



Stability of anterior open bite nonextraction treatment in the permanent dentition

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This study cephalometrically evaluated the long-term stability of anterior open bite nonextraction treatment in the permanent dentition after a mean period of 5 years. The experimental group consisted of 21 patients who had undergone orthodontic treatment with fixed appliances from whom cephalometric headfilms were obtained at the pretreatment, posttreatment, and postretention stages. Two control groups were used. The first, with ages comparable with the experimental group before treatment, was used only to characterize it. The second control group, with normal occlusion, was longitudinally followed for a period comparable with the postretention period and was used to compare the changes between groups during this period. The differences between the observation stages in the experimental group were analyzed with paired *t* tests, and the postretention changes were compared with the changes of the second control group with independent *t* tests. A statistically significant decrease of the obtained anterior overbite was demonstrated at the end of the postretention period. The primary factor that contributed to the overbite decrease was the smaller vertical development of the maxillary and mandibular incisors in the postretention period. Neither the pretreatment anterior open bite amount nor the magnitude of correction was associated with the long-term overbite decrease. However, 61.9% of the sample had a clinically stable open bite correction. (*Am J Orthod Dentofacial Orthop* 2003;124:265-76)

Facial disharmonies in the vertical plane, including the anterior open bite, are great challenges to orthodontists because of the remarkable difficulties of treatment and the instability of the correction, depending on its severity, etiology, and the stage of treatment onset.^{1,2} Early treatment of this malocclusion, during the deciduous or mixed dentition, usually provides the best results with the least relapse,³⁻⁶ probably because spontaneous correction of the open bite in the early ages might be part of the developmental process.^{7,8} Failure to respond successfully to early correction approaches might occur in patients with open bites consequent to Down syndrome or other hereditary or

congenital abnormalities.^{9,10} On the other hand, during the permanent dentition, depending on the severity of skeletal involvement, orthognathic surgery might play an important role when combined with orthodontic treatment to provide stability for correcting this malocclusion.¹¹⁻¹³

In 1985, Lopez-Gavito et al¹⁴ studied the long-term stability of anterior open bite treatment in the permanent dentition and concluded that approximately 35% of patients had more than 3 mm of relapse. Katsaros and Berg¹⁵ reported stability of anterior open bite treatment in 75% of the evaluated patients at least 1 year posttreatment. Incomplete adaptation of tongue posture might lead to a tendency for return of open bite after treatment,¹⁶⁻¹⁸ although tongue and lip pressures during function (eg, swallowing, speaking, and chewing) are relatively unimportant as determinants in malocclusion.¹⁸⁻²⁰ Therefore, myofunctional therapy is usually advocated after treatment to help in eliminating the abnormal tongue posture.^{3,10,21,22} In 1987, Kim²³ described the multiloop edgewise archwire technique (MEAW) and stated that great stability of anterior open bite therapy might be achieved. Similarly, Küçükköles et al²⁴ used nickel-titanium wires in that technique and concluded that this mechanotherapy provides results that are stable and similar to those observed by Kim.²³

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Currently, though some studies^{3,15,25} report successful treatment of this malocclusion with minor relapses, the literature on stability of anterior open bite treatment during the permanent dentition is still scarce. The few studies that do focus on this issue have deficiencies, such as short follow-up periods after treatment,^{15,24,26} small sample sizes or a single clinical case,²⁷⁻³⁰ or absence of differentiation between extraction and nonextraction therapies.^{5,14,26}

In this study, we evaluated the stability of anterior open bite nonextraction treatment in the permanent dentition, after a mean period of 5 years (range, 3.08-9.33), and also clinically significant relapse and stability.

MATERIAL AND METHODS

The experimental group consisted of 21 subjects (16 girls, 5 boys) with a mean pretreatment age of 12.4 years (range, 10.8-16.3), drawn from the files of the Orthodontic Department at Bauru Dental School, University of São Paulo, Brazil. The primary selection criterion for this group was an anterior open bite of at least 1 mm. Additional criteria were all maxillary and mandibular teeth up to the second molars and nonextraction treatment with the edgewise appliance combined with anterior vertical elastics. Initially, the preselected sample consisted of 22 patients, but 1 patient with a Class II malocclusion was omitted to produce a more uniform group (only patients with Class I malocclusions).

Because of the inherent difficulties in finding a suitable longitudinal control group for this type of study, 2 control groups were used. For control group 1, the data from 18 girls, mean age 12 years, with randomized occlusions and without previous orthodontic treatment, were used only to characterize the experimental group at the pretreatment stage. Control group 2 comprised 21 subjects (9 boys, 12 girls) with normal occlusion and an initial mean age of 14.6 years (comparable to the experimental group at the posttreatment stage). These groups were selected from the longitudinal growth study sample of the orthodontic department.

Treatment was conducted with the standard edgewise technique, which is characterized by the use of 0.022 × 0.028-in conventional brackets. For leveling and alignment, the usual wire sequence begins with a 0.015-in twist-flex or a 0.016-in nickel-titanium wire, followed by 0.016, 0.018, and 0.020-in stainless steel round wires. Detailing of tooth position and the finishing procedures were accomplished by either 0.019 × 0.025-in or 0.021 × 0.025-in rectangular wires and 0.018-in round wires, respectively. Intermaxillary elastics (3/16 in) were used to close the anterior open bite

during an average of 4.4 months (range, 15 days–1 year, SD = 3.45 months). No additional auxiliaries were used to control the vertical dimension. Thirteen patients underwent maxillary expansion either with hyrax or Haas appliances to correct posterior crossbites or to provide space in the maxillary arch. After the active treatment period, a Hawley retainer was used in the maxillary arch and a bonded 3 × 3 retainer in the mandibular arch. Myofunctional therapy was recommended to correct tongue posture and function. The mean times were 2.4 years between pretreatment (T1) and immediately after treatment (T2), and 5 years between T2 and follow-up (T3).

