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Quality

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Quality of care, quality assurance, quality measures: these are terms used frequently in discussions of health care. How is quality determined in dental sleep medicine? Quality in research, in continuing professional education and in clinical practice are required to deliver "the right care for every person, every time."[1]

The aim of the Journal of Dental Sleep Medicine is to provide good quality information on all aspects of dental sleep medicine in the broadest sense. Peer-review, while not without limitations, is the best process available to provided good quality information. Our reviewers (unpaid volunteers) check submissions for many things including appropriate methods, analyses, reasonability of conclusions and the use of current references. You as the reader have this assurance of quality in the journal's offerings. Scientific and professional debate is useful and necessary so not everyone will agree on all manuscripts. I look forward to respectful debate on issues raised by journal publications.

There are questions to ask about information published elsewhere. You should know what quality control measures have been employed. Are you reading opinion without evidence? Though not necessarily useless, has it been made clear by the publication?

Few dental schools give undergraduate training in dental sleep medicine.[2]

The challenge of continuing education in dental sleep medicine is that it is entirely a post-dental school endeavor. We don't have a body of basic dental school knowledge to build on. Therefore judging the quality of what is presented can be more difficult than in other areas.

The quality of courses and conferences in dental sleep medicine should also be scrutinized. There are charismatic speakers on the dental sleep medicine circuit whose presentations contain significant amounts of opinion, or fringe information, presented with the same emphasis as information with a solid evidence base.

Because there is no dental school base of information, those of us new to the field are vulnerable to speakers and writers presenting as fact that which is only speculation. The American Academy of Dental Sleep Medicine (AADSM) emphasizes evidence-based presentations in all its continuing education programs and conferences. AADSM offerings may not be as exciting or as flamboyant as presentations "on the circuit" but will be of good quality.

Measurement of quality in clinical practice is coming to all aspects of health care, I encourage you to look at the ADA publication "Quality Measurement in Dentistry: a Guidebook"[3] for an in depth review of quality measurement. The report identifies challenges to measurement, the first being a lack of evidence-based guidelines. As a result of collaboration between the AADSM and the AASM, dental sleep medicine is a leader and an example for other areas of dentistry. Evidence-based guidelines for the use of oral appliances in the treatment of sleep disordered breathing where first developed in 1995 and revisions based on the new evidence published since 2005 are underway.[4,5]

First, the careful, meticulous work of our research colleagues provides the foundation for guidelines. Then the thoughtful and evidence-based presentations of our best educators provide an approachable summary of research findings. Third, the quality measures and clinical guidelines will give us the ability to critically evaluate our clinical processes and outcomes. None of these is likely to be flamboyant, immediately exciting, or lead to rapid revenue, but they will give us the foundation to offer each patient the best quality care possible.

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DISCLOSURE STATEMENT

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

ORIGINAL ARTICLES

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Adenoid Hypertrophy in Pediatric Sleep Disordered Breathing and Craniofacial Growth: The Emerging Role of Dentistry

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Study Objectives: Summarize and synthesize the most recent evidence about adenoid hypertrophy, impact on craniofacial growth, role in sleep disordered breathing, and effects of treatment.

Methods: Literature review of relevant manuscripts from dentistry, orthodontics, otolaryngology, and sleep medicine.

Results: Adenoid hypertrophy is the most common cause of nasopharyngeal obstruction in children; the most common cause of pediatric sleep disordered breathing (SDB); and can be an etiologic cause of altered craniofacial growth characterized by long face, retrusive chin, and narrow maxilla. Early detection and treatment may mitigate or resolve negative effects of adenoid hypertrophy. Adenoidectomy remains a front line treatment for the majority of cases, although alternative treatments must be considered when different SDB etiologies and co-morbidities are present. Best available evidence suggests that rapid maxillary expansion and adenoidectomy work synergistically to resolve SDB symptoms, and often both treatments are necessary for full treatment effect.

Conclusions: Primary care dentists, pediatric dentists, and orthodontists have an important role in early detection of adenoid hypertrophy. Emerging evidence continues to demonstrate dental treatments as playing an increasingly important role in multidisciplinary management of pediatric SDB.

Keywords: adenoids, adenoidectomy, craniofacial growth, diagnosis, palatal expansion, obstructive sleep apnea, orthodontics, sleep disordered breathing

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The adenoids are a collection of lymphatic tissue located in the most superior-posterior aspect of the nasopharynx. They are situated at the inflection point between the horizontally oriented nasal passage and the vertically oriented oropharynx. Being a lymphoid tissue, the adenoids play a role in immunity housing large numbers of immunocompetent cells such as B cells, T cells, lymphocytes, and macrophages.[1] As a result, the adenoids are highly prone to inflammation when an immune response is elicited against foreign antigens.[1]

Even in healthy children, a physiologic amount of adenoid enlargement is a part of normal craniofacial growth and development. The adenoid lymphoid tissue naturally increases to its largest size sometime between age 5-10 years, then continually decreases in size until adulthood.[2,3] Since children of this age range naturally have some element of relative lymph enlargement, additional inflammation—actual inflammatory hypertrophy beyond physiologic adenoid enlargement—can introduce partial or complete nasopharyngeal obstruction.[4]

Epidemiologic studies have reported a high prevalence of adenoid hypertrophy in children. One large study of 1,132 subjects observed a frequency of 27% for children between 5 and 7 years, and 19% to 20% for children between the age of 8 and 14 years.[5] Other smaller studies have observed frequencies of 37.9% among 370 children between 3 and 9 years[6] and 57.7% among 213 children between 6 months and 15 years.[7]

When adenoid hypertrophy occurs in a chronic state, there can be long periods of partial or complete impairment of nasal function,[8] which may lead to mouth breathing to overcome the limited passage of air through the nasopharynx.[6] Chronic nasopharyngeal obstruction is believed to increase the risk for altered craniofacial growth and increase the risk of pediatric sleep disordered breathing.

THE EFFECT OF ADENOID HYPERTROPHY

Adenoid Hypertrophy and Altered Craniofacial Growth

Although previous research studied the link between nasal function and facial pattern, it was Linder-Aronson's seminal work that helped solidify the association between adenoid hypertrophy and altered human craniofacial growth. He noted that adenoid obstruction occurred in all facial types, but children with adenoid hypertrophy presented more frequently with a recurrent craniofacial phenotype. This phenotype was characterized by a narrow maxillary dental arch, posterior dental crossbite, steep mandibular plane, and long anterior face height.[9] Such a craniofacial phenotype was often termed "adenoid facies."

Linder-Aronson acknowledged that, in theory, a genetically driven facial pattern could also cause the nasopharyngeal obstruction. However, he favored a hypothesis that nasopharyngeal obstruction—whether by adenoid hypertrophy or other etiology—increased resistance to nasal airflow such that children were obligated to mouth breathe. The resulting open mouth posture became the driving force behind altered craniofacial growth. He theorized that during mouth breathing, the tongue assumed a lower posture to facilitate oral airflow and therefore no longer rested in the palate. Without the tongue providing internal muscular force, transverse maxillary

development would be hindered, as no expansive force would be present to overcome the external constrictive pressure of the cheek muscles. Put simply: Linder-Aronson viewed maxillary transverse constriction and vertical growth pattern as a result of mouth breathing when there was concomitant evidence of nasopharyngeal obstruction.

While the pathophysiologic mechanism postulated by Linder-Aronson has been debated, the association between facial growth and nasal function has been repeatedly demonstrated.[10-13] Recent studies have demonstrated chronic nasal obstruction by mechanisms other than adenoids—such as deviated septum[14] or chronic rhinitis[15]—also cause the same altered craniofacial growth pattern as adenoid hypertrophy. Two recent systematic reviews and meta-analyses concluded that nasopharyngeal obstruction can be a primary etiologic factor causing "adenoid facies."[16,17]

Adenoid Hypertrophy and Sleep Disordered Breathing

Sleep disordered breathing is a spectrum of disorders unified by respiratory disturbance or inadequate ventilation during sleep.[18] In this context, sleep disordered breathing can range from primary snoring to upper airway resistance syndrome to severe obstructive sleep apnea.[19] In the pediatric population, the epidemiology of sleep disordered breathing is poorly described, as its presence and consequences on overall health and wellness have been widely underappreciated. Only recently has pediatric sleep disordered breathing become more widely acknowledged as a public health problem. Best available estimates suggest the frequency of obstructive sleep apnea is approximately at 1% to 5%,[20] while the frequency of sleep disordered breathing (i.e., snoring) is estimated much higher, ranging from 3% to 27%.[20] The consequences of sleep disordered breathing to overall health can be severe. Neurocognitive dysfunction including attention deficit, hyperactivity, reduced grades in school, and aggression, and cardiovascular dysfunction including hypertension, ventricular hypertrophy, valvular damage, and cor pulmonale and delayed growth have all been reported.[21-27]

Factors such as obesity, asthma, ethnicity, preterm birth, and environmental irritants are all etiologic contributors and comorbidities of pediatric sleep disordered breathing.[1,28-33] However, in the pediatric population, adenoid hypertrophy is the most pervasive primary etiology.[1-3,18,34]

THE EFFECT OF ADENOIDECTOMY

Effect of Adenoidectomy on Craniofacial Growth

Linder-Aronson's work solidified the connection between adenoid hypertrophy and altered craniofacial growth. The next logical step was to investigate whether treating adenoid hypertrophy could normalize craniofacial growth.

