ORIGINAL ARTICLE

The Effect of Continuous Positive Airway Pressure on Basal Metabolism Rate in Patients with Severe Obstructive Sleep Apnoea Syndrome

T Onyilmaz[1], S Argun Baris[2], H Boyaci[2], I Basyigit[2], G Dogru[3], F Yildiz[2]

ABSTRACT

Objective: *To assess the effect of the continuous positive airway pressure (CPAP) treatment on basal metabolism rate (BMR) in patients with severe obstructive sleep apnoea syndrome (OSAS).*

Methods: *Demographic characteristics, body mass index (BMI), apnoea-hypopnoea index (AHI) and smoking history of the patients were recorded. Basal metabolism rate was measured* via *indirect calorimetry in the morning following nights of polysomnography and CPAP titration. Basal metabolism rate, oxygen consumption (VO_2) and carbon dioxide output (VCO_2) levels were compared before and after CPAP administration.*

Results: *A total of 25 patients with a mean age of 51.4 ± 13.7 years were included in the study: 6 (24%) female and 19 (76%) male. A significant reduction in the BMR (*p *= 0.049), VO_2 (*p *= 0.042) and VCO_2 (*p *= 0.008) values were observed after a single night administration of CPAP as compared to before treatment. Furthermore, it was detected that this reduction provided by CPAP treatment was more significant in current smokers, patients with AHI > 60 and BMI ≥ 30.*

Conclusion: *It is suggested that there is a correlation between BMR and the severity of OSAS, and it is possible to provide a significant reduction in BMR with single night administration of CPAP depending on the patient's smoking history, degree of obesity and disease severity.*

Keywords: Carbon dioxide output (VCO_2), continuous positive airway pressure treatment, obstructive sleep apnoea syndrome, oxygen consumption (VO_2)

INTRODUCTION

Obstructive sleep apnoea syndrome (OSAS) is a sleep disorder that is characterized by recurrent episodes of complete or partial upper airway collapse during sleep in the presence of breathing effort. These episodes are associated with recurrent oxyhemoglobin desaturation (1). Obstructive sleep apnoea syndrome is an independent risk factor for the development of several comorbid conditions, especially cardiovascular and metabolic disorders. Basal metabolism rate (BMR) is defined as the energy consumption required to maintain body functions and metabolic activities. A previous study has shown significantly higher energy consumption in patients with OSAS compared to healthy subjects (2). Undesirable outcomes of high BMR include fatigue, tachycardia, arrhythmia, dyspnoea, insomnia, muscle weakness, and mortality (3).

Obstructive sleep apnoea syndrome should be approached as a chronic disease requiring multi-disciplinary management. Even though there are medical, surgical, behavioural and adjunctive treatment strategies, the positive airway pressure (PAP) is the preferred treatment option (4). Alternative treatments may be considered with respect to the patient's anatomy, risk factors and disease severity. The primary goal of the PAP treatment is to normalize the apnoea-hypopnoea index and

From: [1]Department of Pulmonary Diseases, Mardin Government Hospital, Mardin, Turkey, [2]Department of Pulmonary Diseases, Kocaeli University School of Medicine, Kocaeli, Turkey and [3]Department of Pulmonary Diseases, Isparta Government Hospital, Isparta, Turkey.

Correspondence: Dr S Argun Baris, Department of Pulmonary Diseases, Kocaeli University School of Medicine, Kocaeli, Turkey. Email: serap.baris@kocaeli.edu.tr

 DOI: 10.7727/wimj.2015.489

improve sleep quality, thereby preventing the unfavourable outcomes of hypoxemia and hypercapnia during sleep. Previous studies presented that PAP treatment was associated with significant reductions in cerebrovascular and cardiac adverse events in patients with OSAS (5, 6). However, it is not clear whether it has beneficial effects on increased BMR. The aim of this study is to evaluate the effects of short-term CPAP treatment on BMR in patients with severe OSAS.

SUBJECTS AND METHODS

Thirty patients who were admitted to Kocaeli University School of Medicine, Department of Pulmonary Diseases, Turkey, outpatient clinic with the symptoms of snoring, excessive daytime sleepiness and witnessed apnoeas and who would undergo polysomnography to diagnose OSAS were included in the study. In the morning of polysomnography night, BMR was measured *via* indirect calorimetry. Five patients who were diagnosed with mild and moderate OSAS according to polysomnography were excluded from the study. The BMR was re-measured in the remaining 25 patients with severe OSAS in the morning of the CPAP titration night, and the effect of single night CPAP application on BMR was evaluated.