Lateral cephalograms of the experimental group were obtained from each subject at T1, T2, and T3—after a mean follow-up period of 5 years (range, 3.08-9.33). Because of the long time between evaluation stages, lateral headfilms were obtained with various radiograph machines that produced different magnification factors of the images, between 6% and 10.94%.

The cephalometric tracings and landmark identifications were performed on acetate paper by a single investigator (F.P.V.) and then digitized with a DT-11 digitizer (Houston Instruments, Austin, Tex) (Figs 1 and 2). These data were stored on a computer and analyzed with Dentofacial Planner 7.0 (Dentofacial Planner Software, Toronto, Ontario, Canada), which corrected the image magnification factors of the groups.

Because root resorption is usually a concern when open bite malocclusions are treated,³¹⁻³³ a complementary evaluation of root resorption was undertaken to study the effects on the roots of the mechanotherapy used. Root morphology of the maxillary and mandibular central and lateral incisors was evaluated before and after treatment, according to the method of Levander and Malmgren,³¹ in periapical radiographs.

Twenty randomly selected radiographs were retraced, redigitized, and remeasured by the same examiner. The casual error was calculated according to Dahlberg's formula ($Se^2 = \Sigma d^2/2n$),³⁴ and the systematic error with dependent *t* tests, for $P < .05$.³⁵⁻³⁷

To apply the *t* test, a normal distribution of the samples is necessary. This was verified with the Kolmogorov-Smirnov test. Results of this test demonstrated that all variables were normally distributed. Therefore, the *t* test was used to compare the experimental group at T1 with control group 1, and changes during the postretention period (T3 – T2) with the changes during the comparable period for control group 2. Comparisons of changes in variables during the treatment period (T2 – T1) and the postretention

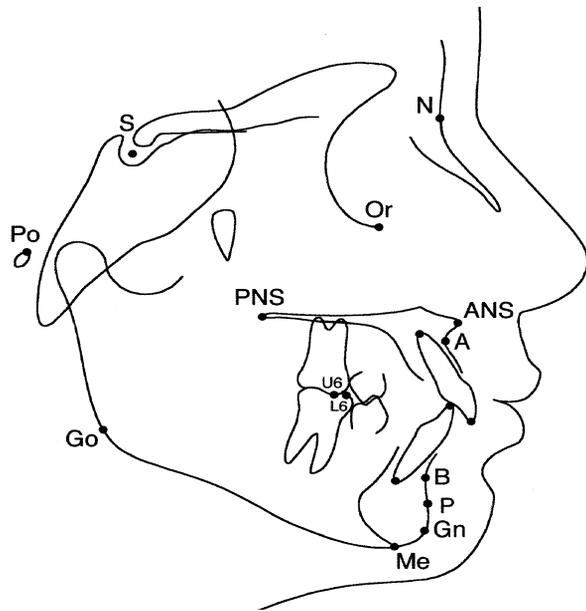


Fig 1. Cephalometric landmarks used on lateral tracings. S, sella turcica; N, nasion; Po, porion; P, pogonion; Or, orbitale; ANS, anterior nasal spine; PNS, posterior nasal spine; A, subspinale; B, supramentale; U6, point located on mesial cusp of maxillary first molar; L6, point located on mesial cusp of mandibular first molar; Go, gonion; Gn, gnathion; Me, menton; points located on apex and incisal edges of maxillary and mandibular incisors.

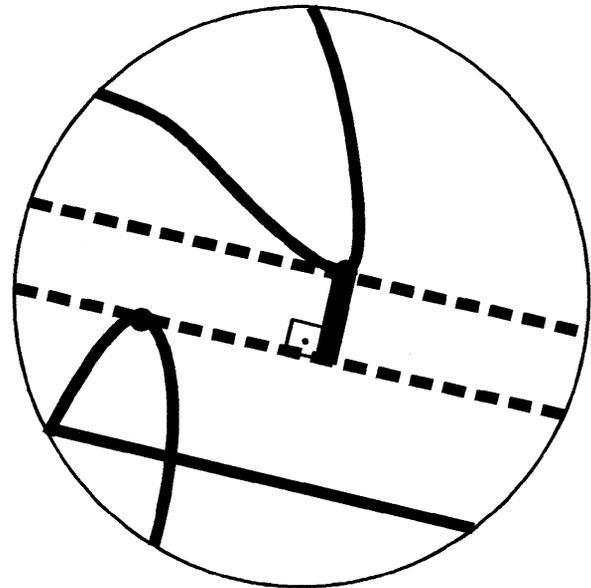


Fig 2. Overbite measurement (magnified). Overbite: distance between incisal edges of maxillary and mandibular central incisors, perpendicular to occlusal plane.

period (T3 – T2) in the experimental group were made with paired *t* tests.

