Review Article

JRAD Journal of Research and Advancement in Dentistry

J Res Adv Dent 2019;9:2s:20-28.

The Role of Periodontist in Orthodontic Therapy

Rosaiah Kanaparthy[1*] Aruna Kanaparthy[2]

[1]Professor and Head, Department of Periodontics, College of Dental Sciences & Hospital, Amargadh, Bhavnagar, Gujarat, India.
[2]Professor, Department of Conservative Dentistry and Endodontics, College of Dental Sciences & Hospital, Amargadh, Bhavnagar, Gujarat, India.

ABSTRACT

Background: Orthodontic tooth movement is brought about by prolonged application of force on the attachment apparatus which can be potentially damaging to the periodontium. Successful orthodontic treatment requires simultaneous care and management of the periodontal health. Thus the role of a periodontist in orthodontic treatment becomes invaluable. This article is an overview on the response of the periodontal ligament to orthodontic forces, some of the problems associated with it, periodontal maintenance of the orthodontic patient and the minor surgeries required to correct the defects affecting the soft and hard tissues of the periodontium.

Keywords: Orthodontic forces, periodontium, attachment apparatus, tooth movement, Supracrestal fiberotomy.

INTRODUCTION

The importance of teamwork in monitoring the periodontal health of patients throughout the course of orthodontic therapy cannot be overemphasized. It is in connection with adult orthodontics that periodontal factors are becoming more important. For best results orthodontic treatment in adults is now being performed by teams of periodontist, restorative dentist and orthodontist. As said by Profit "Orthodontic treatment is based on the principles that if prolonged pressure is applied to a tooth, tooth movement will occur as the bone around the tooth remodels. Because the bony response is mediated by the periodontal ligament, tooth movement is primarily a periodontal ligament phenomenon. (1)

Response of periodontal ligament to orthodontic forces:

Orthodontic forces are known to produce mechanical damage and inflammatory reactions in the periodontium and dental pulp, as well as inflammatory mediators, e.g. prostaglandins(PG), interleukin (IL)-1, IL-6, tumor necrosis factor α, and receptor activator of nuclear factor B ligand, in the periodontal ligament (PDL) and dental pulp. Cyclo-oxygenase-2 is induced in PDL cells by cyclic mechanical stimulation and is responsible for the augmentation of PGE2 production. (2) Experimental studies have shown that when PGE1 was administered locally or systemically to rats, accelerated bone resorption and tooth movement were observed after the application of orthodontic forces. Therefore, PGs play an important role in orthodontic tooth movement. (3)

Several authors have demonstrated that compressive force upregulates RANKL expression and induction of COX-2 in human PDL cells in vitro. PDL cells under mechanical stress may induce osteoclastogenesis through up-regulation of RANKL expression via PGE2 synthesis during orthodontic tooth movement. (4) Shiotani et al. also showed the presence of RANKL in periodontal tissues during experimental tooth movement of rat molars. Therefore it is suggested that RANKL is regulated by inflammatory cytokines in the PDL in response to mechanical stress. (5)

Various studies have reported significant recruitment of mononucleated cells, macrophages, dendritic cells, and MHC class IIIa-expressing cells in the pressure zone incident to orthodontic tooth

Received: Nov. 11, 2018: **Accepted**: Jan. 25, 2019
*Correspondence Dr. Rosaiah Kanaparthy.
Department of Periodontics, College of Dental Sciences & Hospital, Amargadh, Bhavnagar, Gujarat, India.
Email: drrosaiah@gmail.com

pISSN 2278-0076
eISSN 2321-9270

movement.(6, 7) In the tension zone, however,minimal changes in the number and distribution of immune cells have been reported. (8) .Under stress from the orthodontic treatment,there would be changes to the blood flow.(9) Neuropeptides are released from the periodontal nerve endings, which causes neurogenic inflammation in the compressed periodontal ligament.(10) Furthermore, various immunoregulatory molecules, such as interleukin-1 a, interleukin-6, and tumour necrosis factor-a, are released during inflammation and participate in the remodelling of the periodontium. (11)

