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STUDY ON ETIOLOGY AND CLINICAL PROFILE OF CHRONIC COR PULMONALE

Dissertation submitted to

THE TAMIL NADU DR. MGR MEDICAL UNIVERSITY

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CERTIFICATE

This to certify that the dissertation entitled **"STUDY ON ETIOLOGY AND CLINICAL PROFILE OF CHRONIC COR PULMONALE"** is a bonafide original work of **Dr. LAVANYA DEVI. P,** in partial fulfillment of the requirements for M.D. Branch – I (Internal Medicine) Examination of the Tamilnadu Dr.M.G.R Medical University to be held in March 2009.

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DECLARATION

I, **Dr. LAVANYA DEVI. P,** solemnly declare that the dissertation titled, **"STUDY ON ETIOLOGY AND CLINICAL PROFILE OF CHRONIC COR PULMONALE"** is a bonafide work done by me at Institute of Internal Medicine, Madras Medical College, during Dec 2007 to June 2008 under the guidance and supervision of Prof**. Dr. A. R. MALATHY M.D.,** Institute of Internal Medicine. The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in Internal Medicine.

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INTRODUCTION[1-4]

It is traditionally defined as the right ventricular failure secondary to disorders that affect either the structure or function of the lungs.

The association of heart failure with chest diseases was not recognised until the beginning of the 20th century. Osler did not mention it in his Text Book of Medicine published in 1904 but referred to it in later editions.

The term cor pulmonale was first introduced into the medical literature by white (1931). Prior to that it was generally known as emphysema heart, pulmonary heart disease Ayerzas disease and rather eloquently as 'Black cardiacs'[4].

Different terms have been used by different workers, pulmonary failure (Fulton 1953), pulmonary hypertensive heart disease with arterial desaturation (Hecht, 1956), pulmonary heart failure (Stuart Harries and Henley, 1957), secondary pulmonary hypertensive cardiovascular disease (Meltingly, 1962) and chronic pulmonary hypertensive heart disease (Calland, 1963) are some of the terms used for the condition known today as cor puomonale. No satisfactory definition has been provided so far for this condition Strong in 1947 defined cor pulmonale as right ventricular

hypertrophy due to the disorder of pulmonary circulation from any cause.[2, 4]

Fishman et al. (1960) believed that mere presence of pulmonary hypertension does not mean right ventricular disease.

Stuart Harries and Henley (1957) used the term cor pulmonale only when congestive heart failure was present and proposed the name pulmonary heart failure to avoid ambiguous terminology.

Friendberg (1956) defined cor polmonale as hypertension of the right ventricle with or without congestive heart failure resulting from diseases of the lung or of the pulmonary vascular tree.

Ruben et al. (1961) defined cor puomonale as a cardiac enlargement in the form of dilatation or hypertrophy or both secondary to pulmonary diseases, bearing no relation to the presence of pulmonary hypertension.

There is general agreement regarding exclusion of right ventricular involvement secondary to other forms of heart diseases like mitral stenosis and patent ductus arteriosus associated with pulmonary hypertension (Spain and Handler, 1948).

Evans (1964) however defined pulmonary heart diseases as resulting from any lung condition as well as direct involvement of the heart, such as suppurative pericarditis and granulomas like Hodgkin's disease and tuberculosis.

Chronic cor pulmonale is recognised as a serious protracted, ultimately fatal human experience consuming frequently a large segment of the sufferer's life.

As a sociological entity, every case has its etiological and aggravating environmental factors and its many economic implication. This disease, in areas where it is prevalent thus constitutes a serious problem in public health and preventive medicine.[3, 4]

This disease has remained unrecognised so long due probably to a number of causes. For many years the diagnosis was not made. The condition was obscured by the accompanying pulmonary manifestations on one hand, and was identified on the other hand as some other form of heart disease. It is only recently that the clinical physiologists have worked out physiological relationships between chronic pulmonary disease and cor pulmonale and still more recently that adequate methods of diagnosis have been established. Physiologists have now simplified the

principles and methods of diagnosis so that physicians can add them to their clinical analysis.[1,2]

The wide disparities in the reported incidence of the disease in different areas many simply reflect these inconsistencies in the diagnostic terminology and conventions. On the other hand, these reports do indicate real variations in disease experience and may give important clues to those differences in local environment or ways of life, which may underlie the geographical distribution of the disease.[5]

Chronic cor pulmonale can be diagnosed clinically, radiologically, electrocardiographically, by Echocardiography and also by invasive techniques.

