Chapter 5
Discussion
Different factories use different chemicals or ingredients during the process of manufacture of various products which may or may not be harmful to workers. The inhalation of pollutants emerging out of the industries may get associated with respiratory distresses. The by-products or gases expelled from these factories bring about mucosal irritation of the entire respiratory tract. These toxins stimulate or suppress the nerve endings of the respiratory system especially nasal mucosa depending upon the type of pollutants that are released from the factories. The continuous action/effect of these toxins may paralyse the whole respiratory system resulting in respiratory failure.

To arrive at this state, one must consider certain other factors too. Primarily the type of “pollutants”, secondly the “concentration” of these pollutants, thirdly the actual “duration” of exposure, fourthly the “age” of the subject, and finally the “physical conditions” or “clinical condition” of the subject. All these factors will contribute towards the respiratory function/dysfunction. That means, environment plays an important role in the maintenance of an efficient and healthy respiratory conditions/status. The respiratory system will adjust the rate of ventilation as environmental conditions vary, as metabolic demands are altered, or as the physical characteristics of the ventilatory apparatus are modified by growth, senescence or disease (Berne and Levy, 1988).
A continuous supply of oxygen and continuous disposal/removal of carbon dioxide take place during cellular metabolism. This is achieved through the respiratory system by ventilation of the lungs. Under adverse environmental conditions as in the case of industrial workers, the lung volumes and flow rates get reduced in comparison to control (Tables 4.2 and 4.3). Mukhtar and Mohan Rao (1996) were of the opinion that exposure to industrial atmosphere may lead to decrease in vital capacity. Schwartz et al. (1991) reported a negative correlation between pleural thickening and FVC. But Miller et al. (1994) reported that reduction in FVC is not necessarily indicative of restriction caused by deposition of dusts but as a result of trapping of air. A reduction in FEV₁ and flow rates is proportional to the reduction in FVC. Therefore, a reduction in FEV₁/FVC% results in obstruction of air passage (Miller et al., 1994).

At first, pollutants bring about the narrowing of the airways followed by alteration in the mechanical properties of the alveolar walls leading to measurable loss of elastic recoil of lung and finally to the overt development of irreversible air flow limitation. The hypothesis that a sequence of events in the development of chronic air flow obstruction has been proposed by Dosman (1975).

Depending upon the type of chemicals, or gaseous effluents that are released from each factory, the respiratory function status of the workers vary. This was observed in the present findings (Tables 4.2 and 4.3). A significant but varied difference was observed in lung volumes and flow rates i.e. in FVC, FEV₁ and FEV₁/FVC%, PEF, FEF₂₅-₇₅%, FEF₀₂-₁₂, FEF₂₅%, FEF₅₀%, FEF₇₅% among the workers of different industries in comparison to control.

In the present study, to be more specific, the respiratory condition of FACT workers were seen as the most affected followed by workers of Cotton Mill, Indian Rare Earths, and lastly Hindustan Newsprint Ltd., (Tables 4.2 and 4.3; Figures 4.1 and 4.2). This difference might be due to the chemicals either used or produced during the manufacture of their products.
Most of the industries studied either utilizes or produces chemicals which are toxic to human beings. These toxic substances remain suspended in the atmosphere of the factory, varying in concentration from industry to industry due to the difference in the manufacturing processes. Due to the constant contact, the noxious gases enter the blood stream of workers through alveoli along with other gases that are essential for life. The entry of these gases depends on (1) concentration and concentration gradient (atmosphere and blood) (2) diffusibility (3) solubility (4) partial pressure and (5) binding nature of the chemicals. All the above factors are responsible for the entry of the substances into the alveoli (Berne and Levy, 1988).