Pearson correlation coefficients were calculated to determine the relationships between anterior overbite changes in the postretention period and the following: initial severity of the open bite, amount of correction achieved through orthodontic treatment, and postretention changes in all variables. Results were regarded as significant for $P < .05$. These analyses were performed with Statistica for Windows 4.3B (Statsoft, Tulsa, Okla).

A clinically significant relapse of anterior open bite was defined as a negative overbite between the maxillary and mandibular incisors at T3. Therefore, to establish a clinical parameter as to the probability of open bite correction stability, the percentages of patients with and without clinically significant relapses were calculated from the total number of studied patients.

RESULTS

Among the 30 variables, only 5 had a systematic error: SN.OP, LAFH, U1-NA, U1-PP, and L1-NB (see

Table I for definitions of planes and all dental and skeletal landmark abbreviations used in the text). The casual errors were between 0.19 (overjet) and 2.39 (SN.OP).

Results are shown in Tables II through VII. There was a statistically significant negative correlation between the anterior overbite decrease and the increase in dentoalveolar height of the mandibular molar in the postretention period ($r = -.53$, $P = .01$). No other variable change in the postretention period showed a statistically significant correlation with anterior overbite decrease.

No patients had root involvement (grade 0) at T1. At T2, 14 patients (66.7%) had apical blunting (grade 1), and 7 (33.3%) had moderate resorption (grade 2).

Eight patients (38.1%) had a clinically significant relapse of open bite, and 13 (61.9%) did not. Therefore, 61.9% had a clinically stable long-term correction of open bite in the permanent dentition.

DISCUSSION

Pretreatment sample characteristics

Most studies of the stability of anterior open bite correction have not been concerned with dividing the experimental groups into patients treated with or without extraction.^{14,24,26} However, it has been suggested that extraction treatment of the anterior open bite might provide more stable results.³⁸ Therefore, to give a more accurate result regarding the stability

Table I. Definition of some planes and abbreviations for dental and skeletal variables used

Planes	
Frankfort plane:	constructed from Po to Or
Palatal plane:	constructed from ANS to PNS
Functional occlusal plane:	a plane drawn through points of occlusal contact between first permanent molars and first and second premolars ⁵³⁻⁵⁵
Mandibular plane 1:	constructed from Go to Me
Mandibular plane 2:	constructed from Go to Gn
Dental cephalometric variables	
U1.PP:	maxillary incisor long axis to palatal plane angle
U1.NA:	maxillary incisor long axis to NA angle
U1-NA:	distance between most anterior point of crown of maxillary incisor and NA line
U1-PP:	perpendicular distance between incisal edge of maxillary central incisor and palatal plane
U6-PP:	perpendicular distance between mesial cusp of maxillary first molar and palatal plane
L1.NB:	mandibular incisor long axis to NB angle
L1-NB:	distance between most anterior point of crown of mandibular incisor and NB line
IMPA:	incisor mandibular plane angle
L1-MP:	perpendicular distance between incisal edge of mandibular incisor and mandibular plane
L6-MP:	perpendicular distance between mesial cusp of mandibular first molar and mandibular plane
Overbite:	distance between incisal edges of maxillary and mandibular central incisors, perpendicular to occlusal plane (also magnified in Fig 2)
Overjet:	distance between incisal edges of maxillary and mandibular central incisors, parallel to occlusal plane
M.REL. (molar relationship):	distance between mesial cusps of maxillary and mandibular first molars, parallel to functional occlusal plane
Skeletal cephalometric variables	
SNA:	SN to NA angle
Co-A:	condylion to A point distance
A-Nperp:	A point to nasion-perpendicular
SNB:	SN to NB angle
Co-Gn:	condylion to gnathion distance
Go-Gn:	gonion to gnathion distance
Co-Go:	condylion to gonion distance
Co.Go.Me:	ascending ramus to mandibular body angle
P-Nperp:	pogonion to nasion-perpendicular
ANB:	NA to NB angle
Wits:	distance between perpendicular projections of A and B points on functional occlusal plane
Co-A/Co-Gn:	proportion between maxillary and mandibular lengths
FMA:	Frankfort mandibular plane angle
SN.GoGn:	SN to GoGn angle
LAFH (lower anterior face height):	distance from anterior nasal spine to menton
SN.PP:	SN to palatal plane angle
SN.OP:	SN to functional occlusal plane angle

of nonextraction open bite treatment, our sample included only patients who had this type of therapy.

Studies of the stability of open bite correction were carried out 1 to 2 years after treatment, at most.^{24,26} Only Lopez-Gavito et al¹⁴ reported changes after a minimum of 9.6 years after treatment. Consequently, a primary requirement in our sample selection was that patients had completed treatment at least 3 years earlier. The average postretention observation period was 5 years. This is very important, because vertical changes might be observed up to 5 years after treatment, especially in growing patients.¹⁴

To characterize the experimental group at T1, it was compared with a control group of female subjects (Table II). Evidently, this was not ideal.