Subsequent studies suggested a return to normal growth after adenoidectomy was possible. Multiple prospective, non-randomized clinical trials[4,35,36] demonstrated a tendency to a normalization of the mandibular plane angle (i.e., decrease in long face morphology) over 5-year follow-up, but no noticeable change during the first year. Such a finding is not unexpected, as noticeable growth changes take time to occur. Though both studies demonstrated statistical significance, some trends were questionable and the clinical significance was not profound. These findings suggest that some normalization does occur, but growth pattern may not be fully restored to normal.

Current investigations have also produced mixed results. A recent prospective, non-randomized trial[5,37] evaluated growth in a pediatric population (n = 34, mean age 5.6 years) with OSA for 5 years after adenotonsillectomy (A&T). Initially, the treatment group subjects had distinct facial morphology consistent with "adenoid facies," while control subjects did not. Five years after A&T, there was no discernable difference between the treatment and control groups. Conversely, a non-randomized prospective trial evaluated growth differences between treated and untreated controls (n = 80) after 1 year and found no difference.[38] If the conclusions of Linder-Aronson's original intervention study[35] are valid, one year may not be sufficient time to observe a growth change, and Souki et al.[38] may have incorrectly accepted the null hypothesis.

Unfortunately, none of the cited studies had strong methodological features; therefore, inconsistencies between studies may be due to study biases (methodological flaws). Yet the study that reported the strongest results[37] also treated the youngest subjects (mean age 5.6 years). The other studies[35,36] reported subjects mean age 7.5 years and 8.2 years, respectively. A recent cross-sectional study[13] suggested that children with obstructive adenoid hypertrophy should be treated before the age of 6 to achieve total normalization of craniofacial growth. Therefore the spectrum of results across studies may be confounded by an unaccounted covariate—age. We hypothesized that the clinical implication might be that children with nasopharyngeal obstruction should be treated before age 6 for the best prognosis of normalized craniofacial growth.

Effect of Adenoidectomy on Pediatric Sleep Disordered Breathing

At the present time, A&T is the evidence-based, first-line surgical treatment of pediatric obstructive sleep apnea.[18,33] One meta-analysis[39] described an average reduction of 13.9 AHI events following A&T and success rate of 82.9%, while another more recent systematic review estimated a success rate of only 66%.[40] However, the level of evidence generally is low, primarily coming from case series and cohort studies.[39,41]

Even though A&T is the current first surgical step, there are significant questions regarding its universal efficacy. Recent publications have reported failure rates of 49% to 75%.[42,43] Continuous positive airway pressure (CPAP) has become the standard of care treatment for children with failed A&T. A growing body of research suggests that certain populations have a particularly poor prognosis following A&T. The presence of midface deficiency, obesity, family history of SBD, certain ethnicity, asthma, gastroesophageal reflux disease (GERD), septum deviation, and chronic rhinitis all have various degrees of evidence to suggest a more guarded prognosis to A&T treatment.[29,44-47]

Concurrent evidence is growing that alternative treatments are essential for SDB management, such as anti-inflammatory medication,[48,49] proton-pump inhibitors,[50] and orthodontics.[47] Unsurprisingly, each of these treatment alternatives addresses specific comorbidities that may compromise the prognosis of A&T therapy.

In conclusion, recent evidence demonstrating a more guarded prognosis of A&T treatment for pediatric SDB suggests

significant gaps in knowledge in current diagnostic standards. Further research is needed before clinicians can provide consistently accurate, patient-specific prognosis for A&T. Even though the role of A&T requires tailoring, its importance cannot be underestimated in SBD management. Because of the high prevalence of adenoid hypertrophy as a primary etiology in children with SDB, adenotonsillectomy will always remain an important front-line surgical treatment option. Simply put, A&T should be seen as an initial, simple, and important treatment, but no longer viewed as a universal or ultimate surgical treatment for pediatric SDB.

DIAGNOSIS OF ADENOID HYPERTROPHY

Numerous tools are available to evaluate the nasal and nasopharyngeal airway. Clinical exam alone, acoustic rhinometry, lateral cephalometry, multi-row detector CT imaging, video fluoroscopy, and cone beam computed tomography (CBCT) have all been described as methods for evaluating nasopharyngeal patency.[8,51-55] However, each of these methods has significant drawbacks. Clinical exam alone lacks the sensitivity to be useful.[51] Lateral cephalograms provide fair diagnostic value but tend to overestimate adenoid size.[55] Multi-row detector CT scans and video fluoroscopy are both very accurate but require specialized equipment and expose patients to unjustifiably high levels of radiation.[53,54]

Beyond all other diagnostic methods, nasoendoscopy using a standardized grading system is the gold standard for diagnosis of adenoid hypertrophy.[55-57] Nasoendoscopy is minimally invasive, highly reliable, and easy for an otolaryngologist to perform. However, performing nasoendoscopy is outside the scope of practice for other health-care providers concerned with adenoid size, such as orthodontists or sleep medicine specialists. While nasoendoscopy is an excellent diagnostic procedure, gaining access to an otolaryngologist is the most difficult step to getting a reliable diagnosis of adenoid hypertrophy.

A recent study evaluating CBCT has shown promising results.[55] Sensitivity and specificity of 88% and 93%, respectively, were reported. However, there were also challenges. Strong intra-observer and inter-observer repeatability among trained evaluators was observed, but concerns were raised about the diagnostic ability of casual clinicians' interpretation of CBCT images. In addition, there were concerns about unacceptably high ionizing radiation exposure when not used for more medically serious purposes. While CBCT can be very accurate and reliable for diagnosing adenoid size, the results cannot be taken as justification for liberal use of CBCT imaging. Evaluation of adenoid size alone provides insufficient grounds to acquire a CBCT image. However, when CBCT images are acquired for other valid reasons—such as orthodontic records—the images can be secondarily evaluated for adenoid hypertrophy with a high degree of certainty.

ROLE OF THE DENTIST AND ORTHODONTIST IN AIRWAY MANAGEMENT

The dentist has several important roles in airway management. First, for patients with a history of nasopharyngeal obstruction and altered facial growth, orthodontic manipulation of the teeth and skeleton may help normalize the dentofacial appearance, thus improving the patient's esthetics and correcting the associated malocclusion.

Second, a dentist is well situated to play an important role in early detection and screening of certain children with airway dysfunction. Through timely diagnosis, an orthodontist may altogether prevent, or at least limit, the development of malocclusion and altered craniofacial growth. Using the same diagnostic skills, an orthodontist can screen for children with sleep disordered breathing. By facilitating timely referral to an otolaryngologist and/or sleep physician, and orthodontist can substantially improve a patient's overall health and quality of life.

Third, through orthopedic manipulation of the facial skeleton, an orthodontist may be able to contribute to the treatment of specific forms of sleep disordered breathing. Early research in rapid palatal expansion[47] and mandibular repositioning appliances[58] are promising and may in the future become cornerstone treatments for select pediatric sleep disordered breathing patients. However, significant research is still required before an orthodontist can reliably treat sleep disordered breathing.

At the present time dentist's most important role in airway management is to act as an early detector of airway dysfunction, and coordinate timely referral to appropriate health professionals. However, if recent research is any indication of future clinical practice, dentists are likely to gain increasing prominence also in managing specific sleep related problems.

CONCLUSION

In summary, chronic adenoid hypertrophy is the most common etiology of pediatric sleep disordered breathing. It has been strongly implicated in the altered craniofacial growth pattern termed "adenoid facies"—that is, long face, maxillary constriction with an associated dental crossbite, increased overjet, and weak chin projection. Currently adenotonsillectomy is the front-line treatment for pediatric sleep disordered breathing.

A new paradigm is emerging that recognizes a multitude of additional causes for nasal obstruction and pediatric sleep disordered breathing. Recognition of the comorbidities and collaborative disease contributors can be very important for evaluating individual patient risk profiles and prognosis of treatment. Unsurprisingly, new treatment options are emerging as alternative or collaborative therapy modalities that specifically address these alternative disease etiologies.

Dentists and orthodontists have an increasingly important role in the early detection of children with sleep disordered breathing and adenoid hypertrophy. Dentists' ability to recognize altered craniofacial growth patterns and access to alternative diagnostic techniques enables dentists to screen children with sleep disordered breathing with high accuracy. Furthermore, new research suggests that dentists and orthodontists may have a critical role in treating select subgroups of children with sleep disordered breathing.

The understanding of pediatric sleep disordered breathing is evolving to recognize the important diagnostic and unique treatment roles dentists may contribute. Therefore it is important that dentists learn to recognize the signs, symptoms, risk factors, and comorbidities of pediatric sleep disordered

breathing in daily practice. In doing so, dentists can have a positive impact on their patients' overall health and quality of life.

