A cephalometric evaluation of anterior openbite correction with the magnetic active vertical corrector

By Raymond E. Barbre, DDS, MS; and Peter M. Sinclair, DDS, MSD

The diagnosis and treatment of malocclusions characterized by anterior openbite continues to be one of the more difficult problems facing the practicing orthodontist. Anterior openbite has been defined as "that condition where upper incisor crowns fail to overlap the incisal third of the lower incisor crowns when the mandible is brought into full occlusion".[1,2] Many factors have been suggested as potential causes of openbite and they can be divided into three main categories:[3,4]

1. Vertical skeletal growth discrepancies, defined as excessive vertical eruption of the maxillary molars and alveolus that subsequently hinges the mandible down and back.[5-7] In addition, factors such as underdevelopment of the middle cranial fossa height producing an elevation of the glenoid fossa,[6,8] and inadequate alveolar growth in the anterior portion of the maxilla have been suggested as potential causative factors.[8]

2. Abnormal muscular and soft tissue development, particularly when associated with airway problems, may lead to increased anterior facial height openbite.[9-11] In the past, researchers have also credited the postural and swallowing positions of the tongue with causing anterior openbites.[12] A more widely accepted hypothesis is that tongue thrust and orofacial musculature imbalance are secondary factors possibly enhancing or maintaining the underlying skeletal or dental dysplasia, rather than being primary causative factors.[7,13]

3. Many researchers have related and documented thumb sucking and other habits to the formation and maintenance of anterior openbites,[14-17] and have suggested a wide range of treatment techniques directed at eliminating the obvious causative factors.[18-19]

In cases where the openbite etiology is not so clearly defined, treatment has been aimed at controlling the patient's vertical growth. High pull headgear is a popular approach to the correction of anterior openbite, particularly when

Abstract

The purpose of this study was to evaluate treatment effects of the magnetic active vertical corrector appliance (AVC) when used to treat anterior openbites in a sample of growing patients. Twenty-five patients with a mean age of 10 years 8 months underwent AVC treatment for an average of almost 8 months. They had an average of 3 mm of anterior openbite closure during the treatment period, primarily due to maxillary and mandibular molar intrusion. Additional contributions to correction of the openbite were related to maxillary incisor eruption and lingual tipping combined with mandibular incisor lingual movement. A small amount of mandibular rotation with closure of the bite and decrease in anterior force height was observed. Anteroposterior skeletal change attributed to the AVC therapy was minimal.

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Key Words

Anterior openbite • Active vertical corrector • Molar intrusion

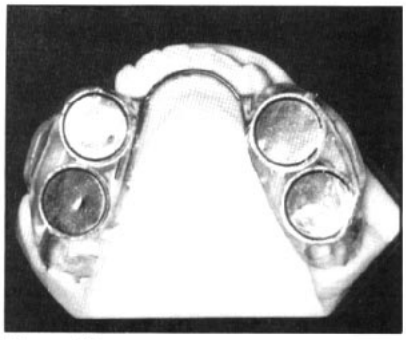

Figure 1A

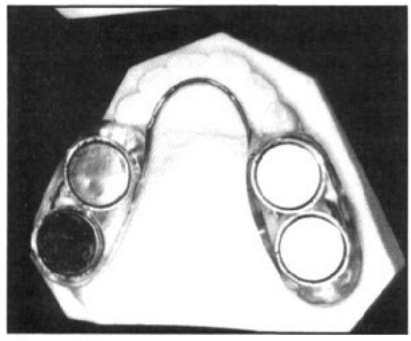

Figure 1B

Figure 1A and B
Occlusal view of the active vertical corrector.

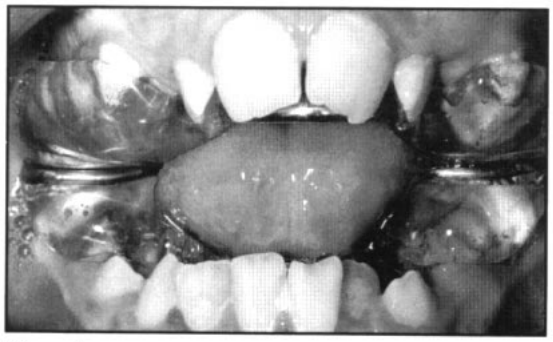

Figure 2

Figure 2
Intraoral view of the active vertical corrector.

