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Craniofacial structure in patients with obstructive sleep apnoea

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Background: *Obstructive sleep apnoea (OSA) is characterised by at least five 10-s episodes of apnoea or markedly shallow breathing per 1 h of sleep, which can lead to severe, sometimes life-threatening complications. It is essential to determine the specific features of the affected patients' craniofacial structure, thus enabling their allocation to risk groups. The aim of the study was to assess the craniofacial structure in OSA patients, comparing the findings with Hasund's and Segner's cephalometric normal values. In addition, the sagittal dimensions of the upper airways, measured at two levels, were compared to McNamara's normal values.*

Materials and methods: *The study covered 41 patients diagnosed polysomnographically with OSA. Lateral cephalograms with cephalometric analysis and the measurements of the upper and lower sagittal dimensions of the upper airways were taken for each patient.*

Results: *The only feature of the patents' facial skeleton that significantly diverged from the normal range was the SNB angle (p = 0.004). Other angles, i.e. SNA, ANB, NL/NSL, NL/ML and NSL/ML, were not significantly different from normal. The average upper cross-sectional area of the upper airways was 10.4 mm; in 97.6% patients, this measurement was below McNamara's normal values. In the majority of patients (75.6%), the average lower sagittal dimension of the upper airways (10.4 mm) was also below the normal.*

Conclusions: *Mandibular retrognathia, manifested by the reduced SNB angle, and the narrowed upper and lower sagittal dimensions of the upper airways can be considered one of OSA prognostic factors.* (Folia Morphol 2016; 75, 3: 311–315)

Key words: obstructive sleep apnoea, cephalometric analysis, airway

INTRODUCTION

Obstructive sleep apnoea (OSA) is a disease characterised by at least five 10-s episodes of apnoea or overly shallow breathing (hypopnoea) per 1 h of sleep, accompanied by a decrease in the arterial blood oxygen saturation by 2–4%, with preserved or even intensified respiratory muscle movements. Hypopnoea is characterised by a decreased airflow in the airways by over 50% (compared to the 2-min period of stable respiratory rhythm) lasting over 10 s or leading to a blood saturation decrease by more than 4% [19]. OSA is diagnosed in 4% of men and 2% of women, in whom the disease develops later, usually after menopause. The disease occurs more frequently in men than in women due to anatomic differences: the longer airway and the related increased predisposition to pharyngeal collapse and greater neck fat deposition [15, 32].

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The severity of OSA is measured using the apnoea-hypopnoea index (AHI), which indicates the average number of apnoeas and hypopnoeas per 1 h of sleep [9, 21, 34].

The aetiology of OSA is multi-factorial. The risk factors include obesity, anatomic and post-inflammatory upper airway abnormalities, inadequate sleep hygiene, as well as maxillary and mandibular hypoplasia [12]. The most common symptoms of OSA include: snoring, gasping or choking at night, frequent sleep pauses, impaired daytime concentration and attention, fatigue or non-restorative sleep [2, 5, 10, 33].

Repeated apnoeas causing a decrease in saturation lead to severe systemic disorders, including life-threatening diseases, such as arterial and pulmonary hypertension, arrhythmias, metabolic disturbances and health consequences of sleepiness-related traffic accidents [4, 6, 7, 13, 31].

Patients with OSA are at higher risk of death; therefore, it is essential to thoroughly analyse OSA risk factors, including the characteristic features of the craniofacial structure. The determination of OSA-related, prognostic anatomic characteristics enables both earlier implementation of prophylactic measures, including education of high-risk patients, and causal treatment, instead of the presently given symptomatic one.

The aim of the present study was to assess the craniofacial structure in OSA patients against the normal values according to Hasund and Segner, and also against the normal upper and lower sagittal dimensions of upper airways according to McNamara. Moreover, the characteristic features of the bony structures in question were determined.

MATERIALS AND METHODS

Medical records of 41 patients with polysomnographically diagnosed OSA (AHI > 4) were analysed. The patients were referred to the Department of Jaw Orthodontics, Medical University of Lublin, Poland in the years 2003–2012 for orthodontic OSA treatment. The patients' records included data regarding age and gender, body mass index (BMI), polysomnographic findings (AHI) and lateral cephalograms with cephalometric analysis; the measurements of the upper and lower pharyngeal depth were marked on the cephalograms (Figs. 1, 2).

