Case Report

Occlusal Side Effects Caused by a Mandibular Advancement Appliance in Patients with Obstructive Sleep Apnea

Edmund C. Rose, Dr med dent, Dr Med[a]; Christina Schnegelsberg, Dr Med Dent[a]; Richard Staats, Dr Med[b]; Irmtrud E. Jonas, Univ-Prof Dr Med Dent[c]

Abstract: Mandibular advancement appliances (MAA) have been established as an alternative treatment option for obstructive sleep apnea (OSA). Although the therapeutic effect of these devices has been proven both clinically and polysomnographically through various studies, there are very few follow-up examinations in existence concerning possible dental side effects caused by the MAA. However, if lifelong treatment of OSA is considered, these follow-up examinations are of utmost importance. This article presents 2 cases with unexpected dental side effects and occlusal alterations caused by MAA therapy. (*Angle Orthod* 2001;71:452–460.)

Key Words: Mandibular advancement appliance; Obstructive sleep apnea; Dental side effects

INTRODUCTION

Obstructive sleep apnea (OSA) is caused by the periodic reduction or cessation of breathing due to narrowing or occlusion of the upper airway during sleep for which there are different therapeutic options. Among the general middle-aged population OSA is present in 4% of men and 2% of women.[1]

In terms of construction design, mandibular advancement appliances (MAA) are predominantly derived from functional orthodontic devices. Treatment with MAA is currently recommended, especially for primary snoring and a mild-to-moderate degree of OSA, in patients for whom improvement of sleep hygiene and the removal of etiological factors are not possible. MAAs are also being used in moderate to severe degrees of OSA when patients refuse nasal continuous positive airway pressure (nCPAP) therapy.

When assessing the treatment effectiveness during nocturnal polysomnographic registration, respiratory, somnographic and subjective improvement of OSA-specific symptoms are considered. These are mainly excessive daytime sleepiness, alertness and snoring. The appliance displaces the mandible and the neighboring soft tissues in an anterior position during sleep. The pharyngeal space, in particular the area of the oro- and hypopharynx is enlarged by this protrusion and kept from nocturnal collapse. Numerous studies have examined the initial effect on respiratory parameters of the MAA; however, there are very few long-term studies concerning respiratory parameters and occlusal side effects.[5–7] This article reports on 2 patients who developed significant occlusal changes during treatment with an MAA.

Mandibular Advancement Appliance

In both cases, a modified activator was used as an MAA (Figure 1). This activator was made of acrylic and split horizontally. Teeth in the lower jaw were covered fully by the acrylic whereas, in the upper dentition, only the premolar and molar regions were covered. In order to leave sufficient space for the tongue, the upper anterior teeth were left uncovered. This also allows for sufficient space for breathing through the mouth. The construction bite for displacing the mandible in the vertical and the sagittal plane was taken individually for each patient.

Recording and Analysis

Medical evaluations of both patients were carried out at admission and at follow-up examinations. These records contained their medical history, a recording of the Epworth Sleeping Scale[8] (ESS) and polysomnographic analyses from the sleep laboratory at Freiburg University Medical Hospital. The ''SleepLab 1000P'' equipment (Jäger Company, Würzburg, Germany) was used in a standard 12-channel recording. The data was evaluated by a physician special-

[a] Research Assistant, Department of Orthodontics, University of Freiburg, Germany.

[b] Head of the sleep laboratory, Robert-Koch-Clinic, Department of Pneumology, University of Freiburg, Germany.

[c] Head and Chairperson, Department of Orthodontics, University of Freiburg, Germany.

Corresponding author: Dr Edmund Rose, Department of Orthodontics, Dental Medical School, University of Freiburg, Hugstetter Straße 55, D-79106 Freiburg i. Br, Germany
(e-mail: rose@zmk2.ukl.uni-freiburg.de).

Accepted: May 2001. Submitted: March 2001.

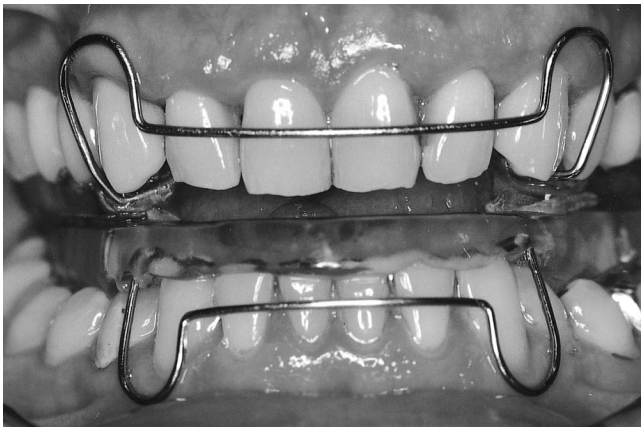


FIGURE 1. Intraoral view of the inserted activator; frontal view.

