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Treatment and posttreatment dentoalveolar changes following intrusion of mandibular molars with application of a skeletal anchorage system (SAS) for open bite correction

The skeletal anchorage system (SAS) consists of titanium anchor plates and monocortical screws that are temporarily implanted in either the maxilla or the mandible as absolute orthodontic anchorage. With SAS, anterior open bite can be improved by the counterclockwise rotation of the mandible, accompanied by the intrusion of molars. The present study was designed to evaluate treatment and posttreatment dentoalveolar changes following the intrusion of mandibular molars. Nine adult open bite patients (7 women and 2 men) successfully treated with SAS were included in the following study. The amount of intrusion, relapse, and dentoalveolar changes were measured on cephalometric radiographs, panoramic radiographs, and dental casts. The results of this study were as follows: (1) the average amount of intrusion of the mandibular first and second molars was 1.7 mm and 2.8 mm, respectively; (2) the average relapse rates were 27.2% at the first molars and 30.3% at the second molars; (3) there were no significant changes in crestal bone heights, clinical crown length, or root length; and (4) counterclockwise rotation of the mandible and decrease of anterior facial height were observed during treatment. Thus, it was concluded that SAS would be a valid modality to intrude mandibular molars for correction of open bite. (Int J Adult Orthod Orthognath Surg 2002;17:243–253)

Skeletal open bite has been recognized as the most difficult malocclusion to correct in clinical orthodontics, because of the high frequency of relapse. Generally, open bite patients are characterized by vertical dentoalveolar excess in the maxilla and/or the mandible. Traditionally, open bite has been corrected by the extrusion of anterior teeth,[1] by surgical maxillary impaction in adult patients,[2,3] or by the inhibition of molar eruption in growing patients.[4–6] Dellinger[7] and Barbre and Sinclair[8] introduced an active vertical corrector (AVC) that used the repellent force of magnets and reported some intrusive effects of molars in growing open bite patients. Kim[9] and Chang and Moon[10] introduced a multiloop edgewise archwire (MEAW) technique for open bite correction, which worked by uprighting the molars and extruding the anterior teeth subsequent to the alteration of the occlusal plane. However, with these kinds of orthodontic mechanics, it is extremely difficult to reduce the excessive lower facial height of open bite patients, because the open bite has been mostly camouflaged by the extrusion of anterior teeth, not by the intrusion of molars.

Recently, we developed the skeletal anchorage system (SAS), which utilizes titanium miniplates and monocortical bone screws as absolute orthodontic anchorages, mostly for correction of skeletal open bite and Class III malocclusion.[11–13] SAS consists

Figs 1a to 1c Intraoral photographs of a typical open bite case treated by using SAS. Photographs were taken *(left)* prior to treatment, *(center)* at debonding, and *(right)* 1 year after debonding.

of a multibracket system with titanium miniplates that are temporarily implanted on the cortical bone of the maxilla and/or the mandible; this enables intrusion, distalization, and protraction of the molars, none of which can be easily achieved with traditional mechanotherapies. As described in our previous report,[13] skeletal open bite characterized by excessive facial height and a large interlabial gap was successfully corrected by the counterclockwise rotation of the mandible, accompanied by intrusion of the mandibular molars.

Although we predicted that the clinical crown length might be shortened by formation of a pseudo-pocket around the intruded molars, our clinical experience actually showed that the clinical crown length and the attachment level of the gingiva have been quite stable in most open bite patients who underwent SAS treatment. In addition, it was suspected that the root apices of the intruded molars might be resorbed or rounded up during orthodontic treatment. In our previous animal study,[14] we found that the root resorption of the mandibular molars after intrusion was minimal. With the above considerations in mind, we aimed to clarify the following points statistically in this study: changes in clinical crown length, changes in crestal bone height, and changes in root length subsequent to molar intrusion.

Retention has long been an issue in orthodontics and is still the subject of a great deal of research.[15–17] We believe that the improvement of long-term stability after orthodontic therapy will be one of the most important research themes in the coming years. While we have developed a system for the intrusion of molars that enables the treatment goals for open bite to be predictably achieved equally as well as in orthognathic surgery, posttreatment stability after intrusion of the mandibular molars is still unclear.

The purposes of the present study were: *(1)* to analyze the changes in the total amount of molar intrusion, clinical crown length, and crestal bone height after treatment; *(2)* to evaluate posttreatment changes of the intruded molars at 1 year after debonding; and *(3)* to elucidate differences in the effects of open bite correction between SAS and MEAW[9,10] as treatment applications.

Materials and methods

Subjects

Nine patients (7 women and 2 men) who had undergone orthodontic treatment with SAS at Tohoku University Dental Hospital were selected. They satisfied the following criteria for case selection: *(1)* diagnosis of anterior open bite with a long face and large interlabial gap, *(2)* successful treatment, *(3)* intrusion of the bilateral first and second mandibular molars, and *(4)* at least 12 months of follow-up after debonding. The average age of the patients at the beginning of treatment was 19.3 years and ranged from 13.3 to 28.9 years. The average treatment period was 27.1 months, ranging from 15 to 40 months, and the period of SAS application averaged 14.9 months, ranging from 9 to 22 months. Figures 1a to 1c show the changes in the occlusion of a typical patient treated with SAS.

