STUDY OF VENTILATORY LUNG FUNCTION TESTS OF BUS DRIVERS IN PUNE CITY

Anuradha Rajiv Joshi*, S M Vaidya, Neha Pimpale*****,

* Associate Professor, ** Professor and Head, *** P G student, Department of Physiology,
Bharati Vidyapeeth (Deemed to be University) Medical College, Pune. India 411043

Abstracts: **Background &Objectives**: Bus drivers are highly exposed to diesel exhaust and other atmospheric pollutants. Thus the study was planned to study ventilatory lung function tests of bus drivers in Pune city.

Methods: Age and height matched control group subjects (n=31) lung function tests were recorded by computerized spirometer and were compared with bus drivers working for 5-10 years (study group I, n=31) and working for 10-20 years (study group II, n=31) Data was analyzed by student's unpaired't' test.

Results &Interpretation: Study group I and Study group II bus drivers showed statistically significant decrease in ERV, FVC and FEV_1 than control group subjects. Study group II showed statistically significant reduction in FVC than study group I subjects. **Conclusion:** Ventilatory lung function tests were decreased in study group subjects. The results suggested combined (obstructive and restrictive) nature of lung diseases.

Keywords: Bus drivers, pulmonary function tests, automobile exhaust

Author for correspondence: Dr Anuradha Rajiv Joshi, Associate Professor, Department of Physiology, Bharati Vidyapeeth (Deemed to be University) Medical College, Pune. India. e- mail: anuradhajoshi30@gmail.com

Introduction:

Pune city is growing rapidly owing to the expanding economic base. This has led to an increase in the use of motor vehicles with a subsequent rise in the levels of air pollution. Among the motor vehicle-generated air pollutants, highly significant percentage of the air particles are emitted by diesel exhaust. Bus drivers are consistently exposed to diesel exhaust and other atmospheric pollutants. [1]

The air quality index shows that Pune city is moderately polluted in all the seasons.[2] Increased number of vehicles result in slow traffic movement which is further compounded by acceleration and de-acceleration of the vehicles on the congested roads. This leads to increased air pollution in the city. Pollutants may be changed by chemical reactions after being emitted. Oxides of nitrogen and volatile organic compounds from automobile exhaust react with sunlight and produce ozone.[3]

Acute effects of the exhaust exposure include irritation of eyes and nose, headache, fatigue, and nausea.[4]Chronic exposure is associated with cough, sputum production, backache, increased stress[5] and decrease in lung function.[6] The most important traffic-related air pollutant is particulate matter (PM). Fine particles of particulate matter (PM 2.5) can reach smaller airways and alveoli leading to decrease in the pulmonary compliance.[7]

Pulmonary function tests (PFTs) are an important tool in the investigation and monitoring of patients with respiratory pathology. Spirometry is the most commonly used lung function screening study. It has been observed that exposure to automobile exhaust and fuel vapor impairs lung function in a time dependent manner. If changes in pulmonary functions can be detected in the earliest stages, then preventive or corrective measures are likely to be more beneficial.

Numerous studies[1.3.4.7] have been done to study effect of air pollution in petrol pump workers and mechanics but it is not studied in bus drivers of public transport workers of Pune city. Thus the present study was aimed to assess ventilatory lung function status of bus drivers in Pune city.

Material and Methods:

The study was approved by institutional ethical committee and the procedure was done in accordance with the Helsinki Declaration of 1975. It was an observational cross-sectional study conducted from May 2015 to May 2016 in public transport bus depot in Pune. It was conducted on 105 volunteers in the age group of 25 to 45 years who were driving the bus from 5 to 10 years to 10 to 20 years. Purpose and procedure of the study was explained to the subjects and a written consent was obtained. History was taken and all subjects were clinically evaluated in detail as per the proforma. Subjects were chosen based on inclusion and exclusion criteria. Subjects with habit of smoking and alcohol consumption, with any acute illness or suffering from respiratory diseases like asthma, bronchitis, COPD or taking medications, such as steroids and bronchodilators were excluded.

Subjects were divided in to two groups. Control group (n=35) consisted of height, weight and age matched indoor office staff without any history of occupational exposure. Study group (n=70) bus drivers were further divided into two groups. Study group I (n=35), bus drivers who had been driving bus for 5 to 10 years and study group II (n=35), bus drivers who had been driving bus for 10 to 20 years.

