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Case Report

Laser irradiation inhibition of open gingival embrasure space after orthodontic treatment

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The purpose of this study was to investigate the inhibitory effect of low-energy laser irradiation on an incidence of open gingival embrasure space after orthodontic treatment. The patient was a 20-year, 7-month-old Japanese female with an Angle Class I malocclusion and crowding in the mandible. Treatment consisted of extraction of maxillary and mandibular first premolars and use of the Edgewise technique.
A Ga-Al-As diode laser was used to irradiate an area of 0.5 cm^2 at the labial and lingual gingival papilla between the canines. The time of exposure was 6 minutes for 3 days, carried out between the relevelling and *en masse* stages of movement. The total energy corresponding to 6 minutes of exposure varied from 1.90 J/cm^2.
There was no further evidence of open gingival embrasure space, except at the mandibular central incisor. Further: an improvement in the gingival inflammation caused by a periodontal disease was observed, and periodontal pocket depth was maintained. These results suggest that low-energy laser irradiation may inhibit the incidence of open gingival embrasure space after orthodontic treatment.
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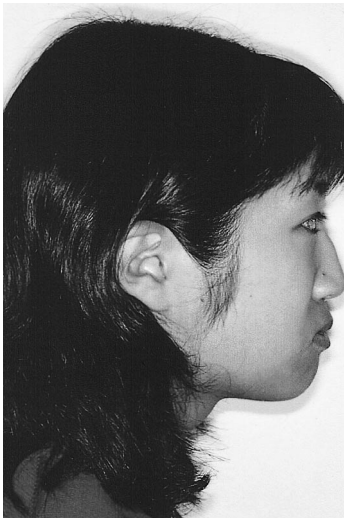
Introduction

The establishment of stability and a good functional relationship of the dentition are high priorities in the finishing stages of orthodontic treatment. Of equal importance are aesthetic demands. One such aesthetic concern is the open gingival embrasure space (black triangle) that develops when the maxillary central and the mandibular central and lateral incisors that were previously crowded are orthodontically aligned. The space looks dark and is usually considered unacceptable.

Burke *et al.* reported that 54 of 129 cases (41.9%) had a black triangular gingival space between the two maxillary central incisors after orthodontic treatment.[1] The cause of the incidence is stretching of the interdental gingival fibres: as the incisor root rotates, separates, and moves distally into alignment, the gingival fibres become tense and stretch as they remain at their physiological level of attachment to the teeth and the alveolar crest. As a result, the papilla is shortened, and the triangular space opens and an incisal portion of the embrasure is unoccupied and looks dark (Figure 1b).

Among the many physiological effects of low-energy laser irradiation, anti-inflammatory effects have been reported. Honmura *et al.* reported the therapeutic effect of Ga-Al-As laser irradiation on experimental carrageenan-induced inflammation.[2,3] Histological evaluations of the effect of laser irradiation on rheumatoid arthritis, a disease associated with bone resorption, have been made.[4] Kana *et al.* in an *in vivo* experiment reported the enhancement of wound healing by laser irradiation.[5] A low-energy laser has been used in the medical treatment of oral soft tissue and positive results in the healing of such diseases as recurrent aphthous stomatitis,[6] acute herpetic stomatitis and erythema multiforme[7] have been reported. Further, Kert



Figures 2a to d. Extra-oral photographs (pretreatment).

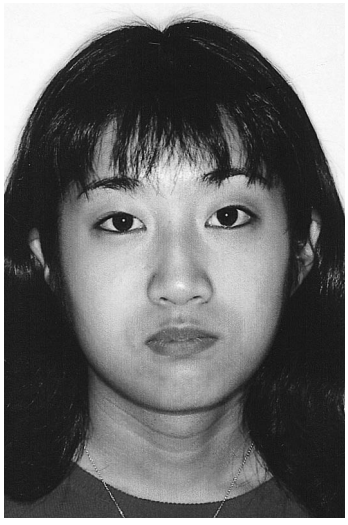


Figure 2b.

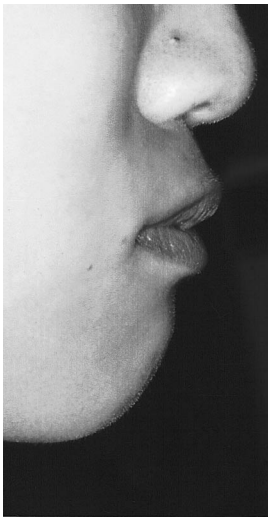
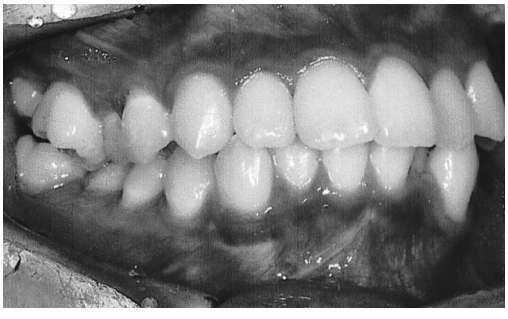


Figure 2c.



Figures 3a to e. Intra-oral photographs (pretreatment).

