Relationship between Smoking and Pulmonary Functions

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Abstracts: Background: Smoking is the most important factor contributing to the development of chronic obstructive pulmonary disease and is one of the major health risks in modern times. Aim: The purpose of the present study was to determine the relationship between cigarette smoking and pulmonary function tests between various groups of smokers and non-smokers. Methods: The study was carried out in 100 male subjects between 19-52 years of age. The subjects were drawn from the community such that they could be grouped as non-smokers (25), mild smokers (25), moderate smokers (25), and chronic smokers (25) according to their questionnaire response. Pulmonary Function Tests were carried out in each subject with a computerized spirometer. The various data was collected, compiled, statistically analyzed and valid conclusions were drawn Results: Results indicate that smoking is generally associated with lower levels of pulmonary functions. . It was established that pulmonary functions decreased with increasing number of pack years. The negative association was evident in most lung functions and capacities, but was largest and most progressive in FEV_1, FEV_1/FVC, $FEF_{25-75\%}$ and PEFR. Conclusion: Pulmonary function data in smokers indicate narrowing of smaller airways, chiefly bronchioles. Rapidly declining pulmonary functions in smokers with increasing number of pack years is predictive of increased risk of development of chronic obstructive pulmonary disease (COPD). The study observed that spirometry was an effective and easy method for detection of COPD in risk group population like smokers and thus promotes smoking cessation efforts to reduce the burden of COPD in the community. [Thaman R G et al NJIRM 2011; 2(4) : 1-6]

Key Words: Smoking, Pulmonary function tests (PFTs), Lung volumes and capacities, Chronic obstructive pulmonary diseases (COPD).

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Introduction: Smoking is a public health problem and a major cause of many preventable diseases and premature deaths all over the world. Cigarette smoking is the most important factor contributing to the development of chronic obstructive pulmonary disease. It is now well established that cigarette smoking for only a few years causes early changes in the peripheral airways of the lung[1]. Soon after commencing the smoking habit, the body becomes used to absorbing so much nicotine regularly that it eventually demands more and more. To obtain the same stimulation, more cigarettes are required as the body becomes incurred to the smaller amounts of nicotine. Also the effect does not last long even if a larger dose is taken in the form of either "Stronger" cigarettes or more cigarettes in shorter time. Thus excessive smoking becomes a vicious circle [2].

Tobacco is the dried leaf of Nicotianna Tobaccum, a plant indigenous to America but now grown in many parts of the world. The poisonous properties of tobacco are due mainly to the presence of nicotine, a heavy oil substance. The amount of nicotine in a pound of tobacco is estimated to be, on an average 377 grains (range 0.5 to 8 %) and this alkaloid is so poisonous that one-tenth of a grain given intravenously can kill a dog in three minutes. Cigarette tobacco contains, on an average 1.5% nicotine and thus an average cigarette of one gram may yield as much as quarter grain to even half grain of the nicotine. When one smokes, heat liberates nicotine in varying degree into smoke; some of the alkaloid is burnt but appreciable quantities gain access to the respiratory tract, depending upon moisture of the tobacco, filtration, heat, rapidity of smoking the depth of inhalation[3].
Bhinde studied the chemical analysis of smoke of Indian cigarettes, bidis and other ingenious forms of smoking levels of steam volatile phenol, hydrogen cyanide and benzopyrene [3]. Besides nicotine, some other specific components of total particulate matter 'TPM' like steam volatile phenol; HCN (hydrogen cyanide) and benzopyrene are known to be hazardous to health. It has been well established that cigarette smoking is a major risk

factor for lung cancer, coronary heart disease and chronic obstructive pulmonary disease (COPD).

Cigarette smoking is the most important factor contributing to the development of chronic obstructive pulmonary disease. It is now well established that cigarette smoking for only a few years causes early changes in the peripheral airways of the lung [4]. The single best thing a smoker can do to improve their lung functions and live a longer life is to stop smoking. It was evident from a recent study that smokers who had their lung functions measured and explained to them in a specific way, were more likely to have quit smoking a year later [5]. The present study has been undertaken to compare between smokers and non-smokers various ventilatory norms using a "Medspior", a computerized spirometer. The spirometer is an effective and easy method for detection of COPD in risk group population like smokers and thus promotes smoking cessation efforts to reduce the burden of COPD and lung cancers in the community [6].

