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Dentoskeletal effects and facial profile changes during activator therapy

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SUMMARY The aim of this retrospective study was to investigate cephalometrically the skeletal, dental, and soft tissue modifications induced by activator treatment in patients with Class II malocclusions caused by mandibular retrognathism. The subjects, all in the mixed dentition, were selected from a single centre and were divided into two groups: 40 patients treated with an incisor double capping activator (20 girls, 20 boys with a mean age of 10 years) and a control group of 30 subjects (15 girls, 15 boys with a mean age of 10 years). The dentoskeletal and aesthetic changes that occurred were compared on lateral cephalograms taken before treatment (T0) and after 18–24 months, when the activator was removed (T1). In the control group the radiographs were obtained before (T0) and after (T1) 21 months (standard deviation ±3 months).

Activator treatment in these growing patients resulted in a correction of the Class II relationship (ANB –2.14°), a restriction of maxillary growth (SNA –0.5°), an advancement of the mandibular structures (SNB +1.64°, FH^NPg +3.39°; OLp-B +5.17 mm, OLp-Pg +5.14 mm, OLp-Go +2.44 mm), a correction of the overjet (–5.03 mm), an improvement in overbite (–1.17 mm) and uprighting of the maxillary incisors (1^FH –5.64°).

The activator appliance was effective in treating growing patients with mandibular deficiency: activator therapy corrected Class II malocclusions by a combination of skeletal and dental changes and improved the soft tissue facial profile.

Introduction

Although functional appliances have been designed to treat all types of malocclusion, their greatest application and success has been in correcting dental and skeletal Class II malocclusions (Owen, 1981; Schmuth, 1983; Carels and van der Linden, 1987; Bishara and Ziaja, 1989).

A major cause of a Class II malocclusion is mandibular retrognathism and, thus, a therapy able to enhance mandibular growth would be desirable (McNamara, 1981).

The hallmark of Class II functional appliances is the construction bite that positions the mandible anteriorly to increase mandibular growth (Moore *et al*., 1989).

Several types of functional appliance are currently in use and are a modification of the Andresen–Haupl activator, so called because of the proposed activation of the muscles and the circulatory system, with a reciprocal and mutual effect between the maxillomandibular complex and the appliance itself (Owen, 1981; Schmuth, 1983).

Many improvements in the design of functional appliances have been introduced to achieve greater effectiveness and patient acceptance.

Many working hypotheses have been conceived to explain the mode of action of the activator. The amount of and interrelationship between sagittal skeletal and dental changes contributing to Class II correction in activator treatment are controversial. Some authors (Luder, 1982; McNamara *et al*., 1985) indicate that the increase in mandibular growth is the distinguishing aspect of functional treatment with respect to other treatment procedures, while others (Björk, 1951; Meach, 1966; Harvold and Vargervik, 1971; Weislander and Lagerström, 1979) suggest that the treatment changes appear to be similar to those caused by growth and that mandibular length is unaltered by functional therapy.

It has been reported that functional therapy with activators restricts horizonal growth of the maxilla (Demisch, 1972; Ahlgren and Laurin, 1976; Forsberg and Odenrick, 1981) or that the effect is solely the result of dentoalveolar remodelling processes (Robertson, 1983; Chadwick *et al*., 2001).

The purpose of this investigation was to evaluate quantitatively on lateral radiographs the skeletal and dental parameters before (T0) and after (T1) 18–24 months of activator appliance treatment. In order to compare the effects of growth versus activator treatment, roentgenogram data taken before (T0) and after (T1) 21 months (standard deviation (SD) ±3 months) from a matching group of untreated Class II subjects were analysed.

Subjects and methods

The subjects for both the study and control groups were selected from a single centre (Department of Orthodontics, University of Rome 'Tor Vergata'). The following selection criteria were used: 9–11 years of age; overjet greater than 5 mm; Class II molar relationship, with at least half a cusp width distal molar relationship; skeletal Class II malocclusion with ANB greater than 5 degrees; retrognathic mandible, with SNB less than 78 degrees; no history of previous orthodontic therapy.

Patients satisfying these criteria were divided into two groups: a control group, 30 subjects (15 girls, 15 boys), who declined activator therapy, and a treatment group, 40 subjects (20 girls, 20 boys), who underwent activator therapy.

The control and treated groups corresponded with respect to initial age, malocculsion and observation period (18–24 months).

The appliance used was an acrylic monobloc attached to the upper jaw by Adams' clasps, with a central screw. The screw was activated only to follow maxillary transversal growth. The activator was designed to avoid undesirable anterior dental movements. The incisal edges and 2 mm of the labial surfaces of the maxillary and mandibular incisors were capped to prevent tipping (Figure 1).

The activator was produced from a construction bite that positioned the mandible anteriorly in an edge-to-edge incisor relationship (Moore *et al*., 1989). The lower jaw was postured forward in a Class I or overcorrected Class I molar relationship to stimulate mandibular growth. As a general rule, the bite registration was obtained 3 mm short of maximum protrusion, with care being taken to ensure that lateral displacement did not occur. The height of the bite exceeded the freeway space by 2–3 mm. During treatment, contact was maintained between the appliance and the maxillary posterior teeth; the mandibular posterior teeth were encouraged to erupt by trimming acrylic on the occlusal and lingual aspect. The patients were instructed to wear the appliance for a minimum of 14 hours in each 24 hour period.

