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"Getting Over" Occlusal Changes

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The growing body evidence confirming the effectiveness of oral appliance therapy (OAT) in disease alleviation should increase demand for treatment.[1] Patients should not be denied access to OAT because of possible side-effects but they need to be informed.

OAT for sleep disordered breathing results in occlusal changes. The evidence of occlusal change occurring with OAT has been growing for years.[2,3] Pliska et al.[2] recently confirmed that occlusal changes continue throughout OAT. Although most individual studies have documented the changes with the use of a particular appliance, all appliances can cause occlusal changes. If clinicians haven't seen change perhaps they are not looking. For years Dr. Alan Lowe has told us to "get over it!"[4] and now we could add, "go forward." What does "go forward" mean from a clinical and a research perspective?

In patients intolerant of CPAP who have moderate to severe OSA, occlusal changes are of relatively minor importance when balanced against the long-term consequences of untreated OSA. Surprisingly, especially given some philosophies in dentistry concerning ideal temporomandibular joint position and occlusion, occlusal changes go unnoticed by many patients. Patients adapt with few problems given that they return for multiple replacement appliances over years of treatment. However, there will be those who will find occlusal changes unacceptable and they should be given the opportunity to refuse treatment. The fact of occlusal change should be part of every treatment plan presentation and the importance placed in context of the individual disease severity.

Clinicians employ a variety of strategies intended to help patients maintain their occlusion. These strategies include: stretching exercises, biting with a jig or occlusal splint and chewing gum. There is some evidence to show the effectiveness of these strategies in the short term[5–7] but no evidence to show the long term effect of any of the occlusal maintenance strategies.

We need to respond with scientific evidence to the patient questions of "if I do the exercises, by what percent will my chance of significant occlusal changes decrease?" "Will the exercises cause any long term detrimental effects to my teeth?" We need the studies looking at strategies to mitigate occlusal changes. Hopefully 2015 will bring some answers to these questions.

CITATION

Dort LC. "Getting over" occlusal changes. *Journal of Dental Sleep Medicine* 2015;2(1):3.

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EDITORIALS

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Setting the Strategic Direction for the American Academy of Dental Sleep Medicine

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Kathleen M. Bennett, DDS, LLC, Cincinnati, OH; President, American Academy of Dental Sleep Medicine

As president of the American Academy of Dental Sleep Medicine (AADSM), one of my chief responsibilities is to support the proper alignment of our organizational priorities and initiatives.[1] For nearly a quarter century, the AADSM has been the leader in advancing dental sleep medicine and promoting recognition of oral appliance therapy.[2] However, the ongoing turbulence in the changing U.S. health care system makes it more important than ever to ensure that the AADSM's route is fixed on its mission to "advance the dentist's role in the treatment of sleep disordered breathing." One way for an organization to keep its bearings and remain on course is through the development of a comprehensive strategic plan.

In September, the AADSM board of directors held a strategic planning meeting, during which we reviewed and discussed the results of an independent membership survey that was conducted to provide a better understanding of the needs and expectations of our membership. Results show that core interests of our members include continuing education, professional standards and policies, and stronger integration between the medical and dental communities.[3] The survey provided a starting point for an extensive strategic planning process, which resulted in a final strategic plan that was approved by the AADSM board of directors at our quarterly meeting in December. I would like to share with you some of the strategic goals that will guide the AADSM over the next three years as we continue to help members excel in the practice of dental sleep medicine to reduce the burden of snoring and sleep apnea.

Define the Scope and Nature for Dental Sleep Medicine Practice

As more dentists begin to treat obstructive sleep apnea every year, and with physicians increasingly becoming aware of the effectiveness of mandibular advancement devices, it is critical for the AADSM to continue to clarify the role of the dentist in the management of patients with sleep disordered breathing. In 2013, the AADSM updated its treatment protocol for oral appliance therapy, providing clear and concise directions to help dentists practice within the limits of our licensure.[4] Currently the AADSM, in partnership with the American Academy of Sleep Medicine (AASM), is finalizing a draft of updated clinical practice guidelines for oral appliance therapy, which will be made available for public comment before being published in 2015. Looking ahead, the AADSM also is planning to conduct a consensus conference to develop a comprehensive standard of care paper for the practice of dental sleep medicine, clearly delineating the role of the dentist in the collaborative care of patients with sleep disordered breathing.

Increase the Number of Providers Trained in Dental Sleep Medicine

In tandem with the obesity epidemic in the U.S., the public health burden of sleep disordered breathing has continued to rise over the last decade.[5] Therefore, it is critical for a greater number of dentists to become involved in providing effective treatment for the millions of people with obstructive sleep apnea. The AADSM will continue to recruit more dentists to join us in our mission while also supporting the work of the American Board of Dental Sleep Medicine in promoting the highest standards of care.

Support and Advance Quality in Dental Sleep Medicine

Health care reform is drawing more attention to quality of care by promoting the Triple Aim of better care for individuals, better health for populations, and lower per capita costs.[6] Because oral appliance therapy addresses a chronic medical disease that is intertwined with a patient's overall health care, it is essential for dental sleep medicine practitioners to demonstrate quality and value in the care that we provide. Therefore, the AADSM is going to develop and evaluate quality measures for oral appliance therapy. Recognizing that scientific research provides a foundation for quality clinical care, the AADSM also is going to explore opportunities to fund new research grants while continuing to publish relevant research in the *Journal of Dental Sleep Medicine*.

Broaden Educational Offerings to Better Meet the Needs of Members

Providing the highest quality of professional education for members of every level of experience continues to be a point of emphasis for the AADSM. We will strengthen and expand our year-round offering of educational opportunities—including the AADSM annual meeting, Essentials of Dental Sleep Medicine course, Practical Demonstration Course, and Study Club—to keep members on the cutting edge of dental sleep medicine.

Strengthen Integration between the Dental and Medical Communities

A clear mandate from the recent membership survey was the need for the AADSM to facilitate greater collaboration between dentists and sleep physicians. One of our top priorities is to streamline patient referrals between AASM accredited sleep centers and qualified dentists. The AADSM also is taking a more proactive approach to educate sleep physicians about recent advances in oral appliance therapy. As a result of our efforts, dental sleep medicine leaders will be presenting a

session at the AASM Sleep Medicine Trends Course in February and a meet-the-professor session at SLEEP 2015, the 29th Annual Meeting of the Associated Professional Sleep Societies, LLC, in June. Recently the AADSM also became a partner in the National Healthy Sleep Awareness Project, which is funded by the Centers for Disease Control and Prevention and led by the AASM. The project promotes the achievement of the Healthy People 2020 sleep health objectives, one of which is to increase the medical evaluation of people with symptoms of obstructive sleep apnea.

I look forward to providing periodic updates to keep members informed of the AADSM's progress as we set our course on the route established by our strategic plan.

CITATION

Bennett K. Setting the strategic direction for the American Academy of Dental Sleep Medicine. *Journal of Dental Sleep Medicine* 2015;2(1):5–6.

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Combination Therapy for Obstructive Sleep Apnea in Order to Achieve Complete Disease Alleviation: from Taboo to New Standard of Care?

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Obstructive sleep apnea (OSA) is a highly prevalent disease with a complex pathophysiology.[1,2] When undiagnosed or untreated OSA leads to subsequent morbidity and significant long-term mortality, also in patients with mild to moderate disease.[3] Treatment with continuous positive airway pressure (CPAP) and oral appliance therapy with mandibular advancement devices (OAm) are the two treatments for OSA whose effects on cardiovascular endpoints have been assessed in randomized controlled trials (RCT).[4–7] However, both therapeutic options have their possible limitations as CPAP treatment often comes with low patients' acceptance, poor tolerance and suboptimal compliance, whereas OAm therapy generally has an inferior efficacy as compared to CPAP.[4,8–10] Indeed, according to the mean disease alleviation (MDA) concept, the risk that the greater efficacy of CPAP is being offset by its inferior compliance relative to OAm therapy is not imaginary, resulting in a similar overall effectiveness for both therapeutic modalities.[4,9] In other words, treatment of OSA with CPAP, OAm or other non-CPAP modalities, as a single treatment, will commonly be associated with an incomplete elimination of the disease with mean values of the MDA index, as a marker of real clinical effectiveness, ranging from 40 to 59 %.[8,11,12]

Supporting data from the literature on the role and impact of adjunct therapy that can be used as needed in order to improve the MDA index of the primary treatment options are scarce.[13] Case series have been published on the combination of CPAP with OAm therapy for the treatment of OSA.[14,15] It has been demonstrated that when mandibular protrusion is limited in case of OAm therapy, a combined approach utilizing both mandibular protrusion and tongue retention is able to provide an effective treatment for moderate-to-severe OSA as the addition of a tongue bulb to the OAm provides further therapeutic effectiveness.[16] Furthermore, adjunct OAm therapy has been shown to be an effective mode of combination therapy to control OSA after failure of upper airway surgery.[2,17] Multilevel surgery, being the combination of more than one surgical technique, can also be considered as combination therapy for OSA.[18–23] In obese patients with severe OSA, CPAP requirements might be decreased after bariatric surgery and these lower CPAP pressures may yet lead to an increased CPAP tolerance and an improved overall compliance with CPAP.[23] The effectiveness of other conceivable and convenient combinations in specific patients needs to be put into perspective and requires further investigation in ongoing studies or future trials.

