ORIGINAL ARTICLE

Long-term stability of anterior open bite extraction treatment in the permanent dentition

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The purpose of this study was to cephalometrically evaluate the long-term stability of anterior open bite extraction treatment in the permanent dentition after a mean period of 8.35 years. Cephalometric headfilms were obtained at pretreatment, posttreatment, and postretention stages from 31 patients who had undergone orthodontic treatment with fixed appliances. Two control groups were used. The first, with an age similar to that of the experimental group before treatment, was used only to characterize it. The second, with normal occlusion, was followed longitudinally for a period comparable with the posttreatment period and was used to compare changes during this period. The differences between the observation stages in the experimental group were analyzed with paired *t* tests, and the posttreatment changes were compared with the changes of the second control group with independent *t* tests. There was no statistically significant decrease of the obtained anterior overbite at the end of the posttreatment period. The primary factors that contributed to the nonsignificant decrease of the overbite were the normal vertical development of the maxillary and mandibular incisors, the smaller vertical development of the mandibular molars, and the consequent smaller increase in lower anterior face height, as compared with the control group in the long-term posttreatment period. Additionally, 74.2% of the sample had a "clinically stable" open bite correction. (Am J Orthod Dentofacial Orthop 2004;125:78-87)

It is agreed that early treatment of the open bite during the deciduous or mixed dentition usually provides the best results with the least relapse,[1-4] although additional scientific evidence is still needed. A reduction in the tendency for the relapse of early treatment might occur because spontaneous correction of the open bite in the early ages constitutes part of the developmental process.[5,6] Failure to respond successfully to early treatment might occur in patients with open bites consequent to neurologic or pathologic abnormalities.[7-9] Few studies have thoroughly investigated the stability of correction of open bites in the permanent dentition. The studies that focus on this issue have deficiencies, such as short follow-up periods after treatment,[10,11] small sample size or single clinical case reports,[12-15] without differentiation between extraction and nonextraction therapies.[11,16,17] Regarding this last factor, there is speculation of a difference in treatment stability of open bite treatment by nonextraction or extraction approaches.[18] To provide additional information comparing the stability of patients treated with or without extractions, we conducted 2 concomitant studies. This is the second of the studies on stability of open bite treatment during the permanent dentition. The first dealt exclusively with nonextraction treatment stability; this study focuses on extraction treatment stability.[19]

This study evaluated the stability of anterior open bite extraction treatment in the permanent dentition after a mean period of 8.35 years (range, 5.25-23.67). An evaluation of "clinically significant" relapse and stability was also performed.

MATERIAL AND METHODS

The experimental group consisted of 31 patients (23 female, 8 male) with a mean age of 13.22 years in the pretreatment stage, drawn from the files of the orthodontic department at Bauru Dental School, University of São Paulo, Brazil. The primary selection criterion for this

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group was an anterior open bite of at least 1 mm. Additional criteria were that the patients had to have all maxillary and mandibular teeth up to the second molars and that they had undergone extraction treatment with the edgewise appliance, associated with anterior vertical elastics. Sixteen patients had Angle Class I and 15 had Class II malocclusions. Twenty-four were treated with 4 first premolar extractions, 2 were treated with 4 second premolar extractions, 1 was treated with 2 second maxillary premolar and 2 first mandibular premolar extractions, and 4 were treated with 2 maxillary premolar extractions. Seven underwent maxillary expansion either with hyrax or Haas appliances to correct posterior crossbites or to help provide space in the maxillary arch.

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 15 female subjects (mean age, 13 years) with randomized occlusions and without previous orthodontic treatment were used only to characterize the experimental group at the pretreatment stage. Control group 2 consisted of 21 subjects (9 male, 12 female) with normal occlusion and an initial mean age of 14.6 years (comparable with 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, characterized by the use of .022 × .028-in conventional brackets associated with extraoral headgear and lip bumper to reinforce anchorage for the maxillary and mandibular teeth, respectively, when necessary. Extraoral headgear was used either to help in correcting the Class II relationship or to reinforce anchorage. Nineteen patients used high-pull headgear, 10 used cervical-pull headgear, and 2 did not use any headgear. For leveling and alignment, the usual wire sequence began with a .015-in twist-flex or .016-in Nitinol wire, followed by .016-, .018-, and .020-in stainless steel round wires. Anterior retraction and the finishing procedures were accomplished by either .019 × .025 or .021 × .025-in rectangular wires and .018-in round wires, respectively. Intermaxillary elastics ($\frac{3}{16}$ in) were also used to help close the anterior open bite. After the active treatment period, a Hawley retainer was used in the maxillary arch and a bonded 3 × 3 retainer in the mandibular arch. The mean treatment time was 2.46 years between pretreatment (T1) and posttreatment (T2) and 8.35 years between T2 and postretention (T3).

Lateral cephalograms of the experimental group were obtained from each subject at T1, T2, and T3 (mean follow-up period, 8.35 years, range 5.25-23.67).

Table I. Skeletal cephalometric variables

Maxillary
SNA: SN to NA angle
Mandibular
SNB: SN to NB angle
Ar.GoMe: ascending ramus to mandibular body angle
Ar-Go: articulare to gonion distance
Maxillomandibular
ANB: NA to NB angle
Growth pattern
FMA: Frankfort mandibular plane angle
SN.GoGn: SN to GoGn angle
LAFH: Lower anterior face height
SN.PP: SN to palatal plane angle
SN.OP: SN to occlusal plane angle
S-Go: sella to gonion distance (Posterior face height — PFH)

S, Sella; *N*, nasion; *A*, A-point; *B*, B-point; *Me*, menton; *Gn*, gnathion.