Both the groups were first interviewed by a questionnaire which included personal data and occupational history. Personal data included history of smoking, tobacco chewing and alcoholism. Occupational history included questions regarding present employment, duration of employment as bus driver, working hours and job responsibilities.

Ventilatory lung function tests were recorded on a computerized portable Schiller lung function unit SP-1(RS 232). Anthropometric parameters like height and weight were recorded as per standard guidelines.[8]The recorded parameters were compared with the inbuilt pulmonary function norms for the Indian population depending upon the age, sex, height, and weight.

Recording of static and dynamic pulmonary function tests was conducted on subjects in standing position as FVC and VC are 2% higher in standing position.

All the pulmonary function tests were recorded at noon before lunch as expiratory flow rates are highest at noon.[9] Anthropometric measurements and lung function tests were taken in standing position on the same day and at one sitting.[8] Three satisfactory reading were taken as per guidelines of American Thoracic Society. The reading showing the maximum effort was selected.

The parameters recorded were Tidal Volume (V_T - Lit), Expiratory Reserve Volume (ERV - Lit), Inspiratory Reserve Volume (IRV-Lit), Forced Vital Capacity (FVC-Lit), Forced expiratory volume at the end of first second (FEV_1- Lit), FEV_1 / FVC ratio.

Statistical analysis:

Data analysis was done using SPSS version 20.0. Data was expressed as Mean ± SD. Student's unpaired 't' test was done and 'p' value was determined. p <0.05 was considered as significant. Spearman correlation test was used to correlate duration of exposure and age.

Results:

Out of 105 subjects only 93 subjects completed the whole study. From control group 4 subjects were unable to meet ideal spirometry FEV_1 standards and from study group subjects 8 suffered viral infection so could not complete the study.

Table 1 shows baseline characteristics of the control and study group subjects such as age, height and weight. Baseline characteristics of all the subjects were similar.

Table:1 Comparison of Anthropometric parameters in control group and study group subjects

Parameters	Control group (n=31)		Study group (n=62)		p value
	Mean	SD	Mean	SD	
Age (years)	39.33	6.58	39.01	7.57	> 0.05
Height (cm)	165.53	4.96	169.93	5.59	> 0.05
Weight (kg)	65.93	8.61	70.09	11.23	> 0.05

p> 0.05 - statistically not significant

Table:2 : Comparison of ventilatory lung function tests in control group and study group I

Parameters	Control group (n=31)		Study group I (n=31)		p value
	Mean	**SD**	**Mean**	**SD**	
VT (L)	0.68	0.17	0.66	0.19	0.348
IRV (L)	1.47	0.27	1.52	0.30	0.518
ERV (L)	1.02	0.12	0.83	0.11	<0.001**
FVC (L)	3.2	0.53	2.58	0.35	<0.001**
SVC (L)	3.01	0.56	2.75	0.56	0.081
FEV1 (L)	2.78	0.42	2.15	0.5	<0.001**
FEV1/FVC	0.9	0.19	0.85	0.24	<0.05*

p< 0.05*- statistically significant

p < 0.001** - statistically highly significant

Table:3 : Comparison of ventilatory lung function tests in control group and study group II

Parameters	Control group (n=31)		Study group II (n=31)		p value
	Mean	SD	Mean	SD	
VT (L)	0.68	0.17	0.66	0.19	0.348
IRV (L)	1.47	0.27	1.52	0.30	0.518
ERV (L)	1.02	0.12	0.83	0.11	<0.001**
FVC (L)	3.2	0.53	2.58	0.35	<0.001**
SVC (L)	3.01	0.56	2.75	0.56	0.081
FEV1 (L)	2.78	0.42	2.15	0.5	<0.001**
FEV1/FVC	0.9	0.19	0.85	0.24	<0.05*

p< 0.05*- statistically significant
p < 0.001** - statistically highly significant

Table:4 : Comparison of ventilatory lung function tests in Study group I and study group II

Parameters	Study group I (n=31)		Study group II (n=31)		p value
	Mean	SD	Mean	SD	
VT (L)	0.66	0.19	0.69	0.21	0.54
IRV (L)	1.52	0.30	1.46	0.34	0.456
ERV (L)	0.83	0.11	0.81	0.16	0.424
FVC (L)	2.58	0.35	2.32	0.23	<0.001**
SVC (L)	2.75	0.56	2.56	0.61	0.231
FEV1 (L)	2.15	0.5	1.96	0.42	0.11
FEV1/FVC	0.85	0.24	0.84	0.16	0.921