Interestingly, in addition, at least half of OSA patients are reported to suffer from supine-dependent or positional OSA (POSA), commonly defined as an apnea/hypopnea-index (AHI) being at least twice as high in the supine sleeping position as compared to the AHI in the non-supine positions.[24,25] While newer methods to treat POSA with higher compliance rates are emerging, the relevance of this finding of POSA being that common is highly relevant.[26–28] Recently, it has been reported that about 1 out of 3 patients still suffer from residual POSA while undergoing OAm therapy.[29] The results of a recently published RCT indicate that in these patients with residual POSA under OAm therapy the combination of OAm and a sleep position trainer leads to a significant improvement of the therapeutic effectiveness with an increase of the MDA index changing from 42% with OAm alone to 70% with the combination treatment of OAm and sleep position trainer, respectively.[30] Similarly, positional treatment might also be an adjunct treatment in patients on CPAP who require higher pressure levels in the supine sleeping position than in the non-supine positions in order to improve CPAP compliance.[31] From a theoretical and conceptual perspective, the combination of positional therapy and surgical upper airway modifications could also result in a significant decrease in OSA severity in patients with POSA.[32,33]

In summary, the combination of different treatment options for the alleviation of OSA is clearly underestimated, undervalued and underinvestigated in the field of sleep medicine. In order to reach the target, preferably an alleviation of the disease, it might indeed be necessary to prescribe two or more therapies with adjunctive therapies used as needed to supplement the primary treatment options.[13] In order to avoid incomplete alleviation of OSA, our field needs to think out of the box and try to get rid of the taboo of combining different treatment options in the individual patients in order to reach a well-defined target correlating with long-term elimination of disease. Further research on the clinical effectiveness of the possible combinations to completely eliminate the patient's OSA is highly needed.

CITATION

Vanderveken OM. Combination therapy for obstructive sleep apnea in order to achieve complete disease alleviation: from taboo to new standard of care. *Journal of Dental Sleep Medicine* 2015;2(1):7–8.

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REVIEW ARTICLES

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Cardiovascular Benefits of Oral Appliance Therapy in Obstructive Sleep Apnea: A Systematic Review

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Study Objectives: To perform a systematic review of the current evidence regarding the cardiovascular benefits of oral appliance (OA) therapy in obstructive sleep apnea (OSA) patients.

Methods: A systematic review of relevant articles retrieved from online databases (PubMed, Web of Science, Medline, OvidSP) was conducted. All relevant studies published prior to January 20, 2013 that examined the effects of OA on any of the cardiovascular parameters were included.

Results: OA therapy could have a beneficial effect on blood pressure (BP), endothelial function (EF), and left ventricular (LV) function of the heart. Eleven articles were included in this systematic review; 7 of 8 studies showed a significant reduction in BP with a mean BP decrease of 4.2 mm Hg, 2 studies showed significant improvement in EF, and 1 study showed significant improvement in LV heart function.

Conclusion: OA therapy showed beneficial effects on the cardiovascular comorbidity in OSA patients. In studies comparing OA to CPAP therapy, effects of OA therapy were in the same order of magnitude as the effect of CPAP therapy.

Keywords: obstructive sleep apnea, cardiovascular, oral appliance therapy, blood pressure

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Obstructive sleep apnea (OSA) is characterized by repetitive episodes of partial or total upper airway obstruction during sleep, resulting in a decrease of oronasal airflow.[1] OSA is acknowledged as a serious health problem,[2,3] and it is the most common sleep-related breathing disorder. The current prevalence estimates of moderate to severe sleep disordered breathing (apnea-hypopnea index [AHI], measured as events/hour, ≥ 15) are 10% among 30- to 49-year-old men, 17% among 50- to 70-year-old men, 3% among 30- to 49-year-old women, and 9% among 50- to 70-year-old women.[4]

The pathophysiology of OSA and its effect on cardiovascular conditions has been reviewed extensively.[2,5,6] During an episode of upper airway obstruction, respiratory effort against the occluded airway generates a negative intrathoracic pressure that increases left ventricular transmural pressure. The increased stress on cardiac muscle stimulates autoregulatory mechanisms that lead to thickening of the left ventricular (LV) wall over time. Negative intrathoracic pressure also increases right ventricular pressure during diastole, and the apnea-induced hypoxia causes pulmonary vasoconstriction, increasing right ventricular afterload.[7] Long-term intermittent hypoxia can induce oxidative stress and activate inflammatory pathways that impair vascular endothelial function (EF).[8,9] The brief arousal from sleep that accompanies apnea termination increases sympathetic activity and suppresses vagal tone. These acute effects do not only lead to oscillations in blood pressure (BP) and heart rate during sleep, but these may also result in daytime hypertension, increased heart rate during the day, and congestive heart failure.[10,11] Hypertension has been found in a large group of OSA patients, and a positive correlation between BP and OSA severity has been shown.[12–14] The repetitive interruptions in breathing cause sleep fragmentation associated with hypoxia and provoke overnight hypertension, leading to atrial fibrillation (AF), myocardial infarction, (MI) and sudden death.[15]

Continuous positive airway pressure (CPAP) is the gold standard treatment for OSA.[16] However, despite its high therapeutic efficacy, CPAP is often not well tolerated by patients, resulting in low compliance rate and limited clinical effectiveness.[17,18] Today, oral appliances, particularly mandibular advancement devices (OAm), are considered to be a valuable non-invasive treatment option for patients with sleep apnea and for patients who do not comply with or refuse CPAP treatment.[19–23] They are worn intraorally at night in order to advance the mandible, thereby reducing the collapsibility of the upper airway.[18,21,24] Studies have shown that OAm patients have been more compliant than CPAP patients. This higher compliance results in a comparable effectiveness.[25–27]

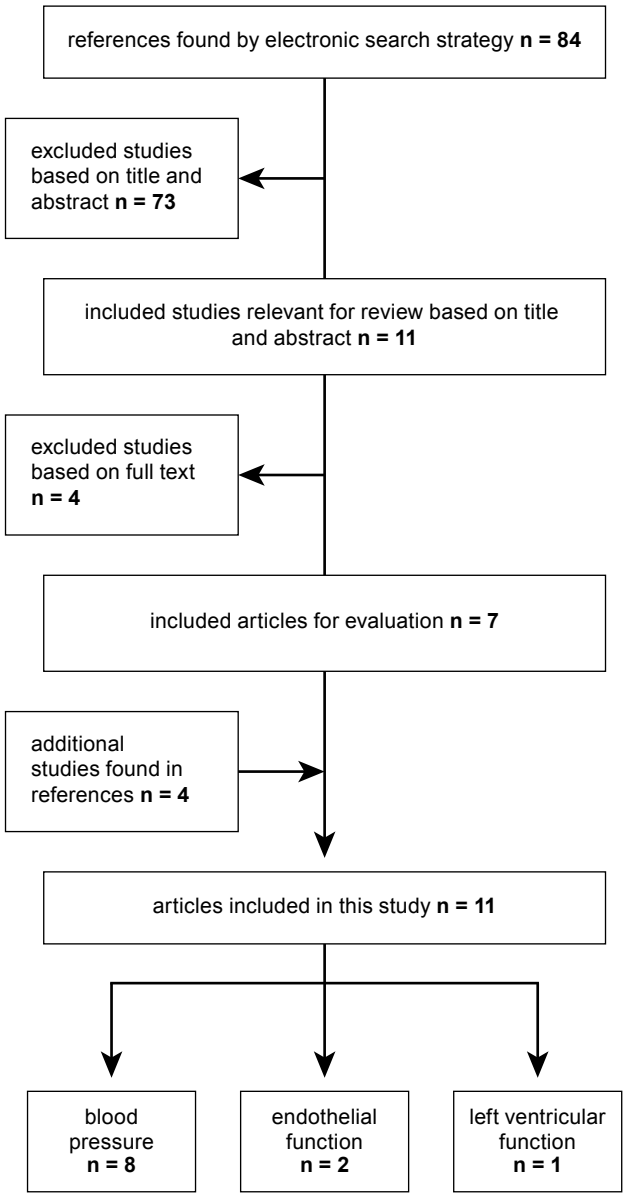
Gaining insight in pathophysiology and treatment of OSA becomes increasingly relevant, as OSA is a common disorder with a range of harmful sequelae. The estimated prevalence rates of OSA represent substantial increases (up to 55%) partly due to increasing awareness and the ongoing obesity epidemic.[4] BP, EF, and LV function of the heart are frequently used to assess cardiovascular morbidity in association with OSA in literature.[19] These objective parameters give an indication of cardiovascular changes during OAm therapy in comparison with baseline measurements, thus clarifying the possible beneficial effect of OAm therapy as an alternative for CPAP. Since the cardiovascular morbidity and mortality is an important feature

in OSA, we performed a systematic review of articles studying cardiovascular changes during OAm therapy to expose the value of OAm.