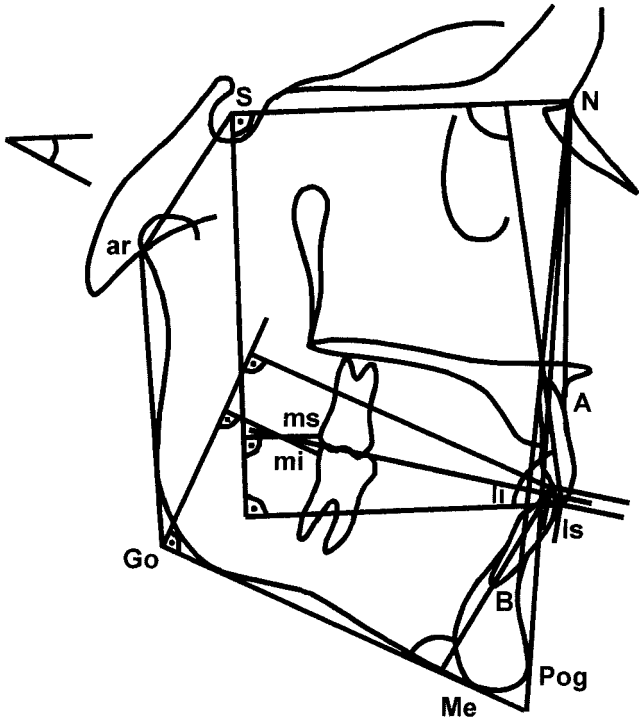


FIGURE 2. Cephalometric parameters. Reference lines: Nasion-Sella line (NS), NS-perpendicular (NSp), Menton-Gonion (MeGo), and MeGo-perpendicular (MeGop). Vertical measurements: Articular angle (SarGo), Gonion angle (arGoMe), Ratio of the anterior (NMe) to the posterior (SGo) facial height, and SNMeGo angle. Anteroposterior measurements: angle SNA, SNB, ANB, SN-Pog. Dental relation: Inclination of the lower incisors (L1 to MeGo), position of the anterior teeth with respect to NPog (U1 to NPog, U1 to NPog). Position of the upper incisors to the SN-perpendicular (SNp–1s) and to the upper molar (SNp–ms), position of the lower incisors to SNp to incision inferior (SNP–1i) and to the lower distal molar (SNp–mi).

ized in the field of somnology. The sleep laboratory, in accordance with the recommendations of the American Academy of Sleep Medicine,[2–9] assessed the severity of the OSA and established the need for an MAA.

The ESS is a questionnaire that assesses the general level of daytime sleepiness. Subjects have to rate the chances that they would doze off or fall asleep in 8 different common situations. A score below 7 is considered as normal.

A dental examination was carried out in the Department of Orthodontics and involved a functional analysis[10] and panoramic and lateral cephalometric radiography in centric occlusion. The same person traced the cephalometric radiographs at different times. We used analyses based on the Freiburg Analysis[11] and a modified method by Björk.[12] Significant landmarks and lines of reference are shown in Figure 2. The cephalometric radiographs were superimposed at the sella-nasion (SN) line. Anteroposterior measurements were carried out referring to the SN-perpendicular (SNp) and to the Menton-Gonion-perpendicular (MeGop).

Case 1

The sleep laboratory for treatment referred a 61-year-old female professor with oral appliances to us. She presented with a polysomnographic diagnosis of moderate OSA accompanied by bruxism events. The patient had refused nCPAP therapy. On direct questioning, she complained of loud snoring, arousal reactions due to apnea at night, and pronounced daytime sleepiness with a score of 16 on the ESS. She complained of tenderness in the temporomandibular joint region caused by bruxism. Her Body Mass Index (BMI) was 25.6 kg/m^2. She did not consume alcohol on a regular basis.

Intraoral examination revealed no need for restorative, periodontal, or prosthetic treatment. The patient had fixed partial dentures in the posterior region of the upper and lower jaw. There was edema of the buccal mucosa and the uvula was hypotonic and prolonged. The patient had a Class II malocclusion with deep overbite; the cephalometric analysis revealed Class II skeletal pattern. Her occlusion was stable and she had no crossbite or forced bite. On palpation, the masseter muscles and the lateral pterygoid muscles were slightly tender on both sides. No clicking, locking, or crepitus could be detected in the temporomandibular joint. The difference between postural rest position was 2 to 3 mm below and behind centric occlusion recording in the canine area.

A modified tooth-borne activator with sagittal mandibular protrusion of 4 mm and a vertical jaw opening of 8 mm was manufactured for the patient. According to the patient, she wore the device continuously for 6 to 7 hours each night. A control polysomnographic registration was carried out 4 weeks after appliance insertion. Subjectively, the patient was content with the treatment effect at the occasion of the follow-up examination. Although mild daytime sleepiness remained, as documented by an ESS of 7, snoring and apnea had ceased. The patient reported increasing tenderness in the temporomandibular joint during the first 2 weeks and difficulty finding occlusion in the mornings. By the time we did the polysomnographic follow-up, these

TABLE 1. Case 1 Polysomnographic Data

	Initial	First Control	Second Control
AI (hour)	8.5	0	2.2
RDI (hour)	23	1	7.2
Desaturation Index (hour)	22.3	4.8	10.5
O_2-basal (%)	96.8	95.9	96.0
O_2-min. (%)	90.7	90.0	90.4
REM (%)	9.4	7.2	10.6
S3 + S4 (%)	22.2	22.7	23.4

Medical examinations were carried out before therapy (initial), after 6 weeks (first control) and 2.8 years (second control) after insertion of the MAA. AI indicates apnea index; RDI, respiratory disturbance index; O_2-basal, basal oxygen saturation; O_2-min, minimal oxygen saturation; REM, phases of rapid eye movement sleep; S3 + S4, sleep stages 3 and 4 corresponding to deep sleep.