Once the gases mentioned above enter the blood, they bind with hemoglobin or other binding proteins. They are transported to the various parts of the body and exert their effect on respiratory centres. These chemical substances act directly or indirectly through chemoreceptors of the sino-aortic mechanism resulting in respiratory distress leading to reduction in lung volumes and flow rates (Berne and Levy, 1988). Transport of these chemicals depends on the (1) concentration of these substances (2) binding nature (i.e., the chemical substances get attached to the membranes and are likely to bring about structural changes in the membranes which are harmful to the normal function of respiration), (3) availability of molecular vehicles of transport, etc. Depending on the concentration and the nature of the substances present in the blood, they either induce or suppress respiration. It will be worth to note that in low concentration these toxic substances can stimulate or induce respiration through respiratory centres, but in higher concentration they suppress respiration (Berne and Levy, 1988). The respiratory centre sends impulses to the various lower centres of respiration including lungs and muscles of respiration in normal condition. Under the influence of these toxic substances it may fail to send impulses in time, resulting in respiratory distress.
The mechanism of action of these pollutants may be either localised or centralised. Localised in the sense that they block the ganglion of nerve endings of mechano receptors, such as irritant receptors and stretch receptors in the lungs and airways. It can also be interpreted, the localised action of these pollutants on nerve endings that are supplied to the respiratory muscles result in the weakness and loss of elasticity of the lungs (Berne and Levy, 1988).

Lung volume has an important effect on airway resistance (West, 1985). At large lung volumes elastic recoil of the lung is high, airways widen and resistance to air flow falls (Berne and Levy, 1988). Compression of the larger airways effectively limit the flow rates. Maximum flow rates will be determined partly by the elastic recoil forces of the lung which in turn will decrease as the lung volume becomes smaller. The main reason for this is that in the enlarged lung the bronchi and bronchioles are held open partially by elastic pull on their outside by lung structural elements. However, as the lung become smaller, these structures are relaxed so that the bronchi and bronchioles are collapsed more easily by external pressure (West, 1985).

The airways are elastic and can be compressed or distended under normal conditions. If the elastic recoil of the lung is reduced by the destruction of alveolar walls by pollutants, airways will be narrow and the resistance to air flow will increase (Berne and Levy, 1988). Elasticity and tone of the bronchial smooth muscle cells that encircle airways affect their calibre hence the air flow resistance is under the control of sympathetic and parasympathetic nerve fibres. Airway pollutants such as cigarette smoke, inhaled dust and noxious gases cause broncho constriction reflexly through stimulation of receptors in the trachea and large bronchi (West, 1985). The decline of respiratory function reported in this study might be due to the above reason (Tables 4.2 and 4.3; Figures 4.1 and 4.2).

The elastic properties of the lungs depend on the physical characteristics of the lung tissue and the surface tension of the film lining of the alveolar wall. With the deposition of pollutants on the alveolar wall and surrounding bronchioles
elastic properties of the lungs are affected resulting in the reduction of lung volumes as reported in the present study (Table 4.2 and Figure 4.1).

The decrease in flow rates in the present work (Table 4.3) may be due to the deposition of dust particles which might increase the inflammatory secretion of mucus which in turn results in increase of the airway resistance. This increase of the airway resistance and mucus secretion obstruct the breathing process. Results of the present study (Table 4.3) supports the earlier observation made by Astrand and Rodalh (1986) that the constriction of bronchi results in decreased flow rates. The increase in the resistance in the upper and lower airways results in the reduction of PEF, FEF$_{25-75}$%, FEF$_{75-85}$%, and FEF$_{0.2-1.2}$ (Govema et al., 1987). Similar observations were also made by Vijayan et al. (1993) and Zuskin et al. (1994).

Though these may the causes of respiratory dysfunction in general, it can vary from industry to industry depending upon the materials handled there. In fact this is what observed in the present study when the industrial workers were compared (Tables 4.4 and 4.5). The decrease in lung volumes and flow rates among FACT workers than all other industries in the present study is an indication of the effect of chemicals present in its environment. Pollutants such as Ammonia, Nitrogen dioxide (NO$_2$), Sulphur dioxide (SO$_2$) and dust in fertiliser industry have been reported to reduce the lung volumes such as FVC, FEV$_1$ and FEV$_1$/FVC% and expiratory flow rates of the workers (Joshi, 1994).