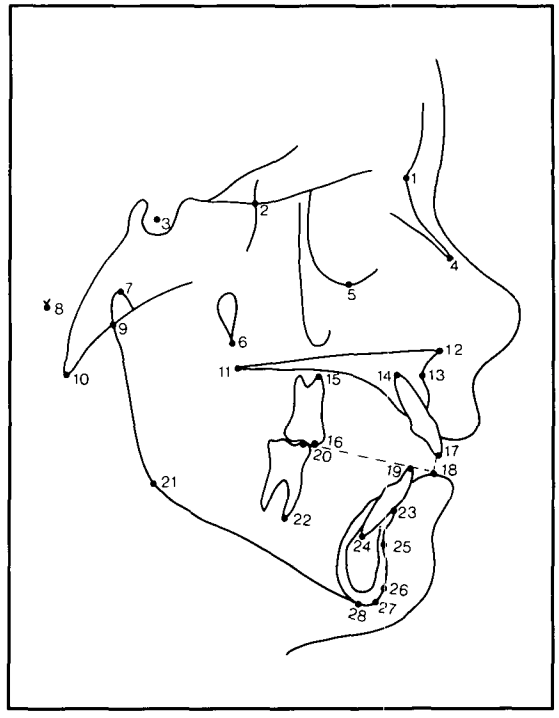

Figure 3

Figure 3
Cephalometric points recorded.

posterior maxillary dentoalveolar excess is suspected of playing a role in the etiology.[6,7,15,20,21] Experimental studies have documented sutural as well as dental changes associated with headgear use.[22,23] Successful high pull headgear therapy has been found to inhibit vertical maxillary development and allow for a forward rotation of the mandible, closing the bite.[21,23,24]

An alternative approach to openbite correction uses posterior bite blocks to achieve inhibition of maxillary posterior dentoalveolar development, which encourages subsequent bite closing mandibular autorotation. This is achieved by preventing the maxillary and mandibular posterior teeth from erupting and by opening the bite several millimeters so as to stretch the posterior muscles of mastication causing them to act as intrusive agents on the maxilla.[25,27] Bite block therapy has included appliances such as activators and bionators which can be thought of as removable posterior bite planes, as well as spring loaded bite planes which deliver an additional mechanical intrusive force to the maxillary posterior segments.[28]

The active vertical corrector (AVC) is an appliance developed for the non-surgical correction of anterior openbites.[29] It uses somarium cobalt magnets embedded in separate maxillary and mandibular bite blocks and delivers intrusive forces to the maxilla and mandible via the repulsive effects of the opposing magnets as well as the conventional bite block effect (Figures 1 and 2). Initial research on the AVC in primates has demonstrated some promising intrusive effects but with the side effect of producing lateral shifts of the mandible.[30,31] To date, no research has been conducted to evaluate the potential of the AVC for correcting anterior openbite in humans. The purpose of this study was to cephalometrically evaluate the treatment effects of a standard AVC design used to treat anterior openbites in a sample of growing patients.

Materials and methods

The sample in this study consisted of 25 patients with anterior openbites. Pretreatment criteria included having had no primary or permanent tooth extractions prior to or during AVC therapy, and no concurrent orthodontic treatment during AVC therapy. All patients had either a skeletal openbite with a high mandibular plane angle, or had previous tongue/thumb crib therapy which did not improve the openbite. At least 6 months separated the end of crib therapy and the insertion of the AVC. The sample's mean pretreatment age was 10 years 8 months (8 yrs 3 mo to 13 yrs 5 mo) and the average treatment time was 7.7 months (2.0 to 17.0 mo). The appliance was worn until the clinician felt that conventional orthodontic therapy could complete the treatment.

Each patient wore a standardized AVC developed by Dr. Eugene Dellinger of Fort Wayne, Indiana and made in his laboratory.* The appliances consisted of somarium cobalt magnets embedded in separate maxillary and mandibular bite blocks (Figure 1) with the right and left quadrants being connected anteriorly by a rigid

*Active Vertical Correctors Inc., Fort Wayne, Indiana.

