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The effects of maxillary protraction and its long-term stability—a clinical trial in Chinese adolescents

LinLing Chen, RongJing Chen, YiMing Yang, GuoPing Ji and Gang Shen
Department of Orthodontics, Ninth People's Hospital, College of Stomatology, Shanghai Jiao Tong University, China

Correspondence to: Professor Gang Shen, Department of Orthodontics, Shanghai Ninth People's Hospital, 639 Zhi-Zao-Ju Road, Shanghai, China. E-mail: gangshen@orthosh.com

SUMMARY The purpose of this study was to evaluate the effect of reverse pull headgear (RPHG) in the treatment of Class III malocclusions in the late mixed and early permanent dentition and its long-term stability at the time when facial growth was close to completion. The treatment group comprised 22 subjects (12 males and 10 females, mean age: 11.38 ± 0.69 years). The control group included 17 subjects (7 males and 10 females, mean age: 11.54 ± 1.07 years). The mean RPHG treatment time was 1.5 ± 0.95 years and the observation time for the control group was 1.75 ± 0.83 years. For each subject, lateral cephalograms were obtained before (T1) and after (T2) treatment or observation. These cephalograms were traced and analyzed and the differences between T1 and T2 values were examined with paired *t*-tests. Of the 22 treated cases, 10 patients were followed up until at the mean age of 16.18 years (T3). Since there was no relapse in anterior crossbite, the long-term effects of RPHG were evaluated by measuring the maxillary and mandibular skeletal changes. The follow-up patients were divided into two groups based on the change in ANB: a stable group (decrease in ANB < 2 degrees) and an unstable group (decrease in ANB > 2 degrees).

The skeletal effects of RPHG in treating Class III anomalies just before or at the beginning of the pubertal growth spurt include protraction of the maxilla and dentition and inhibition of forward growth of the mandible. With regard to the long-term change, a slight alteration in the position of the maxilla and in the position and growth direction of the mandible resulted in a slight decrease in ANB in the stable group. The slight retrusion in the maxilla, combined with the significant protrusion in the mandible and the more horizontal mandibular growth direction, resulted in a decrease in ANB in the unstable group. This indicated that the maxilla remained relatively stable and that the unstable factor was continuing mandibular growth during the pubertal and post-pubertal period. For patients with an excessive mandible, orthopaedic therapy should start at the beginning of pubertal growth and orthodontic fixed appliance should follow immediately after RPHG so that mandibular growth in the sagittal direction during puberty or even after pubertal growth may be effectively inhibited.

Introduction

Skeletal Class III malocclusions in growing children remain one of the most challenging problems in orthodontics. The incidence of this malocclusion in the Chinese population is 14.94 per cent in the primary, 9.65 per cent in the mixed, and 14.98 per cent in the early permanent dentition (Fu *et al.*, 2002). It has been suggested that the majority of subjects with a skeletal Class III malocclusion present with maxillary retrusion and a normal or prognathic mandible (Ellis and McNamara, 1984; Guyer *et al.*, 1986). In China, more than 70 per cent of skeletal Class III patients have a retrognathic maxilla with/without a prognathic mandible (Jin and Lin, 1985). Therefore, maxillary advancement by orthopaedic forces has been considered as a treatment option in young patients. Since the introduction of reverse pull headgear (RPHG) therapy, a number of studies have explored its treatment effects, which include infero-anterior movement of the maxilla and maxillary dentition, downward–backward rotation of the mandible, retroclination of the mandibular incisors, and an increase in lower face height (Hata *et al.*, 1987; Ishii *et al.*, 1987; Mermigos *et al.*, 1990; Baik, 1995; Ngan *et al.*, 1996; Nartallo-Turley and Turley, 1998). Based on clinical trials which compared the orthopaedic changes of treated Class III malocclusion subjects with the natural growth of untreated controls, the orthopaedic interventions were confirmed to be effective (Macdonald *et al.*, 1999; Baccetti *et al.*, 2000; Yüksel *et al.*, 2001; Vaughn *et al.*, 2005; Tortop *et al.*, 2007).

With regard to the optimal timing for orthopaedic treatment, disagreement exists. Many studies in the literature have supported early treatment to maximize maxillary anterior advancement and minimize the dentoalveolar effects (Takada *et al.*, 1993; Chong *et al.*, 1996; Shanker *et al.*, 1996; Kapust *et al.*, 1998; Baccetti et al., 2000). Some investigators, however, have found no relationship between the effect of maxillary protraction and treatment timing during pubertal growth (Takada *et al.*, 1993; Baik, 1995; Sung and Baik, 1998; Cha, 2003).

