ORIGINAL ARTICLE

Periodontal status of mandibular incisors after pronounced orthodontic advancement during adolescence: A follow-up evaluation

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The purpose of this study was to analyze whether pronounced orthodontic advancement of the mandibular incisors during Class II correction in the mixed dentition results in gingival recession. Through mandibular superimposition of the pretreatment and posttreatment cephalograms of 67 Class II patients who were treated with reverse headgear to the mandibular dentition, 45 patients with a minimum of a 1-mm advancement of the cementoenamel junction (CEJ; mean, 2.18 ± 0.87) and a minimum of a 2-mm advancement of the incisal edge (mean, 3.87 ± 1.34) were identified. Using the same protocol in Class II patients, 30 individuals who finished treatment at a similar time and age, but without reverse headgear and with no advancement of the CEJ (mean –0.43, SD 0.53) and a maximum of 1-mm advancement of the incisal edge (mean –0.26 SD 1.15) were identified. Before treatment, the mandibular incisors were more retruded, relative to the line from point A to pogonion and relative to the mandibular plane in the patients with pronounced advancement than in those with no advancement of the mandibular incisors; no differences were found at the time of appliance removal. A total of 30 patients with pronounced advancement and 21 patients with no advancement could meet for a follow-up examination a mean period of 7.83 years (SD, 4.44) and 9.38 years (SD, 4.39) after treatment, respectively. Clinical examinations at the time of follow-up revealed no differences in the amount of recession, the width of attached gingiva, the length of supracrestal connective tissue attachment, the probing pocket depth, and gingival bleeding index or visible plaque index of the mandibular incisors between the patients in the 2 groups. An examination of color slides demonstrated no differences in the number of mandibular incisors that developed recession from before treatment to after treatment and from after treatment to follow-up. Measurement of mandibular incisor crown height on the study models demonstrated no difference in the increase in clinical crown height from after treatment to follow-up between the patients in the 2 groups. It was concluded that pronounced advancement of the mandibular incisors may be performed in adolescent patients with dentoalveolar retrusion without increasing the risk of recession. (Am J Orthod Dentofacial Orthop 2001;119:2-10)

Teeth subjected to orthodontic forces will move in the direction the forces are applied, through alveolar resorption on the pressure side and apposition on the tension side of the periodontal ligament, concomitant with the rearrangement of the periodontal fibers.[1] Experimental studies in dogs suggest that the remodeling occurs without loss of connective tissue attachment and crestal alveolar bone, provided the tooth movement is performed along the dental arch and the supracrestal connective tissue is kept free of inflammation.[2,3] Accordingly, most of the variation in periodontal attachment loss found in groups of orthodontic patients who were treated with fixed appliances[4-6] may be explained by variations in level of oral hygiene performance and resistance to plaque-associated periodontal breakdown among the patients.

Orthodontic tooth movement in vestibular direction may induce bone apposition on the vestibular surface of the alveolar process.[1,7] Accordingly, vestibular crestal bone height may be maintained during orthodontic expansion, as evidenced in experimental studies in humans[7] and monkeys.[8] However, experimental studies in monkeys[9-11] and dogs[12] have demonstrated the formation of alveolar bone dehiscences in the vestibular area of the incisors after excessive anterior movement, particularly if the expansion is combined with extrusion of the teeth.[9] Provided meticulous professional tooth cleaning is performed, loss of connective tissue attachment may not accompany the crestal bone loss during the course of the expansion.[11,12] However, the size of the free gingival unit may be reduced, probably because of the tension that results in minute apical displacement of the gingival margin (GM).[10,11] In areas of clinically detectable inflammation, attachment loss and more pro-

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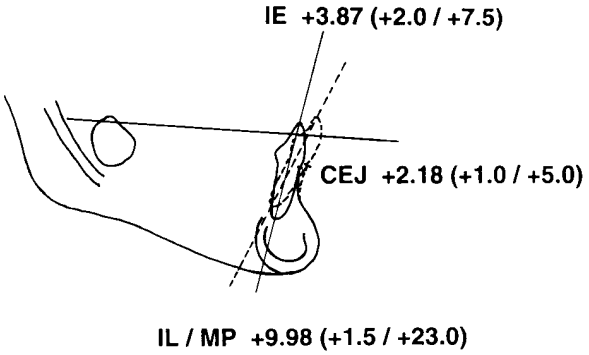

Fig 1. Mandibular superimposition of pretreatment and posttreatment cephalograms of average patient in group that fits criteria for pronounced advancement of the mandibular incisors during appliance therapy. Sample means (ranges in parentheses) of changes in position of IE and CEJ and in IL/MP are indicated. The plus (+) denotes anterior movement or proclination.

