

Respiratory Variables During Wakefulness in Patients with Obstructive Sleep Apnea and Associations with Anthropometric Measurements

Kağan Üçok¹, Abdullah Ayçiçek², Hakan Mollaoğlu¹, Abdurrahman Genç¹, Muzaffer Akkaya¹, Muhsin Toktaş³, Ozan Alper Alkoç³, Mehmet Ünlü⁴

Afyon Kocatepe University, Faculty of Medicine, Departments of Physiology¹, Otorhinolaryngology², Anatomy³, and Pulmonary Medicine⁴, Afyonkarahisar, Turkey.

Eur J Gen Med 2010;7(3):250-258

Received: 30.12.2009

Accepted: 03.03.2010

ABSTRACT

Aim: This study was aimed to investigate the respiratory variables during wakefulness, otorhinolaryngologic and anthropometric differences, and to clarify their possible associations in patients with obstructive sleep apnea syndrome (OSAS).

Method: Apnea-hypopnea index (AHI) was established by overnight polysomnography. Otorhinolaryngologic examination, anthropometric measurements and questionnaire, were performed. Respiratory variables were measured with a cardio-breathing analysis system. Body fat percentages were calculated from the skinfold thicknesses.

Result: Among a total of 83 patients, 43 (32 male and 11 female) patients having an AHI score equal or above 10 were diagnosed as OSA, and 40 (27 male and 13 female) patients having an AHI score under 10 were diagnosed as simple snorers. Apnea with witness, flat soft palate, pharyngeal collapse with Muller maneuver, neck antero-posterior and lateral diameters, thorax antero-posterior diameter, and O₂ pulse values were found significantly higher in OSAS patients than those in simple snorers.

Conclusion: This study suggests that neck antero-posterior and lateral diameters, thorax antero-posterior diameter and O₂ pulse might be predicting factors for OSAS. It also emphasizes that pharyngeal collapse with Muller maneuver can be routine diagnostic procedure in evaluating OSAS patients.

Key words: Obstructive sleep apnea, respiratory variables, anthropometry, Muller maneuver

Correspondence: Dr. Kağan Üçok, Department of Physiology, Faculty of Medicine, Afyon Kocatepe University, 03040, Afyonkarahisar, Turkey

Phone: 902722167901/127

Fax: 902722172029

E-mail: kaganucok@hotmail.com

Uyanık Durumdaki Obstrüktif Uyku Apne Sendromlu Hastalarda Solunumsal Değişkenler ve Antropometrik Ölçümler

Amaç: Bu çalışmada, uyanık durumdaki obstrüktif uyku apne sendromlu hastalardaki solunumsal değişkenlerin, kulak-burun-boğaz ve antropometriyle ilgili farklılıklarının araştırılması ve bunların olası ilişkilerinin aydınlatılması amaçlanmıştır.

Metod: Apne-hipopne indeksi (AHI) polisomnografi ile belirlendi. Kulak-burun-boğaz muayenesi, antropometrik ölçümler ve anket uygulamaları gerçekleştirildi. Solunumsal değişkenler kalp-solunum analiz sistemi ile ölçüldü. Vücut yağ yüzdesi deri kıvrım kalınlıklarından hesaplandı.

Bulgular: Toplam 82 katılımcıdan, AHI skoru 10 ve üzeri olan 43 hasta (32 erkek, 11 kadın) obstrüktif uyku apne sendromu ve AHI skoru 10'un altında olan 40 hasta (27 erkek, 13 kadın) basit horlama tanısı aldı. Obstrüktif uyku apne sendromlu hastalarda tanıklı apne, yaygın yumuşak damak ve Müller manevrasıyla oluşan kollaps, boyun ön-arka ve lateral çapları, göğüs ön-arka çapı ve 'dakika O₂ alımı/kalp hızı' değeri basit horlamalılara göre yüksek bulundu.

Sonuç: : Bu çalışma ile obstrüktif uyku apne sendromlu hastalarda boyun ön-arka ve lateral çapları, göğüs ön-arka çapı ve 'dakika O₂ alımı/kalp hızı' değerinin hastalık prediktörleri olabileceği ileri sürüldü. Aynı zamanda, Müller manevrasıyla oluşan kollapsın obstrüktif uyku apne sendromlu hastalarının rutin muayenesinde tanı amaçlı kullanılabileceği bildirildi.

Anahtar kelimeler: Ostrüktif uyku apne, solunumsal değişkenler, antropometri, Müller manevrası.

