Chapter from the book *Issues in Contemporary Orthodontics*
Downloaded from: http://www.intechopen.com/books/issues-in-contemporary-orthodontics

Interested in publishing with InTechOpen?
Contact us at book.department@intechopen.com
Caries Risk and Prevention in Orthodontic Patients

Anas H. Al-Mulla

1. Introduction

The human oral cavity is a complex ecosystem, inhabited by more than 300 bacterial species, mycoplasmas, protozoa, and yeasts [1]. Any external interference could disturb the balance between components of the microflora in this environment. Fixed orthodontic appliances are an example of such an interference. Bonding of brackets usually includes acid etching of enamel, which results in changes in the morphology and chemical nature of the tooth surface. It has been found that decalcified enamel constitutes good support for adhesion and proliferation of mutans streptococci [2]. It is also known that living cells easily adhere and colonize polymeric surfaces [3-5]. Thus, composite resins, containing polymers used for attaching brackets to etched enamel, provide surfaces especially prone to adhesion and growth of microorganisms [6-8]. In effect, fixed dental appliances induce development and retention of bacterial plaque [9-14]. Development of dental plaque usually leads to an increased level of caries-inducing bacteria in the oral cavity (e.g. mutans streptococci and lactobacilli) [2, 15, 16]. These observations indicate that fixed orthodontic appliances induce a certain risk for development of caries.

2. Caries risk in orthodontic patients

The risk of developing a caries lesion around a bracket, placed on the buccal tooth surfaces during orthodontic treatment with fixed appliances, is high [10, 17, 18]. This risk is attributed to the presence of brackets, arch wires, ligatures, and other orthodontic auxiliaries that complicate conventional oral hygiene measures, which in turn, leads to increased plaque accumulation at the base of the brackets [12, 19]. In the presence of fermentable carbohydrates, demineralization of the enamel around the bracket occurs rapidly [10, 12, 20]. Despite improvements in materials and preventive efforts, demineralization may occur around ortho-
dontic appliances after only one month [11]. Children between 11 and 14 years, the age group in which orthodontic treatment is usually carried out, are considered to be at high risk of developing caries [21]. Fixed orthodontic appliances create extra retention sites, leading to more mutans streptococci soon after the start of treatment [22, 23]. A study concluded that measures including intensive brushing and careful cleaning with dental floss of the spaces around brackets under arch wires and between teeth were insufficient to decrease mutans streptococcus and lactobacillus levels. Thus, patients with fixed appliances carry a high caries risk.

2.1. Caries prevalence in orthodontic patients

The prevalence of new enamel lesions among orthodontic patients treated with fixed appliances and using fluoride toothpaste is reported to range from 13 to 75% [10, 14, 24, 25]. Although demineralized enamel can remineralize after de-bonding, the lesions are often irreversible [11-13, 17]. Inactive demineralized enamel was found to be present five years after the completion of orthodontic treatment [18]. The lesions have been reported to develop on all teeth, but are most frequently observed on the cervical and middle third of the buccal surface of the lateral maxillary incisors, the mandibular canines and the first premolars [10, 17]. The long-term presence of enamel lesions, which appear as white spots, is a concern for both the patient and the orthodontic profession.

2.2. Fluoride toothpaste

Fluoride toothpaste has been widely used for more than four decades and remains a benchmark for the prevention of dental caries [26-28]. It reduces caries in both primary and permanent teeth [28]. For this reason, fluoride toothpaste plays an important role as an effective caries prevention measure worldwide [28]. Topical fluoride (mouth rinses, gels, and varnishes), used in addition to fluoride toothpaste, achieves a modest reduction in caries compared with toothpaste used alone [29]. Numerous studies have shown that even low levels of fluoride, resulting from the regular use of toothpaste, have a profound effect on enamel demineralization and remineralization [30, 31]. Considering the widespread use of fluoride toothpaste during orthodontic treatment, there is little evidence as to which method to deliver the fluoride paste is the most effective.

At least four factors influence the anti-caries efficacy of fluoride toothpaste: (1) frequency of brushing, (2) duration of brushing, (3) fluoride concentration in the toothpaste, and (4) post-brushing rinsing behavior. Brushing should be recommended minimum twice daily [32] and patients should be persuaded to brush for longer periods of time [33]. A recent study by Zero et al. [34] concluded that both brushing time and dentifrice quantity might be important determinants both of fluoride retention in the oral cavity and of consequent enamel remineralization.