However, with only 5 male patients in the experimental group, their influence on the growth pattern variables could be regarded as minimal, because only SN.PP showed a statistically significant difference between the sexes at age 12, as reported in the literature.³⁹ In general, the initial cephalometric characteristics of the experimental group showed only a mild vertical growth tendency, slightly diverging from the literature that reports a stronger predominance of this growth pattern.^{14,40} This was probably because previous studies included patients treated without and with extractions.^{14,24,26} Usually, open-bite patients treated with extraction have a more severe vertical pattern, whereas those treated nonextraction have milder patterns.^{14,24,26}

Table II. Results of *t* test between experimental group at pretreatment stage (T1) and control group 1

Variables	Pretreatment stage (T1)		Control group 1		P
	Mean	SD	Mean	SD	
Maxillary component					
SNA (°)	80.30	3.91	81.1	3.5	.508674
A-Nperp (mm)	-0.12	2.90	-1.1	3.2	.502581
Co-A* (mm)	82.16	4.98	86.9	4.0	.000397
Mandibular component					
SNB (°)	77.44	3.63	78.9	3.1	.176183
P-Nperp (mm)	-3.73	6.09	-4.5	3.0	.622248
Co-Go (mm)	50.98	5.43	53.1	4.2	.183674
Go-Gn (mm)	73.07	4.73	74.2	4.1	.435285
Co-Gn (mm)	110.62	5.96	113.7	5.3	.091286
Co.Go.Me (mm)	128.36	6.40			
Maxillomandibular relationship					
ANB (°)	2.87	2.32	2.2	2.4	.385746
Co-A/Co-Gn (%)	74.29	3.01			
Wits (mm)	-1.94	2.88			
Growth pattern					
FMA (°)	29.78	5.83	27.7	2.9	.172385
SN.PP (°)	7.46	2.33	9.3	3.4	.065428
SN.OP (°)	26.84	6.37	27.5	4.8	.720658
LAFH (mm)	67.75	5.28	64.9	5.8	.114635
SN.GoGn* (°)	36.94	5.66	33.2	5.0	.034681
Maxillary dentoalveolar component					
U1.NA (°)	28.40	4.28	26.1	5.6	.156451
U1-NA* (mm)	5.98	1.89	4.6	1.7	.022986
U1.PP (°)	116.15	6.13	116.4	5.0	.893548
U1-PP (mm)	26.51	2.62	26.9	3.0	.678991
U6-PP (mm)	23.26	2.51	22.2	2.3	.187227
Mandibular dentoalveolar component					
L1.NB (°)	28.25	6.64	26.8	6.3	.495573
L1-NB (mm)	5.45	2.21	4.7	3.0	.372651
IMPA (°)	91.43	7.76	92.4	6.6	.676951
L1-MP (mm)	38.26	2.93	40.0	3.5	.095497
L6-MP* (mm)	29.61	3.26	31.8	2.9	.033671
Dental relationships					
Overjet (mm)	4.43	2.60			
Overbite (mm)	-1.75	0.66			
M.REL. (mm)	0.06	0.36			

*Statistically significant.

Table III. Means and standard deviations of overbite for 3 evaluated stages of experimental group

Stages	Overbite (mm)			
	Mean	Minimum	Maximum	SD
Pretreatment	-1.75	-1.00	-4.10	0.66
Posttreatment	1.43	0.40	2.50	0.50
Postretention	0.07	1.20	-1.40	0.62

Treatment and postretention changes

Overbite changes during the postretention period—the main focus of this study—were compared with control group 2 (Table VI). Subsequently, changes in

overbite and the other variables, during either the treatment or the postretention period, were analyzed to assess whether they could explain the changes in overbite during the postretention period.

There was a statistically significant decrease of anterior overbite in the postretention period. Not only did overbite decrease in the experimental group, but also this decrease was statistically larger than that observed in control group 2; this is an abnormal change of this variable (Tables III and VI). This result contrasts with those of other studies, which have found stability of the obtained anterior overbite in the permanent dentition.^{26,28} However, these reports did not distinguish between patients treated with and without extrac-

Table IV. Results of paired *t* test between pretreatment and posttreatment stages of experimental group (treatment changes, T2 – T1)

Variables	Pretreatment stage (T1)		Posttreatment stage (T2)		Difference	P
	Mean	SD	Mean	SD		
Maxillary component						
SNA (°)	80.30	3.91	80.62	3.49	0.32	.387630
A-Nperp (mm)	-0.12	2.90	-0.10	3.37	0.01	.961943
Co-A (mm)	82.16	4.98	83.00	5.10	0.83	.084448
Mandibular component						
SNB* (°)	77.44	3.63	78.00	3.50	0.56	.040182
P-Nperp (mm)	-3.73	6.09	-3.08	7.35	0.65	.301902
Co-Go* (mm)	50.98	5.43	53.55	4.84	2.56	.002233
Go-Gn* (mm)	73.07	4.73	74.83	5.12	1.76	.012910
Co-Gn* (mm)	110.62	5.96	113.89	6.34	3.27	.000004
Co.Go.Me* (°)	128.36	6.40	127.03	6.19	-1.32	.011189
Maxillomandibular relationship						
ANB (°)	2.87	2.32	2.60	2.05	-0.27	.395539
Co-A/Co-Gn* (%)	74.29	3.01	72.91	3.03	-1.37	.009406
Wits* (mm)	-1.94	2.88	-0.71	2.46	1.22	.026182
Growth pattern						
FMA (°)	29.78	5.83	29.49	5.97	-0.29	.474763
SN.PP (°)	7.46	2.33	7.23	2.24	-0.22	.518752
SN.PO* (°)	16.84	6.37	13.92	4.47	-2.91	.020992
LAFH* (mm)	67.75	5.28	70.20	5.16	2.45	.000314
SN.GoGn (°)	36.94	5.66	36.43	5.36	-0.50	.217737
Maxillary dentoalveolar component						
U1.NA* (°)	28.40	4.28	24.22	4.99	-4.17	.000790
U1-NA (mm)	5.98	1.89	5.30	1.87	-0.68	.169811
U1.PP* (°)	116.15	6.13	112.08	5.55	-4.07	.000821
U1-PP* (mm)	26.51	2.62	29.33	2.64	2.81	.000000
U6-PP* (mm)	23.26	2.51	24.76	2.53	1.50	.000570
Mandibular dentoalveolar component						
L1.NB (°)	28.25	6.64	27.13	5.54	-1.12	.304785
L1-NB (mm)	5.45	2.21	6.04	2.08	0.59	.104591
IMPA (°)	91.43	7.76	90.32	6.56	-1.11	.311171
L1-MP* (mm)	38.26	2.93	40.97	2.74	2.70	.000000
L6-MP* (mm)	29.61	3.26	30.91	2.63	1.30	.000214
Dental relationships						
Overjet* (mm)	4.43	2.60	2.80	1.01	-1.62	.000958
Overbite* (mm)	-1.75	0.66	1.43	0.50	3.19	.000000
M.REL.* (mm)	0.06	0.36	-0.15	0.49	-0.21	.048677