Material and Methods: This study included 100 male subjects between 19-58 years of age. They were further subdivided into following groups:-

Group I (Non-Smokers): 25,Non-Smokers, The subjects having no history of smoking, no current or past history of any Cardio respiratory disorders, exertion dyspnoea, general debility, malnutrition or skeleton deformity were grouped as Controls.
Group II (Smokers): 25,Mild Smokers (< 5 Pack years) **(Group IIa)**; 25,Moderate Smokers (5-10 Pack years) **(Group IIb)**; 25 Chronic Smokers, (>10 Pack years) **(Group IIc).**

1 Pack year = 20 cigarettes/day for one year was considered. A detailed history of smoking was taken;-(1)Type of smoke inhaled, bidi/cigarette; (2) Time since smoking; (3) Number of bidis, cigarettes smoked per day.

The protocol of the study was approved by the ethics committee of our institute. Persons having asthma or chronic infections of lungs, having persistent cough treated recently for any respiratory illness were excluded. The subjects were drawn from amongst the staff and students of the Institute and residents of the city. Written consent was taken from the subjects after explaining the nature of the study and a written bio-data was obtained from them to group them into various groups. A detailed history and physical examination of each subject was carried out. All tests were carried out in the morning during the post absorptive phase. The ventilator tests were carried out with a computerized spirometer "Med-Spiror". It reads the amount of air and the rate of air that is breathed in and out over a specified period of time. Testing procedures were quite simple, non-invasive and harmless to the patient. The subjects were familiarized with the instrument and the technique used.

The readings were taken in standing position. Age, height (without shoes), body weight were recorded. Body Surface Area (BSA) was read from "Nomogram" [7]. The terminology and abbreviations used for different lung function tests carried out are as suggested by Cotes [8].

Each subject was given two trials and three test runs for each test and best of the three test readings was taken. Once the subjects were included in the study, none were subsequently rejected except when they were unable to give the desired co-operation in the experimental procedure.

The parameters studied from the records were; The Anthropometric variables – Age, Height, Weight, Body Surface Are (BSA) and The six Spirometric values- Forced Vital Capacity (FVC), Forced Expiratory Volume in 1sec (FEV_1), FEV_1/FVC%, Peak Expiratory Flow Rate (PEFR), Forced mid Expiratory Flow Rate (FEF_{25-75}) and Maximum Voluntary Ventilation (MVV).

Statistical analysis was carried for all the parameters using SPSF program version 10.0 (Microsoft Corp). 'P' value was determined. P>0.05 was considered as non-significant. Independent student t test was used for between groups comparison.

Result: The mean, standard deviation, t-value and p-value of six Spirometric values and

Anthropometric values have been shown in the observation tables.

Mean values of physical characteristics in non-smokers (Group I) were: - age (34.56+ 10.64yrs), height (168.68 +9.96 cms), weight (65.04 + 11.80 Kg) and Body Surface Area (BSA) (1.74 +.175sqm). Mean values in smokers (Group II) were age (37.16 +10.86), height (164.95 +11.72), weight (60.48 +12.35), BSA (1.66 +0.20) (Table 1).

TABLE 1: Anthropometric Values

	Non-Smoker (GROUP I) 25	Smoker (GROUPII) 75	p – value
Age (years)	34.56 +10.64	37.16 +10.86	N.S.
Height (cm)	168.68 +9.96	164.95 +11.72	N.S.
Weight (kg)	65.04 +11.80	60.48 +12.35	N.S.
BSA (mt2)	1.74 +0.17	0.66 +0.20	N.S.

Table 2 depicts the comparison of mean values of respiratory parameters with standard deviation, t-value and p-value in Group I and Group II.