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a common and often life-altering sleep-related breathing disorder (1). Patients with OSAS have many episodes of increased airway resistance because of repeated collapses of upper airways during night (2). OSAS leads to excessive daytime sleepiness, cognitive dysfunction, impaired work performance, and decrements in health-related quality of life (3). OSAS is associated with increased risk for cardiovascular diseases including hypertension, myocardial ischemia and left ventricular hypertrophy (4).

In the last years a great deal of attention has been paid to this problem due to the fact that the high prevalence of this disease in the general population turns this into a true public health problem (5). Young et al. (6) claimed that the proportion of OSAS patients in the general adult population goes undiagnosed. They estimated that 93% of women and 82% of men with moderate to severe OSAS have not been clinically diagnosed without obvious barriers to health care for sleep disorders. Knowledge of risk factors for obstructive sleep apnea is crucial to properly direct diagnostic attention at those with the highest risk (7). Major risk factors for OSAS are age, excess body weight, sex, race, craniofacial and neck anatomy, familial and genetic predisposition, medical comorbidity, and others (3). Structural features such as retrognathia, tonsillar hypertrophy, enlarged tongue or soft palate, inferiorly positioned hyoid bone, maxillary and mandibular retroposition, and decreased posterior airway space can narrow upper airway dimensions and promote the occurrence of apneas and hypopneas during sleep (8).

Respiratory variables were usually investigated during sleep but not awake in patients with OSAS. Nevertheless, changed respiratory parameters in wakeful patients might be used easily as an indicator of cardio-pulmonary status of OSAS patients.

The aims of present study were to investigate the respiratory variables during wakefulness, anthropometric and otorhinolaryngologic differences, and to clarify possible relationships of them in patients with OSAS.

MATERIALS AND METHODS

Patients

Eighty three patients (59 males, 24 females), who were referred to departments of Otorhinolaryngology and Pulmonary Medicine with suspected OSAS were included consecutively in the study. The study protocol was approved by the Local Ethics Committee, and all patients gave written informed consent to participate in the study. Patients who had craniofacial abnormality, retrognathism, cardiovascular diseases -particularly those who use beta blockers- metabolic disorders such as diabetes mellitus were also excluded from the study.

Otorhinolaryngologic examination

Nasal examination was made using nasal speculum and endoscope. Nasal pathologies (septum deviation, hypertrophy of medium or inferior concha, concha bullosa, nasal polyps, and abnormality of internal and external valves) which block air flow in nasal passage were examined.

Table 1. Apnea-hypopnea index (AHI), clinical examination findings and questionnaires of patients.

	Simple Snorers	OSAS Patients	p value
AHI	3.4±2.3	37.9±23.8	0.000
Age	46.2±8.5	48.9±7.5	0.122
Apnea with witness, %	72.7	97.6	0.002
Nasal obstruction, %	25.8	41.5	0.167
Flat soft palate, %	41.9	82.9	0.000
Wide uvula, %	52.4	58.5	0.644
Pharyngeal collapse (Muller maneuver), %	29.0	95.1	0.000
Smoking (packed-year)	27.3±13.7	18.1±11.6	0.061

Soft palate and uvula were examined when the mouth was open and without pulling the tongue out. Flat, wide and flabby soft palate and uvula which change retro-palatinal distance were evaluated and decided by an expert otolaryngologist (AA) as a result of physical examination. Patients were classified into two groups in terms of nasal obstruction, flat soft palate, wide uvula, and pharyngeal collapse as a result of Muller maneuver. Muller maneuver: Flexible endoscope was inserted into the nose. Patients were instructed to perform forced inspiration with their nose and mouth covered. Pharyngeal intraluminal negative pressure was increased with this maneuver and the velopharyngeal and hypopharyngeal collapse was observed using a flexible endoscope (5). Apnea with witness was determined as apneas by a witnessed bed partner.

Polysomnography

An overnight sleep study was performed in all patients using 18-channel polysomnography (SleepScreen Pro®, Viasys Healthcare, Hoechst, Germany), which included a standard montage of electroencephalogram (EEG), electrooculogram and electromyogram signals together

with pulse oximetry, respiratory impedance and nasal airflow detected by oronasal thermistors. All sleep studies were manually analyzed using computer software by the same physician, who was blinded to the patient's other clinical data. Sleep stages were scored according to the standard criteria of Rechtschaffen and Kales (9). Apneas were defined as complete cessation of airflow for at least 10 s. Hypopnea was defined as $\geq 50\%$ reduction in airflow accompanied by $> 3\%$ desaturation in the following 30 s and a reduction in chest wall movement. EEG arousals were not required to make the diagnosis of a respiratory event. Data were expressed as the apnea-hypopnea index (AHI) based on the number of apneas and hypopneas per hour slept, with an AHI ≥ 10 events/h indicating a positive OSAS diagnosis (10).