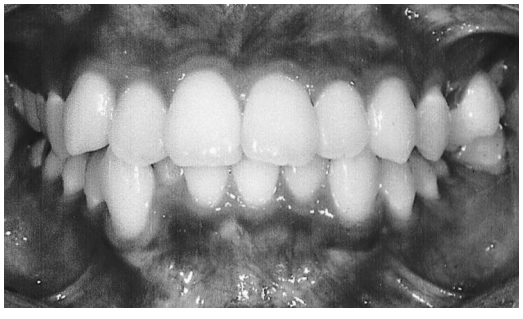


Figure 3b.

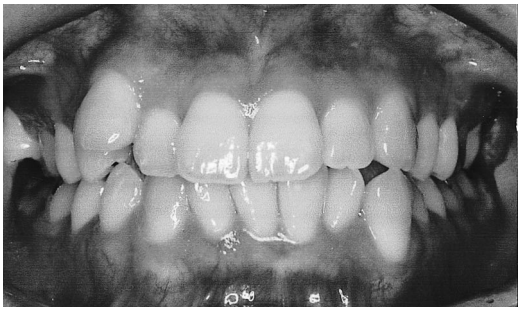


Figure 1a. Pretreatment.

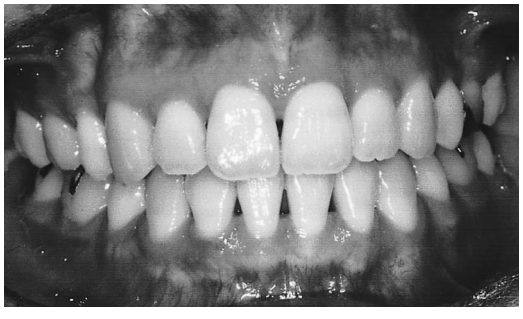


Figure 1b. The incidence of open gingival embrasure space (post-treatment).

and Rose reported that low-energy laser irradiation could rapidly improve the healing of gingival inflammation caused by periodontal diseases.[8]

As little is known about the anti-inflammatory effect of low-energy laser irradiation treatment following orthodontic treatment, this study seeks to further the investigation.

Diagnosis

The patient, a normally developing 20-year, 7-month-old Japanese female, had a Class I malocclusion with 7.0 mm of overjet and 4.0 mm of overbite. Her face was symmetrical and her profile convex (Figures 2a to d). The arch length discrepancies were 5.0 mm in both arches (Figure 3).

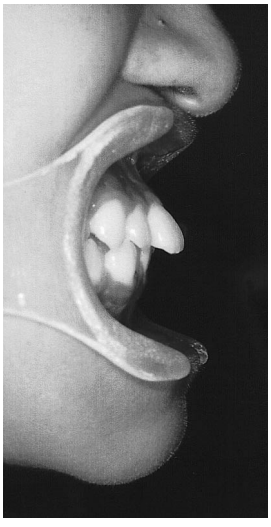


Figure 2d.

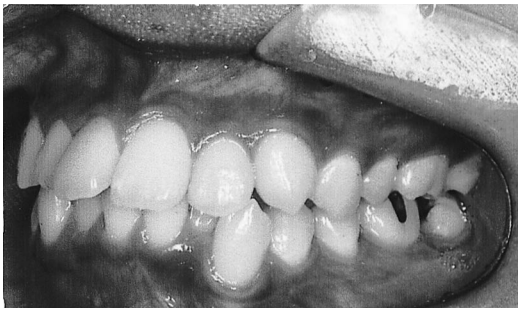


Figure 3c.

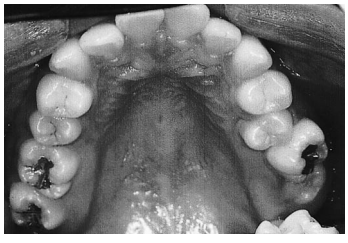


Figure 3d.

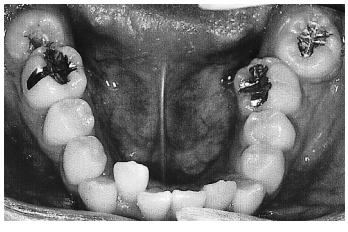


Figure 3e.

Periodontal diagnosis

Oral hygiene was good, but slight inflammation of the gingival papillae was found surrounding the maxillary and mandibular incisors. Probing pocket depths were 2 to 3 mm. The Papillary-Marginal-Attachment (PMA) Index, Gingival Index (GI) and the width of the attached gingiva are shown in Table I.

Treatment objectives

1. Correct to Class I molar and canine relationship.

2. Correct mandibular crowding and gain mandibular and maxillary dental symmetry.

3. Establish an ideal overjet and overbite with a cuspid-protected occlusion.

4. Establish good facial balance and an upper incisor display of approximately 3 mm. Reduce the protrusion of the lips and the strain of the mentalis muscle closure.

5. Following orthodontic treatment, inhibit the incidence of open gingival embrasure by the use of laser irradiation treatment.

Treatment plan

The treatment plan was to extract the maxillary and mandibular first premolars and treat using the Edgewise technique.

