



A STUDY TO ASSESS THE RESPIRATORY IMPAIRMENTS AMONG THREE WHEELER AUTO TAXI DRIVERS

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ABSTRACT

Transportation is the major culprit of air pollution and lungs are more susceptible to it. Auto taxi drivers are exposed to harmful chemicals and toxic substances from their vehicle-exhausts through inhalation. The purpose of the present study was to evaluate pulmonary functions of auto drivers by means of spirometry. The present study was carried out on 96 three wheeler auto taxi drivers of Chinsurah town in Hooghly, a southern district of West Bengal in India and compared it with 90 healthy male residing in the same geographical area those are not occupationally exposed to automobile exhaust. The pulmonary function test (PFT) variables were Forced Vital Capacity (FVC), Forced Expiratory Volume in 1second (FEV1), Ratio of FEV1 and FVC, Peak Expiratory Flow Rate (PEFR) and Forced Expiratory Flow 25-75% (FEF25-75%). All subjects were divided into smokers and nonsmokers. PFT results showed significant low value of pulmonary function indices including forced vital capacity (FVC), forced vital capacity in the first second (FEV1), forced expiratory flow 25-75% (FEF25-75%) and peak expiratory flow rate (PEFR) compared with control group subjects ($p < 0.05$ to $p < 0.001$) except %FEV1/FVC ($p > 0.05$). The restrictive type pulmonary impairment was found in smokers and nonsmokers study subjects. Obstructive and mixed type of pulmonary impairment were noted in smokers study subjects. In conclusion we can assume that petrol engine emission from auto taxi causes respiratory impairment to its drivers. There was higher percentage of respiratory impairment in smoker-drivers than nonsmoker ones in our study.

Key words: Pulmonary function test, Petrol, smoking, occupational hazard, auto taxi driver

INTRODUCTION

The present transport system is the major offender of air pollution which produces a grim hazard to human health (Savile, 1993). Experimental studies indicate that airborne contaminants of automobile fumes cause injury of airways and parenchyma in subjects who are exposed to it as lungs are the major site of contact the body and the environment (Lewis et. al., 1974; Fagerstrom et. al., 1998). Lungs are more susceptible to air pollution as the human lungs encounter approximately 7 liters of air per min

(Ganong, 1981). The vehicular emission include primary reactive species (e.g. carbon monoxide and benzene), particulate matters and secondary reactive species (e.g. ozone and nitrogen oxides. Airborne contaminants like nitric oxide, carbon monoxide, carbon dioxide, sulphur dioxide, hydrocarbons and suspended particulate matters are responsible for injury of airways and lung parenchyma and lead to bronchoconstriction, increased mucus secretion and increased alveolar swelling. Inhalation of nitrogen

dioxide and sulphur dioxide causes bronchoconstriction, mucosal irritation and alveolar swelling leading to obstructive and restrictive pulmonary impairment (Waldron, 1985). Long term employment in the transport industry of bus (bus driving and mechanics) in combination with smoking is linked with development of chronic respiratory symptoms and lung function impairment (Zuskin et. al., 1994). Diesel exhaust contributes COPD in bus drivers (Hart et. al., 2006). Petrol engine emission is ubiquitous sources of particulate matters and non particulate matters. Yet health hazards have received little study in comparison with those of diesel engine emission (Reed et. al., 2008). The present study was carried out on the three wheeler auto taxi drivers to ascertain the effect of petrol exhaust on pulmonary functions.

METHODS

Study population

The present cross sectional study was conducted in Chinsurah, district Hooghly of West Bengal state. The study population included 96 male three wheeler auto drivers comprising of 56 smokers and 40 nonsmokers and 90 control healthy male comprising 40 smokers and 50 nonsmokers having age limit in between 25-50 years. Written consent from the subjects involved in the study was obtained. Exclusion criteria for the subjects included presence of any self reported acute illness, lung diseases like chronic obstructive pulmonary disease, heart failure, malignant diseases, chronic liver or kidney failure and diabetes mellitus. Before spirometry the procedure was explained and demonstrated to each subject.