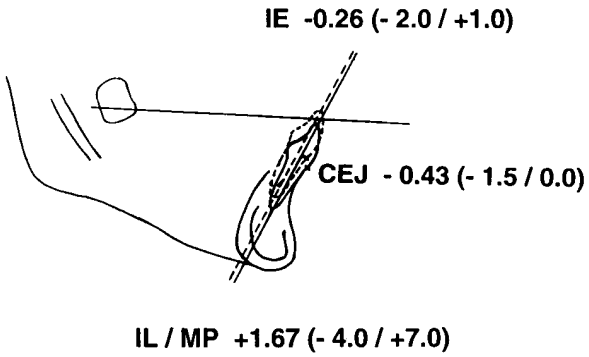

Fig 2. Mandibular superimpositions of pretreatment and posttreatment cephalograms of average patient in group that fits criteria for no advancement of mandibular incisors during appliance therapy. Sample means (ranges in parentheses) of changes in position of IE and CEJ and in IL/MP are indicated. The plus (+) denotes anterior movement or proclination and posterior movement or retroclination.

nounced recession may occur.[10,11] The recession seen in 1 experiment,[9] in which the monkeys were subjected to biweekly tooth cleaning, may therefore reflect that the regime was suboptimal for the prevention of plaque-associated periodontal breakdown.

Vestibular areas with long supracrestal connective tissue attachments produced during orthodontic expansion may be at increased risk of the development of recession over time. Such speculations are reinforced by the high frequency of gingival recession observed on the labial aspect of prominent teeth in persons who have not received orthodontic treatment.[13,14] One reason may be a reduced resistance to tooth-brushing trauma in vestibular areas with thin periodontal structures.[13,15] The resistance against progression of plaque-associated attachment loss may, on the other hand, not be reduced in situations with bone dehiscence.[16] However, if the marginal soft tissue is so thin that the inflammatory lesion occupies the total width of the connective tissue, the periodontal breakdown is likely to be recession rather than pocketing because of proliferation of both oral and dentogingival epithelial cells into the thin and degraded connective tissue.[17,18]

Pronounced orthodontic expansion is necessary in several clinical situations to achieve optimal esthetic and occlusal results. A recent study in adults who needed more than 10° proclination of the mandibular incisors during the orthodontic decompensation before mandibular set-back surgery concluded that such expansion was accompanied with a significant risk of gingival recession, particularly if the alveolar process was thin.[19] Most of the recession occurred during treatment and the first 3 years after surgery. After that time, the progression was similar to that seen in a control group of patients who had not experienced presurgical expansion. These results combined with inferences from the experimental studies led the authors to hypothesize that occasional bone dehiscences were produced during the expansion. The areas with bone dehiscence may then have been subjected to a relatively rapid gingival recession until a normal distance was established between the bottom of the pocket and the crestal bone, which reestablished a structure possibly more conducive to stabilizing the process.[19]

Class II patients with severe dentoalveolar retrusion may also benefit from pronounced orthodontic advancement of the mandibular incisors. Although tissue adaptability may be increased in growing individuals because of the ongoing remodeling of the alveolar processes that accompanies facial growth, case reports indicate a risk of recession after expansion also in adolescent patients.[20] The purpose of this study was to analyze the frequency and severity of gingival recession forming during and after Class II correction among patients who were treated with pronounced mandibular dentoalveolar advancement in the mixed dentition.

