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Changes in alveolar morphology during open bite treatment and prediction of treatment result

Stefan H. Beckmann* and Dietmar Segner**
*The Maurice and Gabriela Goldschleger School of Dental Medicine, Tel Aviv University, Ramat Aviv, Israel, and **Private practice, Hamburg, Germany

SUMMARY It has been postulated that dentoalveolar height is enlarged by a compensation mechanism in long face subjects. In this study, dentoalveolar composition was studied in 83 open bite patients. It was found that increases in overbite during treatment coincided with vertical lengthening of the symphysis, which exceeded increments in lower face height. This vertical growth coincided mainly with an increase in the area of the symphysis. Furthermore, a retrusion of the maxillary incisors enhanced bite deepening.

Stability of the overbite during the retention period was studied in a subset of 22 patients. It was found that retrusion of the maxillary incisors during treatment led to a more stable overbite during the retention period. Vertical lengthening of the symphysis relative to the increase in lower face height seemed to enhance bite opening during retention. Prediction of the overbite may be reliable, if a re-evaluation of the patients takes place during active treatment. The angle NTGoGn had a substantial predictive value (multiple $R = 0.46$) for post-treatment overbite. It is concluded that in open bite patients, a dentoalveolar compensation mechanism results in a stable overbite at the end of treatment by enlarging symphysial height through a moderate increase in symphysial volume. In addition, retrusion of the maxillary incisors contributes to overbite reduction. However, an excessive increase in vertical height of the symphysis relative to lower face height may relapse after active treatment. For prediction of the post-treatment overbite, the angle NTGoGn may be used, although a re-evaluation during treatment is recommended.

Introduction

Functional disorders (thumb sucking and tongue interposition) as well as skeletal factors contribute to the presence of an anterior open bite (AOB). An AOB, which is caused by a sucking habit, has a favourable prognosis, provided that the habit is terminated (Van der Linden and Boersma, 1984; Proffit and Fields, 1993). An open bite can also be associated with a skeletal open bite, which is characterized by an enlarged lower face height and a steep inclined mandibular plane (Nahoum, 1971, 1977; Cangialosi, 1984; Lopez-Gavito *et al*., 1985). A combination of a skeletal and a dental open bite poses severe problems during orthodontic treatment (Nemeth and Isaacson, 1974; Lopez-Gavito *et al*., 1985; Denison *et al*., 1989) and generally leads to poor and unstable results (Safirstein and Burton, 1983; Van der Linden and Boersma, 1984; Segner and Hasund, 1998). It has been suggested that in these subjects, a post-treatment increase in sutural maxillary growth is not matched by a vertical increase in posterior face height (Nemeth and Isaacson, 1974). Thus, a backward rotation of the mandible occurs with a consequent increase in palato-mandibular divergence and with an increase in the vertical distance between the anterior elements of the jaws. If the continuing vertical increase in anterior lower face height is not matched by compensatory growth of the anterior alveolar and basal bone, an open bite will develop. These patients are often treated by orthognathic surgery. The treatment strategy should be decided before the onset of active therapy as pure orthodontic mechanics differs

when compared with treatment that involves orthognathic surgery. Therefore, prediction of the post-treatment overbite may be a useful tool in treatment planning of open bite cases. However, pre-treatment dimensions of the lower face have been shown to be poor cephalometric predictors for post-treatment overbite (Lopez-Gavito *et al*., 1985; Dung and Smith, 1988; Katsaros and Berg, 1993). Whilst associations between post-treatment overbite and the pre-treatment cephalometric measurements have been studied for each measurement separately, the association between a combination of several measurements and post-treatment overbite has not previously been investigated. Only one paper has dealt with the association between overbite and a combination of measurements (Finlay and Richardson, 1995).

Although the skeletal morphology partially determines the degree of overbite after orthodontic treatment, other factors may also be important (Lopez-Gavito *et al*., 1985; Dung and Smith, 1988; Katsaros and Berg, 1993). In some patients with skeletal and dental open bite, elongation of the anterior alveolar and basal bone during treatment may compensate for the enlarged vertical dimensions. This has been described as 'the dentoalveolar compensation mechanism' (Solow, 1980). Other studies have shown that a long face coincides with a narrowed symphysial shape (Aki *et al.*, 1994; Beckmann *et al.*, 1998a) while an open bite is associated with a small symphysial area (Beckmann *et al.*, 1998b). The narrowed symphysial shape in long-faced patients may be associated with a dentoalveolar compensation mechanism, but the symphysial area may be the most important factor in determining overall symphysial size and, consequently, symphysial height, thus having a direct influence upon overbite.

It was decided to examine whether a combination of cephalometric measurements could predict the post-treatment overbite of open bite patients and whether increments in the overbite in these subjects during treatment were associated with a dentoalveolar compensatory mechanism. This would increase the anterior alveolar and basal bone height. In order to achieve this a longitudinal study was performed, in which the following hypotheses were tested:

1. Increments in the overbite of open bite patients during orthodontic treatment are related to a dentoalveolar compensation mechanism which increases the vertical height of the anterior alveolar and basal bones relative to lower face height.
2. The dentoalveolar compensation mechanism acts through vertical growth of the anterior alveolar and basal bone, which is accompanied by an increase in volume.
3. The dentoalveolar compensation mechanism acts through stretching of the anterior alveolar and basal bone, while the volume remains constant.
4. The post-treatment overbite can be predicted from the pre-treatment cephalometric data.
5. The increase in anterior alveolar height is related to the stability of the overbite during the retention phase of treatment.

Material and methods

From the archives of the Department of Orthodontics, University of Hamburg, the records of 380 subjects with a pre-treatment dental AOB were selected from a cohort of 6500 patients. The selection was performed by inspection of the pre-treatment cephalograms. A dental open bite was defined as a missing overlap of the maxillary and the mandibular incisal edges relative to the occlusal plane. From this group of 380 subjects, all those patients: (1) who had completed orthodontic treatment at the unit; (2) whose documentation was complete, including cephalograms taken before treatment and at the end of retention; (3) who were of Caucasian origin; and (4) who did not have severe craniofacial disorders such as cleft palate or hemifacial microsomia, were included. No selection was made on the basis of skeletal configuration, gender, duration of treatment, treatment methods, treatment results, or age before treatment.

The records revealed that some patients who were treated with removable appliances only did not wear their appliances consistently, which prompted the orthodontist to interrupt

treatment. It was decided to exclude these patients, regardless of the skeletal configuration and dental occlusion at the time of treatment interruption, although this posed a risk of selection bias. Furthermore, patients were omitted when orthognathic surgery was originally planned and performed. Other patients who were unsuccessfully treated solely by orthodontics, but where orthognathic surgery was planned, were included in the study. The initial orthodontic treatment was directed at increasing the overbite. The records taken at the end of orthodontic treatment before referral to the orthognathic surgeon were then considered as the records at the end of retention. The final sample included 83 patients (51 female, 32 male).

