PULMONARY FUNCTION TESTS IN THREE WHEELER DIESEL TAXI DRIVERS IN BIKANER CITY

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Background and Methods: Pulmonary Function Tests (PFTs) including spirometry give an important clue in terms of respiratory chronic airway disorders and can predict early damage to pulmonary system. The present study was carried out in 100 diesel taxi drivers (Study group) of Bikaner city and compared it with 100 healthy medicos (control group) in the age group of 20 to 50 years by computerised spirometer. The pulmonary function tests included FVC, FEV_1, FEV_1/FVC, $FEF_{25-75\%}$ and PEFR. All subjects were divided into non-smokers and smokers so that influence of smoking on lungs can be studied. **Results:** The restrictive impairment was found in 87% of study group, of which 50% were smokers and 37% were non-smokers, mixed pattern (both restrictive and early obstructive impairment) was found in only 13% of study group, of which 7% were smokers and 5% non-smokers. **Conclusion:** When all the above five parameters were taken together, they were indicative of mixed pattern (both restrictive and obstructive) lung impairment.

Keyword: Pulmonary function tests, Diesel, Taxi driver, Smoking, Air pollution, Occupational hazard

INTRODUCTION

Nature maintains a balance between land, water, air and all living organisms in the world. Any kind of imbalance in the biosphere results in environmental pollution. Rapid industrialisation, urbanisation, use to motor vehicles, agriculture, nuclear energy program are the major causes of environmental pollution in the world. Experimental studies indicate that due to airborne contaminants of diesel fumes, changes in PFTs are seen due to injury to airways and parenchyma in subjects who are exposed to it, because lungs are the major site of contact between the body and the environment.[1–2]

The human lungs encounter approximately 7 litres of air per minute.[3] Thus it is evident that lungs are a target for adverse effects of noxious gases due to air pollution. The airborne contaminants include: Nitric oxide (NO_2), Carbon mono oxide (CO), Carbon dioxide (CO_2), Ozone (O_3) Sulphur dioxide (SO_2), Hydrocarbons and Suspended particulate matters (SPM). They are responsible for injury to airways and lung parenchyma and lead to bronchoconstriction, increased mucous secretion and increased alveolar swelling. Nitrous fumes may results acute pulmonary oedema.[4] To protect it our body employs several defence mechanisms, e.g., increased mucous secretion. Inhalation of NO_2 and SO_2 causes bronchoconstriction, mucosal irritation and alveolar swelling leading to obstructive and restrictive disorders of lungs.[5]

Bikaner City is almost a non-industrial city. So if there is any cause of air pollution, it is due to the exhaust fumes of diesel taxies, because it is the only mode of public conveyance. Natural air is being continuously polluted by increased urbanisation and quick rise in the number of automobile vehicles. Levels of pollution in various cities of India are shown in Table-1. The taxi drivers who drive for 7–10 hours/day are extensively exposed to this pollution.

The present study was conducted on the diesel taxi drivers of the Bikaner city where the density of taxies on the narrow roads of Bikaner is very high (around 10,000 taxies) in 10×10 km^2 area of the city. Thus, taxi drivers are prone to develop pulmonary function impairments.

Table-1: Level of pollutants in various cites of India[6]

Cities	Particulate Pollution	Gas pollution	
	SPM (200 μg/m^3)	SO_2 (80 μg/m^3)	NO_2 (80 μg/m^3)
Ahmedabad (Ashram Road)	433	7	58
Bangalore (J.C.Road)	313	6	29
Chandigarh (Sector 7)	285	10	22
Chennai (T. Nagar)	180	24	21
Delhi (Lajpat Nagar)	392	10	23
Guwahati (Silpukhuri)	217	7	28
Hyderabad (Greenland)	329	7	34
Kolkata (Shyam Bazar)	174	4	34
Lucknow (Dalibagh)	310	15	34
Mumbai (Chembur)	333	11	29
Bikaner (Rani Bazar)	6676	26	21
Bikaner (Rani Bazar)	3908	26	11
Bikaner (Public Park)	7167	32	22

MATERIAL AND METHODS

In this study, 100 healthy male three-wheeler (diesel taxi) drivers between age 20–50 years were studied in morning hours and 100 normal volunteer medical college staff and students were included in control group.

For study group, smokers as well as non-smokers of age 20–50 years with minimum 7 years of driving out of taxi drivers at different taxi stands of Bikaner city were selected by convenience sampling. For control group, staff and students of Sardar Patel Medical College living in better (healthy) environment than the drivers of same age group were selected.