$p < 0.001^*$ - statistically significant

Discussion: Bus drivers of Pune city are exposed to automobile exhaust and atmospheric pollutants. All the subjects were middle ages individuals. It's a well-established fact that aging physiologically reduces the lung function parameters.[10] As the mean age of control group was matching with the study group (Table no 1), it might prove that occupational exposure was the main culprit for observed effects of lung function parameters in bus drivers. In the present study the subjects having habit of smoking were excluded as smoking has been reported to accelerate the decline in FEV_1. [11]

Present study showed reduction in ERV in bud drivers (Table no 2,3) which was in accordance with the study done by Mahdi Kargarfard et al. [6] They found that ERV was reduced in individuals exposed to polluted air.

Various studies[11,12] observed that ERV and FVC were reduced in restrictive lung diseases. This might be due to alteration in chest mechanics and due to decreased lung compliance. In another study, reduced mechanical properties of breathing were attributed to exposure to benzene in the vapors of petrol.[13,14] FEV_1 was reduced (Table no.2,3) in both the study groups indicating that there might be some obstruction during expiration and decrease in FVC might be due to some degree of restriction being present in the respiratory tract of bus drivers.

The data recorded in the study reflects failure of subject to inhale or exhale completely.

The subjects residual volume might be more as the expiratory flow is so slow that subject cannot exhale long enough to empty lung to residual volume. Decrease in SVC in study group might be indicating restrictive type of pulmonary dysfunction. (Table no.4)

When FVC maneuver is performed, there is high dynamic compression and airway collapse which reduces the ability to mobilize air volume during expiration and therefore causing air trapping. Consequently, FVC values could be lower than SVC values. As SVC is measured through an unforced attempt, there is comparatively less intrathoracic pressure, and, consequently, one could mobilize a larger volume of air. [15]

Chawala et al [16] reported that FEV_1 declined significantly with increasing number of years of work in petrol stations. Similar results have been found in the present study. (Table no 4) In restrictive lung disease as FVC is reduced consequently FEV_1 is also reduced. Thus if only FEV_1 is evaluated this gives a false impression of obstructive airway disease.[17]

FEV_1/FVC indicates the condition of the bronchial musculature. FEV_1/FVC ratio is used for differentiating obstructive pattern from restrictive pattern of lung diseases. In obstructive lung diseases FEV_1 is reduced disproportionately more than FVC, so FEV_1/FVC ratio is reduced. In restrictive lung diseases, FEV_1 is disproportionately less reduced than FVC, so FEV_1/ FVC ratio is normal or increased.[18]

As evident from our study FEV_1/FVC ratio was reduced but was within normal limits. (Table no. 2,3). This might indicate obstructive pattern of lung diseases in our exposed study group subjects. In the present study exposure to automobile exhaust for more than 10 years was associated with a small but statistically significant reduction of FVC without a significant reduction of FEV_1/FVC in study group II as compared to study group I. (Table no. 4)

Similar results have been observed by Solanki et al.[19] They observed that reduction in FVC without any absolute change in FEV_1/FVC might suggest restrictive nature of lung disease.

Similar results have been observed in a study done in transport workers of one of the most polluted city, Dhaka.[20] Results concluded that 41.7% had obstructive and 4.6% had combined features of pulmonary impairment.

Diesel exhaust particles (DEP) constitute major part of atmospheric particulate matter (PM). It has carbonic mixture composed of various organic compounds. The polyaromatic hydrocarbons after deposition in airway mucous surfaces easily pass through epithelial cells resulting in release of pro-inflammatory cytokine and cause inflammatory changes in lungs.[21]

Not only chronic prolonged but also acute episode of exposure to high levels of PM_{10} elevate circulating levels of IL-6. The cytokine induces a systemic response which plays an important role in the pathogenesis of cardiopulmonary adverse health effects associated with atmospheric pollution.[22,23]

$PM_{2.5}$ which is smaller in size inhaled deeply into the lung and exerts their toxic effects on alveolar cells including macrophages, neutrophils and epithelial cells. During the interaction between the $PM_{2.5}$ and alveolar cells, inflammatory mediators are secreted and phagocytosis takes place in order to protect the organism.[24,25]

Ultra-fine particles (UFP) are particles with less than 0.1μm in diameter, primarily produced by combustion in diesel engines. Urban air contains UFP in large number. Due to the smaller size, they are able to penetrate epithelium and vascular walls to enter into the bloodstream. UFP are found to provoke carcinogenicity, autoimmune disorder, and increase cardiovascular disorders.[26]

