 10.8% more of the sealant had been lost. Nearly 4 times the amount of sealant was lost in the first 3 months than the next 4 months. Pit and fissure sealant loss is also greatest during the first 6 months after application,[46] and smooth surface sealant loss is greatest during the first 4 months after application.[49] This suggests that sealant loss is a problem related to the bonding process rather than mechanical abrasion. If sealant loss occurred primarily due to mechanical abrasion, then the loss would likely be more regular over time, with similar amounts of sealant being lost early and late. Previous studies have not looked at sealant loss prior to

4 months, making the 2.8 months in the present study the earliest that retention of smooth sealants has been studied. It is possible that the majority of sealant loss occurs even earlier, perhaps days or weeks after bonding.

Sealant loss differs among teeth. In the present study, the lateral incisors had significantly greater gingival sealant loss than either the canines or central incisors. Chau et al showed that, in-vivo, maxillary canines had greater sealant loss than lateral incisors; the difference was small but statistically significant.[49] This difference could have been caused by their qualitative measurements not being as precise as the current study's quantitative measurements. They suggested that the lateral incisors have less sealant loss because they have less toothbrush abrasion. However, toothbrush abrasion is not likely the primary cause of sealant loss. Previous in-vitro studies showed much less sealant loss than the in-vivo studies, and they simulated two full years of brushing.[42,51] If brushing were the primary cause of sealant loss, those studies would have shown much greater loss than the current study showed after only three months of brushing.

Although no other studies have shown decreased adhesive characteristics of lateral incisors, numerous studies have shown that lateral incisors are one of the most common locations for white spot lesions to occur.[1,3,4,20,61] It has also been shown that the maxillary lateral incisors have a higher percentage of roughened enamel surface after etching than all other permanent teeth.[62] This suggests that there may be something different about the enamel of lateral incisors, which could also affect sealant retention.

It is possible that different teeth and different regions of teeth may etch differently and therefore require more specific etching protocols per tooth. The current

study used a protocol of etching with 37% phosphoric acid for approximately 20 seconds per tooth. The manufacturer's recommendation is 15 seconds. However, surface roughness is actually increased with longer etch times when 37% phosphoric acid is used,[63] and multiple studies have found no differences in bond strength after etching for 15, 30, or 60 seconds.[63-65] Studies are needed that evaluate differences in the distribution of prismless or prismatic enamel and determine how it relates to etching patterns and sealant adhesion.

Gingival retraction cord has an effect on sealant loss, albeit a small effect. The experimental side in the present study had 5.2% less overall sealant loss and the gingival region had 7.6% less loss on the experimental than control side. This indicates that gingival retraction cord has a beneficial effect on sealant retention. The improved sealant retention when using cord may have come from exposing more of the gingival enamel surface prior to sealant application or from improved isolation. No previous studies have evaluated isolation methods for retention of smooth surface sealants. A randomized, split-mouth study found that the rubber dam isolation side had 26% more fully intact pit and fissure sealants than the cotton roll side.[66] However, even with rubber dam isolation, another study reported that 32% of the pit and fissure sealants showed some loss after one year. For cervical restorations, gingival retraction cord has been shown to be as effective as a rubber dam for isolating a tooth during bonding.[50]

The protocol used in the present project was based on the study by Loguercio et al, which placed gingival retraction cord and then etched, rinsed, and bonded.[50] A completely dry gingival sulcus is difficult to achieve. If the cord is placed after etching

and rinsing, there is a risk that blood or gingival crevicular fluid will be pressed out by the cord onto the etched surface. By placing the cord before etching and rinsing, the cord gets wet during the rinsing process. Therefore, for ease of placement, the cord in the present study was wetted before being placed in the sulcus because it was going to eventually be wetted during the rinsing process. Loguercio et al showed that even when wet, cord isolation was as effective as rubber dam isolation.[50]

The small increase in sealant retention when using gingival retraction cord was statistically significant but would not likely be considered clinically significant. However, the large and rapid loss of sealant is vital information for the clinician. While sealants have been shown to be successful at preventing white spot lesions,[39-41] they cannot protect the enamel if they are not retained long term. Clinicians strive to provide excellent orthodontic correction while not causing damage to the teeth during that process. Therefore, prevention rather than restoration must be the standard, and further research needs to be done to achieve such a goal.

