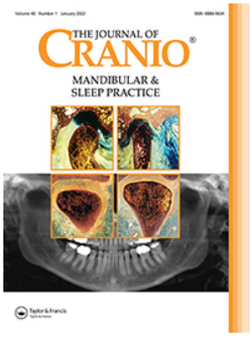

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SLEEP

A retrospective study of the influence of obesity on polysomnography and cephalometric parameters in males with obstructive sleep apnea

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ABSTRACT

Objective: Evaluate the influence of obesity on the polysomnographic and cephalometric parameters in obstructive sleep apnea (OSA).

Methods: Fifty records of male patients with OSA, containing information on dental, medical, polysomnographic, and cephalometric exams were selected. The degree of obesity was based on Body Mass Index (BMI). Group I comprised normal or overweight individuals (BMI ≤ 29.9 kg/m^2), whereas Group II consisted of obese individuals (BMI ≥ 29.9 kg/m^2).

Results: BMI significantly correlated with apnea and hypopnea index ($p < 0.0005$), minimal oxyhemoglobin saturation ($p < 0.0005$), and two cephalometric variables (soft palate length, $p = 0.01$ and width, $p = 0.01$). Group II showed a significant correlation with the position of the hyoid bone ($p = 0.02$). Soft palate length and width significantly differed between groups ($p = 0.014$; 0.016).

Conclusion: Obese males present wider and longer soft palate dimensions, and patients with a greater BMI present a more inferiorly positioned hyoid bone.

KEYWORDS
Sleep apnea; obstructive; cephalometry; obesity; polysomnography

Introduction

Obstructive sleep apnea (OSA) is a public health problem, currently considered to be a chronic, progressive, incapacitating disease with a high mortality rate and cardiovascular morbidity [1]. OSA is characterized by events of upper airway obstruction during sleep, associated with specific clinical signs and symptoms, such as snoring; breathing pauses; excessive daytime sleepiness; restless sleep; morning headache; reduced libido; neurocognitive deficits, causing emotional, social, and professional damage; and harming relationships between couples [2,3]. These recurrent episodes of upper airway obstruction are characterized by a reduction (hypopnea) or complete cessation (apnea) of the airflow, ending with an arousal due to increased respiratory effort, causing sleep fragmentation [4]. Additionally, apnea and hypopnea events may lead to oxyhemoglobin desaturation and hypercapnia. OSA can be associated with severe morbidities, such as hypersomnia, cardiac arrhythmias, arterial hypertension, thromboembolic diseases, among others [5,6]. The severity of this condition is determined by the Apnea and Hypopnea Index (AHI) or Respiratory Disturbance Index (RDI) [7]. Hence, mild OSA is defined by 5 to 15 events of sleep interruption per hour, while 15 to 30 events per hour defines moderate OSA, and in severe OSA, over 30 events of sleep interruption per hour are observed [5,8].

Obesity is an important risk factor for the development of OSA [9], exerting a negative influence on the anatomical and neurofunctional structures of the pharynx. In obese individuals, an increased fat deposition occurs in the tissues of the pharynx, leading to its compression, and the degree of fat deposition correlates with the severity of OSA [10]. The pharyngeal extraluminal pressure, an important determinant of upper airway patency, is influenced by the cervical adipose tissue and by the gravitational forces of the craniofacial structures. Thus, obesity and anatomical changes of the upper airway can increase extraluminal pressure, promoting upper airway collapse [11]. Still, a relationship between obesity and OSA may involve the participation of oxidative stress, inflammatory mediators, and metabolic dysregulation [12].

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Imaging techniques, such as lateral cephalometric radiographs, have been routinely used to help identify airway alterations [6]. Fast execution, low radiation dose (average of 3 μSv), reduced cost, and ease of analysis are some of the reasons cephalometry remains an extremely widespread imaging tool in dental practices, despite being a two-dimensional technique [13]. However, in obese patients with OSA, the number of studies that evaluate airway alterations are very scarce. The present study aimed to use cephalometric radiographs to identify the anatomical structures most influenced by obesity and the associations that may exist between these anatomical alterations and the polysomnographic parameters that determine the presence and severity of OSA.

