Upper airway sagittal dimensions in obstructive sleep apnea (OSA) patients and severity of the disease

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SUMMARY

Objectives. To estimate the soft tissue determined individual features of the upper airway sagittal size which may predispose the patient to snoring and OSA and to find whether there is any relationship between the patient's body-mass index (BMI) and the severity of OSA.

Material and methods. 58 consecutive patients with a mean age of 33.4 years were surgically treated for snoring and OSA. Before the operation they were subject to the following: physical examination, a sleep study to determine the severity of sleep disturbed breathing (SDB); the body mass index (BMI) was calculated, lateral cephalometry (LC) was recorded.

Results. Snoring was found in 25 patients, OSA I (mild) – 15, OSA II (moderate) – 9, OSA III (severe) – in 9 patients. Among patients with obesity, the most widespread types of sleep disorders were OSA II and OSA III. (p=0.029). There were differences in the mean values of the BMI between the group of snorers and the group with OSA II (p=0.007), between snorers and OSA III (p=0.006). Differences in upper airway cephalometric mean values between groups were found, the thickness of the uvula (SPT) between snorers and OSA III (p=0.001), between the OSA I and OSA III groups (p=0.032), and between the OSA II and OSA III groups (p=0.075).

Conclusion. Our study found narrowing in the anterior-posterior dimension of the airway at all levels, correlating with the severity of OSA as well as the BMI. The narrowest place was found in the oropharynx with an extension to the hypopharynx.

Key words: obstructive sleep apnea, lateral cephalometry.

INTRODUCTION

OSA is a significant medical problem affecting up to 4 percent of middle-aged adults [1]. Repetitive cycles of apneas, desaturations [2] and activation of the sympathetic nervous system (SNS) in a longer period play a significant role in the development of hypertension [3], cardiovascular diseases [4] and increase the risk of death from myocardial infarction [5] and cerebral stroke [6]. Although most of the patients are overweighted and have a short,

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malities [8]. A narrow, floppy upper airway provides the pathophysiological basis for OSA. The pharynx will tend to collapse at inspiration due to Bernoulli's effect, which results in partial or complete obstruction. Obstruction persists until sleep is interrupted and muscle tone is restored. With arousal, breathing is restored and after a few breaths deeper sleep returns with recurrence of obstruction as the muscles again relax. In more severe cases of OSA, this cycle of apneas and arousals may occur hundreds of times during the night. In mild cases, it may occur only at certain sleep stages (REM sleep) and at certain postures (supine) or after alcohol consumption. These patients suffer from multiple sleep disruptions because of the regular snoring, airway collapse with apneas and following arousal, which results in fragmented sleep [9]. The consequences are excessive daytime somnolence (EDS), decreased quality of life and an increased car accident risk as a result of somnolence during wakefulness [10]. Multiple radiological tech-

thick neck [7], some are of normal weight but have a small, retrognatic jaw or other anatomical abnor-

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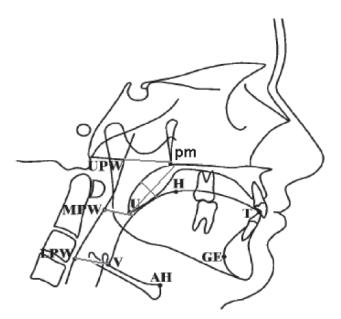


Fig. 1. Cervico-craniofacial landmarks and reference lines

niques – CT, MRI, lateral cephalometry (LC) are used for the examination of the skeletal and soft tissue of the upper airway [11]. Several surgical methods were suggested to relieve the symptoms of OSA. The most frequently used methods among these are uvulopalatoplasty, uvulopalato-pharyngoplasty with or without tonsillectomies; besides, also corrections of nasal breathing, genioglossal advancement and skeletal advancement by bimaxillary surgery have been used [12-14]. The aims of the study were to, by means of LC, estimate soft tissue determined individual features of the upper airway sagittal size which may predispose the patient to snoring and OSA and to find whether there is any relationship between the patient's body mass index (BMI) and the severity of OSA.