The present study showed that when sealants are applied before bonding brackets, almost half of the gingival enamel surface is exposed after less than 3 months of treatment time. Therefore, in order to be effective, sealants must be reapplied regularly, the protocol for etching and bonding sealants must be improved, or the sealant composition must be changed to increase adhesion to enamel surfaces.

CHAPTER V

CONCLUSIONS

1) Whether or not retraction cord is used, large amounts of sealant are lost.
2) Approximately 40% of the sealant is lost within the first 3 months of treatment.
3) There appears to be greater sealant loss in the gingival region than the incisal aspect of teeth.
4) Lateral incisors have greater sealant loss than other maxillary anterior teeth.
5) Gingival retraction cord has a minimally positive effect on preventing sealant loss.

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APPENDIX A

FIGURES

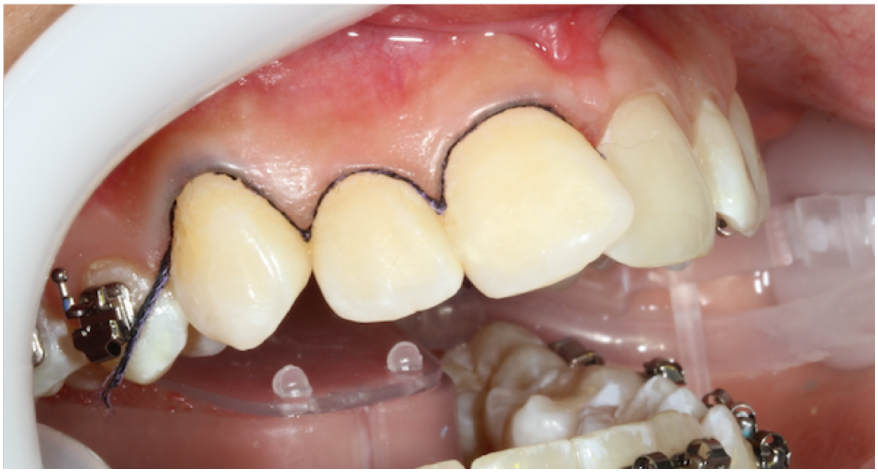

Figure 1. Gingival retraction cord tucked into the gingival sulcus of the central incisor, lateral incisor, and canine, prior to etching and applying the sealant.

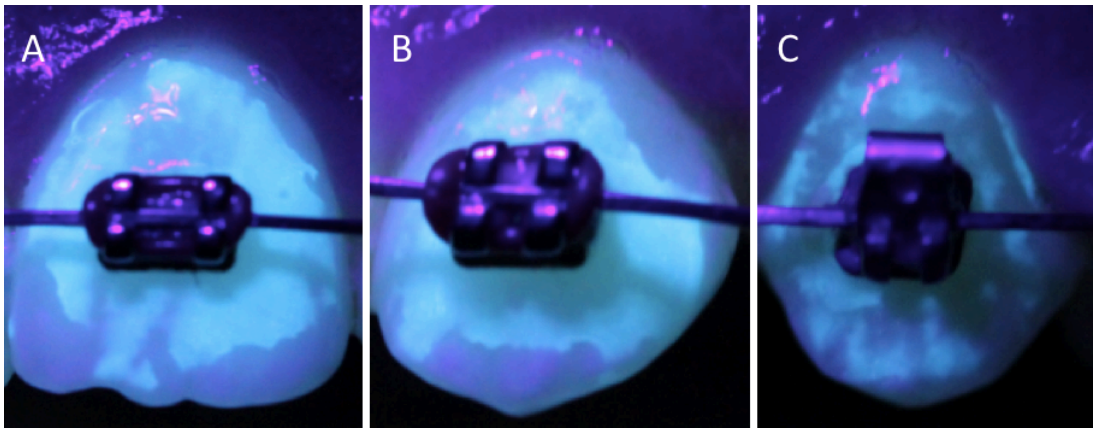

Figure 2 Examples of black light photos of individual teeth, (A) central incisor, (B) lateral incisor and (C) canine.

