chapter 5

discussion
DISCUSSION

Functional status of cardiac patients using lung function test was assessed in 133 cases of different cardiac ailments. The biometric characteristics like race, sex, height and weight of the patients were noted. This was done so, as predicted values of various respiratory parameters for different cardiac cases can only be obtained if such information is available. Information related to patients with reference to residential area occupation, smoking/nonsmoking status and any obvious respiratory disorder suffering from was also considered to be important in view of the possibility that these factors may also interfere with pulmonary function. Patients with known history of any intrinsic respiratory disorder, in residential areas prone to respiratory hazards and heavy smoking habits were summarily excluded to avoid likely complications in precise assessment of functional status of the patients.

Cases with known cardiac disorder were categorised into RHD, IHD and CHD. They were further classified into four functional groups. The grouping was based on the guidelines given by the NYHA (NYHA, 1945). This was done so as some guidelines had to be used as a basis for some preliminary classification. Moreover, as literature indicates this criteria seems to be in wide use. (Aber et al., 1963; Palmer et al., 1963; Patterson et al., 1972; Selzer and Cohn, 1972; Kulkarni, 1977; Franciosa, et al., 1979).

The lung function test was carried out in sitting position. The sitting position was maintained throughout the investigation since the pulmonary performance depends upon the body position (Comroe, 1962). Morgan ELF machine used for this purpose has specifications which far exceed the expectations recommended by the
American Thoracic Society Statement made in the Snowbird Workshop on Standardisation of the Spirometry (Gardner, 1979). The widely acclaimed Epson HX-20 microcomputer which has been listed and electronically enhanced by specially designed solid state circuitry provides the power and versatility to this machine. The precisely engineered robust flow tube made of high quality anodised aluminium which can easily be detached and sterilised ensures the highest standards of surgical hygiene without any risk of corrosion or of damage to the sophisticated electronic sensor. These important features in flowmeter design, in most comparable products have been overlooked. The software incorporated in the machine uses not only the anthropomorphic factors (age, height, weight) but also ethnicity and gender in predicting the normal values of each patient. For deriving the values of residual volume, airway resistance and index of lung elasticity from flow volume variable alone which are of importance in the pulmonary physiology, suitable provisions are available in Morgan ELF. A comprehensive diagnostic interpretation of respiratory disorders reduces the manual interpretation of data obtained. Finally, the compactness and portability of Morgan ELF makes it ideally suited to lung function evaluation of the patient's bedside or wherever the need arises. In order to ensure a maximum benefit of the present study lung function test was also carried out using another portable lung function machine 'Spiroscreen' which has been specially devised to provide only expiratory maneuver. Lung function data obtained from both the machines were then classified into different groups according to the cardiac disorders viz. RHD, IHD, CHD and the functional status of patients in each cardiac disorder in terms as per NYHA classification system (1945).
The details related to various biometric characteristics and pulmonary parameters in each cardiac disease and group for each patient was tabulated. The mean, standard deviation and range of each parameter in each class for different types of cardiac disorders have been calculated and presented. Such a statistical treatment seemed necessary in view of the complex and interrelated nature of the parameters investigated. In addition, the computation involved also helps summarise the obtained data in a meaningful order.

Age factor:

The lowest value of the age in CHD patients (15.76) as compared to RHD (31.47) and IHD (34.80) was observed since the basic cause of these disorders is arrested or defective prenatal development of the heart and great vessels. The development also gets hampered because of the interaction between multifactorial genetic and environmental systems at the time of gestation (Nora and Nora, 1987; Kirby and Waldo, 1990). Higher mean value observed in IHD indicates the prevalence of this disorder in aged subjects. The pathological lesions and clinical forms of coronary heart disease occur predominantly after the age of forty or with increasing frequency as the age is advanced (Narkar, 1970). Observations related to age in RHD patients are similar to that of Padmavati (1958, 1962) and Vakil (1962). These authors reported the peak age incidence of this disorder in third or forth decade of the life.

Sex factor:

Data assimilated by us suggests that more females (58.21%) seem to be affected by RHD than the males (41.79%). These findings are also in good agreement with those of Sepha et al. (1965) and Berry (1973). Berry (1973) asserted that in women,
the mitral stenosis not only is more common but appears earlier; is more severe and it is more likely to be complicated by pulmonary hypertension.

The incidence of IHD in males was noted to be more (65%) than the females (35%). This predominance in males observed was also in line with the previous findings (Joshi et al., 1965; Mathur, 1961; Narkar, 1970). Various explanations have been provided to account for this prevalence. Males are said to be heavy smokers than their opposite sex: are also likely to get more exposed to the competitive stress of industrialised and urban life (Davidson, 1984). However, this explanation does not seem to stand on any scientific grounds. Endogeneous factors such as lesser amount of beta lipoproteins, higher alpha proteins, predominance of esterogen were considered as probable factors for lesser incidence of IHD in females (Farmer and Gotto, 1992).

The incidence of CHD was observed to be of higher order in males (57.69%) than in females (43.31%), similar to the findings reported by Joshi et al. (1965) and Jnathikia (1966). This could probably be explained as follows: The coarctation (3:1), anomalies of the semilunar cusps (5:1), defect of the aortic septum and transposition of great vessels (2:1 or 3:1) are more frequent among males while only patience of the ductus arteriosus occurs more commonly in females (2:1 or 3:1) (Friedberg, 1956).

