Joseph R. Zelk DNP, FNP, BC

Licenses and Certifications

License: Washington State APRN/RN 2003-present

License: Oregon State APN/RN 2006-present

2002 ANCC Board Certified # 374417 2002-2016

2011 - Board eligible for Behavioral Sleep Medicine Certification

2006 - Mini-Fellowship in the Advanced course for Dental Sleep Medicine

2003 – Atlanta School of Sleep Medicine and Technology Program/Clinical Polysomnography

2001 - AANP National Conference for Nurse Practitioners

1998 – Critical Care Consortium

2011, 1997 – ECG Interpretation

ACLS – **1999**-present

BLS – **1991**-present

EDUCATION

Oregon Health & Science University, Portland, Oregon admitted June 2007 completed 2011 - Doctor of Nursing Practice

University of Nevada, Reno, admitted May 2001 complete 2003 - Master of science in Nursing – Family Nurse Practitioner Program

Research Project: *"Rheumatoid Arthritis and Social Support"*
Course Work: Advanced Physiology, Advanced Health Assessment, Management of Acute Illness, Ambulatory Pharmacology, Management of Chronic Illness, Nursing Research, Nursing Theory, over 800 hours of clinical practicum

University of Nevada, Reno, enrolled May 1991 completed May 1997 – Bachelor of Science in Nursing

Employment and Type of Positions Held:

3.01.06 – Present – Clinical Director, Senior Sleep Medicine Consultant, Dental Sleep Medicine Consultant, Co-founder of Sleep Medicine Network
Scope of Clinic Services: Provider of Sleep Medicine, Family Medicine, and Dental Sleep Medicine with interdisciplinary team of Pulmonology, Dentistry and Otolaryngology. National lecturer on Sleep Medicine and Dental Sleep Medicine to include practice management/insurance management.

6.01.06 - 9.01.09 - Sponsored speaker for Cephalon, Takeda, Jazz and Glaxo-Smith-Kline Pharmaceuticals.

9.01.07 - 12.01.09 - Educational Consultant on for Sleep Consultant Network. Sleep Educator – Normal Sleep physiology and Pathophysiology of Sleep Disorders.

1.2005-1.2007 – Invited medical reviewer and consultant for Sleep Disorders for ***The Nurse Practitioner:*** *The American Journal of Primary Healthcare.*
1.10.05 – 1-2006 – Clinical Director of Sleep Health and Wellness NW LLC: Locations at Gresham, Hillsboro, Gateway, Clackamas, Astoria, and Roseburg OR
Scope of Clinic Services: Provider of Sleep Medicine in collaboration with Pulmonary Neurology and Otolaryngology sleep specialist

9.01.2003 – 1.01.2005 – Family Nurse Practitioner in Sleep Disorders Medicine
Eastside Sleep Disorder Center – Formulate plans of care for clients with sleep disorders. Initial consultations, follow up, review of polysomnographic data and in-hospital consultations

5.05.2001 – 8.30.2003 Family Nurse Practitioner in Cardiology Practice
Reno Heart Physicians – Formulate plans of care for cardiology clients, problem-focused and health maintenance; stress testing supervision and interpretation; hospital admissions with MD collaboration; medical management of patient health needs (ischemic risk stratification, cardiovascular risk management, heart failure management, dysrhythmia management); hospital follow up; triage; electrocardiographic interpretation (Holter monitoring, Kin of Hearts monitoring, 12 lead, signal-averaged ECG); advanced knowledge of 2D echocardiography, Doppler flow studies, scintigraphy, angiography

1.2001 – 5.2001 Cardiovascular RN/outpatient
Employed with Reno Heart Physicians – Assistance in patient care and follow up. Supervised Exercise stress testing and nuclear stress testing; Independent study in 2D echocardiography and cardiology

6.1998 – 2001 Critical Care RN
Washoe Medical Center – Intensive care unit: Experience in primary nursing care of trauma, surgery, neurology, cardiology and general medical clients. Management of PICC lines, Epidurals, Swan-ganz catheter, Intracranial Pressure Bolt, ventilator dependent patients, chest tubes, parenteral nutrition, dialysis

12.1997 – 6.1998 Charge and Staff RN
Washoe Medical Center – Orthopedic acute care unit

6.1997 – 12.1997 RN Cardiac Rehab Specialty
Washoe Health System – Cardiac Rehabilitation Center
Responsibilities included monitoring and implementing cardiac rehab program; planning and instruction of Smoking Cessation Program; administering stress tests; performing pulmonary function testing and pulmonary rehabilitation

Professional Publications:

Chief contributing co-author of the Dermatology Chapter for Alice Running & Amy Berndt's: *Management Guidelines for Nurse Practitioners, 2002*.

Sleep Consultant to Wired.MD for the development of a series of health education videos for teaching to the lay person about sleep disorders.

Research and Grants:

Investigator for Pilot Study: Improved CPAP compliance with strapless interface in a cohort of previous CPAP failure with traditional interfaces (to be submitted for publication late 2012). A Respironics, Inc./OHSU collaboration.

Co-patent holder – Nasal dilator splint.

Co-patent holder-SleepAPP^{TM} mandibular advancement device

Co-patent holder – Dental appliance-based nasal CPAP interface and oral obturator

Sub-Investigator for a 12-week, double-blinded, Placebo Controlled, Parallel Group Study to Assess the Efficacy and Safety of Ropinirole XR (Extended Release) in Patients with Restless Legs Syndrome – 2005

DNP Clinical Inquiry Project:

The Use of a Novel Oral Appliance in the Treatment of Moderate and Severe Obstructive Sleep Apnea, completed 5/26/2011

Professional Presentations:

Lecturer for Dental Sleep Medicine to Oregon Health & Science University-affiliated Sleep Medicine Fellowship Program -2007, 2008, 2009, 2010, 2011.

Lecturer for Sleep Medicine Network – 2006, 2007, 2008 *Dental Sleep Medicine, Oral Appliance management and Hybrid CPAP interface management*

Lecturer for Nurse Practitioner of Oregon – 2005, *Insomnia Practice Guidelines*

Lecturer for Sleep Consultant Network 2005, 2006 *Circadian Rhythm Disorders and Novel Hypnotics*

Lecturer for Cephalon, Takeda, SCN 2005, 2006, *Hypersomnolence Management*

Lecturer For Lippincott Williams & Wilkins - 2004

National Conference for Nurse Practitioners, Baltimore Convention Center – *Insomnia Assessment & Management*

National Conference For Nurse Managers – *Employee & Patient Safety: The Effects of Sleep Deprivation*

Lecturer for Sleep Health and Wellness NW LLC *Oregon-2005 Pediatric Sleep Disorders Insulin resistance and Sleep Disordered Breathing*

Lecturer Overlake Hospital Medical Center, Bellevue Washington-2004
Epidemiology and Clinical Presentations of Common Sleep Disorders
Sleep and Aging

Medical Care of the Bariatric Patient

Later School Start Times for Teens - Delayed Sleep Phase

Impacts on QOL related Chronic Insomnia and Fatigue

Lecturer for Eastside Sleep Disorder Center, Bellevue, Washington

EKG Interpretation; Geriatric Sleep Disorders; Work Ergonomics

Lecturer for the Cardiovascular Trends Seminar/Sponsored by Washoe Health Systems – Reno, 2003, *Cardiac Assessment/Heart Sounds/Pathophysiology*

Guest Lecturer for the University of Nevada Master's of
Nursing Program – Fall, 2002
Dermatology, Congestive Heart Failure Management

National Speaker's Panel for Cephalon – *Excessive Somnolence* Astoria, Hillsboro, Portland, Beaverton, and Gresham, Oregon; Reno, NV - 2005
Pacific Northwest Sleep Association- *Basic and Advanced ECG recognition* - 2005

Honors and Awards

Inducted into Sigma Theta Tau National Nursing Honor Society, 2000

Recipient of Sanford Center for Aging Academic Scholarship

Continuing Education Attended:

2011- Sleep Multimedia Text – 114 CME

2011 – Advanced Electrocardiographic interpretation course 55 CME

2011 – 12 case conferences in inter-professional sleep disorders medicine

2009 – Associated Professional Sleep Societies National Sleep Conference, Seattle Washington

2008- First Annual Dental HPAP conference

2007- Associated Professional Sleep Societies National Sleep Conference, Minneapolis, Minnesota, National Sleep Conference

2006 – SCN – Sleep Consultant Network training; ES – Sleep Consultant Congress on Excessive Somnolence (ES) treatment

2005 – Nursing Management Congress

2005 – Nurse Practitioners of Oregon Regional Clinical Conference

2005 - Pacific Northwest Sleep Association conference

2004 – Chest Conference Sleep Medicine Review 2003

2004 - National Nurse Practitioner Conference – Baltimore, Massachusetts

2003 – Basic Pacing and Pacemaker Follow up

2003 – 27th Annual Cardiology Update for Primary Care Physicians

2003 – Cardiovascular Trends Summit

References

Available on request.

Portfolio Executive Summary

Joseph R. Zelk, RN, MS, FNP, BC

Doctor of Nursing Practice Candidate, OHSU School of Nursing

October 28, 2011

The goal for the Doctor of Nursing Practice terminal degree was to fulfill Sleep Disorders Medicine Fellowship and Dental Sleep Medicine Fellowship requirements. My area of expertise for the past several years has been in Sleep Disorders. The components of this portfolio adequately represent the fulfillment of these goals. Sleep medicine fellowship needs to have several interdisciplinary case studies, have training in Polysomnographic signal recognition and interpretation, as well as, medical device and medication management for the treatment of Sleep Disorders. The clinical residency hours combined with the written content of the portfolio represent completed requirements for board eligibility for either the Sleep Medicine and/or the Dental Sleep Medicine board certification examinations.

At this time, only doctorally-prepared clinicians are eligible for sitting for the Behavioral Sleep Medicine board examination. By completed this DNP this requirement will have been fulfilled. The field of Sleep Disorders is relatively young and in need of driving forces for change, innovation and collaboration among health professions. The DNP will allow for me to practice within the advanced practice nursing specialty in a professional and evidence-based manner. The advanced understanding of ethical considerations of health care and delivery of health care has been strengthened by the DNP training.

This doctoral degree has resulted in increased access to underserved people for less well-known treatment modalities for sleep disorders. This DNP has also resulted in quality research study findings that may contribute significantly to the current level of understanding of oral appliance treatment in the management of moderate and severe obstructive sleep apnea through the CIP process. This CIP added to the OSA literature by demonstrating a well-tolerated and efficacious intervention for both moderate and severe OSA. This CIP provides novel data that could lead to more treatment options for patients with moderate and severe OSA. Future steps will be taken to move forward medical reimbursement for oral appliance therapy, explore the relationships between combined treatments for OSA treatment and innovate medical devices needed for the treatment of OSA. This training may result in better training and development of a standard curriculum for future advanced practice nurses aspiring to work in the Sleep Disorders arena.

DNP Portfolio Executive Summary

Joseph R. Zelk, RN, MS, FNP, BC

Doctor of Nursing Practice Candidate, OHSU School of Nurisng

October 28, 2011

The clinical inquiry project (CIP) objective was to evaluate the efficacy of a novel oral appliance (OA) to reduce sleep disordered breathing in patients with moderate and severe obstructive sleep apnea (OSA). The study examined retrospective chart audit date pre-test and post-test results from 120 subjects (mean age 50, mean BMI 34.4, 75% Caucasian). The experimental protocol to fit all subjects with moderate or severe OSA with a novel oral appliance (SleepAPP^{TM}) and advance the mandible per standardized protocol over no longer than 16 weeks. The primary outcome measures pAHI (peripheral arterial tonometry apnea hypopnea index) and total percentage of time of sleep under 90 % of oxygen saturation were assessed with a validated portable sleep monitoring device (WatchPAT100).

The findings indicate that the SleepAPP^{TM} OA reduces OSA and improves sleep quality in patients with moderate and severe OSA as measured by a reduction from a mean pAHI of 38.1 to a mean pAHI of 17.8 ($p < 0.0001$). The device significantly improved oxygen saturation improving 7.5 % to 3.5 % ($p < 0.0001$) of time spent below 90 % of oxygen. The SleepAPP^{TM} OA was well-tolerated as evidenced by 95 % adherence rate and no adverse events. Additional data indicate snore improvement and sleep quality improved in 95 % of the sample population. These data advance the field as they demonstrated efficacy in a higher risk group (moderate to severe OSA) and in a larger sample than is reported in the extant literature. This study is limited by selection bias as subjects enrolled were likely to have failed CPAP therapy or had a family member or friend fail CPAP therapy and thus sought out our specialty practice. A second

limitation is that the WatchPAT100 device resulted in 38 invalid test results due to operator error (patient self-application at home).

Current evidence for the efficacy of OA is for the treatment of mild to moderate OSA. This CIP added to the OSA literature by demonstrating a well-tolerated and efficacious intervention for both moderate and severe OSA. Future trials could compare the SleepAPP™ OA to more traditional CPAP by randomizing OSA patients into either standardized intervention. This CIP provides novel data that could lead to more treatment options for patients with moderate and severe OSA.

Retrospective Chart Review: The Use of a Novel Oral Appliance Toward the Treatment of Moderate and Severe Obstructive Sleep Apnea

Joseph R. Zelk, MS, FNP, BC

Oregon Health & Science University

Nursing 703/Clinical Inquiry

October 28, 2011

Kim Jones RN, MSN, PhD

Results

Sample

The clinical inquiry project (CIP) objective was to evaluate the efficacy of a novel oral appliance (OA) (SleepAPP^{TM}) (Figure 1.) to reduce sleep disordered breathing in patients with moderate and severe obstructive sleep apnea (OSA). The study examined retrospective chart audit date pre-test and post-test results from 120 subjects (mean age 50, mean BMI 34.4, 75% Caucasian). The experimental protocol to fit all subjects with moderate or severe OSA with a novel oral appliance (SleepAPP^{TM}) and advance the mandible per standardized protocol over no longer than 16 weeks. The primary outcome measures pAHI (peripheral arterial tonometry apnea hypopnea index) and total percentage of time of sleep under 90 % of oxygen saturation were assessed with a validated portable sleep monitoring device (WatchPAT100).

Patients with an apnea hypopnea index greater than or equal to 15/h were considered to be eligible candidates. During the study period, 345 patients underwent consultation at the sleep disorders clinic. Of these, 187 patients were found to have moderate or severe OSA and met the inclusion criteria for the study. A consort diagram illustrates the enrollment process in Figure 2. There were 67 patients or thirty-eight percent of the study population that did not have complete data. These patients were not included in the statistical analysis. Review of cause for attrition from the study was accounted for by three reasons. Twelve patients could not be contacted after the fitting appointment of the oral appliance (OA). Seventeen patients were not willing to follow up for objective assessment due to cost of follow up assessment. Thirty-eight patients attempted a follow up assessment that resulted in bad data. These patients were not willing to reattempt the follow up test within the study period.

The baseline demographic and sleep study features for the patients are shown on table 1. Patients with severe OSA were older and heavier than the group in the moderate category. The population consisted mainly of men with a small group being female; this is noted in table 2. The generally accepted criteria for responders to OA treatment is when the Apnea/Hypopnea Index (AHI) is decreased by over 50% or when the AHI decrease is to <10 events an hour (Marklund, 1998) (Kushida, et al., 2006) (Ferguson, et al., 2006). This study combined these criteria to describe successful patient response to treatment. The data collected on this patient cohort was managed by one individual and maintained in a locked storage receptacle. The data used in analysis was de-identified to assure patient privacy.

Statistical Analysis

The means and standard deviations (SD) of continuous variables were compared using two group mean comparison two-tailed t test. Non-normally distributed variables were compared using the Wilcoxon rank-sum test. Ordinal variables were compared using X^2 test. Odds ratios and statistical analysis are presented with 95% confidence intervals, and reported p values are two-tailed. All p values <0.05 were considered statistically significant. All analyses were performed with statistical software (SAS release 9.2; SAS Institute; Cary, NC).

Findings

This clinical inquiry project had primary, secondary and exploratory aims. The primary aim was to evaluate the efficacy of a novel oral appliance to reduce sleep disordered breathing in moderate and severe OSA patients. A secondary aim was to utilize a new portable monitoring (PM) device to test the oral appliance therapy (WatchPAT100) (Figure 2a.). A secondary aim was to assess for change in oxygen saturation in sleep. The assessment measure used was the

amount of time spent during sleep below ninety percent below ninety percent oxygen saturation. Exploratory variables describing the cohort were analyzed.

Fifty-two percent of the subject population was found to have severe OSA, while forty-eight percent had a moderate degree of OSA. Eighty-one percent was male and nineteen percent female. Considering the small proportion of women in the trial, results were pooled for men and women. Ninety-two percent of the group was Caucasian, two percent African American, three percent Asian, and three percent Hispanic. The average age of the entire cohort was 50 years old. The average Body Mass Index (BMI) was 34.41. The average weight was 236 pounds (lbs). The patients with moderate OSA had an average age of 48 years old, with a BMI of 33. The patients with severe OSA the average age were 52 years old, with a BMI of 36.

A total of 47 out of 120 (39.17%) patients were categorized as responders. Eighteen severe patients (38.30%) and twenty-nine (61.70%) moderate. When considering the responders group by moderate or severe, there was a significant intra-group difference ($p=0.02$ at X^2 test). Of the 73 out of 120 (60.83) patients classified as non-responders, 29 patients were moderate and 44 patients severe. There were 71 (59.17%) patients that had => 50% drop of AHI (32/55.17% moderate/39/62.90% severe) ($p=0.39$ at X^2 test). There were 55 (45.83%) patients that had a reduction of AHI to <10 events an hour (37/63.79% moderate/18/29.03% severe) ($p=0.0001$ at X^2 test). There were no cases of OSA aggravation as defined by an increase of more than 10 in the AHI. The responders and non-responders had a significant intra-group difference when tested for weight change ($p=0.0005$ at X^2 test). The non-responders on average gained 4.38 lbs. The responders to treatment lost on average 3.56 lbs.

AHI events improved significantly ($p<0.0001$) with the OA compared to baseline assessment with an average reduction of 20.31(19.87) (Figure 3.). Patients with moderate OSA

exhibited an average decrease in AHI of 11.6 (5.9), from 21.4 (4.6) to 9.8 (7.0) ($p<0.0001$). Patients with severe OSA exhibited an average AHI decrease by 27.90 (20.77) from 53.2 (19.8) to 25.3 (21.6) ($p<0.0001$) (Figure 4.) (Table 4.).

The average AHI decrease for the entire cohort was reduced significantly 20.3(19.9) by the OA from 38.1 (21.7) to 17.8 (18.0) ($p<0.0001$) (Figure 5.). For the entire cohort there was a significant difference in the time spent below SaO_2 90% with an average improvement of 3.93 (9.8) from 7.5 (11.5) to 3.5 (7.9) ($p<0.0001$) (Figure 6.). Moderate OSA patients had significant average change of 0.9 (2.3) from 1.6 (2.1) to 0.7 (2.5) ($p<0.0001$). Severe OSA patients had significant average change of 6.5 (12.1) from 12.7 (13.8) to 6.2 (10.0) ($p<0.0001$) (Figure 7.) (Table 6.). Patients reported improvement on subjective questionnaire for reported snoring improvement and sleep quality improvement. A significant number of patients reported a decrease of snoring (95.8%). A significant number of patients reported improved sleep quality (95.8%). Observance of treatment was high with 95.8 % of patients wearing their oral appliance regularly. The average nightly usage was 6.5 hours per night of use and 6.5 nights a week. Only five patients had an observance that was unsatisfactory (less than 4 h per night and less than 4 days per week). No complaints of side effects were recorded that could have reduced the usage of the therapy as noted during the treatment assessment.

Discussion

Figure 5. represents the significantly improved AHI and percentage of total sleep time spent below 90% of oxygen saturation compared to the published literature on OA. The responder rate of 61.7% for moderate OSA patients and 38.3% for severe OSA patients exceeds the benchmark set by the extant OA research (60% and 25% respectively) ($p<0.0001$) (Marklund, 1998) (Ferguson, et al., 2006) (Figure 6.). Previous studies used a responder criteria

based on either AHI reduction of 50% or reduction of AHI below 10 events per hour. Other studies showed a response rate up to 60 percent; an improved response was found in this study, with a 61.7% response rate using the more rigorous criteria of reduction of AHI below 10 events per hour and AHI reduction of 50% or greater (Figure 7.).

The standard criteria for evaluation of OA treatment response is the use of the AHI and the assessment of nocturnal oxygen saturation. A reliable measurement of nocturnal oxygen saturation is the percent time of oxygen saturation below 90% for total sleep time (TST) (Penzel, 2004). The impact on oxygen saturation improvement sometimes yielded conflicting results from study to study in the literature (Lim, et al., 2006). This study represents a strongly significant improvement in oxygen saturation for treatment responders. The review of literature revealed a lack of consistent treatment criteria (Ferguson, et al., 2006). The oxygen saturation criteria for OA treatment response was less systematically reviewed (Mehta, 2001). Many of the studies found during the review of literature were representative of small sample sizes. The conflicting results from study to study may be attributable to these smaller sample sizes (Kushida, et al., 2006) (Lim, et al., 2006).

In an effort to establish a rigorous and consistent responder criteria for future research this study used a combination of the two common criterions (Marklund, 1998). To the knowledge of the researcher, this study is the first to assess efficacy of OA with the more rigorous benchmark for treatment using both AHI reduction of 50% and AHI below 10 events per hour. It is also the largest sample size to be assessed for device treatment effectiveness in the extant research. Possibly as significant as the inclusion of the stricter criterions for treatment, and the large representative sample size, is the exclusion of the mild forms of OSA from the sample. The specific aim of this clinical inquiry project was to assess the treatment response of OA in a

population of patients with more significant worsening of AHI in the moderate and severe cases of the disorder. The overall treatment response of 52% based on previous criteria may have been diluted by the large representation of mild OSA patients (Ferguson, et al., 2006)(Lim, et al., 2006). This study achieved similar overall treatment response in a population that excluded the mild form of OSA. By excluding the mild OSA population this study may be able to more specifically evaluate the response to OA in the forms of sleep disordered breathing that have a more significant negative impact on cardiovascular health.

The published literature has significant variability in treatment positions for the OA (Franson, 2003). Many studies evaluate OA with a fixed position at the patient's 50% protrusion for the range of motion of the mandible. Several studies explored the patient's treatment response to maxim capacity for mandibular advancement (Hoekema, et al., 2006). The optimal treatment response appeared to be obtained in patients who were able to achieve approximately 75% of protrusive range of motion (Gindre, et al., 2008). The device evaluated in this study was advanced in-office by the clinician. The patient was initially set at 50% of protrusive capacity. The advancement of the mandible was advanced 1-1.5 millimeters each adjustment. The average number of adjustments for the advancement protocol was 2 per patient.

The extant research criticizes OA treatment for a number of treatment fallibilities. The first issue is the concern about the lack of compliance monitoring of patient adherence to therapy (Johal & Battagel, 2001). The measure of adherence to treatment is by patient report of hour's usage per night and day's usage per week. In comparison, continuous positive airway pressure (CPAP) implements compliance monitors to measure patient usage. Patient reports of CPAP use have been reported to be significantly over-estimated compared to objective monitoring. Surgical

treatment does not have a concern for reporting patient usage since the surgical alteration is permanent.

Another concern observed by critics of OA devices is the ability to under-treat the patient with the device unknowingly. The primary problem is the variable patient-to-patient response in effectiveness (Lee, et al., 2009). There are no reliable predictors for patient selection for treatment. This variability in treatment necessitates objective monitoring of the therapy with either polysomnography or portable monitoring (PM). Patient sleep quality can be improved and snore treatment effective in patients despite the presence of persistent OSA (Henke, 2000). This persistent OSA, while having well-treated snore, has been reported in OSA surgical intervention, suboptimal CPAP pressure treatment and OA therapy. This under-treated patient has been labeled to have "silent" OSA (Krishnan, 2008). This silent OSA patient can believe there is effective treatment and remain at risk for sleepy driving accidents or cardiovascular disease (Kushida, et al, 2006)(Lim, et al., 2006).

There is a clinical advantage to the SleepAPP™ over other OA devices that can be adjustable. From a long-term tolerance standpoint, the patient well-treated with less mandibular advancement experiences less transient side effects (Gostopoulos, et al., 2002). If treatment is left to an acceptable subjective treatment response, without considering objective treatment of AHI and low oxygen saturation, the patient will trend toward less mandibular advancement simply to achieve snoring control (Krishnan, 2008). Most comparable OA devices that have the capacity to be advanced over time incrementally can also be easily adjusted by the patient. This patient-driven advancement may result in long-term under-treatment. The novel use of a thermoplastic in an OA allows for clinician adjusted treatment, avoiding the possible loss of

treatment effect by patient home adjustments. The ability to assure that the device is set at a point confirmed to be effective at treating OSA is unique to the SleepAPP™.

The literature recommends key points for future research to evaluate the effect of different appliance designs (Ferguson, et al., 2006). A standard criterion is recommended to be used as a benchmark for future research. Ongoing refinements of appliance design may eventually lead to improved outcomes with fewer complications (Tegelberg, 2003). Published literature now provides evidence for the efficacy of OA into the treatment of mild to moderate OSA (Ferguson, et al., 2006). This study has attempted to contribute to these recommendations. This clinical inquiry project could lead to more appropriate treatment with OA, thus more treatment options for patients with moderate and severe OSA. The clinical inquiry component of the doctor of nursing practice (DNP) represents a unique opportunity to bridge communication between dental professionals and medical professionals to provide improved care for patients with OSA.

Limitations

It is understood that the retrospective design and the clinical characteristics of the trial of this clinical inquiry project reduces the ability to generalize the findings. A randomized control trial (RCT) would be needed to inform cause and effect. The statistical analysis of treatment response was reduced patients who had significant weight gain. The weight gain may have resulted in a worsening of the baseline AHI in these patients. Having an updated baseline assessment for these patients could have provided better insight into the effects of weight gain on OSA severity.

The average attrition in the published literature was 11.5% with a range from 0 to 38% (Ferguson, et al., 2006). This trial was assessed in a clinical practice and revealed a level of

attrition at 38%. The PM studies that had invalid data were a significant source of lost follow up data and significantly higher than the 2% invalid results reported in the published literature for the WP100 (Penzel, et al., 2004). A limitation to the study was that it was performed by the medical device manufacture. The standard practice in OA research has been to have the OA medical device manufacture perform the research. Thus there is a possible bias in reporting of data in the published literature for OA. This trial did assess for sleep improvement and snoring improvement with a simple questionnaire; but did not utilize a validated qualitative assessment tool to monitor OSA symptoms. Recommendation for future research on the SleepAPP™ could be to conduct a randomized controlled trial (RCT) with a similar or larger sample size comparing OA to CPAP, OA to other types of OA, or OA to OSA surgery.

Conclusions

In conclusion, treatment with the SleepAPP™ OA reduces AHI and improves sleep quality in patients with moderate and severe OSA. The SleepAPP™ OA can effectively treat patients of more advanced age and BMI greater than 35 with moderate and severe OSA. Full response was more likely to occur in the moderate degree of OSA severity. Full response was more prevalent in the severe OSA group when compared to the published literature. The SleepAPP™ OA was well-tolerated by the majority of patients in this study. This study supports the currently accepted practice for follow-up sleep testing in patients with moderate and severe OSA. The majority of patients in this study found the OA to acceptably treat snoring and improve sleep quality despite under-treatment in many of the patients. The finding of silent OSA during treatment with OA is a potential concern and thus supports the published literature recommendations for objective monitoring of AHI response to OA (Marklund, 1998) (Kushida, 2006) (Ferguson, 2006). Patients were effectively assessed with the WP100 portable monitoring

unit. The WP100 device was well tolerated, but resulted in a high level of invalid test results in a clinical setting. Current evidence for the efficacy of OA is for the treatment of mild to moderate OSA. This CIP added to the OSA literature by demonstrating a well-tolerated and efficacious intervention for both moderate and severe OSA. Future trials could compare the SleepAPP^{TM} OA to more traditional CPAP by randomizing OSA patients into either standardized intervention. This CIP provides novel data that could lead to more treatment options for patients with moderate and severe OSA.

Acknowledgements

Thank you to my committee for your insight and guidance: Kim Jones, RN, MSN, PhD, Daniel O'Hearn, MD, Scott Mist, PhD, and Richard Moore, DDS.

References

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apnea with mandibular advancement appliances. *British Dental Journal*, 190, 532-536.

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Lee, C. H., Mo, J. H., Choi, I. J., Lee, H. J., Seo, B. S., Kim, D. Y., Yun, P. Y., Yoon, I. Y., Won Lee, H., & Kim, J. W. (2009). The mandibular advancement device and patient selection in the treatment of obstructive sleep apnea. *Archives of Otolaryngology -- Head & Neck Surgery, 135*(5), 439-444.

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Appendix

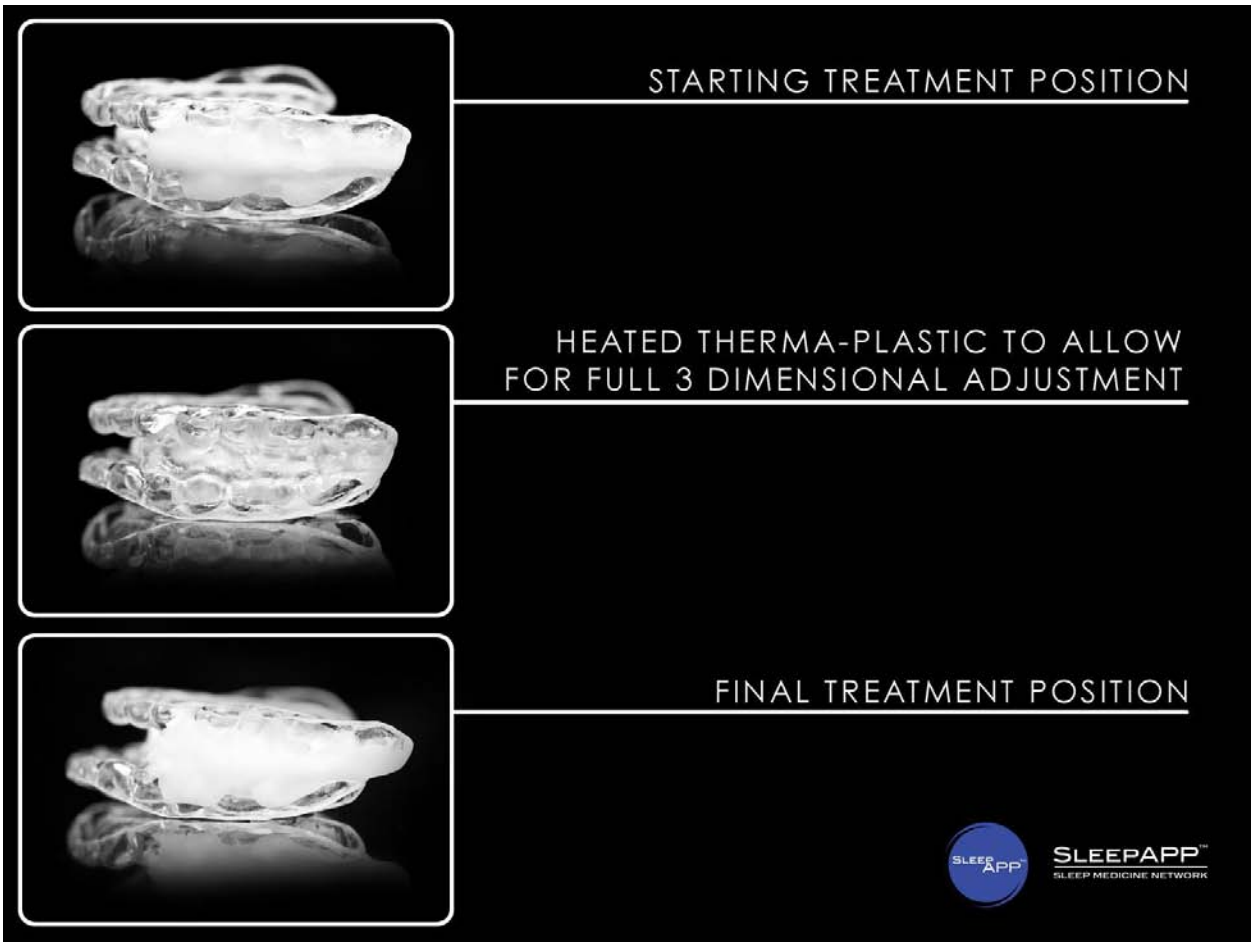

Figure. 1 Illustration of the OA at starting position, adjustment, and final treatment position.

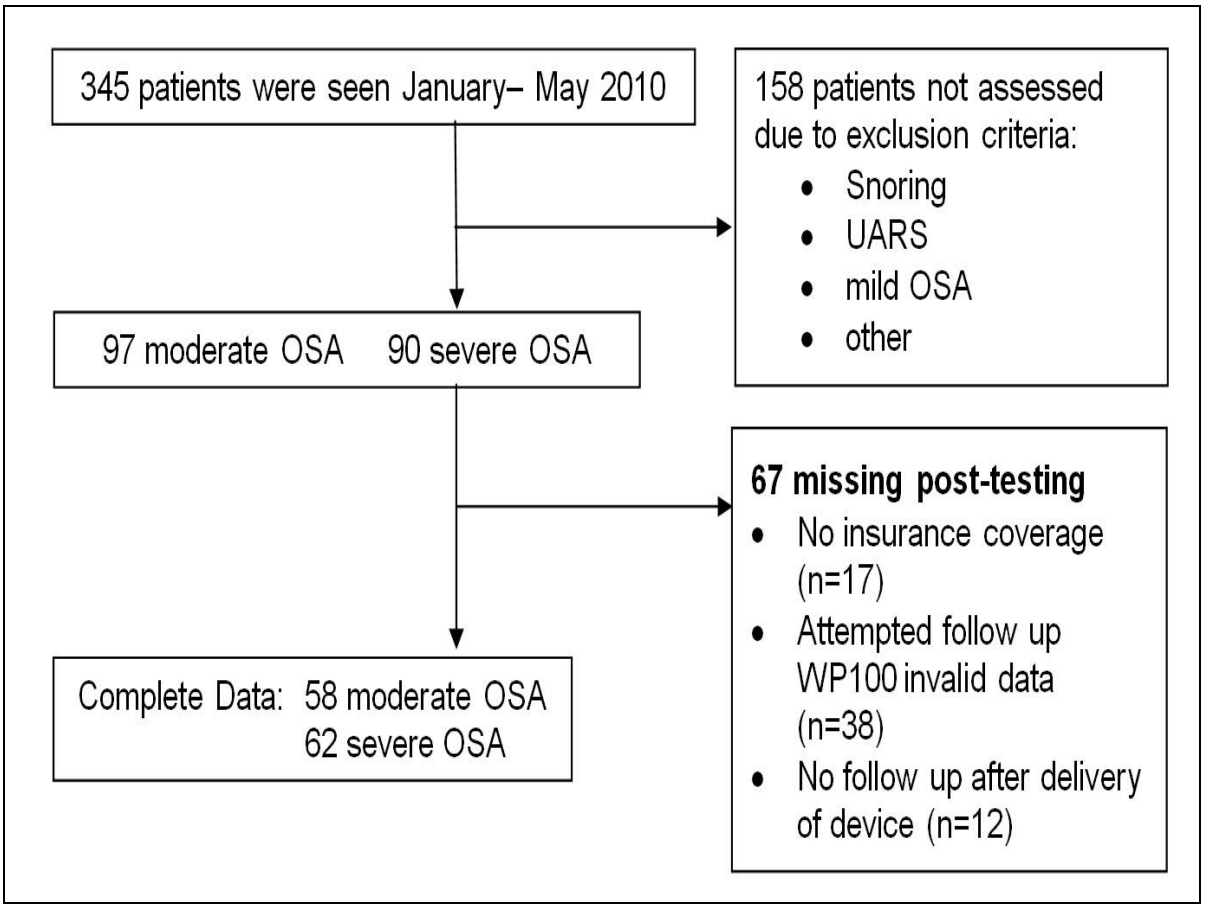

Figure 2. Consort Diagram of patient enrollment process.

Figure 2a. An example illustration of the portable monitor used to assess treatment response of OA.

Moderate	N	Min	Median	Max	Mean
AGE	58	26	46	77	47.48(11.8)
BMI	58	22.1	30.95	55.7	32.75(6.8)
WEIGHT	58	113	216	361	222.26(49.57)

Severe	N	Min	Median	Max	Mean
AGE	62	27	52	82	52.42(11.63)
BMI	62	13.9	34.7	57.4	35.97(8.5)
WEIGHT	62	135	235	450	248.35(62.95)

Table 1. Baseline demographic table for moderate and severe group.

Gender	
Male	n=79 (81.4%)
Female	n=18 (18.6%)
Ethnicity	
Caucasian	n=89 (91.8%)
African American	n=2 (2.1%)
Asian	n=3 (3.1%)
Hispanic	n=3 (3.1%)

Table 2. General demographic characteristics of study participants.

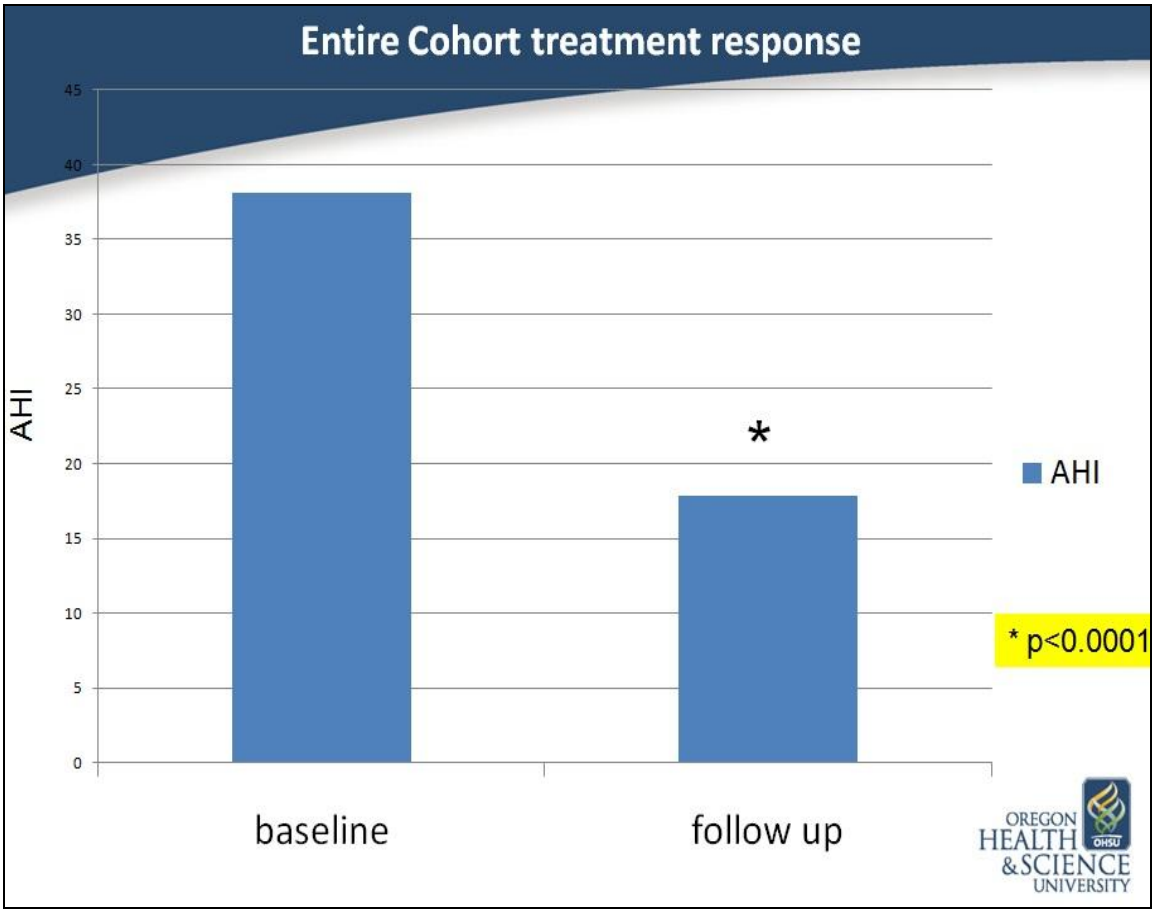

Figure 3. Entire cohort treatment response to OA therapy.

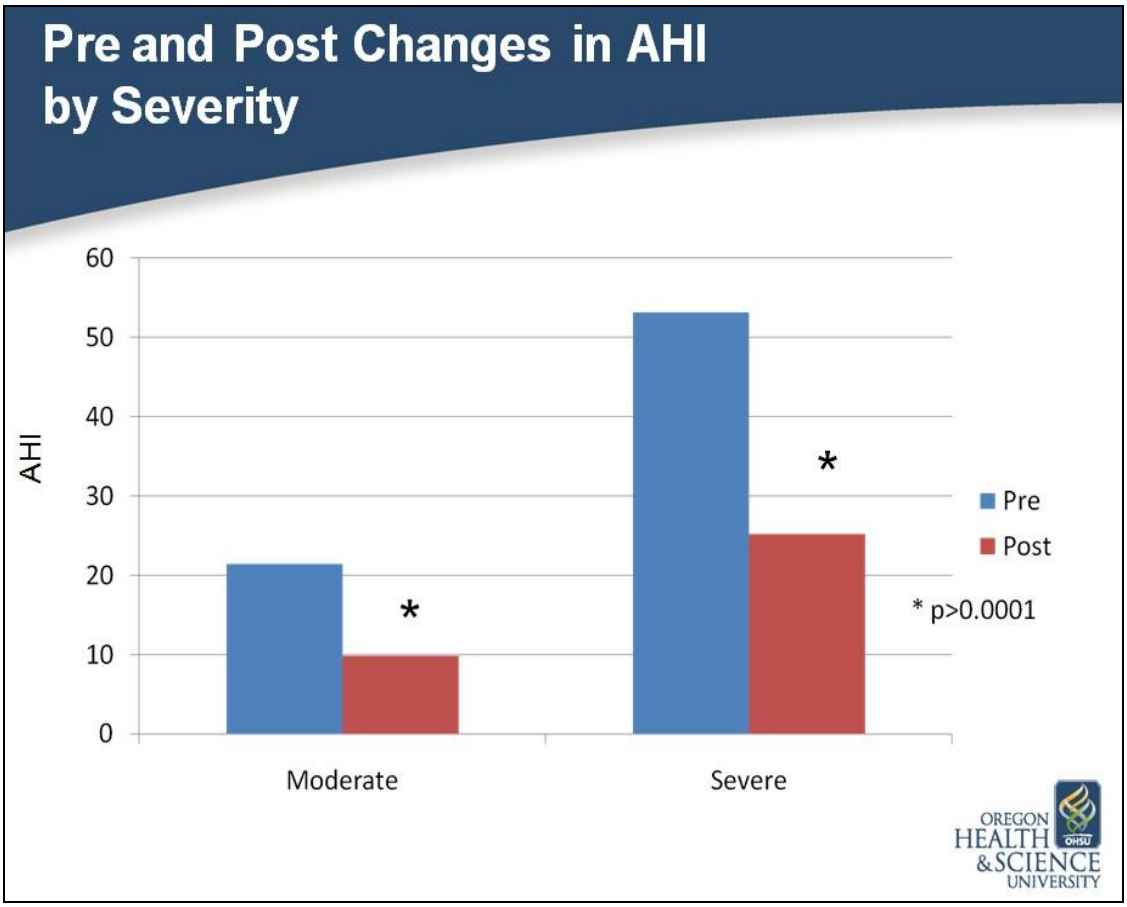

Figure 4. Pre and Post changes in AHI moderate vs severe.

Moderate AHI	N	Mean Std Err	Min	Median	Max	95% CL Mean		P-value
Baseline	58	21.42(4.60)	0.62	10.50	22.00	31.20	20.18	22.66	<.0001
Follow-up	58	9.84(6.98)	0.92	2.20	8.35	35.80	8.00	11.67	
Baseline-Follow-up		11.58(5.94)	1.12				9.39	13.78	

Severe AHI	N	Mean Std Err	Min	Median	Max	95% CL Mean		P-value
Baseline	62	53.15(19.84)	2.54	30.00	46.00	105.00	48.07	58.23	<.0001
Follow-up	62	25.25(21.64)	2.75	0.80	19.00	109.00	19.75	30.75	
Baseline-Follow-up		27.90(20.77)	3.75				20.49	35.32	

Table 4. Baseline and follow up assessment of OA treatment response.

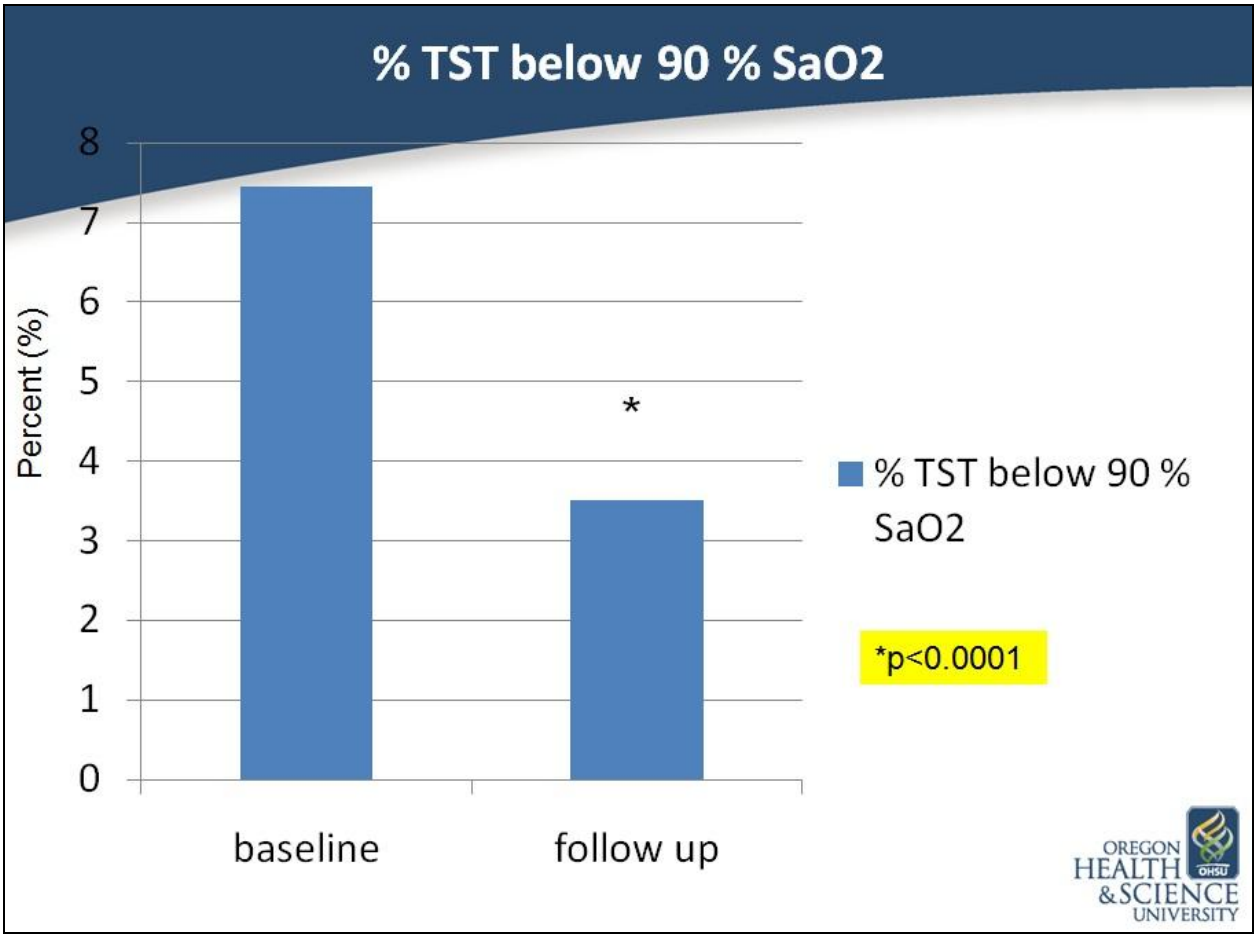

Figure 5. Pre and post test % total sleep time spent below 90% oxygen saturation.

