Evaluation of maximum inspiratory and expiratory pressure in patients with chronic obstructive pulmonary disease

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Abstract  Respiratory muscle dysfunction is a cardinal feature of acute and chronic respiratory failure in COPD. Diaphragm and accessory inspiratory muscles face increased load due to increased lung resistance and elastance, as well as increased ventilatory demand.

Aim of the work:  The objective of this work is to measure maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) in stable COPD patients and to correlate it with degree of airway obstruction and functional disability.

Subjects, methods and result:  Forty known COPD male patients were enrolled in this study with mean age 56.8 ± 7.7. Spirometry was done for all patients with mean FEV1 39.5 ± 15.1%, mean FVC 59.5 ± 19.2%, mean FEV1/FVC 52.9 ± 10.3%. Maximum inspiratory and expiratory pressures were done with mean 43.6 ± 26.9% and 46.8 ± 26% respectively. As regards 6 min walk distance, its mean was 131.41 ± 41.73 m. A quantification of dyspnea using the Modified Medical Research Council Scale (MRC) was done. The results of the present study showed: a highly significant positive statistical correlation between MIP, MEP and 6 min walk distance, also highly significant negative statistical correlation between MIP, MEP and MRC dyspnea scale. We note a
significant positive correlation between MIP and each of PaO$_2$ and SaO$_2$, while MEP has a significant correlation with SaO$_2$, also a highly significant negative statistical correlation between MIP, MEP and PaCO$_2$.

Conclusion: A highly significant positive statistical correlation between MIP, MEP and 6 min walk distance and a highly significant negative statistical correlation between MIP, MEP and subjective dyspnea evaluated by MRC dyspnea scale were found.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extra pulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases [1].

Respiratory, and particularly inspiratory, muscle function is altered in COPD. Many of these alterations are secondary to a mechanical disadvantage related to hyperinflation. Other factors, including corticosteroid therapy and nutritional depletion, are also deleterious to muscle function. Moreover, the imbalance between respiratory muscle function and load is an important determinant of dyspnea and hypercapnia. Because much of the lung and airway derangements are irreversible in COPD, the respiratory muscles appear to be an attractive target for therapeutic interventions [2].

Maximal inspiratory pressure (MIP) is the maximum negative pressure that can be generated from one inspiratory effort starting from functional residual capacity (FRC) or residual volume (RV). Maximal expiratory pressure (MEP) measures the maximum positive pressure that can be generated from one expiratory effort starting from total lung capacity (TLC) or FRC. Unlike inspiratory muscles, expiratory muscles (abdominal and thoracic muscles) reach their optimal force–length relationship at high pulmonary volumes [3].

Respiratory muscle dysfunction is a cardinal feature of acute and chronic respiratory failure in COPD. Diaphragm and accessory inspiratory muscles face increased load due to increased lung resistance and elastance, as well as increased ventilatory demand [4].

Measurement of the maximum static inspiratory pressure that a subject can generate at the mouth (PImax) or the maximum static expiratory pressure (PEmax) is a simple way to gauge inspiratory and expiratory muscle strength. When respiratory muscle weakness occurs, the PImax can be more sensitive than the VC because the relationship between VC and PImax is curvilinear, so that decreases in respiratory muscle strength occur before decreases in lung volume can be identified [5].

Aim of the work

The objective of this work is to measure maximal inspiratory pressure (MIP) and maximal expiratory pressure (MEP) in stable COPD patients and to correlate MIP and MEP values with degree of airway obstruction and functional disability.

Subjects and methods

The present study was conducted upon forty known COPD patients according to GOLD 2010 guidelines at Ain Shams University Hospital in the period between August 2011 and March 2012.

For all patients the following were done

– Full history taking.
– Physical examination.
– Chest X-ray.
– Spirometric pulmonary function.
– Maximal inspiratory pressure and maximal expiratory pressure.
– Arterial blood gases.
– Six minute walking test according to ATS standards [6].