Demographic characteristics of the patients (including smoking history, comorbid conditions, body mass index (BMI), apnoea-hypopnoea index (AHI) and regularly used medications) were recorded.

Polysomnography records were performed with the Compumedics E series system by hospitalizing the patients for one night (between 11 pm and 8 am) in the Sleep Disorders Center of Kocaeli University School of Medicine, Turkey. In polysomnography, electroencephalography, electromyography of jaw and legs, respiratory movement of chest and abdomen, body position, and airflow of oronasal cannula were recorded. A fingertip pulse oximeter was used to monitor oxygen saturation, and snoring was recorded through a tracheal microphone placed on the neck. The number of both apnoeas and hypopnoeas per sleep hour was defined as the apnoea-hypopnoea index. According to AHI, the patients were diagnosed with mild (AHI = 5–15), moderate (AHI = 15–30) and severe OSAS (AHI ≥ 30). The assessment of the sleep records was done by an experienced sleep laboratory specialist. Continuous positive airway pressure titration was performed with the patients in whom diagnosis of OSAS was established, and CPAP treatment was planned. Continuous positive airway pressure titration was done through automatic-CPAP (DEVILBISS Respironics, USA) within the values that have been found convenient by the clinician.

Basal metabolism rate was measured through an indirect calorimetry instrument (N Spire ZAN 600 Ergospirometry) assessing respiratory gas exchange. It was performed after at least eight-hour night sleep when the patients were awake, hungry and in supine position by keeping room temperature constant at 22–24°C.

The study was approved by the local ethical committee of the Faculty of Medicine of Kocaeli University, Turkey (Approval Date of Ethical Committee and Project Number: July 10, 2012 and 2012/55), and all patients provided written informed consent.

Statistical analysis

Statistical Package for Social Sciences Ver. 13.0 software package was used in the statistical analysis of data. Categorical measurements were summarized as number and percent, and numeric measurements were summarized as mean and standard deviation (median and minimum–maximum when necessary). Shapiro–Wilk test was used to examine whether data fit to normal distribution. In the comparison of values before and after PAP treatment, significance tests of the differences between two pairs (test in dependent groups) were applied. Statistical significance level was taken as $p < 0.05$ in all tests.

RESULTS

A total of 25 patients, six female (24%) and 19 male (76%), with a mean age of 51.4 ± 13.7 years, were included in the study. The mean BMI of the patients was 34.06 ± 6.02 kg/m^2 (minimum: 24.6, maximum: 48.4), and the mean AHI was 60.76 ± 15.03 (minimum: 44, maximum: 90). Before- and after-treatment values of BMR, oxygen consumption (VO_2) and carbon dioxide output (VCO_2) of the study population are shown in Figs. 1–3, respectively.

There were 10 non-smokers (40%) and 15 current smokers (60%) in the study population, and the mean AHI index of the smokers was significantly higher than that of the non-smokers (61.5 ± 5.13 *vs* 51.6 ± 6.03; $p < 0.05$).

It was found that the values of BMR, VO_2 and VCO_2 after CPAP administration were significantly lower compared to before treatment evaluation (p = 0.049, 0.042 and 0.008, respectively) for the entire study population (Table 1).

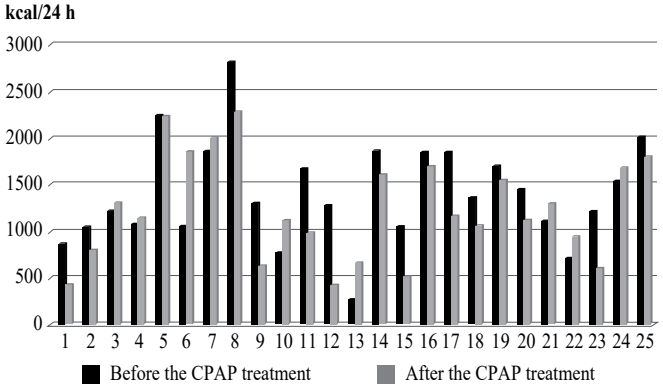

Fig. 1: Basal metabolism rate levels of each patient before and after the continuous positive airway pressure (CPAP) treatment.