Entire Cohort time below 90 % SaO2	N	Mean	Min	Median	Max	P-value
Baseline	120	7.45(11.53)	0.00	2.75	59.00	
Follow-up	120	3.52(7.86)	0.00	0.20	43.40	<.0001
Baseline-Follow-up		3.93(9.83)				

Table 5. Pre and post test % total sleep time spent below 90% oxygen saturation table.

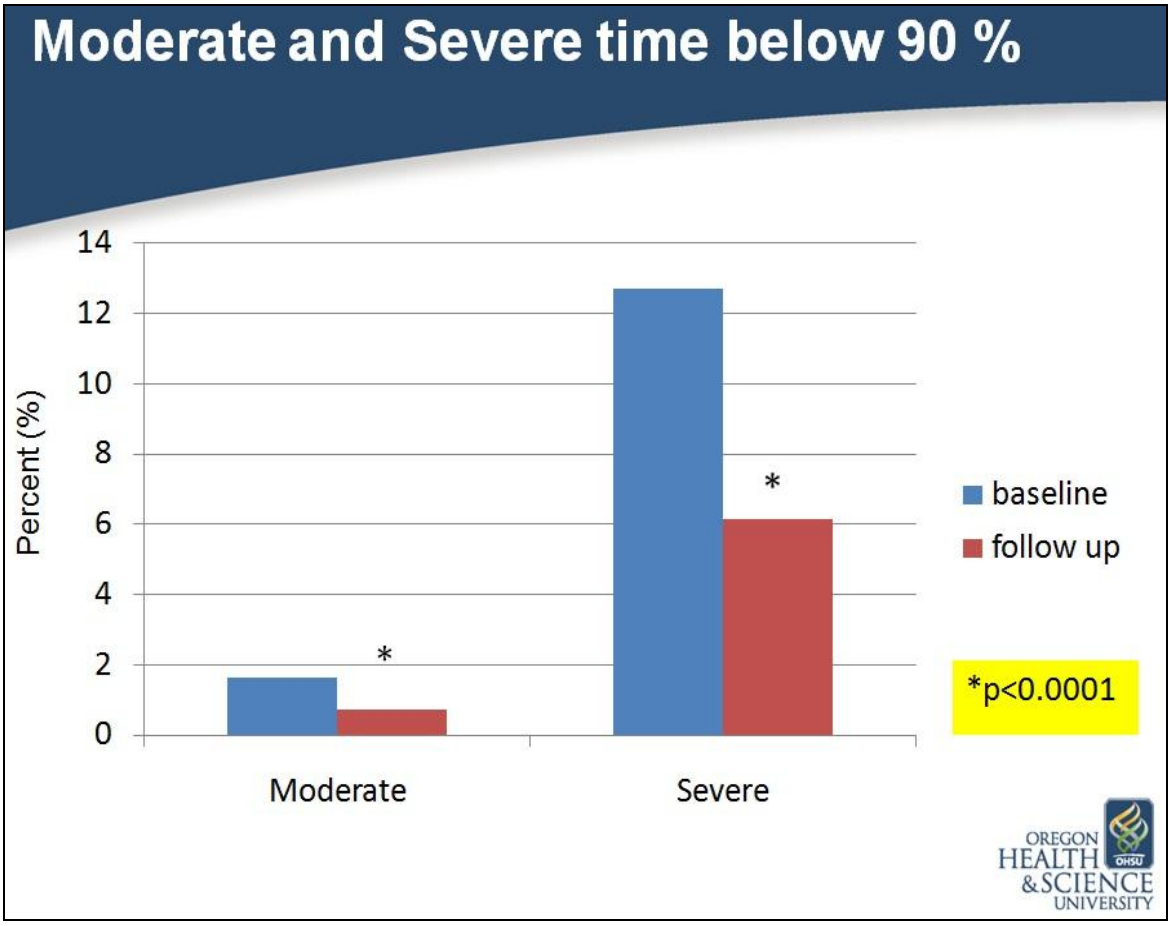

Figure 6. Comparison graph moderate vs severe group response to oxygen saturation.

Moderate Time spent below 90%	N	Mean	Min	Median	Max	P-value
Baseline	58	1.63(2.13)	0.00	0.60	7.60	<.0001
Follow-up	58	0.71(2.52)	0.00	0.00	17.50	
Baseline-Follow-up		0.93(2.34)				

Severe Time spent below 90%	N	Mean	Min	Median	Max	P-value
Baseline	62	12.70(13.84)	0.20	9.10	59.00	<.0001
Follow-up	62	6.16(10.00)	0.00	1.25	43.40	
Baseline-Follow-up		6.54(12.06)				

Table 6. Comparison table moderate vs severe group response to oxygen saturation.

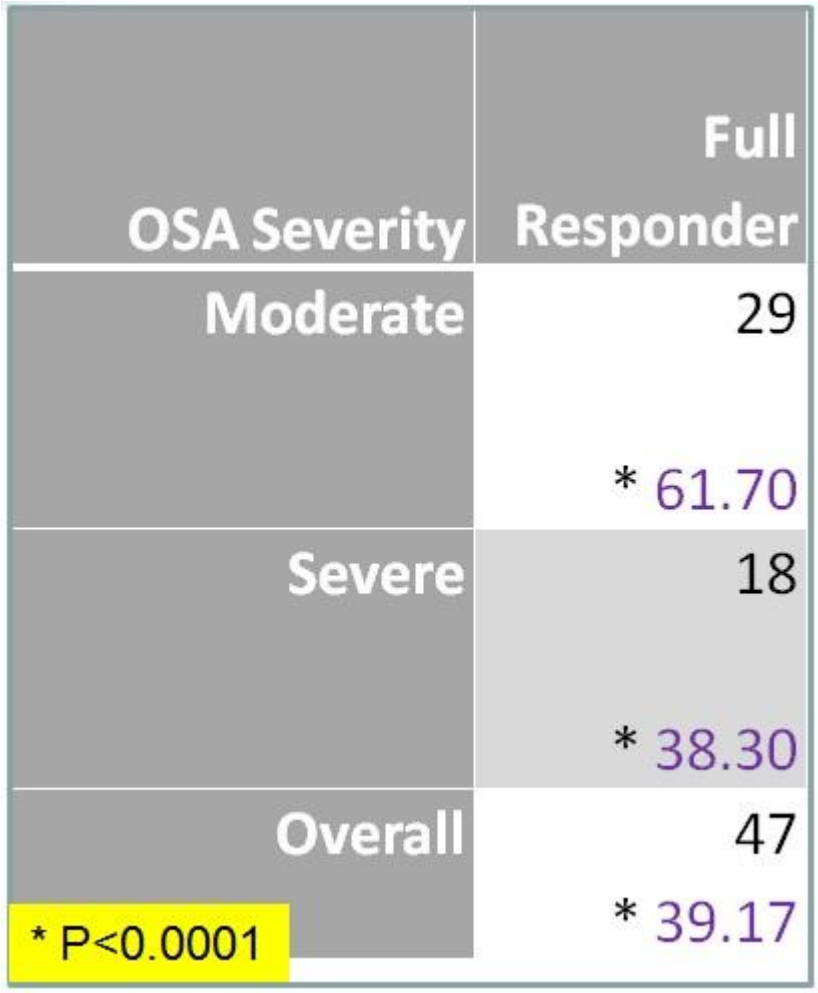

Table 6. Full responder moderate group, severe group and overall response.

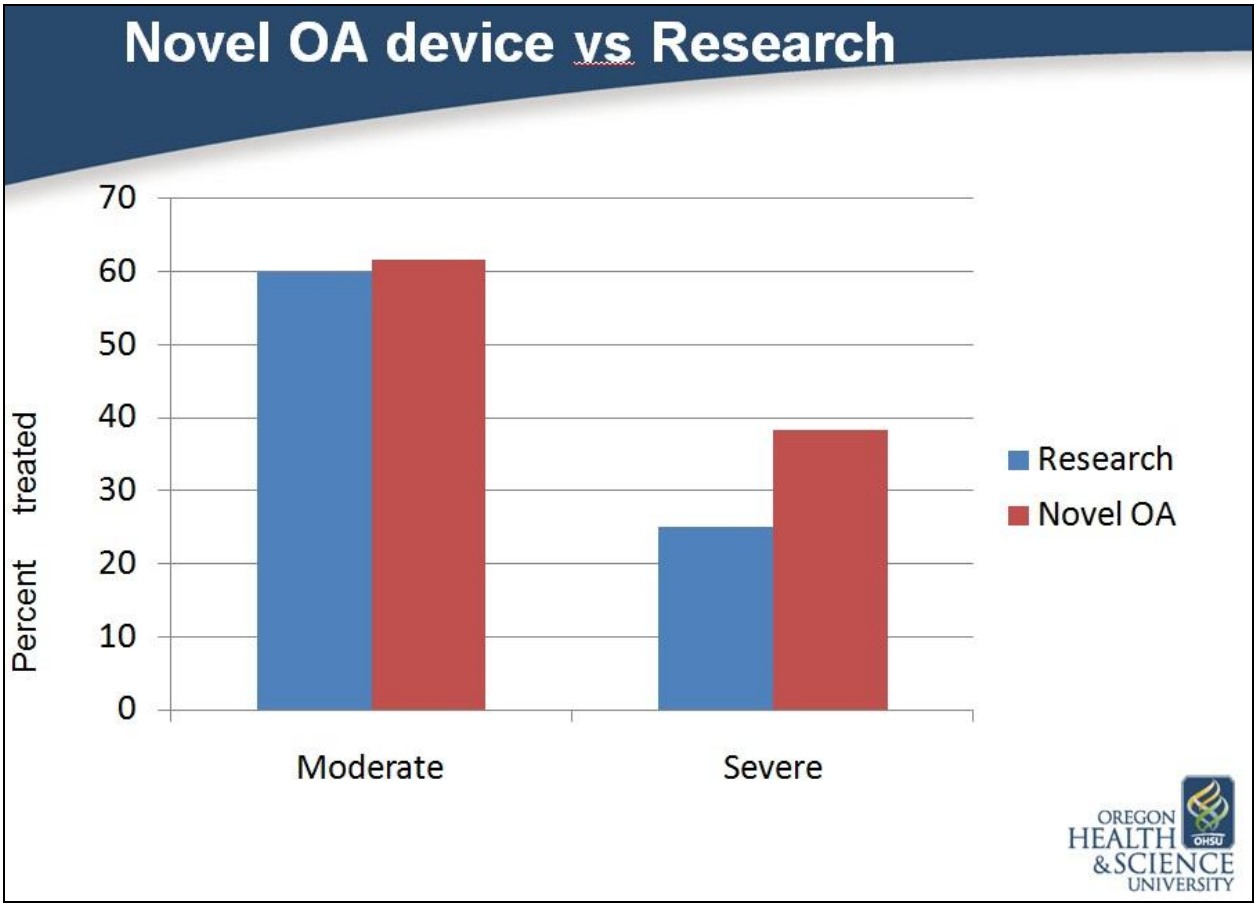

Figure 7. Comparison of treatment response between the novel OA and published research.

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The use of a novel oral appliance in the treatment of moderate and severe obstructive sleep apnea

Joseph Zelk, M.S., F.N.P. B.C.

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA)

Associated with CVA, MI, HTN, arrhythmias, and motor vehicle accidents (Mansfield & Naughton, 2005).

9 % of middle-aged women, 25 % of middle-aged men, 60 % of people between the ages of 50 and 70 (Young, 1993).

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Routine Polysomnograph Montage

- LOC/A2
- ROC/A1
- C3/A2;C4/A1
- O2/A1;O1A2

- Submental/Masseter EMG
- ECG
- Anterior tibialis EMG (R/L)
- Nasal/Oral airflow
- Thoracic respiratory effort
- Abdominal respiratory effort
- O_2 saturation
- Position monitor

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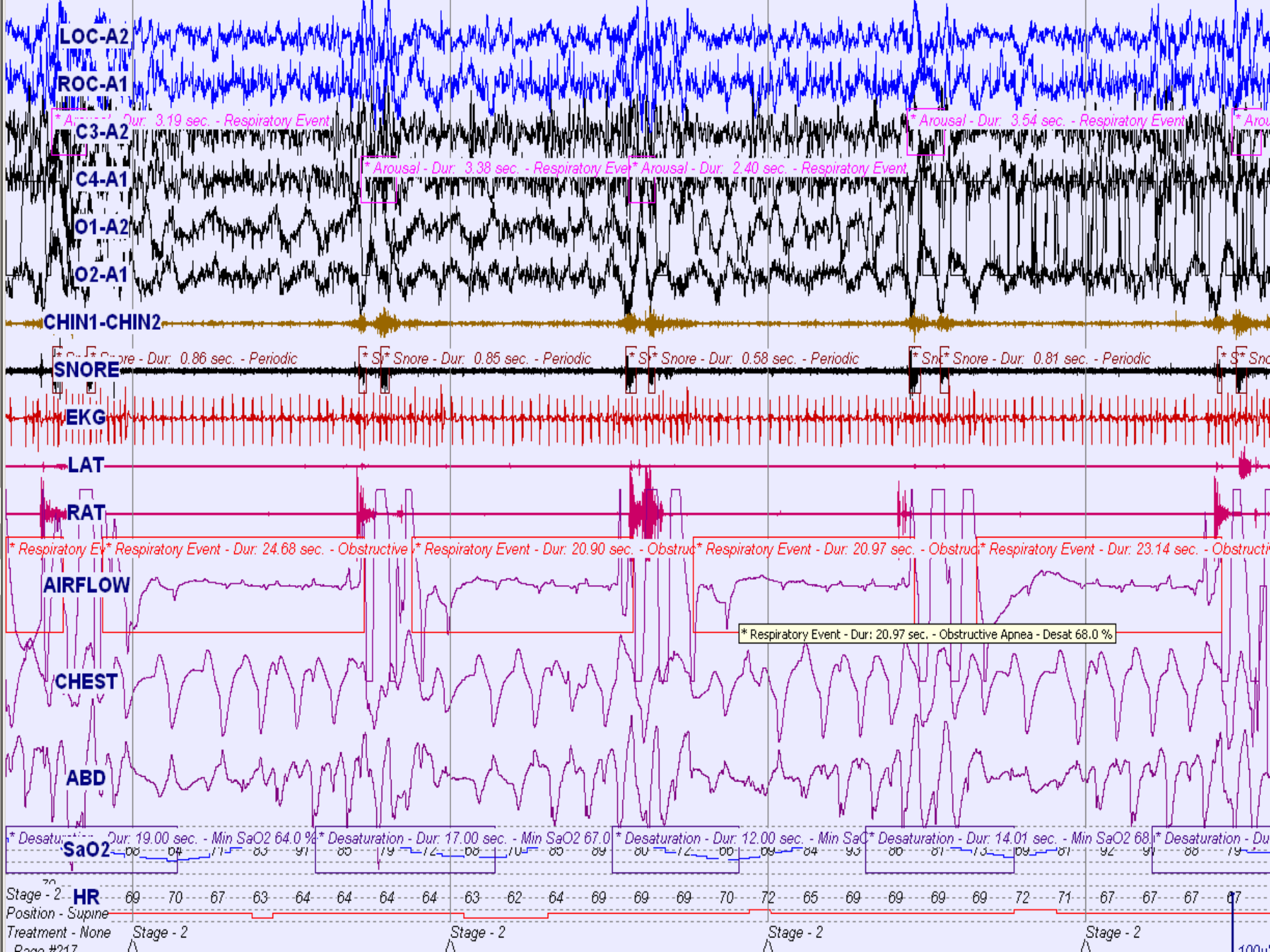
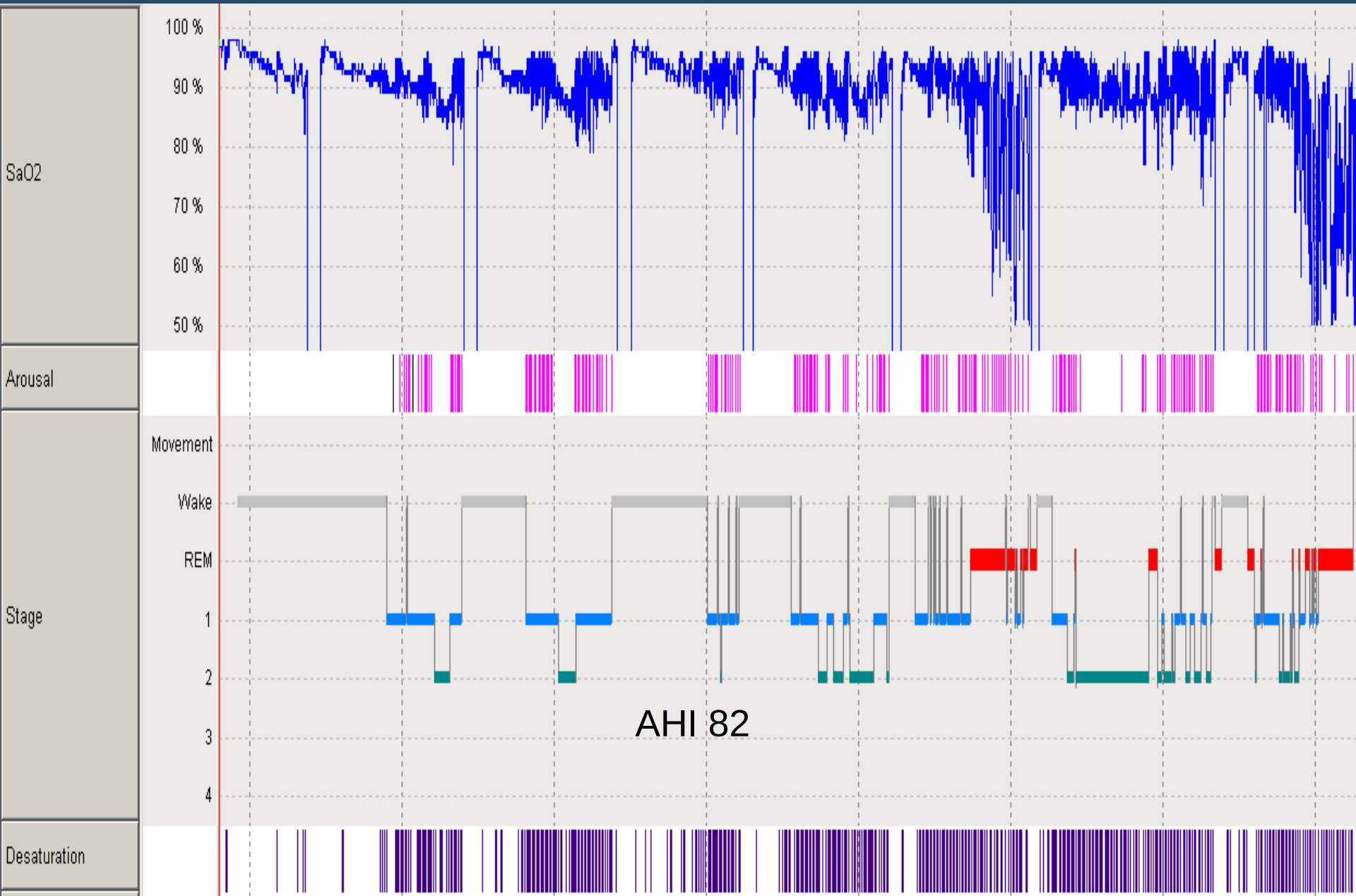
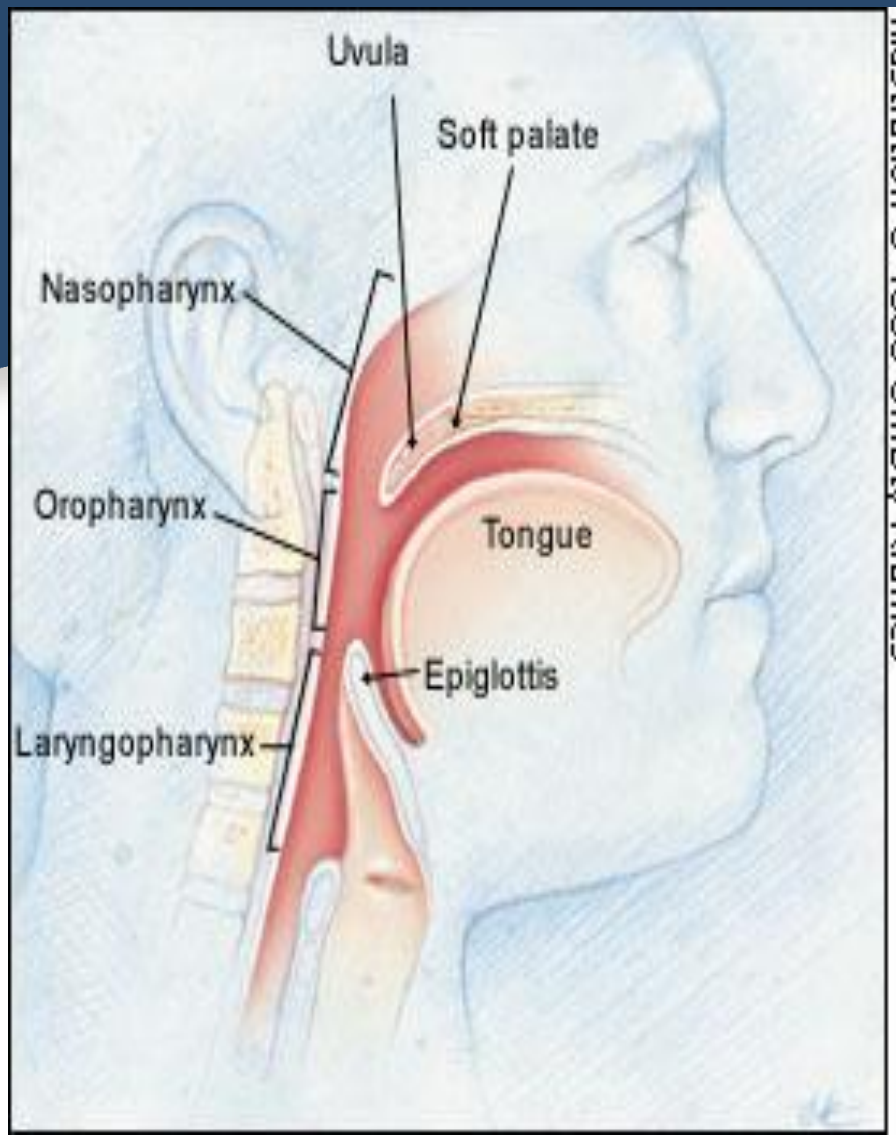

LOC-A2
ROC-A1
C3-A2
C4-A1
O1-A2
O2-A1
CHIN1-CHIN2
SNORE
EKG
LAT
RAT
AIRFLOW
CHEST
ABD
SaO2
HR
* Arousal - Dur: 3.19 sec. - Respiratory Event
* Arousal - Dur: 3.38 sec. - Respiratory Event
* Arousal - Dur: 2.40 sec. - Respiratory Event
* Arousal - Dur: 3.54 sec. - Respiratory Event
* Snore - Dur: 0.85 sec. - Periodic
* Snore - Dur: 0.58 sec. - Periodic
* Snore - Dur: 0.81 sec. - Periodic
* Respiratory Event - Dur: 24.68 sec. - Obstructive
* Respiratory Event - Dur: 20.90 sec. - Obstruc
* Respiratory Event - Dur: 20.97 sec. - Obstruc
* Respiratory Event - Dur: 23.14 sec. - Obstructi
* Respiratory Event - Dur: 20.97 sec. - Obstructive Apnea - Desat 68.0 %
* Desaturation - Dur: 19.00 sec. - Min SaO2 64.0 %
* Desaturation - Dur: 17.00 sec. - Min SaO2 67.0
* Desaturation - Dur: 12.00 sec. - Min Sa
* Desaturation - Dur: 14.01 sec. - Min SaO2 68
Stage - 2
Position - Supine
Treatment - None
Stage - 2
Stage - 2
Stage - 2
Stage - 2

An example of a night of severe OSA





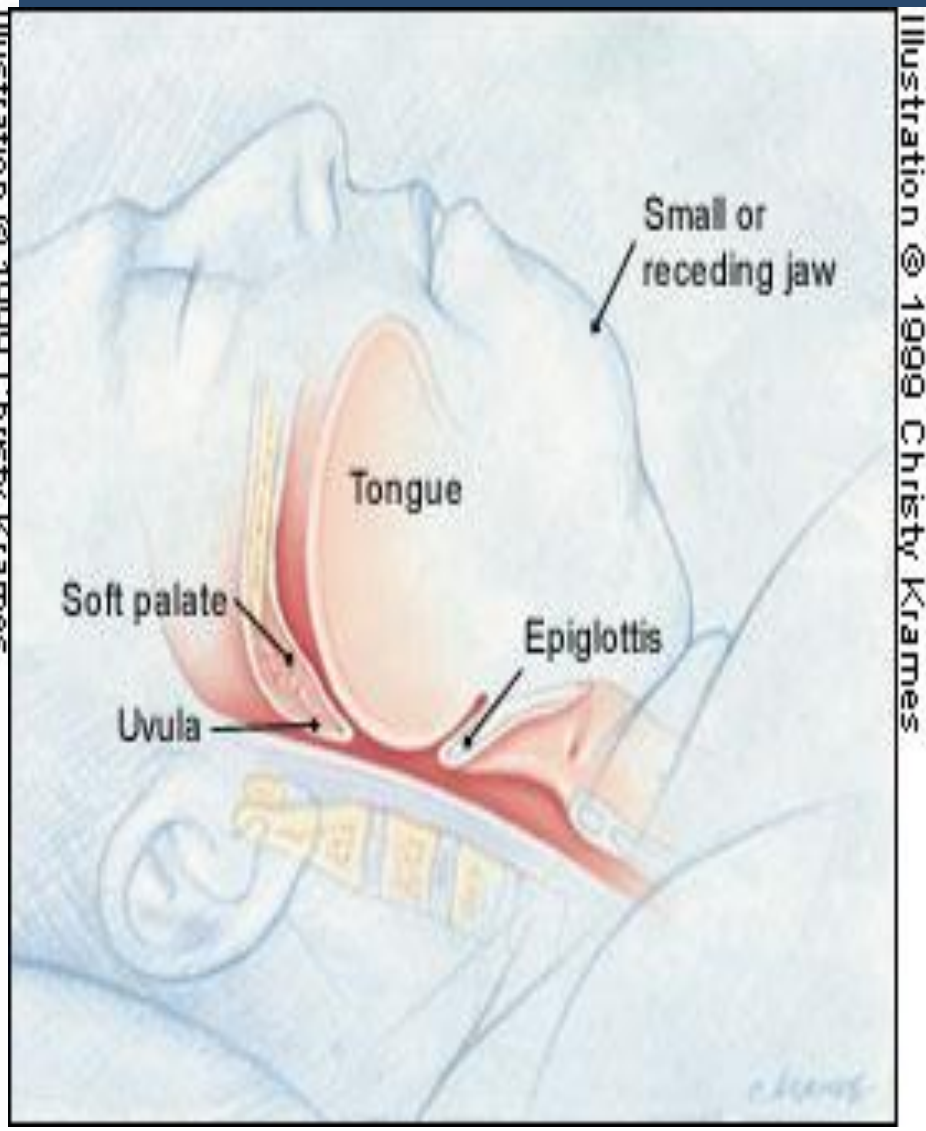
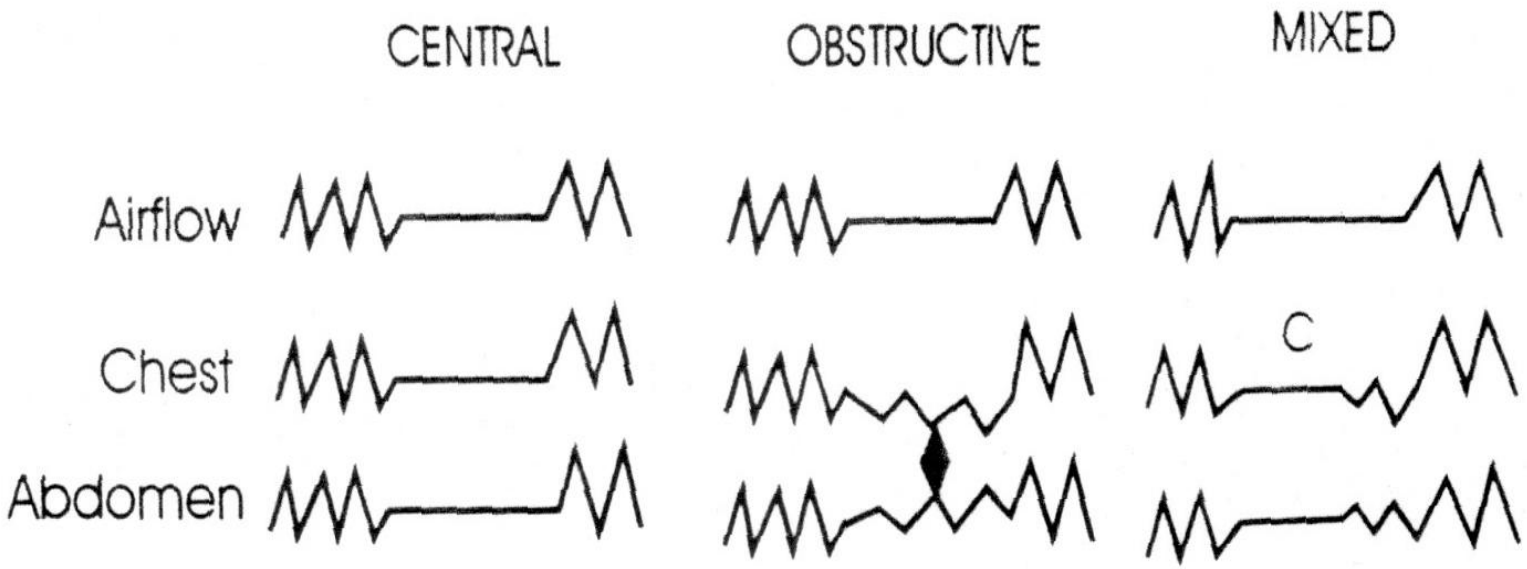


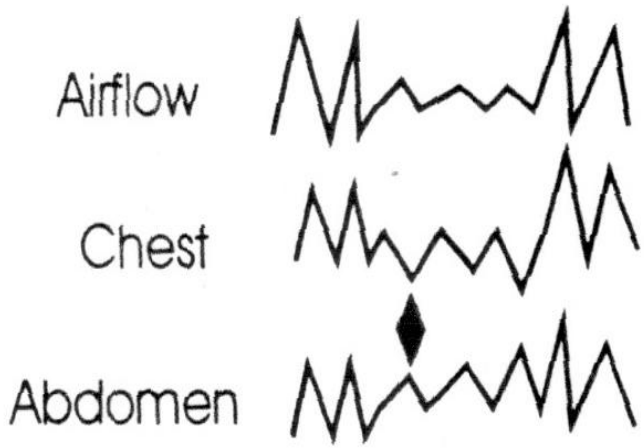
Illustration © 1999 Christy Krames

Pharyngeal collapse is due to a complex interaction of structural and neuromuscular factors.

APNEA TYPES



HYPOPNEA



♦ Paradoxical Movement

Severity Classification of OSA

- Apnea Hypopnea Index (AHI)
- AHI 5-15 = mild
- AHI 15-30 = moderate
- AHI >30 = severe
- Primary Snoring without pathology

Polysomnography vs Portable Monitoring (PM)

Polysomnography average ($): 2400

Portable monitoring average ($): 150-450

WatchPAT 100 sleep diagnostic device

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Clinical Problem

The most common treatment of Obstructive Sleep Apnea is continuous positive airway pressure (CPAP) (Netzer, 2003).

Adherence to CPAP is a concern.

4 hours/night, 5 days/week, 46-83% of patients non-adherent (Weaver, 2008).

Evidence suggests >6 hours a night is needed for treatment of excessive daytime somnolence (Weaver, 2008).

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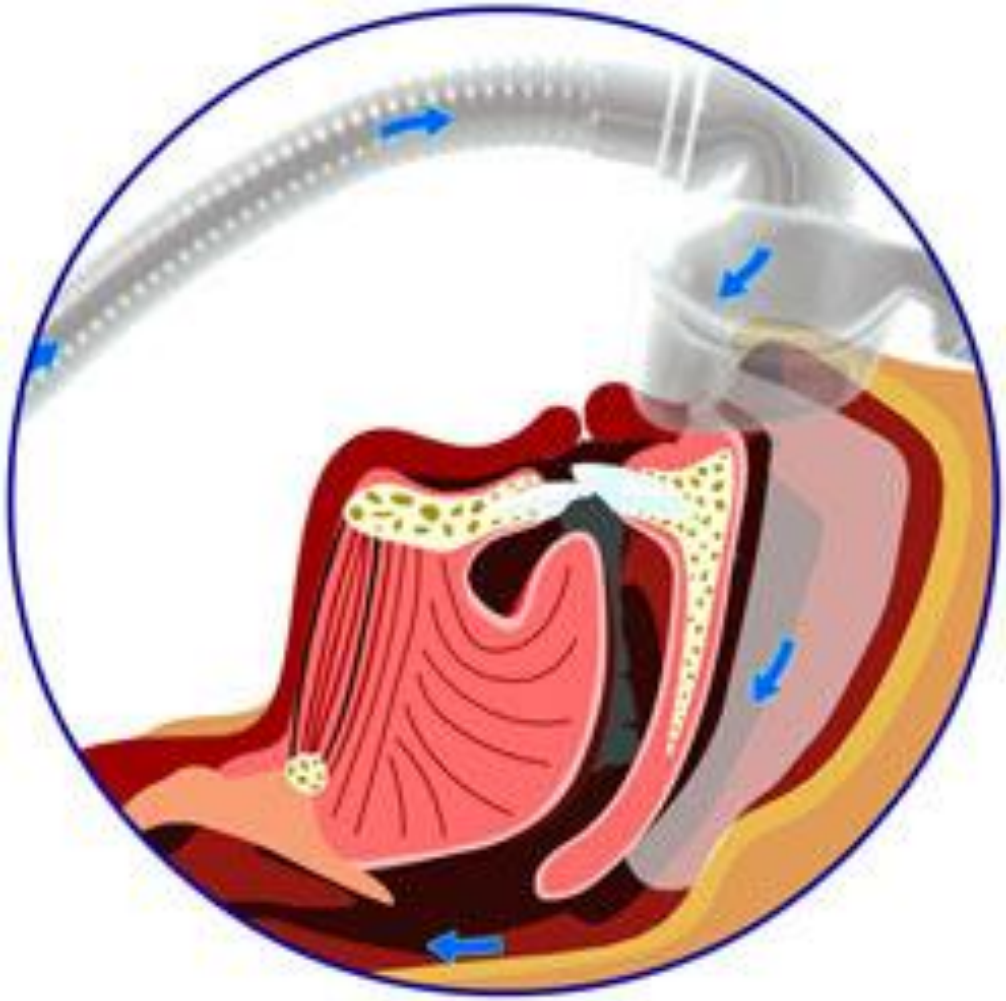
CPAP



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Alternatives to CPAP

- BMI > 30 = weight loss
- Side sleep therapy for supine related OSA
- Surgical intervention
- **Oral Appliance therapy (OA)**

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Aims of Clinical Inquiry

- Evaluate the efficacy of a novel oral appliance (SleepAPP™) to reduce sleep disordered breathing (moderate and severe OSA).

- Utilize new portable monitoring to test OA therapy (WatchPAT 100).

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SleepAPP™

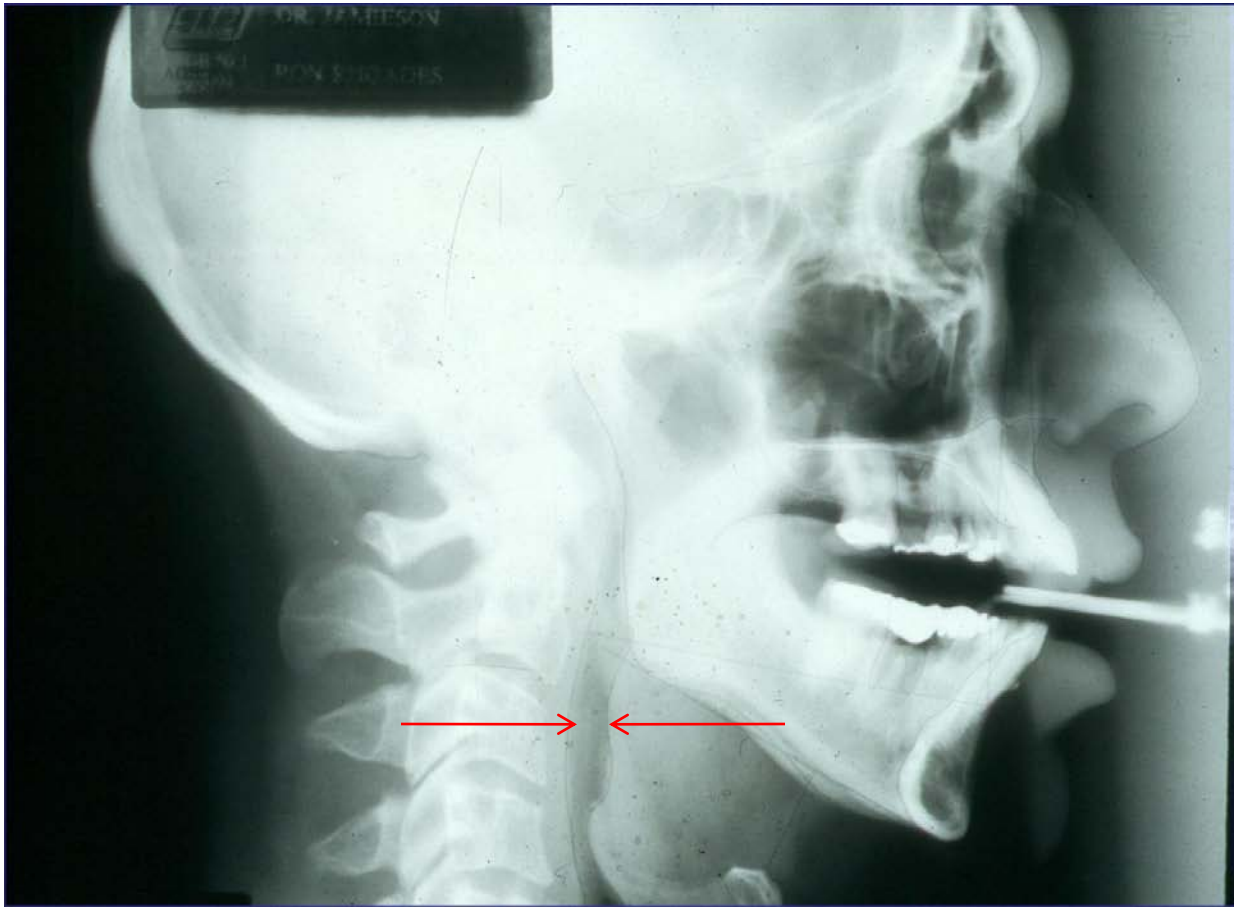
STARTING TREATMENT POSITION

HEATED THERMA-PLASTIC TO ALLOW
FOR FULL 3 DIMENSIONAL ADJUSTMENT

FINAL TREATMENT POSITION

SLEEPAPP™
SLEEP MEDICINE NETWORK

Radiograph – baseline awake



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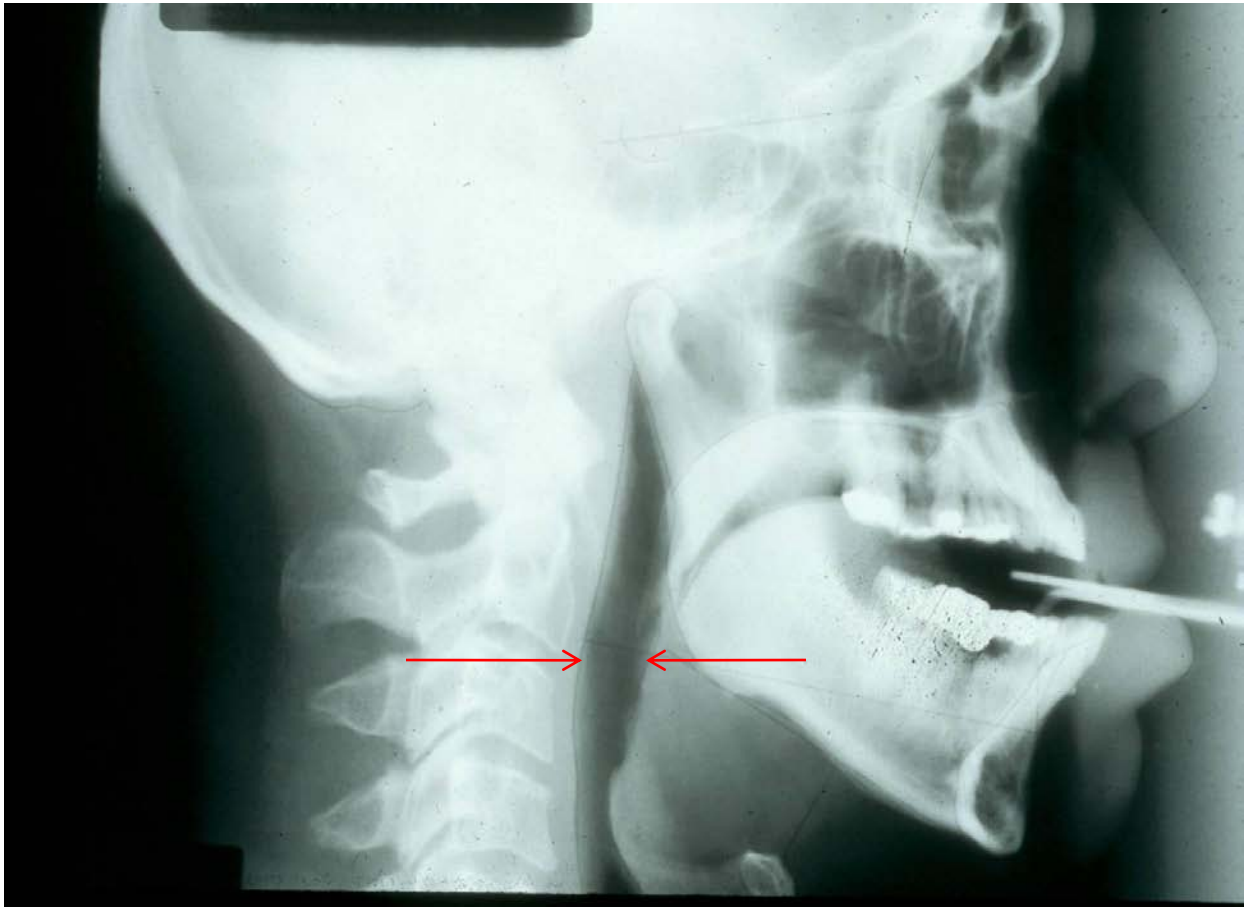
Radiograph – treatment awake



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Hypothesis Statement

The SleepAPP™ oral appliance (OA) will treat moderate or severe obstructive sleep apnea as well as, or more effectively than other oral appliances in the medical literature.

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The Effect of Oral Appliances on OSA

AHI <10

Mild OSA (81%)

Moderate (60%)

Severe (25%)

<50% AHI

Mild OSA (48%)

Moderate (67%)

Severe (38%) (Marklund, 1998)

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Author	Study Design	Sample Size	Severe Y/N
Marklund, 1998	Prospective	47	Y
Vecchierini, 2008		35	N
Lam, 2011		10	N
El-Solh, 2010		34	N
Clark, 1993		15	N
Eveloff, 1994		19	N
O'Sullivan, 1995		51	N
Marklund, 2001		33	N
Aarab, 2011	Retrospective	43	N
Martinez-Gomis, 2010		40	N
Doff, 2010	RCT	51	N
Macguire, 2010		52	N
Zhang, 2009		46	N
Ferguson, 1996		19	N
Bloch, 2000		19	N
Randerath, 2002		20	N
Gotsopoulos, 2002		73	N
Mehta, 2001		28	Y
Engleman, 2002		51	N

Literature Gap Addressed by this Study

- Novel appliance that allows for mandibular advancement by clinician, improved compliance for prescribed treatment
- Larger sample size
- Combined criteria for successful treatment

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Study Design and Setting

- Retrospective analysis of consecutive patients treated with the SleepAPP™.
- Setting: Sleep disorders clinic
- 187 consecutive moderate to severe OSA patients who received treatment with SleepAPP™ seen between January 1, 2010 and May 31, 2010

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Inclusion Criteria

- Adults age 21-85 years
- Patient's with moderate and severe OSA who are either intolerant or refuse treatment with CPAP
- OSA patients who have refused or who are not candidates for tonsillectomy, adenoidectomy, maxillomandibular osteotomy and tracheostomy

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Exclusion criteria

Standard exclusion criteria for watchPAT100 and OA

- Patients with primary CSA, snoring, UARS, mild OSA
- Patients with unstable TMD
- Patients max vertical <25 mm
- Active orthodontics, or surgery
- Advanced COPD, or CHF
- Pacemaker
- Atrial Fibrillation
- >200 mg morphine dose (Equivalents)
- Alpha-adrenergic blocker

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Treatment Protocol

- WatchPAT100 diagnostic testing was done at baseline
- Patient underwent mandibular advancement protocol of OA.
  - Gradual advancement of the appliance starting at 50% of mandibular protrusive capacity with 1 mm adjustments as tolerated.
  - 8-16 weeks after commencement of oral appliance therapy.

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Measures

- Primary outcome variable: AHI - Effective treatment is a reduction of AHI to ‹10 **and** a reduction of AHI by ≥50%

- Secondary outcome variable: percent time spent below 90% SaO_2 of total sleep time

- Exploratory variables: sex, age, ethnicity, height, weight, BMI

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Statistical Methods

- 2 Group Mean Comparison t-test to compare the pretreatment and post-treatment AHI.
- Wilcoxon rank sum test was used to compare the pretreatment and post-treatment percent time below SaO_2 90%.
- Chi-square test to assess for groups differences.