Table 1—Overview of search terms.

Search terms	Results found
Obstructive Sleep Apnea AND oral appliance AND blood pressure	54
Obstructive Sleep Apnea AND oral appliance AND cardiovascular	40
Obstructive Sleep Apnea AND oral appliance AND (cardiovascular benefit OR cardiovascular improvement OR cardiovascular impact)	15
Obstructive Sleep Apnea AND mandibular advancement AND blood pressure	35

Figure 1—Search strategy.



METHODS

We searched 4 online databases: Pubmed, Web of Science, Medline and OvidSP. All relevant studies published prior to January 20, 2013, that examined the effects of OAm on any of the cardiovascular parameters were included. Using the results produced by the search terms listed in Table 1, a first selection was made based on the content of the title and abstract.

A second selection was then based on the evaluation of the content of the manuscripts. Studies that investigated non-cardiovascular effects of OAm or studies that did not include any oral appliance arm were excluded. Figure 1 explains the search strategy in detail. Finally, a total of 11 studies could be included in the present review. Because the topic of this literature study is relatively new, no exclusions were made based on publication date; all studies were published after 2003.

Systematic Review

Apnea-Hypopnea Index (AHI)

One way to measure the outcome of OAm therapy is using the apnea-hypopnea index (AHI) that represents the number of apneas and hypopneas per hour of sleep. An obstructive apnea is defined as an interruption in nocturnal breathing ≥ 10 sec despite continued respiratory effort. Hypopneas are abnormal respiratory events lasting ≥ 10 sec with ≥ 30% reduction in thoracoabdominal movement or airflow, and with ≥ 4% oxygen desaturation.[28] Therefore, several studies not only described the cardiovascular effect of OAm, but also the change in AHI after OAm therapy. Table 2 represents the mean AHI values of the different studies.

Blood Pressure (BP)

As previously mentioned, there is a positive correlation between BP and OSA severity expressed in terms of AHI. The following studies investigated the effect of OAm therapy on a decrease in BP values.

In a randomized clinical trial protocol, Gotsopoulos et al.[29] examined the BP of 61 patients diagnosed with OSA (AHI ≥ 10/h) before and after 4 weeks of OAm treatment. The control group was treated with an OAm without mandibular protrusion. They found a significant reduction of 24-h diastolic blood pressure (DBP) after OAm therapy in comparison to the control group, mainly due to the effect of therapy on daytime DBP. Daytime BP values with OAm were significantly lower than those of the control group: systolic blood pressure (SBP) −3.3 ± 1.1 mm Hg, DBP −3.4 ± 0.9 mm Hg and mean arterial pressure (MAP) −3.6 ± 0.9 mm Hg. The 24-h heart rate (HR) was reduced in comparison to the control group, which was caused by a daytime HR reduction of 4 ± 1 beats per minute in comparison to the control group. The BP reduction after OAm therapy was most pronounced in the early morning, when the risk of a myocardial infarction is highest.[23,30]

Barnes et al.[31] examined 114 patients with mild to moderate OSA (5/h < AHI ≤ 30/h). In this RCT, patients were treated with CPAP, OAm, and placebo for 3 months each. Eighty patients completed all 3 treatments. Patients treated with OAm showed a significant decrease in DBP at night (−2.2 ± 0.7 mm Hg). A significant number of the patients who did not show a BP dip at

Table 2—Summary of OAm treatment studies assessing blood pressure parameters.

Study	Year	N	Age (mean ± SD)	% Male (N)	BMI (mean ± SD)	Duration of intervention	Method of BP measurement	Mean SBP change (mm HG)	Study Type	Pre AHI/h (mean ± SD)	Post AHI/h (mean ± SD)	Δ%
Gotsopoulos et al.[29]	2004	61	48 ± 11	79% (53)	28 ± 5	4 weeks	24-h	−3	RCT	28 ± 17	12 ± 2	57%
Barnes et al.[31]	2004	114	46	79% (67)	31	12 weeks	24-h	0	RCT	21 ± 11	14 ± 10	34%
Yoshida et al.[32]	2006	161	54 ± 14	75% (121)	25 ± 4	15 weeks	Clinical	−5	case series	18 ± 14	6 ± 6	68%
Otsuka et al.[33]	2006	11	52 ± 7	73% (8)	29 ± 4	32 weeks	20-h	−5	case series	25 ± 20	6 ± 4	75%
Andrén et al.[34]	2009	29	57	62% (18)	29 ± 4	3 years	Clinical	−14	case series	16 ± 9	4 ± 3	75%
Andrén et al.[35]	2013	72	58 ± 8	79% (57)	29 ± 4	3 months	24-h	−2	RCT	23 ± 16	8 ± 6	66%
Lam et al.[36]	2007	34	45	76% (26)	27	10 weeks	Clinical	−1	case series	21 ± 10	11 ± 10	49%
Phillips et al.[25]	2013	108	49 ± 11	81% (87)	29 ± 5	1 month	24-h	−2	RCT	26 ± 12	11 ± 12	56%

SD, standard deviation; 24-h (or 20-h), automatic BP measurement during 24 hours a day (or 20 h); Clinical, BP measurement manual or electric; BMI, body mass index (kg/m^2); AHI, apnea-hypopnea index; RCT, randomized controlled trial.

night prior to treatment—a sign associated with OSA—regained this BP dip after OAm therapy. This effect was not found with placebo or CPAP treatment.

Yoshida et al.[32] examined 161 patients in a non-controlled study. These patients were treated for 2 months with OAm. Their BP values before and after treatment were compared. SBP (−4.5 ± 7.4 mm Hg), DBP (−3.0 ± 6.3 mm Hg), and MAP (−3.7 ± 6.1 mm Hg) all decreased significantly during OAm therapy. MAP response significantly correlated with baseline BP and AHI reduction. These results emphasize the importance of effective OSA therapy in the treatment of high BP.

Otsuka et al.[33] studied 11 patients diagnosed with OSA. In this non-controlled study, all patients were treated with OAm, and their BP values were measured before and after titration up to two-thirds of maximum mandibular protrusion. The mean time between these 2 measurements was 5.2 months. They found significant changes in 20-h BP values after OAm therapy: 20-h DBP decreased from 79.5 ± 5.5 to 74.6 ± 6.0 mm Hg, and 20-h MAP decreased from 95.9 ± 5.4 to 91.2 ± 5.9 mm Hg. The 20-h SBP did not change significantly. BP values also changed significantly during sleep: SBP decreased from 118.4 ± 10.0 to 113.7 ± 9.1 mm Hg, DBP decreased from 71.6 ± 8.0 to 67.2 ± 7.9 mm Hg, and MAP decreased from 88.4 ± 8.0 to 83.9 ± 7.5 mm Hg. In this study, there also were decreases in early morning BP values, although these daytime changes were not significant.

Andrén et al.[34] examined 29 patients diagnosed with OSA in a non-controlled study. BP was measured before OAm treatment, after 3 months of treatment and after 3 years of treatment. SBP showed a decrease from 154.9 ± 20.2 before treatment to 140.6 ± 15.9 after 3 months and 139.4 ± 17.5 mm Hg after 3 years of OAm use. DBP decreased from 88.4 ± 10.1 before treatment to 79.8 ± 9.9 after 3 months and 78.1 ± 8.9 after 3 years of OAm use. The changes after 3 months and 3 years of therapy were significantly different from baseline.

Andrén et al.[35] examined 70 patients diagnosed with OSA and systemic hypertension in an RCT. The study group was treated for 3 months with an OAm with mandibular protrusion and the control group with an OAm without mandibular protrusion. The 24-h BP was measured before and after treatment. BP values of the study group decreased slightly but not significantly in comparison to the control group. The greatest change was seen in the 24-h SBP, with a mean reduction of 1.8 mm Hg in comparison to the control group. The BP changes were higher after exclusion of patients with normal baseline BP and also after exclusion of patients with AHI ≤ 15/h.

In the study by Lam et al.,[36] 101 patients were randomized in 3 groups: an OAm group, a CPAP group and a group in which only conservative measures were taken. All patients underwent a full polysomnographic examination before and after intervention, which was maintained for 10 weeks in each group. CPAP as well as OAm decreased early morning DBP significantly in comparison to baseline values, but there was no obvious difference between these 2 groups.

Phillips et al.[25] studied the BP values of 102 OSA patients. Patients were divided into 2 equal groups and treated for 1 month with either OAm or CPAP. Thereafter, a 2-week washout period was scheduled and then patients were treated for 1 month with the other therapy. The authors found a reduction in MAP after 1 month of treatment for both therapies, but there was no significant difference between them. There was no significant reduction in mean BP after 1 month of therapy. These similar results were explained by the greater efficacy of CPAP and the greater compliance with OAm therapy.

A summary of OAm treatment studies assessing BP parameters is given in Table 2. In 8 studies, BP of 590 OSA patients was monitored, with a mean BP decrease of 4.2 mm Hg during OAm therapy.