Materials and methods

Sample selection

The study sample consisted of medical records from 50 white male patients evaluated at the Ambulatory of Respiratory Sleep Disorders of the Faculty of Medicine (Federal University of Ceará, Fortaleza, Brazil) and diagnosed with OSA according to the criteria of the American Association of Sleep Medicine (2005) [5]. These records were blindly and sequentially selected. Information regarding medical and dental history and polysomnographic (PSG) and cephalometric results were collected from records. The study was approved by the Research Ethics Committee involving Human Subjects of the Federal University of Ceará – Brazil, under number 1.559.795. Informed consent was obtained from study subjects.

The study included medical records with information on apnea and hypopnea index, oxyhemoglobin saturation, and body mass index. The examiner did not have access to personal information of volunteers, such as name, initials, date of birth, address, or telephone. Incomplete and illegible medical records were excluded from the study sample. Also, records of patients with the following conditions were excluded from the study: missing posterior teeth; under the use of hypnotics, neuroleptics, or any medication that might induce or reduce sleep, or that modified the electroencephalographic pattern; diagnosis of sleep disorders other than OSA and/or with a history of orthodontic and dentofacial orthopedic treatment; or previous treatment of OSA (use of oral appliances, history of surgeries, or use of continuous positive airway pressure masks [CPAP]). Records of individuals with a BMI below 18.5 kg/m^2 (low weight) were also excluded from the study sample (Figure 1).

For the purpose of the present study, the degree of obesity in these medical records was defined by the Body Mass Index (BMI = Weight (kg)/ Height^2 in meters). Records from normal or overweight but not obese (BMI up to 29.9 kg/m^2) individuals according to the World Health Organization (WHO) [14] were included in Group I. Data from individuals with obesity grades I, II, and III (moderate, severe, and morbid obesity, respectively) were placed in Group II (BMI equal to or greater than 30 kg/m^2).

Polysomnographic analysis

The diagnosis of OSA in these records was confirmed by overnight polysomnography (PSG). All PSG recordings were performed in the same sleeping laboratory by using a computerized "Sleep Analyzer Computer" (SAC – Version 9.2 – Oxford Instruments Inc.), consisting of a central processing unit, a Sleep Respiration Interface (Medilog SAC SRI) amplifier, a coupled unit Patient Function Box (PJB), and accessories (surface electrodes and transducers) that were connected to the patient. This examination recorded the main physiological events during sleep, such as electroencephalogram (EEG), electrooculogram (EOG), electrocardiogram (ECG), submental and tibial electromyogram (EMG), airflow, thoracoabdominal movement, number and duration of respiratory pauses, and oxyhemoglobin saturation, among other parameters. The examination was performed by a PSG technician.

The data analysis contained in the polysomnographic examination, as well as the diagnosis of OSA, were under the responsibility of a previously trained physician. Apnea was defined as a complete cessation of airflow for at least 10 s, whereas hypopnea consisted of a decreased airflow by 30% or more for at least 10 s, followed by a reduction of 4% or more in oxyhemoglobin saturation (SpO_2). The Apnea and Hypopnea Index (AHI) were defined as the number of obstructive events (apnea and hypopnea) occurring per hour of sleep. Minimal oxyhemoglobin saturation was defined as the minimum percent of hemoglobin (Hgb) in the blood carrying oxygen.

Cephalometric analysis

A single operator performed the tracing of all cephalograms, which included anatomical tracing, points, lines, cephalometric planes, and angular and linear cephalometric quantities of the upper airway. The evaluation of the pharyngeal airway dimensions, the length and width of the soft palate, and the position of the hyoid bone were the main focus of this analysis (Table 1).

