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**A Cross-Sectional Study Evaluating the Risk of Obstructive Sleep Apnea in an Orthodontic
Adult Patient Population**

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**UCSF Division of Orthodontics
Class of 2013**

THESIS

Submitted in partial satisfaction of the requirements for the degree of

MASTER OF SCIENCE

in

Oral and Craniofacial Sciences

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GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO

Dedication

I would like to dedicate this work to my mom whose unconditional love and support has been the pillar of my educational achievements. Mom, as my career as a formal student concludes, I know that I couldn't have reached this point without the strength you instilled in me throughout the years. You have been my biggest inspiration and cheerleader. I love you, and I hope that one day I'll give my children a fraction of what you've given me.

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**A Cross-Sectional Study Evaluating the Risk of Obstructive Sleep Apnea in an
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Sara Asghari, DDS

ABSTRACT

Introduction

Obstructive sleep apnea (OSA) is characterized by breathing disturbances during sleep, and daytime sleepiness.¹ Prevalence of OSA has been cited as 3-33% for men and 2-9% for women.²⁻⁴ Risk factors for OSA include obesity, male gender, age, neck circumference, high blood pressure, and specific craniofacial anomalies.^{1,2,5,6} Craniofacial findings in OSA patients include an inferiorly-positioned hyoid relative to the mandibular plane, a relatively shorter maxilla and mandible, larger overjet, an increased mandibular plane angle, and an increased ANB angle.⁷⁻¹⁰ Our hypothesis is that in an adult orthodontic patient population, specific skeletal cephalometric measurements can distinguish between OSA high-risk and low-risk patients, and that there is a preventive effect of orthognathic surgery in young adulthood for developing OSA later in life.

Materials and Methods

We conducted a cross-sectional study evaluating OSA risk in adult orthodontic and orthognathic surgery patients from the UCSF Orthodontic and Oral and Maxillofacial Surgery clinics. We used a questionnaire composed of the Berlin Questionnaire (BQ),

and the Epworth Sleepiness Scale (ESS) to assess the risk of OSA. Each orthodontic subject's lateral cephalogram was digitized. Logistic regression was used to evaluate the association between the subjects' specific cephalometric measurements, and the results of the BQ and ESS.

Results

27 orthodontic subjects and 28 surgical subjects were included. In the orthodontic group, none of the female subjects and 4 of the male subjects scored 'high-risk' on the BQ. Male gender ($p < 0.05$) was found to be significantly associated with being classified as 'high-risk' by the BQ in the orthodontic patient group. No difference was found in the BQ and ESS results of the orthodontic and surgical groups. The mandibular plane to hyoid distance (MPH) was found to be moderately associated with being classified as 'high-risk' by the BQ ($p < 0.1$).

Conclusions

1. Our questionnaire serves as an excellent screening tool for orthodontic patients.
2. Prevalence of probable OSA was found to be 40% for males and in 0% of the females in this study's orthodontic patient population.
3. There is a positive association between likelihood of OSA and the following factors: male gender, and an inferiorly-positioned hyoid.

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INTRODUCTION

Background

Obstructive sleep apnea (OSA) is a condition characterized by breathing disturbances during sleep resulting in daytime sleepiness and an overall reduced quality of life.¹ OSA is differentiated from central sleep apnea by the presence of breathing effort. In OSA, breathing effort against a completely or partially collapsed airway results in multiple episodes of apneas and hypopneas during the hours of sleep. Apnea is defined as complete cessation of airflow for ≥ 10 seconds, and hypopnea is defined as a 25-30% reduction in airflow followed by a reduction in blood oxyhemoglobin saturation levels by $>4\%$ or sleep arousal. The patient is usually unaware of symptoms (*i.e.* cessation of breathing), which are typically brought to attention by a bed partner. The gold standard for diagnosis of sleep apnea is a polysomnogram (PSG) or sleep study, which is conducted in a sleep lab or at home. The PSG yields the apnea-hypopnea index (AHI), which is the sum of episodes of apneas and hypopneas per hour of sleep. Sleep apnea is categorized as mild for $AHI \geq 5$, moderate for $AHI \geq 15$, and severe for $AHI \geq 30$. While a diagnosis of Sleep-Disordered Breathing (SDB) is made based on an AHI index of 5 or more, a diagnosis of sleep apnea syndrome requires that SDB be associated with daytime hypersomnolence.¹¹

OSA Co-morbidities

OSA associated morbidities include excessive daytime sleepiness, cardiovascular disease, stroke, abnormal glucose metabolism, increased cancer mortality, and increased overall mortality.^{6,12-15}

Mortality

An 18-year mortality follow-up of the Wisconsin Sleep Cohort (n=1,522) with men and women spanning ages 30 to 60, found that the adjusted hazard ratio for all-cause mortality with severe versus no SDB independent of age, sex, and BMI was 3.8.¹² A recent study of the Wisconsin Cohort Study shows that after adjusting for age, sex, BMI, and smoking, SDB is associated with increased rate of cancer mortality. The adjusted relative hazards of cancer mortality were 1.1 for mild SDB, 2.0 for moderate SDB, and 4.8 for severe SDB.¹³

Cardiovascular Disease

OSA has been shown to be an independent risk for cardiovascular disease in adults over the age of 40.^{5,6,16} The Sleep Heart Health study found that among men, 40 to 70 years old, with an AHI \geq 30, that they are 68% more likely to develop coronary heart disease and 58% more likely to develop heart failure than those with AHI $<$ 5.⁶ An 18-year follow

up of the Wisconsin Sleep Study cohort found that cardiovascular mortality accounted for 26% of all deaths among persons without SDB at baseline and 42% of all deaths in persons with severe SDB at baseline.¹² The suggested sequence of pathophysiology of OSA is that intermittent hypoxia and sleep fragmentation lead to activation of the sympathetic nervous system, alterations in intrathoracic pressure, and decrease in stroke volume leading to hypertension and cardiovascular disease.^{6,14}

Diabetes

Although a causal relationship has not yet been proven, a growing body of evidence suggests OSA as an independent risk factor for Insulin resistance and onset of type II diabetes mellitus.¹⁴ The autonomic response to OSA also leads to an altered neuroendocrine function, release of inflammatory cytokines and, thus, altered glucose metabolism.¹⁴

Prevalence

The prevalence of OSA is found to be highly variable in the current sleep literature. It is agreed that OSA is largely under-diagnosed; as much as 90% of all those affected remain undiagnosed.^{3,17} Punjabi¹ has derived prevalence for OSA based on large population-based studies using polysomnography for diagnosis in the United States, Australia, Spain, China, Korea, and India. Based on these studies, the prevalence of OSA,

associated with daytime sleepiness is 3-7% in adult men, and 2-5% in adult women in the general population with the prevalence being higher in overweight individuals, minorities, and older individuals.¹ In a study of 602 adult male and female workers in Wisconsin, ages 30 to 60, Young found the prevalence of SDB as defined by an AHI of 5 or higher to be 9% for women and 24% for men as diagnosed by overnight polysomnography.¹¹ In the Young study, prevalence of the sleep apnea syndrome, which requires the minimal diagnosis findings of both SDB and daytime hypersomnolence, was 2% for women and 4% for men. However, since Young mentions that the hypersomnolence was likely underestimated in this study, the prevalence of OSA is also likely to be underestimated. Netzer *et al.*, found the prevalence SDB as indicated by the Berlin questionnaire to be 37.5% in a primary care patient population of 744.⁴ Levendowski *et al.*, used a similar questionnaire in a dental population of 175 men and 156 women, and found that of the 66% of the men and 28% of the women identified as high-risk, 33% of the men and 6% of the women were predicted to have moderate to severe sleep apnea based on the polysomnography results of a subgroup of 105 patients.¹⁸

Risk Factors

The main risk factors for OSA are obesity as measured by body-mass index (BMI), male sex, increased age, menopause, craniofacial abnormalities and lifestyle have been identified as risk factor for OSA.^{1,2,4,11}