SAS preparation and mechanics for intrusion of mandibular molars

The fundamental mechanical setup for intrusion of the mandibular molars is shown in Fig 2. The miniplates (Leibinger) or the newer orthodontic anchor plates (Dentsply-Sankin), both made of pure titanium, were implanted below the root apices around the first and second mandibular molars on the cortical bone of the mandibular body, as described previously.[12,13] Implantation was performed under local anesthesia, and the titanium plates were secured with titanium monocortical miniscrews (Leibinger or Dentsply-Sankin). Intrusive force was applied to each of the mandibular molars by ligating with elastic modules between stiff archwires (0.019-inch × 0.026-inch stainless steel) and the first hook of the miniplate. We began SAS treatment 3 to 4 weeks after implantation, which allowed sufficient time for the wound to heal.

Cephalometric analysis

Lateral cephalometric radiographs were obtained at the beginning of treatment immediately before SAS placement (T1), at debonding (T2), and at 1 year after debonding (T3). Cephalometric radiographs were traced, the right and left structures were averaged, and then cephalometric tracings of the mandible including all teeth at T1, T2, and T3 were superimposed following Bjork and Skieller's method.[18] Six reference points were defined on the mandibular first and second molars, as indicated in Fig 3a. These points were: the most convex point of the distal proximal surface (D), the midpoint of the occlusal surface (O), and the most convex point of the medial proximal surface of the crown (M) for both the first and second mandibular molars. Vertical changes at each reference point were measured at intervals of 0.25 mm perpendicular to the functional occlusal plane (FOP) determined on the T1 tracing. The FOP was defined as the line connecting the tip of the distal cusp of the second molar and the tip of the medial cusp of first molar, as shown in Fig 3b. The vertical distances between each reference point from T1 to T2 and from T1 to T3 were measured separately. Thus, the amount of the intrusion was the vertical change from T1 to T2, and the amount of relapse was calculated as the vertical change from T2 to T3. Then, the relapse rate was calculated as follows: (amount of relapse/amount of intrusion) × 100 (%).

The usual landmarks for cephalometric analysis were identified on the averaged tracing, and linear and angular measurements were taken. Twenty-one cephalometric measurements were evaluated (Table 1). The skeletal changes were analyzed during treatment and during the 1 year after debonding; in addition, the effects of treatment on the skeletal changes were compared between SAS treatment and MEAW treatment.[9,10]

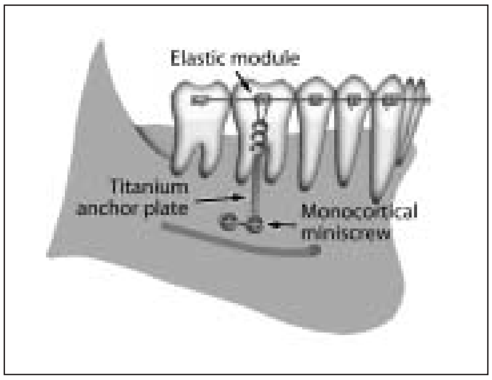
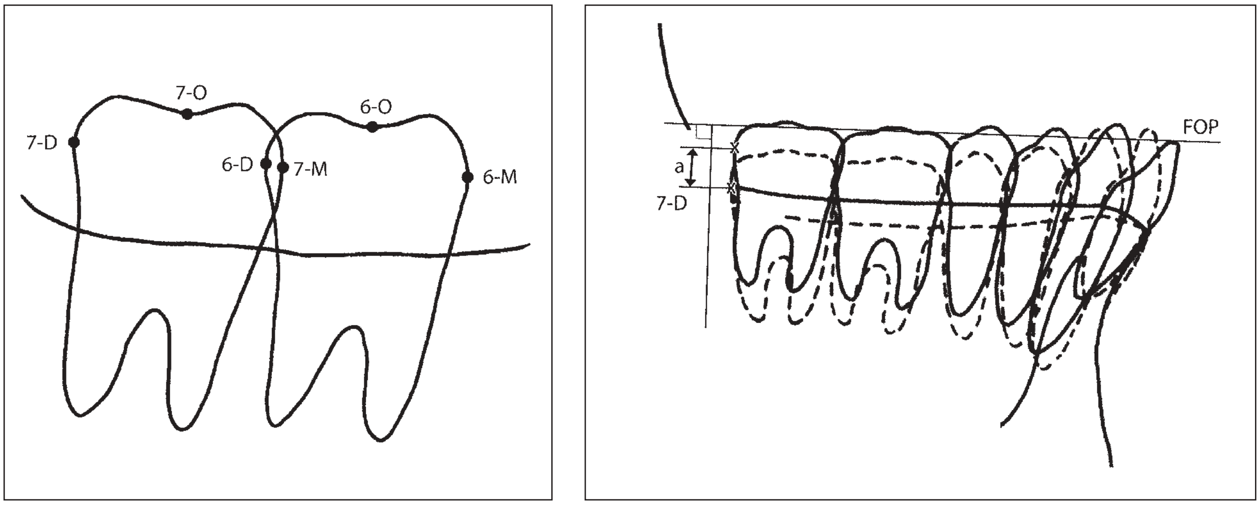