Subjects with history of asthma, chronic bronchitis with purulent sputum, history of haemoptysis, fever, known case of TB, cardiovascular disease, congenital anomalies, obesity and anaemia were excluded from the study.

Computerised Spirometer (for window kit Cosmed Ponygraphic Spirometer) was used to measure respiratory function tests. This is solid state electronic equipment. The subject has to respire in to a sophisticated transducer, which is connected to the instrument by means of a cable. The computer printouts of Forced Vital Capacity (FVC), Forced Expiratory Volume in 1st second (FEV_1), FEV_1/FVC, Forced Expiratory Flow between 25% and 75% of vital capacity ($FEF_{25–75\%}$), and Peak Expiratory Flow Rate (PEFR) with graphic curves were obtained.

The data of pulmonary function tests obtained by the Spirometer were tabulated in master chart; from this chart sub-tables on the basis of age group, duration of exposure, tobacco smoking etc. were prepared. From the control data, same tables were formed as in study group. From all these data, mean values ±standard error were calculated. Finally the results obtained were compared with the reference data. Statistical analysis was done by student's *t*-test.

RESULTS

Table-2 shows the distribution of subjects in control and study groups according to their age and habit of smoking. The study group shows that taxi drivers are more in habit of smoking as compared to control group. Data revealed that smokers were more under the age of 30 years both in study and control groups.

Table-3 shows that values of FVC were statistically significantly lower in study group of both non-smokers and smokers, except above 40 years.

Table-4 shows that values of FEV_1 were lower in study group both in smokers and non-smokers, which were statistically highly significant in age group of up to 40 year in non-smokers and between 31–40 years in smokers.

Table-5 shows that values of FEV_1/FVC ratio, there was no statistically significant differences found in study and control.

Table-6 shows that value of $FEF_{25–75\%}$ were lower in study group of both non-smokers and smokers groups, which were statistically highly significant except above 40 year age group in non-smokers.

Table-7 shows that value of PEFR were lower in study group both in non-smokers and smokers group and the differences was statistically highly significant in smokers above 30 years of age.

Table-2: Distribution of subjects in different groups

Age (yrs)	Control Group			Study Group		
	Non-smokers	Smokers	Total	Non-smokers	Smokers	Total
≤30	69	13	81	24	22	46
31–40	5	3	8	24	17	41
>40	7	4	11	6	7	13
Total	81	19	100	54	46	100

Table-3: Percentage predicted FVC

Groups	Age (yrs)	Control Group		Study Group		*p*
		Mean	SE	Mean	SE	
Non-Smokers	≤30	83.58	1.92	61.39	3.69	<0.01
	31–40	85.94	8.35	60.52	3.10	<0.001
	>40	76.49	5.46	61.08	7.03	<0.05
Smokers	≤30	77.26	5.67	55.43	4.60	<0.01
	31–40	75.17	7.78	51.68	2.58	<0.01
	>40	68.23	2.92	56.61	9.73	NS

Table-4: Percentage predicted FEV_1

Groups	Age (yrs)	Control Group		Study Group		*p*
		Mean	SE	Mean	SE	
Non-Smokers	≤30	91.85	1.77	68.55	5.29	<0.001
	31–40	99.48	8.91	69.70	7.48	<0.001
	>40	87.66	5.93	71.23	18.16	<0.05
Smokers	≤30	85.32	4.17	62.60	5.17	<0.05
	31–40	84.27	3.74	58.65	3.33	<0.001
	>40	61.85	7.45	61.40	9.20	NS

Table-5: Percentage predicted FEV_1/FVC

Groups	Age (yrs)	Control Group		Study Group		*p*
		Mean	SE	Mean	SE	
Non-Smokers	≤30	115.36	0.93	117.68	1.10	NS
	31–40	122.00	0.92	121.10	1.47	NS
	>40	120.00	0.98	122.13	1.58	NS
Smokers	≤30	115.95	1.53	117.99	1.15	NS
	31–40	117.80	4.10	118.34	2.00	NS
	>40	124.35	1.53	115.39	3.74	NS

Table-6: Percentage predicted $FEF_{25–75\%}$

Groups	Age (yrs)	Control Group		Study Group		*p*
		Mean	SE	Mean	SE	
Non-Smokers	≤30	104.87	3.08	88.32	5.55	< 0.01
	31–40	124.12	10.07	95.32	7.39	<0.001
	>40	103.91	724	106.05	9.11	NS
Smokers	≤30	103.79	7.79	80.68	6.27	<0.001
	31–40	121.37	19.79	77.49	8.48	<0.001
	>40	117.48	22.65	62.36	9.77	<0.001