Because of the long time between the evaluation stages, the lateral headfilms were obtained with different x-ray machines, which produced different magnification factors of the images, between 6% and 9.8%.

The cephalometric tracings and landmark identifications were performed on acetate by a single investigator (R.T.S.B.) and then digitized with a Numonics AccuGrid XNT, model A30TLF digitizer (Numonics, Montgomeryville, Pa) (Table I, Figs 1 and 2). These data were stored on a Pentium III 450-Mhz computer (Intel, Santa Clara, Calif) and analyzed with Dentofacial Planner 7.0 (Dentofacial Planner Software, Toronto, Ontario, Canada), which corrected the image magnification factors of the groups.

Eighteen randomly selected radiographs were retraced, redigitized, and remeasured by the same examiner. The casual error was calculated according to Dahlberg's formula ($Se^2 = \Sigma d^2/2n$),[20] where S^2 is the error variance and d is the difference between the 2 determinations of the same variable, and the systematic error with dependent t tests, for $P < .05$.[21-24]

To apply the t test, a normal distribution of the samples was necessary. This was verified with the Kolmogorov-Smirnov test. Results of this test showed that all variables had a normal distribution. Therefore, the t test was used for comparison between the experimental group at T1 and control group 1, and between the changes in the posttreatment period (T3 − T2) and the changes during the comparable period for control group 2. Comparisons of the changes in the variables during the treatment period (T2 − T1) and during the posttreatment period (T3 − T2) within the experimental group were conducted with paired t tests. Because 7 patients underwent rapid maxillary expansion, which is thought to affect the open bite correction stability, the overbite in the 3 different stages of these patients was

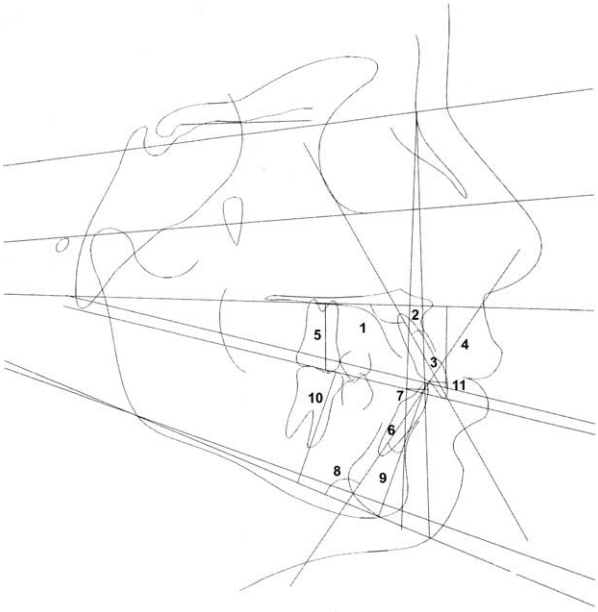

Fig 1. Dental cephalometric variables. Maxillary: *1*, maxillary incisor long axis to palatal plane angle (U1.PP); *2*, maxillary incisor long axis to NA angle (U1.NA); *3*, distance between most anterior point of crown of maxillary incisor and NA line (U1-NA); *4*, perpendicular distance between incisal edge of maxillary central incisor and palatal plane (maxillary incisor dentoalveolar height) (U1-PP); *5*, perpendicular distance between mesial cusp of maxillary first molar and palatal plane (U6-PP). Mandibular: *6*, mandibular incisor long axis to NB line angle (L1.NB); *7*, distance between most anterior point of crown of mandibular incisor and NB line (L1-NB); *8*, incisor mandibular plane angle (IMPA); *9*, perpendicular distance between incisal edge of mandibular incisor and mandibular plane (mandibular incisor dentoalveolar height) (L1-MP); *10*, perpendicular distance between mesial cusp of mandibular first molar and mandibular plane (L6-MP). Maxillomandibular: *11*, overbite: distance between incisal edges of maxillary and mandibular central incisors, perpendicular to functional occlusal plane (also magnified in Fig 2).

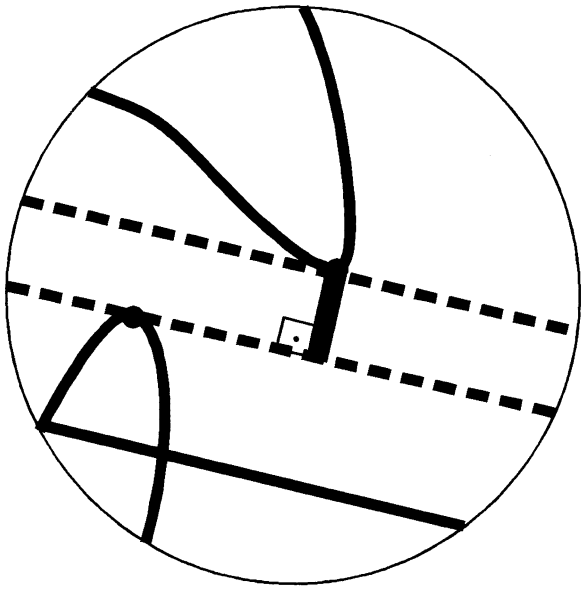

Fig 2. Overbite measurement (magnified). Overbite: distance between incisal edges of maxillary and mandibular central incisors, perpendicular to functional occlusal plane.

calculated separately. The above-mentioned tests conducted for the whole sample were similarly conducted for this subgroup.