There seems to be a correlation between the fluoride concentration of dentifrices and caries prevention [35]. Treatment of demineralized dentin with a toothpaste containing 5, 000 ppm
fluoride reduces mineral loss and lesion depth on exposed dentin [36]. In a randomized clinical trial comparing 5,000 and 1,450 ppm fluoride, the high fluoride toothpaste reversed non-cavitated fissure caries lesions [37]. Moreover, the group using 5,000 ppm fluoride showed a significantly higher decrease in laser fluorescence of enamel than the 1,450 ppm fluoride group. Furthermore, dentifrice containing 5,000 ppm fluoride was significantly better at remineralizing root caries lesions than a dentifrice with 1,100 ppm fluoride [38]. In addition, caries active adolescents using the 5,000 ppm toothpaste showed a significantly lower progression of caries compared to subjects using the 1,450 ppm toothpaste, after two years [39].

Rinsing method after brushing teeth has been found to correlate with caries experience and caries increment [40]. Salivary fluoride concentration measured after dentifrice application decreases significantly with increasing water volume, rinse duration, and frequency of rinsing [41, 42]. A toothpaste technique where a slurry rinse with the toothpaste is carried out after brushing increases the efficacy of fluoride toothpaste and reduces approximal caries in preschool children by an average of 26% [43]. A recent study by Sonbul et al. [43] reported 66% preventive fraction effect on approximal caries between the group that used a modified fluoride toothpaste technique compared to the group who continued with their regular oral hygiene habits. Furthermore, eating immediately after brushing reduces the salivary fluoride level about 12-15 times compared with brushing alone [44]. There is an increase of fluoride in both proximal saliva and plaque, using a dentifrice with 5,000 ppm fluoride without post-brushing water rinsing compared to with rinsing [39]. Post-brushing rinsing habits may play an important role in the oral retention of fluoride from dentifrices that may, in turn, affect their clinical efficacy [41].

The specific aim of the chapter is to test the hypothesis that toothpaste slurry rinsing, combined with some other simple post-brushing advice (in this thesis called modified fluoride toothpaste technique [MFTT]), would reduce the number of decayed and filled tooth surfaces (DFS) in a two-year randomized clinical trial in orthodontic patients.

3. Material and methods

3.1. Subjects

The study population consisted of 150 orthodontic patients at baseline, recruited consecutively during a period of six months at a well-established orthodontic clinic in Riyadh, KSA. They were randomly divided into two groups (test group and control group) with 75 individuals in each group. After six months, we had dropout recession around 33% to achieve a final sample size of 100 orthodontic patients (Figure 1). A power analysis with an assumption significance level of 5%, standard deviations of 3.0 DFS, least detectable difference of 2.0 ΔDFS, and a power for that detection of 90% was performed and produced a minimum sample size of 45 observations per group.
3.2. Examination

The examination consisted of recording the plaque index according to Silness and Løe [45]. Registration of caries was done according to WHO [46] after prophylaxis, flossing, and radiographic exam according to Mejàre et al. [47], which consisted four bitewings. A total of 24 surfaces were included in the radiographic DFS index, from the distal of the first pre-molars to the mesial surface of the second molars. Filled surfaces underlined with caries were scored as recurring caries.
Upon completion of the examination, the patients in both test and control groups received a Colgate Max Cavity fluoride toothpaste containing 1450 ppm (Colgate, Riyadh, KSA).

3.3. Oral hygiene instructions including MFTT

The test groups received verbal and written instructions of the MFTT. They were as follows: (1) use 2 cm (1 gram) of dentifrice on a wet toothbrush, (2) spread the toothpaste evenly in the lower and upper arches, (3) brush all surfaces for 2 min, (4) use a small amount of water, equivalent to a handful, together with the dentifrice remaining in the mouth and filter the dentifrice slurry between the teeth by active cheek movements for 30 s before expectorating, (5) avoid further rinsing with water, (6) avoid drinking or eating for 2 h, (7) brush at least twice a day, after breakfast and at night before going to bed, and (8) abstain from all other types of dentifrice during the treatment and until its completion (Figure 2). To ensure that all patients in both groups had a supply of the toothpaste used in the study, they were supplied with the toothpaste at each visit or on request.