MATERIAL AND METHODS

58 consecutive patients (8 female, 50 male) with a mean age of 33.4 years (the range was from 18 to 78 years) surgically treated for snoring and OSA were included into the study. Before the operation, all patients had a sleep study, underwent physical examination - visualization of the pharynx (Malampati tongue position) and evaluation of nasal breathing. By using questionnaires completed by patients and their bed partners prior to evaluation, we collected anthropometrical data, history of snoring, observed apnea, and medical history [15]. The questionnaires included the Epworth sleepiness scale (ESS). The ESS is a self-administered questionnaire comprising 8 questions. It provides a measure of a person's general level of daytime sleepiness, or their average sleep propensity in daily life. It has become the world standard method for making this assessment. Informed consent was obtained from all the patients.

All patients underwent sleep studies - full night polysomnography (PSG) or polygraphy (PG), which included nasal and mouth airflow, thoracic and abdominal movements recorded by inductive plethysmography, electrocardiogram (ECG), oxygen saturation measured by finger oximetry, body position, snoring events. PSG included PG data, plus electroencephalogram (EEG - C3-A2, C4-A1, O2-A1, O3-A2), bilateral electrooculogram (EEG), chin and leg electromyogram (EMG) [16]. Apneas and hypopneas were scored according to the criteria set by the American Association of Sleep Medicine (AASM). The number of apneas and hypopneas was counted and the apnea hypopnea index (AHI) was calculated. The severity of OSA was determined by AHI, the frequency of apneas and hypopneas per hour during sleep time: snoring ≤ 5 ; mild (OSA) I) corresponds to an AHI value of 5-15, moderate (OSA II) - 15-30, severe $(OSA III) - \leq 30$. BMI was calculated by dividing each patient's weight (in kg) by the square of height (in m). Patients were divided into three groups according to BMI. BMI≤25 was defined as normal weight, BMI 25-30 as overweight and BMI ≥ 30 as obesity.

Patients with significant nasal, oral, pharyngeal, or mandibular abnormalities or diseases were not assigned for the study. For assessment of the upper airway lateral cephalometry, the patients were standing with a natural head position (the mirror technique). At the end-expiration phase they were asked not to swallow. All cephalograms were traced and digitized by a single observer, blinded to AHI and other signs or symptoms of OSA. Randomly chosen 20 LCs were measured repeatedly to assess the measurement error. The cephalometric lines and landmarks used in the study are shown in Figure 1. The following anatomical landmarks were used for measuring the anterior-posterior dimensions: Pm pterygomaxillary fissure, UPW - upper posterior pharyngeal wall, U – uvula tip, MPW – middle posterior pharyngeal wall, V - valecula, LPW lower posterior pharyngeal wall, SPT – maximal thickness of uvula. Cephalometric variables were measured as anterior-posterior dimensions of the airway at: nasopharynx (UPW - Pm), glossopharynx (MPW - U), hypopharynx LPW - V, lengths of the uvula pm-U and maximum thickness of the uvula measured perpendicular to the pm-U line – SPT.

Statistical analysis

Descriptive statistics were used for all the measurements. The mean values and SD were calculated

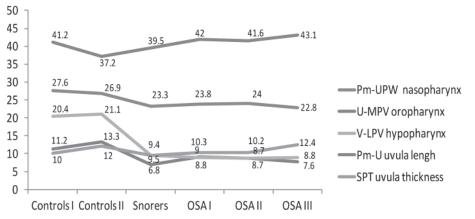


Fig. 2. Comparison of the upper airway dimensions between the patients of the present study and the study by Kollias and Krogstad [26, 27]

for all the measurements and BMI in each OSA group. A comparison of the mean values among groups was performed using one-way ANOVA and Bonferroni adjustment to control the overall type I error rate. ANOVA variance analysis was used to determine the relationship between the upper airway variables, the BMI and AHI as showing OSA severity. The variables were controlled for the normal distribution and included in the analysis as continuous variables. A P value <0.05 was used as the level of statistical significance.