Distribution of cardiac disorders:

The present study indicates that the RHD is a major and commonest form of cardiovascular disease. Amongst the 133 cases studied, RHD was noted in 67 cases (50.38%), followed by IHD in 40 cases (30.07%) and CHD was observed in 26 cases (19.50%). The prevalence of RHD amongst various cardiac disorders has
been reported from various parts of India (Devichand, 1959; Vakil, 1962; Joshi et al., 1965; Reddy and Janbandhu, 1968; Dasgupta et al., 1976); from Marathwada region (Narkar, 1970; Kulkarni, 1977). Our observations confirm higher percentage of RHD in population reporting at the hospital. Banerjea (1965) and Padamavati (1962) pointed out the low living standards and poor economic conditions in our country (leading to the nutritional deficiency) predispose to the development of streptococci infection and consequent attack of rheumatic fever.

The 30.07% incidence of the second important cardiac complication, IHD, observed by us corroborates with the earlier reports of 28.4% (Vakil, 1962); 30.3% (Mathur and Sapru, 1963) and 26.8% and 34.3% (Jhatkia, 1966). Gokhale (1981) stated that the IHD in India has been on a gradual rise during the past 30 years—a trend prevailing in the Western World. Other reports (Mathur, 1961; Jhatkia, 1966; Raghawan, 1962) also pointed out an alarming increase in IHD incidence during the last few decades. Mathur (1961) further has indicated the high incidence of IHD in the higher socioeconomic strata in comparison with lower classes. General awareness of public and medical practitioners to this malady is also considered as an important factor in the rise in the number of reported incidence of IHD (Raghawan, 1962; Mathur and Sapru, 1963). Other explanations afforded for the increase in IHD cases include dietary and smoking habits, stress and strain (Friedberg, 1956).

Congenital heart disease accounted by a 19.55% of the total cardiac cases studied. This is also well comparable to the earlier findings (Vakil, 1949; Malhotra, 1950; Padmavati, 1955).

Analysis of all these cases in the terms of the functional status of the patient reveals a striking, higher percentage in
class II category. This could probably be due to late reporting and admission of the concerned case in the hospital or recovery/improvement in the severity of the disorder under treatment. The lowest percentage observed in class IV strongly relates non reporting on account of lack of operation facilities at the hospital. Patients seem to prefer modern and well equipped cardiac centres.

Cardiothoracic Ratio:

The cardiothoracic ratio concerned with cardiac dilation and hypertrophy (Chen, 1987) as evidence of myocardial, valvular or circulatory disease was determined from the chest X-ray of the patient. The ratio helps indicate the extent of cardiac enlargement (Chen, 1987); the severity of the cardiac disease could be gauged on such a basis (Friedberg, 1956).

Measurements related to CT ratio indicated the mean values above 50% in all the groups of the three types of cardiac disorders investigated. Such higher values tend to indicate a significant cardiac enlargement (Chen, 1987). In case of RHD the maximum value noted was 57.05% in class II patients followed by 56.84% in class IV, 54.86% in class I and 52.75% in class III. The enlargement of the heart thus does not seem to depend on functional status of the patient. Such independence could be due to variations in terms of differential pattern of enlargement of individual heart chamber in various forms of RHD. The differential enlargement reported is of following nature: The enlargement of left atrium in RHD is usually associated with severe mitral insufficiency and (Priest et al., 1962; VanHouten et al., 1974) occasionally with mitral stenosis (Braunwald, 1992). Evidence for left ventricular enlargement also greatly favours predominant mitral regurgitation in combined valvular disease (Paraskos, 1987).
1987), although the evidence may be subtle, specially in the small balanced lesion (Janton et al., 1954; Aravanis, 1965). The variable degree of left atrium enlargement is also independent of the degree of mitral stenosis (Probst et al., 1973) or insufficiency appears to be related to intrinsic differences in the atrial muscles (Friedberg, 1956). The left ventricle is dilated and hypertrophied in case of predominant mitral insufficiency or when prolonged mitral insufficiency precedes stenosis. In hearts with predominant mitral stenosis the left ventricle may be of normal size, but with pronounced and long standing mitral stenosis the left ventricle may become smaller and even atrophic (Friedberg, 1956). The decreased size of the chamber is particularly confined to the posterior half or the so-called inflow tract (Kirch, 1929). An associated aortic valvular disease may produce enlargement of the left ventricle. Several observers repeatedly demonstrated cardiac hypertrophy following the aortic insufficiency (Klatle et al., 1962; Kennedy et al., 1968; Spagnuolo et al., 1971; Alpert, 1987). Bergeron and co-workers (1954) reported a proportional increase in heart weight with the degree of aortic stenosis. The right ventricle usually enlarges and becomes hypertrophied especially after the development of pulmonary hypertension due to left atrial failure; the right atrium also becomes dilated with progressive right heart failure (Friedberg, 1956). Such an extent of differential variation probably is responsible for the lack of any specific order in relation to the functional class of these patients. Variability in the relation between heart and chest diameter of the patient could also be a factor for such inconsistency (Comeau and White, 1942).