Table 1
Pretreatment comparison of AVC and control groups

	Measurement	Experimental ($\bar{X} \pm$ S.D.)	Control ($\bar{X} \pm$ S.D.)	Difference (p)
Anteroposterior skeletal measurements	1. SNA °	82.1±3.3	81.0±0.26	1.1
	2. Ar-A mm	84.0±4.8	— —	—
	3. SNB °	77.4±3.6	77.0±0.5	0.4
	4. ANB °	4.6±2.5	3.9±0.2	0.7
	5. Ar-B mm	94.2±5.8	98.4±3.5	-4.2**
Rotational measurements	6. MPA °	29.8±4.8	28.6±0.9	1.2
	7. SN-MP °	38.4±5.6	34.4±0.6	4.0**
	8. SN-FH °	8.6±3.2	5.8±0.7	2.8***
	9. SN-PP °	3.5±2.7	7.4±0.6	-3.9***
	10. SN-MxOP °	13.9±4.1	— —	—
	11. SN-MnOP °	19.1±5.0	— —	—
Vertical skeletal measurements	12. N-ANS mm	48.3±2.7	52.6±2.2	-4.3***
	13. ANS-Me mm	69.9±4.9	66.8±2.7	3.1*
	14. N-Me mm	116.4±6.6	117.2±4.8	-0.8
	15. ANS-FH mm	19.3±2.2	— —	—
	16. PNS-FH mm	23.6±2.7	— —	—
	17. S-Go mm	72.6±5.8	72.3±3.9	0.3
	18. Ar-Go mm	44.4±4.7	43.4±2.4	1.0
Incisor position measurements	19. U1-SN °	108.9±5.5	104.7±0.8	4.2***
	20. U1-PP °	112.6±5.9	112.2±1.3	0.4*
	21. U1-PP mm	26.9±2.8	27.9±1.8	-1.0
	22. U1-FH mm	46.5±4.2	— —	—
	23. U1-NA mm	5.1±2.5	3.9±0.5	1.2*
	24. U1-L1 °	119.8±8.9	126.5±1.0	-6.7***
	25. L1-MP °	93.4±6.9	94.1±0.7	-0.7
	26. L1-NB mm	5.3±2.0	4.6±0.4	-0.7
	27. L1-MP mm	39.6±2.7	40.7±1.9	-1.1
	28. OB mm	-3.0±1.6	— —	—
	29. OJ mm	4.9±2.2	— —	—
Molar position measurements	30. U6-SN °	72.7±6.3	— —	—
	31. U6-PP °	76.4±6.6	— —	—
	32. UM-PP mm	21.0±2.8	21.0±1.6	0.0
	33. UM-FH mm	43.3±4.0	— —	—
	34. L6-MP °	83.4±4.8	— —	—
	35. LM-MP mm	30.7±2.4	30.8±1.5	-0.1

* indicates significance at $p < 0.05$ level
** indicates significance at $p < 0.01$ level
*** indicates significance at $p < 0.001$ level
— indicates no data available

Table 2
Posttreatment comparison of AVC and control groups

	Measurement	Experimental ($\bar{X} \pm$ S.D.)	Control ($\bar{X} \pm$ S.D.)	Difference (p)
Anteroposterior skeletal measurements	1. SNA °	81.9±3.6	80.9±0.3	1.0
	2. Ar-A mm	84.4±5.0	— —	—
	3. SNB °	78.6±3.6	77.2±0.5	1.4
	4. ANB °	3.3±2.7	3.8±0.3	-0.5
	5. Ar-B mm	96.0±5.8	99.8±3.9	-3.8*
Rotational measurements	6. MPA °	28.8±4.3	28.0±1.3	0.8
	7. SN-MP °	37.4±5.6	34.2±0.7	3.2**
	8. SN-FH °	8.6±2.9	5.8±0.7	2.8***
	9. SN-PP °	3.4±3.1	7.6±0.7	-4.2***
	10. SN-MxOP °	17.0±5.0	— —	—
	11. SN-MnOP °	15.5±6.2	— —	—
Vertical skeletal measurements	12. N-ANS mm	49.0±3.2	53.6±2.4	-4.6***
	13. ANS-Me mm	68.9±4.9	67.7±3.2	1.2
	14. N-Me mm	116.4±7.4	119.0±5.5	-2.6
	15. ANS-FH mm	19.9±2.4	— —	—
	16. PNS-FH mm	24.0±2.8	— —	—
	17. S-Go mm	73.5±5.7	73.9±4.4	-0.4
	18. Ar-Go mm	45.3±4.7	44.4±2.8	0.9
Incisor position measurements	19. U1-SN °	106.4±5.0	104.7±1.0	1.7
	20. U1-PP °	109.7±4.6	112.2±1.4	-2.5*
	21. U1-PP mm	28.0±4.6	28.3±1.7	-0.3
	22. U1-FH mm	48.1±4.2	— —	—
	23. U1-NA mm	4.6±2.5	4.1±0.3	0.5
	24. U1-L1 °	126.9±7.3	126.8±1.4	0.1
	25. L1-MP °	89.5±6.3	94.3±0.6	-4.8
	26. L1-NB mm	4.5±2.2	4.7±0.3	-0.2
	27. L1-MP mm	40.2±2.9	41.4±2.1	-1.2
	28. OB mm	0.0±1.6	— —	—
	29. OJ mm	4.1±2.4	— —	—
Molar position measurements	30. U6-SN °	73.8±4.8	— —	—
	31. U6-PP °	77.5±5.1	— —	—
	32. UM-PP mm	20.4±3.1	21.7±1.8	-1.3
	33. UM-FH mm	43.0±4.5	— —	—
	34. L6-MP °	83.0±5.7	— —	—
	35. LM-MP mm	30.3±2.5	31.4±1.8	-1.1