Protocol

Survey included three phases: i). Interview of the subjects, ii). Anthropometric measurement and iii). pulmonary function test. Interview was done with a structured questionnaire to obtain information on age, occupation, tobacco related behaviors including type of tobacco use and duration of use. Body weight was measured using bathroom scale accurate to 0.5kg. The scale was kept on a flat surface and was adjusted with '0' mark. Then the subject was

requested to step on it in bare feet. Weights were taken in light cloth. Weight was recorded to the nearest 0.5kg. Height was measured using anthropometric rod. Height of the subject was recorded without footwear and expressed to the nearest 0.1cm. Body mass index (BMI) was calculated from the height and weight using following equation: $BMI (kg / m^2) = weight (kg) / height (m)^2$. Subjects having BMI between 18.5-25.0 were selected for pulmonary function tests. Spirometry was done using computerized spirometer (Medikro Spirostar USB Spirometer, Model: M929, Finland). The subject was asked to sit comfortably in a chair. The complete procedure was explained and demonstrated. All doubts if any were cleared. Subject was instructed to breathe in fully by deep inspiration with closed nostril. Three trials were given for each subject. Best of the three was recorded and analyzed. Forced vital capacity (FVC), forced expiratory volume in 1st second (FEV1), Forced expiratory flow between 25% and 75% of forced vital capacity (FEV25-75%) and peak expiratory flow rate (PEFR) were represented in our results. FVC, FEV1 and FEV1/FVC were also expressed as a percentage of predicted to control for the influence of age, gender and height by setting the spirometer according to acceptability standard outlined by Jindal. Test values for the FVC, FEV1 and FEV1/FVC fell below the lower limit of the 95% confidence interval of the predicted value were classified as abnormal (Aaron et. al., 1999). A low spirometric FVC together with a normal or high FEV1/FVC ratio has been classified as a restrictive abnormality (Carpo, 1994; Cheeta et. al., 2004). The fall in FEV1, PEFR and other flow rates indicate obstructive lung changes (Rubeena et. al., 2009).

Data analysis

Data obtained from the study were given as mean \pm SD. The statistical significance was determined by student's t test. Two tailed p values were used throughout and p value less than 0.01 were judged as statistically significant. Chi square test was done to evaluate association between driving occupation and respiratory impairment. The association was considered significant when $p < 0.01$.

RESULTS

In our study both control and study subjects were divided into two categories: smokers and nonsmokers. Table-1 shows the anthropometric values of both experimental and control group subject. There is no significant difference of anthropometric values between control and experimental subjects. The lungs volumes (FVC, FEV1, FEV1%) of control and auto drivers were presented in table-2. It was found that mean values FVC and FEV1 of control subjects were higher than the respective auto drivers. There was no statistically

significant difference of FEV1% between control and auto drivers. Respiratory flow rates (FEF25-75%, PEFR) of smokers and nonsmokers study and control subjects were presented in table-3. Mean values of flow rates were significantly lower in study group of both smokers and nonsmokers in compare to respective control group. The respiratory impairment of control and experimental subjects were evaluated on the basis of values of FVC, FEV1 and percentage of FEV1 to FVC and represented in table 4 and figure1 and 2. Respiratory impairments were found higher among the exposed subjects.

Table-1

Comparison of anthropometric measurements between auto driver and control subjects

Parameters	Smokers			Nonsmokers		
	Control	Experimental	p-value	Control	Experimental	p-value
Age (years)	40.40 ± 6.13	41.25 ± 5.20	>0.05	39.50 ± 2.97	40.00 ± 3.47	>0.05
Height (cm)	164.00 ± 6.25	163.64 ± 5.85	>0.05	164.70 ± 6.21	164.00 ± 5.16	>0.05
Weight (kg)	60.50 ± 5.80	59.00 ± 6.96	>0.05	63.03 ± 6.90	62.60 ± 6.30	>0.05
BMI (kg/m ²)	22.50 ± 2.74	22.03 ± 2.34	>0.05	23.25 ± 2.65	23.20 ± 2.39	>0.05