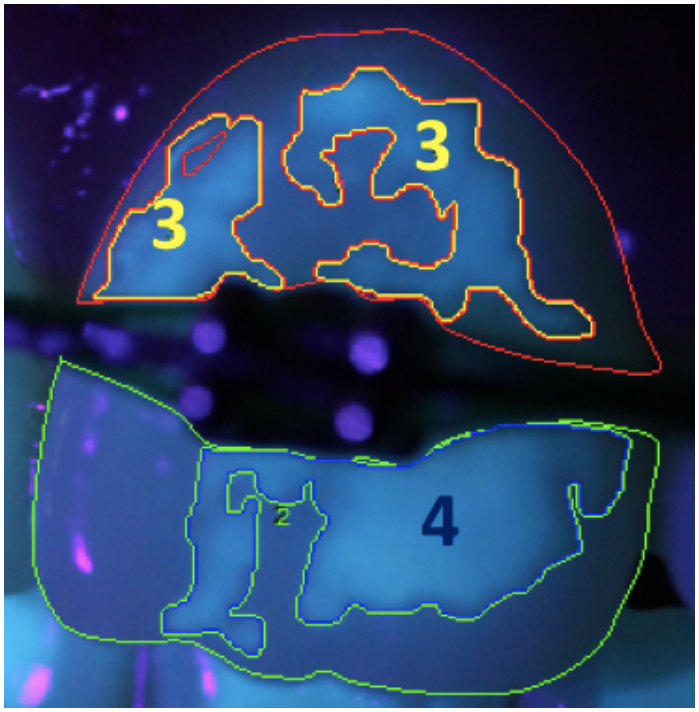

Figure 3 Example of digitally traced sealant. The upper red outline is the gingival half and the lower green outline is the incisal half of the tooth surface. The fluorescent sealant was outlined in each region, indicated by the regions labeled 3 and 4.

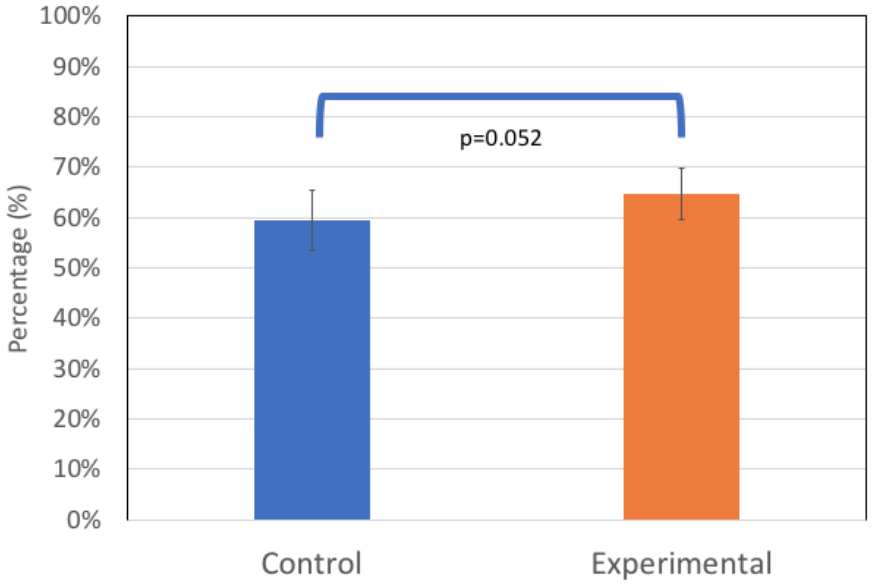

Figure 4 Percentage (± 1.96 S.E.) of sealant remaining on overall tooth surfaces at T1 (2.8 months) on the control (without retraction cord) and experimental (with retraction cord) sides.