Strong forces lead to crushing of periodontal ligament on pressure side leading to degeneration of periodontal ligament which produces hyalinization delaying tooth movement. A moderate force causes strangulation of periodontal ligament leading to delay in bone resorption. Light forces which cause tooth movement continuously with simultaneous bone resorption and formation actually are recommended.(12) The periodontal complications associated with orthodontic therapy mainly include gingivitis, periodontitis, gingival recession or hypertrophy, alveolar bone loss, dehiscences, fenestrations, interdental fold, and dark triangles. (13,14,15, 16)

Periodontal problems associated with orthodontic therapy

1. Loss of periodontal attachment and bone relative to orthodontic therapy

- In the absence of periodontal disease and in the pressure of excellent hygiene, proper orthodontic treatment causes no significant long term effects on periodontal attachment and bone levels. Conversely in patients with active periodontitis (that is, plaque infected deep pockets evidenced by bleeding on probing), orthodontic tooth movement may accelerate the disease process, even when good oral hygiene is practiced Orthodontic bodily movement into plaque induced infra bony defects can be successfully performed, provided that the periodontal lesion is eliminated before tooth movement is begun, and excellent oral hygiene is maintained.
- Tooth movement into areas of edentulous spaces with slow, light orthodontic forces is often possible, depending on the width ratio of tooth to bone, but alveolar bone loss and post treatment space opening can be common sequelae, even under optimal conditions.
- Review of literature suggests that new attachment gain can be predictably achieved with intrusive tooth movement if the intrusion is done in conjunction with a barrier and new attachment procedure.
- Extrusive tooth movement in areas of one and two wall bony pockets leads to a coronal positioning of intact connective tissue attachment and a shallowing of bony defect. These changes in attachment and bone levels are a key factor in up- righting tipped molars. If there is furcation involvement in the tooth to be up righted, orthodontic tooth movement may exacerbate the periodontal problem.

2. Gingival recession due to orthodontic therapy.

Orthodontic tooth movement per se does not cause gingival recession.

- However,during any orthodontic treatment, a slight to moderate degree of gingival overgrowth might be seen. (17,18,19) This can be explained by the accumulation of plaque on the teeth surfaces and the increase of periodontal pathogens.

- In areas of thin labial tissue, labial orthodontic tooth movement can result in bone dehiscence, creating on environment in which plaque / or toothbrush trauma may cause sudden recession.

- Most gingival recessions which occur during an orthodontic treatment had been shown to occur in the regions of the anterior upper and lower teeth.(20,21,22,23)

- Steiner and his colleagues.(24) mentioned that the gingival recessions noted after an orthodontic treatment tend to occur in the regions where the keratinized gingiva and the underlying bone tissues are thin.

- Many researchers and clinicians agree that this thin labial tissue should be augmented before labial orthodontic tooth movement is begun. It

is interesting to note that in areas of a labially positioned tooth with dehiscence, bone may reform and gingival thickness may increase when the tooth moves lingual.

- By increasing the thickness of the covering tissue, the risk of gingival recession during or following orthodontic therapy can be reduced. Numerous grafting techniques have been advocated including free gingival grafts and inter-positional pedicle grafts. The free gingival graft (FGG) provides increased thickness of tissue and increased width of keratinised tissue. However the aesthetic result with the FGG technique is poor. Root coverage with the FGG is unpredictable although this can be addressed via a second surgical procedure involving coronal repositioning of the augmented tissue. Pedicle grafts (laterally repositioned flap LRF and coronally repositioned flap CRF) are dependant on the presence of an adequate width of keratinized tissue either adjacent or apical to the defect. Both the LRF and CRF provide acceptable results in terms of tissue augmentation, aesthetics and root coverage. There is a risk however of induced gingival recession at the donor site with the LRF procedure.(25, 26) it rapidly improves within 48 hours of the appliance being removed.(27,28)

- In regard to relationship between rapid maxillary expansion procedures and gingival recession Graber and Vanarsdall stated that if the maxillary expansion is performed after the mid palatine suture begin to fuse (after approximately 14-16years of age), there is a greater risk of recession of the buccal gingival tissue of the maxillary premolars and molars later in life.(29)

- Periodontal evaluation of a potential adult orthodontic patient must include not only the response to periodontal probing but also level and condition of attached gingiva.