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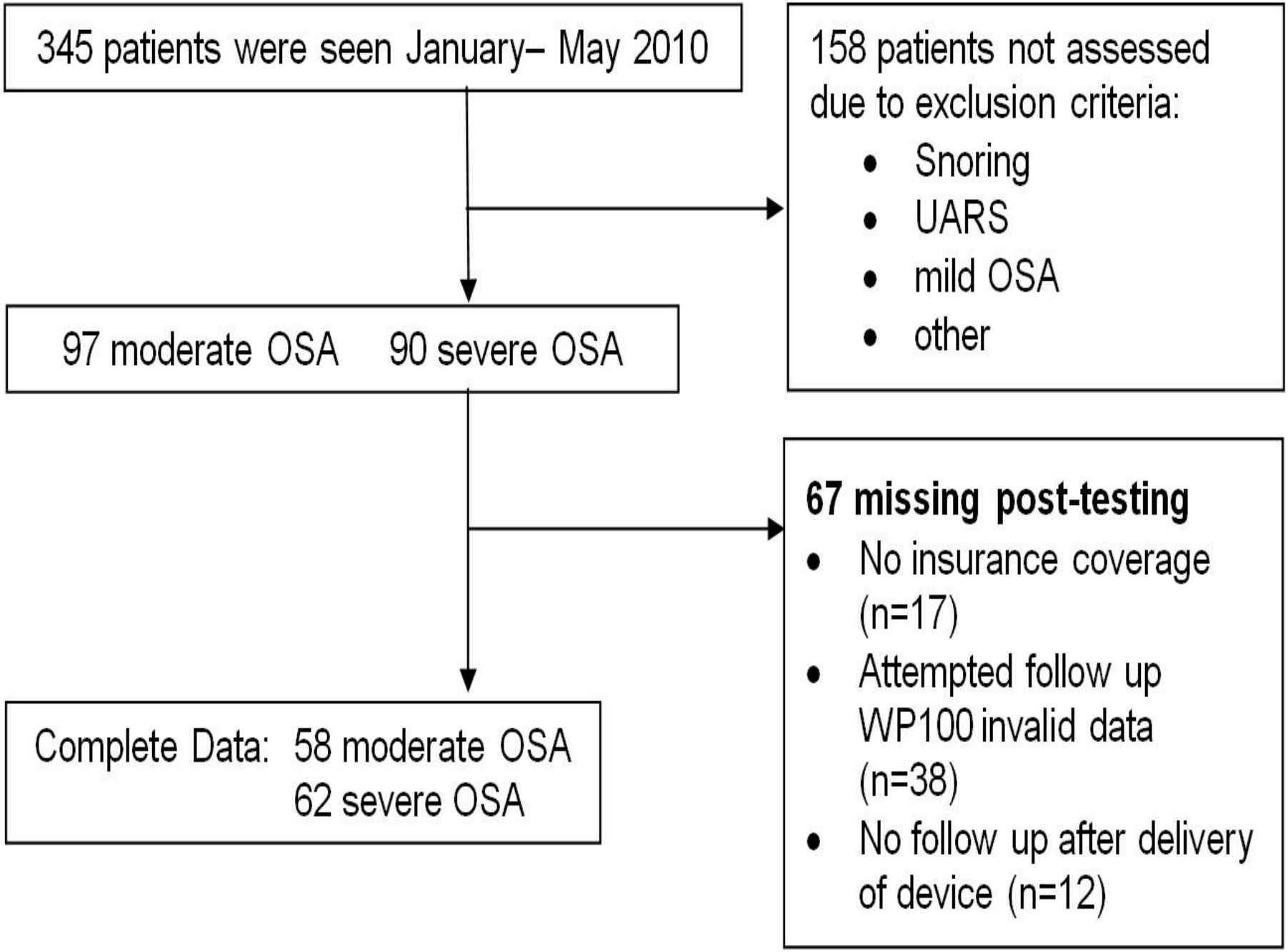
345 patients were seen January– May 2010
158 patients not assessed due to exclusion criteria:
Snoring
UARS
mild OSA
other
97 moderate OSA
90 severe OSA
67 missing post-testing
No insurance coverage (n=17)
Attempted follow up WP100 invalid data (n=38)
No follow up after delivery of device (n=12)
Complete Data: 58 moderate OSA
62 severe OSA

Baseline Group Characteristics

Gender / Severity	Male	Female
Moderate	n=79 (81.44%)	n=18 (18.56%)
Severe	n=78 (86.67%)	n=12 (13.33%)

Severity	
Moderate	n=58 (48.3%)
Severe	n=62 (51.7%)

Gender	
Male	n=79 (81.4%)
Female	n=18 (18.6%)

Ethnicity	
Caucasian	n=89 (91.8%)
African American	n=2 (2.1%)
Asian	n=3 (3.1%)
Hispanic	n=3 (3.1%)

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Baseline Group Characteristics Continued

Moderate	N	Mean (S.D.)
AGE	58	47.5 (11.8)
BMI	58	32.7 5 (6.8)
WEIGHT	58	222.3 (49.6)

Severe	N	Mean (S.D.)
AGE	62	52.4 (11.6)
BMI	62	36.0 (8.5)
WEIGHT	62	248.4 (63.0)

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Baseline Demographics of Population not in Analysis

Variable	N	Mean(S.D.)
AGE	67	46.3(12.1)
BMI	67	34.0(6.1)
WEIGHT	67	239.8(50.7)

Sex	Percent
Male	83.96
Female	16.04

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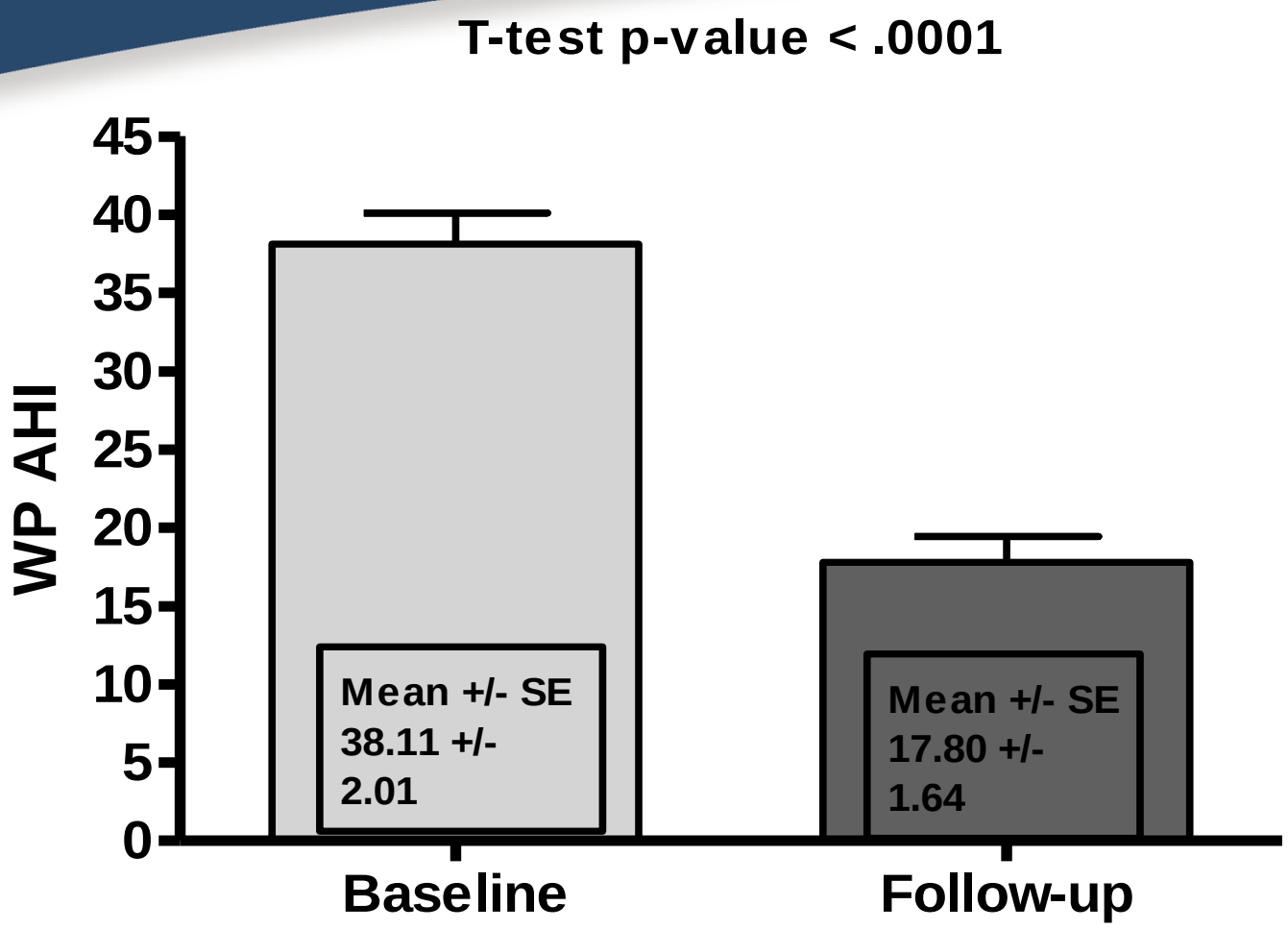
Treatment Response



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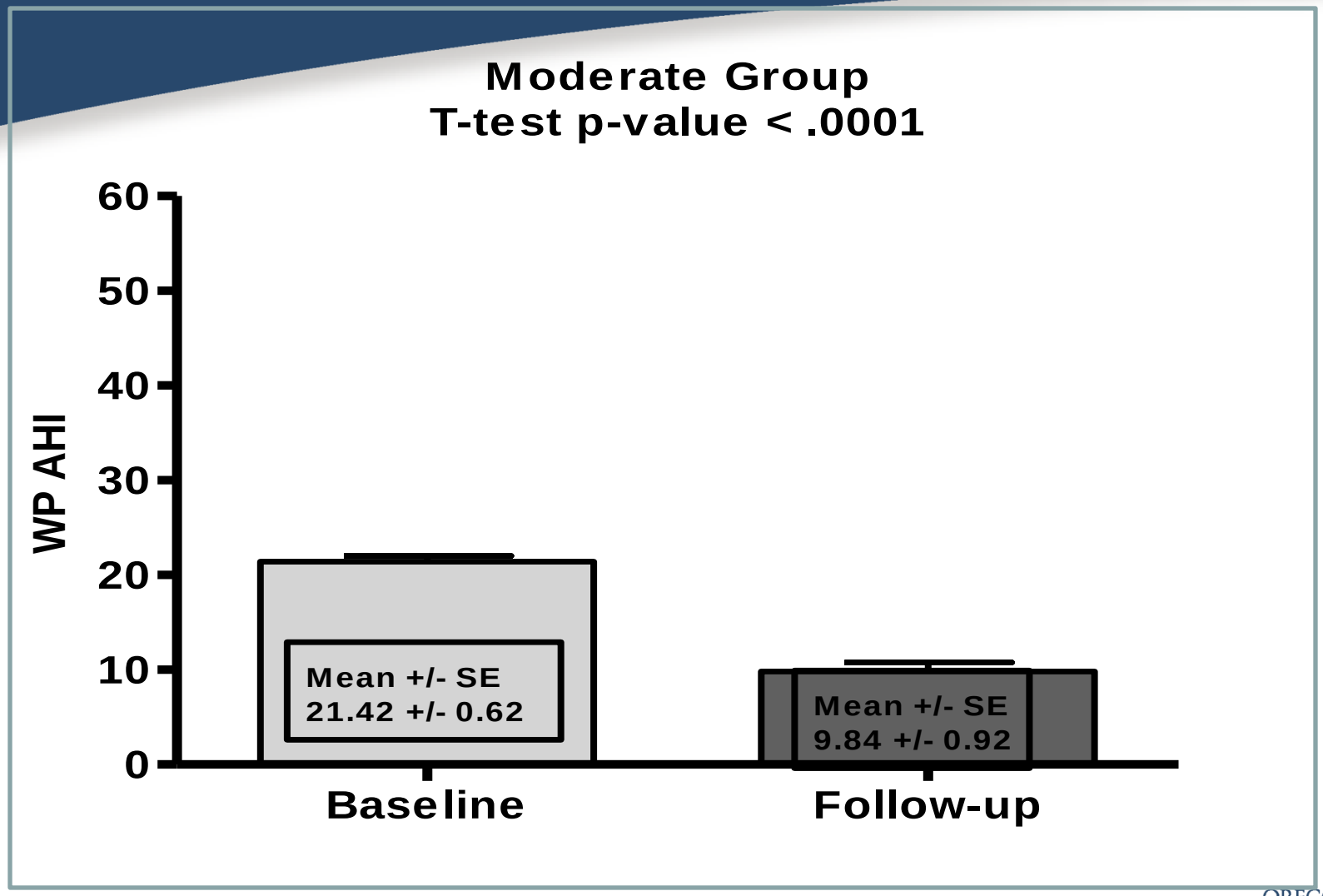
Moderate Group Treatment Response



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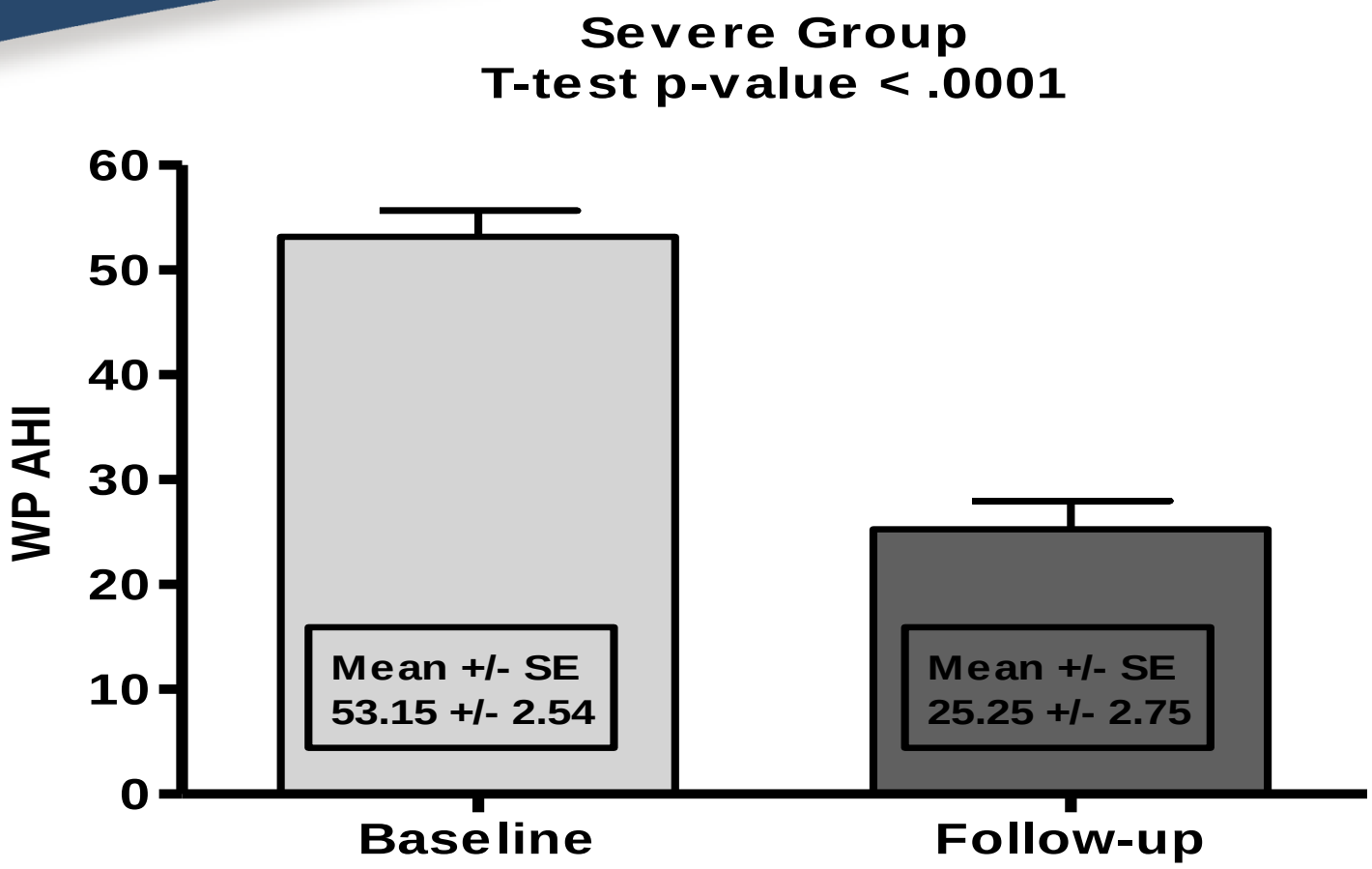
Severe Treatment Response



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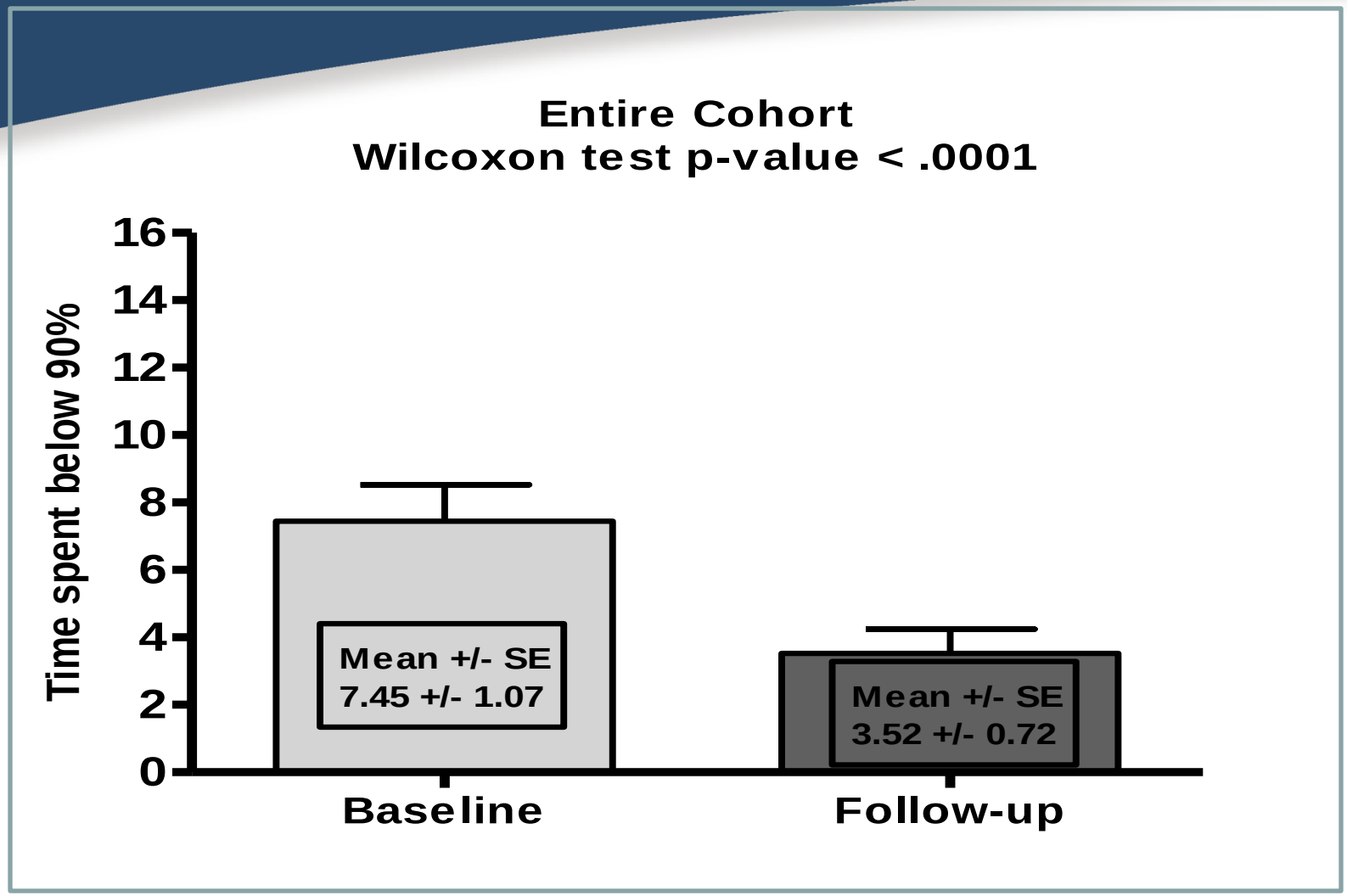
Improvement of % time spent below 90 % SaO_2



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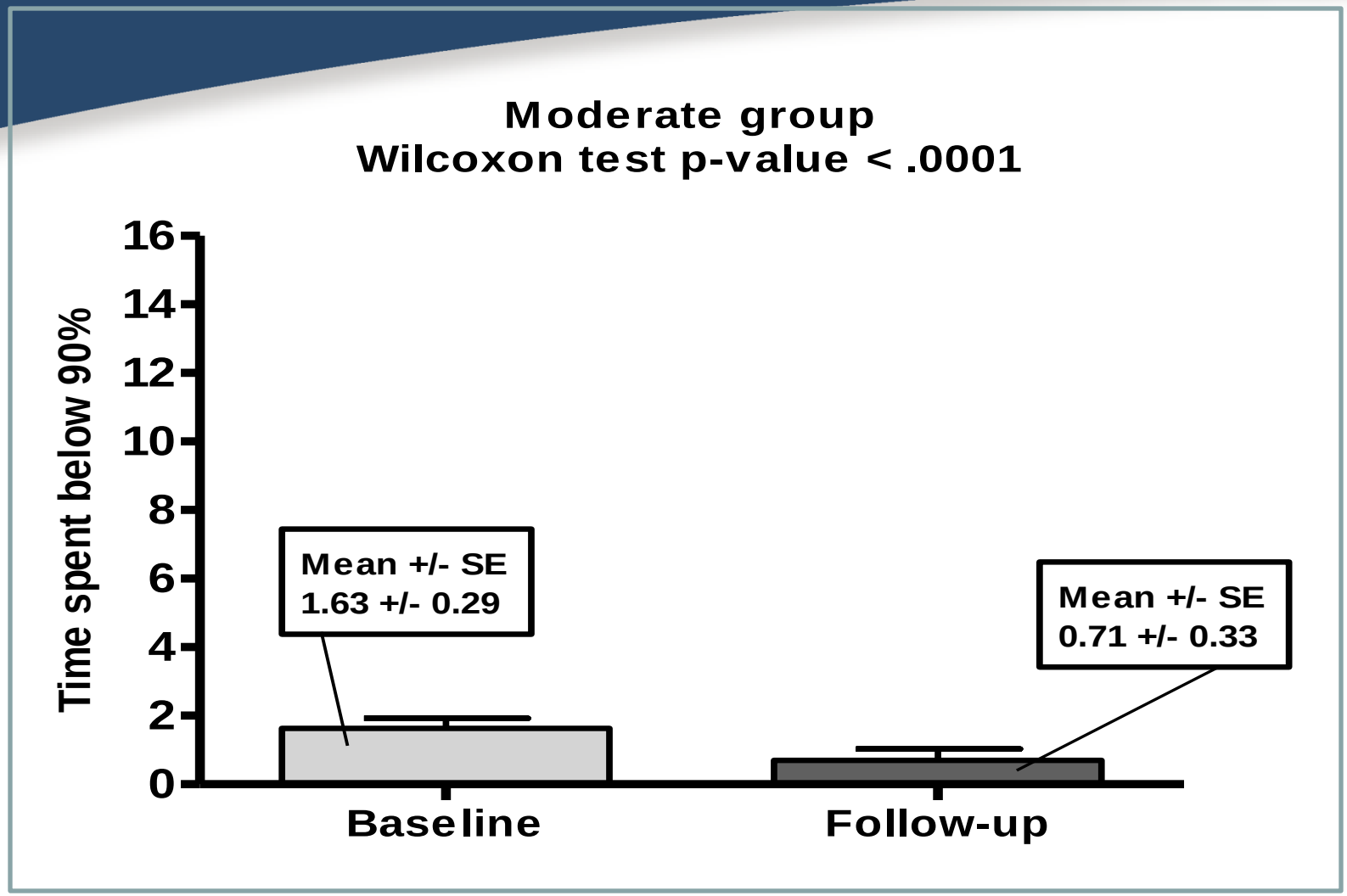
Moderate
Improvement of % time spent below 90 % SaO_2



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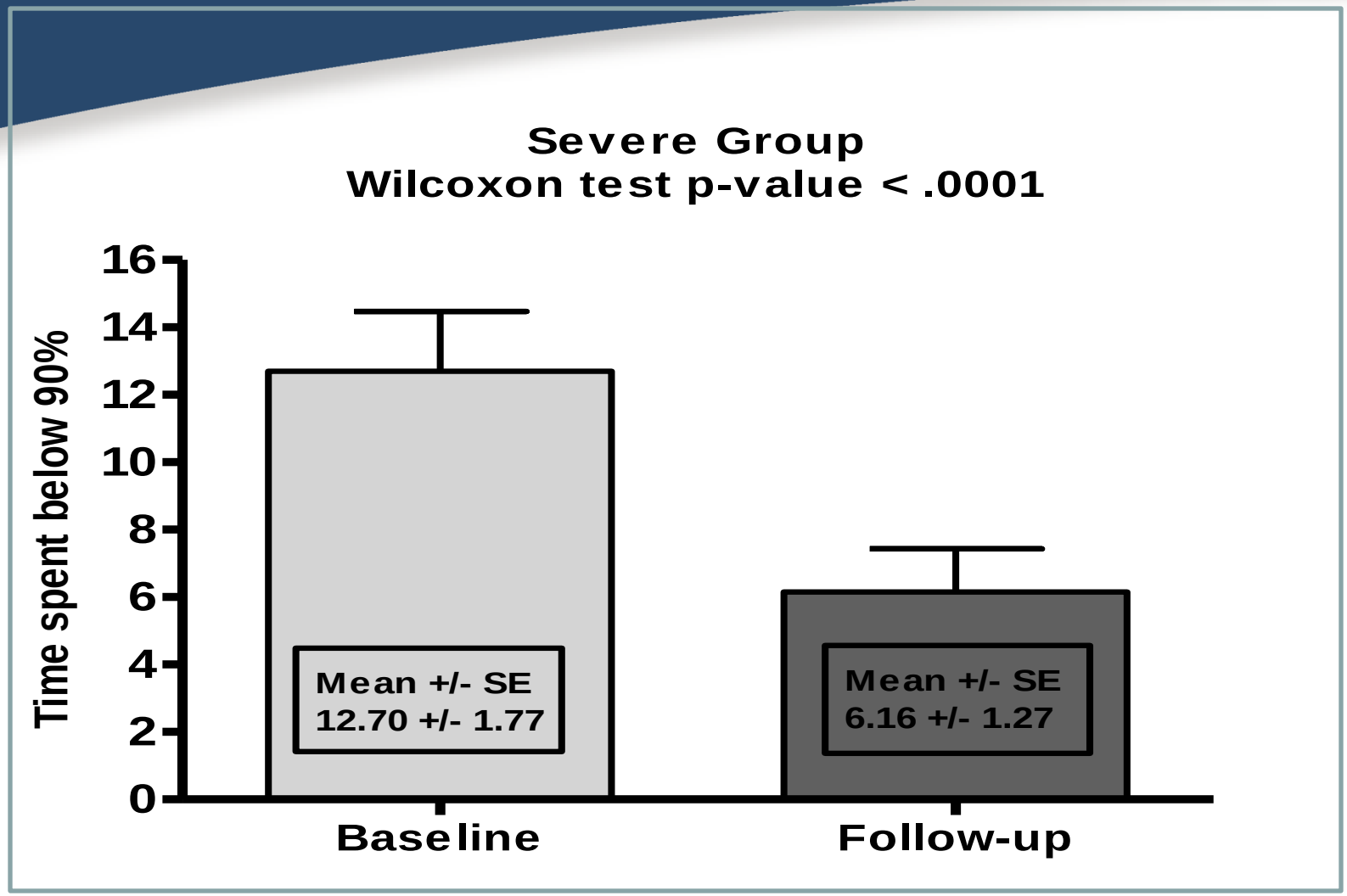
Severe
Improvement of % time spent below 90 % SaO_2



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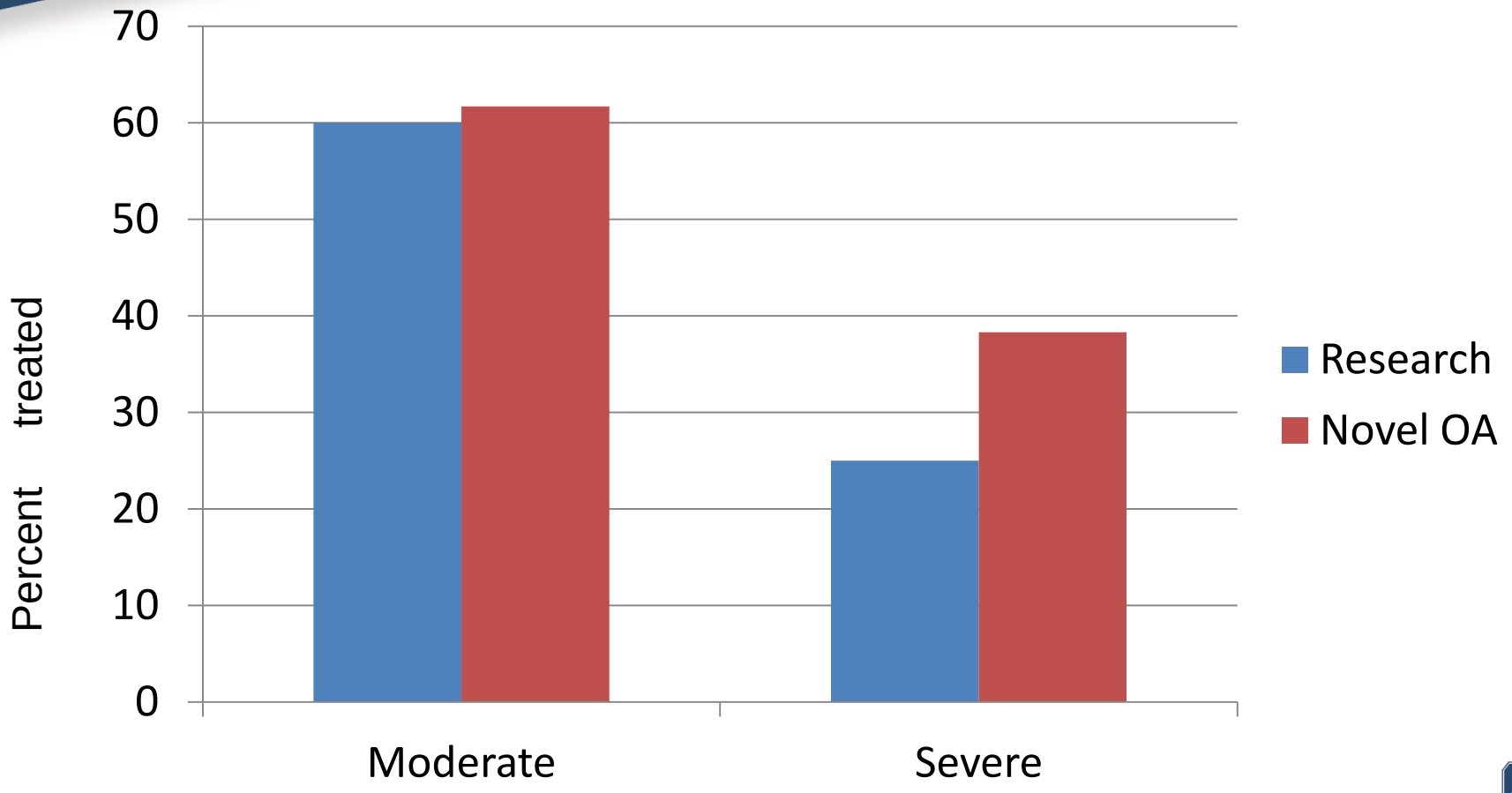
Novel OA device vs Extant Literature



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Treatment response AHI

OSA Severity	Full Responder
Moderate	29 * 61.7
Severe	18 * 38.3
Overall	47 * 39.2

* P<0.0001

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Snoring/Sleep Improvement

Reported	Snoring improvement yes or no	
	Number	Percent
no	5	4.17
yes	115	95.83
Reported	**Improved sleep quality yes/no**	
	Number	**Percent**
no	5	4.17
yes	115	95.83

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Conclusions

The SleepAPP™ device is a successful option for the treatment of sleep disordered breathing.

- Improved AHI as well as or better than published literature.
- Significantly improved nocturnal oxygen saturations.

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Conclusions

- Improved reported average use of therapy compared to CPAP in published literature (>6 hours a night/>6 days a week).
- Subjective improvement in OSA symptoms and snore in responders and non responders.
- Tolerability was good with no reported dropout of patients due to side effects.

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Limitations

- Retrospective assessment of treatment outcome
- No updated baseline assessment in patients with weight gain

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Limitations

- Not a randomized controlled trial
- Lack of quantifiable subjective data in OSA symptomatology
- Attrition: 38% of sample
  - Only 10% lost to follow up

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Future Directions

- Publish the data from study.
- Conduct a randomized controlled trial (RCT) with a similar or larger sample size comparing OA to CPAP.

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- Questions?

- Thank you to my committee for your insight and guidance:
- Dr. Kim Jones – Chair of committee
- Dr. Daniel O’Hearn
- Dr. Scott Mist
- Dr. Richard Moore

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Joseph Zelk

Oregon Health Sciences University

N790: Clinical Residency

Case Study: Patient with Difficult to treat

Obstructive Sleep Apnea Syndrome

Case Presentation

D. T. is an 83 year-old Caucasian male with a history of snoring, severe obstructive sleep apnea, hypertension and excessive daytime sleepiness was seen in the clinic for consultation for difficult to treat obstructive sleep apnea (OSA). Our practice is an inter-professional sleep disorders clinic consisting of a Family Nurse Practitioner, Sleep Technicians and a dentist board certified in dental sleep medicine. The patient's sleep apnea treatment history started with the diagnostic process. He had an initial laboratory-based polysomnogram with split-night protocol in 2002 at Adventist hospital Sleep Diagnostic Center under the care of Dr. Michael McDonald.

Obstructive Sleep Apnea History

The patient's diagnostic polysomnogram study revealed moderate intermittent snoring recorded, poor sleep efficiency at 66 percent (normal >85 percent), substantially elevated stage I sleep at 54 percent (normal is less than 5 percent), absent slow wave sleep (normal > 10 percent) and markedly reduced rapid eye movement sleep (REM, normal 20-25 percent). Respiratory Disturbance Index (RDI) of 42 events per hour (ASDA, 1995). His minimum oxygenation was measured at 79 percent during REM sleep. These results, in light of his symptoms are consistent with severe OSA. An excerpt from the polysomnogram report can be found in Figure 1.

A measurement not commented on during the interpretation report was the patient's persistent low mean oxygen saturation noted to be reduced at 92 percent (normal > 96 percent). The finding of low mean oxygen saturation may reflect an overlap syndrome. Overlap syndrome is present when a patient has possible obstructive lung disease with overlapping OSA. His persistent hypertension on a two medication antihypertensive medication regimen

likely denotes the negative impact of untreated OSA toward suboptimally managed hypertension.

His treatment history relating to OSA started with a continuous positive airway pressure treatment (CPAP) titration study to determine an optimal treatment pressure to effectively manage the OSA. This titration was performed as a split night protocol (Khawaja, Olson, van der Walt, Bukartyk, Somers, Dierkhising, et al., 2010). To improve patient comfort and tolerability of diagnostic testing of OSA, sleep laboratories will often follow a set guideline to initiate CPAP therapy during the latter half of a diagnostic PSG. The patient must demonstrate a minimum of 15 per hour apnea hypopnea index (AHI, normal < 5/hr) during a prescribed period of continuous sleep to meet this criterion.

The patient did not tolerate CPAP well on the study. He could not tolerate a pressure higher than the starting pressure of 5 cmH2O. A CPAP of 5 cmH2O left him under- treated with a RDI of 20.4 and low oxygen saturation of 82 percent. This finding alone, which is known as first-night effect, has been found to be a strong predictor for CPAP failure (Kushida, Chediak, Berry, Brown, Gozal, Iber, et al., 2008). The recommendation from the sleep specialist was to have the patient attempt an auto-adjusting CPAP trial at home to allow the patient more time to acclimate to the therapy. The patient was not able to wear the treatment more than a few hours a night due to unconscious removal of the CPAP mask interface.

The patient worked closely with respiratory therapists at Adventist home medical CPAP department. The patient trialed the use of a nasal CPAP mask, which resulted in excessive mouth leakage. The patient added a chin strap support to encourage nasal breathing and decrease mouth leakage. The adjunct was not effective. He then attempted a full face mask interface, otherwise known as an oronasal mask. This interface covers the mouth and the nose

to allow patients to choose between mouth or nasal breathing. The patient was unable to tolerate this interface due mask leakage and claustrophobia (Massie & Hart, 2003). Dr. McDonald is a board certified Sleep Specialist and Otolaryngologist. Once the patient and his sleep clinician came to the conclusion that the patient was unable to tolerate CPAP therapy, the patient decided to consult with Dr. McDonald on surgical options. At this point in time, the patient was uninterested in mandibular advance appliance treatment, and more interested in attempting surgical correction of his severe OSA.

The literature does not support sufficient treatment of severe OSA with first surgical interventions (Barthel, & Strome, 1999). The mainstay of phase I surgical intervention is uvulopalatopharygoplasty (UPPP) (Epstein, Kristo, Strollo, Friedman, Malhotra, Patil, et al., 2009). UPPP only has a 40-50 percent chance of reducing the AHI by 50 percent. Thus many patients will continue to have an AHI> 20 events per hour after surgery. Tracheostomy is an effective treatment options for OSA but is largely not considered an acceptable options by patients (George,Barber, & Smith, 2007). The surgeon's conclusion was that the patient had a type II obstruction. A type II obstruction is a combined obstruction of both the palatal and hypopharngeal regions of the upper airway (Barthel & Strome, 1999).

The surgical plan resulted in UPPP, genioglossus advancement and hyoid mytomy and suspension. The patient went through two surgeries for a septoplasty and turbinate reduction. The patient's extensive surgical history includes tonsillectomy and adenoidectomy, uvulopalatopharyngoplasty, hyoid myotomy and suspension surgery, genioglossus advancement and retroglossal ablation, nasoseptoplasty, turbinate reduction and somnoplasty. Figure 2, 3 and 4 demonstrate the phase I surgery options. Post surgery the patient reported improvement in snore and excessive daytime somnolence. Six months post-operatively the

patient was found to have an excellent reduction in AHI with follow up polysomnography. Figure 5 depicts the data. Approximately a year after the post-surgical evaluation the patient was referred for a MAD due to recurrent snore and hypersomnolence. The sleep specialist believed the OSA remained treated and therefore was simply dealing with primary snoring. No significant weight gain was noted on examination when compared to initial evaluation. Research supports the use of MAD for primary snoring. A custom-fitted mandibular advancement splint is an oral device that fits to the teeth. The function of the device is to hold forward the mandible during sleep to alleviate upper airway collapse in improve OSA (Chan, Lee, & Cistulli, 2007).

Past Medical History

The patient's past medical history includes obesity, hypertension, hyperlipidemia, low testosterone, long-time snoring, excessive sleepiness and severe obstructive sleep apnea (OSA). He has family history of snoring noted by his father but no recorded OSA. He is a retired, happily married man of 40 years. He is currently the primary care giver for his wife during hospice; she is suffering with terminal breast cancer. He is a devoted Seventh Day Adventist who is a nonsmoker, denies alcohol, nicotine use or recreational drug use. He has a sedentary lifestyle and does not have a standard dietary plan. His weight status has been relatively unchanged for many years. He has no drug allergies and takes 100 mg of Toprol XL and 20 mg Altace for hypertension with moderately persistent hypertensive readings on current medication regimen. He takes 10 mg of Crestor for hypercholesterolemia treating him to recommended cholesterol ratios. He is using topical testosterone for androgen replacement.

Physical examination showed a decrease in the anterior-posterior diameter of her posterior oropharynx space due to enlarged tongue base. He has post surgical findings of the soft palate

that appears reduced noting loss of usual landmarks such as the absence of tonsils on inspection. Patient has evident scarring to the inferior border of the soft palate and surgical removal of the uvula.

By body system findings are as follows: General: Patient is well developed in no acute distress. Patient appears younger than stated age. Eye: Color: Blue. Patient pupils are equal, round and reactive to light. Patient sclera are non-icteric. Ear: Patient external ears, external auditory canals are normal. Throat: Tongue is large, scalloped borders, cheek biting noted. Tongue bulk rises above plane of dental occlusion. Soft palate is status post UPPP reduced to a grade of Mallampati 2 out of 4. Patient has average palatal pillars noted as a Samsoon –Young score of 2 out of 4. Tonsils are surgically absent. Uvula N/A - surgery. Flat hard palate with average skeletal arch form and signs surgical excision of soft tissue. Dentition is in good condition with gold crowns to all molars and noted extraction of teeth 1, 16, 17, 32. Neck: Patient neck is supple, without adenopathy, thyromegaly or jugular venous distention. Neck circumference is 18 inches. Respiratory: Patient chest wall is symmetric with clear breath sounds to auscultation both to anterior and posterior. Musculoskeletal: No muscles tender to palpation. Patient demonstrates normal temperomandibular function. Patient demonstrates mild reduction in lateral cervical range of motion with 8 degree forward head posture. Cardiovascular: Patient has1 plus pulses in the upper extremity without clubbing or cyanosis. Patient has1 plus pulses in feet without edema. Heart tones have regular rate and rhythm with rare ectopy. S1 and S1 sounds are clearly audible without click or pathologic S3 or S4 noted. Neurological: Patient cranial nerves 3-12 are intact. Patient motor strength is symmetric and normal for age. Patient is oriented to person, place, and time. Skin: No rashes, warm dry and intact. Dental Findings: No Joint sounds to auscultation throughout range of motion of TMJ

bilaterally to stethoscopic assessment. Maximum Opening 51 mm, Overjet 2 mm, Overbite 2 mm, Protrusive 16 mm +10/-6 Right Lateral Excursive 11 mm Left Lateral Excursive 11 mm. Mild deviation to the right on mandibular opening. Periodontal exam notes missing teeth of 1, 16, 17, and 32. Mandibular classification of class I occlusion. Periodontal status I without pocketing or gingivitis.

The patient was deemed appropriate for a mandibular advancement device for his primary snoring. Looking back at the follow up testing the patient did have ah AHI greater than 5/hr which is consistent with mild OSA in the presence of symptoms. Since CPAP was attempted and the patient was not open to more upper airway surgical intervention the MAD was considered the patient's last option for treatment. He was started at a 50 percent measurement of his total protrusive range of motion of the mandible. There are many FDA approved devices for the treatment of OSA and snoring (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman, et al., 2006). An example of the MAD used for D. T. can be found in Figure 6. For this patient a device that had a good clinical history of treatment in severe OSA was necessary, thus the TAP II device was selected for treatment in this patient.

He proceeded over 6 weeks to incrementally advance the device a total of 7 mm. The terminal advancement resulted in resolved snoring, reduced sleepiness to normal levels with an ESS of 10/24, and improved hypertension control. He was noted to wear the device 7 nights of the week for 9.5 hours of sleep per night. He had no tooth or muscle discomfort with the device use. He performs daily morning jaw exercises to regain his baseline dental occlusion. At week ten an objective assessment of the device treatment was performed noting a RDI 30, elevated snore was noted on this assessment. The device used to assess oral appliance treatment response was a level III home sleep diagnostic device, Remmers Sleep Recorder (RSR)

(Chesson, Berry & Pack, 2003). Figure 7 represents the data from the RSR assessment of MAD. The patient met many of his personal goals for improved sleep quality, duration, daytime somnolence and snore, but he was still found to be in the moderate range of OSA severity. This outcome study was performed without assessing his current baseline level of OSA. This partial treatment of OSA with the MAD leads us to speculate that his AHI may, in fact be close to his initial baseline AHI prior to the serial OSA surgical interventions. At this point in treatment planning the patient was not open to reattempt CPAP therapy. The patient was followed for a year with the TAP II partial response, and instructed to start lifestyle modification, side sleep therapy to reduce the periods of untreated OSA, and maintain a regular sleep schedule.

The patient felt his treatment responses noted earlier in the year from the oral appliance to be diminished. During this treatment interval a combination device was newly available as a treatment option for patients who had previously been intolerant to CPAP therapy. Since the nationwide compliance rate for CPAP effectiveness has been reported to be 51 percent a stimulus for novel interface approaches had stimulated ideas to improve treatment outcomes (Haniffa, Lasserson & Smith, 2004). In early 2000 a pulmonologist, William Hart, and a dentist, Richard Moore devised a dentally-supported interface to deliver oral airway pressure. This concept leads to interfaces that would be dentally-supported that could supply nasal or oronasal CPAP. D. T. was presented with this treatment options and was willing to move forward.

D.T. case was particularly challenging due to his history of soft palate excision. The loss of this tissue results in less anatomy to direct nasal breathing. This makes it more challenging to deliver nasal CPAP, which is better tolerated than an oronasal mask (Loube, 1997). The

patient opted to try a nasal interface and work on sealing the mouth to stop mouth leakage. Figure 7 denotes and example of a dentally-fixed nasal CPAP interface in situ. Figure 8 demonstrates the whole dentally-fixed CPAP interface . The device is custom molded on the molar aspect of the device to create a functional seal specific to the patient's needs. After 3 visits to make adjustments for comfort and fit the patient found he was able to sleep with CPAP through the whole night. The patient was initially managed at home with an autotitrating CPAP machine (Resmed autoset II) and optilife (Respironics, Inc) nasal interface (Morgenthaler, Aurora, Brown, Zak, Alessi, Boehlecke, et al., 2008). The patient was prescribed heated humidity to decrease the change of oral drying (Massie, Hart, Peralez & Richards,1999).

Noting the patient's history of total CPAP intolerance we chose to start the patient on suboptimal treatment pressures to slowly desensitize the patient to CPAP (Smith & Lasserson, 2009). With each successive visit the patient had a download of the CPAP unit to measure estimated AHI, leak data and compliance. Each visit the pressure was gradually increased by increments of 2 cmH2O as tolerated by the patient. The patient became quickly acclimated to a pressure of 9 cmH2O. At this point the patient was willing to confirm the results obtained on the autotitrating CPAP unit with an laboratory based CPAP titration study at Mt. Scott sleep disorder center. Figure 9. denotes the results from the CPAP titration study that tested the dentally-based interface. The patient had moderately elevated leak noted on the study and at a pressure of 10 cmH2O the AHI was treated to 1.6/hr and low oxygen of 84 percent. This data is consistent with well treated OSA.

Since starting combination therapy or hybridized therapy, the patient has now been monitored for 2 years with continuous objective monitoring. The patient has 100 percent

compliance with average nightly use of 8.5 hours. Figure 10 represents a copy of the objective data collected by the auto-titrating CPAP unit that monitors treatment pressures and interface fit. Good leak management noted by average leak less than 0.30 liters per minute. Patient has had empirical increase in CPAP pressure with auto-titrating CPAP range 9 cmH2O minimum to 13 cmH2O maximum pressure with 95th percentile pressure at 11.4 cmH2O, average pressure of 10.5 cmH2O. The patient continues to experience improved hypersomnolence with ESS less than 8/24. The patient has also been able to achieve optimal blood pressure treatment with current medical management combined with successful CPAP treatment compliance.

Case Analysis

The current model for CPAP delivery has two delivery systems. The oldest and officially sponsored approach by the Centers for Medicare and Medicaid Services (CMS) is durable medical equipment (DME) provider treatment delivery (Ruehland, Rochford, O'Donoghue, Pierce, Singh & Thornton, 2009). The system is relatively slow and vulnerable to interoffice miscommunication between sleep medicine specialty clinics who prescribe the CPAP treatment, and the DME company supplying the therapy. This allows a level of separation between the clinical professionals recommending treatment and the providers of the DME. There is a history of medical practices possibly increasing utilization of medical services when a possible financial conflict of interest is present.

The current system that is experiencing significant growth is the delivery system where the sleep medicine specialist is now dispensing CPAP therapy. Despite the possible financial conflict of interest, the sleep medicine community has largely accepted this delivery system. Sleep specialist and patients with OSA both report in the literature that having access to

treatments at the diagnostic centers improves continuity of care and communication between their sleep clinicians and themselves when problems arise. Prescribing patterns by sleep specialist are significantly slanted toward CPAP treatment for OSA. This preference can be seen in prescribing patterns. Statistically, surgical interventions and mandibular advancement treatments are rarely recommended treatments for OSA by sleep specialists.

In my clinical practice it is quite common to interview patients who have been through extensive efforts to ameliorate side effects of CPAP that are limiting efficacy, where no suggestion for alternative treatments were recommended, even when total CPAP failure was confirmed by the sleep medicine clinical staff. The continued challenge to improve current CPAP noncompliance confronted by sleep healthcare providers requires complex multidisciplinary treatment plans and inter-professional case review panels. Current studies on CPAP outcomes have focused on the CPAP interface to the patient. A mask strapped to the head of a patient elicits a number of stress responses.

Patients that are noncompliant with CPAP may disclose concern about side effects of the interfaces that include; mask leakage, mask dislodgement, claustrophobia, high pressure intolerance, skin breakdown, water aspiration, mucous membrane dryness, restriction in sleep movements, head and shoulder pain related to straps, TMJ soreness with chin straps and full face masks rested on the mandibular fulcrum, unattractiveness, excessive noise and unconscious removal of CPAP mask during sleep. The literature has identified measures that appear to improve patient compliance to CPAP therapy. The ultimate pattern of CPAP use is often established within the first week of treatment. A measure to improve early habits of CPAP use is to institute early intervention. Regular contact with the medical providers and

respiratory therapist or sleep technician is essential to improve the chance of a good outcome (Smith, Nadig & Lasserson, 2009).

Figure 9 illustrates a possible combination of two known treatments that could address side effects that each treatment may be challenged by. The goal is to dilate the upper airway during sleep. A logical step would be to consider combining two or even three treatment options to reduce treatment side effects and improve outcomes. This practice is common in pharmaceutical management of disorders like hypercholesterolemia, hypertension, diabetes mellitus II, heart failure and even oncology.