Pearson correlation coefficients were calculated to determine the relationship between the anterior overbite changes in the posttreatment period and the following: initial severity of the open bite, amount of correction achieved through orthodontic treatment, and posttreatment changes in all the variables. The results were regarded as significant for $P < .05$. These analyses were performed with Statistica software (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 the open bite correction stability, the percentages of patients with and without a "clinically significant" relapse were calculated from the total number of studied patients.

RESULTS

None of the 22 variables presented a systematic error. The casual errors were between 0.38 (SNB) and 1.46 (U1.NA).

The results are shown in Tables II to VII. There was a statistically significant positive correlation between the changes in overbite during the posttreatment period and the initial amount of overbite and the change in maxillary incisor dentoalveolar height during the posttreatment period. There was a statistically significant negative correlation between the changes in overbite during the posttreatment period and its change during treatment and the change in the occlusal plane inclination in the posttreatment period (Table VII).

Eight patients (25.8%) had a "clinically significant" relapse of the open bite, and 23 (74.2%) did not. Thus, 74.2% presented a "clinically stable" correction of the open bite in the permanent dentition in the long term.

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

	T1		Control group 1		
Variables	*Mean*	*SD*	*Mean*	*SD*	P
Maxillary component					
SNA (°)	80.14	3.90	80.60	3.70	.70
Mandibular component					
SNB (°)	76.38	3.60	77.90	3.40	.15
Ar.GoMe (°)	130.06	4.40	124.20	5.70	.00
Ar-Go (mm)*	40.40	3.90	46.80	3.70	.00
Maxillomandibular relationship					
ANB (°)	3.74	2.10	2.60	1.60	.07
Growth pattern					
FMA (°)	30.55	5.00	27.90	2.90	.06
SN.PP* (°)	7.71	3.30	11.00	2.80	.00
SN.OP (°)	20.63	4.90	18.60	4.10	.18
SN.GoGn (°)*	39.10	4.10	34.90	4.60	.00
LAFH (mm)	69.08	5.20	66.40	4.30	.10
S-Go (mm)*	68.23	4.90	74.60	5.20	.00
Maxillary dentoalveolar component					
U1.NA (°)*	29.55	5.60	22.30	6.80	.00
U1-NA (mm)*	7.57	2.45	4.00	2.40	.00
U1.PP (°)	117.41	6.90	114.10	7.20	.14
U1-PP (mm)	27.43	3.00	28.40	2.80	.28
U6-PP (mm)	23.93	2.40	23.30	2.20	.41
Mandibular dentoalveolar component					
L1.NB (°)	28.17	5.40	26.10	4.10	.21
L1-NB (mm)*	6.52	1.50	4.60	1.30	.00
IMPA (°)	90.64	6.10	92.40	5.40	.33
L1-GoMe (mm)*	38.47	2.70	40.90	2.30	.00
L6-GoMe (mm)*	29.60	2.00	32.50	2.90	.00

*Statistically significant at $P < .05$.

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

	Overbite			
Stages	*Mean*	*Minimum*	*Maximum*	*SD*
Pretreatment	−2.73	−1.00	−8.40	1.80
Posttreatment	1.09	3.20	−0.70	0.94
Postretention	1.02	5.10	−2.60	1.62

DISCUSSION

Pretreatment sample characteristics

For this retrospective study, due to very rigid criteria for sample selection, 31 patients were available; they were selected from the files of the orthodontic department, which include more than 2000 treated patients. The primary criterion for patient selection was an open bite of at least 1 mm. No criteria to differentiate between skeletal and dental open bites were used. Because the open bite malocclusions were treated nonsurgically, it could be expected that the skeletal components would be mild, as was confirmed by the comparison of the experimental group with control group 1 (discussed below) (Table II). This criterion is also similar to that used in other studies of open bite correction stability.[11,16,17] Only Küçükkeles et al[10] used a high-angle skeletal pattern (SN.GoMe > 37°) as an additional criterion to select their sample. Thus, the results of this study apply only to patients with similar characteristics as the investigated group.

The mean observation period of 8.35 years is relevant because previous studies on this subject were conducted at most 1 to 2 years after treatment,[10,11] with 1 exception.[17] This is important because vertical changes might be observed up to 5 years after treatment, especially in growing patients.[17]

The size of our patient group (n = 31) can be considered representative: investigations in the literature do not have large homogeneous experimental groups, and many reports concern individual clinical cases.[12-14,25-30] Others have remarkably diverse samples.[10,16,17]

To characterize the experimental group at the pretreatment stage, it was compared with a control group consisting exclusively of female subjects (Table II). This is not ideal. However, because there were only 8 male patients in the experimental group, their influence on the growth pattern variables could be regarded as minimal: only 3 variables (Ar-Go, U1-PP, S-Go) have a statistically significant difference between sexes at age 13, as reported in the literature.[31] In general, the initial cephalometric characteristics of the experimental group showed a mild vertical tendency in the growth pattern. The mandible also showed vertical characteristics, with a larger gonial angle and smaller ramus height. Confirming the vertical growth tendency, the lower posterior face height was smaller, whereas the lower anterior face height was within normal limits. The maxillary and mandibular incisors were protruded, and only the maxillary incisors were labially tipped. These characteristics have usually been observed in open bite patients.[17,32-35] However, the mandibular incisors and molar dentoalveolar heights were smaller than in control group 1. This was probably because these patients did not have an increased lower anterior face height, which is usually associated with increased dentoalveolar heights.[36]

