COMPARATIVE STUDY OF PULMONARY FUNCTION TESTS IN SMOKERS AND NON SMOKERS.

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ABSTRACT

Smoking is one of the most harmful and addictive habits which is wide spread all over the world. Deleterious effects of tobacco are seen on all body systems but most markedly on respiratory and cardiovascular systems. Nicotine present in tobacco is the most dangerous of all psychoactive substances which is harmful to human race. Approximately 40% of cigarette smokers will die prematurely due to cigarette smoking unless they are able to quit.

The present study was conducted on human male subjects comprising of 75 apparently healthy smokers of ages ranging from 20-25, 30-35 & 40-45 years and an equal number of healthy non smokers of the same age groups. Statistical analysis was carried out using standard deviation and chi-square from which ‘p’ value is derived. The FVC, FEV1, PEFR & FVC25-75% are effort dependent and are decreased uniformly in smokers when compared with non-smokers. This suggests smoking effects respiratory system in a significant way.

In this study there is decrease in PFT values with increasing age which suggests natural respiratory changes with age. Smokers showed much more lower values as compared to non-smokers which reflects that smoking has a definite bad impact on the natural age related changes of respiratory system.

KEYWORDS: PFT, Smoking, PEFR, FVC, FEV1, FEF(25-75).

INTRODUCTION

Tobacco is believed to have been introduced in India by the Portuguese in 17\(^{th}\) Century. At present India is the 3\(^{rd}\) largest producer of tobacco and has a dubious distinction of consuming 80% of its production. It is estimated that about 35% of Indian men are smokers. Cigarette smoke plays a predominant role and is undoubtedly the major risk factor for the development of emphysema and chronic bronchitis.

EFFECTS OF CHRONIC SMOKING- The final outcome in chronic smoking is reduced life span. At 35 years of age assessment of expected mortality by 65 years is significantly different in non-smokers ,smokers and heavy smokers.

<table>
<thead>
<tr>
<th>NON SMOKERS</th>
<th>SMOKERS:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 to 14 Cigarettes a day----- 22%</td>
<td></td>
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<tr>
<td>15 to 25 Cigarettes a day - 25%</td>
<td></td>
</tr>
<tr>
<td>25 to more Cigarettes a day- 40%</td>
<td></td>
</tr>
</tbody>
</table>

The risk of death from lung cancer is related to number of cigarettes smoked and the age of commencement of smoking. Earlier in life a person starts to smoke, greater is the risk of early death. Giving up smoking reduces risk of death.

SMOKING AND CANCER: Between 1920 and 1950 an epidemic of bronchial carcinoma occurred. Cigarette smoking satisfied the criteria of an association between bronchial carcinoma and smoking. Pipe and cigar smokers
are found to have lesser risk than cigarette smokers probably because they inhale less.

SMOKING AND CHRONIC LUNG DISEASE: The adverse effects of cigarette smoke on the lungs may be separated into two distinct conditions. These are chronic bronchitis which involves large air ways and usually clears up when the subject stop smoking. While COPD which involves medium and small air ways.

PASSIVE SMOKING: Many non-smokers are exposed to tobacco smoke at home, at working place, on public transport and in public places. People can scarcely avoid breathing air contaminated with tobacco smoke. This mode of smoke inhalation is termed as passive smoking.

THE EFFECTS OF SMOKING ON LUNG FUNCTION: Functional defect in smokers with chronic obstructive pulmonary disease is that of air flow obstruction. Cigarette smoking is overwhelmingly the most important cause of cough and mucus over production.

In the National Health and Nutrition Survey, prevalence of cough and phlegm increased 3 fold in male smokers and 2 fold in female smokers. Both cross sectional and longitudinal studies support in reduction in symptom prevalence in smokers who quit smoking.

Pathogenicity of chronic obstructive pulmonary disease (COPD) depends on risk factors and genetic susceptibility. Exposure to cigarette smoke is the major factor in the pathogenesis of COPD to produce airway obstruction by loss of elastic recoil and airway inflammation.