* indicates significance at $p < 0.05$ level
** indicates significance at $p < 0.01$ level
*** indicates significance at $p < 0.001$ level
— indicates no data available

metal bar. The magnets were embedded in acrylic in such a manner that approximately 600 gr of repelling force per side was generated when brought together. The interocclusal opening required for the appliance was about 7 mm (Figure 2). The AVC was cemented to the teeth and worn 24 hours/day including during eating. No TMJ symptoms were noted and patient compliance (assessed by whether the appliance was in place or not) was good.

Lateral cephalograms were taken prior to treatment (T1), and immediately posttreatment (T2). Twenty-eight points were recorded on the cephalometric tracings (Figure 3) and 35 cephalometric parameters were evaluated, comprising 16 angular and 19 linear measurements (Figure 3). Control cephalometric standards were obtained from the University of Michigan elementary school growth study[32] and each AVC patient's pre- and posttreatment cephalometric values were compared to age and sex matched values from the control group. Each AVC patient's treatment changes were compared to the changes seen in the control group over a similar number of months. Statistical analysis involved the use of means and standard deviations for each parameter followed by student's *t* test for independent samples to determine differences between group means. A significance level of $p < 0.05$ was established. Error analysis from 10 randomly selected and remeasured tracings revealed an average error of 0.13 mm for the linear measurements and 0.27° for the angular measurements.

Results

Overbite (Tables 1,2,3, No 28)

There was a mean decrease of 3.2 mm in overbite during AVC therapy with the reduction ranging from -0.9 mm to -5.7 mm. Nineteen out of the 25 cases (76%) had 1 mm or more of openbite closure and 17 cases (68% of the sample) showed 2.1 mm or more of overbite correction. Treatment time appeared to have little effect on the magnitude of openbite closure produced by the AVC. An average of 2.8 mm of closure was seen in the 13 patients who wore the AVC for 7 months or less, compared to the 3.1 mm noted in the 12 patients who wore the appliance for longer than 7 months. The resulting posttreatment overbite in the AVC group as a whole averaged zero millimeters.

Anteroposterior skeletal measurements

(Tables 1,2,3, Nos. 1-5)

Prior to treatment the AVC group appeared to be slightly more retrognathic than the control group (Ar-B; 94.2 mm versus 98.4 mm $p < 0.01$) and had slightly higher ANB values (4.6° versus 3.9°, p = NS). During AVC treatment the experimental group showed a greater increase than normal in SNB angle (+1.2° versus +0.2°, $p < 0.001$) resulting in a significant reduction in the ANB angle (-1.3° versus -0.1°, $p < 0.001$).

Rotational measurements

(Tables 1,2,3 Nos. 6-11)

Prior to treatment the AVC group demonstrated a significantly higher than normal mandibular plane angle when measured to SN (38.4° versus 34.4°, $p < 0.001$) but not when measured to Frankfort Horizontal (29.8° versus 28.6°, p = NS). The AVC group also demonstrated a steeper palatal plane to SN angle than the control group prior to treatment (3.5° versus 7.4°, $p < 0.001$). The only significant change seen during treatment was a slightly greater than normal reduction in the SN-MP angle (-1.0° versus -0.2°, $p < 0.05$). Of note was the 3.1° clockwise rotation of the maxillary occlusal plane and the 3.6° counterclockwise rotation of the mandibular occlusal plane that occurred during AVC therapy.

Vertical skeletal measurements

(Tables 1,2,3, Nos. 12-18)

Prior to treatment the total facial height (N-Me) was similar in both groups. However, in the AVC group the midface (N-ANS) was shorter by 4.3 mm ($p < 0.001$), while the lower face height (ANS-Me) was longer by 3.1 mm ($p < 0.05$). During treatment the AVC group showed no increase in total facial height, with this finding being significantly different ($p < 0.001$) from the 1.8 mm increase in the N-Me distance normally seen. Lower facial height in the AVC group demonstrated a 1.0 mm decrease during treatment, significantly different from the 0.9 mm increase normally seen ($p < 0.001$). Twenty-two of the 25 AVC cases exhibited less increase in lower facial height than the controls, with the difference being up to -5.3 mm.

