EFFECT OF ISOPRENALINE AND CIGARETTE SMOKING ON AIRWAYS

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(Received on April 8, 1982)

Summary: After two hours of abstinance from smoking pulmonary functions were assessed in twenty-seven asymptomatic male smokers by spirometry and Wright Peak Flow Meter, recording FVC, FEV_1, FEV_1%, FEF_{25-75}%, and FEF_{75-85}% of FVC, MVV and PEFR. These tests were repeated immediately after smoking and again after one hr. Only PEFR decreased significanfly ($P<0.01$) and returned to nearly basal level after one hr.

Sixteen of these smokers were subjected to further study. Isoprenaline inhalation in these smokers caused a significant improvement in most of the flow rates. Smoking of cigarettes at this stage decreased flow rates. After one hr the effect of smoking was over and most of the parameters were between basal and post-isoprenaline levels.

Smoking affects the flow rates more than other parameters. An improvement in FEV_1, FEV_1%, FEF_{25-75}% and FEF_{75-85}% of FVC, after bronchodilator inhalation suggests the presence of some reversible bronchoconsitriction specially of smaller airways basically present in chronic smokers.

Key words : airway response isoprenaline and cigarette smoking

INTRODUCTION

Adverse effect of smoking for prolonged period on lung functions are well known and the relationship between smoking and chronic bronchitis and emphysema is well established. Immediate effects of cigarette smoking on pulmonary functions have been studied by Nadel and Comroe (7), Da silva and Hamosh (2), Zuskin *et al.* (11), Da silva and Hamosh (3) using sophisticated methods, available only in a few well equipped centres. The present study is undertaken to study the acute effects of smoking on airways and an attempt has been made to study the interaction between the effects of isoprenaline (a potent bronchodilator) and cigarette smoking on airways as measured by spirometer and Wright Peak Flow Meter.

MATERIAL AND METHODS

Twentyseven normal, healthy smokers volunteered for the study. They were free from any cardiopulmonary disease. The age and anthropometric parameters were recorded using standard procedure. Detailed history of smoking was recorded. They were directed to abstain from smoking for at least 2 hr before reporting to the laboratory. The subjects were trained well before the study. After determining the basal pulmonary functions each subject was asked to smoke 2 cigarettes without filter of same make, one after the other under the direct supervision of the observer so that he actually inhaled the smoke. Each subject inhaled approximately 30 puffs and took about 10 min for smoking. Peak Expiratory flow rate (PEFR) and spirometric recordings were made immediately and one hr after smoking.

On an other occasion 16 out of these 27 smokers were given 2 puffs of pressurised metered isoprenaline aerosol (after the basal recordings) at an interval of 5 min and pulmonary functions were recorded 15 min after the second inhalation. One puff of pressurised metered isoprenaline aerosol delivers 400 μgm of drug. Then each subject smoked two cigarettes as before. The pulmonary functions were recorded immediately and one hr after smoking.

PEFR was recorded using Wright Peak Flow Meter. Spirometery was done using 9 litre water sealed spirometer for measuring forced vital capacity (FCV), forced expiratory volume in one second (FEV_1), forced expiratory volume in one sec expressed as a percentage of FVC (FEV_1%), forced expiratory flow between 25% and 75% of the FVC (FEF_{25-75}%), forced expiratory flow between 75% and 85% of the FVC (FEF_{75-85}%) and maximum ventilatory volume (MVV). The spirometeric volumes were converted to B.T.P.S. special care was taken to see that none of the subjects smoked again until all the tests were over.

RESULTS

The mean age, height and weight of 27 subjects were 27.7±4.54 (Mean±SD) years, 167.2±6.1 *cms* and 57.5±18.9 *kg* respectively. The duration of smoking was 10.0±5.8 years and quantity of smoking was 15.0±2.8 pack years. The expression pack years of smoking was calculated by multiplying the number of packs consumed daily with duration of smoking habit in years. In this study ten bidis were considered to represent one pack (5).

Acute effects of cigarette smoking in 27 healthy smokers (Pre, post smoking and after 1 hr values) on different parameters are tabulated in Table I. The effect of isoprenaline and then smoking on pulmonary functions, in 16 smokers is shown in Table II. The paired 't' test has been applied to study the statistical significance.

TABLE I : Effect of cigarette smoking on pulmonary functions.

