



Expiratory and Inspiratory Muscle Functions in Obstructive Sleep Apnea Syndrome

Obstrüktif Uyku Apne Sendromunda Ekspiratuvar ve İnspiratuvar Kas Fonksiyonları

Elif Torun Parmaksız, Banu Salepçi, Nesrin Kıral, Ali Fidan, Sevda Cömert, Esma Coşkun, Benan Çağlayan*

Dr. Lütfi Kırdar Kartal Training and Research Hospital, Clinic of Chest Diseases, İstanbul, Turkey

*Koç University Faculty of Medicine, Department of Chest Diseases, İstanbul, Turkey

Abstract

Objective: Obstructive sleep apnea syndrome (OSAS) patients are thought to experience increased resistive load due to both anatomical and functional changes. This can possibly contribute to respiratory muscle dysfunction. We aimed to measure daytime maximal inspiratory and expiratory pressures, in order to find out respiratory muscle functions in OSAS.

Materials and Methods: Patients admitted to our sleep laboratory for one-year period were prospectively analysed. All the cases had undergone overnight polysomnography. All cases had pulmonary function tests performed by the same experienced technician in the morning following sleep study. The study population consisted of 51 (37.8%) female and 84 (62.2%) male patients, with a mean age of 47.

Results: Apnea hypopnea index (AHI) was found to be correlated with forced expiratory volume in one second (FEV1) and forced volume vital capacity (FVC) values and FVC%. FEV1/FVC, maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) values did not seem to be correlated with AHI. FVC (L), FVC%, FEV1, FEV1%, MIP, MIP% and MEP were similar in patients with and without OSAS. OSAS patients had significantly lower MEP% values. FVC, FEV1 and MEP% showed significant differences in different stages of the disease. Other parameters were similar in all groups.

Conclusion: In this study we demonstrated that maximal expiratory muscle strength of awake OSAS patients was lower, whereas inspiratory muscle strength was similar in subjects with and without OSAS.

Keywords: Maximal inspiratory pressure, maximal expiratory pressure, Obstructive sleep apnea syndrome

Öz

Amaç: Obstrüktif uyku apne sendromu (OUAS) hastalarının hem anatomik hem de işlevsel değişiklikler nedeniyle artmış bir direnç yüküne maruz kaldıkları düşünülmektedir. Bu, muhtemel bir solunum kası fonksiyonu bozukluğuna sebep olabilir. OUAS'da solunum kası fonksiyonlarını ortaya çıkarmak için günlük maksimum inspiratuvar ve ekspiratuvar basınçlarını ölçmeyi amaçladık.

Gereç ve Yöntem: Bir yıllık dönemde uyku laboratuvarımıza başvuran hastalar prospektif olarak analiz edildiler. Tüm olgular bir gecelik polisomnografiden geçirildi. Tüm olgulara uyku çalışmasının sabahı aynı deneyimli teknisyen tarafından akciğer fonksiyon testleri uygulandı. Çalışma grubu yaş ortalaması 47 olan 51 (%37,8) kadın ve 84 (%62,2) erkek hastadan oluşuyordu.

Bulgular: Apne hipopne indeksinin (AHI) birinci saniyedeki zorlu vital kapasite (FEV1) ve zorlu vital kapasite (FVC) değerleri ile ve FVC% ile korele olduğu bulunurken FEV1/FVC, maksimum inspiratuvar (MIP) ve maksimum ekspiratuvar basınç (MEP) değerleri AHI ile korele değildi. OUAS olan ve olmayan hastalarda FVC (L), FVC%, FEV1, FEV1%, MIP, MIP% ve MEP değerleri benzerdi. OUAS hastalarında MEP% değerleri önemli derecede düşüktü. FVC, FEV1 ve MEP% değerleri farklı hastalık evrelerinde önemli farklılıklar gösterdi. Diğer parametreler tüm gruplarda benzerdi.