Obesity and Airway

Among all risk factors, it appears that obesity, specifically weight gain, is the most important.¹ Data from the prospective Wisconsin Cohort Study over a 4-year interval shows that a 10% weight gain predicted a 32% increase in the AHI; while, a 10% weight loss predicted a 26% decrease in the AHI.¹⁹ A 10% increase in weight predicted a 6-fold increase in the odds of developing moderate-severe SDB.¹⁹ It is estimated that 58% of moderate or worse SDB (AHI \geq 15) adult cases are attributable to excess weight.²⁰ Neck circumference has been shown to be one of the most significant risk factors for OSA.^{5,7} Although, the mechanism of airway obstruction is not yet fully understood, it is thought that the primary defect in the OSA syndrome is an anatomically small or collapsible pharyngeal airway. Fogel²¹ describes the pathophysiology of OSA syndrome as follows: during wakefulness, the neuromuscular compensatory system maintains a patent airway via increased activity of the pharyngeal dilator muscles. However, the airway collapses at sleep onset due to loss of this reflex. The resulting hypoxemia causes sleep arousal, and the cycle begins again as the patient returns to sleep.²¹ One way of visually measuring the crowdedness of oropharyngeal airway is via Mallampati scores. The Mallampati score measures the crowdedness of the oropharynx during a breath hold at end tidal respiration with the mouth wide open and the tongue maximally protruded without phonation.⁷

Age, Sex and Race

OSA prevalence increases with increasing age. Community-based data from the Sleep Heart Health Study (n=5615) shows that OSA prevalence increases with age, and reaches a plateau after the age of 60 years.²² The highest prevalence is among 40-60 year old men.^{1,6,22} Among women, prevalence is highest in the post-menopausal age group.¹ The sex disparities in prevalence of OSA could be in part because OSA is clinically under-recognized in women since they tend not to report symptoms of loud snoring. Another reason, is gender differences in the anatomical properties of the upper airway between men and women. Hormonal factors influence severity of OSA symptoms as hormone replacement therapy in post-menopausal women reduces symptoms, while androgen therapy in men and women can exaggerate OSA severity.²³ In general, minority populations have a higher prevalence of OSA most likely secondary to a higher prevalence of medical conditions such as obesity, and lower socioeconomic status.¹ Population-based studies show that prevalence of OSA is comparable in Asians and Caucasians, even though obesity is lower among Asians. Differences in craniofacial structures are considered as the etiologic factors for increased risk and severity among Asians.⁷

Craniofacial Anatomy

In studying the craniofacial anatomy in the Caucasian and Asian OSA samples, Lam found that after controlling for ethnicity and obesity, a crowded posterior oropharynx, as indicated by the Mallampati score and a steep thyromental angle, are the most

important predictors for OSA.⁷ Lam found that Chinese patients with OSA have a more crowded upper airway and relative retrognathia compared with their Caucasian counterparts.⁷ Several hard and soft-tissue craniofacial features such as maxillary and mandibular retroposition, tonsillar hypertrophy, enlarged tongue or soft palate, and decreased posterior airway space (PAS) are associated with OSA.²⁴ Certain craniofacial syndromes such as the Pierre Robin sequence, Treacher-Collins, fragile-X, Prader-Willi, and Marphans syndrome are associated with OSA. A study of these syndromes is valuable for understanding the pathophysiology of OSA. In studying OSA in Marfan's syndrome patients, Cistulli *et al.*, found that 13 out of the 15 patients had OSA, and presented with distinct craniofacial patterns.²⁵ Significant abnormalities were bimaxillary retrusion, a reduced maxillary length, an increased total anterior face height, a long lower face height, an obtuse gonial angle, a steep mandibular plane, a reduced posterior nasal airway height, a reduced posterior airway space, and an increased distance from the mandibular plane to the hyoid bone.

Radiographic Imaging Techniques and OSA

Airway Analysis

B.H. Broadbent introduced lateral cephalometry in 1931 for orthodontic diagnostic use in USA. Cephalometric analyses were developed for angular and linear measurements of hard tissue structures. Lateral cephalometry was later used for evaluation of airway

soft tissue, specifically, tonsils and adenoids in orthodontic patients. Since it involves low cost and low radiation dosage, lateral cephalometry has been used as an efficient screening tool for airway analysis in the sagittal plane. However, the main limitation of lateral cephalometry is that it provides no information on the transverse dimensions of the airway. With the advent of 3-D imaging techniques and the popularization of use of cone beam computed tomography (CBCT), the airway can be analyzed in axial as well as sagittal slices; thus, providing information on the cross-sectional area of the airway. A common limitation of both lateral cephalometry and CBCT imaging techniques is that the patient is positioned upright in the machine, which may not be an accurate representation of the airway in the supine position during sleep.

Cephalometric Findings in the OSA Patient

Several studies have found specific cephalometric patterns in OSA patients. In a systematic review, the mandibular body length (Go-Gn) was found to be significantly associated (*i.e.*, shorter) with severity of OSA.⁹ When compared with controls, OSA patients were found to have both a shorter maxilla and mandible⁸. An elongated soft palate, inferiorly positioned hyoid bone, and decreased posterior airway space were found in both Asian and Caucasian OSAS patients.²⁶ An inferiorly positioned hyoid is thought to be an adaptation to accommodate an inferiorly postured tongue to clear the airway in OSA patients.

Treatment Modalities

Excess adipose tissue, a long soft palate, a large tongue, or a short mandible that forces the tongue to fall back into the airway have been proposed as possible mechanisms to induce OSA. Thus, therapies have been aimed at removing the soft tissue obstruction, and clearing the airway during sleep.

Mild OSA

Life style modifications such as weight loss and reduction of alcohol consumption and smoking, and avoidance of supine position are recommended for treatment of mild sleep apnea.

Moderate OSA

The first line of treatment for moderate to severe OSA is Continuous Positive Airway Pressure (CPAP). Although difficult to tolerate for some patients, CPAP has been shown to be effective in reducing AHI. Mandibular anterior repositioning appliances have also been moderately effective in reducing OSA symptoms at the cost of creating occlusal changes.^{27,28} These intraoral appliances have been suggested as good treatment for patients who cannot tolerate CPAP.²⁷

Severe OSA

Surgical therapies such as uvulopalatopharyngoplasty (UPPP) and genioglossal advancement have been moderately successful. To date, the most effective surgical therapy for OSA is maxillomandibular advancement (MMA). A CBCT airway analysis of class II patients (n=10) who underwent MMA reveals that posterior airway space (PAS) significantly increased by an average of 34%, and each patient reported a subjective improvement of breathing.²⁹

Study Significance

As much as 90% of OSA patients remain undiagnosed. To date, there are no studies evaluating the risk of OSA in an orthodontic adult population. There are also no studies investigating a potential preventive effect of orthognathic surgery in young adulthood for developing OSA later on in life. Since orthodontic and orthognathic treatment can significantly alter the craniofacial pattern, orthodontic treatment planning should include a consideration of patient's current and future OSA risk status. Thus, incorporating sleep apnea screening into orthodontics would be a valuable service to patients.

Hypothesis

1. In an adult orthodontic population \geq age 40, specific skeletal cephalometric measurements can distinguish between high-risk and low-risk patients as classified by the BQ and ESS scales.
2. A survey of patients who have had orthognathic surgery five or more years ago reveals that this group has a lower prevalence for probable OSA than their orthodontic patient counterparts.

Study Aims

1. To determine the underlying probable prevalence for OSA in an orthodontic adult population via the BQ and ESS scales
2. To compare the prevalence of OSA between the orthodontic and orthognathic surgery populations
3. To identify cephalometric measurements associated with high-risk OSA patients as classified by the BQ and ESS

METHODS AND MATERIALS

Subject Selection

This is a cross-sectional study evaluating risk of OSA in adult orthodontic and orthognathic surgery patients from the UCSF Orthodontic and Oral and Maxillofacial Surgery clinics. This study was approved by the UCSF Committee of Human Research (CHR; IRB number: 11-06934). The inclusion criteria were patients over the age of 40 currently undergoing active orthodontic treatment or were treated in the past 5 years. Also, included, were patients who had orthognathic surgery for the treatment of malocclusion at least 5 years ago. The patients were categorized into two groups: the orthodontic group and the surgery group. All orthodontic patients must have complete initial records including a lateral cephalogram. The exclusion criteria were patients with a craniofacial anomaly or those who had orthognathic surgery for the treatment of OSA, and those who did not speak or understand fluent English.

Orthodontic patients coming in for routine orthodontic visits were recruited by their orthodontic resident provider. Upon agreement to participate, subjects were handed the recruitment letter, consent form, and the study questionnaire. After review of the consent documents, the subjects were asked to complete a 3-page OSA risk assessment questionnaire. While the patients were given the option of taking the questionnaire home, nearly all orthodontic group patients completed the questionnaire chair-side.