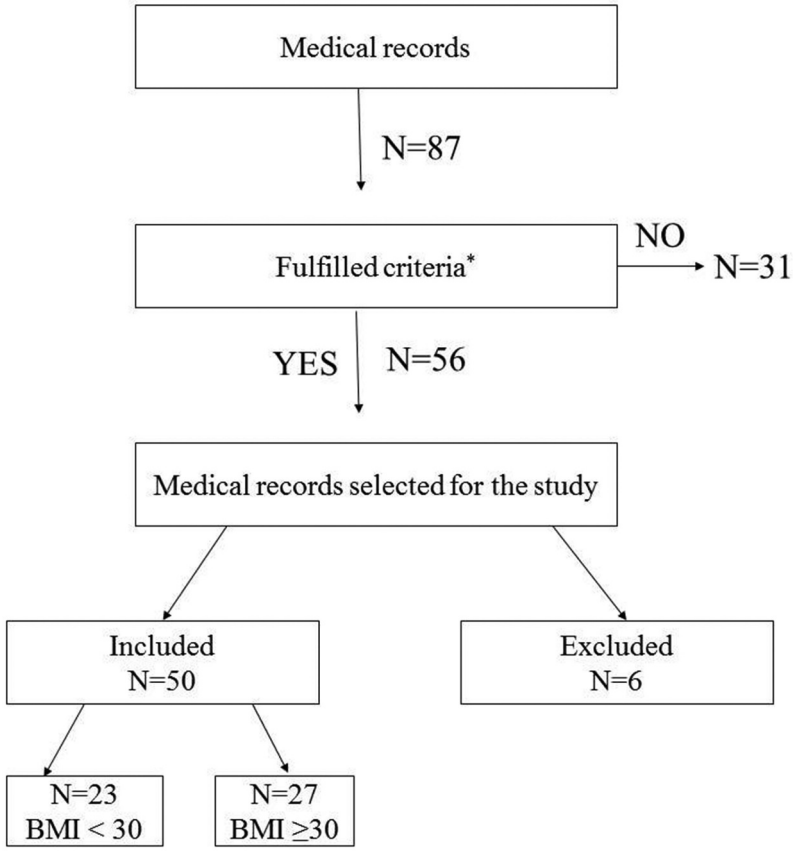

Figure 1. Flowchart for the selection of medical records. *Inclusion criteria: medical records with information on apnea and hypopnea index, oxyhemoglobin saturation and body mass index. The examiner did not have access to personal information of volunteers, such as name, initials, date of birth, address, or telephone. Exclusion criteria: Incomplete and illegible medical records; the records of patients with the following conditions: with loss of posterior dental support; using sleep inducers, hypnotics, neuroleptics, or any medication that might induce or reduce sleep, or that modified the electroencephalic pattern; with sleep disturbances other than OSA and/or with a history of facial orthopedic or orthopedic facial or previous treatment of OSA (oral appliances, surgeries, or use of continuous positive airway pressure masks – CPAP). Records of individuals with a BMI below 18.5 kg/m^2 (low weight).

Statistical analysis

Based on the study by Valarelli et al. [6], which observed the highest apnea scores in patients with an elevated BMI (30.7 ± 0.9) compared to patients with a lower BMI (29.8 ± 1.2), it was estimated that the evaluation of 44 patients would be required for the current study, in order to reject the null hypothesis with 80% power and 95% confidence (Student's *t*-test). A 10% loss due to medical records with incomplete information was expected, so the sample size was increased to n = 50.

To evaluate the reliability and reproducibility of the measurements, an operator calibration and agreement analysis was performed. Data were submitted to Kolmogorov-Smirnov and Friedman normality tests. Pearson's correlation analysis (parametric data) was performed between BMI, polysomnographic (AHI and SpO_2), and cephalometric variables (UPAS, LPAS, PNS-P, SPW and H-MP) for the whole sample (Groups I and II combined) and for Groups I and II, separately. Measurements were taken at two different times, with an interval of 30 days, by the same evaluator. The statistical significance was set at $p < 0.05$. The software used was SPSS version 24.0.

Results

Examiner calibration showed no statistically significant difference between cephalometric airway measurements in the two moments in which the measurements were taken, and a statistically significant correlation between the first and second measurements ($p<0.001$, r =0.948) was observed.

Sample demographics consisted of subjects presenting a mean age of 44.3±10.2years (minimum: 20, maximum: 64years old), mean weight of 89±15.5 kg (minimum: 66, maximum: 143 kg), mean height of 173.7±6.6 cm (minimum: 161, maximum: 188 cm), and a mean BMI of 29.6±5kg/m^2 (minimum: 22.6,

Table 1. Cephalometric points used for analysis.

Point	Description
Pharyngeal airway	
Ba-PNS	Dimension of the pharynx bone. Linear distance between points Ba and PNS.
UPAS	Upper posterior air space. Width of air space posterior to the soft palate along a line parallel to the Goc-B line, which passes through the midpoint of the effective length of the soft palate (PNS-P).
LPAS	Lower posterior air space. Linear distance between a point on the base of the tongue and another point on the posterior pharyngeal wall, both determined by the extension of line B-Goc.
Soft Palate	
PNS-P	Length of Soft Palate
SPW	Soft Palate Width. Maximum of the soft palate width measured on a line parallel to the palatine plane (ANS – PNS).
Hyoid bone	
H-MP	Linear distance along a perpendicular line from the H point to the mandibular plane.