The study group included 7 (17%) women and 34 (83%) men aged 38–74 years (the mean age was 57.6 years). The largest group, i.e., 75% of patients, were 50–65 years old. AHI was 9.1–80.6, on average 30.8, episodes of apnoea and hypopnoea per 1 h of sleep. Seven (17%) patients had mild OSA, 16 (39%) — moderate OSA and 18 (44%) — severe OSA. The sex-related differences in the study group with diagnosed OSA, expressed in AHI, have been analysed.

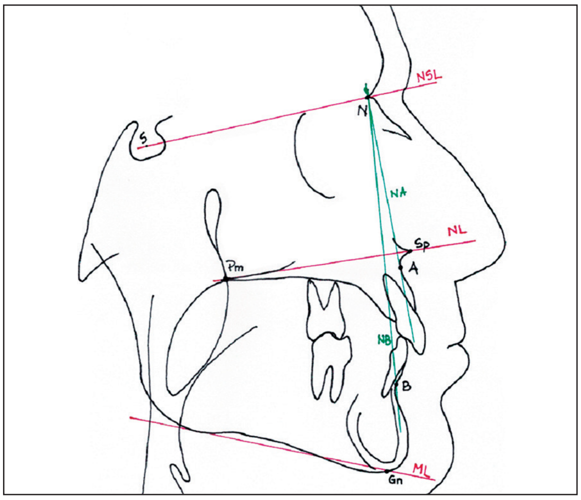

Figure 1. Points and planes used for cephalometric analysis according to Hasund and Segner; S — centre of the sella turcica; N — the most anterior point of the frontonasal suture; Sp — the most anterior point of the anterior nasal spine; A — the deepest point on the anterior outline of the maxillary alveolar process; Pm — the intersection point of the posterior outline of the maxillary body and the outline of the hard and soft palate; B — the deepest point on the anterior outline of the mandibular alveolar process; Gn — the lowest point on the mandibular symphysis. Planes: NSL — denoting the anterior cranial fossa; NL — denoting the maxillary base; ML — denoting the mandibular body; NA — used to describe the sagittal positioning of the maxilla; NB — used to describe the sagittal positioning of the mandible.

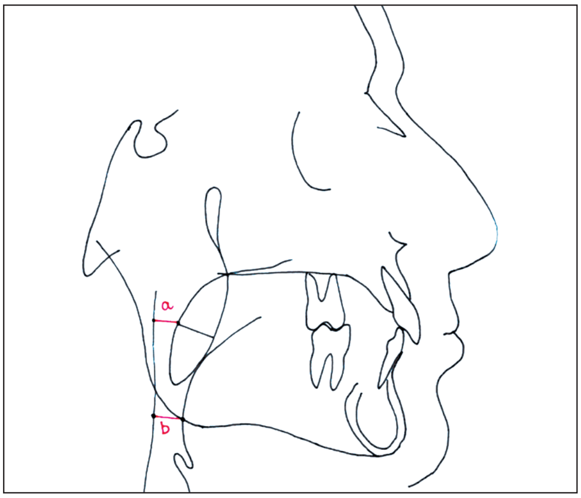

Figure 2. Measurements of the upper airway sagittal dimensions at the soft palate and tongue base levels according to McNamara; segment a — at the palate level; segment b — at the tongue level.

The BMI of the examined patients was 24–41, on average 30.9. No patient was found with underweight; 3 (7%) patients had normal body weight. Other patients were overweight, including 22 (54%) patients classified as obese people. In this group, 2 people had a BMI above 40, which is categorised as extreme obesity people (grade III).

Cephalometric analysis was carried out according to Hasund's and Segner's guidelines (markings and measurements were performed by the same examiner). The results were compared with the cephalometric normal range determined by these researchers (Table 1) [28]. The dimensions of the upper and lower cross-sections of upper airways were measured using the McNamara method and compared with his normal values (Table 1) [17].

Table 1. Range of normal values according to Hasund and Segner, and McNamara

Angles and measurements	Normal range
SNA: Anterior-posterior positioning of the maxilla	82 ± 3º
SNB: Anterior-posterior positioning of the mandible	80 ± 3º
ANB: Skeleton class	2 ± 2º
NSL/ML: Mandibular rotation	28 ± 6º
NSL/NL: Maxillary rotation	8 ± 4º
NL/ML: Inter-maxillary angle	20 ± 7º
∅↑: Upper cross-section of upper airways	17–20 mm
∅↓: Lower cross-section of upper airways	12–13 mm

Statistical analysis

The examined variables were quantitative. The following descriptive parameters were calculated: mean, standard deviation (SD), minimum and maximum value, median (Me) (Table 2). Relationships were tested with the Pearson's r correlation coefficient used to measure the relationship between two variables. The 5% risk of error was assumed. Calculations were performed using Statistica 8.0 software.