Table-2

Comparison of lungs volumes between control and experimental subjects

Lungs volumes	Smokers			Nonsmokers		
	Control (n=40)	Experimental (n=56)	p-value	Control (n=500)	Experimental (n=40)	p-value
FVC (l)	3.26 ± 0.76	2.78 ± 0.65	<0.001	3.81 ± 0.59	3.08 ± 0.45	<0.001
FEV1 (l)	2.78 ± 0.59	2.36 ± 0.57	<0.001	3.36 ± 0.62	2.59 ± 0.22	<0.001
FEV1%	85.40 ± 12.90	84.50 ± 8.28	<0.001	88.30 ± 8.95	84.20 ± 7.67	<0.001

Table-3

Comparison of pulmonary flow rates between control and experimental subjects

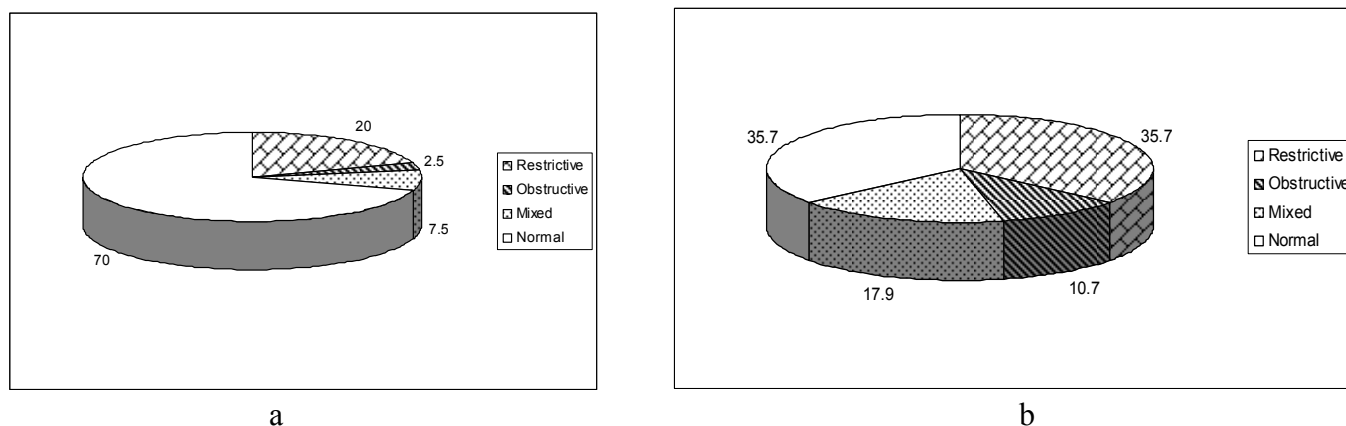
Pulmonary flow rates (l)	Smokers			Nonsmokers		
	Control (n=40)	Experimental (n=56)	p-value	Control (n=50)	Experimental (n=40)	p-value
PEFR	5.29 ± 1.50	4.31 ± 1.31	<0.001	6.03 ± 1.53	5.04 ± 1.36	<0.001
FEF25-75%	3.44 ± 1.18	2.59 ± 0.91	<0.001	3.64 ± 1.04	3.01 ± 0.96	<0.001