The case of Indian Rare Earths Industry was different where monozite, thorium and uranium and their decay products are involved in the day to day activities of the workers. The rare earth plant chemically treat the monozite obtained from the mineral plants for separation of thorium and other rare earth fractions. The acidic and alkaline effluents generated at different stages of chemical processing of monozite contain dissolved solids, suspended solids, phosphates, fluorides and low level of Ra-228. The gaseous effluents contain H$_2$S, HCl and Cl$_2$ in addition to thorium. Inhalation of these gases together with
radiation contributes towards the decline in respiratory function of IRE workers worse than Hindustan Newsprint Ltd. (HNL) workers (Tables 4.4 and 4.5).

In Hindustan Newsprint factory (HNL) and Cotton mill workers, the effect of particular matter of varied size is of great importance, though certain other gases like chlorine, chlorine dioxide, etc. and chemicals like sodium silicate, $\text{H}_2\text{O}_2$, $\text{H}_2\text{SO}_4$ are being utilised in HNL. The less adverse effects of pollution observed in HNL might be due to the clearance of chemical pollutants by mucus secreted for clearance of dust particles, less polluted environment of the factory and lesser exposure duration among these workers. However, it may be assumed that the more adverse effects observed among cotton mill workers than HNL and IRE workers might be due to high amount of minute particular matter in environment by virtue of the difference in the materials handled, and the high amount of mucus secreted to clear them.

Pollutants emitted by different industries remain suspended as aerosols which are trapped by the mucous of the respiratory tract and will get deposited in the small airways because of their density. That is due to sedimentation and tends to occur particularly where the flow velocity is suddenly reduced because of the enormous increase in combined air way cross section. It is also possible that the smaller particle reach the alveoli where some deposition occurs through diffusion. Normally, these particles are removed very efficiently. But in the case of dusty environment mucus secretion may be increased so much that cough and expectoration assist in clearance (West, 1985) depending upon the pollutants from industries. The difference of decrease in respiratory parameters among the industrial workers of the above environment in the present study may be due to the difference in the nature of inhaled gases/chemicals/particulate matter produced/handled (Tables 4.4 and 4.5).

Tables 4.6–4.15 indicate a significant negative correlation between duration of exposure and respiratory parameters. It is clear from the above that as the
duration of exposure of a worker to industrial environment is increased, the lung volumes and flow rates decrease.

Hindustan Newsprint factory (HNL) workers were exposed to various noxious gases and chemicals used in the production of paper and pulp. Of these, SO$_2$ is the most potent allergen (Goodman et al., 1985). SO$_2$ paralyses the cilia and also change the character of mucus. Due to the stimulation followed in irritant receptors, bronchial constriction of the airway occurs as a result of parasympathetic innervation (Goodman et al., 1985). Acute exposure to chlorine gas in the above industry may also cause an allergic response characterised by increased formation of mucous from the upper respiratory tract up to the alveoli, leading to the constriction of bronchioles, by the direct stimulation of bronchioles (Beach et al., 1969, Waldron, 1985). Exposure to dust of Eucalyptus and Bamboo can cause increased secretion of mucus, and it could be attributed towards chronic air flow obstruction. Similarly, upper and lower respiratory tract were excited by the exposure to paper dust (Ericsson et al., 1988).

The decrease in lung volumes in HNL workers (i.e., VC, FVC, FEV$_1$, FEV$_{0.5}$) with increase in duration of exposure to a variety of gases viz., SO$_2$ and Cl and paper and wood dust indicate that elastic recoil forces of the lung is decreased as a result of deposition of these gases and particles leading to the restriction of the airways (Table 4.6). Ericsson et al. (1988) was of the opinion that long term heavy exposure to dust somewhat decreases the vital capacity (VC). Further, heavy exposure to paper dust for at least 10 years had a reduction of FVC and FEV$_1$ compared to men with low exposure (Ericsson et al., 1988). This is in agreement with present findings. But, impairment of pulmonary functions in persons exposed to paper dust for a long period of time has not been observed by Chan-Yeung et al. (1980) and Ferris et al. (1976, 1979). The exposure to “paper dust” was probably much lower in those studies, which may be one explanation of the different results.