With early treatment, there is a significant amount of time between the end of protraction therapy and the cessation of the pubertal growth spurt. Thus, it is important not to ignore the changes occurring after RPHG treatment. In studies on the long-term efficacy of early RPHG therapy, 25–30 per cent of the patients were reported to have relapsed into a reverse overjet as mandibular growth exceeded maxillary growth in the horizontal direction (Hägg *et al.*, 2003; Westwood *et al.*, 2003; Baccetti *et al.*, 2004; Ghiz *et al.*, 2005; Wells *et al.*, 2006). Baccetti *et al.* (2004) reported that an increased posterior face height, an acute cranial base angle and a steep mandibular plane were all indicators of unfavourable long-term outcomes. Hägg *et al.* (2003) found that the indicators of unfavourable growth after orthopaedic treatment included forward positioning of the mandible relative to the cranial base, increased length of the mandibular body and ramus, and an increased gonial angle.

The aim of the present study was to evaluate the effects of RPHG in treating skeletal Class III malocclusions in the late mixed and early permanent dentitions and its long-term stability at the time when facial growth was close to completion.

Subjects and methods

Subjects grouping and treatment modalities

A total of 39 growing children were selected from the patients who presented to the Orthodontic Department of Shanghai Jiao Tong University for Class III malocclusion consultation and treatment (Shanghai Jiao Tong University Ethical Committee Approval No. 720A25). The inclusion criteria included a negative overjet, concave facial profile with maxillary retrusion; ANB < 1 degree; stage 2–4 in the cervical vertebral maturation (CVM) index; no previous orthodontic treatment; and no other craniofacial anomalies. The subjects were designated to either a treatment or control group.

The treatment group comprised 22 subjects (12 males and 10 females), with an age range of 10.1–13.2 years (mean 11.38 ± 0.69 years). Banded palatal expansion appliances were used for seven patients with a posterior crossbite. The expander was activated twice a day until the palatal cusp of the upper first molar contacted the lingual cusp of its lower counterpart. After a retention period of 1 month, the expander was replaced by an orthopaedic appliance, with two hooks in the maxillary canine region for RPHG elastics (Figure 1). This orthopaedic appliance was placed directly for the other 15 patients. The direction of the orthopaedic traction was 15–30 degrees downward from the occlusal plane, delivering a force of 250–300 g at each side. Although the subjects were instructed to wear the RPHG for 12 hours a day, most wore them for only 9–10 hours due to excessive homework and limited sleep. All patients were treated at least to a positive overjet before moving to the fixed appliance (FA) stage. The mean duration of RPHG treatment was 1.5 ± 0.95 years.

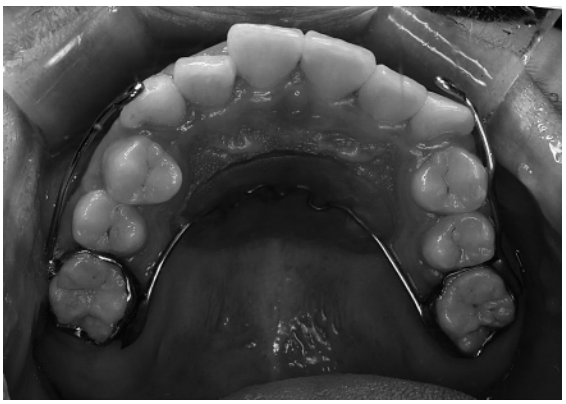

Figure 1 The intraoral appliance for orthopaedic protraction.

On completion of orthopaedic therapy, the subjects were recalled for follow-up. Of 22 treated subjects, only 10 patients (eight of whom underwent the FA phase) presented for the whole follow-up study. The mean follow-up period was 3 years (2–4.4 years) and the mean age 16.18 ± 0.60 years, where the patients were in the post-pubertal growth stage (CVM Index stage 5 or 6).

The control group comprised 17 subjects (seven males and 10 females), with an age range from 9.8 to 13.1 years (mean 11.54 ± 1.07 years) and consisted of those patients who refused or discontinued RPHG therapy. There was no statistically significant difference in average age between the treated and control groups. The average observation period for the control group was 1.75 ± 0.83 years, matching the orthopaedic duration of the treated group.

Data collection and statistical analysis

For each patient, lateral cephalograms were taken before (T1) and after (T2) orthopaedic treatment and at follow-up (T3) in the treated group or before (T1) and after (T2) the observation period in the control group. Each lateral cephalogram was traced and the variables were measured by the same investigator (LLC). The measurements were repeated twice 1 week apart and the mean values of the two measurements were used. Method error (ME) analysis for the cephalometric measurements was calculated using the formula:

$$ME = \sqrt{\frac{\sum d^2}{2n}}$$

where d is the difference between the two registrations of a pair and n is the number of double registrations. Ten measurements were selected randomly from the cephalometric readings for ME analysis. Paired t-tests were used to compare the two registrations. There was no significant difference between the two registrations.