Ba (Basion); PNS (Posterior Nasal Spine); ANS (Anterior Nasal Spine); P (Lowest point of the soft palate); B (Deepest point on the anterior surface of the contour of the mandibular symphysis); Goc (Cephalometric Gonion); Me (Mentonian); H (Superior and anterior point of the hyoid bone).

Table 2. Pearson (R) correlation indices between AHI, SpO_2, and cephalometric variables UPAS, LPAS, PNS-P, SPW, and H-MP.

Variable	AHI	SpO_2
AHI	-	−0.63 (0.00)**
SpO_2	−0.63 (0.00)**	-
UPAS	−0.26 (0.09) NS	0.13 (0.39) NS
LPAS	0.16 (0.29) NS	−0.18 (0.25) NS
PNS-P	0.24 (0.11) NS	−0.32 (0.03) *
H-MP	0.22 (0.15) NS	−0.11 (0.47) NS
SPW	0.45 (0.00) **	−0.49 (0.00) **

NS: not significant; *: significant at 5% ($p < 0.05$); **: significant at 1% ($p < 0.01$).
See Table 1 for definitions.

maximum: 46.1 kg/m^2). The apnea and hypopnea index (AHI/h) mean values were high (50.857±23.678), while mean minimal oxyhemoglobin saturation (SpO_2 min) values were low (67.591±12.675).

There was a significant negative correlation between SpO_2 and AHI (p<0.01), PNS-P and SpO_2 (p=0.03), SPW and AHI (p<0.01), SPW and SpO_2 (p<0.01). A positive correlation was observed between SPW and AHI (p<0.01) (Table 2). Mean and standard deviations of the cephalometric variables for Groups I(BMI <30; n=23) and II (BMI ≥ 30; n=27) are shown in Table 3. The soft palate length (PNS-P) and width (SPW) were statistically different when comparing both groups (Table 3).

Table 3. Mean and standard deviations of the cephalometric variables UPAS, LPAS, PNS-P, SPW, and H-MP for Group I (BMI <30) and for Group II (BMI ≥ 30).

	Group I (BMI < 30)			Group II (BMI ≥ 30)			
Cephalometric variable	Mean	Standard deviation	N	Mean	Standard deviation	N	p-value
UPAS	8.667	4.078	23	7.543	2.345	27	0.238
LPAS	9.881	4.588	23	11.239	2.449	27	0.198
PNS-P	43.548	5.760	23	47.217	4.311	27	0.014*
SPW	12.976	3.100	23	15.239	3.282	27	0.016*
H-MP	26.810	6.355	23	27.174	7.020	27	0.848

*$p < 0.05$: Student´s t-test.
See Table 1 for definitions.

When all study participants from Groups I and II combined (BMI <30 and ≥ 30) were considered, BMI significantly correlated with polysomnographic variables (AHI and SpO_2) and with cephalometric variables (PNS-P and SPW), while Group II presented a significant correlation with the position of the hyoid bone (H-MP) (Table 4).

Discussion

Population studies have shown that OSA is more prevalent among males [1]. In addition, a recent systematic review of the literature found a higher OSA prevalence in association with sex (greater prevalence among males), age advancement, and body mass index [15], and depending on the population, the male-to-female ratio may range from 3:1 to 10:1 [16]. Symptoms, such as daytime fatigue, headaches, depression, or insomnia, are more frequently reported in the female population, whereas snoring has a greater prevalence among men [17,18]. The mean BMI of 29.6 ± 5 kg/m^2 observed among males in the present study was lower than previously reported data in both males and females [18,19]. In the current study population, obese men reported a greater number of abnormal respiratory events (apneas and hypopneas) during sleep, when compared

Table 4. Pearson correlation indices between BMI and AHI, SpO_2, UPAS, LPAS, PNS-P, SPW, and H-MP for the whole sample and for Group I (BMI <30) and Group II (BMI ≥ 30).