Surgical patients were contacted via telephone on behalf of the Director of the UCSF Oral and Maxillofacial Surgery clinic, and upon consent to participate, were asked to complete the sleep apnea risk assessment questionnaire over the telephone. The

patients were also given the option of having a copy of the questionnaire mailed and returned to the clinic at a later time.

Questionnaire

We used a three-page questionnaire to assess the risk of OSA in the UCSF adult orthodontic and orthognathic surgery patient populations. Although, we recognized that an overnight polysomnogram (PSG) is the gold standard for diagnosis of OSA, its use would be impractical for our study. Therefore, we used a questionnaire as a screening tool for identifying patients with likelihood of OSA. We aimed at keeping the questionnaire short and concise so that it could be easily completed by the patient chair-side. The questionnaire is composed of 3 parts: 1) the Berlin questionnaire; 2) patient demographics and health history; and 3) the Epworth Sleepiness Scale (ESS).

The Berlin questionnaire (BQ) on page 1 of the questionnaire (Figure 1) assesses the risk of sleep disordered breathing (SDB) with high sensitivity and specificity.^{4,30} The BQ was developed by the Conference on Sleep in Primary Care in April 1996 in Berlin, Germany which included 120 physicians.⁴ The BQ uses three categories to predict the likelihood of OSA: 1) snoring, 2) daytime fatigue, and 3) hypertension and obesity. If the subject's answers to at least 2 of the 3 above categories fall in the 'high-risk' classification, the subject is placed in the 'high-risk' group for OSA. Thus, based on the results of the Berlin questionnaire, all subjects were divided into 2 groups: high-risk and low-risk.

We included a series of patient demographics and health history including cardiovascular history on page 2 of the questionnaire (Figure 2). We were particularly interested in cardiovascular health, given that OSA has been identified as an independent risk factor for cardiovascular disease in adults over the age of 40.^{2,5,6} Included on this page were also questions regarding history of orthognathic surgery and orthodontic treatment. We did not include a question on neck circumference because as a previous study shows, we made the assumption that most patients would not know this measurement.

The ESS was included on the third page of the questionnaire (Figure 3). The ESS was developed by Dr. Murray Johns in 1990 at the Epworth Hospital in Melbourne, Australia. It is a measure of day-time sleepiness. The ESS asks patients to assign a score of 0 to 3 based on their likelihood of falling asleep in 8 commonly-encountered daily life situations. The total score can range from 0 to 24. A score of 10 or more is indicative of excessive daytime sleepiness or hypersomnolence, and is considered to be associated with OSA.³¹ Based on the results of the ESS, the subjects were divided into high-risk (ESS \geq 10) and low-risk (ESS < 10) groups.

The validity of the BQ and ESS as screening tools for OSA has been well-established in the medical literature. In a recent study, Mungan *et al.*,³² used a combination of ESS and BQ to identify patients at risk for OSA, and found positive correlation between BQ's high risk group as well as the ESS scores and the incidence of post-op atrial fibrillation (POAF).

UCSF Sleep Health Study

Berlin questionnaire

SLEEP EVALUATION

Patient _____

1. Complete the following:

Height _____ age _____
Weight _____ male/female _____

2. Do you snore?

- Yes
 No
 Don't know

If you snore:

3. Your snoring is?

- Slightly louder than breathing
 As loud as talking
 Louder than talking
 Very loud. Can be heard in adjacent rooms

4. How often do you snore?

- Nearly every day
 3-4 times a week
 1-2 times a week
 1-2 times a month
 Never or nearly never

5. Has your snoring ever bothered other people?

- Yes
 No

6. Has anyone noticed that you quit breathing during your sleep?

- Nearly every day
 3-4 times a week
 1-2 times a week
 1-2 times a month
 Never or nearly never

7. How often do you feel tired or fatigued after your sleep?

- Nearly every day
 3-4 times a week
 1-2 times a week
 1-2 times a month
 Never or nearly never

8. During your wake time, do you feel tired, fatigued or not wake up to par?

- Nearly every day
 3-4 times a week
 1-2 times a week
 1-2 times a month
 Never or nearly never

9. Have you ever nodded off or fallen asleep while driving a vehicle?

- Yes
 No

If yes, how often does it occur?

- Nearly every day
 3-4 times a week
 1-2 times a week
 1-2 times a month
 Never or nearly never

10. Do you have high blood pressure?

- Yes
 No
 Don't know

BMI =

Page 1 of 3

Figure 1: The Berlin Questionnaire. Category 1: questions 2 through 6. Category 2: questions 7 through 9. Category 3: question 10. Any answer within the box is a positive response, and any 2 positive responses in a category. Category 1 is positive with 2 or more positive responses. Category 2 is positive with 2 or more positive responses. Category 3 is positive with 1 or more positive responses or BMI>30.

UCSF Sleep Health Study

1. Patient demographics

Age _____

Gender

- Male
- female

Ethnicity

- Caucasian
- Asian
- Latin-American
- African-American

Employment status

- unemployed
- employed
- retired

Education level

- high school graduate
- college degree

2. Patient medical history

Please list any medical problems

Please list your current medications (indicate none if you're not taking any)

Recent Weight loss/gain in the past 5yrs

- Increased
- Decreased
- no change

Do you or have you had any of the following?

High BP

- Yes
- No

Chest pain or angina

- Yes
- No

Heart attack

- Yes
- No

Recent heart-related hospitalization

- Yes
- No

Coronary angioplasty/stent

- Yes
- No

Other cardiac problems including arrhythmias

- Yes _____
- No

3. Surgical history

Please list any surgeries that you've had

Have you had orthognathic/jaw surgery?

- Yes
When? _____
- No

If you've had jaw surgery, please describe the type of surgery

- Single jaw
- Two-jaw
- TMJ

What type of jaw surgery (check all that apply)

- Maxilla
 - Advancement
 - Widened
 - Posterior impaction (open bite correction)
- Mandible
 - Setback
 - Advancement
 - Rotation (asymmetry correction)

4. Orthodontic treatment history

Have you had orthodontic treatment?

- Yes
When? _____
- No

Did you orthodontic treatment involve extraction of premolars (adult teeth)?

- Yes
- No

Figure 2: Patient demographics and health history

UCSF Sleep Health Study

The Epworth Sleepiness Scale (ESS)

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How likely are you to doze off or fall asleep in the following situations in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things, try to work out how they would have affected you.

Use the following scale to choose the most appropriate number for each situation:

0 = would never doze

1 = slight chance of dozing

2 = moderate chance dozing

3 = high chance of dozing

It is important that you answer each question as best you can

<u>Situation</u>	<u>Chance of dozing (0-3)</u>
Sitting & reading	----
Watching TV	----
Sitting inactive in a public place (e.g. a theatre or a meeting)	----
As a passenger in a car for an hour without a break	----
Lying down to rest in the afternoon while circumstances permit	----
Sitting and talking to someone	----
Sitting quietly after a lunch without alcohol	----
In a car, while stopped for a few minutes in the traffic	----

Thank you for your cooperation☺

Page 3 of 3

Figure 3: The Epworth Sleepiness Scale (ESS). The total score ranges from 0 (the least daytime hypersomnolence) to 24 (the most daytime hypersomnolence). A total score of ≥ 10 is predictive for OSA.

Cephalometric analysis

All subjects' had initial lateral head films which were either conventional cephalogram or CBCT-generated. All head films were calibrated to resolve any magnification errors, and brightness and contrast were adjusted for best landmark identification. All head films were then traced and digitized via Total Interactive Orthodontic Planning System (TIOPS, Aarhus, Denmark).

'AirWayMes', a custom-made analysis, was used for measurements. This regimen includes 11 landmarks to measure the anteroposterior dimension of the airway in the nasopharynx, oropharynx, and hypopharynx adopted from Solow (Figure 4).¹⁰ A list of landmarks is described in Table 1. A total of 22 measurements (14 hard tissue and 8 soft tissue) were made (Table 2).