Respiratory variables

The following respiratory variables "tidal volume (VT), respiratory frequency (Rf), ventilation (VE), O_2 uptake (VO_2), CO_2 production (VCO_2), ventilatory equivalent for O_2 and CO_2 (VE/VO_2 and VE/VCO_2), respiratory quotient (VCO_2/VO_2), O_2 pulse (VO_2 /heart rate), heart rate (HR), averaged expiratory concentration of O_2 and CO_2 (FeO_2

Table 2. Anthropometric measurements of patients.

	Simple Snorers	OSAS Patients	p value
BMI (kg/m ²)	31.6±5.2	33.0±5.6	0.221
Fat %	26.1±7.1	25.4±7.2	0.660
Neck antero-posterior diameter (cm)	11.5±1.6	12.7±1.6	0.001
Neck lateral diameter (cm)	11.2±2.0	12.1±1.7	0.032
Mentum-incisura thyroidea superior distance (cm)	6.4±1.0	6.3±1.4	0.689
Mentum-incisura jugularis distance	11.2±1.4	10.8±1.9	0.284
Thorax lateral diameter (cm)	31.7±6.1	33.3±3.5	0.162
Thorax antero-posterior diameter (cm)	21.3±4.1	24.1±3.3	0.001
Biacromial length (cm)	39.1±7.2	41.2±3.4	0.087

*BMI: Body mass index

Table 3. Respiratory variables of patients.

	Simple Snorers	OSAS Patients	p value
VT (tidal volume) (L)	0.7±0.5	0.7±0.4	0.660
Rf (respiratory frequency) (b/min)	16.3±3.9	16.8±4.3	0.582
VE (ventilation) (L/min)	10.7±6.7	12.0±6.3	0.388
VO ₂ (O ₂ uptake) (ml/min)	203.9±102.1	243.9±87.3	0.058
VCO ₂ (CO ₂ production) (ml/min)	193.2±153.7	225.6±92.7	0.244
VE/VO ₂ (ventilatory equivalent for O ₂)	50.0±32.2	44.4±19.6	0.339
VE/VCO ₂ (ventilatory equivalent for CO ₂)	51.5±16.2	47.2±12.1	0.172
VO ₂ /kg (ml/min/kg)	2.4±1.1	2.7±0.8	0.244
R (respiratory quotient=RQ) (VCO ₂ /VO ₂)	0.93±0.29	0.91±0.18	0.779
FeO ₂ (averaged expiratory concentration of O ₂) (%)	17.1±1.2	17.0±1.1	0.667
FeCO ₂ (averaged expiratory concentration of CO ₂) (%)	3.2±0.8	3.4±0.8	0.315
VO ₂ /Heart Rate (O ₂ pulse) (ml/bpm)	2.7±1.6	3.7±1.7	0.019
Heart Rate (bpm)	64.1±12.8	62.1±9.5	0.675
FetO ₂ (end tidal O ₂) (%)	15.6±1.5	15.7±1.6	0.586
FetCO ₂ (end tidal CO ₂) (%)	4.5±1.3	4.6±1.1	0.613
petO ₂ (end tidal PO ₂) (mmHg)	97.1±9.1	98.4±9.9	0.542
petCO ₂ (end tidal CO ₂) (mmHg)	27.4±8.1	27.8±6.8	0.809
FiO ₂ (final inspiratory O ₂ fraction) (%)	20.1±0.2	20.9±0.1	0.089
FiCO ₂ (final inspiratory CO ₂ fraction) (%)	0.05±0.02	0.05±0.03	0.427
Ti (Duration of inspiration) (sec)	1.6±0.6	1.5±0.5	0.462
Te (Duration of expiration) (sec)	2.4±0.8	2.4±0.7	0.977
Ttot (Duration total breathing cycle) (sec)	4.0±1.3	3.9±1.1	0.764
Ti/Ttot	0.4±0.05	0.4±0.06	0.387