Endothelial Function

Although OSA patients may not show signs of cardiovascular disease, they do show early signs of atherosclerosis, such as endothelial dysfunction (ED), increase in intima thickness, increased carotis diameter and increased biomarkers of oxidative stress and inflammation.[8,37–41] These signs are significantly correlated with OSA severity.[37,39,40,42–46]

Itzhaki et al.[47] examined oxidative stress and EF after 3 months and after 1 year of OAm therapy in a controlled study. The study group consisted of 16 patients, 12 of whom completed the 1-year evaluation. The control group consisted of 9 patients, and the study also used a reference group of 10 patients without OSA (AHI ≤ 10/h). The reactive hyperemia peripheral arterial tonometry (RH-PAT) was measured and represents EF. This value improved from 1.77 ± 0.4 to 2.1 ± 0.4 after 3 months and to 2.0 ± 0.3 after 1 year of treatment. The results under OAm therapy did not differ significantly from those of the reference group. The thiobarbituric acid-reactive substance (TBARS) also was measured, representing the oxidative stress, and was

expressed in nanomole malondialdehyde per milliliter plasma. The TBARS value decreased from 18.8 ± 6.2 to 15.8 ± 3.9 after 3 months and remained at 15.5 ± 3.2 after 1 year. There was a correlation between the change in AHI, EF, and TBARS values.

Trzepizur et al.[48] examined the microvascular endothelial function (MVEF) in a study group of 12 patients and a control group of 9 patients (AHI < 15/h). MVEF was measured with laser Doppler flowmetry combined with acetylcholine (ACh) and sodium nitroprusside (SNP) iontophoresis. Cutaneous vascular conductance (CVC) was expressed in AU/mm Hg. CVC values were measured before OAm and CPAP treatment and were compared to control group values and posttreatment values. Baseline CVC did not differ between the control group and the study group before OAm treatment. CVC measurements with ACh showed a significantly higher CVC peak in the control group being 3.8 multiple of baseline conductance (MBC) in comparison to the study group for OAm treatment (2.3 MBC). Correlation analysis of the study and control groups showed that ACh-induced CVC peak was negatively correlated to AHI. CVC values of the study group increased in comparison to baseline after OAm treatment. There was a significant increase in ACh-induced CVC peak after OAm treatment. An increase in SNP-induced CVC peak was also found but was not significant.

Both studies show that EF is correlated to AHI, which emphasizes the importance of an optimal treatment of OSA patients.

Left Ventricle

In patients without cardiovascular diseases, OSA is associated with an elevated incidence of both diastolic and systolic dysfunction and left ventricular hypertrophy.

Hoekema et al.[49] examined the left ventricle (LV) function of 28 patients with mild to moderate OSA (AHI < 20/h) before and after 2–3 months of OAm (15 patients) or CPAP (13 patients) therapy. They evaluated LV function with echocardiography and measurements of the amino-terminal fragment of the pro-brain natriuretic peptide (NT-pro-BNP). The echocardiographic values after OAm treatment were not significantly different from the values before treatment. The concentration NP, which reflects left ventricular wall stress, decreased significantly after OAm treatment; this value increased after CPAP treatment. The changes in these values suggest improved cardiac function after OAm therapy.

Less research was performed in the domain of heart function, which can be monitored by echocardiography. Only 1 study compared the left ventricle function of 15 patients before and after 2–3 months of OAm therapy.

DISCUSSION

In this article we have performed a systematic review of the current evidence regarding the cardiovascular benefits of oral appliance therapy in obstructive sleep apnea patients. After online database research we analyzed 11 relevant articles.

This systematic review demonstrates that the evolution of BP after OAm therapy is studied in detail. BP decreased significantly during OAm therapy in 7 of 8 studies. The two studies comparing OAm with CPAP therapy showed no significant difference in BP results during both therapies. The results during OAm therapy were in the same order of magnitude as after CPAP therapy.[33,38] Phillips et al.[25] explained these similar results by the greater efficacy of CPAP and the greater compliance with OAm therapy.[32,37,38] A clear variability in treatment response rate between the 8 different studies, evaluating BP changes, can be seen in Table 2, with 34% as lowest and 75% as highest treatment response rate. This treatment response is not correlated with the baseline AHI and probably correlated with the decrease in BP. Thereby we assume that the benefit on the heart will be the greatest in optimal OSA treatment.

Less research was performed in the domain of heart function, which can be monitored by echocardiography. Only 1 study compared the left ventricle function of 15 patients before and after 2–3 months of OAm therapy,[49] more extensive research in this topic would be interesting. Both articles studying EF show that EF is correlated to AHI, which emphasizes the importance of an optimal treatment of OSA patients.

In this systematic review, only 3 of 11 studies evaluated their patients after a treatment period longer than 3 months as seen in Table 2. The washout period, which is the period without OSA therapy to eliminate the effects of previous therapy, was not always documented. There are no absolute recommendations on how long OSA therapy should be employed to achieve maximal beneficial cardiac effects or how quickly the beneficial cardiac effects are reversed after discontinuation of the treatment. Cardiac evaluation of patients, with a washout period of at least 3 months and an OAm treatment period of 6 months to 1 year may be worth studying.

Following current recommendations OAm therapy is prescribed for a select group of OSA patients with an AHI < 20/h or in patients for whom CPAP does not work or is not tolerated.[22] Given this recommendation, it would be interesting to study cardiovascular benefit in a patient group with an AHI ≥ 20/h or to make a subanalysis of this group. Secondly only 3 of 11 studies compared cardiovascular effects during OAm therapy with cardiovascular effects during CPAP therapy. Since the cardiovascular morbidity and mortality is an important feature in treating OSA, more evidence comparing these two different treatment modalities is necessary. Large scale studies have demonstrated that CPAP reduces the risk of fatal and non-fatal cardiovascular events in severe OSA.[50,51] In an observational study, Anandam et al.[52,53] examined the cardiovascular mortality in 570 patients with severe OSA treated with either CPAP or OAm. Untreated severe OSA was a strong predictor of cardiovascular death. Cardiovascular death rates of CPAP- and OAm-treated patients were similar (compared to each other) and both significantly lower than the untreated OSA patients. Although residual AHI for OAm-treated patients was significantly higher than CPAP-treated patients, there was no difference in cardiovascular death rate between the two groups. Some studies included in this review demonstrate that patients under OAm therapy report higher subjective compliance than CPAP therapy.[25,36] Objective measurement of compliance of OAm therapy has become feasible using an incorporated sensor in the OAm.[54] Consequently, compliance will become more accurately assessable and will become an important variable in the comparison between CPAP and OAm therapy outcome. Not only efficacy, but also compliance is an important factor in comparing different treatment modalities. The product of

efficacy and compliance can be formulated as the mean disease alleviation (MDA), as described by Vanderveken et al.[54] MDA can be a measure for the net effectiveness of a therapy. Even if CPAP is superior to OAm in reducing AHI, in terms of efficacy the MDA values of both CPAP and OAm therapy are in the same order of magnitude due to the higher compliance to OAm.

All this could be elucidated further by studying the cardiovascular benefits between 6 and 12 months of OAm therapy, including implementation of objective compliance measurement, as well as extensive cardiovascular follow-up of patients, and in patients with more severe OSA and in comparison with CPAP treatment.

CONCLUSION

The results of this concise review indicate that OAm therapy provides a beneficial effect on the cardiovascular comorbidity in OSA patients. Improvement in BP, EF, and left ventricular function are proven in several independent studies. The studies comparing OAm therapy to CPAP therapy even illustrate that the reduction in BP values after both therapies might be of the same order of magnitude. This finding contrasts with the current recommendations for OSA treatment in which OAm therapy is prescribed for a select group of OSA patients with an AHI < 20/h or in patients for whom CPAP does not work or is not tolerated and calls for further research in this field.[22]

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CASE REPORTS

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Oral Appliance Therapy: A Case of False Negatives

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A patient's subjective response to treatment is not necessarily indicative of objective response. This case illustrates treatment with an oral appliance in a CPAP intolerant patient who reported a positive subjective response. Follow-up sleep testing revealed an increase in the severity of sleep disordered breathing related to an emerging medical condition.

Keywords: oral appliances

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The objectives of oral appliance (OA) therapy for obstructive sleep apnea (OSA) include the relief of subjective symptoms such as snoring and sleepiness as well as improvement in objective disease measurements such as apnea-hypopnea index (AHI) and nocturnal oxygen saturation.[1] Subjective symptom relief, while satisfying for patients, does not necessarily indicate improvement in objective disease parameters.[2] Patients may be reluctant to undergo follow-up testing due to convenience and financial factors but objective follow-up testing is an essential component of quality OA therapy. This case illustrates the necessity of follow-up sleep testing.