From all 83 patients, lateral cephalograms at the beginning of treatment (T1) and at the end of the retention period (T3) were available. In 22 subjects, cephalograms were also recorded at the end of active treatment (T2). In these patients, changes in the overbite during the retention period were evaluated separately. Twenty-three anatomical landmarks were recorded and six additional landmarks were constructed. The landmarks were defined according to Segner and Hasund (1998) and Beckmann *et al*. (1998a,b). Thirty-seven measurements were computed. These included standard measurements as described by Segner and Hasund (1998), and measurements to describe the morphology of the symphysis and of the maxillary anterior alveolar and basal bone in terms of height, depth, and total anterior alveolar and basal area. The following indices were calculated:

Index = UFH/LFH
MxAI (Maxillary Alveolar Index) = MxAD/MxABH
MdAI (Mandibular Alveolar Index) = MdAD/MdABH
MxBal (Maxillary Basal Index) = MxBaD/MxABH
MdBal (Mandibular Basal Index) = MdBaD/MdABH
MxIAHI (Maxillary Incisal Alveolar Height Index) = MxIAH/LFH
MdIAHI (Mandibular Incisal Alveolar Height Index) = MdIAH/LFH
MxABAI (Maxillary Alveolar Basal Area Index) = MxABA/LFH
MdABAI (Mandibular Alveolar Basal Area Index) = MdABA/LFH

Four of these described the shape of the anterior alveolar and basal bone by dividing depth into height and the other four the development of the alveolar and basal height and area relative to lower face height development (Figures 1 and 2).

Each cephalogram was digitized twice, the values for each pair of recordings were then averaged, and the statistical analyses were performed with the calculated means of each

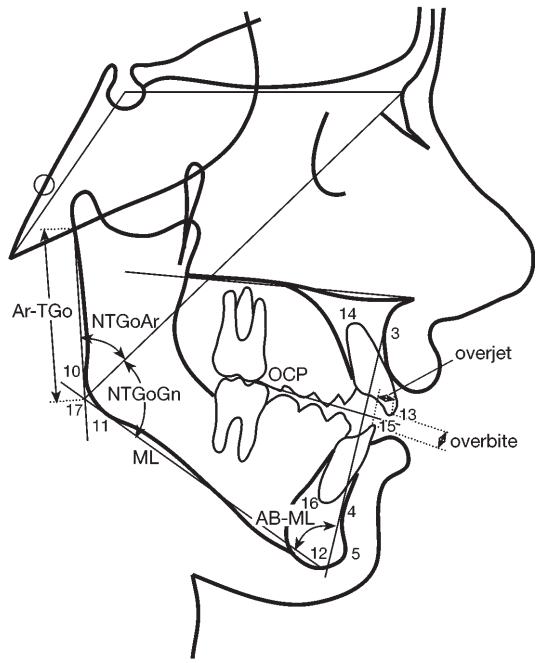

Figure 1 Skeletal landmarks, reference lines and measurements. *Landmarks.* In addition to the measurements as described by Segner and Hasund (1998), the following reference lines and measurements were employed. *Reference lines*—OCP: occlusal plane, connecting the midpoints between the incisal ridges of the central incisors and the midpoint between the mesiobuccal cusps of the first molars. *Measurements*—NTGoGn: the angle between the line N–TGo and the mandibular plane; NTGoAr: the angle between the line N–TGo and the posterior tangent to the ramus; AB–ML: the angle between the line A–B and the mandibular plane; ODI: Modified Overbite Depth Indicator, the difference between the angle AB–ML and NL–NSL according to the definition of Dung and Smith (1988); Ar–TGo: ramus height, the distance between articulare and gonion—tangent point.

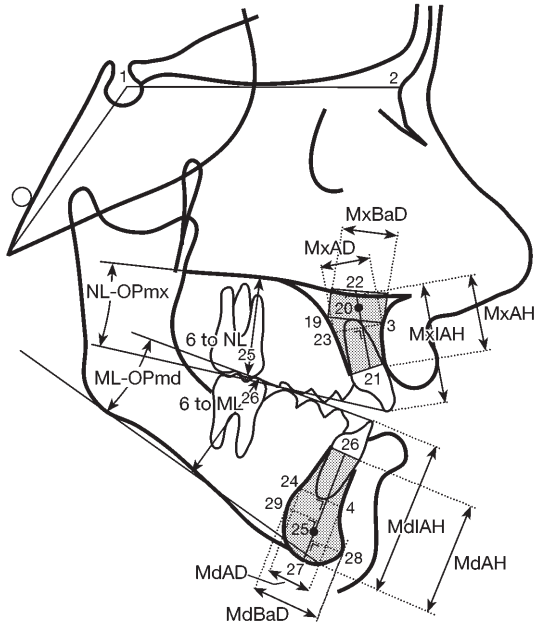

Figure 2 Dentoalveolar landmarks, reference lines and measurements. *Landmarks.* 19: palatal counterpart of point A (2) on the palatal cortical bone at the same distance from the palatal plane as point A. 20: the centre of the rectangle limited by the line 2–12 and the palatal plane. The rectangle represents the mid-sagittal section of the basal bone of the maxilla. This point was defined as the centrepoint of the maxillary alveolus. 21: midpoint of the alveolar meatus of the maxillary central incisor. 22: intersection between the bony hard palate and the maxillary alveolar axis (the maxillary alveolar axis runs from the midpoint of the alveolar meatus of the maxillary central incisor through the centre-point of the maxillary alveolus). 23: the intersection between a perpendicular to the maxillary alveolar axis through point A and the palatal border of the maxillary frontal mid-sagittal alveolar bone. 24: the intersection between a perpendicular to the mandibular alveolar axis through point B and the lingual border of the symphysis. 25: the centerpoint of the basal mid-sagittal bone of the mandible (point D according to Steiner, 1959). 26: midpoint of the alveolar meatus of the mandibular central incisor. 27: intersection between the lower border of the symphysis and the mandibular alveolar axis (the mandibular alveolar axis runs from the midpoint of the alveolar meatus of the mandibular central incisor through the centerpoint of the symphysis). 28: point on the anterior symphysial border, most distant from the mandibular alveolar axis. 29: point on the posterior symphysial border, most distant from the mandibular alveolar axis. *Reference lines*—MxOCP: maxillary occlusal plane, a line through the incisal tip of the most anterior maxillary incisor and the mesiobuccal cusp of the first maxillary molar. MdOCP: mandibular occlusal plane, a line through the incisal tip of the most anterior mandibular incisor and the mesiobuccal cusp of the first mandibular molar. *Measurements*—MxIAH: the distance between the tip of the most anterior maxillary incisor and point 22, measured parallel to the maxillary alveolar axis. MdIAH: the distance between the tip of the most anterior mandibular incisor and point 27, measured parallel to the maxillary alveolar axis. MxAH: maxillary alveolar axis height: the distance between points 21 and 22. MdAH: mandibular alveolar axis height, the distance between points 26 and 27. MxAD: maxillary alveolar depth, the distance between point A and point 23. MxBaD: maxillary basal depth, the distance between point A and point 19. MdAD: mandibular alveolar depth, the distance between point B and point 24. MdBaD: mandibular basal depth, the distance between point 28 and the mandibular alveolar axis, added to the distance between point 29 and the mandibular alveolar axis. 6 to NL: the distance between the most occlusal point of the maxillary first molar and the palatal plane. 6 to ML: the distance between the most occlusal point of the mandibular first molar and the mandibular plane. NL–OPmx: the angle between the maxillary occlusal plane and the palatal plane. ML–OPmd: the angle between the mandibular occlusal plane and the mandibular plane. MxABA: area of the alveolar and basal mid-sagittal cross-section of the maxillary jaw. A line was drawn perpendicular to the palatal plane, intersecting point A (3) and forming the anterior border of the maxillary basal area. From point A, a line was drawn parallel to the nasal plane intersecting the dorsal contour of the maxillary alveolar bone at point 19. A line perpendicular to the nasal plane, intersecting point 19, formed the dorsal border of the maxillary basal area. The area was then measured between these lines and the outer contour of the maxillary alveolar bone (shaded). MdABA: area of the alveolar and basal midsagittal cross-section of the mandible, the area between the outer contour of the symphysis (shaded).