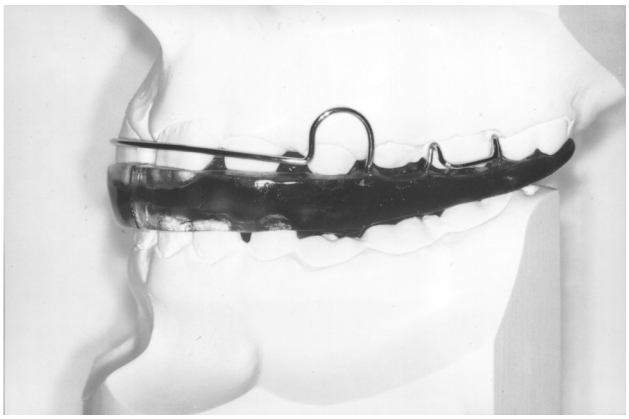

Figure 1 Design of the activator used in the study.

Method

The skeletal and dental changes that occurred were assessed on two lateral cephalometric radiographs. In the treatment group the first cephalogram was taken before treatment (T0) and the second after 21 months (SD ±3 months), when the activator was removed (T1). In the control group radiographs were obtained at the same interval. All cephalograms were taken with the teeth in occlusion and the lips in a relaxed position.

A full cephalometric analysis was carried out using the reference points and lines shown in Figure 2.

The initial cephalometric patterns of the control and treated subjects, as well as the alterations due to growth or treatment, were assessed using the following angles and distances: sagittal analysis: SNA (°), SNB (°), ANB (°), Ao–Bo (mm), Nperp–A (mm), Nperp–Pg (mm), NSCo (°), Co–A (mm), Co–Gn (mm), GoMe (mm), FH^NA (°), FH^NPg (°) (Figure 3); vertical analysis: FMA (°), FH^OL (°), SN^PP (°), PP^GoMe (°), N–ANS (mm), ANS–Me (mm) (Figure 4); dental analysis: $\underline{1}$^FH (°), IMPA (°), interincisal (°), overjet

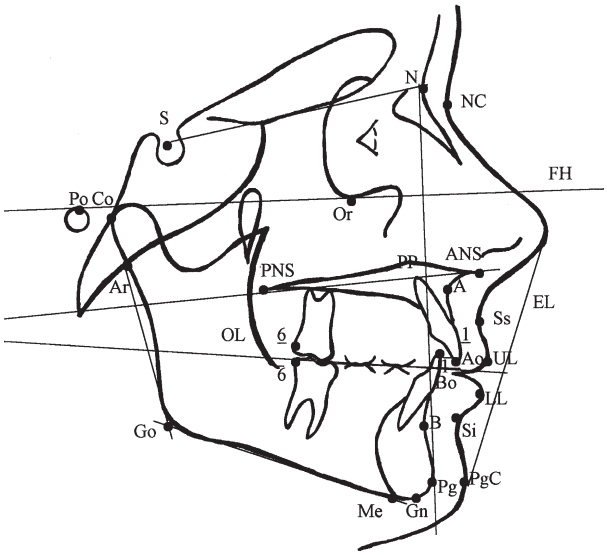

Figure 2 Reference points: sella (S), nasion (N), point A (A), point B (B), perpendicular to the occlusal line through A (Ao), perpendicular to the occlusal line through B (Bo), pogonion (Pg), gnathion (Gn), menton (Me), gonion (Go), articulare (Ar), condylion (Co), porion (Po), orbitale (Or), anterior nasal spine (ANS), posterior nasal spine (PNS), soft tissue nasion (NC), soft tissue pogonion (PgC), upper lip (UL), lower lip (LL), sulcus superius (Ss), sulcus inferius (Si), upper incisor ($\underline{1}$), lower incisor ($\bar{1}$), upper first molar ($\underline{6}$), lower first molar ($\bar{6}$). Reference lines: sella–nasion (SN), Frankfort plane (FH), line perpendiculare to FH passing through point N (Nperp), palatal plane (PP), functional occlusal line (OL), mandibular line (GoMe), aesthetic line (EL).

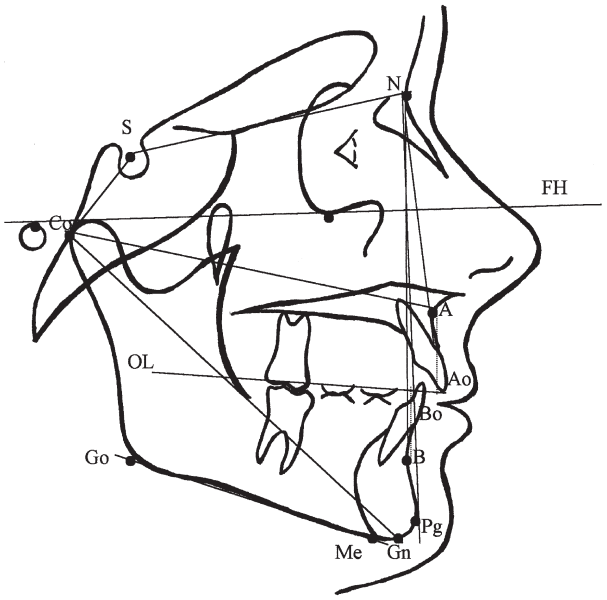

Figure 3 Sagittal analysis: SNA (º), SNB (º), ANB (º), Ao–Bo (mm), Nperp–A (mm), Nperp–Pg (mm), NSCo (º), CoA (mm), CoGn (mm), GoMe (mm), FH^NA (º), FH^NPg (º).