Sonuç: Bu çalışmada uyanıklık sırasındaki maksimal ekspiratuvar kas kuvvetinin OUAS hastalarında daha düşük olduğunu, ancak inspiratuvar kas kuvvetinin ise OUAS'lı olan ve olmayan hastalarda benzer olduğunu gösterdik.

Anahtar Kelimeler: Maksimum inspiratuvar, maksimum ekspiratuvar basınç, Obstrüktif uyku apne sendromu

Introduction

Obstructive sleep apnea syndrome (OSAS) patients are presumed to have some level of inspiratory weakness due to several possible mechanisms. OSAS has been shown to be closely related to increased upper airway resistance in conjunction with reduced upper airway cross sectional area. Therefore,

OSAS patients experience increased resistive load compared to normal subjects. Intermittent hypoxemia also facilitates inspiratory muscle fatigue. Sleep deprivation or fragmentation is another factor which impairs inspiratory muscle endurance. As a result, respiratory muscle pump strength is likely to decrease in the presence of OSAS. Not solely anatomical factors, but also

Address for Correspondence/Yazışma Adresi: Elif Torun Parmaksız MD, Dr. Lütfi Kırdar Kartal Training and Research Hospital, Clinic of Chest Diseases, İstanbul, Turkey

Phone: +90 216 441 39 00 E-mail: dreliftorun@yahoo.com

Received/Geliş Tarihi: 11.10.2016 Accepted/Kabul Tarihi: 16.11.2016

This study was presented in the TUSAD 2012.

©Copyright 2016 by Turkish Sleep Medicine Society / Journal of Turkish Sleep Medicine published by Galenos Publishing House.

functional compromise seem to play role in pathophysiology of the disease (1-3).

OSAS is associated with some changes in contractile function of upper airway dilator muscles. It is possible that these muscles undergo some sort of modification in order to adapt to increased contractile demands. However, whether OSAS patients have lower respiratory effort when measured just following sleep remains unclear. There is no specific data about the measurements of respiratory muscle functions in OSAS cases.

A quick, simple and noninvasive way of assessing respiratory muscle pump strength is measurement of maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP). In order to find out how much impairment, if any, OSAS causes on daytime maximal inspiratory and expiratory pressures, we studied morning respiratory muscle functions of OSAS patients.

Materials and Methods

Patients admitted to our sleep laboratory for one year period were prospectively analysed. Consecutive subjects who were admitted to our sleep laboratory for overnight polysomnography and were compliant to pulmonary function testing were enrolled in the study.

The presenting symptoms were one or more of the following: habitual snoring, witnessed apnea, excessive daytime sleepiness and unsatisfactory sleep. Subjects with diseases that could effect respiratory muscle function such as neuromuscular diseases were not included in the study. Patients with previously diagnosed neuromuscular disease and cases with symptoms reminding of neuromuscular disease such as muscle weakness, muscle loss, balance or movement problems were excluded. The height and weight of each individual were measured and body mass indices were calculated by weight divided by height square. Daytime hypersomnolence was assessed by Epworth sleepiness scale.

All cases had undergone overnight polysomnography with Viasys Sleep Screen (Viasys Healthcare, Germany), Viasys Cephalo (Viasys Healthcare, Germany). Comet apparatus (Grass Technologies, USA) and Sleep Matrix, Somno Star, Grass programmes. Throughout the night electroencephalography (EEG), electrooculography (EOG), electrocardiography (ECG), chin and tibial electromyography (EMG), respiration, snoring, body position and oxygen saturation were monitored. The digitized EEG records were scored in 30-second epochs according to standardized criteria by Rechtschaffen and Kales (4) and American Academy of Sleep Medicine (AASM) 2007 (5). Respiration was scored by certified specialists according to AASM 1999 (6) ve AASM 2007 (5) criteria. Apnea hypopnea index (AHI) was calculated as the sum of apneas and hypopneas during the sleep period divided by total sleep time. Apneas were scored when flow signal amplitudes dropped to $\leq 10\%$ of the stable baseline. Hypopneas were scored when a clearly discernible reduction of the flow signal was terminated by an abrupt recovery and associated with a 4% desaturation. Arousals were scored when clearly discernible reduction of the flow signal or flow limitation was seen and terminated by abrupt recovery associated with EEG arousal in the absence of 4% desaturation. Arousal index was calculated as number of arousals per hour of sleep.