Cephalometric analysis was conducted to identify the changes in eight angles and nine anatomical landmarks (Figure 2). The constructed grid consisted of a horizontal reference line (X), which was 6 degrees downward from the sella–nasion (SN) line at sella and a vertical reference line (Y), which was perpendicular to the horizontal reference line at sella (Cha, 2003). The means and standard deviations of the measurements were calculated and a paired *t*-test was performed using the Statistical Package for Social Sciences version 11.0 (SPSS Inc., Chicago, Illinois, USA).

The 10 follow-up cases had lateral cephalograms taken at T3. One patient had an edge-to-edge incisor relationship, and the others a positive overjet. As eight of the follow-up patients underwent the FA treatment phase and none relapsed to an anterior crossbite, the long-term effects of RPHG could not be evaluated as success or failure by the position of the incisors. It was therefore reasonable to evaluate the orthopaedic effects by measuring the maxillary and mandibular skeletal changes. The follow-up patients were therefore divided into two groups based on the changes in ANB: the stable group with a slight or no decrease in ANB (<2) and the unstable group with an obvious decrease in ANB (>2).

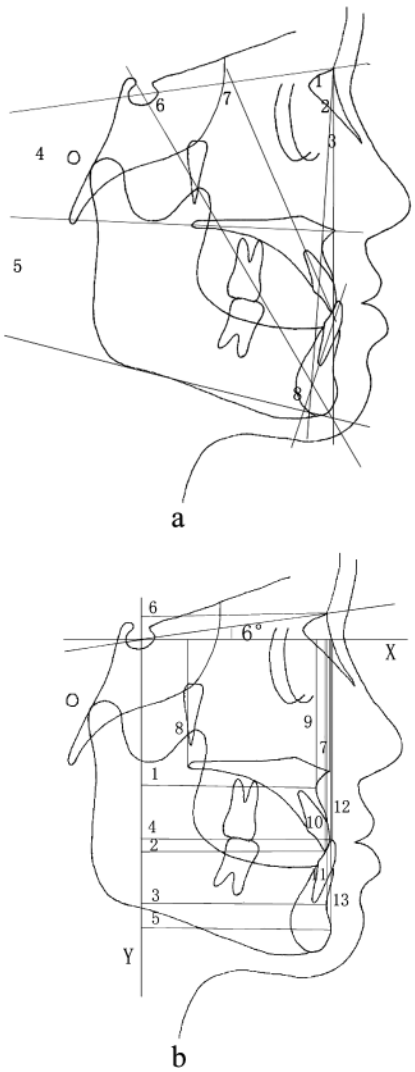

Figure 2 Landmarks and cephalometric measurements used in the study (a) angular (1)SNA (2)SNB (3)ANB (4)SN-PP (5)SN-MP (6)Y-axis (7) U1-SN (8)L1-MP. (b) Linear (1)Y-A (2)Y-Mx1 (3)Y-B (4)Y-Mn1 (5)Y-Pg (6) Y-N (7)X-ANS (8)X-PNS (9)X-A (10)X-Mx1 (11)X-B (12)X-Mn1 (13)X-Pg.

Results

The effects of RPHG

The cephalometric variables at T1 and T2 and the differences between the groups are shown in Tables 1–3.

When compared with the control group, the cephalometric measurements in the treated group revealed statistically significant forward movement of the maxilla, indicated by 3.93 mm forward positioning of point A ($P < 0.05$) and an increase of SNA by 2.25 degrees ($P < 0.001$). The decrease of PP-SN (−1.04 degrees, $P < 0.05$) indicated a statistically significant counterclockwise rotation of the maxilla (Table 3).

Forward growth of the mandible in the treated group was inhibited with slight retrusion evidenced by decreases at point B (−0.52 mm) and pogonion (−0.36 mm). Compared with forward movement at point B (+3.61 mm) and pogonion (+3.82 mm) in the control group, mandibular inhibition in the treated group was highly significant ($P < 0.001$). In the vertical dimension, there was a clockwise rotation of the mandible in the treated group, with a decrease in SNB (−1.18 degrees, $P < 0.001$) and an increase in the Y-axis (+1.63 degrees, $P < 0.01$) and SN-MP (+2.46 degrees, $P < 0.001$); lower face height increased but, compared with the vertical change in the control group, no statistically significant change in linear measurements was found in the treated group (point B: +5.87 mm, Pg: +7.32 mm; $P > 0.05$). The combined maxillary and mandibular changes in the treated subjects resulted in an increase in ANB angle (+3.42 degrees, $P < 0.001$; Table 3).

