Expiratory and Inspiratory Muscle Functions in Obstructive Sleep Apnea Syndrome

Elif Torun Parmaksız, Banu Salepçi, Nesrin Kiral, 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

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
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 ≤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 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.

Statistical Analyses

Statistical analyses were performed with SPSS for Windows
version 17.0 Continuous data are presented as mean ± 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

<table>
<thead>
<tr>
<th></th>
<th>Nonapneic</th>
<th>Mild OSAS</th>
<th>Moderate OSAS</th>
<th>Severe OSAS</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>15</td>
<td>41</td>
<td>31</td>
<td>48</td>
<td>135</td>
</tr>
<tr>
<td>BMI</td>
<td>44.29</td>
<td>29.73</td>
<td>31.34</td>
<td>34.43</td>
<td>33.39</td>
</tr>
<tr>
<td>ESS</td>
<td>9.8</td>
<td>6.78</td>
<td>7.53</td>
<td>11.04</td>
<td>6.44</td>
</tr>
<tr>
<td>AH1</td>
<td>2.90</td>
<td>9.93</td>
<td>22.02</td>
<td>60.91</td>
<td>30.05</td>
</tr>
<tr>
<td>Al</td>
<td>0.76</td>
<td>1.78</td>
<td>5.55</td>
<td>32.29</td>
<td>13.38</td>
</tr>
<tr>
<td>ODI</td>
<td>1.42</td>
<td>6.24</td>
<td>14.34</td>
<td>56.12</td>
<td>25.30</td>
</tr>
<tr>
<td>Minimum saturation</td>
<td>90.06</td>
<td>86.34</td>
<td>83.93</td>
<td>71.1</td>
<td>80.78</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>84.80</td>
<td>84.34</td>
<td>81.79</td>
<td>78.89</td>
<td>81.87</td>
</tr>
<tr>
<td>REM latency</td>
<td>135.7</td>
<td>112.45</td>
<td>138.63</td>
<td>149.48</td>
<td>134.08</td>
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<tr>
<td>Sleep latency</td>
<td>15.73</td>
<td>14.78</td>
<td>15.98</td>
<td>17.7</td>
<td>16.20</td>
</tr>
<tr>
<td>Arousal index</td>
<td>16.65</td>
<td>19.02</td>
<td>24.62</td>
<td>45.52</td>
<td>29.47</td>
</tr>
<tr>
<td>Stage 1 (%)</td>
<td>4.82</td>
<td>4.94</td>
<td>7.38</td>
<td>7.35</td>
<td>6.34</td>
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<tr>
<td>Stage 2 (%)</td>
<td>54.25</td>
<td>57.42</td>
<td>58.25</td>
<td>65.76</td>
<td>60.22</td>
</tr>
<tr>
<td>Stage 3 (%)</td>
<td>19.24</td>
<td>15.56</td>
<td>17.12</td>
<td>9.18</td>
<td>14.13</td>
</tr>
<tr>
<td>REM (%)</td>
<td>14.00</td>
<td>15.70</td>
<td>14.88</td>
<td>11.72</td>
<td>13.92</td>
</tr>
</tbody>
</table>

OSAS: Obstructive sleep apnea syndrome, BMI: Body mass index, ESS: Epworth sleepiness scale, AH1: Apnea hypopnea index, Al: Apnea index, ODI: Oxygenation desaturation index, REM: Rapid eye movement sleep

### Table 2. The results of pulmonary function tests

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

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 Salepci, Esma Coskun,

**Concept:** Elif Torun Parmaksiz, **Design:** Elif Torun Parmaksiz, Nesrin Kiral, **Data Collection or Processing:** Elif Torun Parmaksiz, Banu Salepci, Ali Fidan, **Analysis or Interpretation:** Elif Torun Parmaksiz, Banu Salepci, Sevda Cömert, **Literature Search:** Elif Torun Parmaksiz, **Writing:** Elif Torun Parmaksiz, 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**