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In This Issue

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EDITORIALS

Bias in Dental Sleep Medicine Research: Does it Matter to Clinicians?

Leslie C. Dort, DDS, Diplomate, ABDSM, Editor-in-Chief *Journal of Dental Sleep Medicine*

Calgary, Alberta, Canada

Bias is "systematic error introduced into sampling or testing by selecting or encouraging one outcome or answer over others."[1]

Scientific studies are likely to be subject to various types of bias that could limit the generalizability of the results. As clinicians, we need to be aware of potential sources of bias and how they may be relevant to our specific patient populations.

Recognition and avoidance of bias in scientific research has been very well described.[2,3] Some familiarity with sources of bias gives clinicians tools to critically evaluate the literature.

Study designs of oral appliance outcomes, even randomized controlled trials, may have *selection bias* if the subjects included were limited to those: who have failed CPAP or are CPAP naïve, are of limited age range, are free of the common comorbidities associated with OSA, are compliant with therapy, or have been selected from limited clinical populations. Other designs risk bias when no control group is included. In studies reporting on oral appliance outcomes with no control group, one cannot know—for example—how many subjects may have developed TMJ symptoms or dry mouth over the course of time when not using oral appliances.

There may be systematic differences in the way patients were treated that could introduce *performance bias*. It is very difficult to blind investigators to what treatment subjects are getting and therefore investigators may unintentionally give different attention to one group over another. Some studies report on drop outs and use an intention to treat analysis. Others only include those who complete the protocol—a potential source of *attrition bias*. *Reporting bias* may also be present depending on the criteria used to determine which subjects' data is included in the analysis.

The use of questionnaires that have not been validated may introduce *instrument bias*. Non-validated questionnaires may have questions that inadvertently influence the answers that patients give. Subjects are likely to have *recall bias* when questioned regarding outcomes or side effects depending on time since they occurred. Compared to a general clinic population, subjects who know they are part of a study may be more or less willing to use an oral appliance despite questionable outcomes or significant side effects.

Keeping potential sources of bias in mind will help clinicians interpret study results and consider the relevance of the results to clinical practice.

CITATION

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REFERENCES

1. Bias. Merriam-Webster website. https://www.merriam-webster.com/dictionary/bias. Accessed June 12, 2017.
2. Introduction to sources of bias in clinical trials. In: Higgins JPT, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions*. Version 5.1.0. http://handbook.cochrane.org/chapter_8/8_4_introduction_to_sources_of_bias_in_clinical_trials.htm. Updated March 2011. Accessed June 12, 2017.
3. Pannucci CJ, Wilkins EG. Identifying and avoiding bias in research. *Plast Reconstr Surg*. 2010;126(2):619-625.

SUBMISSION & CORRESPONDENCE INFORMATION

DISCLOSURE STATEMENT

Dr. Dort is Editor-in-Chief of the *Journal of Dental Sleep Medicine*.

Safety and Efficacy of a Novel Oral Appliance in the Treatment of Obstructive Sleep Apnea

Damian Lavery, BDSc[1]; Irene Szollosi, PhD[2]; Stefan Czyniewski, BMedSci[3]; Fiona Beer, MSc[3]; Karen McCloy, BDSc, MS, MSMed; Christopher Hart, MPhil[2]

[1]National Dental Care, Brisbane, Queensland, Australia; [2]Oventus Medical Pty Ltd., Brisbane, Queensland, Australia; [3]Mobius Medical Pty Ltd., Sydney, New South Wales, Australia

Study Objectives: To establish the safety and efficacy of a novel oral appliance (O_2Vent Mono, Oventus Medical Pty Ltd., Brisbane, Australia), that incorporates a built-in enclosed airway, as an alternative treatment for obstructive sleep apnea (OSA).

Methods: A prospective, single-arm, single-center study was performed. Participants had mild-moderate OSA or continuous positive airway pressure (CPAP) intolerant severe OSA. Ambulatory polysomnography (PSG), subjective snoring, and subjective nasal obstruction were assessed at baseline and following acclimatization with the device. Baseline mandibular protrusion was set at 50% and was increased to a maximum of 85% if required as determined by questionnaire and PSG. Participants with a ≥ 50% reduction in apnea-hypopnea index (AHI) were classified as responders. Compliance was recorded via a questionnaire.

Results: In 29 participants (20 males, 9 females), mean ± standard deviation age = 49.3 ± 8.6 years, body mass index = 29.9 ± 6.1 kg/m², AHI decreased from 41.8 ± 26.5 to 16.2 ± 15.4 ($P < .001$) or 62.5 ± 21.1%. Time spent below 90% oxygen saturation as assessed by pulse oximetry improved from 9.3 ± 12.7% to 2.2 ± 3.4% ($P = .001$). Seventeen participants (59%) had subjective nasal obstruction and 22 (75.9%) were classified as responders. Subgroup analysis between those with nasal obstruction (NO) and without nasal obstruction (NNO) revealed no significant difference in percentage of change in AHI from baseline (NO = 66.3 ± 18.1%, NNO = 57.0 ± 24.6%, $P = .280$) or response rate (NO = 76.5%, NNO = 75%, chi-square = 0.930). Overall compliance was 82.8%, and three minor transient device-related adverse events occurred.

Conclusions: This novel device was safe, effective, and well tolerated in a group of participants with relatively severe OSA. There was a clinically and statistically significant reduction in AHI of 62% as well as improvement in oxygen saturation. Importantly, the efficacy and response do not appear to be reduced by the presence of nasal obstruction.

Clinical Trial Registration: Trial name: A prospective, single arm, single center pilot trial to establish the safety and efficacy of the Oventus device to treat mild to moderate obstructive sleep apnea and snoring. URL: https://www.anzctr.org.au/Trial/Registration/TrialReview.aspx?id=367532. Registration number: ACTRN12615000028505

Keywords: mandibular advancement device, mandibular advancement splint, obstructive sleep apnea, oral appliance

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INTRODUCTION

Obstructive sleep apnea (OSA) is characterized by repetitive occlusions of the upper airway during sleep, resulting in sleep fragmentation and oxygen desaturations.[1] The prevalence of OSA is reported to be 34% in men and 17% in women, whereas OSA accompanied by symptoms of sleepiness is reported to occur in 14% of men and 5% of women between 30–70 years old.[2] OSA is an important contributor to cardiovascular disease,[3–7] stroke,[4,8,9] and depression[10] and is a recognized risk factor for motor vehicle accidents,[11–13] workplace injuries, and loss of productivity.[14,15]

Although continuous positive airway pressure (CPAP) remains the treatment of choice for OSA, custom-fit oral appliances that advance the mandible—a mandibular advancement device (MAD)—are emerging as an alternative treatment option. These devices are used primarily in patients with mild to moderate OSA, CPAP intolerant OSA, and primary snorers.[16,17] Studies consistently demonstrate that CPAP reduces the apnea-hypopnea index (AHI) further than oral appliances; however, CPAP efficacy is likely to be offset by reductions in usage and adherence.[18] In support of this, recent studies have demonstrated that the health outcomes in patients with moderate to severe OSA after treatment with CPAP and MAD are similar.[19,20] Patients with high nasal resistance may have difficulty in using both CPAP and traditional oral appliances, with studies reporting high nasal resistance being associated with both CPAP [21] and oral appliance [22] intolerance. High nasal resistance is an indication of reversible or irreversible nasal obstruction and its prevalence is high in OSA.[23]

The novel oral appliance studied in the current study (O_2Vent Mono, Oventus Medical Pty Ltd., Brisbane, Australia) (**Figure 1**) incorporates both mandibular advancement to reduce pharyngeal collapsibility and an enclosed airway that allows airflow through the device to circumvent nasopharyngeal obstruction. Titration is achieved by sequential relining of the upper silicone insert, which is changed over to a more retentive dual laminate material after the optimum level of advancement is achieved. Thus, although a monobloc design, it can be considered as a titratable device during the treatment

Figure 1—Oventus O$_2$Vent Mono device.

The novel oral appliance incorporates an enclosed airway that allows airflow through the device while maintaining a stable jaw position.

optimization and acclimatization period. The aim of the current study was to evaluate the safety and efficacy of this novel oral appliance for the treatment of OSA. In addition, we wanted to examine whether compliance with or the response rate to the device were influenced by the presence of subjective nasal obstruction.

METHODS

This was a single-center, prospective pilot study to establish the safety and efficacy of the oral appliance in treating OSA. The study protocol was approved by an independent Human Research Ethics Committee (Bellberry Limited, Australia), and appropriate informed consent was obtained from all participants. Clinical trial and data management was performed by a contract research organization (Mobius Medical Pty Ltd, Australia) to ensure independent oversight of regulatory compliance, monitoring, and reporting.

Participant Selection

Male and female participants aged 18 years and older were recruited from Turbot Street Medical Centre presenting for consideration of oral appliance therapy. Inclusion criteria were grade 2–3 snoring (regular: more than 3 nights/wk or every night) and either mild to moderate sleep apnea (AHI > 5 and < 30 events/h) and recommended for oral appliance therapy, or CPAP-intolerant patients with moderate to severe sleep apnea (AHI ≥ 15 events/h). Additional inclusion criteria were eligibility to receive a MAD with adequate dentition for retention and the ability to provide written informed consent to all study procedures. Diagnosis was based on polysomnography (PSG) within the past 12 months by a qualified sleep physician. Exclusion criteria were pregnancy or lactation, current participation in another clinical trial, periodontal disease, exaggerated gag reflex, medication usage that could influence respiration or sleep (eg, regular use of sedatives, heavy alcohol consumption), OSA with uncontrolled or untreated cardiovascular disease, central sleep apnea ≥ 5 events/h, previous uvulopalatopharyngoplasty (UPPP), and severe somatic or psychiatric disorders.

Three-dimensional cone beam computed tomography (CBCT) scans were completed to screen for dental pathology and to assess the temporomandibular joints.

Oral Appliance

The initial jaw position at 50% of maximum mandibular protrusion was recorded by a researcher who is a qualified dentist using a George gauge with a 5-mm bite fork (Great Lakes Orthodontics, Tonawanda, New York, United States) to obtain 5 mm of vertical opening. Maxillary and mandibular impressions were taken using Imprint 4 Preliminary VPS Impression Material (3M ESPE, Landsberg am Lech, Germany). The impressions were poured with dental stone, and the models and bite were scanned using TRIOS 3 Scanner (3Shape, Szczecin, Poland) and converted to a sterolithography file. Computer-aided design using proprietary software was used to customize the size and shape of the bimaxillary oral appliance.

The inner core of the oral appliance (**Figure 1**) was printed three-dimensionally using Ti6Al4V ELI Titanium Powder Grade 23 (Arcam AB, Mölndal, Sweden). The titanium core has a customized airway, which divides at the level of the canines, and passes posteriorly between the occlusal surfaces of the maxillary and mandibular teeth to deliver air to the oropharynx in the region of the second molars, with the aim of bypassing nasopharyngeal obstruction during sleep and allowing for breathing through the low-resistance device airway when nasal flow is compromised. The cross-sectional area of the bilateral airway is consistent with the average patent nasal airway and a lip seal can be maintained around the anterior opening.

After polishing the titanium core, a silicon primer was applied (NuSil Technology, Carpinteria, California, United States) to allow for bonding of the maxillary silicone inserts, constructed using Bona-Bite Crystal Vision (DMP Ltd, Markopoulo, Greece). The mandibular insert was a dual laminate material (Erkodent, Wembley, Western Australia, Australia) with a hard outer lining and soft inner lining for greater retention and durability. The upper silicone inserts were positioned with the mandibular protrusion as obtained from the bite record and impressions. Further titration during the protocol was performed by sequential relining and replacement of the upper silicone inserts, resulting in the mandible being in a more advanced position.

Study Protocol

During the baseline visit, demographic information was collected and sleep questionnaires administered. Subjective nasal obstruction was assessed at the baseline visit using a 10-point scale where 0 = no obstruction, 5 = moderate obstruction, and 10 = complete obstruction of nasal airflow. Based on the response to this question, nasal obstruction was recorded as a dichotomous variable with 0 being no, and all other responses as yes. A dental examination was performed to ensure dental suitability for oral appliance therapy by an investigator who is a qualified dentist, who then completed records for construction of the device. Baseline physiological parameters were obtained prior to insertion of the device using a Somte PSG Level II device

Table 1—Sleep and respiratory indices at baseline and following treatment with the oral appliance (n = 29).

	Baseline mean ± SD	O$_2$Vent Mono mean ± SD	P
TST, min	403.0 ± 49.7	399.9 ± 68.7	.801
SE, %	94.2 ± 4.1	92.6 ± 4.4	.129
S1, %	10.5 ± 17.1	5.2 ± 4.1	.093
S2, %	47.4 ± 15.1	50.9 ± 11.1	.240
SWS, %	24.1 ± 15.4	20.7 ± 9.5	.160
REM, %	25.3 ± 25.5	23.2 ± 7.0	.681
Arousal index, events/h	40.6 ± 16.3	22.4 ± 12.4	< .001
AHI total, events/h	41.8 ± 26.5	16.2 ± 15.4	< .001
AHI NREM supine, events/h	36.7 ± 31.9	10.8 ± 17.9	< .001
AHI NREM nonsupine, events/h	36.8 ± 29.2	10.1 ± 15.5	< .001
AHI REM supine, events/h	43.5 ± 30.6	21.1 ± 28.0	.002
AHI REM nonsupine, events/h	43.4 ± 30.8	20.4 ± 20.4	< .001
Minimum SpO$_2$	82.7 ± 8.9	86.3 ± 5.9	.005
% time < 90% SpO$_2$	9.3 ± 12.7	2.2 ± 3.4	.001

Snoring Frequency	Baseline n	O$_2$Vent Mono n	
Rarely	0	2	
Occasionally	5	1	< .001
Often	5	2	
Every night	19	0	

AHI = apnea-hypopnea index, NREM = non-rapid eye movement, REM = rapid eye movement, S1 = Stage 1, S2 = Stage 2, SD = standard deviation, SE = sleep efficiency, SpO$_2$ = saturation of peripheral oxygen, SWS = slow wave sleep, TST = total sleep time.

(Compumedics, Abbotsford, Victoria, Australia) measuring: electroencephalogram, electrooculogram, submental electromyogram, electrocardiogram, oronasal flow using nasal cannula, thoracic and abdominal respiratory effort, snore sound, and body position. All PSG was conducted and scored according to *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications* Version 2.2. The recommended hypopnea rule was adopted (ie, hypopnea defined as a ≥ 3% oxygen desaturation from pre-event baseline and/or the event is associated with an arousal). Analysis was performed by a single registered polysomnographic technologist (RPSGT) and reported by a qualified sleep physician. Participants who fulfilled the inclusion criteria were fitted with the oral appliance and given care and maintenance instructions before being sent home. Following 1 week to 1 month of acclimatization, participants returned for a follow-up visit where a clinical review for compliance, comfort, and efficacy of the device was conducted via questionnaires. If participants were compliant, defined as using the device for at least 4 h/night for at least 5 d/wk, an additional level II PSG was conducted. If participants were nonresponders to treatment (< 50% reduction of AHI from baseline) as determined by PSG, the oral appliance was titrated to 75% maximum protrusion and another clinical review with PSG was performed after 1 week to 1 month. If the second treatment PSG at 75% protrusion indicated nonresponse, and further advancement was tolerated, a final titration to 85% protrusion was performed with a final clinical review and PSG after 1 week to 1 month.

Based on the Simons II stage design, a sample size of 30 participants would yield 80% power with 95% confidence to rule out a 5% response rate in favor of at least a 20% response rate. Assessment of device-related adverse events occurred at each visit and was reported according to ISO 14155-2011. Events of interest included excessive salivation, temporomandibular joint pain, gum irritation, mouth dryness, jaw discomfort, tooth loosening, tooth wear, and jaw set.

RESULTS

Thirty participants were enrolled into the study, with one withdrawal due to personal reasons prior to a device being issued. Results are presented in **Table 1** as mean ± standard deviation for 29 participants (20 males and 9 females) who completed the protocol with age = 49.3 ± 8.6 years, body mass index = 29.9 ± 6.1 kg/m^2. Fifteen participants (51.7%) were initially recommended for CPAP but did not tolerate CPAP prior to inclusion in the study. The study cohort therefore had a greater representation of participants with severe OSA (62%).

Seventeen participants (59%) were advanced to 75% maximum protrusion and four (14%) were advanced to 85%, with a mean protrusion of 69.5 ± 12.7% achieved over a mean follow-up period of 82.1 ± 33.3 days (**Table 2**).

Safety
The device had a favorable safety profile with no serious device-related adverse events reported during the trial. Of the four adverse events reported, only three were device related and all

Table 2—Mandibular advancement summary.

Number of Advancements	n (%)
0 (50% protrusion)	8 (28)
1 (75% protrusion)	17 (59)
2 (85%protrusion)	4 (14)
	mean ± SD
Average final mandibular advancement (%)	69.5 ± 12.7
Average follow-up (days)	82.1 ± 33.3

SD = standard deviation.

Figure 2—AHI at baseline and following treatment with the oral appliance.
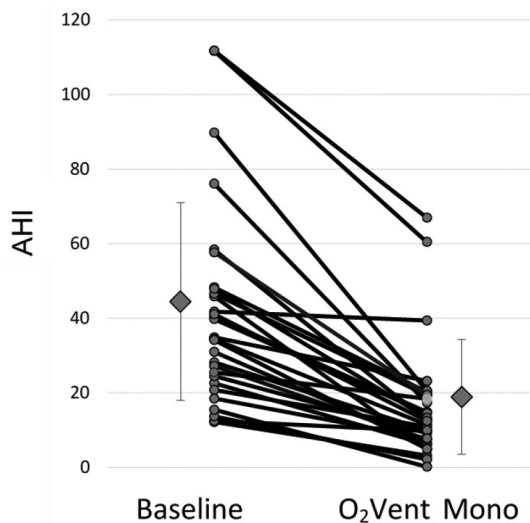
Individual responses are denoted by gray circles while group mean ± standard deviation is denoted by gray diamond. *P* < .001. AHI = apnea-hypopnea index.