The control group was given the routine clinic oral hygiene instructions, which consisted of brushing at least twice a day after breakfast and after dinner before going to bed. At each patient visit to the clinic for the treatment follow-up, the instructions were repeated by the assigned nurse/assistant.

Figure 2. The Modified Fluoride Toothpaste Technique (MFTT).
3.4. Statistical analysis

The Statistical Package for Social Sciences (SPSS Inc., Chicago, IL, USA, version 18.0, Mac OSX) was used for the statistical analysis of the determined measurements in all four studies. ∆DFS and prevented fractions (PF) were calculated according to these two formulas (∆DFS = follow-up DFS - baseline DFS), (PF = [control group ∆DFS - test group ∆DFS]/control group ∆DFS × 100). For the descriptive statistics, the mean values with standard deviations were calculated. To determine statistically significant differences between the groups, the independent sample t-test was applied between the groups. The paired t-test was utilized to check intra-examiner reliability for the radiographic analysis. The 25 randomly selected radiographs were checked within one-week interval. In all the analyses, P < 0.05 was considered statistically significant.

4. Results

4.1. Plaque index, clinical and radiographic DFS

The test and control groups’ baseline and follow-up plaque index, clinical DFS, radiographic DFS, and clinical + radiographic DFS values are shown in Table 1. At baseline, there were no significant differences between the groups. At follow-up, the total number of teeth available was almost the same in both groups (26.9±1.7 test vs. 26.8±1.7 control). At the end of the study, test group patients had a significantly better plaque index in comparison to the control group (P < 0.05). Both groups showed an increase in their DFS index, both clinically and radiographically, with a higher increment in the control group.

|                  | Test (n=51) | Control (n=49) | Test (n=51) | Control (n=49) |
|------------------|------------|---------------|------------|---------------|
| Plaque Index     | 1.4±0.5    | 1.5±0.6       | 1.1±0.8    | 1.6±0.7       |
| Clinical DFS     | 5.6±5.7    | 5.7±5.4       | 5.8±6.0    | 7.4±7.7       |
| Radiographical DFS| 2.7±3.0    | 2.3±3.2       | 3.1±3.0    | 4.1±4.0       |
| Total DFS        | 8.3±7.5    | 8.1±8.4       | 9.0±8.0    | 11.6±10       |

Table 1. Plaque index, clinical DFS, radiographical DFS, and total DFS for test and control groups. Mean±SD are given both at baseline and at follow up. There were no statistical significant differences at baseline and only plaque index was significant at follow-up.

4.2. Caries incidence

The clinical, radiographic, and clinical + radiographic ∆DFS (incidences) are shown in Figure 3. Compared with the test group, the control group patients had > 7 times clinical DFS (P < 0.001), > 4 times radiographic DFS (P < 0.001), and > 5 times clinical + radiographic DFS (P < 0.001), with preventive fraction of 87%, 78%, and 83%, respectively.
5. Discussion

This chapter focuses on caries risk and prevention in orthodontic patients. High concentrated fluoride toothpaste combined with no post-brushing water rinsing and the MFTT could be a suitable regime for patients with high caries risk.

The MFTT aimed to both increase the fluoride concentration and prolong the time during which the fluoride level is elevated in the oral cavity. Spreading dentifrice on the teeth prior to brushing and rinsing with toothpaste slurry immediately after brushing can be expected to produce a more even distribution of the dentifrice and an enhanced fluoride concentration compared with a more conventional technique. Brushing twice daily has been shown to be a very important factor for caries prevention [48], while not eating or drinking for two hours secures a longer time period of elevated fluoride concentration. It must be remembered that the MFTT contains a package of advice. It is possible to speculate about the factor that is most important, but it is not possible to identify a specific one that made our test group patients develop significantly less caries.

Duckworth et al. [49] found that the fluoride concentration in saliva after brushing vanished rapidly as a result of thorough rinsing. Chesters et al. [50] showed that the water-rinsing
pattern among children after brushing influences the cariostatic effect of fluoride toothpaste. They concluded that children who did not use water beakers for rinsing had a significantly lower (16%) caries incidence, during a three-year period than those who used water beakers. Sjögren et al. [43] studied different types of post-brushing behavior; they also evaluated the caries reducing effect of a technique very similar to the one used in Study IV. They concluded that mouth rinsing with the toothpaste foam-water slurry after brushing elevates the concentration of fluoride in saliva for a prolonged period of time as compared to tooth brushing followed by a single or double water rinse; children who used the MFTT developed fewer DFS. These observations are in agreement with Study IV, i.e. the test group patients had a significantly lower mean caries incidence in comparison to the control group. The MFTT technique is easy to teach. Patients can be instructed on how to perform the technique; they can perform it personally in the orthodontic clinic and a pamphlet can be handed to the patients with clear illustrations and instructions.