Table-7: Percentage predicted PEFR

Groups	Age (yrs)	Control Group		Study Group		*p*
		Mean	SE	Mean	SE	
Non Smokers	≤30	87.83	2.60	68.69	3.87	≤0.05
	31–40	86.50	9.25	75.90	5.43	NS
	>40	99.91	8.98	90.35	4.07	NS
Smokers	≤30	78.59	6.89	63.63	5.77	<0.05
	31–40	104.53	1.80	73.33	7.41	<0.001
	>40	106.03	8.60	49.53	10.08	<0.001

DISCUSSION

When all the above five parameters were taken together they all were reduced except FEV_1/FVC ratio that was normal and indicated restrictive lung impairment. The reduced values of PEFR indicate obstructive lung impairment. While the values of FVC, FEV_1 and $FEF_{25–75\%}$ were reduced significantly and indicated mixed restrictive and obstructive impairment. Mixed picture of restrictive and obstructive lung impairment was prevalent amongst taxi drivers (study group). These can be attributed to the adverse effect of SPM, CO, CO_2, NO_2, O_3 and SO_2 content of diesel taxi effluents on respiratory system.

Human lung parenchyma retain $PM_{<2.5}$ (the so called respiratory PM). PM is a highly toxic material because of its small size chemical composition, as suggested by the influx of inflammatory leukocytes into the airspace (30-x). PM has a variety of effect on lung defences. Transition metals contained in PM, particularly iron, damage the airways by generating free radicals and stress.[7] The main cells involved in the initial pro-inflammatory responses to particles are the macrophages and induces oxidative stress only in these cells.[8] The inflammatory effect of PM has been studied in mice, in which the inhalation of 300 μm^{-3} of $PM_{2.5}$ cause increase in TNF-β, interferon-β, IL-6 and transforming growth factor-β.[9] Furthermore, carbon particles, which are important constituents of PM_{10}, cause the release of immature neutrophil from the bone marrow, underlining the chief role of PM in pathogenesis of COPD. Ozone induces decrements in pulmonary function, increasing airway responsiveness and resistance and altering lung volume and flow.

Diesel exhaust particulate constitutes a large proportion of the PM in ambient air. In particular, diesel exhaust fumes cause bronchoconstriction, neutrophilic inflammation and dysfunction of alveolar phagocytosis together with histamine release from mast cell in healthy individuals.

In the present study there was a fall in percentage predicated FVC in study group as compared to control both in non-smokers and smokers. The difference was highly significant ($p<0.001$) in non-smokers in age group 31–40 years. The values were statistically significant ($p<0.01$) except above 40 years smokers group.

The FEV_1 were decreased in study group both in smokers and non-smokers which were statistically highly significant ($p<0.001$) in age group of up to 40 years in non-smokers, and between 31–40 years in smokers while significant ($p<0.05$) in above 40 years in non-smokers and below 30 years in smokers.

Various studies done earlier showed confounding results. In exposed traffic workers as compared to control the value of FVC decreases statistically significantly. Pulmonary function impairment of the workers in garage was mostly of obstructive type and there was deterioration of FVC and FEV_1.[10–12] In cigarette smokers exposed to NO_2, CO and SPM showed the effect seen in the terminal

bronchioles and decreased the pulmonary compliance and reduced FVC.[13]

According to several studies, high level of SO_2 exposure causes higher incidence of chronic bronchitis, in which the values of FEV_1 were reduced.[14] SO_2 and sulphate pollution increases the risk of respiratory infection and causes bronchoconstriction.[15] SO_2 causes bronchoconstriction in part by activating muscarinic afferent nerves. The bronchoconstriction provided by muscarinic effects of SO_2 can be partially inhibited by muscarinic antagonists such as atropine in asthmatic patients.[16]

The frequency of bronchitis had close relation with age, amount and duration of cigarette smoking. In *bidi* smokers it was observed that there is higher incidence of decreased FEV_1 in older subjects. There was higher percentage of subjects in cigarette smokers having abnormal FEV_1.[17]

The $FEF_{25-75\%}$ was lower in study group of both non-smokers and smokers. In smokers of study group value was statistically highly significant ($p<0.001$) in all age groups while in non-smokers, the level of significance increased with age up to 40 years. In some studies lung function test in drivers and mechanics showed lower values of $FEF_{25-75\%}$ as compared to control workers.[18,19]

The PEFR were lower in study group both in non smokers and smokers, difference was statistically highly significant ($p<0.001$) in smokers above 30 years age group. Below 30 years it was statistically significant ($p<0.05$) in control group. A single study showed that PEFR decreased in people living in heavily polluted areas.[10]

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