Figure 3—Distribution of OSA severity pretreatment and posttreatment.
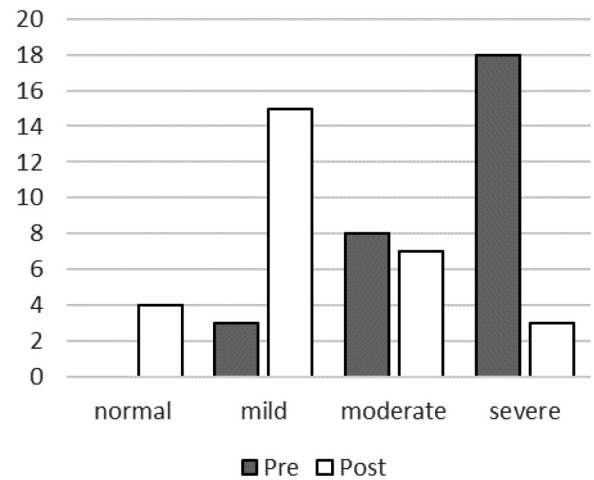
PRE TREATMENT		POST TREATMENT	
mild	3	normal	2
		mild	1
moderate	8	normal	0
		mild	6
		moderate	2
severe	18	normal	2
		mild	7
		moderate	6
		severe	3
		Total	**29**

events were anticipated. These were jaw pain (on the first night only) for one participant whereas another participant experienced two events, sore tongue and mouth ulcers, all of which resolved and were transient in nature.

Effect on OSA

The mean AHI at baseline was 41.8 ± 26.5 (range 12.2 to 111.8) events/h. After the final titration, the group mean AHI was 16.2 ± 15.4 (range 0.2 to 67.0) events/h. The mean reduction in AHI was 24.7 ± 16.8 (range 2.4 to 69.2) events/h, which is statistically and clinically significant (*P* < .001). This represented a reduction in AHI of 62.5 ± 21.1% from baseline (**Figure 2**), with improvement observed in supine and nonsupine rapid eye movement and non-rapid eye movement sleep. Arousal index improved from 40.6 ± 16.3 to 22.4 ± 12.4 events/h (*P* < .001) (**Table 1**).

The time spent below 90% oxygen saturation (T90%) improved from 9.3 ± 12.7% to 2.2 ± 3.4% (*P* = .001) as did the minimum saturation of peripheral oxygen, SpO_2, from 82.7 ± 8.9% to 86.3 ± 5.9% (*P* = .005).

Response rate to treatment according to multiple definitions of AHI reduction were as follows: (1) 22 participants (76%) had

a 50% reduction in AHI compared to baseline, (2) 11 participants (38%) obtained a partial response with AHI < 10, and (3) 4 participants (14%) obtained complete normalization with AHI < 5 events/h. In this cohort, 90% of participants were classified as having moderate or severe OSA (28% and 62%, respectively), and we evaluated the changes in OSA severity pretreatment and posttreatment according to the severity of the AHI (**Figure 3**). Twenty-three participants (79%) obtained a reduction in the classification of OSA severity. Of those with moderate to severe OSA at baseline, 15 of 26 participants (58%) shifted into the normal to mild category.

All participants reported snoring at baseline (**Table 1**) with 19 (66%) reporting snoring every night and 19 (66%) reporting very loud snoring that can be heard in adjacent rooms. At the conclusion of the study, 24 (82%) reported no snoring, and the remaining 5 (18%) all indicated that the frequency and/or intensity of snoring had improved.

Compliance

The device was well tolerated by participants who indicated good compliance with the device and positive feedback when reporting on comfort. Of the 29 participants who received a device, the average usage of the device was 6 nights/wk. Of the nights the device was used, participants averaged 7 hours of usage (range 4–8 hours). Participants were deemed compliant if they used the device for at least 4 hours for at least

5 days a week. Based on the user comfort survey, compliance was 82.8%.

Nasal Obstruction

Seventeen of the 29 participants (59%) were documented to have subjective nasal obstruction. Subgroup analysis between those with nasal obstruction (NO) and without nasal obstruction (NNO) revealed no significant difference in the percentage of reduction in AHI (NO = 66.3 ± 18.1%, NNO = 57.0 ± 24.6%, P = .280) or response rate defined as 50% reduction in AHI (NO = 76.5%, NNO = 75%, chi^2 = 0.928). Importantly, no differences were observed in compliance between those with and without nasal obstruction (NO = 82.4%, NNO = 83.3%, chi-square = 0.945) (**Figure 4**).

DISCUSSION

The results of this study demonstrate that the novel oral appliance is safe and effective in treating patients with mild to moderate OSA as well as CPAP-intolerant severe OSA. Importantly, the efficacy, response and compliance with treatment do not appear to be reduced by the presence of nasal obstruction.

Comparisons of different MADs in the treatment of OSA are complicated by many factors including differences in appliance design, level of protrusion and vertical dimension, definition of success, and duration of follow-up. In a review of mandibular advancement therapy, Marklund et al. reported that the mean reduction of AHI from all studies included in the review was 55% (range 28–80),[18] whereas Sutherland et al. reported success rates in the range of 30% to 85% for posttreatment AHI < 10 events/h.[24] The current study achieved a mean reduction in AHI of 62%, with 38% of the cohort achieving posttreatment AHI < 10 events/h. Although these results are broadly in line with other studies of oral appliance efficacy, it is important to note that the current study included a large proportion of patients (90%) with moderate to severe OSA, making direct comparisons to other studies difficult. However, a recent study by Gjerde et al. reported treatment outcomes for 106 patients with moderate to severe OSA using mixed monobloc and titratable appliances.[25] The authors report 75% of patients achieved treatment success as defined by > 50% reduction in AHI, with a reduction in AHI in those with severe apnea from 41.4 to 17.4 events/h. Using this definition of success, our overall cohort had strikingly similar results both for overall success rate (76%) and decrease in mean AHI from 41.8 to 16.2 events/h. However, complete resolution of OSA with AHI < 5 events/h in the current study was 14% compared to 38% reported by Gjerde et al.,[25] which may be explained by the higher proportion of individuals with severe apnea in our cohort (62% versus 25%) despite mean AHI being similar between groups. The differences of treatment success rates obtained with various definitions as well as the individual variability in treatment responses is well established. Although complete resolution of OSA was not achieved in most participants, it is important to note that 79% had a reduction in OSA severity classification; 50% of those with severe OSA at baseline moved to the normal to mild category after treatment. This is a clinically important result as a dose-response relationship has been reported between OSA severity and all-cause mortality, cardiovascular mortality,[5,7] and

Figure 4—Response rate by nasal obstruction.

No difference observed in response rate between participants with and without nasal obstruction.
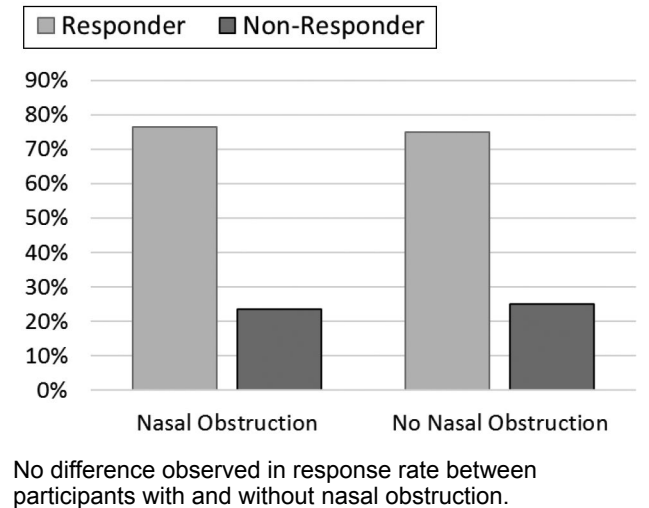
incident events[26] and those with mild OSA may be at relatively low risk of the development of cardiovascular complications. All of the participants with severe OSA were intolerant to CPAP, and a reduction in OSA severity with the device may offer the opportunity to provide health benefits even if the AHI is not completely normalized. The posttreatment AHI may be an important determinant of when combination therapies may be considered, with recent studies reporting CPAP and oral appliances used in combination may be of benefit.[27] Combination therapy using CPAP and MAD may result in lower pressure requirements and improve tolerance and treatment effectiveness in a group that clinically is difficult to treat.

The effect of vertical opening on pharyngeal collapsibility is a topic of current debate and has been evaluated with conflicting results. In one study, increasing the vertical opening was found to have a detrimental effect on pharyngeal collapse as assessed during sleep endoscopy at the base of the tongue[28]; however, in another study of individuals with apnea, there was no statistically significant difference in sagittal airway at 75% protrusion and 5 or 10 mm vertical dimension.[29] In another randomized crossover study, a vertical dimension of 4 or 14 mm did not affect MAD efficacy, although the increase in vertical dimension was associated with a decrease in compliance.[30] The required vertical opening for the device used in the current study was 5 mm obtained with a standard 5-mm bite fork. This is within the lower limits of tested vertical dimensions and therefore unlikely to have an adverse effect on either appliance efficacy or compliance.

Previous studies of oral appliances demonstrate self-reported short-term compliance to be 76% to 95%. Vanderveken et al. conducted a 3-month prospective clinical trial that evaluated oral appliance compliance using an embedded microsensor thermometer to measure adherence. Regular use was defined as > 4 hours per day on > 70% of nights and was achieved in 84% of 43 patients who had a complete dataset in the study.[31] With the same definition of compliance assessed subjectively, 83% of participants in the current study were compliant at the conclusion of the study, which is comparable with previous

research. However, the duration of follow-up was variable and further research is required to demonstrate long-term objective compliance with this device.

Long-term studies show that adverse effects with MADs are common, but are minor and well tolerated, resulting in good compliance.[32] Common short-term side effects include excessive salivation, gum irritation, mouth dryness, and jaw discomfort, with reports that these adverse effects generally last less than 3 weeks.[33–35] Only three device-related adverse events were reported by two participants during this study, all of which were anticipated and consistent with known short-term side effects. These transient events did not affect compliance or the efficacy of the device, with participants continuing to use their device after resolution of the events. Longer term side effects of MAD wear include minor tooth movement, changes in the overbite and overjet relationships, changes in incisal proclination, and an increase in lower facial height.[36–40] Although dental changes may be evident after 6 months of treatment[40] and are generally well tolerated, there is some evidence that these changes are progressive and continue with ongoing treatment.[41] It is anticipated that similar long-term side effects would be seen with the device used in this study; however, a larger cohort with longer follow-up is required to evaluate the long-term changes associated with this device.

As well as demonstrating safety and efficacy of the novel oral appliance, we were able to show that compliance and efficacy, as measured by the response rate (> 50% reduction in AHI), was not different between patients with and without nasal obstruction. This was an interesting finding as increased nasal obstruction is common in OSA[23] and has been associated with both CPAP[21] and oral appliance intolerance.[22] For patients who cannot tolerate CPAP or traditional oral appliances, there are very few treatment options left to consider. We speculate that the similar compliance and response rate of approximately 75% in those with and without subjective nasal obstruction observed in the current study is due to the presence of the built-in device airway, which may improve tolerance and efficacy in patients with increased nasal obstruction who otherwise may find it difficult to breathe exclusively via the nasal route. This, however, needs to be validated in a larger cohort with objective assessment of nasal function.

This was a pilot study that established the safety and efficacy of a novel oral appliance with a built-in enclosed airway in the treatment of mild to moderate OSA and CPAP intolerant OSA. Limitations of the study include small sample size and a relatively short follow-up period. Nasal obstruction was evaluated using subjective symptoms and other health outcomes were not evaluated. Last, the final PSG during the treatment optimization was conducted with silicone as the upper insert material. In clinical practice, this is changed to a more retentive and durable dual laminate material after the optimum position has been identified. An additional sleep study with the change in material at the same level of advancement was not performed and as a consequence it is possible that the final efficacy of this device may have been underestimated. Further investigation is required to determine if treatment effectiveness is affected by the change in material. The results of this study are promising and further research in a larger cohort with

objective assessments of nasal obstruction, health outcomes, and compliance are warranted as are studies to evaluate the contribution of the built-in airway to improvements in efficacy and compliance.

CONCLUSIONS

This novel oral appliance was found to be safe and effective in a cohort of patients with relatively severe OSA who experienced very few side effects and demonstrated good compliance. Use of the device was associated with a clinically and statistically significant reduction in AHI in the order of 62%, which is broadly in line with published literature of oral appliance efficacy in the treatment of OSA. According to the frequently used definition of treatment success (> 50% reduction in AHI), 76% of participants were responders to treatment. Although a complete resolution of OSA was achieved in a small number of participants, this is partly explained by the relatively severe OSA seen in the current cohort. Importantly, we observed a reduction in OSA severity classification that is likely to provide health benefits, and the efficacy, response, and compliance with treatment did not appear to be reduced by the presence of subjective nasal obstruction. Larger studies with long-term follow up and objective assessment of nasal obstruction and compliance are required; however, the results of this study provide encouraging data to support the notion that patients with OSA and increased nasal resistance may also benefit from this device.

REFERENCES

1. White DP. Pathogenesis of obstructive and central sleep apnea. *Am J Respir Crit Care Med*. 2005;172(11):1363–1370.

2. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol*. 2013;177(9):1006–1014.

3. Hla KM, Young T, Hagen EW, et al. Coronary heart disease incidence in sleep disordered breathing: The Wisconsin Sleep Cohort Study. *Sleep*. 2015;38(5):677–684.

4. Young T, Finn L, Peppard PE, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin Sleep Cohort. *Sleep*. 2008;31(8):1071–1078.

5. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med*. 2000;342(19):1378–1384.

6. Punjabi NM, Caffo BS, Goodwin JL, et al. Sleep-disordered breathing and mortality: a prospective cohort study. *PLoS Med*. 2009;6(8):e1000132.

7. Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2001;163(1):19–25.

8. Lipford MC, Flemming KD, Calvin AD, et al. Associations between cardioembolic stroke and obstructive sleep apnea. *Sleep*. 2015;38(11):1699–1705.

9. Redline S, Yenokyan G, Gottlieb DJ, et al. Obstructive sleep apnea-hypopnea and incident stroke: the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2010;182(2):269–277.

10. Gupta MA, Simpson FC, Lyons DCA. The effect of treating obstructive sleep apnea with positive airway pressure on depression and other subjective symptoms: a systematic review and meta-analysis. *Sleep Med Rev*. 2016;28:55–68.

11. Ward KL, Hillman DR, James A, et al. Excessive daytime sleepiness increases the risk of motor vehicle crash in obstructive sleep apnea. *J Clin Sleep Med*. 2013;9(10):1013–1021.

12. Young T, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. *Sleep*. 1997;20(8):608–613.

13. Sassani A, Findley LJ, Kryger M, Goldlust E, George C, Davidson TM. Reducing motor-vehicle collisions, costs, and fatalities by treating obstructive sleep apnea syndrome. *Sleep*. 2004;27(3):453–458.

14. AlGhanim N, Comondore VR, Fleetham J, Marra CA, Ayas NT. The economic impact of obstructive sleep apnea. *Lung*. 2008;186(1):7–12.

15. Deloitte Access Economics. Re-awakening Australia: the economic cost of sleep disorders in Australia, 2010. Sleep Health Foundation website. http://www.sleephealthfoundation.org.au/pdfs/news/Reawakening%20Australia.pdf. Published October 2011. Accessed October 2016.

16. Epstein L, Kristo D, Strollo P, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009;5(3):263–276.

17. Mansfield D, Antic N, McEvoy R. How to assess, diagnose, refer and treat adult obstructive sleep apnoea: a commentary on the choices. *Med J Aust*. 2013;199(8):S21–S26.

18. Marklund M, Verbraecken J, Randerath W. Non-CPAP therapies in obstructive sleep apnoea: mandibular advancement device therapy. *Eur Respir J*. 2012;39(5):1241–1247.

19. Bratton DJ, Gaisl T, Wons AM, Kohler M. CPAP vs mandibular advancement devices and blood pressure in patients with obstructive sleep apnea: a systematic review and meta-analysis. *JAMA*. 2015;314(21):2280–2293.

20. Phillips CL, Grunstein RR, Darendeliler MA, et al. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea. *Am J Respir Crit Care Med*. 2013;187(8):879–887.

21. Sugiura T, Noda A, Nakata S, et al. Influence of nasal resistance on initial acceptance of continuous positive airway pressure in treatment for obstructive sleep apnea syndrome. *Respiration*. 2007;74(1):56–60.

22. Zeng B, Ng AT, Qian J, Petocz P, Darendeliler MA, Cistulli PA. Influence of nasal resistance on oral appliance treatment outcome in obstructive sleep apnea. *Sleep*. 2008;31(4):543–547.

23. Georgalas C. The role of the nose in snoring and obstructive sleep apnoea: an update. *Eur Arch Otorhinolaryngol*. 2011;268(9):1365–1373.

24. Sutherland K, Vanderveken OM, Tsuda H, et al. Oral appliance treatment for obstructive sleep apnea: an update. *J Clin Sleep Med*. 2014;10(2):215–227.

25. Gjerde K, Lehmann S, Berge ME, Johansson AK, Johansson A. Oral appliance treatment in moderate and severe obstructive sleep apnoea patients non-adherent to CPAP. *J Oral Rehabil*. 2016;43(4):249–258.

26. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet*. 2005;365(9464):1046–1053.

27. El-Solh AA, Moitheennazima B, Akinnusi ME, Churder PM, Lafornara AM. Combined oral appliance and positive airway pressure therapy for obstructive sleep apnea: a pilot study. *Sleep Breath*. 2011;15(2):203–208.

28. Vroegop AVMT, Vanderveken OM, Van de Heyning PH, Braem MJ. Effects of vertical opening on pharyngeal dimensions in patients with obstructive sleep apnoea. *Sleep Med*. 2012;13(3):314–316.

29. Piskin B, Karakoc O, Genc H, et al. Effects of varying mandibular protrusion and degrees of vertical opening on upper airway dimensions in apneic dentate subjects. *J Orofac Orthop*. 2015;76(1):51–65.

30. Pitsis AJ, Darendeliler MA, Gotsopoulos H, Petocz P, Cistulli PA. Effect of vertical dimension on efficacy of oral appliance therapy in obstructive sleep apnea. *Am J Respir Crit Care Med*. 2002;166(6):860–864.

31. Vanderveken OM, Dieltjens M, Wouters K, De Backer WA, Van de Heyning PH, Braem MJ. Objective measurement of compliance during oral appliance therapy for sleep-disordered breathing. *Thorax*. 2013;68(1):91–96.

32. Brette C, Ramanantsoa H, Renouardiere J, Renouardiere R, Roisman G, Escourrou P. A mandibular advancement device for the treatment of obstructive sleep apnea: long-term use and tolerance. *Int Orthod*. 2012;10(4):363–376.

33. Mehta A, Qian JIN, Petocz P, Darendeliler MA, Cistulli P. A randomized, controlled study of a mandibular advancement splint for obstructive sleep apnea. *Am J Respir Crit Care Med*. 2001;163(6):1457–1461.

34. 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(6):501–510.

35. Hammond RJ, Gotsopoulos H, Shen G, Petocz P, Cistulli PA, Darendeliler MA. A follow-up study of dental and skeletal changes associated with mandibular advancement splint use in obstructive sleep apnea. *Am J Orthod Dentofacial Orthop*. 2007;132(6):806–814.