TABLE 2 : SPIROMETERIC VALUES

	Non-smoker (GROUP I) 25	Smoker (GROUP II) 75	p-value
FVC (Litres)	3.22 +0.69	2.72 +0.67	<0.01
FEV1 (Litres)	2.98 +0.64	1.64 +0.76	<0.001
PEFR (L/sec)	7.48 +1.67	5.71 +2.17	<0.001
FEF25-75% (L/sec)	4.18 +1.42	3.60 +1.25	<0.001
FEV1/FVC	93.60 +6.31	86.83 +13.71	<0.05
MVV (L/min)	110.24 +46.61	82.96 +17.19	<0.01

Table 3 compares the mean, standard deviation, t-value and p-value of physical characteristics in Group I and Group IIa, II b and Group II c.

TABLE 3 : ANTHROPOMETRIC VALUES

	No.	Non-Smokers (GROUP I)	No	Smokers (GROUPII)	p – value
Age (years)	25	34.56 +10.64	25 25 25	31.36+8.31 37.56+7.24 42.56+13.01	N.S. N.S. <0.05
eight (cms)	25	168.68 +9.96	25 25 25	163.84 +13.38 163.84+9.44 163.52+11.58	N.S. N.S. N.S.
Weight (kg)	25	65.04 +11.80	25 25 25	63.08+13.02 59.28+10.29 59.08+13.12	N.S. N.S. N.S.
BSA (mt2)	25	1.74 +0.17	25 25 25	1.68 +0.22 1.64+0.16 1.66+0.19	N.S. <0.05 N.S.

The comparison of mean age, height, weight and BSA of non-smokers (Group I), mild smokers (Group IIa) were found to be statistically insignificant. The value of mean age in Group IIc in comparison with Group I was found to be statistically significant.

Table 4, depicts the mean values, standard deviation, t-value and p-value of six spirometric values for Group I and Group IIa, Group IIb and Group IIc.

TABLE 4: SPIROMETERIC VALUES

	No	Non-smokers (GROUP I)	No	Smokers (GROUP II)	p-value
FVC (Litres)	25	3.22 +0.69	25 25 25	2.93 +0.70 2.76+0.51 2.46+0.69	N.S. <0.01 <0.001
FEV1 (Litres)	25	2.98 +0.64	25 25 25	2.78+0.68 2.27+0.05 1.90+0.48	N.S. <0.01 <0.001
PEFR (L/sec)	25	7.48 +1.67	25 25 25	7.08+1.63 5.66+2.23 4.38+1.68	N.S. <0.01 <0.001
FEF25-75% (L/sec)	25	4.18 +1.42	25 25 25	3.95 +1.45 3.59+1.34 2.25+1.37	N.S. <0.05 <0.001
FEV1/FVC	25	93.60 +6.31	25 25 25	94.56 +6.91 87.00+12.21 78.98+15.64	N.S. <0.01 <0.001
MVV (L/min)	25	110.24 +46.61	25 25 25	109.68+36.39 77.60+27.67 67.60+29.33	N.S. <0.01 <0.001

Comparison of Group I and Group II revealed significant changes in values of FVC ($p<0.01$), FVC/FEV1 ($p<0.05$) and MVV ($p<0.01$) and highly significant changes in values of FEV1 ($p<0.001$), PEFR ($p<0.001$) and FEF25-75% ($p<0.001$).

Comparison of Group I and Group IIa revealed non-significant changes in most of the spirometric values. Comparison of Group I and Group IIb revealed significantly higher values of FVC ($p< 0.01$), FEV1 ($p<0.01$), PEFR ($P< 0.01$), FEF25-75% ($p<0.05$) and MVV ($p<0.01$).

Comparison of Group I and Group IIc revealed highly significantly value of FVC ($p< 0.001$), FEV1 ($p<0.001$), PEFR ($P< 0.001$), FEF25-75% ($p<0.001$) and MVV ($p<0.001$).