*Statistically significant.

tions;^{14,26} they were isolated clinical case reports²⁸ or had insufficient observation periods, at most 2 years after treatment, to evaluate stability.^{26,38}

Changes in the maxillary component do not seem to have played an important role in the overbite change, because they were not statistically significant during treatment, during the 5-year period after treatment, or in relation to the changes in control group 2 (Tables IV-VI).

Some significant changes were observed regarding the mandibular component during the treatment period (T2 – T1) and in the postretention period (T3 – T2) (Tables IV and V). Mandibular size (Co-Go and Co-Gn) showed statistically significant changes in these

periods, although they were significantly smaller in the posttreatment period (T3 – T2) compared with the normal changes of control group 2 (Table VI). However, because treatment of the open bite is usually not intended to produce growth changes in these structures, they might be regarded as the result of the intrinsic growth of these patients. It is very unlikely that these changes are related to the observed significant decrease of the anterior overbite.^{14,24,26} Additionally, there was no correlation between the decrease of anterior overbite and the changes in these variables during the 5-year postretention period.

Changes in the maxillomandibular relationship in the postretention period were statistically different from

Table V. Result of paired *t* test between posttreatment and postretention stages of experimental group (postretention changes, T3 – T2)

Variables	Posttreatment stage (T2)		Postretention stage (T3)		Difference	P
	Mean	SD	Mean	SD		
Maxillary component						
SNA (°)	80.62	3.49	81.41	3.57	0.78	.054852
A-Nperp (mm)	-0.10	3.37	0.26	3.02	0.36	.427163
Co-A (mm)	83.00	5.10	83.50	5.57	0.51	.212953
Mandibular component						
SNB (°)	78.00	3.50	78.49	4.30	0.48	.071950
P-Nperp (mm)	-3.08	7.35	-2.84	7.83	0.23	.726767
Co-Go* (mm)	53.55	4.84	55.01	6.06	1.46	.047961
Go-Gn (mm)	74.83	5.12	75.43	4.99	0.59	.210799
Co-Gn* (mm)	113.89	6.34	115.01	7.12	1.12	.018849
Co.Go.Me (°)	127.03	6.19	126.18	5.93	-0.84	.105657
Maxillomandibular relationship						
ANB (°)	2.60	2.05	2.93	2.12	0.33	.247511
Co-A/Co-Gn (%)	72.91	3.03	72.64	3.02	-0.27	.407078
Wits (mm)	-0.71	2.46	-0.75	2.79	-0.03	.909942
Growth pattern						
FMA (°)	29.49	5.97	29.27	6.33	-0.21	.606464
SN.PP (°)	7.23	2.24	6.76	2.60	-0.47	.158152
SN.OP (°)	13.92	4.47	13.93	6.77	0.00	.996897
LAFH* (mm)	70.20	5.16	71.69	5.67	1.48	.000681
SN.GoGn* (°)	36.43	5.36	35.41	6.55	-1.02	.022998
Maxillary dentoalveolar component						
U1.NA (°)	24.22	4.99	24.71	4.36	0.48	.474029
U1-NA (mm)	5.30	1.87	5.22	1.91	-0.07	.859075
U1.PP (°)	112.08	5.55	112.88	5.57	0.79	.115308
U1-PP (mm)	29.33	2.64	29.19	2.93	-0.14	.566872
U6-PP* (mm)	24.76	2.53	25.39	2.58	0.63	.033761
Mandibular dentoalveolar component						
L1.NB (°)	27.13	5.54	28.57	6.74	1.44	.073835
L1-NB* (mm)	6.04	2.08	6.53	2.36	0.49	.002788
IMPA (°)	90.32	6.56	91.93	8.15	1.61	.051980
L1-MP (mm)	40.97	2.74	41.31	2.79	0.33	.153841
L6-MP* (mm)	30.91	2.63	31.91	3.06	1.00	.000175
Dental relationships						
Overjet (mm)	2.80	1.01	2.71	1.20	-0.09	.597177
Overbite* (mm)	1.43	0.50	0.07	0.62	-1.36	.000000
M.REL. (mm)	-0.15	0.49	-0.21	0.43	-0.06	.691506

*Statistically significant.

those in control group 2 for ANB (Table VI). It increased by 0.33° in the experimental group and by 0.60° in control group 2. This increase might be explained by a clockwise mandibular rotation after treatment, which in turn might contribute to anterior open bite relapse.^{14,24,26} However, these changes were very small to have significantly influenced the decrease of the anterior overbite.

