Original Article

PULMONARY FUNCTION TESTS AND THEIR REVERSIBILITY IN SMOKERS

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ABSTRACT

Background: Smoking is known as the major cause of chronic obstructive pulmonary disease (COPD) where most of pulmonary function tests (PFTs) especially those indicating the diameter of airways are reduced. In this study, PFTs of smokers were compared with those of nonsmokers, and the effect of bronchodilator inhaler (salbutamol) on PFTs of smokers was also examined.

Methods: Pulmonary function tests were measured in 97 male smokers and compared with 95 male nonsmokers. Forced vital capacity (FVC), forced expiratory volume in one second (FEV1), maximal mid-expiratory flow (MMEF), peak expiratory flow (PEF), maximal expiratory flow at 75%, 50%, and 25% of the FVC (MEF75, MEF50, and MEF25 respectively) were measured. In addition, pulmonary function tests of 33 male smokers before and 10 minutes after administration of 200 μg salbutamol inhaler were measured.

Results: Most values of PFTs in smokers were significantly lower than those of non-smokers (p<0.001 for FVC, FEV1, PEF, MEF75, p<0.01 for MMEF, and p<0.02 for MEF50). Significant correlations between the smoking duration and FEV1, PEF, MEF75, and MEF50 (p<0.05 to p<0.01) was found, but correlations between smoking quantity and values of PFTs were not significant. All values of PFTs were significantly increased after salbutamol administration (p<0.05 to p<0.01).

Conclusion: The profound effect of smoking on PFT showed that smoking leads to constriction of large and medium sized airways which is mostly due to duration not to quantity of smoking. The airway constriction in smokers was reversible which, was mostly seen for medium sized airways.

Key words: Pulmonary function tests, Smoking, Reversibility, Smoking duration, Smoking quantity

INTRODUCTION

It is observed that Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity throughout the world. A large number of people suffer from this disease for years and die prematurely from it or its complications. COPD is currently the fourth leading cause of death in the world¹, and further increases in its prevalence and mortality can be predicted in the coming decades.²

Long term use of Cigarette smoking is the most important risk factor for COPD and the most important way that tobacco contributes to the risk of COPD.³ Cigarette smokers have a higher prevalence of respiratory symptoms, pulmonary function abnormalities, greater annual rate of decline in FEV1, and a greater COPD mortality rate than those of non-smokers.⁴ These differences between cigarette smokers and non-smokers increase in direct proportion to the quantity of smoking. Smoking leads to rapid decline in pulmonary function tests (PFTs) specially those indicating diameter of airways such as forced expiratory flow in one second (FEV1).⁵ Even in teenagers who have smoked only a few years, maximum expiratory flowvolume curves demonstrate decreases in flow rates at small lung volumes.⁶ If smoking causes changes in small airway caliber at such an early age, one might expect that smoking also causes acute changes in these small airways.

Until now, the only welldocumented acute effect of smoking on the airways was the decrease of airway conductance demonstrated by Nadel and Comroe.⁷ The obstruction to airflow that develops in 15 to 20% of heavy smokers is thought to be due to abnormalities in airways with less than 2 mm internal diameter.⁸ Previous studies from several laboratories have shown
that this airway obstruction is associated with chronic inflammatory process in the membranous and respiratory bronchiolus.\textsuperscript{9, 10} It is believed that the airway constriction in COPD and decline in PFT are not reversible. Therefore, in the present study the pulmonary function tests of smokers were compared with those of non-smokers. The effect of quantity and duration of smoking on PFT and the reversibility of PFT were also evaluated in the present study.

\section*{MATERIAL AND METHODS}

\textbf{Subjects:} Ninety seven smokers (age 36.49±13.06, range 19-71 years old and height 171.71±6.68 range 158-190) and ninety five non-smoker men (age 35.56±12.83, range 18-65 years old and height 171.79±8.81 range 154-194) were randomly selected from the visitor of our tertiary care centre (Table 1). All subjects had no history or symptoms of cardiovascular or respiratory diseases that required treatment (excluding the common cold). The protocol was approved by the “Ethics Committee” of our institution, and each subject gave informed consent. Measurement Expiratory flow-volume curves were recorded by a spirometer. The spirometer was calibrated daily for few days at the beginning, at the end and a few intervals during the middle of the study. However, because there were almost no differences in daily calibrations, calibration of the spirometer was carried out weekly in the rest of the study. All tests were conducted by a researcher who was fully trained regarding the procedure of spirometry. Prior to testing, the required manoeuvre was demonstrated by the operator, and subjects were encouraged and supervised throughout the test performance. Studies were performed using the acceptability standards outlined by the “American Thoracic Society” (ATS) with subjects in a standing position and wearing nose clips.\textsuperscript{11} All tests were carried out between 1000 and 1700 hours. In 33 smokers, PFTs were repeated 10 min after 200 $\mu $g inhaled salbutamol. Pulmonary function tests were performed three times in each subject with an acceptable technique. The highest level for forced vital capacity (FVC), forced expiratory volume in one second (FEV1), maximal mid-expiratory flow (MMEF), peak expiratory flow (PEF), and maximal expiratory flow at 75\%, 50\%, and 25\% of the FVC (MEF75, MEF50, and MEF25 respectively) were taken independently from the three curves.

\textbf{Data analysis:} The data of height, age, and pulmonary function parameters were expressed as mean±SD. PFTs of smokers were compared with those of non-smokers using unpaired t-test. PFTs obtained after inhaled salbutamol were compared with the baseline values using paired t-test. The duration and quantity of smoking were related to decrease in their PFT values, using the least square regression. The criterion of significance was $p<0.05$.