and FeCO₂), end tidal O₂ and CO₂ percentages (FetO₂ and FetCO₂), end tidal PO₂ and PCO₂ (petO₂ and petCO₂), final inspiratory O₂ and CO₂ fractions (FiO₂ and FiCO₂), durations of inspiration (Ti), durations of expiration (Te), durations of total breathing cycle (Ttot) were measured using a breathing analysis system (Quark b2, Cosmed S.r.l., Italy) and chest belt telemetry (heart rate monitoring) system (Polar CR2032, CE0682, Monark Exercise AB, Sweden). The subjects were instructed to

avoid food intake 4 hours, no smoking 2 hours and not perform exercise 24 hours before the test. The tests were performed at the same hours (0830-1030) of the day. After resting for 15 minutes, the measurements were applied to the subjects in the laboratory which was silent and at room temperature. The device was calibrated prior to each test. The subjects were put on facemask, lay on supine position and did not move their arms and legs during the test. Respiratory variables re-

Table 4. The correlations with AHI in subjects.

	r value	p value
Age	0.247	0.024
BMI	0.207	ns
Fat %	0.004	ns
Neck A-P Diameter	0.469	0.000
Neck Lateral Diameter	0.321	0.003
Thorax A-P Diameter	0.355	0.001
Thorax Lateral Diameter	0.261	0.017
Apnea with witness	0.296	0.011
Flat soft palate	0.266	0.024
Muller maneuver	0.538	0.000
VO ₂	0.227	0.039
O ₂ Pulse	0.342	0.002
FiCO ₂	0.231	0.036

*ns: Not significant

Table 5. The correlations with O₂ pulse in patients.

	r value	p value
Age	0.088	NS
BMI	0.033	NS
Fat %	0.136	NS
Neck A-P Diameter	0,568	0.000
Neck Lateral Diameter	0,472	0.001
Thorax A-P Diameter	0,498	0.001
Thorax Lateral Diameter	0,351	0.021
Biacromial length (cm)	0,344	0.024

*NS: Not significant

lated O₂ and CO₂ were measured breath by breath for 15 minutes by analyzing the respired gases using a metabolic card. The mean values for 15 minutes were calculated with Excel programme.

Length and diameter measurements

Anthropometric points were fixed and the length and diameter measurements were performed by the same physician all the time. Mentum-incisura tiroidea superior (M-ITS) and mentum-incisura jugularis (M-IJ) lengths were measured at anatomical position using a caliper compass. Diameter measurements of neck and chest were carried out at anatomical position with an anthropometric set (Harpenden, Holtain Ltd., UK).

Determination of body fat percentage

The skinfold thickness measurements were carried out by the same physician using skinfold caliper (Holtain, Holtain Ltd., UK). The skinfold was picked up between the thumb and the index finger so as to include thicknesses of skin and subcutaneous fat (11). Abdomen, triceps, thigh and subscapular skinfold thickness measurements were done twice. When the differences between the two measurements were more than 5 %, the measurements were repeated. Body densities were calculated with Behnke Wilmore (BW) formulas for both men and women (12).

BW formula for calculation of body density for men was;

Body density = 1.08543 - 0.00086 (abdomen skinfold) - 0.0004 (thigh skinfold)

BW formula for calculation of body density for women was;

Body density = 1.06234 - 0.00068 (subscapular skinfold) - 0.00039 (triceps skinfold) - 0.00025 (thigh skinfold)

Body fat percentage was calculated from body density with Siri formula, which is:

Body fat percentage = (4.95/ body density - 4.5)*100

Statistical analysis

The results were presented as mean values±standard deviation. Chi-square test, Mann Whitney-U test, Pearson correlation test, and linear regression analysis were used. The significance level was determined as p<0.05.

RESULTS

Of the total 83 patients, 43 (32 male and 11 female) having an AHI score ≥ 10 were diagnosed as OSAS, and 40 (27 male and 13 female) having an AHI score < 10 were diagnosed as simple snorers. Table 1 shows the mean values of AHI, age, clinical examination and questionnaire findings and smoking status of simple snorers and OSAS patients. AHI, apnea with witness, flat soft palate, and pharyngeal collapse with Muller maneuver values were found significantly higher in OSAS patients than those in simple snorers. The mean values for anthropometric measurements of simple snorers as well as OSAS patients can be seen in Table 2. Neck antero-posterior and lateral diameters, thorax antero-posterior diameter were found significantly higher in OSAS patients than that in simple snorers. Table 3 shows the mean values of respiratory variables for simple snorers and OSAS patients. Only the mean values for O₂ pulse was found to be significantly higher in OSAS patients than the ones in simple snorers.