RESULTS

Measurement*

Pm – UPW

U - MPV

V - LPV

Pm – U

SPT

BMI

AHI

According to clinical signs and symptoms, questionnaires and sleep studies, snoring was found in

Snoring

(n-25)

23.4±34

6.8±3.23

9.4±2.76

39.5±9.55

 9.5 ± 1.47

26.5±3.22

3.5±1.27

Mean

Mean

Mean

Mean

Mean

Mean

Mean

43.1% of patients (n=25); OSA mild – in 25.9% of patients (n=15), OSA moderate – in 15.5% of patients (n=9), OSA severe – in 15.5% of patients (n=9). The mean values and standard deviations of upper airway cephalometric measurements, AHI and BMI in different groups of OSA and snoring are presented in Table 1.

The lowest mean values were observed in the group of patients suffering from snoring. Statistically significant

differences were found for the thickness of the uvula (SPT): between snorers and OSA III patients (p=0.001), between the OSA I and OSA III groups (p=0.032), and between the OSA II and OSA III groups (p=0.075).

Sleep disturbed breathing (SDB) was positively correlated with BMI (p=0.029). Statistically significant differences in the mean values of BMI were observed between snorers and OSA II patients (p=0.007), and between the group of snorers and the OSA III group (p=0.006).

The prevalence of SDB among patients with different BMI is described in Table 2.

Snorers, without significant breathing disturbances, were more prevalent among patients with

> normal weight. There were no OSA II and OSA III cases among patients with normal weight. Among patients with obesity, the most prevalent sleep disorders were OSA II and OSA III.

> Variance analysis confirmed the statistically significant relationship between the severity of SDB and the cephalometrically measured thickness of the uvula (SPT) (p<0.001) and BMI (taken as a continuous variable, p<0.001). The severity of SDB increased with increasing thickness of the uvula and the BMI value.

DISCUSSION

The cause of OSA is very complex and results from an interaction between abnormal

Table 1. Mean sagittal values	$(mm \pm SD)$ of s	sleep disturbed breathing	(SDB)

OSAI

(**n-15**)

23.8±2.55

9.0±2.66

8.8±4.13

42.0±4.98

10.3±1.82

28.3±4.08

12.0±

OSAII

24.0±3.77

8.7±2.95

8.7±2.79

41.6±5.97

10.2±2.13

31.3±2.39

25.7±4.74

(n-9)

OSA III

Pm-UPW

U - MPV

V - LPV

Pm – U

SPT

BMI

AHI

(**n-9**)

Table 2.	Sleep	disturbed	breathing	(SDB)) and BMI

Degree of	BMI				_	Total			
SDB		<25	25-30			>30		- Total	
	Ν	%	Ν	%	Ν	%	Ν	%	
Snoring	7	77.8	15	45.5	3	18.8	25	43.1	
OSA I	2	22.2	10	30.3	3	18.8	15	25.9	
OSA II	0	0	3	9.0	6	37.5	9	15.5	
OSA III	0	0	5	15.2	4	25.0	9	15.5	
Total	9	100	33	100	16	100	58	100	

anatomical features, obesity, and the influence of still other pathophysiological factors is under discussion. Two hypotheses explaining the tendency of obstruction of the upper airway in OSA patients have been proposed and it seems that both are playing a significant role in the development of the disease. The neural hypothesis explains obstruction by reduced neural muscle activity [17], damage of the soft tissues by repetitive cycles of snoring and a struggle of the dilator muscles against the obstructive action of negative inspiratory forces [18]. The anatomic theory suggests an anatomic narrowing of the upper airway [19, 20]. They can be due to congenital abnormalities of the bony structure, yet mostly they are caused by an increase of parapharyngeal fat, pushing the lateral walls of the airway inwards, causing the normally elliptical shape of the upper airway to become more circular and narrower [21]. The narrowed upper airway causes an increase in negative pressure with increased airflow and predisposes the upper airway to collapse, due to Bernoulli's effect.