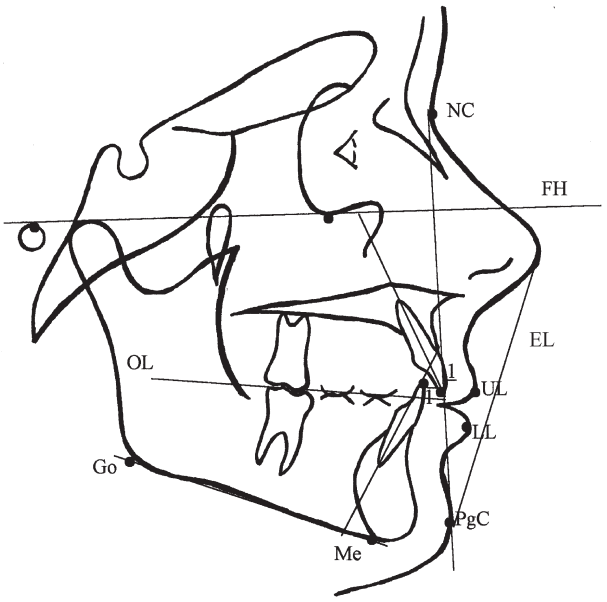

Figure 5 Dental analysis: 1^FH (º), IMPA (º), interincisal angle (º), overjet (mm), overbite mm, $\bar{1}$-OL (mm). Aesthetic analysis: NCPgC^FH (º), UL–EL (mm), LL–EL (mm).

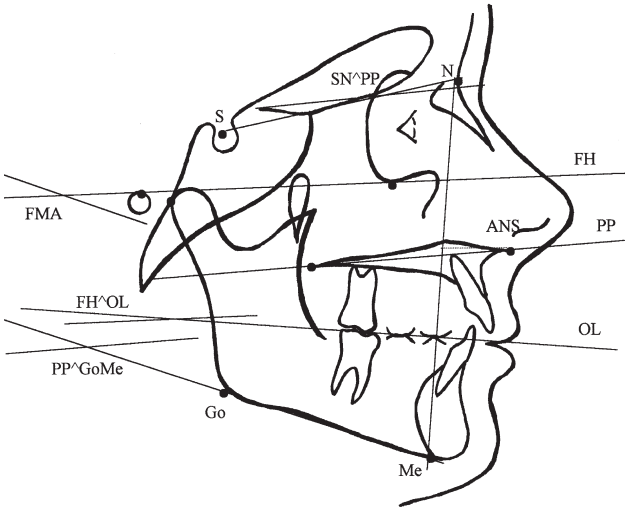

Figure 4 Vertical analysis: FMA (º), FH^OL (º), SN^PP (º), PP^GoMe (º), N–ANS (mm), ANS–Me (mm).

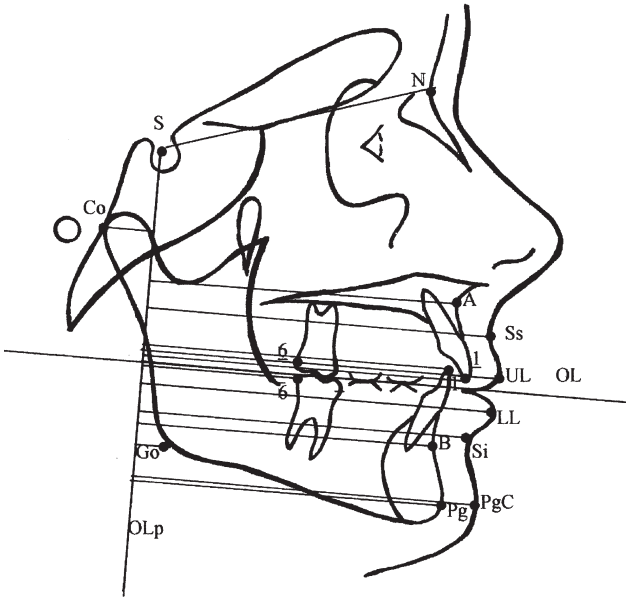

Figure 6 Sagittal registrations performed to OLp and parallel to OL: OLp–Co, OLp–A, OLp–B, OLp–Go, OLp–Pg, OLp–Ss, OLp–UL, OLp–LL, OLp–Si, OLp–PgC, OLp–$\underline{1}$, OLp–$\bar{1}$, OLp–$\underline{6}$, OLp–$\bar{6}$.

(mm), overbite (mm), 1-OL (mm); aesthetic analysis: NCPgC^FH (º), UL–EL (mm), LL–EL (mm) (Figure 5).

Other measuring points and reference lines used were those defined by Pancherz (1984). These linear measurements for the assessment of sagittal relationships were performed using the occlusal line (OL) and the occlusal line perpendicular (OLp) drawn through the sella. The reference grid, taken from the first head film (T0), was transferred to the T1 tracing using the sella–nasion (SN) line, with sella as the registration point.

All sagittal registrations were performed to the same reference line (OLp) and parallel to OL: OLp–Co, OLp–A, OLp–B, OLp–Go, OLp–Pg, OLp–$\underline{1}$, OLp–$\bar{1}$, OLp–$\underline{6}$, OLp–$\bar{6}$.

In addition to these skeletal and dental measurements, a soft tissue analysis was performed using the same reference grid: OLp–Ss, OLp–UL, OLp–LL, OLp–Si, OLp–PgC (Figure 6).

Method error

Each cephalogram was traced and measured by one author (PC). All measurements were repeated after a period of 7 days and the mean value of the two measurements was used. All measurement error coefficients were found to be close to 1.00 and within acceptable limits (Table 1).

Statistical method

Descriptive statistics included mean and standard deviation (SD). The mean intragroup differences in cephalometric measurements at T0 and T1 were examined with Wilcoxon's rank sum test and differences between the control and treated groups with the Mann–Whitney test. The level of significance was $P < 0.05$.

Non-parametric tests were used as the studied variables were not normally distributed.

Table 1 Method error coefficient.