In case of IHD patients a slight increase in CT ratio with
reference to the functional status of the patients was observed. Thus, the values obtained are class I - 52.97, class II - 54.02, class III - 54.92, class IV - 55.02. Cardiac hypertrophy in anaemic condition (Datta and Silver, 1975) or hypertrophy on account of an anomalous coronary artery arising from a pulmonary artery and supplying the heart with unoxegenated blood (Bassis and Sheinkoff, 1955), coronary occlusion and thrombosis (Tennat and Wiggers, 1935; Baxter et al., 1978) could probably explain this trend.

In case of CHD patients the mean value of CT ratio is seen to increase with the functional class of the patients. The findings indicate the value of 54.80% in class I, 55.62% in class II, 56.25% in class III and 59.35% in class IV. Right ventricular hypertrophy (Friedman, 1992), pulmonary hypertension (Edwards, 1966 in CHD, distension of left atrium (Stanger et al. 1969), cardiac enlargement due to a large left to right shunt (Rivkin et al, 1957; Edwards and Burchell, 1960), ventricular septal defect or large patent ductus arteriosus (Bryk and Brooklyn, 1965) could be probable causative factors for the trend observed.

Lung function in cardiac disorders:

Because of the close cooperation and interaction between the cardiovascular and pulmonary system the abnormal functioning of one system often affects the other (Mcfadden and Ingram, 1980). Any primary cardiac disorder may cause significant change in overall lung function in the absence of intrinsic pulmonary disease (Tancredi, 1981). In view of the differential manifestation of pulmonary mechanics in various cardiac disorders, the pulmonary effect in specific disorders viz. RHD, IHD and CHD are discussed separately.
Lung function in rheumatic heart diseases:

The progressive decline in FVC (96.83-17.80), FIVC (99.08-25.80), TLC (102.63-41.20), FRC (110.33-62.80), FEV₁ (104.41-21.40), FEF₂₅ (101.33-28.60), FEF₅₀ (107.91-30.40), FEF₇₅ (150.06-47.00), PEFR (69.83-26.20) and PIFR (76.75-24.20), and considerable increase in Raw (70.66-298.20) from class I to class IV in RHD patients confirm that the natural history of RHD involves progressive changes in pulmonary function. Our findings, in view of the pathological changes described in the lung (Parker and Weiss, 1936) are quite understandable. The pathological changes in the lung are often described under the heading of 'brown induration of the lung' (Bates et al. 1971) which state two striking gross features viz. hemosiderosis and stiff lung (Bates et al., 1971). The hemosideration may be due to intrapulmonary haemorrhage, probably venous in origin (Wood, 1954; Haris and Heath, 1962). Hemosideration seems to be predominant in the upper zones of the lung and may be slight or even absent in the inferior lingular or basal part of the lower lobe even in advanced cases (Gough, 1960). In hemosiderosis the alveolar lining cells get swollen and thickened (Hicks, 1953; Soulie et al., 1953; Harris and Heath, 1962). Schulz and O'Conner (1962) measured these changes accurately by electron microscopy and concluded that in mitral stenosis the basement membrane varies from 2000 Å to 5000 Å in width compared with a value of less than 1600 Å for this structure in the normal human lung. Heath and Hicken (1960) also reported abnormal changes in pulmonary lymphatics. The pulmonary lymphatic was noticed to be tortuous and dilated, the interlobular septa were thickened and edematous, gave rise to the appearance of 'Kerely B lines' (Gough, 1955). This was confirmed by radiological
observations (Jordan et al., 1966). Such changes may lead to pulmonary edema, the organisation of which may result in fibrosis (Bates et al., 1971). In the presence of fibrosis of alveolar septae, various changes in pulmonary vessels were also reported (Harrison, 1958; Harrison, 1960; Harris and Heath, 1962). These include the dilation of main and lobar arteries, severe atherosclerosis, smaller elastic arteries may be normal or slightly dilated, thickening of the media and intimal proliferation of the pulmonary arterioles and small muscular arteries, fibronoid necrosis. Several authors have also noted severe vascular changes even when the pulmonary artery pressure was not greatly elevated (See, Mudd and Wyatt, 1965). Pulmonary venules and small veins showed intimal fibrosis (Harrison, 1958; 1960) and some medial thickening (Harris and Heath, 1962). Varices of the bronchial arteries have also been reported (Ferguson et al., 1944; Bland and Sweet, 1949). It has been suggested that the bronchial artery that anastomoses may be responsible for hemoptysis and further, that may be one of the causes of pulmonary hypertension (Broustet et al., 1957).

As early as 1891 Von Basch showed that with pulmonary vascular congestion in the excised lung of animals, two dominant changes occurred simultaneously. First, there was a reduction in tidal volume for a fixed volume cycling pressure and secondly an increase of the gas volume contained in the lungs. Reduction in the tidal volume has been interpreted as resulting from an increased stiffness of the lungs. This was later confirmed by Christie and Meakins (1934) in patients with heart failure. Frank (1959) studied the recoil force of excised cat's lung with acute pulmonary vascular congestion. He showed that there was an intimate relationship between the volume of the lung and
transpulmonary pressure.