The large difference found in the ∆DFS between the test and control group patients in KSA should not be expected in other countries with a low DFS prevalence. For example, Sweden has long tradition of using fluoride toothpaste and other fluoride products in orthodontic patients and the expected caries reduction after using the MFTT is therefore lower.

5.1. How to treat orthodontic patients in the sagacity of caries risk and prevention - personal reflections

Orthodontic patients are special kind of patients. They usually come to our clinic complaining about mal-alignment, and few come asking us to solve a functional or prophylactic problem. Most of them underestimate the importance of having excellent oral hygiene practices before and throughout their active prolonged orthodontic treatment. For patients with active caries or having a high risk to develop caries, delaying orthodontic treatment would be my preferred choice. For patients with DMFT index above the mean of their corresponding population, orthodontic treatment should be initiated only when they have been followed over a period of time sustaining excellent oral health habits.

There is a huge responsibility on an orthodontist before bonding brackets and ligating wires. It is simplified in something I call PDE (patient diet education). PDE is highly recommended, particularly for patients with a high DMFT index. The cariogenic potential of foods should be explained, the significant effect of fermentable carbohydrates (sugar containing food as an example) on the number of Lactobacilli should be mentioned and the frequency of meals per day and its effect on the pH should be explained.

Patients should be taught the MFTT and should then be asked to demonstrate their ability to perform the technique. They should understand the importance of each step of the technique, brushing after meals while timing themselves when they brush (2 min brushing), rinsing with the toothpaste for 30 seconds and avoiding eating, drinking or further rinsing for 2 h after. The beauty of the MFTT that it can be applied to any regular toothpaste and have significant effect on caries incidence, it can be prescribed in any situation (low, medium or high risk to develop caries) and in any dental clinic (ortho, perio, endo, etc.).
Compared with other dental specialties, orthodontists have a great opportunity to place the emphasis on MFTT in their clinics, as they usually visit the clinic every 6-12 weeks. During active orthodontic treatment, orthodontist and the hygienist should work as a team, continuously reminding the patient of MFTT, encouraging proper diet, and evaluating caries risk on every appointment. Any signs of decalcification or poor oral health status should be documented (taking photos as an example) and potential risks re-explained to the patient and their guardians, in case the patient is a child. If repeated episodes of bad oral hygiene are occurring, wires can be removed and patients are asked to improve their oral hygiene. They should be followed up and evaluated for 3 months. If active caries developed at anytime during treatment, wires and brackets should be removed, preventive caries measures should be explained and applied, and later after 6 months of evaluation, treatment can be re-initiated.

It is not difficult to reduce orthodontic patients’ caries risk and prevent the incidence of new caries lesions, but it is only challenging to an orthodontist to establish a routine regime and spare few more minutes evaluating, educating, and advising his or her patients. Hopefully this chapter will aid orthodontists around the world to achieve these goals.

6. Conclusions

The use of the MFTT significantly reduces the incidence of new caries lesions in orthodontic patients.

Author details

Anas H. Al-Mulla

Address all correspondence to: a.almulla@mac.com

Department of Orthodontics, European University College, Dubai Health Care City, Dubai, United Arab Emirates

No part of this chapter may be reproduced or transmitted, in any form or by any means, without written permission. Dr. Anas Hasan Al-Mulla.

References

[1] Marcotte H, Lavoie MC: Oral microbial ecology and the role of salivary immunoglobulin A. Microb and Molec Bio Rev 1998:63:71-109.
[2] Boyar R M, Thylstrup A, Holmen L, Bowden GH: The microflora associated with the development of initial enamel decalcification below orthodontic bands in vivo in children living in fluorinated-water area. J Dent Res 1989:68:1734-1738.

[3] Zühlke A, Röder B, Widdecke H, Klein J: Synthesis and application of new microcarriers for animal cell culture. Part I: design of polystyrene based microcarriers. J Biom Sci Polymer Edition 1993:5:65-78.

[4] Langer R: Polymers for drug delivery and tissue engineering. Ann Biomed Eng 1995:23:101-111.