AIMS AND OBJECTIVES

Aim of the study is to

1. Identify the probable causes and the primary lung disease, which lead to chronic cor pulmonale.

2. Study the clinical profile including Electrocardiographic and Radiological features.

REVIEW OF LITERATURE

DEFINITION[3,5-7, 9]

Definitions of chronic cor puomonale have been put forward by many authors in clinical, functional or morbid anatomical terms. A clinical definition is considered unsatisfactory, since the chief clinical manifestation is heart failure, which takes a long time to develop.

A functional definition in terms of pulmonary hypertension or raised pulmonary vascular resistance is difficult to measure and is variable, and hypertension may be evanescent, may only occur on exercise and may decline in the terminal phase of the disease.

The WHO committee therefore prefers a definition based upon morbid anatomy, for this provides the only characteristic common to all patients at all stages of the disease.[5]

The definition of cor pulmonale is now fairly universally accepted as 'alteration in structure and function of the right ventricle resulting from diseases affecting the structure and function of the lung or its vasculature. This specifically excludes the alterations resulting from diseases of the left ventricle or congenital heart disease.[9]

Chronic Cor Pulmonale is characterized by hypertrophy and dilatation of the right ventricle secondary to the pulmonary hypertension caused by disease of the pulmonary parenchyma and / or pulmonary vascular system between the origins of the main pulmonary artery and the entry of the pulmonary veins into the left atrium.[3]

ETIOLOGY AND CLASSIFICATION[4, 5]

1. Etiological Classification

The WHO has given the following classification of cor pulmonale according to the diseases, which produce secondary right ventricular hypertrophy.

I. Diseases primarily affecting air passages of the lung and the alveoli.

1. Chronic bronchitis with generalized airways obstruction with or without emphysema.
2. Bronchial asthma
3. Emphysema without bronchitis or asthma
4. Pulmonary fibrosis, with or without emphysema due to:
    a. Tuberculosis
    b. Pneumoconiosis
    c. Bronchiectasis
    d. Other pulmonary infections
    e. Muco viscidosis

5. Pulmonary granulomata and infiltration
    a. Sarcoidosis
    b. Chronic diffuse interstitial fibrosis
    c. Berylliosis
    d. Eosinophilic granuloma or histiocytosis
    e. Malignant infiltration
    f. Scleroderma
    g. Dermatomyositis
    h. Disseminated lupus erythematosus
    i. Alveolar resection
6. Pulmonary resection
7. Congenital cystic disease of the lungs.
8. High attitude hypoxia

II. Disease primarily affecting the movements of the thoracic cage

1. Kyphoscoliosis and other thoracic deformities.
2. Thoracoplasty
3. Pleural fibrosis
4. Chronic neuromuscular weakness e.g. Poliomyelitis.

5. Obesity with alveolar hypoventilation.

6. Idiopathic alveolar hypoventilation

III. Disease primarily affecting the pulmonary vasculature

1. Primary affections of arterial wall.

    a. Primary pulmonary hypertension.

    b. Poly arteritis nodosa

    c. Other arterities

2. Thrombotic disorder.

    a. Primary pulmonary thrombosis

    b. Sickle cell anaemia

3. Embolism

    a. Embolism from thrombosis out side the lungs

    b. Schistosomiasis

    c. Malignant embolism

    d. Other embolism

4. Pressure on main pulmonary arteries and veins by mediastinal tumours, aneurysm, granuloma or fibrosis.

2. Clinical classification of cor pulmonale [4, 5]

Clinically cor pulmonale is classified into acute, subacute and chronic depending on the onset of the disease.