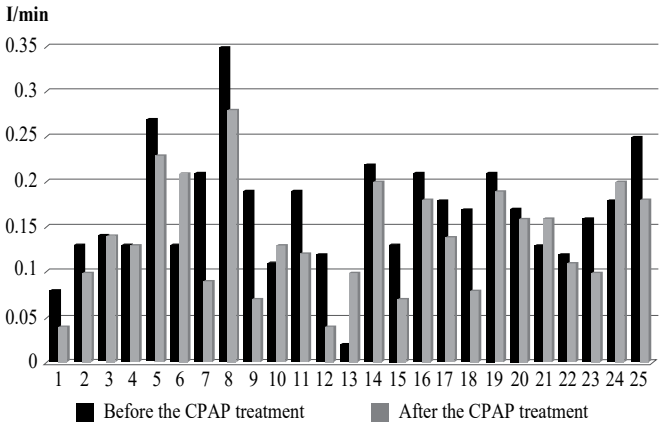

Fig. 3: Carbon dioxide output (VCO_2) levels of the patients before and after the continuous positive airway pressure (CPAP) treatment.

Table 1: The effect of CPAP treatment on BMR, VO_2 and VCO_2

	Before CPAP treatment	After CPAP treatment	*p**
BMR (kcal/24 h)	1414.35 ± 547.7	1245.12 ± 554.47	0.049
VO_2 (L/min)	0.20 ± 0.08	0.17 ± 0.08	0.042
VCO_2 (L/min)	0.17 ± 0.07	0.14 ± 0.06	0.008

*A p value < 0.05 is considered to be statistically significant.
BMR = basal metabolism rate; CPAP = continuous positive airway pressure; VO_2 = oxygen consumption; VCO_2 = carbon dioxide output

We compared the before- and after-treatment values of patients with BMI of ≥ 30 kg/m^2 and < 30 kg/m^2 in order to evaluate the possible effects of BMI on BMR and found a significant association between these parameters. Continuous positive airway pressure treatment for one night significantly reduced the BMR, VO_2 and VCO_2 values in those with BMI of ≥ 30 kg/m^2 (p = 0.001, 0.001 and 0.001, respectively). In those with BMI of < 30 kg/m^2, a slight increase was observed; however, this increase was not statistically significant (Table 2).

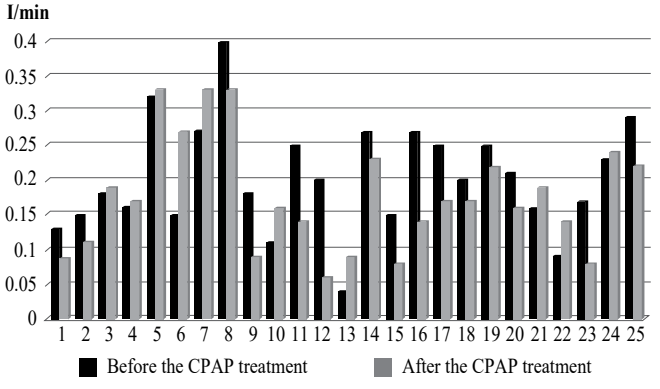

Fig. 2: Oxygen consumption (VO_2) levels of the patients before and after the continuous positive airway pressure (CPAP) treatment.

Table 2: The effect of CPAP treatment on BMR, VO_2 and VCO_2 according to BMI

	BMI	Before CPAP treatment	After CPAP treatment	*p**
BMR (kcal/24 h)	< 30	1266.16 ± 554.95	1450.08 ± 485.16	0.08
	≥ 30	1513.14 ± 538.63	1108.47 ± 570.69	< 0.001
VO_2 (L/min)	< 30	0.18 ± 0.07	0.21 ± 0.07	0.06
	≥ 30	0.21 ± 0.07	0.14 ± 0.07	< 0.001
VCO_2 (L/min)	< 30	0.14 ± 0.06	0.15 ± 0.04	0.75
	≥ 30	0.18 ± 0.06	0.12 ± 0.06	< 0.001

*A p value < 0.05 is considered to be statistically significant.
BMI = body mass index; BMR = basal metabolism rate; CPAP = continuous positive airway pressure; VO_2 = oxygen consumption; VCO_2 = carbon dioxide output

When the relationship between smoking history and the effect of CPAP treatment on BMR was investigated, it was found that a single night of CPAP administration reduced the BMR (p = 0.001), VO_2 (p = 0.004) and VCO_2 (p = 0.001) values in a statistically significant level in current smokers. Although the BMR, VO_2 and VCO_2 values of non-smokers decreased with CPAP administration compared to before treatment, this reduction reached a statistically significance level only in VO_2 value (p = 0.039) (Table 3).