When compared to fine particles, the UFP have a higher deposition probability particularly in small airways and the alveolar region of the lungs. They penetrate into and interact with alveolar epithelial, interstitial, and endothelial cells and are less well phagocytized by alveolar macrophages. They release proinflammatory and anti-inflammatory mediators in abundance. All these characteristic lead the UFP to a high access to the blood circulation, induce more oxidative stress and more inflammatory responses than larger particles.[27,28]

Zuskin E et al[1] in their study observed a significantly higher prevalence of most chronic respiratory symptoms when compared to control workers. Bus drivers and mechanics employed for more than 10 years also exhibited higher frequencies of respiratory symptoms and the ventilatory capacity data demonstrated lower values for all parameters.[29,30]

Vehicular exhaust particularly organic diesel exhaust particle induce reactive oxygen species in macrophages and bronchial epithelial cells which are the key cell types targeted by the particulate matter in the lung. Reactive oxygen species in turn activate the promoters of cytokines and chemokines leading to allergic inflammation through activator protein 1 and nuclear factor kappa B signaling pathways.[31]

These particles also act via mitochondrial pathway, induce apoptosis and necrosis in bronchial epithelial cells. These observations indicate that diesel particles themselves can induce airway inflammation.[30,31] The changes might be in the tissue of the lungs due to chronic irritation by pollutants. Earlier studies in workers exposed to petrol and diesel exhaust, it was observed that the reductions of FEV_1, and FVC were reversible if measures are taken earlier.[13]

Conclusions

Thus considering the results of ventilatory lung function tests it is evident that bus drivers of Pune city might have combined (obstructive and restrictive) nature of lung diseases and long term employment as bus driver may be predominantly associated with restrictive nature of lung diseases. So it is very important to identify vulnerable individuals and take precautionary measures at the earliest.

Limitations:

- Diffusing capacity of the lung for carbon monoxide was not done.
- Sample size was small

Recommendations:

- Periodic evaluation of pulmonary function test and early recognition chronic lung function impairment and if possible change in nature of job from driving to office work
- Compulsory use of protective equipment like nose air filter masks
- Intensive promotion of use of CNG buses by government agencies

Acknowledgment:

We would like to thank all the subjects for active participation in the study without whom study could not be possible. Our sincere thanks to management of the PMPML bus depot for their active cooperation in the research

References:

1. Zuskin E, Mustajbegovic J, Schachter EN. Respiratory symptoms and lung function in bus drivers and mechanics. Am J Ind Med. 1994; 26(6): 771-783.
2. Beig.G.,Sachin D. Ghude , Deshpande A. Scientific evaluation of air quality standards and defining Air Quality Index for India, India Institute of Tropical Meteorology, Pune, India, Ministry of Earth Sciences,Govt.ofIndia.2010,http://www.tropmet.res.in/~lip/Publication/RR-pdf/RR-127.pdf
3. Thomas Gotschi, Joachim Heinrich, Joult Sunyer, Nino Kunzil. Long-Term Effects of Ambient Air Pollution on Lung Function: A Review. Epidemiology 2000; 19(5): 690-701.
4. Tager IB, Balmes J, Lurmann F, Long Ngo, Siana Alcorn. Chronic exposure to ambient ozone and lung function in young adults. Epidemiology 2005 ; 16(6): 751-759.
5. Joshi AR, Vaidya SM. Evaluation of perceived stress in bus drivers of Pune city. Natl J Physiol Pharm Pharmacol. 2017;7(1):90-93.
6. Mahdi Kargarfard, Reza Rouzbahani, Saber Rezanejad, Parinaz Poursafa. The effect of air pollution on cardio respiratory performance of active individuals. Arya Atherosclerosis Journal 2009; 5(2): 69-75.
7. Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 US Cities 1987-1994. The NEJM 2000; 34: 1742-1749.
8. Joshi A R, Singh R, Joshi A R. Correlation of pulmonary function tests with body fat percentage in young individuals. Indian J Physiol Pharmacol. 2008 Oct-Dec;52(4):383-8.
9. Hetzel MR. The pulmonary clock. Thorax. 1981; 36:481.
10. Gulshan Sharma, James Goodwin. Effect of aging on respiratory system physiology and immunology. Clin Interv Aging 2006; 1(3): 253-260.
11. Joshi Anuradha Rajiv, Dhorepatil Aneesh Shrihari. Study of ventilatory lung function tests and aerobic capacity in overweight young adults. National Journal of Integrated Research in Medicine 2014; 5(2): 30-33.
12. Sahebjami H. Dyspnea in obese healthy men. Chest 1998; 114:1373-1377.
13. B Rudell, M-C Ledin, U Hammarstrm, N Stjernberg, B Lundback et al. Effects on symptoms and lung function in humans experimentally exposed to diesel exhaust. Occup Environ Med1996; 53:658-662.
14. Ghose MK, Paul R, Banerjee RK. Assessment of the status of urban air pollution and its impact on human health in the city of Kolkata. Environ Monit Assess.2005;108:151-67.
15. Thomas Gotschi, Joachim Heinrich, Joult Sunyer, Nino Kunzil. Long-Term Effects of Ambient Air Pollution on Lung Function: A Review. Epidemiology 2000; 19(5): 690-701.
16. Anuj Chawla, A.K Lavania. Air Pollution And Fuel Vapour Induced Changes In Lung Functions: Are Fuel Handlers Safe? Indian J Physiol Pharmacol2008; 52(3):255-261.