Acute cor pulmonale is sudden in onset and it follows massive pulmonary embolism.

The subacute variety may range from days to weeks. It is seen in patients with repeated bouts of small pulmonary embolism or endolymphatic carcinomatosis.

Chronic type may take a few months to year for its development and it commonly occurs in association with chronic pulmonary disease.

PRE DISPOSING FACTORS[3, 7, 8, 10-13]

1. AGE

As chronic obstructive pulmonary diseases are common in middle and elderly age groups, the incidence of cor pulmonale is more common between 4th to 6th decade[7].

2. SEX

The disease is more prevalent in males. The most probable cause is high incidence of smoking in males.

3. TOBACCO SMOKING

Smoking is the most commonly identified correlate with cor pulmonale. It was found that mortality rate for chronic bronchitis was significantly higher in cigarette smokers than in non smokers and increased with the amount smoked. In those who stopped smoking, which would include many who stopped for medical reasons, the mortality at first rose but by 10 years was well below that for smokers.

Sample surveys of respiratory symptoms in the population have shown a much higher prevalence of cough and sputum among smokers. In such surveys, chronic bronchitis, whatever the definition used, has

been almost always confined to smokers. For example, a sample survey in urban and rural population in Britain found in males aged 55 to 64 that the prevalence of chronic bronchitis was 17.6% in heavy smokers, 13.9% in light smokers, 4.4% in ex-smokers and nil among non smokers, similar differences were found in the Royal College of General Practitioners survey.[11]

Prevalence studies in at least 10 countries have confirmed the relationship between smoking and chronic bronchitis. A number of these surveys have included simple tests of respiratory function as FEV_1 and PEFR. On the whole, the tests indicated poorer function in smokers than in non smokers. Follow up studies have shown a steeper decline in FEV_1 among smokers than among ex smokers or non smokers and rapid slowing of the rate of decline following cessation of smoking. Diffusing capacity is also decreased in smokers[10].

The effects of cigarette smoke may begin at an early age, as indicated by increased respiratory illness and diminished pulmonary function in children passively exposed to parental cigarette smoke.

Cigarette smoking causes airway obstruction by mucosal gland hypertrophy with resulting increased mucus secretion, coupled with the inhibitory effects on the bronchial ciliary blanket, which predisposes to

the accumulation of mucus in the bronchial tree. Superimposed infection with associated mucosal edema and infiltration with inflammatory cells will exacerbate airways obstruction.

The effect of smoking may be irritant receptor reflex induced phenomenon.

Finally cigarette smoking recruits inflammatory cells to the lungs as indicated by increased numbers of neutrophils and macrophages in bronchoalveolar lavage fluid from smokers' lungs.

Cigarette smoke can stimulate alveolar macrophages to release neutrophil chemo attractants and smoke can also cause elastase release from neutrophils. In addition cigarette smoking can inactivate alpha antitrypsin probably by oxidation, and indeed a diminution of functional alpha antitrypsin in by about 50% has been demonstrated in bronchoalveolar lavage fluids of human smokers. Local inactivation of alpha antitrypsin may also result from the release of oxidants from activated neutrophils and macrophages. In smokers therefore, the scene of protease antiprotease imbalance is well set and it seems likely that this is the cause of emphysema found in association with chronic bronchitis. The emphysema itself, with its loss of supporting lung substance is a major contributor to airway obstruction and cor pulmonale.[3]

4. ATMOSPHERIC POLLUTION[4, 12]

The effect to pollution can begin early in life. An acute form of respiratory distress resembling acute bronchitis and often continuing into a chronic form with predominant wheeze has been described in individuals.

Smoke may be the most important pollutant. The two great London Smogs of 1952 and 1962 were both associated with a rise in bronchitis mortality much higher in 1952 than 1962. The sulphuric oxide concentrations were similar in both years but the smoke concentration was much lower in 1962 and so was the associated respiratory mortality.