pair. Reliability of the measurements was analysed by repeated tracing and digitizing of 20 cephalograms. The second tracing was at an interval of at least two weeks after the first one. Systematic errors were detected by paired Student's *t*-tests. The size of the error (Dahlberg, 1940) and the coefficient of reliability according to Houston (1983) were calculated.

Associations were assessed between overbite changes from T1 to T3 and changes of all other variables from T1 to T3. Furthermore, prediction of the post-treatment overbite was performed by using pre-treatment measurements, as well as a combination of pre-treatment measurements and changes in measurements from T1 to T3. For assessment of these associations, correlation and regression analyses were used on all 83 patients, resulting in a subset of 22 patients who were investigated separately in order to evaluate changes in overbite during the retention period (T2–T3). The associations between changes of the overbite during retention and the measurements at the beginning of treatment as well as during active treatment were also examined by regression analyses.

For all statistical analyses, the confidence level $P < 0.05$ was considered significant.

Results

Sample description

The mean age before the beginning of active treatment was 12 years 11 months (SD: 4 years 8 months). The age distribution was skewed. At the beginning of active treatment, 57 subjects were between 8 and 12 years of age, and 20 patients between 13 and 18 years of age. Six adults were also included (24–27 years of age). The mean duration of total observation time (including treatment and retention) was 5 years 7 months (SD: 2 years 4 months).

A variety of different treatment techniques were used. Forty-four patients were treated solely with removable appliances, and 39 subjects had fixed appliance in one or both arches during certain phases of active treatment. Thirty-four patients had worn occipital headgear and 34 a functional appliance. During retention, Hawley appliances were used, as well as fixed retainers. In some patients an activator was worn during retention.

Error study

The Dahlberg error ranged from 0.23 mm (overbite) to 14.73 mm^2 (maxillary alveolar and basal area). All variables used in the study had coefficients of reliability above 0.97 except for the angles NSBa (0.95), NL–NSL (0.96), for the distance $\underline{1}$ to NA (0.96), for the maxillary alveolar area (0.92), for the maxillary area index (0.93), for the maxillary as well as the mandibular alveolar height index (0.92 and 0.96, respectively), for the maxillary and basal alveolar index (0.89 and 0.85, respectively), and for the maxillary and basal alveolar depth (0.88 and 0.76, respectively).

Systematic errors were found for the variables ML–OPmd, P6 to NL, and MdAH.

Descriptive statistics

Total sample. An increase in overbite was measured in 74 subjects, ranging from 0.20 to 8.70 mm. One patient had no change in overbite and in eight the overbite decreased during treatment. Decreases ranged from –0.04 to –4.81 mm.

Tables 1 and 2 show descriptive statistics for the total group ($n = 83$). In Table 1, mean values of all variables at T1 and T3 for patients with a closed bite (overbite above 0 mm) and with an open bite (overbite smaller than 0 mm) are shown. Table 2 shows the mean values of changes of all variables between T1 and T3 for the same groups. The descriptive statistics of the subset ($n = 22$) are given in Table 3 where the mean values of variables for T1 and changes of all variables between treatment (T1–T2) and between retention (T2–T3) are shown for patients with a closed bite (overbite above 0 mm) and with an open bite (overbite smaller than 0 mm) separately.

Subset. All patients in the subset had some increase in overbite during treatment, ranging from 1.05 to 6.06 mm. However, during the retention period, the overbite decreased in 14 subjects; four had a bite opening of more than

Table 1 Descriptive statistics for groups with treatment success (closed bite at T3, $n = 53$) and treatment failure (open bite at T3, $n = 30$).

	T1 treatment success		T1 treatment failure		T3 treatment success		T3 treatment failure	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	11.67	5.09	12.39	3.70	17.25	4.40	17.97	2.82
SNA	82.16	3.40	80.20	3.91	82.14	2.98	80.15	3.78
NL–NSL	7.13	2.93	7.52	3.25	7.89	2.90	7.82	3.09
NSBa	134.39	4.73	132.71	4.80	134.19	4.76	131.09	5.70
ML–NSL	36.13	5.24	41.90	5.04	33.97	5.54	41.37	5.80
SNB	76.44	3.34	75.04	3.45	77.72	3.19	75.76	4.52
ML–NL	29.00	5.40	34.38	6.20	26.08	5.61	33.56	6.31
UFH	46.35	3.70	48.62	4.25	49.95	2.82	52.15	3.68
LFH	59.97	6.69	65.94	7.43	64.25	5.63	72.90	8.37
Index	77.75	6.13	74.27	7.57	78.18	6.62	72.19	7.48
Ar–TGo	39.80	4.81	40.85	4.58	44.91	4.67	46.21	4.66
ANB	5.72	2.71	5.15	3.21	4.41	2.44	4.40	3.11
ANPg	5.42	2.93	5.33	3.75	3.70	3.04	4.14	3.70
SNPg	76.74	3.38	74.87	3.53	78.44	3.35	76.01	4.62
Pg–NB	0.52	1.52	–0.35	2.33	1.39	2.04	0.47	2.63
NTGoGn	74.47	4.72	80.36	5.58	73.55	4.88	81.45	6.23
NTGoAr	54.01	4.53	51.09	4.85	51.34	3.79	48.69	4.98
ODI	82.35	7.08	77.18	7.85	82.44	6.29	76.52	8.79
NL–OPmx	8.05	3.71	9.61	4.08	10.57	4.09	9.11	3.57
ML–OPmd	16.69	4.81	18.53	4.50	19.53	4.62	20.23	6.65
Overbite	–2.15	1.36	–2.99	1.73	1.72	1.17	–2.15	2.05
Overjet	6.13	2.93	4.91	3.25	3.00	1.40	3.44	2.12
1–T	121.16	8.79	120.82	11.19	130.25	9.66	126.13	10.23
1–NA	25.59	5.26	25.32	5.44	18.48	7.54	22.71	6.50
T–NB	27.53	5.76	28.70	7.66	26.86	6.62	26.76	7.68
1 to NA	4.01	2.24	4.13	2.29	2.01	2.54	3.52	2.58
T to NB	4.69	2.04	5.72	2.58	5.15	2.41	6.21	2.89
P6 to NL	19.59	3.63	21.82	3.96	22.50	2.33	25.34	4.05
M6 to ML	28.11	3.37	30.08	3.28	31.08	2.85	33.44	3.35
MxABA	213.31	41.51	246.11	39.67	232.39	45.26	267.94	48.69
MdABA	243.59	41.71	261.87	47.99	275.23	38.94	287.56	56.02
MxAI	63.36	10.40	59.24	11.51	59.07	10.75	54.98	12.41
MxBaI	82.37	16.15	73.39	17.00	73.22	16.03	65.81	19.03
MdAI	27.60	6.81	23.12	6.79	22.48	6.09	17.20	5.70
MdBaI	52.88	8.34	45.92	7.95	48.03	8.00	41.94	6.61
MxABAI	24.35	1.74	23.88	2.07	23.66	1.67	22.47	1.73
MdABAI	26.09	2.16	24.61	2.41	25.89	2.24	23.31	2.35
MxIAHI	45.82	2.77	45.15	3.10	45.97	2.80	44.00	2.97
MdIAHI	60.78	2.42	60.14	3.15	62.42	2.48	60.17	3.22