36. Almeida FR, Lowe AA, Otsuka R, Fastlicht S, Farbood M, Tsuiki S. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: part 2. study-model analysis. *Am J Orthod Dentofacial Orthop*. 2006;129(2):205–213.

37. Marklund M. Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea. *Am J Orthod Dentofacial Orthop*. 2006;129(2):214–221.

38. Martinez-Gomis J, Willaert E, Nogues L, Pascual M, Somoza M, Monasterio C. Five years of sleep apnea treatment with a mandibular advancement device. Side effects and technical complications. *Angle Orthod*. 2010;80(1):30–36.

39. Doff MH, Finnema KJ, Hoekema A, Wijkstra PJ, de Bont LG, Stegenga B. Long-term oral appliance therapy in obstructive sleep apnea syndrome: a controlled study on dental side effects. *Clin Oral Investig*. 2013;17(2):475–482.

40. Robertson C, Herbison P, Harkness M. Dental and occlusal changes during mandibular advancement splint therapy in sleep disordered patients. *Eur J Orthod*. 2003;25(4):371–376.

41. Pliska BT, Nam H, Chen H, Lowe AA, Almeida FR. Obstructive sleep apnea and mandibular advancement splints: occlusal effects and progression of changes associated with a decade of treatment. *J Clin Sleep Med*. 2014;10(12):1285–1291.

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SUBMISSION & CORRESPONDENCE INFORMATION

DISCLOSURE STATEMENT

The Efficacy of a Titrated Tongue Stabilizing Device on Obstructive Sleep Apnea and the Quality of Life: A Clinical Trial Study Protocol

Kentaro Okuno, PhD[1]; Mona M. Hamoda, BDS, MSc, MHSc[1]; Waled M. Alshhrani, BDS, MS[1,2]; John A. Fleetham, PhD[3];
Najib T. Ayas, PhD[3]; Robert Comey, MD[4]; Alan A. Lowe, DMD, PhD[1]; Benjamin T. Pliska, DDS, MS, FRCD(C)[1];
Fernanda R. Almeida, DDS, MSc, PhD[1]

[1]Department of Oral Health Sciences, Faculty of Dentistry, University of British Columbia, Vancouver, British Columbia, Canada; [2]Department of Prosthetic Dental Sciences, College of Dentistry, King Saud University, Riyadh, Saudi Arabia; [3]Division of Respiratory Medicine, Department of Medicine, University of British Columbia, Vancouver, British Columbia, Canada; [4]Division of Psychiatric Medicine, Department of Medicine, University of British Columbia, Vancouver, British Columbia, Canada

STUDY OBJECTIVES: A tongue stabilization device (TSD) is a preformed appliance that uses suction to maintain the tongue in a protruded position and improves upper airway size and function. TSDs are simple and less expensive than other treatment options currently available for obstructive sleep apnea (OSA), including continuous positive airway pressure (CPAP) and the mandibular advancement device (MAD). The following is a description of an ongoing clinical trial. The main objective of the clinical trial is to determine the efficacy of TSD treatment for sleep-disordered breathing, daytime sleepiness, and quality of life. The second objective is to identify the subjective compliance and side effects of TSD treatment. The third objective is to determine the efficacy of titration of TSDs to compare initial treatment with TSD, 4 mm titrated TSD, and 7 mm titrated TSD.

METHODS: Sixty patients with OSA will be recruited for this study. Each participant will complete a series of validated questionnaires and undergo level III sleep monitoring to evaluate their baseline OSA. The TSD appliances will be provided to each patient and will be titrated to hold the tongue forward in a stepwise fashion using a 4- or 7-mm titration accessory before repeating the questionnaires and sleep monitoring. Finally, we will perform a detailed split-night polysomnography (PSG), half of the night with the TSD and the other half without the device.

CONCLUSIONS: The evidence provided by this trial will improve the management of patients with OSA, especially those who cannot receive or tolerate CPAP and/or a MAD. The results of this trial will reveal the potential of the TSD as a treatment option for OSA.

CLINICAL TRIAL REGISTRATION: United States Clinical Trials Registry, ID: NCT02329925.

KEYWORDS: obstructive sleep apnea, oral appliance, quality of life, tongue stabilization device

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INTRODUCTION

Obstructive sleep apnea (OSA) is characterized by recurrent episodes of partial or complete upper airway collapse during sleep and is highly prevalent in the general population.[1,2] Daytime consequences of OSA include a range of symptoms including excessive sleepiness, neurocognitive impairment, and mood disturbance, which significantly impair quality of life (QOL).[3] In addition, there is an increased incidence of cardiovascular mortality, stroke, and heart attack.[4–6] Hence, OSA is a major public health problem, imposing a financial burden on health care systems.[7,8]

There are a variety of treatment options currently available for OSA, ranging from lifestyle modifications such as weight loss, to invasive soft tissue and/or orthognathic surgery. Continuous positive airway pressure (CPAP) is the most efficient treatment for OSA and has been demonstrated to improve many health outcomes, including sleepiness and QOL, and to reduce the incidence of cardiovascular events.[9,10] Despite these changes, adherence is often poor, with many patients either rejecting treatment or only partially tolerating it, which can result in untreated OSA.[11]

Use of a mandibular advancement device (MAD) is a widely used treatment for OSA. Although the overall effect of these devices on sleep-disordered breathing may be inferior to CPAP, adherence is generally higher.[12] Sleepiness, blood pressure, and disease-specific QOL improve as a result of treatment by MAD and CPAP by similar amounts.[13,14] CPAP and MAD are associated with significant costs.

The tongue stabilization device (TSD) is a preformed appliance that uses suction to hold the tongue in a protruded position, aiming at increasing upper airway size, and is more simple and less expensive than CPAP and MAD. TSD treatment has been reported to improve OSA, with demonstrated reductions in apnea-hypopnea index (AHI) and arousal frequency similar to MAD.[15] However, TSD is associated with poorer adherence when compared to MAD treatment.[15] Unlike CPAP, where the optimal pressure can be titrated, and MAD, which has the ability to titrate the amount of mandibular protrusion, TSD is limited by the inability to standardize the degree of tongue protrusion, which may be important to maximize the treatment effects.

Figure 1—Tongue stabilization device and titration accessories.
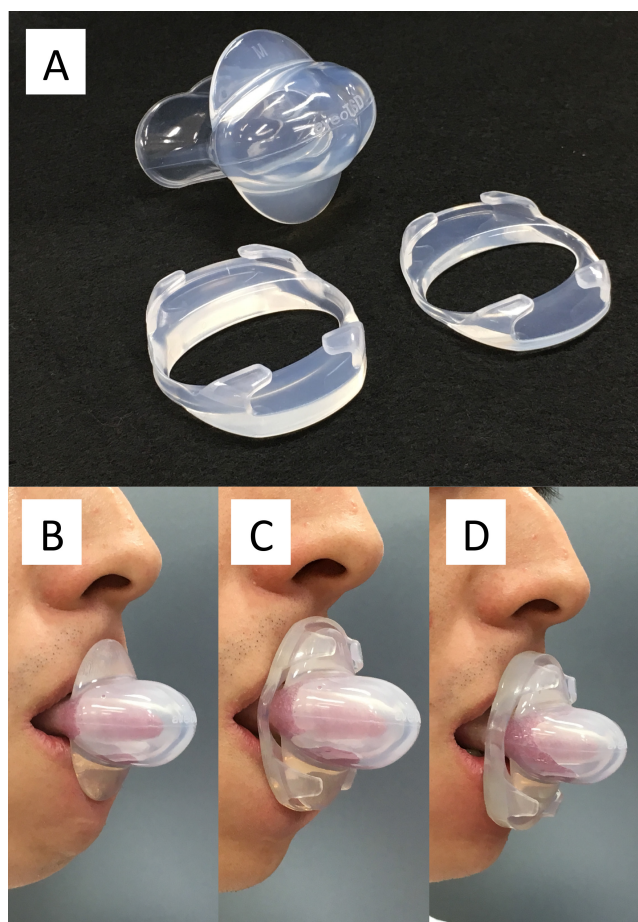
Tongue stabilization device (TSD), 4 mm and 7 mm titration accessory **(A)**. Photograph of the TSD **(B)**, the TSD with 4 mm titration accessory **(C)** and with 7 mm titration accessory **(D)**.

In our ongoing clinical trial, we will use a newly designed TSD, which has thinner material, to improve patient comfort and the ability to provide some level of titration with the use of 4- and 7-mm titration accessories that act to further protrude the tongue. The main objective of the study is to determine the efficacy of titrated TSD treatment for sleep-disordered breathing (as measured by the AHI), daytime sleepiness (as measure by the Epworth Sleepiness Scale [ESS]), and QOL (as measured by the Chalder Fatigue Scale, The Functional Outcomes of Sleep Questionnaire-10 [FOSQ-10], and Medical Outcomes Study 36-Item Short Form [SF-36]). A second objective is to identify the subjective compliance and side effects of TSD treatment. A third objective is to determine the efficacy of titration of TSDs when compared between initial TSD, 4- and 7-mm titrated TSD positions.

METHODS

Ethical Aspects

The study protocol and participant information documents have been approved by the Clinical Research Ethics Board of the University of British Columbia (H14-01333). The trial is registered in the United States Clinical Trials Registry (NCT02329925). Informed consent will be obtained from each eligible participant before proceeding with the trial.

Inclusion and Exclusion Criteria

A total of 60 patients with OSA will be recruited for the study. To be considered for inclusion in the study, the patients must be older than 18 years, have received a diagnosis of OSA (AHI score or oxygen desaturation index of 5 events/h to 50 events/h), have a body mass index of less than 35 kg/m^2. For the purpose of this study, we have accepted all diagnostic tools used in the community, portable monitor levels III and IV, and full PSG. The participants are excluded if they have had previous soft palate surgery, exhibit a neuromuscular disease, and/or are taking medications that disturb sleep.

Tongue Stabilizing Device

The tongue stabilizing device used in this trial is a preformed silicon appliance that uses suction to hold the tongue in a protruded position and improve the size of the upper airway during sleep (Aveo-TSD, Innovative Health Technologies, New Zealand). Patients are instructed to place the flanges of the TSD on the outside of the upper and lower lips, insert the tongue into the bulb as far as is comfortable, then squeeze and release the bulb to generate suction. Patients are advised to increase the suction by protruding the tongue further and squeezing the bulb more should the device loosen or be insufficiently retentive, or conversely decrease the suction should there be excessive discomfort. A titration accessory for the device will also be used, which attaches to the TSD and results in a greater amount of protrusion of 4 and 7 mm (**Figure 1**).

Titration Protocol

Figure 2 provides a flow chart for the baseline assessment, interventions, and follow-up assessment. Before TSD treatment begins, each participant will complete a series of questionnaires and we will perform a limited sleep study with a level III monitor to evaluate the baseline level of OSA. The TSD appliances will then be provided to each patient along with standardized instructions on use and care. After an acclimatization period of at least 2 months, the subjects will undergo a second limited sleep study to determine treatment effectiveness.

A titration protocol will be initiated only for those patients inadequately treated by the initial TSD. The TSD will first be titrated to hold the patient's tongue forward an additional 4 mm, followed by a 1-month acclimatization period and a follow-up limited sleep study. In a similar fashion, an additional 3 mm of advancement with the 7 mm titration accessory will be added as required, followed again by a limited sleep study. All subjects will undergo a split-night laboratory-based PSG and complete the follow-up questionnaires after a satisfactory response to treatment (a reduction in AHI < 10 events/h and > 50% reduction in AHI) or the maximum amount of comfortable titration has been achieved. Finally, we will compare the results of split-night PSG with TSD and without

TSD. We will also analyze and compare the level III portable monitor measurements before and afterward.

Limited Sleep Study

The level III portable monitoring device (MediByte; Braebon Medical Corporation, Ontario, Canada) will be utilized for the titration of the TSD treatment. The portable device consists of two inductance bands for thoracic and abdomen measurement, a nasal cannula pressure transducer airflow signal, finger pulse oximetry, an acoustic microphone for recording snoring sound, and a body position sensor. The description and validation of this portable monitor in 128 valid comparisons of the in-laboratory PSG and portable monitoring device has been previously published.[16] With a preset diagnostic AHI cutoff of < 10, the portable monitoring device derived respiratory disturbance index had a sensitivity and specificity of 79% and 86%, respectively.

Participants will be instructed on how to use a portable monitoring device by a dentist and will be given a portable monitoring device to take home and wear for 1 night, with a preaddressed mailer to return the device to the sleep laboratory. Data from the device are autoscored and then manually reviewed by a trained sleep technologist. Apneas are scored when there is a 95% or more reduction in airflow for at least 10 seconds. Hypopneas are scored based on airflow reduction measured by nasal pressure of 30% to 95% from baseline with an accompanying 3% oxygen desaturation.

Polysomnography

At the end of the trial, patients will have an in-laboratory PSG according to standard criteria.[17,18] PSG recordings will be conducted in a split-night study, half of the night recording the patient not wearing the TSD and the other half of the night with the TSD in place. Each split-night recording will be continued until the detection of at least one rapid eye movement (REM) sleep period. The order of with/without TSD will be randomized.

Standard measurements will include electroencephalography, electrooculography, submental electromyography, electrocardiography, chest and abdominal respiratory impedance plethysmography, arterial oxygen saturation (pulse oximeter), and nasal airflow (nasal cannulae connected to a pressure transducer).

Respiratory events will be scored according to the criteria published by the American Academy of Sleep Medicine.[18] Apnea will be defined as cessation of both nasal and oral airflow with its duration more than 10 seconds. Hypopnea will be defined as a reduction in nasal airflow greater than 30%, with a duration of more than 10 seconds associated with either a decrease in oxygen saturation by at least 3% or an electroencephalography arousal. The AHI will be the mean number of apneas and hypopneas per hour of sleep.

Questionnaires

Specific questionnaires will be used at baseline and after TSD acclimatization and titration according to the protocol. Daytime sleepiness will be assessed by the ESS[19] which is an eight-item, four-point scale (0 to 3). Participants will be asked

Figure 2—Protocol flow chart.
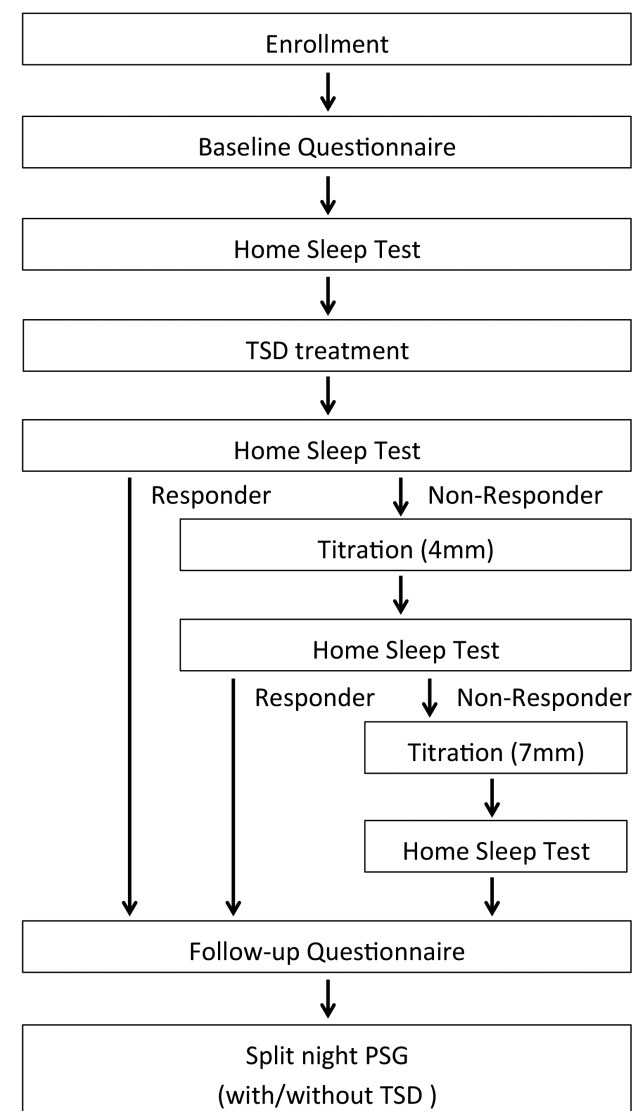
TSD = tongue stabilization device, PSG = polysomnography.

to rate their likelihood of dozing in eight different sedentary situations. The ESS has demonstrated high validity and reliability.[20]

We will use the Chalder Fatigue Scale to measure fatigue. The questionnaire includes questions about symptoms of mental and physical fatigue. The 11 items assess fatigue and are scored on a Likert scale (0, 1, 2, and 3).[21]

FOSQ-10 will be used to assess the effect of excessive sleepiness on daily activities. These 10 items are distributed among 5 subscales as follows: general productivity (2 items), activity level (3 items), vigilance (3 items), social outcomes (1 item), and sexual relationship (1 item).[22] Items are rated on a scale of 1 to 4 (1 = extreme difficulty, 2 = moderate difficulty, 3 = a little difficulty, 4 = no difficulty). The total score ranges from 5 to 20 and higher scores indicate better functional status.

The QOL measurement will be evaluated by SF-36. It is a standard questionnaire assessing QOL, both in the general

healthy population as well as groups of sick patients.[23] It consists of 36 questions grouped into 8 domains measuring different aspects of QOL (Physical Functioning, Role Physical, Bodily Pain, General Health, Vitality, Social Functioning, Role Emotional, and Mental Health). The results are converted into a scale from 0 to 100, where 0 indicates the lowest and 100 indicates the highest QOL. The SF-36 also allows summarization of the results into two summary measures: a physical component summary and a mental component summary.

Patients will be asked to keep a sleep diary with the hours of sleep and hours of TSD usage per night for 30 days. Subjective compliance will be evaluated by measuring hours per night and the number of days per week from a sleep diary. Side effects will be described by participants in terms of subjective side effects, device related side effects and sleep related side effects.

Statistical Analysis

All data will be analyzed by SPSS 15.0 statistical software (SPSS Inc., Chicago, IL, United States). The normality of the data distribution will be assessed using the Kolmogorov-Smirnov test. Descriptive statistics for clinical characteristics will presented as a mean ± standard deviation. Continuous variables will be evaluated with a paired t test or Mann-Whitney U test to compare between baseline and follow-up, as appropriate. The categorical variables will be compared using Pearson chi-square or Fisher exact test depending on the number of events. The Kruskal-Wallis analysis of variance will assess the differences in the variables of level III monitor recordings between the degrees of titration of TSD (initial, 4 mm, 7 mm). When the analysis of variance shows a value of $P < 0.05$, comparisons between the degrees of titration of TSD (initial, 4 mm, 7 mm) will be performed using a Mann-Whitney U test with a Bonferroni correction. A value of $P < .05$ will be used to indicate statistical significance.

