

A Case of Sleep-Breathing Changes Achieved Using the Andresen Activator in a Child With Maxillary Protrusion

Cynthia Concepción Medina, DDS; Hiroshi Ueda, PhD; Yu Matsumura, PhD; Koji Iwai, DDS; Keisuke Sumi, PhD; Kotaro Tanimoto, PhD

Department of Orthodontics, Applied Life Sciences, Institute of Biomedical and Health Sciences, Hiroshima University, Japan

STUDY OBJECTIVES: To evaluate the changes that are brought about by continuous use of an orthodontic activator for the betterment of sleep-breathing in a child.

METHODS: An 8-year-old male who presented with retruded mandible (skeletal class II) and started treatment with the Andresen activator was subjected to at-home polysomnography analysis with a portable sleep monitor as a routine procedure where suspected sleep-disordered breathing signs were first noticed. Radiographic examination showed a slightly narrow upper airway. It was decided to observe this case's progression with the continuous use of the activator, and periodic at-home polysomnographic retests. Cephalometric radiographs were taken at different periods to evaluate the airway's width, the physical changes naturally induced by the activator, and its suspected added benefits.

RESULTS: In addition to the planned and expected physical changes delivered by the activator, there was marked improvement on all assessed indicators of sleep-breathing severity.

CONCLUSIONS: The Andresen activator not only is a useful and long-trusted orthodontic appliance used for the betterment of maxillary protrusion, it also has a positive effect on other aspects of child development by improving the sleep-breathing patterns of children who undergo orthodontic therapy with this appliance.

KEYWORDS: Andresen activator, at-home-sleep monitor, orthodontics, skeletal pattern, sleep-disordered breathing

CITATION: Medina CC, Ueda H, Matsumura Y, Iwai K, Sumi K, Tanimoto K. A case of sleep-breathing changes achieved using the Andresen activator in a child with maxillary protrusion. *Journal of Dental Sleep Medicine*. 2017;4(3):71–75.

INTRODUCTION

It has been generally observed that several conditions, including obesity and abnormal craniofacial characteristics, increase the risk of obstructive sleep apnea (OSA).¹ One of the most frequent craniofacial deformities most associated with OSA is maxillomandibular anteroposterior and vertical disproportion,² which is a result of poor mandibular growth. In children, one of the treatment options includes advancing the mandible forward using fixed or removable orthodontic functional appliances. Recently, it has been suggested that this type of therapy could not only correct the skeletal abnormalities, but could also potentially treat OSA and have a long-term effect that would prevent obstructive events in adulthood.^{3–5} Therefore, we assessed the effects of one such appliance (Andresen activator)⁶ on a child with suspected disordered breathing.

REPORT OF CASE

An 8-year-old boy (weight 47 kg, height 143 cm, body mass index 23 kg/m²; initial overjet 9.4 mm, initial overbite –3.0 mm) (**Figure 1**), was referred to the dental clinic at Hiroshima University Hospital. The chief complaint was maxillary protrusion (SNA 82.6°, SNB 73.6°, ANB 9.0). The overjet was excessive and the maxillary incisors were tipped labially. Cephalometric analysis indicated a mandible positioned posteriorly to the maxilla; from these characteristics the diagnosis of skeletal class II malocclusion was made and it was recommended

that the patient wear a myofunctional appliance to improve this condition. In addition, from the radiographs the upper airway width was deemed narrow. Furthermore, the parents expressed some concern about the child's constant snoring. Also, oropharyngeal crowding assessment was made using the Mallampati score, which showed this child was in class III, with only the base of the uvula visible; because a tonsillectomy procedure had been performed prior to the patient's arrival at Hiroshima University Hospital, tonsil score was 0. At the same time a modified version of the Pediatric Sleep Questionnaire: Sleep-Disordered Breathing Subscale (PSQ: SDB)⁷ and the Epworth Sleepiness Scale (ESS) modified for children were given to both parents and child, respectively. Initial scores were 0.40% for the PSQ: SDB subscale (the threshold for this scale is 0.33%, with this child scoring high on questions concerning mouth breathing and feeling unrefreshed upon waking up in the mornings) and zero for the ESS scale, meaning this child did not appear to suffer from daytime sleepiness.