Table 1: Definition of cephalometric landmarks

Lateral Landmarks	Eur Amer	Definitions
Nasion	n	Antermost point of the frontonasal suture
Sella Anterior	sa	Intersection of the anterior contour of sella turcica and the NSL
Sella	s	Center of Sella Turcica
Basion	ba	Most postero-inferior point on the anterior margin of foramen magnum

Nasal Apex	na	Tip of nasal bone
Articulare	ar	Intersection of the external contour of the cranial base and the posterior contour of the condyle
RamusLine Sup	rls	Deepest point of the posterior contour of the mandibular ramus
RamusLineInf	rli	Tangent point to the posterior contour of the mandibular ramus through the ar
Gonion	go	Intersection of the gonial contour and a line dividing the angle between the ML and RL
Mandibular Line Post	mlp	Tangent point to the inferior contour of the mandible through gn
Antegonion	ag	Superiormost point of the antegonial notch in relation to ML
Mandibular Line Ant	mlla	Tangent point of the inferior contour of the mandible through mlp
Supramentale	sm Bpoint	Posteriormost point of the anterior contour of the mandibular symphysis/lower alveolar process
Suprapogonian	spg	Tangent point to the anterior contour of the mandibular symphysis through sm
Pogonion	pg	Tangent point the anterior contour of the mandibular symphysis through n
Prognathion	pgn	Point on the mandibular symphysis at the greatest distance from the cd
Gnathion	gn	Inferiormost point of the mandibular symphysis
Symphyseon	sym	Posteriormost point of the mandibular symphysis
Mandibular Ref 1	ma 1	Mandibular reference point 1 - Anterior

Mandibular Ref 2	ma 2	Mandibular reference point 2 – Posterior ma1/ma2 should be placed on a line through spg
Pterygomaxillare	pm PNS	Intersection point of the nasal floor and posterior contour of the maxilla
Palation	pal	Point where the asi meets the palatal contour when ILs is rotated with center in isi
Subspinale	ss Apoint	Posteriormost point of the anterior contour of the maxilla / the upper alveolar process
Spinalpoint	sp ANS	Apex of anterior nasal spine
Maxillar Ref 1	mx1	Maxilla Reference point 1 - Anterior
Maxillar Ref 2	mx 2	Maxilla Reference point 2 – Posterior: mx1/mx2 should be placed on the line sp-sa
Incisal Inf Incisor	iii	Midpoint of the incisal edge of the most prominent lower incisor
Apex Inf Incisor	aii	Apex of the lower incisor defined by the apex point of the tooth template
Mesial Inf Molar	mim	Mesial contact point of the average lower molar
Root Inf Molar	rim	Root point of the lower molar defined by the root point of the tooth template
Incisal Sup Incisor	isi	Midpoint of the incisal edge of the most prominent upper incisor
Apex Sup Incisor	asi	Apex of the upper incisor defined by the apex point of the tooth template
Mesial Sup Molar	msm	Mesial contact point of the average upper molar

Root Sup Molar	rsm	Root point of the upper molar defined by the root point of the tooth template
BicuspidOccl Point	pop	Cusp tip of the first lower premolar
Frontal Tangent	ft	Frontal tangent point of NFL
SupraGlbellareSoft	sgs	Deepest point of the soft tissue fossa supraglabellaris
Glabella Soft	gs	Anteriormost point on the soft tissue glabella
Nasion Soft	ns	Deepest point in the soft tissue fronto-nasal curvature
DorsumNasi	dn	Point located at the greatest convexity or concavity of the dorsum nasi
Upper Nasal Tangent	rnt	Nasal tangent point of NFL
Pronasale	prn	Prominent most point on the apex of the nose
Lower Nasal Tangent	lnt	Nasal tangent point of NCL-E line
Nasal Septum Tangent	nst	Anterior tangent point of the tangent to the nasal septum through sn
Subnasale	sn	Deepest point of the naso-labial curvature
subspinaleSoft	sss	Dorsalmost point of the upper lip contour
Labrale Sup	ls	Prominent most point on the prolabium of the upper lip
Labrale Sup Tangent	lst	Tangent point to the prolabium of a tangent parallel to the line ls-sts
Stomion Sup	sts	Most antero-inferior point on the prolabium of the upper lip
Stomion Inf	sti	Most antero-superior point on the prolabium of the lower lip
Labrale Inf Tangent	lit	Tangent point to the prolabium of a tangent parallel to

		the line li-sti
Labrale Inf	li	Prominent most point on the prolabium of the lower lip
Lower Labial Tangent	lit	Superior tangent point to the lower lip through sms
Submentale Soft	sms	Deepest point of the mento-labial sulcus
Pogonion Soft	pgs	Tangent point to the anterior contour of the chin through ns
Chin Tangent	ct	Tangent point to the chin of the NCL-E line
Prognathion Soft	pns	Soft tissue point overlying pgn
Gnation Soft	gns	Soft tissue point overlying gn
Submentale	sme	Deepest point in the submental-neck curvature
Hyoideon	hy	Most antero-superior point of the corpus of the hyoid bone
Tuber Maxillare	tu	Posteriormost point of the maxillary tuberosity
Adenoid Prominence 1	ad1	Point at the shortest distance from tu at the pharyngeal adenoid prominence
Adenoid Prominence 2	ad2	Point at the shortest distance from pm at the pharyngeal adenoid prominence
Adenoid Prominence 3	ad3	Point at the intersection of pharyngeal adenoid prominence and line from pm to ba
Post Vellacula epiglottis	pve	The point on the posterior pharyngeal wall closest to ve
Post Uvula	puv	The point on the posterior pharyngeal wall closest to uv
Post Radis Linguae	prl	The point on the posterior pharyngeal wall closest to

	rl	
Post Velum Palati	pva	The point on the posterior pharyngeal wall closest to va
Vallecula epiglottis	va	The most inferior point on the valley of the epiglottis
Radix linguae	rl	The point on the root of the tongue closest to the dorsal pharyngeal wall
uvula	uv	The tip of the uvula of the soft palate
Velum palati	ve	The point on the soft palate closest to the dorsal pharyngeal wall

Table 2: List of cephalometric measurements

Measurement	Definition
SNA	Angle between S-N and N-A
SNB	Angle between S-N and N-B
ANB	Angle between N-A and N-B
OJ	Horizontal distance between labial surface of mandibular incisor to the incisal edge of maxillary incisor
OB	Vertical distance between incisal edge of mandibular incisor to the incisal edge of maxillary incisor
N-S-Ar	Angle between S-N and S-Ar, cranial base angle
N-S-Ba	Angle between S-N and S-Ba, cranial base angle
NSL/OPT	Angle between S-N and tangent line of distosuperior point and distoinferior point of C2
PP/SN	Angle between palatal plane and S-N

MP/SN	Angle between mandibular plane and S-N
PP/MP	Angle between palatal plane and mandibular plane
U1/PP	Maxillary incisor angulation relative to palatal plane
L1/MP	Mandibular incisor angulation relative to the mandibular plane
MP-H	Distance between hyoid bone and a perpendicular to the mandibular plane
Soft palate length (PNS-uv)	Distance between posterior nasal spine to uvula
Airway 1 (tu-ad1)	Uppermost airway dimension in nasopharynx
Airway 2 (pm-ad2)	Middle airway dimension in nasopharynx
Airway 3 (pm-ad3)	The most inferior airway dimension in nasopharynx
Airway 4 (ve-pve)	The most constricted airway dimension behind the soft palate
Airway 5 (uv-puv)	The most constricted airway dimension between uvula and posterior pharyngeal wall
Airway 6 (rl-prl)	The most constricted airway dimension behind the tongue
Airway 7 (va-pva)	The most constricted airway dimension between vallecula epiglottis and posterior pharyngeal wall

Statistical analysis

The Stata (StataCorp LP, College Station TX) and Microsoft Excel softwares (Microsoft, Redmond, WA) were used for statistical analysis and generation of figures and graphs.

The data were entered onto a Microsoft Excel spread sheath and then imported into

Stata for analysis. A logistic regression model was used to evaluate the association between the subjects' specific cephalometric measurements and the results of the BQ and ESS.