In the treated group, statistically significant forward movement of the maxillary incisors (+7.09 mm, $P < 0.01$) and slight forward positioning of the mandibular incisors (+0.37 mm, $P < 0.01$) was also found, while downward movement of the incisors was not significant ($P > 0.05$; Table 3).

Although the negative overjet was corrected in all treated subjects, it did not necessarily indicate a corresponding skeletal change. Large variations in skeletal changes were observed. These ranged from 0 to 5 degrees for SNA, −4 to 1.5 degrees for SNB, and 1.5–5.5 degrees for ANB (Figure 3A–3C).

Long-term stability of RPHG

Six follow-up patients were allocated to the stable group and the other four to the unstable group (Tables 4 and 5). Overall assessment of SNA (stable group: −0.17 degrees, unstable group: −0.88 degress), SNB (stable group: 0.05

Table 1 Cephalometric changes before (T1) and after (T2) observation in the control group ($n = 17$). SD, standard deviation.

Variables	T1		T2		T2–T1		*P* value
	Mean	SD	Mean	SD	Mean	SD	
SNA(°)	78.77	2.64	78.98	2.46	0.21	1.00	ns
SNB(°)	80.10	2.79	80.98	2.94	0.88	1.25	**
ANB(°)	−1.36	1.62	−2.01	1.70	−0.65	0.73	**
PP-SN(°)	11.21	2.79	11.53	3.36	0.32	1.01	ns
SN-MP(°)	35.82	4.66	35.25	4.98	−0.58	2.45	ns
Y-axis(°)	67.71	3.22	67.53	3.10	−0.18	0.97	ns
U1-SN(°)	106.37	8.90	107.38	8.22	1.02	2.47	ns
L1-MP(°)	86.32	5.89	85.59	5.90	−0.74	3.51	ns
Sagittal							
Y-A(mm)	60.98	3.43	63.37	3.84	2.39	1.94	***
Y-Mx1(mm)	64.77	5.45	68.04	5.83	3.28	2.21	***
Y-B(mm)	60.64	5.59	64.24	6.20	3.61	2.49	***
Y-Mn1(mm)	66.79	5.15	69.91	5.50	3.12	2.11	***
Y-Pg(mm)	61.06	6.13	64.88	7.06	3.82	2.63	***
Y-N(mm)	65.50	2.54	67.79	3.32	2.29	1.77	***
Vertical							
X-ANS(mm)	46.92	3.52	49.12	3.58	2.19	1.86	***
X-PNS(mm)	42.03	3.17	44.17	3.51	2.14	1.79	***
X-A(mm)	52.64	3.56	55.19	3.45	2.56	1.93	***
X-Mx1(mm)	73.78	4.03	77.55	3.71	3.77	2.44	***
X-B(mm)	94.06	6.01	98.07	5.28	4.01	3.06	***
X-Mn1(mm)	71.47	4.71	75.14	4.11	3.66	2.79	***
X-Pg(mm)	103.47	5.32	108.99	4.71	5.52	3.12	***

ns, not significant. $^{**}P < 0.01$; $^{***}P < 0.001$.

Table 2 Cephalometric changes before (T1) and after (T2) reverse pull headgear therapy in the treatment group ($n = 22$). SD, standard deviation.

Variables	T1		T2		T2–T1		*P* value
	Mean	SD	Mean	SD	Mean	SD	
SNA(°)	78.94	3.01	80.76	3.35	2.25	1.81	***
SNB(°)	81.46	3.88	79.76	3.97	−1.18	1.94	***
ANB(°)	−2.52	1.67	1.00	1.72	3.42	1.15	***
PP-SN(°)	10.20	3.00	9.15	2.74	−1.04	1.98	**
SN-MP(°)	32.37	5.42	34.83	5.55	2.46	1.75	***
Y-axis(°)	67.39	3.49	69.02	3.95	1.63	2.04	***
U1-SN(°)	105.02	5.37	112.87	6.35	7.85	5.56	***
L1-MP(°)	87.07	5.99	87.13	6.34	0.06	3.80	ns
Sagittal							
Y-A(mm)	60.29	3.23	64.22	3.99	3.93	2.06	***
Y-Mx1(mm)	63.22	5.04	70.30	4.75	7.09	3.13	***
Y-B(mm)	61.41	4.84	60.89	6.08	−0.52	3.10	ns
Y-Mn1(mm)	66.57	3.83	66.94	4.58	0.37	2.63	ns
Y-Pg(mm)	61.95	5.37	61.59	6.80	−0.36	3.43	ns
Y-N(mm)	64.83	3.42	66.72	3.59	1.89	1.31	***
Vertical							
X-ANS(mm)	45.63	3.23	47.78	3.35	2.15	2.22	***
X-PNS(mm)	43.07	3.28	45.80	3.05	2.74	1.62	***
X-A(mm)	51.54	2.96	53.63	2.89	2.09	2.08	***
X-Mx1(mm)	71.89	3.99	74.87	3.60	2.97	2.76	***
X-B(mm)	90.37	5.05	96.24	5.31	5.87	3.18	***
X-Mn1(mm)	67.94	4.21	72.78	4.97	4.85	3.40	***
X-Pg(mm)	100.70	5.47	108.02	6.13	7.32	3.73	***

ns, not significant. $^{**}P < 0.01$; $^{***}P < 0.001$.