All cases had pulmonary function tests performed by the same experienced technician in the morning following sleep study. Each patient received exactly the same instructions. Spirometry tests were applied and respiratory muscle strength was assessed by measuring maximum inspiratory and expiratory pressures. The patient is asked to exhale slowly and completely after sealing his lips around the mouthpiece and then inhale with maximal effort at residual volume for measuring MIP and exhale at total lung capacity for measuring MEP.

Statistical Analyses

Statistical analyses were performed with SPSS for Windows version 17.0 Continuous data are presented as mean \pm standard deviation. Correlations between parametric data sets were examined using pearson's correlation test. P values less than 0.05 were considered significant for all statistical tests. All reported p values are two-sided.

Results

The study population consisted of 51 (37.8%) female and 84 (62.2%) male patients. Mean age of 135 cases was 47.76 ± 11.88 (18-79).

OSAS was diagnosed in 119 (88.1%) of the cases by polysomnography; 41 (30.4%) had mild, 31 (23.0%) had moderate and 48 (35.6%) had severe OSAS. The detailed polysomnographic data, body mass index and endoscopic ultrasound of apneics and nonapneics are categorised and shown in the Table 1.

The results of pulmonary function tests in each group are shown in Table 2 and Figure 1.

AHI was found to be correlated with forced expiratory volume in one second (FEV1) and forced volume vital capacity (FVC) values and FVC% (p and r for FEV1: 0.002, -0.26, respectively; p and r for FVC: 0.002, -0.26, respectively; p and r for FVC%: 0.017, -0.20, respectively). FEV1/FVC, MIP and MEP values did not seem to be correlated with AHI (p and r for FEV1/FVC: 0.77, -0.025, respectively; p and r for MIP%: 0.54, -0.073 respectively; p and r for MEP% 0.10, 0.20 respectively).

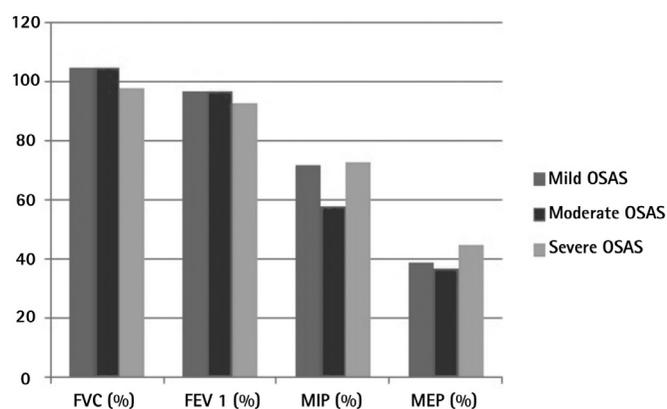


Figure 1. The distribution of spirometric values due to disease severity

FVC: Forced volume vital capacity, FEV1: Forced expiratory volume in one second, MIP: Maximal inspiratory pressure, MEP: Maximal expiratory pressure, OSAS: Obstructive sleep apnea syndrome

FVC (L), FVC%, FEV1, FEV1%, MIP, MIP% and MEP were similar in patients with and without OSAS ($p=0.27, 1.00, 0.75, 0.76, 0.89, 0.08, 0.53$, respectively).

When OSAS and non-OSAS groups were compared, FEV1/FVC was significantly lower in apneic cases compared to nonapneic cases ($p=0.04$). OSAS patients also demonstrated significantly lower MEP% values ($p=0.04$).