Fig 2 Schematic representation of the mechanics used for open bite correction by SAS. Heavy archwire ligated in the full arch is tied to the first hook of a titanium miniplate by an elastic module.

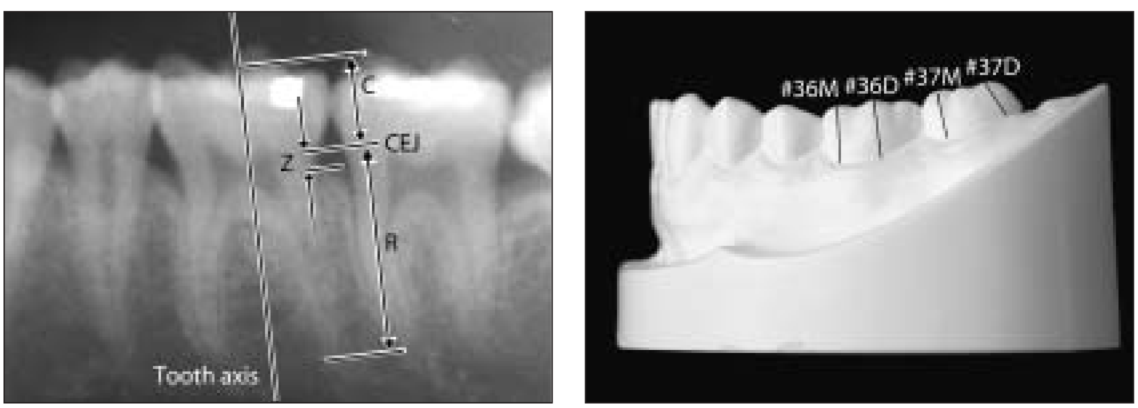
Panoramic radiograph analysis

As shown in Fig 3c, the crestal bone height (Z) and the root length (R) of each molar were measured on panoramic radiographs.[19,20] To standardize the images on panoramic radiographs, crown length (C), which did not change during the treatment, was used as the scale for measurements. The crown lengths at T1 (C1) and at T2 (C2) were measured from the cementoenamel junction (CEJ) to the midpoint of the medial and distal cusps parallel to the tooth axis.

The crestal bone heights at T1 (Z1) and T2 (Z2) were measured parallel to the long axis of the tooth, from the CEJ to the highest point of the crestal bone immediately



Figs 3a and 3b Reference points used for measurement and the method of stability analysis are indicated. Vertical changes were measured at each point perpendicular to the FOP. D = distal; O = occlusal; M = medial; FOP = functional occlusal plane; a = amount of vertical movement of the reference point.



Figs 3c and 3d Schematic drawing for the measurements of alveolar bone height, crown length, and root length and method for the measurement of clinical crown length on the dental cast. All parameters were measured from the CEJ as a zero point parallel to the long axis of the tooth. Clinical crown length was measured at all 4 cusps on the first and second molars. #36 = lower left first molar; #37 = lower left second molar; CEJ = cementoenamel junction; Z = alveolar bone height; C = crown length; R = root length.

medial to the first and second molars and immediately distal to the first molars according to the Baxter method.[21] Standardized Z2 (relative crestal bone height at T2 to Z1) was calculated as Rel Z2 by the following formula: Rel Z2 = Z2 × (C1/C2). The root length was measured along the long axis of the teeth, from the root apices to the CEJ in the medial and distal roots. Similar to the relative crestal bone height, Rel Z2 (relative rooth length at T2) was calculated as Rel R2 by the following formula: Rel R2 = R2 × (C1/C2).

The change in the crestal bone height relative to the original root length was calculated by the following formula on the basis of Melsen's study[22]: Percentage change in crestal bone height against original root length = (Rel Z2 – Z1) × 100/R1 (%). The amount of root resorption was calculated as follows according to the reports of Dermaut and De Munck[23] and Costopoulos and Nanda[24]: Percentage change in root length = (Rel R2 – R1) × 100/R1 (%).