Variables	*R*
SNA (º)	0.99
SNB (º)	0.98
ANB (º)	0.98
Ao–Bo (mm)	0.99
Nperp–A (mm)	0.99
Nperp–Pg (mm)	0.99
NSCo (º)	0.97
Co–A (mm)	0.98
Co–Gn (mm)	0.98
GoMe (mm)	0.99
FH^NA (º)	0.97
FH^NPg (º)	0.99
FMA (º)	0.98
FH^OL (º)	0.98
SN^PP (º)	0.99
PP^GoMe (º)	0.98
N–ANS (mm)	0.97
ANS–Me (mm)	0.98
$\underline{1}$^FH (º)	0.98
IMPA (º)	0.97
Interincisal angle (º)	0.97
Overjet (mm)	0.98
Overbite (mm)	0.99
$\overline{1}$–OL (mm)	0.97
NCPgC^FH (º)	0.99
UL–EL (mm)	0.98
LL–EL (mm)	0.98
OLp–Co (mm)	0.97
OLp–A (mm)	0.99
OLp–B (mm)	0.98
OLp–Go (mm)	0.99
OLp–Pg (mm)	0.98
OLp–Ss (mm)	0.98
OLp–UL (mm)	0.99
OLp–LL (mm)	0.99
OLp–Si (mm)	0.98
OLp–PgC (mm)	0.98
OLp–$\underline{1}$ (mm)	0.98
OLp–$\overline{1}$ (mm)	0.99
OLp–$\underline{6}$ (mm)	0.99
OLp–$\overline{6}$ (mm)	0.97

Results

Before treatment all patients had a Class II molar relationship with an increased overjet. At T0 there were no significant differences between the two groups for any of the cephalometric variables investigated, except for FMA, which was lower in the control group. The changes observed in the measured variables are shown in Tables 2 and 3, which present the average and SD of each cephalometric measurement considered before (T0) and after the treatment/observation period (T1).

Sagittal analysis

The cephalometric values before (T0) and after (T1) treatment showed a significant improvement in the sagittal jaw relationship. ANB diminished during therapy on average 2.14 degrees (SD 1.00, $P < 0.001$), while the control group showed an increase of 0.13 degrees (SD 0.61, ns). The relative sagittal position of the jaws, when measured along the OL (Ao–Bo), showed an average reduction of 3.33 mm (SD 1.43, $P < 0.001$) after treatment and an increase of 0.4 mm (SD 0.51, $P < 0.05$) in the control group.

In the treated group the increase in SNB was on average 1.64 degrees (SD 1.3, $P < 0.001$), while in the control group there was a slight increase of 0.17 degrees (SD 0.41, ns).

There was a significant increase in the horizontal measurements at pogonion after treatment. The variables NperpPg and FH^NPg increased on average 2.17 mm (SD 2.09, $P < 0.001$) and 3.39 degrees (SD 4.16, $P < 0.001$), while in the control group there was a non-significant reduction of 0.77 mm (SD 2.06) and 0.67 degrees (SD 1.76), respectively.

The distance Co–Gn increased significantly in both groups, but more after therapy (treatment group: 5.67 mm, SD 4.85, $P < 0.001$; control group: 3.00 mm, SD 3.68, $P < 0.01$).

The angle NSCo, an expression of the position of the articular complex, increased significantly in the control group (0.93º, SD 0.8, $P < 0.01$), while in the treated group it decreased, but not significantly (–0.39º, SD 2.95).

The linear measurements performed to OLp and parallel to OL showed horizontal changes in the position of the mandible. Condylion (OLp–Co) moved forward in the treated group but not significantly (–0.5 mm, SD 1.16), while it moved backward in the controls (0.80 mm, SD 0.82, $P < 0.01$).

There was a significant advancement of the mandibular structures in the treated group (GoMe: 2.36 mm, SD 1.76, $P < 0.001$; OLp–B: 5.17 mm, SD 3.27, $P < 0.001$; OLp–Pg: 5.14 mm, SD 3.25, $P < 0.001$); the distance OLp–Go also increased (2.44 mm, SD 2.26, $P < 0.01$). In the control group only the increase in GoMe value was significant (2.17 mm, SD 1.71, $P < 0.01$).

Table 2 Treatment group (n = 40): average and standard deviations (SD) before (T0) and after (T1) treatment.

