Effects of passive smoking on pulmonary functions of individuals in an urban area

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ABSTRACT

Background: Passive smoking is a world health problem and part of the tobacco epidemic which victimizes mostly adolescents. Research articles have addressed the relation between passive smoking and respiratory efficiency in adult population and escalated the hazard of chronic obstructive pulmonary disease and lung cancer among adult passive smokers. The present study was conducted to analyze the effects of exposure to passive smoke on pulmonary functions among nonsmoking individuals.

Aim and Objectives: The study was determined to assess the effects of passive smoking on pulmonary functions among individuals in urban region.

Materials and Methods: The present study was conducted on smokers, passive smokers, and nonsmokers (control), aged between 17 and 25 years. Ninety healthy individuals were placed in three different groups on the basis of questionnaire (Group A: active smoker, Group B: passive smoker, Group C: control). Study was done with the help of spirometer and pulse oximeter.

Results: Forced expiratory flow \(25–75\%\), Forced expiratory volume in 1 second/Forced vital capacity % values were significantly reduced in passive smokers as compared to control individuals \(P < 0.001, P < 0.05\). Sp\(O_2\) level was decreased in passive smokers and active smokers, but it was not statistically significant.

Conclusions: The present study showed a strong association between passive smoking and pulmonary abnormalities in individuals exposed to passive smoke. Health hazards caused by passive smoking should be controlled by creating awareness and regular health check-ups among people.

KEY WORDS: Passive Smoking; Active Smoking; Forced Expiratory Volume in 1 Second/Forced Vital Capacity %, Sp\(O_2\)

INTRODUCTION

Tobacco smoking has been identified as the major important source of premature mortality.¹ Experts says that tobacco smoke contains over 4000 different compounds in the forms of particles and gases.² When burned, cigarettes release more than 5,000 different chemicals. Many of these chemical compounds are poisonous at least 70 of these chemicals are known to cause cancer, and many are toxic. Passive smoking also known as environmental tobacco smoke (ETS) exposure comprises the amount of tobacco smoke which comes from the burning of tobacco product such as cigarettes or cigar and smokes that is exhaled by smokers.

In closed room, accumulation of smoke and its concentration varies with the number of active smokers, and it is also depends on type of smoking, distance from the active smokers, size of the room and ventilation. Passive smoking or ETS appears to include most tobacco combustion byproducts, including irritants, and cancer producing substances when compared to active smoking.³

“Bidi” (beedi) is the most popular type of tobacco smoke habit in Indian villages and small towns. Bidi, a hand rolled cigarette filled with tobacco stuff and wrapped into Tendu leaf
and tied with a small thread. Another popular smoking product used in various parts of India particularly in northern region is “hookah also known as chillum,” which resembles a pipe. The concentration of nicotine and tobacco chemicals present in the mainstream smoke of these various tobacco products is likely to be different from those present in the mainstream smoke of standard cigarettes and cigar because of the differences in their design. Tobacco Smoke causes a raise of number of macrophages in pulmonary area and bronchiole. Macrophages release a chemical compound that attracts white blood cells in lung. White blood cells in turn release substance known as proteases and elastase which attack elastic connective tissue in lung which is important for elasticity and extensibility property of lung tissue. In general, alpha-1-antitrypsin is a protein produced in liver tissue is inactivated by oxygen radicals released from white blood cells which results in formation of imbalance between protease and antiprotease functional activity leading to destruction of lung tissue. Exposure to passive smoking is linked with various health effects in nonsmoking individuals such as heart disease, lung cancer, bronchial asthma, and chronic obstructive pulmonary disease (COPD).[3] Several studies have been conducted to investigate the effect of ETS on pulmonary function in developing countries like India. Environmental conditions such as congested areas, poor ventilated homes, household crowding, the health effects of ETS exposure may be even more strongly marked. Besides, the effects of ETS exposure on young adults are important, as their pulmonary and immune system are still in developmental stages, and comes under high risk category of being affected by harmful chemicals from passive smoke. Such environment is more common in developing countries like India, because of its large population. The present study was undertaken to evaluate the effects of exposure to passive smoking among young adults.

