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International Journal of Current Research
Vol. 10, Issue, 02, pp.65116-65119, February, 2018

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

RESEARCH ARTICLE

EFFECT OF CHEMICAL PARTICLES OF CIGARETTE SMOKE ON VENTILATORY FUNCTION TEST

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ARTICLE INFO

Article History:
Received 19th November, 2017
Received in revised form
23rd December, 2017
Accepted 20th January, 2018
Published online 18th February, 2018

Key words:
Free radical, Smokers,
Ventilatory Function Test.

ABSTRACT

The objective of this study was to assess the effect of chemical particle present in cigarette smoke on ventilatory functions on smokers to analyse the extent of impairment in the airways. Parameters recorded were PEFR, FEV1, FVC, $FEF_{25-75\%}$ and FEV1/FVC%. For each participant chest X-ray was also taken. We have done comparison of ventilatory functions in non-smokers and smokers. It was observed that the values PEFR, FEV1, FVC, $FEF_{25-75\%}$ and FEV1/FVC% significantly decreased in smokers (p value<0.001) when compared to non-smokers. Smokers are at high risk of developing obstructive lung disease. Tobacco smoke contains abundant reactive oxygen "Free radical" which deplete antioxidant mechanism thereby inciting tissue damage. Activated neutrophils also added to the pool of reactive oxygen species in the alveoli. The free radical causes inflammation which results in narrowing of airways.

Citation: Vishal Goel and Ashish Arvind, 2018. "Effect of chemical particles of cigarette smoke on ventilatory function test", *International Journal of Current Research*, 10, (02), 65116-65119.

INTRODUCTION

Tobacco smoking is widely prevalent all over the world and it continues to rise in developing countries. It is expected that the developing world will have 7million deaths annually from tobacco use by 2030 (Abdulla, 2004). Exposure to air pollutants is known to be harmful to health, in general, and to the lungs in particular (Gupta *et al*., 2011). Cigarette smoke contains two very different populations of free radicals, one in tar and one in the gas phase. The tar phase contains several relatively stable free radicals; such as a quinine/ hyroquinonecomplex (Q/QH2) held in tarry matrix. Q/QH2 polymer is an active redox system that is capable of reducing molecular oxygen to produce superoxide, eventually leading to hydrogen peroxide and hydroxyl radicals. The gas phase of cigarette smoke contains small oxygen and carbon- centered radicals that are much more reactive than are the tar phase radicals. These gas- phase radicals are produced in a steady state by oxidation of NO to NO_2, Which then reacts with reactive species in smoke such as isoprene. These radicals and the metastable products derived from these radical reactions may be responsible for the inactivation of a proteinase inhibitor by fresh smoke. Nitrogen dioxide (NO_2), a reactive free radical, emitted from cigarette smoke that has been shown experimentally to cause epithelial damage and to induce inflammation in the airways (Daniel *et al*., 1985).

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Inhaled cigarette smoke contains thousands of particles, many of which are irritants that cause bronchial inflammation and destruction of ciliary activity. The excess mucus that accumulates because of decreased ciliary activity increases patient vulnerability to secondary bronchial infections, further compromising the already inflamed bronchial mucosa (Jardin, 2011). Pulmonary function tests help in determining the presence or absence of obstructive, restrictive or mixed airway diseases for effective therapeutic intervention (Chabra, 1999). Pulmonary function tests and chest X-rays are common diagnostic tools for diagnosing respiratory diseases. The objective of this study was to compare ventilatory functions in nonsmokers and smokers to analyze the extent of impairment in the airways.

MATERIALS AND METHODS

This study was conducted in Department of Physiology Maharishi MarkandeshwerInstitute of Medical Sciences and Research Mullana, Ambala in collaboration with department Pulmonary Medicine and Radiology. The pulmonary function tests were conducted on the spiro-exel Medicaid system in 50 subjects of which 25 were taken as the control group and other 25 as the study group. The control group subjects were non-smoker males randomly selected from general population. The study group included smoker males randomly selected from general population.

Inclusion criteria

Non-smoker: those who did not smoke at all.

Smokers: those who smoked any tobacco products daily for more than 10 years.

Exclusion criteria

Patients suffering from tuberculosis, pneumonia, pulmonary effusion, lung carcinoma, ex-smoker or post smoker were excluded from the study. After taking full informed written consent the procedure was explained to the subjects. Theywereasked to sit comfortably and asked to breathe in and out to familiarize to the equipment. They were then asked to inhale to their maximum capacity and forcefully blow out into sensor (nose clipped) as hard as possible. This procedure was repeated and the best of three readings were considered for analysis.

Parameter recorded were

- Peak expiratory flow rate (PEFR)
- Forced expiratory volume in 1^{ST} second
- Forced vital capacity
- FEV1/FVC%
- Forced mid expiratory flow ($FEF_{25-75\%}$)

For each participant chest X-ray (PA- view) 35*35cm were also taken.

RESULTS

The anthropometric measurement including age, weight and height of control group and study groups were comparable.