RESULTS

Apnoea-hypopnoea index for men ranged from 9.1 to 80.6, the mean AHI value was 32.56 ± 18.89, and in half of the men it did not exceed 27.95 (Me = 27.95). For women AHI ranged from 11.6 to 31.1, the mean AHI value was 22.23 ± 8.82, and in half of the female group it did not exceed 24.7 (Me = 24.7). The observed differences were not statistically significant (t-test with a grouping variable t = 1.40, df = 39, p = 0.17).

The comparison of facial skeleton measurements in OSA patients with the cephalometric normal values according to Segner and Hasund revealed that only the SNB angle, representing the anterior-posterior positioning of the mandible, statistically significantly differed from normal (p = 0.004). The remaining angles, i.e. SNA, ANB, NL/NSL, NL/ML and NSL/ML, were not significantly different from the cephalometric normal range. The distribution of cephalometric measurements in relation to the normal range is presented in Table 3. The upper sagittal dimension of the upper airways ranged from 6 mm to 17 mm (10.4 mm, on average). The above measurement was lower than the norm by McNamara in almost all the patients (40–97.6%). In 1 patient, the measurement

Table 2. Descriptive parameters of variables

Variable	Descriptive parameters					
	N	Mean	Median	Minimum	Maximum	SD
AHI	41	30.8	27.9	9.1	80.6	17.9
SNA	41	80.8	80.8	71.3	89.6	4.2
SNB	41	78.1	78.0	68.4	87.8	3.9
ANB	41	2.7	2.2	–1.7	9.0	2.5
NL/NSL	41	7.8	8.3	–1.7	15.9	3.6
NL/ML	41	20.8	20.0	7.0	34.0	7.2
NSL/ML	41	28.4	28.6	11.2	45.5	7.6
∅↑	41	10.4	11.0	6.0	17.0	2.6
∅↓	41	10.4	10.0	5.0	19.5	3.1

AHI — apnoea-hypopnoea index; SD — standard deviation; remaining abbreviations as in Table 1

Table 3. Distribution of cephalometric findings in relation to norm values.

Angle	Number of individuals above the normal range (%)	Number of individuals within the normal range (%)	Number of individuals below the normal ranges (%)
SNA	6 (14.6%)	24 (58.6%)	11 (26.84%)
SNB	4 (9.8%)	21 (51.2%)	16 (39%)
ANB	10 (24.4%)	26 (63.4%)	5 (12.2%)
NL/NSL	4 (9.8%)	32 (78%)	5 (12.2%)
NL/ML	19 (46.3%)	9 (22%)	13 (31.7%)
NSL/ML	8 (19.5%)	25 (61%)	8 (19.5%)

Abbreviation as in Table 1

was within the normal values. The lower sagittal dimension of the upper airways ranged from 5 mm to 19.5 mm (10.4 mm, on average) and was found to be lower than normal in the majority of patients (31–75.6%); in 2 patients this dimension was within the normal range, while in 8 patients it was higher than normal.

DISCUSSION

Obstructive sleep apnoea is a disease of multifactorial aetiology. In a vast majority of cases, it develops when several factors contributing to the upper airways collapse occur simultaneously. The tone of muscles involved in the maintenance of airway patency markedly decreases during sleep. The size of the nasopharyngeal lumen depends then, to a large degree, on the structure of pharyngeal tissues, facial skeleton and cross-sectional sizes of the upper airways. The factors reducing the cross-sectional area lead to the development of OSA. The above is confirmed by the reports on apnoea in patients with neoplastic tumours of the upper airways [11], hypertrophied tonsils [29], and macroglossia [8, 20]. The size of the upper airways cross-sectional area is also related to the craniofacial structure, which was demonstrated in earlier reports concerning narrowed cross-sections of the pharynx in patients with extreme micrognathia [27]. In our population of patients, the comparison of craniofacial structure with the normal values according to Hasund and Segner showed that the only characteristic that statistically significantly differed from the normal range was the SNB angle, describing the anterior-posterior location of the mandible. The SNB angle size is smaller in OSA patients with abnormal posterior positioning of the mandible, which increases a tendency to upper airway collapse. According to Rivlin et al. [26], patients with OSA are characterised by abnormal positioning of the mandible, which correlates with AHI [14, 16, 18, 26]. The decreased sizes of the SNB angle in patients with OSA were also found by other authors [14]. Moreover, Lowe et al. [14] reported the decreased values of the SNA angle. Another characteristic of OSA patients is a reduced sagittal dimension of the upper airway. Compared with the normal values according to McNamara, 97.6% of our patients with OSA had reduced sagittal dimensions at the soft palate level, whereas 75.6% — at the tongue base level [18]. The reduced values of the sagittal dimensions of the upper airways were reported by many authors [3, 22–25, 30]. Only Abu Allhaija et al. [1] did not find a correlation between the changes in the ANB angle dimensions and anterior-posterior dimensions of the upper airway.

