Obstructive Sleep Apnea in Association with Periodontitis: a Case–Control Study

Nuha Ejaz Ahmad, BSDH, MSDH; Anne E. Sanders, PhD; Rose Sheats, DMD, MPH; Jennifer L. Brame, MSDH; Greg K. Essick, DDS, PhD

Introduction

Periodontitis results from an interaction between bacterial infection and host immunological and inflammatory responses. The disease is characterized by destruction of the tooth–supporting tissues leading to the development of periodontal pockets, alveolar bone loss and tooth mobility.[1] Epidemiologic data from NHANES 1999 to 2004 estimate the prevalence of moderate or severe periodontitis among dentate adults aged 20 to 64 years to be 9.3%, and among older adults to be 26.6%.[2]

Periodontitis is a suspected risk factor for low–birth weight and for many systemic diseases, such as cardiovascular disease, stroke, diabetes mellitus, respiratory disease, and rheumatoid arthritis.[3–9] The inflammation surrounding the teeth occurs as the immune system responds to the accumulation of bacterial plaque on the teeth and gums, and is modified by behavioral factors, systemic disease, medications and immune and hematological disorders.[10] Furthermore, the presence of systemic disorders can also affect the efficacy of periodontal therapy.[10]

The prevalence and severity of periodontitis increases with age, and is greater among male than female adults and greater in adults with low socio–economic status (SES) relative to their higher SES counterparts.[11–13] Obesity, nasal breathing difficulty, cigarette smoking and alcohol consumption are positively associated with periodontitis.[14–19]

Abstract

Purpose: Periodontitis is associated with several cardio–metabolic disorders that are co–morbid with sleep–disordered breathing. A relationship between periodontitis and obstructive sleep apnea (OSA) is plausible, but has received little attention. This study investigated the strength of association between periodontitis and risk for OSA.

Methods: In this case–control study, cases had moderate or severe periodontitis (n=50, 32.5%) and controls had gingivitis or slight periodontitis (n=104, 67.5%). Sixty–one males (39.6%) and 93 females (60.4%) with a mean age of 61 years were sampled from the dental hygiene preventive care clinic in the School of Dentistry at the University of North Carolina at Chapel Hill between February and April 2011. Patients received a full mouth periodontal examination that included probing pocket depths and clinical attachment levels at 6 sites per tooth. The case definition for moderate or severe periodontitis was that of the American Dental Association (ADA). Risk for OSA was determined by the 4 item "STOP" OSA screening questionnaire, which assesses self–reported snoring, excessive daytime sleepiness, witnessed apnea during sleep and history of hypertension. Demographic, general health and orofacial characteristics were recorded that were considered putative predictors of either periodontitis or OSA. A multivariate binary logistic regression assessed odds of moderate or severe periodontitis according to OSA risk with adjustment for potential confounders.

Results: In all, 59 patients (38.3%) screened at high risk for OSA by providing 2 or more affirmative responses on the STOP questionnaire. Sixty percent of periodontitis cases (n=30) screened high risk of OSA compared with only 28% of controls (n=29). Cases were 4.1 times more likely (95% CI: 1.9, 11.4) to be at high risk for OSA than controls (p=0.007) after adjustment for potential confounders.

Conclusion: A significant association was observed between moderate or severe periodontitis and risk for OSA.

Keywords: obstructive sleep apnea, periodontitis, STOP questionnaire, epidemiology, human

This study supports the NDHRA priority area, **Clinical Dental Hygiene Care:** Investigate the links between oral and systemic health.

Of most relevance to the present study are findings from a small exploratory study conducted in Australia by Gunaratnam, et al.[20] The investigators reported a significant positive relationship between polysomnography–derived apnea hypopnea index with less than 5 events/hour and periodontitis.[20] In that study, the case definition of moderate or severe periodontitis was that of the Centers for Disease Control and Prevention and the American Academy of Periodontology (CDC/AAP), which defines moderate or severe periodontitis as the presence of two or more interproximal sites with ≥4 mm clinical attachment level, not on the same tooth, or 2 or more interproximal sites with periodontal pocket depth ≥5 mm, not on the same tooth.[10] The investigators found that the prevalence of periodontitis was 77% in the study group, which was 4 times the national average for a representative sample of the adult Australian population using the same CDC/AAP periodontitis case classification. The authors suggested that because both obstructive sleep apnea (OSA) and periodontitis share an underlying inflammatory basis, a common biological pathway for the association was plausible. However, the study's findings were limited by a lack of data from a comparison group without OSA.[20]

OSA is the most common form of sleep–disordered breathing, estimated to affect 18 million individuals in the U.S,[21] Data from the Wisconsin Cohort Study indicates that the prevalence of OSA in people between the ages of 30 and 60 years was 9% to 24% for men and 4% to 9% for women.[22] Characterized by repetitive full or partial collapse of the upper airway, OSA results in periods of asphyxia, hypoxia, arousal from sleep, stimulation of the sympathetic nervous system and altered immunity.[23-25] Clinically, the condition frequently presents as habitual loud snoring, apnea witnessed by a bed partner, arousal during sleep and sleepiness and fatigue during the day.[26] As a life–threatening condition, OSA is associated with hypertension, congestive heart failure, coronary artery disease, myocardial infarction, cardiac arrhythmia, stroke, impaired glucose tolerance and type II diabetes mellitus.[27–32]