1 mm, three maintained a negative overbite until the end of treatment and during the retention period, and one showed an increase in overbite during treatment as well as during retention; however, the increase was not sufficient to close the large open bite in that specific subject (–7 mm). Increases in the overbites of the other patients were matched by equally large amounts of bite opening during retention. In three patients, the bite was closed during treatment, but opened again during retention. All these patients showed some deepening of the bite during treatment, but a decrease in the overbite during retention, which was larger than the increase gained during treatment.

Correlation analyses, total sample (n = 83)

Pearson's correlations were calculated between the overbite at T3 and all measurements at T1

Table 2 Descriptive statistics for groups with treatment success (closed bite at T3, n = 53) and treatment failure (open bite at T3, n = 30).

	$_{\Delta}$T1–T3 treatment success		$_{\Delta}$T1–T3 treatment failure	
	Mean	SD	Mean	SD
Age	5.58	2.16	5.58	2.58
SNA	–0.03	1.61	–0.05	1.80
NL–NSL	0.77	1.71	0.30	1.74
NSBa	–0.20	1.73	–1.62	2.84
ML–NSL	–2.16	2.62	–0.53	2.44
SNB	1.28	1.94	0.71	2.30
ML–NL	–2.92	2.90	–0.82	2.74
UFH	3.60	2.74	3.53	2.42
LFH	4.28	3.58	6.96	5.15
Index	0.43	2.52	–2.09	3.29
Ar–TGo	5.11	4.32	5.36	4.03
ANB	–1.31	1.81	–0.76	1.45
ANPg	–1.72	1.97	–1.19	1.50
SNPg	1.70	2.02	1.14	2.46
Pg–NB	0.87	1.11	0.82	1.16
NTGoGn	–0.92	1.63	1.09	1.83
NTGoAr	–2.67	2.30	–2.40	2.58
ODI	0.10	2.89	–0.66	3.83
NL–OPmx	2.53	4.13	–0.50	3.82
ML–OPmd	2.84	3.93	1.70	3.83
Overbite	3.88	1.74	0.84	2.22
Overjet	–3.13	2.69	–1.48	2.66
1–T	9.09	8.88	5.31	7.67
1–NA	–7.11	7.01	–2.61	5.96
T–NB	–0.67	5.29	–1.94	5.02
1 to NA	–2.00	2.30	–0.62	2.26
T to NB	0.45	1.43	0.49	1.54
P6 to NL	2.91	2.69	3.52	3.03
M6 to ML	2.96	2.18	3.36	3.00
MxABA	19.08	23.88	21.83	33.80
MdABA	31.64	24.97	25.69	27.33
MxAI	–4.29	9.83	–4.26	10.51
MxBaI	–9.15	11.48	–7.58	13.02
MdAI	–5.12	4.86	–5.92	4.14
MdBaI	–4.86	4.32	–3.99	3.63
MxABAI	–0.69	1.15	–1.41	1.65
MdABAI	–0.20	0.81	–1.30	1.03
MxIAHI	0.15	2.29	–1.15	2.15
MdIAHI	1.64	1.99	0.02	1.98

(Table 4). The largest correlations were found between overbite and NTGoGn, LFH, MdBaI, and MdABAI. Assessment of the correlations between changes of overbite between T1 and T3 and changes of measurements during the same period revealed significant correlations above 0.40 for the following variables: mandibular alveolar height index, mandibular alveolar area index, angle ML–NL, index, angles ML–OPmd and NTGoGn, and the inter-incisal angle.

Regression analyses—relative importance of all measurements in changes of the overbite during treatment (total sample, n = 83)

Prediction of the overbite change between T1 and T3 with changes of variables between T1 and T3 showed that 82.5 per cent of the variance of the overbite (R = 0.91) could be explained mainly by a combination of the mandibular and maxillary incisal alveolar height index, the angles 1–NA, ANPg, and the Overbite Depth Indicator (ODI; Table 5). In patients with an increase in mandibular incisal alveolar height relative to lower face height, with retrusion of the maxillary incisors, a decrease in the angle ANPg, an increase in the maxillary incisal alveolar height relative to lower face height and an increase in the ODI, can be expected to result in an increase in overbite during treatment. The contribution of the ODI is very small, although still significant.

Regression equation:

$$_{\Delta}\text{overbite} = 0.495 + 0.626(_{\Delta}\text{MdIAHI}) - 0.162(_{\Delta}\text{1–NA}) - 0.570(_{\Delta}\text{ANPg}) + 0.367(_{\Delta}\text{MxIAHI}) + 0.106(_{\Delta}\text{ODI})$$

Regression analyses—importance of lower face height, size and shape of the anterior mandibular alveolar and basal bone and incisor inclination in bite-closure during treatment (total sample, n = 83)

A regression analysis was performed to predict overbite changes between T1 and T3 from changes of the anterior alveolar and basal bone, and incisor inclination and lower face height between T1 and T3. The following variables were used: lower face height, alveolar and basal area, and alveolar and basal indices. The results showed that 63.2 per cent of the variance of the overbite (R = 0.80) could be explained by a combination of changes between T1 and T3 of the inclination of the maxillary incisor relative to NA, the lower face height, the mandibular alveolar and basal area, and the maxillary basal, mandibular basal, and mandibular alveolar

Table 3 Descriptive statistics for groups with stable overbite at T3 (n = 16) and with relapse of open bite at T3 (n = 6).