Treatment progress

After extraction of the first premolars, a maxillary Nance holding arch was placed. Bands and brackets with 0.018" x 0.025" slots were used. Initial levelling was accomplished with the use of nickel titanium archwires over 4 months. The arches were levelled using a series of round wires. In the maxillary and mandibular arches, the canines were retracted with elastometric chains on a stopped 0.018" round wire, and the incisors were retracted with a 0.017" x 0.025" closing-loop archwire.

Table I. Periodontal diagnosis (pretreatment).

Maxillary								
PMA Index				P	P	P		
Probing pocket depth (mm).	Facial	323	333	323	323	323	333	
	Lingual	333	323	333	333	323	323	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		2	2	2	2	2	3	
	R	3	2	1	1	2	3	L
Mandibular								
PMA Index			P	P	P	P	P	
Probing pocket depth (mm).	Facial	323	323	233	333	323	333	
	Lingual	333	322	323	323	223	333	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		2	2	2	2	2	2	
	R	3	2	1	1	2	3	L

Table II. Periodontal diagnosis (post-treatment).

Maxillary								
PMA Index			P				P	
Probing pocket depth (mm).	Facial	323	333	323	323	323	333	
	Lingual	333	323	333	333	323	323	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		3	2	2	2	2	3	
	R	3	2	1	1	2	3	L
Mandibular								
PMA Index							P	
Probing pocket depth (mm).	Facial	323	323	222	222	323	333	
	Lingual	333	322	222	222	223	333	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		3	2	2	2	2	2	
	R	3	2	1	1	2	3	L

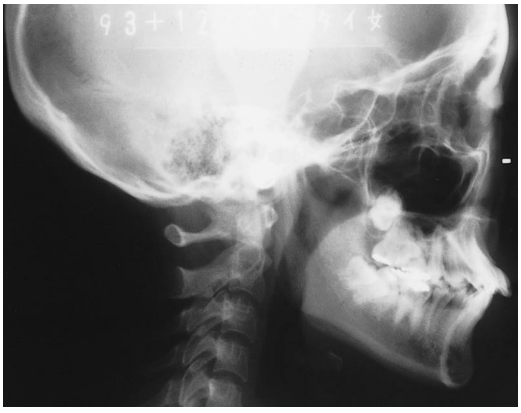


Figure 4a. Cephalometric radiograph (pretreatment).

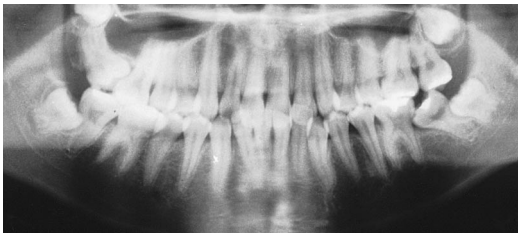


Figure 4b. Panoramic radiograph (pretreatment).

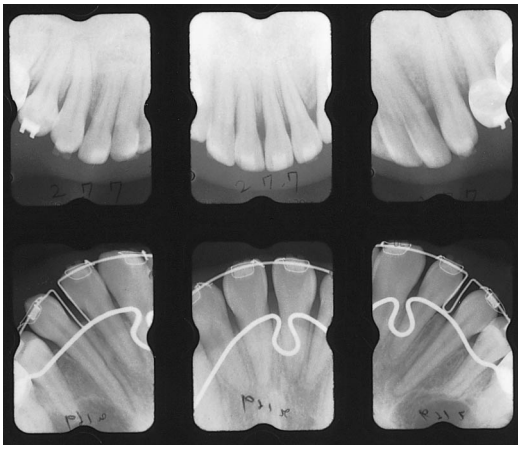


Figure 4c. Periapical radiographs (before *en masse* movement).

Co-ordinated 0.017" x 0.025" wires were placed, and the arches were detailed with finishing bends. After 25 months of treatment, the appliance was removed and retainers placed. A maxillary circumferential removable retainer was used to retain the maxillary arch, and a lingual bonded retainer from canine to canine was used to retain the mandibular arch.

Laser irradiation

A Ga-Al-As diode laser* that has a continuous wavelength of 830 nm and a maximum power output of 700 mW (Figure 5) was used in this study. The laser beam was delivered by an optical fibre 0.6 mm in diameter that expanded at the tip of the fibre and irradiated a circular area 130 mm in diameter at the level of the cell layer. The power density of the laser beam in the central area (65 mm in diameter) of the irradiation circle was uniform as measured by a laser power meter. Laser irradiation was applied to an area of 0.5 cm^2 at the labial gingival papilla, between the lateral and central incisors in the mandible. The time of exposure was for 3 minutes, once a day for 3 days (immediately, and at 24 and 48 hours) carried out between the relevelling and *en masse* stages of movement. The total energy corresponding to 6 minutes exposure varied from 1.90 J/cm^2 (Figure 6).

Figure 5. Ga-A1-As diode laser (Panalas-1000).