Shared risk factors for periodontitis and OSA include male sex, older age, obesity, oral breathing, cigarette smoking and alcohol consumption.[33–39] OSA is formally diagnosed during an overnight sleep study using polysomnography to grade the severity of the disorder according to the frequency of airway collapse, expressed as the apnea–hypopnea index.[40] However, screening tools exist that assess an individual's risk for OSA in various, often pre–surgical, settings.[23,41]

Given the reported high prevalence of periodontitis in patients with confirmed OSA, the current study questioned whether the prevalence of OSA was associated with the severity of periodontitis. To address this question, the risk for OSA in patients with different degrees of periodontitis severity as determined during the patients' scheduled dental visit was evaluated. The risk for OSA was assessed using the STOP questionnaire, a validated screening questionnaire.[41] Data was obtained for potentially confounding factors, such as age and gender, which are associated with increased risk for periodontitis and/or OSA.

Methods and Materials

Study Design

In this case control study, cases had moderate or severe periodontitis (case types III and IV). Dentate patients having gingivitis or slight (early) periodontitis (case types I and II) were classified as controls (Table I).

Study Population and Recruitment Procedures

Study participants were patients treated between February and April 2011 in the dental hygiene preventive care clinic at the University of North Carolina (UNC) School of Dentistry. Inclusion criteria were being dentate and aged 18 years or older. Edentulous patients and patients with healthy periodontium were excluded from participation. Potential patients were approached by the first author (hereafter referred to as the investigator) after the patients' scheduled dental cleaning was completed by the assigned dental hygiene student. Each patient was given a brief description of the study along with educational materials on OSA, after which verbal consent was obtained. The protocol was approved by the Institutional Review Board at the UNC–Chapel Hill.

Data collection

Following consent, each patient completed a sociodemographic/behavioral questionnaire while the investigator completed a clinical characteristics questionnaire. Both questionnaires were processed using Cardiff TeleForm® (version 10.5.1© 2010 Verity, Inc.), an optical scanning system that efficiently transfers penciled information to an electronic format for statistical analysis.[42] The questionnaires were designed to obtain data for this study as well as for a separate analysis of sociodemographic and clinical characteristics associated with high risk of OSA, an analysis that is not included in this publi-

Table I: Periodontal Case Type Definition

Healthy Periodontium, Type N	Gingivitis Type I	Slight* Periodontitis Type II	Moderate Periodontitis Type III	Severe Periodontitis Type IV
• No alveolar bone loss • Healthy gingiva and alveolar bone level • No bleeding • 1 to 2 mm probing depths	• No alveolar bone loss • Bleeding and/or suppuration may be present • 2 to 3 mm probing depths	• Slight attachment loss • Slight bone loss is evident, especially in alveolar crest • 3 to 4 mm probing depths	• Moderate alveolar bone and attachment loss • 5 to 6 mm probing depths	• Severe alveolar bone and attachment loss • 7+ mm probing depths
• No clinical attachment loss (CAL)	• No clinical attachment loss (CAL)	• 1 to 2 mm clinical attachment loss (CAL)	• 3 to 4 mm clinical attachment loss (CAL)	• Greater than or equal to 5 mm clinical attachment loss (CAL)
• No mobility • No furcation	• No mobility • No furcation	• No mobility • No furcation	• Mobility is possible • Furcation involvement is possible	• Mobility is possible • Furcation involvement is possible

Please Note: Periodontal classification is based on clinical and radiographic findings. Findings must be generalized in order to be classified in a higher case type.

localized: <30% of sites involved   generalized: >30% of sites involved

*Note that the American Dental Association (ADA) uses the term "early" in the definition of case–type II periodontitis, where the School of Dentistry at the University of North Carolina at Chapel Hill uses the term "slight " in the definition of case–type II periodontitis

cation. No identifying data was included on either questionnaire.

Sociodemographic/Behavioral Questionnaire

The questionnaire was used to obtain information about the patient's age, gender, weight, height, education, annual household income, sleep quality (snoring, observed apnea, tiredness during the day, Epworth Sleepiness Scale,[43] teeth grinding and nasal–breathing difficulty), social habits (smoking, alcohol and sedative consumption before bedtime) and medical history (high blood pressure, diabetes mellitus, previous diagnosis with OSA or any form of sleep–disordered breathing and any previous or current treatment for OSA).

Clinical Characteristics Questionnaire

The investigator completed this questionnaire, recording relevant clinical observations, some of which were directly observed by the investigator. Other items were observations made by the dental hygiene student, confirmed by the clinical instructor and recorded in the patient's dental chart, from which the investigator extracted pertinent information. On the questionnaire, physical measurements were recorded of the patient's blood pressure (taken by the assigned dental hygiene student), body–mass index (BMI) (calculated by the investigator using the patient's self–reported weight and height) and neck circumference (measured by the investigator using a disposable tape measure). Observations of the oral soft tissue included the presence of dry mouth (as assessed by the moistness of the oral mucosa, and whether the mouth mirror easily stuck to the buccal mucosa as reported by the dental hygiene student[44]), the presence of macroglossia or large tongue (as assessed by the investigator examining the lateral and anterior borders of the tongue)[45] and the Mallampati score and tonsils grade, both of which were assessed by the investigator while the patient was in a seated position, with the head in full extension, the tongue out and with phonation.[46–48] Observations of oral hard tissue included the attrition of teeth (as measured by the investigator using the Basic Erosive Wear Examination using the highest score per sextant),[49] overjet (as measured by the dental hygiene student using a calibrated dental probe) and the morphology of the maxillary arch (as assessed by the investigator observing the shape of the hard palate).