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ABSTRACTS

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POSTER #001

Patients' and Bed Partners' Quality of Life Assessment of Oral Appliance Therapy in Obstructive Sleep Apnea

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Introduction: It is reported that Continuous Positive Airway Pressure (CPAP), often considered as a first choice therapy for OSA, could affect improvements in the QOL of bed partners (Kiely JL, 1997). In Oral Appliance (OA) studies, some reports suggest effectiveness in patient's QOL variables (Phillips CL, 2013). Although a previous study reported improvements of patients' and bed partner's QOL retrospectively (Tegelberg A, 2012), there is no comparative study to estimate the effect of bed partner's QOL. The aim of this study was to examine the effects on the QOL of both parties after the patients were treated with an OA.

Methods: This study consists of a simple questionnaire survey administered by asking the patient and bed partner to complete it at his/her home before and after OA therapy. The questionnaire consists of the Short Form 36 (SF-36), the Epworth sleepiness scale (ESS) and general questions requesting information such as snoring habits and sharing the bedroom details. The protocol had the prior approval of the clinical research ethics board, UBC Office of Research Services. Wilcoxon Signed Ranks test and Spearman test were used for statistical analysis. A $P < 0.05$ was considered as significant.

Results: A total of twenty patients (65% male, age 52.9 ± 9.9 years, BMI 26.3 ± 4.8 kg/m^2, baseline ESS 7.1 ± 3.1) and ten partners (10% male, age 49.9 ± 9.7 years, BMI 25.1 ± 8.6 kg/m^2, baseline ESS 4.4 ± 3.0) completed the data collection. Eighty five percent of patients had bed partner after OA therapy, and seventy five percent of patients and bed partners slept in the same room. In addition to significant improvements in patients ESS (median 6.5 to 5.5, $p = 0.04$) and bed partner's physical function (median 92.5 to 97.5, $P = 0.039$), a higher adherence (frequency of wearing an OA nights/week) correlated with an improvement in patients QOL variables (role physical $r = 0.512$, vitality $r = 0.465$, role emotional $r = 0.488$, and mental health $r = 0.485$, $p < 0.05$). The greater the snoring reduction correlated with an improvement in bed partners ESS score ($r = -0.744$, $p < 0.05$) and QOL variables (role physical $r = 0.632$, mental health $r = 0.848$, physical health $r = 0.848$ and total SF36 score $r = 0.780$, $p < 0.05$).

Conclusion: OSA patients with better adherence to OA therapy experienced an improvement in their own QOL. Bed partners who reported a greater reduction in patient's snoring exhibited an improvement in their own sleepiness and QOL. An assessment of changes in sleepiness and QOL variables in bed partners based on a larger sample size is warranted to further evaluate the potential changes that occur.

POSTER #002

Fabrication of an Oral Sleep Appliance without Dental Impressions

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Introduction: The fabrication of custom oral sleep appliance has historically required dental impressions. This procedure in general is one that patients do not look forward to and may even forgo a dental appliance just to avoid it. Recently, intra oral scanners have proliferated giving the patient the option of digital impressions without any type of impression material in their mouth. A series of scans are taken of the patient's dental arch and through software, stitched together to create a digital dental model. Manufactures also claim that appliances made from these models are more accurate than conventional dental impressions. The purpose of this study was to use digital scans of the dental arches and a therapeutic bite which were then electronically submitted to a dental lab for fabrication of a dental sleep appliance. In addition, the sleep appliance effectiveness was evaluated with a home sleep test.

Methods: An initial home sleep test was given to the subject to verify the diagnosis of mild sleep apnea and to gather baseline data for comparison of the effectiveness of the oral appliance. The subject's upper and lower arches were scanned. For the construction bite the subject was fitted with a jig to determine the greatest vertical opening which was comfortable and didn't cause a chin strain. From there the subject was ask to move the lower jaw forward into the jig while making snoring sounds until diminished with the jaw in a comfortable position where the lips could still seal. With the subject biting on the jig, the right and left buccal segments were scanned to record the construction bite. The STL files were then exported to the Dental Lab for fabrication of a Sleep Herbst appliance. At appliance delivery it was subjectively tested for fit and ability to make snoring sounds. A home sleep test was then given once the subject was comfortable with the appliance and reported improvement of symptoms. Before and after indexes were objectively compared.

Results: Subjectively the appliance fit very well and position of mandible with the appliance in was accurate to the construction bite. Subject reports he was comfortable with the appliance at delivery. The before/after pRDI 11.68/9.24, pAHI 8.57/1.78 and ODI 6.85/1.13 were compared. The comparison of before/after total events for the three indexes were; pRDI 75/57, pAHI 55/11 and ODI 44/7. Both the after AHI and ODI were reduced into

the normal range. The after RDI, although not in the normal range, was also reduced.

Conclusion: Although this study had only one subject, this practitioners' experience with digital scans for fabrication of orthodontic appliances has been very successful and this study indicates it will also be true for impression less fabrication of dental sleep appliances. The fabrication of oral sleep appliances without dental impressions are as effective and more patient friendly than traditional methods and may become the new standard of care.

POSTER #003

ORCADES, a Prospective Multicenter Cohort Study of Obstructive Sleep Apnea (OSA) Patients Treated with a Custom-Made Mandibular Repositioning Device (MRD)

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Introduction: Use of an MRD is an alternative therapy for OSA in patients noncompliant with continuous positive airway pressure (CPAP). ORCADES is a French, prospective, multicenter, long-term, observational cohort study in 360 OSA patients who refused or did not tolerate CPAP and then were treated with a custom-made MRD. Results for the first patients treated are shown.

Methods: OSA patients screened by sleep physicians were referred to a dental specialist who fitted a custom-made MRD (CadCam; Narval) in eligible patients and did gradual mandibular advancement (MA) titration. Objective sleep data, clinical symptoms, quality of life, side effects and compliance were evaluated. Treatment success was defined as a ≥ 50% decrease from baseline in the apnea-hypopnea index (AHI).

Results: Interim analysis was performed on the first 232 patients fitted with an MRD (71% male, age 53.2 ± 11.9 years, mean AHI 29 ± 15/h, body mass index 27 ± 4 kg/m^2, 50.2% who refused CPAP and 49.8% previously treated with CPAP). Baseline AHI was 5-15/h (14% of patients), 15-30/h (44%) or > 30/h (42%); 42% of patients had severe snoring. Mean final MA was 7 ± 2 mm (75% of maximum MA; 2 ± 1 titrations). Among the 143 patients who have undergone a 3-month assessment, treatment success rate was 84% regardless of OSA severity. AHI was < 10/h in 63% of patients. Epworth Sleepiness Scale score decreased from 12 ± 5 to 8 ± 5, and loud snoring disappeared in 90% of patients affected. Nocturnal polyuria and sexual dysfunction resolved completely in > 50% of patients. Quality of life and fatigue score improved significantly from baseline. Compliance was high, with MRD use of mean 6.7 h/night on mean 6.6 days/wk. During the first 9 months of treatment, 61 patients (27%) have reported side effects. These included gum irritation or pain (9.5%, n = 22), dental or periodontal pain (8%, n = 19), temporomandibular joint pain or stiffness (7%, n = 16), cheek or tongue irritation (3%, n = 8), excessive salivation (2.5%, n = 6), dry mouth (2.5%, n = 6). To date, the rate of occlusion change or dental mobility was low (< 2%). Only 10 patients (4%) stopped treatment early as a result of side effects.

Conclusion: A custom-made MRD is an effective therapy for OSA that can be used successfully in patients who refuse, or are noncompliant with, CPAP.

POSTER #004

Oral Appliance Therapy Versus Nasal CPAP in Obstructive Sleep Apnea: a Randomised, Placebo-Controlled Trial on Psychological Distress

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Introduction: Obstructive sleep apnea is associated with a high prevalence of psychological distress symptoms, including depression and somatisation. The aim of the present study was to compare the effects of a mandibular advancement device (MAD) with those of nasal Continuous Positive Airway Pressure (nCPAP) on psychological outcomes.

Methods: This study is part of a randomized placebo-controlled trial, in which different treatment effects of a titrated MAD are compared with those of nCPAP and an intra-oral placebo appliance in a parallel design. Sixty-four mild/moderate OSA patients (52.0 ± 9.6 years) were randomly assigned to these three parallel groups. All patients filled out twice the Dutch version of the Symptom Checklist-90-Revised (SCL-90-R): one before treatment and one after six months of treatment. The SCL-90-R is a multidimensional symptom inventory designed to measure symptomatic psychological distress over the past week (e.g., depression and somatisation). Linear mixed model analyses were performed to study differences between the groups for the different dimensions of the SCL-90-R over time.

Results: The three groups showed higher average values of psychological distress at baseline than the reported normal values for the Dutch population (P = 0.001). The baseline values of the different dimensions of the SCL-90-R questionnaire did not differ significantly between the three groups (P = 0.305–0.987). The changes in the different dimensions from baseline to therapy evaluation were not significantly different between the three groups (P = 0.175–0.950), while the pooled data of the three groups showed significant improvements in the dimensions "somatisation," "Insufficiency of thinking and acting," "agoraphobia," "anxiety," "depression," and "sleeping problems," over time (F = 4.14–16.73, P = 0.048–0.0002).