The patient was recommended to use the Andresen activator (**Figure 2**), an orthodontic functional appliance for assisting proper mandible growth, with a mandibular advancement of 6.2 mm at construction bite. As per the hospital guidelines ideal use was recommended to be 10 to 12 hours every day, particularly at night. Two years after initial use of the activator the subject was also provided with a type 4 at-home sleep monitor (Brizzy Nomics, Liege, Belgium) with instructions to use it with and without wearing the activator in the mouth as to properly evaluate the changes that may happen. The data

Figure 1—Initial facial and oral photographs.



Figure 2—The Andresen activator.

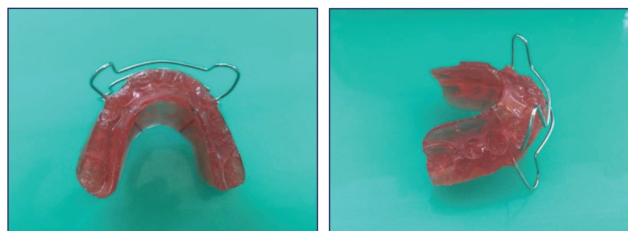


Table 1—Indicators of severity changes in Andresen activator treatment by at-home sleep monitor.

	T0 Treatment Start (Activator Out)	T1 Six Months After T0 (Activator In)	T2 A Year After T0 (Activator Out)
RDI (n/h)	15.5	1.8	8.7
ARL (n/h)	21.4	12.5	16.7
CT (%)	27.3	10.3	13.2
ODI (n/h)	7.9	5.9	8.3

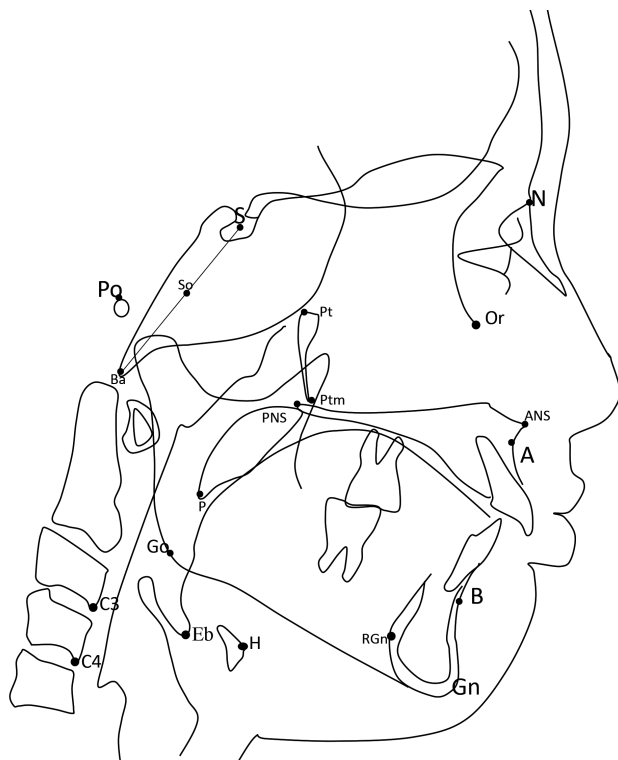
ARL = sleep fragmentation index, CT = cumulative time in respiratory effort, ODI = oxygen desaturation index, RDI = respiratory disturbance index.

were analyzed with the equipment’s proprietary software (APIOS, Nomics, Liege, Belgium). The provided variables were used as indicators of sleep-disordered breathing severity; these were respiratory disturbance index (RDI), sleep fragmentation index (ARL), cumulative time in respiratory effort (CT), and oxygen desaturation index (ODI). The RDI, ARL, and ODI scores represented the number of events per hour of recording time where the number of obstructive, central, and mixed events per hour of sleep were detected, the number of arousals or discontinuity per hour of sleep, and the times where blood oxygen levels decreased below a delimited threshold respectively, and finally CT, which refers to all periods of abnormal respiratory effort expressed as a percentage of the total sleep time. Three different recordings were recovered (T0 to T2) (Table 1), and from these results the patient was suspected of mild disordered sleep-breathing.