Subjective dyspnea scale

A quantification of dyspnea using the Modified Medical Research Council Scale (MRC) is indicated since it predicts quality of life and survival (Functional Dyspnea):

Exclusion criteria

– Patients with primary muscular or neuromuscular diseases.
– Patients with clinically significant co morbidities those are likely to affect test results.
– COPD patients in exacerbation.
– NB: Spirometry, MIP and MEP were done using Master Screen PFT with built in program for measuring MIP and MEP.

Measurement of MIP and MEP

We can measure MIP and MEP according to [7].

Subject preparations

The subjects were instructed not to engage in heavy exercise immediately before testing.

Equipment

System description. We use a pulmonary function testing instrument with software and hardware adaptations that allow for the measurement of respiratory muscle pressures.
Evaluation of maximum inspiratory and expiratory pressure in patients

Procedure.
- The maneuver was explained and demonstrated to the subjects using a spare mouthpiece.
- The subjects were instructed to sit upright.
- The subjects were instructed to keep a tight lip seal and to give maximum effort.
- A tight-fitting nose clip was attached to the patient’s nose to prevent leakage.

Steps.
(1) A tight-fitting disposable rubber mouth piece was placed firmly onto the mouth piece adapter.
(2) MIP: from tidal breathing the patient slowly exhales as deeply as possible. During expiration the measurement is started manually. The shutter will be set as soon as the patient starts to breathe in. Now the patient is asked to inspire as fast and as powerful as possible against the shutter. The maximal inspiratory pressure will be reached after about 0.5–1 s. The shutter opens automatically.
(3) MEP: from tidal breathing the patient slowly breathes in as deeply as possible. The shutter will be closed with expiration onset. Now the patient is asked to expire as fast and as powerful as possible against the shutter. The shutter will be opened automatically.
(4) Three trials were obtained.
(5) The subjects were allowed to rest for 30 to 60 s between trials.

Reporting results. The most negative values of MIP and MEP were reported in (k pa).

Statistical methodology

Data management and analysis:
The collected data were revised, coded, tabulated and introduced to a PC using Statistical package for Social Science (SPSS 15.0.1 for windows; SPSS Inc, Chicago, IL, 2001). Data were presented and suitable analysis was done according to the type of data obtained for each parameter.

Descriptive statistics:
(1) Mean, Standard deviation (±SD) and range for parametric numerical data
(2) Frequency and percentage of non-numerical data.

Analytical statistics:
(1) Correlation analysis (using Pearson’s method): To assess the strength of association between two quantitative variables. The correlation coefficient denoted symbolically “r” defines the strength and direction of the linear relationship between two variables.
(2) ANOVA test was used to assess the statistical significance of the difference between more than two study group means.

P-value: level of significance:  P > 0.05: Non significant (NS).

Results
Forty known COPD male patients were enrolled in this study with mean age 56.8 ± 7.7 and smoking index mean 30 ± 10.1. Spirometry was done for all patients with mean FEV1 39.5 ± 15.1%, mean FVC 59.5 ± 19.2%, mean FEV1/FVC 52.9 ± 10.3%. Maximum inspiratory & expiratory pressures were determined with mean 43.6 ± 26.9% and 46.8 ± 26% respectively mean PH 7.40 ± 0.03, mean PaCO2 40.09 ± 4.23 mmHg, mean PaO2 71.89 ± 9.35 mmHg, mean SaO2 93.43 ± 2.80%. As regards 6 min walk distance, its mean was 131.41 ± 41.73 m.

- Correlation between MIP and each of FEV1 %, FVC%, FEV1/FVC%, 6MWD, PaO2, PaCO2, SaO2% and dyspnea scale is shown in Table 1. There was no significant statistical correlation between MIP and each of FEV1 %, FVC%, but highly significant positive statistical correlation between MIP and 6MWD, significant positive correlation between MIP and each of PaO2 and SaO2%, highly significant negative statistical correlation between MIP and PaCO2 and highly significant negative statistical correlation between MIP and dyspnea scale.
- There was a significant positive correlation between maximal inspiratory pressure and PaO2 as shown in Fig. 1, and a highly significant negative statistical correlation between maximal inspiratory pressure and PaCO2 as shown in Fig. 2.
- There was a highly significant negative statistical correlation between maximal inspiratory pressure and dyspnea scale as shown in Fig. 3.
- Correlations between MEP and each of FEV1 %, FVC%, FEV1/FVC%, 6MWD, PaO2, PaCO2 and SaO2% (Table 2). There was no significant statistical correlation between