During treatment, there was a significant decrease in the SN.OP angle and an increase in LAFH. The SN.OP angle showed a reduction, probably due to the extrusion of the mandibular incisors during the open bite correction. The increase in LAFH is usually expected during orthodontic treatment. After treatment,

the SN.OP angle did not show a decrease similar to the control group, probably because of relapse of the mandibular incisor extrusion, discussed below. On the other hand, LAFH continued to increase in the normal pattern (Tables IV-VI). Therefore, changes in growth pattern consisted of unfavorable changes at the dentoalveolar structures; these can contribute to a decrease of the anterior overbite.

Results from the treatment period (T2 – T1) indicated that correction of anterior open bite was possible primarily due to the extrusion and uprighting of the maxillary incisors and extrusion of the mandibular incisors, as demonstrated by the U1-PP, U1.PP, and L1-MP variables, which had statistically significant

Table VI. Results of *t* test between changes during postretention period of experimental group (T3 – T2) and changes in control group 2 during comparable period

Variables	Postretention period (T3 – T2)		Control group 2		P
	Mean	SD	Mean	SD	
Maxillary component					
SNA (°)	0.78	1.76	0.30	1.24	.318674
A-Nperp (mm)	0.36	2.07	–0.38	1.32	.169102
CoA (mm)	0.50	1.81	1.70	2.14	.059408
Mandibular component					
SNB (°)	0.48	1.16	0.90	1.32	.282986
P-Nperp (mm)	0.23	3.01	0.60	2.75	.675541
Co-Go* (mm)	1.46	3.17	3.74	2.66	.015788
Go-Gn* (mm)	0.59	2.10	1.90	1.88	.040653
Co-Gn* (mm)	1.12	2.01	4.28	3.12	.000361
Co.Go.Me (°)	–0.84	2.29	–1.24	1.84	.536641
Maxillomandibular relationship					
ANB* (°)	0.33	1.28	–0.60	0.95	.010413
CoA/CoGn* (%)	–0.27	1.46	–1.30	1.00	.010847
Wits (mm)	–0.03	1.52	0.59	2.03	.264427
Growth pattern					
FMA (°)	–0.21	1.87	–1.04	1.62	.134349
SN.PP (°)	–0.47	1.47	–0.69	1.60	.647633
SN.OP* (°)	0.00	5.54	–3.44	3.99	.026025
LAFH (mm)	1.48	1.69	2.43	2.30	.132659
SN.GoGn (°)	–1.02	1.90	–1.64	2.04	.312912
Maxillary dentoalveolar component					
U1.NA (°)	0.48	3.05	0.57	2.28	.913896
U1-NA (mm)	–0.07	1.81	0.78	0.98	.065186
U1.PP (°)	0.79	2.21	0.19	2.08	.371767
U1-PP* (mm)	–0.14	1.16	0.83	0.79	.002599
U6-PP* (mm)	0.63	1.27	1.90	1.31	.002791
Mandibular dentoalveolar component					
L1.NB (°)	1.44	3.50	–0.04	2.45	.119332
L1-NB* (mm)	0.49	0.65	0.08	0.63	.047645
IMPA (°)	1.61	3.57	0.44	2.43	.096548
L1-MP* (mm)	0.33	1.04	1.27	1.12	.007845
L6-MP (mm)	1.00	0.99	1.38	1.57	.347919
Dental relationships					
Overjet (mm)	–0.09	0.81	–0.08	0.43	.962408
Overbite* (mm)	–1.36	0.54	–0.31	0.83	.000022
M.REL. (mm)	–0.06	0.70	–0.28	1.06	.436653

*Statistically significant.

Table VII. Analysis of correlation between decrease of anterior overbite in postretention period (overbite T3 – T2) and initial severity of anterior open bite (overbite T1) and amount of correction achieved through treatment (overbite T2 – T1)

Variables	Overbite T3 – T2 <i>r</i>	P
Overbite T2 – T1	–.09	.695372
Overbite T1	–.21	.357618

changes during treatment—changes that were also observed by most other authors.^{14,24,26-28} Maxillary molar dentoalveolar height showed a statistically sig-

nificant increase (1.5 mm) during orthodontic treatment (T2 – T1) and the postretention period (T3 – T2). However, its vertical development in the latter period was significantly smaller than in control group 2 (Tables IV-VI). It could be speculated that the significant increase in maxillary molar dentoalveolar height during treatment could be a consequence of the rapid palatal expansion that 13 patients had. However, the extrusion of the maxillary posterior teeth that usually occurs after rapid palatal expansion is noticeable only immediately after the procedure. At the end of the complete fixed appliance treatment time, extrusion of these teeth is similar to that of the control group.⁴¹⁻⁴³ In this study, because no control group was used to