CONCLUSIONS

1. Patients with OSA have abnormal posterior positioning of the mandible (a decreased SNB angle).
2. In patients with OSA the narrowed upper and lower sagittal dimensions of the upper airways were found.
3. The above-described deviations observed in the anatomic craniofacial structure can be considered as one of the prognostic risk factors of OSA. This points out the need for monitoring and possible orthodontic treatment, especially in patients with such abnormalities at the developmental age.

REFERENCES

1. Abu Allhaija ES, Al-Khateeb NS (2005) Uvulo-glosso-pharyngeal dimensions in different anteroposterior skeletal patterns. Angle Orthod, 75: 1012–1018.

2. American Academy of Sleep Medicine Task Force (1999) Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep, 22: 667–689.
3. Bacon W., Turlot JC, Krieger J, Stierle JL (1990) Cephalometric evaluation of pharyngeal obstructive factors in patients with sleep apnea syndrome. Angle Orthod, 60: 115–121.
4. Barreiro B, Garcia L, Lozano L, Almagro P, Quintana S, Alsina M, Heredia JL (2013) Obstructive sleep apnea and metabolic syndrome in Spanish population. Open Respir Med J, 18: 71–76. doi: 10.2174/1874306401307010071.
5. Buda AJ, Schroeder JS, Guilleminault C (1989) Abnormalities of pulmonary artery wedge pressure in sleep-induced apnea. Int J Cardiol, 1: 67–74.
6. Catarino R, Spratley J, Catarino I, Lunet N, Pais-Clemente M (2014) Sleepiness and sleep-disordered breathing in truck drivers: risk analysis of road accidents. Sleep Breath, 18: 59–68. doi: 10.1007/s11325-013-0848-x.
7. Dumitrascu R, Tiede H, Eckermann J, Mayer K, Reichenberger F, Ghofrani HA, Seeger W, Heitmann I, Schulz R (2013) Sleep apnea in precapillary pulmonary hypertension. Sleep Med, 14: 247–251. doi: 10.1016/j.sleep.2012.11.013.
8. Friedel ME, Johnston DR, Singhal S, Al Khalili K, Farrel CJ, Evans JJ, Nyquist GG, Rosen MR (2013) Airway management and perioperative concerns in acromegaly patients undergoing endoscopic transsphenoidal surgery for pituitary tumors. Otolaryngol Head Neck Surg, 149: 840–844. doi: 10.1177/0194599813507236.
9. Guilleminault C, Quera-Salva M, Partinen M, Jamieson A (1988) Women and the obstructive sleep apnea syndrome. Chest, 93: 104–109.
10. Hoffstein V, Szalai JP (1993) Predictive value of clinical features in diagnosing obstructive sleep apnea. Sleep, 6: 118–122.
11. Jung HJ, Kim JW, Lee CH, Chung YJ, Mo J-H (2013) A thyroglossal duct cyst causing obstructive sleep apnea in adult. Clin Exp Otorhinolaryngol, 6: 187–190. doi: 10.3342/ceo.2013.6.3.187.
12. Krieger J (1998) Clinical presentations of sleep apnoea. Eur Respir Mon, 3: 75–105.
13. Levy P, Ryan S, Oldenburg O, Parati G (2013) Sleep apnoea and the heart. Eur Respir Rev, 22: 333–352.
14. Lowe AA, Santamaria JD, Fleetham JA, Price C (1986) Facial morphology and obstructive sleep apnea. Am J Orthod Dentofac Orthop, 90: 484–491.
15. Malthotra A, Huang Y, Fogel RB, Pillar G, Edwards JK, Kikinis R, Loring SH, White DP (2002) The male predisposition to pharyngeal collapse: importanceof airway length. Am J Resp Crit Care Med, 166: 1388–1395.