	T1 treatment success		T1 treatment failure		$_{\Delta}$T1–T2 treatment success		$_{\Delta}$T1–T2 treatment failure		$_{\Delta}$T2–T3 treatment success		$_{\Delta}$T2–T3 treatment failure	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	14.31	6.37	14.08	1.69	2.92	1.58	2.92	1.50	1.97	1.20	1.55	0.67
SNA	81.71	2.89	80.88	5.97	0.41	2.18	–0.90	1.57	–0.52	1.35	–0.09	0.65
NL–NSL	7.55	2.86	6.61	4.16	–0.07	1.96	1.01	1.41	0.56	1.16	–0.66	1.21
NSBa	133.50	4.87	129.47	7.60	–0.70	2.13	–0.30	0.91	0.57	1.52	–0.69	1.43
ML–NSL	36.38	5.99	40.36	1.64	–1.23	1.47	–0.58	0.94	–0.55	1.58	0.31	0.81
SNB	76.25	3.19	75.42	3.40	1.01	1.31	–0.06	1.79	–0.26	0.55	–0.17	0.52
ML–NL	28.83	6.72	33.74	5.15	–1.16	2.01	–1.59	1.41	–1.11	1.76	0.98	1.14
UFH	48.26	3.75	50.01	3.39	1.52	2.23	1.55	1.08	0.63	1.51	0.63	1.00
LFH	63.68	7.04	68.60	6.40	2.17	2.54	2.47	2.70	0.60	1.32	1.12	1.11
ANB	5.46	2.72	5.46	5.20	–0.60	1.77	–0.85	1.45	–0.26	1.25	0.09	0.61
ANPg	5.05	3.14	5.06	5.96	–0.88	1.79	–1.20	1.17	–0.55	1.51	0.12	0.69
SNPg	76.66	3.42	75.83	3.21	1.29	1.39	0.29	1.75	0.02	0.69	–0.21	0.61
Pg–NB	0.74	1.26	0.76	2.42	0.55	0.86	0.72	0.82	0.60	0.68	–0.06	0.63
Overbite	–2.11	1.10	–3.07	2.06	3.74	1.07	2.54	1.28	–0.01	1.11	–0.75	0.91
1–NA	25.47	4.90	27.32	5.36	–8.78	6.08	–6.82	8.35	1.72	6.32	0.54	2.55
T–NB	26.64	4.88	29.29	13.14	0.36	6.18	–1.50	8.56	–0.24	3.79	1.33	3.54
1 to NA	4.20	2.17	4.98	2.65	–2.64	2.18	–1.97	2.73	0.36	1.66	0.19	0.81
T to NB	4.83	2.23	6.65	4.44	0.75	1.54	0.23	2.78	–0.13	1.06	0.32	0.75
MxABAI	23.73	1.64	23.72	2.25	–0.06	1.22	–1.74	2.06	–0.36	1.02	0.12	0.91
MdABAI	25.96	2.47	23.83	3.07	–0.30	0.92	–0.58	0.86	–0.11	0.44	–0.70	0.22
MxIAHI	45.24	1.97	46.71	4.64	0.78	1.19	–1.55	3.25	–0.61	1.23	0.25	1.10
MdIAHI	60.94	1.85	59.24	2.03	1.38	1.17	1.66	1.24	0.43	1.37	–0.99	0.86

indices (Table 6). A marked increase in the overbite mainly coincided with a slight increase in lower face height, an increase in the mandibular alveolar and basal area, a retrusion of the maxillary incisor relative to the line NA, a decrease in the maxillary and mandibular basal index, and a decrease in the mandibular alveolar index.

Regression equation:

$$_{\Delta}\text{overbite} = 1.460 - 0.551(_{\Delta}\text{LFH}) - 0.120(_{\Delta}\underline{1}\text{–NA}) - 0.041(_{\Delta}\text{MxBaI}) - 0.149(_{\Delta}\text{MdAI}) - 0.144(_{\Delta}\text{MdBaI}) + 0.059(_{\Delta}\text{MdABA})$$

Regression analyses—the relative importance of all measurements in prediction of the overbite at the end of retention (total sample, n = 83)

In order to predict the overbite at T3 with the initial variables at T1, all variables were considered in a multiple stepwise regression analysis. The results showed that 40.9 per cent of the variance of the overbite (R = 0.64) could be explained by a combination of the angles NTGoGn and ML–OPmd, and upper face height and the mandibular basal index (Table 7). Patients with a large NTGoGn angle, a large upper face height, a small ML–OPmd angle, and a narrow mandibular symphysis base pre-treatment, will have a small or negative overbite at the end of retention. This can be deduced from the positive and negative B and β-values. The contributions of the upper face height and mandibular basal index are very small, although still significant.

Regression equation:

$$\text{Overbite T3} = 15.93 - 0.217(\text{NTGoGn}) + 0.255(\text{ML–OPmd}) - 0.151(\text{N–Sp}') + 0.074(\text{MdBaI})$$

Prediction of the overbite at T3 with changes of variables between T1 and T3 showed that

Table 4 Correlations between overbite and all other variables, $n = 83$.

Variables	Corr. var. T1 and overbite T3	Corr. var. $_{\Delta}$T1–T3 and overbite T3	Corr. var. $_{\Delta}$T1–T3 and overbite $_{\Delta}$T1–T3
Age	0.24*	0.03	0.11
SNA	0.24*	0.01	0.01
NL–NSL	0.13	0.20	0.29**
NSBa	0.14	0.26*	0.17
ML–NSL	0.41***	0.31**	0.36**
SNB	0.16	0.19	0.26*
ML–NL	0.33**	0.38***	0.49***
UFH	0.38***	0.09	0.08
LFH	0.43***	0.28*	0.27*
Index	0.20	0.48***	0.49***
Ar–TGo	0.23*	0.05	0.02
ANB	0.11	0.24*	0.31**
ANPg	0.05	0.22	0.30**
SNPg	0.20	0.17	0.26*
Pg–NB	0.14	0.02	0.06
NTGoGn	0.46***	0.46***	0.47***
NTGoAr	0.24*	0.13	0.07
ODI	0.27*	0.08	0.14
NL Opmx	0.10	0.39***	0.36**
ML OPmd	0.08	0.30**	0.48**
Overbite	0.32**	0.80***	1.00***
Overjet	0.20	0.29**	0.27*
1–T	0.06	0.29**	0.41***
1–NA	0.10	0.35**	0.33**
T–NB	0.05	0.07	0.12
1 to NA	0.03	0.22*	0.22*
T to NB	0.17	0.02	0.09
P6 to NL	0.37***	0.04	0.01
M6 to ML	0.32**	0.07	0.19
MxABA	0.21	0.03	0.16
MdABA	0.31**	0.09	0.14
MxAI	0.37***	0.02	0.05
MxBaI	0.43***	0.23*	0.20
MdAI	0.32**	0.16	0.11
MdBaI	0.13	0.13	0.32**
MxABAI	0.25*	0.10	0.11
MdABAI	0.43***	0.48***	0.50***
MxIAHI	0.26*	0.31**	0.38***
MdIAHI	0.23*	0.51***	0.70***

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

61.6 per cent of the variance of the overbite ($R = 0.79$) could be explained by a combination of the mandibular and maxillary incisal alveolar height index, the angles 1–NA, NTGoGn, ANB, NSBa, and NTGoAr (Table 8). Patients with an increase in the mandibular alveolar height index, a retrusion of the maxillary incisors, a decrease in the angle NTGoGn, an increase in the maxillary alveolar height index, a decrease in the ANB angle, an increase in the NSBa angle, and a decrease in the NTGoAr angle will have a deeper overbite at the end of retention. This can be deduced from the positive and negative B and β-values.