Control group 2 was comparable with the experimental group, with minor differences. Its mean age at the posttreatment evaluation stage was 14.6 years (15.68 years in the experimental group). The posttreatment observation period for the experimental group was 8.35 years; control group 2 had a mean observation period of 5

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

	T1		*T2*			
Variables	*Mean*	*SD*	*Mean*	*SD*	*Mean difference*	P
Maxillary component						
SNA (°)	80.14	3.97	79.72	3.30	−0.41	.296937
Mandibular component						
SNB (°)	76.38	3.60	76.31	3.52	−0.07	.783886
Ar.GoMe (°)	130.06	4.45	130.02	5.41	−0.03	.947966
Ar-Go (mm)*	40.40	3.92	41.57	4.36	1.16	.008506
Maxillomandibular relationship						
ANB (°)	3.74	2.18	3.41	2.04	−0.32	.390649
Growth pattern						
FMA (°)	30.55	5.05	31.47	5.17	0.91	.177242
SN.PP (°)	7.71	3.30	7.97	3.09	0.25	.557700
SN.OP (°)*	20.63	4.49	17.06	3.93	−3.57	.000001
SN.GoGn (°)	39.10	4.16	39.46	4.32	0.35	.232383
LAFH (mm)*	69.08	5.22	71.39	5.35	2.30	.000449
S-Go (mm)*	68.23	4.94	70.44	5.27	2.20	.000002
Maxillary dentoalveolar component						
U1.NA (°)*	29.55	5.67	23.24	6.33	−6.31	.000138
U1-NA (mm)*	7.57	2.45	4.04	2.91	−3.52	.000002
U1.PP (°)*	117.41	6.98	109.33	11.13	−8.08	.000033
U1-PP (°)*	27.43	3.04	30.00	2.97	2.57	.000001
U6-PP (°)*	23.93	2.47	25.72	2.64	1.78	.000014
Mandibular dentoalveolar component						
L1.NB (°)*	28.17	5.48	23.10	5.10	−5.06	.000000
L1-NB (mm)*	6.52	1.59	5.66	1.35	−0.86	.001147
IMPA (°)*	90.64	6.10	85.20	6.21	−5.44	.000000
L1-GoMe (mm)*	38.47	2.70	40.15	2.54	1.68	.000001
L6-GoMe (mm)*	29.60	2.06	31.42	1.85	1.82	.000000
Dental relationship						
Overbite (mm)*	−2.73	1.80	1.09	0.94	3.83	.000000
Overbite (Exp) (mm)*	−3.32	2.37	1.07	1.18	4.39	.001404

Exp, Expansion patients.
*Statistically significant at $P < .05$.

years. Also, sex distribution was not equal between the groups. However, because the most important variable to be compared was overbite, which does not differ significantly at this age range and between sexes, it was thought that this control group would provide a good comparative parameter. As to the changes in the other variables, we did not expect much difference because, after 19 years of age, the changes with growth are minimal.[37] The use of a normal occlusion control group for this evaluation period seemed more appropriate than an open bite malocclusion group, because, once the malocclusion is corrected, it should have characteristics and behavior similar to normal occlusion. Consequently, it allows a comparison of the corrected overbite changes with time with the behavior of the overbite in a normal occlusion group.

The use of 2 control groups is not ideal. However, because of the difficulties in finding a suitable control group for this type of study, it was decided 2 control groups would be satisfactory, despite the limitations. Control group 1 was used only to characterize the experimental group at the pretreatment stage. Control group 2, used to compare the changes during the main period under study, is the more important control group. The difficulty in finding a suitable control group is evident in the literature: other studies that evaluated the stability of open bite correction did not use a control group.[10,11,16,38] Lopez-Gavito et al[17] used a control group to characterize the experimental group at the pretreatment stage but did not use a control group to compare the posttreatment changes.

Treatment and posttreatment changes

Overbite changes were analyzed primarily during the posttreatment period and compared with control group 2 (Table VI). Subsequently, changes in the overbite during treatment and in the other variables were analyzed to determine whether they could explain

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

	T2		T3			
Variables	*Mean*	*SD*	*Mean*	*SD*	*Mean difference*	P
Maxillary component						
SNA (°)	79.72	3.30	79.38	3.63	−0.33	.293793
Mandibular component						
SNB (°)	76.31	3.52	76.07	3.79	−0.24	.319108
Ar.GoMe (°)*	130.02	5.41	128.16	5.46	−1.86	.003343
Ar-Go (mm)*	41.57	4.36	43.69	3.46	2.11	.001763
Maxillomandibular relationship						
ANB (°)	3.41	2.04	3.30	2.29	−0.11	.707566
Growth pattern						
FMA (°)*	31.47	5.17	30.17	4.59	−1.29	.017777
SN.PP (°)	7.97	3.09	8.10	3.45	0.12	.633317
SN.OP (°)	17.06	3.93	16.44	4.29	−0.61	.228076
SN.GoGn (°)	39.46	4.32	38.90	4.51	−0.56	.144621
LAFH (mm)	71.39	5.35	72.12	5.44	0.73	.092604
S-Go (mm)*	70.44	5.27	72.40	5.19	−1.96	.002685
Maxillary dentoalveolar component						
U1.NA (°)	23.24	6.33	24.41	6.60	1.17	.243711
U1-NA (mm)*	4.04	2.91	5.21	2.62	1.17	.040000
U1.PP (°)	109.33	11.13	111.38	7.46	2.05	.241477
U1-PP (mm)	30.00	2.97	30.36	2.79	0.36	.144714
U6-PP (mm)*	25.72	2.64	26.47	2.15	0.75	.036712
Mandibular dentoalveolar component						
L1.NB (°)*	23.10	5.10	25.05	4.29	1.95	.009646
L1-NB (mm)*	5.66	1.35	6.21	1.53	0.55	.002062
IMPA (°)*	85.20	6.21	88.00	5.42	2.79	.001191
L1-GoMe (mm)*	40.15	2.54	40.74	2.59	0.59	.022880
L6-GoMe (mm)*	31.42	1.85	32.40	1.86	0.97	.000030
Dental relationship						
Overbite (mm)	1.09	0.94	1.02	1.62	−0.06	.803535
Overbite (Exp) (mm)	1.07	1.18	0.47	1.92	−0.60	.324981