The decrease in FEF$_{25-75\%}$, FEF$_{75-85\%}$, FEF$_{0.2-1.2}$, FEF$_{50\%}$ and FEF$_{75\%}$ in the present study can be explained on the basis of increased resistance to air in the
large and small airways which occur as a result of bronchoconstriction due to Cl and SO₂ (Table 4.7). Kennedy et al. (1991) explained this as a result of inflammatory reactions occurring in small airways in response to chlorine gas. The indication of airflow obstruction in workers exposed to wood dust was also reported earlier (Vedal et al., 1986; Rastogi, et al., 1989 and Shamssain, 1992). The present work can be further supported by Sahle et al. (1990) that exposure to paper dust was found to cause an obstructive airway reaction resulting in the decline of flow rates.

A negative and significant reduction of lung volume (i.e., VC, FVC, FEV₁, FEV₀.₅) with increase in duration of exposure was observed among IRE workers (Table 4.8). Here the workers are subjected to inhalation of short lived gaseous thorium and its decay materials. The α-particles emitted by radon and the decaying thorium are carcinogenic agents and can damage bronchial epithelium and alveoli of lungs. Tumours of the bronchi were observed among the uranium, silver and nickel miners, where there was very high level of radioactivity (Waldron, 1985). Significant decrease in lung volume in these workers indicated a non-obstructive, but a pure restrictive ventilatory defect of pulmonary origin which is usually due to decrease in the distensibility of the lung, i.e., the lung compliance is reduced. This is a diminution in ventilatory capacity which is not due to limitation of airflow but due to changes within the lung or chest wall including weakness of respiratory muscles (Cotes, 1979).

The acidic and alkaline effluents generated at different stages of chemical processing of monazite, containing dissolved solids, suspended solids, phosphates, fluorides and low levels of Ra₂₂₈, might damage the respiratory epithelium and alveolar capillaries. This might result in decrease in the elastic recoil forces of the lung and its volume (Table 4.8). The efficiency of muco-ciliary apparatus fails to clear these toxic substances which reduces the lung volume by decreasing elastic resistance (Goodman et al., 1985).
The flow rates were not found to be significantly declined due to increased exposure to chemical pollutants along with radiation (Table 4.9) in IRE workers. This observation is unparallel, because there have not been any comparable studies of respiratory function among radiation exposed workers.

Workers employed in FACT are always exposed to a large variety of gases viz., CO₂, CO, SO₂, chlorine, formaldehyde, ammonia, various oxides of nitrogen, H₂S, sulphuric acid and particulate matter of varying size. The acute effect of inhaled dust is dependent on its size, distribution, nature and dose (Ganguli, 1992). It causes bronchitis with dyspnea, pulmonary oedema, cough with a frothy sputum, etc. (Waldron, 1985). Sulphuric acid increases airway resistance which affects the flow rate (Goodman et al., 1985). Nitrous fumes cause sudden bronchospasm and death from respiratory failure at a concentration of 100-500 ppm. Irritation to upper respiratory tract along with severe alveolar oedema occurs as a result of these fumes (Waldron, 1985). These noxious gases, along with dust particles get deposited in the surfactant layer of alveoli, decrease the elastic recoil forces of the lung which in turn increases the airway resistance. This results in the decrease in lung volumes (Tables 4.10 and 4.12) along with decrease in flow rates of these workers (Tables 4.11 and 4.13).

Polyvinylchloride (PVC) workers exposed to HCl, CO, CO₂ and benzene show acute and chronic obstructive ventilatory changes (Baser et al., 1985). But Huang et al. (1991) reported that short term exposure to low concentration of SO₂ and NOₓ did not affect lung function which opposes the findings of Kolarzk et al. (1990). The present work is in agreement with the Kolarzk's findings that exposure to SO₂ and NOₓ gases causes reduction in FEV₁ and FEF₂₅-₇₅%, (Tables 4.10–4.13).