REPORT OF CASE

A 51-year-old male presented for OA consultation. His chief concern was loud snoring that kept his wife awake. He had two polysomnographs (PSGs); a full night four years previously and a split night study ten months previously. The first PSG showed an AHI of 29 events/hour and the split night an AHI of 13 events/hour. During the split night study his oxygen saturation did not fall below 90%. Continuous positive airway pressure (CPAP) therapy at 10 centimeters of water effectively treated his OSA.

At the time of OA consultation the patient had become intolerant of CPAP therapy. He reported that he routinely removed the mask after only one or two hours use. He did not take the CPAP during frequent work-related travel.

His BMI was 35.7 kg/m^2, neck circumference 42 cm, and blood pressure 110/81. He had been diagnosed with atrial fibrillation and had a pacemaker.

He was fitted with an OA and reported immediate control of snoring. Over the following eight weeks, he was seen three times to make minor adjustments to his OA. He and his wife reported ongoing successful results during this time.

A follow-up PSG was scheduled to assess his progress with his OA in place. He elected to postpone his follow-up study for 10 months.

The follow-up PSG results were unexpected. His sleep efficiency was good at 89% with a sleep onset latency of 11 minutes. Breathing during sleep was characterized by repetitive hypopneas. These occurred with a waxing and waning pattern most consistent with Cheyne-Stokes breathing (i.e., "breathing pattern characterized by regular 'crescendo-decrescendo' fluctuations in respiratory rate and tidal volume").[3] There were obstructive apneas and some central apneas seen near the end of the study (although these were infrequent). His oxygen saturation during rapid eye movement (REM) was observed to fall as low as 71%, despite the presence of his OA and in contrast to PSGs prior to treatment. During the PSG incremental adjustments were made to the patient's OA increasing protrusion by 1.25 mm. The overall AHI during this study was 81 per hour with a range of 50–90 per hour depending on the level of OA adjustment. Contrary to what was expected, disturbed breathing persisted despite OA therapy.

The marked increase in sleep disordered breathing made this sleep study distinctly different from those observed previously. The pattern was more consistent with Cheyne-Stokes respiration than obstructive or central sleep apnea. The patient had a history of cardiomyopathy being followed by his cardiologist, and had gained approximately 30 pounds since the previous sleep studies.

Treatment was begun directed at congestive heart failure. The medical sleep specialist recommended continued use of the OA and placed the patient on nightly oxygen therapy. He further recommended immediate follow-up with his cardiologist. The sleep specialist reported that it was not obstructive apnea, but documentation of heart failure occurring during the recent PSG. The patient's cardiologist agreed with this assessment and began treatment to improve his heart function.

DISCUSSION

This case highlights the importance of working as a part of a medical team, which includes close interactions with the patient's physicians. The patient's positive subjective OA therapy results could have lulled me into a false sense of security; however, the absence of anticipated findings through follow-up studies over time indicated a larger health problem. Regular communication with the patients' physicians enabled diagnosis and treatment of a significant new health problem.

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CASE REPORTS

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Effect of Beer Ingestion on a Patient with OSA while Using Oral Appliance Therapy

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It has been shown that alcohol prior to sleep in obstructive sleep apnea (OSA) patients exacerbates the frequency and severity of hypoxic events and shortens the time from sleep onset to the most severe event. Ingestion of a moderate amount of alcohol does not decrease the efficacy of continuous positive airway pressure in treatment of OSA. Oral appliance therapy has been gaining in use as an effective option in the treatment of OSA. The question arises as to whether oral appliance use will protect the patient from the deleterious effects of alcohol ingestion on respiration. In this case report, the OSA patient had ingested alcohol approximately 3 hours before bedtime and then used an overnight oximeter while wearing her oral appliance. She showed significant oxyhemoglobin desaturations soon after retiring for the night. The period of instability lasted for approximately 1.5 hours and was not repeated for the remainder of the night.

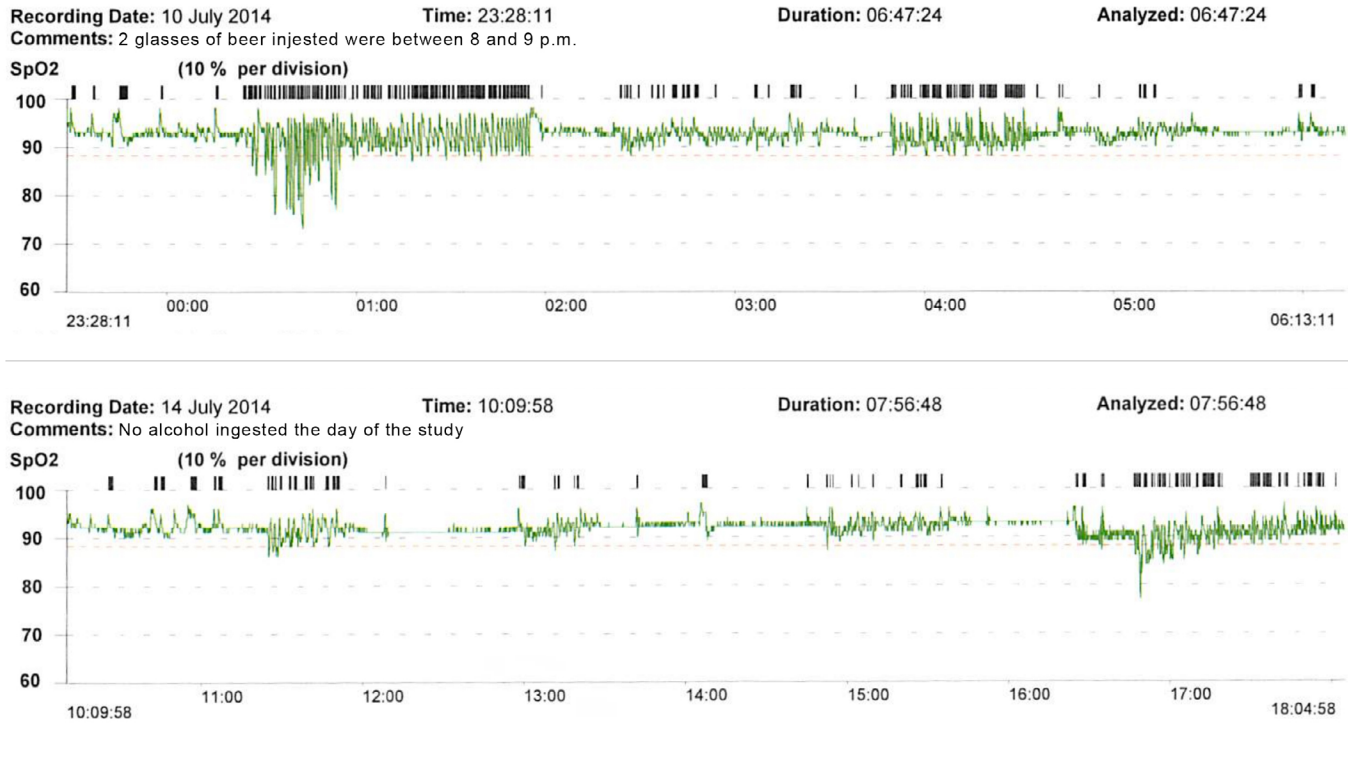
Keywords: oral appliance therapy, alcohol, oxygen saturation

Citation: Katz S. Effect of beer ingestion on a patient with OSA while using oral appliance therapy. *Journal of Dental Sleep Medicine* 2015;2(1):17–18.

REPORT OF CASE

A 63-year-old female was referred for complaints of snoring, teeth grinding, acting out dreams, and a morning dry throat. She had a history of dyslipidemia, hypertension, hypothyroidism, and gastroesophageal reflux disease. Her medications included levothyroxine, triamterene, and omeprazole. Her body mass index was 33.2, and her Epworth Sleepiness Scale score was 5/24. The patient had a complete evaluation and polysomnogram which showed respiratory disturbance index (RDI) 22.8, apnea-hypopnea index 15.8, oxyhemoglobin nadir 71%, hypoxic burden of 3.2, supine RDI 22.8, and REM sleep RDI 88.7. Based on these findings, she was diagnosed with moderate obstructive sleep apnea (OSA). The severity of the sleep disordered breathing was significantly worse during REM. She was prescribed continuous positive airway pressure (CPAP) but was told that she would need a full face mask, and felt she would not be able to tolerate it.

Figure 1



The patient was referred for an evaluation for oral appliance therapy (OAT) for the management of her OSA. The oral evaluation revealed that her tongue level was high, she had a Mallampati III soft palate classification, and her tonsils were grade 1. No other significant abnormalities were noted. These findings were reviewed with the patient and it was agreed that she was a good candidate for OAT. Impressions and records were made, and an oral appliance was fitted and delivered one month later. The patient began using the oral appliance. Appropriate adjustments were made to resolve the patient's symptoms of snoring, morning headaches, and dry throat. Overnight oximetry was performed with the oral appliance in place. This showed significant oxygen desaturations starting one hour into the study, lasting 1.5 hours (Figure 1). It was noted that the patient had 2 beers between 20:00 and 21:00 on the night of the study. A subsequent study, with no alcohol, was done 1 month later. No such events occurred. The patient is feeling more alert during the day and is not snoring. She has a home sleep study scheduled to validate the efficacy of the oral appliance's current position.