DISCUSSION

This trial will advance the understanding of the effectiveness of titrated TSD treatment for OSA. Because of the high costs associated with the main forms of OSA treatment such as CPAP and MAD, from a public health perspective there is a strong desire to investigate more economic forms of therapy including the TSD.

This trial has a limitation about a split-night PSG. The method of a split-night PSGs has the potential to include low sleep efficiency and short duration or lack of REM sleep. Therefore, each split night recording of half will be continued until the detection of at least one REM sleep period to close to the same sleep stages pattern among split-night PSG ideally. To add to the efficacy assessment, we will also assess before and after TSD with a level 3 portable monitor.

Furthermore, patient populations unsuitable for MAD treatment such as edentulous patients or those with advanced periodontal disease who lack the dental support to tolerate the mandibular protrusion are also likely to benefit from TSD therapy. The evidence provided by this trial will help patients with OSA—especially those who cannot tolerate or afford

CPAP and/or MAD—and reveal the potential of the TSD as a treatment option for OSA.

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(17):1230–1235.

2. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep*. 1997;20(9):705–706.

3. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: a population health perspective. *Am J Respir Crit Care Med*. 2002;165(9):1217–1239.

4. Young T, Finn L, Peppard P, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep*. 2008;31(8):1071–1078.

5. Gottlieb DJ, Yenokyan G, Newman AB, et al. Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: the Sleep Heart Health Study. *Circulation*. 2010;122(4):352–360.

6. Redline S, Yenokyan G, Gottlieb DJ, et al. Obstructive sleep apnea hypopnea and incident stroke: the Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2010;182(2):269–277.

7. Hillman DR, Murphy AS, Pezzullo L. The economic cost of sleep disorders. *Sleep*. 2006;29(3):299–305.

8. Jennum P, Kjellberg J. Health, social and economical consequences of sleep-disordered breathing: a controlled national study. *Thorax*. 2011;66(7):560–566.

9. McDaid C, Durée KH, Griffin SC, et al. A systematic review of continuous positive airway pressure for obstructive sleep apnoea-hypopnoea syndrome. *Sleep Med Rev*. 2009;13(6):427–436.

10. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet*. 2005;365(9464):1046–1053.

11. Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. *Proc Am Thorac Soc*. 2008;5(2):173–178.

12. Lim J, Lasserson TJ, Fleetham J, Wright J. Oral appliances for obstructive sleep apnoea. *Cochrane Database Syst Rev*. 2004;(4):CD004435.

13. Phillips CL, Grunstein RR, Darendeliler MA, et al. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: a randomized controlled trial. *Am J Respir Crit Care Med*. 2013;187(8):879–887.

14. Bratton DJ, Gaisl T, Wons AM, Kohler M. CPAP vs mandibular advancement devices and blood pressure in patients with obstructive sleep apnea: a systematic review and meta-analysis. *JAMA*. 2015;314(21):2280–2293.

15. Deane SA, Cistulli PA, Ng AT, Zeng B, Petocz P, Darendeliler MA. Comparison of mandibular advancement splint and tongue stabilizing device in obstructive sleep apnea: a randomized controlled trial. *Sleep*. 2009;32(5):648–653.

16. Pereira EJ, Driver HS, Stewart SC, Fitzpatrick MF. Comparing a combination of validated questionnaires and level III portable monitor with polysomnography to diagnose and exclude sleep apnea. *J Clin Sleep Med*. 2013;9(12):1259–1266.

17. Rechtschaffen A, Kales A. *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Brain Information Service/Brain Research Institute, University of California; 1968.

18. Iber C, Ancoli-Israel S, Chesson AL, Quan SF; for the American Academy of Sleep Medicine. *The AASM Manual for the Scoring of Sleep Associated Events: Rules, Terminology and Technical Specifications*. 1st ed. Westchester, IL: American Academy of Sleep Medicine; 2007.

19. Johns MW. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*. 1991;14(6):540–545.

20. Johns MW. Reliability and factor analysis of the Epworth Sleepiness Scale. *Sleep*. 1992;15(4):376–381.

21. Chalder T, Berelowitz G, Pawlikowska T, et al. Development of a fatigue scale. *J Psychosom Res*. 1993;37(2):147–153.

22. Chasens ER, Ratcliffe SJ, Weaver TE. Development of the FOSQ-10: a short version of the functional outcomes of sleep questionnaire. *Sleep.* 2009;32(7):915–919.

23. Ware JE, Kosinski M, Keller SD. *SF-36 Physical and Mental Health Summary Scales: A User's Manual.* Boston, MA: The Health Institute, New England Medical Center; 1994.

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SUBMISSION & CORRESPONDENCE INFORMATION

Address correspondence to: Kentaro Okuno, DDS, PhD, Department of Oral Health Sciences, Faculty of Dentistry, The University of British Columbia, 2199 Wesbrook Mall, Vancouver, BC, Canada V6T 1Z3; Email: okuno-kentaro-ig@alumni.osaka-u.ac.jp

DISCLOSURE STATEMENT

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

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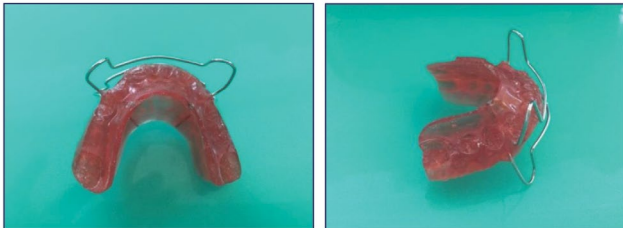
INTRODUCTION

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/m[2]; 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 airways[8–10] (**Figure 3** to **Figure 6**).

Figure 3—Anatomical points of reference used for the cephalometric analysis.
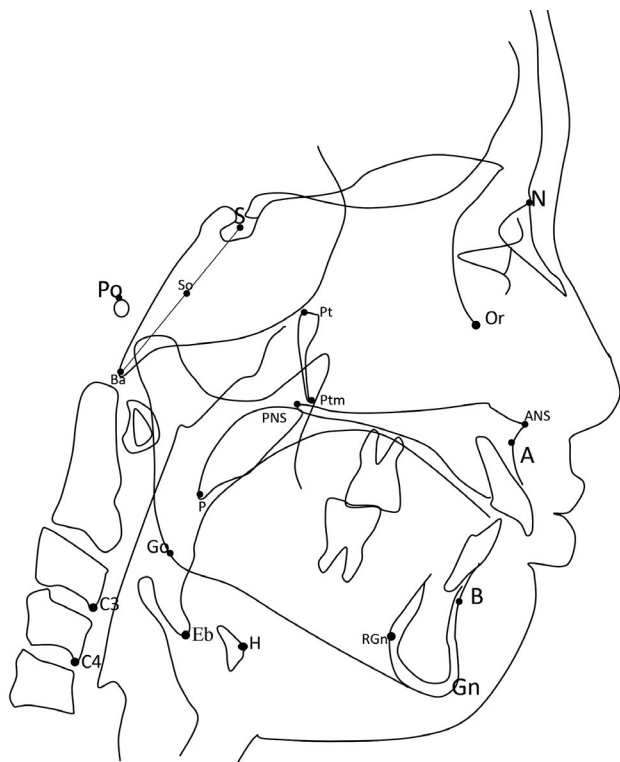
A = most posterior point on the anterior contour of the upper alveolar process, ANS = apex of the anterior nasal spine, B = most posterior point on the anterior contour of the lower alveolar process, Ba = the most posteroinferior point on the clivus of occipital, C3 = third cervical vertebra, C4 = fourth 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.
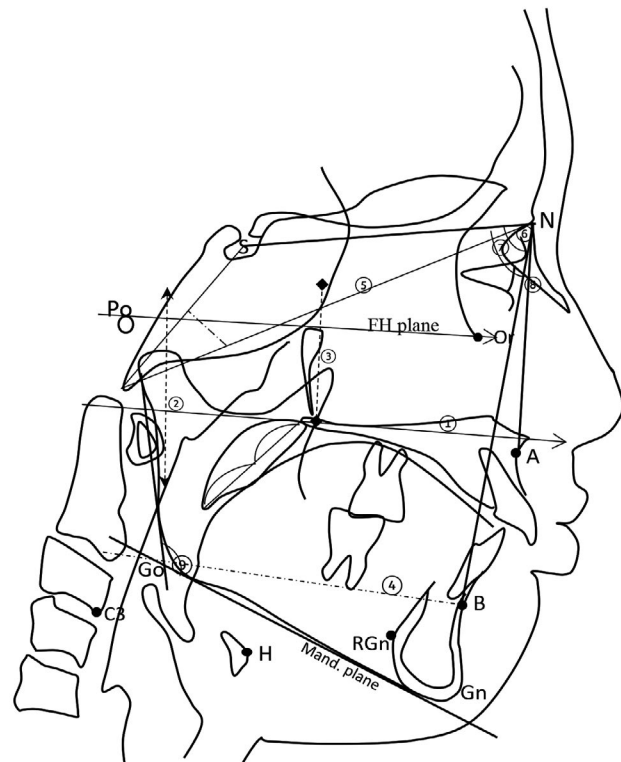
Figure 4—Reference lines and angles.

(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.
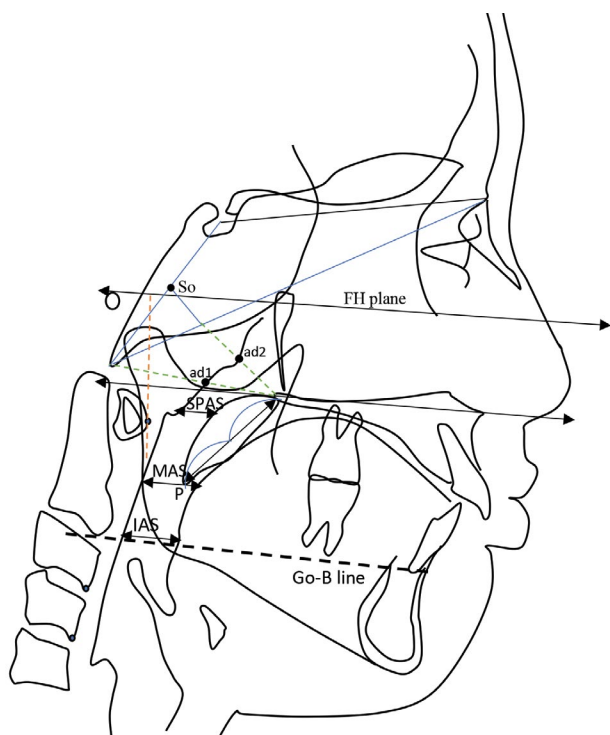
ad1 = the intersection of the line Pt-Ba and the posterior nasopharyngeal wall, ad2 = the intersection of the posterior nasopharyngeal wall and the line Pt-So, IAS = the thickness of the airway along a line extended through Go-B, MAS = the thickness of the airway along a line parallel to Go-B through 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.
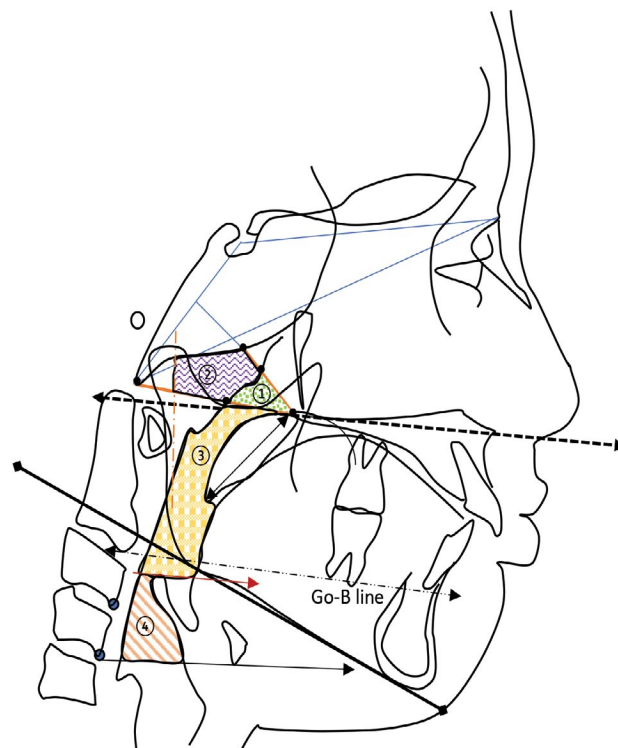
(1) NP area (nasopharyngeal area) = corresponds to the nasopharyngeal area in vertical and anteroposterior dimensions from ad1-Ba-ad2-So, where the posterior wall is delimited by the anterior atlas line, measured in mm^2. **(2)** Adenoid area = corresponds to the adenoid area in vertical and anteroposterior dimensions from ad1-So-Ba-ad2, measured in mm^2. **(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 mm^2. **(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 mm^2.

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.
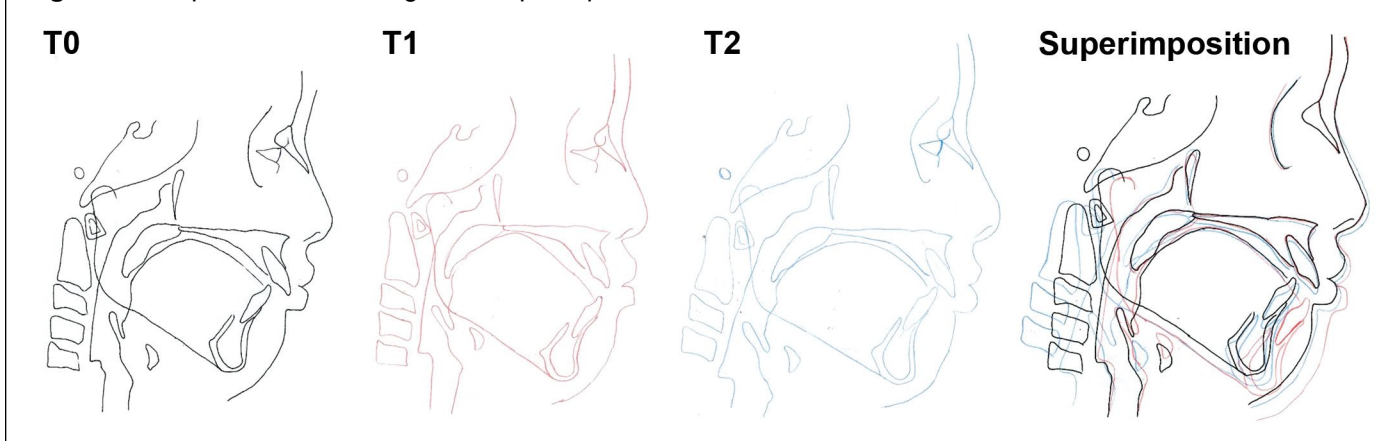
Despite the mentioned setbacks, this case presented interesting outcomes throughout its evaluation time, with an observed betterment of sleep-breathing that was also somewhat maintained over time. This may be explained as the patient grows the changes brought upon by the activator are maintained, not 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. Horihata 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. *Comtemp Clin Dent.* 2010;1(2):66–69.

SUBMISSION & CORRESPONDENCE INFORMATION

DISCLOSURE STATEMENT

Using a Lingual Frenulum Depressor to Create an Airway in a Patient With Obstructive Sleep Apnea: A New Method

Hiroshi Suzuki, DDS, PhD[1]; Taiga Fukuda, DDS[2]; Satoru Tsuiki, DDS, PhD[3]; Yoshihiro Iwata, DDS, PhD[1];
Mayuko Yoshimura, DDS[1]; Tatsuo Sakamaki, MD, PhD[3,4]; Takashi Kaneda, DDS, PhD[2]; Misao Kawara, DDS, PhD[1]

[1]Department of Oral Function and Rehabilitation, Nihon University School of Dentistry at Matsudo, Chiba, Japan; [2]Department of Radiology, Nihon University School of Dentistry at Matsudo, Chiba, Japan; [3]Department of Internal Medicine, Nihon University School of Dentistry at Matsudo, Chiba, Japan; [4]Japan Somnology Center, Institute of Neuropsychiatry, Tokyo, Japan

This report presents the case of a patient with obstructive sleep apnea (OSA) whose symptoms improved with a lingual frenulum depressor (LFD) attached to an oral appliance (OA) to noninvasively secure the breathing route. A diagnosis of mild OSA was made using polysomnography in a 25-year-old man (body mass index = 31.1 kg/m²). A monobloc-type OA with an LFD posterior to the lower front teeth was fabricated to depress the lingual frenulum posteroinferiorly. With this appliance, apnea-hypopnea index decreased (12.9 events/h to 5.8 events/h), the 3% desaturation index decreased (9.6 to 5.6), the lowest percutaneous saturation index increased (83% to 89%), and arousal index improved (15.3 events/h to 7.58 events/h). Coronal magnetic resonance images showed no gap between the tongue and soft palate without the OA with LFD, but a gap was present with the OA inserted. We conclude that the LFD created a "crevasse" that acted as a breathing route through the oral cavity by changing the tongue shape. This is a new approach to OSA treatment.

KEYWORDS: airway, lingual frenulum depressor, obstructive sleep apnea, oral appliance, velopharynx

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INTRODUCTION

The velopharynx is the most common site of occlusion in patients with obstructive sleep apnea (OSA).[1,2] In an experiment in which obstructive apnea was experimentally induced, Isono et al. found that the tongue pushes the soft palate dorsally with inspiratory effort, which results in a narrowed velopharynx.[3] This indicates that there is no airway present between the soft palate and the tongue during apneic events. Therefore, although both nasal continuous positive airway pressure and mandibular advancement devices are known to effectively enlarge the constricted retropalatal airway,[4,5] "an additional airway through the oral cavity" might help maintain airway patency independent of the occluded velopharynx. Based on this hypothesis, the case of a patient with OSA in whom a newly developed lingual frenulum depressor (LFD) successfully created an oral bypass airway that improved the OSA is presented.

REPORT OF CASE

A 25-year-old man complaining of heavy snoring and daytime sleepiness visited our hospital. His height was 170 cm and he weighed 90 kg (body mass index = 31.1 kg/m²), and he had normal occlusion without subjective or objective abnormalities in the stomatognathic system (**Figure 1A**). There were no past or current temporomandibular joint (TMJ) disorders or motor function abnormalities of the trunk or limbs. In addition, the patient had allergic rhinitis. Based on the results of polysomnography (PSG), a diagnosis of mild OSA was made, and the patient's apnea-hypopnea index (AHI) was 12.9 events/h. A

mandibular advancement device was then fabricated for routine treatment of OSA in our hospital. However, the patient discontinued its use after several days due to TMJ soreness. Subsequently, we attempted treatment using new methodology. First, in accordance with our hospital's procedures, a monobloc-type mandibular advancement device was fabricated. Next, posterior to the lower front teeth and parallel to the occlusal plane, an L-shaped (width, 2 cm; right angle, 3 cm) lingual frenulum depressor (LFD) was added to the lower oral appliance (OA) to depress the lingual frenulum posteroinferiorly (**Figure 1B** and **Figure 1C**). Finally, the upper and lower OA was fixed (**Figure 1D**); an edge-to-edge relationship of the upper and lower incisors was achieved that yielded a 2-mm advanced mandibular position. The patient was instructed to minimize tongue movement, to not bite the appliance firmly, and to cease appliance use if any discomfort that prevented sleep occurred.