Exp, Expansion patients.
*Statistically significant at $P < .05$.

the changes in overbite during the posttreatment period (Tables III, IV, and V).

There was no statistically significant change of the anterior overbite in the posttreatment period. Not only did the overbite show no statistically significant change in the experimental group, but its change was similar to that observed in control group 2, thus demonstrating a normal behavior of this variable (Tables V and VI). This result is similar to those from other studies that have found stability of the obtained anterior overbite from treatment in the permanent dentition.[11,13,28] However, these reports do not distinguish between patients treated with and without extractions,[11,17] are reports of single cases,[13,28] or involve an insufficient observation period (at most 2 years posttreatment) to evaluate stability.[11,18] Therefore, the current results are more specific and might contribute to understanding the long-term behavior of the overbite in extraction treatment of open bite malocclusion in the permanent dentition.

Changes in the maxillary component do not seem to have played an important role in overbite improvement during treatment, because they were not statistically significant during this period (Table IV). The absence of significant anteroposterior changes in the maxillary component during the mean period of 8.35 years posttreatment, or in relation to the changes in control group 2, might have positively influenced the stability of the overbite in the posttreatment period (Tables V and VI).

Some significant changes were observed in the mandibular component during the treatment period (T2 − T1) and the posttreatment period (T3 − T2) (Tables IV and V). Mandibular ramus height (Ar-Go) increased significantly in these periods, and the gonial angle (Ar.Go.Me) decreased during the posttreatment period. Because the observed changes in these periods tend to produce a counterclockwise rotation of the mandible, it is possible that they might have contributed to the stability of the overbite after treatment. However, there was no correla-

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

	T3–T2		*Control group 2*		
Variables	*Mean*	*SD*	*Mean*	*SD*	P
Maxillary component					
SNA (°)	−0.33	1.74	0.30	1.24	.151456
Mandibular component					
SNB (°)*	−0.24	1.32	0.90	1.32	.003771
Maxillomandibular relationship					
ANB (°)	−0.11	1.65	−0.60	0.95	.226291
Growth pattern					
FMA (°)	−1.29	2.87	−1.04	1.62	.716090
SN.PP (°)	0.12	1.41	−0.69	1.60	.060021
SN.OP (°)*	−0.61	2.77	−3.44	3.99	.004004
SN.GoGn (°)	−0.56	2.08	−1.64	2.04	.069365
LAFH (mm)*	0.73	2.35	2.43	2.30	.012898
Maxillary dentoalveolar component					
U1.NA (°)	1.17	5.51	0.57	2.28	.639047
U1-NA (mm)	1.17	3.03	0.78	0.98	.577964
U1.PP (°)	2.05	9.55	0.19	2.08	.386704
U1-PP (mm)	0.36	1.34	0.83	0.79	.150312
U6-PP (mm)*	0.75	1.92	1.90	1.31	.020808
Mandibular dentoalveolar component					
L1.NB (°)*	1.95	3.93	−0.04	2.45	.044075
L1-NB (mm)*	0.55	0.91	0.08	0.63	.045253
IMPA (°)*	2.79	4.34	0.44	2.43	.029328
L1-GoMe (mm)	0.59	1.38	1.27	1.12	.067758
L6-GoMe (mm)	0.97	1.10	1.38	1.57	.276709
Dental relationship					
Overbite (mm)	−0.06	1.50	−0.31	0.83	.498362
Overbite (Exp) (mm)	−0.60	1.48	−0.31	0.83	.519616

Exp, Expansion patients.
*Statisically significant at $P < .05$.

Table VII. Correlation results

	Overbite T3–T2	
Variables	r	P
Overbite T1	.63	.000131
U1-PP T3–T2	.37	.035095
Overbite T2–T1	−.67	.000030
SN.OP T3–T2	−.38	.031909

tion between changes of the anterior overbite during the posttreatment period and the changes in these variables during these periods. Comparisons of the mandibular ramus height, gonial angle, and posterior face height changes in the posttreatment period with control group 2 were not performed, because these variables were not available for control group 2.