DISCUSSION

Because these observations were not made by polysomnography, it is not possible to draw definitive conclusions regarding the patients' awake and sleep time and her sleep stages. However, the most significant desaturations appear to be at the beginning of the night, within the time frame described by Scrima et al., when he described the increase of severity of OSA after alcohol ingestion: "The most severe hypoxic events occurred within 80–160 minutes after sleep onset."[1]

Nigri et al.[2] and Teschler et al.[3] reported that "CPAP can offset the adverse effects of alcohol on the upper airway." This case study suggested that oral appliance therapy may not be able to offer that same protection. Krol et al. demonstrated that the "neural mechanisms underlying the respiratory activity of the genioglossus are more susceptible to depression by alcohol than those serving the muscles of the ventilatory pump."[4]

Further research is warranted to confirm and possibly expand on this observation. If oral appliances are shown to inadequately protect the airway when under the influence of alcohol, patients must be counseled to this effect; CPAP may be a better option for patients that choose to use alcohol regularly.

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CASE REPORTS

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Oral Appliance Impact on Complex Sleep Apnea: A Case Report

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Complex sleep apnea is most often treated with adaptive servo-ventilation (ASV), a compensatory form of positive airway pressure therapy. What happens when ASV is not successful or tolerated? Is there a role for oral appliance therapy in the management of complex sleep apnea? A case is presented that explores these issues in a complex sleep apnea patient with comorbidities.

Keywords: complex sleep apnea, oral appliance, mandibular advancement, sleep apnea, oral splint

Citation: Scherr SC. Oral appliance impact on complex sleep apnea: a case report. *Journal of Dental Sleep Medicine* 2015;2(1):19–20.

There is robust evidence supporting the use of mandibular advancement oral appliances in the treatment of obstructive sleep apnea for those patients preferring an oral appliance over continuous positive airway pressure (CPAP).[1] Some obstructive sleep apnea patients with no daytime hypercapnia are known to develop central apnea events during non-REM sleep, or a Cheyne-Stokes respiration pattern, with initial CPAP titration after resolution of airway obstruction events. This phenomenon has been termed complex sleep apnea. Some central events with initial CPAP titration may be transient and resolve with continued CPAP use. However, complex sleep apnea patients often have poor initial experiences with CPAP and become noncompliant. The cause of complex sleep apnea is currently unknown, although it may be associated with ventilatory control system instability (high loop gain). Though most commonly associated with the introduction of CPAP,[2] there are also case reports of treatment-related development of central sleep apnea following tracheostomy, maxillomandibular advancement surgery, and oral appliance therapy.[3–6]

The prevalence of complex sleep apnea has been reported in a range of 1% to 20%.[7,8] Risk factors may include congestive heart failure,[9] though most patients exhibiting complex sleep apnea are not readily identifiable in advance. Successful treatment requires correction of both airway obstruction and ventilatory dysregulation. First-line treatment is adaptive servo-ventilation (ASV), a compensatory form of bilevel PAP that is less prone to precipitating central apneas. Not all patients are responsive to ASV, and alternative avenues for treatment are needed. There are few reports in the literature regarding the use of mandibular advancement oral appliances in the treatment of complex sleep apnea. The following is a case involving just such a treatment.

REPORT OF CASE

A 77-year-old Caucasian male was referred for consideration of oral appliance therapy by a board-certified sleep physician/pulmonologist. His body mass index was 30 kg/m^2 and his neck size was 16 inches at initial presentation. He expressed concerns about snoring, difficulty maintaining sleep, nightmares, night thrashing, and feeling tired all the time. His Epworth Sleepiness Scale score was 20. The relevant medical history included high blood pressure, Barrett's esophagitis, arthritis, and recurrent liposarcoma. Medications included metoprolol, valsartan, amlodipine, duloxetine, omeprazole, allopurinol, fentanyl transdermal patch, and supplemental oxygen. The patient had completed a baseline split polysomnogram at an American Academy of Sleep Medicine accredited sleep center and received a diagnosis of severe obstructive sleep apnea with an apnea hypoxia index (AHI) of 69.8/h and moderate oxyhemoglobin desaturations to a nadir of 80% and T90 of 38.8%. During the CPAP titration portion of the study, central apneas were exchanged for obstructive apneas and a diagnosis was made of severe complex sleep apnea. He subsequently completed a bilevel PAP titration polysomnogram, but central apneas developed with increasing pressures and bilevel PAP treatment was not successful. He next received an ASV titration polysomnogram, but severe mask leaks were noted at all ASV pressures despite a trial of multiple interfaces and use of a chin strap. Treatment with ASV was not successful. It was at this point that the patient was referred for consideration of oral appliance therapy.

The patient had received routine dental work including a number of stable fillings and crowns. His overbite was 9 mm and overjet was 4 mm. There were no diastemas or abfractions, though moderate occlusal wear was noted. He exhibited moderate periodontitis including mild-moderate mobility of the incisor teeth. His skeletal profile was retrognathic (Class II), and occlusal classification was Class II Division 2 on the right and Class I on the left. The oropharynx was characterized by a very low-arch palate (Mallampati IV). He had a large tongue with scalloping of the lateral borders. The temporomandibular joints, muscles of mastication, and mandibular range of motion were within normal limits. He was deemed a suitable candidate for treatment with a mandibular advancement oral appliance and was encouraged to improve his oral hygiene as well as work with his general dentist to improve his periodontal health. The selected oral appliance was a TAP 3 because of the patient's compromised manual dexterity, as gradual home mandibular advancement adjustments are easily accomplished by the patient with this design. In addition, the TAP 3 design restricts the degree of mandibular opening, reducing the risk of further airway obstruction during wide opening.

The patient made steady progress with gradual home mandibular advancement calibration of the oral appliance. However, he reached a point where placement of the oral appliance became a challenge for him. He was instructed to reverse several appliance adjustments prior to seating the device and then readjust the appliance to the treatment position after placement. He was

unable to comply due to difficulty placing the adjustment key into the device after placement. The adjustment key was then permanently attached to the oral appliance with cyanoacrylate adhesive. This allowed the patient to comfortably follow through with the recommended protocol and the oral appliance calibration continued until reaching the patient's comfortable limit. His wife reported snoring was well controlled at this setting. However, the patient described only mild improvement in sleep continuity and significant ongoing daytime sleepiness. The patient consulted with board-certified sleep physicians at several major area accredited sleep centers before presenting for a split-night polysomnogram, with the first half of the night dedicated to ASV titration and the balance for TAP 3 oral appliance confirmation/calibration.

With ASV, the patient's AHI was 60.8 disordered breathing events (DBE) per hour which included a mixture of central and obstructive events. His blood oxygen desaturation nadir was 83%. Once again, ASV was unsuccessful. During the oral appliance portion of the test, the AHI was 6.9/h at the most optimal oral appliance setting (1 mm of additional mandibular advancement), and blood oxygen desaturation nadir was 96%. The interpreting physician made a recommendation of home therapy with use of the oral appliance and supplemental oxygen. The patient found his sleep continuity was improved. However, he continued to complain of nightmares, memory loss, and sleeping most of the day. Several months later, he completed a new oral appliance confirmation/calibration polysomnography (PSG) which found an AHI of 45/h and blood oxygen desaturation nadir of 87%. It was thought that a combination of oral appliance therapy with ASV might be an option, so an additional ASV titration PSG in the presence of the oral appliance was completed. The test results included an AHI of 58.8/h and blood oxygen desaturation nadir of 89%. There were minimal central events this time and the desaturation nadir was much improved compared to the baseline polysomnogram.

At the patient's next visit, he seemed much more alert and upbeat and said he was feeling better than he had in a long time. He described sleeping fairly well with the combination of his oral appliance simultaneous with ASV, and was feeling much more vigilant since his dose of fentanyl was recently reduced by a significant amount.

DISCUSSION

The clinical management of a challenging patient diagnosed with complex obstructive sleep apnea while being managed for chronic pain related to multiple surgeries for recurrent liposarcoma is described. This patient was resistant to established therapy for the complex sleep apnea condition. Oral appliance therapy was initially effective at improving polysomnographic variables when combined with supplemental oxygen. However, significant daytime symptoms remained and subsequent overnight sleep studies found treatment with oral appliance therapy/O_2 to be subtherapeutic. Combination oral appliance therapy/ASV was considered to be the most stable treatment for this patient. Yet his sleep-related symptoms improved only after his dose of fentanyl transdermal was significantly reduced. Fentanyl is an opioid analgesic prescribed for the management of chronic pain.[10] Known side effects include difficulty sleeping, daytime drowsiness, and respiratory depression. Side effects may be more pronounced in the elderly. It is not known if fentanyl may have contributed to objective signs of sleep disordered breathing, since no further sleep studies were obtained after the dose was reduced.