MATERIAL AND METHODS

Screening of subjects

Records of Class II cases treated by 1 of the authors (D.G.) were screened. Only cases started in the mixed dentition with an activator and extraoral traction combination for orthopedic correction and followed by a period of fixed appliance therapy to achieve ideal tooth positions[21] were selected. An attempt was made to

Table I. Age and cephalometric measurements before (*T-1*) and after (*T-2*) treatment and at time of follow-up (*T-3*) in patients with pronounced (n = 30 patients) and no (n = 21 patients) advancement of the mandibular incisors from T-1 to T-2

Variables	*Pronounced advancement (mean ± SD)*	*No advancement (mean ± SD)*	*Difference*	P *value*
T-1				
Age (y)	10.29 ± 1.39	10.07 ± 1.76	0.22	.63
Symphysis (mm)	9.90 ± 1.28	10.09 ± 1.37	0.19	.60
IE/APg (mm)	–3.47 ± 2.08	–0.64 ± 1.94	–2.83	<.01
IL/MP (°)	89.13 ± 6.09	94.55 ± 5.48	5.42	<.01
MP/SNL (°)	30.43 ± 3.22	35.60 ± 13.54	5.17	.10
T-2				
Age (y)	14.48 ±1.86	14.50 ± 1.37	0.02	.98
Symphysis (mm)	8.83 ± 1.18	9.02 ± 1.68	0.19	.64
IE/APg (mm)	2.27 ± 1.51	1.69 ± 1.85	0.58	.23
IL/MP (°)	99.12 ± 5.43	96.21 ± 4.72	2.91	.06
MP/SNL (°)	30.60 ± 3.59	35.74 ± 14.19	5.14	.12
T-3				
Age (y)	22.32 ± 4.65	23.88 ± 4.44	1.56	.24
Symphysis (mm)	8.58 ± 1.15	8.53 ± 1.90	0.05	.90
IE/APg (mm)	1.08 ± 1.93	1.50 ± 2.38	0.42	.50
IL/MP (°)	96.35 ± 6.51	95.70 ± 5.29	0.65	.71
MP/SNL (°)	28.53 ± 4.28	33.35 ± 13.91	4.82	.15

Table II. Changes in cephalometric measurements of incisor position and number of incisors developing recession from before to after treatment in patients with pronounced (n = 30 patients) and no (n = 21 patients) advancement of the mandibular incisors during treatment

	Pronounced advancement (mean ± SD)	*No advancement (mean ± SD)*	*Difference*	P *value*
IE-1/IE-2 (mm)	3.87 ± 1.34	–0.26 ± 1.15	4.13	<.01
CEJ-1/CEJ-2 (mm)	2.18 ± 0.87	–0.43 ± 0.53	2.61	<.01
IE/APg (mm)	5.73 ± 1.81	2.33 ± 1.35	3.40	<.01
IL/MP (°)	9.98 ± 5.56	1.67 ± 3.42	8.31	<.01
Recession (mm)	0.40 ± 0.76	0.08 ± 0.32	0.32	.06

examine all cases that were treated with reverse headgear to the mandibular dentition during the orthopedic phase in the period from 1975 to 1990.[21] Mandibular superimposition of cephalograms made before (T-1) and after (T-2) active treatment was performed on the 67 cases identified. The mandibles were traced on the cephalograms at T-1, which emphasized the identification of the natural structures in the corpus that were used as reference for superimposition,[22] the incisal edge (IE-1), the labial projection of the cementoenamel junction (CEJ-1), and the occlusal plane (OP-1). The cephalometric tracing at T-1 was superimposed on the corresponding cephalogram at T-2, according to the "best anatomic fit" of the natural structures.[22] Then IE-2 and CEJ-2 of the incisor at T-2 were traced. The distances from IE-1 to IE-2 and from CEJ-1 to CEJ-2 parallel to OP-1 were measured to the nearest 0.5 mm (Figs 1 and 2) with a transparent grid. Pronounced advancement of the mandibular incisors (defined as a minimum of 1.0 mm anterior movement of CEJ and 2.0 mm anterior movement of IE) was diagnosed in 45 of the 67 cases. Following the same protocol, measurements were made on cephalograms of Class II patients who finished treatment at similar time and age, but without the use of reverse headgear. No advancement of the mandibular incisors (defined as no anterior movement of CEJ and a maximum of 1.0 mm anterior movement of IE) was diagnosed in 30 patients.

Selection of subjects

A total of 30 patients with pronounced advancement and 21 patients with no advancement of the mandibular incisors could meet for a follow-up examination. At that time (T-3), study models, cephalograms, and intraoral slides were made, and clinical examinations were performed. The percentages of female patients were 67.7 and 71.4 in the 2 groups. There were no differences in age at the 3 time periods (Table I). The differences in measurements IE-1 to IE-2 and CEJ-1 to CEJ-2 were significant between the groups ($P < .01$; Table II; Figs 1 and 2).