Thus at T_2 those in the AVC group still had significantly shorter midfaces than the control group but had lower facial heights within the normal range. Interestingly, posterior facial dimensions were similar in both groups and showed little change with treatment.

Incisor position measurements

(Tables 1,2,3, Nos. 19-29)

Prior to treatment the position of the maxillary incisors in the AVC group was more protrusive than the control group relative to both SN (108.9° versus 104.7°, $p < 0.001$) and the NA line (5.1 mm versus 3.9 mm, $p < 0.001$), while

Table 3
AVC group treatment changes compared to control group changes

	Measurement	Experimental ($\bar{X} \pm$ S.D.)	Control ($\bar{X} \pm$ S.D.)	Difference (p)
Anteroposterior skeletal measurements	1. SNA °	-0.2±1.3	-0.1±0.3	0.1
	2. Ar-A mm	0.4±1.3	— —	—
	3. SNB °	1.2±1.1	0.2±0.2	1.0***
	4. ANB °	-1.3±1.4	-0.1±0.2	1.2***
	5. Ar-B mm	1.8±1.8	1.4±1.0	0.4
Rotational measurements	6. MPA °	-1.0±2.0	-0.6±0.8	0.4
	7. SN-MP °	-1.0±1.4	-0.2±0.3	0.8*
	8. SN-FH °	0.0±1.6	0.0±0.3	—
	9. SN-PP °	-0.1±1.3	0.2±0.3	0.3
	10. SN-MxOP °	3.1±3.1	— —	—
	11. SN-MnOP °	-3.6±3.8	— —	—
Vertical skeletal measurements	12. N-ANS mm	0.7±1.1	1.0±0.67	0.3
	13. ANS-Me mm	-1.0±1.6	0.9±0.72	1.9***
	14. N-Me mm	0.0±2.4	1.8±1.3	1.8***
	15. ANS-FH mm	0.6±1.2	— —	—
	16. PNS-FH mm	0.4±1.1	— —	—
	17. S-Go mm	0.9±1.5	1.6±1.1	0.7
	18. Ar-Go mm	0.9±1.9	1.0±0.9	0.1
Incisor position measurements	19. U1-SN °	-2.5±4.2	0.0±0.8	2.5**
	20. U1-PP °	-2.9±4.2	0.0±0.7	2.9**
	21. U1-PP mm	1.1±1.0	0.4±0.3	0.7**
	22. U1-FH mm	1.6±1.8	— —	—
	23. U1-NA mm	-0.5±1.5	0.2±0.3	0.7*
	24. U1-L1 °	7.1±6.2	0.3±1.1	6.8**
	25. L1-MP °	-3.9±4.5	0.2±0.5	4.1***
	26. L1-NB mm	-0.8±1.1	0.1±0.2	0.9***
	27. L1-MP mm	0.6±1.0	0.7±0.6	0.1
	28. OB mm	-3.2±1.4	— —	—
	29. OJ mm	-0.8±1.6	— —	—
Molar position measurements	30. U6-SN °	1.1±5.1	— —	—
	31. U6-PP °	1.1±5.9	— —	—
	32. UM-PP mm	-0.6±0.9	0.7±0.5	1.4***
	33. UM-FH mm	-0.3±1.2	— —	—
	34. L6-MP °	-0.4±4.4	— —	—
	35. LM-MP mm	-0.4±1.6	0.6±1.8	1.0*

* indicates significance at $p < 0.05$ level
** indicates significance at $p < 0.01$ level
*** indicates significance at $p < 0.001$ level
— indicates no data available

the position of the mandibular incisors was similar in both groups. During AVC treatment the maxillary incisors were retracted an average of 2.5° relative to SN ($p < 0.01$) in contrast to the 0.0° normally seen. The maxillary incisors also erupted 1.4 mm relative to palatal plane during treatment, compared to the 0.4 mm normally seen ($p < 0.01$). The mandibular incisors demonstrated almost 4° of lingual tipping during treatment ($p < 0.001$) which was substantially more than the 0.2° normally seen. The mandibular incisors also demonstrated almost 1 mm of lingual movement during AVC therapy which was significantly different ($p < 0.001$) from the 1 mm of labial movement normally seen. However, in contrast to the maxillary incisors, the mandibular incisors did not demonstrate an increased degree of eruption during AVC treatment.