Table 1. Smoking index among smokers

Parameter	Smoker
Mean duration of smoking (years)	13.8 ± 10.3
Average frequency of smoking per day	14 ± 6.3
Smoking index (frequency×duration)	182

Smoking index is equal to multiplication of average number of cigarettes/ bidis smoked per day and duration (in years) of tobacco smoking.

Table 2. Pulmonary function test findings

Parameter	Control	Smokers
PEFR (L/s)	9.549 ± 1.1084	7.362 ± 2.1072
FEV1 (L/s)	3.527 ± 0.2399	2.751 ± 0.7303
FVC (L/s)	3.863 ± 0.252	3.165± 0.693
FEF(25-75%)(L/S)	5.648±0.7342	4.207±1.621
FEV1/FVC (%)	91.352 ± 3.5515	85.873 ± 7.5247

X=mean, SD= Standard deviation
Comparison of ventilatory function test between Smoker and Non Smoker

Table 3. Peak expiratory flow rate (PEFR)

Parameter	Control	Smokers
PEFR (L/s)	9.549 ± 1.1084	7.362 □ 2.1072

PEFR in control group is significantly higher when compared with smoker group (p< 0.0001).

Table 4. Forced expiratory volume in first second (FEV1)

Parameter	Control	Smokers
FEF1(L)	3.527 ± 0.2399	2.751 ± 0.7303

X=Mean Value, SD= Standard deviation

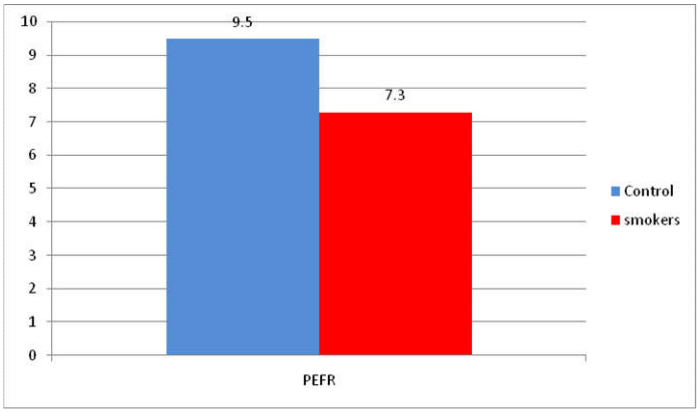


Figure 1.

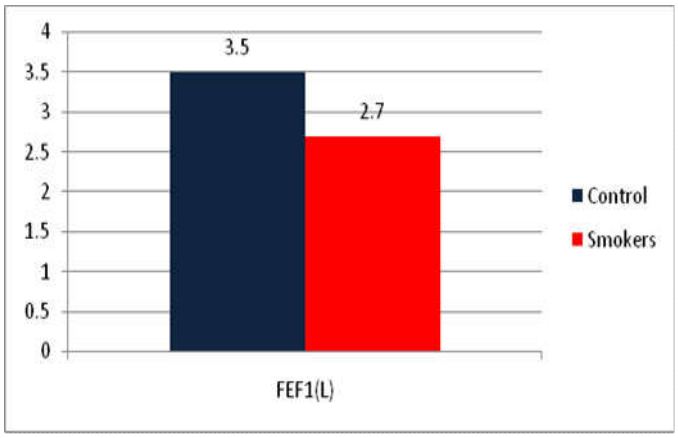
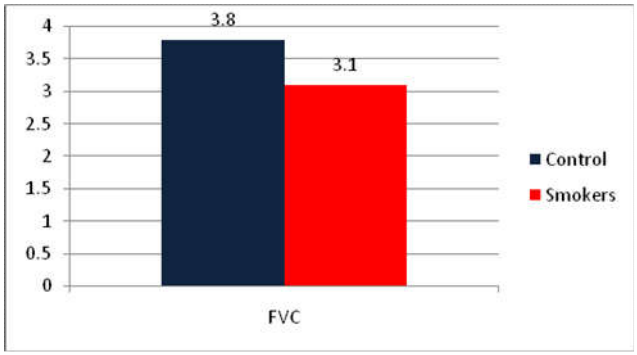


Figure 2

FEV1 in control group is significantly higher as compared to smoker group (p< 0.0001).

Table 5. Forced Vital Capacity (FVC)

Parameter	Control	Smokers
FVC (L)	3.863 ± 0.252	3.165 ± 0.693



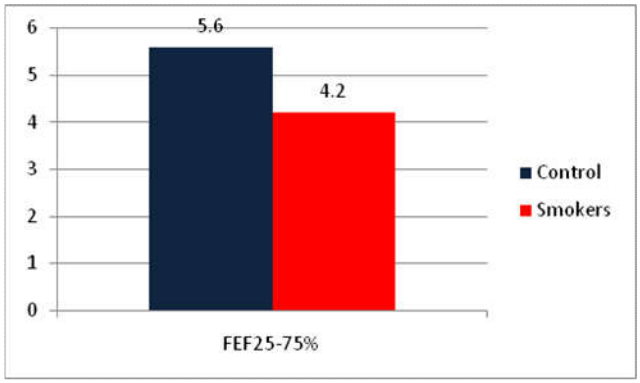
X= Mean value, SD= Standard deviation

Figure 3.