In Table 4, the correlations observed with AHI in subjects were given. That is, only pharyngeal collapse with Muller maneuver was found to be strongly correlated with AHI. Linear regression analysis of the measurements revealed that pharyngeal collapse with Muller maneuver (OR=20.7, CI=10.3-31.0, P=0.000) value was the most important predictor of OSAS.

Table 5 shows the correlations observed with O₂ pulse in patients. Biacromial length, and thorax and neck antero-posterior and lateral diameters were significantly correlated with O₂ pulse in OSAS patients. But no correlation was found between O₂ pulse and anthropometric measurements in simple snorers.

DISCUSSION

The abnormal properties of respiratory movement were shown during apneic sleep in OSAS patients (13). Is it possible that abnormal breathing during sleep might affect respiratory variables after awakening in patients with OSAS? Ashraf et al. (14) analyzed spirometric indices of forced expiratory volume in 1 second / forced expiratory volume, maximum mid-expiratory flow, peak expiratory flow, forced expiratory flow 50, and forced inspiratory flow 50 in 138 patients with OSAS. They reported that spirometric abnormalities were not common in OSAS patients. Radwan et al. (2) postulated that

OSAS patients have impaired respiratory compensation of additional inspiratory load, which was demonstrated during hypercapnic rebreathing test. Fuse et al. (15) examined ventilatory responses to hypoxia and to hypercapnia before and after sleep of 19 OSAS patients and 12 normal subjects. They found that the values of resting ventilation, minute ventilation at arterial oxygen saturation of 80% in hypoxia and minute ventilation at end-tidal partial pressure of CO₂ in arterial blood of 60 mmHg in hypercapnia were significantly smaller in after sleep than those in before sleep; also end-tidal partial pressure of CO₂ in arterial blood rose significantly from before sleep to after sleep in the OSAS group. They suggested a hypothesis that repeated episodes of nocturnal hypoxia and hypercapnia may modify the regulation of ventilation after awakening in patients with OSAS (15). In light of above literatures, we planned to investigate the respiratory variables comprehensively, and as a result of the study, we found that O₂ pulse values elevated during wakefulness in patients with OSAS. We also investigated some anthropometric measurements for craniofacial, neck and chest regions, and performed otorhinolaryngologic examination. In current study, we found as a first time that increased thorax antero-posterior diameter value was significantly higher in OSAS patients than that in simple snorers.

It was shown that, respiratory timing is modified in patient with OSAS during sleep (16-18). Stoohs and Guilleminault (18) monitored and compared respiratory timing parameters of eight OSAS patients awake and during nocturnal sleep. They found an immediate increase in Ti, Te and Ttot with the onset of snoring. Also, through the duration of the snoring period, a further increase in Ti and a decrease in Te were noted, with a mean change in Ti/Ttot (18). Bittencourt et al. (19) studied moderately obese, sleep-deprived OSAS patients presenting daytime hypersomnolence, with normocapnia and no clinical or spirometric evidence of pulmonary disease. They found that average rest (wakeful) ventilatory response, inspiratory occlusion pressure, and ventilatory pattern (VT/Ti, Ti/Ttot) were within the normal range. Except for respiratory timing, other respiratory variables during wakefulness were not investigated in patients with OSAS previously. We found that the respiratory variables studied excepting O₂ pulse were not significantly different OSAS patients than those in simple snorers (Table 3). O₂ pulse reflects the capacity of the heart to deliver O₂ per heartbeat.