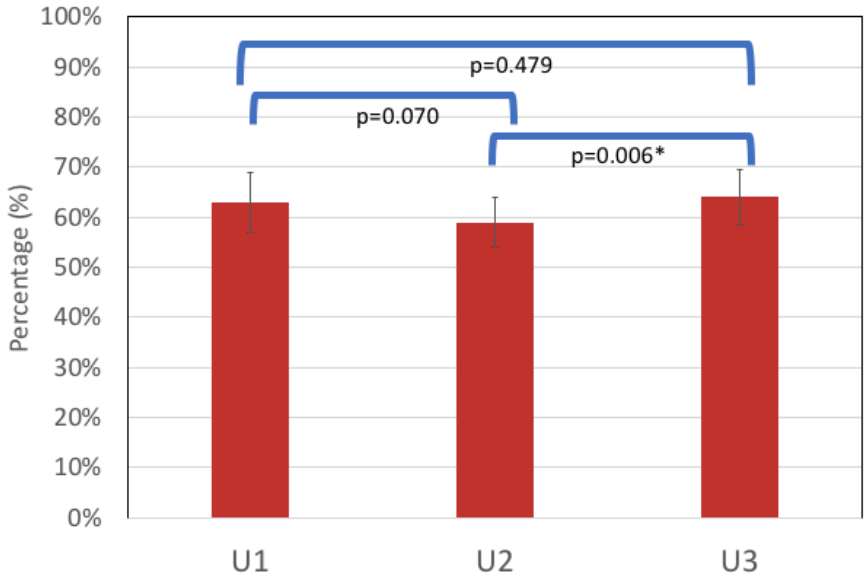

Figure 5 Percent (± 1.96 S.E.) of sealant remaining on overall tooth surfaces at T1 (2.8 months) on the U1 (central incisor), U2 (lateral incisor) and U3 (canine).

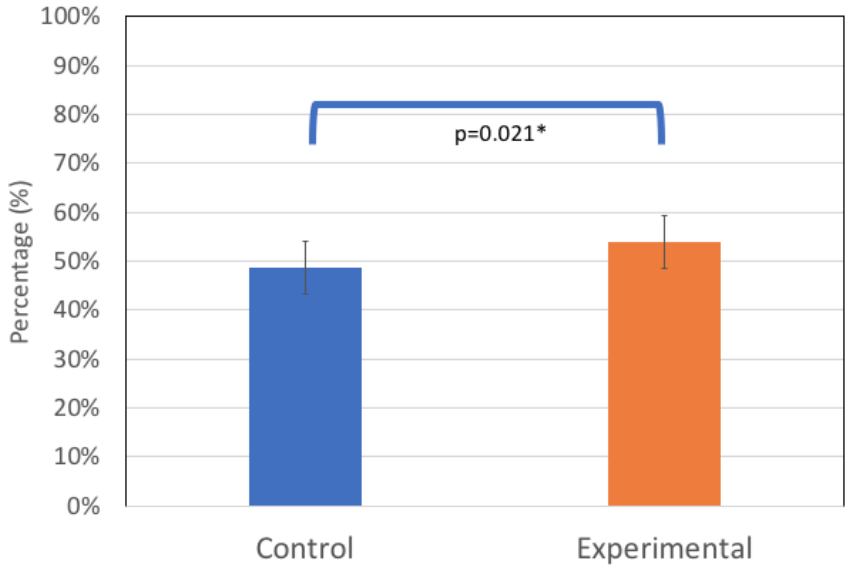

Figure 6 Percentage (± 1.96 S.E.) of sealant remaining on overall tooth surfaces at T2 (7.3 months) on the control (without retraction cord) and experimental (with retraction cord) sides.