After 2 weeks, PSG was conducted in the same way. Insertion of the OA with the LFD decreased the AHI from 12.9 events/h to 5.8 events/h. Moreover, this OA revealed that, in comparison to baseline results, the 3% desaturation index decreased (9.6 to 5.6), the lowest percutaneous saturation index increased (83% to 89%), and the arousal index improved (15.3 events/h to 7.58 events/h). To clarify the effect of the OA with the LFD in the pharynx, magnetic resonance imaging was performed. Experiments were conducted under two conditions: with and without the patient wearing the OA with the LFD. On sagittal images, no gap was present in the oropharynx whether the patient was wearing the OA or not. On coronal images, no gap was seen in the velopharynx without the OA, but a gap was present when it was worn (**Figure 2A** and **Figure 2B**).

Figure 1
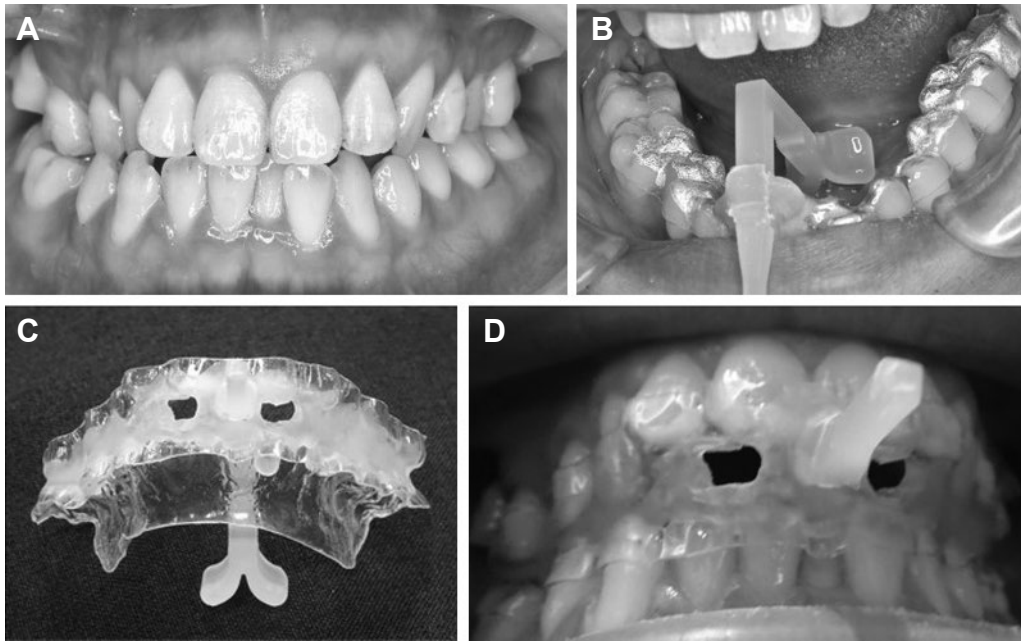
(A) The patient with normal occlusion, without subjective or objective abnormalities in the stomatognathic system. **(B)** The lingual frenulum depressor. **(C)** Depression of the lingual frenulum posteroinferiorly by the lingual frenulum depressor. **(D)** The oral appliance was fixed after an edge-to-edge relationship of the upper and lower incisors was achieved.

Figure 2—Coronal magnetic resonance images of the patient.
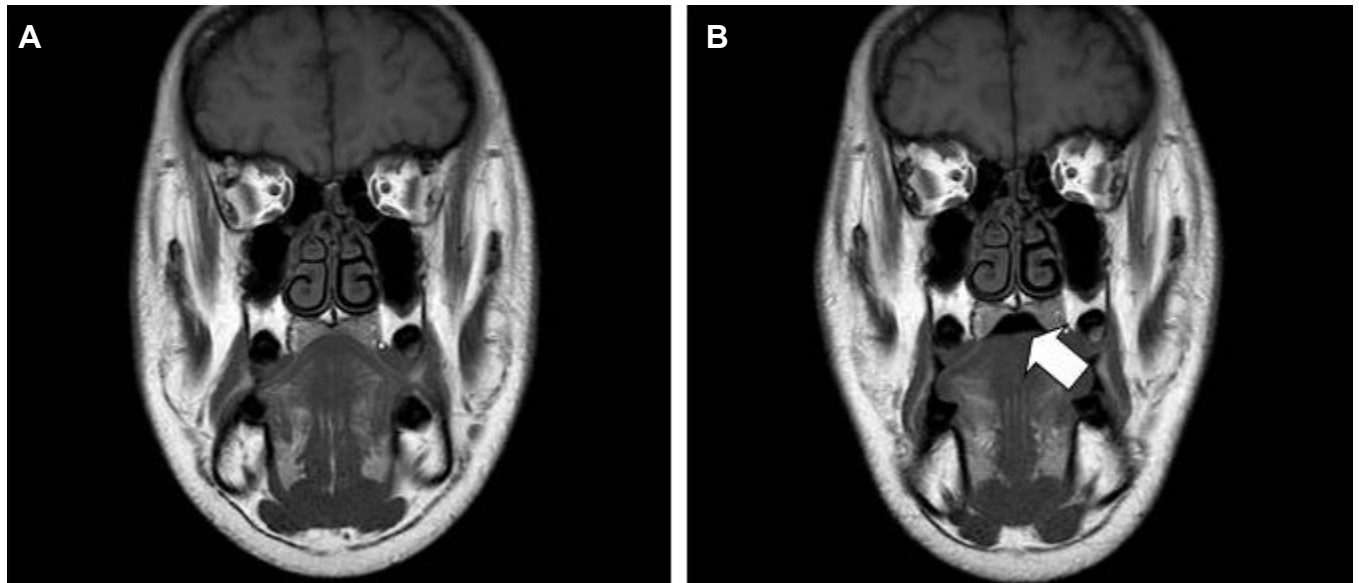
Images show the patient **(A)** not wearing and **(B)** wearing the oral appliance with the lingual frenulum depressor.

This work was conducted in accordance with the Declaration of Helsinki. The subject received explanation regarding the content of the study, and he provided informed consent for all examinations, testing, and treatment. The patient was advised that following the study, if he desired, a new, standard monobloc-type OA would be fabricated and fitted as treatment. This study was approved by the Ethics Committee of Nihon University School of Dentistry at Matsudo (EC 12-012).

DISCUSSION

To the best of our knowledge, there is no published literature reporting attempts to relieve OSA by altering tongue shape to secure a breathing route. This simple, noninvasive, and inexpensive method has potential clinical applications, either alone or in conjunction with an OA.

This LFD has two important advantages. First, the LFD is not dependent on the status of the velopharynx (ie, patent

or occluded), because the experimentally induced "crevasse" (**Figure 2B**) between the tongue and the soft palate directly connects the oropharynx with the outside of the mouth. The genioglossus muscle and the inferior longitudinal muscle of the tongue are depressed posteroinferiorly when the lingual frenulum is depressed posteroinferiorly by the LFD. Consequently, the lingual septum caves in, and the base of the tongue becomes rounded. This is thought to create a crevasse in the pharyngeal region of the upper portion of the base of the tongue.

Second, similar to treatment with a tongue position controller, patients with nasal congestion who are likely to cease continuous positive airway pressure and OA use would be good candidates for this simple treatment for the aforementioned reasons.[6,7] Patients with temporomandibular disorders, those exhibiting partial or complete edentulism, and those with severe nasal congestion would likely be good candidates for this treatment. In fact, the current patient's nasal airway was completely obstructed due to rhinostenosis from allergic rhinitis. Absence of a breathing route could lead to treatment failure and reduced adherence to the appliance. Therefore, it was necessary to secure a "crevasse" as a breathing route using the OA with the LFD to achieve a favorable response.

Additionally, there was no soft-tissue discomfort, specifically of the lingual frenulum or floor of the mouth, no mouth dryness or in the TMJ or masticatory muscles, following removal of the new OA after PSG. This is likely because of the appliance being worn with almost no anterior mandibular displacement. Moreover, no damage to the appliance was seen after use. However, long-term use of this device may result in stomatitis of the lingual frenulum, discomfort in the floor of the mouth, and inhibition of tongue movement, in addition to the usual side effects of an OA.[8]

This case study had several limitations. First, imaging was conducted while the patient was awake, whereas actual usage of the device occurred while the patient was sleeping. Second, only one case was evaluated. However, the results demonstrated that occlusion of the airway can be prevented. In the future, it will be necessary to recruit a larger patient sample to verify if wearing the OA with the LFD while sleeping improves OSA, and to clarify the advantages and disadvantages of its long-term use.

In conclusion, treatment of OSA using this newly developed OA with an LFD changed the shape of the tongue, creating a gap in the throat that allowed the airway to remain patent and improved sleep status.

REFERENCES

1. Kuna ST, Remmers JE. Anatomy and physiology of upper airway obstruction. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* 3rd ed. Philadelphia, PA: WB Saunders; 2000:840–858.

2. Enciso R, Shigeta Y, Nguyen Y, Clark GT. Comparison of cone-beam computed tomography incidental findings between patients with moderate/severe obstructive sleep apnea and mild obstructive sleep apnea/healthy patients. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2012;114(3):373–381.

3. Isono S, Tanaka A, Nishino T. Dynamic interaction between the tongue and soft palate during obstructive apnea in anesthetized patients with sleep-disordered breathing. *J Appl Physiol.* 2003;95(6):2257–2264.

4. Gordon P, Sanders MH. Sleep.7: positive airway pressure therapy for obstructive sleep apnoea/hypopnoea syndrome. *Thorax.* 2005;60(1):68–75.

5. Ferguson KA, Cartwright R, Rogers R, Schmidt-Nowara W. Oral appliances for snoring and obstructive sleep apnea: a review. *Sleep.* 2006;29(2):244–262.

6. Tsuiki S, Isono S, Minamino O, et al. Tongue position controller as an alternative treatment for obstructive sleep apnea. *Sleep Breath.* 2012;16(4):957–960.

7. Richard GN, Cistulli PA, Ungar RG, Berthon-Jones M, Sullivan CE. Mouth leak with nasal continuous positive airway pressure increases nasal airway resistance. *Am J Respir Crit Care Med.* 2000;154(1):182–186.

8. de Almeida FR, Lowe AA, Tsuiki S, et al. Long-term compliance and side effects of oral appliances used for the treatment of snoring and obstructive sleep apnea syndrome. *J Clin Sleep Med.* 2005;15(2):143–152.

DISCLOSURE STATEMENT

26th Annual Meeting of the American Academy of Dental Sleep Medicine: Boston, MA, June 2–4, 2017

POSTER #001

LONG-TERM SIDE EFFECTS OF SLEEP APNEA TREATMENT WITH ORAL APPLIANCES

Hamoda MM, Almeida FR, Pliska BT

Department of Oral Health Sciences, Faculty of Dentistry, University of British Columbia, Vancouver, BC, Canada

Introduction: Oral appliances for the treatment of obstructive sleep apnea (OSA) reduce upper airway collapse by advancing the mandible (OA_m) and decreasing the collapsibility of the upper airway. OA_m are well tolerated, still these appliances have known side effects, with the most serious being dental movement. Previous studies have shown that the amounts of dentoalveolar changes are not related to appliance design but to the duration of therapy. It is not yet clear whether there are also irreversible skeletal changes related to OA use.

As oral appliance treatment for OSA is a life-long therapy, careful and extended follow-up of patients is required to evaluate possible side effects with prolonged durations of oral appliance use. The objective of this study is to evaluate both the magnitude and progression of the dental and skeletal changes associated with long-term OA_m treatment.

Methods: Retrospective study of lateral cephalograms of adults treated for primary snoring or mild to severe OSA with an OA_m for a minimum of 8 years. All patients were treated with a custom made titratable oral appliance; the number of lateral cephalograms obtained for each patient ranged between 2 to 9 and the baseline cephalogram for each patient was obtained prior to the initiation of treatment. The cephalograms were analyzed for skeletal, dental and soft tissue facial changes. The progression of these changes over time was determined and initial patient and dental characteristics were evaluated as possible predictors of the observed dental side effects of treatment. Approval for the study was obtained from the UBC Clinical Research Ethics Board H11-01661.

Results: A total of 62 patients (average age at start of treatment: 49 ± 8.6 years) were included in this study and average treatment length was 12.6 years (range: 8-21 years). Over the total treatment interval evaluated there was a significant ($P < .001$) reduction in overjet (3.5 ± 2.5 mm) and overbite (2.2 ± 2.3 mm), as well as maxillary incisor retroclination and mandibular incisor proclination. All changes seemed to continuously progress except for overbite reduction which levelled off with time. Although some statistically significant ($P < .001$) skeletal changes were noted in the SNB and mandibular plane angles, measured differences were less than a 1° and were deemed not clinically significant.

Conclusions: This study represents the longest OA_m side effects follow up duration conducted to date. It confirms that there are significant and progressive dental changes with prolonged OA_m use and no clinically significant skeletal changes.

POSTER #002

IS SNORING DURING PEDIATRIC DENTAL PROCEDURES INDICATIVE OF OBSTRUCTIVE SLEEP APNEA?: A PILOT STUDY

Chiang HK[1], Best AM[1], Leszczyszyn DJ[2]

[1]School of Dentistry, Virginia Commonwealth University, Richmond, Virginia; [2]School of Medicine, Virginia Commonwealth University, Richmond, Virginia

Introduction: The American Academy of Pediatric Dentistry estimated over 300,000 children are sedated annually during pediatric dental care. Pharmacologic and physiologic effects of sedation and anesthesia simulate the effects of sleep and can uncover the propensity for snoring. Snoring duration and loudness are associated with obstructive sleep apnea (OSA) severity. Therefore, sedation for dental procedures presents a unique opportunity to use snoring to identify children at higher risk for OSA. The purpose of this pilot was to determine whether pediatric snoring could be used as a screening tool for OSA.

Methods: The case-control study was approved by the VCU-IRB (HM 15315). All participants were pediatric dental patients requiring moderate sedation. Snoring cases were identified by significant snoring (audible, turbulent sounds lasting longer than 2 breaths) during dental care. Non-snoring (control) participants were selected from those without significant snoring. OSA was diagnosed using a portable home sleep monitoring device. Parents filled out two questionnaires: Pediatric Sleep Questionnaire (PSQ) and the Pediatric Symptom Checklist (PSC). Groups were compared using a *t*-test.

Results: In the 19 children screened for eligibility, there were 10 snoring cases (59%) and 7 controls recruited and completed the home monitoring portion of the study. The 9 males and 8 females had an average age of 5.5 years (range = 4 to 8) and had an average BMI percentile of 59.3 (range = 2nd to 99th percentile). Fifty-seven percent of the children were black and 29% were white. The snorers and non-snorers did not differ on demographics. The primary outcome variable was the respiratory disturbance index (RDI) indicator of OSA. Although the children who snored during pediatric dental surgery had a nominally higher RDI than the control children who did not snore (mean = 5.3 vs. 4.3), this difference was not statistically significant ($P > .7$). Using a cut-off of RDI > 5 to indicate significant OSA, there were 5 of 8 in the snorer group and 2 of 4 in the non-snorer group who met this criteria. Similar results were obtained using the snore index (SI), and type 1&2 snoring percentages; specifically that the snorers had nominally higher

but statistically non-significant averages. On the other hand, one non-snoring participant had a maximum relative loudness of 27dB and this resulted in the group mean being nominally higher than the snoring group's average. The results from the questionnaires were more hopeful. Both the PSQ and PSC average was 5 points higher in the snoring group than the average of the non-snorers but the difference was not statistically significant (PSQ *P* = .056, PSC *P* > .4).

Conclusions: Snoring during pediatric dental procedures does not appear to be indicative of obstructive sleep apnea on its own. Future studies could explore whether this clinical finding would add to the screening capability of existing questionnaires such as the PSQ.

Support: This study was funded by Virginia Commonwealth University Clinical Research fund.

POSTER #003

MANAGEMENT OF COMPLEX SLEEP APNEA WITH MANDIBULAR ADVANCEMENT DEVICE: A CASE REPORT

Li S, Correa L, Mehta NR

Tufts University School of Dental Sleep Medicine, Boston, Massachusetts

Introduction: Complex sleep apnea may be defined as the presence of both obstructive and repetitive central sleep apnea occurring in the same individual during the same night (R.J. Castriotta, R. Majid, 2013). During REM sleep, the behavioral system is dominant, with impaired ventilator response to hypercapnia, especially during phasic REM sleep (E.A. Phillipson, 1977). During non-REM sleep, in contrast, the metabolic control of breathing is dominant, with minimal to absent behavioral control (B.R. Fink, 1961). For this reason, in adults, significant central apnea is more frequently and sometimes exclusively seen during non-REM sleep, whereas obstructive apnea is more frequently seen in REM sleep (E. Lugaresi et al., 1972). Some authors state that the number of central apnea events may increase with initiation of positive airway pressure (PAP) therapy due to increased alveolar ventilation with a decrease of CO_2 lower than the apneic threshold (D. Neu et al., 2015). However, there is a lack of data on the influence of oral appliance therapy (OAT) on central component of complex sleep apnea. The purpose of this case report is to show result of management of complex sleep apnea with OAT.

Methods: A 59-year-old male with long-term symptoms of sleep disturbance was referred by his sleep physician to explore the option mandibular advancement device (MAD) due to intolerance to PAP therapy. The patient underwent home sleep study with type 3 home sleep test device. He was diagnosed with Complex Sleep Apnea (AHI = 31.4 events/h) with large amount of central apnea (CA) events (CA index = 20.2 events/h). The overall respiratory events index (REI) was 38.2 events/h with the majority of the events happening in supine position. Baseline oxygen saturation was 97.4% with lowest O_2 saturation of 84.2%. A custom, adjustable MAD was fabricated and fitted with 80% jaw protrusion; titration of the MAD

was reached at 90% during follow up appointment. Morning jaw repositioning device (MRD) was provided to minimize potential development of occlusal side effects. After achieving subjective success, the patient was referred back to the sleep physician to objectively assess the efficacy of the MAD.