MATERIALS AND METHODS

The study was carried out at department of physiology, ESIC Medical College and PGIMSR, Chennai. It was a cross-sectional study. Active smokers, passive smokers, and nonsmokers (control), aged between 17 and 25 years were included in the present study. Only healthy male subjects were included in the present study. No female subjects were included. The volunteers for the present study were recruited from our institution. Ninety healthy individuals were placed in three different groups on the basis of questionnaire (Group A active smoker, Group B passive smoker Group C control). The study was approved by Institutional ethics committee (Approval No: 16-03/07/2015) and an informed consent was taken from all the subjects after explaining the test procedures and the goal of the study in local language. Experiments were done in accordance with Helsinki declaration of 2000.

Study Group

Ninety healthy age matched volunteers were divided into three groups. Individuals with history of cigarette smoking more than 3 year were considered as active smokers in group A (Active smokers-male), Non-smoking male subjects those who stayed with at least one smoker or interacted regularly with smokers at work place for at least 3 years before the study were included in Group B (Passive smokers-male), Subjects who were neither passive nor active smokers were included in Group C (control group-male). Each group consists of 30 individuals.

Inclusion Criteria

Healthy individuals in the age group of 17–25 years.

Exclusion Criteria

Subjects on medication for respiratory, cardiovascular and central nervous system disorders. Subjects with past and present history of cardio pulmonary disorders, diabetes, psychiatric illness and drug abusing.

Test parameters chosen for this study are:

- Peak expiratory flow rate (PEFR) (L/S), forced expiratory volume in first second of FVC (FEV1) (L), vital capacity (VC) (L), forced vital capacity (FVC) (L), FEVI/FVC (%), forced expiratory flow (FEF) 25-75%, maximum voluntary ventilation (MVV), oxygen saturation (SpO2).

Study Procedure

Study was done with the help of spirometer (RMS Helios 401). The whole procedure was explained to the subject thoroughly. Anthropometric measurements (height, weight, Body Mass Index [BMI], body surface area [BSA]) were recorded on each individual. Appropriate data (name, age, sex, height, weight) were registered in computer program. The subjects were given proper instructions before each parameter. Pulmonary function tests were performed with all subjects in upright position wearing nose clips.[4,5] The subjects were asked to take deep inspiration and then to expire as forcefully and as fast as he can inside the mouthpiece. Tests were repeated 3 times and the best efforts were recorded for statistical analysis. SpO2 refers to the percentage (%) of the hemoglobin binding with oxygen (%). Pulse oximetry was part of our extended examination. SpO2 was measured with a non invasive digital handheld pulse oximeter (BPL Smart Oxy fingertip pulse Oximeter, India). The examinees were asked to relax at least 10–15 min before examination. SpO2 was then measured for about 1 min by attaching the sensor to the middle finger. SpO2 higher than 95% are listed normal, values below 93% shows that oxygen therapy is necessary and require close observation and monitoring of the person.

Statistical Analysis

Statistical analysis was performed with Statistical Package for Social Sciences software (version 17.0). The data are
expressed as mean±Standard deviation, and the statistical analysis of data was done using one-way analysis of variance ANOVA with 5% level of significance.

RESULTS

In the present study, Table 1 showed mean value of anthropometric parameters (physical parameters) of Group A (active smokers), Group B (passive smokers), and Group C (control). There was no significant difference in the mean physical parameters such as BMI and body surface area etc among Group A, Group B, and Group C (control). Figure 1 shows lung functions (FVC, PEFR, FEV1, VC) of Group A, B and C. The mean values of FVC (P = 0.787), PEFR (P = 0.408), FEV1 (P = 0.926), and VC were decreased in Group A and Group B as compared to Group C (control individuals). Mean values of VC were decreased significantly in active smokers (P = 0.010); however, there were no significant changes in FVC, PEFR, and FEV1 values among Group A and Group B as compared to Group C (control subjects) [Figure 1]. Figure 2 MVV shows mean values of MVV among Group A, B and C (control subjects) but there were no significant change among these three groups (P = 0.269). Figure 3 shows FEV1/FVC % of Group A (Active smokers), Group B (Passive smokers), and Group C (Control subjects). It was significantly decreased in active smokers as compared to control individuals (P = 0.022). Figure 4 shows FEF 25–75% among group A, B and C. FEF 25–75% values were highly significant in Group B as compared to Group C (control) (P < 0.001). The mean value of S$_{pO2}$ in group A was decreased than group B and C (control subjects) but it was not statistically significant [Table 2].