The remaining items (approximately half) on the clinical characteristics questionnaire pertained to the patient's periodontal status. Recorded periodontal indices included the Plaque Index, Gingival Index and Bleeding Index (all of which were measured by the dental hygiene student).[50] Periodontal measurements, also assessed by the dental hygiene student using a calibrated probe, included the peri-

Table II: Percent of Periodontitis Cases According to Number of Affirmative Responses on the STOP Obstructive Sleep Apnea Screening Questionnaire

Number of affirmative responses to STOP items	Gingivitis or slight periodontitis (%)	Moderate or severe periodontitis (%)	p–value
0	77.1	22.9	0.002
1	80.0	20.0	
2	52.4	47.6	
3	46.7	53.3	
4	0.0	100.0	

odontal pocket depth (the distance from the gingival margin to the base of the pocket) and gingival recession (the distance from the cemento–enamel junction to the gingival margin). The patient's periodontal case classification, based on a detailed clinical periodontal charting with radiographic interpretation of the bone levels as prescribed by the American Dental Association, was obtained directly from the patients' chart.[51] Based primarily on the severity of attachment loss, the patient was classified into 1 of 4 categories: case type I: gingivitis, case type II: slight (early) periodontitis, case type III: moderate periodontitis or case type IV: advanced (severe) periodontitis (Table I).

Data Analysis and Statistical Methods

Using relevant patient–reported responses to the Symptom/Health Questionnaire, the patient's risk of OSA was calculated as described for the validated OSA screening instrument known as STOP questionnaire. The 4 questions assess the presence of loud snoring, frequent daytime sleepiness and tiredness, observed apnea during sleeping and high blood pressure. Responses to each question are "Yes" or "No." According to the questionnaire's scoring algorithm, affirmative responses to any 2 or more of these 4 questions denotes high risk for OSA, while affirmative responses to fewer than 2 questions denotes low risk for OSA.

Descriptive statistics summed the number of patients responding affirmatively to none, 1, 2, 3 or 4 of the STOP OSA screening items. The number and proportion of patients at high risk for OSA were calculated. Exploratory analyses were performed to describe the distribution of affirmative responses to each of the 4 STOP OSA screening items. The association between the study population's socio–demographic, behavioural and clinical characteristics and risk for OSA was tested for statistical significance using Fisher's exact test for dichotomized risk indicators and Pearson's chi–square test for categorical risk indicators. Potential covariates were limited to characteristics that have been associated with risk of periodontitis and/or

OSA: gender, age, BMI, educational attainment, annual household income, cigarette smoking, alcohol/sedative consumption before bed, diabetes mellitus, impaired nasal breathing and dry mouth.

Covariates were included in multivariable binary logistic regression models if their associations in unadjusted analyses with either outcome, OSA risk or periodontitis, were significant at the p<0.2 level. Multivariable binary logistic regression was used to estimate the odds ratio and 95% confidence interval associating moderate/severe periodontitis with high risk for OSA with adjustment for confounding. Analyses were conducted using STATA/IC software version 12.0 (StataCorp. 2011).

Results

The study population of 154 patients comprised 50 cases (32.5%) and 104 controls (67.5%). Based on the STOP questionnaire, 38.3% of study patients were at high risk for OSA, providing affirmative responses to 2 items (27.7%), 3 items (9.7%) or all 4 items (1.2%). The likelihood of being classified a case increased with the number of affirmative responses (p=0.002, Table II), suggesting a crude association between OSA and periodontitis.

A higher percentage of cases than controls were at high risk for OSA for each STOP questionnaire item (Table III). Moreover, cases and controls differed significantly on the report of high blood pressure (p=0.006) and tiredness/sleepiness during the day (p=0.016). The proportion of cases was almost twice that of controls for these 2 STOP questionnaire items.

Univariate Analyses

Univariate analyses were undertaken to determine if each of 8 characteristics was associated with the risk of OSA (Table IV). A significant positive association was observed with age (p=0.018), self–reported diabetes mellitus (p=0.008) and dry mouth (p=0.002).

Table III: Response to Individual Items on the STOP OSA Screening Questionnaire[a] and Relationship With Periodontitis Case Status

	STOP screening questionnaire items	Response	n (%)	Moderate or severe periodontitis (%)	p–value[b]
S	Do you snore loudly?	No	124 (80.5)	29.8	0.157
		Yes	30 (19.5)	43.3	
T	Do you often feel tired, fatigued, or sleepy during the day?	No	83 (53.9)	24.1	0.016
		Yes	71 (46.1)	42.3	
O	Has anyone observed you stop breathing during your sleep?	No	132 (85.7)	31.1	0.361
		Yes	22 (14.3)	40.9	
P	Do you have or are you being treated for high blood pressure?	No	80 (51.9)	22.5	0.006
		Yes	74 (48.1)	43.2	

a – The STOP questionnaire classifies persons as high risk for OSA with affirmative responses to ≥2 STOP questions; low risk is defined as <2 affirmative responses
b – p–value tests the null hypothesis that no difference exists between response to the OSA screening question and periodontitis case status

Univariate analyses were undertaken to determine if each of the 10 characteristics was associated with moderate or severe periodontitis (Table V). Odds ratios of moderate or severe periodontitis increased with age, more prevalent among current cigarette smokers and diabetic patients.