Results: Follow-up polysomnography demonstrated reduction of obstructive and central sleep apnea with the use of the MAD with residual apnea in the supine position (AHI = 8.8, CA index = 2), mean O_2 saturation = 94%, with lowest O_2 saturation = 90%. Subjective improvement included feeling more refreshed at the morning and improved quality of sleep. Epworth Sleepiness Scale score was reduced from 13/24 to 4/24. Patient was satisfied with the MAD and reported minor bite symptoms at the morning after removal of the MAD, which were eliminated by using the MRD for 15-20 minutes.

Conclusions: This case report demonstrated improvement of obstructive and central apnea components with the MAD. A possible mechanism of reduction of central apnea events could be associated with stabilization of the ventilator control system and improving the sleep architecture.

POSTER #004

CESSATION OF SNORING WITH AN INCREASED APNEA HYPOPNEA INDEX DURING MANDIBULAR ADVANCEMENT DEVICE THERAPY: A CASE REPORT

Ebato A[1], Tsuiki S[2-4], Almeida F[4], Kohzuka Y[4], Suzuki H[1], Inoue Y[2]

[1]Department of Oral Function and Rehabilitation, Nihon University School of Dentistry at Matsudo, Japan; [2]Japan Somnology Center, Institute of Neuropsychiatry, Japan; [3]Aging and Geriatric Dentistry, Faculty of Dentistry, Tohoku University, Japan; [4]Department of Oral Health Sciences, Faculty of Dentistry, The University of British Columbia, Canada

Introduction: Snoring is a typical chief complaint in patients with mild obstructive sleep apnea (OSA) who initiate oral appliance therapy. The cessation of snoring after a prescription of a mandibular advancement device (MAD) can often be assumed to reflect a patent upper airway following mandibular advancement, which would simultaneously support the effectiveness of MAD. However, we recently experienced a case of mild OSA in which snoring disappeared with the use of an MAD, but the apnea-hypopnea index (AHI) increased. This case highlights the importance of objective follow-up evaluation of MAD treatment.

Methods: A 41-year-old male (body mass index [BMI] = 22.2 kg/m^2) was diagnosed with mild OSA with an AHI of 8.5 events/h and was referred to the Sleep Apnea Dental Clinic, Yoyogi Sleep Disorder Center, Tokyo, for treatment with MAD. The patient complained of mild insomnia and light sleep and his wife reported loud snoring with episodes of apnea. The score on the Japanese version of the Epworth Sleepiness Scale at his first visit was 7. The patient did not complain of dozing-off during work. A custom-made monobloc MAD was fabricated at 50% of the maximum mandibular protrusion. After 3 months of MAD treatment with additional advancement

of the mandible as necessary, he used his MAD 6-7 nights a week and his wife no longer reported his snoring, although he did not feel any subjective improvement of his sleep. He went through follow-up polysomnography with the MAD.

Results: Follow-up polysomnography with the MAD in place revealed that his snoring had indeed improved from 14.8% to 0.4% of the total sleep time. However, the AHI had increased from 8.5 events/h to 11.6 events/h and the nadir $SlpO_2$ fell from 93% to 87%. Notably, the AHI during rapid eye movement sleep increased from 8.8 events/h to 33.4 events/h.

Conclusions: While an MAD has been reported to predominantly enlarge the velopharynx, which is where snoring originates, it is interesting to find a patient in whom OSA worsened despite the presence of a typical sign of improved upper airway patency (ie, cessation of snoring). Although the reason for the increased AHI in this case is unknown, we speculate that a slight increase in BMI may have resulted in an increase in the collapsibility of the oropharynx and/or hypopharynx. To avoid adverse health outcomes such as hypoxemia even after treatment, we emphasize the need for the objective follow-up evaluation of MAD treatment using polysomnography.

POSTER #005

THE ROLE OF A NOVEL ORAL APPLIANCE THERAPY DEVICE ON PHARYNGEAL PRESSURE SWINGS AND CPAP REQUIREMENTS DURING SLEEP IN OBSTRUCTIVE SLEEP APNEA: A PILOT STUDY

Amatoury J[1,2], Tong B[1], Nguyen C[1], Szollosi I[3], Eckert DJ[1,2]

[1]Neuroscience Research Australia (NeuRA), Sydney, Australia; [2]School of Medical Sciences, University of New South Wales, Sydney, Australia; [3]Oventus Medical Ltd, Brisbane, Australia

Introduction: Oral appliance (OA) therapy is often an effective alternative to continuous positive airway pressure (CPAP) for the treatment of obstructive sleep apnea (OSA). However, OA treatment efficacy varies and is often poorly tolerated in people with high nasal resistance. A new OA therapy device that incorporates an opening to the oral cavity (Oventus, O_2Vent T) allows breathing through the device airway, which may reduce the high inspiratory pressures required to drive air through the pharynx in people with nasal obstruction. The goals of the current study were to: (1) assess pharyngeal pressure swings during sleep with and without the OA therapy device and (2) determine the effect of the device on the CPAP requirements to minimize pharyngeal pressure swings and abolish residual events.

Methods: Four individuals were studied overnight in the sleep physiology laboratory (3 male; age: 43-62 years, BMI: 28-33 kg/m[2]). In addition to standard polysomnography, subjects were fitted with a nasal CPAP mask and pneumotachograph to quantify airflow. Choanal pressure (Pcho) and epiglottic pressure (Pepi) were measured using pressure transducer-tipped catheters. Nasal resistance was quantified during quiet nasal breathing awake (Pcho/flow@200ml/s). Nasal CPAP was carefully titrated during NREM sleep to determine "therapeutic

CPAP" level. Participants were studied under the following conditions during supine NREM sleep: (1) no OA and no CPAP (baseline), (2) OA only, (3) CPAP only, and (4) OA and CPAP combination therapy. The degree of mandibular advancement with the OA therapy device was sub-optimal to allow expression of residual events that required combination OA and CPAP therapy. The apnea-hypopnea index (AHI) and nadir Pepi swings were determined during each condition. CPAP levels with OA therapy to achieve nadir Pepi values equivalent with therapeutic CPAP without the OA was also determined.

Results: Awake nasal resistance ranged between 2.8 and 20.6 $cmH_2O/mL/s$ (12.3 ± 8.2 $cmH_2O/mL/s$, mean ± SD). 3/4 participants had high nasal resistance (> 3 $cmH_2O/mL/s$). Baseline AHI was 37 ± 35 events/h, which decreased with OA therapy to 8 ± 10 events/h. Average nadir Pepi swings during baseline NREM sleep were -8.5 ± 2.5 cmH_2O, falling to -4.1 ± 1.6 cmH_2O with OA therapy. The therapeutic CPAP level required to abolish respiratory events during NREM sleep without OA therapy was 6.8 ± 2.4 cmH_2O with corresponding nadir Pepi swings of -4.0 ± 2.6 cmH_2O. With OA and CPAP combination therapy, a CPAP level of 2.3 ± 0.9 cmH_2O abolished respiratory events and resulted in further reduction of the nadir Pepi swings to -2.7 ± 1.1 cmH_2O.

Conclusions: The oral appliance device with built in airway reduces pharyngeal pressure swings and the CPAP requirements necessary to achieve stable breathing during sleep. These options may be viable alternatives for the treatment of OSA in people with high nasal resistance.

POSTER #006

WHEN TO PERFORM FOLLOW-UP POLYSOMNOGRAPHIC EVALUATION OF ORAL APPLIANCE THERAPY FOR OBSTRUCTIVE SLEEP APNEA

Eno Y[1,2], Tsuiki S[3,4], Fukuda T[3], Taga H[1,5], Inoue Y[3]

[1]JR Tokyo General Hospital, Japan; [2]School of Dentistry, Meikai University, Japan; [3]Japan Somnology Center, Institute of Neuropsychiatry, Japan; [4]Aging and Geriatric Dentistry, Faculty of Dentistry, Tohoku University, Japan; [5]Department of Anesthesiology, Fujigaoka Hospital, Showa University, Japan

Introduction: There is no concise tool for deciding when to perform follow-up polysomnography in oral appliance therapy for obstructive sleep apnea (OSA). However, the timing of this evaluation is clinically important because frequent polysomnographic studies are impractical with regard to both the patient's inconvenience and cost. The STOP questionnaire consists of 4 yes/no questions (ie, snoring, tiredness during daytime, observed apnea, high blood pressure), and is used to identify subjects who may be at risk of OSA if they answer yes to 2 or more questions (Chung et al. Anesthesiology 2008). We hypothesized that a reduction in the STOP score (ie, fewer yes responses), in patients undergoing oral appliance treatment could be an indicator of the best timing for follow-up polysomnography.

Methods: The study protocol was approved by the ethics committee of the Institute of Neuropsychiatry. Among patients who were diagnosed with OSA at a single sleep center from June 2011 to December 2015, OSA patients who were to be prescribed a monobloc were consecutively targeted and prospectively recruited into this study (ie, STOP group). Subjects who lived alone were excluded. The STOP score was recorded at every patient visit, while the upper and lower parts of the appliance were separated and later reattached as necessary. We considered that the appropriate timing for follow-up polysomnography was when the STOP score stopped changing. A historical control group was also established from consecutive patients without a STOP score for whom we had both baseline and follow-up polysomnographic studies (n = 228, June 2005 to May 2011).

Results: The median (interquartile range) STOP scores significantly decreased with oral appliance treatment from 3 (2-3) to 0 (0-1) in the STOP group (n = 69) ($P < .05$, Wilcoxon signed-rank test). The duration from appliance prescription to follow-up polysomnography in the STOP group was longer than that in the control group (147 [115-215] vs 113 [70-198] days, $P < .05$, Mann-Whitney U test). The apnea-hypopnea index (AHI) was improved in both the STOP group (16 [10-22] to 3 [2-7] events/h, $P < .05$) and the control group (18 [13-26] to 6 [3-11] events/h, $P < .05$). However, a greater percentage of subjects achieved a follow-up AHI of < 5 events/h with a > 50% reduction in baseline AHI in the STOP group (44 of 69 patients, 64%) than in the control group (94 of 228 subjects, 41%) ($P < .05$, chi square test).

Conclusions: These findings suggest that recording of the STOP score is helpful for determining the timing of follow-up polysomnography in oral appliance treatment: this may reflect satisfactory adherence to and successful adjustment of oral appliances. We conclude that this simple, quick, and inexpensive evaluation facilitates proper and necessary follow-up diagnosis in oral appliance therapy.

POSTER #007

PEDIATRIC SLEEP-DISORDERED BREATHING: THE DENTAL MIRACLE

White JR[1], Boota A[2], Alexander N[3], Hooks K[4]

[1]*Dental Sleep Medicine-SC, Greenville, South Carolina; [2]Palmetto Pulmonary and Critical Care, PA, St. Francis Hospital, Greenville, South Carolina; [3]Greenville ENT, Greenville Hospital System, Greenville, South Carolina; [4]Medbridge Healthcare, Greenville, South Carolina*

Introduction: Oral respiration, abnormal cranio-facial growth and pediatric obstructive sleep apnea are truly a medical/dental highball with a twist of myofunctional therapy. The constant interaction between oral-facial muscles and oral-facial structural growth starts early in development and continue thru childhood. Chronic open mouth posture and breathing, as Guilleminault tell us, is an important clinical marker of oral-facial dysfunction which over time will alter normal naso-maxillary development. Oral respiration, deficient maxilla and malocclusion are not historical characteristics of the human genotype and suggest that environmental factors have

a significant role and may alter the phenotype. To correct deficient maxilla and malocclusion the dental profession has used rapid maxillary expansion (RME) first introduced by Angell in 1860 and brought back into favor by Haas in 1961. RME consist of application of orthopedic forces to the midpalatal suture with the forces dissipating across the cranial and circum-maxillary sutures. This results in a larger maxillary arch which will accommodate the teeth in a pleasing arrangement but also significantly increase the nasal volume. This increase in nasal volume will decrease airway resistance which among variables causing soft tissue collapse trumps.

Methods: Data were available for two female 9 year old twins. Excessively loud snoring, enlarged tonsils, and excessive daytime sleepiness. Pediatrician disregarded Mother's concerns, and ENT denied adenotonsillectony. Mother presented video of girls snoring, gasping with apneas to a dental sleep professional. Upon examination the dental professional found the girls to be retrognathic and maxillary deficient using a visual clinical assessment; White/Hooks survey. Patients underwent HST with a 4 channel device (effort, flow, pulse, and saturation were monitored), at the request of the dental professional to asses sleep. Increase in heart rate, frequent arousals, and a high number of respiratory events were noted (AHI 83&35). A trial of CPAP at 7 cmH$_2$O was conducted to provide a baseline for treatment results. Patients improved cognition and temperament improved overnight and maintained with continued use of CPAP. RME was performed by the dental professional with continued CPAP use during the expansion. Patient's adenoids and tonsils were removed by pediatric ENT post expansion. Post expansion, adenotonsillectony positive subjective results mimicked those during CPAP trial.

Results: HST was conducted on both girls during treatment. Diagnostically (83&35); With CPAP (0&0); Post RME without CPAP (11&7); Post T&A and RME (5&3).

Conclusions: These positive results should bring appreciation to the complex interplay between normal respiration, craniofacial growth and development and its contribution to pediatric obstructive sleep apnea.

POSTER #008

PREDICTORS OF SUCCESS FOR ORAL APPLIANCE (OA) THERAPY IN OBSTRUCTIVE SLEEP APNEA (OSA) PATIENTS BASED ON INITIAL CRANIOFACIAL CHARACTERISTICS

Khojah M, Correa LP, Finkelman M, Trotman CA, Kanavakis G

Tufts University School of Dental Medicine, Boston, Massachusetts

Introduction: The aim of this investigation was to explore hard and soft tissue cephalometric predictors for the success of oral appliance therapy, in patients with varying severity of OSA.

Methods: A review of 108 consecutively treated patients with OSA was performed at the Dental Sleep Medicine Clinic at Tufts University School of Dental Medicine. Fifty-two subjects, all treated with OA therapy were included. Our predictive factors included BMI, age, gender, mandibular plane angle (MP), vertical distance between MP and the most superior

point of the hyoid bone (MP-H), ANB angle (ANB), soft tissue ANB angle (S.T. ANB), anterior-posterior upper lip position (UL-VL), anterior-posterior lower lip position (LL-VL), and anterior-posterior soft tissue chin position (C-VL). Treatment success was defined in three ways: (1) At least 50% reduction in initial AHI, (2) Residual AHI ≤ 10 after treatment, and (3) Residual AHI ≤ 5. A multiple regression model was developed to study the effect of various variables on success. The level of statistical significance was set at .05.

Results: No statistically significant differences were found between subjects with mild, moderate and severe OSA ($P > .05$). BMI (median = 28.3, IQR = 5.9) was weakly correlated to AHI ($rs = 0.28$, $P = .045$). OA therapy resulted in 51.9%, 55.7% and 30.7% successful outcomes, using the first, second and third methods of defining success, respectively. MP and C-VL were positively associated with treatment success ($AUC_{MP} = 0.67$ and $AUC_{C-VL} = 0.71$).

Conclusions: A weak positive correlation was found between BMI and OSA severity. The MP and C-VL were significantly correlated to the outcome of OA therapy, but showed a weak to moderate predictability for the success of OA therapy. The results should be interpreted with caution and their clinical significance should be investigated in future studies.

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POSTER #009

THE EFFECT OF THE CUSTOM FACE MASK ON THERAPEUTIC CPAP PRESSURE

Prehn RS[1,2,3], Simmons JH[3,4]

[1]*Director Restore TMJ & Sleep Therapy, PA, The Woodlands, Texas;* [2]*Adjunct Professor, University of Texas School of Dentistry, Houston, Texas;* [3]*Sleep Education Consortium, Houston, Texas;* [4]*Comprehensive Sleep Medicine Associates, Houston, Texas*

Introduction: The purpose of this study was to investigate the effect of therapeutic CPAP pressures needed to resolve OSA when patients fabricated a custom face mask (CFM), which is used in combination therapy to treat OSA in patients who presented to a dental sleep center. The CFM is a custom CPAP face mask that is fabricated from an impression of the face. This CFM is then connected to the post attached to an oral appliance. This strapless CPAP face mask features a CPAP interface with mandibular stabilization.

Methods: A retrospective chart review of 35 CFM patients on combination therapy from 2006-2012 was conducted in 2015 to determine changes in therapeutic CPAP pressures caused by utilizing the CFM in combination therapy.

Results: Average CPAP pressures before CFM: 14 cmH$_2$O (± 4). Average CPAP pressures after CFM: 13 cmH$_2$O (± 3). Average reduction in CPAP pressures for those that had reduction (n = 15): 4.3 cmH$_2$O. Average increase in CPAP pressures for those that had increase (n = 12): 1.6 cmH$_2$O. No change in CPAP pressures (n = 8)

Most patients (n = 15) in this follow-up survey that had reduction of CPAP pressures after the fabrication of the CFM, had

significant reduction of pressures (average 4.3 cmH$_2$O). The patients (n = 12) that had increased CPAP pressures after the fabrication of the CFM had only minor increases of CPAP pressure settings (1.6 cmH$_2$O).

The reason the 12 patients had an increase in CPAP pressures (and the 8 patients who had no change) after the fabrication of the CFM, is most likely that the OSA disorder may have actually become worse (nothing to do with the CFM) and the CFM is helping to keep these patients in effective therapy with the higher pressures. These patients were also on the higher end of BMI compared to the ones who had reduction in CPAP pressures. The other possibility is that these patients were under-titrated to begin with (intolerance of high pressures) and now they are able to tolerate these higher pressures with the CFM.

A unique aspect of the CFM is that leakage around the mask is significantly reduced by providing an improved seal of the mask against the face. The design of the mask, by virtue of the way it is fabricated from an accurate impression of the face specifically in line with facial and nasal contours, enables the pressure of the PAP to expand the facial skin outwards towards the mask. This creates a more reliable seal. In the majority of patients in the study, the result was a significant reduction of CPAP pressures allowing a more comfortable experience improving satisfaction and compliance.

Conclusions: The CFM should be considered for patients that require high therapeutic CPAP pressures in order to resolve OSA. The CFM is not only able to handle high pressures, but is also able to provide a secure CPAP interface by its direct attachment to an oral appliance, providing mandibular stabilization and advancement. This will increase effectiveness of combination therapy along with increased patient satisfaction and compliance.

POSTER #010

AERODYNAMIC CHARACTERISTICS OF THE UPPER AIRWAY: OBSTRUCTIVE SLEEP APNEA PATIENTS VERSUS CONTROL SUBJECTS

Chen H, Li Y, Aarab G, Reiber JHC, de Lange J, Lobbezoo F, Tu S, van der Stelt P

Academic Centre for Dentistry Amsterdam (ACTA)

Introduction: To determine the difference in aerodynamic characteristics of the upper airway between obstructive sleep apnea (OSA) patients and their controls.