Variables	T0	SD	T1	SD	Mean	SD	*P*
SNA (°)	81.83	3.03	81.33	2.81	–0.5	1	ns
SNB (°)	74.75	2.81	76.39	2.95	1.64	1.3	***
ANB (°)	7.08	1.81	4.94	1.40	–2.14	1	***
Ao–Bo (mm)	4.30	2.12	0.97	1.60	–3.33	1.43	***
Nperp–A (mm)	1.50	2.82	1.11	2.55	–0.39	1.22	ns
Nperp–Pg (mm)	–7.30	4.18	–5.14	5.01	2.17	2.09	***
NSCo (°)	135.11	4.62	134.72	4.55	–0.39	2.95	ns
Co–A (mm)	86.94	4.95	87.61	5.00	0.67	1.88	ns
Co–Gn (mm)	101.69	6.32	107.36	7.44	5.67	4.85	***
GoMe (mm)	65.61	4.88	67.97	5.39	2.36	1.76	***
FH^NA (°)	91.83	2.73	91.39	2.68	–0.44	1.2	ns
FH^NPg (°)	84.30	4.13	87.69	2.68	3.39	4.16	***
FMA (°)	23.28	4.01	23.75	4.42	0.47	1.83	ns
FH^OL (°)	10.02	2.14	11.28	2.69	1.25	2.24	*
SN^PP (°)	7.36	3.11	8.94	3.59	1.58	2.38	*
PP^GoMe (°)	26.39	5.03	26.14	4.61	–0.25	3.27	ns
N–ANS (mm)	48.55	3.72	51.00	3.82	2.44	2.07	***
ANS–Me (mm)	61.44	4.15	63.55	3.46	2.11	2.64	**
$\underline{1}$^FH (°)	115.25	4.60	109.61	4.51	–5.64	4.12	***
IMPA (°)	95.67	5.57	97.22	4.76	1.55	3.09	ns
Interincisal angle (°)	125.44	6.1	128.86	5.52	3.42	6.4	*
Overjet (mm)	8.72	1.83	3.69	1.46	–5.03	1.45	***
Overbite (mm)	4.53	2.00	3.36	1.61	–1.17	2	*
$\overline{1}$–OL (mm)	2.42	1.76	1.89	1.05	–0.53	1.41	ns
NCPgC^FH (°)	92.22	4.29	93.97	3.01	1.75	2.73	***
UL–EL (mm)	0.86	2.20	–1.14	1.97	–2	1.77	**
LL–EL (mm)	0.78	2.53	0.39	2.32	–0.39	2.3	ns
OLp–Co (mm)	10.89	2.11	10.39	2.17	–0.50	1.16	ns
OLp–A (mm)	75.53	3.71	76.50	3.92	0.97	1.55	*
OLp–B (mm)	70.67	3.6	75.83	4.28	5.17	3.27	***
OLp–Go (mm)	5.39	3.26	7.83	3.62	2.44	2.26	**
OLp–Pg (mm)	73.83	4.29	78.97	5.17	5.14	3.25	***
OLp–Ss (mm)	88.94	4.77	91.19	4.9	2.25	2.93	*
OLp–UL (mm)	93.25	4.9	95.00	5.28	1.75	3.41	ns
OLp–LL (mm)	90.44	4.70	94.19	5.18	3.75	3.40	***
OLp–Si (mm)	81.64	4.24	86.94	4.47	5.30	3.66	***
OLp–PgC (mm)	83.94	3.55	89.92	5.44	5.97	3.86	***
OLp–$\underline{\underline{1}}$ (mm)	82.67	4.60	82.86	4.29	0.19	3.10	ns
OLp–$\overline{1}$ (mm)	73.05	4.02	78.64	6.35	5.59	4.83	***
OLp–$\underline{6}$ (mm)	47.50	3.39	49.44	4.14	1.94	2.34	**
OLp–$\overline{6}$ (mm)	44.47	3.32	49.11	4.33	4.64	2.57	***

ns, not significant.
*P < 0.05; **P < 0.01; ***P < 0.001.

When the control findings were compared with those of the treated group, the activator therapy seemed to inhibit maxillary growth. OLp–A increased in both the control and treated groups, but the amount was greater in the controls (treatment group: 0.97 mm, SD 1.55, P < 0.05; control group: 2.23 mm, SD 1.37, P < 0.001).

The variable Co–A increased significantly in the control group (5.13 mm, SD 2.36, P < 0.001), but not in the treated subjects (0.67 mm, SD 1.88).

According to the variables SNA, NperpA and FH^NA, a restriction of forward growth of the maxilla occurred in the treated group, but this was not significant.

Vertical analysis

The measurements reflecting vertical development were similar in the treated and control groups.

Lower anterior (ANS–Me) and total anterior (N–Me) face height increased significantly in both groups.

FMA and FH^OL increased in the treated group (FMA: 0.47°, SD 1.83, ns; FH^OL: 1.25°, SD 2.24, P < 0.05) but decreased in the controls (FMA: –1.33°, SD 5.22; FH^OL: –0.13°, SD 1.90). This was not significant.

The inclination of the palatal plane (SN^PP) increased in the treated (1.58°, SD 2.38, P < 0.05) but not in the control group (–0.67°, SD 1.29, ns).

Dental analysis

Activator therapy moved the maxillary incisors palatally ($\underline{1}$^FH: –5.64°, SD 4.12, P < 0.001) and the significant correction in overjet, which averaged –5.03 mm (SD 1.45, P < 0.001), was due almost entirely to this palatal movement.

Table 3 Control group (n = 30): average and standard deviations (SD) before (T0) and after (T1) treatment.

Variables	T0	SD	T1	SD	Mean	SD	*P*
SNA (º)	83	1.64	83.33	1.48	0.33	0.49	ns
SNB (º)	77.33	2.25	77.50	2.28	0.17	0.41	ns
ANB (º)	5.70	1.03	5.83	1.57	0.13	0.61	ns
Ao–Bo (mm)	2.1	2.5	2.5	2.56	0.4	0.51	*
Nperp–A (mm)	3.13	0.72	3.07	1.53	–0.07	1.45	ns
Nperp–Pg (mm)	–2.47	2.41	–3.23	1.33	–0.77	2.06	ns
NSCo (º)	130.07	4.59	131.0	3.90	0.93	0.8	**
Co–A (mm)	86.33	4.32	91.47	2.92	5.13	2.36	***
Co–Gn (mm)	108.73	3.65	111.73	6.83	3	3.68	**
GoMe (mm)	69.83	4.50	72	3.75	2.17	1.71	**
FH^NA (º)	93.67	1.16	93.33	1.48	–0.33	1.63	ns
FH^NPg (º)	88.60	1.79	87.93	1.08	–0.67	1.76	ns
FMA (º)	20.67	1.48	19.33	5.23	–1.33	5.22	ns
FH^OL (º)	8.80	1.77	8.67	1.48	–0.13	1.90	ns
SN^PP (º)	8.67	1.91	8	1.84	–0.67	1.29	ns
PP^GoMe (º)	22.33	2.55	21.60	1.95	–0.73	1.71	ns
N–ANS (mm)	52.13	2.47	55.47	1.67	3.33	2.97	**
ANS–Me (mm)	64.67	3.28	67.33	4.4	2.67	1.95	**
$\underline{1}$^FH (º)	113.07	7.49	112.33	5.21	–0.73	4.08	ns
IMPA (º)	96.4	7.75	94.73	6.49	–1.67	1.29	**
Interincisal angle (º)	128	12.47	133.33	8.91	5.33	4.17	***
Overjet (mm)	6	1.84	5.87	2.5	–0.13	0.88	ns
Overbite (mm)	3.33	2.25	6.67	2.12	3.33	1.18	***
$\overline{1}$–OL (mm)	2.57	1.16	4.27	2.11	1.7	1.49	***
NCPgC^FH (º)	95.4	2.22	95.37	1.57	–0.03	1.8	ns
UL–EL (mm)	–0.90	1.59	–2.77	3.58	–1.87	2.19	*
LL–EL (mm)	–1.1	1.68	–1.03	2.46	0.07	3.54	ns
OLp–Co (mm)	9.8	2.64	10.6	1.95	0.80	0.82	**
OLp–A (mm)	76.83	3.99	79.07	3.88	2.23	1.37	***
OLp–B (mm)	74.67	5.55	76.33	4.93	1.67	2.97	ns
OLp–Go (mm)	4.13	3.96	4.3	4.75	0.17	3.55	ns
OLp–Pg (mm)	77.67	5.55	79.67	3.97	2	3.05	ns
OLp–Ss (mm)	90.73	3.57	92.67	2.68	1.93	2.22	*
OLp–UL (mm)	94.67	4.23	96.4	3.35	1.73	0.96	***
OLp–LL (mm)	91.67	5.55	94.93	4.28	3.27	1.33	***
OLp–Si (mm)	85.33	5.98	87.27	5.57	1.93	3.03	*
OLp–PgC (mm)	88.13	5.74	91.33	4.80	3.2	3.31	*
OLp–$\underline{1}$ (mm)	84	4.53	86	3.46	2	1.69	**
OLp–$\overline{1}$ (mm)	77.67	4.4	78.4	3.6	0.73	2.05	ns
OLp–$\underline{6}$ (mm)	50.17	4.21	53	2.35	2.83	2	***
OLp–$\overline{6}$ (mm)	48	4.76	50.67	3.92	2.67	2.55	**