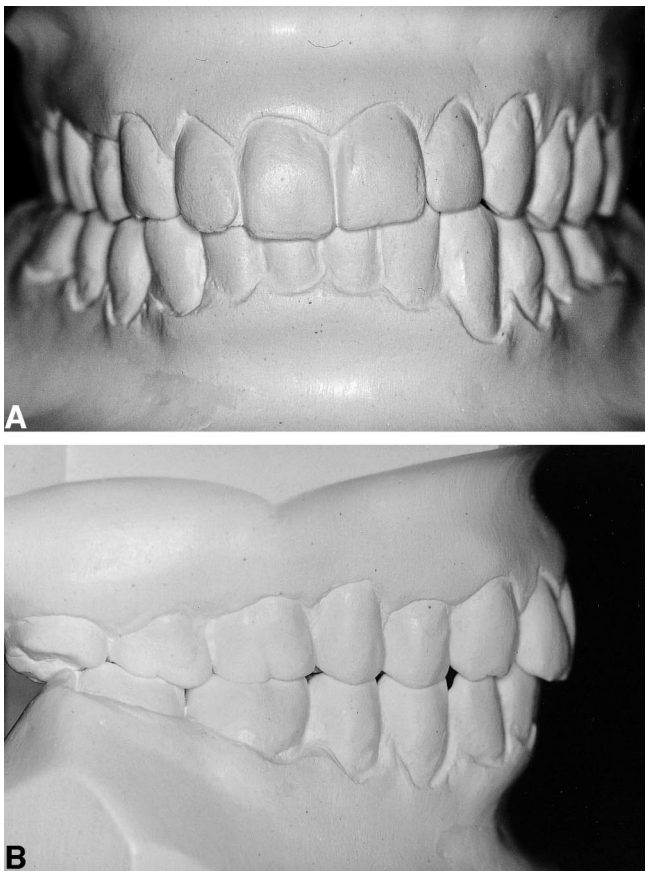


FIGURE 3. Case 1: Initial study casts. (A) Frontal view. (B) Lateral view.

symptoms had ceased. Respiratory improvement of the recorded parameters and an increased amount of sleep stage III and IV reinforced the subjectively perceived treatment success (Table 1).

Upon her dentist's advice, the patient returned after 2.8 years. She reported having worn the device for approximately 6 to 7 hours each night during the previous 2 years. Due to a reduction in symptoms and a general lack of complaints, she had not felt it necessary to appear at any of her follow-up appointments. She had no daytime sleepiness (ESS of 4) and snoring and night arousals had ceased. Temporomandibular tenderness was overcome. From time to time she still had brief difficulty finding occlusion in the mornings. Upon clinical examination, we found an alteration of the occlusion primarily in the sagittal and the transverse relationship. The deep bite was raised and there was a Class I relationship with a lateral open bite in the molar and premolar region on both sides. Model analysis showed a decrease in overbite from 5.8 mm to 2 mm, with the overjet being reduced from 5.8 mm to 2.7 mm. A forward displacement of the lower teeth of 2.8 mm in both the canine and molar regions on each side had taken place. In order to elucidate the occlusal changes, both the pretreatment and follow-up models are shown in Figures 3 A,B and 4 A,B. Clinical examination revealed no change in the distance between habitual occlusion and postural rest position. The mandible showed no anterior sliding action, nor could it be forced manually into a more backward position. Maximum jaw opening was unlimited with a value of 50 mm. There was neither clicking nor crepitus in the area of the temporomandibular joints. Neither the masseter muscle nor the temporal muscle was tender on palpation.

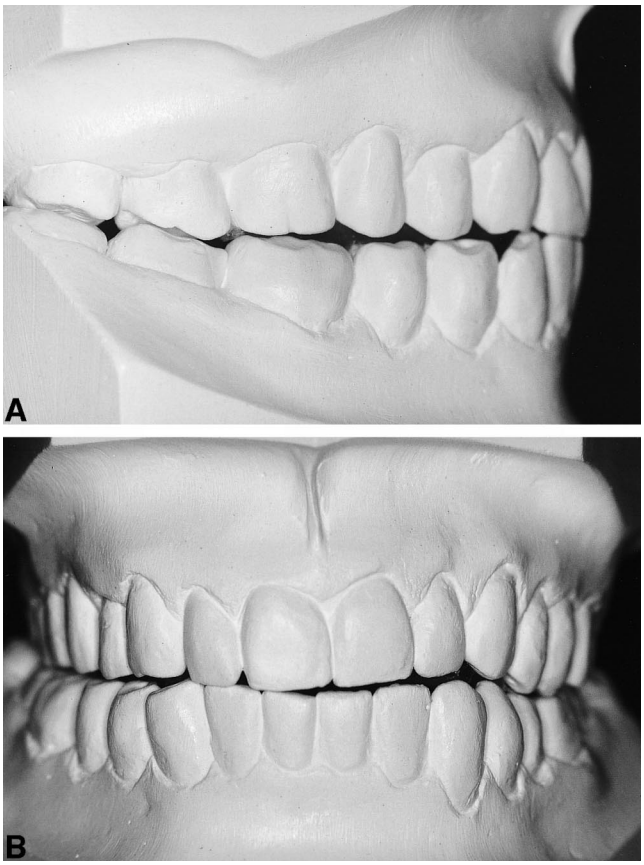


FIGURE 4. Case 1: Follow-up study casts. (A) Frontal view. (A) Lateral view.