Molar position measurements

(Tables 1,2,3, Nos. 30-35)

Prior to treatment both groups showed similar molar heights while the posttreatment evaluation revealed that the maxillary molars in the AVC group had undergone 0.6 mm of intrusion relative to palatal plane, a highly significant 1.3 mm difference from the 0.7 mm of molar eruption normally seen ($p < 0.001$). Seventeen of the 25 cases (68%) demonstrated some degree of intrusion, ranging from 0.1 mm to a maximum of 2.5 mm.

Mandibular molar heights started with similar dimensions at T_1, but by T_2 there had been an average of 0.4 mm of intrusion in the AVC group compared to the 0.6 mm of eruption normally seen ($p < 0.001$) with 12 cases showing some mandibular molar intrusion which ranged up to -3.7 mm.

Discussion

The overall impression gained from the data gathered in this study was that the AVC was effective at reducing openbites, with 19 out of the 25 cases demonstrating over 2 mm of bite closure. The mean bite closure for the group as a whole was 3.0 mm. These changes were achieved primarily by maxillary and mandibular molar intrusion combined with a significant amount of maxillary incisor retraction and eruption. There was some lingual tipping of the mandibular incisors and a small degree of counterclockwise rotation of the mandible.

The AVC's prinicipal effect appeared to be its ability to restrict the normal eruption of the maxillary and mandibular first molars. The 0.6 mm of maxillary molar intrusion seen in this study was in marked contrast to the 0.7 mm of eruption that might normally have occurred over the 8 months of the AVC treatment.

Similarly, the 0.4 mm of mandibular molar intrusion demonstrated in the study was significantly different from the 0.6 mm of eruption normally seen over a similar period. This study's findings in this area were similar to those of Woods and Nanda who demonstrated posterior dental intrusion in a study that used the active vertical corrector in baboons.[31] They also saw similar responses with non-magnetic bite blocks and hypothesized that the depression of posterior segments seen could be attributed to increased muscular forces occurring as much in response to the increased posterior vertical dimension as to the presence of the repelling magnets. This hypothesis might be supported by Carlson and Schneiderman's data which demonstrated 3.3 mm of posterior molar intrusion simply due to the placement of posterior bite splints in monkeys.[26] However, as has been pointed out, extrapolating results from baboons to humans is particularly dangerous in this area as the dolichofacial pattern frequently seen in human openbite patients is rarely seen in the primates used in these studies.[31]

In contrast, the posterior bite splint work of McNamara failed to demonstrate any significant dental intrusion[27] and studies of the effects of high-pull headgear, while producing significant inhibition of vertical skeletal development, have demonstrated only minimal (0.1 mm to 0.2 mm) dental intrusion.[21,22,33,34]

Once inhibition of vertical development is achieved, either skeletally or dentally, the clinical effect is usually to allow anterior rotation of the mandible to close the bite. In this study only 1° of reduction in the SN to MP angle was noted. A possible explanation for this finding could lie in the occurrence of a significant amount of remodeling at the mandibular gonial angle in response to the increased muscular tension which thereby masked a bite-closing mandibular rotation.[29-31] This theory might be supported by the findings that the AVC group's posterior face height (as measured from sella to gonion) increased at just over one-half the normal rate while the AVC group's lower anterior face height reduced significantly during treatment. It is interesting to note that with the total of 1 mm of molar intrusion seen in this study, Kuhn's formula for the effects of molar instrusion on openbite correction would predict that 2.8 mm of openbite closure should occur due to mandibular rotation. In fact, a very similar average of 3.0 mm of openbite closure was found in this study.[20]

These rotational effects may thus account for the 1.9 mm reduction in lower anterior face

height seen in this study; a highly significant finding when compared to the increase of 1 mm normally seen over the same time period. Similar findings have been reported both with bite splints,[29,30] and with some functional appliances utilized to treat openbites.[35] It is also interesting to note that the AVC had no beneficial effect on the shortened midface (N-ANS) seen initially in the AVC group.

Significant additional contributions to closure of the openbite seen in the AVC group may well have come from the changes seen in the position of the maxillary incisors. With 2.5° of retroclination and over a millimeter of eruption, the maxillary incisors contribution to the openbite closure may have been considerable. Similar changes have been noted by McNamara, as well as by Carlson and Schneiderman, and Woods and Nanda in their bite splint studies.[26,27,31] However, Hoenie, who used a splint that covered the incisors, failed to demonstrate increased incisor eruption.[30] In that study the mandibular incisors, while showing a significant amount of lingual tipping, failed to demonstrate any increase in eruption. Although the reasons for these incisor changes is not clear, they perhaps were in response to increased pressure being placed on the labial surface of the incisors by the circumoral musculature stretching and thus producing a disturbance in soft tissue equilibrium in response to the 7 mm of bite opening induced by the placement of the AVC appliance.[16,36] However, it is not known how many subjects still had a habit at the time of the AVC placement. The presence of the appliance might have stopped the habit, thus contributing to the reduction in openbite.