Measurements on cephalograms

The cephalograms at T-1, T-2, and T-3 were traced and superimposed by hand. The inclination of the mandibular incisor relative to the mandibular plane (IL/MP) and the inclination of the mandibular plane relative to the line from nasion to sella (MP/SNL) were measured to the nearest 0.5° with a protractor. The distance from the IE to the line from point A to Pogonion (APg) and the width of the symphysis at the level of the apex of the incisors[19] were measured to the nearest 0.5 mm with a transparent ruler. Mandibular superimposition of the cephalograms at T-2 and T-3 was performed to measure the movement of IEs from T-2 to T-3 (IE-2 to IE-3) along OP at T-2 (OP-2).

Measurements on study models

The clinical crown height of the mandibular incisors at T-2 and T-3 were measured to the nearest 0.1 mm with digital calipers. The distances were measured from the deepest point of the curvature of the vestibulogingival margin to the IE-1 of the crown of the incisor.

Examination of intraoral color slides

Color intraoral slides at T-1, T-2, and T-3 were projected onto a screen. The gingival tissue was recorded as recessive if the labial CEJ was exposed.[23] U-shaped recessions of the gingiva combined with hyperplasia or V-shaped recessions were also registered.[23]

Clinical examinations

Scoring was performed on the mid labial aspect of the 4 mandibular incisors at T-3. All measurements were made with a Marquis probe. Hygiene status and gingival health were scored according to the visible plaque index and gingival bleeding index systems.[24] When CEJ was exposed, the amount of recession was measured to the nearest 0.5 mm as the distance from GM to CEJ. Otherwise recession was scored as 0. Width of keratinized tissue was measured as the distance from GM to the mucogingival junction (MGJ) and the probing pocket depth as the distance from the GM to the bottom of the pocket (BP). The distance from alveolar bone (AB) to GM was measured after forcing the tip of the probe through the connective tissue until definite resistance was met.[25] All three measurements were rounded to the nearest millimeter. The length of the supracrestal connective tissue attachment was calculated by subtracting the distance GM-BP from the distance GM-AB, and the width of attached gingiva by subtracting the distance GM-BP from the distance GM-MGJ (Fig 3).

Error of the method

The reproducibility of the measurements on study models and cephalograms was assessed by statistically analyzing the difference between double measurements made 5 days apart on 20 randomly selected study models, cephalograms, and superimpositions. The cephalograms were retraced and superimposed before the second measurement. The method error was calculated from the equation:

$$S_x = \frac{\sum D^2}{2N}$$

where D is the difference between duplicated measurements and N is the number of double measurements.[26] The errors for angular and linear measurements on the cephalograms did not exceed 0.48° and 0.42 mm; the error for measurement of crown height was 0.09 mm.

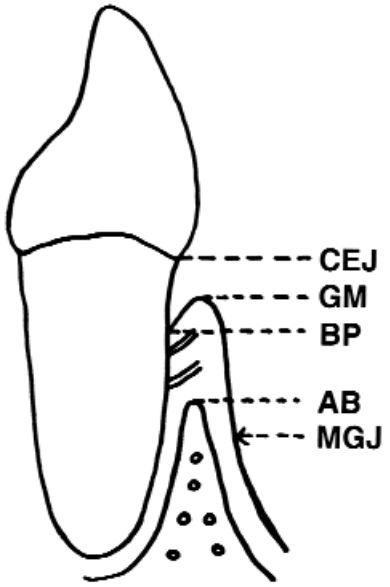

Fig 3. Line drawing indicates location of CEJ, GM, BP, AB, and MGJ. Recession measured as distance CEJ-GM when CEJ was exposed, probing pocket depth as distance GM-BP, length of supracrestal connective tissue attachment as distance BP-AB, and width of attached gingiva as distance BP-MGJ. Clinical crown height measured on study models as distance from IE-1 to GM.