Changes in the maxillomandibular relationship during treatment in the posttreatment period and compared with control group 2 were not statistically significant, and therefore this variable does not seem to have influenced the overbite changes (Table VI). Additionally, there was no correlation between the changes in overbite and maxillomandibular relationship in the posttreatment period. Previous studies have also found no correlation between these variables.[10,17,39]

During treatment, there was a significant decrease in the SN.OP angle and an increase in LAFH and posterior face height (S-Go) (Table IV). The SN.OP angle showed a reduction, probably due to the extrusion of the mandibular incisors during the open bite correction that usually takes place.[10,17,39] The increase in LAFH is usually expected as a result of growth during orthodontic treatment, and the increase in ramus height is consequent to growth.[40] After treatment, the SN.OP angle did not show a decrease similar to the that in the control group, probably because of the slightly smaller (nonsignificant) vertical development of the mandibular incisors as compared with control group 2 and also because of the greater vertical growth pattern component of the experimental group. Changes in this variable were inversely correlated with changes in the overbite, as previously reported,[16] although with a low

correlation coefficient. Nevertheless, attention should be paid to the factors that could cause an increase in the occlusal plane inclination, to provide a better stability of the obtained overbite. The LAFH increase in the posttreatment period was not statistically significant and was less than that of control group 2, which might have contributed to the stability of the obtained overbite, associated with the significant increase of the posterior face height during the posttreatment period (Tables V and VI). The FMA angle did not change during treatment but decreased in the posttreatment period, similar to control group 2. Thus, most changes in the growth pattern variables in the posttreatment period favored stability of the obtained overbite.

The results from the treatment period (T2 − T1) indicated that correction of the anterior open bite was possible primarily because of the statistically significant extrusion, retrusion, and uprighting of the maxillary and mandibular incisors, as demonstrated by the U1.NA, U1-NA, U1.PP, U1-PP, L1.NB, L1-NB, IMPA, and L1-GoMe variables, which presented statistically significant changes during treatment. This has also been observed by many authors.[10-13,17] These changes were primarily consequent to the anterior segment retraction during space closure and to use of vertical intermaxillary elastics to aid in closing the open bite.[11,17] The maxillary molars' dentoalveolar height showed a statistically significant increase during orthodontic treatment and the posttreatment period (Tables V and VI). Their vertical development during this last period was smaller than that in control group 2. It could be speculated that the significant increase in the maxillary molars' dentoalveolar height during treatment could be a consequence of the rapid palatal expansion in 7 patients. However, the extrusion of the maxillary posterior teeth that usually occurs after rapid palatal expansion is transitory and noticeable only immediately after the procedure. At the end of the complete fixed appliance treatment, extrusion of these teeth is similar to that seen in control groups.[41-45] Besides, an increase in the vertical height of the maxillary molars during orthodontic treatment in adolescents is usually observed.[46] In this study, because no control group was used to compare the changes during treatment, it cannot be determined whether the significant increase in the maxillary molars' dentoalveolar height was a direct consequence of the mechanotherapy or of normal growth. Therefore, despite deserving further investigation, it is unlikely that the rapid palatal expansion in these 7 patients played an important role in the observed changes. Because it is commonly thought that relapse of expansion could contribute to relapse in open bite, the overbite changes in these 7 patients during treatment and the posttreatment period were separately investigated in a pilot study. Comparison of the posttreatment changes was also performed with control group 2. The results demonstrated the same tendency as in the whole sample (Tables IV, V, and VI). Thus, the expansion procedure does not seem to have affected the stability of the open bite correction, but further investigation is still needed. Previous studies have not addressed the influence of rapid palatal expansion on open bite correction stability.[10,11,16,17] Although the maxillary incisors showed a statistically significant vertical development during orthodontic therapy, this was not observed during the posttreatment period (Tables IV and V). During the posttreatment period, the vertical development of these teeth was similar to that in control group 2 (Table VI). A smaller-than-normal vertical development of the maxillary molars and a normal vertical development of the maxillary incisors during the posttreatment period are favorable factors for the stability of open bite correction. There was a statistically significant correlation between the vertical development of the maxillary incisors and the overbite changes in the posttreatment period ($r = .37$, $P = .03$). This result coincides with that reported by Huang et al.[16]

A behavior similar to that of the maxillary incisors was observed for the mandibular incisors during treatment. Vertical development of the mandibular incisors and molars was statistically significant during the treatment and posttreatment periods (Tables IV and V). During the posttreatment period, vertical development of the mandibular incisors and molars was similar to that in control group 2 (Table VI). This shows that the mandibular teeth do not contribute significantly to overbite decrease in the posttreatment period. There was no statistically significant correlation between the changes in the mandibular teeth and the changes in overbite in the posttreatment period. Some unfavorable changes of the mandibular incisors toward overbite stability were the greater labial tipping and protrusion during the posttreatment period and in relation to control group 2, which consist of a relapse tendency to the original position (Tables V and VI). The observed tendency is that variables that had the greatest influence during treatment will usually show smaller changes after treatment. Some will be favorable, and some will not.[47-50]

Kim et al[11] stated that the remarkable vertical development of the posterior teeth, combined with the changes at the incisors during and after orthodontic treatment, is the main reason for the decrease of the anterior overbite, because it causes a mandibular clockwise rotation. Most authors studying treatment stability of anterior open bite agree with this statement.[10,17] Because in this study the maxillary posterior teeth showed a smaller vertical development, and the mandibular posterior teeth showed a

normal vertical development during the posttreatment period in relation to control group 2, it could be speculated that this might have contributed to the absence of a statistically significant decrease of the overbite in the posttreatment period. The amount of overbite change in the posttreatment period was positively correlated with the initial overbite amount and negatively correlated with the amount of overbite change during treatment (Table VII). These results confirm previous statements that the initial open bite severity and the amount of open bite correction play important roles in open bite relapse.[12,16,51,52] However, although this correlation was observed in this extraction treatment group, it was not observed in a concomitant study of exclusively nonextraction open bite treatment in the permanent dentition.[19]