Table 3 Comparison of changes between the control (n = 17) and treatment (n = 22) groups before (T1) and after (T2) observation or reverse pull headgear therapy. SD, standard deviation.

Variables	Control: T2–T1		Treatment: T2–T1		*P* value
	Mean	SD	Mean	SD	
SNA(°)	0.21	1.00	2.25	1.81	***
SNB(°)	0.88	1.25	−1.18	1.94	***
ANB(°)	−0.65	0.73	3.42	1.15	***
PP-SN(°)	0.32	1.01	−1.04	1.98	*
SN-MP(°)	−0.58	2.45	2.46	1.75	***
Y-axis(°)	−0.18	0.97	1.63	2.04	**
U1-SN(°)	1.02	2.47	7.85	5.56	***
L1-MP(°)	−0.74	3.51	0.06	3.80	ns
Sagittal					
Y-A(mm)	2.39	1.94	3.93	2.06	*
Y-Mx1(mm)	3.28	2.21	7.09	3.13	**
Y-B(mm)	3.61	2.49	−0.52	3.10	***
Y-Mn1(mm)	3.12	2.11	0.37	2.63	**
Y-Pg(mm)	3.82	2.63	−0.36	3.43	***
Y-N(mm)	2.29	1.77	1.89	1.31	ns
Vertical					
X-ANS(mm)	2.19	1.86	2.15	2.22	ns
X-PNS(mm)	2.14	1.79	2.74	1.62	ns
X-A(mm)	2.56	1.93	2.09	2.08	ns
X-Mx1(mm)	3.77	2.44	2.97	2.76	ns
X-B(mm)	4.01	3.06	5.87	3.18	ns
X-Mn1(mm)	3.66	2.79	4.85	3.40	ns
X-Pg(mm)	5.52	3.12	7.32	3.73	ns

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

degrees, unstable group: 2.50 degrees), and Y-axis (stable group: −0.01 degrees, unstable group: −2.38 degrees; Figure 3) at T2 and T3 indicated that the position of the maxilla and the position and growth direction of the mandible in the stable group remained almost unchanged, apart from a slight decrease in ANB, while in the unstable group, the maxilla became more retrusive, combined with a marked protrusive mandible and a more horizontal mandibular growth direction, resulting in a significant decrease in ANB. Although the changes in the unstable group were unfavourable, the low growth potential greatly reduced the risk of relapse.

Discussion

This study revealed that RPHG therapy protruded the maxilla and inhibited forward growth of the mandible. Compared with the obvious forward movement in the control group, mandibular inhibition in sagittal growth in the treated group was very effective (Table 3, Figure 3B). The counterclockwise-rotated maxilla and clockwise-rotated mandible resulted in downward and backward repositioning of the chin. Although lower face height increased, no statistically significant change was found in linear measurements compared with the control group. This was due to the significant forward and downward mandibular

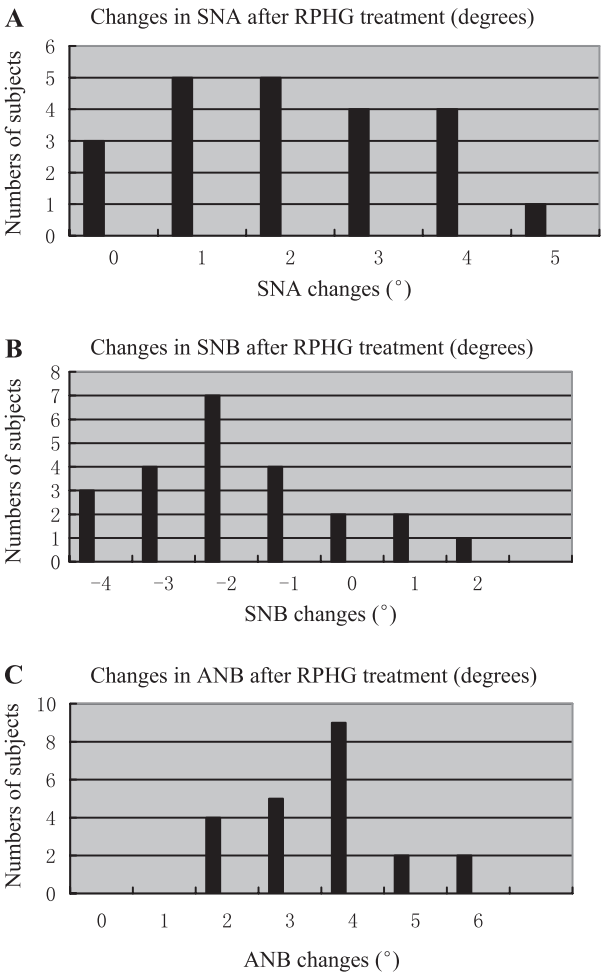

Figure 3 Changes in (A) SNA, (B) SNB, and (C) ANB after reverse pull headgear therapy.