Conclusion: Within the limits of this study, it can be concluded that there is no significant difference between MAD and nCPAP in the effects on psychological outcomes. Moreover, placebo effects probably play an important role in the significant improvements of psychological distress symptoms with these therapies.

POSTER #005

Dental Side-Effects Following Mandibular Advancement Therapy: Assessment of Changes in Overjet as a Function of the Type of Occlusion

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Introduction: Oral appliance (OA) therapy with mandibular advancement devices (OAm) is a lifelong treatment. Therefore, the prevalence of possible side effects as well as their nature need to be evaluated for each patient. The present study describes the prevalence of changes in overjet (OJ) as a function of the type of occlusion.

Methods: A retrospective study was conducted on 93 patients (baseline: apnea/hypopnea index (AHI) 20.0 (13.7) /hr; body mass index (BMI) 26.8 (4.2) kg/m^2; male/female ratio 77.4%; age 47.2 (9.1) years) treated for 1 year with a custom-made titratable OAm without labial protection at the incisors (treatment: AHI 7.5 (8.5) /hr; BMI 26.8 (4.2) kg/m^2; Δprotrusion 8.8 (2.6) mm; subjective use 47 (10.2) h/week). Patients titrated until maximum comfortable protrusion or resolution of the subjective symptoms.

Patients were excluded after one year because of (a) missing data (n = 1), (b) irregular OAm use (n = 4), (c) dental prosthetics or orthodontic wires (n = 14) on incisors and/or canines which could inhibit teeth movement. Finally 78 patients were subdivided in six groups based on the measured OJ and overbite: normal bite (NB; n = 21), deep bite (DB; n = 16), large OJ (LOJ; n = 21), LOJ plus DB (LOJDB; n = 13), small overjet (SOJ; n = 4), edge-to-edge (ETE; n = 2), and open bite (OPB; n = 1). SOJ, ETE and OPB were excluded due to a small sample size.

The effects of age, BMI, ΔAHI, Δprotrusion and number of supporting teeth were studied in R (ANOVA or Kruskal-Wallis analysis; level of significance $p < 0.05$; normal distribution verified using Shapiro-Wilk). The effect of the type of occlusion on ΔOJ after one year of treatment was calculated with a multiple linear regression model and a step-down Bonferroni analysis, corrected for both the amount of protrusion and the degree of subjective use of the OAm.

Results: One year of OAm treatment caused in 79.5% of all patients a change in OJ. No significant difference was found between the number of patients with a changed OJ per subgroup. However, only 60% of patients starting with a DB demonstrated changes in OJ as opposed to 90%, 81% and 85% of patients with a LOJ, NB and LOJDB, respectively. Multiple regression analysis revealed a significant difference in changes in OJ between the NB and DB subgroup ($p = 0.027$) and the LOJ and DB subgroup ($p = 0.012$).

Conclusion: The present results confirm a high prevalence of changes in overjet (OJ). Further, the presence of a deep bite will result in smaller changes in OJ after one year compared to a normal bite or a large OJ. This protective deep bite effect applies only to those patients starting with a normal or small OJ.

POSTER #006

Comparison of Mandibular Repositioning Device Outcomes for the Treatment of Obstructive Sleep Apnea Using Alternative Approaches for Determining the Optimal Jaw-Forward Position

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Introduction: Successful outcomes from oral appliance therapy require an optimized positioning of the jaw in both the vertical and protrusive planes. This study provides a comparison of mandibular repositioning device outcomes (MRD) using two methods for defining the optimal jaw position.

Methods: Method A (N = 28) results were derived from a retrospective analysis of patient charts from those who had a dental sleep medicine office. The George Gauge was used to define protrusion of 60%. Patients were instructed then to advance the MRD until snoring stopped and/or they felt less tired. If the patient had not advanced the MRD or subjective reports indicated additional titration was required, additional office visits were scheduled for adjustments. Method B (N = 23) patients were evaluated prospectively. The Apnea Guard device (AG) was used to determine correct jaw position based on 70% protrusion, and a predictive algorithm for vertical separation (VDO) based on gender and tongue size. AG outcomes were measured at 30 days with no additional adjustments to the MRD.

Results: Improvement in sleep parameters: The mean pre and post-treatment AHI for Method A were 24 ± 10.7 and 8 ± 6.8. The comparative change in AHI for Method B was 22 ± 10.2 to 6 ± 4.1. Additionally, for those in Method A Group, 64.3% (18) achieved an AHI < 10 and an overall reduction of 50%. In Method B, 78.3% of patients achieved the goal of < 10AHI and a 50% reduction. 14.3% and 4.3% of patients respectively failed to reach treatment goals using Method A versus Method B. No statistically significant differences in outcomes were observed between the two methods based on the Chi-Squared statistic. One of the two patients who failed the endpoint with Method B appeared to be a non-responder to oral appliance therapy (rather than a failure of the protocol). Improved time to successful treatment: Group A displayed a mean time to successful treatment of 136 ± 53.1 while Group B displayed an improved time to successful treatment outcome of 33 ± 15 days. The average time to achieve an efficacious outcome with Method B (i.e., 34 days) excluded delays resulting from patient rescheduling.

Conclusion: Methods A and B both achieved clinically important treatment endpoints for the vast majority of patients, while Method B provided fewer treatment failures. A jaw-forward

position that optimizes treatment outcomes with the MRD was predicted with the AG. Method B allowed therapy to be initiated immediately, reduced the treatment time by an average of 100 days, and decreased the cost for delivery and repeat visits. A cost analysis showed that Method B reduced the treatment time by an average of 100 days, and decreased the cost for delivery of an efficacious outcome by an average of $300 per patient in this setting.

POSTER #007

Effects of Response Criteria on the Success Rate of Oral Appliance Treatment for Obstructive Sleep Apnea

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Introduction: One clinical issue in oral appliance therapy for obstructive sleep apnea (OSA) is that treatment success is defined rather arbitrarily. Although a given treatment may be regarded as "successful" under the liberal definition of a response, an increase in residual respiratory events after treatment could possibly lead to adverse health outcomes including OSA-related hypoxemia. We hypothesized that the success rate of treatment with an oral appliance would vary considerably with the selection of the response criteria. We further sought to identify clinically-relevant criteria for treatment success with oral appliances by focusing on oxygenation in addition to Apnea Hypopnea Index (AHI).

Methods: The study protocol was approved by the ethics committee of the Neuropsychiatric Research Institute, Japan. The effects of an oral appliance on AHI and nadir percutaneous oxygen (SpO_2) were investigated in 224 Japanese OSA subjects (47 ± 12 years, 25 ± 4 kg/m^2). Treatment success was defined as a reduction in AHI to < 5/h with a > 50% reduction in baseline AHI (criterion 1), a > 50% reduction in baseline AHI alone (criterion 2), or a > 50% reduction in baseline AHI with the nadir SpO_2 above 90% (criterion 3). Paired t-tests were used to compare the differences between the baseline and follow-up AHI. 2×4 χ^2 tests followed by residual analyses were also used to evaluate the responder-nonresponder distributions.

Results: The baseline AHI was reduced with an oral appliance in place, compared with the follow-up value (23 ± 11 to 8.5 ± 8.7 /h, $p < 0.05$) in all of the patients. There were fewer responders under criterion 1 (41%, $|R_{adjusted}| = 1.8$, $p < 0.01$) while more responders under criterion 2 (72%, $|R_{adjusted}| = 0.8$, $p < 0.01$). Based on criterion 2, there were 161 responders, in whom the baseline AHI decreased from 23 ± 12 /h to 5 ± 4 /h ($p < 0.01$). However, 43% and 6% of these responders did not satisfy criteria 1 and 3, respectively. In every OSA subgroup, the success rate under criterion 2 [75% (mild), 71% (moderate), and 70% (severe)] was greater than that under criterion 1 (53%, 40%, and 24%, respectively). Nevertheless, responders under criterion 2 in the severe OSA subgroup were still hypoxemic with a nadir SpO_2 of 87 ± 8% even after treatment. This situation was improved by the use of criterion 3, where a satisfactory improvement in AHI (from 38 ± 11 to 1 ± 1 /h, $p < 0.01$) was associated with a sufficient increase in the nadir SpO_2 (from 75 ± 8 to 93 ± 2%, $p < 0.01$).

Conclusion: This is the first study to demonstrate that the success rate of OSA treatment with oral appliance can vary remarkably with selection of the response criteria. We propose that, to avoid adverse health outcomes, an adjunct definition of treatment success using SpO_2 may be effective for patients who have more severe OSA.

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POSTER #008

Postural Movements of the Mandible Associated with Inspiratory Effort during Sleep in Obstructive Sleep Apnea Patients

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Introduction: Mandibular posture in patients with obstructive sleep apnea (OSA) has received greater interest with the increased use of oral appliances that alter the posture of the mandible. However, postural movements of the mandible associated with respiratory effort during sleep remains poorly understood. The aim of this study was to characterize the relationship of the postural movements of the mandible with inspiratory effort during sleep in patients with various levels of OSA severity.