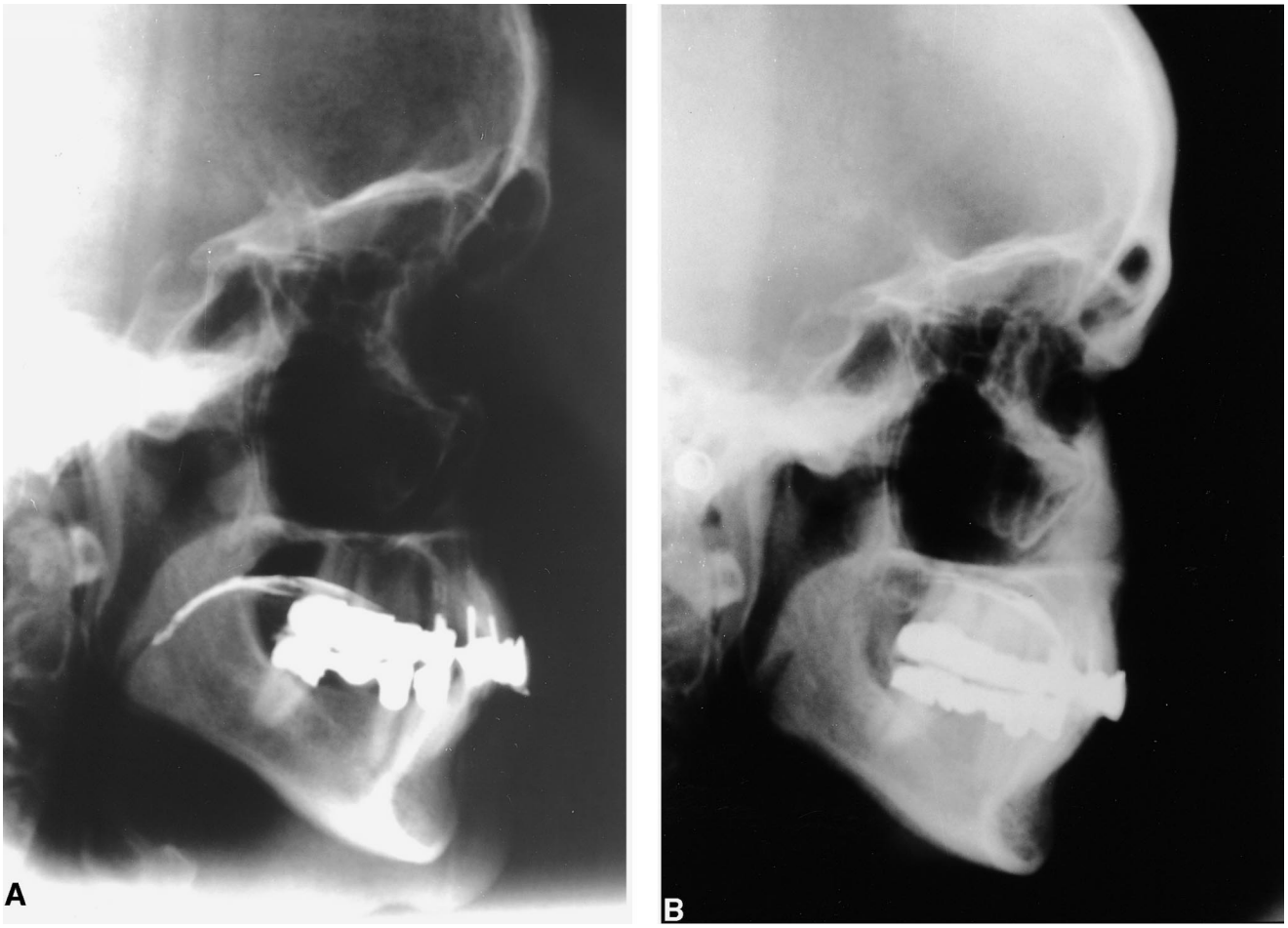


FIGURE 5. Case 1: Lateral cephalograms. (A) Initial x-ray. (B) Follow-up x-ray after 2.8 years of MAA-treatment.

A second cephalometic analysis was carried out 2.8 years after insertion of the MAA (Table 2)(Figure 5). Whereas skeletal parameters were not altered substantially, the mandible did rotate slightly downward and backward. This can be deduced from changes of the SNMeGo angle, articular angle, facial height ratio, and the SNPog angle. The upper incisors tipped lingually from 104° to 96° in reference to SN. The lower front teeth showed labial tipping of 5° to the reference line Menton-Gonion (MeGo). These alterations resulted in a change of the interincisal angle of 4.5° from 127.5° to 131°. Although the upper incisal point tipped to the lingual, correlation to SNp increased by 2 mm. Changes were minimal in the posterior region. Labial tipping of the mandibular front teeth is also responsible for the enhanced antepositioning of the lower incisors with respect to NPog (L1 to NPog 4 mm) (Figure 6).

In order to reevaluate the treatment indication, we conducted a control polysomnography. The therapeutic effect of the MAA treatment was still guaranteed but was decreased, particularly with regard to the apnea-hypopnea index (Table 1).

At the beginning of treatment the patient had been informed about the possible dental side effects caused by this therapeutic approach. Indication for a continuation of treatment continues. The patient has refused a change to n-CPAP-therapy for the time being, due to fear of recurring tenderness in the temporomandibular joint. For extrusion of the posterior region, the activator was trimmed in the molar and premolar areas. Regular recalls have been arranged with the patient.

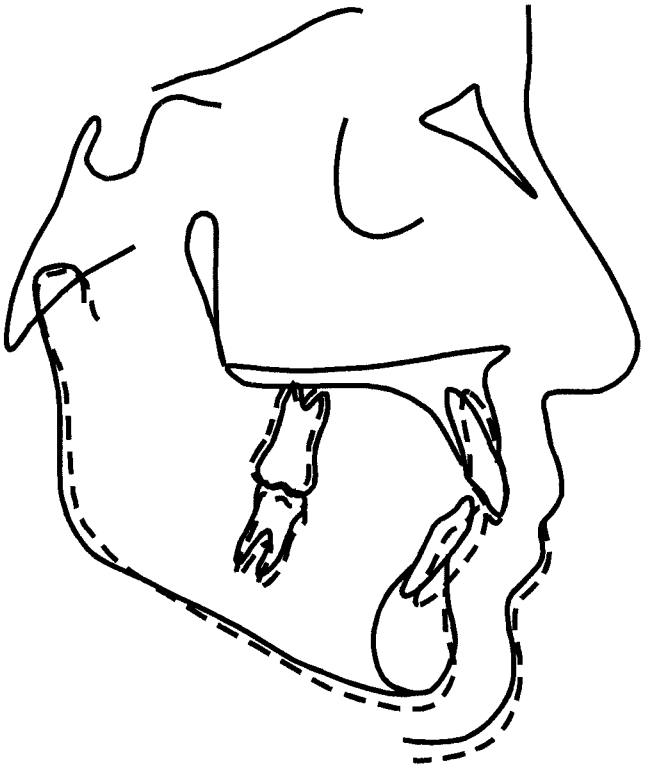
Case 2

A 53-year-old man complained of heavy snoring and night arousals which were observed by his wife for many years. Due to the noise, his wife slept in a different bedroom. The patient had no daytime sleepiness (ESS of 6). He was undergoing treatment for arrhythmia. His BMI was not excessive with a value of 24.3 kg/m^2. Oral examination showed inflamed and edematously changed pharyngeal mucosa. The polysomnograph findings were consistent with a diagnosis of moderate OSA (Table 3). The patient was wary of nCPAP therapy and elected MAA treatment.