ns, not significant.
*P < 0.05; **P < 0.01; ***P < 0.001.

The mandibular incisors moved labially, but while IMPA increased this was not significant (1.55º, SD 3.09).

Dental measurements performed to the reference line (OLp) and parallel to OL showed a forward movement of the lower incisors in the treated group (OLp-$\overline{1}$: 5.59 mm, SD 4.83, P < 0.001), while the upper incisors seemed stable (OLp-$\underline{1}$: 0.19 mm, SD 3.10, ns). In the treatment group, the variables interincisal angle, overbite and $\overline{1}$-OL also improved (interincisal angle: 3.42º, SD 6.4, P < 0.05; overbite: –1.17 mm, SD 2, P < 0.05; $\overline{1}$-OL: –0.53 mm, SD 1.41, ns).

In the control group, the position of the maxillary incisors and overjet did not change significantly ($\underline{1}$^FH: –0.73 mm, SD 4.08; overjet: –0.13 mm, SD 0.88); the lower incisors moved lingually (IMPA: –1.67º, SD 1.29, P < 0.01). By examining OLp-$\overline{1}$, the lower incisors did not show significant modifications in their position (OLp-$\overline{1}$: 0.73 mm, SD 2.05, ns) while OLp-$\underline{1}$ expressed a significant forward movement of the upper incisors (OLp-$\underline{1}$: 2 mm, SD 1.69, P < 0.01).

In the controls, the variables interincisal angle, overbite and $\overline{1}$-OL increased (interincisal angle: 5.33º, SD 4.17, P < 0.001: overbite 3.33 mm, SD 1.18, P < 0.001; $\overline{1}$-OL: 1.7 mm, SD 1.49, P < 0.001).

The mandibular (OLp-$\overline{6}$) and maxillary (OLp-$\underline{6}$) molars moved forward in both groups, but the amount was greater for the mandibular molars in the treated group (4.64 mm, SD 2.57, P < 0.001), while in the controls it was lower (2.67 mm, SD 2.55, P < 0.01).

Aesthetic analysis

The positive effects of treatment on the facial profile were accompanied by an increase in the aesthetic facial

angle, NCPgC^FH (1.75º, SD 2.73, $P < 0.001$), while in the control group there was a reduction in this parameter (–0.03º, SD 1.8, ns).

Although the soft tissue profile landmarks (OLp–PgC, OLp–LL, OLp–Si, OLp–Ss) showed forward growth in both groups, the amount was greater in the treated subjects. The soft tissue profile in the mandibular area (PgC, Si) was positioned significantly further anteriorly in the treated group.

The upper lip landmark (OLp–UL) did not move as far forward in the treated group as in the controls (treatment group: 1.75 mm, SD 3.41, ns; control group: 1.73 mm, SD 0.96, $P < 0.001$). The linear measurement UL–EL reduced more in the activator therapy subjects (–2 mm, SD 1.77, $P < 0.01$) than in the controls (–1.87 mm, SD 2.19, $P < 0.05$).

The horizontal position of the lower lip remained relatively unchanged in both groups, according to LL–EL (treatment group: –0.39 mm, SD 2.3, ns; control group: 0.07 mm, SD 3.54, ns), while the variable OLp–LL showed a significant anterior movement in both groups (treatment group: 3.75 mm, SD 3.40, $P < 0.001$; control group: 3.27 mm, SD 1.33, $P < 0.001$).

Table 4 shows the differences between the treatment and control groups using the Mann–Whitney test.

Table 4 Changes in the control and treatment groups from before (T0) to after (T1) treatment.