There could be an alternate explanation to the variations observed in respiratory parameters with respect to the functional status of the cardiac disorder- RHD. These changes besides the pathological observations, can also be explained on the basis of increased pulmonary venous pressure due to elevated left ventricular end diastolic and/or left atrial pressures (Marshall et al., 1954; Frank et al., 1957; White et al., 1958; Saxton et al., 1965; Hughes et al., 1969; Luepker et al., 1971; Wood et al., 1971; James et al., 1971; Gump et al., 1972; Pain et al., 1972; Giuntini et al., 1974; Glauser et al., 1974; Collins et al., 1975; Dawson et al., 1976). Patients with chronically elevated pulmonary venous pressure can be characterised as having restrictive ventilatory defects i.e. loss of lung volumes (Mcfadden and Ingram, 1984). Decrease in various capacities like FVC, FIVC, FRC and TLC as observed in the present work with the functional status of the patient suggest a definite relationship between pulmonary restriction and functional class of the patient.

Vital capacity has been noted to diminish in obstructive lung diseases. The decreased TLC precisely therefore is of prime importance in understanding such a restrictive pattern in RHD (Hercules et al., 1979). The reduction in TLC may result from replacement of air in the lung with either blood or interstitial fluid. These changes affect the elastic properties of the pulmonary parenchyma (Hercules et al., 1979). As pulmonary venous hypertension and hence interstitial and alveolar wall edema advances the lung becomes less compliant (Wade, 1977). As this process worsens with increased severity of the disease air trapping could occur because of earlier than normal closer of
dependent airways. Residual volume then may increase as TLC decreases (Collins et al., 1975). This explains the small increase in RV in absolute term in class II patients. Similar findings have been reported earlier (Krautwald et al., 1961; Murtz and Eckern, 1961; Englert and Denolin, 1965). In class III patients the reduction in RV observed may be due to the decreased TLC as suggested (Collins et al., 1975). Normal values of RV noted in these studies in class I and IV similar to the previous observations (Bates, 1954; Friedman et al., 1959; Hamm and Scholmerich, 1964) could be explained as follows. Measurement of the static pressure volume (recoil) properties assume greater significance in understanding the mechanism involved in such variations (Wood et al., 1971; De Troyer et al., 1980). These authors reported that the elastic recoil generally increases at higher lung volume, is normal at functional residual capacity, becomes abnormally low as RV is approached. They have further argued that vascular plethora accounts for the loss of recoil at small lung volumes and that pulmonary fibrosis produces the increased retractive forces at the larger volume. Regardless the mechanism involved, this type of change in elastic properties probably helps to understand why FRC and RV could remain normal in the presence of reduced TLC. In case of FRC, the resting mechanical balance of the respiratory system set by the tendency of the lung to recoil and the chest wall to the spring outward appears intact (Hercules et al., 1979). With respect to RV it can be seen that two forces (airway compression from enlargement or edema of the vascular sheath and loss of recoil with resultant loss of radial traction of the airway) combine to promote airway closure with airway trapping (Collins et al., 1975). In addition to these abnormalities, disturbances in
associated respiratory muscles in patients with mitral stenosis could also lead to increased RV (De Troyer et al., 1980).

Variation in RV may also be due to the complex abnormalities in the pulmonary system. Restrictive types of abnormalities reduce the RV while obstructions increase RV (Hercules et al., 1979).

The RV/TLC ratio has been observed to increase with severity of disorder, the exception being that the mean value observed in class III seems to be slightly lower than the class II patients. The increase in RV/TLC could be explained as follows: As the inspiratory capacity reduces, the TLC too decreases in many of the patients (Frank et al., 1953; Bates, 1954). Consequently the RV/TLC ratio gets elevated. Others also have documented such increase in RV/TLC (Bishop et al., 1961, 1962). The cardiac enlargement in patients could account for the part of the fall in TLC. The pathological changes in the lungs, however seem to provide a better argument to understand the related changes both in VC and TLC. The observed lower value of RV/TLC in class III compared to class II are understandable when one observes the lesser values of CT ratio and RV in these types.

The FEV$_1$% did not vary with reference to the functional class of the RHD patient. The values observed are normal for the four functional classes studied. These observations indicate a proportional decrease in FEV$_1$ and TLC in all the groups under investigation. Such proportional decrease also represents restrictive type of disorder in such patients (Grant et al., 1978; Cotes, 1979).

Sharp decrease in forced vital capacity, forced expiratory volumes, flow rates and corresponding increase in airway resistance with respect to the functional class was observed. Such variations could be related to an obstructive type of
pulmonary disorder. Observations related to FEV₁% discussed above are an evidence for the airway obstruction, which are within normal limits. This implies that in these patients lungs were emptying normally but from a lower volume (Collins et al., 1975). This further explains the observed progressive fall in t @ 50% and t @ 75% from class II to IV. Class I patients exhibited lower values when compared with the class II. The lung emptying seemed to prolong in class II as compared to class I. This could be due to obstruction. This obstruction is understandable from the low value of FEV₁% and high value of Raw in respective groups.

The sharp decline in various respiratory capacities such as FVC, FIVC and TLC from Class I to Class IV are comparable to the earlier reports (Bates, 1954; Friedman et al., 1959; Krautwald et al., 1961; Palmer et al., 1963; De Troyer et al., 1980). Decrease in vital capacity observed by these authors were correlated to the severity of the disease. The present findings which are consistent with the observation of the Fragminingham study (Kannel et al., 1974) clearly indicate that any reduction in VC could certainly predict the development of a congestive heart failure.