Table-4

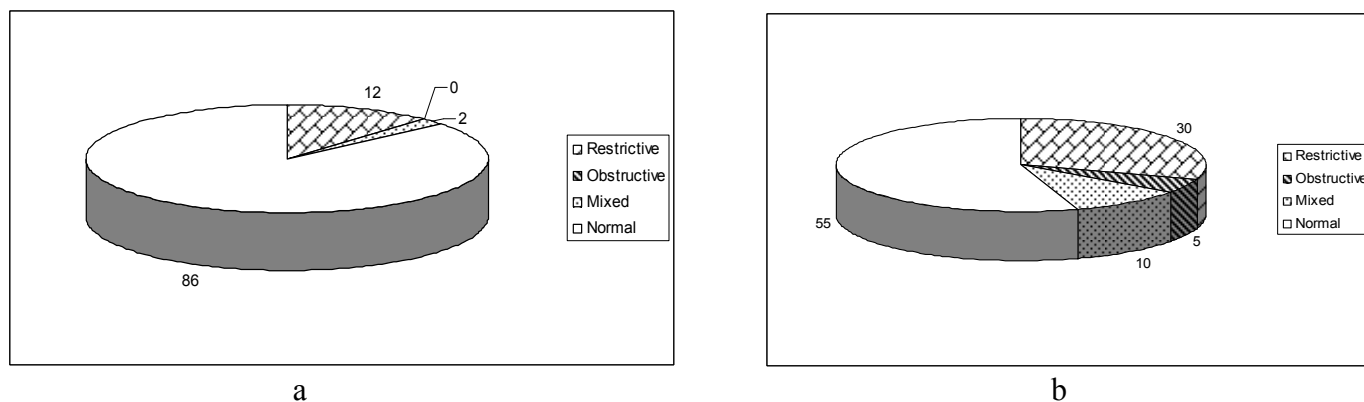
Comparison of lung functions tests in control and experimental subjects

Lung functions	Smokers		Nonsmokers	
	Control	Experimental	Control	Experimental
Normal	28	20	43	22
Restrictive	8	20	6	12
Obstructive	1	6	0	2
Mixed	3	10	1	4
total	40	56	50	40

* $p < 0.001$ in comparison of respiratory impairment between control and experimental group

**Figure 1**

Comparison of lung functions tests in smoker control (a) and auto taxi drivers

**Figure 2**

Comparison of lung functions tests in nonsmoker control (a) and auto taxi drivers

DISCUSSION

There was no significant difference in physical parameters like age, height, weight and BMI and thereby showing proper matching of control and experimental subjects. We tested five respiratory parameters viz. FVC, FEV₁, percentage of FEV₁ to FVC (FEV₁%), PEFR and FEV₂₅₋₇₅%. Out of these parameters except FEV₁% all were significantly reduced in experimental subjects both in smokers and nonsmokers. Test values for the FVC, FEV₁ and FEV₁% fell below the lower limit of the 95% confidence interval of the predicted value were classified as abnormal (Aaron et. al., 1999). A low spirometric FVC together with a normal or high FEV₁% has been classified as a restrictive

abnormality (Carpo, 1994; Cheeta et. al., 2004). The values of FVC, FEV₁, PEFR and FEF₂₅₋₇₅% were reduced significantly and indicated both obstructive and restrictive lung impairment. Thus mixed picture of restrictive and obstructive pulmonary impairment was prevalent among auto driver. Like diesel engine exhaust emission from petrol engine contains carbon monoxide, hydro carbon, sulphur dioxide, nitrogen oxides and particulate matters. However concentration of CO and hydro carbon are much more in petrol engine emission where as particulate matters is low in petrol engine emission than diesel engine emission (Weisenberger, 1984). There is no difference in the concentration of sulphur dioxide

and nitrogen oxides in petrol and diesel engine emission (Weisenberger, 1984). Low FVC in auto drivers may be due to carbon monoxide and nitrogen oxides in petrol engine emission. Subjects exposed to carbon monoxide and nitrogen oxides showed low FVC (Rao et. al., 1991). According to several studies, high level of sulphur dioxide causes higher incidence of chronic bronchitis in which the values of FEV1 were reduced (Godhkhindfi and Doshi, 1984). SO₂ and sulphate pollution increase the risk of respiratory infection and bronchoconstriction (Kamat et. al., 1984). Thus low FEV1 in auto drivers may be due to sulphur dioxide from their vehicles. In cigarette smokers exposed to NO₂, CO and SPM showed the effect in the terminal bronchioles (Rao et.al., 1991). The FEF_{25-75%} was lower in study group of both smokers and nonsmokers. This observation was supported by some studies on lung function test in drivers and mechanics (Chattopadhyaya et. al., 2003; Zuskin et. al., 1994). Low PEFR and FEV_{25-75%} in auto drivers may be