Dental cast analysis

As shown in Fig 3d, clinical crown lengths were measured at T1 and T2 on the dental

Table 1 Cephalometric parameters used in the present study

Measurement	Interpretation
SNA (deg)	Sella-nasion to point A
SNB (deg)	Sella-nasion to point B
ANB (deg)	SNA minus SNB
Wits (mm)	Wits appraisal
FH to MP (deg)	Frankfort horizontal plane to mandibular plane
ATFH (mm)	Anterior total facial height
ALFH (mm)	Anterior lower facial height
Overbite (mm)	Horizontal distance between incisor superius (Is) and incisor inferius (Ii)
Overjet (mm)	Vertical distance between Is and Ii
U1 to FH (deg)	Upper incisor axis to Frankfort horizontal plane
U6 to PP (deg)	Upper first molar axis to palatal plane
L1 to MP (deg)	Lower incisor axis to mandibular plane
L6 to MP (deg)	Lower first molar axis to mandibular plane
FMIA (deg)	Frankfort horizontal plane to mandibular incisor axis
U1-PP (mm)	Vertical distance between Is and palatal plane
U6-PP (mm)	Vertical distance between upper first molar tip and palatal plane
U7-PP (mm)	Vertical distance between upper second molar tip and palatal plane
L1-MP (mm)	Vertical distance between Is and mandibular plane
L6-MP (mm)	Vertical distance between upper first molar tip and mandibular plane
L7-MP (mm)	Vertical distance between upper second molar tip and mandibular plane
Facial angle (deg)	Nasion–anterior nasal spine to pogonion

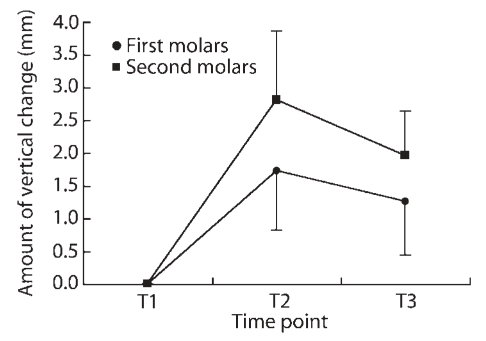


Fig 4 Average vertical changes and standard deviation (vertical bars) during the treatment and 1 year posttreatment. T1 = pretreatment; T2 = debonding; T3 = 1 year after debonding.

cast by calipers at every 0.05 mm following the method of Zachrisson and Alnaes.[25] The highest point of the marginal gingiva to the tip of the mediobuccal cusp parallel to the axis of the mediobuccal cusp was measured. The same procedure was applied to measure the clinical crown length at the mediolingual, distobuccal, and distolingual cusps.

Statistical analysis

Longitudinal changes between T1 and T2 or T2 and T3 for each measurement described above were statistically evaluated with a paired *t* test at *P* levels of .01 and .05.

Results

Treatment and posttreatment changes in molar intrusion

The average amount of intrusion was 1.7 mm (SD 0.91) and 2.8 mm (SD 1.05) at the first and second molars, respectively (Fig 4). The average amount of relapse was 0.5 mm and 0.9 mm at first and second molars, respectively. There was no statistically significant difference between the changes at T1–T2 and T1–T3 at both the first and second molars. The average relapse rates

Table 2 Cephalometric changes during treatment (mean ± SD)

Variables	TI	T2	T3	T2–T1 Mean	T2–T1 P	T3–T1 Mean	T3–T1 P
Skeletal							
Sagittal							
ANB	4.5 ± 2.0	3.8 ± 1.9	4.0 ± 1.9	–0.7	NS	–0.6	NS
SNA	83.5 ± 2.7	83.2 ± 3.0	82.9 ± 2.8	–0.3	NS	–0.6	NS
SNB	79.0 ± 2.4	79.4 ± 2.4	79.0 ± 2.3	0.4	NS	0.0	NS
Wits	–2.4 ± 3.2	–1.3 ± 2.7	–2.0 ± 1.7	1.1	NS	0.4	NS
Vertical							
FH to MP	33.1 ± 2.1	31.7 ± 2.4	32.2 ± 3.0	–1.3	*	–0.9	NS
ATFH	129.5 ± 7.4	128.5 ± 7.4	128.8 ± 7.4	–1.0	NS	–0.7	NS
ALFH	76.1 ± 5.8	74.6 ± 6.0	75.2 ± 5.8	–1.5	**	–0.9	NS
Dental							
Overbite	–2.8 ± 1.8	2.1 ± 0.8	1.2 ± 0.8	4.9	**	4.0	**
Overjet	1.7 ± 1.5	3.0 ± 0.8	2.2 ± 0.5	1.3	NS	0.5	NS
Angular							
U1 to FH	117.8 ± 5.4	114.7 ± 5.2	115.7 ± 5.9	–3.0	*	–2.1	NS
L1 to MP	94.1 ± 4.5	89.8 ± 4.9	92.2 ± 4.7	–4.3	*	–1.9	NS
FMIA	52.8 ± 4.8	58.5 ± 4.7	55.6 ± 5.3	5.6	**	2.7	*
Vertical							
U1-PP	29.8 ± 3.1	30.9 ± 3.3	30.7 ± 3.1	1.1	*	0.9	**
U6-PP	24.0 ± 3.0	25.0 ± 2.8	25.1 ± 2.5	1.0	**	1.1	**
U7-PP	21.7 ± 3.0	23.2 ± 2.6	22.9 ± 2.7	1.5	**	1.2	**
L1-MP	44.5 ± 3.9	45.8 ± 4.1	45.3 ± 4.3	1.3	*	0.9	NS
L6-MP	35.7 ± 4.1	33.9 ± 4.1	34.2 ± 4.4	–1.8	**	–1.6	**
L7-MP	33.1 ± 4.1	29.8 ± 4.8	30.9 ± 4.6	–3.3	**	–2.2	**