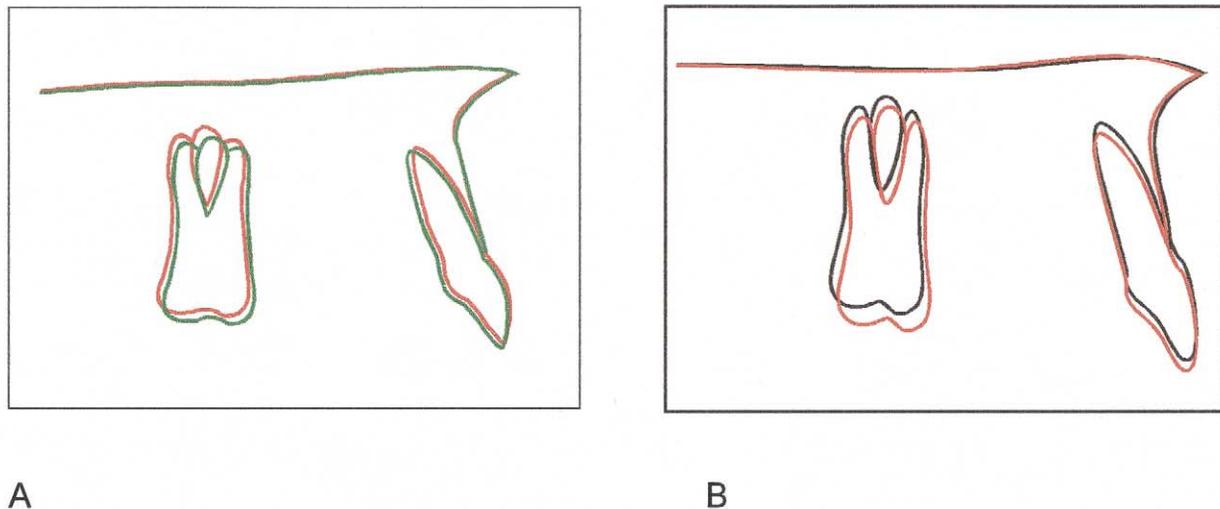


Fig 3. A, Superimposition on palatal plane, registered on anterior nasal spine, of posttreatment (*red*) and postretention (*green*) mean tracings of experimental group; **B**, same superimposition of initial (*black*) and final (*red*) mean tracings of control group 2, which compare with stages of experimental group shown in **A**.

compare the changes during treatment, it cannot be determined whether the significant increase in maxillary molar dentoalveolar height was a direct consequence of the mechanotherapy or of normal growth. Therefore, even though further investigation is needed, it is unlikely that the rapid palatal expansion in these 13 patients played an important role in the observed changes. Although the maxillary incisors demonstrated a statistically significant vertical development during orthodontic therapy, this was not observed during the postretention period (Tables IV and V). During the postretention period, the vertical development of these teeth was smaller than in control group 2 (Fig 3 and Table VI). Whereas the smaller vertical development of the maxillary molars is a favorable factor for stability of open bite correction, this is not true for the maxillary incisor. The observed tendency is that variables that demonstrated the greatest influence during treatment will have smaller changes after treatment. Some will be favorable, and some will not.^{44,45}

A similar behavior was observed for the mandibular teeth. Vertical development of the mandibular molars was statistically significant during the treatment (T2 – T1) and postretention (T3 – T2) periods (Tables IV and V). The mandibular incisors showed a statistically significant vertical development only during treatment. During the postretention period, only the vertical development of the mandibular incisors was statistically smaller than in control group 2 (Fig 4 and Table VI). Although the vertical development of the mandibular

molars was smaller than that of control group 2, the difference was not statistically significant. This demonstrates another factor contributing to open bite relapse—the smaller vertical development of the mandibular incisors in the postretention period—which directly contributes to overbite decrease. Additionally, although the vertical development of the mandibular molars was similar to that of the control group, it showed a significant negative correlation with decrease of the anterior overbite in the postretention period. Therefore, effective retention appliances to control this vertical development should be used to ensure greater stability of open bite correction. A possible explanation for the smaller vertical development of the maxillary and mandibular incisors in the postretention period in relation to the control group is the lack of tongue adaptation; this causes the tongue to remain positioned between these teeth at rest.¹⁶ To minimize this relapse factor, myofunctional therapy is usually recommended after orthodontic treatment.^{3,10,21,22} The treatment protocol of the experimental sample included myofunctional therapy after treatment. However, because this is a retrospective study, it could not be ascertained from the clinical charts whether all patients followed the recommendations and underwent such therapy. Others recommend 1 to 2 years of crib or sharp spur therapy, hoping to cause the tongue to adapt to its space.^{5,46} Partial glossectomy is another therapy prescribed to improve the stability of open bite therapy. Although some case reports show good short-term results, no