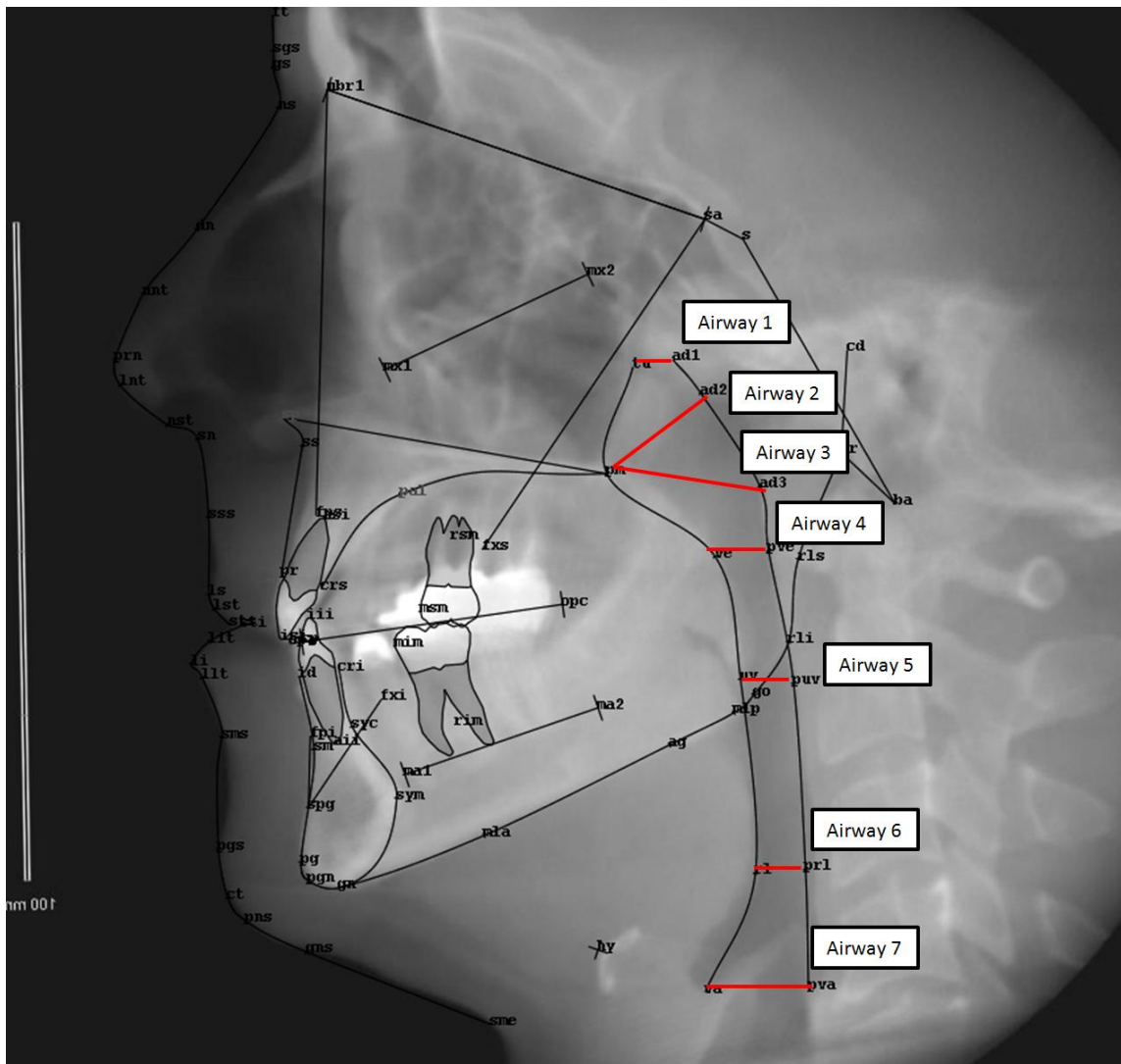


Figure 4: Cephalometric Analysis. Example of a cephalogram generated from a CBCT scan of a 'high-risk' male orthodontic patient. The ceph was imported into TIOPS, traced and digitized. The measurements were made by the 'AirWayMes' ysis. Digitized are 72 landmarks and 22 measurements including 7 airway measurements.

RESULTS

A total of 27 orthodontic subjects (13 females, 10 males), and 28 (13 females, 15 males) surgical subjects completed the questionnaire. The survey data for the orthodontic and surgical groups are shown in Tables 3 and 4, respectively. The mean data for the orthodontic and surgical groups, males and females, are shown in Tables 5 through 10.

In the orthodontic group, none of the female subjects and 4 of the male subjects scored high-risk on the BQ. The Fisher's exact test indicates ($p= 0.012$) that male sex is significantly associated with a high-risk score on the BQ in the orthodontic patient group.

Table 3. Orthodontic subjects raw data (n=27)

Subject	Age	BMI	Sex	Berlin	ESS	CV
1	42	28.9	1	1	5	1
2	63	27	1	1	8	1
3	42	24.7	1	0	9	0
4	45	32.6	1	1	4	1
5	43	25.7	1	0	4	1
6	61	20.6	0	0	10	0
7	59	18.9	0	0	4	0

8	60	25.8	0	0	3	0
9	49	23.4	0	0	7	0
10	45	29	0	0	0	0
11	60	22.7	1	0	3	0
12	55	23.3	1	0	16	0
13	48	25.6	0	0	18	0
14	69	26.4	1	0	10	0
15	44	20.6	0	0	3	0
16	49	21.9	1	0	4	0
17	60	25.1	0	0	1	0
18	63	19.2	1	1	2	1
19	47	24.3	0	0	5	0
20	47	26.6	0	0	9	0
21	44	21.9	0	0	2	0
22	51	23.8	0	0	1	0
23	43	20.8	0	0	3	0
24	61	23.6	0	0	1	0
25	42	25	0	0	4	0
26	60	24.6	0	0	1	0

27	52	22.3	0	0	2	0
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Sex: 1 = male, 0 = female, Berlin: 1 = high-risk, 0 = low-risk, CV (cardiovascular): 1 = yes, 0 = no

Table 4: Surgical subjects raw data (n=28)

Subject	Age	Age at surgery	BMI	Sex	Berlin	ESS	CV	Treatment
1	31	21	22.4	1	0	8	0	Mx adv, md setback, post impac
2	39	19	24.4	1	0	4	0	Mx adv, exp, md setback
3	56	42	19.6	0	0	1	0	Md adv
4	58	45	27.1	0	0	4	1	Md adv
5	52	43	45.4	0	1	7	1	Md adv
6	31	22	25.1	1	0	0	0	Mx adv
7	26	18	18.8	1	0	6	0	Mx post impac
8	28	17	23.4	0	1	17	0	Mx post impac
9	36	20	21.3	1	0	4	0	Md setback, Mx adv
10	40	19	17.9	1		5	0	Mx adv
11	51	28	23.7	0		1	0	SARPE
12	33	24	22.9	1		9	0	Mx adv
13	63	36	23.7	0		1	0	Md adv

14	44	19	20.5	0		4	0	Md adv
15	42	21	26.6	0		5	1	Md setback
16	32	21	24.4	1	0	5	0	Md setback, Mx adv
17	28	17	24.3	0	0	5	0	Md adv
18	40	29.0	23.8	1	0	1	0	MMA
19	43	32	26.4	1	0	2	0	SARPE
20	35	24	26.1	0	0	1	0	Asymmetry Correction
21	31	22	21.6	1	0	1	0	Mx adv
22	64	55	25.1	0	1	3	1	Md adv
23	51	41	28.6	1	0	0	0	Md setback
24	62	52	30.8	1	0	3	1	Mx adv
25	50	41	38.7	1	1	7	1	Mx adv
26	47	31	16.0	1	0	3	0	SARPE
27	41	20	20.4	0	0	10	0	Md setback
28	49	20	23.9	0	0	0	0	Md adv

Sex: 1 = male, 0 = female, Berlin: 1 = high-risk, 0 = low-risk, CV (cardiovascular): 1 = yes, 0 = no. Md: mandible, Mx: maxilla, adv: advancement, exp: expansion, SARPE: surgically-assisted rapid palatal expansion, MMA: maxilla-mandibular advancement, post impact: posterior impaction.