Data analysis

Group means and SDs were calculated for each variable at each time period and for changes of each variable between time periods, with the averaged value per tooth per patient of the clinical measurements and the measurements of crown height and of the number of teeth per patient with development of recession. The Student *t* test for independent means was used to test for intergroup differences. Such tests were also used to test for differences in the width of symphysis at each

Table III. Changes in cephalometric measurements of incisor position, number of incisors developing recession, and average increase in clinical crown height of the incisors from after treatment to time of follow-up in patients with pronounced (n = 30 patients) and no (n = 21 patients) advancement of the mandibular incisors during treatment

	Pronounced advancement (mean ± SD)	*No advancement (mean ± SD)*	*Difference*	P *value*
IE-2/IE-3 (mm)	–1.23 ± 1.33	–0.50 ± 1.45	–0.73	.07
CEJ–2/CEJ–3 (mm)	–0.36 ± 0.56	–0.26 ± 0.41	–0.10	.63
IE/APg (mm)	–1.18 ± 1.26	–0.22 ± 1.41	–0.96	<.05
IL/MP (°)	–2.77 ± 3.10	–0.62 ± 3.86	–2.15	<.05
Recession (mm)	0.56 ± 0.80	0.64 ± 1.12	0.08	.71
Crown height (mm)	0.55 ± 0.48	0.45 ± 0.81	0.10	.65

Table IV. Averaged clinical scores of incisors at time of follow-up in patients with pronounced (n = 30 patients) and no (n = 21 patients) advancement of the mandibular incisors during treatment

	Pronounced advancement (mean ± SD)	*No advancement (mean ± SD)*	*Difference*	P *value*
Visible plaque index (score 0,1,2)	0.23 ± 0.39	0.14 ± 0.23	0.09	.35
Gingival bleeding index (score 0,1,2)	0.15 ± 0.29	0.12 ± 0.18	0.03	.65
Probing pocket depth (mm)	1.20 ± 0.26	1.20 ± 0.26	0.00	.93
Length of supracrestal connective tissue (mm)	1.08 ± 0.27	1.01 ± 0.04	0.07	.21
Width of attached gingiva (mm)	2.35 ± 0.98	2.84 ± 1.06	0.49	.10
Recession (mm)	0.27 ± 0.52	0.17 ± 0.32	0.10	.41

time period between the subsamples of patients with and without teeth with the presence of recession at T-3. Finally, the subset of patients who had teeth both with and without recession at T-2 was selected from the group with pronounced advancement. In these patients, the mean increase in clinical crown height from T-2 to T-3 was calculated separately for the teeth with and without the presence of recession at T-2; the *t* test for dependent means was used to test for significance.

RESULTS

Cephalometric measurements

At T-1, IE was in a more retruded position relative to APg, and IL was in a more retroclined position relative to MP in the patients with pronounced advancement than in those patients with no advancement of the mandibular incisors ($P < .01$; Table I). No other intergroup differences in cephalometric measurements were found at the 3 time periods. Few patients with larger than normal angle MP/SNL were seen among the patients with pronounced advancement (Table I). From T-1 to T-2, more protrusion of IE relative to APg and more proclination of IL relative to MP occurred in the patients with pronounced advancement than in those patients with no advancement ($P < .01$; Table II). From T-2 to T-3, more retrusion of IE relative to APg and more retroclination of IL relative to MP occurred in the patients with pronounced advancement than in those patients with no advancement ($P < .05$; Table III). However, no intergroup differences in posterior movement of IE or CEJ were found from T-2 to T-3 when measured on the superimposition (Table III).

Development of recession

Recession was present on 12 teeth in 8 of the 30 patients with pronounced advancement versus on 2 teeth in 2 of the 21 patients with no advancement at T-2 and on 28 teeth in 15 patients versus 16 teeth in 8 patients, respectively, at T-3. These differences were not significant, and no intergroup difference in increase in crown height was found from T-2 to T-3 (Tables II and III). Also, in the 8 patients with pronounced advancement who had teeth with recession at T-2, the increase in crown height from T-2 to T-3 was not larger for the 12 teeth with recession at T2 (mean, 0.28 ± 0.65 mm) than for the 20 teeth without (mean, 0.85 ± 0.97 mm). In the group with pronounced advancement, the mean width of the symphysis was similar at T-1 (9.50 ± 0.89 mm and 10.30 ± 1.50 mm), at T-2 (8.67 ± 1.22 mm and 9.00 ± 1.16 mm), and at T-3 (8.43 ± 1.16 mm