NS = $P > .05$; *$P < .05$; **$P < .01$.

were 27.2% and 30.3% at the first and second molars, respectively.

Cephalometric changes during treatment and posttreatment period

The cephalometric measurements at each time point and changes in measurements during treatment and the posttreatment period are indicated in Table 2. During SAS treatment, the anterior lower facial height (ALFH) ($P < .01$), mandibular plane angle (FH to MP) ($P < .05$), and the lower molar height at the first and second molars (L6-MP and L7-MP) ($P < .01$) were reduced significantly. On the other hand, overbite ($P < .01$) and maxillary molar height (U6-PP) ($P < .05$) increased significantly. The extrusion of the upper and lower incisors was minimal. Most of these significant differences were maintained until 1 year after debonding on average; however, FH to MP and the ALFH increased by 0.4 degree and 0.6 mm on average, respectively.

Changes in crestal bone height and root length during treatment

As shown in Table 3, no statistically significant differences were observed between Z1 and Rel Z2, except at #46D. The average percentage of change in crestal bone height was 1.7%. The difference in the percentage of change in crestal bone height versus the original root length, even at #46D, did not exceed 2.7%. Taken together, the relative position of the crestal bone to the CEJ was stable during treatment.

Table 3 Average changes in crestal bone height during treatment

	Z1 (mm)		Rel Z2 (mm)		% change in R1	
Variables	Mean	SD	Mean	SD	Mean	SD
#36M	1.4	0.8	1.7	0.6	2.4(NS)	4.5
#36D	1.0	0.5	1.2	0.8	1.4(NS)	6.8
#37M	1.3	0.9	1.4	0.5	0.8(NS)	6.9
#46M	1.3	0.5	1.4	0.3	0.4(NS)	3.4
#46D	1.3	0.4	1.7	0.4	2.7*	3.3
#47M	1.2	0.5	1.6	0.4	2.7(NS)	4.9
Mean	1.3	0.6	1.5	0.5	1.7	5.0

NS = $P > .05$; *$P < .05$; **$P < .01$.
Z1 = alveolar bone height at T1; Z2 = alveolar bone height at T2; R1 = root length at T1; M and D = medial and distal crestal bone height.

Table 4 Changes in root length during treatment

	R1 (mm)		Rel R2 (mm)		% change in R1	
Variables	Mean	SD	Mean	SD	Mean	SD
#36M	15.9	2.0	15.0	1.7	–5.2*	6.6
#36D	15.1	1.5	14.3	1.9	–4.8(NS)	8.9
#37M	15.1	1.2	13.5	1.7	–10.6**	6.6
#37D	13.8	1.3	13.0	1.2	–5.6*	5.3
#46M	6.3	1.8	15.7	1.7	–3.6*	4.0
#46D	15.0	1.5	14.6	2.0	–2.5(NS)	12.4
#47M	15.8	1.4	14.8	2.0	–6.1*	7.0
#47D	14.1	0.9	13.1	1.6	–7.2(NS)	9.6
Mean	15.1	1.4	14.3	1.7	–5.7	7.5

NS = $P > .05$; *$P < .05$; **$P < .01$.
R1 = root length at T1; R2 = root length at T2; M and D = medial and distal root length.

Statistically significant differences between root lengths at intruded molars between R1 and Rel R2 were observed at #36M, #37M, #37D, #46M, and #47M (Table 4). The average amount of root resorption was 0.9 mm, ranging from 0.4 mm at #46D to 1.6 mm at #37M. The average percentage change in root length was 5.7%, relative to R1.

Changes in clinical crown length during treatment

A very small but insignificant amount of change was noted in clinical crown length, as measured on the dental casts. The average clinical crown length of first and second mandibular molars was 4.6 mm at T1 and 4.6 mm at T2 (Table 5). The small amount of change noted was in the second decimal point, so it can be considered negligible. A slight elongation of the clinical crown length was observed at the buccal side of the molars, but no significant differences were observed.