A construction-bite revealed a sagittal mandibular pro-

TABLE 2. Pre- and Posttreatment Data of the Roentgenocephalometric Analysis of Both Patients

	Standard Value	Case 1		Case 2	
		Initial	Control	Initial	Control
Skeletal relation-vertical					
SarGo (°)	143 ± 6	148	150	143	147
arGoMe (°)	130 ± 7	121	121	119	117
SNMeGo (°)	34	33	35	29	31
Facial height ratio (%)	62 − 65	67	69	67	68.5
Skeletal relation-sagittal					
SNA (°)	81	82	82	84	84
SNB (°)	79	78	77	80	79
ANB (°)	2	4	5	4	5
ind. ANB (°)		3.9	4.3	4.3	4.7
SNPog (°)	80	81	79	80	81.5
Metric					
OK (mm)		73	73	45.5	45.5
UK (mm)		46	46	81	81
R.a. (mm)		57	57	61	61
Dental relationships					
Overjet (mm)	2	6	3	3	0
Overbite (mm)	2	5.5	2	2	0
Interincisal angle (°)	135	127.5	131	133	128.5
U1 to SN (°)	102 ± 2	104	96	100	97
L1 to MeGo (°)	90 ± 3	95	100	91	104
U1 to NPog (mm)	+2− +4	8.5	8	6	7
L1 to NPog (mm)	−2− +2	2.0	6	2	4
SNp-Is (mm)		54	56	63	60
SNp-ms (mm)		16	16.5	5	5
MeGop-Ii (mm)		56.7	66	68	72
MeGop-mi (mm)		23.5	27.5	11	14



←

FIGURE 6. Superimpositions of the cephalometric tracing of the initial and follow-up X-ray. The superimposition on NS shows that the upper incisors were retruded and tipped to the lingual and the mandible was rotated in a downward and backward direction.

TABLE 3. Case 2: Polysomnographic Data

	Initial	First Control	Second Control
AI (/h)	5.8	3	1.2
RDI (/h)	20	4.1	7.5
Desaturation Index (/h)	19.6	7.2	11.2
O_2-basal (%)	95.5	95.1	94.3
O_2-min. (%)	93.1	90.8	92.8
REM (%)	8.0	5.2	8.4
S3 + S4 (%)	18.9	29.4	23.6

Medical examinations were carried out at the following times: before treatment (initial), 8 weeks (first control) and 2.4 years (second control) after insertion of the MAA. AI indicates apnea index; RDI, respiratory disturbance index; O02-basal, basal oxygen saturation; O_2-min, minimal oxygen saturation; REM, rapid eye movement; S3 + S4, sleep stages 3 and 4 corresponding to deep sleep.

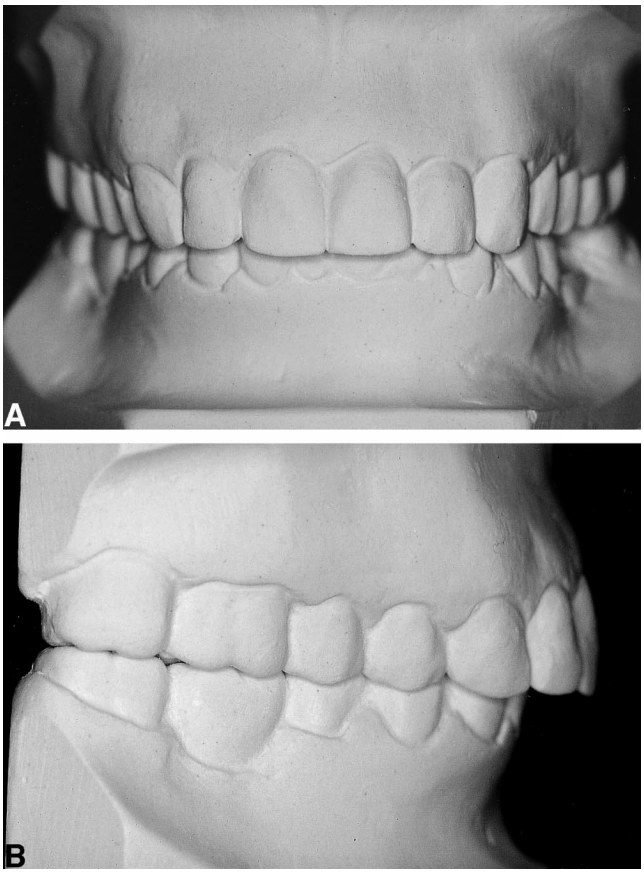


FIGURE 7. Case 2: Initial study casts. (A) Frontal view. (A) Lateral view.