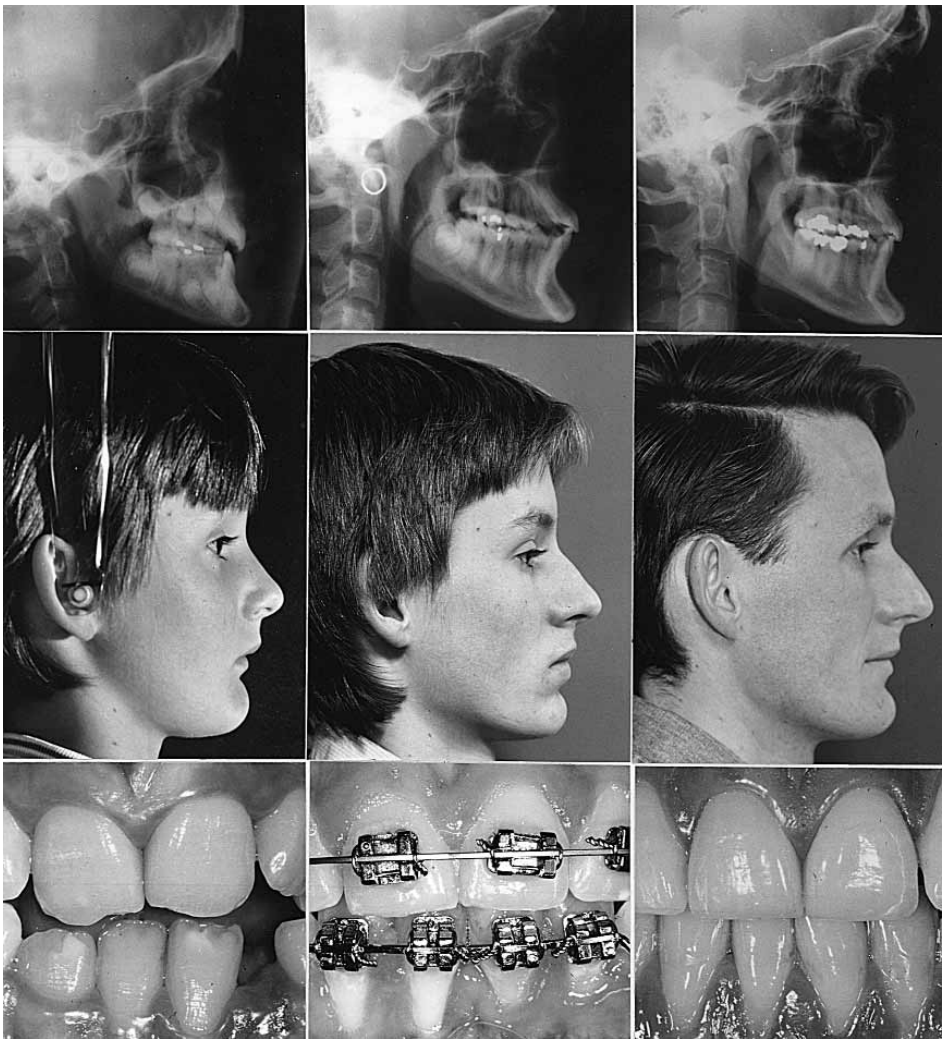

Fig 4. Cephalograms, extraoral photographs, and intraoral photographs made before, after, and 10 years after treatment of patient with pronounced advancement of mandibular incisors during appliance therapy. Note development of recession during appliance therapy on mandibular right central and lateral incisors. Also note minimal increase in recession on those teeth during posttreatment and development of similar amount of recession on mandibular left central and lateral incisors.

and 8.73 ± 1.16 mm) for the patients with and without recession at T-3, respectively. However, in the group with no advancement, the symphysis was significantly narrower at T-1 (9.00 ± 0.93 mm and 10.77 ± 1.15 mm), at T-2 (7.56 ± 0.86 mm and 9.92 ± 1.41 mm), and at T-3 (7.06 ± 1.29 mm and 9.50 ± 1.61 mm) in the patients with recession at T-3 than in those patients without ($P < .01$), respectively.

Clinical scores at time of follow-up

No significant differences in clinical scores were found at T-3 between the patients in the 2 groups (Table IV). The visible plaque index score 2 was assigned to 1 tooth in each of 3 patients with pronounced advancement and to no teeth in the patients with no advancement of the incisors. No gingival bleeding index score 2 was given. The probing pocket depths were 1 and 2 mm, and the maximum length of supracrestal connective tissue attachment was 2 mm. The recession ranged from 0.5 to 3.0 mm in the 8 patients with pronounced advancement who had some recession at T-2, both on the 12 teeth that developed recession from T-1 to T-2 and on the 7 teeth of 4 of the patients who developed recession from T-2 to T-3 (Figs 4 and 5). In contrast, the recession was 0.5 mm on all 9 teeth in the patients with pronounced advancement who did not have recession at T-2.