Table 4 Variables before (T1), after (T2) reverse pull headgear treatment and at follow-up (T3) in the stable group.

Case		Age (years)	SNA (°)	SNB (°)	ANB (°)	Y-axis (°)
1. Male	T1	13.6	81.0	83.0	−2.0	67.0
	T2	14.7	85.0	81.5	3.5	69.0
	T3	16.7	85.0	82.0	3.0	69.0
2. Male	T1	12.2	79.0	81.0	−2.0	69.0
	T2	13.8	80.0	78.0	2.0	74.0
	T3	16.1	81.0	79.5	1.5	72.0
3. Female	T1	10.9	79.0	81.0	−2.0	68.0
	T2	12.0	81.0	79.0	2.0	69.5
	T3	16.0	79.5	79.0	0.5	69.5
4. Female	T1	12.7	76.5	80.5	−4.0	66.5
	T2	13.6	78.0	79.0	−1.0	67.5
	T3	15.6	78.0	79.3	−1.3	66.5
5. Male	T1	12.3	76.5	80.0	−3.5	69.5
	T2	13.3	77.5	76.0	1.5	73.0
	T3	15.7	79.0	76.0	3.0	73.0
6. Male	T1	10.5	77.0	81.0	−4.0	66.5
	T2	14.1	80.0	78.0	2.0	70.0
	T3	17.5	78.0	76.0	2.0	72.5

Table 5 Variables before (T1), after (T2) reverse pull headgear treatment, and at follow-up (T3) in the unstable group.

Case		Age (years)	SNA (°)	SNB (°)	ANB (°)	Y-axis (°)
7. Female	T1	10.1	82.0	82.0	0.0	64.5
	T2	11.1	85.5	81.5	4.0	64.5
	T3	15.5	85.0	84.5	0.5	61.5
8. Male	T1	12.0	81.0	86.0	−5.0	61.5
	T2	13.5	84.0	84.0	0.0	65.0
	T3	16.5	83.0	85.0	−2.0	63.5
9. Female	T1	11.7	75.5	75.0	0.5	74.0
	T2	12.5	75.5	72.0	3.5	77.0
	T3	15.9	74.0	75.5	−1.5	73.0
10. Male	T1	11.3	74.5	74.5	0.0	73.0
	T2	13.2	77.5	75.5	2.0	72.0
	T3	16.3	77.0	78.0	−1.0	71.0

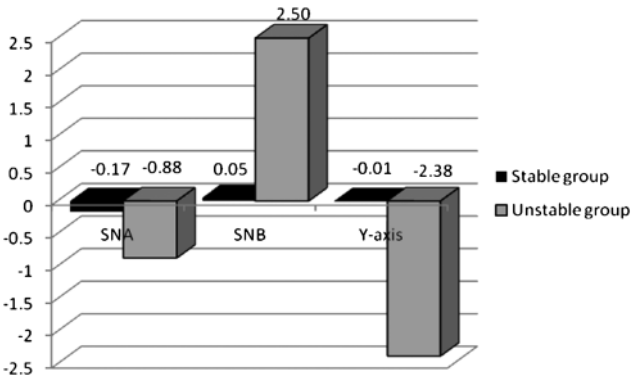

Figure 4 Changes in SNA, SNB, and *Y*-axis in the stable and unstable groups after reverse pull headgear treatment and at the end of follow-up.

growth occurring in the majority of the control subjects. The change in ANB was mainly caused by forward movement of the maxilla, suggesting that effective skeletal change can be obtained with RPHG therapy even though treatment started at the beginning of the growth spurt. The changes in SNA, SNB, and ANB in this study were similar to those of subjects treated during the pre-pubertal growth peak (Cha, 2003). Contrary to other studies (Baccetti *et al.*, 1998; Kapust *et al.*, 1998; Macdonald *et al.*, 1999), there was no retroclination of the mandibular incisors perhaps due to the relatively lower orthopaedic force and short daily wear of the RPHG. It has been emphasized that treatment changes provoked by orthopaedic forces include treatment effects and natural growth, especially for those treated over a long period of time. Many researchers have found that if a Class III malocclusion is allowed to develop without orthopaedic intervention, the skeletal pattern worsens (Battagel, 1993; Miyajima *et al.*, 1997; Deguchi *et al.*, 2002). In this study, it was also noted that in the control group, SNB increased more than SNA, resulting in a decrease of ANB during the observation period (Table 1). It is therefore reasonable to assume that by subtracting natural growth from the skeletal changes, the increase in SNA and decrease in SNB observed resulted from the effects of orthopaedic treatment.