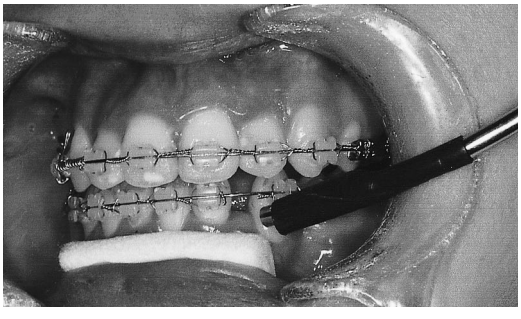
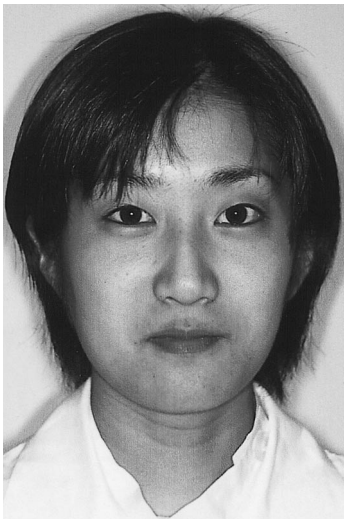


Figure 6. Laser irradiation.

* Model: Panalas-1000; Matsushita, Tokyo, Japan.



Figures 7a to d. Extra-oral photographs (post-treatment).

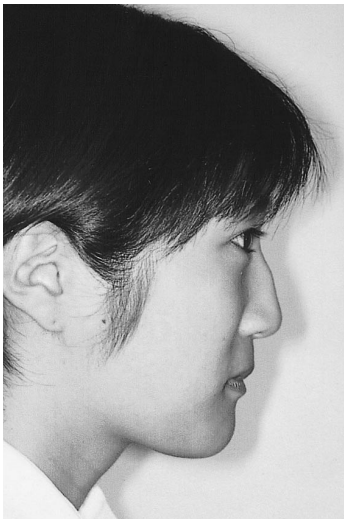


Figure 7b.

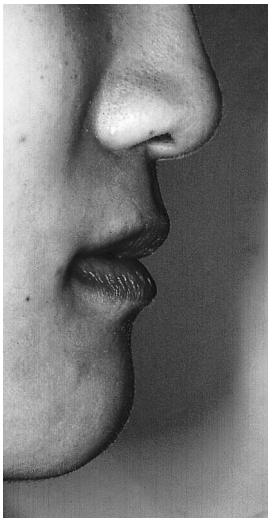
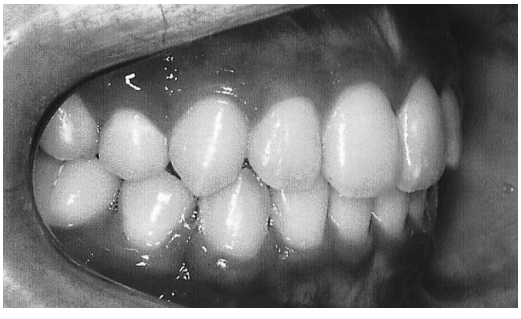


Figure 7c.



Figures 8a to e. Intra-oral photographs (post-treatment).

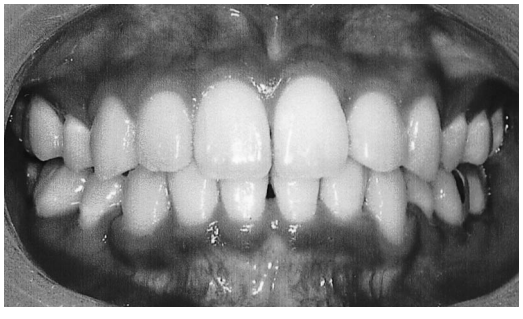


Figure 8b.

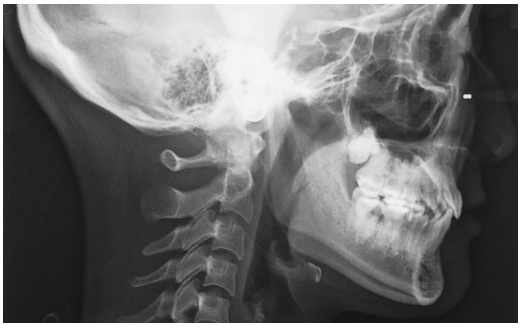


Figure 9a. Cephalometric radiograph (post-treatment).

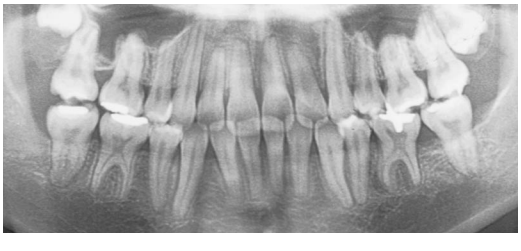


Figure 9b. Panoramic radiograph (post-treatment).

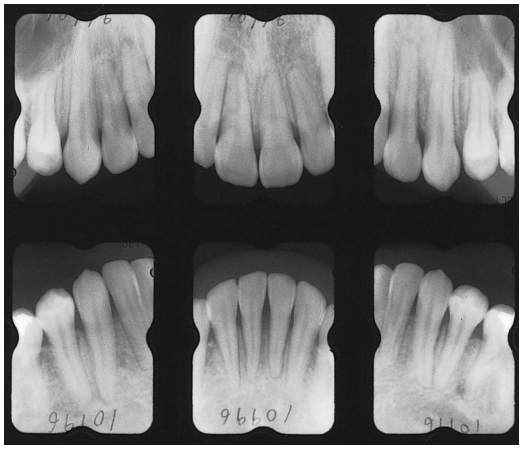


Figure 9c. Periapical radiographs (post-treatment).