	Basal	*Immediately after smoking*	*After one hr. of smoking*
	Mean± SD	Mean± SD	Mean±SD
FVC (Lt)	3.76±0.46	3.77±0.48	3.83±0.42
FEV_1 (Lt)	3.07±0.34	3.06±1.44	3.15±0.39
FEV_1%	81.80±9.45	81.20±10.96	82.20±9.76
PEFR (Lt/Mt)	500.00±67.65	480.70±73.11**	494.80±61.69
MVV (Lt/Mt)	125.90±22.44	126.00±23.20	127.60±23.44
FEF_{25-75}%(Lt/Sec)	3.81±0.91	3.69±0.97	3.97±1.37
FEF_{75-85}%(Lt/Sec)	1.54±0.69	1.52±0.75	1.66±0.91

Statistical analysis by 'paired t test' : ** $P<.01$

TABLE II : Effects of cigarette smoking on pulmonary functions after isoprenaline inhalation.

	Basal	*Effect of isoprenaline*	*Effect of smoking*	
			Immediate	*after 1 hr.*
	Colm I	*Colm II*	*Colm III*	*Colm IV*
	Mean± SD	Mean± SD	Mean±D	Mean±SD
FVC (Lt)	3.72±0.41	3.83±0.47	3.75±0.44	3.83± 0.42
FEV_1 (Lt)	2.95±0.37	3.2±0.39***	2.97±0.33*	3.01± 0.49
FEV_1%	79.35±8.99	83.7±7.24**	79.5±8.92*	78.7±10.48
PEFR (Lt/Mt)	499.3±67.07	503.3±72.39	499.3±70.32	497.6±71.77
MVV (Lt/Mt)	123.0±19.23	126.5±19.95	120.7±22.87*	124.9±22.30
FEF_{25-75}% (Lt/Sec	3.12±0.81	3.76± 0.94*	3.66± 0.95	3.45± 0.92
FEF_{75-85}% (Lt/Sec)	1.35±0.52	1.68± 0.89*	1.47± 0.73	1.45± 0.63

Statistical analysis by 'paired t test' : Effect of isoprenaline (Colm II) on pulmonary functions from basal (Colm I). Immediate effect of smoking (Colm III) from improved pulmonary functions after isoprenaline inhalation (Colm II).

*$P<0.05$ **$P<0.01$ ***$P<0.001$

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DISCUSSION

Cigarette smoking is the most important factor contributing to the development of chronic obstructive pulmonary disease. Significant pulmonary function abnormalities may be present even in teenage smokers (6).

Walter (9) observed a fall in FVC, FEV_1 and PEFR immediately after smoking. We observed a significant fall in PEFR only, which is in agreement with the observations of Gupta and Tandon (4). However, all the parameters were within one SD range one hr after smoking and varied statistically insignificantly from the basal. Thus probably the immediate effect of smoking was bronchoconstriction of larger air passages and was over within one hr as it was PEFR which got significantly lowered and there was no significant effect on $FEF_{75-85}\%$ of FVC.

A statistically significant, improvement in FEV_1, $FEV_1\%$, $FEF_{25-75}\%$, and $FEF_{75-85}\%$ was observed after isoprenaline inhalation in smokers which is in agreement with the observations of Watenabe *et al.* (10). Neither PEFR nor spirometric FVC and MVV showed significant improvement. These observations point towards the presence of reversible bronchoconstriction basically present in smokers, specially in small and medium sized bronchioles.

On smoking two cigarettes after isoprenaline inhalation, there was a fall in all the spirometric parameters from post isoprenaline level (improved levels) but mostly they were still higher than basic levels. From improved level the fall in pulmonary functions was significant only in FEV_1, $FEV_1\%$ and MVV. After one hr the spirometric parameters were near or more than basal levels. This was probably due to the fact that some effect of isoprenaline was still present while that of smoking was over.

Clarke (1) observed a decrease in airways conductance even with filtered cigarettes. Nadel and Comroe (7) observed decrease in airways conductance on acute inhalation of cigarette smoking and reversal or prevention of effect with isoprenaline. Our observations support and indicate that basically smokers have increased airways resistance and smoking causes further narrowing of larger air passages. This immediate bronchoconstriction produced by smoking is probably due to direct irritating effect of smoke on bronchial trees or it may be mediated through liberation of histamine (8).

The present observations show that isoprenaline does cause bronchodilatation

in smokers but is unable to block completely the immediate bronchoconstrictive effect of cigarette smoking specially on large air passages.

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