A commonly used criterion to assess the success of treatment and its long-term stability in Class III malocclusion subjects is a positive overjet. However, this only represents a correction of the dental relationship that may not necessarily indicate a correction of the skeletal discrepancy. In this study, the changes in SNA and SNB after RPHG therapy revealed that in three subjects, there was no improvement in the position of the maxilla (Figure 3A) while mandibular protrusion remained in the other three subjects (Figure 3B). However, when assessing skeletal change by ANB angle, at least 1.5 degrees of improvement was achieved (Figure 3C). Therefore, a comprehensive approach must be adopted to evaluate the overall skeletal effects of RPHG, i.e., subjects with no maxillary protraction should be also evaluated by taking the mandibular retrusive effect into account and *vice versa*

It is recognized that the growth pattern of the maxilla modified by orthopaedic protraction may not be sustainable and may return to its original Class III growth pattern (Chong *et al.*, 1996; Shanker *et al.*, 1996; Miyajima *et al.*, 1997; Ngan *et al.*, 1997; Gallagher *et al.*, 1998; Macdonald *et al.*, 1999). This is supported by the findings of the present study where a slight retrusive maxillary position was identified in the stable and unstable groups during the follow-up stage (Figure 4).

Large variations in the maxillary growth pattern after RPHG were found in some patients: a mild response to treatment was followed by continuing improvement of maxillary growth (cases 2 and 5); however, a good response was followed by either an unchanged growth pattern (cases 1, 7, and 10) or even an unfavourable growth pattern (cases 3, 6, and 8). Similar scenarios were also found in the post-RPHG mandibular growth pattern, suggesting that there is no correlation between maxillary and mandibular response and the subsequent growth patterns (Tables 4 and 5). This adds to the difficulty in predicting growth tendency. Some researchers have reported that downward–backward rotation of the mandible during RPHG increases the risk of long-term treatment failure (Hägg *et al.*, 2003; Wells *et al.*, 2006). This was not supported by the present findings, where an obvious rotation of the mandible during RPHG was found in cases 2, 5, and 9, but their follow-up changes varied. In case 2, mild forward growth of the mandible caused a slight decrease in ANB; in case 5, the mandibular position was stable after treatment and the maxilla had more forward growth, resulting in an increase in ANB; and in case 9, excessive sagittal mandibular growth led to a significant decrease in ANB (Tables 4 and 5).

Many researchers recommend early treatment, i.e., during the primary or early mixed dentition in order to obtain more maxillary skeletal effects. Late treatment, alternatively, may result in more mandibular rotation and alveolar change (Takada *et al.*, 1993; Shanker *et al.*, 1996;

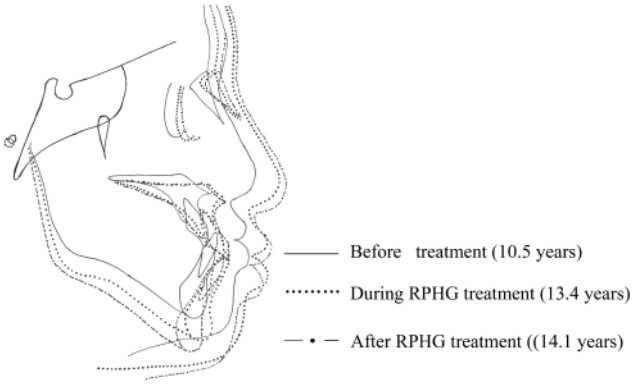

Figure 5 Case no. 6. Cephalometric tracings before, during, and after reverse pull headgear treatment superimposed on the nasion–sella line with sella as the registration point.