<table>
<thead>
<tr>
<th>Variable</th>
<th>MIP%</th>
</tr>
</thead>
<tbody>
<tr>
<td>r-Value</td>
<td>P-Value</td>
</tr>
<tr>
<td>FEV1 %</td>
<td>-0.145</td>
</tr>
<tr>
<td>FVC%</td>
<td>-0.159</td>
</tr>
<tr>
<td>FEV1/FVC%</td>
<td>-0.86</td>
</tr>
<tr>
<td>6MWD (m)</td>
<td>0.546</td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>0.339</td>
</tr>
<tr>
<td>PaCO2 (mmHg)</td>
<td>-0.492</td>
</tr>
<tr>
<td>SaO2%</td>
<td>0.381</td>
</tr>
<tr>
<td>Dyspnea scale</td>
<td>-0.954</td>
</tr>
</tbody>
</table>

This table shows that there was: No significant statistical correlation between MIP and each of FEV1%, FVC% and FEV1/FVC%.
Highly significant positive statistical correlation between MIP and 6MWD. Significant positive correlation between MIP and each of PaO2 and SaO2%.
Highly significant negative statistical correlation between MIP and PaCO2.
Highly significant negative statistical correlation between MIP and dyspnea scale.
MEP and each of FEV1%, FVC%, FEV1/FVC% and PaO2 but highly significant positive statistical correlation between MEP and 6MWD also significant positive correlation between MEP and SaO2%, highly significant negative statistical correlation between MEP and PaCO2 and highly significant negative statistical correlation between MEP and dyspnea scale.

- There is a highly significant positive statistical correlation between maximal expiratory pressure and 6 min walk distance as shown in Fig. 4, and a highly significant negative statistical correlation between maximal expiratory pressure and dyspnea scale as shown in Fig. 5.

- Table 3 shows that there was a highly significant positive correlation between FEV1% and each of PaO2 and SaO2% but highly significant negative statistical correlation between FEV1% and PaCO2 and highly significant positive statistical correlation between FEV1% and 6MWD.

- Table 4 shows that there was a highly significant positive statistical correlation between FVC% and 6 min walk distance.

Table 2 Correlations between MEP and each of FEV1%, FVC%, FEV1/FVC%, 6MWD, PaO2, PaCO2 and SaO2%.

<table>
<thead>
<tr>
<th>Variable</th>
<th>MEP%</th>
<th>r-Value</th>
<th>P-Value</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1%</td>
<td>-0.168</td>
<td>0.321</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>FVC%</td>
<td>-0.234</td>
<td>0.164</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>FEV1/FVC%</td>
<td>-0.460</td>
<td>0.785</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>6MWD (m)</td>
<td>0.523</td>
<td>0.001</td>
<td>HS</td>
<td></td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>0.310</td>
<td>0.062</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>PaCO2 (mmHg)</td>
<td>-0.459</td>
<td>0.004</td>
<td>HS</td>
<td></td>
</tr>
<tr>
<td>SaO2%</td>
<td>0.398</td>
<td>0.015</td>
<td>S</td>
<td></td>
</tr>
<tr>
<td>Dyspnea scale</td>
<td>-0.905</td>
<td>0.0001</td>
<td>HS</td>
<td></td>
</tr>
</tbody>
</table>

This table shows that there was:
- No significant statistical correlation between MEP and each of FEV1%, FVC%, FEV1/FVC% and PaO2.
- Highly significant positive statistical correlation between MEP and 6MWD.
- Significant positive correlation between MEP and SaO2%.
- Highly significant negative statistical correlation between MEP and PaCO2.
- Highly significant negative statistical correlation between MEP and dyspnea scale.
Discussion

In the present study, it was found that there was no correlation between maximal inspiratory and expiratory pressures with spirometric pulmonary functions regarding FEV1%, FVC% and FEV1/FVC%.

These correlations were studied before and disagree with our results, Kabitz et al., (2007) [8] found a positive statistical correlation between maximal inspiratory pressure and lung function parameter (FEV1%) who conducted their study upon stable COPD population compared to healthy, non-smoking men as a control.