Discussion

Open bite correction by intrusion of molars

The anterior open bite has been considered one of the most difficult malocclusions to correct in orthodontic treatment, and various treatment modalities have been developed over the years.[2,3,26] Because extrusion of anterior teeth is basic to all of the traditional mechanotherapies for open bite correction, and because the anterior teeth

Table 5 Changes in clinical crown length (mm)

	T1		T2			
Variables	Mean	SD	Mean	SD	Change (mm)	*P*
#36MB	5.2	0.5	5.7	0.8	0.5	NS
#36DB	5.4	0.6	5.4	0.7	0.0	NS
#37MB	4.5	1.0	4.3	1.0	–0.2	NS
#37DB	3.7	1.6	3.3	1.5	–0.4	NS
#36ML	5.0	1.1	5.1	0.9	0.1	NS
#36DL	5.0	0.9	5.1	0.9	0.1	NS
#37ML	4.4	0.8	4.3	0.9	–0.1	NS
#37DL	4.0	1.2	3.9	1.3	–0.1	NS
#46MB	5.6	1.0	6.0	0.9	0.4	NS
#46DB	5.5	0.9	5.6	0.8	0.1	NS
#47MB	4.3	0.9	4.5	0.5	0.2	NS
#47DB	3.1	0.8	3.5	1.0	0.4	NS
#46ML	4.7	0.6	4.5	0.7	–0.2	NS
#46DL	4.8	0.4	4.7	0.5	–0.1	NS
#47ML	4.2	0.7	3.9	1.0	–0.3	NS
#47DL	4.0	0.8	3.7	0.9	–0.3	NS
Mean	4.6	0.9	4.6	0.9	0.0	

NS = $P > .05$.
MB = mediobuccal cusp; DB = distobuccal cusp; ML = mediolingual cusp; DL = distolingual cusp.

of skeletal open bite are usually over-erupted due to the dentoalveolar compensatory mechanism for excessive anterior facial height, the stability of all of these treatment options remains questionable. Intrusion of the molars can be achieved with application of MEAW in adult patients[27]; however, the amount of intrusion of molars using this method has been found to be minimal. Since intrusion of the molars using SAS is a new modality for open bite correction, both its treatment effects and post-treatment stability have not yet been clarified. We demonstrated the advantages of SAS for intrusion of the mandibular molars in the present study. With application of SAS, the molars were predictably intruded, and the counterclockwise rotation of mandibles resulted in the reduction of lower facial height and the large interlabial gap. Additionally, this treatment influenced the anteroposterior jaw relationship because of the autorotation of the mandible following the intrusion of the molars. When the treatment effects of the SAS were compared to MEAW,[10,27] as shown in Table 6, it is clear that SAS achieved a considerable amount of intrusion of molars with minimal extrusion of incisors, while MEAW did not achieve counterclockwise rotation of the mandible and resulted in a considerable amount of extrusion of the incisors. Thus, intrusion of the molars seems to be the most rational treatment procedure for skeletal open bite patients who show long face types with skeletal Class I or mild skeletal Class II jaw relationships.

The amount of mandibular autorotation following the intrusion of molars was smaller than expected. Because the maxillary molars were not vertically controlled during SAS treatment, some amount of extrusion of the maxillary molars was observed. This may be a result of the physiologic mechanisms to maintain the maxillomandibular distance or the application of Class I maxillomandibular elastics at the detailing and finishing stage. Further investigation is needed to clarify the reason for extrusion of the maxillary molars.

Table 6 Comparison of treatment changs between SAS and MEAW (Kim[9] or Chang and Moon[10])

Variables	SAS	Kim	*P*	Chang and Moon	*P*
Skeletal					
Sagittal					
ANB	–0.7	–0.1	NS		
Wits	1.1				
Facial angle	0.8	–0.5	NS		
Vertical					
FH to MP	–1.3	–0.2	NS		
ATFH	–1.0	0.5	NS		
ALFH	–1.5	–0.2	NS		
Dental					
Overbite	4.9	4.0	**	5.4	**
Angular					
U6 to PP	–1.2	4.5	**	–1.7	NS
L1 to MP	–4.3	–1.4	NS		
L6 to MP	4.0	4.5	**	–9.2	**
Vertical					
U1-PP	1.1	1.3	**	2.0	**
U6-PP	1.0	–0.7	*		
U7-PP	1.5			1.1	NS
L1-MP	1.3	1.9	*		
L6-MP	–1.8	0.5	NS		
L7-MP	–3.3			–1.0	**

NS = $P > .05$; *$P < .05$; **$P < .01$.

Recently, to avoid the extrusion of the opposing molars, we have begun to apply 2 anchor plates to each jaw.