O₂ pulse, as an important index of cardiovascular efficiency, is closely related to health and cardiopulmonary function (20). Lavie et al. (21) used peak O₂ pulse and lean body mass-adjusted O₂ pulse for predicting prognosis in patients with systolic heart failure. We found that O₂ pulse value was significantly higher in OSAS patients than the one in simple snorers (Table 3). In addition, It was correlated with AHI (Table 4). In this study, interestingly, O₂ pulse was statistically different between OSAS patients and simple snorers, but VO₂, VO₂/kg, VCO₂ and HR values were not (Table 3). Some other factors might affect O₂ pulse in OSAS patients both at apneic and non-apneic conditions. We think that elevated O₂ pulse in OSAS patients could be due to increased sympathetic activity and respiratory effort. Insidious changes such as increased sympathetic activity within the respiratory and cardiovascular systems occur over time in OSAS (22,23). Sympathetic over-activity at both acute and diurnal (non-apneic) conditions were demonstrated with urinary and plasma catecholamines, and intra-neuronal recordings of muscle sympathetic nerve activity in patients with OSAS (24). Increased respiratory effort may cause to develop of anaerobic muscle metabolism in OSAS patients. Bonanni et al. (25) found that exercise lactate levels were significantly higher, and an earlier lactate threshold was detected, in the patients with OSAS compared to normal controls. Saulea et al. (26) applied a needle biopsy of the quadriceps femoris and found that the diameter of type II fibres and protein content were higher in patients with OSAS than healthy controls. The type II fibres of skeletal muscle uses mainly anaerobic metabolism. Uçok et al. (27) found that aerobic capacity was significantly lower in OSAS patients than in controls. These findings in the literature mentioned above (25-27) might be interpreted that the aerobic capacity was lower and the anaerobic metabolism was more developed in the patients with OSAS than normal subjects. Lactic acid, a product of anaerobic metabolism, largely is broken down with using O₂ during non rapid muscle activity. However, it seems difficult to establish a direct link between metabolic status and O₂ pulse in OSAS patients. We suggest that, possibly the finding of elevated O₂ pulse might be an indicator for OSAS during wakefulness for the first time. It is accepted that some morphological differences are associated with the narrowing of the upper respiratory tract in patients for OSAS. During physical examination of patients with suspected OSAS, a comment is frequently

made that they appear to have a short and fat neck (7). Iriundo Bedialauneta et al. (5) said that in a first visual exam, we should focus on the patient's morphotype, very probably that of an obese individual, with a short, thick neck. They also found that patients with OSAS have a greater neck circumference, and more frequent pharyngeal collapse in Muller's maneuver than simple snorers, nonetheless, nasal pathology was not different between OSAS and simple snorer. Neck circumference has been identified as the best predictor of sleep apnea (7,28-31). Similarly, we found that neck antero-posterior and lateral diameters were significantly higher in OSAS patients than those in simple snorers, and they correlated with AHI (Table 2 and 4). We investigated detailed anthropometric measurements of neck and chest. Unexpectedly, the result for M-IJ distance which also reflects neck length was not significantly lower in OSAS patients than that in simple snorers (Table 2). We first show that thorax antero-posterior diameter value was found to be increased in OSAS patients (Table 2). The increase in thorax antero-posterior diameter of patients with OSAS might be considered due to increased respiratory effort during sleep in these patients.

Ozbulut et al. (32) investigated the correlations between 39 anthropometric measurements and pulmonary functions tests in the patients with depressive, anxiety and psychotic disorders. They found that in patients groups, 12 sub-measurements in depressive disorder, and 5 in anxiety disorder were to be correlated significantly with pulmonary functions tests. However, they found no positive correlation between any anthropometric measurements and pulmonary functions tests in patients with schizophrenia. They suggest that these data might be an early indicator of a highly increased tendency for poor health status which might be an additional finding making these patients prone to some illness (32). Despite the fact that this investigation was carried out in a different patient group, we similarly studied the correlations between anthropometric measurements and respiratory variables, and found that O₂ pulse value was significantly correlated with neck and thorax anthropometric measurements in OSAS patients (Table 5) but not in simple snorers. It might be speculated that the functional and anatomical differences may have affected each other during the course of OSAS development.

We found that apnea with witness, flat soft palate and pharyngeal collapse with Muller maneuver values were significantly higher OSAS patients than those in simple

snorers and they correlated with AHI (Table 1 and 4). But only the value of pharyngeal collapse with Muller maneuver was strongly correlated with AHI (Table 4). To our knowledge, it was the most predicting factor for OSAS confirming the previous studies (5,33-35). Nasal continuous positive airway pressure is effective in moderate to severe OSAS patients (36). The patient undergoes evaluation by all the related specialties but the otolaryngologist plays the most important role and the purpose of otolaryngologic evaluation is to diagnose the problems causing the obstructive sleep apnea syndrome besides identifying the problems that may interfere with the use of continuous positive airway pressure devices (37). Apnea with witness, soft palate examination, and especially pharyngeal collapse with Muller maneuver might be used for routine diagnostic procedure in evaluating OSAS patients. Obesity is a potent risk factor for development and progression of sleep apnea, and clearly contributes to the incidence of sleep-disordered breathing (38,39). In our study neither BMI nor fat % values were found correlated with AHI in all patients (Table 4). These might be because of the fact that both simple snorers and OSAS patients were obese (Table 2).