- Wennstrom et al (1987) found no relationship between initial width of keratinized gingiva and tendency for development of gingival recession during orthodontic tooth movement in monkeys. (30) .Instead it is the buccolingual thickness which maybe the determining factor for development of gingival recession and attachment loss at sites with gingivitis during orthodontic treatment.

Jon Artun (2001) conducted a study to analyze whether pronounced orthodontic advancement of mandibular ncisors during class II correction in mixed dentition resulted in gingival recession. They used 67 patients treated with reverse headgear. They concluded that pronounced advancement of mandibular incisors might be performed in adolescents with dentoalveolar retrusion without increasing the risk of recession.(31)

Boyd et al found no differences in post orthodontic development of mucogingival problems between groups of patients who had under gone tooth extraction and those who had not. For adult orthodontic patients it is much better to prevent gingival recession than to try and correct it later. (32) The protective effect of a FGG may be more in increasing the thickness at keratinized tissue than increasing the width of the same. It should be particularly considered in a patient who requires moderate arch expansion to align incisors and those who will have surgical mandibular advancement or genioplasty.

2. Inter-dental recession

From an esthetic view point it would appear that inter dental recession, manifesting as pronounced empty spares (dark triangles) between teeth would be equally or perhaps more important. It would be visible both in rest portion of the lips and upon smiling. Since quality of life (esthetic and lack of pain) has become increasingly more important in recent years for the selection of periodontal therapies disfigurement of the gingival papillae during orthodontic treatment should be avoided. Norland and Tarnow (1998)-classification of papillary height adjacent to natural teeth.(33)

Normal:-The interdental papilla occupies the entire embrasure space apical to the interdental contact point / area.

Classification I:- The lip of the interdental papilla is located between the interdental contact point and the level of the CEJ on the proximal surface of the tooth.

Classification II:- The tip of the interdental papilla is located between the interdental contact point and the level of the CEJ on the proximal surface of the tooth, but coronal to the level of the CEJ mid-buccally.

Classification III:- The tip of the interdental papilla is located at or apical to the level of the CEJ mid-buccally.

Causes

1. Advancement PDL disease, by the tissue destruction or due to to pocket elimination by surgery,
2. Triangular tooth shape due to abnormal interproximal wear of teeth in crowded positions before orthodontic treatment.
3. Divergent roots due to improper bracket placement

Various theories have been advanced to account for open gingival embrasures following orthodontic treatment. One proposal implicates shortening of the interdental papillae due to stretching of the interdental gingival fibers as crowded incisors are aligned (Artun J. 1986). (34) Tanaka has given a detailed explanation of open gingival embrasures and said that loss of gingival embrasures increases with age. (35)

Clinical options for treatment:

1. Open gingival embrasures could be closed by torqueing the central incisor roots mesially as the proximal contacts are maintained.
2. Mesiodistal enamel reduction or Proximal stripping or Slicing in which 0.5 - 0.75 mm of enamel is removed and contact area lengthened.
3. Mucogingival surgery using coronally repositioned flaps and guided tissue regeneration techniques. (Pini Prato et al 1995). (36)
4. Provisions of a gingival prosthesis.

Gingival hyperplasia.

- ❖ In the presence of excellent oral hygiene no significantly hyperplasia should develop as a result of orthodontic tooth movement in adolescents or adults.
- ❖ However fixed orthodontic appliances in the presence of consistently poor oral hygiene can lead to moderate to severe hyperplasia, especially in the lower incisor region. Severe cases of gingival hyperplasia may leads to bone loss. Gingival hyperplasia is often reversible especially after removal of orthodontic appliances.
- ❖ However, Graber suggested that some patients may benefit from removal of excessive tissue from the crown of the teeth, which may lead to the stability of the orthodontic correction.

Management

- Gingivectomy
- Intercession + incisal restoration or porcelain veneer.
- Extrusion + fiberotomy + porcelain crown
- Surgical crown lengthening by flap procedure at osteotomy / osteoplasty of bone (Bragger et al 1992). (37)
- Use of lasers has proved to be beneficial due to superior hemostasis less postoperative discomfort, pain or oedema, better tolerance from the patient, less complicated procedures, suturing and dressing avoided and decreased chances of postoperative bacteraemia.