Data recovered from the monitor were analyzed at three time points with 6 months of difference between each, showing that when the activator is inserted in the mouth while sleeping (T1) there is a marked improvement of all indicators of severity and even though the results seen on T2 are still not desirable there is, nonetheless, an improvement when comparing to initial data (T0), (Table 1) which could be inferred to be carried throughout growth.

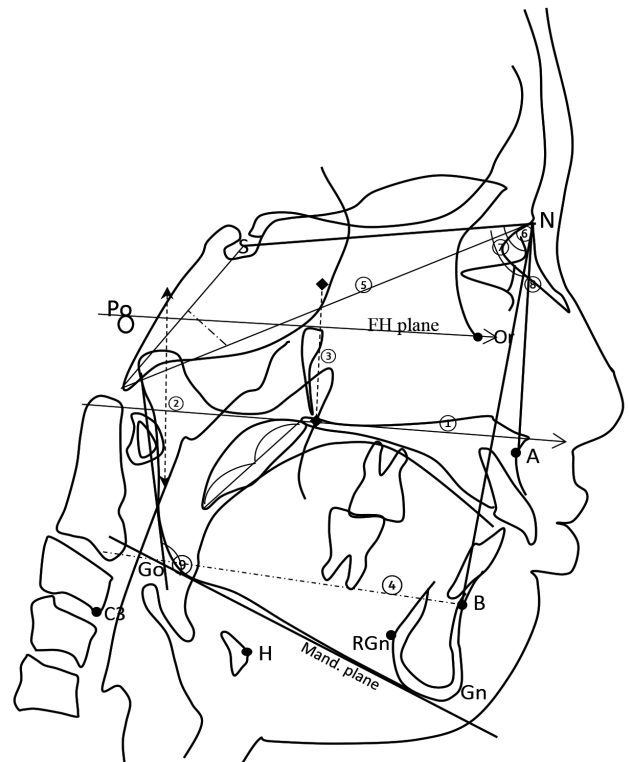
To fully assess the development of this case, and to further confirm if through use of the Andresen activator the upper airways are truly widened, a series of lateral cephalometric radiographs were taken, which included an amalgam of different analysis used in previous studies of the upper airways⁸⁻¹⁰ (Figure 3 to Figure 6).

Figure 3—Anatomical points of reference used for the cephalometric analysis.



A = most posterior point on the anterior contour of the upper alveolar process, ANS = apex of the anterior nasal spine, B = most posterior point on the anterior contour of the lower alveolar process, Ba = the most posteroinferior point on the clivus of occipital, C3 = third cervical vertebra, C4 = fourth cervical vertebra, Eb = most anterior-inferior point of the epiglottic fold, Et = tip of the epiglottis: the most superior point of the epiglottis, Gn = point on the chin determined by bisecting the angle formed by the facial and mandibular planes, Go = most posterior-inferior point on the convexity of the angle of the mandible, H = most anterior-superior point on the body of the hyoid bone, N = most anterior point of the frontonasal suture, Or = lowest point on the average left and right inferior borders of the bony orbit, P = lowest point of the soft palate, PNS = intersection between the nasal floor and the posterior contour of the maxilla, Po = highest point on the superior surface of soft tissue of the external auditory meatus, Pog = most anterior point of the contour of the chin, Pt = pterygoid point, Ptm = the intersection between the nasal floor and the posterior contour of the maxilla, RGN = retrognathion: the most posterior point of the mandibular symphysis along the FH plane, S = center of sella turcica, So = the midpoint on the line joining sella and basion.

Figure 4—Reference lines and angles.



(1) Palatal line (PL) = line representing palatal plane passing the most superior point on dens axis from ANS to PNS. (2) Anterior atlas line (AAL) = line perpendicular to palatal plane tangent to anterior surfaces of dens axis. (3) Pterygomaxillary line (PML) = line perpendicular to palatal plane that intersects palatal plane at pterygomaxillary fissure. (4) Go-B line = distance from Go to B. (5) Sphenoid line (SPL) = line tangent to lower border of sphenoid bone registered at basion. (6) SNA = angle between S-N and N-A. (7) SNB = angle between S-N and N-B. (8) ANB = difference between SNA and SNB. (9) Gonial angle = angle between ramus plane and mandibular plane.