It is known that sleep-disordered breathing is very common and is associated with an increased risk of cardiovascular disease, cardiac arrhythmia and stroke (40). OSAS is insidious and patients are often unaware of the associated symptoms. Compounding the lack of patient awareness, health care professionals in most medical specialties not having received the necessary training to help expedite case finding and institute early intervention; it can be emphasized that early recognition and appropriate therapy can ameliorate the neurobehavioral consequences as well as quality of life and may also have favorable effects on cardiovascular health (3,41,42). The current study might shed on light some new aspects of physio-pathological status of OSAS and might contribute to management of OSAS if it is supported in another set of patients.

In conclusion, we suggest that neck antero-posterior and lateral diameters, thorax antero-posterior diameter might be predicting factors for OSAS. This study also emphasizes that pharyngeal collapse with Muller maneuver can be used as a routine diagnostic procedure in evaluating OSAS patients. We further suggest as first time that elevated O₂ pulse might be a possible cardiopulmonary indicator in patients with OSAS during wakefulness.

REFERENCES

1. Shah N, Roux F, Mohsenin V. Improving Health-Related Quality of Life in Patients with Obstructive Sleep Apnea: What are the Available Options? *Treat Respir Med* 2006;5:235-44.
2. Radwan L, Koziarowski A, Maszczyk Z, et al. Respiratory response to inspiratory resistive load changes in patients with obstructive sleep apnea syndrome. *Pneumonol Alergol Pol* 2000;68:44-56.
3. Punjabi NM. The epidemiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 2008;5:136-43.
4. Leung RST, Bradley TD. Sleep apnea and cardiovascular disease. *Am J Respir Crit Care Med* 2001;164:2147-65.
5. Iriondo Bedialauneta JR, Santaolalla Montoya F, Moreno Alonso E, Martínez Ibargüen A, Sánchez Fernández JM. Analysis of the Anthropometric, Epidemiological, and Clinical Parameters in Patients with Snoring and Obstructive Sleep Apnoea. *Acta Otorrinolaringol Esp* 2007;58:413-20.
6. 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;20:705-6.
7. Katz I, Stradling J, Slutsky AS. Do patients with obstructive sleep apnea have thick necks? *Am Rev Respir Dis* 1990;141:1228-31.
8. Cistulli PA. Craniofacial abnormalities in obstructive sleep apnoea: implications for treatment. *Respirology* 1996;1:167-74.
9. Rechtschaffen A, Kales A. (1968) *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*. Public Health Services, NIH Publication no. 204, US Government Printing Office, Washington DC.
10. Lavie P, Herer P, Hoffstein V. Obstructive sleep apnoea syndrome as a risk factor for hypertension: population study. *BMJ* 2000;320:479-82.
11. Hurson M, Corish C. Evaluation of lifestyle, food consumption and nutrient intake patterns among Irish teenagers. *Ir J Med Sci* 1997;166:225-30.
12. Behnke AR, Wilmore JH. (1974) *Evaluation and Regulation of Body Build and Composition*. Prentice-Hall, Englewood Cliffs, New Jersey.
13. Miyata M, Burioka N, Suyama H, et al. Non-linear behaviour of respiratory movement in obstructive sleep apnoea syndrome. *Clin Physiol Funct Imaging* 2002;22:320-7.
14. Ashraf M, Shaffi SA, BaHammam AS. Spirometry and flow-volume curve in patients with obstructive sleep apnea. *Saudi Med J* 2008;29:198-202.
15. Fuse K, Satoh M, Yokota T, et al. Regulation of ventilation before and after sleep in patients with obstructive sleep apnoea. *Respirology* 1999;4:125-30.
16. Onal E, Lopata M. Respiratory timing during NREM sleep in patients with occlusive sleep apnea. *J Appl Physiol* 1986;61:1444-8.
17. Cibella F, Marrone O, Sanci S, Bellia V, Bonsignore G. Expiratory timing in obstructive sleep apnoeas. *Eur Respir J* 1990;3:293-8.
18. Stoohs R, Guilleminault C. Snoring during NREM sleep: respiratory timing, esophageal pressure and EEG arousal. *Respir Physiol* 1991;85:151-67.
19. Bittencourt LR, Moura SM, Bagnato MC, Gregório LC, Tufik S, Nery LE. Assessment of ventilatory neuromuscular drive in patients with obstructive sleep apnea. *Braz J Med Biol Res* 1998;31:505-13.
20. Wasserman K, Hansen J, Sue DY, Casaburi R, Whipp BJ. (2004) *Principles of exercise testing and interpretation*. 4th ed. Lippincott Williams and Wilkins, Philadelphia.
21. Lavie CJ, Milani RV, Mehra MR. Peak exercise oxygen pulse and prognosis in chronic heart failure. *Am J Cardiol* 2004;93:588-93.
22. Bradley TD, Phillipson EA. Pathogenesis and pathophysiology of the obstructive sleep apnea syndrome. *Med Clin North Am* 1985;69:1169-85.
23. Lee DS. Respiratory and cardiac manifestations of obstructive sleep apnea. *Nurs Clin North Am* 2008;43:55-76.
24. Fletcher EC. Sympathetic over activity in the etiology of hypertension of obstructive sleep apnea. *Sleep* 2003;26:15-9.
25. Bonanni E, Pasquali L, Manca ML, et al. Lactate production and catecholamine profile during aerobic exercise in normotensive OSASS patients. *Sleep Med* 2004;5:137-45.
26. Sauleda J, Garcia-Palmer FJ, Tarraga S, Maimó A, Palou A, Agustí AG. Skeletal muscle changes in patients with obstructive sleep apnoea syndrome. *Respir Med* 2003;97:804-10.
27. Uçok K, Aycicek A, Sezer M, et al. Aerobic and anaerobic exercise capacities in obstructive sleep apnea and associations with subcutaneous fat distributions. *Lung* 2009;187:29-36.
28. Davies RJ, Stradling JR. The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnea syndrome. *Eur Respir J* 1990;3:509-14.
29. Stradling JR, Crosby JH. Predictors and prevalence of obstructive sleep apnea and snoring in 1001 middle aged men. *Thorax* 1991;46:85-90.
30. Dixon JB, Schachter LM, O'Brien PE. Predicting sleep apnea and excessive day sleepiness in the severely obese: indicators for polysomnography. *Chest* 2003;123:1134-41.
31. Lam B, Ip MS, Tench E, Ryan CF. Craniofacial profile in Asian and white subjects with obstructive sleep apnoea. *Thorax* 2005;60:504-10.
32. Ozbulut O, Uçok K, Mollaoglu H, et al. Assessment of anthropometric measurements and pulmonary functions in patients with psychiatric disorder. *Neurol Psychiatr Brain Res* 2007;14:95-101.
33. Naya M, Vicente E, Llorente E, Marín C, Damborenea J. Predictive value of the Muller maneuver in obstructive sleep apnea syndrome. *Acta Otorrinolaringol Esp* 2000;51:40-5.
34. Terris DJ, Hanasono MM, Liu YC. Reliability of the Muller maneuver and its association with sleep-disordered