Methods: We prospectively selected thirteen OSA patients (age = 43.1 ± 9.9, apnea-hypopnea index (AHI) = 14.9 ± 7.1 events/h) and ten control subjects (age = 44.7 ± 15.3). The diagnosis of OSA patients was based on an overnight poly-somnographic recording. To exclude the presence of OSA, the control subjects filled out the validated Philips questionnaire. NewTom5G cone beam computed tomography (CBCT) scans were obtained from OSA patients and control subjects. Computational models of the upper airway of OSA patients and their controls were reconstructed on basis of their CBCT images. Using computational fluid dynamics (CFD) analysis with these models, we characterized the aerodynamic features within the

upper airway (ie, velocity, wall shear stress, wall static stress, and airway resistance).

Results: There was no significant difference in age, gender and body mass index between the OSA patients and controls. In OSA patients, the airway resistance during inspiration (R_{in}) and expiration (R_{ex}) was significantly higher than that in the controls (Z_{in} = -2.2, P_{in} = .03; Z_{ex} = -2.4, P_{ex} = .02).

Conclusions: During respiration, the airway resistance (R) of OSA patients was higher than that in controls. The repetitive collapse of the upper airway in OSA patients can be explained by the higher airway resistance (R) in their upper airway.

POSTER #011

THE VALIDITY OF USING A PHARYNGOMETER FOR AIRWAY MEASUREMENT COMPARED TO CBCT MEASUREMENTS

Ananthan S, Patel S, Kanti V, Kabaria A, Creanga A

Rutgers School of Dental Medicine, Newark, New Jersey

Introduction: An acoustic pharyngometer is a simple device that offers a cost effective, quick and noninvasive method of measuring the upper airway. It is commonly used in a dental sleep clinic to determine the risks of obstructive sleep apnea (OSA). The pharyngometer works on the following principle: an audible sound signal is generated at the bottom of a tubular probe and is transmitted into the cavity via an anatomically fitted coupler. The acoustic pulse is partially reflected when it encounters an area change. The amplitude and temporal changes in the reflected pulse compared with the incident pulse are used to calculate the changes in airway cross sectional area. A computer performs the calculation. Although the pharyngometer is used extensively in airway studies, its use has previously not been validated. The aim of the present study is to establish the validity of the use of a pharyngometer as compared to Cone Beam Computed Tomography (CBCT) measurements.

Methods: Patients who were referred to the Division of Oral Radiology at the Rutgers School of Dental Medicine, Newark, New Jersey for full volume (whole head) CBCT imaging for various reasons were recruited for the study. The iCAT 17-19 (Imaging Science International, Hatfield, Pennsylvania) was used for CBCT imaging. The Eccovision pharyngometer (Sleep Group Solutions, Hollywood, Florida) was used to measure the volume of oropharyngeal space. The volumetric readings from the pharyngometer were compared to the volumetric readings of CBCT oropharyngeal space analysis obtained from InVivo dental imaging software version 5.4.4 (Anatomage, San Jose, California). SPSS version 24 (IBM, Armonk, New York) was used for the data analysis. A Pearson's correlation test was performed.

Results: 25 subjects were recruited for the study. 21 subjects were included in the final analysis. The ages of the subjects ranged from 16-74 years. 16 subjects were female and 5 were male. The Pearson correlation coefficient was r = 0.978.

Conclusions: The correlation between the two modalities of measuring the airway volume is strong. The pharyngometer may be a suitable device for readings in the same patient, between treatment visits, in settings such as sleep clinics (before and after the delivery of a mandibular advancement device), orthodontic settings (before and after palatal expansion), and some instances when airway analysis is desired without radiation exposure. The pharyngometer is a valid tool to use for airway measurement when compared to CBCT readings.

POSTER #012

ASSESSMENT OF THE STOP-BANG SCREENING QUESTIONNAIRES IN DETECTING SLEEP-DISORDERED BREATHING AMONG DENTAL PATIENTS IN A COMMUNITY PRACTICE SETTING

Dillow KD, Sanders AE, Essick GK

University of North Carolina School of Dentistry, Chapel Hill, North Carolina

Introduction: Obstructive sleep apnea (OSA) is widely under-diagnosed, despite simple-to-administer screening questionnaires. One questionnaire that is often recommended is the STOP-BANG. In studies of non-dental patient populations, the STOP-BANG—designed to screen patients for OSA prior to surgery—was found to exhibit high sensitivity but low specificity. We sought to determine if the STOP-BANG is suitable to screen dental patients for sleep-disordered breathing (SDB) by comparing its outcomes with those from nocturnal pulse oximetry.

Methods: Flyers were used to recruit a convenience sample of adults at a community-based dental practice in Raleigh, North Carolina. A dental hygienist administered the STOP-BANG screening questionnaires and issued instructions for overnight pulse oximetry. High-risk for SDB was defined by $\geq$ 3 of the following eight items: loud snoring; daytime tiredness; witnessed apnea; hypertension; BMI > 35; age > 50; neck circumference > 40 cm; male gender (original 2008 scoring criteria). Based on a recent recommendation to improve the specificity of the STOP-BANG, high-risk was alternatively defined as the presence of $\geq$ 5 of the eight items, or $\geq$ 3 items that included at least one of "BMI > 35," "neck > 40 cm," or "male" (2016 scoring criteria). Overnight pulse oximetry classified dental patients according to SDB severity defined by oxygen desaturation index (ODI) at cut-points of $\geq$ 5, $\geq$ 15 and $\geq$ 30 events/h in which oxyhemoglobin saturation decreased $\geq$ 3% from baseline. The sensitivity, specificity, and diagnostic accuracy (area under the ROC curve) of the STOP-BANG were calculated for each of the three levels of ODI severity using each of the two sets of scoring criteria.

Results: Among 119 dental patients studied (mean age = 51 years), 47.9% were male and 24.4% were obese. Over half screened high-risk using the 2008 criteria (67.2%) or the 2016 criteria (56.3%). The percentage of patients with SDB decreased ten-fold with increased ODI severity from 66.4% (ODI $\geq$ 5) to 26.9% (ODI $\geq$ 15) to 6.7% (ODI $\geq$ 30). Across the three levels of SDB severity, the sensitivity of the STOP-BANG in detecting SDB averaged 80.7% (2008 criteria) and

74.2% (2016 criteria); the specificity averaged 39.1% and 51%; and the diagnostic accuracy averaged 0.60 and 0.63, respectively. The accuracy was similar for the different cut-points of ODI severity, whereas the sensitivity and specificity covaried in magnitude.

Conclusions: An unexpectedly high percentage of patients (more than half) in a general dental practice screened high-risk for SDB based on the STOP-BANG questionnaire. ROC curve analysis produced areas under the curve in the range of 0.6 to 0.7, indicating fair diagnostic accuracy when using pulse oximetry as the gold standard. Use of the 2016 scoring criteria resulted in a higher specificity, and thus can be used to minimize the number of dental patients without SDB who are referred for follow-up evaluation by a sleep physician.

POSTER #013

INVESTIGATING THE CORRELATION BETWEEN CHANGES IN THE SEVERITY OF OBSTRUCTIVE SLEEP APNEA AND CHANGES IN PATIENT'S QUALITY OF LIFE USING SAQLI QUESTIONNAIRE

Lamia A, Baflah LA, Correa LP, Mehta NR, Kulich R, Alghanem T, Maloney GE

Tufts University, Boston, Massachusetts

Introduction: The purpose of this study was to investigate whether reduction in apnea-hypopnea index (AHI) would result in an improved self-reported of quality of life for patients using mandibular advancement devices (MAD) for the management of obstructive sleep apnea (OSA).

Methods: Thirty-two (32) subjects were recruited from the Dental Sleep Medicine Clinic, Tufts University Dental School. Data were collected at baseline and at a posttreatment visit following a minimum of 4 weeks of intervention. Data for the SAQLI questionnaire were collected using an Audio Computer-Assisted Self Interviewing survey system and via in-person interviews. Pre- and post-mean AHI scores and Sleep Apnea Quality of Life Index (SAQLI) scores were compared using a paired-sample t-test. The association between the change in SAQLI scores and in AHI between pretreatment and posttreatment were determined using Pearson correlation analysis, as the data were distributed normally. A P value < .05 was considered statistically significant.

Results: A total of 32 subjects were recruited for the study, 18 females and 14 males, with an average age of 53.88 (SD = 12.36) years. Twenty-five subjects, 13 females and 12 males, completed the SAQLI questionnaire and were included in the analysis. A paired t-test was conducted and showed statistical significant results in reduction of AHI values (P = .02), AHI pretreatment 18.41 (15.95) and posttreatment 10.24 (9.74). SAQLI scores were not statistical significant (P = .14). Some patients' quality of life improved to a certain degree, social interaction and symptoms domains were largely improved.

Conclusions: AHI values after intervention showed significant changes; however, the change in severity of AHI was not correlated with the change in severity of SAQLI scores. Results are

discussed with respect the impact of other variables such as depression and anxiety.

POSTER #014

COMBINATION OF MANDIBULAR ADVANCEMENT DEVICE AND POSITIONAL THERAPY IN THE MANAGEMENT OF SEVERE OSA: A CASE REPORT

Karimi N, Correa L, Mehta NR

Tufts University School of Dental Medicine, Boston, Massachusetts

Introduction: The purpose of this study was to present the results of positional therapy (PT) as adjunct option for management of residual OSA in a patient utilizing mandibular advancement device (MAD).

Methods: A 78-year-old male referred by a sleep physician to the Dental Sleep Clinic at Tufts Dental School for the use of MAD due to BIPAP therapy intolerance. Diagnostic sleep study (Type I) revealed severe obstructive sleep apnea (AHI = 59.72, Nadir O_2 = 84%). REM sleep 23.3%. BIPAP titration (18/7 cm) with persistent obstructive events and emergence of central apneas. Examination of masticatory muscles and TMJ within normal findings with a baseline discomfort Visual Analog Scale (VAS) of 0/10. A MAD was fabricated and fitted with 80% maximum jaw protrusion, reaching 90% at the last visit with discomfort VAS scale 0/10 at the end of therapy.

Results: Type III sleep test (WP-200) read and interpreted by sleep physician showed a reduction of OSA severity (AHI = 21.2) and increased Nadir O_2 = 89%, REM sleep 23% with MAD. Combination of MAD and positional therapy revealed additional reduction to normal values (AHI = 3.9) Nadir O_2 = 92%, REM sleep = 23%. Patient was scheduled to follow up his sleep physician and at the dental sleep clinic for long-term follow-ups as standard clinical guidelines.

Conclusions: This case report showed the benefit of body position as an adjunct therapy for residual OSA in patients with mandibular advancement devices. Evidence of positional therapy efficacy is emerging and in combination with MAD could be an important armamentarium in the management of OSA patients in dental practice.

POSTER #015

WEARABLE NON-INVASIVE VIBROACOUSTIC STIMULATION IMPROVES CRANIAL CIRCULATION, QUALITY OF SLEEP AND MINIMIZES NEGATIVE EFFECTS OF APNEIC EVENTS

Aharon NJ[1], Uryash A[2]

[1]*Aharon and Associates, Pittsburgh, Pennsylvania;* [2]*Parallel Biotech, Miami Beach, Florida*

Introduction: Obstructive sleep apnea (OSA) affects 10% of adults and remains an important cause of morbidity leading to progressing functional decline of patients and high healthcare expenditures. OSA is a condition characterized by symptoms of brain dysfunction such as extreme daytime sleepiness, depression, anxiety and memory problems. Recent studies suggest that one of the major causes of brain dysfunction of

sleep apnea sufferers is due to weaker cranial blood flow. Wearable devices that address chronic medical conditions will have a positive impact on disease management and cost.

Vibroacoustic stimulation (VAS) is the noninvasive delivery of sonic stimulation to regional arteries via speaker embedded in a wearable neck applicator. VAS induces pulsatile shear stress on the vascular endothelium via penetrating rhythmic sound waves. This leads to upregulation of a transcriptionally regulated cellular mechanotransduction system and endothelial nitric oxide synthase (eNOS). This in turn increases availability of nitric oxide (NO) and vasodilation.

We hypothesize that physical acoustic forces from VAS may induce increase of cranial blood flow, decrease desaturation, improve quality of sleep and contribute to improvement in brain function in OSA patients.

Methods: We investigated the effects of VAS System (Parallel Biotech, Miami, Florida) on cranial circulation, brain function and quality of sleep in OSA volunteer patients, in compliance with IRB requirements. OSA patients were randomized to receive daily 10 minutes VAS before falling asleep and during REM phase (OSA-VAS) (n = 5) or control (OSA-C) (n = 5) for 2 weeks. Blood flow/oxygenation relationships were analyzed using an infrared-doppler, plethysmography and pulse-oximetry. Sleep phases and quality were analyzed by digital monitoring.

Results: VAS decreased desaturation during REM and NREM sleep, significantly increased distal blood flow and improved quality of sleep. Patients also reported better mood and alertness during wakefulness. VAS increased peripheral cranial perfusion in OSA patients by 15% ($P < .01$). Oxygenation levels were 8% higher in OSA-VAS group ($P < .01$). Data: * $P < .01$ (OSA-C vs. OSA-VAS).

Conclusions: In OSA patients, a wearable non-invasive neck vibroacoustic stimulator (VAS) markedly improved cranial blood flow, quality of sleep and reduced desaturation, possibly via modulation of vertebral and carotid arterial circulation. These findings have implications for prevention and treatment of OSA using non-surgical, cost-effective and sleep-friendly solution.

POSTER #016

COMBINATION THERAPY FOR SEVERE OSA AND RELIEF OF TMD UMBRELLA SYMPTOMS: A CASE REPORT

Mansouri N, Correa L, Mehta NR

Tufts University School of Dental Medicine, Boston, Massachusetts

Introduction: The purpose of this case report is to present the effects of mandibular advancement device (MAD) on a patient with severe obstructive sleep apnea (OSA) and chronic history of TMJ pain, headaches and neck pain and the effects of a combination therapy of continuous positive airway pressure (CPAP) and MAD on OSA patients.

Methods: A 45 year-old male referred by a sleep physician to the Dental Sleep Clinic at Tufts Dental School for the use of

MAD due to PAP therapy intolerance. Diagnostic split night sleep study (Type I) revealed severe obstructive sleep apnea (AHI = 71.6, central apneas = 3, lowest SpO_2: 89%, REM = 0%) in the first half of the night and moderate obstructive sleep apnea and emergent central apneas when using the CPAP in the second half (AHI = 25.4, central apneas = 15, lowest SpO_2 = 86%, REM = 1.1%) . Epworth Sleepiness Scale (ESS) 6/24, PAP therapy was prescribed but he experienced air leakage and developed neck pain when using it. He also reported frequent transmeridian travels for work and having difficulty carrying the PAP machine. Clinical history intake and examination revealed chronic symptoms of TMJ pain, headaches, ear pain, and neck pain with a Visual Analogue Scale (VAS) score of 6/10. A MAD was fabricated and fitted with 80% maximum jaw protrusion

Results: Patient reported improvement in the overall quality of sleep and a significant reduction in TMJ pain (VAS 0/10), ear pain (0/10), headaches (2/10) and neck pain (VAS 2/10) with ESS reduction 2/24. Follow-up split night sleep study (Type 1) interpreted by a board certified sleep physician with the MAD in place for the first half of the night showed a > 60% reduction of OSA severity (AHI = 28.46, central apneas = 2, lowest SpO_2 = 89%, REM = 13.4%) while the second half of the night showed (AHI = 11.57, central apneas = 5, lowest SpO_2 = 92%, REM = 8.4%) while using the MAD and PAP therapy. Combination therapy of MAD and PAP was recommended, long-term follow-ups were scheduled at 6 months and 1-year as standard clinical guidelines.

Conclusions: This case report showed a reduction of OSA from severe to mild, an improvement in REM sleep cycle and resolution of TMD symptoms and chronic headaches. Studies support the direct effect of anterior jaw repositioning and increase of VDO over neck, masticatory muscles, and unloading of TMJ area. The use of MAD for OSA may be beneficial on patients with pre-existing TMD symptoms, and PAP emergent central apneas, in severe OSA cases combination therapy may help to improve PAP compliance by assisting on reducing air pressure. Patient selection is a key factor when using MAD for severe OSA, as anatomical features and BMI are potential clinical predictors for oral appliance success.

POSTER #017

NORMAL RANGE OF MAXIMUM MANDIBULAR PROTRUSION: FIRST STEP IN THE DESIGN AND CONSTRUCTION OF A CUSTOMIZED MANDIBULAR ADVANCE DEVICE

Mayoral Sanz P, Martín JV, Romero MM, Reyes MG

[1]*Alfonso X University of Madrid, Spain*

Introduction: One important aspect of the construction of a mandibular advance device (MAD) is the advancement of the mandible. Frequently, with higher level of advancement, better treatment effect can be obtained, although potential increase of side effects should be considered and balanced. The degree of advancement is usually expressed in % of maximum protrusive capacity or/and in millimeters (mm). Percentage of maximum protrusive capacity is used in reference to potential

side effects and percentage or millimeters to effectiveness in opening the upper airway. Among the studies that address the mandibular movements, just few of them have determined the normal range of this movement. Therefore, the aim of this study was to estimate the range of mandibular advance in a representative sample of adult population.

Methods: 100 students of dentistry 20–24-years-old were included in this study. Measurement of mandibular border paths of Posselt movement was carried out with the aid of a marker fixed to the mandible and recorded using a video camera and then processed by a computer program capable of detecting the marker on the images and reconstructing its trajectory graphically in the 2D space. Maximum protrusion, maximum retrusion and total mandibular advance **were** measured in each patient with 2 mm and 5 mm George Gauge. The statistical analysis was made with the Wilcoxon signed-rank test for paired data.

Results: Mandibular total advance was 13.62 mm with the 2 mm fork of a George Gauge and 12.51 with the 5 mm one 11.37 mm linear and 14.35 mm curved border path of Posselt. The full range of mandibular protrusion movement measured through the border paths of Posselt Diagram of a healthy subject starts with a mandibular downward movement guided by sliding of the mandible head through the posterior wall of the articular eminence and the incisor edge of lower incisors across the lingual surface of upper incisors till the border-to-border position with 2 mm upward and 3 mm forward mandibular displacement. Following this initial movement, there is a 3 mm upward and 9 mm forward mandibular displacement and, at the end of the movement, a small curve is observed.

Conclusions: The values of mandibular motion range for the population studied can serve as a reference parameter for its use in functional evaluation of the mandibular protrusion and in the customization of MAD for treatment of obstructive sleep apnea. The amount of bite opening should be minimized to improve patient tolerance and increase the beneficial effect on upper airway dimensions, since increase in bite opening reduces the range of mandibular advancement.