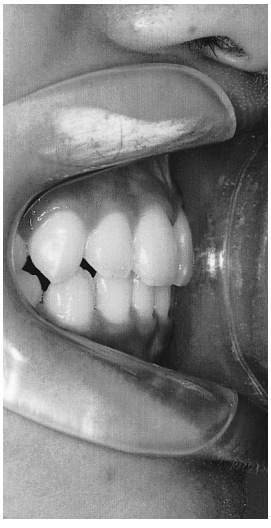


Figure 7d.

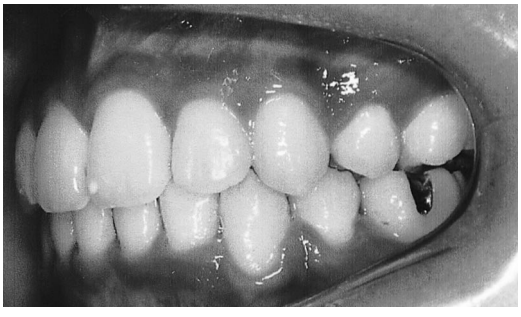


Figure 8c.

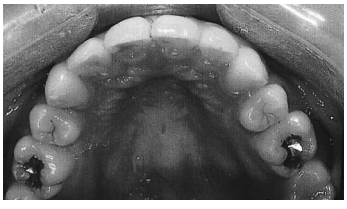


Figure 8d.

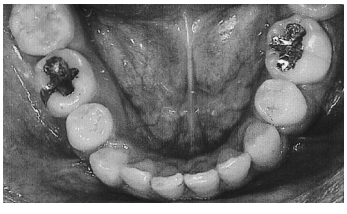


Figure 8e.

Treatment results

Facial aesthetics: Overall facial aesthetics were improved (Figures 7a to c).

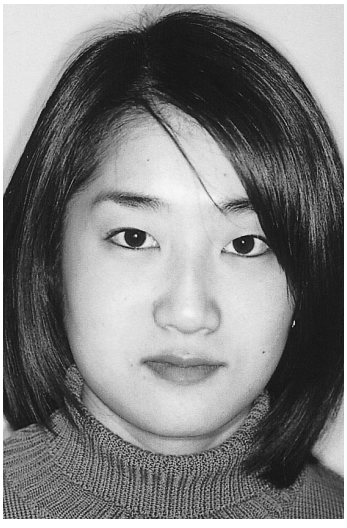
Occlusion: The Class I molar relationship was maintained, and the Class I canine relationship improved. The overjet and overbite were reduced (Figure 7d).

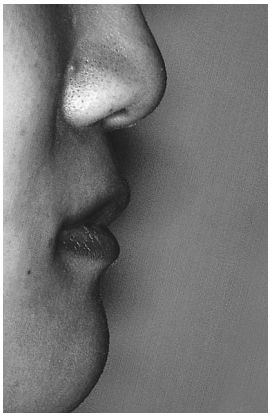
Maxillary and mandibular dentition: The arch length deficiency was eliminated, and the incisors were proclined. The mesial inclination of the mandibular second molar and the cross bite of the right first molar were improved (Figures 8a to e). The pretreatment (Figure 4a) and post-treatment cephalometric radiographs (Figure 9a) and the cephalometric summary (Table IV) demonstrate significant positional changes to the maxillary and mandibular incisors. The maxillary incisors were proclined during treatment from a pretreatment U1-SN angle of 121.0 degrees to a post-treatment angle of 102.5 degrees. The mandibular incisors were proclined from an initial FMIA angle of 60.0 degrees to a final measurement of 65.0 degrees.

Periodontal condition: No incidence of open gingival embrasure space was found, apart from the mandibular central incisor (Figure 8). Further, improvement of the gingival inflammation associated with the periodontal disease was observed. The post-treatment PMA Index had improved when compared with the pretreatment Index, and periodontal pocket depth was maintained (Table II).

The treatment results met all the initial objectives, and the patient was pleased with the overall results.

After retention, regular visits were maintained for twelve months. The open gingival embrasure space of the mandibular central incisor, the PMA Index, the GI, the width of attached gingiva and the periodontal pocket depth were all maintained during this period (Figures 10, 11 and 12, and Table III).

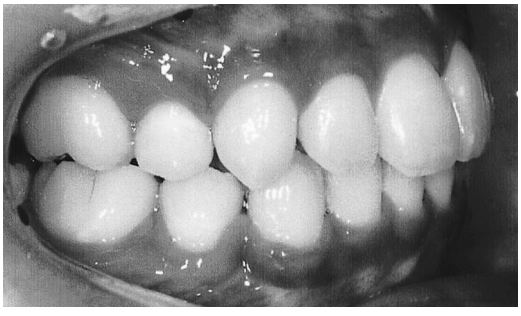


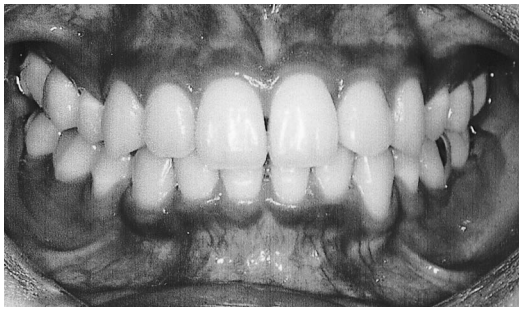


Figures 10a to d. Extra-oral photographs (retention).