Methods: Forty-three patients (11 non-OSA patients, 18 mild and moderate, and 14 severe OSA) were studied in a hospital sleep laboratory using standard video-polysomnography. The posture of the mandible was monitored with a magnetic resonance field transducer placed over the forehead and chin (JAWSENS, Nomics, Belgium). Inspiratory effort was monitored with an inductance plethysmograph on the ribcage and abdomen. The jaw-closing or jaw-opening movement synchronized with inspiratory effort was visually scored in each epoch (30 seconds). A synchronized movement was considered to be positive when it lasted more than a half of the epoch and when the amplitude of the jaw movement was greater than 1.0 millimeter. The percentage of the positive epochs per total sleep epochs was calculated and P values of < 0.05 were considered significant.

Results: Two types of postural movement of the mandible were synchronized with inspiratory effort; type A) jaw-closuring movement during inspiratory effort, and type B) jaw-opening movement during inspiratory effort. There was no significant difference in the percentage of epochs that contained type A movement (non-OSA: 1.1 ± 2.3%; mild and moderate OSA: 5.7 ± 14.7%; severe OSA: 3.3 ± 7.5%). In contrast, the percentage of epochs that contained type B movement (synchronized

jaw-opening during inspiratory effort) was significantly higher in the severe OSA patients compared to non-OSA patients (non-OSA: 3.1 ± 6.7%; mild and moderate OSA: 9.3 ± 19.9%; severe OSA: 23.6 ± 27.2%). The increase in the percentage of type B movements was positively correlated with an increase in the Apnea Hypopnea Index ($P < 0.05$).

Conclusion: Our findings suggest that there is an increase in synchronized jaw-opening associated with inspiratory effort that increases with OSA severity. The observed unstable mandibular posture with increased OSA severity could represent a reduced response of the jaw-closing muscles to airflow stimuli in the pharynx. The observed postural movement may contribute to our understanding of the mode of action of oral appliance therapy.

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POSTER #009

Risk Factors Associated with Obstructive Sleep Apnea: A Case-Control Study

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Introduction: Obstructive sleep apnea (OSA) is a type of breathing disorders which is related to snoring and repeated obstruction at the pharyngeal area. The consequences of OSA impact quality of life and are associated with systemic diseases such as hypertension, diabetes mellitus and cardiovascular disease. OSA is a complex disease with multifactor etiology. However, the association between oral manifestration and etiology of OSA is still not clear.

Methods: We conducted a case-control study to investigate the association between physiologic (age, gender, BMI and neck circumference), systemic (diabetes and hypertension) and oral anatomic factors (tongue size, torus mandibularis, Mallampati's score, palatal vault and presence of lateral pharyngeal wall) with OSA. A total of 154 subjects including 78 OSA diagnosed patients (43 males, 35 females) and 78 general non-OSA patients (37 males, 41 females) were enrolled in the case and control groups, respectively. History-taking together with physical and oral examinations were thoroughly conducted according to the criteria of each factor classification.

Results: Descriptive statistic together with inferential statistic for bivariate analysis using chi square and multiple logistic regression revealed that there are three factors significantly associated with OSA. First, patients with Mallampati's score level 3 and 4 had risk for OSA 29.470 times higher than patients with lower level of Mallampati's score (OR = 29.470 95% CI 5.217–33.741). Second, patients with neck circumference > 40 cm had risk for OSA 10.861 times higher than patients with smaller neck circumference (OR = 10.861 95% CI 2.032–16.280). Third, patients with larger tongue had risk for OSA 5.34 times higher than patients with smaller tongue (OR = 5.341 95% CI 1.187–8.052). However, there is no statistical significant correlation between OSA and palatal width and depth, torus mandibularis size and presence of lateral pharyngeal wall.

Conclusion: Collaboration between physicians and dentists is crucial for management of OSA. As larger neck circumference and oral manifestation (high Mallampati's and larger tongue) can be found during physical and oral examination by dental practitioners, dentists should be able to aware, identify, give advice or refer patients who have history of snoring to get further examination, diagnosis and proper management if they have OSA from physician.

Support: Neuroscience Research and Development group (NRD), Faculty of Dentistry, Khon Kaen University, Thailand

POSTER #010

Practice Management Implications of Leading Custom Mandibular Advancement Devices

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Introduction: Mandibular Advancement Device (MAD) selection may impact patient outcomes and the business economics of a dental practice. Midwest Dental Sleep Center (MDSC) began recording the type of MAD selected by all dentists in the practice for treatment of OSA. Results of the patient/dentist reported problems and dental economic implications are reported for 309 consecutive patients treated by 4 dentists.

Methods: Patients were previously diagnosed by a sleep physician and subsequently referred to MDSC for a custom MAD. The dentists completed new patient consultations to determine candidacy for treatment and selected a custom MAD based on the overall clinical assessment. All findings were recorded in the patient's medical record allowing for data retrieval and analysis. Any unplanned patient visits due to patient reported problems, broken MADs and appointments for redelivery of repaired or replaced MAD's were tracked from 15–198 days.

Results: Across 309 cases, six different custom MADs were provided that met the inclusion criteria of 5 or more patients). There was no correlation of AHI severity or gender in the appliances selected. Patients call the practice with problems and when it cannot be resolved with phone consultation, appointments are scheduled. During the 198 day period, 21 percent (66/309) required a problem appointment. The rate of patient reported problem appointments ranged from 15% to 57% (of new patient deliveries) reported by MAD type. On average these are 30 minute appointments that are not reimbursed and represent a $200.00 loss in production per appointment. Therefore, during the 198 day period, patient reported problem appointments cost the practice $13,200.00, ranging from $1,600.00 to $4,000.00 depending on MAD type. Thirteen percent (41/309) of all cases required a lab repair or remake. The combined repair/remake rate by MAD type ranged from 0% to 22%. On average repairs/remakes take 3 weeks, which is

significant in lost therapy nights. Appointments for redeliveries are 45 minutes and represent a $300.00 loss in production per appointment. Excluding the lab fee for the remake or warranty coverage, redeliveries cost the practice $12,300.00 ranging from $600.00 to $4,800.00 depending on MAD type.

Conclusion: There are material differences in custom Mandibular Advancement Device rates of patient reported problems, repair and remake rates and the subsequent financial impacts on the operation of the dental practice. These have not previously been known and quantified.

POSTER #011

Feasibility Pilot Evaluating the Use of Pre-Fabricated Titratable Mandibular Advancement Device for Management of Obstructive Sleep Apnea

Dennis Hwang,[1] Samina Farooqi,[1] Nehemiah Chang,[1] Edgar El Sayad,[1] Anthony Daclan,[1] Olufemi Adenuga,[1] Kendra A. Becker,[1] Julie DeWitte,[1] Pat Maley,[2] Rosa Woodrum[1]

[1]Sleep Medicine, Kaiser Permanente, Fontana, CA, USA, [2]Apnea Sciences, Kaiser Permanente, Fontana, CA, USA

Introduction: The effectiveness of oral appliance therapy (OA) for OSA is variable, but ability to predict response based on baseline characteristics is limited. This is a feasibility pilot evaluating the use of a prefabricated titratable mandibular advancement device of PFMAD (ApneaRX, Apnea Sciences). If successfully used in a clinical setting, we can anticipate evaluating its utility as a possible predictive mechanism.

Methods: PFMAD used is a "one size fits all" boil-and-bite device with ability to incrementally adjust mandibular position (1 mm is typical neutral position and able to advance to 10 mm). Appropriate candidates for OA therapy at Kaiser Permanente Sleep Center (Fontana) were created a PFMAD by a respiratory therapist. Patients were instructed to start at 3 mm and advanced nightly by 1 mm as tolerated. At maximum tolerable advancement, repeat polysomnography (post-PSG with portable monitor) was performed. We assessed device fit, maximum tolerable advancement, efficacy, and acceptance of therapy.

Results: 76 patients attended OA Class. 73 patients were fit for PFMAD; 3 opted out due to being poor dental candidates. 52 patients returned for post-PSG while 21 declined further PFMAD trial (15 for discomfort, 3 failed to follow up, 2 re-tried CPAP, 1 no symptomatic benefit). Of the 52 that returned for post-PSG, 69% were "responders" (AHI4% improvement and post-AHI < 15). Response rate based on OSA severity: mild 63% (17/27); moderate 65% (13/20); severe 100% (5/5). Overall, AHI improved from 17.9 ± 12.7 to 7.1 ± 8.4 ($p < 0.01$) with median mandibular advancement of 6 mm (range 4-9 mm). In responders, there was essential normalization of AHI from 19.4 ± 14.3 to 2.9 ± 2.4 ($p < 0.01$). There were no significant differences in baseline characteristics between responders and non-responders. 24 patients were referred for custom oral appliance; comparison of efficacy with PFMAD is pending.

Conclusion: PFMAD can be feasibly created by non-dental sleep center providers, able to be worn by most patients, and able to be titrated by patients at home. We anticipate studying its utility in predicting efficacy and acceptance of customized OA.