When spirometric parameters were compared between nonapneics, mild, moderate and severe OSAS patients, FVC and FEV1 showed significant difference between groups ($p=0.007, 0.02$, respectively). MEP% was also significantly different between groups ($p=0.02$). Other parameters were similar in all groups (Table 2).

Discussion

In this study we demonstrated that maximal expiratory muscle strength during wakefulness is lower in OSAS patients, whereas

inspiratory muscle strength is similar in subjects with and without OSAS. This may possibly suggest that inspiratory muscles are more resistant to increased contractile demands than expiratory muscles. The MIP reflects the strength of the diaphragm and other inspiratory muscles, while the MEP reflects the strength of the abdominal muscles and other expiratory muscles.

Until recently, few studies have been published on the effects of daytime respiratory muscle functions. Repetitive upper airway collapse in OSAS leads to increased resistive load on respiratory muscles, intermittent hypoxia and sleep deprivation and fragmentation, which may eventually result in some decrease in respiratory muscle pump strength.

In most OSAS cases, the optimal treatment modality is non-invasive mechanical ventilation. Continuous positive air pressure or bi-level positive air pressure machines are prescribed to a substantial amount of patients. Determination of MIP and MEP

Table 1. The polysomnographic data, body mass index and Epworth Sleepiness Scale of the population

	Nonapneic	Mild OSAS	Moderate OSAS	Severe OSAS	Total
n	15	41	31	48	135
BMI	44.29	29.73	31.34	34.43	33.39
ESS	9.8	6.78	7.53	11.04	6.44
AHI	2.90	9.93	22.02	60.91	30.05
AI	0.76	1.78	5.55	32.29	13.38
ODI	1.42	6.24	14.34	56.12	25.30
Minimum saturation	90.06	86.34	83.93	71.1	80.78
Sleep efficiency	84.80	84.34	81.79	78.89	81.87
REM latency	135.7	112.45	138.63	149.48	134.08
Sleep latency	15.73	14.78	15.98	17.7	16.20
Arousal index	16.65	19.02	24.62	45.52	29.47
Stage 1 (%)	4.82	4.94	7.38	7.35	6.34
Stage 2 (%)	54.25	57.42	58.25	65.76	60.22
Stage 3 (%)	19.24	15.56	17.12	9.18	14.13
REM (%)	14.00	15.70	14.88	11.72	13.92

OSAS: Obstructive sleep apnea syndrome, BMI: Body mass index, ESS: Epworth sleepiness scale, AHI: Apnea hypopnea index, AI: Apnea index, ODI: Oxygenation desaturation index, REM: Rapid eye movement sleep

Table 2. The results of pulmonary function tests

	Nonapneics	Mild OSAS	Moderate OSAS	Severe OSAS	Total
FVC (L)	3.59	4.19	3.92	3.51	3.82
FVC (%)	101.73	105.22	105.23	98.83	102.56
FEV1 (L)	2.93	3.25	3.02	2.72	2.97
FEV1 (%)	96.47	97.93	97.35	93.73	96.14
FEV1/FVC	81.47	77.46	76.81	77.77	77.87
MIP	74.13	79.00	67.76	71.35	73.20
MIP (%)	88.78	72.00	58.67	73.37	71.83
MEP	79.00	83.27	80.11	84.47	82.48
MEP (%)	52.00	39.55	37.23	45.64	43.04

OSAS: Obstructive sleep apnea syndrome, FVC: Forced volume vital capacity, FEV1: Forced expiratory volume in one second, MIP: Maximal inspiratory pressure, MEP: Maximal expiratory pressure

helps to assess the ventilatory functions. Serial measurements of the MIP and MEP can be used to evaluate whether the respiratory muscle weakness has improved, remained stable, or worsened with the treatment chosen.

In a study conducted to find out respiratory muscle strength before and after sleep, the authors measured MIP and MEP in cases with sleep disordered breathing. They have found higher values in the morning when compared with night, and had come up with the fact that this was attributed to the restorative effect of nocturnal sleep. The diurnal difference in respiratory muscle strength was not correlated with the severity of the disease (7).