In FVC no significant difference of values is observed when value of control group and smoker group were compared.

Table 6. FEF25-75%

Parameter	Control	Smokers
FEF25-75% (L/s)	5.648±0.7342	4.207±1.621



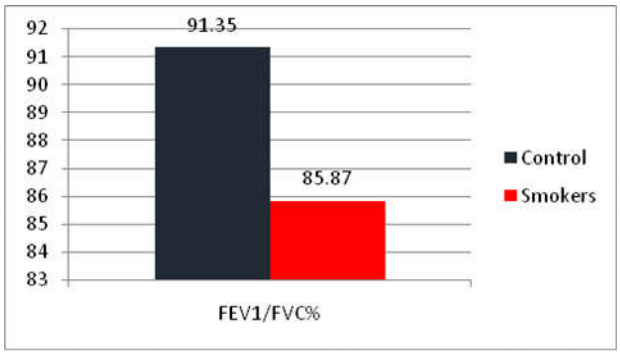
X=mean value, SD= Standard deviation

Figure 4. FEF25-75% of control group is significantly higher than smoker group (p< 0.0001)

FEF25-75% of control group is significantly higher than smoker group (p< 0.0001).

Table 7. FEV1/FVC%

Parameter	Control	Smokers
FEV1/FVC%	91.352±3.5515	85.873±7.5247



FEV1/FVC%

Figure 5.

FEV1/FVC% in control group is significantly higher as compared to smoker group (p<0.0001).

X-Ray Findings
X-ray shows hilar lymph node enlargement and increase in bronchovascular marking in study group when compare to control.

DISCUSSION

In this study all the parameter of pulmonary function like PEFR, FEV1, FEV1/FVC, $FEF_{25-75\%}$ are significantly decreased in smokers when compared with control (nonsmokers) by applying unpaired t test of significance. Analysis of relation between smoking status and ventilatory functions revealed the significant decrease in PEFR, FEV1, $FEF_{25-75\%}$. So it is suggested that smoking may play a role in narrowing of airways .But there is not significant decrease in FVC. Fall in FEV1, PEFR and $FEF_{25-75\%}$ indicates obstructive lung changes. The results of this study for the Pulmonary functions impairment in smokers is similar to the observation reported by Anglo *et al*. (1973), Malo *et al*. (1975), Gupta *et al* (1977), Mahajan *et al*. (1983). As they also observed fall in all parameter (PEFR, FEV1, FEV1/FVC% and FEF25-75%) but there is no significant difference in FVC in smoker.

Normally, the lung contains a healthy complement of antioxidants (superoxide dismutase, glutathione) that keep the oxidative damage to minimum. Tobacco smoke contains abundant reactive oxygen "Free radical" which depletesantioxidant mechanism thereby inciting tissue damage. Activated neutrophils also added to the pool of reactive oxygen species in the alveoli. Asecondary consequence of oxidative injury is inactivation of native antiproteases, resulting in "Functional" ATT (α_1 –antitrypsin) deficiency even in patients without enzyme deficiency (Husain *et al*., 2010). Lung elastin, a major structural protein that supports the alveolar wall of the lungs, is normally protected by ATT, a protein that opposes the degradative threat of neutrophil elastase, a protein contained within the neutrophils that is released when neutrophils are attracted to the lung during inflammation and infection. Under normal circumstances of an adequate amount of ATT, neutrophil elastase is counteracted so as to digest lung elastin. However, in the face of severe deficiency of ATT, neutrophil elastase may go unchecked, causing breakdown of elastin resulting in dissolution of alveolar walls.

It is likely that impaction of smoke particles, predominantly at the bifurcation of respiratory bronchioles, results in the influx of neutrophils and macrophages, both of which secrete proteases. Tissue breakdown is enhanced as a consequence of inactivation of protective antiproteases by reactive oxygen species in cigarette smoke. This scheme also explains the additive influence of smoking and ATT deficiency in inducing serious obstructive airway diseases (Kacmarek *et al*., 2009).

Conclusion

Smokers are at high risk of developing obstructive airway diseases. It is due to the effect of free radical produced by cigarette smoke. The free radical causes inflammation which results in narrowing of airways.

Abbreviations

Where
PEFR= Peak expiratory flow rate i.e. the maximum velocity of flow in liters per minute with which air can be forced out of the lungs.

FEV1= Forced expiratory volume in one second, that is volume of air exhaled in first second during forced vital capacity effort.

FVC= Forced vital capacity, that is the maximum amount of air that can be exhaled following a maximal inspiratory effort.
FEV1/FVC ratio

(FEF25-75%): Forced mid expiratory flow- This is the maximum flow achieved during middle third of total expired volume.

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