POSTER #012

Sleep Medicine in Dental Hygiene Education

Brittany Minichbauer, Rose Sheats, Rebecca Wilder, Ceib Phillips, Gregory Essick

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

Introduction: According to the National Sleep Foundation, 70 million Americans chronically suffer from over 80 medically recognized sleep disorders. Many of these individuals remain undiagnosed. In order to effectively address this issue, health care professionals must collaboratively work to educate, identify, and treat patients with sleep disorders. However, medical and dental clinicians do not receive adequate education in sleep medicine. On the frontline regarding prevention and counseling, dental hygienists can play an important role in patient education, screening, and management of sleep disorders. The purpose of this study was to assess the amount of sleep medicine content in dental hygiene education programs across the US, to determine if the institutional setting or geographic region had an effect on the amount of sleep medicine content taught, and to solicit opinions from dental hygiene faculty on the importance of sleep medicine content in the dental hygiene curriculum.

Methods: An electronic survey was emailed via Qualtrics to all 332 accredited dental hygiene programs in the US. The 18-question survey assessed the sleep medicine content presented during the 2012/2013 academic year. Follow-up emails and phone calls were made to non-responding programs. Email addresses and phone numbers were obtained from the American Dental Hygienists' Association (ADHA) website.

Results: Thirty-six percent (36%) of the programs responded. The mean number of hours devoted to sleep medicine education was 1.55 hours (SD = 1.38). Seventy-four percent (74%) of the responding programs reported spending time on sleep bruxism (mean = 1.38 hours, SD = 0.85). However, only 39% of the responding programs reported spending time on other topics such as snoring and obstructive sleep apnea (mean = 1.39 hours, SD = 0.72). Other topics such as snoring and obstructive sleep apnea were more likely to be taught in university-based programs without a dental school than in those with a dental school or in community-based programs ($p < 0.05$). Seventy-one percent (71%) of respondents agreed that sleep medicine content should be incorporated into the dental hygiene curriculum. Eighty-five percent (85%) of respondents indicated an interest in learning more about sleep medicine.

Conclusion: Sleep medicine education is included in the majority of US dental hygiene programs, but this is largely limited to a superficial coverage of sleep bruxism and occlusal guards. Current training is inadequate to prepare dental hygienists for their potential role in patient education, screening, treatment and management of sleep-related breathing disorders. Dental hygiene faculty agree that sleep medicine is an important issue in health care and are interested in learning more about the subject.

POSTER #013

Physician Evaluation among Dental Patients who Screen High-Risk for Sleep Apnea

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Introduction: Obstructive sleep apnea (OSA) is increasing in prevalence, widely undiagnosed, a precursor of significant pathology and responsive to therapy. Collectively these features point to the salience of OSA screening. This study sought to investigate the utility and public acceptability of screening for OSA risk in a dental practice setting and to examine the response of patients to a recommendation for physician evaluation.

Methods: A convenience sample of 124 adults was recruited by dental hygienists at a community-based dental practice in Raleigh, NC, using flyers. OSA risk was assessed using two methods. (1) The 4-item STOP screening questionnaire classified high-risk as the presence of ≥ 2 of: loud snoring; daytime tiredness; witnessed apnea; hypertension. (2) Overnight pulse oximetry classified high-risk as the presence of either: oxyhemoglobin saturation below 90% for ≥ 1% cumulative recording time; or oxygen desaturation index ≥ 10 events/hour in which oxyhemoglobin saturation decreased ≥ 3% from baseline. Patients were notified of their OSA risk status according to each instrument in a mailed letter. Those classified as high-risk on one or both instruments were advised to seek physician evaluation within 3 months. Three months later, patients were asked by telephone if they had sought physician evaluation. Prevalence ratios (PR) with 95% confidence limits (95% CL) were estimated using a log-binomial regression model in which the binary dependent variable was physician evaluation. The independent variable was OSA risk classified as low-risk on both instruments or high-risk on: STOP only; pulse oximeter only; or both instruments. Covariates were age, sex, body mass index and daytime sleepiness.

Results: Among 124 patients, 48.4% was male, 23.9% was obese and the mean age was 51 years. Fifty percent screened high-risk on STOP questions and 31.5% screened high-risk on both instruments. Physician consultation information was obtained from 114 patients (91.9%). Of those patients who screened high-risk for OSA, 46.2% (n = 42) sought physician evaluation. Compared to patients screening low-risk on both instruments, those at high-risk on the STOP questions alone were nine times as likely to seek physician evaluation (adjusted PR = 9.2, CL: 1.1, 76.2); similar to patients screening high-risk by pulse oximetry alone (adjusted PR = 9.7, 95% CL: 1.2, 77.4). Patients high-risk on both instruments were 13 times as likely to seek physician evaluation as those at low-risk (adjusted PR = 13.0, 95% CL: 1.6, 103.4) but this was not significantly greater than that of patients at risk based on one instrument alone.

Conclusion: Patients who screened high-risk for OSA in a dental setting were receptive to advice to seek physician evaluation. Probability of care seeking was similar for patients at high-risk by simple questions or pulse oximetry when instruments were jointly administered. Findings, including the unexpectedly high percentage of patients screening high-risk for OSA based on published criteria, have implications for the establishment of recommendations for OSA screening in the dental office.

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Patient Complaint of "Sore Mouth" after Placement of a Mandibular Advancement Device: What Do You Do?

B. Gail Demko, DMD, Dip ABDSM

Sleep Apnea Dentists of New England, Weston, MA; Past-President, American Academy of Dental Sleep Medicine

The patient is a 58-year-old college professor who had a medical history positive for hypertension, dyslipidemia, vertigo, and chronic prostatitis. He is allergic to penicillin. His medications consisted of atenolol and simvastatin, and he had recently been switched to a third antibiotic (Cipro XR) because of chronic prostatitis. His dental history was negative for orthodontic therapy and his 28-tooth dentition was well restored. He has a long history of bruxism and, prior to placement of his mandibular advancement device, was compliant with use of a Michigan splint maxillary full arch night guard.

The patient was originally diagnosed with moderate obstructive sleep apnea (OSA). His apnea hypopnea index (AHI) was 25, with an apnea index (AI) of 5.4. Snoring was soft to moderate, and his oxygen nadir was 87% (mean 96%). The patient was intolerant of positive airway pressure (PAP) because of severe upper airway dryness; he was referred for evaluation by an otolaryngologist. He then underwent laser assisted uvuloplasty (LAUP) twice; follow-up polysomnography revealed severe obstructive sleep apnea with an AHI of 63.2, an AI 58.8, and an oxygen nadir of 72% (mean 93%). The otolaryngologist referred the patient for oral appliance therapy (OAT).

Evaluation of the Michigan splint revealed significant lateral wear facets, and the device of choice was believed to be a TAP 1 since this device allows easy lateral mandibular movement with the device in place.

The patient received his oral device and, at his one week follow-up evaluation reported excellent results with significant decrease in snoring, no witnessed apneic events, and significant improvement in Epworth Sleepiness Scale (ESS) score. His only complaint was sensitivity on his lips that had started on the upper lip, at the wet-dry line, and was now present on his lower lip. He was advised to use a lip emollient and return in one week.

The patient returned in one week with the complaint that "my lips are so sore I can't drink wine." He stated that "I love my new device but my lips are killing me." He also complained of xerostomia and the feeling of having a "cotton mouth." Clinical evaluation of the oral cavity showed obvious erythema and mild edema at the wet-dry line of both the upper and lower lip. The most likely diagnosis appeared to be irritation from the extra-oral knob on the TAP 1; the knob was removed and the patient was prescribed Kenalog in Orabase ointment to soothe the irritated labial tissue. The patient was appointed to return in one week.

When the patient returned, his complaints had escalated. Clinical evaluation showed significant erythema of the inner lining of the lips and erythema on the anterior one-third of the tongue with enlargement of the fungiform papillae; the visual picture was similar to an oral burn (see Figures 1 & 2). The patient was extremely uncomfortable and complained of problems with eating and speaking.

QUESTION: What is the differential diagnosis?