\section*{RESULTS}

\textbf{Duration and quantity of smoking:} Mean duration of smoking was 17.41±4.68 years (range 2-50 years) and mean quantity of smoking was 12.09±9.68 cigarettes per day (range 0.25-50), (Table 1).

\textbf{Pulmonary function tests:} All values of pulmonary function tests in smokers were significantly lower than those of non-smoker subjects ($p<0.02$ to $P<0.001$) except MEF25 (Table 2).

\textbf{Correlation between duration and quantity of smoking with PFTs in smokers:} There were significant negative correlation between duration of smoking and decrease in FEV1, PEF, MEF75, and MEF50 ($p<0.05$ to $p<0.01$). However, the correlations between the quantity of smoking and values of PFT were not significant.

\textbf{Low PFTs among smoker and nonsmoker subjects:} The percentage of low values of most PFTs (lower than 80\% predictive values) among smoker was significantly more than those of normal subjects (Table 3). Only 0-10.6\% of non-smokers had low PFT values while in 21.6-42.3\% of smokers PFT values were lower than normal range.

\section*{Table 1: Characteristics of study population}

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non Smokers (n=95)</th>
<th>Smokers (n=97)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>Mean±SD</td>
<td>Range</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>154-194</td>
<td>171.79±8.81</td>
</tr>
<tr>
<td>Age (year)</td>
<td>18-65</td>
<td>35.56±12.83</td>
</tr>
<tr>
<td>Amount (cigarettes per day)</td>
<td>0.25-50</td>
<td>12.09±9.68</td>
</tr>
<tr>
<td>Duration (years)</td>
<td>2-50</td>
<td>17.41±9.68</td>
</tr>
<tr>
<td>FVC</td>
<td>71.36-134.50</td>
<td>95.71±12.22</td>
</tr>
<tr>
<td>FEV1</td>
<td>80.90-184.40</td>
<td>102.04±17.29</td>
</tr>
<tr>
<td>MMEF</td>
<td>69.70-239.60</td>
<td>104.15±20.33</td>
</tr>
<tr>
<td>PEF</td>
<td>61.50-150.90</td>
<td>100.13±16.84</td>
</tr>
<tr>
<td>MEF75</td>
<td>51.50-170.50</td>
<td>105.62±21.65</td>
</tr>
<tr>
<td>MEF50</td>
<td>52.10-213.30</td>
<td>104.28±28.89</td>
</tr>
<tr>
<td>MEF25</td>
<td>68.50-223.80</td>
<td>110.48±27.58</td>
</tr>
</tbody>
</table>

FEV1: forced expiratory volume in one second; FVC: forced vital capacity; MMEF: maximal mid-expiratory flow; PEF: peak expiratory flow; MEF75, MEF50, and MEF25: maximal expiratory flow at 75\%, 50\%, and 25\% of the FVC, respectively.

All values of PFTs were quoted as percentage predicted.
Effect of salbutamol on PFTs of smokers

Pulmonary function tests of 33 male smokers (height 172.79±11.94 cm, age 38.30±6.65 years) before and 10 min. after administration of 200 μg salbutamol inhaler were measured. All values of PFT in smokers significantly increased 10 min. after 200 μg inhaled salbutamol (p< 0.005). The enhancement in PEF, MEF75, and MEF50, was around 12% and that of MEF25 was 17% (Table 4).

**DISCUSSION**

This study has shown reduction of all values of pulmonary function testvalues in smokers as compared to those nonsmoker subjects. Although the mean values of PFTs in smokers was in normal range (83.78±16.83 to 110.84±42.42), they were significantly lower than PFT values in normal subjects. However, in 21.6-42.3 % of smokers, the values of PFT were lower than normal range, while only 0-10.6% of normal subjects had low values of PFT. In addition, relatively younger smoker subjects had low values of PFT compared to normal subjects. Previous studies also showed reduction of different values of PFT among smokers comparing to normal subjects.

The result of the present study showed the reduction in PEF and MEF75 among smoker subjects was significantly more than other values of PFT. These results may indicate that in smoker subjects medium and large airways are affected more than other airways. The results of our study were supported by previous studies indicating reduction of PFTs in smokers. However, there is some evidence that small airways are affected more by smoking. The present study also showed negative correlation between decrease in most values of PFT and duration of smoking.

However, the relationships between decrease in PFTs and quantities of smoking were not significant. These results showed that duration of smoking has more profound effect on airways than quantity of smoking. The studies of Sherrill et al. and Verschakelen et al. also showed correlation between smoking and reduction in most values of PFT which support the results of the present study. In addition, Burrows et al. also showed quantitative relationship between cigarette smoking and reduction in values of PFT.

Furthermore, the results of the present study, showed that the values of PFT of smokers were significantly increased due to 200 μg inhaled salbutamol indicating some degree of reversibility of the airway constriction in smokers. Although the mean value of MEF25 among smokers was normal, increase in this value of PFT due to salbutamol administration was more than other values of PFT. This may indicate that in smokers small airways are more liable to reversible constriction. It is believed that airway constriction of COPD patients is not reversible, or there is very small reversibility of airways in these patients. However, the results of our study demonstrated a relatively large component of reversibility of airways in smokers, which is a novel finding of the present study.

**CONCLUSION**

The results of the present study demonstrated the profound effect of smoking on PFT and therefore, indicated that smoking leads to constriction of large and
medium airways, which is mostly due to duration, not to quantity of smoking. The airway constriction in smokers was reversible which was mostly seen for medium and small sized airways.

REFERENCES