DISCUSSION

At present, tobacco smoking is treated to be one of the detrimental to human behaviors and the most dangerous of addictions. Recently, sizeable attention has been fixed on the harmful effect of “passive” smoking, confirming ETS as a serious public health problem, with specific impact on respiratory health of growing children. Some research articles have also stated this effect in adults.[6] Exposure to passive smoke, results in development of diseases in lower and upper airways such as bronchial asthma, wheeze.[7,8] Nowadays, the risk of development of COPD is significantly

<p>| Table 1: Physical characteristics of active smokers (A. Smokers), Passive smokers (P. Smokers) and control subjects |</p>
<table>
<thead>
<tr>
<th>Variables</th>
<th>A. Smoker Mean±SD*</th>
<th>P. Smoker Mean±SD*</th>
<th>Control Mean±SD*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24.10±10.11</td>
<td>23.19±12.09</td>
<td>23.25±11.08</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.70±0.19</td>
<td>1.71±0.22</td>
<td>1.71±0.12</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>59.5±9.20</td>
<td>61.4±12.06</td>
<td>60±10.02</td>
</tr>
<tr>
<td>Body mass index</td>
<td>21.7±2.21</td>
<td>21±3.02</td>
<td>21.5±2.10</td>
</tr>
<tr>
<td>Body surface area (m$^2$)</td>
<td>1.70±0.09</td>
<td>1.72±0.02</td>
<td>1.70±0.06</td>
</tr>
</tbody>
</table>

SD*: Standard deviation

| Figure 1: Pulmonary function tests (forced vital capacity, peak expiratory flow, forced expiratory volume in 1 second and vital capacity) among active smokers (A. Smokers), Passive smokers (P. Smokers) and control subjects |
| Figure 2: Maximum voluntary ventilation among active smokers (A. Smokers), passive smokers (P. Smokers) and control subjects |
| Figure 3: Forced expiratory volume in 1 second/forced vital capacity ratio (Forced expiratory volume in 1 second/Forced vital capacity %) among active smokers (A. Smokers), passive smokers (P. Smokers) and control subjects |
| Figure 4: Forces expiratory flow (FEF 25–75%) among active smokers (A. Smokers), passive smokers (P. Smokers) and control subjects |
higher in passive smokers. Worldwide, many individuals such as children, male and female non-smokers were exposed to ETS in 2004. This disease is most likely to be the top third cause of mortality.[19]

NF 25–75 measures airway flow rates on an FVC segment. The present study showed significant reduction of NF 25–75 values in passive smokers (P = 0.001) [Figure 4: NF 25–75% among Active smokers, Passive smokers and Control subjects]. It is in agreement with previous studies[10,11] showing that NF 25–75% values were significantly decreased in passive smokers. White et al.[12] reported small airways dysfunction with a 14% reduction in NF 25–75% in non-smokers exposed to ETS; Kauffman et al.[13] studied the effect of exposure to cigarette smoke at home on lung function in French men and showed a 6% fall in NF 25–75% Masi et al.[14] found adverse effects of passive smoking on NF 25–75% in young men (aged 15–35). Our results were in agreement the findings of White et al., Kauffman et al., Masi et al. Another study done by Casale et al.,[15] who investigated the effects of passive smoking on lung function of children aged 6–11 years old, and found the NF 25–75% to be significantly reduced in study group.

MVV (L/min) is a dynamic and scientific method for measurements of respiration muscles capacity and indeed, is a test for evaluation of human respiration system. The MVV was formerly called the maximum breathing capacity is the largest volume of gas that can be moved in and out of the lungs in 1 min by voluntary effort. The normal MVV is 125–170 l/min. In the present study, the MVV was decreased when compare to control group, but it was not statistically significant [Figure 2: MVV among Active smokers, Passive smokers and Control subjects]. In the present study, VC was decreased in passive smokers and smokers, but it did not reach the level of statistical significance in passive smokers but it was significantly decreased in active smokers. It is in agreement with Gupta.[16] who could not able to find out significant changes in VC among passive smokers.