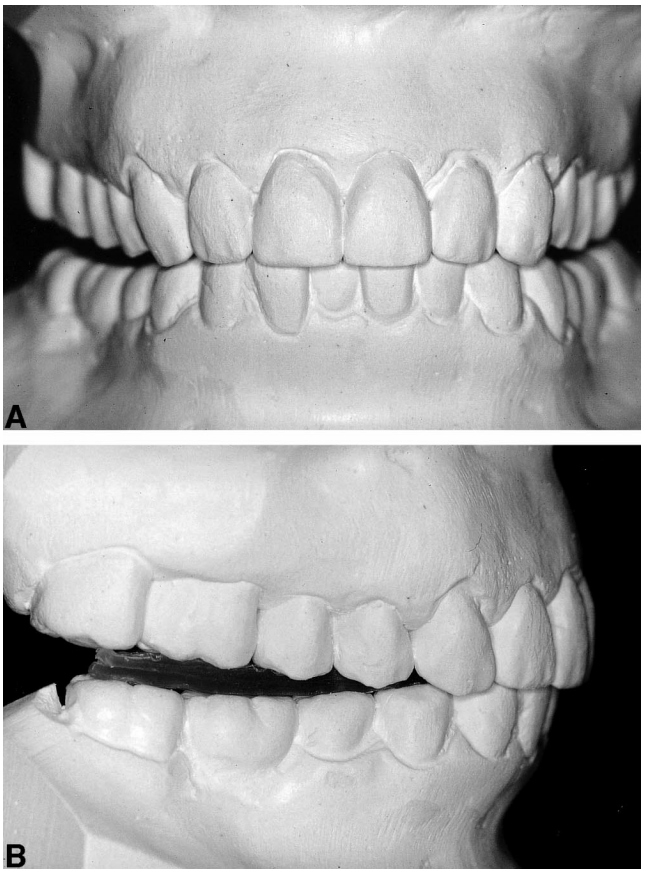


FIGURE 8. Case 2: Follow-up study casts. (A) Frontal view. (A) Lateral view.

trusion of 5.5 mm and a vertical opening of 10 mm. Two months after the MAA was inserted he returned to the sleep laboratory for a control polysomnography which revealed a relatively well-adjusted OSA (Table 3). Snoring was essentially reduced, so that he and his wife could again share the same bedroom. Treatment with the MAA continued. The patient did not return for follow-up appointments.

After 2.3 years, the patient returned complaining of new increased snoring, particularly when sleeping on his back. His wife had not observed any more night arousals. No temporomandibular joint symptoms were present. BMI had increased slightly to 25.2 kg/m^2. The patient underwent polysomnography in the sleep laboratory once more with the intraoral device in place. Respiratory parameters were substantially reduced compared to baseline results. The therapeutic effect had, however, deteriorated in relation to the initial clinical findings; in particular, the desaturation and apnea-hypopnea index had increased (Table 3).

We discovered both a lateral open bite and a displacement of the bite in this case. The front teeth were positioned in an edge-to-edge-relationship. The patient himself was not aware of these changes and continued to feel fine. Overjet was reduced by 2.8 mm, the overbite by 2.3 mm. Measuring in the canine region, a 2 mm mesial displacement of the lower dentition in the sagittal plane could be diagnosed on both sides (Figures 7A,B and 8 A,B).

Changes similar to those seen in Case 1, were found by roentgenocephalometric analysis (Table 2). There was a discrete downward and backward rotation of the mandible, with an increase in the facial height ratio and the SNMeGo angle. Clinical dental findings showed a change in overbite. The upper incisors had tipped to the lingual (U1 to SN $-3°$) and the lower front teeth had tipped to the labial (L1 to MeGo $+13°$). Overbite was reduced from 2 mm to 0 mm and the overjet from 3 mm to 0 mm (Figures 9A, B and Figure 10).

The patient was informed about the occlusal alterations but still refused nCPAP therapy. Due to the dental changes and the simultaneous tendency of worsening in the respiratory parameters, further close follow-up appointments have been arranged with the patient.

DISCUSSION

Oral protrusion appliances have been established since the mid-1980s as a form of treatment for obstructive sleep disorders.[4,13–15] The effectiveness of this therapeutic ap-