Figure 1

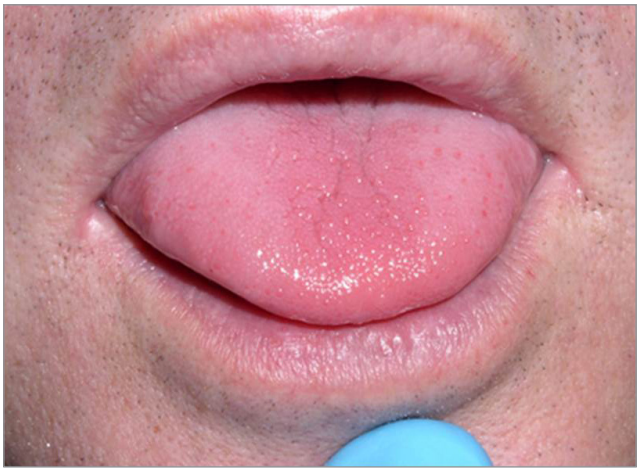
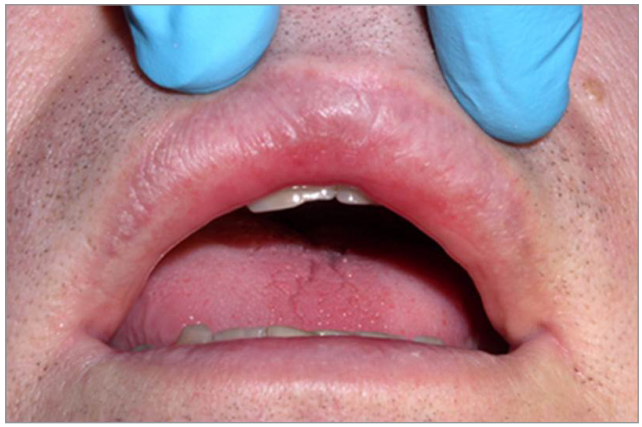


Figure 2



ANSWER:

- Burning mouth syndrome
- Allergic reaction to the components of the device or cleaning solution
- Allergic reaction to medication
- Candidiasis

Burning mouth syndrome normally presents as a moderate-to-severe burning sensation in the mouth, which may persist for many months. It often varies in intensity throughout the day and may subside at night. Anxiety and depression are common in patients with this syndrome, which may be result of the severe pain. There are multiple causes for this syndrome, the majority of which are never identified. This syndrome is classically identified with middle-aged women but can be found at any age or either gender. The oral mucosa often appears normal to visual examination.[1]

Allergic reaction to the components of the oral device can occur in any patient with any device. Polymethyl methacrylate (PMMA),[2] polycarbonate, nickel metal, dyes, and latex can all create an allergic reaction when in contact with oral mucosa. It is common for topical allergic reactions to occur after greater than three weeks of exposure to the allergen, but it is possible that the patient could have previously been exposed and sensitized to the allergen. An allergic reaction to a specific topical allergen should show coincident tissue reaction wherever that material contacts oral mucosa. Patients using a device with latex elastics will normally show reaction directly over the latex. Those who are allergic to nickel metal will respond with a reaction over the metal component. The oral cavity has very thin mucosa and is not as well protected from topical allergens as is highly keratinized tissue such as skin.

Allergic reaction to medications can occur at any time. Atenolol has been known to cause side effects of dizziness, lightheadedness, and nausea. It is more unusual for it to cause mood alterations, depression dizziness, and trouble breathing. Simvastatin has been known to cause muscle pain and tenderness, pain or burning during urination, headache, skin rash, or symptoms of upper respiratory infection.[3] Cephalosporins can create a range of hypersensitivity reactions from mild delay-onset cutaneous reactions to life-threatening anaphylaxis. It is also been tied to oral candidiasis, fever, and vomiting. All cephalosporin medications can cause the development of oral sores. These often occur along the gingival margin, on the buccal mucosa, or the tongue. Some patients respond with skin lesions on the lips or around the mouth.[4] The side effects are more likely to occur the longer the patient takes the cephalosporin.

Oral candidiasis is an opportunistic oral and genital infection caused by *Candida albicans*. While *C. albicans* is a normal component of gut flora, patients who are immunocompromised, under significant stress, or diabetic may be more open to attack by this this diploid fungus. The clinical appearance may be *pseudomembranous* with the "classic appearance" of oral candidiasis with an overgrowth of hyphae on the dorsum of the tongue and oral cavity often called "thrush," or *erythematous* in which the oral mucosa appears red and raw with atrophy of tissue structures and may precede the formation of the pseudo-membrane. *Hyperplastic candidiasis* appears as a persistent white plaque which does not rub off. Hyperplastic candidiasis is found most commonly at the commissures of the mouth.[5]

In this case, the patient was experiencing a reaction to long-term cephalosporin therapy for chronic prostatitis. The patient's oral lesions resolved within two weeks of discontinuing Cipro XR, and he has been successful with OAT for 10 years.

This case history is used to exemplify the need to have an accurate and in-depth medical history of the patient treated with OAT. While allergic reactions to device components can occur at any time, the treating dentist must be aware of other differential diagnoses that may be the cause of the patient's complaints.

CITATION

Demko BG. Patient complaint of "sore mouth" after placement of a mandibular advancement device: what do you do? *Journal of Dental Sleep Medicine* 2014;1(2):97–98.

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LETTERS TO THE EDITOR

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Macroglossia and Down Syndrome: Don't Be Fooled

Brian Hockel, DDS

Walnut Creek, CA

Congratulations on the new journal! After reading in the inaugural issue of *Journal of Dental Sleep Medicine* the case report of the patient with Down Syndrome by B. Gail Demko,[1] I thought a common misunderstanding could be clarified.

The article stated, "The overall goal of treatment was to provide increased interincisal distance and mild advancement to allow the macroglossic tongue to move forward out of the upper airway." It has been shown, however, that for people with Down Syndrome, it's the shorter cranial base that accounts for the lack of space for the tongue, rather than macroglossia. It turns out that the tongue is usually a normal size. Macroglossia is generally not the problem. The face is simply too shallow in depth to accommodate the normally-sized tongue. Then the poor rest oral posture leads to an underdevelopment of the size of the palate and forward growth of both jaws.

The conclusion of the work done by Guimaraes et al. was, "Children with Down Syndrome do not have true macroglossia but have relatively large tongues compared to the bony confines of the oral cavity."[2]

That's why many of us focus on orthopedic increase of oral volume and forward growth of the jaws at an early age, to try to avoid oral appliances, and perhaps OSA itself, at later ages.

CITATION

Hockel B. Macroglossia and Down Syndrome: don't be fooled. *Journal of Dental Sleep Medicine* 2014;1(2):99.

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LETTERS TO THE EDITOR

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Macroglossia and Down Syndrome

B. Gail Demko, DMD, Dip ABDSM

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This letter is to send kudos to Dr. Brian Hockel.[1] Because of his strong training in orthodontics and knowledge of the literature, he immediately picked up on the incorrect use of the term "macroglossia" when describing patients with Down Syndrome. Indeed the correct term should have been "relative macroglossia."

For more than 50 years, medical and dental literature has correctly defined patients with Down Syndrome as having a relatively normal size tongue in an underdeveloped skeletal surround. Not only is the maxilla underdeveloped with midface hypoplasia, these syndromal patients often lack frontal and maxillary sinuses. The high narrow palate represents a normal palatal height with thickened lateral aspects only giving the impression of a high narrow palate. These patients are often obligate mouth breathers because of a small nasopharynx and frequent upper respiratory infections which dries the dorsum of the tongue creating a fissured effect and strengthening the impression of macroglossia.

Ardran et al.[2] used two dimensional radiographs to show that Down Syndrome patients had relative macroglossia in 1972, a topic more elegantly researched using 3-D imaging by Guimaraes et al.[3]

Patients with syndromal deficiencies are often best treated young with orthodontic and orthopedic correction of the underlying bony abnormalities.

CITATION

Demko BG. Macroglossia and Down Syndrome. *Journal of Dental Sleep Medicine* 2014;1(2):101.

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AADSM News and Updates

Reported by Donald Farquhar, DDS; Stacey Kreutz, DDS; Rose Sheats, DMD, MPH; Leslie C. Dort, DDS, Dip ABDSM

AADSM 23RD ANNUAL MEETING

Dr. Gail Demko, President of the AADSM, welcomed members and attendees to the 23rd Annual Meeting of the AADSM on Thursday, May 29, 2014. The meeting began with the presentation of the AADSM awards. Dr. Satoru Tsuiki, DDS, PhD received the Pierre Robin Academic Award, Dr. Nancy Addy, DDS, Dip, ABDSM received the Distinguished Service Award and Dr. Charles Czeisler, MD, PhD was given the Honorary Membership Award for 2014.

Dr. Greg Essick, Chair of the AADSM Research Committee presented the following abstract awards:

- Clinical Excellence – Marie-Francoise Vecchierini, MD
- Clinical Research – Ghizlane Aarab, DDS, PhD
- Clinical Research – Scott Craig
- Student Excellence Award – Kristin Dillow, RDH
- Graduate Student Research Award – Tatsuya Fukuda, DDS
- Graduate Student Research Award – Brittany Minichbauer, RDH, BS

Selected Meeting Highlights

Pre-meeting Educational Courses

Mini Board Review Course

Todd Morgan, DMD; Robert Rogers, DMD; David Schartz, DDS

Drs. Morgan, Rogers and Schwartz provided a brief review of the sleep literature with specific reference to oral appliance therapy. Through didactic presentation and a number mock board review questions participants got an idea of what may be expected on future Board certification exams. For those planning on taking the 2014 American Board of Dental Sleep Medicine exam the session provided an opportunity to consolidate their knowledge base.