Posttreatment stability

The posttreatment stability of the various orthodontic mechanotherapies developed to date is considered questionable as well, because there is still growth to be experienced in AVC cases[8] and there is a possibility of relapse of the extruded anterior teeth in MEAW cases.[9] In the present study, we indicated the short-term stability of SAS following the intrusion of molars. Although the relapse rate of the intruded molars reached approximately 30% on average, the skeletal profiles of the treated patients were maintained 1 year after debonding. That means the relapse of the intruded molars was not directly related either to the skeletal changes or to the recurrence of open bite. In the literature to date,[15–17] with regard to the amount of relapse after tooth movement, intrusion is less stable than mediodistal movement and rotation. This could be explained by the following: *(1)* while the other type of tooth movement is accompanied by new bone formation that prevents relapse, intrusion does not induce new bone formation during tooth movement; *(2)* periodontal fibers, which are generally thought to resist occlusal forces, can also strongly resist intrusive forces; *(3)* apical periodontal tissues are reorganized more slowly than that in other sites; and *(4)* an effective method for retention has not been established for the intruded molars. In previous studies,[28] it was demonstrated that the application of bisphosphonates in animals was an effective pharmacologic method of retention that inhibited the bone from remodeling around the moved teeth. Therefore, it may be that the administration of bisphosphonate has the effect of stabilizing the intruded molars in human patients as well.

Changes in crestal bone height and clinical crown length

Clinical crown length and crestal bone height were not affected during the intrusion of the molars, as indicated in the results. Many studies have been conducted for both of these concerns in orthodontics[22,29–32]; however, there is still a lot of controversy. Some of the researchers have demonstrated a loss of crestal bone height after orthodontic treatment. Melsen[22] indicated in an animal study that intrusion of teeth did not induce a loss of marginal alveolar bone. In our previous study,[14] we indicated that the remodeling of crestal bone during intrusion did not promote crestal bone loss in dogs. We predicted that intrusion of the molars would produce pseudo-pockets during treatment, resulting in a decrease in clinical crown length. However, this was not observed. While the procedures employed in the present study might not be accurate enough to evaluate these data, it was apparent that there were no clinical problems that emerged after the intrusion of the molars in this context. Clinical and animal studies are ongoing in our laboratory and clinics to evaluate the periodontal conditions of the intruded molars.

Root resorption

Root resorption is an irritative iatrogenic problem basic to orthodontic treatment. In previous studies,[33–36] intrusion of the incisors induced a considerable amount of root resorption. Dermaut and De Munck[23] reported 18% root resorption after 3.6 mm of intrusion of incisors, and McFadden et al[36] reported 13.2% and 4.3% root resorption after maxillary and mandibular incisor intrusion, respectively. In contrast, both DeShields[37] and Kaley and Phillips[38] failed to detect a correlation between the amount of intrusion and root resorption in incisors. In the present study, we found 5.7% root shortening after molar intrusion, which was insignificant and apparently fell within the range of previous studies. Since previous studies were focused on monorooted incisors or premolars, there could be a difference in the pattern of root resorption of molars, since they have a large bifurcation area. Indeed, Bondevik[29] found a large amount of root resorption in the bifurcation area in rats when the molars were intruded, rather than around the root apex. Although the amount of root resorption did not exceed that of previous studies, careful follow-up studies are needed.

Conclusions

The skeletal and dentoalveolar changes produced by the intrusion of mandibular molars with application of SAS were apparently effective for open bite correction. However, a considerable relapse rate of approximately 30% of the intruded mandibular molars was observed. It suggests that overcorrection may be necessary.

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References

1. Kucukkeles N, Acar A, Demirkaya AA, Evrenol B, Enacar A. Cephalometric evaluation of open bite treatment with Ni-Ti arch wires and anterior elastics. Am J Orthod Dentofacial Orthop 1999;116:555–562.
2. Epker BN, Fish L. Surgical-orthodontic correction of open-bite deformity. Am J Orthod 1977;71:278–299.
3. Bell WH, Creekmore TD, Alexander RG. Surgical correction of the long face syndrome. Am J Orthod 1977;71:40–67.
4. Pearson LE. Vertical control through use of mandibular posterior intrusive forces. Angle Orthod 1973;43:194–200.
5. Pearson LE. Vertical control in treatment of patients having backward-rotational growth tendencies. Angle Orthod 1978;48:132–140.
6. Kuster R, Ingervall B. The effect of treatment of skeletal open bite with two types of bite-blocks. Eur J Orthod 1992;14:489–499.
7. Dellinger EL. A clinical assessment of the Active Vertical Corrector—A nonsurgical alternative for skeletal open bite treatment. Am J Orthod 1986;89:428–436.