Periodontal maintenance of orthodontic patients:

It has been observed that as a result of the orthodontic treatment a shift in the composition and type of bacteria can be expected. (38) Orthodontic treatment is known to affect the equilibrium of oral microflora by increasing bacteria retention. In a study done by Ristic et al. (39) An increase in the value of periodontal indices and growth of periodontopathogenic bacteria was observed in adolescent patients undergoing fixed orthodontic treatment. Before orthodontic treatment is begun, each member of the dental team

must be sure that the patient is practicing excellent oral hygiene and is free of active periodontal disease.

Oral hygiene instructions must be given along with demonstration of modified Bass brushing technique. Chlorhexidine mouthwashes, as an adjunct to tooth brushing, have been found effective in the control of gingival inflammation, although prolonged use may cause problems with staining as Chlorhexidine rinses can potentially stain the margins of composite restorations that cannot be easily removed. More recently, pre-brushing rinses have been introduced, though these show no differences in effect on plaque accumulation or gingival health. Chlorhexidine is also useful for patients after orthognathic surgery, especially when intermaxillary fixation is to be used.

On the other hand, Fluoride mouth rinses significantly reduce the extent of enamel decalcification and gingival inflammation during orthodontic treatment. A number of studies evaluated the effect of mechanical aids, as compared with manual tooth brushing, on oral hygiene in orthodontic patients and it has been shown that the use of electric toothbrushes brought a significant improvement in oral hygiene. (40,41,42,43,44,45)

Suggested protocol before orthodontic therapy:

Ideally the periodontist at the time of referral should provide the orthodontist with the following.

1. A copy of patients recent periodontal charting.
2. Recent anterior IOPA's and bite-wing radiograph
3. A periodontal clearance statement. (Serves as valuable document for medico legal patient as well as acts as reinforcement to the patient).
4 If surgery is contemplated a period of 4-6 months for healing and observation should be allowed before ortho treatment.

Suggestive protocol during and after orthodontic therapy:

- Once orthodontic therapy begins, the periodontist, restorative dentist and orthodontist should re-evaluate all patients and ensure that there is excellent plaque control and no inflammation especially in those with a history of periodontal disease for any sign of recurrence.
- It has been observed that some patients tend to adopt a more aggressive brushing habit during orthodontic therapy. They should be advised of toothbrush trauma resulting in gingival recession due to over zealous brushing.

Minor surgeries associated with orthodontic therapy.

Minor periodontal surgery maybe used to improve or stabilize results achieved by orthodontic treatment for malocclusion. Edwards 1977 first introduced frenotomy and few other clinical procedures to prevent relapse of derotated teeth and extraction spaces. (46)

Fiberotomy: Methods to reduce the occurrence of rotational relapse may include

- Complete correction or over correction of rotated teeth.
- Stable long-term retention with bonded lingual retainers.
- Use of fiberotomy Rationale for fiberotomy:

A major cause of rebound after orthodontic therapy is due to stretched gingival fibers. As teeth are moved to a new position, these fibers tend to stretch and remodel very slowly. If the pull of these elastic fibers could be eliminated, a major cause of relapse of previously irregular and rotated teeth could be eliminated. In fact, if the supracrestal fibers are sectioned and allowed to heal while the teeth are held in the proper position, relapse caused by gingival elasticity will be greatly reduced.

Circumferential Supracrestal fiberotomy,(CSF) technique: First developed by Edwards.Technique-After infiltration with a local anesthetic the procedure consists of inserting the sharp point of a time blade (No.11) into sulcular area and severing the epithelial attachment surrounding the involved tooth. No periodontal pack is necessary, and there is only minor discomfort after the procedure.

Papilla Split procedure by Profitt

An alternative method is to make an incision in the centre of each papilla, sparing the margin but separating the papilla from just below the margin to 1-2 mm below the height of bone buccally and lingually.

Advantages

It is easier to perform with the orthodontic appliance still in place.