	AHI	SpO_2	UPAS	LPAS	PNS-P	SPW	H-MP
BMI	0.57 (0.00) **	−0.63 (0.00) **	−0.13 (0.40)	0.18 (0.25)	0.39 (0.01) *	0.40 (0.01)*	0.23 (0.13)
BMI <30 N = 23	0.20 (0.38)	−0.35 (0.12)	−0.11 (0.63)	0.11 (0.62)	0.02 (0.93)	−0.03 (0.91)	−0.12 (0.60)
BMI ≥30 N = 27	0.32 (0.14)	−0.36 (0.09)	0.06 (0.78)	0.05 (0.83)	0.35 (0.10)	0.36 (0.09)	0.48 (0.02)*

NS: not significant; *: significant at 5% ($p < 0.05$); **: significant at 1% ($p < 0.01$).
See Table 1 for definitions.

to data on obese women in other studies. This result may be explained by the fact that women increase their chemo-responsiveness (hyperventilatory response to blood gas changes) by increasing their weight to a greater extent than men [20], requiring a greater fat deposition to develop OSA [21]. It is also important to note that OSA is especially observed among postmenopausal women [22].

The polysomnographic parameters included in this study, Apnea and Hypopnea Index (AHI) and the minimal oxyhemoglobin saturation (SpO_2 min), were chosen to investigate how BMI and cephalometric parameters may explain decreased airflow (AHI) followed by a reduction in oxyhemoglobin saturation (SpO_2). A negative correlation was found between these two polysomnographic variables, indicating that the higher the AHI, the lower the SpO_2. This occurs due to the fact that, in patients with high AHI, respiratory pauses are more frequent and of longer duration, resulting in a decrease in arterial oxygen and, consequently, lower values of SpO_2. Oxyhemoglobin saturation is also a polysomnographic parameter of great importance due to the systemic repercussions of the reduction of arterial oxygen during sleep. In patients with OSA, arterial oxygen saturation often drops to below 80%, whereas in patients with OSA, accentuated with prolonged apneic episodes, saturation levels reach 60%. In normal patients, these levels are not lower than 92% [23]. The minimal oxyhemoglobin saturation (SpO_2 min) used in this study as one of the polysomnographic parameters confirms the reduction of arterial oxygen levels during periods of apnea and hypopnea. In the present study, the mean SpO_2 was of 67.5%, reflecting the desaturations that accompany this condition. These desaturation events are related to the number and duration of abnormal respiratory events resulting from repetitive obstructions of the upper airways during sleep [24].

Cephalometry has been used for many years by orthodontists and oral maxillofacial surgeons to identify and quantify skeletal and soft tissue abnormalities, aiding in orthodontic or surgical diagnosis and treatment [25]. But, it was not until the early 1980s that cephalometrics was used with the aim of verifying possible anatomical alterations involved in sleep disorders, more specifically in OSA [26]. Cephalometric studies have demonstrated a wide variety of craniofacial anomalies and, more specifically, in the soft tissue anatomy of the upper pharyngeal air space, which may predispose to airway collapse in patients with OSA [27]. The pharyngeal airway of individuals with OSA was evaluated cephalometrically in the retropalatal region (or velopharynx), corresponding to the region posterior to the soft palate and to the retroglossal region posterior to the base of the tongue. The soft palate was analyzed in its width and length. The soft palate width (SPW) demonstrated a significant correlation with AHI. This study's findings also revealed a significant correlation between the soft palate length (PNS-P) and the SpO_2. This result indicates that an increased soft palate size may generate an increase in the apnea and hypopnea index (AHI) and a decrease in the minimal oxyhemoglobin saturation (SpO_2).

Obesity is one of the most important risk factors for the development of OSA. North American data showed that 79.1% of men aged 40 to 59 years had a BMI (BMI) greater than 25 kg/m^2, which shows a high percentage of overweight and obese men [28]. However, a previous study showed that there was no statistically significant difference in the severity of OSA when obese and non-obese individuals were compared. Even so, those with BMI> 25 kg/m^2 had a larger tongue size and width of the dental arches [29].

In order to better analyze the influence of obesity on cephalometric variables, cephalometric measurements were chosen, representing the pharyngeal airway space, soft palate, and position of the hyoid bone, since these anatomic sites may be more susceptible to changes caused by increase in BMI. When the whole sample was considered (BMI <30 and ≥ 30), there was a significant correlation of BMI with the polysomnographic variables AHI and SpO_2 and with the cephalometric variables PNS-P and SPW, while Group II (BMI ≥ 30) presented correlation with the cephalometric variable H-MP. Corroborating these findings, Kim et al. [30] observed statistically significant differences in the AHI parameter and the H-MP measurement when comparing obese and non-obese patient groups [30].