Variables	T1–T0 control group ($n = 30$)	SD	T1–T0 treated group ($n = 40$)	SD	*P*
SNA (º)	0.33	0.49	–0.5	1	*
SNB (º)	0.17	0.41	1.64	1.3	***
ANB (º)	0.13	0.61	–2.14	1	***
Ao–Bo (mm)	0.4	0.51	–3.33	1.43	***
Nperp–A (mm)	–0.07	1.45	–0.39	1.22	ns
Nperp–Pg (mm)	–0.77	2.06	2.17	2.09	**
NSCo (º)	0.93	0.8	–0.39	2.95	*
Co–A (mm)	5.13	2.36	0.67	1.88	***
Co–Gn (mm)	3	3.68	5.67	4.85	ns
GoMe (mm)	2.17	1.71	2.36	1.76	ns
FH^NA (º)	–0.33	1.63	–0.44	1.2	ns
FH^NPg (º)	–0.67	1.76	3.39	4.16	***
FMA (º)	–1.33	5.22	0.47	1.83	ns
FH^OL (º)	–0.13	1.90	1.25	2.24	ns
SN^PP (º)	–0.67	1.29	1.58	2.38	**
PP^GoMe (º)	–0.73	1.71	–0.25	3.27	ns
N–ANS (mm)	3.33	2.97	2.44	2.07	ns
ANS–Me (mm)	2.67	1.95	2.11	2.64	ns
$\underline{1}$^FH (º)	–0.73	4.08	–5.64	4.12	**
IMPA (º)	–1.67	1.29	1.55	3.09	***
Interincisal angle (º)	5.33	4.17	3.42	6.4	ns
Overjet (mm)	–0.13	0.88	–5.03	1.45	***
Overbite (mm)	3.33	1.18	–1.17	2	***
$\overline{1}$–OL (mm)	1.7	1.49	–0.53	1.41	***
NCPgC^FH (º)	–0.03	1.8	1.75	2.73	*
UL–EL (mm)	–1.87	2.19	–2	1.77	ns
LL–EL (mm)	0.07	3.54	–0.39	2.3	ns
OLp–Co (mm)	0.80	0.82	–0.50	1.16	**
OLp–A (mm)	2.23	1.37	0.97	1.55	*
OLp–B (mm)	1.67	2.97	5.17	3.27	*
OLp–Go (mm)	0.17	3.55	2.44	2.26	ns
OLp–Pg (mm)	2	3.05	5.14	3.25	*
OLp–Ss (mm)	1.93	2.22	2.25	2.93	ns
OLp–UL (mm)	1.73	0.96	1.75	3.41	ns
OLp–LL (mm)	3.27	1.33	3.75	3.40	ns
OLp–Si (mm)	1.93	3.03	5.30	3.66	*
OLp–PgC (mm)	3.2	3.31	5.97	3.86	*
OLp–$\underline{1}$ (mm)	2	1.69	0.19	3.10	*
OLp–$\overline{1}$ (mm)	0.73	2.05	5.59	4.83	***
OLp–$\underline{6}$ (mm)	2.83	2	1.94	2.34	ns
OLp–$\underline{\overline{6}}$ (mm)	2.67	2.55	4.64	2.57	ns

ns, not significant; SD, standard deviation.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

Discussion

There is a lack of consensus regarding the relative orthodontic/orthopaedic correction obtained by functional appliances during Class II treatment.

The purpose of the present study was to evaluate the changes occurring in subjects treated with an activator. In an attempt to determine if there are significant growth changes above what can be expected, a control group was examined. This appears to be the best method to differentiate growth changes from treatment changes.

Maxillary effects

The results show that an orthopaedic retraction of the maxillary complex seemed to be consistent. Point A was prevented from moving forward by 1.26 mm when measured parallel to OL in the activator group. This finding is in agreement with Pancherz (1984), who found that forward movement of point ANS was reduced 1.7 mm in the treated group in comparison with Bolton standards.

In the present analysis the angular measurement SNA decreased (–0.50º, SD 1) in the treated group, while in the controls it increased (0.33º, SD 0.49), even if the amounts were not significant.

During treatment with functional appliances, it has been claimed that forward growth of the maxilla may be inhibited (Demisch, 1972; Ahlgren and Laurin, 1976; Forsberg and Odenrick, 1981; Owen 1981; Luder, 1982; Bass, 1983; Creekmore and Radney, 1983; Pancherz, 1984; Vargervik and Harvold, 1985; Bishara and Ziaja, 1989; Moore *et al*., 1989), but dentoalveolar effects cannot be excluded (Björk, 1951; Weislander and Lagerström, 1979; Robertson, 1983; Derringer, 1990; Courtney *et al*., 1996).

In the present study dentoalveolar changes occurred in all treated patients, which contributed to the sagittal correction: lingual tipping of the maxillary incisors caused a resorption of point A.

Mandibular effects

It has been stated that an increase in mandibular growth is the distinguishing aspect of functional therapy with respect to other treatment modalities (Demisch, 1972; Eirew *et al*., Owen, 1981; Luder, 1982; McNamara *et al*., 1985; Perillo *et al*., 1996; Toth and McNamara, 1999), while others believe that mandibular length is unaltered by functional appliance therapy (Harvold and Vargervik, 1971; Weislander and Lagerström, 1979; Vargervik and Harvold, 1985; Jakobsson and Paulin, 1990) and that the treatment changes appear to be similar to those caused by growth (Björk, 1951; Forsberg and Odenrick, 1981).

In the present study there was advancement of mandibular structures in the activator group, when the cephalometric values related to the lower jaw were compared with the controls. Activator treatment resulted in approximately 3 mm of anterior mandibular displacement (OLp–B, OLp–Pg) when compared with the controls.

The increase in SNB of 1.64 degrees (SD 1.3) in the treated group, compared with the slight increase of 0.17 degrees (SD 0.41) in the controls was statistically significant. Mandibular length, expressed as GoMe, increased in both groups, but the difference was not significant.

The results of the present study were unable to show a significant additional increase in mandibular length. However they indicate an anterior displacement of the mandibular structures, which improved the jaw relationship and soft tissue facial profile.