8. Barbre RE, Sinclair PM. A cephalometric evaluation of anterior open bite correction with the magnetic active vertical corrector. Angle Orthod 1991;61:93–102.
9. Kim YH. Anterior open bite and its treatment with multiloop edgewise archwire. Angle Orthod 1987;57:290–321.
10. Chang YI, Moon SC. Cephalometric evaluation of the anterior open bite treatment. Am J Orthod Dentofac Orthop 1999;115:29–38.
11. Sugawara J, Umemori M, Mitani H, Nagasaka H, Kawamura H. Orthodontic treatment system for Class III malocculusion using titanium miniplate as an anchorage. Jpn J Orthod Soc 1998;57: 25–35.
12. Nagasaka H, Sugawara J, Kawamura H, Kasahara T, Umemori M, Mitani H. A clinical evaluation on the efficacy of titanium miniplates as an orthodontic anchorage. Jpn J Orthod Soc 1999;58: 136–147.
13. Umemori M, Sugawara J, Mitani H, Nagasaka H, Kawamura H. Skeletal anchorage system for open-bite correction. Am J Orthod Dentofac Orthop 1999;115:166–174.
14. Daimaruya T, Nagasaka H, Umemori M, Sugawara J, Mitani H. The influences of molar intrusion on the inferior alveolar neurovascular bundle and root using the skeletal anchorage system in dogs. Angle Orthod 2001;71:60–70.
15. Takahashi H. Stability of orthodontically rotated teeth in dogs; the effect of mechanical retention and gingival fiber transection. Jpn J Orthod Soc 1976;35:1–18.
16. Kawarada T. Experimental study on the effect of the mechanical retention of the tooth in dogs. Jpn J Orthod Soc 1978;37:8–36.
17. Ohtani N. A study of the relapse movement of the intruded teeth and the effects of mechanical retention and gingival transection. Jpn J Orthod Soc 1980;39:390–406.
18. Bjork A, Skieller V. Normal and abnormal growth of the mandible. A synthesis of longitudinal cephalometric implant studies over a period of 25 years. Eur J Orthod 1983;5:1–46.
19. Harris EF, Baker WC. Loss of root length and crestal bone height before and during treatment in adolescent and adult orthodontic patients. Am J Orthod Dentofac Orthop 1990;98:463–469.
20. Faltin RM, Arana-Chavez VE, Faltin K, Sander FG, Wichelhaus A. Root resorptions in upper first premolars after application of continuous intrusive forces. Intra-individual study. J Orofac Orthop 1998;59:208–219.
21. Baxter DH. The effect of orthodontic treatment on alveolar bone adjacent to the cemento-enamel junction. Angle Orthod 1967;37:35–47.
22. Melsen B. Tissue reaction following application of extrusive and intrusive forces to teeth in adult monkeys. Am J Orthod 1986;89:469–475.
23. Dermaut LR, De Munck A. Apical root resorption of upper incisors caused by intrusive tooth movement: A radiographic study. Am J Orthod Dentofac Orthop 1986;90:321–326.
24. Costopoulos G, Nanda R. An evaluation of root resorption incident to orthodontic intrusion. Am J Orthod Dentofac Orthop 1996;109:543–548.
25. Zachrisson BU, Alnaes L. Periodontal condition in orthodontically treated and untreated individuals. I. Loss of attachment, gingival pocket depth and clinical crown height. Angle Orthod 1973;43:402–411.
26. Creekmore TD, Eklund MK. The possibility of skeletal anchorage. J Clin Orthod 1983;17: 266–269.
27. Kim YH, Han UK, Lim DD, Serraon ML. Stability of anterior open bite correction with multiloop edgewise archwire therapy: A cephalometric follow-up study. Am J Orthod Dentofac Orthop 2000;118:43–54.
28. Igarashi K, Mitani H, Adachi H, Shinoda H. Anchorage and retentive effects of a bisphosphonate (AHBuBP) on tooth movements in rats. Am J Orthod Dentofac Orthop 1994;106:279–289.
29. Bondevik O. Tissue changes in the rat molar periodontium following application of intrusive forces. Eur J Orthod 1980;2:41–49.
30. Melsen B, Agerbaek N, Eriksen J, Terp S. New attachment through periodontal treatment and orthodontic intrusion. Am J Orthod Dentofac Orthop 1988;94:104–116.
31. Melsen B, Agerbaek N, Markenstam G. Intrusion of incisors in adult patients with marginal bone loss. Am J Orthod Dentofac Orthop 1989;96: 232–241.
32. Murakami T, Yokota S, Takahama Y. Periodontal changes after experimentally induced intrusion of the upper incisors in *Macaca fuscata* monkeys. Am J Orthod Dentofac Orthop 1989;95:115–126.
33. Henry JL, Weimann JP. The pattern of resorption and repair of human cementum. J Am Dent Assoc 1951;42:270–290.
34. Harry MR, Sims MR. Root resorption in bicuspid intrusion. A scanning electron microscope study. Angle Orthod 1982;52:235–258.
35. Linge BO, Linge L. Apical root resorption in upper anterior teeth. Eur J Orthod 1983;5:173–183.
36. McFadden WM, Engstrom C, Engstrom H, Anholm JM. A study of the relationship between incisor intrusion and root shortening. Am J Orthod Dentofac Orthop 1989;96:390–396.
37. DeShields RW. A study of root resorption in treated Class II, Division I malocclusions. Angle Orthod 1969;39:231–245.
38. Kaley J, Phillips C. Factors related to root resorption in edgewise practice. Angle Orthod 1991;61: 125–132.