POSTER #018

CBCT IN THE STUDY OF DIFFERENT PHENOTYPES OF RESPONDERS AND NON-RESPONDERS OF MANDIBULAR ADVANCE DEVICE TREATMENT: A PRELIMINARY STUDY

Mayoral Sanz P[1], Contreras MM[2], Domínguez-Mompell R[3]

[1]Alfonso X University of Madrid, Spain; [2]Rey Juan Carlos University of Madrid, Spain; [3]University of California Los Angeles (UCLA)

Introduction: Mandibular advancement devices (MAD) are increasingly being used in the treatment of obstructive sleep apnea (OSA) as an effective alternative to continuous positive airway pressure (CPAP). MAD protrude the mandible with the aim of increasing upper airway calibre and thereby preventing collapse of the upper airway during sleep. However, the mechanisms by which MAD improve OSA are not well understood. Limited studies have identified an effect of mandibular

advancement on aspects of the structure and function of the upper airway. Therefore, the aim of this study was to study the changes on the airway structures and determine the different phenotype of responders and non-responders of MAD treatment.

Methods: 10 patients mild to moderate OSA treated with MAD, 7 responders and 3 non-responders, were included in this study. A custom-made two-piece MAS Orthoapnea was used. Home sleep monitoring was performed at baseline and 6 weeks after of treatment. Cone-beam computed tomography (CBCT) scans were obtained for all patients with and without MAD.

Results: Mean mandibular advancement was 8.2 ± 1.6 mm (mean ± standard deviation). This produced movement through a connection from the ramus of the mandible to the pharyngeal lateral walls in all subjects. In the sagittal plane, 2 patterns of posterior tongue deformation were seen with mandibular advancement—(A) bidirectional motion pattern in responders and (B) minimal anterior movement in non-responders. Baseline AHI (events/h) responders 19.7 ± 8.3 non-responders 20.6 ± 8.5 and AHI with MAS (events/h) responders 7.7 ± 4.3 and non-responders 22.0 ± 7.8

Conclusions: Mandibular advancement in responders has two mechanisms of action which increase airway size: forward movement of the tongue and lateral airway expansion. CBCT is useful in identifying upper airway form and size changes of MAD treatment.

POSTER #019

DIFFERENCE IN DENTAL ARCH SIZE BETWEEN JAPANESE AND CAUCASIAN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA: AN INTERNATIONAL COMPARISON STUDY

Kohzuka Y[1,2], Taga H[3], Almeida F[1,2], Tsuiki S[1,4]

[1]Department of Orthodontics, Faculty of Dentistry, The University of British Columbia, Canada; [2]Frontier Clinical Research Centre, Canada; [3]JR Tokyo General Hospital, Japan; [4]Institute of Neuropsychiatry, Japan

Introduction: Obesity is a major factor which increases the collapsibility of the upper airway in Caucasian patients with obstructive sleep apnea (OSA). In contrast, cranio- and dento-facial factors could be mainly involved in the development of OSA in Asian people who are less obese in comparison with Caucasians. However, clinically, not all Caucasian OSA patients are obese, similarly not all Japanese OSA patients have OSA due to craniofacial factors. We hypothesized that, when the severity of OSA in addition to obesity was matched between the two races, there would be lesser anatomic differences between Japanese and Caucasian OSA patients.

Methods: The study protocol was approved by the both ethics committee of the Institute of Neuropsychiatry, Tokyo and the University of British Columbia, Vancouver. Amongst OSA patients who visited each Sleep Apnea Dental Clinic, male OSA patients were recruited to the study. As per clinical protocol for each clinic, an upright lateral cephalogram was undertaken to

evaluate maxillomandibular dimensions while study models were fabricated for an analysis of dental arches. After matching age, body mass index (BMI), and apnea-hypopnea index (AHI), a total of 31 Japanese and 32 Caucasian OSA patients were selected for final analysis. The maxillomandibular dimensions as well as the dental arch width and length both in the upper and lower dentitions were compared between the two ethinic groups using unpaired t-tests.

Results: There were no statistically significant differences between Japanese patients and Caucasian patients with regard to age (48 ± 8 vs 48 ± 7 years, $P = .93$), BMI (28 ± 2 vs 29 ± 3 kg/ m^2, $P = .10$), and AHI (25 ± 16 vs 25 ± 12 events/h, $P = .99$). No significant difference was also found with respect to SNA (81 ± 3 vs 82 ± 5 degrees, $P = .39$) and SNB (79 ± 3 vs 78 ± 7 degrees, $P = .63$), and sagittal tongue size (39 ± 3 vs 38 ± 4 cm^2, $P = .79$) between Japanese and Caucasians. In turn, upper intercanine width (27 ± 2 vs 25 ± 2 mm, $P < .01$), upper interfirst molar width (41 ± 6 vs 36 ± 3 mm, $P < .01$), and lower interfirst molar width (37 ± 3 vs 35 ± 3 mm, $P < .01$) were significant larger in Japanese patients than Caucasian patients. Upper (39 ± 5 vs 37 ± 3 mm, $P = .09$) and lower (33 ± 3 vs 32 ± 2 mm, $P = .63$) dental arch lengths were not different between Japanese OSA patients and Caucasian patients.

Conclusions: There is a lateral dilation in the dental arches, in Japanese patients with OSA. We conclude that, although minor, these dental changes in Japanese OSA patients may play an important role in the understanding of craniofacial differences and OSA incidence between Japanese and Caucasian, where Japanese arches are wider but more retropositioned.

POSTER #020

PHARYNGEAL AIRWAY DIMENSIONS WHILE AWAKE CORRELATE WEAKLY WITH OUTCOME OF ORAL APPLIANCE THERAPY FOR OSA

Remmers JE[1,2], Charkhandeh S[2], Zareian Jahromi SA[1,2]

[1]University of Calgary, Calgary, Canada; [2]Zephyr Sleep Technologies, Calgary, Canada

Introduction: While continuous positive airway pressure (CPAP) has been standard medical therapy for obstructive sleep apnea (OSA), this status has recently been called into question by evidence that the therapy does not reduce the incidence of cardiovascular events. Oral appliance therapy (OAT) has higher compliance and is preferred over CPAP by most patients. However, OAT resolves OSA in only 50% to 60% of patients. This highlights the need to prospectively identify therapeutic responders to mandibular protruding OAT.

Imaging the pharyngeal airway may provide a basis for selecting favorable candidates for OAT. Mandibular protrusion changes the mechanics of the collapsible regions of pharyngeal airway, but the protrusion-induced changes in the size of segments of the pharyngeal airway, measured while awake, have not been convincingly linked to patients' responses to OAT. The objective of this research is to assess the relation between such changes in airway geometry and OAT response.

Methods: Twenty-eight individuals with OSA underwent cone beam computed tomography (CBCT) of the upper airway and were treated with a mandibular protruding oral appliance (MicrO$_2$). Baseline oxyhemoglobin desaturation index (ODI) was measured as the mean value determined from two nights of home monitoring with a validated recorder. Therapeutic ODI was similarly measured as the mean value determined from two home sleep tests while wearing a custom-made OA adjusted to a final protrusive position. OAT outcome was calculated as the fractional reduction in ODI comparing baseline and therapeutic values.

A scan was performed while awake with the mandible at each of two positions: centric occlusion (CO) and mandibular advancement (MA). No appliance was in the mouth for CO scan. The MA scan was performed with a temporary oral appliance in place and set at 90% of full protrusion. The scans were taken while the participants were seated and relaxed. Five geometric measurements were obtained from each of the two pharyngeal segments, velopharynx and oropharynx, namely: volume (V), minimum cross-sectional area (MCA), anterior-posterior (A-P) and lateral-lateral (L-L) distances at MCA, and mid-sagittal area (MSA). The relative protrusion-induced change in each geometric variable was correlated with OAT outcome.

Results: Mandibular protrusion significantly increased V and L-L of the velopharynx and the oropharynx ($P < .05$). MCA, A-P, and MSA did not differ statistically between CO and MA scans for either segment. A linear regression analysis comparing OAT outcome with fractional change in each of the ten variables revealed no significant correlation. Weak, non-significant correlations were observed for OAT outcome versus velopharyngeal V ($r = -.33$; $P = .08$) and OAT outcome versus oropharyngeal MSA ($r = .32$; $P = .09$). A multi-variable regression illustrated a weak, but significant correlation between all features and OAT outcome ($r = .51$; $P = .006$).

Conclusions: We demonstrate in OSA individuals a significant, but weak, correlation between the protrusion-induced increase in pharyngeal dimensions while awake and OAT outcome.

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POSTER #021

UTILIZING A FULLY DIGITAL CLINICAL WORKFLOW FOR ORAL APPLIANCE THERAPY WITH AN AUTO-TITRATING MANDIBULAR POSITIONER (AMP): A FEASIBILITY STUDY

Charkhandeh S[1], Vranjes N[2], Kuhns D[3], Mosca E[1], Bruehlmann S[1]

[1]Zephyr Sleep Technologies, Calgary, Canada; [2]The Snore Centre, Calgary, Canada; [3]ProSomnus Sleep Technologies, Dublin, California

Introduction: It is estimated that in the US alone, the number of patients on oral appliance therapy (OAT) will be over one million by 2023. To keep up with the rising demand, more efficient and precise workflow models are required to minimize inaccuracies and costs associated with delivery of care. The

purpose of this study is to evaluate the feasibility of a novel, fully digital workflow model, utilizing intra-oral digital scanning and CAD/CAM device manufacturing, in combination with an AMP for patient selection and effective target protrusion (ETP) prediction to minimize inefficiencies and improve quality of care.

Methods: In the first study group (Group A: n = 30), the workflow impact of placing participants prospectively determined to be successful with OAT directly at a pre-selected target was evaluated. A CAD/CAM MRD (MicrO$_2$ Sleep Device) was inserted at the pre-determined ETP or if required, at a lower protrusion with instructions to adjust in 1-2 mm increments to ETP at home. During the first year, additional follow-up appointments requested by the participants were recorded.

In the second study group (Group B: n = 5), we evaluated the feasibility of utilizing existing technologies to create a fully digital clinical workflow for manufacturing MRDs at a pre-selected target. Two CAD/CAM MRDs (MicrO$_2$) per patient were manufactured using a conventional method (PVS impressions and bite registration) and a digital method using an intraoral digital scanner (iTero) and a "digital open-bite registration". Each patient received both appliances and the dental fit, occlusal fit and patient preference were recorded.

Results: Group A: The median ETP was 63% (range: 36% to 100%). 67% of participants had their OA inserted directly at ETP, including 2 who had an ETP > 80%. All participants self-calibrated at home to achieve ETP, where 86% were a therapeutic success. 4 participants required in-office appointments for calibration of the OA to achieve success. Once therapeutic success had been achieved, 12 participants required 1-2 non-calibration follow-up appointments for reasons such as new dental restorations, polishing, repair, or discomfort. The remainder of participants did not require additional dental chair time.

Group B: In the conventional workflow, 2 appliances required minor dental adjustments & 3 appliances required occlusal adjustments. In the digital workflow, no dental or occlusal adjustments were required. All patients preferred the digital MRDs in terms of comfort.

Conclusions: Utilizing the existing tools and technologies, it may be possible to create new workflow models for OAT that are more accurate, require less follow-up and chair time, and improve patient satisfaction. In combination with an AMP test to select suitable patients for OAT and identify an ETP, these models may improve the quality and delivery of care. Further well-controlled studies are required to test the complete workflow.

POSTER #022

CLINICAL SIGNIFICANCE OF PATIENT PHENOTYPE IN OUTCOME PREDICTION FOR ORAL APPLIANCE THERAPY AND THERAPEUTIC PROTRUSIVE POSITION (TPP) BASED ON AN AUTO-TITRATING MANDIBULAR POSITIONER (AMP) TEST: A RETROSPECTIVE ANALYSIS

Charkhandeh S[1], Zareian Jahromi SA[1,2], Bruehlmann S[1], Mosca E[1]

[1]*Zephyr Sleep Technologies, Calgary, Canada;* [2]*University of Calgary, Calgary, Canada*

Introduction: Oral appliance therapy (OAT) remains an underutilized treatment, likely due to its inconsistent efficacy and the lack of a standard objective titration method to reach an effective therapeutic protrusive position (TPP). We have previously demonstrated the accuracy of an at-home auto-titrating mandibular positioner (AMP) in prospectively identifying OAT outcome in > 150 patients. The objective of this study was to retrospectively analyze the data set to evaluate the correlation between patients' physical characteristics (OSA severity, weight, and dental anatomy) and OAT outcome and TPP.

Methods: Participants (n = 48) with obstructive sleep apnea (OSA) participated in a study to evaluate the accuracy of the AMP device. Following the AMP test with a temporary dental appliance, each participant was treated with a custom oral appliance and advanced until therapeutic success was achieved as determined by outcome sleep tests. The final protrusion was determined the TPP. In this study, characteristics of baseline ODI, BMI, neck circumference (NC), age, dental overbite (OB) & overjet (OJ) were evaluated for correlation with OAT and TPP in the group that achieved therapeutic success (ODI < 10 events/h).

Results: The values for correlations between TPP and baseline characteristics for the group who achieved therapeutic success were: baseline ODI, $r = -.30$, $P = .09$; BMI, $r = .51$, $P = .01$; NC, $r = .65$, $P < .01$; OJ, $r = -.14$, $P = .44$; OB, $r = -.07$, $P = .70$; and age, $r = -.23$, $P = .20$. For the group who achieved therapeutic success, the values for correlation between TPP and ΔODI were $r = .25$, $P = .15$, and between TPP and final ODI were $r = .16$, $P = .36$.

There were no significant differences found between individuals who were successfully treated with OAT and those who were not for any of the following characteristics: BMI, $P = .42$; NC, $P = .27$; OB, $P = .62$; OJ, $P = .13$. A significant difference was found between groups for baseline ODI, with the group who experienced therapeutic success having a lower baseline ODI than the group who did not ($P < .001$).

Conclusions: There were no significant correlations between TPP and baseline ODI, OJ, OB, age, ΔODI, or final ODI for participants who achieved therapeutic success. Weak, yet significant correlations were found between TPP and BMI and NC among those known to be therapeutic successes; however, as there was no correlation with outcome with any measures the finding is not clinically relevant. For prediction of outcome, despite there being a significant difference between the therapeutic success and failure groups for baseline ODI, there was no correlation between therapeutic position and ODI for those who achieved therapeutic success. The results of the analysis show that none of the factors can individually be used as a reliable tool to predict outcome or the amount of protrusion required to achieve efficacious treatment with OAT.

POSTER #023

CUSHING'S DISEASE CUSHIONING THE PATIENT'S AIRWAY

Patel IY[1], Patel SI[2], Carr AB[1]

[1]Department of Dental Specialties, Mayo Clinic College of Medicine, Rochester, Minnesota; [2]Center for Sleep Medicine, Mayo Clinic College of Medicine, Rochester, Minnesota

Introduction: Cushing's disease occurs as results of hypersecretion of cortisol in the blood. Cushing's disease occurs in about 15 per million people with average age of diagnosis 20-50 years old. Obstructive sleep apnea is common in Cushing's disease. It is thought that fat accumulation in the parapharyngeal space is the cause of obstruction. The treatment of Cushing's disease impacts the severity of obstructive sleep apnea.

Methods: We reviewed a case of Cushing's disease with a large pituitary microadenoma that underwent transphenoidal resection and the impact of surgery on OSA.

Results: A 58-year-old with a past medical history of Cushing's disease, large pituitary microadenoma status post transphenoidal resection was referred for oral appliance therapy to treat the residual obstructive sleep apnea.

He reported increase in blood pressure over the last 3-5 years, weight gain, hypogonadism, and presence of obstructive sleep apnea for the last 14 years. The first polysomnography (PSG) revealed an apnea-hypopnea index (AHI) of 9 and respiratory disturbance index (RDI) of 32. After symptoms onset of Cushing's disease, PSG showed AHI 66 and RDI of 67.

Polysomnography performed four years after transphenoidal surgery, revealed AHI 8 and RDI 13. Pre-operative MRI was notable for pituitary microadenoma 7-8 mm. Post-operative MRI had no evidence of residual neoplasm.

His Epworth Sleepiness Scale score was 4/24. He was utilizing an over the counter oral appliance with residual symptoms of snoring.

Physical examination revealed Friedman's class III oropharynx, neck circumference 41 cm, maximum interincisal opening of 53 mm, right excursion of 12 mm and left excursion of 14 mm. No temporomandibular disorder.

A custom-fitted oral appliance was fabricated and dispensed. The mandible was advanced 60%. Snoring resolved. A follow-up sleep test is pending.

Conclusions: OSA in some patients with Cushing's disease secondary to a pituitary microadenoma may improve or resolve after surgery. As illustrated by this case, Cushing's disease patients with OSA preoperatively should be retested a few months after successful surgery to determine whether OSA is still present. Oral appliance can be an effective treatment modality depending on the severity of the disease.

POSTER #024

DENTAL SIDE-EFFECTS OF LONG-TERM OBSTRUCTIVE SLEEP APNEA THERAPY: A 10-YEAR FOLLOW-UP STUDY

Uniken Venema JAM[1,3], Hoekema A[2], Sokolova D[3], Doff M[3]

[1]Rijksuniversiteit Groningen, Groningen, Netherlands; [2]ACTA, Amsterdam, Netherlands; [3]Universitair Medisch Centrum Groningen, Groningen, Netherlands

Introduction: Obstructive sleep apnea (OSA) is a sleep-related breathing disorder. OSA is characterized by repetitive obstruction of the upper airway during sleep. Patients are usually treated with either continuous positive airway pressure (CPAP) or oral appliance therapy. The objective of this study is to evaluate changes in dental occlusion, which are associated with long-term oral-appliance and CPAP therapy.

Methods: 29 OSA patients using an anterior traction oral appliance and 34 patients using CPAP therapy, were evaluated. Data was analyzed at baseline, 2-year and 10-year follow-up. Changes in dental occlusion were manually analyzed from dental plaster casts using a digital sliding caliper.

Results: At 2-year follow-up, oral appliance therapy resulted in significant dental changes as compared to CPAP therapy. Overjet and overbite decreased on average with 1.5 mm (SD ± 1.5 mm) and 1.2 mm (SD ± 1.1 mm), respectively. The anterior-posterior change in occlusion was significantly larger in the oral appliance group (–1.3 ± 1.5 mm) as compared to the CPAP group (–0.1 ± 0.6 mm). Both groups showed a significant decrease in number of occlusal contact points in the (pre)molar region. At the 10-year follow-up, higher significant changes were seen in overjet and overbite, but also in anterior-posterior change and in the number of contact points in the (pre)molar region. Definitive analysis are currently being conducted and will follow.

Conclusions: This study confirms that oral appliance and CPAP therapy changes dental occlusion significantly. These changes appear more pronounced with an anterior traction oral appliance as compared to CPAP therapy.