Many methods have been used to identify the sites of obstruction in OSA patients, such as endoscopy, fluoroscopy, catheters, CT scanning, MRI, LC. By choosing lateral cephalometry, we were looking for a noninvasive, inexpensive, simple technique with minimum radiation exposure [22]. It has been shown to be reliable narrowing in sagittal dimensions of the airway at all levels correlated with OSA severity and BMI. The narrowest place was found at the oropharynx with an extension to the laryngopharynx, which is in agreement with other

studies that have used different radiological techniques [23 - 25, 21]. These patients have not only a smaller cross sectional size of the airway but also an enlarged uvula as compared with the controls. The mean values of the upper airway found in our study and the mean values of the airways of dental students without any breathing disturbances reported by Kollias and Krogstad [26, 27] are shown in Fig. 2.

In literature, we find controversial opinions as to the effectiveness of LC for predicting the surgical outcome. Ryan and his colleagues [28] found that alternative cephalometric measurements were helpful in predicting the success of surgery. Gislason and colleagues [29] found that none of the cephalometric variables helped to predict the surgical outcome. Howewer, the common opinion is that the severity of OSA correlates with the surgical outcome. [28, 30].

CONCLUSIONS

1. Patients with sleep disturbed breathing have reduced soft tissue determined upper airway size at all levels in anterior-posterior dimensions; it correlates with the severity of OSA.

2. There is a statistically significant correlation between the severity of OSA and the thickness of the uvula.

3. The narrowest sagittal dimension of the upper airway in OSA patients was observed at the oropharyngeal level.

4. There was a correlation between the body mass index and the severity of sleep disturbed breathing.

REFERENCES

- 1. Lindberg E, Gislason T. Epidemiology of sleep-related obstructive breathing. *Sleep Med Rev* 2000;4:411-33.
- Ryan CM, Bradly TD: Pathogenesis of obstructive sleep apnea. J Appl Physiol 2005;99:2440-50.
- 3. Nieto F, Young T, Lind B, Shahar E, Samet JM, Redline S, et al. Association of sleep disordered breathing, sleep apnea and hypertension in a large community based study. *JAMA* 2000;238:1829–36.
- Peker Y, Carlson J, Hedner J. Increased incidence of coronary artery disease in sleep apnoea: a long-term follow-up. *Eur Respir J* 2006;28:596–602.
- Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. *Lancet* 2009;373:82-93.
- Parra O, Arbiox A, Bechich S, Grasia-Eroles L, Montserrat JM, Lopea JA, et al. Time course of sleep-related breathing disorders in first-ever stroke or transient ischemic attack. *Am J Respir Crit Care Med* 2000;161:375-80.
- Mayer P. Pepin JL, Bettega G, Veale D, Ferretti G, Deschaux C, et al. Relationship between body mass index, age and upper airway measurements in snorers and sleep apnoea patients. *Eur Respir J* 1996;9:1801-9.
 Johns FR, Strollo PJ, Buckley M, Constantino J. The influence
- Johns FR, Strollo PJ, Buckley M, Constantino J. The influence of craniofacial structure on obstructive sleep apnea in young adults. *J Oral Maxillofac Surg* 1998;56:596–602.
- 9. Kimoff RJ. Sleep fragmentation in obstructive sleep apnea.

Sleep 1996;19:61-6.