Multivariable Analyses

A multivariate binary logistic regression was performed with case status (1=case, moderate or severe periodontitis, 0=control, gingivitis or slight periodontitis) as the dependent variable. Independent variables were those patient characteristics that met criteria based on the univariate analyses (Table VI). It was found that patients at high risk of OSA had 4.1 times greater odds (95% CI: 1.5, 11.4) of moderate or severe periodontitis than patients at low risk, after adjustment for potential confounders. Significant covariates included age, smoking and nasal–breathing difficulty. Diabetic patients had 2.7 times greater odds for having moderate or severe periodontitis, but the 95% confidence interval was equivocal (0.6, 11.4).

Discussion

The results of this study extend the finding of Gunaratnam et al that prevalence of periodontitis may be higher in patients with OSA.[20] Specifically, the present study suggests that odds of moderate or severe periodontitis were elevated 4.1–fold (95% CI: 1.5, 11.4) among patients screening high risk relative to low risk for OSA after adjustment for potential confounders. The 2 studies, however, differ in a number of ways. Gunaratnam et al studied patients who were clinically diagnosed with OSA.[20] In contrast, the current study compared periodontitis

cases and controls and estimated their risks of OSA using a validated questionnaire.[41] Unlike Gunaratnam et al, it remained unknown whether the patients in the present study, in general, had OSA.[20] In both studies, periodontitis was diagnosed and classified by clinical examination. While findings of both studies suggest that the prevalence of periodontitis is greater in patients with OSA, the case control study design used in the present study provided a higher level of evidence in support of this association.

Mechanisms Underlying Association Between Periodontitis and OSA

Gunaratnam et al suggested that the increased prevalence of periodontitis in OSA patients could be due to a true association between OSA and periodontitis: OSA could act as an inflammatory mediator for periodontitis or vice versa.[20] It is also possible that an increased prevalence of mouth breathing in patients with OSA could exacerbate periodontitis and underlie the association.[20] However, in the present study, there was no association between the presence of dry mouth and periodontitis. Moreover, patients who reported more difficulty with nasal breathing (presumably favoring oral to nasal breathing) were less likely to exhibit moderate or severe periodontitis. Given that periodontitis and OSA are co–morbid with a large number of pathological conditions, it is also possible that their association is not causative but rather a reflection of their relationship to these common co–morbid conditions.[20]

Confounding variables

The multivariable analysis took into account a number of factors that have been associated with

Table IV: Relationship of study participant characteristics and high risk for obstructive sleep apnea[a]

Characteristic	n (%)	Percent with High Risk OSA	p–value
Total	154 (100.0)	38.3	
Gender			
• Female • Male	93 (60.4) 61 (39.6)	37.6 39.3	0.831
Age (years)			
• 18 to 49 • 50 to 59 • 60 to 69 • ≥70	32 (20.8) 26 (16.9) 49 (31.8) 47 (30.5)	15.6 34.6 46.9 46.8	0.018
BMI (kg/m2)[b]			
• Underweight/normal • Overweight • Obese • Missing	53 (34.4) 52 (33.8) 47 (30.5) 2 (1.3)	26.4 44.2 42.6 –	0.117
Cigarette smoking status			
• Current • Former • Never • Missing	10 (6.5) 64 (41.6) 77 (50.0) 3 (2.0)	30.0 35.9 41.6 –	0.675
Alcohol/sedatives before bed			
• Yes • No • Missing	126 (81.8) 27 (17.5) 1 (0.7)	38.1 40.7 –	0.798
Self–reported diabetes status			
• No diabetes • Diabetes	134 (87.0) 20 (13.0)	34.3 65.0	0.008
Nasal breathing status			
• No difficulty • Difficulty • Missing	138 (89.6) 15 (9.7) 1 (0.7)	39.1 33.3 –	0.661
Dry mouth			
• No dry mouth • Dry mouth	115 (74.7) 39 (25.3)	31.3 59.0	0.002

a – The STOP questionnaire classifies persons as high risk for OSA with affirmative responses to ≥2 questions; low risk is defined as <2 affirmative responses
b – World Health Organization International Classification: underweight (<18.50); normal (18.50–24.99); overweight (25.00–29.99); obese (≥30.00)

periodontitis, some of which have also been associated with sleep–disordered breathing. These are briefly discussed in the sections below in the order observed in Table VI.