The recommended interventions that have been found to improve compliance to CPAP therapy thus far are intensive education about importance of treatment and treatment alternatives before and after the PSG; careful mask fitting, testing for leaks/comfort before PSG; pretreatment of nasal congestion; use of heated humidification; involvement of spouse in education of CPAP training; early contact for follow up; early interventions for side effects; regular objective evaluation of adherence; and regular follow up with sleep medicine provider (Smith, Nadig, & Lasserson, 2009).

Most DME and sleep medicine clinics are performing these recommended interventions consistently. The challenge of addressing persistent side effects of CPAP needs more attention from inter-professional teams of clinicians. Increased education about the effects of OSA on health has limited influence on a patient's decision to continue therapy when the therapy may be causing pain or discomfort.

It is not uncommon to see patients with OSA dealing with chronic nasal restriction (Chandrashekariah, Shaman & Auckley, 2008) (Zeng, Ng, Qian, Petocz, Darendeliler & Cistulli, 2008). It is also quite common to see prescribed CPAP necessary to treat the AHI

related to a patient's OSA, but find it to too high to be tolerated by the patient. Improving fit of a mask will not ameliorate a high pressure problem that may result in claustrophobia, aerophagia, headaches, sinus infections or irritation, oral dryness, ear fullness and pressure and end expiratory pressure discomfort (Smith & Lasserson, 2009).

Currently, the standard of care is not to add mandibular advancement or surgical reduction of soft tissue to CPAP to lower treatment pressures. The current standard is to separate inspiratory positive pressure (IPAP) and expiratory positive pressure (EPAP), called bilevel PAP (BiPAP). By lowering the EPAP it is postulated to improve comfort level of PAP if the expiratory pressure is decreased. Review of the literature does not reveal convincing evidence that BiPAP improves PAP compliance (Kushida, Littner, Hirshkowitz, Morgenthaler, Alessi, Bailey, et al., 2006).

To expedite treatment of these common side effects and improve compliance rates, coordination between the sleep clinician, the otolaryngologist and the dental sleep medicine practice is a wise direction to develop inter-professional case reviews. There is an opportunity to identify patients who have nasal restriction and refer to surgical consult. Patients who demonstrate poor sleep efficiency during the CPAP titration study or need CPAP higher than 15 cmH2O are in a high risk group of patients for CPAP failure (Kushida, Littner, Hirshkowitz, Morgenthaler, Alessi, Bailey, et al., 2006).

There is anecdotal evidence that mandibular advancement with an oral appliance may be able to reduce effective CPAP to improve patient comfort (Denbar, 2002). There are small preliminary studies that have demonstrated improved CPAP comfort and resolution of hypersomnolence when compared to strapped CPAP mask delivery systems. Better efforts by sleep clinicians to initiate early intervention to reduce treatment pressures with mandibular

advancement may be a promising are for inter-professional teams to develop into a possible standard of care for CPAP therapy.

The American Academy of Sleep Medicine is the scientific body that recommends evidence-based treatment guidelines for the treatment of OSA (Ruehland, Rochford, O'Donoghue, Pierce, Singh & Thornton, 2009). Comfort measures to improve CPAP compliance are clearly receiving greater attention to improve OSA outcomes (Abo-Khatwa, Osman, Hill, Lee & Osborne, 2008). A step to toward developing review panels consisting of sleep medicine practitioners, otolaryngologists and dental sleep medicine practitioners could accelerate research in combination of treatment modalities to address many CPAP side effects. Research is warranted in combination therapies of nasopharyngeal surgical intervention with CPAP, mandibular advancement devices with CPAP, and/or mandibular advancement devices and phase I or II surgical interventions. There is one case report of a patient who was undertreated with MMO and improved outcome with mandibular advancement appliance (Hoekema, de Vries & Stegenga, 2007).

MAD is an effective treatment modality for the patient with mild to moderate OSA per these guidelines (Chan, Lee & Cistulli, 2007). Increasing referral patterns to dental sleep medicine for this patient population, before attempting CPAP in patients who prefer MAD, would reduce the number of CPAP failures (Chervin, Moyer, Palmisano, Avidan, Robinson, Garetz & Helman, 2003). This outcome would be a component of improved appropriate patient selection for any given OSA treatment (Bian, 2004). There continues to be a dearth of professionals who are well-trained in Dental Sleep Medicine.

At present, the recommended delivery system for MAD is through the dental practitioner. Currently, access to care is limited due to poor coverage of MAD by medical insurance

companies when provided by dental professionals. This barrier to reimbursement in part, is due to dental clinics reliance on an insurance management system that needs less staff than a standard medical clinic. The logistical mismatch between dental reimbursement and medical reimbursement has proven to be a large barrier to care. Without an sufficiently staffed administrative team, a clinician is unable to perform predetermination of medical benefits or appeal possible inaccurate denial determinations that may occur with third party payers. Lack of medical insurance coverage for a therapy often leaves this therapy inaccessible financially for most patients. The lack of medical coverage for a given treatment may psychologically bias patients and medical providers away from seeking an evidence-based treatment. Attempts are currently being made to train these clinicians in medical insurance management techniques. The progress is limited largely due the hectic pace of general dentistry and time constraints to devote more patient time to this group of therapies.

An alternative delivery system could be to mimic the practice patterns currently being practiced with CPAP treatments. The MAD was classified as a medical device in 1995 by the Food and Drug Administration. With the appropriate training, a medical provider may be qualified to provide medical treatments such as MAD. Integration of this treatment modality into the sleep medicine clinic could improve access to both MAD and combination treatments of MAD with CPAP to improve comfort and compliance. My practice has developed a version of this inter-professional sleep medicine practice. This model has improved our access to medical insurance coverage for MAD and combination CPAP treatments. The dissemination of this OSA treatment delivery system by a Doctor of Nursing Practice may have significant implications for nursing practice.

Reflection on Practice

Sleep apnea is a medical problem with serious health consequences like cardiovascular disease, fatigue, and excessive hypersomnolence which can result in avoidable morbidity and mortality associated with motor vehicle accidents (He,Kryger, Zorick, Conway & Roth, 1988). Currently a pulmonologist or neurologist sleep specialist is considered the coordinating provider for sleep-related treatments. This case study reviewed the need for inter-professional consultation and management of OSA. This model of care could translate into a true medical home for the sleep apnea patient. This type of inter-professional practice could treat the patient comprehensively and provide multiple treatment options, or even combinations, tailored to a patient's preference.

Not only can OSA decrease quality of life, it can also worsen survival outcomes (He, Kryger, Zorick, Conway & Roth, 1988). In my clinical practice, I have seen a multitude of patients who were partially treated when measuring post-treatment AHI, but felt fully treated for their OSA and rested. This effect carries long-term in many of these patients out-lasting possible placebo effect. On the other hand, I have treated many patients with CPAP to a normal AHI (AHI < 5 per hour) who never felt any improvement in quality of life, or even felt less rested with treatment. To achieve both goals, a sleep clinician needs to strive to continue to reduce those side effects related to any given treatment for OSA that will result in either under-treatment of AHI or persistent hypersomnolence. These often confusing outcomes can be frustrating to the sleep clinician. Edward M. Weaver, Chief of Sleep Surgery and Associate Professor of Otolaryngology-Head and Neck Surgery at University of Washington School of Medicine in Seattle, believes there is no set criterion that adequately defines success or failure of treatment. The AHI does not correlate well with many important clinical features of OSA (Haniffa, Lasserson & Smith, 2004).

This case study reveals a growing concern in the general patient population; what alternatives to CPAP are there for me? If a patient has indeed failed surgery or MAD therapy does reattempting CPAP pose a good treatment option. Patients who end up needing to reattempt CPAP with the same treatment pressures or the same nasal interfaces have a low likelihood of long term success with CPAP. A lower pressure with less invasive interface may benefit patients who have demonstrated significant side effects to their initial CPAP treatment and now find themselves needing to make another attempt at CPAP.

Summary/Conclusion

This case study reveals a potential for enhancement to the current practice patterns by clinicians participating in OSA management. Many sleep medicine practices have brought in nurse practitioners to help keep pace with the intensive follow up needed to improve CPAP outcomes. This naturally occurring trend of collaborative practice of the ANP and sleep specialist allows for a window of opportunity for the DNP to facilitate development of more complex treatment modalities for the challenging OSA patient.

The Essentials of Doctoral Education for Advanced Nursing Practice (AACN, 2004) recognized the direct care role for advanced practice nurses. The doctor of nursing practice (DNP) is described as a clinician with expanded responsibility and accountability in the care and management of individuals and families and is also educated to identify and improve provision of care for a particular population at the aggregate and systems level. This case study demonstrates specifically two core competencies as defined by National Organization of Nurse Practitioner Faculties (NONPF) (Smolowitz & Honig, 2008). This specific competency states for the DNP to identify gaps in access and/or reimbursement that compromise patient's optimal

care and apply current knowledge of the organization and financing of health care systems in order to ameliorate negative impact and/or reduce barriers to patient access.

The other competency for DNP is to establish and utilize a collaborative network of specialists while maintaining primary responsibility for patient care and accept referrals from other health professionals and agencies to provide optimum care (Smolowitz & Honig, 2008). This new concept for the sleep medicine medical home could achieve such a goal. By doing so, the DNP can begin development of a referral network between the primary care APN and DNP sleep medicine clinician to improve the likelihood for treatment success in this patient population.

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Appendices

SLEEP STAGE DISTRIBUTION: The patient shows a marked increase in wake-after-sleep onset at 33%. Stage I is substantially elevated at 54%. Slow-wave sleep is absent and REM sleep is markedly reduced.

APNEA SUMMARY: The total RDI during this portion was 42. The events were evenly distributed between obstructive apneas and obstructive hypopneas. They occurred in all positions. They were associated with substantial oxygen desaturation and with microarousal activity.

OXIMETRY SUMMARY: The baseline SaO2 was 93 with a minimum of 79, the lowest desaturations occurring during REM-related events.

PLMS SUMMARY: There were substantial non-wake leg movements with an index of 40. There were a number of arousals associated with leg movements for an index of 10.2

HEART RATE SUMMARY: The patient is in normal sinus rhythm with multiple premature ventricular contractions of multifocal etiology.

SNORING SUMMARY: The patient does snore intermittently throughout the first portion of this study.

Figure 1. Excerpt from baseline polysomnogram. The study was performed on 1-15-02. Retrieved from personal archives with permission.

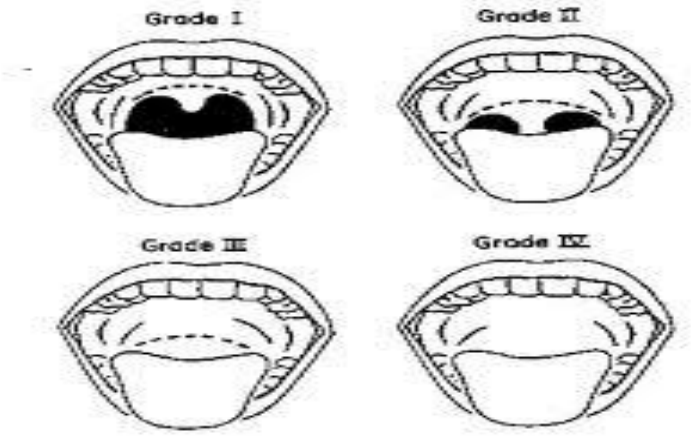

Figure 2. A depiction of the anatomical region to perform UPPP. Retrieved from www.medscape.com

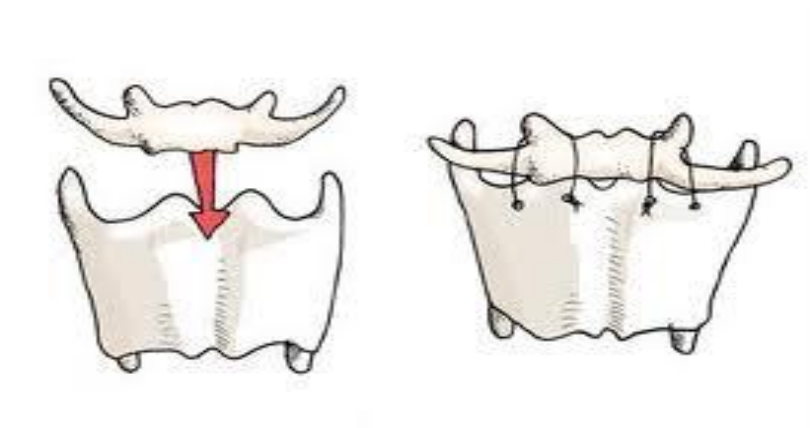

Figure 3. Depiction of myohyoid suspension surgery for OSA. Retrieved from

http://personal.inet.fi/tiede/tapiovaara/kevatkokous2008.htm

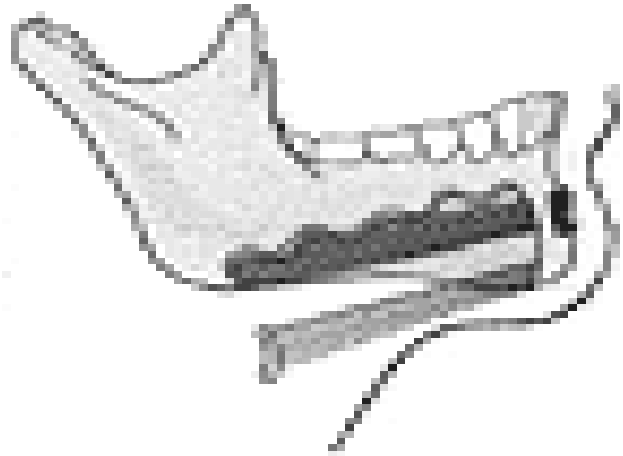

Figure 4. Depiction of genioglossal advancement. Retrieved from

http://www.medscape.com/viewarticle/501708_3

SLEEP STAGE DISTRIBUTION:
The patient shows a marked increase in wake-after-sleep onset. Stage 1 is substantially elevated. Stage 2 is reduced. Slow-wave sleep is absent. REM sleep is mildly reduced.

APNEA SUMMARY:
The overall apnea-hypopnea index was 7.3 and the events were primarily central apneas, many of these occurring when the patient was in transition sleep. The patient was in supine sleep and REM sleep but not simultaneously REM and supine sleep.

OXIMETRY SUMMARY:
The baseline SaO2 was 92 with a minimum SaO2 of 81 and the lowest saturations occurring during the central-related apneas.

Figure 5. Excerpt from follow up sleep testing 6 months postoperative surgical intervention for OSA. The study was performed on 2-18-04. Retrieved from personal archives with permission.

Figure 6. These are examples of the line of TAP II mandibular advancement devices that are FDA-approved to treat obstructive sleep apnea and snoring. Retrieved from www.amisleep.com

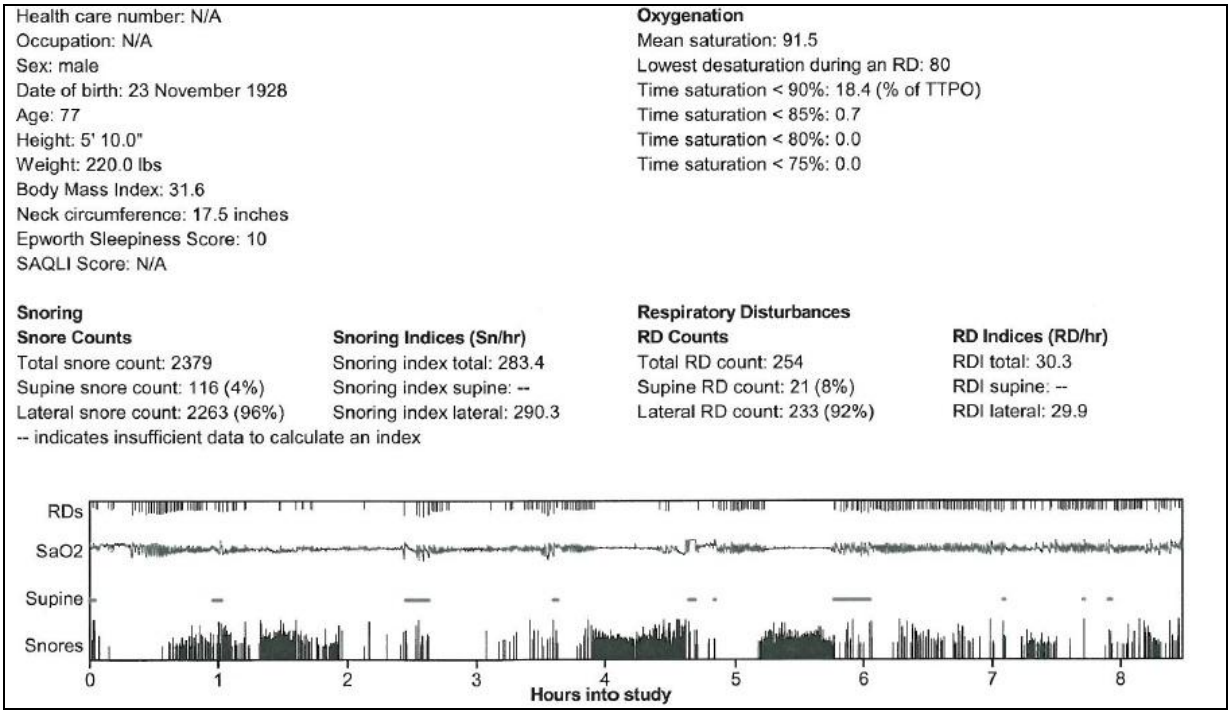

Figure 7. Excerpt from follow up Level III sleep testing 2years postoperative surgical intervention for OSA. The study was performed on 7/25/06. Of note, patient weighs 3 lb less than postoperative polysomnogram. Retrieved from personal archives with permission.

Figure 8. Depiction of the dentally-fixed CPAP interface in situ. Retrieved from personal archives with permission.

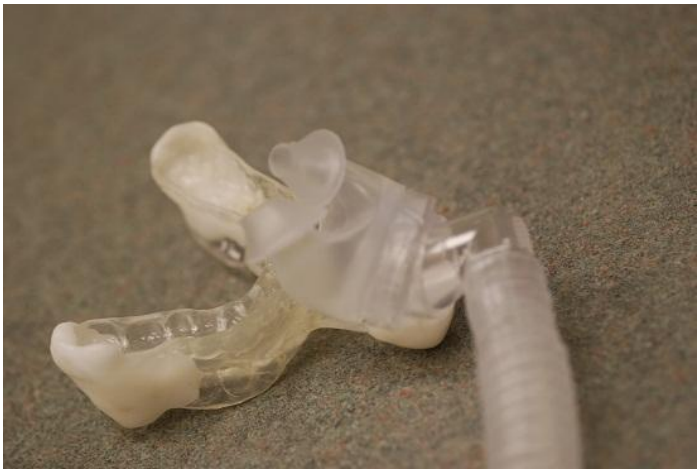

Figure 9. Depiction of the dentally-fixed CPAP interface demonstrating the posterior seals that are functionally fitted to the patient. Retrieved from personal archives with permission.

A total of 10 respiratory events were recorded, giving an overall Apnea / Hypopnea Index (**AHI**) of 1.6/hour. A total of 18.0 minutes of stage REM sleep was recorded, with a REM AHI of 0.0/hr. The longest recorded apneic event was 0.0 seconds long. The average blood oxygen saturation was 92% during the night, with the lowest desaturation with a respiratory event at 84%, and with a lowest

Figure 10. Excerpt from follow up dentally-fixed CPAP interface at CPAP of 11 cmH2O. The study was performed on 1/4/07. Retrieved from personal archives with permission.

Product: S8 AutoSet Spirit

10/1/2010 - 10/27/2010

Device Settings		
Therapy Mode: AUTOSET	Minimum Pressure: 9.0 cmH2O	Maximum Pressure: 13.0 cmH2O
Pressure - cmH2O		
Median: 10.2	95th Percentile: 11.4	Maximum: 12.4
Leak - L/min		
Median: 16.8	95th Percentile: 30.0	Maximum: 37.2
AHI & AI - Events/hr		
Apnea index: 0.7	AHI: 4.8	% Time in Apnea: 0.2
Hypopnea index: 4.1		
Usage		
Used Days >= 4 hrs : 27	Used Days < 4 hrs : 0	% Used Days >= 4 hrs : 100
Days not used: 0	Total days: 27	Median daily usage: 8:50
Total hours used: 236:15	Average daily usage: 8:45	

Figure 11. Excerpt from most recent download of auto-titrating CPAP representing excellent treatment response with dentally-fixed interface. Retrieved from personal archives with permission.

Case Study: Chronic headache Syndrome Improved with Sleep APP™ Oral Appliance

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Case Study: Chronic Headache Syndrome Improved with Sleep APP™ Oral Appliance

Headache is a common medical complaint seen in both primary and specialty practice. Headache is generally categorized into one of three syndromes. Headache can be treated as a complex syndrome when more than one of the syndromes occurs concurrently. Migraine can be characterized by recurrent headache, nausea, vomiting, photophobia, aura, unilateral and bilateral distribution with likely throbbing intensity (Bigal & Lipton, 2009). Tension-type headache is mild to moderate in intensity, often bilateral, non-throbbing with few other associated features. Cluster headache is unilateral, has abrupt onset that can result in severe pain within minutes (Bender, 2007).

Differential diagnosis for etiology of headache syndromes may include sinus-type headache, facial pain syndrome, post-traumatic brain injury, medication over-use, dental pain, obstructive sleep apnea, nocturnal bruxism, temporomandibular disorders, cancer, hydrocephalus and other metabolic imbalances (Wright & Jundt, 2006). This case study reveals the impact of sleep disordered breathing and obstructive sleep apnea (OSA) on headache syndrome occurrence. OSA has been estimated to occur in up to 33 percent of the general population and noted to be more than twice as common in the population of patients who see a primary care provider regularly. OSA is significantly under-diagnosed with estimates predicted to be as high as eighty-five percent still left undiscovered (Chesson, Berry & Pack, 2003).

Inter-professional treatment planning for headache syndromes appears to be occurring more rapidly in the recent past. Complementary and alternative therapies to treat headache are becoming more common in medical treatment planning (Mauskop, 2001). These therapies may include acupuncture, aromatherapy, biofeedback, cognitive behavioral therapy, hypnosis,

chiropractic, cranial electrical stimulation, hyperbaric oxygen therapy, massage, yoga, nutrition and intra-oral devices (Graff-Radford, & Newman, 2002).

Proponents for intra-oral appliances for treatment of headaches affirm that stimulation of the trigeminal nerve through increased cervical and mandibular activity, such as nocturnal and awake bruxism and clenching, may trigger headaches (Lamey, Steele & Aitchison, 1996). The nociceptive trigeminal inhibition – tension suppression system (NTI-tss) intra-oral appliance is now cleared by the Food and Drug Administration (FDA) since 2001 for the prevention of migraine headaches, associated tension-type headaches, chronic daily headaches (CDH) and prevention of bruxism and TMJ injury (Bender, 2007). Since 2001, there have been case studies demonstrating this effect in intra-oral devices that treat snoring and OSA (Blumenfeld, 2007).

Signs and symptoms of parafunction are listed on figure 1. More recent research has been demonstrated that teeth grinding consists of an increase in sympathetic nervous system activity; cortex activation; heart rate increase; and increase in jaw depressor muscle activity (Bigal & Lipton, 2009). Sixty-eighty percent of sleep bruxism episodes tend to occur along with cyclic alternating patterns (CAPs) (Lavigne, et al., 2001). CAPs consist of activation of electroencephalogram (EEG) and electrocardiogram (ECG) patterns and occur approximately every 20-60 seconds during non-REM sleep (Lavigne, et al., 2001). The following case study may demonstrate the benefit of this treatment to prevent headaches for those with OSA.

Case Presentation

J. M. is a 39 year-old Caucasian male with a history of snoring, severe obstructive sleep apnea, and chronic morning headache and morning grogginess. The patient's past medical history includes obesity, long-time snoring, morning headache and severe obstructive sleep apnea (OSA). His chief concern is to treat his loud snoring and improve his morning perception

of being excessively tired. The sleep disorder treatment history for J. M. started with a patient initiated complaint of snoring from his primary care provider (PCP) that resulted in a referral to a board-certified sleep specialist. He had an initial laboratory-based polysomnogram (PSG) prior to 2006 at Providence St. Vincent's Hospital sleep disorder center. Report of PSG is noted in figure 2. He was offered only continuous positive airway pressure (CPAP) as a treatment option at that time by his primary care provider. Patient declined this treatment option due to its appearance of obtrusiveness to his intimacy with his wife.

J.M. presented to our office approximately four years after his diagnosis of OSA and declining treatment of his OSA at the time. He was seen in our sleep clinic by self-referral for consultation to treat severe snoring and obstructive sleep apnea (OSA). Our sleep clinic is an inter-professional sleep disorders center consisting of a Family Nurse Practitioner, sleep technicians and a dentist, board-certified in dental sleep medicine. The practice has a niche in sleep medicine by focusing on home sleep diagnostics, therapeutic assessments, PAP treatment and oral appliance treatments for OSA and complex sleep disorders.

The patient's symptoms have persisted and the snoring has continued to affect his sleep environment resulting in a restless and unacceptable experience for his wife. The two bed-partners are now resorting to sleeping in different rooms for the last six months. Review of systems reveals no chronic medical conditions that are under any medical treatment. Patient does have noted sleepiness with driving, an ability to nap in the afternoon, daily morning headaches, nightly nocturia, and heartburn symptoms occurring weekly and morning dry throat and hoarseness.

Sleep-related history includes Epworth Sleepiness Scale (ESS) of seven out of a possible twenty-four points which is consistent with normal alertness per parameters of this test. This

patient's history of present illness (HPI) report of sleepiness is not in line with the score from the ESS. Occasionally he will nap due to sleep pressure. He will awaken two to three times a night for nocturia and unknown reasons. Patient goes to bed 10:30 p.m. and gets up at 6 a.m. and on weekends at 8 a.m. His sleep onset latency is minutes most nights. He denies chronic pain, cataplexy, restless legs symptoms, hypnogagic hallucinations or parasomnias.

Diagnostic polysomnogram from 2006 was consistent with a severely elevated apnea/hypopnea index of 36 events per hour. The mean hypopnea duration was 32 seconds and the longest was 56 seconds. Mild snoring was noted on the study. Sleep staging was significant for marked reduction of rapid eye movement (REM) sleep to 4 percent. The patient was exposed to CPAP the latter half of the study. The pressure of 7 centimeters of water resolved patient's AHI to normal levels (<5/hr). Patient had a rebound of REM sleep on treatment portion of study to normal levels (25 percent of sleep time). Improved sleep quality noted upon awakening with persistent morning headache.

The patient has attempted use of non-steroidal anti-inflammatory (NSAIDS) for morning headaches with little improvement of pain. The most common NSAID regimen was 800 milligrams of ibuprofen two to three times a day. The use of the NSAIDS was terminated due to exacerbation of occasional heartburn symptoms. Patient reports headache occurrence to be daily often lasting 1-2 hours in am with throbbing quality, starting in right temporal area/behind the right eye, photophobia with visual analog scale (VAS) of six to seven on a scale of one to ten with ten being severe. The pain will diminish to three to four for the remainder of most days and becomes more global across the cranium. Increased sleep time, caffeine cessation and known trigger avoidance has not resulted in headache improvement. This regimen was attempted after consultation with PCP.

J.M. has a surgical history of remote adenotonsillectomy and pediatric myringotomy bilaterally for chronic otitis media. He has family history of snoring noted by his father, mother history of breast cancer. He is the manager of a tire company and is married. He is Christian, reports being a non-smoker, he drinks an average of 1-2 cups of coffee a day, and denies nicotine use or recreational drug use. Rare alcohol intake reported. He is not physically active and does not have a standard dietary plan. His weight status has been relatively unchanged for many years. He has no drug allergies to medications but noted likely allergy to monosodium glutamate (MSG).

Physical examination shows an average anterior-posterior diameter of his posterior oropharynx. There is noted long soft palate that appeared moderately redundant. Tonsils on inspection surgically removed. Patient has reddened uvula noted on inspection. Posterior airspace is easily visualized upon inspection.

Physical examination by body systems is as follows: Most recent vital signs: Height, 69 inches; Weight of 211 pounds; Body Mass Index, 32; Blood Pressure, 122/72; Heart Rate 72 beats per minute with regular rate and rhythm. General: Patient is well developed in no acute distress. Patient appears stated age. Eye: Color: Brown. Patient pupils are equal, round and reactive to light. Patient sclera are non-icteric. Ear: Patient external ears, external auditory canals are normal. Throat: Tongue is large with scalloped borders. Tongue bulk rises above plane of dental occlusion consistent with Friedman classification of 3/4. Soft palate is a grade of Mallampati 3/4. Patient has average palatal pillars width noted as a Samsoon –Young score of 2/4. Tonsils are surgically absent. Uvula long and reddened. Average hard palate width and skeletal arch form. Dentition is in good condition with no crowns or fillings to two molars and noted extraction of teeth 1, 16, 17, 32. Neck: Patient neck is supple, without adenopathy,

thyromegaly or jugular venous distention. Neck circumference is 16.5 inches. Respiratory: Patient chest wall is symmetric with clear breath sounds to auscultation both to anterior and posterior. Musculoskeletal: No muscles tender to palpation aside from mild palpation initiated discomfort to bilateral temporal regions. Patient demonstrates normal temperomandibular (TMJ) function with no click to TMJ. Patient has enlarged masseter bulk bilaterally with mild tenderness to direct palpation of lateral pterygoids bilaterally.

Cardiovascular: Patient has 2 plus pulses in the upper extremity without clubbing or cyanosis. Patient has2 plus pulses in lower extremities without edema. Heart tones have regular rate and rhythm without ectopy. S1 and S1 sounds are clearly audible without click or pathologic S3 or S4 noted. Neurological: Patient cranial nerves 3-12 are intact. Patient motor strength is symmetric and normal for age. Patient is oriented to person, place, and time. Skin: No rashes, warm dry and intact. Dental Findings: Normal range of motion of TMJ bilaterally to stethoscopic assessment. Maximum Opening 42 mm, Overjet 2 mm, Overbite 3 mm, Protrusive 10 mm +7/-3 Right Lateral Excursive 12 mm Left Lateral Excursive 12 mm. Periodontal exam notes missing teeth of 1, 16, 17, and 32. Mandibular classification of class 2 occlusion. Periodontal status I without pocketing or gingivitis. No evidence of facets to dentition or grinding.

Oral Appliance Treatment

Patients with less severe obstructive sleep apnea (OSA) are usually prescribed oral appliances. The American Academy of Sleep Medicine practice parameters recommends oral appliance therapy as the second line treatment in patients with severe OSA (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman et al., 2006). The guidelines specifically discuss patient preference as one element to treatment selection for OSA. Noting the patient's treatment

history the patient was deemed appropriate for a mandibular advancement device for his severe OSA.

One study has tried to define predictive criteria for optimal response to mandibular advancement (MAD) in patients who have failed CPAP. The study conclusion was that patients with CPAP treatment pressure less than 10.5 cmH2O were more likely to respond to MAD (Tsuiki, et al., 2010). It may be logical to apply the same predictive criteria possibly to patients who suffer from more severe OSA, but respond to lower CPAP. Larger clinical trials are needed to better evaluate these criteria.

Prior to initiating treatment the patient underwent a more current home sleep diagnostic assessment of the patient's OSA severity due to variability of OSA severity that may occur over 4 years. The device used to assess oral appliance treatment response in this practice is a level III home sleep diagnostic device, WatchPAT 100 (WP100) (Chesson, Berry & Pack, 2003). This study demonstrated similar findings to the patient's PSG assessment. Figure 2 demonstrates the WatchPAT100 unit. Figure 3 represents the WatchPAT100 report. J.M. was started at a 40 percent measurement of his total protrusive range of motion of the mandible. The treatment response to MAD for severe OSA is unpredictable. There are many FDA approved devices for the treatment of OSA and snoring (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman, et al., 2006). An example of the MAD used for J.M. is found in Figure 4. The device used to treat this patient is named the SleepAPP™.

He had a second adjustment to a total of 1 millimeter forward from incisal edge to edge occlusion. The terminal advancement resulted in resolved snoring, resolution of chronic morning headaches and reduced sleepiness to normal levels with an ESS falling from 7/24 to 1/24. He was noted to wear the device 7 nights of the week for 6 hours of sleep per night. He had no tooth

or muscle discomfort with the device use. Goals of treatment that were achieved include improved hypersomnolence, resolution of chronic headache syndrome, snoring and nocturia. Patient also felt more refreshed on awakening. He performs daily morning jaw exercises to regain his baseline dental occlusion. In dental sleep medicine treatments it is recommended to perform objective assessment of OSA after subjective improvements in sleep are reported by the patient (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman et al., 2006).

At week 9 an objective assessment of the device treatment was performed noting a pAHI of 6, low oxygen of 91 percent and well-treated snore, see Figure 5. This study reveals well treated OSA. The patient met many of his personal goals for improved sleep quality, duration, daytime somnolence and snore. The goal for complete response to the oral appliance is a pAHI of less than 10 (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman et al., 2006). All patients with OSA should be counseled on weight loss, avoidance of sedatives near sleep period, side sleep therapy and regular sleep hygiene measures (Ruehland, Rochford, O'Donoghue, Pierce, Singh & Thornton, 2009).

Case Analysis

Chronic daily headache (CDH) includes chronic migraine, and tension-type headache. Dr. Goadsby (2003) has described migraine as a disorder of trigeminal sensory modulation. This modulation may occur indirectly and directly and result in pain and sensory input processing. This trigeminal motor hyperactivity and dysfunction through the third division can result in persistent afferent discharges of pain that may result in central sensitization (Malick & Burstein, 2000). Dr. Blumenfeld (2007) is a neurologist who has worked closely with Dr Boyd the inventor of the NTI-tss. He has been a prominent proponent of directed non-systemic treatments for CDH. Current preventative pharmacotherapies modulate the trigeminovascular system

indirectly, by modifying events that lead to trigeminovascular nociception (Milanov & Bogdanova, 2003). Many patients are reluctant to use pharmacotherapeutic treatments due to their systemic side effects and concerns about increased body weight and sedation.

Dr. Blumenfeld (2007) offers targeted, non-systemic approach to prevent pain by reducing trigeminally-innervated muscular hyperactivity of the temporomandibular system. Two emerging examples of directed treatments are the use of Botox injections and intra-oral devices (Silberstein et al., 2006). Botox appears to alter afferent trigeminal activity to the sensory nucleus. The other option is the traditional mouthpiece (intra-oral devices) intended to treat temporomandibular disorders designed to reduce strain and load on the TM joint (Silberstein et al., 2006). Dr. Lamey (1996) and colleagues have shown that a dental splint can reduce migraine frequency by an average of 60 percent.

The growing level of evidence to support the use of intra-oral devices as adjuvant treatment for CDH is compelling. Pathologic nocturnal hyperactivity of the trigeminal motor nucleus results in intense contraction of the temporalis (Milanov & Bogdanova, 2003). Jaw clenching in sleep provides an unnatural resistance to the disclusion of the teeth. This excessive isometric contraction of the lateral pterygoids results in noxious afferent activity that is transmitted to the pterygoid plates of the sphenoid bone. A properly designed occlusal device can re-direct pathologic strain and load away from the TM joint. A device providing minimal condylar distraction during occlusding events reduces trigeminal motor activity to 25 percent of voluntary maximum and minimizes strain and load to the TM joints (Baad-Hansen, Jadidi, Castrillon et al., 2007). Dr. Bender (2007), a dentist who works in the field of headache management reports that his typical patient has seen six to nine specialists before coming to him, and Dr. Bender estimated that his center's success rate with NTI-tss or other dental appliances

may reach as high as the mid- to high 90% range, based on quality-of-life measures, VAS scores, and subjective reports.

Reflection on Practice

Despite the approval for the prevention of medically-diagnosed migraine pain, the NTI-tss and similar intra-oral devices are yet to become an accepted standard of adjunctive treatment for either pain specialty or primary care practice. Commonly, the reasons for this slow acceptance rate of intra-oral devices include prior experiences of patients, seen in headache clinics, who report a history of negative outcome from improperly placed dental splints, as well the lack of knowledge by providers of this alternative option (Mauskop, 2001). One plausible reason for the suboptimal treatment of some patients may be the continued low screening rate of undiagnosed OSA.

A recent study monitored nocturnal parafunction during the treatment of OSA with CPAP. A majority of patients monitored had a significant reduction in electromyographical activity of the masseter (Oksenberg & Arons, 2002). The possible cross-over of these syndromes present a plausible avenue for improved treatment outcomes. Improved education of these two syndromes to the medical teams managing CDH could improve patient access to these promising treatments.

Conclusion

Etiology of CDH has multiple variables contributing to its occurrence. Parafunctional activity may result in various pain presentations and have deleterious effects on oral structures and CDH management. Oral appliances can be used as an aid in reaching a diagnosis of parafunction, as well as be utilized in the management and prevention of these disorders. Case-appropriate selection should consider relevant scientific evidence available. Providing patients

with an appropriate intra-oral device compliance with prescribed treatments for CDH will be improved. The end result of this inter-professional treatment planning ultimately results in an improved quality of life due to improved CDH prevention. The added benefit of looking at parafunctional etiologies of CDH will in turn improve screening habits toward persistent snore and result in more appropriate diagnosis and treatment of OSA. Figure 6 denotes a list of signs and symptoms commonly present in patients diagnosed to have nocturnal parafunction.

An improvement in efficacy for intra-oral device for CDH may be improved by reducing the extraneous confounding variables. In one crossover, controlled, polysomnographic study comparing full maxillary tooth coverage appliances with a palatal splint without tooth coverage, a 40-50 percent reduction in the number of sleep bruxism episodes was observed (Baad-Hansen, Jadidi, Castrillon et al., 2007). One study found that maxillary oral appliances may aggravate OSA (Gagnon et al., 2004). Figure 7 demonstrates a full maxillary tooth coverage appliance without mandibular advancement. Patients with OSA and CDH may need treatment with an oral appliance that improves not only sleep bruxism, but needs to improve the treatment of OSA. In these select patients an intra-oral device that improve OSA and sleep bruxism may improve the percentage of successfully treated CDH patients offered intra-oral devices as a treatment option.

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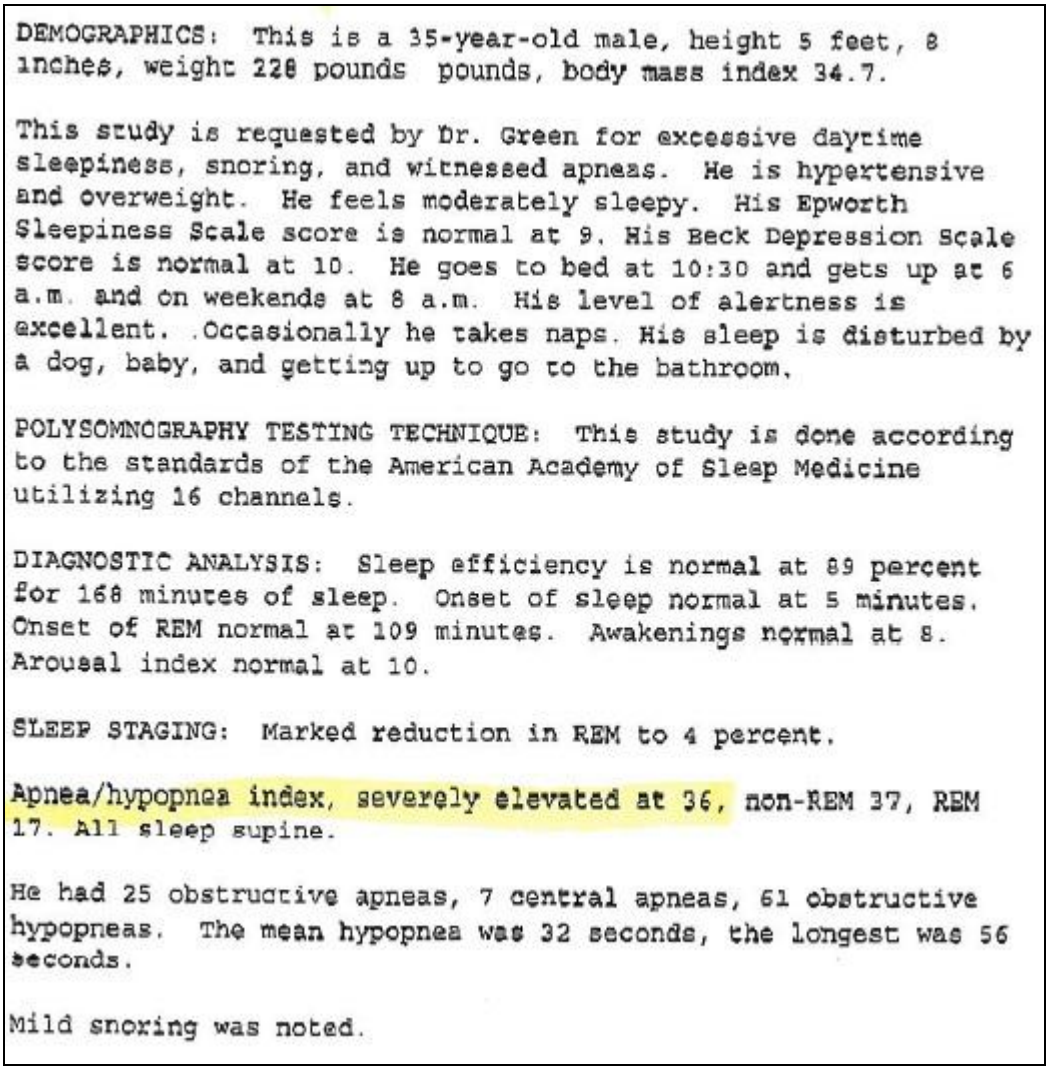
Appendix



DEMOGRAPHICS: This is a 35-year-old male, height 5 feet, 8 inches, weight 228 pounds pounds, body mass index 34.7.

This study is requested by Dr. Green for excessive daytime sleepiness, snoring, and witnessed apneas. He is hypertensive and overweight. He feels moderately sleepy. His Epworth Sleepiness Scale score is normal at 9. His Beck Depression Scale score is normal at 10. He goes to bed at 10:30 and gets up at 6 a.m. and on weekends at 8 a.m. His level of alertness is excellent. .Occasionally he takes naps. His sleep is disturbed by a dog, baby, and getting up to go to the bathroom.

POLYSOMNOGRAPHY TESTING TECHNIQUE: This study is done according to the standards of the American Academy of Sleep Medicine utilizing 16 channels.

DIAGNOSTIC ANALYSIS: Sleep efficiency is normal at 89 percent for 168 minutes of sleep. Onset of sleep normal at 5 minutes. Onset of REM normal at 109 minutes. Awakenings normal at 8. Arousal index normal at 10.

SLEEP STAGING: Marked reduction in REM to 4 percent.

Apnea/hypopnea index, severely elevated at 36, non-REM 37, REM 17. All sleep supine.

He had 25 obstructive apneas, 7 central apneas, 61 obstructive hypopneas. The mean hypopnea was 32 seconds, the longest was 56 seconds.

Mild snoring was noted.

Figure 1. Excerpt from baseline polysomnogram. Retrieved from personal archives with permission.

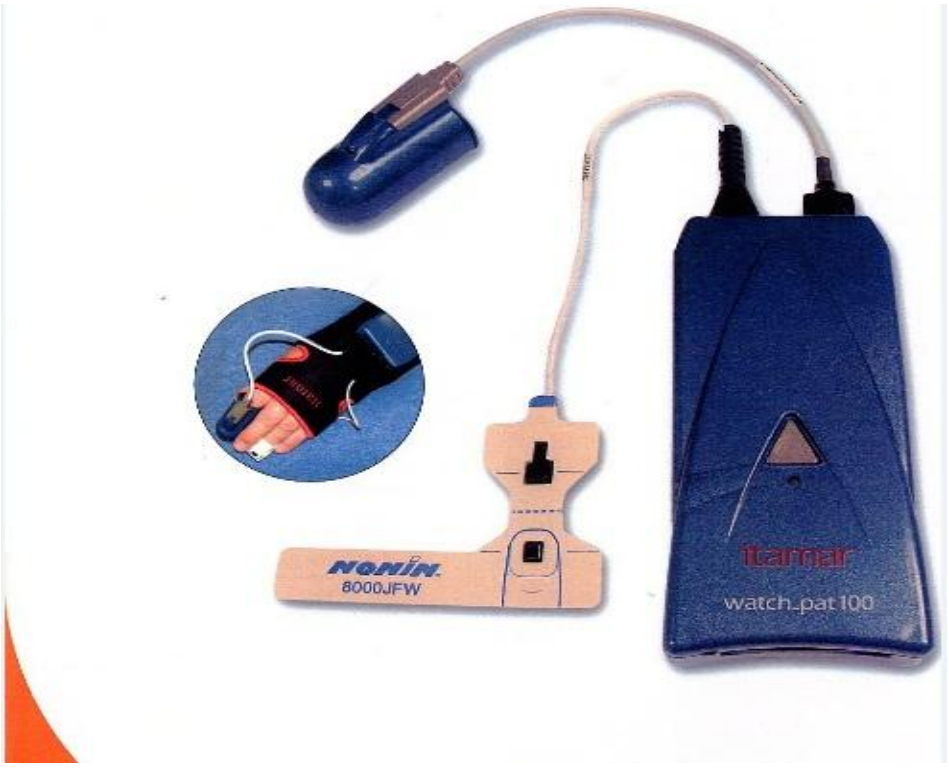


Figure 2. Image of WatchPAT 100 device. Retrieved from personal archives with permission.

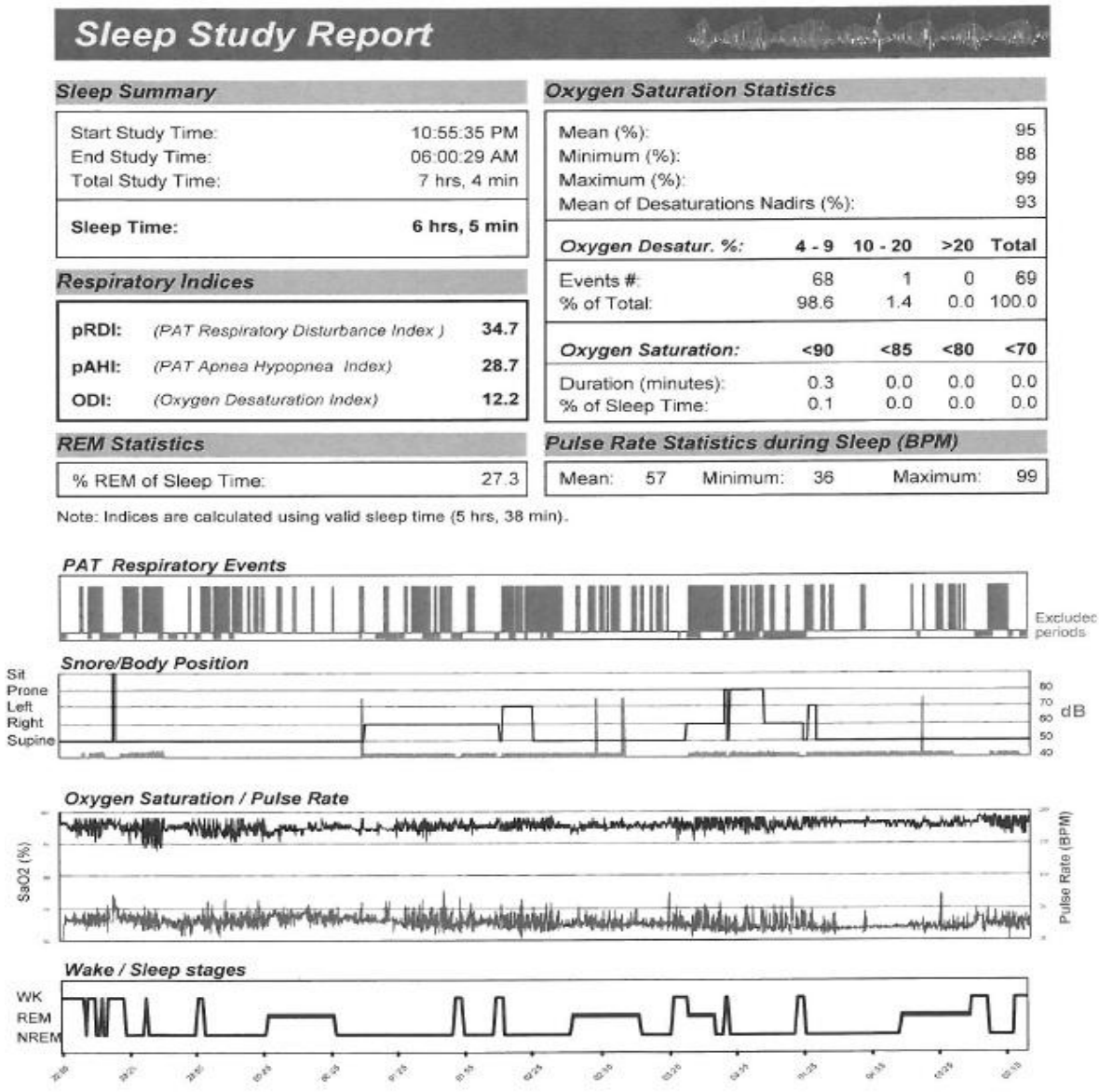


Figure 3. Excerpt from baseline WatchPAT100. Retrieved from personal archives with permission.