These results may be related to changes in the condylar–glenoid fossa complex: remodelling and anterior relocation of the glenoid fossa may have contributed to the correction of the skeletal Class II malocclusion, as evidenced by the NSCo angle, which decreased significantly, and by linear measurement OLp–Co, which moved forward (Woodside *et al*., 1987; Ruf *et al*., 2001).

Effects on the dentition

Many authors (Ahlgren and Laurin, 1976; Pancherz, 1984; Courtney *et al*., 1996; Weiland *et al*., 1997) have observed significant dentoalveolar changes during activator treatment. In the present study, correction of upper incisor prominence appeared significant in the treated group. The overjet correction was due to a combined maxillary and mandibular orthopaedic effect, in addition to lingual movement of the upper dentition, in spite of the teeth being notched in the acrylic.

Activator therapy retroclined the maxillary incisors by 5.64 degrees (1^FH) and reduced the overjet by 5.03 mm, while the control group showed no significant differences during the observation period.

The upper dental component of the overjet correction is similar to data in the literature (Ahlgren and Laurin, 1976; Vargervik and Harvold, 1985; Derringer, 1990; Courtney *et al*., 1996; Weiland *et al*., 1997). Pancherz (1984) found that more than 50 per cent of overjet correction was produced by upper incisor tipping. The amount of upper incisor tipping may be significant during activator treatment: this means that the initial angulation of the upper incisors is of importance in influencing treatment outcome (Barton and Cook, 1997).

OLp-1, used for assessing the position of the upper incisors, demonstrated a statistically significant forward moving in the control group.

The mandibular incisors slightly proclined in the treated group and IMPA increased by 1.55 degrees, but this was not significant. Other studies have reported that the mandibular incisors procline or advance significantly during functional appliance treatment (Luder, 1982; Chang *et al*., 1989; Jakobsson and Paulin, 1990; Weiland *et al*., 1997), in spite of capping (Ahlgren and Laurin, 1976; Pancherz, 1984; Nelson *et al*., 1993). The incisal capping used in the design of the activator in this study prevented the amount of mandibular incisor proclination.

The dentoalveolar changes included intrusion of the maxillary and mandibular incisors, which improved the overbite.

Activator therapy caused dentoalveolar changes in the molar area. The acrylic did not contact the occlusal surfaces of the lower posterior teeth. Tooth contact with the appliance was maintained at the gingival margin distally on the mandibular and mesially on the maxillary premolars and molars.

In the present analysis, activator appliances resulted in approximately 2 mm of additional forward movement of the mandibular molars (when measured parallel to OL) but it was not statistically significant. The finding is in agreement with Weiland *et al*. (1997), Malmgren *et al*. (1987) and Vargervik and Harvold (1985) who concluded that the mandibular molars come forward with the mandible and not just by tooth migration.

Forward movement of the maxillary molars was reduced by 0.89 mm in the treated group in comparison with the controls, but the difference was not significant.

Effects on the vertical growth of the jaws and dentition

Activator therapy appears to increase vertical development of the mandible. A number of authors have found that the majority of mandibular growth is expressed vertically because of backward rotation of the mandible (Williams and Melsen, 1982; Creekmore and Radney, 1983; McNamara *et al*., 1985; Ruf *et al*., 2001).

An increase in face height in the first molar region disturbs the balance of vertical development and

thereby influences displacement of pogonion in a backward direction; variations in the vertical dimensions of the maxilla are thus related to the sagittal discrepancy. For this reason it appears that control of the vertical dimension is imperative for an optimal forward displacement of the mandible in the correction of a skeletal Class II malocclusion.

The results of the present study did now show significant modifications in the vertical development of the maxillomandibular complex: the angular measurements indicated a slight increase in angles FH^OL and SN^PP in the treated subjects, while FMA angle did not change significantly; in the controls the vertical relationship also seemed stable.

The vertical dental relationship expressed by the overbite is an important feature in functional therapy, associated with a good prognosis for treatment outcome (Charron, 1989). During activator therapy the incisors were passively prevented from erupting by the double capping as the molars erupted: these dental movements resulted in a statistically significant correction of the overbite in the treated group, while in the controls, overbite increased significantly ($\bar{1}$-OL: 1.7 mm, SD 1.49; overbite: 3.33 mm, SD 1.18).

Effects on aesthetics

The activator was constructed for the purpose of 'stimulating' mandibular growth and improving the patient's profile characterized by retrognathic mandible.

According to the observation of Bishara and Ziaja (1989), Forsberg and Odenrick (1981) and Remmer *et al.* (1985) soft tissue pogonion appears significantly further anteriorly due solely to the advancement of the mandible.

The aesthetic facial angle NCPgC^FH and the variable OLp–PgC showed an additional increase of 1.78 degrees and 2.77 mm, respectively, in the activator group in comparison with the controls.

Maxillary retraction and lingual movement of the maxillary incisors played an important role in upper lip retrusion, which appeared more prevalent in the treated group than in the controls. The lower lip moved anteriorly in both groups.

The final clinical effect was an improvement in facial appearance, due to anterior displacement of the mandibular structures, and lip balance.

Conclusions

The results indicate that the activator appliance is effective in treating mandibular deficiency. Functional therapy is of clinical benefit in actively growing patients and should be initiated during the middle to late mixed dentition period. Patient co-operation and the age at which functional appliance treatment is instituted are important for satisfactory correction of Class II malocclusions.

When the activator patients were compared with the untreated controls subjects, the results showed that therapy promoted a combination of skeletal and dental changes and improved the soft tissue facial profile.

Dentoalveolar effects seemed to play an important role in this correction, but a relative maxillomandibular displacement, mainly a mandibular advancement, was also determinant.

Further investigations on changes occurring in the condylar–glenoid fossa relationship might provide information concerning the remodelling processes responsible for Class II correction following activator treatment.

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