Original Article

Effect of 2-5 Years of Tobacco Smoking on Peak Expiratory Flow Rate: A Comparative Study among Medical Students Smokers and Non-Smokers in KIMS, Hubballi

Bajentri.A.L[1], Baljoshi.V.S[2]

[1]Associate Professor, [2]Professor, Department of Physiology, KIMS, Hubballi

ABSTRACT

Introduction : Tobacco smoking in India has been increasing alarmingly.

Aims and Objectives: The present work is undertaken to study the effect of 2-5 years of tobacco smoking on ventilatory functions.

Material and Methods: The study group consisted of 25 male, young, healthy medical students, free from cardiopulmonary diseases and with history of smoking of 2-5 years duration, on an average of 10 cigarettes per day. The control group consisted of 25 males age matched healthy individuals who had never smoked tobacco. Peak expiratory flow rate is measured using Wright's peak flow meter.

Result : PEFR is decreased in tobacco smoking students compared to non-smoking students.

Conclusion: This study shows that 2-5 years of tobacco smoking leads to a definite tendency to narrowing of small airways.

Key words: Peak expiratory flow rate, smokers, medical students, Wright's peak flow meter.

INTRODUCTION:

Tobacco use is the leading single preventable cause of deaths worldwide. Each year an estimated seven million deaths are attributed to the use of tobacco[1]. PEFR is a useful parameter to monitor airway obstruction, assess its severity and variation and evaluate the effects of treatment.[2] Several studies have reported that PEFR was significantly lower in smokers than in non-smokers.[3,4] It is difficult to establish national norms in India for healthy men and women as the lung function varies with socio-economic, geographic, climatic, environmental and nutritional conditions. Various authors have used regression analysis to explore the relationship between PEFR and age, height and weight.[5] Obstruction to airflow that develops in 15-20% of heavy smokers is thought to be due to abnormalities in airways with less than 2mm internal diameter.[6] It is estimated that around 13% of cardiovascular disease deaths are due to tobacco smoking.[7]

With the above background, it is clear that smoking is hazardous for health. The first impact of smoking is on lungs where it starts decreasing the lung capacity of the smokers. Hence if we can identify this decrease in lung capacity, at an early stage among smokers, we can try to prevent further effects of smoking on all body systems. And as mentioned above, this can be simply and easily measured by PEFR.

Address for correspondence:
A.L.Bajentri, Associate Professor of Physiology, KIMS, Hubballi. Email- ashokbajentri@gmail.com, Phone no.9448029507

http://dx.doi.org/10.31975/NJBMS.2018.9204

MATERIAL AND METHODS:

The present study was conducted in the Department of physiology, Karnataka institute of medical sciences Hubballi, on two groups (study and control) comparable in age and sex.

All the subjects who participated in the study gave informed consent after the procedure of the non-invasive investigation to be carried out was explained to them. They were subjected to detailed clinical examination to rule out the presence of any underlying disease.

CRITERIA FOR THE STUDY GROUPS:

INCLUSION CRITERIA:-

1) Age 20-25 years
2) For cases, minimum duration of smoking of 2 years
3) Willing to participate in the study

EXCLUSION CRITERIA:-

1) Presence of acute or chronic respiratory illness, diabetes mellitus, hypertension or any other systemic illness
2) Passive smokers

The control (non-smoker) group comprised of age and sex matched healthy individuals who had never smoked tobacco even once in their lifetime.

The details of the anthropometric data of the two groups are shown in Table 1. Subjects were made familiar to the working of the instrument and the test protocol maneuver was explained in brief.

PEFR is a simple method of measuring airway obstruction and it will detect moderate or severe disease. The simplicity of the method is its main advantage. It is measured using a Wright peak flow meter. The needle was always reset to zero before PEFR was measured. The test was conducted while the subjects were sitting comfortably in a chair and had rested sufficiently for a period of 15 minutes during the same time of the day (9-11am) under similar atmospheric conditions. Statistical analysis was done by student-t test.

RESULTS:

The mean age of smokers and non-smokers were comparable and there was no statistically significant difference in the mean values of them. This is because they were matched for age and BMI. (Table-1)

Table 1. Anthropometric and vital data of the study group and controls (Mean+/- SD)

Group	Age (yrs)	Height (cm)	Weight (kg)	BMI Kg/m^2	Body Surface Area (m^2)	Pulse Rate (/min)	Respiratory rate (/min)	Systolic BP Diastolic BP (mmHg)
Controls (n=25)	22.20 +/- 1.75	164.23 +/-4.33	60.70 +/-3.34	20.72 +/-1.40	1.68 +/-0.06	78.06 +/-3.76	18.24 +/-1.47	117.82 +/-3.85 78.40 +/-3.88
Smokers (n=25)	22.63 +/- 1.73	163.90 +/-2.82	62.74 +/-3.20	21.86 +/-1.45	1.67 +/-0.05	77.06 +/-3.62	18.40 +/-1.63	118.04 +/-3.48 80.84 +/-5.93

Table-2: Showing comparison of PEFR in Controls and the study groups-

Parameters	Control group (C) (mean +/- SD)	Study group (S) (mean +/- SD)
PEFR (L/min)	370.6+/-90.65	316.7+/-92.35

Following abbreviations have been used throughout the study.
C- Control group, S-Study group

It can be observed from table 2 that the mean PEFR in smokers was significantly low compared to non-smokers.