The infiltration of fat into the muscles that form the walls of the upper airway can directly interfere with their functional mechanism, modifying the capacity of the pharyngeal dilator muscles to maintain airway space dimensions during sleep, increasing the number of abnormal respiratory events, raising the index of apnea and hypopnea, decreasing minimal oxyhemoglobin saturation, and contributing significantly to OSA severity [31,32]. The correlation of BMI with OSA severity has also been shown to be related to the increase in tissue volume adjacent to the pharyngeal airway space, as evidenced in the current results by the increase in soft palate length (PNS-P) and width (SPW). Such an increase in soft palate length and width may lead to oropharyngeal narrowing during sleep, indicating that the retropalatal region is increased in the majority of obese patients with OSA [33]. In addition, a previous study demonstrated that the reduction of BMI by

approximately 7% over a 6-month follow-up period correlated with a significant decrease in the apnea and hypopnea indexes [34]. The distance from the hyoid bone to the mandibular plane (H-MP) was greater in the group of patients with BMI ≥ 30, showing that the hyoid bone had a more inferior position among patients with a higher BMI, probably due to the presence of a larger deposit of cervical fat. The inferior displacement of the hyoid bone contributes to the reduction of pharyngeal airway dimension [35]. Tangugsorn et al. [36] also demonstrated that obese patients with OSA presented a more inferiorly and anteriorly positioned hyoid bone in relation to the mandibular plane and the third cervical vertebra, respectively.

The main limitations of the present study relate to the two-dimensional imaging (cephalometry) used to evaluate the airways. Nonetheless, the cost-effective and practical method investigated in the present study allowed the identification of important anatomical alterations in the soft palate and hyoid bone of obese patients that are correlated with polysomnographic findings and BMI of OSA patients.

Conclusion

Obesity influenced the soft tissue dimensions of the pharynx. Specifically, soft palate width and length significantly correlated with AHI and SpO_2 among obese patients with OSA. The increase in the width and length of the soft palate seems to contribute to the aggravation of OSA, and in these patients, the hyoid bone is more inferiorly positioned, contributing to airway space reduction. These findings demonstrate the importance of performing a cephalometric evaluation with focus on pharyngeal airway space, soft palate, and position of the hyoid bone, while investigating obese males with suspected OSA. This type of assessment may help clinicians and researchers better understand the underlying causes of OSA, enabling adequate management of this condition.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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References

[1] 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.

[2] Bachour P, Bachour A, Kauppi P, et al. Oral appliance in sleep apnea treatment: respiratory and clinical effects and long-term adherence. Sleep Breath. 2016;20 (2):805–812.

[3] Alencar e Rangel AJ, Barros VM, Seraidarian PI. Snoring and obstructive sleep apnea syndrome: a reflection on the role of dentistry in the current scientific scenario. Dental Press J Orthod. 2012;17:58–63.

[4] Chaves Junior CM, Dal-Fabbro C, Bruin VMSD, et al. Brazilian consensus of snoring and sleep apnea: aspects of interest for orthodontists. Dental Press J Orthod. 2011;16:1–10.

[5] American Academy of Sleep Medicine. International classification of sleep disorders. 3rd ed. Darien (IL); 2014.

[6] Valarelli LP, Corradi AMB, Grechi TH, et al. Cephalometric, muscular and swallowing changes in patients with OSAS. J Oral Rehabil. 2018;45 (9):692–701.

[7] Shamim-Uzzaman QA, Singh S, Chowdhuri S. Hypopnea definitions, determinants and dilemmas: a focused review. Sleep Sci Prac. 2018;2(1):1–12.

[8] Tsara V, Amfilochiou A, Papagrigorakis MJ, et al. Guidelines for diagnosis and treatment of sleep-related breathing disorders in adults and children. Definition and classification of sleep related breathing disorders in adults: different types and indications for sleep studies (Part 1). Hippokratia 2009;13(3):187–191.

[9] Tuomilehto H, Seppä J, Uusitupa M. Obesity and obstructive sleep apnea – clinical significance of weight loss. Sleep Med Rev. 2013;17(5):321–329.

[10] Weaver JM. Increased anesthetic risk for patients with obesity and obstructive sleep apnea. Anesth Prog. 2004;51(3):75.

[11] Horner RL, Mohiaddin RH, Lowell DG, et al. Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnea and weight matched controls. Eur Respir J. 1989;2(7):613–622.

[12] Kuvat N, Tanriverdi H, Armutcu F. The relationship between obstructive sleep apnea syndrome and obesity: a new perspective on the pathogenesis in terms of organ crosstalk. Clin Respir J. 2020;14(7):595–604.