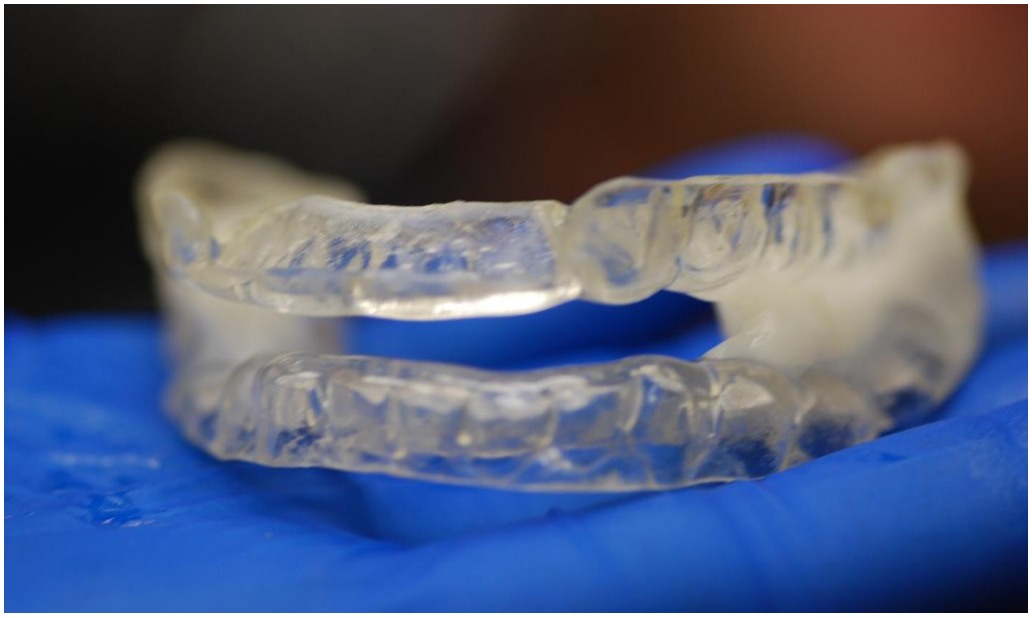


Figure 4. Image of SleepApp mandibular advancement device. Retrieved from personal archives with permission.

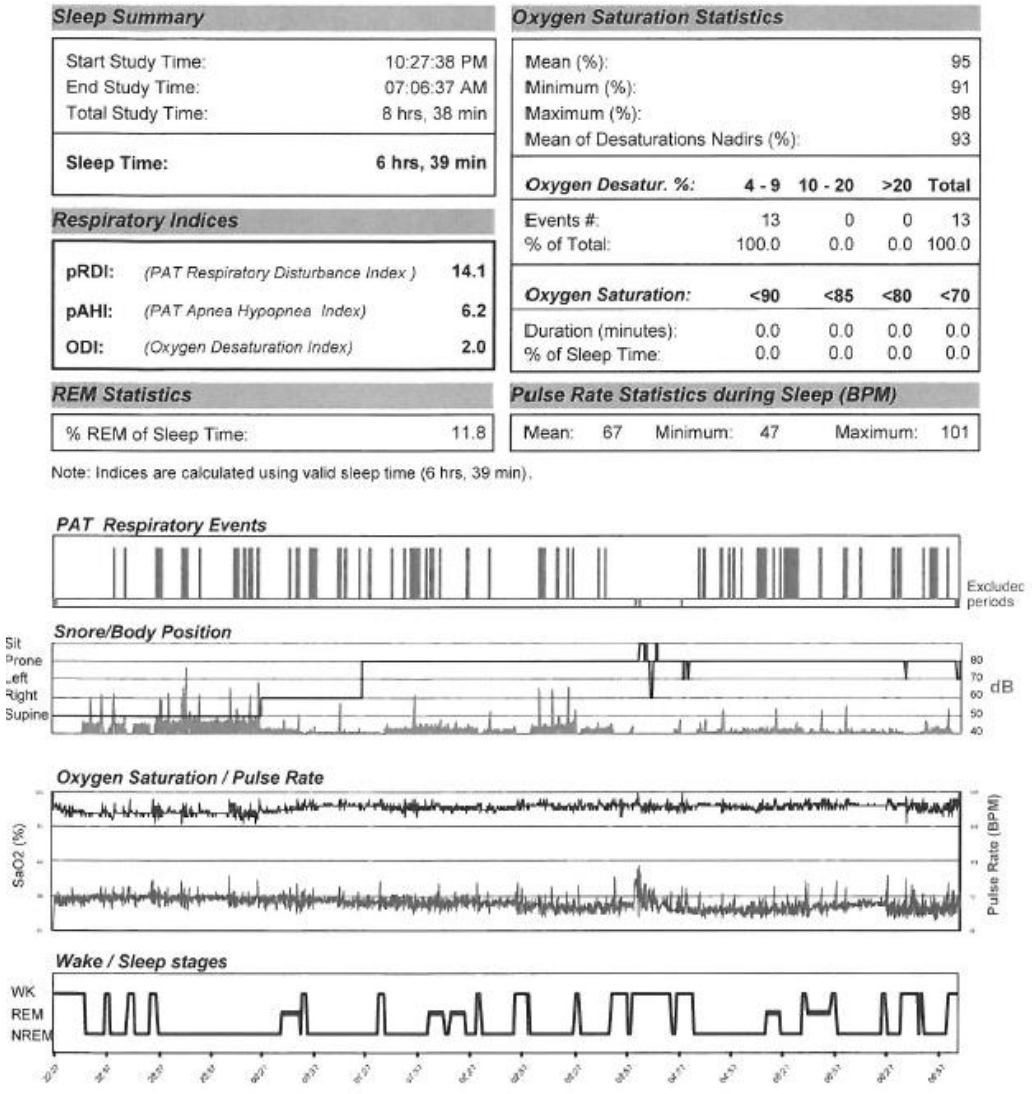


Figure 5. Excerpt from follow up WatchPAT100. Retrieved from personal archives with permission.

Tooth wear –Facets
Tooth sensitivity or heat or cold intolerance
Abfractions
Tooth mobility
Scalloping of lateral borders of the tongue
Ridged keratin development of buccal mucosa

Maxillary or mandibular exostoses
Jaw pain, easily fatigued or muscle stiffness and limited ROM
Radiographic changes of the condylar region
Hypertrophy or muscles of mastication (masseter, lateral pterygoids and temporalis)
Headaches
Neck pain and stiffness, decreased ROM

Figure 6. List of signs and symptoms of parafunction and bruxism.

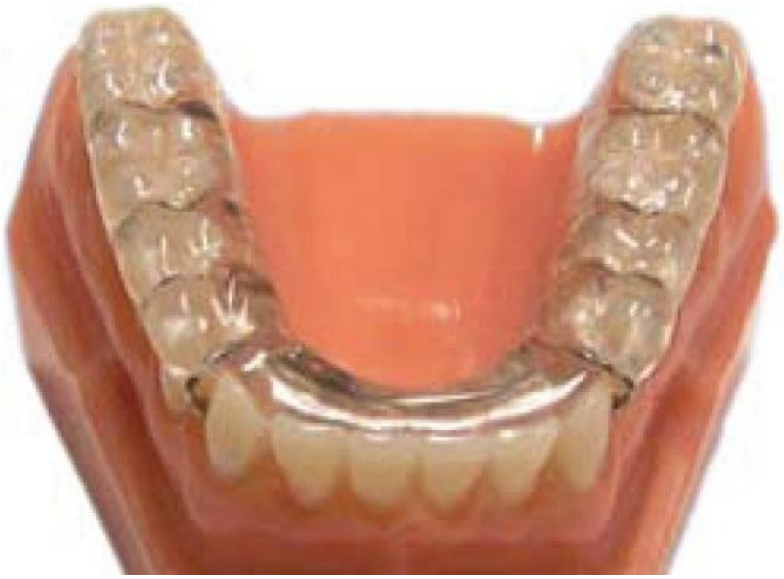


Figure 7. Example image of full maxillary arch appliance without mandibular advancement. Retrieved from personal archives with permission.

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An Investigation into Craniofacial Anomalies and Obstructive Sleep Apnea

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Abstract

Introduction The purpose of this article is to explore the relationship of genetics, environment and epigenetics/epigenomics to health and healthcare practice relating to developing theories for craniofacial factors that may contribute to Obstructive Sleep Apnea/Sleep Disordered Breathing (OSA/SDB) for the pediatric population in the United States. Modern dentistry has for, nearly a century, attempted to redirect jaw growth forward in an effort to correct noted deficiency to facial growth in children. Until recently, attempts have been limited to achievements related to better facial symmetry and balance.

Objective This article strives to connect current understanding of SDB and how it may relate to insufficient craniofacial development of the modern human face. Exploration of current theoretical frameworks and a review of current standard treatment options for OSA in the pediatric population will be reviewed.

A brief discussion will cover the implications of the current divided system of fee-for-service payment of Dentistry and Medical Care reimbursement models for Medicine. A review of literature was performed using keywords in the Cochrane, Medline, and Pubmed databases. Literature both in medical and dental journals were reviewed relating to facial development topics and OSA.

Conclusion The impact of altered facial development in civilized humans on the development of OSA continues to garner increased attention for an interdisciplinary group of health care practitioners. The interest appears to be growing in the dental community and the medical community in a parallel fashion. Much of the inquiry is lacking coordination of efforts between physicians and dentists. The same separation is apparent in the respective academic institutions.

Keywords Obstructive sleep apnea - Craniofacial growth and development - Functional matrix hypothesis - Negative pressure in the chest - Growth of the mandible - Orthodontic treatment - Airway patency – Prevention – Sleep apnea treatments – adenotonsillectomy – Mandibular advancement device – Continuous positive airway pressure – snoring – nasal restriction – healthcare access

Introduction

Christian Guilleminault [1], a leading sleep researcher once said, "Prevention of Sleep Disordered Breathing (Obstructive Sleep Apnea/OSAS/SDB) should be a public health priority… A good understanding of the underlying mechanisms associated with the development of SDB may allow early detection and, possibly, correction of anatomical risk factors." He also felt that "Current goals are to improve the early detection of SDB and to develop a preventative strategy. Prevention requires identification of the genetic factors associated with SDB." These statements were made by a physician with a vast insight of a syndrome that remains little understood by most clinicians. The current move toward prevention of OSA is through knowing not only the genetic influences of facial growth, but the epigenetic influences of our behaviors and environment on development [1].

There is an unfortunate chasm between those who can implement preventative measures and those who manage the patient populations at risk. To clarify, facial development interventions need concerted and meaningful partnerships between primary care providers, who monitor children's health, and dental practitioners who can expertly supply the early interventions needed to

prevent SDB [2]. There is currently a dichotomy between pediatric aesthetic interventions and monitoring of guidance and promotion of normal growth and development. The orthodontist and pediatric dentist focus on a discrete set of functions for dental concerns. The pediatrician, with little communication with either the pediatric dentist or the orthodontist monitors the medical management of the child's growth and development [3].

My hypothesis is that with better cooperation between the primary care givers to children, synergistic improvement of both dental and medical outcomes can be achieved. When adequate cross pollination of ideas is effectively performed several benchmarks will be achieved those benchmarks will include monitoring known epigenetic factors that may lead to OSA. Some areas of concern in the literature are the lack of early identification of mouth breathing habits, which can be related to bottle feeding infants, food allergies, congenitally associated craniofacial anomalies, and high nasal restriction. Another area of concern would be early identification of pathologic dental and skeletal occlusal classifications [4]. Seventy-five percent of facial growth is achieved prior to the age of five years old and ninety-five percent is achieved by the age of twelve years old [4]. Genetic counseling should be discussed for families who have known OSAS in the parents or genealogy and assessing for craniofacial, ethnic or lifestyle related etiologies for those family members with OSAS. If a particular lifestyle alteration is obesity, then dietary and exercise patterns should be addressed [4]. Children with no previously identified genetic linkage to craniofacial anomalies can initially have normal craniofacial development and are able to diverge to pathologic development when exposed to environmental factors, such as, obesity, tonsillar hypertrophy, adenoidal hypertrophy, nasal allergies, malnutrition and thumb sucking [4].

In the 1930s, Doctor Weston Price traveled all over the world visiting "uncivilized tribes" trying to determine why Americans had so much decay in their teeth and these cultures did not [4]. He discovered that the people had minimal decay, had healthy dentition, well-formed dental occlusions, and symmetrical facial forms and could breathe well. His initial conclusions were that these tribes had access to good, whole nutrition and non-processed foods. An additional postulation that has gained support over the years was that some benefit was also related to their breast-feeding habits versus those of the bottle feeding for civilized countries. Natural selection in less civilized tribes access to vaccination or medicinal treatments for infectious occurrence may be limited and lead only those with robust genetic expressions to severe such outbreaks. Those progeny likely would have the same craniofacial genetic expressions that lead to more oxygen intake and adequate feeding [5].

Skulls of recent civilized cultures have noted a rapid departure in craniofacial development from broad symmetrical jaws to asymmetrical and narrow arches. Anthropological assessments of modern skulls demonstrate a preponderance of abnormal findings including: crowded dentition, narrow dental arches, high palates, retrognathia, prognathia, smaller posterior nasal apertures and crowded dentition [10]. The advent of wisdom tooth extraction has been noted to be largely a civilized country intervention. Refer to Figure 1a/1b. And note the symmetrical round and broad dental arch of the uncivilized human jaw. Figure 2. will illustrate obvious asymmetry, misalignment and jaw width narrowing. There is a growing body of knowledge that high risk malocclusions in infancy lead to a higher risk occurrence of sleep apnea. In order to achieve this goal, improved access to health care provider's knowledge in this paradigm and

reduction of the barriers to health care associated with early intervention of craniofacial anomalies is needed.

In the current American health care system the access to such extensive care is out of reach to most patients. The current system for dental insurance is largely fee-for-service. This system leaves a significant personal financial responsibility to the parent of the child to pay for interventional treatments. Patients have a stereotype toward dental care being purely aesthetics and not health care interventions. In the case of facial development, the public has been groomed to believe that traditional wire-and-bracket extraction orthodontics is the mainstay for dental misalignment treatment. Most patients that are candidates for orthodontics should start therapy with functional treatment planning. Functional treatment includes mandibular advancement procedures for retrognathia and palate expansion for patients with bilateral crossbites and narrow palatal arches. In a competitive fee-for-service market place where low cost competition drives patient volume the decision by most Americans is to delay dental interventions until a child's pre-pubertal years of approximately eleven to twelve years of age, where facial growth capacity has diminished.

To support the contention that the current system limits access to care for minorities one only need to quote King, et al., [4] who contend, substantial racial/ethnic disparities in self-reported orthodontic visits exist for Black and Hispanic children. Children from lower-income families and those without private health insurance are noted to less likely report an orthodontic visit in the United States [5].

As the debate in the orthodontic community shifts from a discussion of mere aesthetic alignment of teeth, to an early intervention for the promotion of health in a population at risk for OSA, the impetus for change should occur

quickly. But in the current reimbursement dental insurance scheme for reimbursement of dental care major barriers to health care access will remain despite the growth of medical and dental professional partnerships [9]. Thus the interventions undertaken to treat OSA should be managed under the medical insurance reimbursement schemes. Precedence has been laid for the medical necessity to treat OSA by continuous positive airway pressure, mandibular advancement devices and various soft tissue removal surgeries of the upper airways [11].

Obstructive Sleep Apnea Syndrome (OSAS) is a potentially life-threatening medical disorder caused by repetitive narrowing or collapse and blockage of the upper airway during sleep, resulting in snoring, repetitive episodes of apnea and hypopnea, daytime inattention, hyperactivity or even hypersomnolence and may diminish quality of life for the developing child [2 6 7 12 13]. Craniofacial abnormalities are more frequently being recognized to occur commonly in patients with OSAS. Much interest is developing to investigate the relationship between limited upper airway dimensions and the development of OSAS. Notable anatomical variance from normal may include mandibular deficiency, greater flexion of the cranial base, an inferiorly placed hyoid bone, and maxillary and mandibular transverse deficiencies [9]. By acknowledging these deviations from normal anatomical development and disseminating the information to the medical community, the role of craniofacial abnormalities in the development of OSAS will continue to lead to treatment strategies to correct or improve craniofacial structures. As prospective data is collected on the effectiveness of early intervention orthopedics for the treatment of OSAS, then the medical necessity should cover this new area of pediatric craniofacial interventions for the prevention and treatment of OSA.

Theoretical Framework: Much the current understanding of normal craniofacial development has been proposed by Melvin Moss [12]. The theory best accepted by Dentist is called, the functional matrix hypothesis. It describes the functional cranial components whose size, shape, and position are relatively independent of each other. This was an early finding in his career. Other aspects include comments on cellular theory. He feels that there is mechanosensory role of osteocytes in the bone [11]. The notion that genetics is the strongest determining factor of facial development has not been supported by Dr. Moss [10]. Moss asserts that the growth of the functional component, irrespective of their ossification mechanism, is entirely dependent on the growth and function of the functional matrices. Moss denies any intrinsic regulatory control in growing bony tissues themselves. Control, he believes, of bone growth is by either local epigenetic factors or, environmental factors.

Some research data suggests patient populations with craniofacial pattern such as Dolicofacial pattern or increased anterior facial height or more prone to the development of OSA. Another anomaly is often retropositioned mandible, long soft palate, decreased airway space, and lowered position of the hyoid bone. Moss [11] feels that regulatory roles of intrinsic (genomic) and extrinsic (epigenetic) factors in cephalic growth and thus the functional matrix hypothesis [8]. There are two parts to the growth hypothesis. The first is that of the periosteal matrix. An example of periosteal matrix is a notation that the coronoid process is dependent on the strength of the temporalis muscle Moss, [11]. The capsular or second part of the theory is exemplified by the relationship between the brain and the cranium. The size of the cranium is dependent on the size of the brain. The periosteal matrix works on the bone directly, like bone resorption and deposition,

and the capsular matrix works on the bone indirectly. This is the difference between the periosteal and capsular matrices.

Review of Literature: In 2002, Haim Reuveni and his team undertook an analysis of health care services untilization in children with obstructive sleep apnea syndrome (OSAS). After evaluating 287 consecutively recruited children from the ages one to eighteen with OSAS in a cross-sectional study against control group matched by age, gender and geographic location, they found some surprising results. There was a 226 percent increase in health care utilization noted among children with OSAS compared to healthy controls. There recommendation was for early diagnosis and intervention to improve cost-effective health care delivery.

OSAS is closely linked to obesity and Carolyn levers-Landis and Susan Redline (2006) note there is a rise in this problem in the American pediatric population. They reported that data from the Cleveland Family Study, which evaluated children aged 4 to 18 years, indicate that children who were overweight were at a 4.6-fold increased risk for OSAS than children who were normal weight [8]. The most current epidemiological assessment of OSAS in the pediatric population was a review of literature looking at data from 1999 to 1950. The limited data suggest that the disorder affects 2 to 3 percent of middle-school children and as many as 13 percent of children aged 3 to 6 years [9]. The prevalence may be two to fourfold higher in vulnerable populations, such as blacks and in children who were born preterm and symptoms appear to be increased in Hispanic children. Recent data suggests OSAS is more common in children from poor neighborhoods.

The chronic comorbidities associated with untreated pediatric OSAS include cognitive deficits, attention-deficit/hyperactivity disorder, excessive daytime sleepiness, impaired school performance, poor quality of life and enuresis. The area of assessment does not take into account the stark increase in obesity over the last ten years [3]. Several large epidemiologic studies have demonstrated that small changes in weight lead to changes in the apnea-hypopnea index (AHI) or the grading criteria for severity of OSAS. A 1 percent change in body mass index (BMI) has been estimated to lead to a 3 percent change in AHI and a 10 percent increase in BMI increases incident OSAS by six-fold [8]. The fat deposition is believed to alter pharyngeal size and shape, increasing the vulnerability of the upper airway to collapse. The impact of obesity on the craniofacial development in the pediatric population has not been explored. The mechanism of soft tissues crowding the airway due to fat deposition likely mimics the effect of tonsillar hypertrophy on the risk of OSAS. Both etiologies of airway restriction lead to mouth breathing and negatively impact facial development. It is logical to deduce that early childhood OSAS, if left untreated, could exacerbate a known chronic disease and negatively affect long term growth potential.

A recent study performed by Banabilh et al (2009) looks into this question of how craniofacial obesity may help in recognizing OSA in patients. These authors postulate that the current failure to recognize OSA is in part due to the limited availability of diagnostic facilities and to the non-specific nature of symptoms that can be associated with OSA [19]. The low specificity of these symptoms is related to the fact that other disorders can cause similar findings. These authors note that there are little supported consistent physical findings that are predictive for OSA. Some craniofacial differences that have been discussed as the findings that may turn out to be the most useful for identifying OSA could

include noted mandibular deficiency, bimaxillary retrusion, shortened cranial base, reduced cranial base angle, reduced mandibular length, increased lower anterior facial height, altered craniocervical angulation, an inferiorly positioned hyoid bone, and enlargement of the soft palate [14 15 16]. One modestly predictive physical finding that has been supported in data collected and reported on in the Wisconsin Sleep Cohort Study is that reported obesity increases the risk of OSA in both male and female adults.

To assist the medical community in establishing predictive facial landmarks when evaluating a patient's for OSA, one need only look at an animal study performed on monkeys that were experimentally monitored after surgically-induced nasal restriction. In this trial, young monkeys who had their noses surgically blocked developed abnormal facial growth and malocclusions. The hypothesis regarding the connection between poor nasal function and facial development specifically relates to its ability to support normal muscle function. Breathing, swallowing, chewing, and talking causes us to use groups of muscles in an incongruent manner [20]. The current dental perspective of myofunction purports that bone in the mid-face responds to the requirements of the muscles attached to them. Abnormal muscle function can modify and distort the shape of the midfacial bones and the teeth related to them.

The incongruence between obligate mouth breathing and abnormal facial development is the related low tongue posture of this developing child. The evidence suggests that when the tongue does not rest and function in the palate then both the maxilla and mandible do not form sufficiently forward. The limited forward momentum of the face results in a short length of the respective jaws limiting potential available space for soft tissues like the soft palate and the tongue [21]. There is evidence that orthopedic devices that are used to correct

malocclusion have the potential to re-establish nasal function barring severe nasal allergies or trauma related septal deviation.

The most effective treatment modality to treat OSA in children is adenotonsillectomy [10]. This is the mainstay of treatment for patient identified with snoring and gasping. The vast majority of children with OSA that would be appropriate for facial development intervention are not identified and thus are evaluated only for surgical intervention. What happens to those children that may have untreated OSA as adults? Is there a possibility this soft tissue obstruction could negatively affect normal facial development? Zettergren-Wijk et al (2006) tested the affect on dentofacial morphology after adenotonsillectomy in young children with OSA. The findings noted that after a 5-year-period of follow up 17 children with OSA were compared against 14 children without breathing disorder. OSA children exhibited a more posteriorly inclined mandible, a more anteriorly inclined maxilla, a greater lower anterior face height, a shorter anterior cranial base, retroclined upper and lower incisors, reduced airway space, and a less pronounced nose.

The findings noted that OSA children that underwent adenotonsillectomy at 5 years had no statistically significant differences between the groups except for the lengths of the anterior cranial base and the nose which were still shorter [19]. This study supports the hypothesis that OSA untreated has unfavorable effect on the development of several dental and facial components. The encouraging finding was that many of these abnormal findings can be consistently reduced to a point that they are statistically insignificant if intervention is performed as a young child. A similar study has been demonstrated that treatment of nasal obstruction in growing individuals results in a more normal pattern of dentofacial

development. This interventional study was performed with nasal steroid treatment to decrease nasal atopic symptoms.

It is not part of general guidelines recommendations for OSA treatment to monitor ongoing dentofacial characteristics after adenotonsillectomy. These results may lend credence for clinician to assess nasal breathing to assess if there may be relapse in a post surgery intervention for OSA. There is documented 25 percent relapse rate or incomplete response rate for children who undergo surgical treatment. Children tend to be treated with nasal CPAP as an adjunct to surgery if the patient is partially treated. There is some data being compiled in adult OSA study that may possibly show midfacial retrusion with long term use of nasal interfaces. The change in facial anatomy could reduce the functional size of the airway and theoretically exacerbate the OSA in a developing child. Since many children who undergo adenotonsillectomy at later intervals and may have some more pronounced altered facial development than those who are non-obese and intervened on in young childhood. Those facial changes in a child with OSA may be propelled into such a low efficiency airway that the adult level of OSA may be more severe than otherwise expected. Patient who are significantly obese in early life could possibly mimic the effect that excessively large lymph tissue size does to pediatric airway development.

It sounds logical that removable devices that would help establish or make up for the deficiency caused by the mouth breathing habit could possibly treat OSA. In 2004, Pirelli et al performed a prospective interventional trial that tested the effectiveness of rapid maxillary expansion in children with poor nasal breathing and OSA. The conclusion of that trial was that this treatment was useful and reduced and in many children resolved their OSA. The implications of epigenetic responses to inefficient breathing can result in facial changes is a

powerful example of epigenetic influences on facial development and a possibility for exacerbating functions to OSA as a fully-developed adult. There is a growing agreement by sleep researchers that significant obesity in children can mimic airway collapsibility that occurs with large lymph tissue. Thus even in adenotonsillar correction in the pediatric population weight management should be considered as part of the long-term interventional follow up.

Traditional wire and bracket orthodontics has the opposite effect on palate development than rapid palatal expansion. This finding may need to be weighed by orthodontists. To help screen for snoring and OSA before orthodontic consultation the orthodontist should possibly need to consider these patients as contraindicated for retrusive orthodontic treatments.

A final study to review in this article is an interventional study that consisted of early orthodontic treatment for a patient with an overbite or Class II malocclusion [13]. It this trial a Herbst mandibular advancement device and interceptive removal appliance were used asynchronously to help the child's maxilla and mandibular jaws develop more forward positions. In this trial the child began with OSA and was treated to an acceptable level of normal night time breathing. This trial is encouraging and likely to help move interdisciplinary partnerships more in the future. The conclusion of this trial is that snoring patients with a significant overbite or skeletal Class II malocclusion with deficient mandible may be good candidates for orthopedic intervention [19]. There is data that surgical mandibular advancement is helpful in reducing OSA in children. The challenge of this intervention is its significant recovery time, pain and cost.

Discussion

The mounting data consists of mediocre extrapolation level power. The sample sizes are small and these studies should be considered along the lines of

pilot research. The promising results of these trials should be proposed for replication with larger treatment groups. Current pediatric treatment guidelines are restrictive and recommend limited interventions. The mainstay for treatment is adenotonsillectomy or CPAP. Both of these interventions have the possibility of causing certain sequelae. Surgery has its risk for injury and CPAP has a low compliance rate and may negatively impact developing pediatric profiles. Understanding epigenetic influences on health in this paradiagm of growth and development can help the medical/dental team to better coordinate the needed increased efficiency of screening, and early intervention for children with OSA. This will likely increase needed diagnostic efforts to more closely close the gap between current screening levels and those levels that need to be achieved.

Luckily in this arena of health, medical devices have been developed with the idea of epigenetics at its core. The functional development of the face with these rapid palatal expanders actually appears to be a total departure from much of the traditional orthodontic treatment protocols being recommended today. There is a need to increase debate and investigation on what the impact of wire and bracket facial limiting interventions. The field of Dental Sleep Medicine has a robust call to arms. The partnership of Sleep Medicine and Dental Sleep Medicine stand to make a significant impact on a prolifically common epigenetic influence of rampant nasal allergies, asthma and associated malocclusions.

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Figure 1. Prehistoric skull with normal palate and larger posterior nasal aperture.

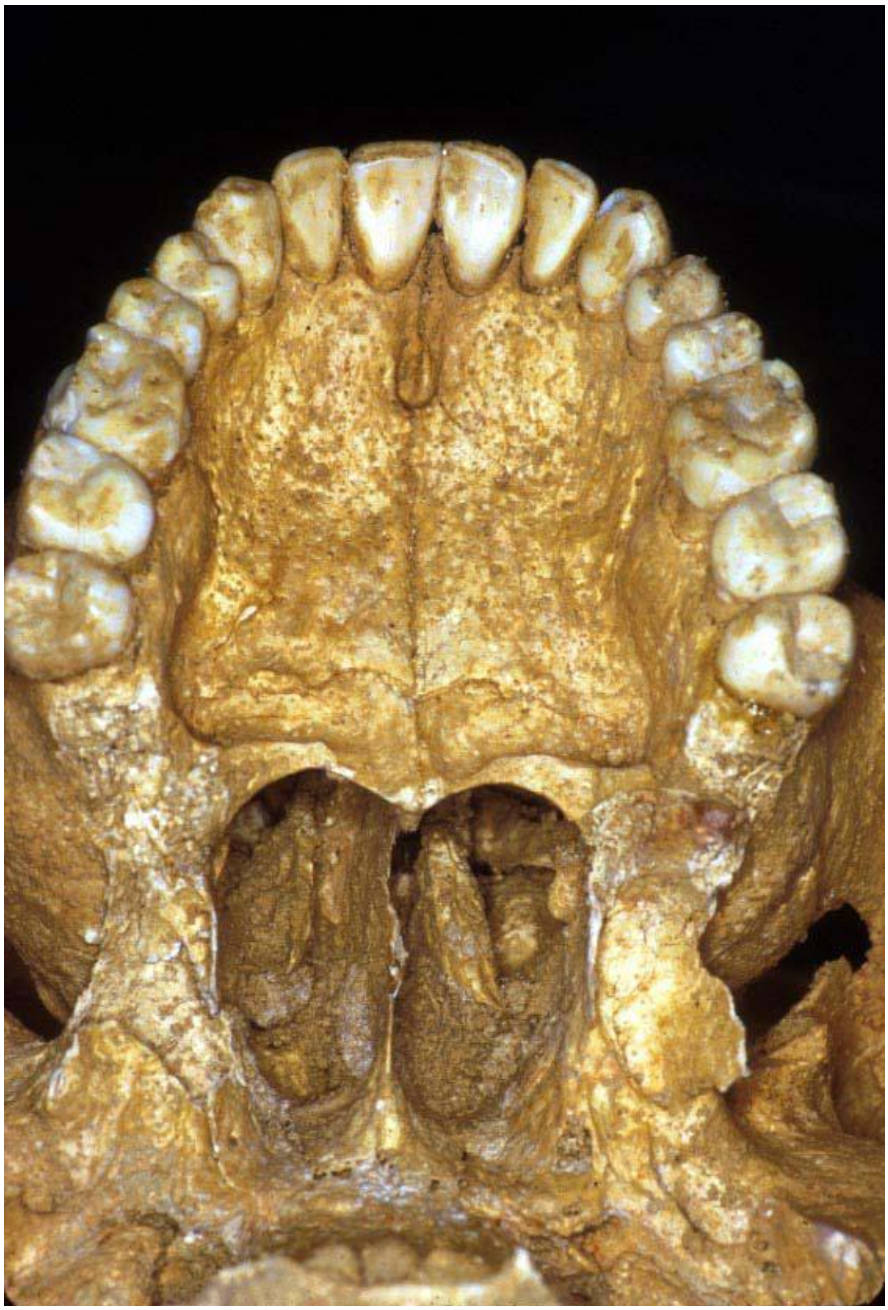


Figure 1b. 1940 Modern jaw that has narrowed the arch and nasal aperture.

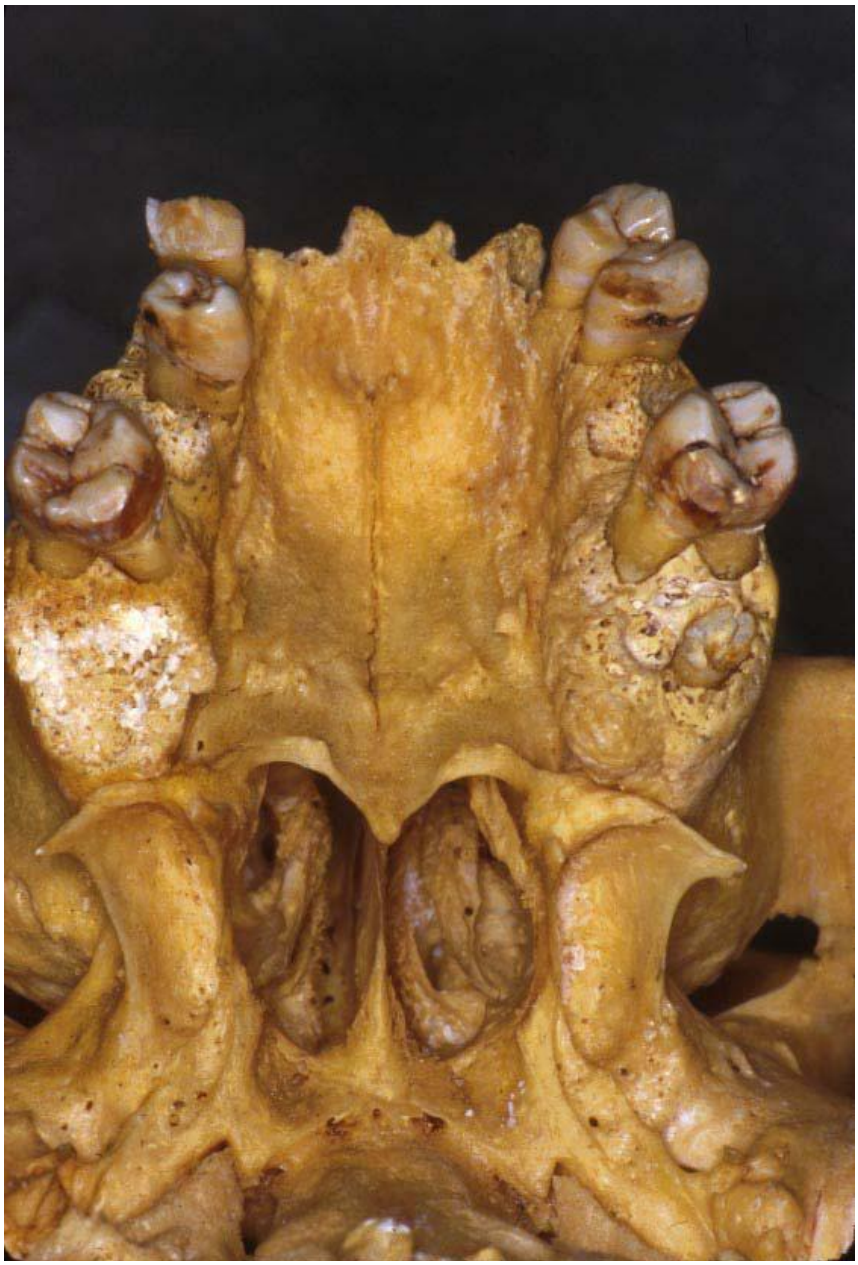
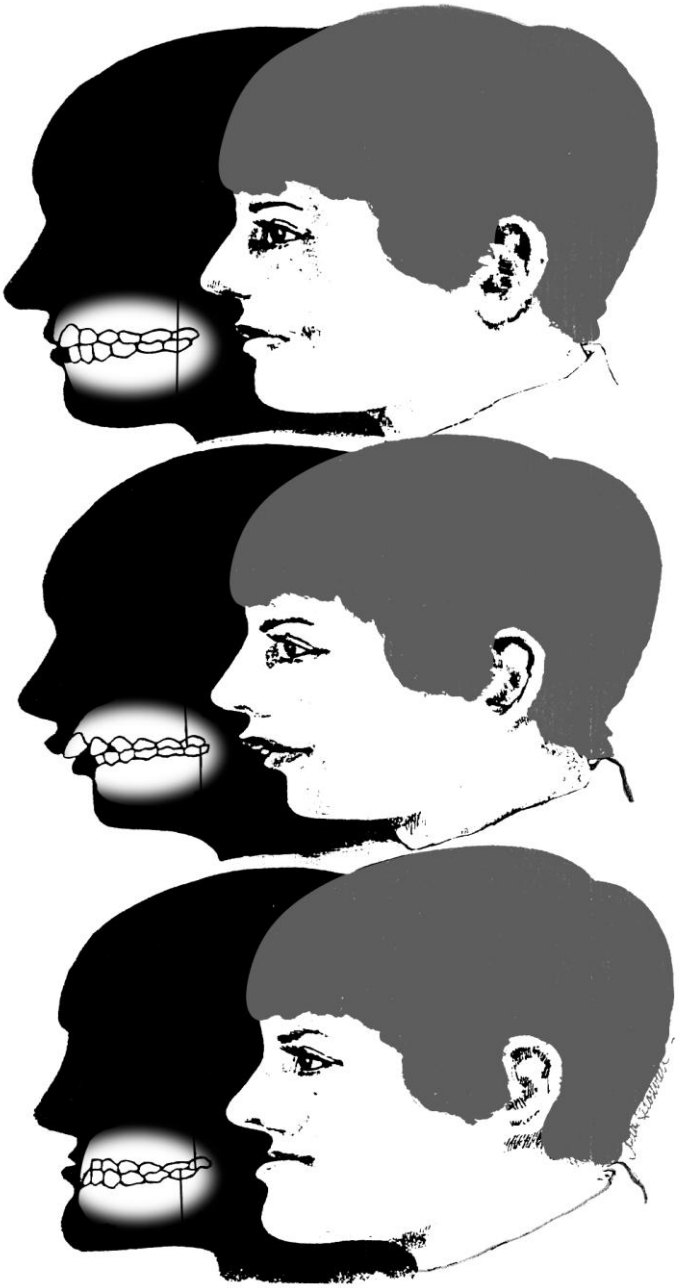


Figure 2. Examples of class I, II and III occlusions. Source google images.



Running head: SLEEPY DRIVING: ETHICAL DILEMMA IN THE PATIENT WITH OBSTRUCTIVE SLEEP APNEA AND PERSISTENT SOMNOLENCE

Sleepy Driving: Ethical Dilemma in the Patient under Treatment for Obstructive Sleep Apnea and Persistent Somnolence

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Case and Dilemma

F.R. is morbidly obese, 55 year old, Caucasian male with a BMI of 55 whose chief complaint on initial consultation says, "I am so tired all the time, especially when driving. I have to snack on foods to stay awake while driving now-a-days." He is having increasing level of fatigue and exhaustion over the last 6 months. These symptoms lead to his referral by his chiropractor to our office. The specific indication was the chiropractor's concern over the patient's extreme exhaustion and hypersomnolence (EDS). These factors have lead to a recent single car motor vehicle accident immediately following a dozing episode.

Previous to this intake the patient's primary care provider added a thyroid supplement after a TSH assessment showed normal serum levels and low normal T4 levels. His primary care provider had seen him on a monthly basis to treat his comorbid gastroesophageal reflux and mild hypertension, but has ignored complaints of fatigue, aside from recommending an exercise regimen and weight loss. Tachycardia, persistent hypertension were noted on exam despite polypharmaceutical management of antihypertensives. No stress testing or echocardiography was recommended at this time by primary care provider (PCP).

On presentation to my office for outpatient evaluation for possible obstructive sleep apnea, which was not addressed in any conversation previously by his primary care provider, he was found to be in biventricular failure and acute pulmonary edema. Without any prior cardiovascular comorbidity diagnosed, or family history of early cardiovascular disease, there were several differential diagnoses to consider. His body habitus did point

to a high level of suspicion for alveolar hypoventilation variant of obstructive sleep apnea as a likely contributor to his current acute condition, if not the sole etiology.

Vital signs 120 heart rate at rest, 30 respiratory rate and room air oximetry of 84 percent saturation. He was diaphoretic and in distress. He was alert, oriented to person, place and time; his Epworth Sleepiness Scale was 19/24 (normal is <8). Central cyanosis noted with prolonged capillary refill, no lung sounds noted to two thirds of his lung fields bilaterally and positive for egophony and pectoriloquoy. S1 and S2 heart sounds with S4 noted, all heart tones were reduced to auscultation. Because family nurse practitioners are not readily able to be on medical staff, or to have hospital privileges, he was referred to the emergency department with explicit written referral instructions from my office. His primary care was contacted through his answering service to notify him of the patient developments. The PCP was not aware of the above stated prevalence data at the time of the telephone conversation. The patient was cautioned to avoid driving until medically stable, as well as while he is sleepy, which appears to be a constant the last six months with reported frequent sleep attacks daily; last of which resulted in a motor vehicle accident (MVA). Once stable the patient was found to be driving daily despite questionable compliance to treatment and persistent EDS.

Obstructive sleep apnea (OSA) is a Department of Transportation (DOT) listed medical disorder for mandatory reporting (Lavie, 2006). If this patient is continuing to drive, then he has placed me in a legal and ethical dilemma now that his EDS is documented this leaves the patient unfit to drive even with limited compliance to treatment or my recommendations (Devarajan, et al., 2007). In 2000, the AMA adopted ethical opinion to address physicians' ethical obligations in this regard (Gupta, 2007). If

clear evidence of substantial driving impairment implies a “strong threat” to patient and public safety, and if the patient ignores the advice to discontinue driving, then the AMA believes it is desirable and ethical for the physician to notify the applicable DOT; however, the opinion clarifies that the physician must follow state law if reporting is required (Ellen, et al., 2006). The opinion also advises that physicians should disclose and explain their responsibility to report to their patients (Felmet and Petersen, 2006).

Should this patient be reported to the department of motor vehicles regarding his persistent hypersomnolence since he has not altered his driving habits? If not, am I liable for not discussing the eight-fold risk for motor vehicle accident in patient’s with severe obstructive sleep apnea reported by Gislason, et al. (1997). If he has a motor vehicle accident what is my liability? What is my duty to patient confidentiality versus my duty, as a clinician, to public health and to the patient’s own risk stratification for further injury related to a repeat MVA? What if the dilemma stems from noncompliance to therapy, in this case the patient has adequate improvement of fatigue when compliant to therapy, should other therapies or a combination of therapies be pursued?

Medical Indication:

This patient was found to have a very severe case of obstructive sleep apnea (OSA). This is a common finding in the ultra morbidly obese. Acutely, the patient has been stabilized by aggressive medical intervention to include bilevel positive airwary pressure (BiPAP). To grade the severity of OSA one must be clear on the apnea hypopnea index (AHI). Apnea hypopnea index, is defined as number of apneas and hypopneas per hour of sleep. Sleep-disordered breathing is defined by an AHI of at least

10 (mild to moderate), 15 to 30 (moderate severe), with greater than 30 events an hour being severe regardless of level of oxygen desaturations (Giles, et al., 2006). In the population of ultra morbidly obese (greater than 100 pounds heavier than ideal weight) the prevalence of OSA has been reported consistently at sixty to seventy percent having significant obstructive sleep apnea (Fritscher, et al., 2007). Recurrent episodic hypoxia, large swings in intrathoracic pressure, and sleep fragmentation all contribute to autonomnic dysfunction, causing an abnormal increase in sympathetic tone and leading to fatigue, hypertension and end organ disease (Chwan, Freedman & Sindone, 2007) (Epstein, 2007). Nocturnal angina, cardiac arrhythmias, including asystole, ventricular tachycardia, atrial fibrillation, and conduction blocks, are symptoms that are common in sleep apnea patients when breathing disturbances and hypoxia are mild, moderate or severe (Caples, Garcia-Touchard & Somers, 2007). Treatment with nasal CPAP, dental appliances and phase two maxillo-mandibular osteotomy have been shown to normalize sympathetic tone in this patient population (Culebras, 2007; Ferguson, et al., 2006). Weaver (2006) has noted that survival and excessive daytime somnolence cannot be fully managed unless compliance to therapy meets, or exceeds, six hours of therapy use per night, all nights of the week. This patient is not meeting this medical benchmark. His current, less than optimal CPAP use, will likely benefit in the short-term in reducing his fatigue symptoms; but risks for heart failure progression remains a guarded long-term prognosis.

The cardiovascular impacts are important to consider in the management of this patient's long-term prognosis (Chrostowska, Szczech & Narkiewicz, 2006). His current concern medically is his high risk behavior of sleepy driving. His risks of driving when

sleepy are more dangerous than drunk drivers (Tobin, 2007; Pandi-Perumal, 2006; Barbe, 1998). His risk of being involved in an occupational accident is increased by 50 percent (Pakola, Dinges & Pack, 1995; Nowak, Kornhuber & Meyrer, 2006). Statistically he is more likely to have multiple accidents in 5 years (odds ratio = 7.3) (Tippin, 2007; NSF national poll, 2007). The American Medical Association in 1999 adopted a new ethical guideline stating that it is "desirable and ethical" for physicians to notify a state licensing authority about patients who, because of a medical condition, may be unsafe drivers (Hartenbaum, et al., 2006). This will place the patient's right to privacy in a tenuous position, straining our patient-clinician confidentiality relationship.

His long-term risks are, exacerbation of heart failure, hypertension, risk of a coronary heart disease, and stroke. Patients with sleep apnea have a two to four times increased relative risk of heart failure and atrial fibrillation. His sleep apnea will continue to cause increased levels of inflammatory, oxidative and vascular endothelial damage associated with atherogenesis (Chwan, Freedman & Sindone, 2007). Patient has an option for other treatments, which alone have not been proven to be as reliably successful as CPAP at reaching treatment success. These treatments include mandibular advancement appliances, tracheostomy and maxillo-mandibular osteotomy. Most patients decline these surgical options due to surgical risk of long-term side effects. Mandibular advancement appliances are well tolerated, but do not fully treat the AHI alone in the very severe cases of OSA (Veasey, 2006). The possible side effects to consider for CPAP that lead to 60% compliance to therapy at one year are: nasal bridge decubitus, mucosal pain, aerophagia, tinnitus, disequilibrium, ear fullness, head aches, nasal congestion, claustrophobia, mucosal dryness, sinus pressure, eye irritation, continued hypersomnolence, insomnia

and rarely aspiration (Weaver, 2006; Basner, 2007; Chai, Pathinathan & Smith, 2006). The concern over likely long term attrition from therapy adherence is real.

Patient Preferences:

F.R. was quite amenable to seeking acute care and followed clinical directions quite well during the E.R. admission. He chose to be admitted to the hospital for 3 days and underwent in-hospital polysomnography. He has used the BiPAP therapy on most nights since his discharge. His use has decreased due to side effects such as unconscious removal and severe dry mouth. He has also reported some ulcerations to his nasal bridge related to the strap pressures of the CPAP mask. This has resulted in reduced usage of the therapy. He has elected to use BiPAP for its noninvasive nature of the therapy. He found improvement in his sleepiness symptoms initially, but now, due to decrease use his sleepiness is progressing. He has questioned on many occasion if his problem is really that bad. He is finding difficult to attribute all his recent problems to a problem with his sleep. He has always felt that he is a "sound sleeper." His primary care provider has given little input on this disorder either, and has focused on the medication list recommended post discharge from the hospital due to his discomfort level with this therapy. F.R. discusses his willingness to maintain this regimen religiously, aside from the diuretics.