Clinically significant relapse and stability

The overbite decrease previously mentioned only demonstrates whether the changes in the overbite between the end of treatment and an average of 8.35 years later are "statistically significant" within the experimental group and in relation to a control group. Although this mathematic jargon can be very useful and understandable to the researcher, it does not demonstrate to the clinician whether the patients had a negative overbite (ie, perceptible by the patient and possibly the cause of complaints) again after this period. It also does not show the percentages of patients who might or might not show a negative overbite again in the long term. For these reasons, evaluation of the "clinically significant" relapse and stability of the open bite treatment was performed. It was observed that only 8 patients (25.8%) showed a "clinically significant" relapse of the open bite (ie, a negative overbite at T3). Consequently, 74.2% of the patients in the experimental group showed a "clinically significant" stability of the anterior open bite correction in the long term. Lopez-Gavito et al,[17] using a different evaluation method, found that 63.5% of patients showed stability of the open bite correction in the long term. Besides the differences in evaluation methodology, her sample also combined nonextraction and extraction treatment protocols. This might explain the slightly fewer stable patients. Results from the current study seem to confirm earlier speculation that there is a difference in treatment stability of open bite treated by nonextraction and extraction approaches.[18] The extraction approach seems to be more stable than nonextraction. As previously mentioned, a concomitant study has been undertaken on patients treated only with nonextraction, and the results point toward this tendency.[19]

Even though the decrease in overbite in the posttreatment period was not statistically significant, it was observed that 25.8% of patients might have varying degrees of open bite in the long term. Although treatment of open bite with extractions seems to provide a better prognosis for correction stability, some patients might still show relapse of the open bite in the long term. Therefore, every procedure designed to provide stability of the correction should be undertaken, such as overcorrection, active retention with bite-blocks, and speech therapy. Future studies comparing the stability of nonextraction and extraction open bite treatments are worthwhile.

CONCLUSIONS

On the basis of the present results and according to the methodology used to evaluate the stability of extraction therapy for the anterior open bite in the permanent dentition an average of 8.35 years after treatment, it was possible to conclude the following:

1. The decrease in the overbite obtained with treatment was not statistically significant.
2. The primary factors that contributed to the nonsignificant decrease of the overbite were the normal vertical development of the maxillary and mandibular incisors, the smaller vertical development of the mandibular molars, and the consequent smaller increase in lower anterior face height, as compared with the control group in the long-term posttreatment period.
3. There was a statistically significant positive correlation between the changes in overbite during the posttreatment period and the initial amount of overbite and the change in maxillary incisor dentoalveolar height during the posttreatment period. There was a statistically significant negative correlation between the changes in overbite during the posttreatment period and its change during treatment and the change in the occlusal plane inclination in the posttreatment period.
4. There was "clinically significant" stability in 74.2% of patients receiving extraction open bite treatment in the permanent dentition.

REFERENCES

1. Fränkel R, Fränkel C. A functional approach to treatment of skeletal open bite. Am J Orthod 1983;84:54-68.
2. Graber TM. Etiology of malocclusion: extrinsic or general factors. In: Graber TM, editor. Orthodontics, principles and practice. 2nd ed. Philadelphia: W. B. Saunders; 1966. p. 249-325.
3. Ngan P, Fields HW. Open bite: a review of etiology and management. Pediatr Dent 1997;19:91-8.
4. Parker JH. The interception of the open bite in the early growth period. Angle Orthod 1971;41:24-44.
5. Klocke A, Nanda RS, Kahl-Nieke B. Anterior open bite in the deciduous dentition: longitudinal follow-up and craniofacial growth considerations. Am J Orthod Dentofacial Orthop 2002; 122:353-8.