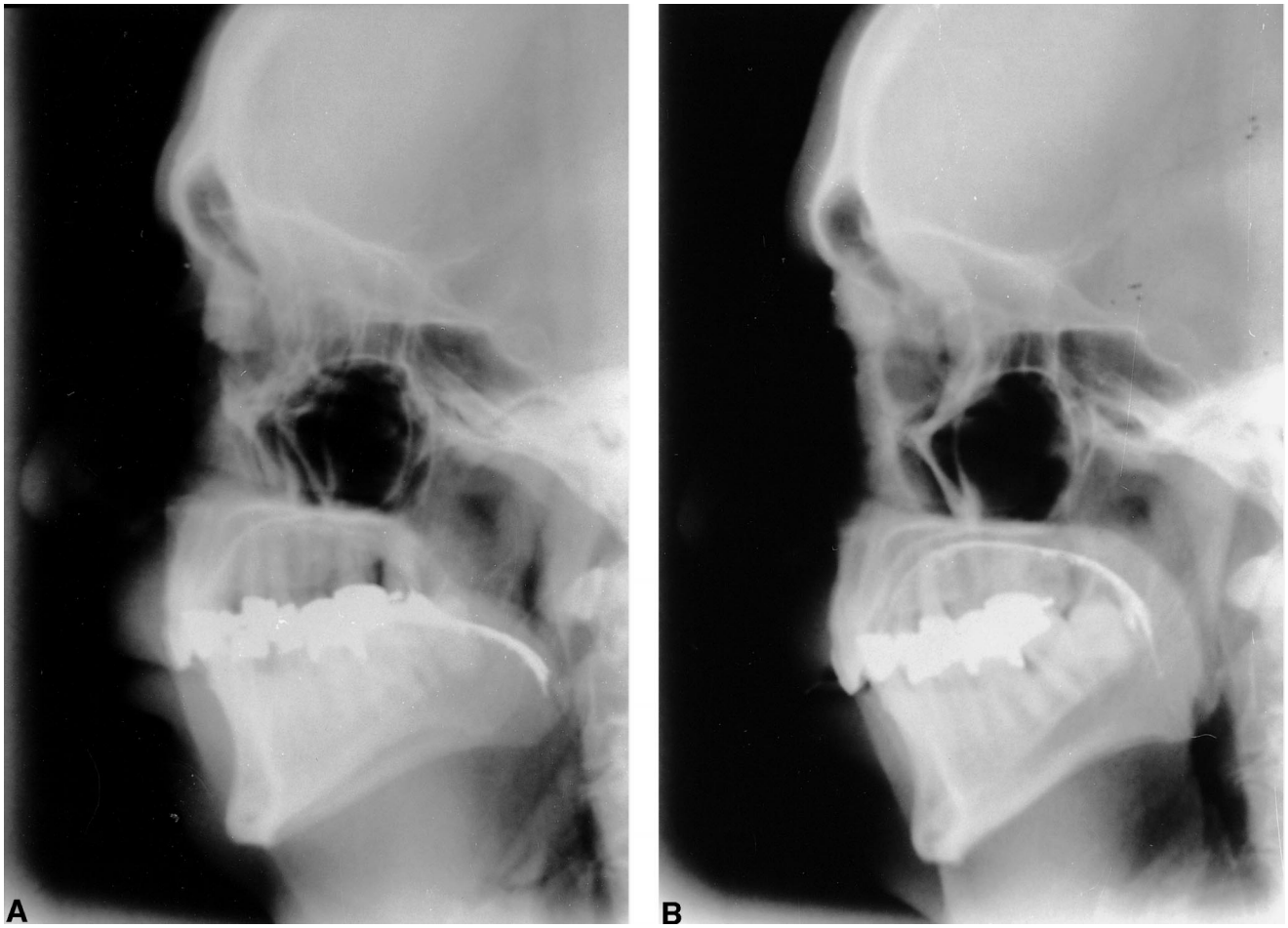


FIGURE 9. Case 2: Lateral cephalograms. (A) Initial x-ray. (B) Follow-up x-ray after 2.4 years of MAA-treatment with the MAA.

proach has been assessed in numerous studies.[3,7,13,16–18] The initial effect of the devices that were applied in our two case reports has been verified by means of polysomnography. There was a tendency for a decrease in effectiveness of the MAA at the second control polysomnography taking place after 2.3 and 2.8 years, respectively, as compared to the initial results. However, the therapeutic effect was still sufficient, even if limited. One major risk factor of OSA is the body mass. Both patients were relatively thin with a BMI of less than 25 kg/m^2. Since many patients tend to gain weight over time, a further lessening of the treatment's effect might be expected. Within this context we must consider whether MAA treatment can be carried out on a lifelong basis or whether it should be used only as a transitional treatment in preparation for nCPAP-therapy.

In terms of their design, MAA are derived historically from functional orthodontic appliances since they move the mandible into a protruded and fixed position. Functional orthodontic appliances are used to treat skeletal and dental changes in all three dimensions. Skeletal changes, particularly in the temporomandibular joint region, are not to be expected once condylar growth has stopped in adults. For this reason functional appliances are also used for retention after orthodontic treatment. Apart from skeletal effects, these devices have also shown dental effects. The transition of the force on the teeth can be minimized in these cases by covering the teeth, especially the occlusal surface, with acrylic.

Roentgenocephalometric studies before and 2 years after treatment with an intraoral device identified few skeletal changes,[19] particularly in the position of the mandible. So far, the dental side effects of MAA have been described as minor.[7] Only in the case report study of Panula and Keski-Nisula[6] have irreversible changes in the occlusion been reported. Similar changes occurred in our cases presented herein.

In order to achieve sufficient opening of the pharyngeal space, we used devices with a sagittal mandibular protrusion of one-half to three-fourths premolar width and a vertical opening of 8 to 10 mm. This substantial advancement of the mandible evokes reciprocal forces that are transferred onto the teeth. Because of muscular activation, tenderness is often felt at the beginning of treatment in the masticatory muscle. These findings appear in general during the adaptation period. In addition, retruding forces act on the upper front teeth and protruding forces on the lower front teeth.

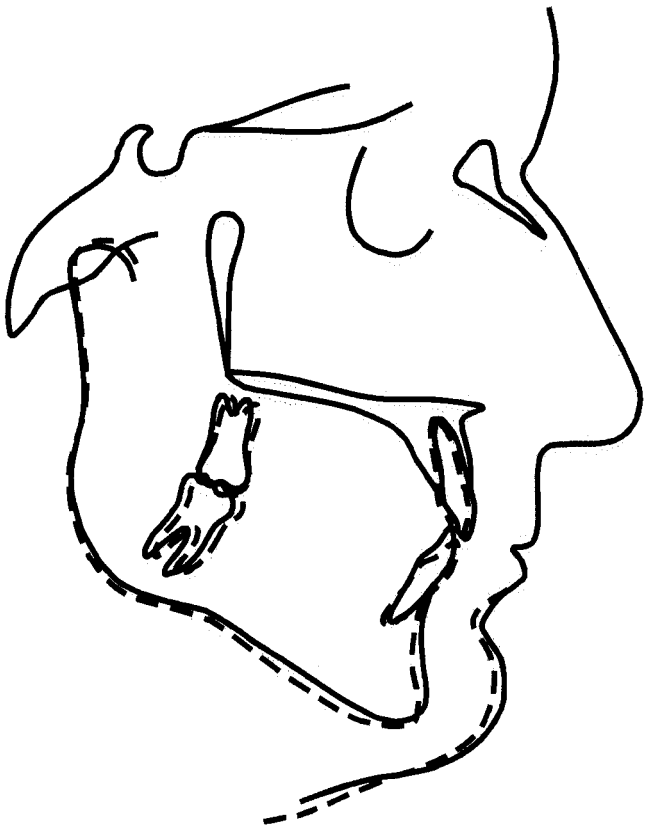


FIGURE 10. Superimposition of the cephalometric tracing of the initial and control x-ray. The superimposition on NS reveals that the upper incisors were retruded and tipped lingually. The mandible underwent a downward and backward rotation.