Discussion: Comparison between various groups of smokers, mild/moderate/chronic was undertaken to assess the lung functions tests using a computerized spirometer. Comparisons were also drawn between non-smokers and smokers in relation to the lung functions. The study observed that spirometry was an effective and easy method for detection of COPD in risk group population like smokers. In a recent study it was seen that, using the psychological tool of performing spirometry of smokers to show them the apparent premature ageing of their lungs, improves the likelihood of them quitting smoking, but the mechanism by which this intervention achieves its effect is unclear [5].

By analysis of the spirogram, determination of vital capacity, timed vital capacity, maximum ventilator volume and maximum mid expiratory flow rate it is usually possible to accurately detect and evaluate the underlying pathophysiology. Pulmonary function data in smokers indicate narrowing of smaller airways chiefly bronchioles which lead to slowly progressive COPD. It is inflammatory response of lungs to noxious gases or particles. Oxidative stress induced by smoking also induces COPD.

In the present study, the results of the lung function values were recorded and compared amongst the various groups. The results were also compared with the studies carried out previously.

The physical parameters of the present study showed insignificant results though body surface area value was significant ($p < 0.01$) amongst the non-smokers and smokers (Table 1). The above finding is in agreement with the findings of Rai and Nancy **[9].** There is also comparative reduction in weight of chronic smokers though statistically insignificant (Table 3), the findings are in agreement with Dhand and Malik **[10].** The decrease in the body weight in chronic smokers may be due to the fact that absorbed nicotine interferes with the appetite and food intake and it also alters the balance between body protein and body fat.

The results of FVC in smokers is on the lower side compared to non-smokers as shown in Table1, p-value is statistically significant. The results of the present study are lower than that reported by Nag and Dey **[11].** Non-smokers (3.676+0.440 and smokers 3.412± 0.55); $p < 0.01$. Intensity wise analysis (Table 2) shows the FVC in mild smokers is lower compared to non-smokers but the p-value is statistically insignificant. The FVC in moderate and chronic smokers is less than non-smokers and the p-value is statistically significant p, 0.001. The results of the present study are comparable to studies by Mosharraf-Hossain KM et al [6]. Sherril DL et a [12] and Chhabra SK et al **[13].** In these studies too it was apparent that smoking has negative impact on most measures of lung functions. The results indicated that the respiratory symptoms are generally associated with lower levels of FVC. But the results of the present study are not in agreement with those of Nancy NR and Rai US **[14].** in which there was non-significant change in the value of FVC between smokers (3.074+0.950) and non-smokers (3.055+0.825).

FEV_1 in smokers is on the lower side compared to non-smokers and p-value is statistically significant ($p<0.001$), as shown in Table 1. The above findings are comparable to the findings of Nancy and Rai and Nag and Dey **[11, 14].** Intensity wise analysis (Table3) reveals the value of FEV_1 in mild smokers is on the lower side in comparison to the control group and p-value so calculated to be insignificant. The value of FEV_1 of moderate and chronic smokers is on the lower side compared to non-smokers and the p-value came out to be statistically significant, $p<0.001$. The above findings are in agreement with the findings of Underdorben M et al [15], Sherril DL et al [12], Siatkowska H etal [16], Islam SS and Schottenfeld D [17]. These studies also reiterate that chronic smoking related changes in pulmonary function are reflected as accelerated decrease in FEV_1. The lung functions also showed a decline with increasing number of pack years. Smokers with decreased value of FEV_1 were more likely to develop such diseases as systemic hypertension, coronary diseases and COPD. Study by the Westerners showed higher value of FEV_1 as compared to the Indian studies, the reason being: difference in the nutrition status and the body build racial differences, geographical distribution and the economic status of the subjects. Another study showed that with each decline in FEV_1 of 100ml/year, lung cancer incidence density

increased 1.16 per 1000 person-years [17]. Rapidly declining lung functions in current smokers is predictive of increased risk of COPD and lung cancers and correlates with cumulative years of cigarette smoking.