Regression equation:

$$\text{Overbite T3} = -1.484 + 0.396(_{\Delta}\text{MdIAHI}) - 0.139(_{\Delta}\underline{1}\text{–NA}) - 0.217(_{\Delta}\text{NTGoGn}) + 0.304(_{\Delta}\text{MxIAHI}) - 0.350(_{\Delta}\text{ANB}) + 0.209(_{\Delta}\text{NSBa}) - 0.172(_{\Delta}\text{NTGoAr})$$

Prediction of the post-treatment overbite from the combination of pre-treatment measurements and changes of variables during treatment showed that 86.7 per cent of the variance of the overbite ($R = 0.93$) could be explained by a combination of the following variables: overbite, NL–NSL, and MdAI before treatment, changes of MdIAHI, MxIAHI, 1–NA, ANPg, and LFH during treatment (Table 9). Patients with a large NL–NSL, a large open bite, and a small MdAI pre-treatment, increases in LFH, ANPg, 1–NA, and decreases in MxIAHI and MdIAHI during treatment generally had an open bite at the end of retention. This can be presumed from the positive and negative B and β-values.

Regression equation:

$$\text{Overbite T3} = 0.917(\text{overbite T1}) + 0.530(_{\Delta}\text{MdIAHI}) - 0.148(_{\Delta}\underline{1}\text{–NA}) + 0.347(_{\Delta}\text{MxIAHI}) - 0.403(_{\Delta}\text{ANPg}) - 0.162(\text{NL–NSL}) + 0.059(\text{MdAI}) - 0.072(_{\Delta}\text{LFH})$$

Correlation analyses (subset, n = 22)

Correlations were calculated between changes in overbite during retention and a selection of pre-treatment variables, as well as changes of variables during active treatment (Table 10). Significant correlations with pre-treatment variables were only found for the angles SNA and 1–NA. Correlations with changes of variables during active treatment were found to be significant for the ANB angle, for Pg–NB and for the mandibular alveolar height index. All other correlations were not significant.

Table 5 Results of regression analysis.

Variable	R	R^2	SE	B	β	T	P
MdIAHI	0.697	0.485	1.742	0.626	0.551	11.047	***
1–NA	0.760	0.578	1.588	–0.162	–0.468	–9.272	***
ANPg	0.827	0.683	1.384	–0.570	–0.431	–7.954	***
MxIAHI	0.899	0.808	1.083	0.367	0.351	6.956	***
ODI	0.908	0.825	1.043	0.106	0.143	2.670	***
Constant				0.495		2.520	*

*$P < 0.05$; ***$P < 0.001$.
Dependent variable: overbite $_{\Delta}$T1–T3; independent variable: all variables $_{\Delta}$T1–T3, $n = 83$. R, R^2, and SE are displayed after the corresponding variable is added to the regression equation; R, R^2, and SE (R) are given for the total equation.

Table 6 Results of regression analysis.

Variable	R	R^2	SE	B	β	T	P
1–NA	0.334	0.112	2.289	–0.120	–0.346	–4.798	***
MdBaI	0.494	0.244	2.124	–0.144	–0.244	–2.629	*
LFH	0.637	0.405	1.896	–0.551	–1.000	–8.621	***
MdABA	0.737	0.543	1.674	0.059	0.633	5.850	***
MdAI	0.773	0.580	1.579	–0.149	–0.283	–2.739	**
MxBaI	0.795	0.632	1.521	–0.041	–0.202	–2.640	*
Constant				1.460		4.213	***

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
Dependent variable: overbite $_{\Delta}$T1–T3; independent variable: changes of selected variables between T1 and T3, $n = 83$. R, R^2, and SE are displayed for each variable after being added to the regression equation; R, R^2, and SE (R) are given for the total equation.

Table 7 Results of regression analysis.

Variable	R	R^2	SE	B	β	T	P
NTGoGn	0.456	0.208	2.168	–0.217	–0.517	–3.989	***
ML–OPmd	0.543	0.295	2.058	0.255	0.500	4.194	***
UFH	0.610	0.372	1.955	–0.151	–0.252	–2.611	*
MdBaI	0.639	0.409	1.909	0.074	0.269	2.197	*
Constant				15.930		2.830	**

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
Dependent variable: overbite T3; independent variable: all variables T1, $n = 83$; R, R^2, and SE are displayed after the corresponding variable is added to the regression; R, R^2, and SE (R) are given for the total equation.

Regression analyses (subset, n = 22)

Prediction of overbite changes during retention (T2–T3) with variables at the beginning of active treatment (T1) showed that 60.5 per cent of the variance of the overbite ($R = 0.78$) could be explained by a combination of the angle SNA and the inclination of the mandibular incisor relative to NB (Table 11). Bite opening during retention coincided with a retrognathic maxilla

Table 8 Results of regression analysis.

Variable	*R*	R^2	SE	B	β	T	*P*
MdIAHI	0.508	0.258	2.099	0.396	0.347	4.405	***
1–NA	0.602	0.362	1.958	–0.139	–0.400	–5.315	***
NTGoGn	0.686	0.470	1.795	–0.217	–0.175	–2.029	*
MxIAHI	0.722	0.521	1.719	0.304	0.291	3.682	***
ANB	0.754	0.569	1.641	–0.350	–0.246	–3.121	**
NSBa	0.769	0.591	1.608	0.209	0.197	2.488	*
NTGoAr	0.785	0.616	1.568	–0.172	–0.170	–2.216	*
Constant				–1.484		–4.808	***

*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.
Dependent variable: overbite T3; independent variable: all variables $_{\Delta}$T1–T3, $n = 83$; *R*, R^2, and SE are displayed after the corresponding variable is added to the regression; *R*, R^2, and SE (*R*) are given for the total equation.

Table 9 Results of regression analysis.