Gender: Although the present study did not demonstrate a significant association between gender and the severity of periodontitis, several studies have reported that the prevalence of periodontitis is greater in males than in females.[11,12] For example, Albandar et al reported that in individuals 30 to 54 years of age, a prevalence of periodontitis was found in 34% of males but only 23% of females.[11] In individuals 55 to 90 years of age, the prevalence was 56% in males and 44% in females.[11]

Age: Age is an important risk factor for periodontitis in many studies. Both the prevalence and extent of periodontitis increase with age.[11] For example, Albanadar et al showed that 29% of persons in the aged 30 to 54 years old had periodontitis, compared with 50% aged 55 to 90 years old.[11] The present study confirmed a positive association between age and periodontitis and found that patients

Table V: Relationship of Study Participant Characteristics and the Presence of Moderate or Severe Periodontitis

Characteristic	n (%) with moderate or severe periodontitis	Odds ratio for moderate or severe periodontitis	95% CI
Total	50 (32.5)		
Gender			
• Female	27 (29.0)	Ref	–
• Male	23 (37.7)	1.48	0.75, 2.93
Age (years)			
• 18–49	6 (18.8)	Ref	–
• 50–59	4 (15.4)	0.78	0.20, 3.15
• 60–69	13 (26.5)	1.56	0.52, 4.6
• ≥70	27 (57.5)	5.85	2.02, 16.87
BMI (kg/m2)[a]			
• Underweight/normal	14 (26.4)	Ref	–
• Overweight	17 (32.7)	1.35	0.58, 3.14
• Obese	18 (38.3)	1.73	0.74, 4.04
Cigarette smoking status			
• Current	5 (50.0)	2.35	0.62, 8.90
• Former	22 (34.4)	1.23	0.60, 2.50
• Never	23 (29.9)	Ref	–
Alcohol/sedatives before bed			
• Yes	44 (34.9)	Ref	–
• No	6 (22.2)	0.53	0.20, 1.42
Self–reported diabetes status			
• No diabetes	96 (71.6)	Ref	–
• Diabetes	8 (40.0)	3.79	1.44, 10.00
Nasal breathing status			
• No difficulty	49 (35.5)	Ref	–
• Difficulty	1 (6.7)	0.13	0.02, 1.02
Dry mouth			
• No dry mouth	36 (31.3)	Ref	–
• Dry mouth	14 (35.9)	1.23	0.57, 2.64
Educational attainment			
• <College	29 (35.4)	Ref	–
• ≥College	21 (29.2)	0.75	0.38, 1.49
Annual household income (USD)			
• <50,000	28 (37.3)	Ref	–
• ≥50,000	15 (25.0)	0.56	0.26, 1.18

a – World Health Organization International Classification: underweight (<18.50), normal (18.50 to 24.99), overweight (25.00 to 29.99) and obese (≥30.00)

who were 70 years and older had 1.76 times greater odds (95% CI: 1.17–2.64) of moderate or severe periodontitis than patients who were between 18 to 49 years old.

BMI: The present study did not find a significant association between BMI and the severity of periodontitis. This stands in contrast to reported findings by other investigators.[14,15] Chaffee conducted a systematic review and meta analysis of 57 independent studies on the association between BMI and periodontitis, and identified a slight linear increase in the odds of periodontitis with increasing BMI.[14]

Cigarette smoking status: This study demonstrated a significant association between current cigarette smoking and the severity of periodontitis, where current smokers had 24.9 times greater odds

Table VI: Multivariate Binary Logistic Regression Modelling Odds of Moderate or Severe Periodontitis With 95% Confidence Interval (95% CI), (n=132)

	Odds ratio	95% CI
Male gender (ref=female)	1.78	0.64, 4.94
Age in decades[a]	1.76	1.17, 2.64
Body mass index (continuous variable)	1.06	0.96, 1.16
Current smoker (ref=never smoked)	24.68	3.19, 191.20
Former smoker (ref=never smoked)	0.72	0.24, 2.11
Alcohol/sedative before bed (ref=no alcohol/sedative)	0.72	0.19, 2.71
Diabetes (ref=no diabetes)	2.70	0.64, 11.37
Nasal breathing difficulty (ref=no difficulty)	0.09	0.01, 0.88
Dry mouth (ref=no dry mouth)	0.36	0.10, 1.39
Educational attainment ≥college (ref≤college)	0.63	0.22, 1.82
Annual household income ≥$50,000 (ref≤USD$50,000)	0.63	0.24, 1.63
High risk for obstructive sleep apnea on STOP questionnaire (ref=low risk)	4.11	1.48, 11.45
Constant	0.00	0.00, 0.18

a – Age measured in years is not substantively meaningful; hence age was rescaled in units of 10 to denote decade–sized units

(95% CI: 3.2–191.2) of having moderate or severe periodontitis than those who never smoked. The lack of precision was attributed to the small sample size. A similar finding has been observed by many previous epidemiological studies reporting smoking as a significant risk factor for periodontitis.[18,52] For example, Bergström found that smokers have 2.5 to 3.5 times greater risk of severe periodontal attachment loss than never smokers.[52]

Alcohol/Sedative use Before Bedtime: Although the current study did not find significant associations between alcohol consumption before bedtime and severity of periodontitis, several studies have suggested that alcohol consumption in general, with no restriction to bedtime, is a risk factor for oral diseases, including periodontitis, as it increases the host's susceptibility to infection.[53] The findings in the current study may not have demonstrated an association due to limiting the question to a specific time of day.

