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Effect of Smoking on PEFR: A Comparative Study among Smoker and Non-Smokers

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Abstract

Background: Smoking is the most important factor contributing to the development of COPD and is one of the health risks in modern time. The purpose of the present study was to determine the relationship between cigarette/biri smoking and PEFR 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. Equipment used computerized spirometer. *Results:* PEFR is decreased in cigarette smokers and magnitude of decline was higher in chronic smokers. *Conclusion:* The intensity of cigarette smoking [pack years] emerged as the main variable to influence airway obstruction in smokers that caused reduction in PEFR.

Keywords: Smoking; PEFR.

Smoking is apublic health problem and amajor cause of many preventable diseases premature deaths all over the world. It is now well established that cigarette smoking for only a few years causes early changes in peripheral airways of the lung. The primary objective of the study was to investigate whether PEFR differs between cigarette smokers compared to non smokers.

Cigarette smoking has been identified to be the most important determinant of ventilatory impairment.

Although it is known that smoking causes respiratory dysfunction, but very few works have been actually done on the dose and time dependent effect of smoking on lungs.

Objective is to know whether the chronic heavy smoking start deteriorating the pulmonary function test as early as 5years of smoking habit. Only parameter selected is PEFR as it can be monitored by smoker himself. PEFR asses the severity and variation of disease and evaluates the effects of treatment.

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 vicious circle.

Tobacco is dried leaf of nicotianna tabacum, 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 and this alkaloid is so poisonous that of again given intra venously can kill a dog in three minutes. Cigarette tobacco contains, on an average 1.55 nicotine and thus an average cigarette of one gram may yield as much as quarter grain to even half grain of nicotine. When one smokes, heat liberates nicotine in varying degree into smoke, some of the alkaloid is burnt but appreciable quantities gain access to respiratory tract. Depending upon moisture of the tobacco filtration, heat, rapidity of smoking the depth of inhalation.

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

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other specific components of total particulate matter TPM like steam volatile phenol, HCN 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 and COPD.

Cigarette smoking is the most important factor contributing to the development of COPD. It is now well established that cigarette smoking for only a few years causes early changes in the peripheral airways of lung [4]. The single best thing a smoker can do to improve their lung functions and live a longer life is to stop smoking. Or to monitor the severity of disease by peak flow meter. And can decrease the smoking and see the response to treatment.the single best thing asmoker can do to improve their lung functions and live alonger 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 ayear later [5]. The present study has been undertaken to compare between smokers and non smokers the PEFR using a medspiror acomputerized 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]. The single best thing asmoker can do to improve their lung function and live a longer life is to stop smoking and to monitor the severity of disease by measuring PEFR [5].

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 lla); 25, Moderate Smokers (5-10 pack years) (Group llb); 25 chronic smokers,(.10 pack years) (Group llc).

1 Pack years =20 cigarettes/day for one years was considered. A detailed history of smoking was taken; (1) Type of smoking 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. Person having asthma or chromic of our infection 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 residence of the city. Written consent was taken from 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 subjects was carried out. All testes 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 the air that is breathed in and out over a specified period of time. Testing procedures were quite simple, non-invasive and harmless to the patent. The subjects were familiarized with instrument and technique used.

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

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

The parameters studies from the records were; The Anthropometric variable - Age, Height, Weight, Body surface Are (BSA) peak Expiratory Flow Rate (PEFR)

Statistical analysis was carried 'P' value was determined. $p > 0.05$ considered as non-significance. Independent student test was used for between groups comparison.

Result

The mean, standard deviation, t-value and p-value of PEFR 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 + 10.86), height (164.95 + 11.72), weight (60.48 + 12.35), BSA (1.66 + 0.20) Table 1).

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 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.

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 statically significant.

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 PEFR (P<0.01).

Comparison of Group I and Group IIc revealed PEFR (P<0.001).

Table 1: Anthropometric values

	Non-Smoker (Group I) 25	Smoker (Group II) 75	p-value
Age (years)	34.56 + 10.64	37.16 + 1.86	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: Spirometric values

	Non-Smoker (Group I) 25	Smoker (Group II) 75	p-value
PEFR	7.48 + 1.67	5.71 + 2.71	<0.001

Table 3: Anthropometric values

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

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

	No.	Non-Smokers (GROUP I)	No	Smokers (GROUP II)	p-value
PEFR	25	7.48 + 1.67	25	7.08 + 1.63	N.S.
			25	5.66 + 2.23	<0.05
			25	4.38 + 1.68	<0.001

Comparison of Group I and Group II revealed in PEFR (p<0.001).

Discussion

Comparison between various groups of smokers, mild/ moderate / chronic was undertaken to assess the lung function tests using a computerized spirometer. Comparisons were also drawn between non smokers and smokers in relation to lung functions. The study observed that spirometry was an effective and easy method for detection of copd in risk group population like smokers.

Pulmonary function data in smokers indicate narrowing of smaller airways chiefly bronchioles which lead to slowly 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 lung function 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 Dand 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.

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 pvalue 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 [11]. The present study comprises of 75 mild, moderate and chronic smokers. Intensity wise analysis showed that the values 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 valuesas we proceeded from non smokers to heavy smokers [11,18].

One possible reason for decrease in PEFR could be inflammation which is common and constant pathological finding in cigarette smokers [22].

Inflammation either directly or by increasing smooth muscle tones indirectly may cause airway fibroses. All these changes provide wall thickness leading to air way narrowing and flow limitation. In addition inflammation causes destruction of the alveolar walls attached to the airway contributing further to airflow limitation by deforming and narrowing the airway lumen [21].

Overall our findings are consistent with others that the intensity of cigarette smoking [pack years] emerged as the main variable to INFLUENCE air way obstruction in smokers.

Constituents of tobacco smoke cause damage throughout the respiratory tree from the main airways [bronchi] to the peripheral airways [bronchioles] right down to the terminal alveoli [air pockets] as well as to immune system.

Loss of cilia and mucus glands hypertrophy occur in the upper airways, inflammation, epithelial changes fibross secretory congestion occur in the peripheral airways and alveoli are destroyed with loss of gas exchange area and airway flexibility.

Conclusion

Study concludes smokers can be considered one of major risk factors for COPD and lungs diseases. Which can be prevented by avoiding smoking habits and secondly chronic smokers may benefits from regular peak flow monitoring. PEFR is useful parameter to monitor airway obstruction, assess the severity and variation of diseases of also to evaluate the effects of treatment.

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