His dietary intake has not changed or his physical activity. He feels he is not in an emotional condition to add one more task to his laundry list of new medications and this "machine." He is taking most of his new medications, but skips his diuretic due to the few "missed opportunities to the bathroom." The concerns of his ongoing sleepiness and the documented relationship between his OSA and high risk for motor vehicle accident

were discussed. He feels driving has actually improved and he does not have to eat "snack foods" to maintain alertness while driving now. He does admit to daily lapses of attention. He may have suffered from a mildly obtunded affect on acute presentation and inability to accurately assess his level of impairment, but now in the stabilized phase of his disease, he feels he is alert fully in command of his mental capacities.

In a patient-centered manner of care the patient was asked if he felt he could alternate his driving patterns to allow for car-pooling or more opportunities for his wife to drive. This suggestion was dismissed and the patient feels he is as good of a driver as he has ever been, especially now that he isn't as sleepy. His follow up Epworth score was 12/24 consistent with excessive sleepiness. He denied objective measure of his sleepiness with a multiple sleep latency test stating that he feels "fine." He only falls asleep overtly in church, which is still embarrassing, but not a new occurrence for him. He is quite devout in his Baptist beliefs and feels God intervened to let us spare him to do his work. He has only dozed at the wheel once since his discharge after a long day at work. He attributes this to, "getting back in the saddle too quickly."

Quality of Life:

Following his hospital discharge, F.R. found a new vigor for life that he felt had escaped him due to the process of aging and obesity. He feels that his quality of life has changed "180 degrees." His wife reports less snore noise, suffocating or snorting. The patient awakens with less coffee and snacking in the morning. His wife is not as frightened of his driving now, though she reports he is not fully recovered in this venue.

He is more active in church and at work. He feels one hundred percent better than before the hospitalization.

His shortness of breath persists, as well as his exercise intolerance. He struggles with his chronically painful right knee with walking. The chronic lower extremity edema is improved, but not resolved. He continues to feel self-conscious of his weight, which has actually increased by ten pounds after discharge now that he is, "awake enough to eat better." He is concerned about his weight now that he has more time to think about it with his increased alertness. He actually feels there is a double-standard for the thin people and the overweight people. He is frustrated with knee pain that he feels keeps him from being more active. He isn't snacking as much while driving, but more so at home watching TV, now that he can stay awake better.

Contextual Features:

The patient is sound financially, has a supportive spiritual network and has few strains to his marriage compared to most he reports. He has to deal with higher car insurance premiums and has dealt with higher health insurance premiums now that he has a diagnosis of obstructive sleep apnea. He has thought about life insurance and the five companies he looked into wouldn't cover him with his sleep apnea diagnosis and documented less than recommended treatment compliance. His preacher has asked him to talk about this life changing experience to help his fellow parishioners, but he declined since he is not an orator. He feels better and is not falling asleep in conversations, but continues to struggle with sleepy driving occasionally.

The chiropractor that referred him is a family friend and has been talking about his miraculous changes. The patient wishes his friend would be less free with the information, he is a bit embarrassed. His wife has chided him about the informed seriousness of his condition as it relates to his driving. He has not reported this diagnosis to the DOT/DMV. He would like further discussion or sharing of the information to be limited. He has indicated this in the latest authorization of health information.

Case Analysis and Recommendations

F.R. did have difficulty controlling his congestive heart failure and persistent hypersomnolence status post diagnosis much of this is due to the underuse of the BiPAP prescription on discharge from the hospital. It was found that the patient had been noncompliant of BiPAP therapy. After education and desensitization he was able to tolerate the therapy most of the night, five days a week on average. Patient continued to have significant sleepiness despite this use. Patient has not avoided driving despite his daytime sleepiness recalcitrant to therapy. Treating daytime sleepiness is not consciously the priority for F.R., and his current use of BiPAP is to treat his shortness of breath symptoms, hypertension and heart failure.

It is noted that in the refractory to control hypertensive population eighty percent of these patients may have contributing untreated obstructive sleep apnea to the disorder (Epstein, 2007). It is noted that upward of seventy percent of the ultra-obese population suffers from obstructive sleep apnea and/or alveolar hypoventilation (Fritscher, Mottin, Canani, & Chatkin, 2007). The new prevalence of OSA and the impact of this disorder on many of the most common causes for primary care visits have not been broadly adopted

by the national medical community. This delay presents a public health risk to the driving community since the scope of the undiagnosed population with OSA will not only increase use of primary care, but also acute care due to the car accidents not prevented by the primary care community.

In 1999 the American Medical Association (AMA, 2005) House of Delegates reported that sleepiness behind the wheel of a motor vehicle as a major public health issue; this is my priority ethical dilemma in the management of this patient. To demonstrate the scope of this debate one should cite a measure taken by the state of New Jersey who was the first to label death related to driver sleepiness a criminal offense to be punished as felony vehicular homicide (Philip & Akerstedt, 2006). More importantly for my practice in the state of Oregon, it should be noted that the state of Oregon has a bill in process that requires Department of Transportation (DOT) to include consequences of driving while fatigued in examination for driver licensing (NSF, 2007).

Findings from the Wisconsin Cohort trial notes that relative to stable weight, a 10% weight gain predicted an approximate 32% increase in the AHI. A 10% increase in weight predicted a 6-fold increase in the odds of developing moderate-to-severe SDB (Collop, 1996). Sleep apnea sufferers have a significantly increased risk of motor vehicle accidents (from four to six times) (Sigurdson & Ayas, 2007; Sisley, 2007). This trend may present another layer to this dilemma. The growing prevelance of sleep apnea related to the obesity trends will lead to more sleepy drivers on the road (Vivoli, et al., 2006). It is estimated by the National Highway Traffic Safety Administration (NHTSA) in 1998 that there were annually in the USA 310,000 OSA-related collisions causing 1,400 fatalities and $15.9 billion in collision costs (Taylor & Dorn, 2006). The increased risk of

collisions due to OSA is removed when patients are treated with CPAP and EDS is ameliorated (Santamaria, Iranzo, Montserrat & de Pablo, 2007).

The AMA stance on the responsibility of the clinician in managing the public health risk of sleepy driving is that: (1) the physician should actively seek to prevent sleep-related injuries and deaths by assessment and follow-up of excessive sleepiness in their patients and discuss the possible health and safety implications; (2) the physician should be alert that patients may be impaired by the side effects of our medical therapies and other medical conditions that affect driving capabilities (AMA, 2005; Hartenbaum, et al., 2006; Philip & Akerstedt, 2006; Taylor & Dorn, 2006).

As stated earlier in this paper in Oregon, a physician is required to report specific disorders of their patients to the DOT. Obstructive Sleep Apnea is a DOT listed medical disorder for mandatory reporting. This patient's driving habit has placed me in a legal and ethical dilemma that King (1992) justifies the action of the clinician if he was to deem the patient as unfit to drive due to lack of symptom improvement. The competing interests between my respect for the patient's autonomy and loyalty or fairness to the patient are salient. This dilemma of whether to breach the confidentiality between clinician and patient to fulfill my responsibility to protect the innocent public from a known health concern is the focus of this analysis. The question at hand is the physician-patient confidentiality relationship subservient to the greater good? To jeopardize the patient's ability to drive could diminish his level of autonomy and right to self-determination. Noting the patient's stated preferences personally and goals for optimal quality of life, it would be prudent to intercede on his decision to place himself in a

position that would not minimize possible harms. To fulfill my duty to the principle of beneficience I need to make an effort to secure my patient's well-being. I would be neglecting any level of my commitment to the principle of justice if I did not take into the equation the merit of the situation, or the competence of the patient to appreciate the caliber of the health risk at hand.

Measures to educate the patient about the concerns related to state regulations and measures that can be agreed to, short of reporting to the department of motor vehicles regarding his persistent hypersomnolence, is an option. My duty to the patient confidentiality versus my duty as a clinician to public health and to the patient's own risk stratification for injury is the predominating concern. The dilemma can be initially addressed through education, a patient-clinician contract noting measures taken to address any persistent hypersomnolence associated with this disorder. Heroic efforts to ensure a compatible treatment for his OSA would be appropriate as well. Referral for objective assessment of alertness can give the patient a level of control over the measures to be taken to address this health risk for him, as well as, the dangers for the public that share the road with him. Making it clear about open communication regarding any new compromise of compliance to therapy so as to offer early investigation of alternate OSA treatments. Lastly, the adjunctive treatment of alerting agents and psychostimulants poses an avenue to explore to meet needs of both parties (Rapoport & Banina, 2007).

Ethical Essay

The prevailing ethical dilemma for this case study is the short term risk to the patient's health relating to sleepy driving due to the recalcitrant somnolence caused by

obstructive sleep apnea. The text by Jonsen et al (2006) refers to the principles of ethical treatment discussed in the seminal reference written by Beauchamp and Childress from 1994. The conundrum to prevail is the patient's right to self-determination tempered against the principle of justice for the common good. The action taken in this case study has been supported legally the obligation of the provider to act to protect the public if a risk is assessed. To avoid unintentionally compromising this patient's quality of life for the patient's medical indications for intervention, a strategy to meet the global index of needs should be formulated.

To protect the public it would be a simple matter to remove the patient's driving privileges. To protect the patient's autonomy it would be a simple matter of omission to let him walk out of the exam room with a cursory discussion of the inherent risks of driving under his current neurocognitive state. To address the principle of nonmaleficence it is required for the clinician to not intentionally create a needless harm or injury to the patient, either through acts of commission or omission (Jonsen, Siegler, & Winslade, 2006). My goal as the clinician is to provide a proper standard of care that avoids the risk of harm to the patient, and supports by my moral convictions, which in this case are simultaneously supported by laws of society.

This treatment contract meets the criteria for all the aforementioned principles, both to the needs of the patient, and the public wealth fare. By choosing the educational route, the clinical contract and maintaining a more frequent and diligent follow up of neurocognitive status, as well as, compliance to medical therapy for OSA, the multilayered needs to this case study should be met.

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N721: Genomics in Healthcare

Genomics Case Study on a Patient with Oral Cleft and Obstructive Sleep Apnea Syndrome

Introduction

S.G. is a 20 year old Hispanic-American female who presented to my office with a history of Cleft Lip/Cleft Palate Syndrome (CLCP) and history of chronic snoring. Other than the CLCP, she is a normally developed and mentally capable young adult. She presented to our practice by referral from her Primary Care Practitioner (PCP) to rule out sleep disordered breathing. The patient has history of excessive daytime sleepiness (EDS), anhedonia and loud persistent snoring. My practice is limited to Sleep Disorders Medicine and Dental Sleep Medicine, thus the patient was referred for consultation for treatment alternative to Continuous Positive Airway Pressure (CPAP). The treatment believed to be the most appropriate for her snoring and high pretest likelihood for Sleep Disordered Breathing (SDB) was a mandibular advancement device (MAD).

The patient has a partially corrected antral fistula of the oral palate, which would preclude the use of CPAP due to oral pressure incompetence. Oral clefts, such as, CLCP are one of the most common birth defects in the United States. The problems associated with oral clefts include the following: breathing, skeletal growth and development, hearing, speech and language ability, learning difficulties, and social integration. There is a growing body of evidence supporting an association with CLCP and SDB. More discouraging data supports a likely exacerbation of a baseline level of SDB with corrective surgeries performed to aid CLCP rehabilitation (Leiberman, Tal, Sofer, 1987). The standard of care for interdisciplinary coordination of services for this population includes anesthesiology, audiology, diagnostic medical imaging/radiology, genetic counseling, genetics/dysmorphology, neurology, neurosurgery, nursing, ophthalmology, oral and maxillofacial surgery, orthodontics, otolaryngology, pediatrics, pediatric dentistry, physical anthropology, plastic surgery,

prosthodontics, psychiatry, psychology, social work, and speech-language pathology (Cobourne, 2004). The medical effectiveness of treatments for CLCP can be improved by adding Dental Sleep Medicine Specialty care to the current interdisciplinary team that works on the CLCP population.

Case Presentation

S.G. is a 20 year-old Hispanic-American female with a history of snoring and excessive daytime tiredness and sleepiness was seen in the clinic for consultation to rule out Obstructive Sleep Apnea. The standard tool for assessing sleepiness is the Epworth Sleepiness Scale than ranges from low score of 1 to maximum score of 24. A score above 10 is considerate pathologic sleepiness. She presented with a score of 12/24. Patient had a weight gain of 40 pounds over the last five years to a body mass index (BMI) of 35 (normal <25). She had an initial level III home sleep diagnostic study (WATCHPAT100 device) performed after initial consultation. The study revealed severe snoring recorded at 74 decibels, Respiratory Disturbance Index (pRDI) of 13 events per hour and an Apnea Hypopnea Index (pAHI) of 6 events per hour (ASDA, 1995). Her minimum oxygenation was measured at 93 percent during sleep. These results, in light of her symptoms of ESS are consistent with mild OSA. The study revealed an exacerbation of sleep disordered breathing of a moderate degree in supine and Rapid Eye Movement (pREM) sleep. Her sleep efficiency was reduced measured at 77 percent (normal is >85 %) noting fragmented sleep.

The patient's past medical history includes long time snoring and excessive sleepiness, likely long standing undiagnosed obstructive sleep apnea (OSA) that may have complicated her bipolar depression diagnosis. The patient's extensive surgical history includes

tonsillectomy and adenoidectomy, antral palate fistula repair, and sixteen serial surgical attempts to correct her bilateral cleft palate and cleft lip defects over an eighteen year period. She has an unknown family history due to being adopted in a closed adoption. She is a single college student who lives alone. She is a nonsmoker, she also denies alcohol, nicotine use or recreational drug use. She has a sedentary lifestyle and does not have a standard dietary plan. She has no drug allergies and takes Abilify 10 milligrams.

Physical examination showed a decrease in the anterior-posterior diameter of her posterior oropharynx space and retrognathia. She has post surgical findings of the soft palate that appears reconstructed noting the loss of usual landmarks such as the absence of tonsils on inspection. By body system findings are as follows: General: Patient is well developed in no acute distress. Patient appears stated age.Eye: Patient pupils are equal, round and reactive to light. Patient sclera are non-icteric. Ear: Patient external ears, external auditory canals are normal. Throat: Tongue is large, scalloped borders, cheek biting noted. Soft palate is small with grade of Mallampati 1 out of 4. Patient has narrow palatal pillars noted as a Samsoon – Young score of 2 out of 4. Tonsils are surgically absent. Uvula N/A - surgery. Flat hard palate with narrowing arch form and signs of cleft palate surgery with oral Aatral fistula. Neck: Patient neck is supple, without adenopathy, thyromegaly or jugular venous distention. Respiratory: Patient chest wall is symmetric with clear breath sounds to auscultation. Musculoskeletal: No muscles tender to palpation. Patient demonstrates normal temperomandibular function. Patient demonstrates normal cervical range of motion. Cardiovascular: Patient has1 plus pulses in the upper extremity without clubbing or cyanosis. Patient has1 plus pulses in feet without edema. Neurological: Patient cranial nerves 3-12 are intact. Patient motor strength is symmetric and normal for age. Patient is oriented to person,

place, and time. Skin: No rashes, warm dry and intact. Dental Findings: No Joint sounds throughout range of motion of TMJ bilaterally to stethoscopic assessment. Maximum Opening 43 mm, Overjet 1 mm, Overbite 1 mm, Protrusive 10 mm +6/-4 Right Lateral Excursive 9 mm Left Lateral Excursive 9 mm. Mild deviation to the right on mandibular opening. Periodontal exam notes missing teeth of 1, 6, 11, 16, 17, and 32. Mandibular classification of class II occlusion. Periodontal status I without pocketing or gingivitis.

The patient was deemed appropriate for a mandibular advancement device for her recently diagnosed moderate snoring, mild OSA with significant sleepiness and tiredness symptoms. She was started at a sixty percent measurement of her total protrusive range of motion of the mandible. There are many FDA approved devices for the treatment of OSA and snoring. An example of the MAD used for S. G. can be found in Figure 2. For this patient a device that had less retention to the upper arch of teeth and could be fabricated to occlude the antral fistula was the SomnMed device. She proceeded over 4 weeks to incrementally advance the device a total of 2.5 mm. The terminal advancement resulted in resolved snoring, reduced sleepiness to normal levels with an ESS of 6/24, discontinuation of Abilify due to improved mood stability, resolved nocturia and fatigue. She was noted to wear the device 7 nights of the week for 8 hours of sleep per night. She had no tooth or muscle discomfort with the device use. At week six of treatment she was noted to have lost ten pounds due to more motivation and energy. At week six objective assessment of the device treatment was performed noting a reduction of pRDI to 4, pAHI to 0.1 and snore to well treated 42 decibels. Her sleep efficiency increased to 85 percent.

CLCP are common craniofacial anomaly, requiring complex multidisciplinary treatment and having lifelong implications for these affected people. The etiology of both CLCP is

thought to be multifactorial, with both genetic and environmental factors playing a role (Spritz, 2004). Figure 1 illustrates the process in which the palate and the alveolus and lip are formed in one process. The failure of this formation may result in a cleft that involves all or part of the lip, the alveolus and the palate up to the incisive foremen. The secondary palate is formed by fusion of two shelves that grow medially from the maxillary arches. The incidence of these defects varies according to geographical location, ethnicity and socioeconomic status, but as an average occurs one in eight hundred live births to one in one thousand live births (Gorlin et al., 2001). The complex phenotype represents a breakdown in the normal mechanisms involved during early embryological development of the face. The clinical manifestations of these defects range from cleft of the lip, alveolus and palate. The most common form of CLCP is non-syndromic, occurring as an isolated condition unassociated with any other recognizable anomalies (Prescott, 2001).

Case Analysis

Current scientific understanding of the etiology of these conditions remains relatively poor. This is a reflection of the complexity and diversity of the mechanisms involved during embryogenesis, with genetic and environmental factors playing an important role (Murray, 2002). Embryological origins of the midline facial structures starts with development of the lateral nasal processes, then the alae and sides of the nose, while the medical nasal processes from the intermaxillary segment, composed of the upper lip philtrum, the primary palate and the four incisor teeth. The maxillary process forms the remainder of the upper lip and the secondary palate, consisting of the hard palate and associated dentition anteriorly and posteriorly, and the soft palate (Spritz, 2001).

Many risk factors for OSA are being investigated in pediatrics with the best known being adenotonsillar hypertrophy. Other causes are craniofacial anomalies like CLCP, nasal septal deviation, nasal hematoma, foreign bodies and neuromuscular weakness (Broullette, 1982). A study that analyzed a common treatment for CLCP known as pharyngeal flap surgery was noted to have caused OSA (Leiberman, 1987). This case study notes that S.G. did in fact undergo adenotonsillectomy and several revisions of the pharyngeal flap surgery. Long-term follow up did not include monitoring for OSA by her CLCP interdisciplinary team and was likely present throughout her formative years. Including a dental sleep medicine professional would likely have helped her manage and improve her symptoms of SDB which may have contributed to her labile mood disorder. Instituting the MAD was effective in treating her OSA.

The American Academy of Sleep Medicine is the scientific body that recommends evidence-based treatment guidelines for the treatment of OSA. The MAD is an effective treatment modality for the patient with mild to moderate OSA per these guidelines. There is a dearth of professionals who are competent and well-trained in Dental Sleep Medicine. This is one of the first medical guidelines in medical history that recommend dental professionals to be included into the interdisciplinary team of health care providers to treat a common medical disorder with a FDA approved medical device. The first disorder that dentistry began to manage medically was temporomandibular disorders (TMD). Currently, access to care is limited due to poor coverage of MAD by medical insurance companies. Lack of medical insurance coverage for a therapy often leaves this therapy inaccessible financially for most patients. The lack of medical coverage for a given treatment often psychologically biases patients away from seeking a recommended treatment. My practice is the first to develop a truly interdisciplinary medical insurance management protocol to provide care through medical

reimbursement. We have noted a dramatic increase in patient volume since the advent of active medical insurance management for MAD. The end result has been a thirty-fold higher patient volume than other well-known Dental Sleep Medicine Practices. This increase in patient volume is an obvious reflection of improved access to MAD. The dissemination of this treatment by a Doctor of Nursing Practice has significant implications for nursing practice.

Reflection on Practice

Shortly before the initial patient consultation we were made aware of connections between CLCP and OSA. Discussion regarding a theory called functional matrix hypothesis (Moss, 1997) was being taught as part of a dental sleep medicine course for the treatment of OSA in the pediatric population. The orthopedic medical device was innovated by interdisciplinary research team that demonstrated correction of facial asymmetry of CLCP patients with removal orthopedic devices. This topic introduced the connection between craniofacial hypoplasia as a substrate for OSA. The theory postulates that the "roles of intrinsic (genomic) and extrinsic (epigenetic) factors interact in cephalic growth. The functional matrix hypothesis stresses epigenetic primacy. Simply put, external forces are more significant in the size, shape, form and location of the maxilla than genetic influence." (Belfor, 2008, p. 86).

Despite the significant impacts of this theory and the work being done in this field of orthopedic devices for correction of craniofacial anomalies, there is a dearth of research occurring in this form of treatment for OSA in children. Functional orthopedic appliances area rarely ordered or managed by the sleep specialist. The low utilization for this promising treatment comes down to simple ignorance of the treatment option. The lack of familiarity and knowledge of appropriate referral by the sleep specialist has stifled the implementation of these exciting treatments. Another main barrier to care and innovation is the financial constraints of

the dental insurance reimbursement schemes for medical treatments. Encouraging cross pollination of the treatment and screening of these disorders would dramatically accelerate the entrance of this treatment option to the medical community managing these disorders. There are efforts being made currently to improve public awareness of OSA in children due to the obesity epidemic. These efforts may in fact encourage research and interdisciplinary cooperation for screening and treatment of OSA in patients with CLCP. Medical coverage for medical disorders is generally influenced at the state level. Bill 1634 submitted by the state of California, discusses just this topic of interdisciplinary work between dental professionals and medical teams for improved access to orthopedic treatments for CLCP.

Summary/Conclusion

This case study reveals a serious deficiency in the long-term management of patients with CLCP and pediatric diagnosis of OSA. The presumption that adenotonsillectomy is the sole treatment for patients with craniofacial anomalies or those with obesity is inaccurate. Tauman et al (2006) reported that only 25 percent of children treated for OSA with adenotonsillectomy had complete postoperative normalization of symptoms. Use of MAD is significantly limited in the mainstream treatment of uncomplicated OSA. The use of MAD for OSA in special populations, like this example, is appropriate and effective. Dissemination to Pediatricians and Family Practitioners of current evidence-based guidelines to treat OSA is needed to improve outcomes for children and adults with OSA.

To support this assertion, I would like to note that the National Institutes for Health has designated innovation in treatment and outcomes for CLCP anomalies as “Highest Priority Challenge Topic” for grant monies as referenced by 05-DE-103 for calendar year 2009. The statement mandates that, “practices associated with best outcomes need to be identified.

Projects that answer the challenge could address: Pre-surgical appliances, whether to use and what type." Including screening and dual treatment of CLCP and OSA would significantly contribute to best possible outcomes for this patient population as per this NIH recommendation. Eventually, early orthopedic intervention may contribute to early correction of mid-facial hypoplasia in patients with craniofacial anomalies. With more refined treatment devices and monitoring techniques for OSA this early treatment may correct the facial deficiency, improve airway dynamics and preclude long-term management with MADs. Our practice has plans to implement and disseminate treatment protocols, medical reimbursement protocols and reach out to other specialist to improve access to treatments such as MADs and orthopedic medical devices for craniofacial anomalies.

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Appendices

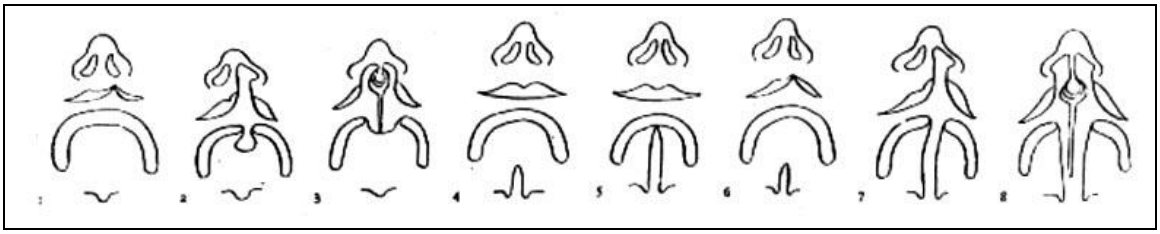

Figure 1. Palate and the alveolus and lip are formed in one process. Adapted from lecture by Dr. Surajit Bhattacharya.

Figure 2. These are examples of the line of Somnomed mandibular advancement devices that are FDA-approved to treat obstructive sleep apnea and snoring. Used by permission from www.somnomed.com.

Obstructive Sleep Apnea and Sleep Disturbances Awareness Policy Analysis

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Obstructive Sleep Apnea and Sleep Disturbances Awareness Policy Analysis

Context

In 1837 Charles Dickens likely described one of the first published descriptions of sleep disordered breathing. In the "Posthumous Papers of the Pickwick Club" one of Dickens' characters, Fat Joe, was described as follows, "…on the box sat a fat and red-faced boy, in a state of somnolency" Sleep Multimedia, 2001). In 1918 Sir William Osler coined the term "Pickwickian" to refer to obese, hypersomnolent patients. In 1956, Dr. Burwell and colleagues in described several obese, hypersomnolent patients with respiratory and cardiovascular collapse that they referred to as "Pickwickian Syndrome" (Sleep Multimedia, 2001). Dr. Gastault, a French investigator, with colleagues made the important observation in 1965 that "Pickwickian" patients had repetitive apnea events while asleep. Dr. Kuhlo and colleagues in 1969 described a significant improvement in patients hypersomnolence and respiratory failure with tracheostomy. This response to treatment lead Dr. Kuhlo and colleagues to deduce that the major reason why apneas occur is the collapse and closure of the upper airway breathing lumen during sleep (Sleep Multimedia, 2001).

In 1978, Dr. John Remmers and colleagues described the interaction between sleep, the breathing muscles of the chest, and the muscles of the upper airway which explained, to a large degree, why the upper airway collapses during sleep (Sleep Multimedia, 2001). This noted finding of airway collapsibility lead to the description of Obstructive Sleep Apnea Syndrome. In 1981 Dr. Sullivan from Australia published the first account of treating Obstructive Sleep Apnea (OSA) with continuous positive airway pressure (CPAP). Soon after the advent of CPAP, the evolution of surgical interventions of the upper airways tissues as a treatment for OSA became more common place. The low compliance with CPAP and less than optimal outcomes of surgery for OSA lead dentists to introduce basic life support (BLS) mandibular advancing devices (MAD) to reduce snoring and apnea recurrence (Thorpy,

2007). An MAD is a series of both upper and lower trays of retainers. The upper and lower trays are approximated together with some sort of material, whether it be metal or plastic, to keep the mandible in a forward positioned while a patient sleeps.

OSA is a significant medical problem affecting up to 4 percent middle-aged adults as noted in the most recent epidemiologic assessment performed in 1999 (Victor, 1999). More current estimates of OSA prevalence estimate 5-15 percent in middle-aged adults (Devulapally, 2009). Many cardiovascular authorities have revealed strong associations for untreated OSA with incidence and poor outcome of hypertension, coronary artery disease, arrhythmia, heart failure, and stroke (WHO, 2002). Therefore, OSA is poised to gain increasing public health importance with the rising prevalence of obesity. Patients with OSA suffer from fragmented sleep and may develop cardiovascular abnormalities because of repetitive cycles of snoring, airway collapse and arousal (Cassar, et al, 2007). Although most patients are overweight and have a short, thick neck, some are of normal weight but have craniofacial abnormalities. Initially OSA was deemed only necessary to treat in patients who suffered hypersomnolence (Beebe, 2006). Like prehypertension and prediabetes severe OSA can significantly worsen health without obvious hypersomnolence. As it turns out, the quintessential excessive sleepiness criterion for clinicians to identify OSA is not very specific to the disease. Just as frustrating for clinicians is the impaired ability for patients to identify hypersomnolence, or in other words excessive sleepiness symptoms. Figure 3. Illustrates the data supporting linkages between OSA and common medical disorders.

Undiagnosed sleep disorders, sleep deprivation and sleep-disordered breathing have impact not only on cardiovascular disease, but also, fatigue-related errors that result in work related morbidity and transportation related mortality (Young, 1993). . Figure 1 describes the increased risk for motor vehicle accidents for patients with untreated OSA. Statistics show that 100,000 police-reported crashes, 71,000

injuries, and 1,550 deaths occurred due to drowsy driving each year in the United States. This statistic is believed to be severely underestimated due to lack of standardized data collection methods. One significant problem with the issue of drowsy driving is the lack of enforcement mechanisms for the worst offenders. A recent report found that every state includes regulations that limit a person's right to drive due to certain medical conditions, with nearly 30 percent of states directly considering sleep disorders. The National Highway Traffic Safety Administration (2004) conservatively estimates that 100,000 police-reported crashes are the direct result of driver fatigue each year. The monetary loss of sleepy driving is estimated at 12.5 billion dollars annually. Like driving under the influence, fatigue needs to be addressed as a public health issue by dealing with the underlying causes of sleep deprivation such as lifestyles, work hours, shift work, or untreated sleep disorders.

Typically, public opinion has to precede the change related to sleepy driving. There has to be sentiment in the public that something is wrong akin to the revelation of drunk driving and its negative impacts on public safety (WHO, 2002). With increased awareness of the OSA problem and its effects on driver safety will be the response to change toward driving habits that occurred with drunk driving in the early 1970's. It takes awareness efforts to make change. Advocacy groups often need to bring the issue to the forefront of public consciousness. One exemplar for possible initial movement toward change may be the recent passage of the "Maggie's Law" in the state of New Jersey in 2003. The CDC recently reported that chronic sleep loss is an under-recognized public health problem that has cumulative effect on physical and mental health (CDC, 2008). Sleep loss and sleep disorders can reduce quality of life and productivity, increase use of health-care services and result in injuries, illness, or deaths. Epidemiologic surveys suggest that mean sleep duration among United States (U.S.) adults has decreased during the past two decades. An estimated 50-70 million persons in the U.S. have chronic sleep and wakefulness disorders. Sleep disorders and sleep loss are associated with mental distress, depression, anxiety,

obesity, hypertension, diabetes, high cholesterol, and adverse health behaviors such as cigarette smoking, physical inactivity and heavy drinking.

According to a 2005 National Sleep Foundation poll, U.S. adults sleep an average of 6.9 hours per night, and 40 percent report sleeping less than 7 hours on weekdays. The National Sleep Foundation reports that most adults need 7-9 hours of sleep each night to feel fully rested, children aged 5-12 years require 9-11 hours, and adolescents need 7-9 hours of sleep each night to feel fully rested excluding the presence of a primary dissomnia. Few formal clinical practice guidelines are yet available for assessing and treating rest or sleep insufficiency and sleeping disorders. The WHO (2002) suggests that some areas of behavioral change are likely to be adopted relatively easily once information becomes available, assuming that the technology is affordable. Other types of change will benefit from active government intervention, particularly those with low rates of risk aversion. Risk reduction may be best promoted through changes in the law or financial incentives and disincentives. An issue tied to sleepy driving education may best emulate current measures for public safety regarding road safety. Under this example of risk a significant number of people might not choose to drive safely, or use a seat belt or motorcycle helmet, but government action can encourage the public to do so, thereby preventing injuries to themselves and to others.

OSA prevalence is largely ignored and undiagnosed in special populations. In 2005, the American Academy of Pediatrics guidelines recommend screening for snoring for all children. The impetus was epidemiological study performed in 1999 that demonstrated a 10 percent occurrence of OSA in children (Li, 2008). The same report noted that nearly 100 percent of children have gone undiagnosed (Beebe, 2006). The untreated OSA in children has been associated with Attention Deficit Hyperactivity Disorder, reduced IQ compared to control children, higher incidence of diabetes mellitus type II, Asthma, failure to thrive, gastroesophageal reflux disease (GERD), arrhythmia and dental

malocclusion and altered craniofacial development (Halbower, 2007). The incidence of OSA in the geriatric population is known to double compared to middle-aged patients (Persson,1993). The rate of screening in this population is only marginally better than comparable cohorts that have fewer occurrences (Rosamond, 2008). Finally, obesity in many cases does not cause sleep disordered breathing. In most cases the disorder is present prior to morbid obesity; that being said, the crowding of the airway increases exponentially with increase in overall Body Mass Index (BMI), particularly in patients with truncal and neck obesity. At a BMI of 35 or greater patients without OSA at normal weight will develop OSA. Patients over the BMI of 40 will likely develop severe OSA. Patients who are ultra-obese with a BMI greater than 50 can develop an average Apnea Hypopnea Index (AHI) of greater than 100 incidents per hour which is three times higher than the minimum criteria for severe OSA. The current obesity epidemic has resulted in a dramatic increase in the incidence of OSA. Any discussion of Obesity must include a discussion of OSA. With the high correlation, any discussion of obesity must include a discussion of OSA. This education is not occurring because many lack knowledge about the disorder from medical professionals, the general public and governmental policy makers.

Congress Bilirakis submitted House Resolution 384 which was referred to the Committee on Energy and Commerce on April 30, 2009. The resolution is an expedited protocol that is not subject to debate or modification. The mandate of the resolution is to generate recognition of the importance of increased awareness of sleep apnea. It (1) supports raising public awareness of sleep apnea; and (2) encourages all Americans to educate themselves and others about the consequences of sleep apnea and its potential treatments. The major stakeholders include politicians, American citizens, patients, public media, medical professionals, Federal and State agencies, transportation industry, and the insurance industry. The most entrenched stakeholder is the medical device manufactures of CPAP machines. These entities include companies like Respironics, Resmed, Tyco, and Embla. These entities combined

due a business volume of over 10 billion dollars a year in the U.S. alone. These same industry leaders manufacture the majority of devices that are used to screen and diagnose OSA and sleep disorders. The will of these stakeholders have been the most visible during the rise of OSA into the public conscious. The combating stakeholder to the rise of OSA awareness is the pharmaceutical industry. The ubiquitous relevance of OSA to major disease management protocols have been mysteriously lacking in industry sponsored medical management protocols. This trend may be true in many other medical guidelines where medical device treatments may be appropriate, but seem to lack representation when a pharmaceutical equivalent is available. An interesting twist to that may supplant the primacy of pharmaceutical companies could be their own distributors; pharmacies. In Australia, Dr. Tran (2009) headed a network of pharmacies in a screening and treatment distribution program for CPAP to patients diagnosed with OSA. The mere mention that the supply chain for the major confronting stakeholder to dissemination efforts for OSA awareness could be the sleep researcher's best ally.

The employer-sponsored health care system will drive the changes to health care the fastest. Examples used in this paper will highlight how rapid new information can be implemented when it results in better profitability versus simple altruistic awareness campaigns.

Problem statement

Too many people have undiagnosed OSA and sleep disorders causing increased motor vehicle accidents, lost business productivity, over-utilization of medical insurance benefits, work errors, physical disability, property damages and lowered health and disease.

Review of Evidence

The review of the literature relating to sleep disorders breathing is comprehensive and substantial. Public safety and transportation agencies have noted the negative impact of undiagnosed sleep disorders (Gurubhagavatula, et al, 2008). Most of the medical academies have found reason to

recommend in actively looking for sleep disordered breathing (SDB) in their guidelines. The WHO (2002) has recommended screening for SDB in patients recently diagnosed with Diabetes Mellitis type II (DM II). Insurance companies have now started adding SDB to list of diseases that needed to be mitigated for when calculated premiums to purchase life, vehicle, workmen's compensation and health insurance policies (Elshaug, 2007). Data is thus divided into medical implications, public safety, worker safety, and disease management categories.

What appears to be the greatest barrier to screening and diagnosis for SDB is the slow assimilation to guidelines by clinicians. The problem is not relegated to only U.S. medical providers, but also other industrialized countries as well. A revealing study performed by Vigg, Vigg, Vigg (2005) assessed the knowledge of practicing chest physicians on the issues of related to SDB. The findings were that basic knowledge was poor. The conclusion was that overall awareness about SDB amongst practicing physicians was poor in urbanized areas of India. Lack of awareness amongst physicians and lack of recent formal epidemiological data can hinder evidence based medicine (EBM) application to medical care (Halbower et al., 2007). Dr. Vigg (2005) has noted that data published twenty-five years ago from the western world clearly showed a poor perception about SDB amongst the practicing physician. The awareness of the disorder and its impact on primary care has improved, but persists in being severely under-diagnosed. Epstein, et al. (2009) with the American Academy of Sleep Medicine has compiled a clinical guideline for the evaluation, management and long-term care of OSA in Adults. The Task Force Members strongly urge that "Patient education should optimally be delivered as part of a multidisciplinary chronic disease management team including the sleep physician, the referring provider and allied health care providers" (p. 268). In 1999 the institutes of Medicine recommended that professional societies raise the standard of practice, improve clinical outcomes, and improved patient

safety by developing evidence-based guidelines. The first consensus report came out in 2005 and has now been currently updated in 2009.

Despite advances in basic sciences and increasing demand for clinical services, very few healthcare professionals receive adequate training or education in sleep medicine. In the last 30 years, a number of national surveys have shown a poor level of training in sleep medicine in American Medical Schools. These same studies have noted a six-fold increase in referral of patients to regional sleep centers for further evaluation after intensive training and education of practicing physicians. The National Commission on Sleep Disorders Research (NCSDR) highlighted the lack of progress relating SDB and general medicine fifteen years ago saying, "What we are dealing with are two gigantic problems of your society: An epidemic of undiagnosed and untreated sleep disorders and pervasive sleep deprivation with all its consequences for errors, accidents, disability, damages and health." Dr. Vigg stated, "if we think pragmatically, the impact of sleep disturbances on health and welfare of our society rivals that of smoking."

To underscore the statement that SDB impacts health on the level of smoking one need only review tragedies of industry. The National Transportation Safety Board has documented many studies that note fatigue to be the leading cause of heavy truck accidents. The sheer magnitude of such catastrophes such as, the grounding of Exxon Valdez, the near meltdown at Three Mile Island, the Bhophal catastrophe, and the explosion of the space shuttle Challenger, all caused totally or in part by sleepy people convinced Commissioners that the consequences of inadequate sleep must be addressed in all components of society. These devastating tragedies forced congress to look into sleep disorders medicine in 1992. During this inquiry research established the national prevalence of Obstructive Sleep Apnea as a potentially fatal illness in which victims stop breathing hundreds of times during their sleep. In the 1992 commission assessment of sleep disorders impact on health Congress passed Public Law

103-43 to establish the National Center on Sleep Disorders Research (NCSDR) to improve the visibility of sleep disorders. Specifically the mandate that came from 1992 National Commission with the passing of Public Law 103-43 was to eliminate the noted absence of awareness that was so pervasive and complete, that it could only be changed by strong, Federal initiative. Now over fifteen years later the level of public awareness has only been marginal.

The current effort of H. Res 384 by Congressman Bilirakis as a House Resolution, which is an expedited protocol that is not subject to debate or modification. The mandate of the resolution is to generate recognition of the importance of increased awareness of sleep apnea, and for other purposes. It (1) supports raising public awareness of sleep apnea; and (2) encourages all Americans to educate themselves and others about the consequences of sleep apnea and its potential treatments. Information relayed by the health liaison for Congressman Bilirakis explains that the measure is a cursory Resolution that is merely an initial step in highlighting the concern of OSA regarding its negative impacts toward the health of Americans. I contend that by appealing directly to the public, the possibility for improving visibility of SDB is more likely than previous attempts made in 1992. As it turns out, the initiative of the NCSDR was derailed in the 1990s due to budget constraints. The monies dedicated for this effort was limited by line-item budget changes made to the Budget Reconciliation Act of 1993. The noted authority, William Dement, known as the father of Sleep Medicine, has stated that public awareness campaigns that mimic the impact of Acquired Immunodeficiency Syndrome (AIDS) awareness need to be emulated to publicize the concerns of SDB. Grassroots efforts were made to educate the public on, what was a once mysterious disease in the 1980s and now are very well known as, HIV infection. What he stated as an issue in 1992 is still in many cases true. William Dement stated our society has little knowledge; the wrong knowledge; we have mythology; we have erroneous attributions, and mostly

plainly stated ignorance. The previous legislation passed in 1993 was found to support National Institute of Health (NIH) research initiatives. This inception of funding and research may have resulted in real resolution of OSA ignorance. But funding was limited and research has not been used by clinicians as it was expected to be. Now, a sign of how futile the effort for awareness as become, in 2009 a meager House Resolution has been suggested to increase awareness in Congress of the issues with OSA. There has been not recommendation for increased funding or any real initiatives to improve access to education on sleep disorders.

Alternatives

Alternatives to the current trend for SDB awareness should include NIH funding initiatives and State mandates. The opportunity available to medical innovation seems to be intimately linked to NIH initiatives. Efforts that align with this agency are most likely to feasibly develop over time. The attempts in the past for the NCSDR that failed appear to be linked to the inability of Congress to fund the Public Law 103-43 adequately due to budget constraints and concerns dealt with during 1992. Policy driven through established avenues have an advantage over novel suggestions for policy change. The alternative NIH initiative most closely resembles what the current trend has followed for SDB awareness. The alternative of making policy changes at the State level also has some traction in place. Noting the current State run Department of Transportation responsibility for ensuring safe driving practices and safe thoroughfares one can logically surmise some traction can be made for SDB awareness. Now that there has been just under twenty years of evidence showing significant health concerns associated with SDB many stakeholders should be more open to accepting the need for SDB awareness.

The numerous stakeholders come from different sections of society. The growing research shows that there may be a combination of scientific and political processes that may be allowing intersectoral actions by different agencies and opportunities for open communication and dialogue. Both alternatives should take into account the fact that SDB will be accepted as a well known, substantial and widespread risk factor for disease (Schechter, 2002). The alternative should also entail cost-effective risk reduction strategies. A partnership between governmental agencies and the private sector are likely to more easily take traction as well in public policy. The national context is very important for assessing the options for risk prevention in SDB. This disease process could, if handled blandly, become low priority for any political action. The situations for public awareness of risk factors for SDB and its sequelae may be best attended to if the knowledge about the most dangerous risk factors is brought openly to public attention. The “Maggie’s Law” discussed early would be the best spring board to disseminate state-based policy. Working with state agencies to assimilate well planned driver’s safety laws would decrease the impact of untreated SDB on motor vehicle accidents. Establishing and enforcing the new laws concerning those who chose to drive with untreated SDB is important as well.

Coordinating efforts with the private sector and governmental agencies could improve public awareness of SDB. An exemplar for this alternative would be the current initiative between Snyder trucking and the States that the company does business. Data gathered from internal screening of truck drivers for Snyder has resulted in new and profound statistics. Data found by Sassanai, et al., (2004) were, (1) 28 percent of truck drivers have sleep apnea; (2) 2-7 times increased risk for crash with sleep apnea; (3) treatment cannot only improve safety and driving performance, but other health outcomes; (4) treating all US drivers suffering from apnea would save $11.1 billion dollars in collision costs and save 980 lives annually). Robust improvements in safety and health outcomes in this private sector initiative

would be used to support data that would lead to increased penetration of “Maggie’s Law” type legislation into more representative State laws. Suggested campaign goals that are currently associated with this exemplar include: (1) Increase driver and industry awareness about sleep apnea and other sleep disorders including associated dangers, risk factors, symptoms and treatments. (2) encourage drivers and others in the trucking and motorcoach industries to seek medical help if they are experiencing symptoms of sleep apnea or other sleep disorders. (3) Ease driver and industry fears around sleep apnea by communicating that treatment will help reduce crash risk and by clarifying guidelines regarding fitness for duty (NSF, 2006). “Get on the road to better health” was the joint program of the Federal Motor Carrier Safety Administration and the National Sleep Foundation (Pagel, 2008). This joint effort between government and private sector resulted in tangible progress in the form of a new industry standard for worker safety in trucking. The industry “Joint Statement” issued in September 2006 recommended that all CMV drivers with typical signs of OSA be tested and treated, including: BMI greater than 35, neck size greater than 17 inches, hypertension, snoring, excessive daytime fatigue or witnessed apneas and a positive Epworth Sleepiness Scale of greater than ten (Hartenbaum, et al., 2006). This joint statement is now being considered by multiple state departments of transportation. Schneider trucking found some revealing data during its National OSA Screening Program which included: (1) 30 percent accident reduction in the OSA treatment group; (2) $500-700 dollars per driver per month health care savings twelve months post CPAP as compared to twelve months pre-CPAP in the cohort; (3) retention in the treatment group at the time of the study showed twice the retention as in regular fleet resulting in less turnover in employment (Marin et al., 2005). As part of the Schneider OSA screening Program there was a recommended education arm. Monthly newsletters, text messages were sent to

drivers to remind them of sleep testing and follow up appointments with the clinician, and driver handbooks.

Project Outcomes

Comparison of current trend and alternatives strongly urges acceptance of alternative measures. The projected outcomes associated with increased awareness of SDB would be primarily to increase the measured number of screened, diagnosed and then treated patients with SDB. The most visible public outcome would be to show evidence-based treatments for OSA into current mainstream practice guidelines for appropriate co-morbid disease processes (Khayat et al., 2009). Currently, OSA is recommended as a differential diagnosis for DM II, hypertension, Atrial Fibrillation, Depression, ADHD, Obesity, Myocardial Infarction (MI), Cerebral Vascular Accident (CVA) and metabolic syndrome. Currently none of these pharmaceutical sponsored treatment guidelines depict OSA in the treatment decision tree (Hla et al., 2002). Projected outcomes should include decreased annual motor vehicle accident occurrence, improved heart failure outcomes, improved rehabilitation and prevention of CVA. Improved scores for surveyed clinicians for cursory SDB related facts. The NCSDR recommended specific outcomes to measure improved SDB awareness. Large-scale public awareness campaigns are needed to change behaviors or conditions that lead to family dysfunction, workplace accidents, automobile crashes, lost education and income opportunities, disability and premature death related to an untreated major cardiovascular risk factor (Hartenbaum, et al., 2006; Talmage et al., 2008)). Support of educational programs aimed at researchers, accident investigators, and law enforcement officials in order to identify sleep-related transportation and workplace accidents properly (Pack et al., 1995). Establishing guidelines for how officers should respond to non-commercial drivers who are impaired by fatigue (Philibert, 2005). The continued positive results, found in ongoing research,

of the effects of how CPAP improves alertness in drivers with OSA will codify current commercial driving safety program efforts. Table 2 illustrates the decreased risk for motor vehicle accidents in patient treated with CPAP for OSA.

Apply Evaluative Criteria

The standards used to measure the projected outcomes for the listed alternatives include, whether the alternative meets the health needs of the population and is it consistent with current national priorities, will the alternative reduce the projected risks of OSA, is the alternative costly to implement, will the alternative increase the rates of initiation/compliance with OSA treatments, and does the evidence base support the alternative (preliminary data). The core challenge with the current state of OSA awareness is the main challenge that the proposed legislation is attempting to address. Review the literature it appears this current effort falls short from addressing the real national priorities for improved health care delivery. The need for improved OSA awareness has been compared to the dire situation that was experienced prior to the mass dissemination of information relating to Human Immunodefiiciency Virus (HIV) infections twenty years previous.

The experts in sleep medicine feel they are fighting an uphill battle with OSA awareness. The auspices of sleep medicine and OSA have left the effort for awareness to be hampered by the stereotypes held by both the lay public and health professionals not trained in sleep disorders medicine toward sleep disorders and their lack of representation in other medical management guidelines. With continued research in to OSA, or in other words, “sleep suffocation,” it turns out that the disorder is just as important to treat to improve cardiovascular disorders as it is to get a “good night’s sleep.” The following are measured on the following table. Collins (2001) describes “three Es” effectiveness,

efficiency and equity to evaluate policy issues and their viability to come to fruition. The following table 1. describes the pros and cons for both the H. Res. 384 versus the recommended alternative courses of action to improve screen and diagnosis of OSA.