Diabetes: The present study found that patients who reported being diabetic had 2.70 times greater odds (95% CI: 0.64–11.37) of having moderate to severe periodontitis than patients who did not report being diabetic. This finding has been supported by many studies that indicate that diabetes and poor glycemic control are significant risk factors for periodontitis and vice versa.[7,54–56]

Nasal Breathing Difficulty (Mouth Breathing): The results of this study indicate that difficulty with nasal breathing favored slight periodontitis or gingivi-tis rather than moderate or severe periodontitis: Patients with nasal breathing difficulty had 0.09 times the odds (95% CI: 0.01–0.88) of having moderate or severe periodontitis of patients with no nasal breathing difficulty. No studies have addressed the relationship between mouth breathing and periodontitis in adults, however, a relationship has been reported in teenagers.[17]

Dry Mouth: The results of this study did not demonstrate a significant relationship between dry mouth and the severity of periodontitis. Moreover, there is little published evidence to suggest that dry mouth has a direct influence on periodontitis. This may be because saliva does not enter into the periodontal pockets where the bacterial pathogens are located.[57]

Educational attainment: Although this study did not demonstrate a significant association between educational attainment and the severity of periodontitis, a meta–analysis conducted by Boillot concluded that low educational attainment is associated with increased risk for periodontitis.[58]

SES: The results of this study did not demonstrate an association between SES and the severity of periodontitis. However, a significant association has been reported in several studies. Specifically, patients with high SES have been reported to have healthier periodontium than patients with low SES.[13]

An important strength of this study is that peri-

odontitis cases and controls were drawn from the same source population during the same time interval. All patients underwent the same measurements of periodontal health and OSA screening which were performed by the same clinicians. This enhances the principal of comparability, by ensuring less variability and a reduced risk of confounding.

Limitations of Study

This study relied on periodontal data collected by multiple examiners. However, all examiners were calibrated on the periodontal assessment and classification, and the supervising faculty confirmed the assessments before they were entered into the patients' records. Each classification was defined by precise measurements of periodontal pocket depth, clinical attachment level, mobility and/or furcation involvement and by whether these conditions were localized (<30% of the site) or generalized (≥30% of the site). This reduced the risk of case status misclassification.

The major limitation of this study is the uncertainty in the proportions of the cases and controls with and without OSA, respectively. The STOP questionnaire is a measure of risk for sleep apnea but falls short of clinical diagnosis. This questionnaire was developed for the pre–surgical assessment of patients prior to general anesthesia and administration of agents that depress respiration. Its diagnostic usefulness has been determined for patients with different severity levels of OSA based on the apnea hypopnea index. Specifically, its sensitivity and specificity with the apnea hypopnea index cutoff >5 events/hour are estimated to be 65.5% and 60%, respectively, >15 events/hour, 74.3% and 53.3%, and >30 events/hour, 79.5% and 48.6%. Though some other screening questionnaires have been shown to have higher diagnostic sensitivity, such as the STOP–BANG questionnaire,[41] the STOP questionnaire was chosen for this study because of its higher specificity. However, if misclassification of OSA status is non–differential, i.e. unrelated to periodontitis status, which is believed to be the case, then the effect of any misclassification bias is towards the null. What this means is that the odds ratio for the strength of the relationship between OSA risk and periodontitis shifts toward 1.0, making the initial estimate more conservative than it would otherwise be.

Relevance to Dental Hygienists: Patients visit the dental office more frequently than the physician's office. Moreover, dental cleaning is the most common periodically performed procedure in dentistry. One of the dental hygienists' responsibilities is to assess patients' overall well–being as well as to facilitate health promotion, disease prevention and patient education. The STOP screening questionnaire is an easy, time efficient questionnaire that, if included with the health history and periodic recall assessments, could help identify patients at high risk for a serious life–threatening condition. Moreover, the dental hygienist is optimally positioned in the dental practice to make soft tissue observations that have been associated with increased risk for OSA. By screening patients for sleep–disordered breathing, the dental hygienist supports the practice in fulfilling a greater role in the public health of the community.[21,59,60]

Conclusion

This case–control study found a positive significant association between moderate or severe periodontitis and high risk for OSA, based on the STOP questionnaire, after adjustment for potential confounders. Further investigation of this association using objective measures of OSA (polysomnography or home sleep test) is warranted. The STOP questionnaire is a simple, inexpensive screening tool could be included with the health history forms and used to identify dental patients at high risk for a life–threatening medical condition. Further investigation should include sleep study to confirm the presence or absence of OSA in patients with and without OSA. As well as to further investigate the underlying pathophysiological mechanisms of both conditions that can better explain the significant association.

Nuha Ejaz Ahmad, BSDH, MSDH, currently works at the King Saud University, College of Applied Medical Sciences, Dental Health Department, Riyadh, Saudi Arabia. At the time of this study, she was a graduate student in the Master of Science Degree Program in Dental Hygiene Education at the University of North Carolina. Anne E. Sanders, PhD, is an assistant professor, Department of Dental Ecology. Rose Sheats, DMD, MPH, is a Graduate Program Director, Department of Orthodontics. Jennifer L. Brame, RDH, MSDH, is a Clinical Assistant Professor, Department of Dental Ecology. Greg K. Essick, DDS, PhD, is a Professor, Department of Prosthodontics and Center for Neurosensory Disorders. All four are at the School of Dentistry at the University of North Carolina Chapel Hill.

References

1. Kim J, Amar S. Periodontal disease and systemic conditions: A bidirectional relationship. *Odontology*. 2006;94(1):10–21.

2. Dye BA, Tan S, Smith V, et al. Trends in oral health status: United states, 1988–1994 and 1999–2004. *Vital Health Stat 11*. 2007;(248):1–92.