[5] West JL, Hubbell JA: Polymeric biomaterials with degradation sites for proteases involved in cell migration. Macromolecules 1999:32:241-244.

[6] Weitman RT, Eames WB: Plaque accumulation on composite surfaces after various finishing procedures. J Am Den Assoc 1975:91:101-106.

[7] Gwinnett AJ, Ceen RF: Plaque distribution on bonded brackets: a scanning microscope study. Am J Orthod 1979:84:667-677.

[8] Sukontapatipark W, El-Agroudi MA, Selliseth NJ, Thunold K, Selvig KA: Bacterial colonization associated with fixed orthodontic appliances. A scanning electron microscopy study. Eur J Orthod 2001:23:475-484.

[9] Zachrisson BU: Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. Am J of Orthod 1976:69:285-300.

[10] Gorelick L, Geiger AM, Gwinnett AJ: Incidence of white spot formation after bonding and banding. Am J Orthod Dentofacial Orthop 1982;81:93-98.

[11] O’Reilly MM, Featherstone JDB: Demineralization and remineralization around orthodontic appliances: an in vivo study. Am J Orthod 1987:92:33-40.

[12] Øgaard B, Rolla G, Arends J: Orthodontic appliances and enamel demineralization. Part 1: Lesion development. Am J Orthod Dentofacial Orthop 1988a;94:68-73.

[13] Øgaard B, Rolla G, Arends J, ten Cate JM: Orthodontic appliances and enamel demineralization. Part 2: Prevention and treatment of lesions. Am J Orthod Dentofacial Orthop 1988b; 94:123-128.

[14] Mitchell L: Decalcification during orthodontic treatment with fixed appliances: An overview. Br J Orthod 1992;19:199-205.

[15] Balenseifien J W, Madonia J V: Study of dental plaque in orthodontic patients. J Dent Res 1970:49:320-324.

[16] Diamandi-Kiopioti A, Gusberti F A, Lang N P: Clinical and microbiological effects of fixed orthodontic appliances. J Clin Periodontol 1987:14:326-333.

[17] Årtun J, Brobakken BO: Prevalence of carious white spots after orthodontic treatment with multibonded appliances. Eur J Orthod 1986;8:229-234.
[18] Øgaard B: Prevalence of white spot lesions in 19-year-olds: A study on untreated and orthodontically treated persons 5 years after treatment. Am J Orthod Dentofacial Orthop 1989;96:423-427.

[19] Chang HS, Walsh LJ, Freer TJ: Enamel demineralization during orthodontic treatment: Aetiology and prevention. Austr Dent J 1997;42:322-327.

[20] Mizrahi E: Surface distribution of enamel opacities following orthodontic treatment. Am J Orthod Dentofacial Orthop 1983;84:323-331.

[21] Axelsson P: An introduction to risk prediction and preventive dentistry. Carol Stream, Quintessence Publishing, 1999, pp 107-111.

[22] Scheie AA, Arneberg P, Krogstad O: Effect of orthodontic treatment on prevalence of Streptococcus mutans in plaque and saliva. Scand J Dent Res 1984;92:211-217.

[23] Jordan C, LeBlanc DJ: Influences of orthodontic appliances on oral populations of mutans streptococci. Oral Microbiol Immunol 2002;17:65-71.

[24] Wenderoth CJ, Weinstein M, Borislow AJ: Effectiveness of a fluoride-releasing sealant in reducing decalcification during orthodontic treatment. Am J Orthod Dentofacial Orthop 1999;116:629-634.

[25] Fornell AC, Sköld-Larsson K, Hallgren A, Bergstrand F, Twetman S: Effect of a hydrophobic tooth coating on gingival health, mutans streptococci, and enamel demineralization in adolescents with fixed orthodontic appliances. Acta Odontol Scand 2002;60:37-41.

[26] Marinho VC, Higgins JP, Sheiham A, Logan S: Fluoride toothpastes for preventing dental caries in children and adolescents. Cochrane Database Syst Rev 2003:CD002278.

[27] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, Lingström P, Mejàre I, Nordenram G, Norlund A, Petersson LG, Söder B: Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003;61:347-355.

[28] Twetman S: Caries prevention with fluoride toothpaste in children: An update. Eur Arch Paediatr Dent 2009;10:162-167.