Table 5: All orthodontic subjects (n=27)

Variable	Mean	SD	Range
Age (years)	52.0	8.3	42-69
BMI	24.2	3.1	18.9-32.6
ESS	5.1	4.5	0-18

Table 6: Male orthodontic subjects (n=10)

Variable	Mean	SD	Range
Age	53.1	10.2	42-69
BMI	25.2	3.8	19.2-32.6
ESS	6.5	4.3	2-16

Table 7: Female Orthodontic subjects (n=17)

Variable	Mean	SD	Range
Age	51.3	7.2	42-61
BMI	23.6	2.6	18.9-29
ESS	4.3	4.5	0-18

Table 8: All surgical subject (n=28)

Variable	Mean	SD	Range
Age (years)	43.0	11.3	26-64
BMI	24.7	5.9	16-45.4

ESS	8.0	3.7	0-17
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Table 9: Male surgical subjects (n=15)

Variable	Mean	SD	Range
Age (years)	39.5	9.7	26-62
BMI	24.2	5.6	16-38.7
ESS	4.0	2.8	0-9

Table 10: Female surgical subjects (n=13)

Variable	Mean	SD	Range
Age (years)	47	12	28-64
BMI	25.4	6.5	19.6-45.4
ESS	4.5	4.7	0-17

The cephalometric measurements of the orthodontic subjects are shown in Table 11. Logistic regression analysis of results of the BQ was compared against mandibular body length (Go-Gn), and mandibular plane to hyoid distance (MPH). An increase in MPH was found to be moderately associated ($P < 0.1$) with 'high-risk' classification by the BQ (Figure 5). In comparison of MPH with ESS scores, a weak association was found. Figure 6 shows this association before and after the elimination of an outlier (subject 13).

Table 11: Cephalometric measurements of orthodontic subjects (n=27)

Variable	Mean	SD	Range	Population norms*
SNA	83.0	4.5	72.9-93.9	82.0
SNB	79.6	4.5	71.5-91.8	80.0
ANB	3.4	2.5	-3.2-7	2.0
L1-MP	90.6	9.1	68.9-107.1	95.0
U1-PP	109.7	10.4	95.1-134.4	110.0
PP-MP	24.5	7.8	6.5-44.6	25.0
SN-MP	31.0	8.9	11.2-49.6	33.0
OJ	3.7	2.7	-4-10.5	2.5
OB	3.6	2.3	0-8.7	2.5
Go-Gn	70.6	5.9	60.3-83.1	75.0
MPH	13.7	5.4	3.8-24.5	N/A
ANS-PNS	53.9	6.1	42.0-64.9	N/A
SN-Ba	125.0	7.4	112.3-139.7	N/A
PNS-UV	34.4	3.5	28.0-40.0	N/A
Airway 1	8.1	2.5	5.5-18.8	N/A
Airway 3	23.0	3.5	16.2-31.0	N/A
Airway 4	8.8	2.9	3.9-15.1	N/A
Airway 5	8.7	2.6	4.2-15.9	N/A

Airway 6	9.3	3.6	3.5-19.7	N/A
Airway 7	15.1	4.7	8.4-32.0	N/A

*Population norms adopted from Dolphin Imaging (Patterson Dental Supply, Inc.)

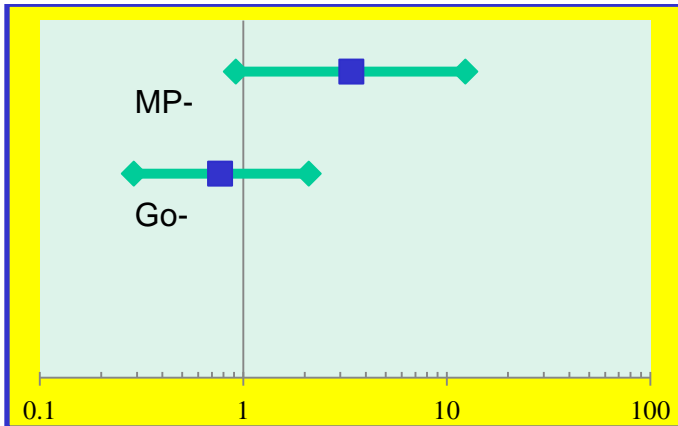


Figure 5: Forest plot for association between subjects ‘high-risk’ classification by the BQ and the cephalometric measurements of MPH and GoO-Gn. For every 5 mm increase from the mean (13.7 mm) in hyoid to mandibular plane distance, there is a 3.4 times increase in odds of being classified as OSA “high-risk” by the BQ. Note the weak, but negative association of mandibular body length (Go-Gn) with results of BQ. MP-H: $p = 0.070$, $OR = 3.0$, $CI: 0.92- 12.35$. Go-Gn: $p = 0.611$, $OR = 0.77$, $CI: 0.29-2.07$

We investigated correlations of airway cephalometric measurements of the orthodontic patients with BMI and ESS. Figure 6 (A through G) displays linear correlation graphs between ESS, BMI and airway measurements 1 through 7 with the corresponding r values. These variables do not seem to be related except for the lower airway measurements to each other (Figure 7).

In the surgical group, the Berlin questionnaire was not completed for subjects 10 through 15 because in the original design of this study, only the ESS scales was included in the survey.

A comparison of ESS results of the surgical and orthodontic groups are shown in Tables 12 and 13. Table 12 shows a comparison of ESS results of all subjects included. Table 13 shows the ESS results of the two groups with the inclusion of only 18 surgical subjects (#3-6, 10-14, 17, 19, 21-26, 28) who had maxillary or mandibular advancement or SARPE (Surgically Assisted Rapid Palatal Expansion). Subjects who had mandibular set-back, asymmetry correction, maxillary posterior impaction, or MMA (Maxillomandibular Advancement) for treatment of OSA were excluded from this analysis.

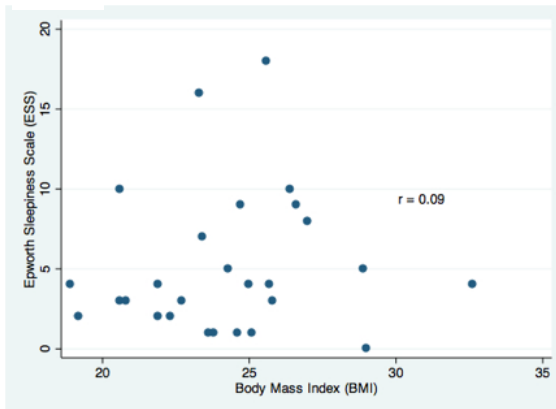
Table 12: Descriptive statistics of surgical subjects (n=28) and orthodontic subjects (n=27)

Group	Age	BMI	ESS
Orthodontic	52 ± 8	24 ± 3	5 ± 5
Surgical	43 ± 11	25 ± 6	8 ± 4

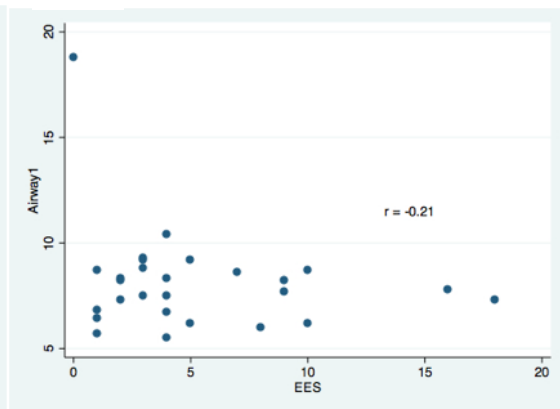
Table 13: Descriptive statistics of surgical advancement procedure subjects (n=18) and orthodontic subjects (n=27).

Group	Age	BMI	ESS
Orthodontic	52 ± 8	24 ± 3	5 ± 5
Surgical	47 ± 11	26 ± 7	3 ± 3

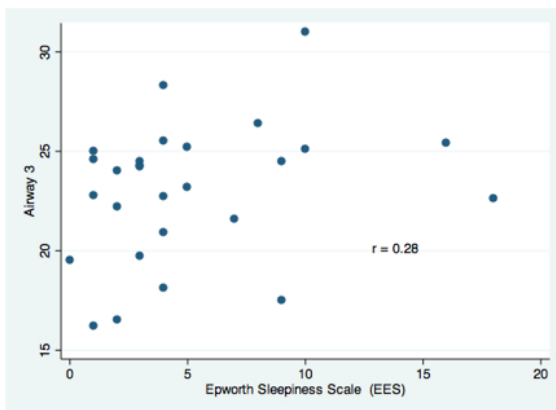
A)



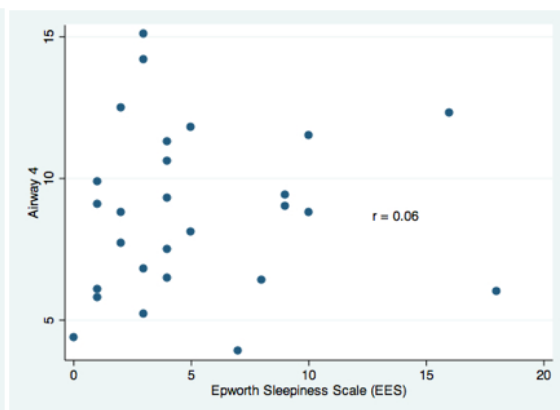
B)