- breathing. *Laryngoscope* 2000;110:1819-23.
35. Dreher A, de la Chaux R, Klemens C, et al. Correlation between otorhinolaryngologic evaluation and severity of obstructive sleep apnea syndrome in snorers. *Arch Otolaryngol Head Neck Surg* 2005;131:95-8.
 36. Lin CC, Luo SK, Hsu MT, Chen IH, Huang WC. Effects of nasal continuous positive airway pressure on patients with obstructive sleep apnea syndrome. *J Formos Med Assoc* 1991;90:296-9.
 37. Coskun HH. Clinical evaluation in obstructive sleep apnea syndrome: special considerations in physical examination (the otolaryngologist's perspective). *Turkiye Klinikleri J Surg Med Sci* 2007;3:42-4.
 38. Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. *J Appl Physiol* 2005;99:1592-9.
 39. Schwartz AR, Patil SP, Laffan AM, Polotsky V, Schneider H, Smith PL. Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. *Proc Am Thorac Soc* 2008;5:185-92.
 40. Bounhoure JP, Galinier M, Didier A, et al. Sleep apnea syndromes and cardiovascular disease. *Bull Acad Natl Med* 2005;189:445-59.
 41. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005;365:1046-53.
 42. Aktunc E, Altin R, Demircan N, Tor M, Unalacak M, Kart L. The diagnostic effectiveness of three cardinal symptoms in sleep apnea syndrome when asked routinely in out-patient visits. *Eur J Gen Med* 2006;3:121-5.