- Ellen RL, Marshall SC, Palayew M, Molnar FJ, Wilson KG, Man-Son-Hing M. Systematic review of motor vehicle crash risk in persons with sleep apnea. J Clin Sleep Med 2006;15:193-200.
- 11. Faber CE, Grymer L. Available techniques for objective assessment of upper airway narrowing in snoring sleep apnea. *Sleep Breath* 2003;7:77-86.
- Won CHJ, Li KK, Guilleminault C. Surgical treatment of obstructive sleep apnea. Proc Am Thoracic Soc 2008;5:193-9.
- Franklin KA, Antilla H, Axelsson S, Gislason A, Maasilta P, Myhre KI, et all. Effects and side-effects of surgery for snoring and obstructive sleep apnea – A systematic review. *Sleep* 2009;32:27-36.
- Lowe AA, Fleetham, JA, Adachi S, Ryan CF. Cephalometric and computed tomographic predictor of obstructive sleep apnea severity. *Am J Orthod Dentofacial Orthop* 1995;107:589-95.
- 15. Pang KP, Terris DJ, Screening for obstructive sleep apnea: an evidence based analysis. *Am J Otolaringol* 2006;27:112-8.
- 16. Practice parameters for the indications for polysomnography and related procedures. Polysomnography Task Force, American Sleep Disorders Association Standards of Practice Committee. *Sleep* 1997;20:406–22.
- 17. Horner RL, JA Innes, K Murphy, and A Guz. Evidence of refflex upper airway dilator muscle activation by sudden negative

presure in man. J Physiol 1991;436:15-29.

- Friberg D. Heavy snorer's disease: a progressive local neuropathy. Acta Otolaryngol 1999;119:925-33.
- 19. Abramson Z., Susarla S., August M., Troulis M., Kaban L. Abranson Z., Susana S., August M., Houns M., Kadar E., Three-Dimensional Computed Tomographic Analysis of Air-way Anatomy in Patients With Obstructive Obstructive Sleep Apnea. *Chest* 2002;122:1139-47.
 Sforza E, Bacon W, Weiss T, Thibault A, Petiau C, Krieger J. Upper airway collapsibility and cephalometric variables in
- patients with obstructive sleep apnea. Am J Respir Crit Care Med 2000;161:347-52
- 21. Rodenstein DO, Dooms G, Thomas Y, Liistro G, C.Stanescu D, Culee C, et al. Pharyngeal shape and dimensions in healthy subjects, snorers, and patients with obstructive sleep apnoea. Thorax 1990;45:722-
- 22. Rama AN, Tekwani SH, Kushida C.A. Sites of Obstruction in Obstructive Sleep Apnea. Chest 2002;122;1139-47.
- 23. Battagel JM, Johal A. A cephalometric comparison of nor-*Radiography* 2000;6:283–92.
- 24. Faber CE, Grymer L. Available techniques for objective assessment of upper airway narrowing in snoring sleep apnea. Sleep Breath 2003;7:77-86.

- 25. Schwab JR, Pasirstein M, Pierson R. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. Am J Respir Crit Care Med 2003;168:522-30.
- 26. Tangugsorn V, Skatvedt O, Krogstad O, Lyberg T. Obstructive sleep apnoea: a cephalometric study. Part II. Eur J Orthodontic 1995,17:57-67.
- 27. Kollias I, Krogstad O. Adult craniofacial and pharyngeal changes - a longitudinal cephalometric study between 22 and 42 of age. Part II: morphologycal uvulo-glosospharyngeal changes. *Eur J Orthodontic* 1999;21:345-55.
- 28. Ryan CF, Dickson RI, Lowe AA, Blokmanis A, Fleetham JA. Upper airway measurements predict response to uvulopalatopharyngoplasty in obstructive sleep apnea. Laryngoscope 1990;100:248-53
- 29. Gislason T, Lindholm CE, Almqvist M, Birring E, Boman G, Eriksson G, et al. Uvulopalatopharyngoplasty in the sleep apnea syndrome. Arch Otolaryngol Head Neck Surg 1988;114:45-51.
- 30. Janson C, Gislason T, Bengtsson H, Eriksson G, Lindberg E, Lindholm CE,, et al. Long-term follow-up of patients with obstructive sleep apnea treated with uvulopalatopharyngoplasty. Arch Otolaryngol Head Neck Surg 1997;123:257-62.

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