6. Worms FW, Meskin FH, Isaacson RJ. Open-bite. Am J Orthod 1971;59:589-95.
7. Takeyama H, Honzawa O, Hozaki T, Kiyomura H. A case of open bite with Turner's syndrome. Am J Orthod Dentofacial Orthop 1990;97:505-9.
8. Nagahara K, Miyajima K, Nakamura S, Iizuka T. Orthodontic treatment of an open bite patient with oral-facial-digital syndrome. Am J Orthod Dentofacial Orthop 1996;110:137-44.
9. Subtelny JD, Subtelny JD. Oral habits—studies in form, function, and therapy. Angle Orthod 1973;43:347-83.
10. Küçükkeles N, Acar A, Demirkaya AA, Evrenol B, Enacar A. Cephalometric evaluation of open bite treatment with NiTi arch wires and anterior elastics. Am J Orthod Dentofacial Orthop 1999;116:555-62.
11. Kim YH, Han UK, Lim DD, Serraon MLP. Stability of anterior openbite correction with multiloop edgewise archwire therapy: a cephalometric follow-up study. Am J Orthod Dentofacial Orthop 2000;118:43-54.
12. Enacar A, Ugur T, Toruglu S. A method for correction of open bite. J Clin Orthod 1996;30:43-8.
13. Goto S, Boyd RL, Nielsen IL, Iizuka T. Case report: nonsurgical treatment of an adult with severe anterior open bite. Angle Orthod 1994;64:311-8.
14. Lee BW. Case report: treatment of anterior open bite with tongue thrust and associated temporo-mandibular joint symptoms. Aust Orthod J 1993;12:246-9.
15. Safirstein GR, Burton DJ. Open bite—a case report. Am J Orthod 1983;83:47-55.
16. Huang GJ, Justus R, Kennedy DB, Kokich VG. Stability of anterior open bite treated with crib therapy. Angle Orthod 1990;60:17-26.
17. Lopez-Gavito G, Wallen TR, Little RM, Joondeph DR. Anterior openbite malocclusion. A longitudinal 10-year postretention evaluation of orthodontically treated patients. Am J Orthod 1985;87:175-86.
18. Chang Y, Moon SC. Cephalometric evaluation of the anterior open bite treatment. Am J Orthod Dentofacial Orthop 1999;115:29-38.
19. Janson G, Valarelli FP, Henriques JFC, Freitas MR, Cançado RH. Stability of anterior open bite nonextraction treatment in the permanent dentition. Am J Orthod Dentofacial Orthop 2003;124:265-76.
20. Dahlberg G. Statistical methods for medical and biological students. New York: Interscience; 1940.
21. Houston WJB. The analysis of errors in orthodontic measurements. Am J Orthod 1983;83:382-90.
22. Gravely JF, Benzies PM. The clinical significance of tracing error in cephalometry. Br J Orthod 1974;1:95-101.
23. Baumrind S, Frantz R. The reliability of head film measurements. 1. Landmark identification. Am J Orthod 1971;60:111-27.
24. Richardson A. A comparison of traditional and computerized methods of cephalometric analysis. Eur J Orthod 1981;3:15-20.
25. Cho HJ. Patient with severe skeletal Class III malocclusion and severe open bite treated by orthodontics and orthognathic surgery—a case report. Am J Orthod Dentofacial Orthop 1996;110: 155-62.
26. Jackson AF. Open bite. Report of a case. Am J Orthod 1951;37: 623-8.
27. Insoft MD, Hocevar RA, Gibbs CH. The nonsurgical treatment of a Class II open bite malocclusion. Am J Orthod Dentofacial Orthop 1996;110:598-605.
28. Miyajima K, Iizuka T. Treatment mechanics in a Class III open bite malocclusion with Tip Edge technique. Am J Orthod Dentofacial Orthop 1996;110:1-7.
29. Lieberman MA, Gazit E. Correction of Class I skeletal open bite malocclusion. Angle Orthod 1978;48:206-9.
30. Pearson LE. Treatment of a severe openbite excessive vertical pattern with an eclectic non-surgical approach. Angle Orthod 1991;61:71-6.
31. Riolo ML, Moyers RE, McNamara JA, Hunter WS. An atlas of craniofacial growth: cephalometric standards from The University School Growth Study, The University of Michigan. Monograph 2, Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development, The University of Michigan; 1974.
32. Subtelny JD. Openbite: diagnosis and treatment. Am J Orthod 1964;50:337-58.
33. Sassouni V. A classification of skeletal facial types. Am J Orthod 1969;55:109-23.
34. Nemeth RB, Isaacson RJ. Vertical anterior relapse. Am J Orthod 1974;65:565-85.
35. Frost DE, Fonseca RJ, Turvey TA, Hall DJ. Cephalometric diagnosis and surgical-orthodontic correction of apertognathia. Am J Orthod 1980;78:657-69.
36. Janson GRP, Metaxas A, Woodside DG. Variation in maxillary and mandibular molar and incisor vertical dimension in 12-year-old subjects with excess, normal, and short lower anterior facial height. Am J Orthod Dentofacial Orthop 1994;106:409-18.
37. Snodell SF, Nanda RS, Currier GF. A longitudinal cephalometric study of transverse and vertical craniofacial growth. Am J Orthod Dentofacial Orthop 1993;104:471-83.
38. Katsaros C, Berg R. Anterior open bite malocclusion: a follow-up study of orthodontic treatment effects. Eur J Orthod 1993;15:273-80.
39. Kim YH. Anterior open bite and its treatment with multiloop edgewise archwire. Angle Orthod 1987;57:290-321.
40. Tsang WM, Cheung LK, Samman N. Cephalometric characteristics of anterior open bite in a southern Chinese population. Am J Orthod Dentofacial Orthop 1998;113:165-72.
41. Linder-Aronson S, Lindgren J. The skeletal and dental effects of rapid maxillary expansion. Br J Orthod 1979;6:25-9.
42. Wertz RA. Skeletal and dental changes accompanying rapid midpalatal suture opening. Am J Orthod 1970;58:41-66.
43. Adkins MA, Nanda RS, Currier GF. Arch perimeter changes on rapid palatal expansion. Am J Orthod Dentofacial Orthop 1990; 97:194-9.
44. Haas AJ. Long-term posttreatment evaluation of rapid palatal expansion. Angle Orthod 1980;50:189-217.
45. Mew J. Relapse following maxillary expansion. A study of twenty-five consecutive cases. Am J Orthod 1983;83:56-61.
46. Gardner RA, Harris EF, Vaden JL. Postorthodontic dental changes: a longitudinal study. Am J Orthod Dentofacial Orthop 1998;114:582-7.
47. Franchi L, Baccetti T, McNamara JA. Treatment and posttreatment effects of acrylic splint Herbst appliance therapy. Am J Orthod Dentofacial Orthop 1999;115:429-38.
48. Pancherz H, Hansen K. Occlusal changes during and after Herbst treatment: a cephalometric investigation. Eur J Orthod 1986;8: 215-28.
49. Vargervik K, Harvold EP. Response to activator treatment in Class II malocclusions. Am J Orthod 1985;88:242-51.
50. Woodside DG. Do functional appliances have an orthopedic effect? Am J Orthod Dentofacial Orthop 1998;113:11-4.
51. Nahoum HI. Vertical proportions: a guide for prognosis and treatment in anterior open bite. Am J Orthod 1977;72:128-46.
52. Nielsen H. Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment. Angle Orthod 1991;61:247-60.