Exacerbation of bronchitis is clearly related to periods of heavy pollution with sulphur dioxide. Nitrogen dioxide can produce small airway obstruction in experimental animals exposed to high concentration of nitrogen dioxide.

5. OCCUPATION[10, 12]

Chronic obstructive lung diseases are more prevalent in workers who are engaged in occupations exposing them to either inorganic or organic dusts or to noxious gases. Epidemiologic surveys have succeeded

in demonstrating accelerated decline in lung function of many such workers.

6. INFECTION[1, 2, 13]

Katten et al. have demonstrated evidence of small airways dysfunction 10 years after an attack of bronchiolitis in infancy, having as far as possible excluded asthmatics.

In a community study in Arizona, Lebowitz and Burrows found a highly significant correlation between poor respiratory function and a history of childhood respiratory illness or of recurrent respiratory infections in adult life. It makes an even bigger contribution to the development of chronic lung disease in developing countries with poor social conditions and poor medical services.

Earlier pathological studies, very reasonably showed that each of the infective exacerbation produced a little further lung damage and added a little more to the permanent impairment of respiratory function. A number of studies have also shown a correlation between deterioration of lung function and frequency of exacerbation.

The viruses concerned were those causing respiratory infection in the general population and included influenza, parainfluenza and respiratory syncitial viruses.

Mycoplasma pneumoniae infection has been reported in association with exacerbation of chronic bronchitis in a small number of cases.

Bacterial infection with encapsulated strains of hemophilus influenza or with streptococcus pneumoniae plays a major part in exacerbations of chronic bronchitis associated with pus in the sputum, though these infections may only be a complication of an exacerbation initiated by a viral infection. More recently Branhamella catarrhalis has been implicated in exacerbations of chronic bronchitis.

Hemophilus and streptococcus pneumoniae are more frequently isolated from mucopurulent than from mucoid sputum in chronic bronchitis. They are frequently present in the nasopharynx of normal people but tend to spread into the lower respiratory tract in the winter after an upper respiratory tract infection.

It is not necessary that all exacerbations are infective in origin. In two Edinburgh studies pathogens were identified in only about half of the

cases. Some may be largely chemical or irritative in origin even though secondary infection is common.

7. GENETIC FACTORS[14]

Alpha–1 antitrypsin deficiency: Eriksson established that this deficiency is inherited and associated with development of lung disease in early adulthood.

Genetic variations in human β - defesin an endogenous antimicrobial peptide in the airway has also been found to be significantly associated with development of COPD where the component of chronic bronchitis is predominant.[14]

8. IMMUNOLOGICAL FACTORS

In 1970 Dutch workers proposed that individuals with asthma and smokers with chronic irreversible airflow obstruction shared a common allergic constitution and increased non specific bronchial reactivity.

Numerous workers have found that smokers have higher IgE levels and higher eosinophil counts than non smokers although the levels do not approach those seen in asthmatics. Atopic status does not appear to differ between smokers and nonsmokers.

It is interesting that histamine, SRS-4 and IgE can be demonstrated in significant levels in bronchitic sputum and that there is a significant inverse correlation between these levels and the minor variations in PEFP.

It remains possible that mediators of type I hypersensitivity may make a small but nevertheless significant contribution to the pathophysiology of cor pulmonale.

Tumour Necrosis Factor α a potent pro-inflammatory cytokine may be associated with development of COPD. Polymorphisms of TNF - α gene promoter is also found be associated with COPD.[15]

9. ABNORMALITIES OF THORACIC CAGE[17]

Scoliosis: In scoliosis the total lung volume is reduced and the lungs differ in size with distortion of lobar shape due to deformity. Alveolar size may vary, and if scoliosis has been present from an early age alveolar numbers per acinus may correspond to the developmental stage. In severe scoliosis progressing to cor pulmonale the pulmonary changes associated with marked vascular medial hypertrophy are indistinguishable from the changes seen in other causes of cor pulmonale.