Table 3. Association between smoking and PEFR

	Abnormal PEFR	Normal PEFR	Total
Smokers	21(84%)	04(16%)	25(100%)
Non-smokers	15(60%)	10(40%)	25(100%)
Total	36(72%)	14(28%)	50(100%)

It can be seen from table 3 that smoking is strongly associated with the abnormal PEFR. The percentage of abnormal PEFR was 84% among smokers as compared to 60% among on smokers. The smokers were found to be 3.3 times more at risk of having abnormal PEFR than non smokers. This association was statistically significant ($p<0.05$).

DISCUSSION:

Peak expiratory flow rate (PEFR) value can be used as an indicator of a person's health and it's a clinical tool in diagnosis, management and follow up for respiratory diseases.[8] Peak flow measurements can be used to measure the strength of muscles of respiration and thus can be used to monitor respiratory impairment. Diurnal variation of PEFR is used to diagnose and in the management of bronchial asthma.[9]

PEFR is measured by peak expiratory flow meter which is a simple and relatively cheap device. It has great diagnostic and prognostic value in patients with hyperactive airway disease.[10]

Many factors can affect the reading of PEFR .The age, sex, and height are important variables upon which PEFR depends.[11] One possible reason for the decrease in PEFR is inflammation which is a common and constant pathological finding in cigarette smokers.[12]

Earlier studies have reported that airway flow limitation occurs due to bronchial constriction caused by mediators of inflammation.[13] Inflammation either directly or by increasing smooth muscle tone, indirectly, may cause airway fibrosis. All these changes promote wall thickness leading to airway narrowing and flow limitation.[14]

The prevalence of abnormal PEFR was 84% among tobacco smokers as compared to 60% among non tobacco smokers .The tobacco smokers were found to be 3.33 times more at risk of having abnormal PEFR than non tobacco smokers .This association was statistically significant ($p<0.05$)

Ferris and Cotes showed a decrease in diffusing capacity in cigarette smokers and this was probably related to a lower pulmonary capillary blood volume in smokers compared to non smokers.[14] Chatterji S et al found out that value of MVV and PEFR is significantly lower in smokers than non smokers.[15]

Sawant GV et al showed that PEFR is significantly reduced in smokers compared to non smokers and abnormal PEFR was found significantly more in smokers as compared to non smokers.[8]

CONCLUSION:

The smokers were found to have reduced lung capacity compared to non-smokers. Smoking is known to reduce the lung capacity. This study has proved that smoking adversely affects the normal functioning of lungs which leads to increased morbidity and mortality among smokers. Thus smoking was found to be directly related to reduced lung capacity measured in terms of PEFR.

REFERENCES:

1. WHO report on the global tobacco epidemic, 2017: monitoring tobacco use and prevention policies. Geneva: World Health Organization; 2017. License: CC BY-NC-SA 3.0 IGO.

2. Quanjer P, Lebowitz MD, Gregg I, Miller MR, Pedersen OF. Peak expiratory flow: conclusions and recommendations of a Working Party of the European Respiratory Society. European respiratory journal. 1997 Feb 1;10(24):2s.

3. Karia RM. Comparative study of peak expiratory flow rate and maximum voluntary ventilation between smokers and non smokers. National Journal of Medical Research. 2012;2(2):191-3.

4. Vaidya P, Kashyap S, Sharma A, Gupta D, Mohapatra PR. Respiratory symptoms and pulmonary function tests in school teachers of Shimla. Lung India. 2007 Jan 1;24(1):6.

5. Mathur N, Rastogi SK, Husain T, Gupta BN. Lung function norms in healthy working women. Indian journal of physiology and pharmacology. 1998 Apr;42:245-51.

6. Hogg JC ,Macklem PT , Thurlbeck MW.Site and nature of airway obstruction in chronic obstructive lung disease . New England J Med. 1968;278(25): 1355-60.

7. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez AD. The burden of disease and injury in Australia 2003; PHE 82.

8. Gokuldas V Sawanth, saurabh P Kubde, Prashant R Kokiwar. Effect of smoking on PEFR;A comparative study among smokers and non smokers in an urban sum of community of Hyderabad, India; International Journal of Community Medicine and Public Health. 2016;3:46-250.

9. Katz DN. The mini Wright peak flow meter for evaluating airway obstruction in a family practice. J FamPract 1983;17: 51-7.

10. British Thoracic Association. Death from asthma in two regions of England. British Med J. 1983; 285: 1251-5.

11. British Thoracic Society. Research unit of the Royal college of Physicians of London.Guidelines for the management of asthma in adults with chronic persistent asthma. British med J.1982 Oct 30; 285(6350): 1251–1255.

12. Vanhoutte PM. Airway epithelium and bronchialreactivity.Can J PhysiolPharmacol. 1987;65: 448-50.

13. Berend N. Lobar distribution of bronchiolar inflammation in emphysema. American Review of Respiratory Disease. 1981 Sep;124(3):218-20.

14. Van Ganse WF, Ferris Jr BG, Cotes JE. Cigarette smoking and pulmonary diffusing capacity (transfer factor). American Review of Respiratory Disease. 1972 Jan;105(1):30-41.

15. Chatterjee S Nag SK,Dey SK. Spirometric standards for non smokers and smokers of India (eastern region).Japanese J Physiol. 1988;38:283-93.

Received on 01/12/2018 **Revised** on 10/12/2018 **Accepted** on 17/12/2018