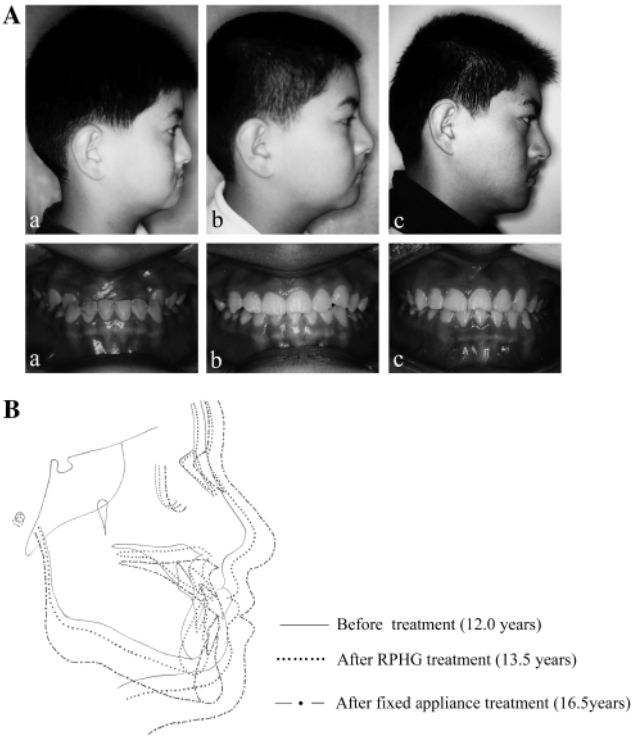

Figure 6 Case no. 8. (A) The profile and incisor relationship (a) before and (b) after reverse pull headgear therapy and (c) after fixed appliance (FA) treatment. (B) Cephalometric tracings before, after reverse pull headgear treatment, and after FA treatment superimposed on the nasion–sella line with sella as the registration point.

Chong *et al.*, 1996; Kapust *et al.*, 1998; Baccetti *et al.*, 2000). It is important that the timing of early treatment is prior to the pubertal growth spurt; at this point, forward growth of the maxilla falls behind that of the mandible (Miyajima *et al.*, 1997; Deguchi *et al.*, 2002). Thus, an immediate effect from early treatment may not necessarily lead to long-term skeletal correction due to the differential growth rate in the jaws during pubertal growth. The long-term effects of Class III correction can be assessed only if the subjects have reached the skeletal age of the post-pubertal growth spurt. Many studies support the view that the change in SNB after RPHG therapy usually decides the long-term stability of Class III malocclusions (Sugawara *et al.*, 1990; Ishikawa *et al.*, 1998; Hägg *et al.*, 2003; Westwood *et al.*, 2003; Baccetti *et al.*, 2004; Ghiz *et al.*, 2005; Wells *et al.*, 2006); this was verified in the present study, where in the stable group, SNB slightly increased, whereas in the unstable group, it markedly increased (Figure 4). This further suggests that the major factor that determines long-term success with RPHG is not the response of the maxilla to forward traction but the amount and direction of mandibular growth during and after adolescence.

It is therefore essential to restrict excessive mandibular growth in the horizontal direction when treating a Class III malocclusion. In the present study, the inhibition of mandibular horizontal growth by RPHG proved to be effective, and the cephalometric parameters of the patients with a long treatment duration showed complete inhibition of mandibular sagittal growth (Figure 5). This may suggest a positive correlation between the length of treatment and the inhibiting effects of RPHG. More importantly, the present study demonstrated a favourable maxillary response to orthopaedic force even at the early permanent dentition stage. This finding agrees with some studies that found no relationship between the effect of maxillary protraction and treatment timing before the post-pubertal growth peak (Takada *et al.*, 1993; Baik, 1995; Sung and Baik, 1998; Cha, 2003). Furthermore, if possible, FA treatment should start immediately after RPHG therapy at the peak or post-peak stage. Lin and Gu (2006) reported that mandibular horizontal growth was inhibited even in severe Class III malocclusion subjects through FA. Thus, by controlling mandibular growth direction through RPHG and then FAs, sagittal growth of the mandible during the pubertal or even post-pubertal stage can be inhibited, and the risk of relapse can be reduced. As shown in case 8 in this study, despite effective RPHG treatment, excessive mandibular growth made this case susceptible to relapse. However, the mandibular growth direction was controlled through FAs with molar extrusion during the post-pubertal growth peak. Cephalometric superimposition showed that there was significant mandibular growth after RPHG treatment, and growth in the horizontal direction was less than that in the vertical direction. The positive overjet was thus maintained (Figure 6).

Conclusions

1. Reliable skeletal effects in treating Class III anomalies can be achieved by starting RPHG treatment just before or at the beginning of the pubertal growth spurt. The effects include protrusion of the maxilla and dentition and inhibition of forward growth of the mandible. The longer the period of orthopaedic treatment, the greater the long-term effects.

2. In general, SNA was stable during the follow-up stage and there seems to be no relationship between the maxillary response and the follow-up growth pattern. The change in SNB after RPHG therapy is associated with the mandibular sagittal growth pattern during the pubertal and post-pubertal stage.
3. For patients with an excessive mandible, it is suggested that orthopaedic therapy should start at the beginning of pubertal growth. FA treatment should follow immediately after the completion of RPHG therapy so that mandibular growth in the sagittal direction during pubertal growth may be effectively inhibited.

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