Figure 10b.

Figure 10c.





Figures 11 a to e. Intra-oral photographs (retention).

Figure 11b.

Table III. Periodontal diagnosis (retention period).

Maxillary								
PMA Index				P	P	P		
Probing pocket depth	f	323	333	323	323	323	333	
	l	333	323	333	333	323	323	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		2	2	2	2	2	2	
	R	3	2	1	1	2	3	L
Mandibular								
PMA Index			P	P	P			
Probing pocket depth	f	323	323	233	333	323	333	
	l	333	322	323	323	223	333	
Gingival Index (GI)		0	0	0	0	0	0	
Width of attached gingiva		2	2	2	2	2	2	
	R	3	2	1	1	2	3	L

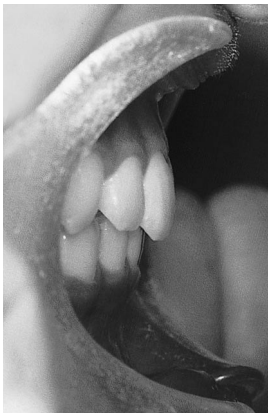


Figure 10d.

Table IV. Analysis of cephalograms.

	Pretreatment (20 years,) 7months	Post-treatment (23 years, 0 months)	Retention (24 years, 0 months)
SNA	81.0	81.0	81.0
SNB	80.0	80.0	80.0
ANB	1.0	1.0	1.0
FMIA	60.0°	65.0°	65.0°
U1-SN	121.0°	102.5°	102.5°
FMA	24.5	25.0	25.0

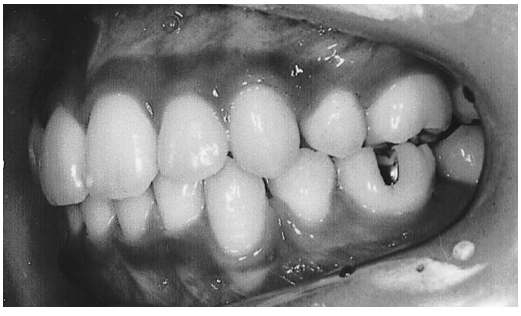


Figure 11c.

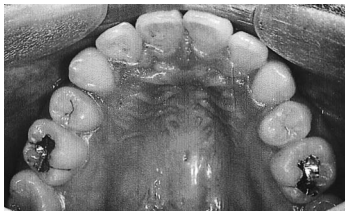


Figure 11d.

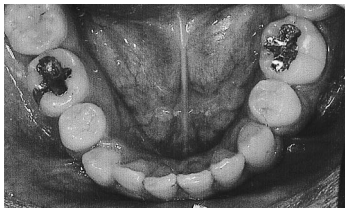


Figure 11e.

Discussion

No incidence of an open gingival embrasure space was found after this particular orthodontic treatment, except at the mandibular central incisor. Further: a reduction of the gingival inflammation associated with a periodontal disease was observed.

Zachrisson and Alnaes measured the mean distance between the cemento-enamel junction and alveolar crest at 1.11mm in an orthodontically treated group of adolescents and 0.88 mm in an untreated control group.[9] Kennedy *et al.* reported that alveolar bone heights were reduced by orthodontic treatment.[10] Therefore, the incidence of open gingival embrasure space may be caused by alveolar bone loss in response to orthodontic treatment. Burke *et al.* suggested that the triangular gingival embrasure space can frequently be closed by torquing the central incisor roots towards each other, decreasing the width and shortening the cervico-incisal length of the gingival embrasure as the mesioproximal crown contacts are maintained.[1]

Polishing mesioproximal enamel from the contact areas of the crowns of both central incisors and moving the two together will further decrease the width of the gingival embrasure. However, although this procedure may effectively close a slight triangular gingival embrasure space between the central incisors, it may be not able to close a marked space with alveolar bone loss (such as in Figure 1b). Therefore, laser irradiation may be useful only for inhibition of the incidence of open gingival embrasure spaces that are due to bone loss and gingival recession.

Yamaguchi *et al.* reported that mechanical stress induced the production of prostaglandin E_2 (PGE_2) and plasminogen activator (PA) activity by human periodontal ligament cells.[11,12] Further,

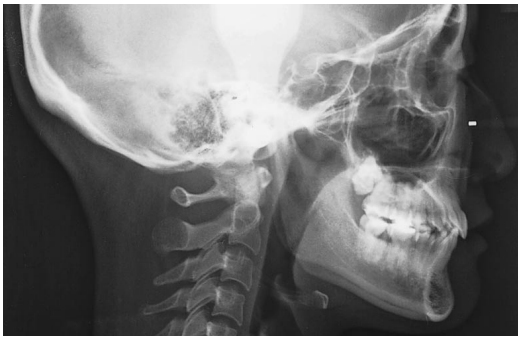


Figure 12a. Cephalometric radiograph (retention).