Smoke causes an increase in the number of pulmonary alveolar macrophages. macrophages release a chemical substance that attracts leucocytes in lung. Leucocytes in turn release proteases including elastase which attack elastic tissue in lung. Normally alpha-1antitrypsin inactivates elastase and other proteases. Alpha-1 antitrypsin is inactivated by oxygen radicals which are released by the leucocytes leading to protease-antiprotease imbalance with increased destruction of lung tissue.

COMPOSITION OF TOBACCO SMOKE: Cigarette smoke is a heterogeneous mixture of gases and particulate substances produced by burning of tobacco leaf. A large number of chemical components are released from the burning end of the cigarette. About 4000 compounds have been identified, some of them are carcinogenic and others have injurious effects. Well known carcinogenic substances present are nitrosamines and hydrazine. Tobacco smoke contains many ciliotoxins, which damage cilia. Pharmacologically active compound is nicotine which causes acute effects on respiratory and cardiovascular systems.

Tobacco smoke contains 1 to 5% carbon monoxide. Habitual smokers have 3 to 7% and heavy smokers as much as 15% of their haemoglobin as carboxyhaemoglobin which cannot carry oxygen. This is sufficient to reduce exercise capacity in patients with angina pectoris.

TOBACCO DEPENDENCE: There is no major personality difference between smokers and non-smokers, cigarette smokers tend to be more extroverted, less rigid and more prone to anti-social tendencies. Psychological dependence is extremely strong and accounts largely for the difficulty in stopping smoking.

MATERIALS AND METHODS

The study was conducted in the pulmonary laboratory of department of tuberculosis and chest diseases, at Government Fever Hospital, Guntur. Lung function tests of the particular study was done using Computerized Spirometer model no “SPIRO 232” P.K. Morgan Medical Ltd. The subject was asked to stand in front of the spirometer and was asked to open his mouth. The mouth piece of the spirometer was introduced into his mouth and he was asked to close the lips around the mouth piece tightly. His nostrils were pinched with nasal clip. Immediately after the deep inspiration he was asked to blow out the air as fast as possible into the mouth place. Soon after this he was asked to take a deep or maximal inspiration, it was ensured that the inspiration was full and unhurried and the expiration once begun was continued without pause.

TEST PARAMETERS chosen for this study are:

1. FORCED VITAL CAPACITY (FVC):-This is the volume of the air that can be expired force fully and maximally after taking a deep and maximal inspiration.

Normal value ----3.5-5.5 liters.

2. FORCED EXPIRATORY VOLUME IN 1ST SECOND (FEV1): It is the amount of air that can be expired forcefully and maximally in the 1st second after a maximal inspiration. Normal values 80%-85% or 4-4.5 liters.

3. PEAK EXPIRATORY FLOW RATE (PEFR):- It is the amount of air that can blown out of fully inflated lungs as rapidly as possible. Normal Values 6-15 liters/sec.In adults is 400 liters/ mt.
RESULTS AND DISCUSSION

The forced vital capacity in the smokers of 20-25 years of age (Group I B) was compared with the non smokers of the same age group (Group-I A) was reduced by 11% (P<0.001). The forced vital capacity in the smokers of age 30-35 years (Group II B) was compared with the non smokers of the same age (Group II A) showed a decrease of 13% (p<0.001) in smokers. The forced vital capacity in the smokers of 40-45 years of age (Group III B) was compared with the non smokers of the same age group (Group-III A) was reduced by 17% (P<0.001).

The forced expiratory volume in 1st second (FEV1) of 20-25 years of age (Group-I B) was compared with the non smokers of the same age group (Group-I A) was reduced by 13% (p<0.001). The forced expiratory volume in 1st second (FEV1) in the smokers of age group (Group-II B) was compared with the non smokers of the same age group i.e., (Group-II A) showed a decrease of 14% (p<0.001). The forced expiratory volume in 1st second (FEV1) of 40-45 years of age (Group-III B) was compared with the non smokers of the same age group (Group-III A) was reduced by 15% (p<0.001).