The AVC appeared to have minimal effects in the anteroposterior plane of space. No significant effects were seen on maxillary growth, unlike other reports on bite splint effects[26,27,31,37] and while there was a 1.2° reduction in the SNB angle, this was not due to any additional mandibular growth and seemed to be attributable to the forward, closing rotation of the mandible.[38]

In evaluating the overall effects of the AVC appliance it should be noted that although the openbite had been significantly reduced and now averaged 0 mm, the mandibles of the patients in the AVC group still remained somewhat deficient. Patients in this group still had high mandibular plane angles and they were shorter than normal in the midface. The dental corrections seen, while initially impressive, have yet to undergo long-term evaluation which must be the ultimate test of any treatment regime.

Conclusions

From a study of pre- and posttreatment cephalometric radiographs of 25 openbite cases treated with the active vertical corrector, the following conclusions were reached:

1. AVC therapy produced an average of 3 mm of anterior openbite closure over an 8 month treatment period.
2. The bite closure was due in large part to maxillary and mandibular molar intrusion, resulting in bite closing mandibular rotation.
3. Additional significant contributions to the correction of the openbite were due to maxillary incisor eruption and lingual tipping combined with mandibular incisor lingual movement.
4. A small amount of mandibular bite closing rotation and decrease in anterior facial height were seen, but there were only minimal skeletal changes in the sagittal direction attributable to AVC therapy.

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Author Address

Dr. Peter M. Sinclair
University of North Carolina
School of Dentistry
Orthodontic Department
Chapel Hill, NC 27599-7450

R.E. Barbre is in private practice in Arlington, Texas. This article is based on his thesis, submitted to the Department of Orthodontics, Baylor College of Dentistry, Baylor University in partial fulfillment of the requirements for a Master of Science degree.

P.M. Sinclair is an Associate Professor in the Department of Orthodontics, University of North Carolina and formerly was Associate Professor, Baylor College of Dentistry.