Results related to FEV₁, FEF₂₅, FEF₅₀, FEF₇₅, PEFR, PIFR and Raw suggested the very good correlation with the functional status of the patient. All flow volumes and rates decreased from class I to Class IV with consistent increase in Raw. Similar observations related to the FEV are reported by others in such patients (Stock and Kennedy, 1953; Krautwald et al., 1961; Palmer et al., 1963). Stock and Kennedy (1953) related the decrease in these parameters with the stage of the disease as indicated by the clinical grading. This was very much similar to the measurement
of MMF, which showed no overlap between the least severe and most advanced groups (Palmer et al., 1963).

The close relationship of the raw mid expiratory flow rate with the functional status of the patients which seems to be a very significant observation in this study. These patients seem to have true airway obstructions since the measured airway resistance was seen to progressively increase. Palmer et al. (1963) related such increase with high incidence of bronchospasm, other referred to it as 'cardiac asthma' in more severe cases. He also pointed out a very close relationship between airway obstruction measured by MMFR and pulmonary hypertension in RHD patients. In this connection Friedman et al. (1959) has confirmed that maximum breathing capacity progressively decreases with each advancing stage of the disease. Krautwald et al. (1961) in their detailed studies of about 153 patients with mitral stenosis also suggested that ventilatory defect is related to pulmonary vascular resistance. Such findings related to impairment of ventilatory function and increased airway resistance in a patient with mitral stenosis whose haemodynamic changes are but slight suggest the possibility that chronic bronchitis seems to play a predominant part in the production of dyspnea in these specific cases.

The reduction observed in Kst (L) amongst all the four functional classes studied is similar to those reported earlier (Wood, 1971; Yernault et al., 1979). These reduced values of Kst (L) could be due to reduced lung compliance and increased elastic recoil (a prominent feature of the restrictive disorder) (Colebatch et al., 1979). Fall in lung compliance may be associated with pulmonary congestion (Saxton et al., 1985) or pathological changes like fibrosis and vascular obliteration.
Lung function in ischaemic heart diseases:

Significant reduction observed in the percent predicted mean values of FVC (91.88-22.66), FIVC (92.22-30.66), TLC (89.00-48.66), FEV₁ (106.55-22.66), FEF₂₅ (123.44-22.66), FEF₅₀ (135.22-23.33), FEF₇₅ (208.33-34.33), PEFR (109.00-29.00), and PIFR (75.44-44.66) with corresponding increase in RV/TLC ratio (106.88-214.00) and Raw (77.33-326.00) revealed a good correlation with the functional status of the IHD patients. These findings indicate a close similarity in the effect of IHD on pulmonary mechanics as was observed in RHD (McFadden and Ingram, 1984). The pulmonary manifestation in IHD patient could be explained on the basis of changes in pulmonary vascular pressure by left ventricular failure which is most frequently associated with coronary artery disease and its complication (Ross, 1971). Measurement of pulmonary hypertension in acute myocardial infarction (Fluck et al., 1967; Sjogren, 1970) and left heart pressure in patient with myocardial infarction (Kirby et al., 1968; Begg et al., 1969; Hodges et al., 1969; Russell et al., 1969; Bradley et al., 1970) indicate that in the majority of these patients left atrial or left ventricular and diastolic pressure has been raised under these conditions. Pulmonary hypertension is generally considered to be a passive reflection of raised left atrial pressure (Ross, 1971). Left ventricular failure may occur rapidly in the early stages of acute myocardial infarction or it may also develop slowly and insidiously as a result of myocardial fibrosis secondary to previous infarction and chronic ischaemia (Tancredi, 1981). Generally, left ventricular failure is characterised by ventricular hypertrophy and dilation with increase in end-systolic and end-diastolic volumes and decrease
of the ejection fraction leading to acute elevation in end diastolic pressure with subsequent development of pulmonary congestion and edema (Ross, 1971). The severity of edema varies from that of early interstitial edema to that of florid and rapidly fatal alveolar edema (Tattersfield et al., 1972). With the onset of this edema, arterioles and bronchioles get compressed (Fig. 7), causing the same type of pathological sequence as discussed earlier in case RHD patients. As this occurs, widespread closure of dependent airways with resultant arterial hypoxemia takes place (Demedts et al., 1974; Hales and Kazemi, 1974). There is a significant evidence to suggest that the arterial hypoxemia may be related to ventilation-perfusion imbalance secondary to pulmonary congestion from left ventricular failure (Valentine et al., 1966; Mackenzie et al., 1964; McNicol et al., 1965; Higgs, 1968; Sukumalchantra et al., 1969; Sjorgen, 1970). Most of these authors attributed this disturbance in ventilation-perfusion to the accumulation of interstitial or alveolar fluid in the lungs. Such abnormal ventilation-perfusion has been explained by several authors (Demedts et al., 1974; Hales and Kazemi, 1974. These authors have suggested that a myocardial infarction or during an episode of prolonged myocardial ischaemia, the lung volume at which airway closure begins can encroach upon or even exceed FRC). Therefore, during normal respiration some alveoli are not ventilated and could act as an anatomical shunt (McFadden and Ingram, 1984). In addition to alteration in gas exchange myocardial ischaemia and or infarction may cause acute elevation in airway resistance and reduction in pulmonary compliance (Pepine and Wiener, 1972; Interiano et al., 1973). The decreased lung compliance then causes a decrease in vital capacity and total lung volume in such patients. This
explains progressive falls in FVC, FIVC, TLC and other related parameters in relation to the functional status of the IHD patient investigated in the present work.