Table 3: The effect of CPAP treatment on BMR, VO_2 and VCO_2 according to smoking history

	Smoking history	Before CPAP treatment	After CPAP treatment	*p**
BMR (kcal/24 h)	(+)	1574.76 ± 532.95	1328.24 ± 643.26	0.001
	(–)	1173.74 ± 500.91	1120.43 ± 383.88	0.05
VO_2 (L/min)	(+)	0.23 ± 0.07	0.18 ± 0.09	0.004
	(–)	0.17 ± 0.07	0.17 ± 0.06	0.039
VCO_2 (L/min)	(+)	0.19 ± 0.07	0.15 ± 0.07	0.001
	(–)	0.14 ± 0.05	0.12 ± 0.03	0.98

*A p value < 0.05 is considered to be statistically significant.
BMR = basal metabolism rate; CPAP = continuous positive airway pressure; VO_2 = oxygen consumption; VCO_2 = carbon dioxide output

A similar association was also observed in patients with higher AHI (> 60) compared to patients with AHI lower than this level. A single night of CPAP administration achieved a significant reduction in BMR ($p = 0.00$), VO_2 ($p = 0.014$) and VCO_2 ($p < 0.001$) values in patients with AHI greater than 60 while the decrease was not statistically significant in all study parameters in patients with AHI lower than 60 (Table 4).

Table 4: The effect of CPAP treatment on BMR, VO_2 and VCO_2 according to AHI

	AHI	Before CPAP treatment	After CPAP treatment	p*
BMR (kcal/24 h)	≤ 60	1326.39 ± 518.41	1288.45 ± 474.57	0.74
	> 60	1526.3 ± 588.19	1189.96 ± 662.63	0.008
VO_2 (L/min)	≤ 60	0.19 ± 0.07	0.18 ± 0.06	0.73
	> 60	0.21 ± 0.08	0.16 ± 0.09	0.014
VCO_2 (L/min)	≤ 60	0.15 ± 0.05	0.15 ± 0.04	0.77
	> 60	0.18 ± 0.07	0.12 ± 0.07	< 0.001

A p value < 0.05 is considered to be statistically significant.
AHI = apnoea-hypopnoea index; BMI = body mass index; BMR = basal metabolism rate; CPAP = continuous positive airway pressure; VO_2 = oxygen consumption; VCO_2 = carbon dioxide output

DISCUSSION

This study demonstrated that CPAP administration for one night reduced BMR, VO_2 and VCO_2 levels in patients with severe OSAS, and this reduction was especially remarkable in current smokers, patients with an AHI level greater than 60 and a BMI greater than 30.

Obstructive sleep apnoea syndrome is an independent risk factor for the development of several comorbid conditions, especially cardiovascular and metabolic disorders. A previous study that investigated the exercise metabolism in patients with OSAS demonstrated that the maximum oxygen consumption (VO_2 max) levels of those with OSAS were lower compared to the control group (7). However, studies investigating the effects of CPAP treatment in VO_2 max levels reported incompatible results (8, 9). These different results might be related to patients' comorbid diseases, drug usage and severity of OSAS. Even though there are few studies investigating the relationship between OSAS and BMR, it was demonstrated that BMR was higher in patients with OSAS (2, 10, 11). This study suggested that this unfavourable outcome of OSAS that occurred in basal energy metabolism might be improved with CPAP treatment and showed significant reductions in BMR with one-night administration of CPAP.

Basal metabolism rate is the amount of energy consumption required to maintain body functions and metabolic activities. Total energy consumptions of an individual consist of three parts. The first is the BMR, and this constitutes approximately 70% of total daily energy consumption; the second part is the energy consumption related to physical activity, and this is approximately 20%; and the last part is the thermal effect formed by the foods, and this constitutes 10% of the general consumption (12). Many factors such as physical activity, thermogenesis depending on diet, gender, age, height, weight, heredity, race, sleep, body temperature, environment temperature, sympathetic stimulation, thyroid and growth hormones, and pregnancy can be counted as parameters affecting BMR. In our study, environmental factors were minimized by performing BMR measurements of the patients following a 12-hour hunger between 8.30 am and 10.30 am, in a silent room with a 22–24°C medium temperature. Pregnant females and those with thyroid disease were not included in the study.