Variable	*R*	R^2	SE	B	β	T	*P*
$_{\Delta}$MdIAHI	0.508	0.258	2.099	0.530	0.465	9.150	***
overbite	0.709	0.502	1.730	0.917	0.586	12.298	***
$_{\Delta}$1–NA	0.772	0.596	1.567	–0.148	–0.426	–9.354	***
$_{\Delta}$MxIAHI	0.833	0.694	1.373	0.347	0.331	6.808	***
$_{\Delta}$ANPg	0.902	0.814	1.077	–0.403	–0.304	–6.148	***
NL–NSL	0.917	0.841	1.004	–0.162	–0.203	–4.316	***
MdAI	0.926	0.857	0.957	0.059	0.174	3.577	***
$_{\Delta}$LFH	0.931	0.867	0.930	–0.072	–0.131	–2.325	*
Constant				0.717		1.264	

*$P < 0.05$; ***$P < 0.001$.
Dependent variable: overbite T3; independent variable: all variables T1 and $_{\Delta}$T1–T3, $n = 83$; *R*, R^2, and SE are displayed after the corresponding variable is added to the regression; *R*, R^2, and SE (*R*) are given for the total equation.

and proclined mandibular incisors before treatment. The most important variable was the angle SNA, followed by the angle T–NB.

Regression equation:

$$_{\Delta T2–T3}\text{overbite} = –16.552 + 0.227(\text{SNA}) – 0.078(\text{T–NB})$$

Prediction of overbite changes between the retention T2 and T3 with changes of variables between T1 and T2 showed that 42.3 per cent of the variance of overbite ($R = 0.65$) could be explained by a combination of the mandibular incisal alveolar height index and the distance of Pg to the line NB (Table 12). A bite opening during the retention period coincided with a large increase in mandibular dentoalveolar height in relation to increases in lower face height and with an increase in prominence of the bony chin. Both variables were equally important.

Regression equation:

$$_{\Delta T2–T3}\text{overbite} = 0.800 – 0.450(_{\Delta T1–T2}\text{MdAIHI}) – 0.594(_{\Delta T1–T2}\text{Pg–NB})$$

Discussion

Generally, overbite deepening did not occur in cases where vertical growth of the anterior lower face exceeded vertical growth of the symphysis during treatment. In the maxilla, the association between vertical growth of the anterior maxillary

Table 10 Correlations between variables T1 and the overbite at $_{\Delta}$T2–T3 and correlations between variables $_{\Delta}$T1–T2 and the overbite $_{\Delta}$T2–T3.

Variables	Correlation variables T1 – overbite $_{\Delta}$T2–T3	Correlation variables $_{\Delta}$T1–T2 – overbite $_{\Delta}$T2–T3
Age	–0.10	0.21
SNA	0.58**	–0.20
NL–NSL	–0.04	0.08
NSBa	–0.04	–0.11
ML–NSL	–0.30	0.10
SNB	0.31	0.24
ML–NL	–0.23	0.00
UFH	–0.04	0.14
LFH	–0.16	0.00
ANB	0.36	–0.46*
ANPg	0.28	–0.36
SNPg	0.34	0.12
Pg–NB	0.15	–0.44*
Overbite	–0.17	0.00
1–NA	–0.42*	0.06
T–NB	–0.25	0.34
1 to NA	–0.40	–0.10
T to NB	–0.21	0.06
MxABAI	–0.13	0.22
MdABAI	0.13	0.08
MxIAHI	–0.12	0.22
MdIAHI	–0.02	–0.46*

*$P < 0.05$; **$P < 0.01$.

alveolar and basal bone, and vertical growth of the anterior lower face was much smaller.

If vertical growth of the lower face was independent from vertical growth of the symphysis, a low correlation between the increase in alveolar and basal height of the symphysis and the increase in lower face height would be expected. The correlation is plotted in Figure 3 and was found to be highly significant. The high correlation coefficient and its significance indicate that vertical growth of the lower face and of the symphysis are related; an increase in lower face height was generally matched by a compensatory lengthening of the symphysis. Increases in alveolar and basal height were not associated with increases in overbite, but if the increase in lower face height was taken into account (as in the incisal alveolar height index), changes in the mandibular alveolar height index correlated significantly with overbite during treatment. It may be concluded that in patients where bite deepening occurred during treatment, a compensatory mechanism enhanced the bite deepening by vertical growth of the mandibular alveolar height that exceeded the increase in lower face height. The first hypothesis may thus be accepted. This compensation appeared to be achieved

Table 11 Results of regression analysis.

Variable	R	R^2	SE	B	β	T	P
SNA	0.584	0.341	0.905	0.227	0.796	5.108	***
T–NB	0.778	0.605	0.710	–0.078	–0.557	–3.569	**
Constant				–16.552		–4.814	***

**$P < 0.01$; ***$P < 0.001$.
Dependent variable: overbite $_{\Delta}$T2–T3; independent variable: variables at T1, $n = 22$; R, R^2, and SE are displayed for each variable after being added to the regression equation; B and $_{\beta}$-values are given for the total equation.

Table 12 Results of regression analysis.

Variable	R	R^2	SE	B	β	T	P
MdIAHI	0.465	0.216	0.987	–0.450	–0.483	–2.772	*
Pg–NB	0.650	0.423	0.869	–0.594	–0.455	–2.610	*
Constant				0.800		2.396	*

*$P < 0.05$.
Dependent variable: overbite $_{\Delta}$T2–T3; independent variables $_{\Delta}$T1–T2, $n = 22$; R, R^2, and SE are displayed for each variable after being added to the regression equation; B and $_{\beta}$-values are given for the total equation.

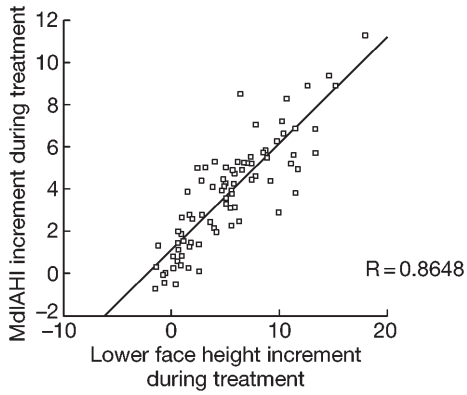

Figure 3 The correlation between increments in lower face height and increments in the mandibular incisal alveolar height relative to LFH is plotted. Note the large correlation coefficient (0.86).

mainly by growth of the symphysis. From Table 6 it can be seen that the second important factor in determining changes in overbite, besides the increase in lower face height, is a change of the area of the symphysis. Although this measurement is only two-dimensional, it may be assumed that it reflects the amount of increase in the volume of the symphysis.

The second hypothesis postulated that the compensatory mechanism acts through growth of the volume of the symphysis, while according to the third hypothesis, the symphysis gains vertical height through altering its shape by narrowing and concomitant lengthening. The results show that a narrowing and stretching of the symphysis was a factor of only minor importance. These results confirm the findings of Beckmann *et al.* (1998b), who concluded that symphysial area and shape were related to overbite in such a way that an open bite was associated with a small symphysial area, and secondly, with a wide, shortened symphysial shape. According to Beckmann *et al.* (1998a), symphysial shape is associated mainly with lower facial height, being narrowed and lengthened in long-faced patients with normal overbite. Aki *et al.* (1994) showed that a narrow shaped symphysis is mainly associated with a posterior rotation of the mandible. The results of the current study suggest that a wide symphysis may have more potential to compensate for an enlarged lower face height than a narrow symphysis. Patients with posterior growth rotation during treatment also had a narrow symphysis pre-treatment, which does not allow for dentoalveolar compensation of the enlarged lower face height.