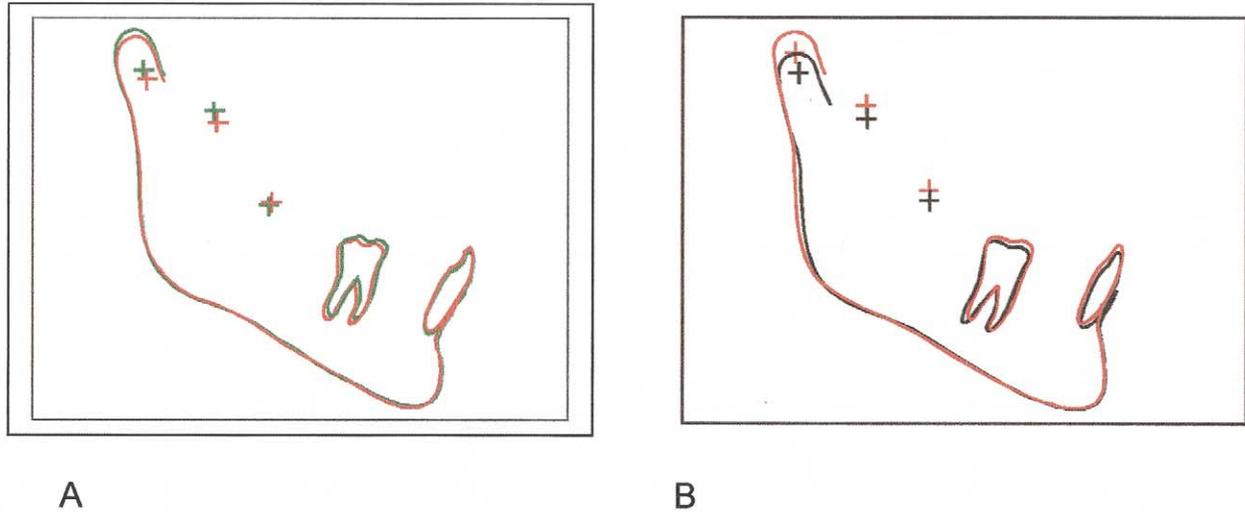


Fig 4. **A**, Superimposition on mandibular plane, registered on symphysis, of posttreatment (*red*) and postretention (*green*) mean tracings of experimental group; **B**, same superimposition of initial (*black*) and final (*red*) mean tracings of control group 2, which compare with stages of experimental group shown in **A**.

long-term data support its general use, especially in light of the potential morbidity.^{47,48}

Only the vertical development of the mandibular molars in the postretention period was inversely correlated to the decrease of anterior overbite. Kim et al²⁶ stated that the remarkable vertical development of the posterior teeth, combined with the changes in the incisors during and after orthodontic treatment, is the main reason for the decrease of anterior overbite, because it causes a clockwise mandibular rotation. Most authors studying treatment stability of anterior open bite agree with this statement.^{14,24} An interesting result was the absence of correlation between the decrease of anterior overbite and the initial open bite severity or the amount of open bite correction (Table VII). Most authors state that the initial open bite severity and the amount of open bite correction play an important role in open bite relapse.^{2,5,27} Perhaps this absence of correlation might be influenced by excluding patients treated with extractions. Preliminary results from a concurrent study that includes only extraction subjects have demonstrated a correlation between open bite relapse and initial open bite severity and amount of open bite correction.

Regarding root resorption at the posttreatment stage, 14 patients (66.7%) showed apical blunting (grade 1), and 7 (33.3%) showed moderate resorption (grade 2). No patient had either accentuated (grade 3) or extreme (grade 4) resorption. These results are similar to other investigations^{32,49-52} in patients with

varying malocclusions, and, therefore, the observed resorption could be considered within normal limits. Consequently, the mechanotherapy used to treat these open bite patients did not produce a greater collateral effect than expected during fixed orthodontic treatment.

Clinically significant relapse and stability

The overbite decrease previously mentioned tells only whether the changes in the overbite between T2 and T3 are statistically significant in the experimental group and in relation to a control group. Although this mathematic jargon can be useful and understandable to the researcher, it does not tell the clinician whether the patients had a negative overbite (that is perceptible by the patient and might be the reason for complaints) again after this period. It also does not give the percentage of patients who might or might not have a negative overbite again in the long term. For these reasons, we evaluated the clinically significant relapse and the stability of open bite treatment. It was observed that only 8 patients had a clinically significant relapse of the open bite, ie, a negative overbite at T3. Consequently, 61.9% of the patients in the experimental group had clinically significant stability of anterior open bite correction in the long term. Lopez-Gavito et al,¹⁴ using a different evaluation method, found that 63.5% of patients had stable open bite corrections in the long term. In addition to the differences in evaluation methodology, their sample combined nonextraction and extraction treatment protocols. This might explain the

slightly greater amount of stability. The use of another evaluation method and combining nonextraction and extraction treatments might also be the reason that Katsaros and Berg¹⁵ found stability in 75% of the patients investigated, although their observation period was rather short. Results from the current study seem to confirm earlier speculation that there is a difference in stability between open bite treatments that involve nonextraction versus extraction approaches.³⁸ The extraction approach seems to be more stable. As previously mentioned, a concomitant study is being undertaken on patients treated only with extractions, and the preliminary results confirm this finding.

CONCLUSIONS

On the basis of the present results and according to the methodology used to evaluate the stability of nonextraction therapy for anterior open bite in the permanent dentition an average of 5 years after treatment, we conclude the following:

1. A statistically significant decrease of the overbite was obtained with treatment.
2. The primary factors contributing to the decrease of anterior overbite were the smaller vertical development of the maxillary and mandibular incisors as compared with the control group and the vertical development of the posterior mandibular teeth during the postretention period.
3. No correlation was found between the decrease of anterior overbite in the postretention period and the initial open bite severity or the amount of open bite correction.
4. There was clinically significant stability of nonextraction open bite treatment in the permanent dentition in 61.9% of the patients.

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