The peak expiratory flow rate of 20-25 years age group in smokers (Group-I B) was compared with peak expiratory flow rate of non smokers of the same age group (Group-I A) was reduced by 8% (p<0.001). The peak expiratory flow rate of 30-35 years age group in smokers (Group-II B) was compared with peak expiratory flow rate of non smokers of the same age group i.e., (Group-II A) showed a decrease of 12% (p<0.001). The peak expiratory flow rate of 40-45 years age group in smokers (Group-III B) was compared with peak expiratory flow rate of non smokers of the same age group (Group-III A) was reduced by 15% (p<0.001).

The forced expiratory flow 25 to 75% of the 20-25 years age group in smokers (Group-I B) was compared with the same age group of non smokers in Group-I A showed a decrease of about 8% (p<0.001). The forced expiratory flow rate 25 to 75% of 30-35 years age group in smokers (Group-II B) was compared with the same age group of non smokers (Group-II A) showed a decrease of 13% (P<0.001). The forced expiratory flow 25 to 75% of the 40-45 years age group in smokers (Group-III B) was compared with the same age group of non smokers in Group-III A showed a decrease of about 16% (p<0.001).

There is significant reduction in the flow rates was in consistent with the study of Beck G.J,Byerley.P.M,Weitz.CA,Rivhards.F et al showed that cigarette smoking causes significant reduction in the pulmonary functions, which is also in agreement with our study. 

The present study is based on two very similar groups of people. The smokers and non-smokers are comparable. Nevertheless, there were some marked differences in the results in these two groups. The lung capacities, volumes and ventilatory flow rates have shown significant differences between smokers and non-smokers; lower values are obtained in the former group. When the subjects were grouped, these differences persisted both in the young and older subjects. These observations are in accordance with those of others. [9] WALTER et al. (1979) showed significant changes in lung function in young smokers. Moreover, in this study these changes were dose-related. The early changes in smokers are probably due to narrowing of the small airways. The present observations are in accordance with this view which also coincide with WALTER et al. (1979). 

The present study illustrates the usefulness of the forced expiratory spirogram in evaluating the early changes in lung function in smokers. Judging by the number of smokers who had abnormal results, FEF25-75% was found to be the most sensitive indicator for this purpose, whereas FEV1, FEV1%, FEF75-85% also appeared to be sensitive but not to the extent of FEF25-75%.
parameter is a sensitive indicator for the detection of expiratory flow obstruction. The significant reduction in the flow rates was consistent with the study of Beck G.J. Doyle L.A. Schachter N.

The present study was consistent with the study of Millat W.A. Elganon F.M. who also showed the significant lower values of forced expiratory flow 25-75% and PEFR peak expiratory flow rate in the study with smokers the study of Gold D.R. Wangx, Wypli D. et al on the effect of cigarette smoking on lung function is consistent with our study by reduction in forced expiratory flow FEF(25-75%) and forced expiratory volume in 1st second (FEV1). The decline in physical fitness and lung function among healthy middle aged men was considerably greater among smokers than non smokers by Sandvik L, Brikssen G, Thalow E was also consistent with our study. The decrease in the forced expiratory volume in 1st second (FEV1) which was seen in our study is found consistent with the study of Burchael C.M. Marcus E.B. Curb J.D et al. The decrease in FEV1 in smokers in our study was in agreement with work done by Seersholm N; Kokjonson A who found the development of emphysema in smokers at younger age with α1-antitrypsin deficiency subjects.