Present study conforms to the findings of Gardiner et al. (1993) that long term exposure to dust and noxious gases can cause damage to parenchyma tissue of lung with a characteristic change in the transfer of gases and elastic recoil resulting in pulmonary fibrosis.
Workers exposed to cotton dust also experience breathing disorder "Byssinosis"—a chronic respiratory disease characterised by the tightness of chest and or shortness of breath (Parikh et al., 1990). The causative agent for abnormal pulmonary function is present in the bracts of the cotton bolls rather than in the cotton linters and seeds. The acute effect upon the lung airways by inhalation of cotton dust is constriction of small airways (Cotes, 1979).

A decline in lung volume, i.e., FEV₁/FVC% and flow rates especially FEF₂₅%, FEF₅₀%, FEF₇₅%, and PEF, seen in cotton mill workers is indicative of obstruction in small and large airways (Tables 4.14 and 4.15). Broncho constriction may result in decreased flow rates in cotton workers due to high airway resistances by the release of mediators from the mast cells lying beneath the airway epithelium (Cotes, 1979). Obstruction in small airways owing to exposure to cotton dust can be detected by mid and end expiratory flow rates (Hayes et al., 1994). Reduced FEV₁/FVC% among cotton mill workers in the present study may be due to an obstructive type of ventilatory defect associated with premature closure of airways as suggested by Cotes (1979).

The epithelial lining the airway acts as a barrier between noxious gases in the airstream and the cellular components. These noxious gases, dusts or fumes can disrupt the epithelial barrier resulting in the constriction of the airway (Berne and Levy, 1988) by obstruction and restriction in the alveolar walls of the lung. The decrease in lung function of industrial workers with increase in exposure duration (Tables 4.6-4.15) could be explained on the basis of this concept. There is evidence from epidemiological studies that exposure to dust over a periods of years causes permanent impairment of lung function associated with chronic obstructive pulmonary disease (COPD) (Cotes, 1979).

Respiratory function may be further complicated by additional exposure to other injurious agents like cigarette smoke which results in obstructive lung diseases (Sue et al., 1985). The action of the cigarette smoke is attributed to the nicotine present in it. During respiration nicotine present in the cigarette smoke,
compete with other toxic gases released from industries for the receptor site in the respiratory tract. This will result in receptor-nicotine complex, leaving behind all other gases/noxious gases. Another important aspect of nicotine is its muscarinic effect. It is worth to note at this juncture, that the receptors present in the smooth muscles of the airways i.e. α and β receptors help in either constriction or dilation of the bronchioles. That means β-adrenergic receptors in the smooth muscle cell membrane dilate the bronchioles and α receptors in the cell membrane constrict the airways. It is probable that nicotine induce the action of α-receptor and thereby induce airway resistance (Berne and Levy, 1989).

The damaging effect of cigarette smoke on the entire respiratory tract probably produces a vicious cycle leading to serious disruption of defence mechanism by changing proteolytic and anteproteolytic forces in the lung and develop obstructive lung disease in smokers (Sherman, 1992). This has significant detrimental effect on both structure and function of the lung (Weiss, 1984, Sherman, 1992, Özdener et al., 1995).