This results in lingual tipping of the upper incisors and in a reciprocal labial movement of the lower incisors.

The bite was raised in both of our cases and the mandible underwent a backward rotation. This effect can be derived from changes in the SNMeGo angle, the sum angle, and an increase in the facial height ratio and the SNPog angle. Since the premolars and molars did not erupt, a lateral open bite manifested. No further differential diagnostics such as magnetic resonance tomography (MRT) have been carried out on the patients. It remains speculative as to what extent changes were triggered by direct osseous adaptation in the temporomandibular region.

To what extent an initially existing posterior functional shift of the mandible, accompanied by condylar compression, was present in the first case cannot be determined in retrospect. However, improvement in the temporomandibular joint symptoms at follow-up indicates that such changes did indeed occur.

The incidence of OSA increases with age, thus periodontal disorders and sufficient anchorage of the device must be considered before deciding upon treatment with MAA. After the sleep laboratory has determined the medical indication for MAA treatment, somnographic control examinations are regarded as a standard means of assessing treatment effects. As demonstrated by our case reports, the patient must be carefully monitored through regular follow-up visits in order to recognize eventual dental and occlusal changes within the stomatognathic system as early as possible.

Final Evaluation

In summary, these cases prove that patients have to be informed about possible dental side effects resulting in substantial occlusal disorders. If side effects occur, continuation of treatment must be reevaluated by the patient and the somnologists in charge. For overall evaluation of the efficacy of OSA treatment with MAA, further studies concerning the long-term effects of the appliance on both respiratory parameters and dentition are essential.

REFERENCES

1. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med.* 1993;328:1230–1235.
2. American Sleep Disorder Association. Practice parameter for treatment of snoring and obstructive sleep apnea with oral appliances. *Sleep.* 1995;18:511–513.
3. Schmidt-Nowara WW, Meade TE, Hays MB. Treatment of snoring and obstructive sleep apnea with a dental orthosis. *Chest.* 1991;99:1378–1385.
4. Lowe A. Dental appliances for the treatment of snoring and obstructive sleep apnea. In: Kryger MH, Roth T, Dement W, eds. *Principles and Practice of Sleep Medicine.* Philadelphia, Penn: WB Saunders Co; 2000:929–939.
5. Bondemark L, Lindman R. Craniomandibular status and function in patients with habitual snoring and obstructive sleep apnoea after nocturnal treatment with a mandibular advancement splint: a 2-year follow-up. *Eur J Orthod.* 2000;22:53–60.
6. Panula K, Keski-Nisula K. Irreversible alteration in occlusion caused by a mandibular advancement appliance: an unexpected complication of sleep apnea treatment. *Int J Adult Orthodon Orthognath Surg.* 2000;15:192–196.
7. Tegelberg A, Wilhelmsson B, Walker-Engstrom ML, Ringqvist M, Andersson L, Krekmanov L, Ringqvist I. Effects and adverse events of a dental appliance for treatment of obstructive sleep apnoea. *Swed Dent J.* 1999;23:117–126.
8. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep.* 1991;14:540–545.
9. American Academy of-Sleep Medicine Task Force. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep.* 1999;22:667–689.
10. Rakosi T, Jonas I, Graber T, eds. *Orthodontic Diagnosis. Color Atlas of Dental Medicine.* New York, NY: Georg Thieme Verlag; 1993.
11. Rakosi T. Der Konstruktionsbiß. In: Rakosi T, ed. *Funktionelle Therapie in der Kieferorthopädie.* München-Wien: Carl Hanser Verlag; 1985.
12. Bosch C, Athanasiou AE. Landmarks, variables and norms of various numerical cephalometric analyses—cephalometric morphologic and growth data references. In: Athanasiou AE, ed. *Orthodontic Cephalometry.* London: Mosby-Wolfe; 1995:245–246.
13. Clark GT, Arand D, Chung E, Tong D. Effect of anterior mandibular positioning on obstructive sleep apnea. *Am Rev Respir Dis.* 1993;147:624–629.

14. Lavigne GJ, Goulet JP, Zuconni M, Morrison F, Lobbezoo F. Sleep disorders and the dental patient: an overview. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1999;88:257–272.
15. Meyer-Ewert K, Schäfer H, Kloß W. Treatment of sleep apnea by mandibular protracting device [abstract]. Munich: 7th European Congress of Sleep Research. 1984:217.
16. Bonham PE, Currier GF, Orr WC, Othman J, Nanda RS. The effect of a modified functional appliance on obstructive sleep apnea. *Am J Orthod Dentofacial Orthop.* 1988;94:384–392.
17. Marklund M, Franklin KA, Sahlin C, Lundgren R. The effect of a mandibular advancement device on apneas and sleep in patients with obstructive sleep apnea. *Chest.* 1998;113:707–713.
18. Schmidt-Nowara W, Lowe A, Wiegand L, Cartwright R, Perez-Guerra F, Menn S. Oral appliances for the treatment of snoring and obstructive sleep apnea: a review. *Sleep*. 1995;18:501–510.
19. Bondemark L. Does 2 years' nocturnal treatment with a mandibular advancement splint in adult patients with snoring and OSAS cause a change in the posture of the mandible? *Am J Orthod Dentofacial Orthop.* 1999;116:621–628.