**Graph – 1**
Comparison of PFT in Group -1
20-25 Yrs

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Non Smokers Mean</th>
<th>Non Smokers SD</th>
<th>Smokers Mean</th>
<th>Smokers SD</th>
<th>T Value</th>
<th>P Value</th>
<th>Crude Value</th>
<th>↓</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>4.976</td>
<td>0.149</td>
<td>4.467</td>
<td>0.743</td>
<td>14.927</td>
<td>0.001</td>
<td>0.509</td>
<td>10%↓</td>
</tr>
<tr>
<td>FEV1</td>
<td>4.077</td>
<td>0.115</td>
<td>3.585</td>
<td>0.051</td>
<td>19.790</td>
<td>0.001</td>
<td>0.492</td>
<td>12%↓</td>
</tr>
<tr>
<td>PEFR</td>
<td>4.678</td>
<td>0.127</td>
<td>4.312</td>
<td>0.093</td>
<td>11.990</td>
<td>0.001</td>
<td>0.366</td>
<td>08%↓</td>
</tr>
<tr>
<td>FEF 25-75%</td>
<td>4.462</td>
<td>0.108</td>
<td>4.104</td>
<td>0.079</td>
<td>13.807</td>
<td>0.001</td>
<td>0.358</td>
<td>08%↓</td>
</tr>
</tbody>
</table>
### Table 2: Comparison of PFT in Group II A & B Male Non Smokers and Smokers of 30-35 Yrs

<table>
<thead>
<tr>
<th></th>
<th>Non Smokers</th>
<th>Smokers</th>
<th>T Value</th>
<th>P Value</th>
<th>Crude Value</th>
<th>↓</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>4.152</td>
<td>3.616</td>
<td>5.336</td>
<td>0.001</td>
<td>0.536</td>
<td>13% ↓</td>
</tr>
<tr>
<td>FEV1</td>
<td>3.254</td>
<td>2.796</td>
<td>5.668</td>
<td>0.001</td>
<td>0.464</td>
<td>14% ↓</td>
</tr>
<tr>
<td>PEFR</td>
<td>4.323</td>
<td>3.632</td>
<td>2.958</td>
<td>0.01</td>
<td>0.690</td>
<td>16% ↓</td>
</tr>
<tr>
<td>FEF 25-75 %</td>
<td>4.154</td>
<td>3.426</td>
<td>3.130</td>
<td>0.01</td>
<td>0.728</td>
<td>18% ↓</td>
</tr>
</tbody>
</table>

**Graph 2: Comparison of PFT in Group II 30-35 Yrs**
TABLE – 3: COMPARISON OF PFT IN GROUP III A & B
MALE NON SMOKERS AND SMOKERS OF 40-45 Yrs

<table>
<thead>
<tr>
<th></th>
<th>NON SMOKERS</th>
<th>SMOKERS</th>
<th>T Value</th>
<th>P Value</th>
<th>Crude Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC</td>
<td>2.490</td>
<td>2.064</td>
<td>4.462</td>
<td>0.001</td>
<td>0.432</td>
</tr>
<tr>
<td>FEV1</td>
<td>2.021</td>
<td>1.714</td>
<td>6.193</td>
<td>0.001</td>
<td>0.307</td>
</tr>
<tr>
<td>PEFR</td>
<td>3.102</td>
<td>2.636</td>
<td>19.640</td>
<td>0.001</td>
<td>0.466</td>
</tr>
<tr>
<td>FEF 25-75%</td>
<td>2.912</td>
<td>2.436</td>
<td>18.776</td>
<td>0.001</td>
<td>0.476</td>
</tr>
</tbody>
</table>

CONCLUSION

Tobacco smoking is an important causative factor in a number of lung diseases. Studies revealed that smoking accounts for 80-90% risk of developing chronic obstructive pulmonary disease and 80-85% of Bronchogenic Carcinoma. Tobacco smoke causes wide influence on the general health. In smokers, changes occur in respiratory system due to inflammation and fibrosis. So all the dynamic pulmonary parameters under consideration are significantly lower than the normal values.

The FVC, FEV1, PEFR & FVC25-75% are effort dependent and are decreased uniformly in smokers when compared with non-smokers. This suggests smoking effects respiratory system in a significant way. In this study there is decrease in PFT values with increasing age which suggests natural respiratory changes with age. Smokers showed much more lower values as compared to non-smokers.
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REFERENCES