Lung volumes are reduced with a restrictive pattern even in asymptomatic adolescents with scoliosis.

The higher the dorsal spine curve, the more severe is its effect on function.

The residual volume may be maintained by increasing RV/TLC ratio. The FEV_1/FVC ratio is normal unless obstructive disease supervenes.

Lung compliance in scoliosis is commonly low, perhaps reflecting occurrence of small airway collapse due to inability to take deep breath or a sigh.

In patients with mid or high thoracic curve greater than about 100°, progression to pulmonary hypertension with or without right ventricular failure often occurs in fourth decade.

Pectus excavatum or funnel chest: In this condition, the body of the sternum is angled backwards towards the spine. Minor decreases in vital capacity, total lung capacity and maximal breathing capacity have been reported.

10. OTHER RARE CAUSES[16]

Some rare causes have been reported where corpulmonale occurred secondary to muscle weakness. A case was reported where a 46 year old female admitted for decompensated corpulmonale was found to have metabolic myopathy due to acid maltase deficiency as the primary cause.

PHYSIOLOGICAL DERANGEMENTS IN CHRONIC COR PULMONALE[17-19]

The physiological disturbances in this group of diseases comprise those related to the respiratory function (gaseous exchange) and those connected with the hemodynamics of the pulmonary circulation. Though individual diseases can be classified broadly according to predominant physiological disturbances, it must be recognised that these frequently over lap and are present to a variable extent at different stages of the diseases.

Disturbances in the respiratory function:

The alterations in the respiratory function that can be recognised are four in number.

1. Obstructive ventilatory impairment:

Impairment due to obstruction to airflow somewhere within the tracheobronchial tree.

2. Restrictive ventilatory impairment:

Impairment due to reduction of ventilatory capacity without obstruction to airflow.

3. Impairment in pulmonary gas diffusion:

Disturbance in gaseous exchange between alveoli and pulmonary capillary due to anatomical or functional alterations.

4. Reduction in the ventilation perfusion ratio:

This implies that some of the blood traversing the lungs passes through areas of diminished or absent ventilation or through arteriovenous pulmonary shunts.

The final effect of these functional alterations is seen by reference to the arterial blood and to the respective tensions there in of O_2 and CO_2. The interactions in various alteration in function may be seen by reference to the following examples.

In chronic bronchitis with emphysema the main disturbance is that of obstructed ventilation but this may be accompanied by various degrees of impairment of the pulmonary gas diffusion and by reduction in the ventilation perfusion ratio.

In severe pulmonary fibrosis the main disturbance is one of the restricted ventilation but this may be accompanied by reduction in pulmonary gas diffusion and in the ventilation perfusion ratio.

PATHOGENESIS[9,20,23]

Pulmonary hypertension is the "sine qua non" of cor pulmonale. Accordingly, the mechanisms of cor pulmonale are first those of pulmonary hypertension. In chronic respiratory diseases pulmonary hypertension results from increased pulmonary vascular resistance (PVR) whereas cardiac output and pulmonary "capillary" wedge pressure are normal; pulmonary hypertension is said to be precapillary.

The factors leading to an increased PVR in chronic respiratory disease are numerous but alveolar hypoxia is by far the most predominant, at least in COPD, kyphoscoliosis, and the obesity – hypoventilation syndrome. Two distinct mechanisms of action of alveolar hypoxia must be considered. Acute hypoxia causes pulmonary vasoconstriction, and chronic longstanding hypoxia induces structural changes in the pulmonary vascular bed (pulmonary vascular remodelling).

Hypoxic pulmonary vasoconstriction (HPV) has been known since the studies in 1946 of Von Euler and Liljestrand on the cat. HPV explains the rise of PVR and PAP observed in humans, and in almost all species of mammals, during acute hypoxia. The vasoconstriction is localised in the small precapillary arteries. Its precise mechanism is not fully understood.