Edwards and Rinaldi. (47,48) have demonstrated that sectioning the gingival fibers is an effective method to control rotational relapse but does not control the tendency for crowded incisors to again become irregular. When performed in healthy tissues after ortho treatment negligible loss of attachment (0.1 – 0.3 mm). In a 15-years follow up study on CSF found it to be effective in severe cases of rotated teeth and more successful in maxillary anterior than mandibular ones.

Frenotomy

Bergstrom et al (1973). (49) stated that the probability for diastema in the long run is the same whether or not frenectomy is preformed. Earlier frenectomy extending into palatal surface was advocated. But this leads to loss of inter dental papilla between upper central incisors. So, the frenotomy by Edwards (1977). (46) was introduced, which represents a more gentle operation, with only partial removal of frenum and with the purpose of relocating the attachment in an apical direction. Procedure consists of V shaped incision along the frenal borders, removal of frenum tip, before cutting fibers and periosteum with some vertical and horizontal incisions.

Removal of gingival invagination (Clefts):

- Incomplete adaptation of supporting structures during orthodontic closure of extraction spaces in adults may result in infolding or invagination of the gingiva. The clinical appearance of such invaginations may range from a minor surface crease to deep clefts that extend across the interdental papilla.

- There is general trend toward some resolution of these defects with time, but many invaginations persist for 5 years, or more after completion of orthodontic therapy Edward (1971). (50) suggested that simple removal of only the excess gingiva in the buccal and lingual area of approximated teeth would be sufficient to enhance re-establishment a more normal connective tissue.

Gingivectomy

If gingival margin discrepancy is present, but the patients lip does not move upward to expose the discrepancy upon smiling, it does not require correction.However if the defect is visible,corrective gingivectomy can be carried out.Laser gingivectomy is frequently used to correct gingival discrepancies. (51)

CONCLUSION

There exists a close relationship between orthodontic treatment and periodontal health. The success or failure of orthodontic treatment in adults depends on the health of the periodontium. Orthodontic treatment can be carried out with predictable success in adults by ensuring that physiological forces are used. Studies show that there is decreased motivation among patients as the duration of treatment increases resulting in increased incidence of gingival enlargements. (52) The periodontist must monitor the case to make sure that the patient is maintaining excellent oral hygiene thereby preventing gingival inflammation and periodontal disease.

CONFLICT OF INTEREST

No potential academic conflict of interest relevant to the above article was reported.

REFERENCES

1. ProfittWR, Fields HW, Contemporary orthodontics,2nd ed, St. Loius, Missourie: Mosby publications 1997.

2. Yamaguchi M, Kasai K. Inflammation in periodontal tissues in response to mechanical force. Arch Immunol Ther Exp 2005; 53: 388–398.

3. Yamasaki K., Shibata Y., Imai S., Tani Y., Shibasaki Y. and Fukuhara T. Clinical application of prostaglandin E1 (PGE1) upon

orthodontic tooth movement. Am. J. Orthod1984, 85, 508–518

4. Kanzaki H., Chiba M., Shimizu Y. and Mitani H. Periodontal ligament cells under mechanical stress induce osteoclastogenesis by receptor activator of nuclear factor kappaB ligand up-regulation via prostaglandin E2 synthesis. J. Bone Miner. Res.2002, 17, 210–220.

5. Shiotani A., Shibasaki Y. and Sasaki T. Localization of receptor activator of NFkappaB ligand, RANKL, in periodontal tissues during experimental movement of rat molars. J. Electron Microsc.2001,50, 365–369.

6. Vandevska-Radunovic V,Kvinnsland I H,S. Kvinnsland H S,and Jonsson R, "Immunocompetent cells in rat periodontal ligament and their recruitment incident to experimental orthodontic tooth Movement European Journal of Oral Sciences,1997,105, (1),36–44.

7. Brudvikand P.Rygh,"Non-clastcellsstartorthodonticroot resorption in the periphery of hyalinized zones," European Journal of Orthodontics, 1993,15,(6),467–480.

8. Nakamura K, Sahara N, and Deguchi T, "Temporal changes in the distribution and number of macrophage-lineage cells in the periodontal membrane of the rat molar in response to experimental tooth movement "Archives of Oral Biology ,2001,46,(7)593–607.