Table. 1

Alternatives	Meet health need of target population/consistent with policies/priorities?	Reduce risk of OSA (reduce expense from OSA)	Resource: expenditure of intervention	Increase identification of OSA	Evidence to support outcomes
Current Trend	Below average	Unknown	Low expense	below average	unknown
NIH Funding	Below average	Unknown	high expense	Below average	unknown
State Legislation/ Private Sector	meets	meets	high expense	exceeds	exceeds

Make the Decision

The best supported effort to improve OSA awareness out of the presented alternatives is the government and private sector partnerships. Noting the over a decade slow pace of poor advances in awareness for OSA, the current trend alternative appears to be insufficient. The H. Res. 384 doesn't have much depth or substance to it. It is interesting to note that the sponsor of the bill is the son of the congressman who listened to the sleep researchers back in the 1990s. This factoid is highlighted to reflect the deep rooted lack of understanding by politicians and the public on the impact of untreated OSA. The admitted aim by the sponsor of the bill is merely to raise general awareness with Federal

Politicians and the general public (J. Gray, personal communication, November 9, 2009). No funding is tied to the measure and leaves its impact to be small in its current form. The current stakeholders may induce a significant number of conflicting interests that could result in recommended testing measures, treatment options and research methodologies that may have intrinsic conflicts depending on the stakeholder's likelihood to financially benefit from political mandates. A national employer who is self-insured both for medical and workman compensation insurance has resulted in the best measurable outcomes relating to increased OSA screening, diagnosis and treatment. The driving forces and converging streams appear to have facilitated this partnership When there is a confluence of streams, a window of opportunity is opened (Blankenau, 2001). The partnership with the business industry in the U.S. is a good example of this window of opportunity. Capitalism is the strongest driving force for political change. When business stakeholders are able to become more efficient by reducing cost related to injuries and lost productivity, then the motivational to change the current status quote regarding OSA treatments will materialize (Tregear et al., 2007). Blankenau (2001) believes for change to be viable then, solutions must be acceptable to the policy community such that they have technical feasibility, are affordable, and are within constraints of values of the policy system. Emulating efforts like the departments of transportation and Schneider trucking company may lead to the most expeditious vehicle for policy change regarding OSA awareness campaigns.

The H. Resolution 384 is a good example of the willingness of the U.S. Congress to acknowledge a problem without measurable action. The mere mention of OSA by policy makers denotes an open opportunity for public support for OSA advocacy. The fact that industry leaders such as the transportation industry and corresponding state departments of transportation are actively implementing and achieving improvements in a measurable fashion better safety and decreased disability for

constituents allows for a growth of public interest in this disorder in the near future. The current climate for thrift, cost-savings and overhead reduction for business makes the likelihood for a catalyzed partnership between industry and government inevitable. The evidence is irrefutable and as the epidemic of AIDS was the galvanizing health concern of the 1980s-90s, so too will OSA become our shared beacon in the coming decades.

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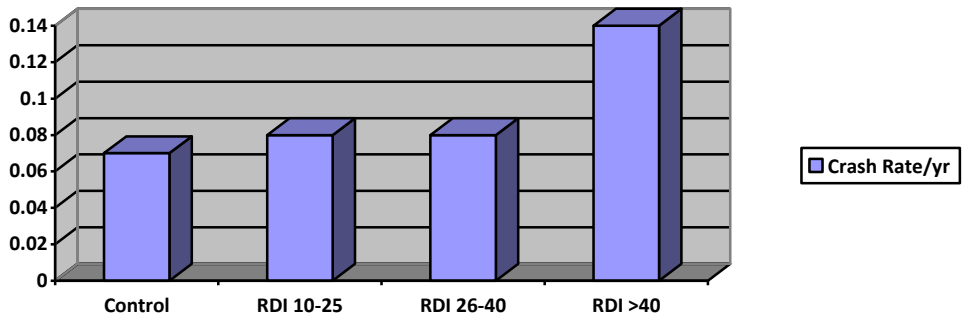

Figure 1. Relationship between severity of OSA and crash risk. (George, 2001)

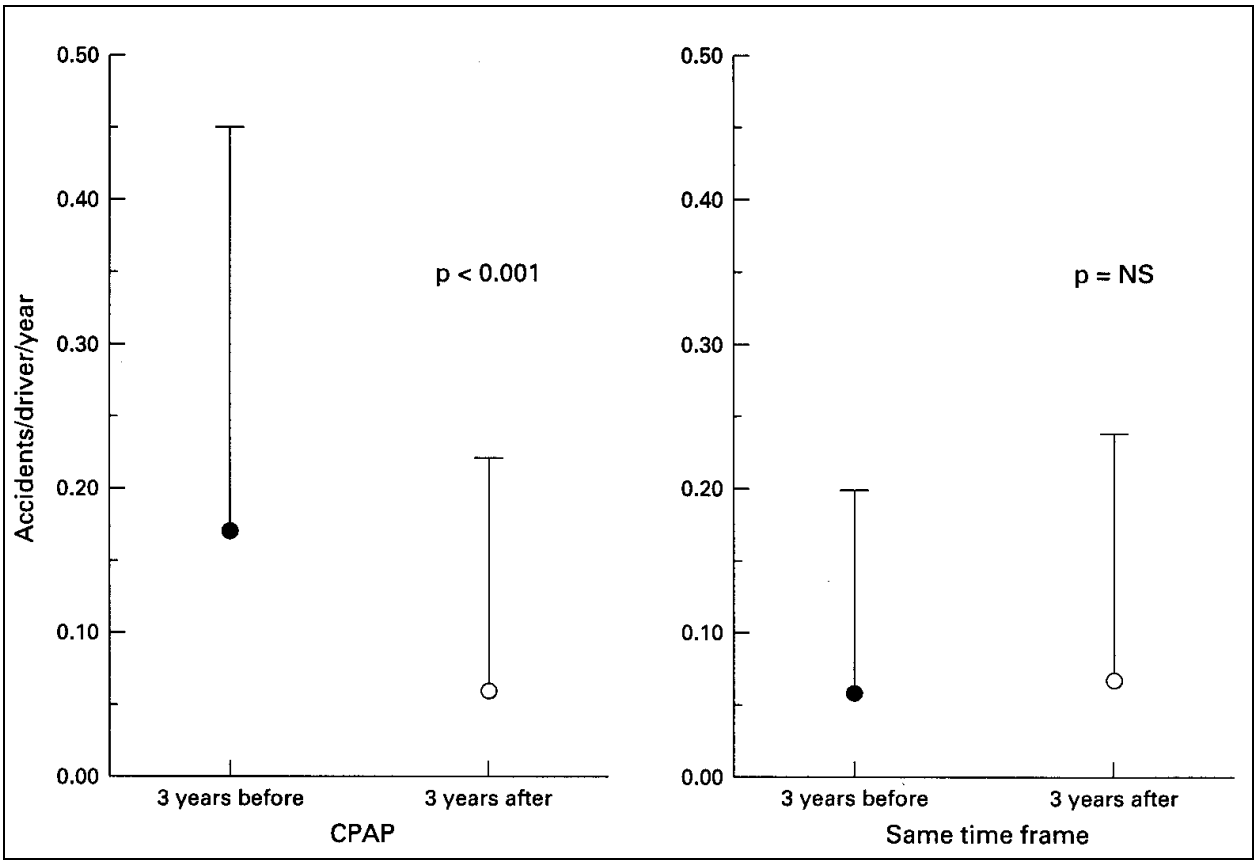

Figure 2. Most definitive study – crash rates before and after CPAP and in controls. (George, 2001)

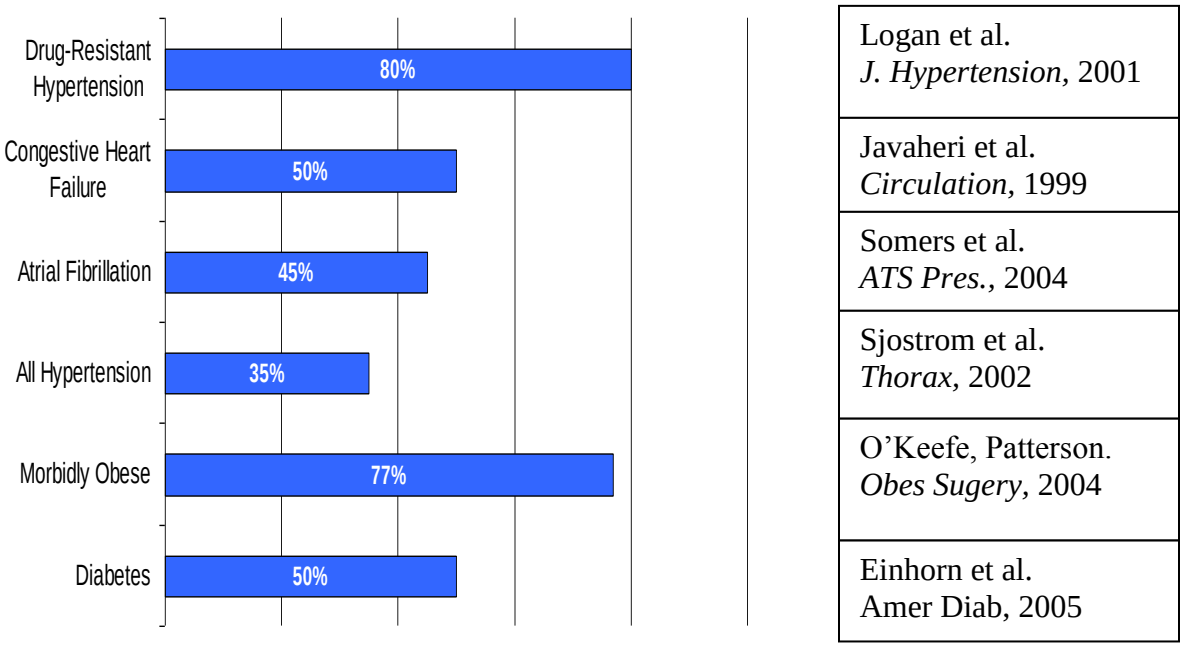

Figure 3. Prevalence of obstructive sleep apnea and its co-morbidities.

Inter-professional Sleep Diagnostic Center

Business Plan

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March 18, 2011

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Introduction

The current national physician provider mix is gravitating toward providing greater numbers of specialists and fewer primary care physicians, while the opposite trend is occurring in sleep medicine (Lacey, 2009). With sleep board examinations now being administered by the American Board of Internal Medicine (ABIM), the window of opportunity is narrowing (and closing after the 2011 exam) for non-fellowship-trained physicians to qualify for board eligibility, which will eventually result in fewer physicians being board certified yearly in the specialty of sleep medicine (Lacey, 2009). But with the growth of sleep science and importance of treating sleep disorders, the clinical expertise needed to properly evaluate, diagnose, and manage more complex sleep patients requires practices whose primary clinical specialty is sleep medicine. This paper introduces an innovative model of multi-disciplined and inter-professional sleep specialty practice using a new innovative model for care delivery. Delivery of alternative treatments to CPAP, as well as CPAP therapies and portable testing to meet sleep diagnostic demands in current society is the goal of this paper.

Name of Organization: Sleep Medicine Network

Sleep Medicine Network (SMN) is a full service sleep disorders center, designed to provide a multi-disciplined approach to the evaluation and treatment of sleep disorders, as well as create an atmosphere of trust in a managed care environment. Quality of sleep has a direct effect upon waking performance, which includes attitude, and the overall quality of life. Usually the causes of poor, non-restorative sleep is often unknown to or unheeded by the sleeper. The standard sleep center relies heavily in on-site polysomnography (Strollo, et al., 1996). Rapid advancements in technology have expanded the range of available methods for testing patients with

sleep disordered breathing/obstructive sleep apnea (OSA) to quality portable testing (Chesson, 2003) (Redline, Tosteson, Boucher & Millman, 1991).

This paper reviews the advantages and limitations of unattended at-home evaluations for patients with suspected sleep disordered breathing. Thriving in today's economy demands that sleep professionals adopt fiscally conscious practices and innovative ways to improve and streamline patient care. Increasing the number of patients seen daily has become an economic necessity and the utilization of nurse practitioners in specialty practice has become more common place, thus integrated practice with FNP will be reviewed in this business plan. It also follows patient driven treatments for sleep disordered breathing beyond continuous positive airway pressure (CPAP). Currently, CPAP treatment is offered to the vast majority of patients with OSA (Campos-Rodriguez, F. et al., 2005). Adherence rates to CPAP can be low without close monitoring of treatment (Standards of practice committee of the American sleep disorders association, 2005). Clearly action is needed.

CPAP therapy has been the gold standard for decades and, due to recent scientific advances, dramatic improvements near interfaces and PAP technologies to improve patient compliance have been implemented (Ruehland, 2009) (Morgenthaler, 2008). There has been another major development in OSA compliance such as variable PAP devices and smaller better fitting nasal interfaces (Pusalavidyasagar, 2006) (Thomas, 2005). Recently oral appliances have been provided by an emerging group in dental sleep medicine (Kushida, 2006). These oral appliances can be used alone as an amelioration for OSA or they can be used to compliment CPAP.

A sleep center is a healthcare organization in which several sleep problems including sleep apnea, snoring, sleeplessness, insomnia, daytime sleepiness, narcolepsy, nightmares/terrors, restless leg syndrome, etc., are given diagnostic evaluation and treatment (Ruehland, 2009).

Studies have shown that these disorders are much higher in the populations than earlier anticipated (Fletcher, 2003). About 40 percent of the people have day time sleepiness such that it interferes with their work, and about 18 percent reported this more frequently (Dyken, 1996). About 34 percent of the population experiences snoring a few days a week, and about 16 percent of the male and 10 percent of the female population experience snoring more frequently (Frost and Sullivan Marketing Report, 2001). More than 50 percent of the population experience insomnia and about 62 percent of the population have some disturbance in their sleep (NSF, 2001).

A variety of testing options are available for the diagnosis of sleep disordered breathing (Kuzniar, 2008) (Lain, 2003). To date, standard attended polysomnography is still the gold standard (Kryger, 2000). The higher level of complexity in sleep testing is considered level 1, or 15 channel polysomnography (Kushida, et al., (2006). Many ambulatory level 2 and 3 testing devices have been validated for use in the home, and they are suitable for selected patients. Level 4 is a single channel sleep parameter such as nocturnal oximetry. This modality has been deemed poor in specificity and sensitivity for OSA (Lain, 2003). As awareness of sleep disordered breathing increases and the demand for testing rises, there has been a move to improve access to testing and to reduce its cost (Chesson, 2003). One approach has been to conduct sleep evaluations in the home. This portable testing can be as complex as a laboratory evaluation and measure all of the same parameters. Portable testing is providing less complex testing limited to assessing just a few parameters (Chesson, 2003). The provider who orders unattended ambulatory testing for sleep disordered breathing should be knowledgeable about other sleep disorders (Bradley, 2000).

Sleep disorders are a group of syndromes characterized by disturbance in the patient's

amount of sleep, quality or timing of sleep, or in behaviors or physiological conditions associated with sleep. The aims of testing a patient with suspected sleep disordered breathing are to confirm the diagnosis, to determine the frequency and severity of respiratory events, and to evaluate the physiologic consequences during sleep that occur secondary to the respiratory events (Bradley, 2000). The physiologic variables that are evaluated in a standard polysomnogram include changes in oxygen saturation oxygen saturation, cardiac arrhythmias, arousals caused by respiratory events, and sleep fragmentation caused by frequent arousals (Fletcher, 2003) (Gyulay, 1993) (Gyulay, 1987) (Fleury, 1996) Gugger, 1995) (Standards of Practice Committee of the American Sleep Disorders Association, 2005). The physiologic consequences of untreated OSA can be daytime sleepiness, memory loss, irritability, psychomotor impairment (Zikek, 2002).

Continuous positive airway pressure (CPAP), bilevel positive airway pressure (BiPAP), and auto-titrating CPAP are important treatment options for OSA (Berry, 1999) (Bradley, 2003) Fletcher, 2003) (Morgenhaler, 2008) (Gugger, 1995). Currently the use of alternative treatments to CPAP for OSA has been under-utilized by sleep specialty clinics. These alternative therapies are sought after by the patient outside the sleep specialty system with otolaryngologists and general dentists for those patients who do not want to attempt CPAP treatments. Sleep clinics are rapidly becoming associated with CPAP as their exclusive treatment option. Dental sleep medicine has a significant spot to fill on the sleep lab team, yet most sleep labs are missing this patient service and business opportunity (Feedback research services, 2001). The growing need for sleep testing, lack of treatment options on initial diagnosis of OSA, and growing burden of health care costs transferred to the patient in the form of high deductibles and copayments to insurance coverage represent a compelling need for the services of a company willing to move in the direction of portable testing like Sleep Medicine Network (Yamashiro & Kryger, 1995) (Frost

and Sullivan Marketing Report, 2001).

Description of product or service

The sleep center will provide both diagnostic and treatment sleep studies, as well as provide consultations, follow-up visits, and personal treatment plans for a wide variety of sleep complaints. The team of medical professionals contracted with SMN will include a Family Nurse Practitioner with curriculum vitae in sleep disorders medicine and a dentist with a board certification in dental sleep medicine. The dentist has been well acquainted with this region as the sole provider of dental sleep medicine services. This allows the center to handle all sleep disorders in-house including sleep disordered breathing, insomnia, circadian rhythm disorders, excessive daytime sleepiness, and parasomnias. The center will sell sleep related breathing equipment and mandibular advancement devices as a means of providing both additional revenue to the center and improve patient convenience and monitoring of treatment, “one stop shop” service option.

Target Market

Our primary market strategy will focus on distinct consumer groups; sleep specialist, primary care physicians and persons within the community complaining of sleep disorder symptoms. This will occur first within the market area of Portland, Oregon and Vancouver, Washington. Once this market is clearly established, we will continue to expand our service to new markets including northwest Washington and southern Oregon.

Targeting referring physicians is a priority goal; without the support of the primary care physicians within those target markets, targeting other groups will have a diminished effect. Also, this specific market segment has the ability to create a steady stream of both new and returning patient referrals, which in turn created a source of return revenue for our center, SMN

intends to operate within a managed care market, and this system allows primary care physicians (PCP) to control the flow of many potential customers. Even if a patient requested a referral to our facility, their PCP makes the final decision.

Another target market includes those consumers who are suffering from a sleep disorder that have not been successful with previous CPAP treatments and possible sleep disorder and desire a sleep evaluation and treatment plan. For those patients with a fee-for-service carrier, SMN intends to offer lower pricing and courtesy discounts to reduce the barrier of higher deductibles. Those consumers can range in age from 14 years of age to the "very" elderly with any number of associated comorbidities (ASDA, 2005).

Potential patients of SMN
Patients with arthritis
ADHD/Psychiatrics/Depressions
Elderly
Diabetes Type 2
Impotence
Pre- Eclampsia
Fibromyalgia
Pain Management
Shift- Workers
High Risk Occupations
Males/ Females over age 55 (estimate 4-20% of general populations
Patients with a BMI greater than 28 (*weight- loss groups*)
Cardiology patients (specifically CHF, hypertension)
Those patients within all adult age groups who complain of:
Non-restorative sleep
Morning headaches
Excessive daytime sleepiness
Frequent awakening
Gasping at night
Snoring
Nighttime urination
Chronic fatigue

Inability to sleep at night

Channels of Distribution

Accessing the sleep center will be an easy step for the primary care physicians; they may chose to either fax or phone over their request. All other steps will be handled internally, including contacting and scheduling patients, mailing educational material/questionnaires to patients, conducting all pre-authorizations, and verifying that all patients are scheduled for a follow- up visit with their referring physician within one week of the test. For patients who have fallen out of the traditional sleep disorder management due to treatment failure or missed OSA screening opportunity through primary care services direct patient education will be implemented. The major strategy for reaching these consumers will be through radio broadcast media.

Our distribution will expand by opening new sleep centers throughout the region. Several other forms of distribution will be used. We will have an active website that will allow customers to view our service and locate the closest sleep center to them. They will also be able to use this website to view the latest sleep disorder treatments and educational materials. In addition, we will offer community health screenings, as well as attend local conference in which we will address sleep disorders. Offering less invasive treatment options, in-network treatment delivery and portable testing this facility will increase market share in a saturated market that currently has twenty in-lab bound sleep diagnostic centers that have chosen to not move into portable testing modalities.

Advertising

Need: assignment of company spokesperson, radio personality
Radio: cost and demographics research

Research/validation trials

Requip XR, phase III trial
Dental sleep medicine training facility
Adjunct faculty to (American Academy of Sleep Medicine) AASM sleep medicine fellowship
OPAP (Dental oral interface for nasally restricted patients)
Nasal Valve Dilating Appliance
Invention of SleepApp mandibular advancement device
Training facility for Dental Sleep Medicine in the commercial market
Invention of HPAP (pressure reduction interface for PAP treatments)

Administration

An LLC will be created with 3 founding members; the division of this LLC will be determined between the founding members, and this will achieve the following outcomes: the LLC will maintain a primary ownership of no less than 14% of all outside clinics, and no less than 67% on internally sponsored clinics. Internal sponsored clinics include any clinics that the LLC opens directly as opposed to "consulting" on new clinics (Stolze, 1996).

The administration of SMN will be organized in such a way to allow SMN to maximize profits while supplying patients with superior customer service. The company will be broken into three key divisions that will include customer service, finance, general operations, and marketing and promotions.

The center's managing director will provide the highest level of direction and supervision within SMN. This person will maintain responsibility for hiring and training all team members, including both technical and support staff positions. This training will occur during the first 30 days after the initial hire date, and all employees will be on probationary status during that time.

The center managing director will provide the employee with an exit review upon completion of the 30-day probationary period; this will include a service and sales skill checklist, which must be "passed" by the managing director.

Internal selling

The entire staff of the center, however, will participate in the selling and promotion of our services as a regular component of their employment with SMN. All staff must understand and agree to the following the below guidelines as a part of their regular duties. This includes the following service components as related to job description:

Administrative Assistant: (1.0 FTE)

1. All incoming calls will be answered in a positive and friendly manner, the name of the organization will be given, the caller will be allowed to express his needs without interruptions, and they will be thanked for calling SMN at the end of the conversation.
2. All requests for assistance in calling insurance carriers, adjusting test times, and requesting educational materials will be honored. If the request does not seem appropriate, the caller will be asked if they can hold and the call will be given to the lead technologist present.
3. The complete test will be faxed and mailed to the referring physicians the day it is received; all faxes will contain a hand written thank-you on the cover. Request for a second treatment sleep study and DME equipment will be done via telephone. This call will be documented in the patients chart along with the physician's response; the center's managing director will review all denials for patterns and potential service issues.

Sleep Technologist: (3.0 FTE)

1. This position maintains primary responsibility for greeting the patients as they arrive for their overnight sleep study. Staff will converse with the patients in a warm and friendly manner

and make certain they are at ease in the center.

2. The study data will be collected following AASM guidelines; all study tracing will be of the highest level of quality with a 95% match on sleep scoring rules.

External

In an effort to increasing patient growth, SMN will utilize an external sales organization to compliment its own internal sales policies. These two organizations will work towards a common goal of increasing company profits. This includes increasing the volume of patients who access our service and by finding ways within the organization to maintain and/or cut costs. SMN will consider the idea of hiring professional representatives in a consulting capacity to market and promote company service.

Potential barriers to market entry

Barriers include a lack of public awareness regarding the importance of testing and treating sleep disorders. SMN is a business that is not readily identifiable to consumers, and public education as to our services will be at the forefront. Another potential barrier includes our ability to contract with regional health plans this needs to include a blend of fee for service and managed care contracts. A significant barrier will also be the use of a Nurse Practitioner as the medical director of the practice. This is outside the usual direction of medical practice in the United States. A growing number of states are providing FNP practice opportunities to provide medical care autonomously from physician supervision. In Oregon and Washington a FNP can manage their own practice. SMN will begin as this form of inter-professional practice. Once the concept is proven, the goal for growth will be to align the practice with industry leading sleep specialists and otolaryngologists.

We will reach our target populations by forming a close alliance to the various internal

and family medicine groups within our target region. We will need to reach these medical populations by dispatching sales reps to inform and educate them regarding both the importance of sleep screenings, as well as the positive services SMN can uniquely provide.

If there is already a competing center within the target region, SMN will investigate to see if that center has more referrals than access, and if the demand is high for the types and level of service that we will be providing. If the current center is booked out two months or more, we can gauge the demand for our service by their waiting list. We will in initially target their overflow to sustain our business expenses, while we work on building our own customer base.

Customer Satisfaction

Customer satisfaction is the single most important issue to SMN. If customers are not satisfied with the service they receive, they will look to another practice to fulfill their needs. Several techniques will be used to measure the satisfaction of patients.

- SMN will monitor and maintain a patient database than list patient outcomes. It is our goal to effectively resolve the patient's subjective complaints within 6 months of the initial visit.
- SMN will monitor the amount of the time patients spend in the waiting room before being seen. SMN will limit this time to 15 minutes or less. Patients must feel that their personal time is respected.
- SMN will send satisfaction surveys to all patients within one week after their appointments. These surveys will be utilized by SMN to determine what areas, in any, need improvement.

Outcomes Measurement

It is the policy of the center to conduct random audits within all operational areas to ensure that the above guidelines are being followed. This will consist of confidential peer review

evaluations, patient follow up calls, and solicited criticism and comments from outside referrals sources. As well, random charts will be pulled to review charting, billing, and exit interview information. These charts will be a random sampling of each member of the team. This audit is to be conducted by a panel review team, which will include the centers manager, the center's medical director, and rotating members of the billing and sleep staff team members.

Public Relations

For SMN, the public consist of patients with sleep disorders and the various clinicians that encounter those patients. It is with those two separate publics that strong relationships must be established and maintained in order to build and grow the center. The following strategy will attempt to maintain the public relation goals of SMN.

The first step includes a series of print ads in local newspapers. Those ads will be brief, but include visuals of the center, pictures or real patients, and information relating to the importance of screening sleep disorders. The campaign will also focus on the facility, its state of art testing equipment, portable convenient diagnostic options, varied treatment options and in-network provider of diagnostics and treatments.

Secondly, a radio campaign will be prepared. This will include a series of 60 second ads. These ads will portray individuals with sleep disorders, their pretreatment symptoms and their post-treatment satisfaction. Additional ads, which are purely information and contain real statistics regarding the effects of daytime sleepiness and treatment options, will be available at the radio center as additional references.

To further penetrate the physicians market, the center will develop a 15- minute informational video, which will be provided to all physicians within the market area who treat or potentially treat sleep disorder patients. It will focus on the facility, various treatment programs,

referral programs, research information and follow-up care and support groups for patients. The video will accompany support documents that provide the details for referring and contacting the center.

Lastly, the public relations department will develop a website which will address both physicians and patient's specific needs. It will also serve as a site for patients to obtain information regarding sleep disorders and treatment options. A survey will be on site for patients to complete, screening them for several possible sleep disorders.

Sleep Management Services

Creation of operating sleep/DME center
Scheduling
Referral and preauthorization tracking
Submission and review of fee tracking
Review and tracking of sleep orders
Maintenance of sleep medical records
Clinical sleep testing
Clinical sleep scoring
Preliminary Reports
Transcription
Provider's dictation and interpretations
Medical Director – FNP/Dental Director, ABDSM
DME set- ups, follow ups, sales
DME support clinic- no charge
Clinical sleep staff
Quality assurance
Provider education and assistance
Sleep management and strategic planning.

Compensation Guidelines
12-month agreement with automatic renewal
90 days notice for withdrawal, plus 90 days worth of management fees as a buy out.
Initiation/ training fee
Anticipated labor costs as agreed per budget, paid one month in advance. Management fees equal to 15% of TC and private pay
Primary Start-Up Needs (minimum per center): 3-Bed
Provider within target state eligible to read studies
1.2 daytime PSG lead
1.0 overnight PSG lead
Carrier contracts
Billing/Registration/Transcription
Marketing/Supervisor

Budget

The following budget proposal outlines the financial expectations for a three diagnostic bed facility, including 4 portable testing units, these revenues assume 12 months at 75% fill rate. The budget proposal below overestimates costs and provides conservative productions volumes in order to produce a conservative financial Performa. The Performa includes a column for year one (running lower production) as well as the expected revenues for three-bed sleep center running at 80% fill rate (Dollinger, 1999). This business plan does not discuss the income associated with patient encounters for the clinical staff following up on patient care.

Sleep Financial Performa:	**3-beds: prorated 12 month Projected:**	**3-beds Full 12 months: Projected**
# of studies attended # of studies unattended	598 100	832 200
Average charge: attended Average charge unattended	2200 **(1,315,600)** 700**(70,00)**	2200 **(1,830,400)** 700 **(1,40,00)**
# of DME rentals: # of DME purchases: # of DME supplies	418 **(62,700)** 350 **(472,500)** 418 **(188,100)**	600 **(90,000)** 500 **(675,000)** 600 **(270,000)**

Average charge: rentals Average charge: purchase Average charge: supplies:	150 1350 450	150 1350 450
Total Gross Charges:	**2'108,900**	**3,005,400**
Reimbursement rate:	65.00%	65.00%
Net Revenue:	**1,370,785**	**1,953,510**

Expense: Employees		
Salary:	162,800	244,960
Payroll Taxes	13,838	20,821
Flex Credit (Benefits)	12,500	15,000
Expense: Svcs & General	**189,138**	**(280,781)**
Phone: Janitorial:	6,000 5,000	8,000 7,000
Laundry: Equipment Lease:	1,500 24,000	2,000 30,000
Transcription: Patient Education:	15,00 36,00	20,000 40,000
Building rent: Medical supplies/Forms:	78,000 20,00	78,00 35,00
Supplies: DME Depreciation: Marketing:	209,000 20,000 80,000	270,00 27,000 80,000
Total Operating Expense:	**683,638**	**877,781**
Net Income before Provider Expense:	707,147	1.095,729
Total Net Income Profit Margin (Net income/ net revenue)	**707,147** 51.58%	**1,095,729** 56.09%

HCPCS - Description
E0601 - CPAP machine purchase E0561 - CPAP Passover Humidifier E0562 - CPAP Heated Humidifier E0470 - BiPAP purchase E0471 - BiPAP-ST purchase A7034 - CPAP nasal mask A7032 - CPAP nasal mask cushion A7030 - CPAP Full Face mask

A7031 - CPAP Full Face mask cushion A7044 - CPAP Full Oral Interface A7046 - CPAP Humidifier Chamber A4604 - CPAP tubing, heated breathing tube A7037 - CPAP tubing, long and short hoses A7038 - CPAP disposable filter A7039 - CPAP foam filter A7035 - CPAP headgear A7033 - CPAP nasal pillows A7036 - CPAP chinstrap E1399 - CPAP Miscellaneous

CPT codes
99205 - Consultation with sleep specialist
99214 - Patient follow up by sleep specialist, comprehensive complexity
99213 - Patient follow up by sleep specialist, medium complexity
95810 - Diagnostic polysomnogram
95806 - Portable diagnostics
E0486 - Mandibular advancement orthotic

Indications for testing and consultation
327.23 Obstructive sleep apnea
327.25 Sleep related hypoventilation/hypoxemia
327.21 Primary central sleep apnea
347.0 Narcolepsy
327.14Hypersomnia due to medical condition (OSA)
491.21 COPD
333.99 Restless legs syndrome
327.51 Periodic limb movement disorder
327.33 Irregular sleep wake type
327.42 REM sleep behavior disorder
250.0 Diabetes
401.9 Hypertension

Conclusion

The sleep disorders business in its peak commonly experienced up to thirty percent annual growth (Reporter Link, 2009). The growth of the industry has declined to about five percent over the last several years. Despite the challenges, the sleep industry has a bright future

ahead. People and medical experts are more aware of issues surrounding sleep than ever before (Reporter Link, 2009). Obstructive sleep apnea (OSA) a potentially life-threatening condition characterized by episodes of breathing cessation during sleep alternating with snoring receives regular news coverage alongside other conditions such as diabetes (Campos-Rodriguez, F. et al., 2005). Regardless of how the economy turns, the second half of the year tends to be the time patients seek diagnostic testing such as sleep-testing. Patients often schedule sleep studies once they've met their health plan's deductible. Income taxation, an item that may be subtracted from gross income or adjusted gross income in determining taxable income (e.g., interest expenses, charitable contributions, certain taxes) or co-insurance requirement.

There are many strategies sleep labs can use to navigate uncertain financial times. Far more than simply staying lean and mean now is the time to fine-tune and be ready to seize opportunity from recession. Thriving in today's sleep industry takes more than simply setting up a lab with equipment. With competition growing, managers need to become savvy marketers, too. Moving toward portable testing can reduce per study production cost considerably. The in-lab test costs per study can range 150 to 250 dollars per study. If a lab averages 12 to 14 patients per month, the total cost per study for rented equipment over the life of the equipment would average $34.50. The comparable cost per study in portable testing could be $20-55 test (Reporter Link, 2009).

Another essential for a start-up is a third-party payer analysis to make sure you can participate in third-party payer sleep service contracts and that they contract for reimbursements that will allow you to be profitable (Harper, 1991). The goals for sleep diagnostics in this current health care market are to perform equivalent quality sleep testing and treatments at a cheaper price. That price includes delivering not only efficacious treatments, but effective treatments that

are actually used by patients to improve disease management outcomes. Currently sleep diagnostic is an expensive cost that is seeing less referral by a PCP demographic that annoyed due to high cost of testing. To engage this demographic actively educating and providing more affordable portable testing is crucial. Educating and offering alternative treatments to CPAP such as, weight loss management, sleep hygiene, behavioral sleep medicine services for insomnia management, dental sleep medicine, surgical treatments for OSA and complex CPAP compliance management. This business plan meets many of these identified challenges to maintaining the needed growth of sleep services and treatments to meet the future demand.

Implications for Practice

Adding non-physician health care providers to a busy sleep medicine practice can significantly benefit aspects of patient care; sleep histories, medical examinations, CPAP compliance enhancement, mandibular advancement therapy and assisting with behavioral medicine interventions. Advanced practice nurses also can be beneficial in the areas of medication management and supervision of sleep diagnostic staff. There is already ample evidence of the value of physician assistants and nurse practitioners in primary care, specialty medicine practices, emergency departments, and working with hospitalists. Physician assistants and nurse practitioners improve service by reducing waiting times until appointments are available, managing follow-ups, addressing needs for medication renewals, and helping with patient callbacks of test results. They also are often able to spend more time with patients than physician's schedules may allow, affording patients the opportunity to have more time with a health care practitioner. With the explosive realization of the prevalence of sleep disorders, its public health implications, and the benefits of good sleep, the demand for sleep medical services is growing exponentially. This kind of innovative testing and treatment facility can help meet the

growing need for sleep specialty care.

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Case Study: Trigeminal Neuralgia

Improved with NTI – tss Intra-Oral Appliance

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Case Study: Trigeminal Neuralgia

Improved with NTI – tss Intra-Oral Appliance

Trigeminal neuralgia (TN) is a pain syndrome that results in a unilateral facial pain. A patient experiencing TN may have significant pain from mild stimulation of the face, such as brushing teeth, chewing food, or movement of the mandible (Zakrzewska, 2004). TN affects women more often than men (2:3), and is more likely to occur in people who are middle-aged (40-50 years of age). TN occurs in approximately 14,000 people each year in the United States (Cheshire, 2005). The nature of pain in TN is stabbing or an electric shock-like sensation and often can be severe. Pain is brief, a few seconds to a few minutes, and paroxysmal. Pain may occur several times a day or during irritation of the area affected (Cheshire, 2005).

TN is recognizable by patient history alone. The presentation can vary regarding the distribution of the pain. The pain often follows the unilateral sensory distribution of trigeminal nerve (V), typically radiating to the maxillary or mandibular area (Cruccu, et al, 2008) (Milanov & Bogdanova, 2003). Physical examination findings are typically normal, although mild light touch or pin perception loss has been described. Significant sensory loss suggests that the pain syndrome is secondary to another process thus would require neuro-imaging (Zakrzewska, 2004). To confirm the diagnosis, other conditions should be investigated. Other conditions that may result in TN-type symptoms include tumors, aneurysms or multiple sclerosis (Nobrega, 2007). A focused dental examination may also reveal whether a problem with the teeth or gums is the cause of TN pain (Mauskop, 2001) (ADA, 2006).

The goal of therapy is to reduce pain for the patient. Medical management largely relies on anticonvulsant medications (carbamazepine, neurontin, oxcarbazepine) (Silberstein, 2006). Additional agents that may benefit the patient may include muscle relaxants (baclofen and

klonipin) (Malick & Burstein, 2000). Surgical management may be indicated to reduce causes of compression of the trigeminal nerve such as mass lesions, large vessels or vascular malformations. Procedures that have been tested and recommended per TN guidelines include microvascular decompression, stereotactic radiosurgery, nerve blocks and ablative procedures such as electrocoagulation (Cruccu, et al, 2008).

Complementary treatments would include chiropractic adjustment, acupuncture, and intra-oral appliances (Silberstein, 2006). Dentists and neurologists familiar with hyperactive masticatory function will include this treatment option into their armamentarium (Lavigne, et al., 2001) (Lamey, Steele & Aitchison, 1996). Nocturnal and wake masticatory function may negatively impact trigeminal nerve through increased cervical and mandibular activity, such as nocturnal and awake bruxism and clenching, may trigger headaches (Bender, 2007).

The nociceptive trigeminal inhibition – tension suppression system (NTI) intra- oral appliance is cleared by the Food and Drug Administration (FDA) since 2001 for the prevention of migraine headaches, associated tension-type headaches, and prevention of bruxism and TMJ injury (Wright & Jundt, 2006). An example of the (NTI) is seen in figure 1. Displacing clenching forces to the incisors reduces the impressive forces that can be placed on molar dentition by the masticatory muscle system that can be seen on Figure 2. Advocates of intra-oral appliances for the treatment of TN believe controlling clenching and grinding of the teeth may decrease the possibility of noxious stimulation of the trigeminal nerve system (Blumenfeld, 2007). An example of the theoretical model is depicted in figure 3.

Case Presentation

V. A. a 51 year-old East Indian female with a history of obesity, hypertension, and diabetes mellitus type two, presented to our office for complaint of facial and dental pain. Her

recent medical history was significant for a recent outpatient surgery. Since her surgical recovery she had a gradually worsening facial pain felt in her left ear and lower mandible attributed to be dental pain. She was referred to our clinic after initial standard treatments were recommended by her primary care provider and her general dentist of record. Our sleep clinic is an inter-professional sleep disorders clinic consisting of a Family Nurse Practitioner, sleep technicians and a dentist, board-certified in dental sleep medicine. The practice has experience with oral appliance treatments for obstructive sleep apnea and snoring (OSA), facial pain syndromes, headache syndromes, nocturnal bruxism and complex sleep disorders.

The history of the present illness notes that the patient underwent elective cataract surgery two months prior to presenting to our office. The intervention was performed and achieved aims and goals of the procedure. The patient admitted to a high level of emotional stress leading up to the surgery. The patient recovered without incident. The patient realized during the first week of recovery that she would awaken with occasional recurrent frontal headaches. She also noted new onset dental pain along tooth 13 and 14 during chewing and felt the pain radiated to both the left temporal region and the ramus along the left mandible. Cold sensitivity was noted as well. The patient sought care with her dentist of record who found no acute issues with her dental status.

She tried to manage the pain with ibuprofen sparingly taken over a few weeks. The patient's pain persisted and continued to radiate to her temporal region and left ear. The pain was reported as sharp, lancinating pain in the inner ear also extending to the left scalene and trapezius region of the neck. Associated with the pain was a feeling of fullness in the ear. The primary care provider (PCP) initially treated this as an inner ear infection with an empirical trial of oral antibiotics. The patient finished the course without resolution of symptoms. During the treatment

course she reported to her PCP noting the symptoms were not improving. The PCP re-inspected the inner ear with an impression of no current otitis media. The patient discontinued antibiotics and was recommended to take hydrocone with acetaminophen as needed for pain.

Pain was initially local pain with chewing, jaw movements, light touch in the form of lying on the face, brushing lightly against the left facial region and then progressed to referred lancinating pains along the left facial region in both a cephalic pattern and inferiorly to the neck. The patient reported constant throbbing with episodes of severe short burst of lancinating pain the patient could not attribute to any antecedents or causes. The severe bursts of pain were progressive increasing in frequency over the five weeks leading to her referral to our practice. The patient reported one to two that had progressed to ten or more a day up to the day of our consultation. The patient was not taking the opiate pain medication due to the nausea and continued pain experienced when she attempted the treatment regimen.

The patient sought another consult with her general dentist that resulted in a prescription of Valium 5 mg tablets, without improvement and referred to Walgreens for an over the counter night guard for bruxing an example seen on figure 4. The patient attempted the device but the size of the device caused worsening of pain due to pressure of the buccal surface of the cheek. Patient reported ringing in ears, light sleep and moderate snoring. Patient reported averaging 5 hours per day of sleep which she has reported as adequate for her. Patient reported more pain in am and cold sensitivity to left teeth.

The patient's past medical history obesity, diabetes mellitus type two, and long-time snoring. A noted family history of diabetes mellitus type two, snoring and heart disease on the patient's paternal side of the family. She is a homemaker, has two adult children and is happily married. Her creed is Hindu, and reports being a nonsmoker, she is abstinent of alcohol, and

denies nicotine use or recreational drug use. She is inactive with rare physical activity and does not have a standard dietary plan. Her weight status has been relatively unchanged for many years. She has no drug allergies and takes metformin 1000 milligrams a day and glyburide 5 milligrams a day.

Review of systems was as follows: Constitutional: Denies night sweats sensitivity to heat or cold; Eyes: Denies vision changes; Ears: Reports tinnitus; Nose: Denies sinus infections; Mouth: Denies sore gums or sore tongue; Throat: Denies soreness or hoarseness; Cardiovascular: Denies high blood pressure, angina, swelling in ankles or palpitations; Respiratory: Denies cough-persistent, yellow or green sputum.

Physical examination by body systems are as follows: Most recent vital signs: Height, 64 inches; Weight 170 pounds; Body Mass Index 29; Blood Pressure, 132/82; Heart Rate 64 beats per minute with regular rate and rhythm. General: Patient is well developed in no acute distress. Patient appears younger than stated age. Eye: Color: Brown. Patient pupils are equal, round and reactive to light. Patient sclera are non-icteric. Ear: Patient external ears, external auditory canals are normal. Throat: Tongue is large with scalloped borders. Oral exam showed a narrowed anterior-posterior diameter of her posterior oropharynx space due to small diameter lumen, narrowed pharyngeal width, and enlarged tongue base. Small tonsillar tissue noted in orophaynx. Posterior airspace is easily visualized upon inspection.

Tongue bulk rises above plane of dental occlusion. Soft palate is not enlarged with pink healthy mucosa. Mallampati 1 out of 4. Patient has narrowed palatal pillar width noted as a Samsoon –Young score of 3 out of 4. Tonsils are small remnants bilaterally. Uvula small and non-reddened. Average arch to hard palate and average skeletal arch form. Dentition has significant wear pattern in both upper and lower incisors. Patient has average condition molar

occlusal surface with moderate abfraction of the noted condition with a total of 4 crowns, two gold and two porcelain with silver fillings to two molars and noted extraction of teeth 1, 2, 16, 17, 18, 31 32. There is advanced mobility to upper central incisors graded at two to three on scale of four. Neck: Patient neck is supple, without adenopathy, thyromegaly or jugular venous distention. Neck circumference is 15 inches. Respiratory: Patient chest wall is symmetric with clear breath sounds to auscultation both to anterior and posterior.

Musculoskeletal: No muscles tender to palpation. Patient demonstrates abnormal temporomandibular joint (TMJ) function with mild click to left and right TMJ throughout all directions of range of motion. The patient has guarded pain response to palpation of the left TMJ. The patient can elicit pain with light touch to right hemisphere of the face. The foci are for maximum discomfort is 1 mm dorsal to the left TMJ. The patient has limited maximum opening due to pain in the lateral, opening and protrusive movements. The resting pain is a throbbing quality of 6 out of 10 on the visual analog pain scale. The pain is 10 out of 10 with direct palpation of limited compression. The patient has had limited chewing, due to pain related to mastication. The pain is both focal and radiates upward and downward.

Cardiovascular: Patient has1 plus pulses in the upper extremity without clubbing or cyanosis. Patient has1 plus pulses in feet without edema. Heart tones have regular rate and rhythm without ectopy. S1 and S1 sounds are clearly audible without click or pathologic S3 or S4 noted.

Neurological: Patient cranial nerves 3-12 are intact. Patient motor strength is symmetric and normal for age. Patient is oriented to person, place, and time. Skin: No rashes, warm dry and intact. Dental Findings: As noted above in musculoskeletal section. Limited range of motion of TMJ bilaterally noted on visual assessment. Maximum Opening 25 mm, Overjet 5 mm, Overbite 3 mm, Protrusive 8 mm +5/-3 Right Lateral Excursive 6 mm Left Lateral Excursive 4 mm.

Periodontal exam notes areas of pocketing of the gingival margin greater than 4 millimeters. Mandibular classification of class 2 occlusion.

Occlusal Splint Treatment

The initial consult day included intake and expanded dental examination with our dental director and Family Nurse Practitioner (FNP) with seven years of splint treatment experience. The patient symptoms appeared to be a complex of left joint derangement; this resulted in limited maximum opening of the mouth. The pain syndrome was consistent with TN. The patient's full coverage over-the-counter appliance was assessed and found to be lacking in any ability to inhibit occlusal function. A temporary NTI was fabricated out of polycaprolactone is seen on Figure 5. Ultrasound physical therapy modality with a standard treatment protocol was applied in office. The regimen consisted of 0.8 mV at 3 mHz delivered over 3 intervals of 10 minutes to each TM. The ultrasound is depicted in Figure 6. The end of visit pain score was reported to be 30 percent reduced from the splint placement and ultrasound treatment.

The second day of treatment was approximately one week after initial consultation. Patient presented for follow up on NTI treatment adherence and response to pain reduction. Patient reported wearing the device for most of the day and night averaging 15 hours. Patient reported that she stopped taking ibuprofen 4 days post initial consult due to improved pain severity and frequency. Patient reported the NTI being quite helpful with facial pain. Swelling in left lateral ptyregoid muscles were reduced significantly. No changes in bite, no changes in medical history were noted at this assessment. The patient had a repeat of the ultrasound therapy at this visit as well.

The third day of treatment, three weeks following the second appointment, the patient reported to the office for a more permanent and lower profile maxillary full-arch NTI appliance

figure 7). At this visit the patient reported the pain to left side had resolved completely. Overall, the patient was deemed to be responding well to treatment.

The fourth day of treatment, 6 weeks after her initial intake to our practice the patient no longer needs to use Advil even as needed. The patient reported mild bite change. The patient transitioned to wearing the initial NTI appliance at night, all night. The maxillary device was still being worn during the day most days. The patient reported meeting treatment goals with total resolution of TN pain syndrome. The patient eventually transitioned to wearing the NTI simply at night with long term follow up on treatment response.

Conclusion

Modern western medicine has thoroughly investigated the pharmacotherapeutic modalities for treatment of medical disorders. Clinicians report good outcomes with these regimens. The positive outcomes from pharmacotherapeutics can be lost if the possible risk of systemic side effects due occur. In another subset portion of patients offered pharmacotherapeutics this group can lack efficacious results (Silberstein, 2006). The current preventative pharmacotherapies modulate the trigeminovascular system indirectly attempting to modify events that lead to trigeminovascular nociception (Malick & Burstein, 2000).

The alternative medical referral for TN is generally for surgical consultation. The surgical options include peripheral nerve blocks or ablation, gasserian ganglion and retrogasserian ablative procedures, microsvascular decompression and stereotactic radiosurgery (Crucca, 2008). These are effective treatments, but also have the possibility for complications such as infection and further injury.

The TMJ and the apparatus of the muscles of mastication can contribute significantly to common complaints of headache, facial pain and the less common TN (Cascone, Fatone,

Paparo, Arangio, & Lannetti, 2010) (Baad-Hansen, Jadidi, Castrillon, et al., 2007). This region of the body needs better inter-professional understanding of how it affects medical outcomes. The general understanding of this area of the body is quite limited in the generalist medical clinician knowledge set.

There is a possibility for great strides in pain management that responds to adjunctive treatment planning. Increased access to basic identification and screening techniques of parafunctional etiologies for the medical practitioner would be beneficial for patients with pain (Blumenfeld, 2007). The skills regarding these techniques to better treat facial pain syndromes could result in an increase in the percentage of treated patients presenting with pain syndromes to the primary care practice. The primary care clinician could be a valuable member of the pain management team if made to be familiar with modalities often used in TMD treatments.