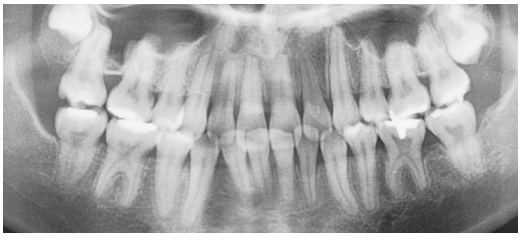


Figure 12b. Panoramic radiograph (retention).

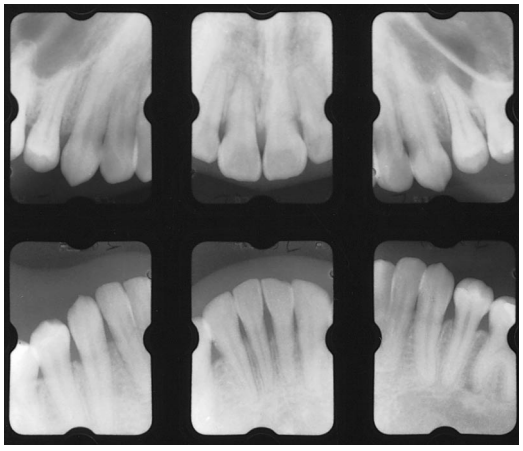


Figure 12c. Periapical radiographs (retention).

Grieve *et al.* reported that the PGE levels in gingival crevicular fluid were elevated during orthodontic treatment.[13] There is a possibility that prostaglandins may play a role in the bone metabolism[14] and in inflammation.[15]

Klein and Raisz found that PGE_2 is a potent stimulator of bone resorption.[16] The relationship between PGE_2 and the progression of periodontal diseases has been intensively studied. PGE_2 concentration increases in the periodontal connective tissues of adult periodontitis lesions.[17–20] Further, PGE_2 levels in the crevicular fluid can serve as a static assessment of ongoing disease activity.[21, 22]

PA, a wide-spectrum serine protease that is secreted from various types of cells including fibroblasts, activates circulating plasminogen to produce plasmin. Plasmin is capable of degrading extracellular matrix proteins and cell adhesive molecules[23] through activation of matrix metalloproteases.[24] Furthermore, plasmin activates the kinin cascade through the conversion of prekallikrein into kallikrein.[25] Therefore, PA is considered to play a central role in the breakdown of the extracellular matrix during tissue destruction.[26, 27] The relationship between periodontal disease and the PA-plasmin system has been investigated. Hidaka *et al.* reported that the activities of plasmin and PA are increased in the gingival fluid in periodontal disease,[28] with plasmin activity being markedly decreased by periodontal treatment.[29] In an *in vitro* study of the effects of laser irradiation, Shimizu *et al.* reported that laser irradiation inhibited the mechanical stress induced production in human periodontal ligament cells of prostaglandin E_2.[30] Further, it is also reported that laser irradiation inhibits PA activity and PGE_2 production by lypopolysaccharide of periodontal pathogen.[31, 32] These findings suggest that laser irradiation may reduce PGE_2 and PA production in gingival tissues that become inflamed during orthodontic treatment.

Although there are various factors that contribute to the incidence of black triangles, no useful methods of treating the problem have been reported. Therefore, it is important to prevent black triangles from occurring, and laser-irradiation is one preventative method. This study shows that the effects of laser irradiation can be clinically evaluated. Further research in this field is being conducted by the authors.