The most valuable tool to measure BMR is indirect calorimetry. Indirect calorimetry determines BMR by measuring oxygen consumption and carbohydrate production. Since it measures caloric burning rate with oxygen intake, it is referred to as indirect (13). In our study, the BMRs of the patients were measured through indirect calorimetry methods by ensuring optimum conditions. Studies conducted in either animals or humans have demonstrated that an experimental interruption of sleep was related to increasing energy expenditure (14, 15). Repetitive apnoea and hypopnoea episodes in patients with OSAS not only disrupt normal respiration but also increase energy consumption (11). Ryan *et al* have found higher energy consumption in patients with OSAS compared to the control group (2). Similarly, Stenölf *et al* have reported that patients with OSAS spent higher energy compared to the control group and that energy expenditure was reduced following CPAP treatment for three months (10). In our study, the acute response of CPAP treatment was evaluated, and it was found that CPAP treatment reduced BMR.

Male gender predominance is a well-known demographic feature in patients with OSAS. Bixler *et al* have found a male/female ratio of 3.3/1 in patients with sleep apnoea (16). In a study conducted by Young *et al*, prevalence was detected as 2% in females and as 4% in males (17). In our study, the male/female ratio was 3/1. The BMR in males was significantly higher than that in females in this study. However, since the number of female patients was low, statistical comparison could not be performed. No difference was observed between genders in terms of the effect of CPAP treatment on BMR.

It is suggested that smoking is a risk factor in the development of apnoea by causing nasal congestion (18). Wetter *et al* studied the relationship between respiratory disorders in sleep and smoking in 811 cases and found that the prevalence of simple snoring and sleep-related respiratory disorders were significantly higher in smokers (19). Kashyap *et al* compared 108 patients with OSAS and an AHI greater than 10 with 106 simple snoring patients with an AHI less than five and found that smoking prevalence was higher in the OSAS group (20). In our study, 60% of the patients were current smokers, and it was noted that AHI values of smokers were higher than non-smokers. Furthermore, it was observed that the BMR was higher in smokers and that the reduction in BMR was more significant in these patients after CPAP treatment.

The relationship between obesity and OSAS has been demonstrated in many studies. Wolk *et al* (21) reported that 70% of patients with OSAS were obese and 40% of obese people have OSAS. Moreover, it has been reported that gaining weight by 10% was associated with a six-fold increase in the risk of sleep apnoea development. Since sleepless nights seen in OSAS will reduce daytime physical activity, it has been stated that OSAS has an important effect on increasing obesity (21). In our study, a BMI of ≥ 30 was found in 60% of the patients. When the relationship between BMR and BMI was studied, a statistically significant reduction was observed in BMR after CPAP treatment in patients with a BMI of ≥ 30. On the other hand, in patients with a BMI of < 30, even though it was not statistically significant, an elevation in BMR was seen after CPAP treatment. This finding suggested that since the effect of OSAS on BMR was more prominent in patients with a BMI greater than 30, the beneficial effect of CPAP treatment was more significant in these patients.

Basal metabolism rate was also shown to be correlated with severity of OSAS as evaluated by AHI (22). In this study, the BMR of the patients with an AHI of > 60 was higher compared with those with an AHI of < 60. Furthermore, a statistically significant reduction was observed in the BMR of the patients with an AHI of > 60 following CPAP treatment. Similarly, a significant beneficial effect of CPAP treatment on BMR was not noted in patients with an AHI of lower than 60.

The limitations of this study were the limited number of patients, the inhomogeneity of the gender distribution and the evaluation of only one-night effect of CPAP on BMR.

CONCLUSION

It is suggested that there is a correlation between BMR and the severity of OSAS and it is possible to provide a significant reduction in BMR with a single night administration of CPAP depending on the patient's smoking history, degree of obesity and disease severity. Future studies including more patients are required to determine long-term outcomes of high BMR observed in patients with OSAS and possible beneficial effects of CPAP treatment.