FEV1 is an important part of evaluating COPD and monitoring progression of the condition. Some studies shows that the FEV1 values of children were significantly reduced by parental smoking.[17] More recent findings reported by Merghani and Saeed[18] who studied 135 young male students (9–14 years old) in Khartoum, Sudan, and found the FEV1 and FVC to be significantly lower in the passive smoke exposed group than the nonsmoker control group. Our study does not show significant difference in FEV1 and FVC values. Kentner et al.[19] carried out an investigation involving 1,351 white collar workers. He could not able to find out significance difference in FVC. Our findings were in agreement with Kentner et al. Another study from Mohammad-Reza Masjedi et al. Shows significant reduction of FEV1 and FVC values in male passive smoker.[20] Results of previous studies regarding FEV1 are more consistent, with a majority showing a lower FEV in children exposed to parental smoking.[21] In the present study, we did not find any significant changes in FEV1 and FVC parameters in passive smokers but it shows lower values in passive smokers however it did not show statistical significance. It might be due to nature of exposure of passive smoking, many individuals in our study were exposed by passive smoking in open air. This might not produce changes in FEV1 and FVC. The FEV1/FVC ratio (FEV1/FVC%), also called Tiffeneau-Pinelli index,[22] is a calculated ratio used in the diagnosis of obstructive and restrictive lung disease.[23] It represents the proportion of a person’s VC that they are able to expire in the first second of forced expiration FEV1 to the full, FVC. According to recent studies, even 1 h of passive smoking exposure can induce a significant decrease in FEV1 and FEV1/FVC ratio along with cytokine releases.[24] The present study showed significant changes in FEV1/FVC ratio (P = 0.02) [Figure 3: FEV1/FVC ratio (FEV1/FVC %) among Active smokers, Passive smokers and Control subjects]. It is in agreement with Meenakshi Kalyan et al.[25] Smoking in an enclosed area increases the concentration of respirable particles such as nicotine, polyacrylic hydrocarbons, carbon monoxide (CO), acrolein and nitrogen dioxide. The impact of smoking on indoor air quality depends on the number of smokers, the intensity of smoking, the size of the indoor space, the rate of exchange of the air of the indoor space with the outdoor air, and the use of air cleaning devices. In our study less number of people has exposure on passive smoking in enclosed area. There are some limitations that may influence the interpretation of our cross sectional results. We were unable to determine the extent of indoor air pollution in the homes of the subjects. In addition, we lacked information about status of air pollution in their living area and we couldn’t be able to get proper details regarding ventilation, population in their places. Some reports have linked cooking energy sources to impairment of ventilatory function, whereas others have not found such an effect.[26] It needs further detailed analysis. Smoking is one of the most important causes of cardiovascular diseases and deaths from these diseases. The most effective components of cigarette smoke in the cardiovascular system are nicotine and CO. Both molecules affect oxygen requirement in heart muscle. CO reduces

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study sample</th>
<th>Mean±SD</th>
<th>f-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S_{O_2}</td>
<td>A. Smoker</td>
<td>97.56±0.50</td>
<td>f=1.93682</td>
<td>0.15032&lt;sup&gt;0.04&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>P. Smoker</td>
<td>97.7±0.46</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>97.8±0.40</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SD*: Standard deviation, S_{O_2}: Oxygen saturation, NS: Not significant

Table 2: S_{O_2} among active smokers (A. Smokers), passive smokers (P. Smokers) and control subjects.
oxygen supply to tissues of the body by binding to proteins such as hemoglobin and form carboxyhemoglobin. This carboxyhemoglobin level generally increases 2–15 times in active smokers than compare to non smokers. Mustafa Özda et al. Studied SpO₂ among male and female smokers and found significant difference.\(^{(27)}\) Low SpO₂ and partial pressure of oxygen in arterial blood (PaO₂) in smokers have been shown in previous studies.\(^{(28)}\) Heavy smokers may have their saturation overestimated, since high carboxy hemoglobin levels may give overestimation of true SpO₂. In our study, we couldn’t be able to find out any significant difference among smokers, passive smokers and non smokers (control). This might be due to most of the subjects in our study belongs to adolescent category with less exposure to cigarette smoking.

**Strength and Limitations of the Present Study**

There are a few limitations of the study. In the present study, only male subjects had participated in the research. Hence, in feature, we would like to include female subjects to reach concrete conclusion. The Present study will give an impact to understand about passive smoking exposure in relation to the lung functions such as COPD among participants.

**CONCLUSIONS**

The present study suggests a significant reduction of lung functions in individuals exposed to passive smoke. Passive smoke exposure is an independent risk factor for developing COPD and passive smokers are at similar risk like active smokers. Pulse oxymeter is a simple affordable and reliable technique and helps to evaluate SpO₂. It may be particularly useful in risk assessment with spirometry. In light of this, further research is needed to understand the status of SpO₂ in chronic smokers.

**REFERENCES**


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