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References

1. Burke S, Burch JG, Tetz J A. Incidence and size of pre-treatment overlap and post-treatment gingival embrasure space between maxillary central incisors. Am J Orthod Dentofac Orthop 1994; 105: 506–11.
2. Honmura A, Yanase M, Obata J, Haruki E. Therapeutic effect of Ga-Al-As diode laser irradiation on experimentally induced inflammation in rats. Lasers Surg Med 1992; 12: 441–9.
3. Honmura A, Ishii A, Yanase M, Obata J, Haruki E. Analgesic effect of Ga-Al-As diode laser irradiation on hyperalgesia in carrageenan-induced inflammation in rats. Lasers Surg Med 1992; 13: 463–9.
4. Amano A, Miyagi K, Azuma T, Ishihara Y, Katsube S, Aoyama I, Saito I. Histological studies on the rheumatoid synovial membrane irradiated with a low energy laser. Lasers Surg Med 1994; 15: 290–4.
5. Kana JS, Hutschenreiter G, Haina D, Waidelich W. Effect of low-power density laser radiation on healing of open skin wounds in rats. Arch Surg, 1981; 116: 293–6.
6. Korynyi DL. Use of a helium-neon laser in therapeutic stomatology. Stomatologiia 1978; 57: 21–6.
7. Lutsyk LA, Rybert IA, Svishch MP. Use of a helium-neon laser in the combined treatment of oral mucosal diseases in children. Stomatologiia 1981; 60: 6–15.
8. Kert J and Rose L. Klinisk laserbehandling-low level laser therapy. Scandinavian Medical Laser Technology. Osterbrogade 1989; 135: 2100 Kobenhavn.
9. Zachrisson BU, Alnaes L. Periodontal condition in orthodontically treated and untreated individuals. II. Alveolar bone loss: radiographic findings. Angle Orthod 1974; 44: 48–55.
10. Kennedy DB, Joondoph DR, Osterberg SK, Little RM. The effect of extraction and orthodontic treatment on dentoalveolar support. Am J Orthod 1983; 84: 183–90.
11. Yamaguchi M, Shimizu N, Goseki T, Shibata Y, Takiguchi H, Iwasawa T, Abiko Y. Effect of different magnitudes of tension force on Prostaglandin E2 production by periodontal ligament cells. Arch of Oral Biol 1994; 39: 877–84.
12. Yamaguchi M, Shimizu N, Ozawa Y, Saito K, Miura S, Iwasawa T, Takiguchi T, Abiko Y. Effect of tension-force on plasminogen activator activity from human periodontal ligament cells. J Period Res 1997; 32: 308–14.
13. Grieve III WG, Johnson GK, Moor RN, Reinhardt RA, DuBois LM. Prostaglandin E (PGE) and interleukin-1β (IL-1β) levels in gingival crevicular fluid during human orthodontic tooth movement. Am J Orthod Dentof Orthop 1994; 105: 369–74.
14. Chase LR, Aurbach GD. The effect of parathyroid hormone on the concentration of adenosine3,',5'-monophosphate inskeletal tissue in vitro. J Biol Chem 1970; 245: 1520–6.
15. Lewis GP. The role of prostaglandins in inflammation. Vienna: Hans Hiber, 1976.
16. Klein DC, Raisz LG. Prostaglandins: stimulation of bone resorption in tissue culture. Endocrinology 1970; 86: 1436–40.
17. Goodson JM, Dewhirst FE, Brunetti A. Prostaglandin E2 levels and human periodontal disease. Prostaglandins 1974; 6: 81–5.
18. Loning TH, Albers H-K, Lisboa BP, Burkhardt A, Caselits J. Prostaglandin E and the local immune response in chronic periodontal disease: immunohistochemical and radioimmunological observations. J Periodont Res 1980; 15: 525–35.
19. Offenbacher S, Heasman PA, Coolins JG. Modulation of host PGE2 secretion as a determinant of periodontal disease expression. J Periodontol 1993; 64: 432–44.
20. Salvi GE, Beck JD. PGE2, IL-1β, and TNF-αl in diabetics as modifiers of periodontal disease expression. Ann Periodontol 1998; 3: 40–50.
21. Offenbacher S, Odle BM, van Dyke TE. The use of crevicular fluid prostaglandin E2 levels as a predictor of periodontal attachment loss. J Periodont Res 1986; 21: 101–12.
22. Cavanaugh PF Jr, Meredith MP, Buchanan W, Doyle MJ, Reddy MS, Jeffcoat MK. Coordinate production of PGE2 and IL-1β the gingival crevicular fluid of adults with periodontitis: its relationship to alveolar bone loss and disruption by twice daily treatment with ketorolactromethamine oral rinse. J Periodont Res 1998; 33: 75–82.
23. Castellino FJ. Biochemistry of human plasminogen. Semin Thromb Hemost 1984; 10: 18–23.
24. Werb Z, Mainardi CL, Vater CA, Harris Jr ED. Endogeneous activation of latent collagenase by rheumatiod synovial cells. N Engl J Med 1977; 296: 1017–23.
25. Vogt W. Kinin formation by plasmin: an indirect process mediated by activation of kallikrein. J Physiol 1964; 170: 153–66.
26. Saksela O. Plasminogen activation and regulation of pericellular proteolysis. Biochem Biophys Acta 1985; 823: 35–65.
27. Dano K, Andreasen PA, Grondahl-Hansen J, Kristensen P, Nielsen LS, Skriver L, Plasminogen activators, tissue degradation and cancer. Adv Cancer Res 1985; 44: 139–266.
28. Hidaka N, Maeda K, Kawakami C, Aono M, Okada H. Fibrinolytic activity in periodontal disease: the relationship between fibrinolytic activity and severity of periodontal disease. J Periodontol 1981; 52: 181–6.
29. Talonpoika J, Soderling E, Tiekso J, Paunio K. Gingival crevicular fluid plasmin activity in different clinical conditions and after periodontal treatment. Proc Finn Dent Soc 1991; 87: 329–37.
30. Shimizu N, Yamaguchi M, Goseki T, Shibata Y, Takiguchi H, Iwasawa T, Abiko Y. Inhibition of prostaglandin E2 and interleukin-1β production by low-power laser irradiation in stretched human periodontal ligament cells. J Dent Res 1995; 74: 1382–8.
31. Takema T, Yamaguchi M, Abiko Y. Reduction of plasminogen activator activity stimulated by lipopolysaccharide from periodontal pathogen in human gingival fibroblasts by low-power laser irradiation. Laseres Med Sci 2000; 15: 35–42.
32. Sakurai Y, Yamaguchi M, Abiko Y. Inhibitory effect of low-level laser irradiation on LPS-stimulated prostaglandin E2 production and cyclo-oxygenase-2 in human gingival fibroblasts. Eur J Oral Sci 2000; 108: 29–34.