Weibel (1972) was of the opinion that nicotine present in the cigarette smoke causes abolition of phagocytic activity of the macrophages present in the respiratory tract especially alveoli. In normal conditions, only very few particles are likely to reach the alveoli which are practically sterile and possess amoeboid mobility and phagocytic activity and take dust particles and bacteria. These dust particles and bacteria are phagocytosed into digestive tract via the airways or via blood or lymphatics. This phagocytic capacity and normal clearance mechanism can be diminished by the influence of the smoking (Astrand and Rodalh, 1986). Once the normal structure of the airway is damaged due to smoking narrowing of the small airways occur leading to the increased airway resistance which result in respiratory distress (Comroe, 1966b). The deposition of nicotine particles in the alveolar wall increases its thickness and results in muscular hypertrophy associated with a decrease in maximal mid expiratory flow (MMEF) and elastic recoil pressure (Cosio et al., 1977).
Tables 4.16–4.25 shows the lung volumes and flow rates of control and non-smoker and smoker workers of various industries. There is significant decrease in lung volumes and flow rates in non-smokers and smokers of the industries in comparison to control. The difference between smokers and non-smokers of various industries was insignificant though the smokers deviate differently from the control.

Significant difference in FVC, PEF, FEF25–75%, FEF50%, and FEF75% among smoker workers of HNL from that of control indicate that both cigarette smoke and exposure to pollutants in industry contribute towards reduction in lung volumes and flow rates (Tables 4.16 and 4.17). But among non-smoker workers reduction of FVC, FEV1, PEF and FEF75% suggests that even only the exposure to industrial environment have influenced large airway function which leads to overall mild obstruction.

Smoking along with radiation exposure among IRE workers were observed to cause decrease in FEV1/FVC%, PEF, FEF25–75%, FEF2, PEF, FEF25%, FEF50%, and FEF75% (Tables 4.18 and 4.19). These findings suggest reduction in upper and small airway function. This is an agreement with Joshi (1994). Deiels et al. (1987) suggested that adverse effects of long term exposure to oxidant pollution may occur mainly in the large airways in both smokers and nonsmokers. This supports the present observation of significant reduction in PEF, FEF25–75%, FEF25%, FEF50%, and FEF75% among nonsmokers of IRE as well (Tables 4.18 and 4.19).

Among FACT workers (CD and UD divisions) significant reduction in lung volume and flow rates of smoker and non-smoker workers establish the fact that chronic industrial exposure along with or without smoking is seen associated with small and large airway obstruction and restrictive condition of lung (Tables 4.20–4.23). Similar was the observation among cotton mill workers except FEV1 which was not found to be influenced either by cotton dust alone or by cigarette smoke and cotton dust (Table 4.24 and 4.25). This is probably due to hypertrophy of mucosal cells due to irritation by cotton dust and smoke which
results in increased secretion of mucus and formation of mucosal plugs causing obstruction (Culver and Butler, 1985). Beck et al. (1984) suggested that cotton dust and smoking were additive and equally important. They also reported that both affect different regions of the lung. Smoking of cigarettes has been shown to affect small airways leading to airway obstruction (Bouhuys, 1974; Dosman, et al., 1975) and cotton dust may be affecting the large airways as suggested by the FVC and FEV_{1} results and similar findings for PEFR (Beck et al., 1984). Decline in FEV_{1} leading to the development of chronic air flow limitation by cigarette smoking have been earlier reported (Peat et al., 1990). The present study does not support the observation of Beck et al. (1984) and Peat et al. (1990) that cigarette smoking and atmospheric pollution have additive effect on lung function.

Reduction in FVC in all the industrial workers irrespective of smoking habit indicates that severe restrictive condition occurs among them. But Joshi (1994) was of opinion that reduction in FVC is largely due to the effect of smoking rather than exposure to industrial environment. Kreiss et al. (1989) observed that smoking had a modifying effect on the pattern of respiratory function impairment. In the present study, no significant difference is observed between smokers and nonsmokers, though more deterioration is observed in smokers.

Under different exposure groups, smoker and non-smoker workers of different factories show significant changes in both lung volumes and flow rates (Tables 4.26–4.31). The difference in the lung function was due to the differences in the degree of damage to the respiratory system by exogenous agent.