9. Santamaria Jr M., Milagres D, Sasso Stuani A, Sasso Stuani M.B, and de Oliveira Ruellas A.C, "Initial changes in pulpal microvasculature during Orthodontic tooth movement: astereological study, European Journal of Orthodontics, 2006,28,(3),.217–220.

10. Vandevska-Radunovic V, "Neural modulation of inflammatory reactions in dental tissues incident to orthodontic tooth movement. A review of the literature," European Journal of Orthodontics,1999, vol.21,(3),231–247.

11. Bletsa A, Berggreen E, and Brudvik P, "Interleukin-1α and tumor necrosis factorα expression during the early phases of orthodontic tooth movement in rats,"European Journal of Oral Sciences,2006,vol.114,(5),423–429.

12. Kein Robert G, Esthetics in clinical orthodontic periodontic interactions. Periodontol 2000; 27: 2001

13. Preoteasa C.T, Ionescu E, Preoteasa E, Risks and Complications Associated with Orthodontic Treatment,2012.

14. Talic N.F,"Adverse effects of orthodontic treatment:aclinical perspective, SaudiDentalJournal,2011,23,(2).55–59.

15. Bragger U, and Lang N.P, "The significance of bone in periodontal disease,"Seminars in Orthodontics,1996,vol.2.(1) 31–38.

16. Romero M,"Surgical solutions to periodontal complications of orthodontic therapy,"Journal of Clinical Pediatric Dentistry,2000,vol. 24,(3).159–163,

17. Trossello VK, Gianelly AA. Orthodontic treatment and periodontal status. J Periodontol. 1979 Dec;50(12):665-671.

18. Zachrisson BU, Alnaes L. Periodontal condition in orthodontically treated and untreated individuals. II. Alveolar bone loss: radiographic findings. Angle Orthod. 1974 Jan;44(1):48-55.

19. Zhao H, Xie Y, Meng H. [Effect of fixed appliance on periodontal status of patients with malocclusion]. Zhonghua Kou Qiang Yi Xue Za Zhi. 2000 Jul;35(4):286-288

20. Hall WB. The current status of mucogingival problems and their therapy. J Periodontol. 1981 Sep;52(9):569-575.

21. Pearson LE. Gingival height of lower central incisors, orthodontically treated and untreated. Angle Orthod. 1968 Oct;38(4):337-339.

22. Polson AM, Reed BE. Long-term effect of orthodontic treatment on crestal alveolar bone levels. J Periodontol. 1984 Jan;55(1):28-34.

23. Sadowsky C, BeGole EA. Long-term effects of orthodontic treatment on periodontal health. Am J Orthod. 1981 Aug;80(2):156-172.

24. Steiner GG, Pearson JK, Ainamo J. Changes of the marginal periodontium as a result of labial tooth movement in monkeys. J Periodontol. 1981 Jun;52(6):314-320

25. Monfeldt I and Zachrison BU: Adjustment of clinical crown height by gingivectomy Following orthodontic space. Angle Orthodontics.1977; 47:408-413.

26. Carranza F and Newmann MG: Clinical Periodontology 8th Ed.pp.323 Contemporary Periodontics.2nd ed.William R Proffit .593-596

27. Baer PN, Coccaro PJ. Gingival enlargement coincident with orthodontic therapy. J Periodontol 1964;35:436-439.

28. Alexander SA. Effects of orthodontic attachments on the gingival health of permanent second molars. Am J Orthod Dentofacial Orthop 1991, 100:337-40.

29. Graber TM. Orthodontics principles and practice. 3rd ed. Philadelphia: WB Saunders; 1972,

30. Wennstrom,Adult orthodontics by Birte Melsen,.p87,Wiley Blackwell

31. Jon Årtun, at.al, Periodontal status of mandibular incisors after pronounced orthodontic advancement during adolescence: A follow-up evaluation, American Journal of Orthodontics and Dentofacial Orthopedics 2001Volume 119,(1), 2–10

32. Boyd RL. Mucogingival considerations and their relationship to orthodontics. J Periodontol 1978;49:67-76

33. Nordland Wp, Tarnow Dpa, Classification System For Loss Of Papillary Height. J Periodontol. 1998 Oct;69(10):1124-1126.