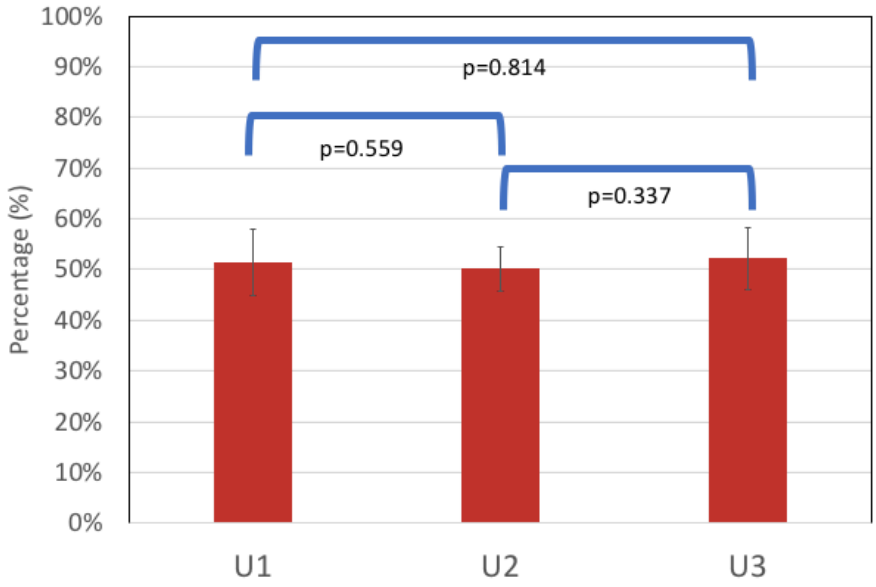

Figure 7 Percentage (± 1.96 S.E.) of sealant remaining on overall tooth surfaces at T2 (7.3 months) on the U1 (central incisor), U2 (lateral incisor) and U3 (canine).

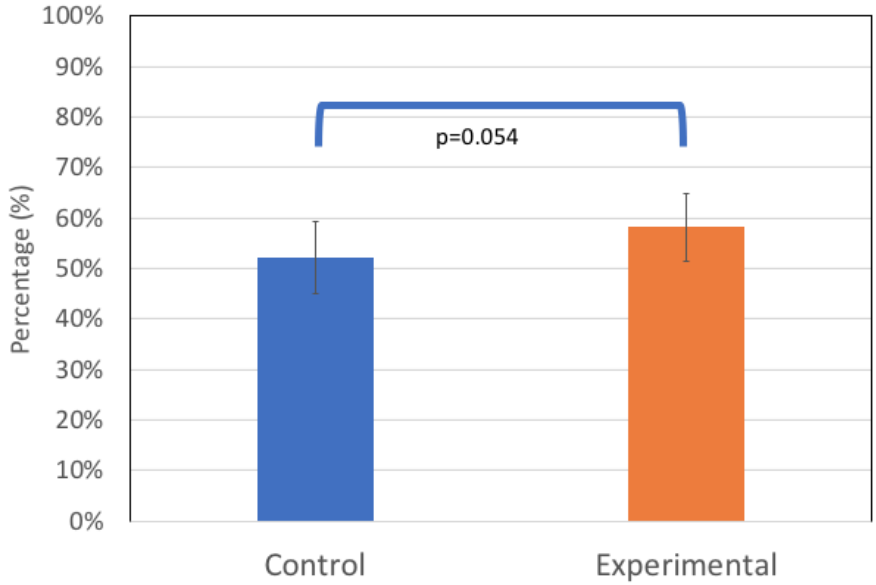

Figure 8 Percentage (± 1.96 S.E.) of sealant remaining on gingival tooth surfaces at T1 (2.8 months) on the control (without retraction cord) and experimental (with retraction cord) sides.