3. Offenbacher S, Boggess KA, Murtha AP, et al. Progressive periodontal disease and risk of very preterm delivery. *Obstet Gynecol*. 2006;107(1):29–36.

4. Joshipura KJ, Rimm EB, Douglass CW, Trichopoulos D, Ascherio A, Willett WC. Poor oral health and coronary heart disease. *J Dent Res*. 1996;75(9):1631–1636.

5. Pradeep AR, Hadge P, Arjun Raju P, Shetty SR, Shareef K, Guruprasad CN. Periodontitis as a risk factor for cerebrovascular accident: A case–control study in the indian population. *J Periodontal Res*. 2010;45(2):223–228.

6. Mealey BL, Oates TW, American Academy of Periodontology. Diabetes mellitus and periodontal diseases. *J Periodontol*. 2006;77(8):1289–1303.

7. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and diabetes: A two–way relationship. *Diabetologia*. 2012;55(1):21–23.

8. El Attar MM, Zaghloup MZ, Elmenoufr HS. Role of periodontitis in hospital–acquired pneumonia. *East Mediterr Health J*. 2010;16(5):563–569.

9. Scardina GA, Messina P. Microvascular periodontal alterations: A possible relationship between periodontitis and rheumatoid arthritis. *Clin Hemorheol Microcirc*. 2007;37(3):229–235.

10. Page RC, Eke PI. Case definitions for use in population–based surveillance of periodontitis. *J Periodontol*. 2007;78(7 Suppl):1387–1399.

11. Albandar JM, Brunelle JA, Kingman A. Destructive periodontal disease in adults 30 years of age and older in the united states, 1988–1994. *J Periodontol*. 1999;70(1):13–29.

12. Desvarieux M, Schwahn C, Völzke H, et al. Gender differences in the relationship between periodontal disease, tooth loss, and atherosclerosis. *Stroke*. 2004;35(9):2029–2035.

13. Chandra Shekar BR, Reddy C. Oral health status in relation to socioeconomic factors among the municipal employees of mysore city. *Indian J Dent Res*. 2011;22(3):410–418.

14. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: A systematic review and meta–analysis. *J Periodontol*. 2010;81(12):1708–1724.

15. Mathur LK, Manohar B, Shankarapillai R, Pandya D. Obesity and periodontitis: A clinical study. *J Indian Soc Periodontol*. 2011;15(3):240–244.

16. Al–Zahrani MS, Bissada NF, Borawskit EA. Obesity and periodontal disease in young, middle–aged, and older adults. *J Periodontol*. 2003;74(5):610–615.

17. Wagaiyu EG, Ashley FP. Mouthbreathing, lip seal and upper lip coverage and their relationship with gingival inflammation in 11–14 year–old schoolchildren. *J Clin Periodontol*. 1991;18(9):698–702.

18. Moimaz SA, Zina LG, Saliba O, Garbin CA. Smoking and periodontal disease: Clinical evidence for an association. *Oral Health Prev Dent*. 2009;7(4):369–376.

19. Amaral Cda S, Luiz RR, Leão AT. The relationship between alcohol dependence and periodontal disease. *J Periodontol*. 2008;79(6):993–998.

20. Gunaratnam K, Taylor B, Curtis B, Cistulli P. Obstructive sleep apnoea and periodontitis: A novel association? *Sleep Breath*. 2009;13(3):233–239.

21. Barsh LI. The recognition and management of sleep–breathing disorders: A mandate for dentistry. *Sleep Breath*. 2009;13(1):1–2.

22. Young T, Finn L, Peppard PE, et al. Sleep disordered breathing and mortality: Eighteen–year follow–up of the wisconsin sleep cohort. *Sleep*. 2008;31(8):1071–1078.

23. Enciso R, Clark GT. Comparing the berlin and the ARES questionnaire to identify patients with obstructive sleep apnea in a dental setting. *Sleep Breath*. 2011;15(1):83–89.

24. Bradford A, McGuire M, O'Halloran KD. Does episodic hypoxia affect upper airway dilator muscle function? implications for the pathophysiology of obstructive sleep apnoea. *Respir Physiol Neurobiol*. 2005;147(2–3):223–234.

25. Remmers JE, deGroot WJ, Sauerland EK, Anch AM. Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol*. 1978;44(6):931–938.

26. Ward Flemons W, McNicholas WT. Clinical prediction of the sleep apnea syndrome. *Sleep Med Rev*. 1997;1(1):19–32.

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

28. Shahar E, Whitney CW, Redline S, et al. Sleep–disordered breathing and cardiovascular disease: Cross–sectional results of the sleep heart health study. *Am J Respir Crit Care Med*. 2001;163(1):19–25.

29. Lee CH, Khoo SM, Chan MY, et al. Severe obstructive sleep apnea and outcomes following myocardial infarction. *J Clin Sleep Med*. 2011;7(6):616–621.

30. Mehra R, Benjamin EJ, Shahar E, et al. Association of nocturnal arrhythmias with sleep–disordered breathing: The Sleep Heart Health Study. *Am J Respir Crit Care Med*. 2006;173(8):910–916.

31. Capampangan DJ, Wellik KE, Parish JM, et al. Is obstructive sleep apnea an independent risk factor for stroke? A critically appraised topic. *Neurologist*. 2010;16(4):269–273.