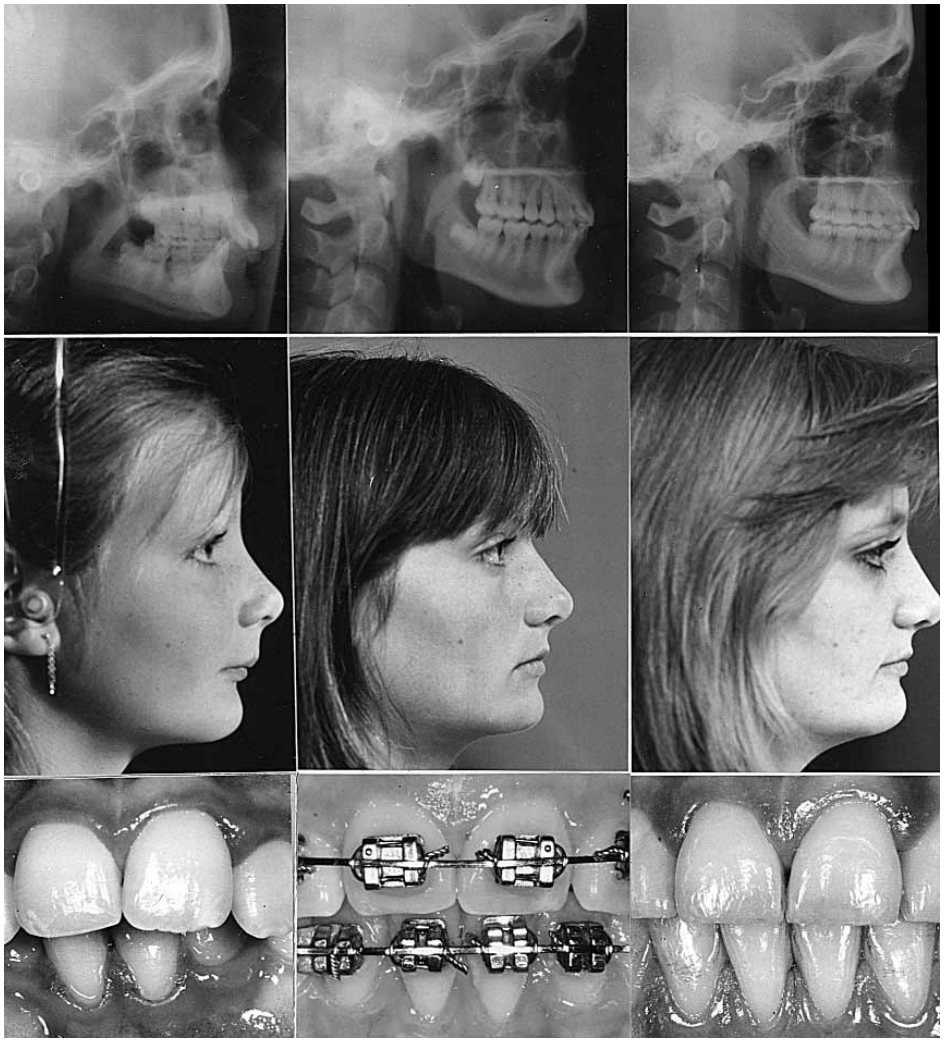

Fig 5. Cephalograms, extraoral photographs, and intraoral photographs made before, after, and 9 years after treatment of patient with pronounced advancement during appliance therapy. Note development of small amount of recession on mandibular central incisors during appliance therapy and minimal change posttreatment.

DISCUSSION

The patients who met our criteria for pronounced advancement of the mandibular incisors during treatment had upright and retruded mandibular incisors relative to established norms and relative to the patients included in the control group. In addition, most of the patients had an inclination of the mandibular plane that fell within the lower range of the population norm; few patients demonstrated a narrow alveolar process in the area of the incisors. It should also be stressed that although the incisors were advanced extensively during treatment, the actual position of the teeth was not anterior to that found in the control subjects at time of appliance removal. Under such conditions, it may be concluded that the mandibular incisors may be advanced considerably during active treatment of Class II malocclusions in young adolescent patients without an increase in the risk of gingival recession.