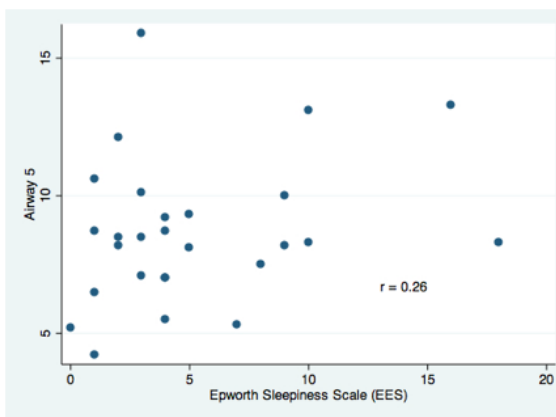
C)



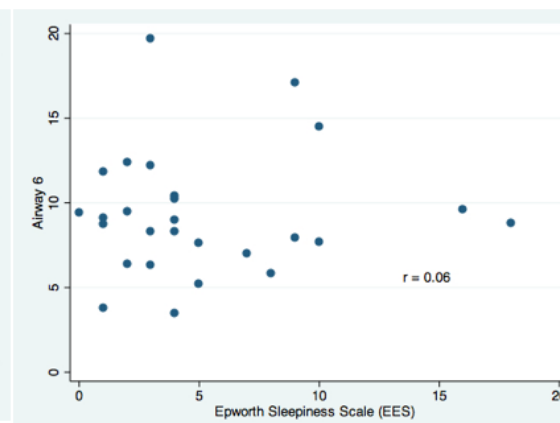
D)



E)



F)



G)

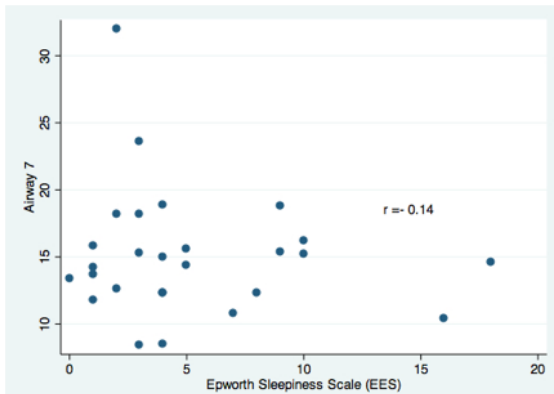


Figure 6 (A-G): Linear correlation graphs of ESS, BMI, Airway 1-7. None of the relationships displayed by graphs A through G are significant.

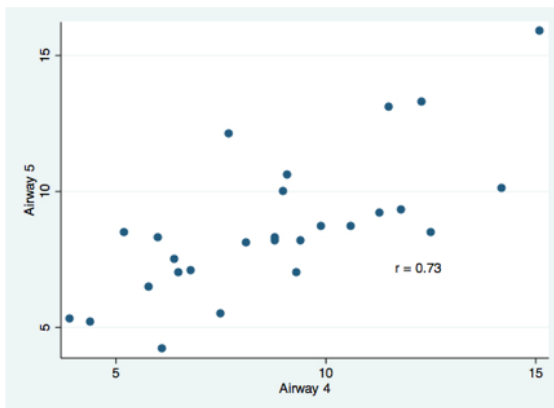


Figure 7: Linear correlation graphs Airway 1-7 with each other. Airway 4 to Airway 5 ($r = 0.73$), Airway 5 to Airway 6 ($r = 0.60$), Airway 6 to Airway 7 ($r = 0.65$) are significantly correlated.

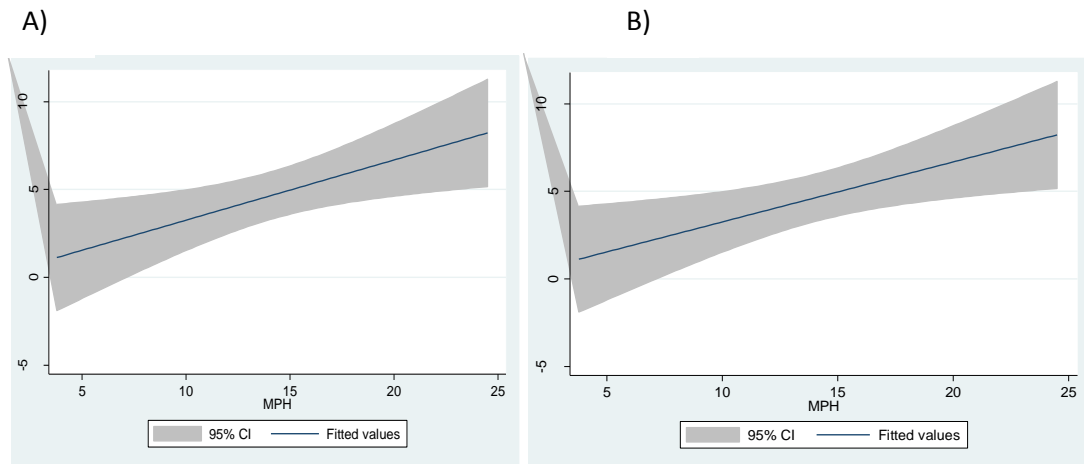


Figure 8: A) Mandibular plane to hyoid distance VS. ESS scores (all orthodontic subjects n=27). B) same plot after elimination of outlier subject 13.

DISCUSSION

In the orthodontic group, prevalence of probable OSA as classified by the BQ was found to be in 40% of the males with a mean age of 53 years. None of the female subjects in our orthodontic population were found to be at high risk for OSA as classified by the BQ. In investigating OSA prevalence in a San Diego dental population (175 men and 156 women), Levendowski *et al.*¹⁸ used the Apnea Risk Evaluation Survey (ARES™), which includes the ESS and questions about snoring, and cardiovascular disease, similar to BQ. They confirmed the results of the survey with a sleep study in a subset of their study population. Levendowski *et al.* identified 67% of the men and 28% of the women to have high risk of at least mild OSA. The prevalence of OSA in the Levendowski dental

population is higher than in our orthodontic patient population. The BMI of the Levendowski's dental population was 28 and 25 for males and females respectively, indicating that that specific population is more overweight than our orthodontic population with respective BMI's of 25 and 24. We hypothesize that the orthodontic patient population is relatively more health-conscious than the general dental population.

In the orthodontic group, two male and two female subjects had an ESS score ≥ 10 ; none of who, however, had a 'high-risk' BQ result. In the orthodontic group, the subjects who were classified as 'high-risk' by the BQ did not have an ESS score ≥ 10 , while those who had an ESS score ≥ 10 , were classified as 'low-risk' by the BQ. This result could be explained by the fact that ESS is a measure of day-time sleepiness which could be caused by OSA and other sleep disorders, while the BQ more specifically targets OSA. It has also been hypothesized that females generally, tend to under-report snoring symptoms, while males have a tendency to respond to snoring questions more truthfully.²³

In our orthodontic population, male gender was shown to be significantly associated with being classified as 'high-risk' by the BQ ($P < 0.05$). This finding is consistent with the current literature identifying male gender as a significant risk factor for OSA.^{1,2,5,6,16}

Although the BQ and ESS are only capable of identifying at-risk individuals for OSA rather than diagnosis, they are efficient screening tools to use in the clinical orthodontic setting. Our study has shown that the BQ and ESS can be implemented into orthodontic

patient evaluation as a useful screening tool for OSA. Once high risk individuals are identified, appropriate referral and subsequent treatment planning considerations should be made by the orthodontist.

In investigation of cephalometric measurements associated with risk of OSA, we found that increased mandibular plane to hyoid distance is positively associated with high OSA risk with moderate statistical significance ($p < 0.1$). The estimated odds ratio of 3.4 was not statistically significant ($p=0.070$), but was large enough to possibly be considered important. The confidence interval argues against a substantial beneficial effect (at best 0.91), but leaves open the possibility of a clearly substantial harmful effect (up to 12). This finding is consistent with other studies which have found a significantly inferiorly-positioned hyoid bone relative to the mandible in OSA patients.^{26,33} These studies used patients with confirmed diagnosis of OSA to arrive at the findings. We found a positive association between MP-H distance and likelihood of OSA, which consistent with current literature, is interesting. This finding validates the accuracy of the BQ in predicting OSA. It is hypothesized in the literature that the inferior position of the hyoid bone is a consequence of the adaptive inferior positioning of the tongue to clear the airway in OSA patients.²⁶

Based on this study's set of orthodontic patients, there does not seem to be a correlation between ESS and BMI and the cephalometric airway linear measurements. However, there is a relationship between the following airway measurements with each

other: Airway 4 to Airway 5 ($r = 0.73$), Airway 5 to Airway 6 ($r = 0.60$), Airway 6 to Airway 7 ($r = 0.65$).