32. Mahmood K, Akhter N, Eldeirawi K, et al. Prevalence of type 2 diabetes in patients with obstructive sleep apnea in a multi–ethnic sample. *J Clin Sleep Med*. 2009;5(3):215–221.

33. Kapsimalis F, Kryger MH. Gender and obstructive sleep apnea syndrome, part 1: Clinical features. *Sleep*. 2002;25(4):412–419.

34. Young T, Skatrud J, Peppard PE. Risk factors for obstructive sleep apnea in adults. *JAMA*. 2004;291(16):2013–2016.

35. Young T, Shahar E, Nieto FJ, et al. Predictors of sleep–disordered breathing in community–dwelling adults: The sleep heart health study. *Arch Intern Med*. 2002;162(8):893–900.

36. Koutsourelakis I, Vagiakis E, Roussos C, Zakynthinos S. Obstructive sleep apnoea and oral breathing in patients free of nasal obstruction. *Eur Respir J*. 2006;28(6):1222–1228.

37. Wetter DW, Young TB, Bidwell TR, Badr MS, Palta M. Smoking as a risk factor for sleep–disordered breathing. *Arch Intern Med*. 1994;154(19):2219–2224.

38. Ekici M, Ekici A, Keles H, et al. Risk factors and correlates of snoring and observed apnea. *Sleep Med*. 2008;9(3):290–296.

39. Mitler MM, Dawson A, Henriksen SJ, Sobers M, Bloom FE. Bedtime ethanol increases resistance of upper airways and produces sleep apneas in asymptomatic snorers. *Alcohol Clin Exp Res*. 1988;12(6):801–805.

40. Sleep–related breathing disorders in adults: Recommendations for syndrome definition and measurement techniques in clinical research. The report of an american academy of sleep medicine task force. *Sleep*. 1999;22(5):667–689.

41. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: A tool to screen patients for obstructive sleep apnea. *Anesthesiology*. 2008;108(5):812–821.

42. Quan KH, Vigano A, Fainsinger RL. Evaluation of a data collection tool (TELEform) for palliative care research. *J Palliat Med*. 2003;6(3):401–408.

43. Johns MW. A new method for measuring daytime sleepiness: The epworth sleepiness scale. *Sleep*. 1991;14(6):540–545.

44. Navazesh M, ADA Council on Scientific Affairs and Division of Science. How can oral health care providers determine if patients have dry mouth? *J Am Dent Assoc*. 2003;134(5):613–620.

45. Ueyama Y, Mano T, Nishiyama A, Tsukamoto G, Shintani S, Matsumura T. Effects of surgical reduction of the tongue. *Br J Oral Maxillofac Surg*. 1999;37(6):490–495.

46. Yagi H, Nakata S, Tsuge H, et al. Morphological examination of upper airway in obstructive sleep apnea. *Auris Nasus Larynx*. 2009;36(4):444–449.

47. Friedman M, Tanyeri H, La Rosa M, et al. Clinical predictors of obstructive sleep apnea. *Laryngoscope*. 1999;109(12):1901–1907.

48. Lewis M, Keramati S, Benumof JL, Berry CC. What is the best way to determine oropharyngeal classification and mandibular space length to predict difficult laryngoscopy? *Anesthesiology*. 1994;81(1):69–75.

49. Bartlett D, Ganss C, Lussi A. Basic erosive wear examination (BEWE): A new scoring system for scientific and clinical needs. *Clin Oral Investig*. 2008;12(Suppl 1):S65–S68.

50. Wilkins EM. Indices and Scoring Methods In: Clinical Practice of the Dental Hygienist. Eighth ed. Philadelphia: Lippincott Williams and Wilkins. 1999. 298 p.

51. Risk Management Series: Diagnosing and Managing the Periodontal Patient. *J Am Dent Assoc*. 1986;12–16.

52. Bergström J. Cigarette smoking as risk factor in chronic periodontal disease. *Community Dent Oral Epidemiol*. 1989;17(5):245–247.

53. Hornecker E, Muuss T, Ehrenreich H, Mausberg RF. A pilot study on the oral conditions of severely alcohol addicted persons. *J Contemp Dent Pract*. 2003;4(2):51–59.

54. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: An epidemiologic perspective. *Ann Periodontol*. 2001;6(1):99–112.

55. Taylor GW, Borgnakke WS. Periodontal disease: Associations with diabetes, glycemic control and complications. *Oral Dis*. 2008;14(3):191–203.

56. Taylor GW, Burt BA, Becker MP, et al. Severe periodontitis and risk for poor glycemic control in patients with non–insulin–dependent diabetes mellitus. J Periodontol. 1996;67(Suppl 10):1085–1093.

57. Dawes C. Salivary flow patterns and the health of hard and soft oral tissues. *J Am Dent Assoc*. 2008;139(Suppl):18S–24S.

58. Boillot A, El Halabi B, Batty GD, Rangé H, Czernichow S, Bouchard P. Education as a predictor of chronic periodontitis: A systematic review with meta–analysis population–based studies. *PLoS One*. 2011;6(7):e21508.

59. Barsh LI. Responsibilities of the dental profession in the recognition and treatment of sleep breathing disorders. *Sleep Breath*. 1997;2(2):41–44.

60. Barsh LI. Dentistry's role in the recognition and treatment of sleep–breathing disorders: The need for cooperation with the medical community. *J Calif Dent Assoc*. 1998;26(8):591–598.