In order to maintain upper airway patency, the dilator muscles are activated thereby reducing resistance. However, the anatomical compromise is not sufficient to explain OSAS pathophysiology. The functional changes, their relationship with lung volumes, pressures and flow, and altered neuromuscular responses predisposing collapse still remain poorly defined (3).

It is well-known that the cross-sectional measurement of OSAS patients during wakefulness tend to be lower than normal subjects (8). In a group of patients with moderate OSAS, the effects of muscle training was studied and it was found to result in improvement in snoring, daytime sleepiness, complaints of the partners as well as reduction in AHI (9).

Although training respiratory muscles was proposed to decrease the severity of OSAS, it is not likely to be sufficient for prevention of airway collapse during sleep (8).

One limitation of the study is lack of usage of more sensitive and accurate methods for measuring respiratory muscle strength, such as transdiaphragmatic pressure measurement. The difficulty in application of such sophisticated techniques limit their utility and MIP-MEP measurement is a simple, quick indicator of respiratory muscle function.

Another possible limitation is that MIP and MEP require cooperation and compliance of the patient. Still many studies emphasize the role of their measurement for evaluation of respiratory muscle function. MIP and MEP values below normal limits do not always point impaired muscle function; however normal test results are helpful in ruling out severe functional loss of respiratory muscles.

Conclusion

MEP% were lower in mild-moderate OSAS cases and higher in obese patients. MIP values, on the other hand, were similar in the whole group. On the basis of the findings described in this study, larger populations need to be evaluated with

similar techniques in addition with transdiaphragmatic muscle measurements.

Ethics

Informed Consent: It was taken.

Peer-review: Internally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Banu Salepçi, Esmâ Coşkun, **Concept:** Elif Torun Parmaksız, **Design:** Elif Torun Parmaksız, Nesrin Kırıl, **Data Collection or Processing:** Elif Torun Parmaksız, Banu Salepçi, Ali Fidan, **Analysis or Interpretation:** Elif Torun Parmaksız, Banu Salepçi, Sevda Cömert, **Literature Search:** Elif Torun Parmaksız, **Writing:** Elif Torun Parmaksız, Benan Çağlayan.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Kimoff RJ. Upper airway myopathy is important in the pathophysiology of obstructive sleep apnea. *J Clin Sleep Med* 2007;3:567-9.
2. Arens R, Sin S, McDonough JM, Palmer JM, Dominguez T, Meyer H, Wootton DM, Pack AI. Changes in upper airway size during tidal breathing in children with obstructive sleep apnea syndrome. *Am J Respir Crit Care Med* 2005;171:1298-304.
3. Owens RL, Eckert DJ, Yeh SY, Malhotra A. Upper airway function in the pathogenesis of obstructive sleep apnea: a review of the current literature. *Curr Opin Pulm Med* 2008;14:519-54.
4. Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques and Scoring System For Sleep Stages of Human Subjects. Eds. Washington DC: US. Government Printing Office 1968.
5. Iber C, Ancoli-Israel S, Chesson AL, Quan SF. The AASM Manual for the Scoring of Sleep and Associated Events. Rules, Terminology and Technical Specifications. Westchester, IL: American Academy of Sleep Medicine 2007.
6. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22:667-89.
7. Su MC, Chin CH, Chen YC, Hsieh YT, Wang CC, Huang YC, Lin MC. Diurnal change of respiratory muscle strength in patients with sleep-disordered breathing. *Chang Gung Med J* 2008;31:297-303.
8. Eckert DJ, Malhotra A. Pathophysiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 2008;15;5:144-53.
9. Puhan MA, Suarez A, Lo Cascio C, Zahn A, Heitz M, Braendli O. Didgeridoo playing as alternative treatment for obstructive sleep apnoea syndrome: randomised controlled trial. *BMJ* 2006;332:266-70.