References

1. Nahoum, H.I.: Anterior open bite: a cephalometric analysis and suggested treatment procedures. Am. J. Orthod., 67:513-521, 1975.
2. Ellis, E., McNamara, J.A. Jr., Lawrence, T.M.: Components of adult Class II openbite malocclusion. J. Oral Maxillofac. Surg., 43:92-105, 1985.
3. Lopez-Gavito, G., Wallen, T., Little, R., Joondeph, D.: Anterior openbite malocclusion: a longitudinal 10 year postretention evaluation of orthodontically treated patients. Am. J. Orthod., 87: 175-186, 1985.
4. Subtelny, J., Sakuda, M.: Openbite: diagnosis and treatment. Am. J. Orthod., 50:337-358, 1964.
5. Isaacson, J., Isaacson, R., Speidel, T., Worms, F.: Extreme variation in vertical facial growth and associated variation in skeletal and dental relations. Angle Orthod., 44:219-229, 1971.
6. Sassouni, V., Nanda, S.: Analysis of dentofacial vertical proportions. Am. J. Orthod., 50:801-823, 1964.
7. Speidel, T., Isaacson, R., Worms, F.: Tongue thrust therapy and anterior dental openbite. Am. J. Orthod., 62:287-295, 1972.
8. Björk, A.: Prediction of mandibular growth rotation. Am. J. Orthod., 55:585-599, 1969.
9. Linder-Aaronson, S., Backstrom, A.: A comparison between mouth breathers and nose breathers with respect to occlusion and facial dimensions. Odontol. Revy., 11:343-376, 1960.
10. Bresolin, D., Shapiro, P.A., Shapiro, G.G., Chapko, M.K., Dassel, S.: Mouthbreathing in children: its relationship to dentofacial development. Am. J. Orthod., 83:334-340, 1983.
11. Trask, G.M., Shapiro, G.G., Shapiro, P.A.: The effects of perennial allergic rhenitis on dental and skeletal development: a comparison of sibling pairs. Am. J. Orthod., 92:286-293, 1987.
12. Garliner, D.: Myofunctional therapy in dental practice. Abnormal swallowing habits, diagnosis, practitioner and speech pathologist. Bartel Dental Book Co., Inc., Brooklyn, NY, 1971.
13. Proffit, W.R., Mason, R.: Myofunctional therapy for tongue thrusting: background and recommendations. J. Am. Dent. Assoc., 90:403-411, 1975.
14. Gershatner, M.: The proper perspective of openbite. Angle Orthod., 42:263-272, 1972.
15. Mizrahi, E.: A review of anterior openbite. Brit. J. Orthod., 5:21-27, 1978.
16. Proffit, W.R.: Equilibrium theory revisited: factors influencing position of the teeth. Am. J. Orthod., 48:175-186, 1978.
17. Willmot, D.: Thumbsucking habit and associated dental differences in one of monozygous twins. Brit. J. Orthod., 11:195-199, 1984.
18. Haryett, R., Hansen, F., Davidson, P., Sandilands, M.: Chronic thumbsucking: the psychologic effects and the relative effectiveness of various methods of treatment. Am. J. Orthod., 53:569-585, 1967.
19. Parker, J.H.: The interception of the openbite in the early growth period. Angle Orthod., 41:24-44, 1971.
20. Kuhn, R.: Control of anterior vertical dimension and proper selection of extraoral anchorage. Angle Orthod., 38:340-349, 1968.
21. Badell, M.: An evaluation of extraoral combined high-pull traction and cervical traction to the maxilla. Am. J. Orthod., 69:431-446, 1976.
22. Meldrum, R.: Alterations in the upper facial growth of Macaca mulatta resulting from high-pull head gear. Am. J. Orthod., 67:393-441, 1975.
23. Brandt, H., Shapiro, P., Kokich, V.: Experimental and post-experimental effects of posteriorly directed extra-oral traction in adult Macaca fascicularis. Am. J. Orthod., 75:301-317, 1979.
24. Watson, W.G.: Openbite — a multifactorial event. Am. J. Orthod., 80:443-446, 1981.
25. Breitner, C.: Further investigations of bone changes resulting from experienced orthodontic treatment. Am. J. Orthod., 27:605-632, 1941.
26. Carlson, D.S., Schneiderman, E.D.: Cephalometric analysis of adaptations after lengthening of masseter muscle in adult rhesus monkeys, Macaca mulatta. Arch. Oral Biol., 27:627-637, 1983.
27. McNamara, J.A.: An experimental study of increased vertical dimension in the growing face. Am. J. Orthod., 71:382-395, 1977.
28. Woodside, D., Aronson, S.: Progressive increase in lower anterior face height and the use of posterior occlusal bite-block in its management. Treatment and technique principles. In: Graber, L.W. (ed.) Orthodontics, State of the Art: Essence of the Science. St. Louis, C.V. Mosby, 200-221, 1986.
29. Dellinger, E.: A clinical assessment of the active vertical corrector — a nonsurgical alternative for skeletal openbite treatment. Am. J. Orthod., 89: 428-436, 1986.
30. Hoenie, D.C.: The effect of interocclusal repelling magnets in a bite opening splint on the growth of the craniofacial complex in juvenile Macaca mulatta. MS Thesis, University of Michigan, 1986.
31. Woods, M.C., Nanda, R.S.: Intrusion of posterior teeth with magnets — an experiment in growing baboons. Angle Orthod., 58:136-150, 1988.
32. Riolo, M.L., Moyers, R., McNamara, J., Hunter, W.: An atlas of craniofacial growth. University of Michigan, Ann Arbor, Michigan, 1974.
33. Baumrind, S., Korn, E.L., Isaacson, R.J., West, E.E., Molthen, R.: Quantitative analysis of the orthodontic and orthopedic effects of maxillary traction. Am. J. Orthod., 384-398, 1983.
34. Damon, D.H.: A clinical study of extraoral high-pull traction to the maxilla utilizing a heavy force. MSD Thesis, University of Washington, 1970.

35. Pearson, L.E.: Vertical control in treatment of patients having backward-rotational growth tendencies. Angle Orthod., 48(2):132-140, 1978.
36. Fränkel, R., Fränkel, C.: A functional approach to treatment of skeletal openbite. Am. J. Orthod., 84:54-68, 1983.
37. Lowe, A.: Correlations between orofacial muscle activity and craniofacial morphology in a sample of central and anterior openbite subject. Am. J. Orthod., 78:89-98, 1980.
38. McNamara, J.A.: Neuromuscular and skeletal adaptations to altered function in the orofacial region. Am. J. Orthod., 64:578-606, 1973.
39. West, R.A., Epker, B.N.: The posterior maxillary osteotomy: its place in the treatment of selected dentofacial deformities. J. Oral Surg., 30:562, 1972.