16. Mayer P, Pepin G, Bettega A, Veale D, Ferretti G, Deschaux C, Levy P (1996) Relationship between body mass index, age and upper airway measurements in snorers and sleep apnoea patients. Eur Respir J, 9: 1801–1809.
17. McNamara JA (1981) Influence of respiratory pattern on craniofacial growth. Angle Orthod, 51: 269–300.
18. McNamara J, Burdon W (1994) Orthodontic and orthopedic treatment in mixed dentition. Needham Press, Ann Arbor, MS.
19. McNicholas WT (2008) Diagnosis of obstructive sleep apnea in adults. Proc Am Thorac Soc, 5: 154–160.
20. Mete T, Yalcin Y, Berker D, Ciftici B, Guven Firat S, Popaloglu O, Cinar Yavus H, Guler S (2013) Relationship between obstructive sleep apnea syndrome and thyroid diseases. Endocrine, 44: 723–728. doi: 10.1007/s12020-013-9927-9.
21. Ojoo JC, Kastelik JA, Mori AH (2001) The respiratory system and the menopause. Menopause Int, 7: 168–173.
22. Ono T, Lowe AA, Ferguson KA, Fleetham JA (1996). Associations between upper airway structure, body position and obesity in skeletal Class I male patients with obstructive sleep apnea. Am J Orthod Dentofac Orthop, 109: 625–634.
23. Özbek MM, Keisuke M, Lowe AA, Fleetham JA (1998) Natural head posture, upper airway morphology and obstructive sleep apnoea severity in adults. Eur J Orthodontics, 20: 133–143.
24. Pae EK, Lowe AA, Fleetham JA (1997) A role of pharyngeal length in obstructive sleep apnea patients. Am J Orthod Dentofac Orthop, 111: 12–17.
25. Pracharktam N, Nelson S, Hans MG, Broadbent BH, Redline S, Rosenberg C, Strohl KP (1996) Cephalometric assessment in obstructive sleep apnea. Am J Orthod Dentofac Orthop, 109: 410–419.
26. Rivlin J, Hoffstein V, Kalbfleisch J, McNicholas W, Zamel N, Bryan AC (1984) Upper airway morphology in patients with idiopathic obstructive sleep apnea. Am Rev Respir Dis, 129: 355–360.
27. Sedaghat AR, Anderson IC, McGinley BM, Rossberg MI, Redett RJ, Ishman SL (2012) Characterization of obstructive sleep apnea before and after tongue-lip adhesion In children with micrognathia. Cleft Palate Craniofac J, 49: 21-26, doi: 10.1597/10-240.
28. Segner D, Hasund A (1996) Indywidualna kefalometria. Med Tour Press International, Wydawnictwo Medyczne, Warszawa.
29. Sung MW, Lee WH, Wee JH, Lee CH, Kim E, Kim JW (2013) Factors associated with hypertrophy of the lingual tonsils in adults in sleep-disordered breathing. JAMA Otolarymgol Head Neck Surg, 139: 598–603. doi: 10.1001/jamaoto.2013.3263.
30. Tangugsorn V, Skatvedt O, Krogstadt O, Lyberg T (1995) Obstructive sleep apnoea: a cephalometric study. Part I. Cervico-craniofacial skeletal morphology. Eur J Orthod, 17: 45–56.
31. Tasali E, Ip MSP (2008) Obstructive sleep apnea and metabolic syndrome: Alteration in glucose metabolism and inflammation. Proceedings ATS, 5: 207–217. doi: 10.1513/pats.200708-139MG.
32. Whittle AT, Marshall I, Mortimore IL, Wraith PK, Sellar RJ, Douglas NJ (1999) Neck soft tissue and fat distribution: comparison between normal men and women by magnetic resonance imaging. Thorax, 54: 323–328.
33. Whyte KF, Allen MB, Jeffrey AA, Gould GA, Douglas NJ (1989) Clinical feature of the sleep apnea/hypopnea syndrome. Q J Med, 72: 659–666.
34. Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S (1993) The occurrence of sleep-disordered breathing among middle-aged adults. N Engl J Med, 328: 1230–1235.