It is evident from the present observation, that, no significant difference in the lung volumes and flow rates between non-smokers and smokers of industrial workers does occur, in any three exposure groups though the non-smokers had slightly better values on most counts. Pulmonary function study in urea plant workers of a fertiliser industry suggests that only exposure to plant environment probably does not have any influence on the large airway function, but association of smoking with chronic exposure to polluted environment leads to overall mild
obstruction of large airways (Joshi, 1994). The present study in which both of the categories had low/significantly low levels of lung volume and flow rates when compared to control (Tables 4.16–4.25), is not in agreement with Joshi (1994). Smoking together with the inhalation of noxious gases from the different industrial plants brings about a restrictive and obstructive disturbance of ventilation (Tables 4.16–4.25, 4.26–4.31) resulting in decline of lung volumes and flow rates.

Dutton et al. (1993) reported that the industrial exposure contribute only weakly and inconsistently to the well documented reduction in lung function that occurs from smoking and there are no sufficient exposure related reduction in mean pulmonary function in smokers and nonsmokers. Korn et al. (1987) did pulmonary function tests and reported no evidence that smoking modified the effect of occupational exposure on pulmonary function. That means some industrial dust exposure may be associated with change in pulmonary function. Smoking accentuates these changes. Fishwick et al. (1996) was of the opinion that smoking and dust exposure interact to cause loss of lung function in cotton textile workers. But Graham et al. (1984) reported no significant interaction between working in the potash industry and cigarette smoking which lower pulmonary function.

Cigarette smoking and noxious gases together potentiate each other which is responsible for chronic obstructive lung disease (Mikaelsson et al., 1982). Apart from the pollutants present in chemical factories, smoking produce restrictive and obstructive disturbances of ventilation (Lewin-Kowalik et al., 1994). Several reports are in agreement with the fact that decrease in pulmonary function is greater in smokers than in non-smokers (Manfreda, 1982; Samet et al., 1984; Attfield, 1985; Governa, et al., 1987) while Gupta et al. (1991) fails to find any link between respiratory impairment and duration of exposure with smoking among industrial workers.

It is clear from the present study that even though the mean lung volumes and flow rates of smoker and non-smoker industrial workers were different, but insignificant, (Tables 4.16–4.25) the lowest levels being observed among smoker
industrial workers. It is interesting to note that the average FVC and FEV1 of HNL, IRE and cotton mill smoker workers are better than those values of non-smoker workers. But, the above values show no difference in the FACT-UD and FACT-CD workers when grouped as smokers and non-smokers. The factories HNL and IRE are less polluted than FACT divisions as evidenced from better lung function averages (Tables 4.2 and 4.3 and Figures 4.1 and 4.2). This could be a pollution-countering mechanism shown by cigarette smoke efficient only in less polluted areas as well as lower duration of exposure. With increase in pollution smoker-non-smoker difference disappears. It may be assumed that it is apparently analogous with the status of smokers with more haemoglobin than nonsmokers.

The present findings indicate that lung volumes and flow rates are affected by smoking and industrial exposure (Tables 4.16–4.25) while Kamat et al. (1982) and Chattopadhyay et al. (1992) could find its effect only in flow rates of smokers. Marine et al. (1988) reported that the workers exposed to various dusts provide evidence that the decline in pulmonary function is greater in smokers than non-smokers. Miller et al. (1994) observed a decrease in FVC and FEV1/FVC% which was influenced by cigarette smoking. Earlier, Zuzkin et al. (1969) observed no smoking-effect on acute ventilatory function. The difference in FEV1 between smoker and non-smoker is very small (Merchant et al., 1973; Parikh et al., 1990).

In the Surgeon Generals Report of 1985 from US Public Health Service on Cancer and Chronic Lung Disease in the Work Place, it is mentioned that interaction between smoking and the occupational exposure is complex and needs further development of techniques to study these interactions. The present study also highlights the above plea. The evidence of interaction between these two factors has not been consistent. It is demonstrated in some studies and not in others (Higgins, 1973).