In investigating potential preventive effects of orthognathic surgery for OSA, we found that the mean ESS scores for both groups were comparable and below 10; the score above which the patient is considered to have a high likelihood of OSA. The mean ESS score of the surgical group decreased from 8 to 3 after exclusion of patients who had a mandibular setback procedure included in their surgical treatment. This decrease in ESS score of 5 may be significant, however, due to the small sample size, we are not able to make conclusions about the risk of OSA and history of orthognathic surgery for correction of malocclusion. Mandibular and maxillary body lengths have shown to be associated with OSA.^{8,9} Mandibular anterior positioning devices and surgical advancement of mandible are shown to be effective in reducing symptoms of OSA.²⁷ Thus, we hypothesize that patients who have had corrective mandibular or maxillary advancement in young adulthood, are at lower risk for OSA later on in life. A larger epidemiologic study is required to further investigate this hypothesis.

Clearly, the relationship of craniofacial structures to OSA is quite complex and multifactorial. A single craniofacial pattern such as mandibular retrognathism in the normal population cannot be solely responsible for developing OSA, however, it is one of multiple risk factors.

Future Studies

Our hypothesis on the potential preventive effect of mandibular or maxillary advancement in young adulthood on developing OSA later in life needs to be investigated on a larger epidemiologic scale. Our prediction model for OSA based on cephalometric measurements should be tested on a larger group of patients.

CONCLUSIONS

1. The OSA risk assessment questionnaire serves as an excellent screening tool for orthodontic patients. This questionnaire can be easily completed chair-side in about 5 minutes with excellent response rate.
2. Prevalence of SDB and likely OSA was found to be in 40% of the males and 0% of the females in our orthodontic patient population based on the results of the BQ, but in 20% of the males and 20% of the females based on the results of the ESS.
3. There is a positive correlation between the following and the likelihood of OSA: male gender, and a more inferiorly-positioned hyoid relative to the mandible.

REFERENCES

1. Punjabi NM. The epidemiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 2008;5:136-143.
2. Kirkness JP, Schwartz AR, Schneider H, Punjabi NM, Maly JJ, Laffan AM et al. Contribution of male sex, age, and obesity to mechanical instability of the upper airway during sleep. *J Appl Physiol* 2008;104:1618-1624.
3. Young T, Evans L, Finn L, Palta M. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. *Sleep* 1997;20:705-706.
4. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med* 1999;131:485-491.
5. Caffo B, Diener-West M, Punjabi NM, Samet J. A novel approach to prediction of mild obstructive sleep disordered breathing in a population-based sample: the Sleep Heart Health Study. *Sleep*;33:1641-1648.
6. Gottlieb DJ, Yenokyan G, Newman AB, O'Connor GT, Punjabi NM, Quan SF et al. Prospective study of obstructive sleep apnea and incident coronary heart disease and heart failure: the sleep heart health study. *Circulation*;122:352-360.
7. Lam B, Ip MS, Tench E, Ryan CF. Craniofacial profile in Asian and white subjects with obstructive sleep apnoea. *Thorax* 2005;60:504-510.
8. Lowe AA, Santamaria JD, Fleetham JA, Price C. Facial morphology and obstructive sleep apnea. *Am J Orthod Dentofacial Orthop* 1986;90:484-491.

9. Miles PG, Vig PS, Weyant RJ, Forrest TD, Rockette HE, Jr. Craniofacial structure and obstructive sleep apnea syndrome--a qualitative analysis and meta-analysis of the literature. *Am J Orthod Dentofacial Orthop* 1996;109:163-172.
10. Solow B, Skov S, Ovesen J, Norup PW, Wildschiodtz G. Airway dimensions and head posture in obstructive sleep apnoea. *Eur J Orthod* 1996;18:571-579.
11. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230-1235.
12. Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep* 2008;31:1071-1078.
13. Nieto FJ, Peppard PE, Young T, Finn L, Hla KM, Farre R. Sleep-disordered breathing and cancer mortality: results from the Wisconsin Sleep Cohort Study. *Am J Respir Crit Care Med*;186:190-194.
14. Punjabi NM, Polotsky VY. Disorders of glucose metabolism in sleep apnea. *J Appl Physiol* 2005;99:1998-2007.
15. Redline S, Yenokyan G, Gottlieb DJ, Shahar E, O'Connor GT, Resnick HE et al. Obstructive sleep apnea-hypopnea and incident stroke: the sleep heart health study. *Am J Respir Crit Care Med*;182:269-277.
16. Punjabi NM, Caffo BS, Goodwin JL, Gottlieb DJ, Newman AB, O'Connor GT et al. Sleep-disordered breathing and mortality: a prospective cohort study. *PLoS Med* 2009;6:e1000132.

17. Kapur V, Strohl KP, Redline S, Iber C, O'Connor G, Nieto J. Underdiagnosis of sleep apnea syndrome in U.S. communities. *Sleep Breath* 2002;6:49-54.
18. Levendowski DJ, Morgan T, Montague J, Melzer V, Berka C, Westbrook PR. Prevalence of probable obstructive sleep apnea risk and severity in a population of dental patients. *Sleep Breath* 2008;12:303-309.
19. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 2000;284:3015-3021.
20. Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. *J Appl Physiol* 2005;99:1592-1599.
21. Fogel RB, Malhotra A, White DP. Sleep. 2: pathophysiology of obstructive sleep apnoea/hypopnoea syndrome. *Thorax* 2004;59:159-163.
22. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ et al. Predictors of sleep-disordered breathing in community-dwelling adults: the Sleep Heart Health Study. *Arch Intern Med* 2002;162:893-900.
23. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A et al. Prevalence of sleep-disordered breathing in women: effects of gender. *Am J Respir Crit Care Med* 2001;163:608-613.
24. Cistulli PA. Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment. *Respirology* 1996;1:167-174.
25. Cistulli PA, Gotsopoulos H, Sullivan CE. Relationship between craniofacial abnormalities and sleep-disordered breathing in Marfan's syndrome. *Chest* 2001;120:1455-1460.

26. Li KK, Kushida C, Powell NB, Riley RW, Guilleminault C. Obstructive sleep apnea syndrome: a comparison between Far-East Asian and white men. *Laryngoscope* 2000;110:1689-1693.
27. Almeida FR, Lowe AA, Otsuka R, Fastlicht S, Farbood M, Tsuiki S. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: Part 2. Study-model analysis. *Am J Orthod Dentofacial Orthop* 2006;129:205-213.
28. Almeida FR, Lowe AA, Sung JO, Tsuiki S, Otsuka R. Long-term sequellae of oral appliance therapy in obstructive sleep apnea patients: Part 1. Cephalometric analysis. *Am J Orthod Dentofacial Orthop* 2006;129:195-204.
29. Raffaini M, Pisani C. Clinical and cone-beam computed tomography evaluation of the three-dimensional increase in pharyngeal airway space following maxillo-mandibular rotation-advancement for Class II-correction in patients without sleep apnoea (OSA). *J Craniomaxillofac Surg*.
30. Harding SM. Prediction formulae for sleep-disordered breathing. *Curr Opin Pulm Med* 2001;7:381-385.
31. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540-545.
32. Mungan U, Ozeke O, Mavioglu L, Ertan C, Karaca IO, Keskin G et al. The Role of the Preoperative Screening of Sleep Apnoea by Berlin Questionnaire and Epworth Sleepiness Scale for Postoperative Atrial Fibrillation. *Heart Lung Circ*.


33. George JR, Chung S, Nielsen I, Goldberg AN, Miller A, Kezirian EJ. Comparison of drug-induced sleep endoscopy and lateral cephalometry in obstructive sleep apnea. *Laryngoscope*;122:2600-2605.

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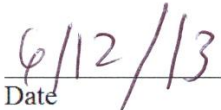
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