In IHD patients the observed mean values for FRC in class I (91.88) and class II (103.75) are within normal limits, class III (75.50) and class IV (68.00) patients showed slight reduction in relation to the severity of the disease. Such normal values observed are understandable on the basis of airway closure as a result of possible air trappings (Collins et al., 1975) especially in class I and class II patients. The mechanism involved has been discussed earlier in the section related to RHD.

No correlation was observed in the measurement of RV and functional status of the concerned patients. The changes in RV in these patients also indicate the complex mechanism involved in the developed disorder as suggested in case of RHD patients. Measured RV/TLC ratio increases from class I to class IV (106.68-214.00). This is probably due to a significant reduction in TLC with respect to the severity of the disease under investigation.

Other two parameters viz. t @ 50 and t @ 75 also showed a similar trend in relation to the functional status as observed in RHD patients. Both these parameters initially increased from class I to class II and later decreased up to class IV. The mechanism involved basically seems to be of similar type as has been explained in case of RHD.

Observations related to forced vital capacity, forced expiratory volume in one second, and FEV₁ % are similar to the findings reported by Collins et al. (1975), who evaluated airway function in 72 patients with left sided valvular and ischaemic heart disease. These patients were free of the related
MYOCARDIAL INFARCTION

↑ LVEDP

↑ PULMONARY VENOUS PRESSURE

NORMAL

ARTERIOLE

VENULE

BRONCHIOLE

VASCULAR ENGORGEMENT WITH COMPRESSION OF BRONCHIOLES

EDEMA AROUND EXTRAALVEOLAR VESSELS AND AIRWAYS

FIG. 7.
symptoms and as a group, these individuals had lower values for FVC and FEV₁ in comparison to the healthy subjects. In same patients the averaged value of 0.75 percent for FEV₁ was noticed to be within normal limits. Fall in FVC and FEV₁ and normal recording of FEV₁% as was also the case in RHD discussed earlier. All these findings lead to being the causative for obstruction in IHD patients.

Observed decrease in FEV₁ (106.55-22.66), FEF₂₅ (123.44-22.66), FEF₅₀ (35.22-23.33), FEF₇₅ (208.33-34.33), PEFR (109.00-29.00) and PIFR (75.44-44.66) in relation to the functional class may be explained on the basis of change in elastic recoil properties of the lung. This is acceptable as one observes smooth increase in the values of kst (L) (65.77-122.00) in IHD patients. This clearly indicates the reduction in elastic recoil of lung in such patients. The reduction in elastic recoil further flattens the pressure volume curve indicating the restrictive type of pulmonary abnormality. Such extent was seen to increase with the severity of the disease.

A close relation was observed in case of Raw with reference to the functional status of the patient. Raw progressively increased from class I to class IV (77.33-328.00). This increase can be explained in terms of the competition between dilated arteries, veins and airways for space in edematous lung (Fig. 7) (Maloney et al., 1970).

The changes observed in various pulmonary parameters are often associated with the cardiac enlargement in IHD patient. The observed values of CT ratio (class I: 52.97, Class II: 54.02, Class III: 54.92, Class IV: 55.02) in these patients indicated a smooth enlargement of heart in relation to the functional class of the disorder. Cardiac hypertrophy observed in
FIG. 8.

COMPRESSION OF LEFT MAINSTEM BRONCHUS
these patients can be interpreted as a direct consequence of the ischaemia due to coronary occlusion or thrombosis (Baxter et al., 1978). Such hypertrophy can also be related to similar conditions like anaemia (Datta and Silver, 1975) or coronary artery ligation (Tennat and Wiggers, 1935) or anomalous coronary artery arising from a pulmonary artery and supplying the heart with relatively unoxygenated blood (Bassis and Sheinkoff, 1955). It is also believed that when cardiac hypertrophy follow a coronary occlusion, it is due to either present or preexistent hypertension associated with cardiovascular disease due to congestive heart failure by severe extensive myocardial damage (Friedberg, 1956).

This study involved hearts at autopsy of about hundred cases of acute coronary occlusion and helped determine the relationship of this condition to cardiac hypertrophy. Smith (1928) evaluated the presence or absence of hypertrophy on the basis of the weight of the heart. He also related the presence of hypertrophy in various cardiac disorders to observed hypertension. In contrast Davis and Blumgart (1937) related the hypertrophy to congestive heart failure even in the absence of hypertension, valvular disease or other accepted causes of hypertrophy. They reported 14 out of 15 cases of congestive heart failure associated with advanced coronary atherosclerosis, the weights of hearts were above 400 gms, and in 11 of these above 500 gms and in 5 above 600 gms. Such cardiac enlargement may cause compression of mediastinal structure, airways or lungs (Fig. 8). This may reflect the changes in the pulmonary function. With massive cardiomegaly the main bronchus may be completely obstructed, with resulting atelectasis of the left lung (Stanger et al., 1969).
Lastly, attention needs to be focused on the respiratory features of angina as have been summarised in Osler's Lumleian lectures (Osler, 1910). He has listed four important special features affecting the pathophysiology of the lungs. i) distension of lungs (acute emphysema) with limited inspiratory excursion and prolonged expiration, ii) at times, actual wheezing and other physical signs like those of an acute attack of bronchial asthma, iii) occasionally, transient acute pulmonary edema and iv) rarely hemoptysis.