Also Terzano et al. [3] found a significant positive correlation between maximal inspiratory and expiratory pressures with FEV1% and FVC%. This study was conducted upon patients with stable COPD and age-matched healthy subjects. Moreover Nishimura et al. [9] and Heijdra et al. [10] found a correlation between MIP and FEV1 in COPD patients. Also a study done by Awad et al. [11] upon patients with a wide range of the severity of the disease found a significant positive correlation between FEV1%, FVC% and FEV1/FVC% and MIP.

We can explain the differences in results between our study and others; by the valuable observation by Rochester [12] who suggested that the observed values of MIP in patients with COPD should be compared with the values that normal subjects would achieve at similar lung volumes. For normal subjects was expected 80–90% of the predicted MIP at 60% TLC. This means that MIP, after correction for lung volume, was not lower than the values in normal subjects as was suggested in previous studies. Also the current study was done upon non homogenous group.

In this study, a significant positive statistical correlation was found between maximal inspiratory and expiratory pres-
In agreement with the present study, Singer et al. [13] and Tudorache et al. [14] have found the same statistical correlations. In our study we correlate between MIP and MEP to MRC dyspnea scale to define degree of patient’s disabilities. A negative significant correlation was found between MIP and MEP and MRC dyspnea scale grades, which is in agreement with Gigliotti et al. [15]. The present study showed also a correlation between MIP values with PaO2, PaCO2 and SaO2, while MEP correlates only with PaCO2 and SaO2.

Kabitz et al. [8] found a significant correlation between MIP and PCO2. Another study by Heijdra et al. [10] found a correlation between MIP and PaO2, PaCO2 and SaO2, while MEP is not. Rochester and Braun [16] in a study done upon 32 COPD patients found a negative correlation between MIP and PaCO2. Awad et al. [11] in their study found a significant correlation between arterial blood gases (PH, PaCO2, PaO2, and SaO2%) and MIP.

Kabitz et al. [8] proposed that the PaCO2 will rise because of reduced inspiratory muscle strength in COPD by two mechanisms:

- First, reduced diaphragmatic contractility beginning in early stages of the disease that is independent of hyperinflation.
- Secondly, reduced diaphragmatic force generation due to hyperinflation in severe disease stages only.

Heijdra et al. [10] found that the impairment in inspiratory muscle strength causes micro-atelectasis resulting in a decrease of PaO2 and the hypoxemia decreases the work of the respiratory muscles.

The current study showed a significant positive correlation between FEV1 and PaO2, and SaO2. Also a negative correlation with PaCO2. This is in agreement with Casanova et al. [17] who found a positive correlation between FEV1 and each of PaO2 and SaO2 and Fard and Zarezadeh [18] found a correlation between FEV1 and PaO2, PaCO2.

The current study shows a significant positive correlation between FEV1 and 6 min walk distance. Also Casanova et al. [17] showed a highly significant positive correlation between FEV1 and 6 min walk distance in COPD patients and Fujimoto et al. [19] too showed a positive correlation between FEV1 and 6 min walk distance. Another study by Mak et al. [20] upon 42 COPD patients found a positive correlation between FEV1 and FVC with 6 min walking distance.

Rate of desaturation after 6 min walk test was evaluated in only 8 patients and showed non-significant results and due to small sample size we could not rely on these results which needs further investigational studies.

Conclusion

In the present study, it was found that:

- No significant statistical correlation between MIP, MEP and each of FEV1%, FVC% and FEV1/FVC%.
- Highly significant positive statistical correlation between MIP, MEP and 6 min walk distance.
- Highly significant negative statistical correlation between MIP, MEP and subjective dyspnea evaluated by MRC dyspnea scale.
- Significant positive correlation between MIP and each of PaO2 and SaO2, while MEP has a significant correlation with SaO2 only.
- Highly significant negative statistical correlation between MIP, MEP and PaCO2.
- FEV1% shows a highly significant correlation with 6 min walk distance, PaO2, PaCO2 and SaO2.
- FVC% shows a highly significant correlation with 6 min walk distance.

Conflicts of interest

None declared.

References

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