Analysis of the actual process through which the recessions developed would have required multiple examinations at standardized time periods during and after treatment, which were not performed in this study. However, inferences from experimental studies may lead to the hypothesis that, initially, a bone dehiscence was produced at the labial tooth aspects in question because of a lack of compensatory bone apposition during the orthodontic expansion.[9-12] The consequential long supracrestal connective tissue attachment in those areas

may then have offered less resistance to retraction of the GM. This theory implies a pronounced individual variation in the development of bone dehiscence with expansion; a satisfactory explanation for such variation is lacking. There are suggestions that the orthodontic force system is the decisive variable. The mechanism offered is that correctly planned forces will cause tooth movement "with" rather than "through" bone.[27,28] This concept is speculative, however, and appears to have been derived from the well-documented finding that excessive orthodontic forces invariably produce undermining bone resorption, starting in the marrow spaces or at the alveolar bone wall around the cell free tissue,[1,29] rather than direct surface resorption. If the mechanism[27,28] is correct, orthodontic tooth movement along the dental arch should be associated with crestal bone loss because periods of undermining resorption are difficult to avoid.[1,29] However, experimental studies clearly demonstrate that this is not so, provided the tissues are kept free of inflammation.[2,3] Another well-documented finding is that tooth movement in a vestibular direction is associated with simultaneous formation of compensatory bone layers on the periosteal side[1,7,29] and that there are indications that the force level may not be an important variable in this respect.[7]

A more likely explanation for the variation in formation of bone dehiscences may be that the biologic structure represents a limit for how much the teeth can be moved with concomitant bony apposition on the periosteal side. The mechanism may be that the vestibular tissue eventually becomes too thin for the osteogenic progenitor cells to form new bone. This theory is supported by the fact that part of the bone dehiscences produced during excessive expansion may repair if the teeth are moved back to normal labiolingual positions because of an increase in the crestal bone height and a reduction of the elongated supracrestal connective tissue attachment.[12] Similarly, experimental studies in monkeys have shown that the root apices can be moved through the cortical plate, despite the use of biologic force systems, and that the perforation site repaired completely with the movement of the roots back to the normal position.[30]

The teeth with photographic evidence of recession at time of appliance removal experienced only a small increase in clinical crown height during the relatively long posttreatment period, which ranged from 1.7 to 14.8 years (Figs 4 and 5). This finding supports a previous study[19] that concluded that gingival recession that is associated with orthodontic expansion is not progressive. It should be stressed, however, that any abrasion of the IEs may have caused an underestimation of the gingival retraction. The fact that no bone dehiscences were found at follow-up may suggest that the process stabilizes once a normal distance between attachment level and bone level is reestablished. Another finding of clinical interest was that few new teeth developed pronounced recession during the follow-up period and only in patients with other teeth that experienced recession during appliance therapy (Fig 4). Our results may therefore suggest that adolescent patients without signs of recession during orthodontic expansion and finishing are not likely to develop recession of more than 0.5 mm during the first years after appliance removal.

Recessions in young healthy individuals with good oral hygiene are frequently accompanied by hard tissue lesions in the cervical area, shallow pocket depths with occasional clefts or festoons in the GM, and low plaque scores.[23] Such findings suggest that overzealous tooth brushing may be an important etiologic factor.[23] However, recession may also be a response to plaque-associated periodontal destruction. The resistance to such destruction may not be reduced in areas with long supracrestal connective tissue attachment,[16] but the attachment loss is likely to be recession rather than pocketing if the tissues are thin and without cortical bone.[17,18] The patients in our sample did not demonstrate hard and soft tissue lesions in the cervical area, and we did not observe any apparent differences in plaque score or pocket depth between areas with and without recession. Also, few patients had poor oral hygiene at follow-up, and obvious signs of gingival inflammation were not observed in any of the areas examined. A likely reason for the recessions in our sample may therefore be that areas with long supracrestal connective tissue attachment offer reduced resistance to normal tooth-brushing procedures.

CONCLUSION

Adolescent orthodontic patients with dentoalveolar retrusion may be treated with pronounced advancement of the mandibular incisors without increasing the risk of recession. Recession that occurs during the period of active treatment is not progressive. However, patients in whom recession develops on individual teeth during appliance therapy are at risk of experiencing recession on other teeth after treatment.

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