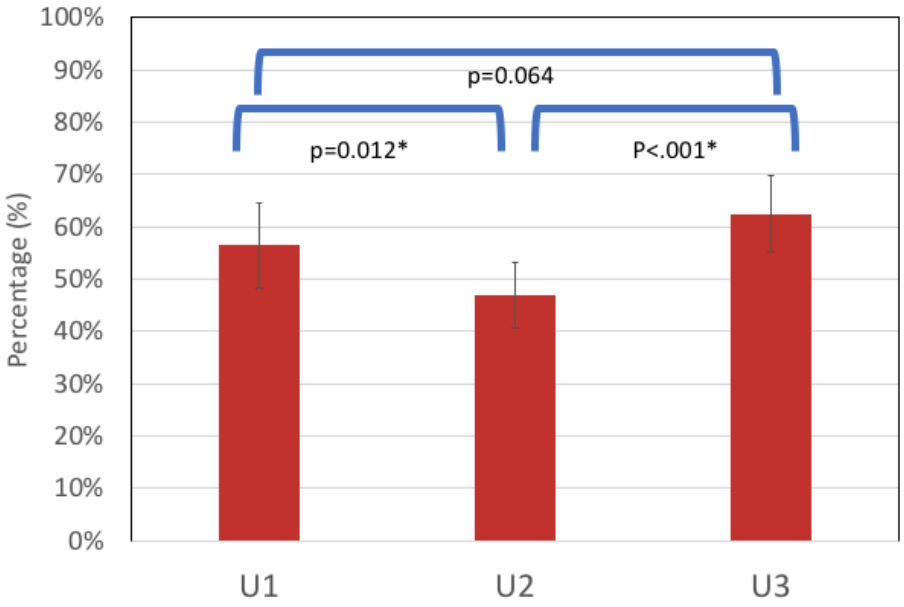

Figure 9 Percentage (± 1.96 S.E.) of sealant remaining on gingival tooth surfaces at T1 (2.8 months) on the U1 (central incisor), U2 (lateral incisor) and U3 (canine).

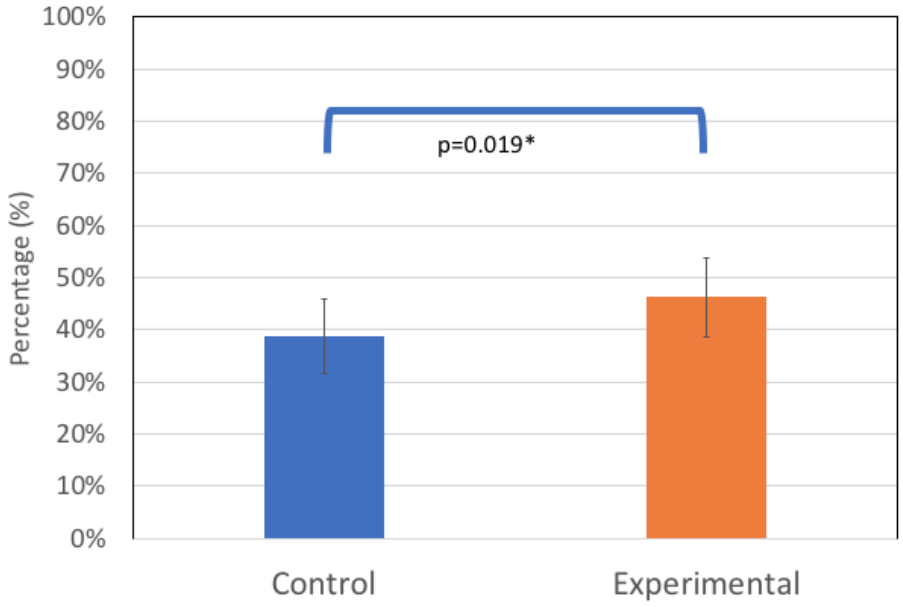

Figure 10 Percentage (± 1.96 S.E.) of sealant remaining on gingival tooth surfaces at T2 (7.3 months) on the control (without retraction cord) and experimental (with retraction cord) sides.