The care of this patient demonstrates a novel multidisciplinary approach to management of multifactorial symptomatology. This case demonstrates that oral appliance therapy has the potential to be both a precipitating factor in the development of complex sleep apnea and part of the solution. In addition, residual sleep-related symptoms invite further scrutiny of the patient's medical history and pharmacology. Comprehensive and coordinated care best enhances quality of life outcomes.

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SPECIAL ARTICLES

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Health Advisories

National Healthy Sleep Awareness Project

NATIONAL HEALTHY SLEEP AWARENESS PROJECT

Dental sleep medicine clinicians have an obligation to educate the public regarding healthy sleep practices in addition to providing oral appliance therapy. Healthy sleep practices enhance treatment outcomes as well as general health and public safety.[1,2]

The first health advisories issued by the National Healthy Sleep Awareness Project (NHSAP) follow below. The health advisories on childhood sleep duration, school start times for teens, childhood insomnia and drowsy driving are for public education in any clinical practice.

The primary goals of the NHSAP are to: expand sleep health promotion and sleep prevention awareness; develop partnerships and collaborations to improve education and awareness about sleep hygiene; improve provider knowledge and skills about sleep health promotion; recommend a sleep health data collection model; and identify and disseminate sleep health-related policies.[3]

NHSAP is a 5 year cooperative project awarded to the American Academy of Sleep Medicine by the Centers for Disease Control and Prevention. The American Academy of Dental Sleep Medicine is a part of the collaboration that is focusing on the Sleep Health Objectives of Healthy People 2020. These objectives are to increase the proportion of symptomatic people who seek evaluation for obstructive sleep apnea, reduce the rate of vehicular crashes due to drowsy driving and increase the proportion of adults and teens who get sufficient sleep.

Other advisories will follow that are the results of reviews of scientific literature and expert consensus by the multiple partners in the NHSAP. Please use them to educate the public in promoting healthy sleep.

HEALTH ADVISORIES

The following health advisories have been developed by the American Academy of Sleep Medicine and endorsed by the Sleep Research Society and other partners in the National Healthy Sleep Awareness Project, a collaborative project funded by the Centers for Disease Control and Prevention. Learn more at www.projecthealthysleep.org.

Child Sleep Duration

The nightly duration of healthy sleep needed by children varies by age and individual biology. In general the American Academy of Sleep Medicine recommends that school-aged children and pre-teens get about 10 to 11 hours of sleep each night for optimal health, daytime alertness and school performance.

Teen Sleep Duration and School Start Times

The American Academy of Sleep Medicine recommends that adolescents get a little more than nine hours of nightly sleep for optimal health and daytime alertness during the critical transition from childhood to adulthood. In puberty a natural shift occurs in the timing of the body's internal "circadian" clock, causing most teens to have a biological preference for a late-night bedtime. It is important that parents and local schoolboards work together to implement high school start times that allow teens to get the healthy sleep they need to meet their full potential.

Figure 1—National Healthy Sleep Awareness Project logo.

The National Healthy Sleep Awareness Project promotes improved sleep health in the U.S. It was established by a cooperative agreement between the Centers for Disease Control and Prevention and the American Academy of Sleep Medicine.

Insomnia in Children

Behavioral interventions should be the first treatment option for healthy children who struggle with bedtime resistance or nighttime awakenings. For those who fail to respond, or for children with more complex medical problems, evaluation by a sleep physician is recommended.

Drowsy Driving

Drowsy driving is common on U.S. roads and represents a pervasive threat to public health and transportation safety. Drowsiness is similar to alcohol in the way that it compromises driving ability by reducing alertness and attentiveness, delaying reaction times, and hindering decision-making skills. The severe driving impairment caused by drowsiness increases the risk of motor-vehicle accidents, injuries and fatalities.

The American Academy of Sleep Medicine (AASM) recommends that states mandate instruction in drowsy driving education as a requirement for driver's education programs, provide comprehensive information about drowsy driving in state curricula and driver's manuals, and include questions related to drowsy driving on driver's license exams. The AASM also advises transportation companies to promote public safety by adhering to hours-of-service regulations, scheduling work shifts based on sleep need and circadian timing, implementing an evidence-based fatigue management system, and screening drivers for sleep disorders such as obstructive sleep apnea.

The AASM encourages every driver to take responsibility for staying "Awake at the Wheel" by making it a daily priority to get sufficient sleep, refusing to drive when sleep-deprived, recognizing the signs of drowsiness, and pulling off the road to a safe location when sleepy.

CITATION

National Healthy Sleep Awareness Project. Health advisories. *Journal of Dental Sleep Medicine* 2015;2(1):21–22.

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SPECIAL ARTICLES

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The Dentist-Physician Partnership

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Collaboration among dentists and dental specialists is not new. Collaboration among physicians and physician specialists is not new. What is novel is the need for a different kind of partnership—that of the dentist and the physician—for the sake of patients suffering from obstructive sleep apnea (OSA). Some have entered into this novel partnership with ease and grace; for others it has been more of a struggle. The two fields have a host of differences: education and training, terminology, practice regulations, types of insurance and billing, as well as documentation styles. Dental sleep medicine requires the dentist to leave part of the dental world behind and embrace the medical world with regard to communication, documentation, and insurance/billing. This can be a challenge, but the satisfaction of contributing to the health and happiness of sleep apnea patients is well worth the effort.

Exposed to dental sleep medicine since the time of my fellowship training, I have long advocated for the use of oral appliance therapy (OAT) in appropriately selected patients. We know that the medical and surgical treatment options for OSA are not embraced or suitable for all patients. It has been a huge benefit to my patients for me to be able to offer OAT. At the time of this publication, there are only 197 board certified dental sleep medicine practitioners in the entire United States (and 17 in Canada). This number is simply insufficient to manage the expansive need. I would strongly encourage interested dentists to study sleep medicine and embrace board certification.

So…maybe you are new to the idea of practicing dental sleep medicine or maybe you have taken a course or even made an appliance for yourself as a trial. What is next? Although you did not have any business courses in dental school, it might be time to develop a business plan to see if including dental sleep medicine within your practice is a viable option. In addition to a budget, this would likely include some sort of "SWOT" analysis of your strengths (S), weaknesses (W), opportunities (O), and threats (T) with regard to the new endeavor. Only you will know the SWOT components as they relate to your particular practice. One important factor is whether your office is prepared to manage medical insurance in this ever-changing landscape that is health care. As for the external environment, in addition to evaluating population statistics in your geographic area and other site-specific information, you will want to size up the competition. Who are the interested dentists in your area? Have you checked the American Academy of Dental Sleep Medicine (AADSM) website (www.aadsm.org/findadentist.aspx)? Are your competitors board certified in dental sleep medicine? What kind of training have they had? How long have they been practicing dental sleep medicine? How many different appliances do they use? Do they have relationships with physicians/centers/laboratories? Read the websites of your potential competitors to get an idea.

In addition, you will want to know who your physician colleagues might be. The Internet is your friend here again. Do your homework. Find out how many board certified sleep medicine physicians there are in your area. The website sleepeducation.com is a good starting place. You can search for American Academy of Sleep Medicine (AASM) accredited sleep centers in your area by full address, zip code, or city/state. Most of the listings include the website of each individual sleep center. The websites can help with basic information about the sleep centers and the doctors who are affiliated with them. You will want to know how many sleep centers/laboratories are in your area. How many beds does each center have? Can you get a sense of the volume of home sleep apnea testing (HSAT) they perform? How many and who are the doctors who work with them? Are there dentists on the team already? Although it would make life in reconnaissance easier, you may not be able to discern whether or not the physician has an association with a dental sleep medicine practitioner. Note that if the physician is affiliated with a large hospital network or university, there may be a dentist working within that network (or even in the sleep medicine clinic) already.

It is also important to remember one thing when researching the physicians: sleep medicine is an amalgamated field of medicine (do I get dental points for that one?). There are pulmonologists, neurologists, internists, psychiatrists, otolaryngologists, anesthesiologists, pediatricians, and others who work in the field of sleep medicine. Some have elected to practice sleep medicine full time. Others may continue to practice in their primary field as well. Additional Internet research may be required at this point. Be aware that many of the transparency websites out there pull data from various sources that may or may not be correct or up to date. Reading patient reviews can be helpful as well. Unfortunately, there is not a "master list" of all board certified sleep medicine physicians available on the World Wide Web. Prior to 2007, the American Board of Sleep Medicine (ABSM) was responsible for administering the Sleep Medicine Specialty Examination. The list of physicians who achieved board certification through the ABSM is available at the time of this printing at www.absm.org/sleepmedicinespecialty.aspx. In 2007, several member boards of the American Board of Medical Specialties began administering subspecialty certification examinations in sleep medicine. The ABSM web address above also has a listing of these member boards (and their websites) which includes the following: American Board of Anesthesiology, American Board of Family Medicine, American Board of Internal Medicine, American Board of Pediatrics, American Board of Psychiatry and Neurology, and the American Board of Otolaryngology. If a physician has achieved board certification through one of the above boards, that information may be available on that particular board's website. For example, the American Board of Anesthesiology has a "Verify a Physician's Certification" on the home page for the organization.