due to NO₂ and CO from their vehicle emission. From the above discussion we can presume that petrol engine emission from auto taxi causes respiratory impairment to its drivers. There was higher percentage of respiratory impairment in smoker-drivers than its nonsmoker-counterpart in our study. Thus auto driving with smoking habit can be termed as a dreadful combination in respect to pulmonary health.

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REFERENCES

1. Aaron SD, Dales RE, Cardinal P. How accurate is spirometry at predicting restrictive pulmonary impairment? CHEST. 1999; 115: 869-873.
2. Carpo R. Pulmonary function testing. New Eng J Med. 1994; 331: 25-30.
3. Cheeta A, Marangio E, Olivieri D. Pulmonary function testing in interstitial lung diseases. Respiration. 2004; 71: 209-213.
4. Chattopadhyaya BP, Alam J, Roy Chowdhary A. Pulmonary function abnormalities associated with exposure to automobile exhaust in a diesel bus garage and roads. Lung. 2003; 181: 291-302.
5. Fagerstrom KO, Ramstrom L. Can smokeless tobacco rid us of tobacco smoke? Am J Med. 1998; 104: 501-503.
6. Ganong WF. Review of Medical Physiology. Lang Medical Publication (10th Edition) 1981, p-507.
7. Godhkhindfi KD and Doshi VB. A cross sectional comparative study between three urban communities (inclusive of slums) with different air pollution levels and a rural community for health morbidity and lung functions. Lung India. 1984; 2: 21-28.
8. Hart JE, Laden F, Schenker MB and Garshick E. Chronic obstructive pulmonary disease and mortality in diesel exposed rail-road workers. Env Health Perspect. 2006; 114(7): 1013-1017.
9. Kamat SK, Doshi VB, Patade VD, Naik M. Third year analysis of regularly followed sample of Bombay air pollution study population and correlation with other factory. Lung India. 1984; 2: 110-130.
10. Lewis TR, Moorman WJ, Yang YY, Stara JF. Long term exposure to auto exhaust and other pollutants mixture. Arch Env Health. 1974; 21: 102-106.
11. Malik SK. Chronic bronchitis in bidi smokers. Indian J Chest Diseases. 1974; 16: 94-99.
12. Rao NM, Petel TS, Raiyani CV, Kulkarni PK, Agarwal AL and Kashyap SK. A dose response relationship between pollution index and pulmonary function in shopkeepers exposed to auto exhaust. Indian J Environ Protect. 1991; 11: 737-740.

13. Reed MD, Barrett EG, Campen MJ, Diven KK, Gigliotti AP, McDonald JD, Seagrave JC, Mauderly JL, Seilkop SK and Swenberg JA. Health effect of subchronic inhalation exposure to gasoline engine exhaust. *Inhal Toxicol.* 2008; 20(13): 1125-1143.
14. Rubeena B , Mahagaonkar AM , Kulkarni NB , Nadeem A, Nighute S . Study of pulmonary function tests among smokers and nonsmokers in a rural area. *Pravara Med Rev* 2009; 4(1): 11-16
15. Savile SB. Automobile options and quality management in developing countries. *Industrial Env.* 1993; 16: 20-32.
16. Waldron HA. Lecture notes on Occupational Medicine, 3rd Edn. Oxford. Blackwell Scientific Publication; 1985, p 47-163.
17. Weisenberger BL. Health effects of diesel emission –an update. *Occupational Med.* 1984; 34(3): 90-92.
18. Zuskin E, Mustajbegovic J and Schachter EN. Respiratory symptoms and lung function in bus drivers and mechanics. *Am J Industrial Med* 1994; 26(6): 771-783.