It therefore appears that the dentoalveolar compensation mechanism acts through an increase in symphysial volume, which leads to acceptance of the second and rejection of the third hypothesis. However, the shape of the symphysis may determine the maximum amount of symphysial growth.

As bite deepening was associated with a basally wide symphysis, it may be that growth potential is reflected in the basal part of the symphysis. A narrow basal symphysis may have less potential for vertical growth, compared with a wide basal symphysis. Future studies should investigate the role of the basal part of the symphysis in determining overbite.

Uprighting of the maxillary incisors was found to be beneficial for bite deepening; this is in agreement with the results of Katsaros and Berg (1993). Protruded maxillary incisors, which may be associated with thumb-sucking habits and can be easily treated, as uprighting in these cases may occur spontaneously. In all but one of the adult open bite patients, the bite deepening coincided with maxillary incisal retrusion of at least 6 degrees.

The present study showed several pre-treatment measurements that correlated significantly with post-treatment overbite. Finlay and Richardson (1995) recently reported that spontaneous bite closure in open bite patients could be predicted by a combination of cephalometric measurements. In this study, a similar design was used to predict the post-treatment overbite with a combination of unconventional measurements in open bite subjects who underwent treatment. This approach considerably enlarges the predictive potential of cephalometric measurements. The large correlation between the angle NTGoGn and the post-treatment overbite was also found by Dozet (1979). The angle NTGoGn not only represents the inclination of the mandibular plane but also reflects the magnitude of total face height. Several studies have shown associations between

overbite and inclination of the mandible as well as lower face height (Nahoum, 1971, 1977; Cangialosi, 1984; Ellis and McNamara, 1984; Lopez-Gavito *et al*., 1985). Therefore, it may be expected that a measurement that combines both variables shows a higher correlation with overbite than each measurement separately. The contribution of the difference between the occlusal and the mandibular plane (ML–OPmd) for prediction of the post-treatment overbite may indicate that the rates of symphysial and molar vertical growth are determined individually and remain constant during treatment. Consequently, the prognosis for an open bite subject with a hyper-divergent mandible may still be favourable if the amount of vertical symphysial growth exceeds vertical growth of the mandible in the molar region, thus compensating for the unfavourable skeletal conditions.

Prediction of the post-treatment overbite by changes of measurements during treatment is more reliable compared with that by pre-treatment measurements only. If both pre-treatment measurements and changes during treatment are used as predictor-variables, the post-treatment overbite is largely dependent on changes in cephalometric structures during treatment. This may indicate that it might be useful to re-evaluate open bite patients during treatment by analysing growth of the symphysis and lower face height, as well as the amount of retrusion of the maxillary incisors. The amount of vertical growth of the symphysis should be compared with the increase in vertical skeletal height.

The fact that the maxillary anterior alveolar and basal bone is much less associated with the post-treatment overbite shows that this area is more independent from the overbite. The post-treatment overbite was only very slightly related to the pre-treatment inclination of the palatal plane, which may be expected as it has been frequently reported in the literature that the inclination of the maxilla can be influenced by therapeutic measurements, such as a high pull headgear (Merrifield and Cross, 1970; Duterloo, 1981; Carels and van der Linden, 1987). The pre-treatment inclination of the mandible is more strongly related to the overbite at the end of retention. This shows that therapeutic correction of the pattern of mandibular growth is difficult to achieve, which was also stated by Segner and Hasund (1998).

The low correlation between the ODI and the post-treatment overbite is in contrast to the results of Kim (1974) and Dung and Smith (1988). The ODI combines the palato-mandibular angle with the sagittal relationships between the maxilla and the mandible. As the correlation between overbite and sagittal jaw angle was low (0.11), it seems that the sagittal relationship between the jaws is not related to the vertical overbite. However, a decrease in the ANB angle seems to be beneficial for overbite at the end of retention. The favourable effect of a reduction in ANB angle may be related to retrusion of the maxillary incisors that also causes a dorsal repositioning of point A. The prognostic value of the maxillary incisal inclination was also confirmed by Katsaros and Berg (1993). Protruded maxillary incisors may also be associated with a reduced maxillary alveolar height which explains the contribution of this measurement in the regression analysis.

As age did not show a significant contribution in the regression analyses, it appears that compensatory growth of the alveolar bone of the symphysis can also take place after the pubertal growth spurt. Some of the patients in the sample were already in their late adolescence at the beginning of treatment and achieved a normal overbite at the end of retention. An active or passive extrusion of the anterior teeth seems to be associated with lengthening of the alveolar process. In some cases, this was also associated with an increase in the area of the symphysis. It may be that similar growth of the alveolar bone occurs in teeth whose antagonists are lost and which then continue to erupt (Proffit and Fields, 1993).

The treatment results indicate that the overbite of patients with a pre-treatment prognathic maxilla and retrusion of the mandibular incisors was generally more stable. This again confirmed the fact that retrognathic subjects are generally more difficult to treat. In contrast, a retrusion of the mandibular incisor during active treatment seemed to enhance relapse. This may be due to the fact that in subjects where the mandibular

incisors were retruded during treatment, the tongue pushed against the incisors in the retention phase, forcing them back into protrusion, which enhanced bite opening. Furthermore, the results show that an extrusion of the mandibular incisors, which exceeds the increase in the lower face height, may cause relapse, which confirms the fifth hypothesis. Active extrusion of anterior teeth in order to close an open bite is generally not advisable (Subtelny and Sakuda, 1964; Subtelny, 1975; Dellinger, 1986). The results of this study suggest that active extrusion may be possible, but it should not exceed the vertical growth of the lower face. Therefore, extrusion of the anterior teeth during treatment, which is often undertaken using vertical elastics, should be performed with care.

Conclusion

In patients with an open bite, the overbite may be corrected by a dentoalveolar compensation mechanism, which appears to enlarge the symphysial height by an increase in symphysial volume. Patients with a basally wide symphysis may have a larger potential for symphysial vertical growth.

Prediction of the overbite after treatment may be reliable, if a re-evaluation takes place during active treatment. The angle NTGoGn has a substantial predictive value for the post-treatment overbite. A stable overbite at the end of treatment may be achieved through retrusion of the maxillary incisors and growth of the symphysis. An increase in lower face height during treatment tends to diminish the amount of bite deepening. An excessive increase in vertical height of the symphysis may enhance relapse after active treatment and thus seems inadvisable.

Address for correspondence

Stefan H. Beckmann
The Maurice and Gabriela Goldschleger School of Dental Medicine
Section of Orthodontics
Tel Aviv University
Ramat Aviv
69978 Tel Aviv
Israel

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