Age is also an important determinant of respiratory function. The elastic properties of the lung depends on the physical characteristics of lung tissue and the surface tension of the film lining the alveolar walls. The elasticity of the lung tissue
arises from the elastin and collagen fibres in the alveolar walls, surrounding the bronchioles and the pulmonary capillaries. The elastin fibres can be stretched to double their resting length. The collagen fibres resist stretch and act mainly to limit further expansion at large lung volumes. The lung expands during breathing through an unfolding and geometric rearrangement of fibres in the alveolar walls. Changes in the arrangement and physico chemical properties of the elastin and collagen fibres in the lung account for the increasing dispensability of the lung with advancing age (Berne and Levy, 1988) and it would be enhanced by industrial pollutants.

With advance in age, the chest wall stiffens because of structural changes in the bones and joints of the rib cage. The compliance in lung and chest wall changes in opposite directions, and therefore the total lung capacity as functional residual capacity changes little with senescence (West, 1985). The strength of the respiratory muscle decreases in elders along with the internal surface area of the lung. This in turn diminishes lung volume and forced expiratory flow rate (FEF) of the lung. The alveoli becomes wider and more shallow. The dead space enlarges, and the diffusing capacity decreases. The closing volume of airways increases progressively with advance in age reflecting a greater unevenness in the distribution of ventilation. Hence, the pulmonary performance declines with advance in age.

In the present study, lung volume and flow rates of workers decreased with advance in age irrespective of the industries and smoking. (Tables 4.32a&b–4.41a&b). Since all the dynamic ventilatory functions depend upon the compliance of the thorax-lung system, airway resistance and muscular strength rather than the absolute anatomical lung volumes, the deterioration in the lung function with advancing age, is mainly caused by change in these factors (Jain and Ramiah, 1969). The age related loss in respiratory function is due to decrease in lung compliance, increase in airway resistance and reduction in the strength of respiratory muscles associated with changes in the elastic recoil of the lung and increase in the stiffness of the thoracic cage (Cotes, 1975). Cotes (1979) reported
that ageing together with pollutants and smoking aggregates the restriction on respiration leading to limitation in inspiration and expiration. It is clear from the present observation that effect of ageing was aggravated by industrial pollutants (Tables 4.32a&b-4.41a&b) with no significant effect of smoking added to industrial pollutants. The findings of the present study are similar to those of Bass (1973), Udwadia et al. (1986) and Vijayan et al. (1993).

In the present observation lung volumes such as FVC and FEV1 decreased only after the age of 45 in IRE and cotton mill non-smoker workers. Similar was the observation in control group, while the same trend was observed in nonsmokers of the cotton mill and smoker and non-smoker workers of IRE with respect to flow rates. It can be explained as a consequence of impairment of lung function which expected to originate around 40, but become more pronounced after 50 years of age as suggested by Jain and Gupta (1967). But the smokers show deterioration (Tables 4.40a and 4.41a) of lung functions earlier to 40 years of age. In the case of lung volumes the peak functional status is in the 35-45 age group in control and non-smoker workers whereas the peak is in <35 age group in smoker workers (Table 4.40a). But the peak of flow rates is in <35 age group in control as well as smoker workers and it is in 35-45 age group in non-smoker workers (Table 4.41a). However, this interesting observation is not of much significance since always the lung functions are significantly lower in non-smoker and smoker workers groups than the control group.

Significant difference in the present study among workers of different industries suggest that respiratory function changes become more pronounced after 45 years. In the present study it is observed that the combined effect of industrial exposure, smoking and ageing had caused impairment of respiratory function of industrial workers.

The outcome of the present study indicated major loss of small and large airway function in chronically exposed workers of different industries. Therefore, we recommend the following measures, implementation of which may help in early
detection as well as in prevention of further loss of pulmonary function in these workers.

1. Periodical pulmonary function assessment, especially tests concerned with early detection of large and small airway obstruction in these workers after 20 years of their working in industries.

2. Industrial workers may be motivated to become and remain non-smokers.

3. Use of appropriate safety measures like wearing masks may be enforced.

4. Workers with severe impairment of lung function may be moved away from the immediate vicinity of polluting areas as far as possible.