What if you have now decided to take the plunge? You have enough information to put more than a toe in the proverbial

water. How in the world are you going to establish contact with your would-be partners—the physicians? Reaching out may feel foreign and very uncomfortable. You could approach it one of several ways. The options include writing a letter of interest, knowing that you run the risk that it might never see the eyes of the physician in the modern medical office setting, instead ending up in the file that is circular. You could call to speak with the physician or set up a meeting to do so. Either way, once you get your foot in the door, it is time to express your interest to be involved in the care of the OSA patient. Let the physician know where you are in the process. Are you board certified in dental sleep medicine? As you know, we can look it up. Are you involved with the AADSM? Is your office accredited by the AADSM? How many appliances have you fabricated? How many different appliances do you use? What have you done in the name of education? Have you tracked your patients? How do you maintain records? Do you send the patients back to the physician for repeat study to determine efficacy and to potentially calibrate the positioning? What is your success rate and how do you define success?

If you have gotten this far, then try to get an understanding of the physician's familiarity and current utilization of OAT. Make sure you are well aware of the current practice parameters and standards of practice regarding OAT. A literature search and review would be optimal. Know your stuff and bring everything that you might need (but you may or may not use in the discussion with the physician). Firstly, you may want to discuss the efficacy data of OAT from the literature. Be mindful however that in determining the effectiveness of positive airway pressure therapy (PAP), the efficacy is measured in the device's ability to suppress the apnea-hypopnea index (AHI) below 5 events per hour of sleep. Until recently, many efficacy studies in dental sleep medicine used alternative markers of success such as improvement in patient symptoms, reduction in AHI by a certain percentage, suppression of the AHI below 10 events per hour, or the like. Be sure to compare apples to apples in this scenario. Secondly, once you have discussed the efficacy of OAT in mild, moderate, and severe OSA, you will want to discuss that last group in more detail. There is little room for debate that positive airway pressure is the first line treatment for the more severe cases of OSA. However, if a patient is intolerant to positive airway pressure, other options must be explored. Options include weight loss, surgery, oral pressure therapy, expiratory positive airway pressure (EPAP) via nasal valve, and hypoglossal nerve stimulation. Although the goal is full control of the OSA, some patients may be referred to you for "salvage therapy." This means that the expectation may not be full control of the OSA, but instead as much control as possible in an effort to reduce the negative impact of the OSA on the health and quality of life. You will also want to have some familiarity regarding the use of combination therapy (OAT and PAP at the same time). In addition to the above, be prepared to talk about the ideal patient characteristics for OAT, the contraindications (absolute and relative ones) to OAT, the side effects and how they are managed, medical billing and the concept of bundled services, protocols for follow-up, medical insurance coverage, and the general cost.

If the conversation is turning into a partnership, there are other components to address. How will this all work? You will want to know how to refer a patient for evaluation (generally to rule out OSA) to the physician. What paperwork is needed for the referral? What insurance plans are accepted? Are both in-laboratory polysomnography and HSAT offered? In some cases, you will not be referring to an individual physician but instead to a sleep center which then assigns the referral to one of their physicians by center protocol. You will want to understand the proper channels going forward. Also, you must be mindful of the regulatory aspects of the Health Insurance Portability and Accountability Act (HIPAA), including for example that referrals emailed on the World Wide Web are not considered "protected" information.

On the flipside, you may want to discuss how the physician is going to make a referral to you for a patient with known OSA. In addition to a referral, the physician will send you a prescription for the OAT, the medical indication, and medical necessity for the device. This may be in the form of preprinted prescription pads, a web-based document, a simple Word document or form, or may be encompassed in the electronic health record. There are certain elements that the prescription must contain and one should be aware of federal and state laws with regards to such. Similar to a prescription for PAP durable medical equipment (DME) to a DME company, additional information such as the pertinent sleep study report, clinic notes, insurance information, and full demographics is required.

After a referral is made in either direction, it is important to know what is to happen next. If you refer a patient to a physician for an evaluation, it is standard practice that the physician send a consult note back to you. This note generally consists of a chief complaint/reason for visit, a history of present illness and sleep history, past medical and surgical history, current medications, allergies, social history, family history, and a physical examination including vital signs. Lastly, there is a section of the documentation called "Assessment and Plan." In this section, the physician provides a brief summary and assessment of the case as well as the plan for work-up and/or treatment. It will be important for you to familiarize yourself with the typical medical documentation. Your partners in medicine will likely have the expectation that you document in the medical format (you are treating a medical disorder after all). Not only will this be important for your initial visit with a patient, but on follow-up patient visits as well. One common approach to the documentation of a follow-up patient visit is that of a "SOAP" note. The acronym stands for the four basic elements of the note itself: subjective, objective, assessment, and plan. The subjective component is the patient report with regards to the problem at hand. This might include details such as how they are tolerating treatment, current sleep quality, daytime symptoms, side effects of treatment, and the like. The objective component is the clinician's observations such as the patient's vital signs, physical examination findings, and other objective information (adherence to therapy data, recent laboratory tests, x-ray results, etc). The assessment is just that—the clinician's assessment of the current situation. The plan is the discussion or outline of the next steps in the treatment of the patient. Whether it is an initial evaluation or follow-up evaluation, it is general practice that the clinician send a copy of that documentation to other team members involved in the care of the patient. This documentation is your lifeline of communication.

Now that we have the documentation out of the way, I am going to talk about the elephant in the room—sleep testing—and more specifically HSAT. HSAT, in my humble opinion, is the greatest threat to the physician-dentist relationship. Confusion about the "who" is what leads to the discontent (who orders, who provides, who interprets, who bills, who explains, etc). As a point of education, according to the Current Procedural Terminology (CPT) Codebook, a sleep test is defined as "the continuous simultaneous monitoring of physiological parameters during sleep." This includes the most simple home pulse oximetry study that determines oxygen levels during sleep to the most complicated in-laboratory polysomnography study with full EEG. With regards to sleep testing, including that done in the home, the position both of the AASM and the AADSM is that OSA is a medical disease that must be diagnosed by a physician. Furthermore, because snoring is a common symptom of OSA, a diagnosis of primary snoring can be made only by a physician who has ruled out OSA. The best treatment outcomes will result from a like-minded partnership where the physician diagnoses OSA, and in the appropriately chosen patient, requests that a qualified dentist provide an oral appliance if it is suitable from a dental point of view. Two clinicians coming to the same mutual decision on behalf of the OSA patient represents the true collaborative spirit. Subsequently, the dental follow-up should be by the dentist, and the sleep apnea and sleep medicine follow-up should be by the physician. The latter is paramount when considering that it is not uncommon for OSA patients to have ongoing sleep disordered breathing despite symptom improvement with OAT. In this case, follow-up testing may indicate that further advancement of the mandible is necessary for full control of the OSA. When advancement is not possible or not tolerated, inadequate control of sleep apnea may indicate that other treatment options should be explored and potentially prescribed. Lastly, even with adequate control of OSA, the sleep apnea patient may also require ongoing sleep medicine follow-up for residual hypersomnia and/or comorbid sleep disorders such as insomnia, restless legs syndrome, and the like.

It is not just the national organizations that have drawn the line in the sand. Although the debate rages on in some states, in others, state dental boards have weighed in regarding the use of sleep tests by dentists. For example, the New Jersey State Board of Dentistry responded to inquiries posed by the New Jersey Dental Association as to whether it is within the scope of practice of a New Jersey licensed dentist to perform a sleep study, interpret a sleep study, and/or dispense the machinery to perform a sleep study. The Board ruled "no" to all three, but did note that the dentist is permitted to work with other medical professionals to treat sleep apnea and be within their scope of practice (New Jersey State Board of Dentistry, Public Session Minutes 4/1/2009). With this and other rulings in mind, you are advised to contact the dental licensing board in your state to verify the approved scope of practice. For each of your patients, you are also encouraged to review the specific insurance policy requirements related to who may order, perform, and read sleep tests.

We each have an incredibly valuable role to play in the treatment of the OSA patient. The sleep medicine physician should perform a complete sleep medicine evaluation and, if medically indicated, perform the appropriate diagnostic testing for OSA. In turn, it is the dentist who is trained in dental sleep medicine, who should provide safe, effective, and well-tolerated oral appliance treatment. Dual ownership is the avenue to success. Coordination, communication, and collaboration between dental sleep medicine practitioners and sleep medicine physicians ensures that we achieve the ultimate goal—the highest quality of care for patients with OSA.

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NEWS AND UPDATES

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AADSM 2015 Educational Calendar of Events

AADSM Staff

AADSM National Office, Darien, IL

February 3–April 14
Spring Study Club Program (live, web-based seminars)

February 28–March 1
Essentials of Dental Sleep Medicine Course
New Orleans, LA – Hilton New Orleans Riverside

March 21
Practical Demonstration Course
Darien, IL – AADSM National Office

March 21–March 22
Board Review Course
Chicago, IL – Hilton O'Hare

June 4–June 6
24th Annual Meeting
Seattle, WA – Washington State Convention Center