The clinical situations that bear the closest analogy with acute hypoxic challenges are probably exacerbations of COPD leading to acute respiratory failure, and the sleep related episodes of worsening hypoxaemia.

Furthermore, other functional factors must be considered, namely hypercapnic acidosis and hyperviscosity caused by polycythaemia, but their role seems small when compared to that of alveolar hypoxia. In idiopathic pulmonary fibrosis the increase of PVR is caused by anatomical factors: loss of pulmonary vascular bed or compression of arterioles and capillaries by the fibrosing process.

Pulmonary hypertension increases the work of the right ventricle, which leads more or less rapidly to right ventricular enlargement (associating hypertrophy and dilatation) which can result in ventricular dysfunction (systolic, diastolic). Later, right heart failure (RHF) characterised by the presence of peripheral oedema can be observed, at least in some respiratory patients. The interval between the onset of pulmonary hypertension and the appearance of RHF is not known and may vary from one patient to another. There is a relation between the severity of pulmonary hypertension and the development of RHF.

HEMODYNAMIC FEATURES[4, 24 - 26]

The hemodynamic observations in cor pulmonale depend somewhat on the cause and duration of lung disease. In patients with multiple pulmonary emboli or primary pulmonary hypertension, pulmonary arterial pressures are more apt to reach systemic levels than are seen in patients with chronic bronchitis and emphysema during an acute respiratory infection. Patients with diffuse interstitial disease tend to have moderate levels of pulmonary arterial hypertension until late in the disease, when ventilation perfusion abnormalities become sufficiently deranged to cause arterial hypoxaemia and respiratory acidosis.

Mean pulmonary arterial pressures may then reach 60 to 80 mmHg. The cardiac output tends to be normal at rest, except late in the disease, when it is apt to be low. In instances of cor pulmonale in which hypoxaemia is a prominent feature, cardiac output tends to be at upper normal limits or slightly greater than normal, recovery from heart failure is associated with a decrease in cardiac output towards normal, presumably as a consequence of the relief of hypoxaemia and acidosis. During exercise, the increase in cardiac output restricted pulmonary vascular tree, particularly if associated with arterial hypoxaemia, generally elicits large increments in pulmonary arterial pressure.

The left atrial pressure is almost consistently normal in cor pulmonale. Exceptions may occur in any type of heart failure that is

associated with an increase in circulating blood volume or, more specifically, when cor pulmonale is complicated by left ventricular failure. Pulmonary hypertension being the common denominator in all types of cor pulmonale, may lead to right ventricular failure. Particular hemodynamic features characterize different stages in this sequence. Thus, before the stage of cor pulmonale, the cardiac output is normal at rest and increases normally during exercise; the filling pressures of the right ventricle (end diastolic pressures) remain normal. Though levels of pulmonary hypertension may vary according to the cardiac output and level of hypoxaemia.

The next stage of cor pulmonale, is characterised by abnormally high filling pressures to accomplish normal increments in cardiac output. Pulmonary arterial pressures may then increase considerably or remain unchanged, depending on the increase in cardiac output and degree of arterial hypoxemia. Finally, should the right ventricle fail, end diastolic pressures in the right ventricle become abnormally high, and the cardiac output which may be normal at rest, fails to increase normally during exercise. At this time, systemic venous congestion manifest, and the circulating plasma volume increases as in other types of heart failure. Lung water also increases.

Both circulating plasma volume and lung water decrease as right ventricular failure and systemic venous congestion are relieved.

CLINICAL FEATURES OF COR PULMONALE[4, 6, 27, 28]

Clinical recognition of cor pulmonale:

All too often the clinical diagnosis of cor pulmonale is first entertained only after the right heart has failed and has evolved systemic venous hypertension, congestive hepatomegaly and peripheral edema. Indeed, many clinicians continue erroneously to reserve the diagnosis of cor puomonale until the stage of right ventricular failure has materialized.