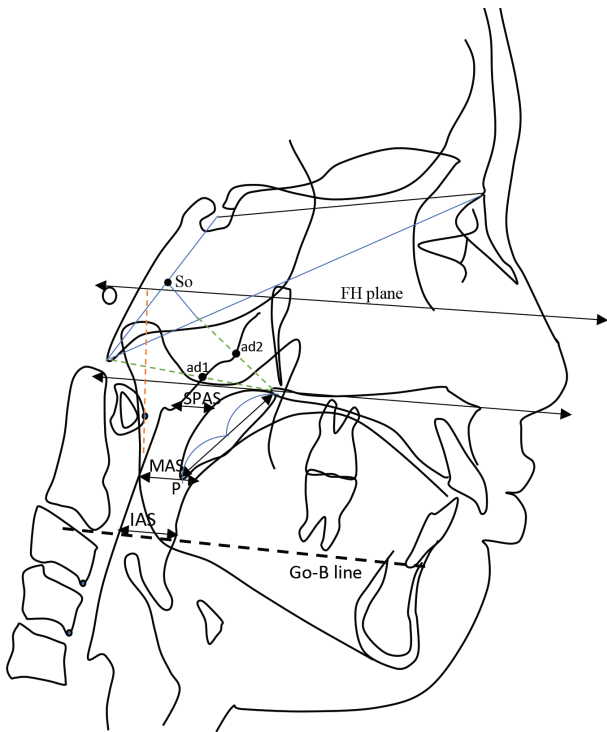
From these cephalometric radiographs the upper airways width (Table 2) was shown to have increased from T0 to T2, thus contributing to better sleep-breathing patterns. Also, the upper airways area seems to have increased slightly after activator therapy. Adenoid size was also measured at two points (T0 and T2) in which a small reduction of its size could be witnessed. The upper airways total area, as seen on the radiographs, showed an increased size, especially the areas delimited as oropharynx and hypopharynx. (Table 2). Serial lateral cephalograms as well as a superimposition of all three points (T0, T1 and T2) can be seen in Figure 7.

It is important to clarify that this patient was selected from a pool of patients who had volunteered to be part of broader

clinical research that included only healthy children who share the same characteristics of being skeletal class II and were recommended to use the Andresen activator to improve their occlusal and facial-skeletal discrepancy. The case mentioned in this report was chosen because it showed signs of being worse than the norm for all the other cases examined; thus, it was considered for a longer observation period of evaluation. After the first analysis was done and results explained to the parents, they decided to wait before deciding not to seek a specialized opinion even though they expressed some concern at the beginning of treatment about the child's constant snoring, of which they noticed a slight reduction from wearing the appliance.

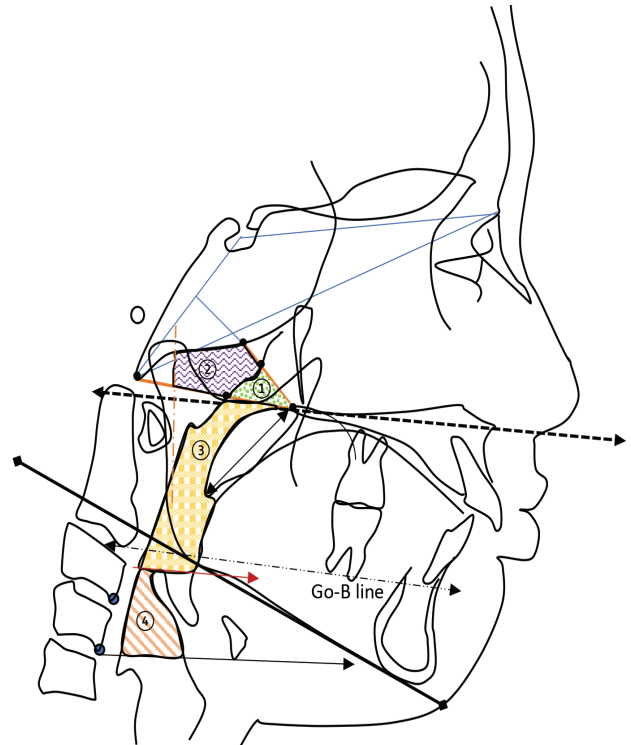
At the time of the final checkup the patient was 12 years old (weight 53 kg, height 151 cm, body mass index 23.2). Final overjet was measured at 6.7 mm and overbite at 1.2 mm. Final PSQ: SDB score was 0.36%, showing a small improvement in the child's sleep breathing as perceived by his parents, and a final ESS score of zero, showing that the case, at least concerning daytime sleepiness as perceived by the child, did not deteriorate throughout its observation time.