Intraoral appliance use is a reversible and non-invasive treatment modality that has shown to be effective in treating patients with facial pain syndromes (Dupont, 2003). The primary care clinician has been integrating acupuncture, massage, chiropractic and physical therapy adjunctive treatments more commonly over the past several years. Understanding when to include the option of the NTI and other occlusal splints would benefit many patients not currently offered this treatment (Cheshire, 2005). Diagnosis of head pain syndromes is difficult because sensory fibers of the cervical spine as well as branches of the facial nerve, glossopharyngeal nerve, and vagus nerve are intimately placed near the fibers of the trigeminal spinal tract nucleus (Forssell, Tenovuo, Silvoniemi, & Jaaskelainen, 2007) (Milanov & Bogdanova 2003). Ultimately, since the likelihood of a clinician being exposed to facial pain syndromes is common, becoming familiar with the most common differential diagnosis improves the possibility learning the definitive diagnosis.

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Appendix

Figure 1. Nociceptive Trigeminal Inhibition splint example. Example from www.nti-tss.com.

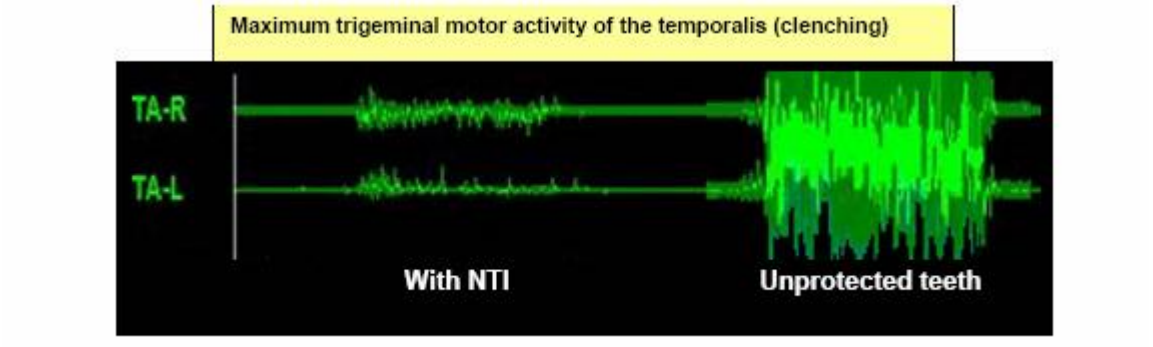

Figure 2. Example of EMG activity comparing the occlusal forces of molar contact and anterior dental contact alone. Example from www.nti-tss.com

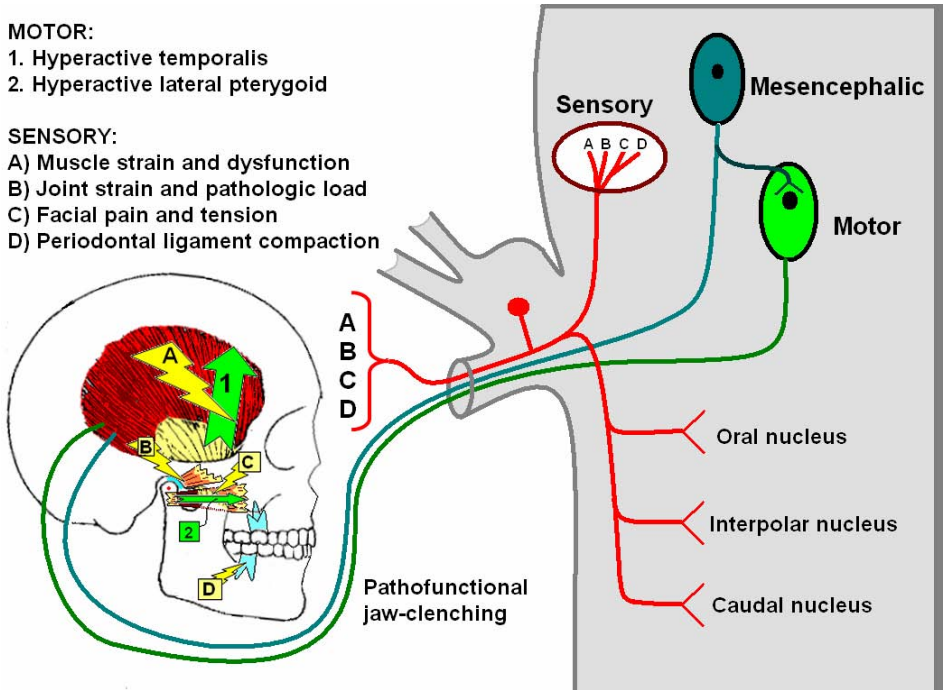

Figure 3. Theoretical framework for the effect from inhibiting masticatory function. Example from www.nti-tss.com

Figure 4. Over-the-counter night guard. Example from www.walgreens.com

Figure 5. Example of initial splint placed on consultation visit. Example from www.nti-tss.com.

Figure 6. Example of the ultrasound modality. Example from www.spineuniverse.com

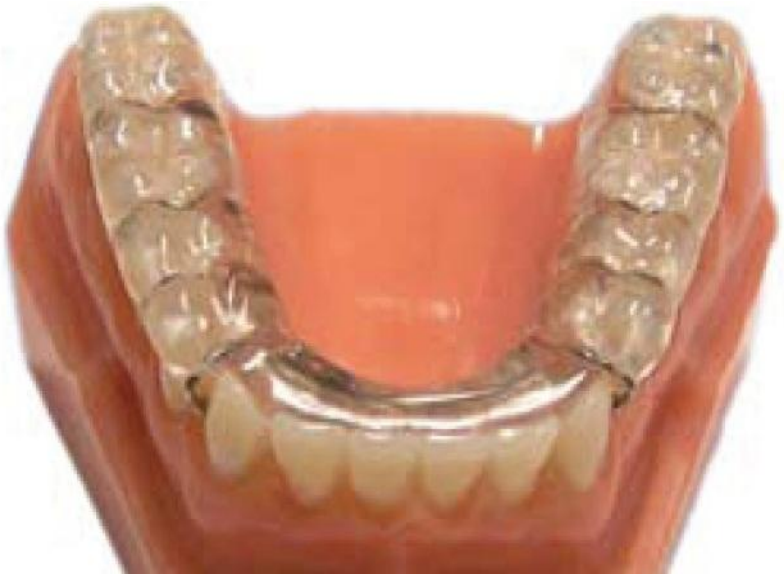

Figure 7. Example of long term daytime/night-time splint used by the patient.

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N790: Clinical Residency

Case Study: Efficacy of an Oral Appliance in the Treatment of Persistent Obstructive Sleep Apnea After Uvulopalatopharyngoplasty

Case Presentation

T. S. is a 47 year-old Caucasian male with a history of snoring, severe obstructive sleep apnea, poorly controlled complex headache syndrome refractory to conventional medical therapy, hypertension and excessive daytime sleepiness. He was seen in sleep clinic for consultation for difficult to treat obstructive sleep apnea (OSA). Our sleep clinic is an inter-professional sleep disorders clinic consisting of a Family Nurse Practitioner, sleep technicians and a dentist, board-certified in dental sleep medicine. The practice has a niche in sleep medicine by focusing on home sleep diagnostics, therapeutic assessments, PAP treatment and oral appliance treatments for OSA and complex sleep disorders.

The sleep disorder treatment history for T.S. started with screening from his primary care provider (PCP) with referral to a board-certified sleep specialist. He had an initial laboratory-based polysomnogram prior to 2008 at an unspecified sleep disorder center that prompted his first treatment attempt of his OSA. T.S. presented to our office approximately a year after an attempt to treat his severe OSA with the uvulopalatopharyngoplasty (UPPP). Figure 1 and figure 2 demonstrate a schematic of the UPPP and the status post surgery appearance of the patient's airway. The most recent treatment assessment polysomnogram performed by Dr. Joshua Ramseyer in 2008 (NPSG) revealed residual severe OSA and significant oxygen desaturations.

Obstructive Sleep Apnea History

The patient's diagnostic polysomnographic study revealed severe persistent snoring recorded, normal sleep efficiency at 94 percent (normal >85 percent), stage two sleep at 81 percent (normal 55-65 percent), stage 3 and 4 NREM delta sleep at 13 percent (normal > 10

percent) and no rapid eye movement (REM) sleep (REM, normal 20-25 percent). Apnea-hypopnea index of 96 events per hour (ASDA, 1995). His minimum oxygenation was measured at 74 percent during sleep. These results, in light of his symptoms are consistent with severe OSA. An excerpt from the polysomnogram report can be found in Figure 3.

His treatment history relating to OSA started with a continuous positive airway pressure treatment (CPAP) titration study to determine an optimal treatment pressure to effectively manage the OSA. The "gold standard" treatment for adults with OSA is positive airway pressure (PAP). Although extremely effective, tolerance and compliance remain considerable hurdles to successful PAP treatment. Certain interventions may promote compliance to PAP, such as correction of nasal obstruction, attention to mask-fit, desensitization for claustrophobia, heated humidification, patient education, regular follow-ups, compliance software, and support groups. Despite these measures, CPAP therapy remains a considerable challenge for many individuals, and surgical intervention is often an effective therapeutic alternative (Won, C. L., Kasey, K. L., & Guilleminault, C., 2008).

His initial CPAP attempt resulted in complete intolerance due to claustrophobia. CPAP is considered the "gold standard" therapy for OSA, yet CPAP users achieve only approximately 50% of ideal use. Therefore, today's definition of surgical success is achieving a greater than 50% reduction of the AHI and an AHI of less than 20 events per hour. This is a standard definition to monitor surgical success rate (Won, C. L., Kasey, K. L., & Guilleminault, C., 2008). Uvulopalatopharyngoplasty (UPPP) aims to enlarge the retropalatal airway by trimming and reorienting the posterior and anterior lateral pharyngeal pillars, and by excising the uvula and posterior portion of the palate. This surgery is often performed in conjunction with adenotonsillectomy. UPPP has a reported success rate of approximately 40–50% for improving

mild to moderate OSA, although surgical efficacy appears to decrease over time (Walker-Engstrom, 2002). UPPP may be associated with significant complications, including velopharyngeal insufficiency, dysphagia, persistent dryness, and nasopharyngeal stenosis time (Walker-Engstrom, 2002).

T. S. sought consultation with Dr. Lippman, an otolaryngologist with experience in surgical treatments for OSA. Surgery aims to reduce anatomical upper airway obstruction in the nose, oropharynx, and hypopharynx. Successful surgery depends on proper patient selection, proper procedure selection, and experience of the surgeon. Most surgeries are done in combination and in a multistep manner, with maxillomandibular advancement typically being reserved for refractory or severe OSA, or for those with obvious and significant maxillomandibular deficiency (Won, C. L., Kasey, K. L., & Guilleminault, C., 2008). The patient chose to undergo tonsillectomy and adenoidectomy, as well as, UPPP.

The American academy of sleep medicine has found that mandibular advancement splints are an acceptable treatment option for patients with severe OSA when they cannot tolerate CPAP. The function of the device is to hold forward the mandible during sleep to alleviate upper airway collapse in improve OSA (Chan, Lee, & Cistulli, 2007). This patient was not presented with mandibular advancement splint therapy as a treatment option prior to his decision to undergo surgical intervention. The patient has verbalized that if the MAD was offered as an initial non-invasive option, he who would have been interested in the management plan prior to undergoing OSA surgery. His long-term side effects from the surgery included persistent snore and excessive daytime sleepiness, significant xerostomia, and velopharyngeal incompetence and post-surgical pain continued longer than six months postoperatively.

The CPAP titration that was performed after the patient's UPPP surgery during a split-night protocol (Khawaja, Olson, van der Walt, Bukartyk, Somers, Dierkhising, et al., 2010). To improve patient comfort and tolerability of diagnostic testing of OSA, sleep laboratories will often follow a set guideline to initiate CPAP therapy during the latter half of a diagnostic PSG. The patient must demonstrate a minimum of 15 per hour apnea hypopnea index (AHI, normal < 5/hr) during a prescribed period of continuous sleep to meet this criterion (Khawaja, Olson, van der Walt, Bukartyk, Somers, Dierkhising, et al., 2010). This patient far exceeded minimal criteria to have CPAP initiated. The titration of CPAP resulted in a prescription for 7 centimeters of water pressure (cmH2O). His AHI was treated to less than 6/hr.

The patient was not able tolerate CPAP well at home after his second titration study despite is documented physiologic improvement during the study. After the CPAP failure for the second time the patient was resigned to non-treatment of his OSA. The patient was referred to our practice after consultation with his primary care provider (PCP) for treatment alternatives to CPAP. The literature does not support treatment of severe OSA with phase I surgical interventions (Barthel, & Strome, 1999). Surgical treatment recommendation for a patient with severe OSA is to undergo go phase I and II surgery. Figure 4 and 5 demonstrates examples of phase II surgeries. The phase I surgical intervention is uvulopalatopharygoplasty (UPPP) (Epstein, Kristo, Strollo, Friedman, Malhotra, Patil, et al., 2009). UPPP only has a 40-50 percent chance of reducing the AHI by 50 percent. Thus many patients will continue to have an AHI> 20 events per hour after surgery.

In this patient's case, there was a worsening of OSA after phase I surgery. The patient was unwilling to undergo further surgery after experiencing excessive side effects and no efficacy with phase I surgery for OSA. Phase II surgery may include genioglossus advancement and

hyoid mytomy and suspension. Tracheostomy is an effective treatment options for OSA but is largely not considered an acceptable options by patients (George, Barber, & Smith, 2007). Figure 2, 3 and 4 demonstrate the phase I and II surgery options.

Past Medical History

The patient's past medical history includes obesity, hypertension, hyperlipidemia, long-time snoring, excessive sleepiness and severe obstructive sleep apnea (OSA). He has family history of snoring noted by his father; his brother also has severe OSA. He owns his own excavation company and is happily married. He denies any specific creed, and reports being a nonsmoker, he drinks an average of two whiskeys to get to sleep and moderate social drinking on weekends, and denies nicotine use or recreational drug use. He is active and tries to aerobically exercise three times a week and does not have a standard dietary plan. His weight status has been relatively unchanged for many years. He has no drug allergies and takes 50 mg of Cozaar and 10 mg of Norvasc for hypertension. He takes 40 mg of Simvastatin for hypercholesterolemia. Prior to current treatment with MAD the patient relied on two shots of hard alcohol and 10 mg of Zolpidem for early awakening insomnia.

Physical examination showed a narrowed anterior-posterior diameter of her posterior oropharynx space due to enlarged tongue base. There was noted post-surgical findings of the soft palate that appeared reduced along with non visualized tonsils on inspection. Patient has evident scarring to the inferior border of the soft palate and surgical removal of the uvula. Posterior airspace is easily visualized upon inspection.

Physical examination by body systems are as follows: Most recent vital signs: Height, 69 inches; Weight reduced from initial intake of 192 down to current weight of 162 pounds; Body Mass Index, initial intake of 28 and currently down to 25; Blood Pressure, 128/78; Heart Rate

68 beats per minute with regular rate and rhythm. General: Patient is well developed in no acute distress. Patient appears younger than stated age. Eye: Color: Blue. Patient pupils are equal, round and reactive to light. Patient sclera are non-icteric. Ear: Patient external ears, external auditory canals are normal. Throat: Tongue is large with scalloped borders. Tongue bulk rises above plane of dental occlusion. Soft palate is status post UPPP reduced to a grade of Mallampati 2 out of 4. Patient has average palatal pillars width noted as a Samsoon –Young score of 2 out of 4. Tonsils are surgically absent. Uvula N/A - surgery. High-arched palate with narrowed skeletal arch form and signs surgical excision of soft tissue. Dentition is in good condition with two silver fillings to two molars and noted extraction of teeth 1, 16, 17, 32. Neck: Patient neck is supple, without adenopathy, thyromegaly or jugular venous distention. Neck circumference is 17 inches. Respiratory: Patient chest wall is symmetric with clear breath sounds to auscultation both to anterior and posterior. Musculoskeletal: No muscles tender to palpation. Patient demonstrates normal temperomandibular (TMJ) function with mild click to left TMJ.

Cardiovascular: Patient has1 plus pulses in the upper extremity without clubbing or cyanosis. Patient has1 plus pulses in feet without edema. Heart tones have regular rate and rhythm without ectopy. S1 and S1 sounds are clearly audible without click or pathologic S3 or S4 noted. Neurological: Patient cranial nerves 3-12 are intact. Patient motor strength is symmetric and normal for age. Patient is oriented to person, place, and time. Skin: No rashes, warm dry and intact. Dental Findings: Normal range of motion of TMJ bilaterally to stethoscopic assessment. Maximum Opening 44 mm, Overjet 3 mm, Overbite 3 mm, Protrusive 13 mm +8/-5 Right Lateral Excursive 12 mm Left Lateral Excursive 12 mm. Periodontal exam notes

missing teeth of 1, 16, 17, and 32. Mandibular classification of class I occlusion. Periodontal status I without pocketing or gingivitis.

CPAP was attempted was not tolerated, the patient was not open to more upper airway surgical intervention noting his previous experience, the MAD was considered the patient's last conservative option for treatment. Patients with less severe obstructive sleep apnea (OSA) are usually prescribed oral appliances. A recent study tried to define predictive criteria for optimal response to MAD in patients who have failed CPAP. The study conclusion was that patients with CPAP treatment pressure less than 10.5 cmH2O were more likely to respond to MAD (Tsuiki, et al., 2010). It may be logical to apply this same predictive criteria may possibly apply to patients who suffer from more severe OSA, but respond to lower CPAP. Noting the patient's treatment history the patient was deemed appropriate for a mandibular advancement device for his severe OSA.

Prior to initiating treatment the patient underwent a more current home sleep diagnostic assessment of the patient's OSA severity. The device used to assess oral appliance treatment response was a level III home sleep diagnostic device, WatchPAT 100 (WP100) (Chesson, Berry & Pack, 2003). This study demonstrated similar findings to the patient's PSG assessment. Figure 6 represents the WatchPAT100 report. T.S. was started at a 50 percent measurement of his total protrusive range of motion of the mandible. The treatment response to MAD for severe OSA is unpredictable. There are many FDA approved devices for the treatment of OSA and snoring (Kushida, Morgenthaler, Littner, Alessi, Bailey, Coleman, et al., 2006). An example of the MAD used for T. S. can be found in Figure 7. For this patient a device that had a good clinical history of treatment in severe OSA was necessary, thus the TAP T device was selected for treatment in this patient.

He proceeded over 6 weeks to incrementally advance the device a total of 7 mm. The terminal advancement resulted in resolved snoring, resolution of chronic morning headaches and reduced sleepiness to normal levels with an ESS falling from 15/24 to 9/24. He was noted to wear the device 7 nights of the week for 6 hours of sleep per night. He had no tooth or muscle discomfort with the device use. Goals of treatment that were achieved include improved hypersomnolence, resolution of chronic headache syndrome, snoring and insomnia syndrome. Patient also found he did not need Zolpidem or alcohol at bedtime to fall asleep or stay asleep. He performs daily morning jaw exercises to regain his baseline dental occlusion.

At week ten an objective assessment of the device treatment was performed noting a pAHI of 19, low oxygen of 89 percent and well-treated snore. This study reveals a 77 percent reduction of the OSA. The patient met many of his personal goals for improved sleep quality, duration, daytime somnolence and snore. The goal for complete response to the oral appliance is an pAHI of less than 10. The patient was counseled on weight loss, side sleep therapy and regular sleep hygiene measures.

The patient has undergone serial sleep tests to monitor long term treatment outcome of MAD (Figure 8). The patient experienced a failure of the advancing mechanism to the TAP T and subsequently was transitioned to a different oral appliance. The patient has been treated with the Sleep APP ^{TM} MAD (Figure 9).The patient felt his treatment response has continued to improve due to recent adjunctive weight loss related to dietary changes. The patient has now been monitored for two years; and has noted fifteen pounds of weight loss from initial evaluation. The patient continues to experience improved hypersomnolence with ESS less than 8/24. The patient has also been able to achieve optimal blood pressure treatment with current

medical management combined with successful OSA treatment compliance. Most recent level III testing reveals a pAHI of 2/hr and low oxygen saturation of 92 percent (Figure 10).

Case Analysis

Undiagnosed and untreated obstructive sleep apnea (OSA) causes health and safety risks. Recent estimates indicate that more than 3 million men and 1.5 million women have some form of OSA (Westbrook, 2005). Despite many patients presenting with clear symptoms of OSA, an estimated 80%-90% of OSA cases are undiagnosed (Chervin, Moyer, Palmisano, Avidan, Robinson, Garetz & Helman, 2003). According to a report from the National Sleep Foundation (Westbrook, 2005), individuals with undiagnosed or untreated OSA have up to a seven-fold increased risk of falling asleep while operating a motor vehicle. The American Academy of Sleep Medicine is the scientific body that recommends evidence-based treatment guidelines for the treatment of OSA (Ruehland, Rochford, O'Donoghue, Pierce, Singh & Thornton, 2009).

Nevertheless, the disappointing long-term compliance rates of 40 to 60% among PAP users have to be regarded as a major challenge warranting more aggressive exploration of both surgical and nonsurgical alternatives for OSA therapy. It is important to mention that a multidisciplinary approach involving a sleep specialist, otolaryngologist, maxillofacial surgeon, dental sleep medicine practitioner and primary care providers be better developed and implemented to improve OSA screening, diagnostic measures and appropriate treatment selection. Research is warranted in combination therapies that include mandibular advancement devices and phase I or II surgical interventions.

There is one case report of a patient who was undertreated with MMO and improved outcome with mandibular advancement appliance (Hoekema, de Vries & Stegenga, 2007). There was a 1998 small case series review of 24 patients treated with UPPP and Herbst MAD found in the literature. Eighteen of a sample of twenty-four patients had control of the OSA with the Herbst appliance with a fall in the AHI to < 10. There were, in addition, two partial responders as defined by an AHI of <20 and a >50% fall in AHI compared with baseline and post-UPPP values. All but one of the responders and partial responders had complete resolution of subjective symptoms of daytime sleepiness with the appliance. An adjustable oral appliance appears to be an effective mode of therapy to control OSA after an unsuccessful UPPP (Millman, Rosenberg, Carlisle, Kramer, Kahn, & Bonitati, 1998).

MAD is an effective treatment modality for the patient with mild to moderate OSA per these guidelines (Chan, Lee & Cistulli, 2007). Increasing referral patterns to dental sleep medicine for this patient population, before attempting CPAP in patients who prefer MAD, would reduce the number of CPAP failures (Chervin, Moyer, Palmisano, Avidan, Robinson, Garetz & Helman, 2003). This outcome would be a component of improved appropriate patient selection for any given OSA treatment (Bian, 2004). There continues to be a dearth of professionals who are well-trained in Dental Sleep Medicine.

An alternative delivery system could be to mimic the practice patterns currently being practiced with surgical treatments. The MAD was classified as a medical device in 1995 by the Food and Drug Administration. With the appropriate training, a medical provider may be qualified to provide medical treatments such as MAD. CPAP treatment addresses several areas of soft tissue collapse of the air. The most common airway obstruction (ignoring nasal level)

are mixed involving the soft palate and tongue base (43.5%), followed by soft palate alone (34%), and tongue base alone (22.5%). (Won, Li, & Guilleminault, 2008).

To approach a more reliable treatment alternative to CPAP, more collaboration between otolaryngologists and dental sleep medicine practitioners could be initiated. The aim would be to develop combination treatments of MAD and surgery that would more predictably manage all levels of OSA. A study in 2005 demonstrated the ability of an otolaryngologist to manage MAD for patients with OSA. The study reported prospectively treatment outcomes from forty-four patients with OSA who failed or refused CPAP or surgery for OSA. The patients were treated with a custom MAD, 68 percent of the enrolled OSA patients were noted to be cured or substantially improved (Maurer, Huber, Verse, Hormann, & Stuck, 2005).

Integration of this treatment modality into the sleep medicine clinic or the otolaryngologist office could improve access to both MAD; combination treatments of MAD with surgical treatments or CPAP to improve comfort and compliance of these therapies. These measures could offer more hope and choices to patients diagnosed with OSA. The sleep medicine team can more effectively trouble shoot common barriers to CPAP success through exploration of CPAP alternatives. Simultaneously, knowledge of alternatives to CPAP guides the sleep medicine team to offer more appropriate treatment selections for OSA patients. My practice has developed a version of this inter-professional sleep medicine practice. This model has improved our access to medical insurance coverage for MAD and combination CPAP treatments. The dissemination of this OSA treatment delivery system by a Doctor of Nursing Practice may have significant implications for nursing and medical practice.

Reflection on Practice

Sleep apnea is a medical problem with serious health consequences like cardiovascular disease, fatigue, and excessive hypersomnolence which can result in avoidable morbidity and mortality associated with motor vehicle accidents (He,Kryger, Zorick, Conway & Roth, 1988). Not only can OSA decrease quality of life, it can also worsen survival outcomes (He, Kryger, Zorick, Conway & Roth, 1988). The efforts to promote the use of evidence-based medicine to treat patients with OSA have been partially implemented. The lack of dissemination of the comprehensive panel of treatment alternatives for OSA, such as less invasive OSA surgery, MAD treatment, or combinations of the three accepted treatment modalities continue to block efforts to improve treatment outcomes. Sleep specialists, otolaryngologists, durable medical equipment companies and medical insurance carriers are the major stakeholders responsible for the current state of OSA treatment. To have this treatment model more rapidly developed, these stakeholders will need to be reached and convinced of the need for more effective delivery of OSA treatments.

Summary/Conclusion

This case study reveals a potential for enhancement to the current practice patterns by clinicians participating in OSA management. Many otolaryngology practices have recruited nurse practitioners and physician assistants to improve access to care. This naturally occurring trend of collaborative practice of the ANP and otolaryngology allows for a window of opportunity for the DNP to facilitate development of more complex treatment modalities for the challenging OSA patient.

The Essentials of Doctoral Education for Advanced Nursing Practice (AACN, 2004) recognized the direct care role for advanced practice nurses. The doctor of nursing practice (DNP) is described as a clinician with expanded responsibility and accountability in the care

and management of individuals and families and is also educated to identify and improve provision of care for a particular population at the aggregate and systems level. This case study demonstrates specifically two core competencies as defined by National Organization of Nurse Practitioner Faculties (NONPF) (Smolowitz & Honig, 2008). This specific competency states for the DNP to identify gaps in access and/or reimbursement that compromise patient's optimal care and apply current knowledge of the organization and financing of health care systems in order to ameliorate negative impact and/or reduce barriers to patient access.

The other competency for DNP is to establish and utilize a collaborative network of specialists while maintaining primary responsibility for patient care and accept referrals from other health professionals and agencies to provide optimum care (Smolowitz & Honig, 2008). By doing so, the DNP can begin development of a referral network between the primary care and the other clinicians participating in the sleep medicine team to improve the likelihood for treatment success in this patient population.

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Appendices

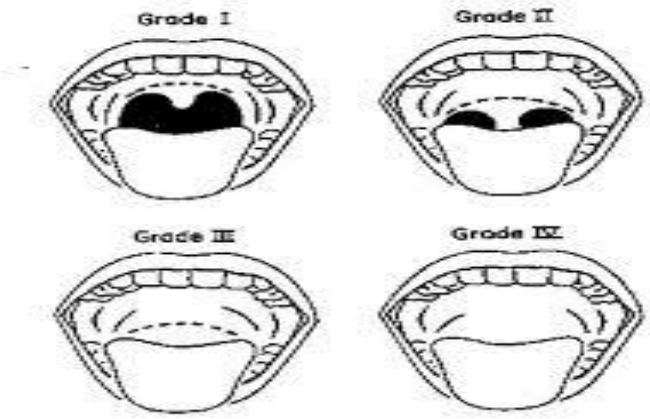


Figure 1. A depiction of the anatomical region to perform UPPP. Retrieved from www.medscape.com

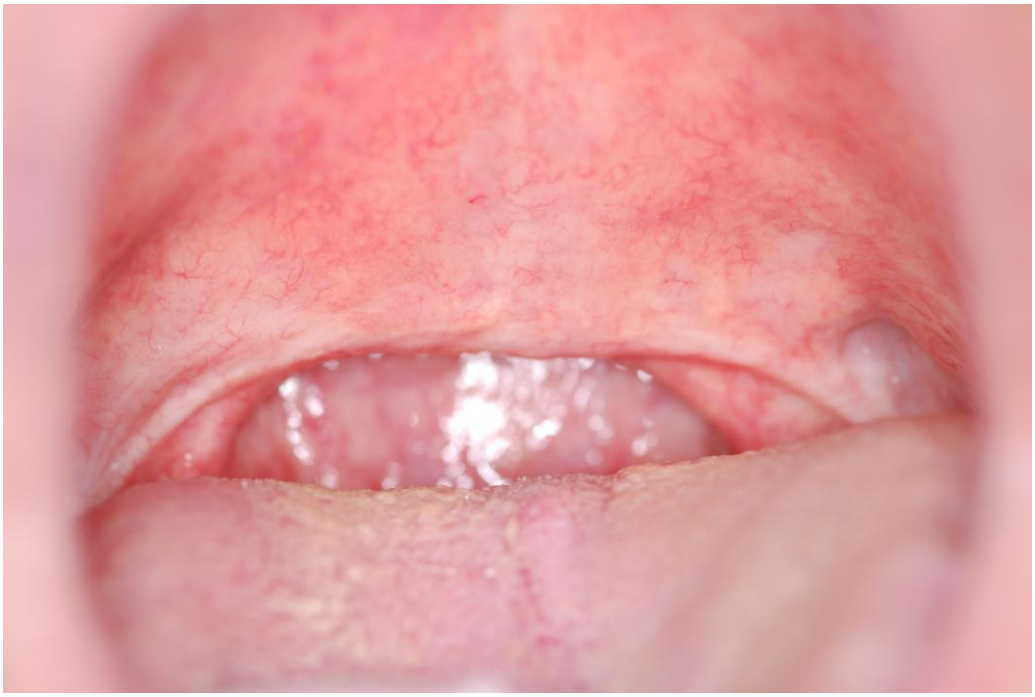


Figure 2. A depiction of the post-surgical appears of the patient's oral cavity. Retrieved from personal archive with permission.

ATTENDING PHYSICIAN: JOSHUA A RAMSEYER, MD
NAME OF SERVICE: POLYSOMNOGRAM AND CPAP TITRATION

HISTORY OF PRESENT ILLNESS
The patient is a 45-year-old male with a known history of obstructive slee
apnea. The patient has already had a uvulopalatopharyngoplasty which was
not successful in treating his daytime sleepiness or his snoring. The
purpose of this study was to re-establish a diagnosis of apnea and treat
him with CPAP.

PROCEDURE DETAILS
This study was started at 11:06 p.m., and the patient took 1.3 minutes to
fall asleep. He then slept for 126.5 minutes. On this baseline study, the
patient had a 94% sleep efficiency. He spent 81% of his time in stage 2 of
non-REM sleep, 13% in stage 3/4 non-REM sleep, and he did not obtain any
REM sleep.

The patient had extraordinarily severe obstructive sleep apnea with an
overall apnea-hypopnea index of 96 events per hour. This was associated
with hypoxia, with oxygen desaturations down to 74%.

Because of the tremendous severity of his obstructive sleep apnea, the
patient was woken up at 1:21 in the morning and placed on CPAP. On CPAP, h

Figure 3. Excerpt from baseline polysomnogram. The study was performed on 03-05-2008. Retrieved from personal archives with permission.

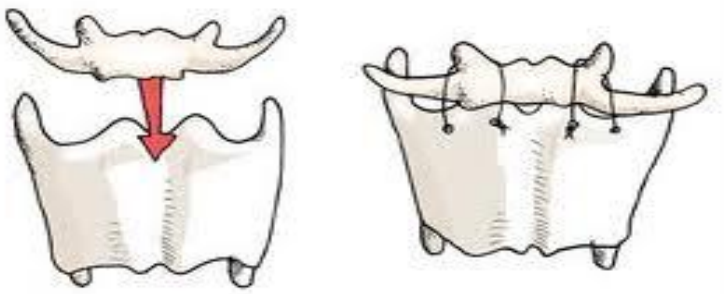


Figure 4. Depiction of myohyoid suspension surgery for OSA. Retrieved from http://personal.inet.fi/tiede/tapiovaara/kevatkokous2008.htm

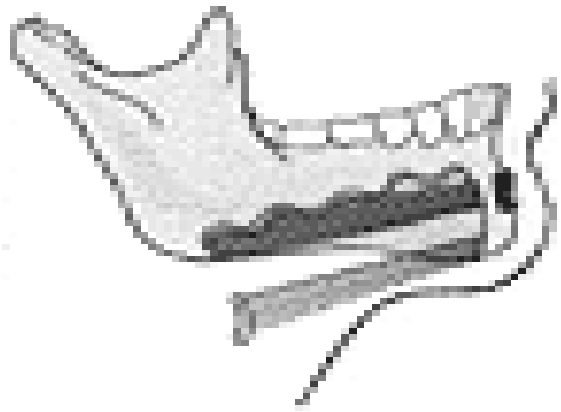


Figure 5. Depiction of genioglossal advancement. Retrieved from

http://www.medscape.com/viewarticle/501708_3

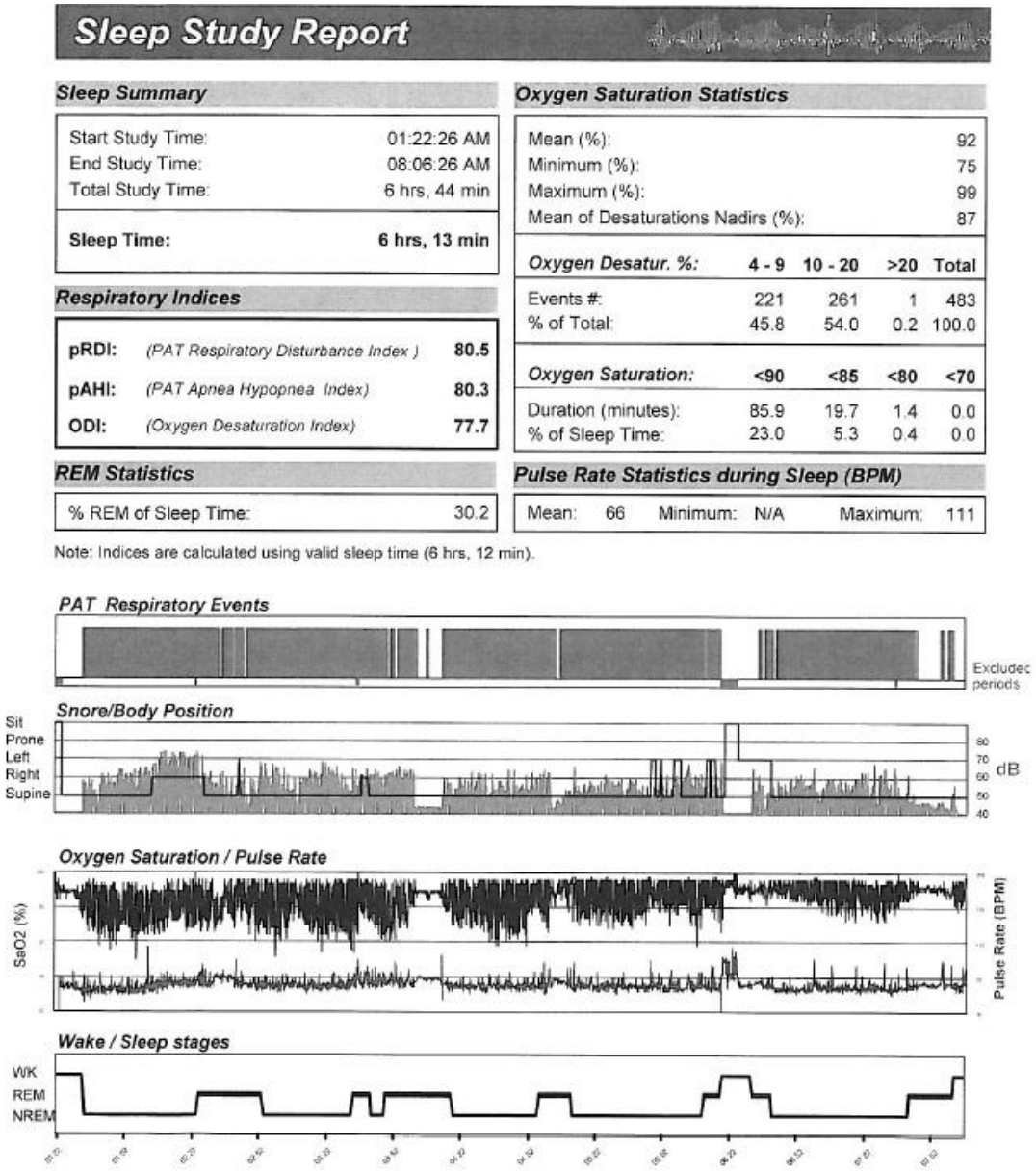
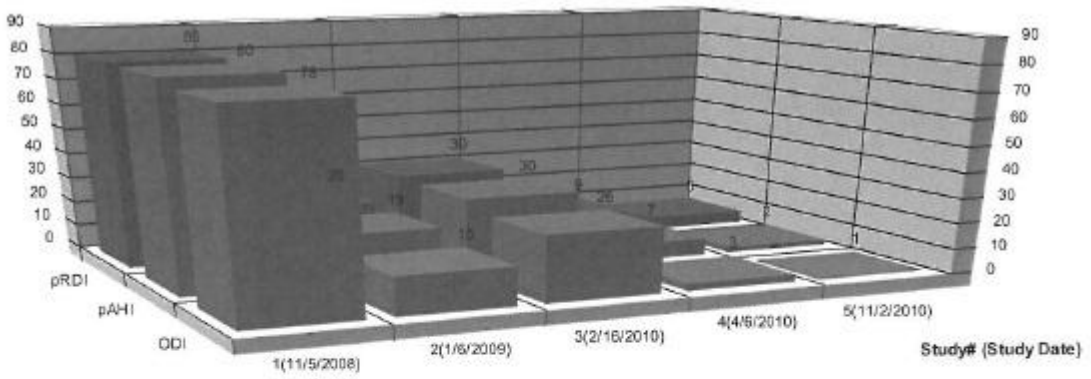


Figure 6. Excerpt from baseline home sleep testing performed prior to MAD intervention for OSA. The study was performed on 11-05-2008. Retrieved from personal archives with permission.

Figure 7. These are examples of the line of TAP II mandibular advancement devices that are FDA-approved to treat obstructive sleep apnea and snoring. Retrieved from www.amisleep.com



Study #:	Study Date	Ref. Physician	pRDI	pAHI	ODI
1 Diagnosis:	05-Nov-2008	Josh Ramseyer	80.5	80.3	77.7
2 Diagnosis:	06-Jan-2009	Josh Ramseyer	20.2	19.3	15.1
3 Diagnosis:	16-Feb-2010	Josh Ramseyer	30.5	29.7	25.9
4 Diagnosis:	06-Apr-2010	Josh Ramseyer	9.0	6.8	2.9
5 Diagnosis:	02-Nov-2010	Josh Ramseyer	5.0	2.0	0.5

Total Number of Studies: 5

Figure 8. Serial level III home sleep testing observations over a two year span. Retrieved from personal archives with permission.

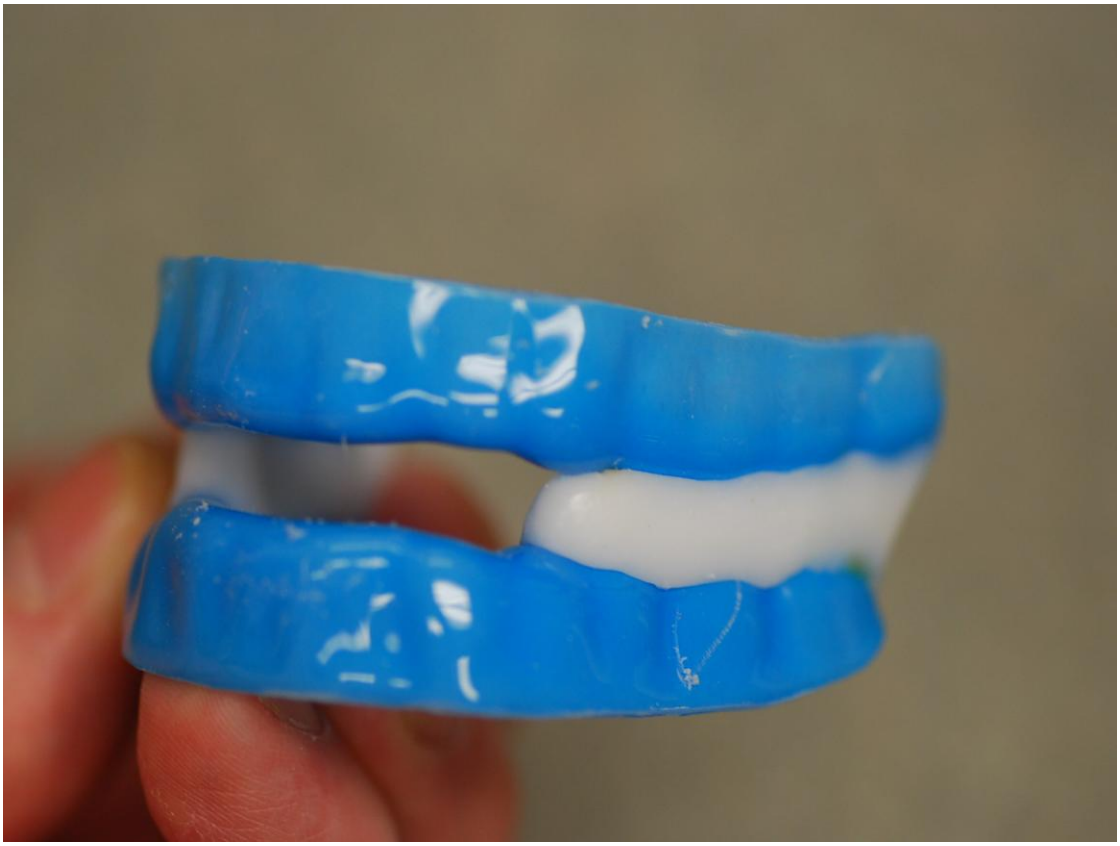
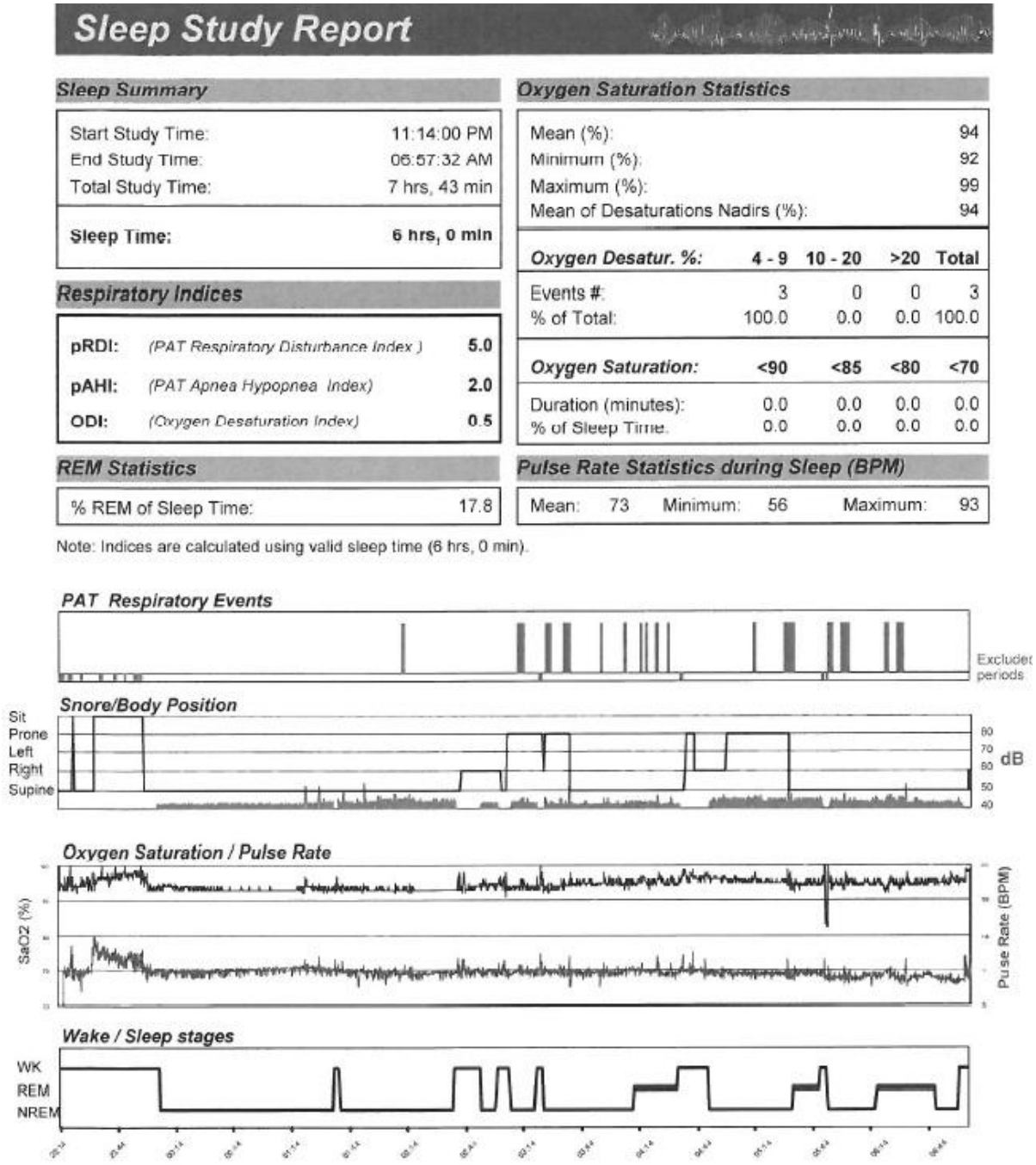


Figure 9. This is an example of the SleepApp oral appliance. This oral device was assessed on the most recent level III home sleep test. Retrieved from personal archives with permission.



Sleep Study Report

Sleep Summary

Start Study Time:	11:14:00 PM
End Study Time:	05:57:32 AM
Total Study Time:	7 hrs, 43 min
Sleep Time:	**6 hrs, 0 min**

Respiratory Indices

pRDI:	*(PAT Respiratory Disturbance Index)*	**5.0**
pAHI:	*(PAT Apnea Hypopnea Index)*	**2.0**
ODI:	*(Oxygen Desaturation Index)*	**0.5**

REM Statistics

% REM of Sleep Time:	17.8

Oxygen Saturation Statistics

Mean (%):	94
Minimum (%):	92
Maximum (%):	99
Mean of Desaturations Nadirs (%):	94

Oxygen Desatur. %:	**4 - 9**	**10 - 20**	**>20**	**Total**
Events #:	3	0	0	3
% of Total:	100.0	0.0	0.0	100.0

Oxygen Saturation:	**<90**	**<85**	**<80**	**<70**
Duration (minutes):	0.0	0.0	0.0	0.0
% of Sleep Time:	0.0	0.0	0.0	0.0

Pulse Rate Statistics during Sleep (BPM)

Mean:	73	Minimum:	56	Maximum:	93

Note: Indices are calculated using valid sleep time (6 hrs, 0 min).

PAT Respiratory Events

Excluded periods

Snore/Body Position

Sit
Prone
Left
Right
Supine

80
70
60
50
40
dB

Oxygen Saturation / Pulse Rate

SaO2 (%)

Pulse Rate (BPM)

Wake / Sleep stages

WK
REM
NREM

Figure 10. Excerpt from MAD treatment evaluated by Level III home sleep testing for OSA. The study was performed on 11-02-2010. Retrieved from personal archives with permission.

OREGON HEALTH & SCIENCE UNIVERSITY

SCHOOL OF NURSING
Ashland Klamath Falls La Grande Portland

DNP Clinical Inquiry Project Report & DNP Portfolio Approval

Student Name: Joseph Zelk

Degree: Doctor of Nursing Practice

Title of Study:

Title: Retrospective chart review: De-identified patient data from an outpatient clinic relating to OSA treatment with Sleep App™ oral appliance

APPROVED:

Date: [redacted]

Submit completed originol form to the Groduate Program office.

Revised 4/2009