[29] Marinho VC: Cochrane reviews of randomized trials of fluoride therapies for preventing dental caries. Eur Arch Paediatr Dent 2009;10:183-191.

[30] Bowen WH: The role of fluoride toothpastes in the prevention of dental caries. J R Soc Med 1995;88:505-507.

[31] Lynch RJ, Navada R, Walia R: Low-levels of fluoride in plaque and saliva and their effects on the demineralisation and remineralisation of enamel; role of fluoride toothpastes. Int Dent J 2004;54:304-309.
[32] Davies RM, Ellwood RP, Davies GM: The rational use of fluoride toothpaste. Int J Dent Hyg 2003;1:3-8.

[33] Creeth JE, Gallagher A, Sowinski J, Bowman J, Barrett K, Lowe S, Patel K, Bosma ML: The effect of brushing time and dentifrice on dental plaque removal in vivo. J Dent Hyg 2009;83:111-116.

[34] Zero DT, Creeth JE, Bosma ML, Butler A, Guibert RG, Karwal R, Lynch RJ, Martinez-Mier EA, Gonzalez-Cabezas C, Kelly SA: The effect of brushing time and dentifrice quantity on fluoride delivery in vivo and enamel surface microhardness in situ. Caries Res 2010;44:90-100.

[35] Travess H, Roberts-Harry D, Sandy J: Orthodontics. Part 6: Risks in orthodontic treatment. Br Dent J 2004;196:71-77.

[36] Bizhang M, Chun YH, Winterfeld MT, Altenburger MJ, Raab WH, Zimmer S: Effect of a 5000 ppm fluoride toothpaste and a 250 ppm fluoride mouth rinse on the demineralisation of dentin surfaces. BMC Res Notes 2009;2:147-151.

[37] Schirrmeister JF, Gebrande JP, Altenburger MJ, Monting JS, Hellwig E: Effect of dentifrice containing 5000 ppm fluoride on non-cavitated fissure carious lesions in vivo after 2 weeks. Am J Dent 2007;20:212-216.

[38] Baysan A, Lynch E, Ellwood R, Davies R, Petersson L, Borsboom P: Reversal of primary root caries using dentifrices containing 5,000 and 1,100 ppm fluoride. Caries Res 2001;35:41-46.

[39] Nordström A, Birkhed D: Fluoride retention in proximal plaque and saliva using two NaF dentifrices containing 5,000 and 1,450 ppm F with and without water rinsing. Caries Res 2009;43:64-69.

[40] Chestnutt IG, Schafer F, Jacobson AP, Stephen KW: The influence of toothbrushing frequency and post-brushing rinsing on caries experience in a caries clinical trial. Community Dent Oral Epidemiol 1998;26:406-411.

[41] Duckworth RM, Knoop DT, Stephen KW: Effect of mouthrinsing after toothbrushing with a fluoride dentifrice on human salivary fluoride levels. Caries Res 1991;25:287-291.

[42] Attin T, Hellwig E: Salivary fluoride content after toothbrushing with a sodium fluoride and an amine fluoride dentifrice followed by different mouthrinsing procedures. J Clin Dent 1996;7:6-8.

[43] Sjögren K, Birkhed D, Rangmar B: Effect of a modified toothpaste technique on approximal caries in preschool children. Caries Res 1995;29:435-441.

[44] Sjögren K, Birkhed D: Effect of various post-brushing activities on salivary fluoride concentration after toothbrushing with a sodium fluoride dentifrice. Caries Res 1994;28:127-131.
[45] Silness J, Löe H: Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. Acta Odontol Scand 1964;22:121-135.

[46] World Health Organization. Oral health surveys: Basic methods, 4th ed. Geneva: World Health Organization, 1997.

[47] Mejàre I, Källestål C, Stenlund H, Johansson H: Caries development from 11 to 22 years of age: A prospective radiographic study. Prevalence and distribution. Caries Res 1998;32:10-16.

[48] Attin T, Hornecker E: Tooth brushing and oral health: How frequently and when should tooth brushing be performed? Oral Health Prev Dent 2005;3:135-140.

[49] Duckworth RM, Jones Y, Nicholson J, Jacobson AP, Chestnutt IG: Studies on plaque fluoride after use of F-containing dentifrices. Adv Dent Res 1994;8:202-207.

[50] Chesters RK, Huntington E, Burchell CK, Stephen KW: Effect of oral care habits on caries in adolescents. Caries Res 1992;26:299-304.