Figure 5—Cephalometric variables of linear measurements.



ad1 = the intersection of the line Pt-Ba and the posterior nasopharyngeal wall, ad2 = the intersection of the posterior nasopharyngeal wall and the line Pt-So, IAS = the thickness of the airway along a line extended through Go-B, MAS = the thickness of the airway along a line parallel to Go-B through P, PNS-ad1 = linear distance from the point PNS to the point ad1, PNS-ad2 = linear distance from the point PNS to the point ad2, SPAS = the thickness of the airway behind the soft palate along a line parallel to Go-B.

Figure 6—Upper airways area measurements.

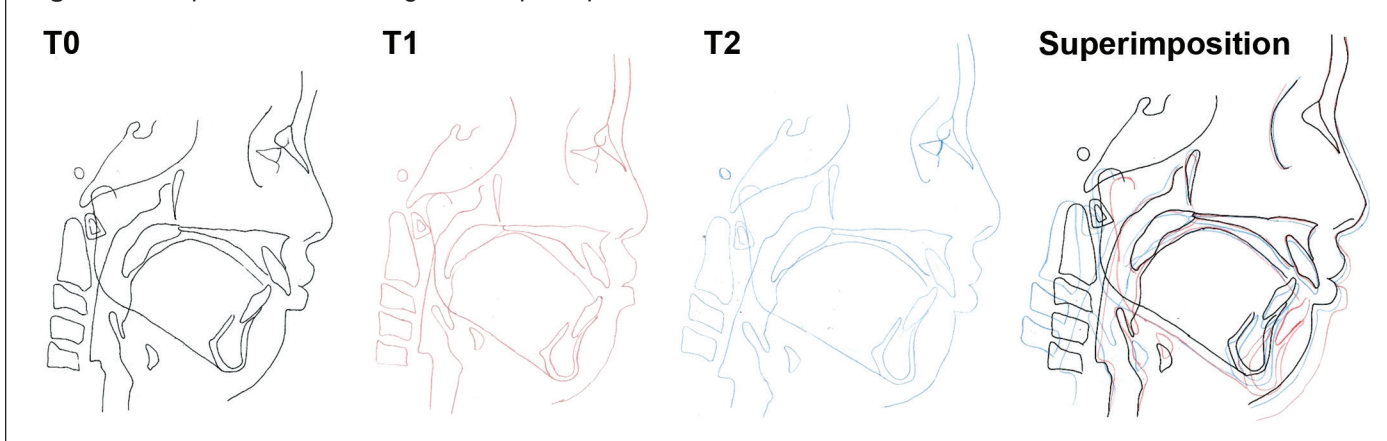


(1) NP area (nasopharyngeal area) = corresponds to the nasopharyngeal area in vertical and anteroposterior dimensions from ad1-Ba-ad2-So, where the posterior wall is delimited by the anterior atlas line, measured in mm². (2) Adenoid area = corresponds to the adenoid area in vertical and anteroposterior dimensions from ad1-So-Ba-ad2, measured in mm². (3) Oropharynx = the area outlined by the inferior border of the nasopharynx, the posterior surface of the soft palate and tongue, a line parallel to the palatal plane through point Et, and the posterior pharyngeal wall, measured in mm². (4) Hypopharynx = the area outlined by the inferior border of the oropharynx, the posterior surface of the epiglottis, a line parallel to the palatal plane through point C4, and the posterior pharyngeal wall, measured in mm².

Table 2—Changes in the cephalometric values before and after using the activator.