34. Artun J,Osterberg SK,Kokich VG.Long term effect of thin interdental alveolar bone on periodontal health after orthodontic treatment.J Periodontal, 1986.57.341-346

35. Tanaka OM,Furquim BDA,Pascotto RC,Ribeiro GLU,Boscio JA,Maruo H.The dilemma of the open gingival embrasures between maxillary central incisors.J Contemp Dent Pract 2008 ;(9)6:92-98

36. Pini-Prato G, Clauser C, Magnani C, Cortellini P. Resorbable membrane in the treatment of human buccal recession: A nine-case report. Int J Periodont Restor Dent. 1995;15:258–267

37. Bragger, U., Lauchenauer, D. & Lang, N. P. Surgical crown lengthening of the clinical crown. Journal of Clinical Periodontology 1992, 19, 58–63.

38. Petti S, Barbato E, and Simonetti D'Arca A, "Effect of orthodontic therapy with fixed and removable appliances on oral microbiota: A six-month longitudinal study," New Microbiologica,1997.vol.20,(.1),.55–62.

39. Ristic M, VlahovicSvabic M, Sasic M,and .Zelic O,"Clinical and microbiological effects of fixed orthodontic appliances on periodontal tissues in adolescents,"Orthodontics & Craniofacial Research,2007,vol.10,(.4).187–195,

40. Wilcoxon DB, Ackerman RJ, Jr., Killoy WJ, Love JW, Sakumura JS, Tira DE. The effectiveness of a counterrotational-action power toothbrush on plaque control in orthodontic patients. Am J Orthod Dentofacial Orthop. 1991 Jan;99(1):7-14.

41. Brightman LJ, Terezhalmy GT, Greenwell H, Jacobs M, Enlow DH. The effects of a 0.12% chlorhexidine gluconate mouthrinse on orthodontic patients aged 11 through 17 with established gingivitis. Am J Orthod Dentofacial Orthop. 1991 Oct;100(4):324-329.

42. Pontier JP, Pine C, Jackson DL, DiDonato AK, Close J, Moore PA. Efficacy of a prebrushing rinse for orthodontic patients. Clin Prev Dent. 1990 Aug-Sep;12(3):12-17.

43. Boyd RL. Two-year longitudinal study of a peroxide-fluoride rinse on decalcification in adolescent orthodontic patients. J Clin Dent. 1992;3(3):83-87.

44. Boyd RL, Chun YS. Eighteen-month evaluation of the effects of a 0.4% stannous fluoride gel on gingivitis in orthodontic patients. Am J Orthod Dentofacial Orthop. 1994 Jan;105(1):35-41.

45. Denes J, Gabris K. Results of a 3-year oral hygiene programme, including amine fluoride products, in patients treated with fixed orthodontic appliances. Eur J Orthod. 1991 Apr;13(2):129-133.

46. Edwards JG: The diastema, the frenum, frenotomy. A clinical study. Am J Orthod 1977; 71:489-508

47. Rinaldi S A: Changes in free gingival level and sulcus depth of the human periodontium following circumferential supracrestal fiberotomy. Am.J.Orthod 1979;75:46-53.

48. Edwards JG: A Long term prospective evaluation of the circumferential Supracrestal fiberotomy in alleviating orthodontic relapse. Am J Orthod Dentofacial Orthop 1988; 93:380-387.

49. Bergstrom K, Jensen R, Martensson B. The effect of superior labial frenectomy in cases with midline diastema. Am J Orthod, 1973; 63: 633–638.

50. Edwards JG, The prevention of relapse in extraction cases. Am J Orthod. 1971,60:128–144

51. Prabhu M , Ramesh A ,Thomas Treatment of orthodontically induced gingival hyperplasia by diode laser. Nitte University Journal of Health Science 2015, 5,66-68.

52. Pinto et al,Gingival enlargement in orthodontic patients: Effect of treatment duration.Am J Orthod Dentofacial Orthop 2017;152:477-82.