REFERENCES

1. Kryger MH. Fat, sleep, and Charles Dickens: literary and medical contributions to the understanding of sleep apnea. Clin Chest Med 1985; **6:** 555–62.
2. Ryan CF, Love LL, Buckley PA. Energy expenditure in obstructive sleep apnea. Sleep 1995; **18:** 180–7.
3. Ruggiero C, Metter EJ, Melenovsky V, Cherubini A, Najjar SS, Ble A et al. High basal metabolic rate is a risk factor for mortality: the Baltimore longitudinal study of aging. J Gerontol 2008; **63:** 698–706.
4. Qaseem A, Holty JE, Owens DK, Dallas P, Starkey M, Shekelle P et al. Management of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. Ann Intern Med 2013; **159:** 471–83.
5. Capodanno D, Milazzo G, Cumbo M, Marchese A, Salemi A, Quartarone L et al. Positive airway pressure in patients with coronary artery disease and obstructive sleep apnea syndrome. J Cardiovasc Med (Hagerstown) 2014; **15:** 402–6.
6. Destors M, Tamisier R, Baguet JP, Levy P, Pepin JL. Cardiovascular morbidity associated with obstructive sleep apnea syndrome. Rev Mal Respir 2014; **31:** 375–85.
7. Lin CC, Hsieh WY, Chou CS, Liaw SF. Cardiopulmonary exercise testing in obstructive sleep apnea syndrome. Respir Physiol Neurobiol 2006; **150:** 27–34.
8. Lin CC, Lin CK, Wu KM, Chou CS. Effect of treatment by nasal CPAP on cardiopulmonary exercise test in obstructive sleep apnea syndrome. Lung 2004; **182:** 199–212.
9. Alonso-Fernández A, García-Río F, Arias MA, Mediano O, Pino JM, Martínez I et al. Obstructive sleep apnoea-hypoapnoea syndrome reversibly depresses cardiac response to exercise. Eur Heart J 2006; **27:** 207–15.
10. Stenölf K, Grustein R, Hedner J, Sjöström L. Energy expenditure in obstructive sleep apnea: effects of treatment with continuous positive airway pressure. Am J Physiol 1996; **271:** 1036–43.
11. Marcus CL, Carroll JL, Koerner CB Hamer A, Lutz J, Loughlin GM. Determinants of growth in children with the obstructive sleep apnea syndrome. J Pediatr 1994; **125:** 556–62.
12. Larson PR, Kronenberg HM, Melmed S, Polonsky KS (eds) Williams textbook of endocrinology. 10th ed. Philadelphia: Saunders Publishers; 2003.
13. Caliyurt O, Altiay G. Resting energy expenditure in manic episode. Bipolar Disord 2009; **11:** 102–6.
14. Bergmann BM, Everson CA, Kushida CA, Fang VS, Leitch CA, Schoeller DA et al. Sleep deprivation in the rat: V. Energy use and mediation. Sleep 1989; **12:** 31–41.
15. Bonnet MH, Berry RB, Arand DL. Metabolism during normal, fragmented, and recovery. J Appl Physiol 1991; **71:** 1112–8.
16. Bixler EO, Vgontzas AN, Lin HM, Ten Have T, Rein J, Vela-Bueno A et al. Prevalence of sleep-disordered breathing in women: effects of gender. Am J Respir Crit Care Med 2001; **163:** 608–13.
17. Young T, Shahar E, Nieto FJ, Redline S, Newman AB, Gottlieb DJ et al. Predictors of sleep-disordered breathing in community dwelling adults: the sleep heart health study. Arch Intern Med 2002; **162:** 893–900.

18. Kauffmann F, Annesi I, Neukirch F, Oryszczyn MP, Alpérovitch A. The relation between snoring and smoking, body mass index, age, alcohol consumption and respiratory symptoms. Eur Respir J 1989; **2:** 599–603.
19. Wetter DW, Young TB, Bidwell TR, Badr MS, Palta M. Smoking as a risk factor for sleep-disordered breathing. Arch Intern Med 1994; **154:** 2219–24.
20. Kashyap R, Hock LM, Bowman TJ. Higher prevalence of smoking in patients diagnosed as having obstructive sleep apnea. Sleep Breath 2001; **5:** 167–72.
21. Wolk R, Shamsuzzaman ASM, Somers VK. Obesity, sleep apnea and hypertension. Hypertension 2003; **42:** 1067–74.
22. Ucok K, Aycicek A, Sezer M, Fidan F, Akgun L, Akkaya M et al. Resting metabolic rate and anthropometric measurements in male sleep apnea patients. Intern Med 2011; **50:** 833–8.