Lung function in congenital heart diseases:

In this group the cases that come across (both ASD and VSD) were mainly of left to right shunt category. This was probably so as pressures in the left of the heart are always more than in the right side of the heart (Heath and Edwards, 1958; Wagenvoort et al., 1964; Naeve, 1966). The variations observed in the mean of percent predicted values of different respiratory parameters in relation to the functional status of the patient are very significant. A progressive decline in the mean values of FVC (88.5-19.5), FIVC (88.5-19.5), RV (148.25-78), TLC (100-30), FRC (114.75-35.50), FEV₁ (97.75-20.50), FEF₂₅ (126.50-27.0), FEF₅₀ (111.5-27.0), FEF₇₅ (123.75-35.00), PEFR (106.75-24.50), PIFR (86.75-30.0) was observed from class I to class IV, while two parameters viz. RV/TLC (145.75-257.50), Raw (53.00-289.50) were seen to increase with respect to the functional class of the patients.

The observed changes in pulmonary mechanics are not surprising in view of the observation reported by Hauge and Waller (1975) in such patients. They showed that an increase in pulmonary blood volume lowers the lung compliance even in the absence of significant interstitial fluid increase. Eisenmenger (136
1898) was probably the first person who showed that in ventricular septal defect the pulmonary circulation overfills the lungs. As a result the lungs become stiff (Howlett, 1972).

Similarly, the effect of the altered pulmonary haemodynamics upon the mechanical behaviour of the lung has also been investigated by several workers in animals (Borst et al., 1957; Cook et al., 1959), children (Othinishi, 1970; Howlett, 1972) and adults (Saxton et al., 1956; Davies et al., 1962; Davies and Gazetopohlos, 1967). Mills (1949) observed that altering the pulmonary blood volume in the adult subjects altered the vital capacity. Harda (1959) studied healthy adult dogs before and after lung resection and found a positive correlation between post operative changes and decrease in the effective pulmonary compliance. Wallgren et al. (1960) suggested that the reduction in lung compliance found in presence of left to right shunt might be due to a reduction in the aerated lung volume.

Alterations in regional pulmonary blood have also been recorded in left to right shunt patients with both atrial and ventricular septal defects (Friedman et al., 1968; Sade et al., 1976). The report indicates that the abnormalities characterised by increased pulmonary blood flow or elevated pulmonary atrial pressures or both lead to an increased ratio of pulmonary blood flow in the lung apices relative to that in the dependent lung zones. Studies of regional lung function in patients with VSD have demonstrated mildly abnormal ventilation perfusion relationship. Ventilation to the left lung tend to get depressed slightly as perfusion gets slightly increased (Sade et al., 1976).

De Troyer et al. (1977) extensively studied the consequences of a chronic increase in pulmonary blood flow from
arterial septal defect and related it to the levels of pulmonary hypertension in these patients. Further, the authors showed that, the patients in whom the mean pulmonary artery pressure is normal, the only change observed is an increase in diffusing capacity. Thus, when pulmonary blood flow is abnormally high but vascular pressures are normal, pulmonary mechanics are normal. There is customarily no alteration in the subdivisions of lung volume (Jonsson et al., 1957) or slight reduction in TLC and VC. However, a relative increase in RV could be noted (Linde et al., 1964; Estevez et al., 1967). No impairment of maximum breathing capacity (Bedell, 1961) and no effect in inert gas distribution (Bedell, 1961; Estevez et al., 1967) was noticed. However, when both blood flow and pressure are increased the lung function is seen to deteriorate. Consequently, in those patients with modest pulmonary hypertension there is an overt decrease in maximum expiratory flows at all lung volumes and some reduction in static compliance, in addition to the increased diffusing capacity (Kanagami et al., 1962). When pulmonary hypertension becomes severe air flow rates are markedly depressed, elastic recoil and lung volumes are sharply reduced, airway resistance is elevated and diffusing capacity becomes normal (McFadden and Ingram, 1984). Therefore, the progressive reduction in FVC, FIVC, FRC and TLC and corresponding increase in RV/TLC ratio can be explained on the basis of such increased pulmonary hypertension in the patients investigated. Increased pulmonary hypertension further increases the airway resistance, decreases the maximum expiratory flow owing to the significant decrease in various lung volumes and capacities. Thus, indicating the presence of both the obstructive as well as restrictive abnormalities in these patients.
A progressive increase in RV/TLC in relation to the functional status of the patients was observed. This may be due to decrease in TLC despite a slight increase in RV as observed in class II patients. This observation is very similar to the previous finding in case of atrial septal defect. (Troyer et al., 1977). Normal values of RV were reported in other classes when investigated.