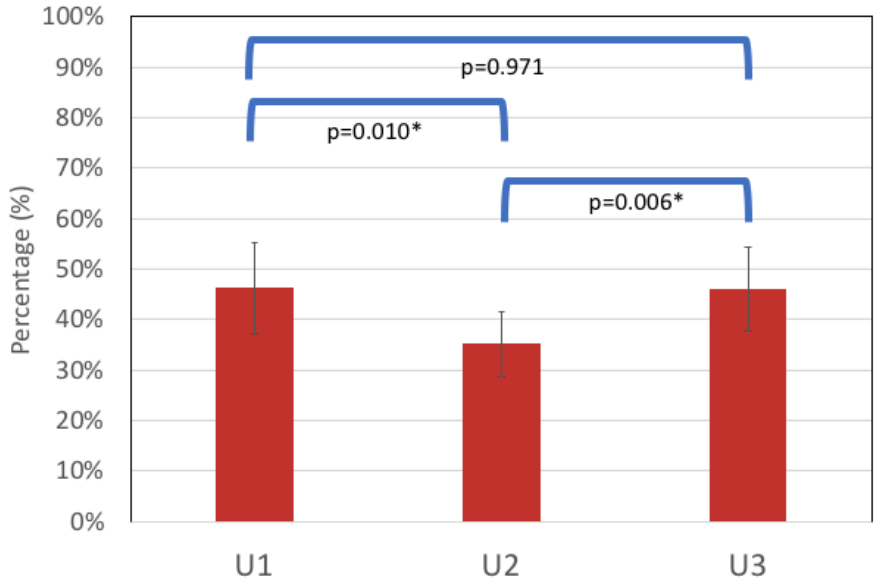

Figure 11 Percentage (± 1.96 S.E.) of sealant remaining on gingival tooth surfaces at T2 (7.3 months) on the U1 (central incisor), U2 (lateral incisor) and U3 (canine).

APPENDIX B

TABLES

	Control		Experimental		Difference	
	Mean	SE	Mean	SE	Mean	P-value
Overall surface T1	59.5	3.0	64.6	2.6	5.1	0.052
Overall surface T2	48.7	2.8	53.9	2.7	5.2	0.021*
Gingival surface T1	52.3	3.7	58.2	3.4	5.9	0.054
Gingival surface T2	38.7	3.6	46.3	3.9	7.6	0.019*

Table 1 Comparisons of percentage (%) sealant remaining on the control versus experimental sides. Control, without retraction cord, experimental, with retraction cord. * $P < 0.05$, statistical significance

	U1		U2		U3	
	Mean	SE	Mean	SE	Mean	SE
Overall surface T1	62.7	3.0	59.0	2.5	64.4	2.8
Overall surface T2	51.5	3.3	50.2	2.2	52.2	3.1
Gingival surface T1	56.5	4.2	46.9	3.2	62.5	3.7
Gingival surface T2	46.3	4.6	35.2	3.3	46.1	4.3

Table 2 Comparisons of percentages (%) of sealant remaining on different teeth. U1, central incisor, U2, lateral incisor, U3, canine.

	U1-U2	U2-U3	U1-U3
Overall surface T1	0.070	0.006*	0.479
Overall surface T2	0.559	0.337	0.814
Gingival surface T1	0.012*	<.001*	0.064
Gingival surface T2	0.010*	0.006*	0.971

Table 3 Pairwise comparisons of percentage (%) sealant remaining between different teeth. U1, central incisor, U2, lateral incisor, U3, canine.