The value of FEV_1/FVC in smokers is on the lower side as compared to the non-smokers and the p-value is statistically significant. The findings are in agreement with the findings of Nancy et al **[18].** During intensity wise analysis (Table 4) it was studied that the values of FEV_1/FVC in mild smokers is lower as compared to the control group and p value is statistically insignificant. The value of FEV_1/FVC in moderate and chronic smokers is much lower in comparison to non-smokers and the p-value is highly significant. The findings are comparable to the findings of some previous studies. In these studies also FEV_1/FVC showed significantly greater airway obstruction in smokers as compared to non-smokers. The negative impact of smoking was apparent in most measures but was most progressive in FEV_1/FVC ratio [12, 13, 16].

In the present study it was reported that the value of PEFR in smokers is lower than that in non-smokers as shown in Table 2 and p-value is statistically significant. The above study is not in agreement with an earlier study by Nag and Dey because the study undertook the comparison study between equal number of smokers and non-smokers and the age group was different (45-49 yrs) [11]. The present study comprises of 75 mild, moderate and chronic smokers. Intensity wise analysis showed that the values of PEFR in moderate and chronic smokers is lower than the control group and the p-value is statistically significant ($p < 0.001$). The results of the present study are comparable to earlier studies which reported decreasing trends in the values as we proceeded from non-smokers to heavy smokers [11, 18].

The value of $FEF_{25-75\%}$ in smokers is less in comparison to non-smokers as shown in Table1 and the p-value is statistically significant ($p < 0.001$). The above findings are in agreement with the findings of some earlier studies which showed significant reduction in the values of smokers in comparison to non-smokers [11, 18]. The results of the present study are higher compared to Dhand et al because the age group chosen by the study was 60 yrs (old men) and the subjects were all chronic smokers [10]. Intensity wise analysis shows that the value of $FEF_{25-75\%}$ in mild and moderate smokers is less as compared to non-smokers and the p- value is statistically significant ($p < 0.01$). The $FEF_{25-75\%}$ value in chronic smokers is much less and p-value is statistically highly significant ($p < 0.001$). The above findings are in agreement with the findings of Marq Minette and Walter and Nancy which show a decreasing trend as we proceed from non-smokers to chronic smoker [18, 19.] Few other studies also show significantly lower $FEF_{25-75\%}$ in cigarette smokers than in non-smokers [15, 20]. The data indicate acute and reversible effects of cigarette smoke exposures and no-smoking on mid to small size pulmonary airways in dose dependent manner. In fact decrease of $FEF_{25-75\%}$ values is the foremost change in people having COPD, even before changes in other respiratory parameters become apparent. The result of MVV in smokers is less than that in non-smokers and the p-value is statistically significant. The findings are by Nag and Dey also shows similar results [11]. Intensity wise analysis (Table 2) shows that the value of MVV in moderate and chronic smokers is less than that of non-smokers and the p-value is statistically significant ($p < 0.001$).

In the present study the lung capacities, volume and ventilator flow rates have shown significant difference between smokers and non-smokers. Pulmonary function data in smokers indicate narrowing of smaller airways chiefly bronchioles. The onset of COPD leads to a fixed narrowing of the airways and destruction of alveoli maintained in the peripheral part of the lungs. The negative impact of smoking was apparent in most measure, but was largest and most progressive in FEV_1 and FEV_1/FVC ratio. Non-smokers in all cases had better lung function values than smokers. Correlation between smoking habits and dyspnoea, morning cough, sputum production was confirmed. It was also established that lung functions decreased with increasing number of pack years. Rapidly declining lung functions in smokers is predictive of COPD and increased risk of lung cancers, systemic hypertension and coronary diseases and correlates with cumulative levels of exposure to cigarette

smoking. The study observed that spirometry was an effective and easy method for detection of COPD in risk group population like smokers and thus promotes smoking cessation efforts to reduce the burden of COPD in the community.

Conclusion: The observations serve to emphasize that rapidly declining lung functions in smokers is predictive of COPD and correlates with cumulative levels of exposure to cigarette smoking. There is limitation to rely solely on symptoms to confirm cases of COPD. The study observed that spirometry was an effective and easy method for detection of COPD in risk group populations like smokers and it thus promotes smoking cessation efforts to reduce the burden of COPD in the community.

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