The decrease in FEV₁, FEF 25, FEF 50, FEF 75, PIFR and PEFR can be easily explained on the basis of an increased resistance of the intrapulmonary airways as suggested in forced expiration model (Mead et al., 1967; Pride et al., 1967). This increase in resistance of small airways might be attributed to a competition for space between vessels and airway in the bronchovascular sheath as suggested by Hogg et al. (1972) (Fig. 7).

Increasing pulmonary artery pressure could be associated with an overt decrease in the FEV₁ in relation to the functional class of the patient as observed. Such reduction in FEV₁ also remained significant when expressed as function of vital capacity. The values of FEV₁% are near about same and around normal in all the classes investigated. The normal values of FEV₁% also confirm the pulmonary restriction in these patients.

Sign of obstructive lung disease such as wheezing, use of accessory muscle of respiration, lobar emphysema and atelectasis were also reported in patients with ventricular septal defect with large left to right shunt (Bryk and Brooklyn, 1965; Howlett, 1972; Hordof et al., 1977; Moss and McDonald, 1977). The airway obstruction in these patients can be due to compression of airway by enlarged pulmonary arteries or cardiac chambers [See Fig. 8, 9] (Krabbenhoft and Evans, 1954; Rivkin et al., 1957; Edwards
and Burchell, 1960; Bryk and Brooklyn, 1965; Stanger et al., 1969) and due to an increase in the small airway resistance as a result of accumulation of peribronchiolar fluid (Stanger et al., 1969). All these changes lead to decrease in pulmonary compliance in CHD patients (Bancalari et al., 1977). Lack of a significant correlation between Kst (L) and functional status of CHD patients in the present study confirms the previous findings in which specific compliance was correlated to the pulmonary artery pressure (Saxton et al., 1956; Wallgren et al., 1960; Davies et al., 1962; Davies and Gazetopolous, 1967; Howlett, 1972). The explanation remains uncertain and could even be coincidental. It could be suggested that interstitial changes such as incipient or actual pulmonary edema could have reduced the compliance. However, this dose not apply to our studies. No changes were observed in Kst (L) with the functional status of the patients. Moreover one would not expect pulmonary resistance to be normal in the face of major interstitial changes reported. (Howlett, 1972). Our observation related to airways resistance indicates a very close relation with respect to the functional class of the patients. Progressive increase in Raw was observed in class I to class IV.

No significant changes were observed between two constants of lung emptying viz. t @ 50 and t @ 75. However, the trend shows little increase from class I to class III and in class IV observed values are lowest among all the classes investigated.

Data obtained from Spiroscreen related to various parameters like FVC, FEV₁, FEV₁%, FEF₅₀, FEF₇₅, PEFR and MMF in three cardiac disorders showed similar trends as observed in case of Morgan ELF. The data obtained by these two machines is not combined since the standard norms used for determination of
percent predicted values of respiratory parameters in one machine slightly differs from the other (See, respective operating manuals). The weight factor has not been taken into consideration in case of Spiroscreen as existence of complex nonlinear relation of lung function to weight is known (Hutchinson, 1948). The parameters FVC, FEV\textsubscript{1}, FEF\textsubscript{50}, FEF\textsubscript{75} and PEFR show a striking fall in the mean percent predicted values observed in relation to the functional class of patients in all the cardiac disorders investigated, while FEV\textsubscript{1\%} showed no change with respect to the functional status of the patients. The close relation of MMF with functional status of the patients seemed to be a very significant observation in this recording. A sharp decrease was noticed from class I to class IV patients in RHD (85.53-5.90), IHD (100.53-11) as well as in CHD patients (87.26-17.50). This finding confirms the observation reported by Palmer et al. (1963) in the measurement of airway obstruction by MMFR.

On the basis of variations in the mean percent predicted values of various parameters of the respiratory system recorded on Morgan ELF and Spiroscreen, a new, simple noninvasive system is suggested for the assessment of the functional status of the various cardiac disorders. Those parameters that have higher values of coefficient of correlation (more than 50\%) with respect to functional class of the patient are selected for this purpose. Three cardiac disorders viz. RHD, IHD and CHD investigated showed such higher values for following parameters: FVC, FIVC, TLC, RV/TLC, FRC, FEV\textsubscript{1}, FEF\textsubscript{25}, FEF\textsubscript{50}, FEF\textsubscript{75}, PEFR, PIFR and Raw. Other parameters that exhibit normal values (FEV\textsubscript{1\%}) or fluctuating values (RV, kst (L)) or without specific trends (t @ 50, t @ 75) are omitted for such
classification. Observed values of coefficient of correlation for these parameters also indicate very poor correlation of these parameters with functional status of the patients.

In the proposed system the patients studied are classified into four major groups for each cardiac disorders viz RHD, IHD and CHD. The upper and lower limit of each group for various parameters are determined from the patient predicted mean values of parameters obtained for each functional class as per the NYHA. Lower or upper limit of the group I is determined by taking the mean of mean values of class I and class II, the lower or upper limits of group II is determined by taking the mean of mean values of class I and class II and class III. Similarly the upper or lower limits of group III is determined from mean values of class II and class III and class III and class IV and for group III the mean value of class III and class IV. This procedure has put forth a simpler albeit a precise model for categorisation of functional status of patients in various cardiac disorders.