Variable	T0 (Activator Out)	T1 (Activator In)	T2 (Activator Out)
SNA	82.6°	82.5°	82.7°
SNB	73.6°	79.4°	76.3°
ANB	9.0°	3.1°	6.4°
PNS-ad1 (mm)	21.74	*	16.81
PNS-ad2 (mm)	15.22	*	12.48
SPAS (mm)	10.24	11.57	12.12
MAS (mm)	14.18	14.58	14.34
IAS (mm)	13.03	13.16	13.47
Adenoid area (mm ²)	45.20	*	24.60
Nasopharynx area (mm ²)	7.61	*	5.25
Oropharynx area (mm ²)	46.46	41.91	48.75
Hypopharynx area (mm ²)	14.03	15.55	21.00

* = no data. ANB = difference between SNA and SNB, IAS = the thickness of the airway along a line extended through Go-B, MAS = the thickness of the airway along a line parallel to Go-B through P, PNS-ad1 = linear distance from the point PNS to the point ad1, PNS-ad2 = linear distance from the point PNS to the point ad2, SNA = angle between S-N and N-A, SNB = angle between S-N and N-B, SPAS = the thickness of the airway behind the soft palate along a line parallel to Go-B.

Figure 7—Cephalometric tracings and superimposition from T0 to T2.

Despite the mentioned setbacks, this case presented interesting outcomes throughout its evaluation time, with an observed betterment of sleep-breathing that was also somewhat maintained over time. This may be explained as the patient grows the changes brought upon by the activator are maintained, not only in the development of the mandible, but also the widening of the airways.

CONCLUSIONS

Even though this case is far from full health, the extended and uninterrupted use of the Andresen activator could not only bring about the necessary changes for a harmonious occlusion, it also may improve sleep-breathing of children undergoing this type of orthopedic therapy.

REFERENCES

1. Subramani Y, Singh M, Wong J, Kushida CA, Malhotra A, Chung F. Understanding phenotypes of obstructive sleep apnea: applications in anesthesia, surgery, and perioperative medicine. *Anesth Analg*. 2017;124(1):179–191.
2. Zhong Z, Tang Z, Gao X, Zeng XL. A comparison study of upper airway among different skeletal craniofacial patterns in non-snoring Chinese children. *Angle Orthod*. 2010;80(2):267–274.
3. Amaral Júnior R, Kim LJ, Tufik S, Andersen ML. Is it possible to prevent sleep apnea with maxillomandibular orthopedic treatment during childhood? *Sleep Breath*. 2014;18(4):675–676.
4. Singh GD, Garcia-Motta AV, Hang WM. Evaluation of the posterior airway space following biobloc therapy: geometric morphometrics. *Cranio*. 2007;25(2):84–89.
5. Horiata A, Ueda H, Koh M, Watanabe G, Tanne K. Enhanced increase in pharyngeal airway size in Japanese class II children following a 1-year treatment with an activator appliance. *Int J Orthod Milwaukee*. 2013;24(4):35–40.

6. Hossain MZ. Technique training of myofunctional appliance: activators. *Orthopedics (BJO and DFO)*. 2011;2:(1).
7. Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. *Sleep Med*. 2000;1(1):21–32.
8. Iwamoto T, Takata Y, Kitamura N, Hasebe D, Kobayashi T, Saito C. Prognostic predictors on the efficacy of oral appliance therapy for obstructive sleep apnea syndrome. *Open J Stomatology*. 2012;2(3):201–221.
9. Lowe AA, Ono T, Ferguson KA, Pae EK, Ryan CF, Fleetham JA. Cephalometric comparisons of craniofacial and upper airway structure by skeletal subtype and gender in patients with obstructive sleep apnea. *Am J Orthod Dentofacial Orthop*. 1996;110(6):653–664.
10. Grewal N, Godhane AV. Lateral cephalometry: a simple clinical guide for assessment of nasopharyngeal free airway space in mouth breathers. *Contemp Clin Dent*. 2010;1(2):66–69.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication February 28, 2017
 Submitted in final revised form May 23, 2017
 Accepted for publication May 26, 2017
 Address correspondence to: Hiroshi Ueda PhD, DDS, Associate Professor, Department of Orthodontics, Hiroshima University, 1-2-3 Kasumi, Minami-ku, Hiroshima, Japan; Tel: +81-82-257-8656; Email: milim@hiroshima-u.ac.jp

DISCLOSURE STATEMENT

This clinical case was treated at Hiroshima University Hospital. All authors declare to have seen and approved of this case report. Authors declare no conflict of interest. Authors declare no financial support was received.