Advanced Dental Sleep Medicine

Rose Sheats, DMD; Reshma Amin, MD; Shalini Paruthi, MD; Benjamin Pliska, DDS, MS; Carol Rosen, MD

The advanced course was focused on pediatric sleep medicine. The dentist as "first responder" in the identification of sleep disordered breathing was a theme that was introduced at this course and echoed by many speakers throughout the meeting. Dr. Paruthi began the session reviewing the risk factor for sleep disordered breathing in children: adenotonsillar hypertrophy, obesity, cranial facial disorders and neuromuslcular disorders. She encouraged dentists and pediatricians who both see children regularly to ask parents about snoring. Dr. Carol Rosen addressed the complicated association between sleep disordered breathing and ADHD. Dentists were encouraged to: screen for snoring and SDB by asking questions, get to know the experts in the community, refer for objective evaluations when ADHD or SDB are suspected, and provide treatment in selected cases. Dr. Amin reviewed the many treatment possibilities for SDB in children. Dr. Pliska presented the dental and orthodontic treatment option for children with OSA. He discussed the limited yet promising evidence for mandibular advancement therapy as an orthodontic option for OSA.

General Sessions

Dr. Bruce Templeton, Chair of the Annual Meeting committee introduced Charles Czeisler, MD, PhD who gave the keynote address.

Keynote Address – Prevalence of Sleep Disorders in American Workers: Public Policy Implications

Charles Czeisler, MD, PhD

Dr. Czeisler gave a compelling summary of the impact of poor sleep on society. He dramatically highlighted the positive impact on productivity to be gained from minor changes to shift scheduling. He noted that the shocking statistic showing 1.75 million people fall asleep at the wheel every week was a call for awareness. He challenged dentists to be the "first responder" in helping to identify those at risk of having obstructive sleep apnea.

Comparative effectiveness of CPAP and Oral Appliance Therapy in Obstructive Sleep Apnea

Peter Cistulli, MBBS, PhD

Dr. Cistulli presented a summary of the recent research comparing CPAP and OA effectiveness. CPAP is more effective in reducing AHI but OA's have an effect similar to CPAP when comparing improvement in blood pressure and subjective outcomes. Information on objective compliance is now available for OA use. OA adherence objectively is longer than for CPAP and OA users are more accurate than CPAP users when reporting use subjectively. These results were found to be consistent for those with mild, moderate, and severe OSA

Sleep Medicine Malpractice: "Highway to the Danger Zone"

Ken Berley, DDS, JD

An attorney as well as a dentist who has been practicing dental sleep medicine for over 5 years, Ken Berley sent shock waves through the audience as he warned attendees of the "unusual level of risk" they assume when they treat patients with sleep disordered breathing. After receiving a whirlwind primer on relevant legal issues, dentists left Dr. Berley's gripping presentation determined to review their Informed Consent forms to ensure that their forms specify "explicit consent" rather than "implied consent." Furthermore, Dr. Berley exhorted practitioners to develop a detailed "Waiver and Indemnification Form" to limit their liability in the event of patient non-adherence

to treatment recommendations including follow-up with their referring physicians.

Role of Adenotonsillectomy in the Management of Pediatric OSAS: Findings from the CHAT Study That May Inform Your Dental Sleep Medicine Practice

Carol Rosen, MD

Results from this landmark randomized controlled trial on the potential benefits of early adenotonsillectomy (AT) to manage pediatric obstructive sleep apnea syndrome were published in the *New England Journal of Medicine* in 2013 (Marcus CL et al. N Engl J Med. 2013;368(25):2366-2376). The multi-center trial enrolled 453 children aged 5-9 years old who were confirmed on PSG to have moderate OSAS ($2 \leq$ oAHI ≤ 30 or $1 \leq$ oAI ≤ 20). Children were randomized to either early AT or Watchful Waiting with Supportive Care (WWSC) for 7 months. Using validated instruments, the investigators demonstrated no difference between groups in attention/executive functions. However sleep parameters as well as neuropsychogical and health outcomes significantly improved in the AT group. Overall, the AHI normalized in approximately 80% of the AT group, but it is worth noting that 46% of the WWSC group normalized in the 7 month period. Disease severity, race and obesity were significantly associated with poorer outcomes.

Insomnia Assessment and Management "In the Trenches"

Donald Townsend, PhD

Dr. Townsend discussed several options for treating perpetuating factors that keep patients above the insomnia threshold, including medication, cognitive behavioral treatment (CBT) and non-traditional methods. He noted that CBT involves sleep restriction, stimulus control, relaxation therapy, sleepy hygiene education, and cognitive restructuring. He added that the CBT must be matched to the contributing factor.

Temporomandibular Disorders (TMD): Evidence for an Association with OSA

Gregory Essick, DDS

Dr. Essick described evidence for five hypothetical causative mechanisms underlying the observed association between TMD and OSA. Hypothetical causal pathways based on the available literature include the possibilities of impaired baroreflex sensitivity (impaired pain processing), increased sympathetic nervous system activity, activation of systemic pro-inflammatory pathways, psychological distress, sleep bruxism, and awake bruxism. These hypotheses require further research and evaluation.

In discussing findings from the ongoing large-scale NIH-supported "Orofacial Pain: Prospective Evaluation and Risk Assessment" study, he explained that this multi-center study is including investigation of the association of sleep disordered breathing and TMD disease in the initiation of first onset TMD as well as the odds of being at high risk for OSA in chronic TMD cases.

The Correlation between OSA and Nasal Breathing

Neal Nay, RPSGT, RST; John Tucker, DMD

Drs. Nay and Tuscker discussed how nasal airway obstruction caused by external or internal valve collapse, deviated septum or inflammation can affect CPAP and OAT therapy. Treating nasal restriction increases airflow should be considered as an adjunctive therapy.

Hypoglossal Nerve Stimulation Therapy for OSA

Ryan Soose, MD

Hypoglossal nerve stimulation is a new emerging treatment option for OSA. It involves implanting muscle stimulator subcutaneously on the patient's right side that stimulates the hypoglossal nerve via a pulmonary feedback lead. The tongue is stimulated and is displaced anteriorly, opening the airway. This is a new treatment option that requires further testing.

Dr. Soose presented the early results of patients who had received a hypoglossal nerve stimulating implant. The multi-center trial has had considerable success in reducing OSA in patients who received the device without major side-effects. Future work will help identify the appropriate patients to receive the device and to develop smaller devices to minimize impact on the implant on daily living.

Selection of OSA Patients for CPAP or Oral Appliance Therapy: Can Studies Performed During Sleep Guide Management Decisions?

Olivier Vanderveken, MD, PhD

Dr. Vandereken addressed the need for evidence based prediction of OA outcomes. He reviewed the results of studies using techniques during sleep to predict OA outcome. There is evidence to support a significant association between protrusive position and outcome but not between chin lift and treatment outcome. Results from drug induced sleep endoscopy (DISE) and area of airway collapse highly predictive of OA outcome.

Physician/Dentist Relations

Kelly Carden, MD, MBA

A common concern reported by dentists beginning the practice of dental sleep medicine is the difficulty in getting referrals from medical colleagues. Although well-educated in dental sleep medicine, many clinicians report difficulty in establishing referral networks that would give patients access to oral appliance treatment. Dr. Carden presented a process for establishing relationships with physicians. She gave many valuable suggestions for beginning and maintaining relationships with physicians to facilitate the multi-disciplinary treatment of OSA.

New Insights into the Pathogenesis of Obstructive and Central Sleep Apnea and Obesity Hypoventilation Syndrome

Richard Schwab, MD

This fascinating presentation reviewed imaging modalities that may help clarify the role of anatomic features in the pathogenesis of obstructive sleep apnea. Included in his summary of obesity findings, Dr. Schwab presented new research about alterations in distribution of tongue fat in apneics.

AADSM Annual Membership Meeting

On Friday, May 30, 2014 the new leadership of the AADSM was introduced at the Annual Membership Meeting of the AADSM. Dr. Gail Demko became immediate past-president and introduced the AADSM board for 2014-2015.

The AADSM Board for 2014-2015

Officers:
President: Kathleen Bennett, DDS, Dip ABDSM
President Elect: Harold Smith, DDS, Dip ABDSM
Past-President: Gail Demko, DMD, Dip ABDSM
Secretary/Treasurer: Leslie Dort, DDS, Dip ABDSM

Directors:
Grant Hensley, DDS
Kevin Postol, DDS, Dip ABDSM
Steve Scherr, DDS, Dip ABDSM
David Schwartz, DDS, Dip ABDSM
Rose Sheats, DDS, MPH
Thomas Schell, DMD, Dip ABDSM

Completing their terms were Todd Morgan, DMD, Dip ABDSM and Michael Simmons, DMD, Dip ABDSM.

American Board of Dental Sleep Medicine (ABDSM)
This year the largest number of candidates sat for the ABDSM diploma exam. On Sunday, June 1 2014 fifty-five individuals sat for the exam. All members are encouraged to begin the process of becoming a diplomate. Visit ABDSM.org for information.

The ABDSM Board for 2014-2015

Officers:
President: Nancy Addy, DDS, Dip ABDSM
President Elect: Steve Scherr, DDS, Dip ABDSM
Past President: Jonathan Parker, DDS, Dip ABDSM
Secretary-Treasurer: Ron Prehn, DDS, Dip ABDSM

Directors:
Leila Chahine: DDS, Dip ABDSM
Katherine Phillips: DDS, Dip ABDSM
Flavia Sreshta: DDS, Dip ABDSM