17. Muhammad Zafar, Asmatullah and Muhammad Khalil, Ahmad Khan. Exposure of vehicular pollution in altering the pulmonary functions in traffic policemen in Multan city. Punjab Univ. J. Zool.2015; 30(1):1-5.
18. Amit H Makwana, Jayesh D Solanki, Pradnya A Gokhale, Hemant B Mehta, Chinmay J Shah, Bhakti Gadhavi. Study of computerized spirometric parameters of traffic police personnel of Saurashtra region, Gujarat, Lung India 2015; 32(5):457-461.
19. Ravi B Solanki, Anjali R Bhise, Bharat M Dangi. A study on spiromentry in petrol pump workers of Ahmedabad, India. Lung India 2015; 33(4): 347-352.
20. Mahfuz, M, Ahmed, T, Ahmad, S.A. and Khan, M.H. Altered Pulmonary Function among the Transport Workers in Dhaka City. Health 2014; 6: 2144-2153.
21. G. Mazzarella, F.Ferraraccio., M.V.Prati, S. Annunziata, A. Bianco et al. Effects of diesel exhaust particles on human lung epithelial cells: An in vitro study. Respiratory Medicine 2007; 101: 1155-1162.
22. Eeden V, Tan S.F, Suwa W.C, Mukae T, Terashima H, Fujii T. Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM10). Am J Respir Crit Care Med 2001; 164: 826-830.
23. Salvi S, Holgate S. Mechanism of particulate matter toxicity. Clinical Experimental Allergy 1999; 29:1187-1194.
24. Pozzi R, Berardis D.B, Paoletti L, Guastadisegni C. Inflammatory mediators induced by coarse (PM2.5-1.0) and fine (PM2.5) urban air particles in RAW 2647 cells. Toxicology 2003; 183:243-254.
25. Krelying W.G, Semmler M, Erbe F, Mayer P, Takenaka S,et al. Translocation of ultrafine insoluble iridium particles from lung epithelium to extrapulmonary organs is size dependent but very low. J Toxicol Environ Health A 2002; 65: 1513-1530.
26. Ibald-Mulli A, Wichmann H.E. Epidemiological evidence on health effects of ultrafine particles. Journal of Aerosol Medicine 2002; 15:189-201.
27. Oberdorster G, Ferin J, Gelein R, Soderholm S.C, Finkelstein J.N. Role of the alveolar macrophage in lung injury: studies with ultrafine particles. Environ Health Perspect 1992; 97:193-199.
28. Seaton A, Mac Nee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. Lancet 1995; 345:176-178.
29. Chattopadhyay BP, Alam J, Roychowdhury A. Pulmonary function abnormalities associated with exposure to automobile exhaust in a diesel bus garage and roads. Lung India 2003; 181 (5): 291 - 302.
30. Jimenez L.A, Thompson J, Brown D.A, Rahman I, Antonicelli F,Duffin R et al. Activation of NF-Kappa B by PM10 occurs via an iron mediated mechanism in the absence of I kappa B degradation. Toxicol Appl Pharmacol. 2000 ;166(2):101-10
31. Wei Zhou, Dong Yuan, Shunhua Ye, Pingping Qi, Congying Fu. Health Effects of Occupational Exposures to Vehicle Emissions in Shanghai. Int J Occup Environ Health 2001; 7(1):25-30.

Disclosure: No conflicts of interest, financial, or otherwise are declared by authors