[13] Kim KB. How has our interest in the airway changed over 100 years? Am J Orthod Dentofacial Orthop. 2015;148(5):740–747.

[14] World Health Organization. Obesity: preventing and managing the global epidemic. Report of a World Health Organization Consultation. Geneva (Switzerland): WHO; 2000.

[15] Senaratna CV, Perret JL, Lodge CJ, et al. Prevalence of obstructive sleep apnea in the general population: a systematic review. Sleep Med Rev. 2017;34:70–81.

[16] Wimms A, Woehrle H, Ketheeswaran S, et al. Obstructive sleep apnea in women: specific issues and interventions. Biomed Res Int. 2016;2016:1764837.

[17] Bonsignore MR, Saaresranta T, Riha RL. Sex differences in obstructive sleep apnoea. Eur Respir Rev. 2019;28 (154):190030.

[18] Young T, Hutton R, Finn L, et al. The gender bias in sleep apnea diagnosis. Are women missed because they

have different symptoms? Ach Intern Med. 1996;156:2445–2451.
[19] Redline S, Kump K, Tishler P, et al. Gender differences in sleep disordered breathing in a community-based sample. Am J Respir Crit Care Med. 1994;149(3 Pt 1):722–726.
[20] Kunitomo F, Kimura H, Tatsumi K, et al. Sex differences in awake ventilatory drive and abnormal breathing during sleep in eucapnic obesity. Chest. 1988;93 (5):968–976.
[21] Dancey DR, Hanly PJ, Soong C, et al. Gender differences in sleep apnea: the role of neck circumference. Chest J. 2003;123(5):1544–1550.
[22] Kapsimalis F, Kryger MH. Gender and obstructive sleep apnea syndrome, part 1: clinical features. Sleep. 2002;25 (4):412–419.
[23] Meyer JB Jr, Knudson RC. The sleep apnea syndrome. Part. I: diagnosis. J Prosthet Dent. 1989;62(6):675–679.
[24] Kulkas A, Duce B, Leppänen T, et al. Severity of desaturation events differs between hypopnea and obstructive apnea events and is modulated by their duration in obstructive sleep apnea. Sleep Breath. 2017;21(4):829–835.
[25] Fleetham JA. Upper airway imaging in relation to obstructive sleep apnea. Clin Chest Med. 1992;13 (3):399–416.
[26] Riley R, Guilleminault C, Herran J, et al. Cephalometric analysis and flow-volume loops in obstructive sleep apnea patients. Sleep. 1983;6(4):303–311.
[27] Conley RS. Evidence for dental and dental specialty treatment of obstructive sleep apnea. Part 1: the adult OSA patient and Part 2: the paediatric and adolescent patient. J Oral Rehabil. 2011;38(2):136–156.
[28] Ogden CL, Carroll MD, Kit BK. Prevalence of childhood and adult obesity in the United States, 2011-2012. JAMA. 2014;311(8):806–814.
[29] Maeda K, Tsuiki S, Isono S, et al. Difference in dental arch size between obese and non-obese patients with obstructive sleep apneoea. J Oral Rehabil. 2012;39 (2):111–117.
[30] Kim TH, Chun BS, Lee HW, et al. Differences of upper airway morphology according to obesity: study with cephalometry and dynamic MD-CT. Clin Exp Otorhinlaryngol. 2010;3(3):147–152.
[31] Guilleminault C, Quera-Salva MA, Partinen M, et al. Women and the obstructive sleep apnea syndrome. Chest. 1988;93(1):104–109.
[32] Vollmer ME. The obstructive sleep apnea syndrome: pathophysiology, diagnosis and treatment. Indiana Med. 1987;80(12):1174–1177.
[33] Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. Sleep. 1996;19(2):104–115.
[34] Sutherland K, Phillips CL, Yee BJ, et al. Maxillomandibular volume influences the relationship between weight loss and improvement in obstructive sleep apnea. Sleep. 2016;39(1):43–49.
[35] Sutherland K, Lee RWW, Cistulli PA. Obesity and craniofacial structure as risk factors for obstructive sleep apnea: impact of ethnicity. Respirology. 2012;17 (2):213–222.
[36] Tangugsorn V, Krogstad O, and Espeland L, et al. Obstructive sleep apnea: multiple comparisons of cephalometric variables of obese and non-obese patients. J Craniomaxiloffac Surg. 2000;28(4):204–212.