The diagnosis of cor pulmonale is liable to be overlooked unless each patient with pulmonary abnormalities that predispose to pulmonary hypertension is regarded as a likely candidate and clinical acuman is directed towards uncovering early signs of right ventricular enlargement.

Some predisposing conditions are easier to recognise than others.

It is possible to think of COPD as a continum, with chronic bronchitis at one extreme and emphysema at the other end and the majority of patients having features of both conditions.

The distinction between the two groups is presented in the following table.

Comparison of clinical and physiological features of emphysema and chronic bronchitis

	Emphysema	**Chronic bronchitis**
Signs and symptoms:		
Cough and sputum	Scanty	Marked
Dyspnoea at rest	Marked	Usually absent
Recurrent chest infections	Unusual	Frequent
Cyanosis	No	Yes
Edema	No	Yes
Increase AP diameter of thorax	Marked	Mild
Hyperresonance to percussion	Marked	Mild
Breath sounds	Absent to depressed	Rales, rhonchi
Chest X-ray	Hyper inflation	No hyperinflation
	No cardiomegaly	Cardiomegaly
Pulmonary Gas Exchange:		
Haematocrit	Normal	Elevated
Pa O_2	Slight reduction	Markedly reduced
Pa Co_2	Low or normal	Elevated
Diffusing capacity	Markedly decreased	Normal or rest
Pulmonary mechanisms		
Expiratory flow rate	Reduced	Reduced
Elastic recoil	Markedly reduced	Normal or slightly reduced
Lung volume	Marked hyperinflation	Mild hyperinflation
Pulmonary circulation		
Pulmonary hypertension at rest	None or mild	Marked
With exercise	Moderate	Marked
Rt. Heart failure	Terminal	Repeated

CLINICAL MANIFESTATIONS OF COR PULMONALE WITH HEART FAILURE

The presenting symptoms include

1. Dyspnoea
2. Paroxysmal cough
3. Fluid retention with edema and sometimes ascites

Most commonly observed findings are

1. Central cyanosis
2. Distended neck veins which exhibit prominent 'a' and 'v' waves and do not collapse with inspiration.
3. Palpable parasternal or subxiphoid heave
4. Loud pulmonic second sound
5. S3 gallop on right side accentuated by inspiration
6. Holo systolic murmur along the lower left parasternal edge accentuated by inspiration

7. Examination of lung reveals diffuse inspiratory and expiratory rhonchi.

8. Liver is enlarged, tender and frequently pulsatile

9. In acute respiratory failure papilledema, confusion, hyperkinetic circulation and asterexis may be present.

ELECTROCARDIOGRAPHY[29 –34 ,36-39]

Electrocardiography is quite essential in the diagnosis of pulmonary heart disease and in following its course.

The ECG findings suggestive of cardiac involvement in chronic pulmonary disease can be best understood if they are divided in the following groups.

a) ECG findings in right ventricular hypertrophy without any associated emphysema.

b) ECG findings when right ventricular hypertrophy is associated with emphysema.

c) ECG findings that are purely due to emphysema without any right ventricular hypertrophy.

d) ECG findings that may mimic coronary artery disease, which may be due to emphysema and/or cor pulmonale as proved at autopsy. Before dealing with these specific problems some relevant points in this connection are worth mentioning.

Right ventricular hypertrophy may be due to increased volume load or due to increased pressure load.

In increased volume load the crista supraventricularis shows hypertrophy, the free right ventricular wall may be normal. In the ECG this is seen as rsR' in V1. In case of increased pressure load hypertrophy of the entire right ventricular wall and of the crista superaventricularis is seen. Here in addition to a dominant R in V1 there is wide S in I, II, III, V5, V6.

In cor pulmonale the hypertrophy of the right ventricle is due to both the pressure and the volume overload, more so the former as it occurs chiefly as a result of pulmonary hypertension.

However, rarely hypertrophy may be due primarily to a volume load as shown by the hypertrophy of the crista superventricularis with the involement of the free right ventricular wall.[34] Much of the prevailing confusion regarding the ECG in corpulmonale can be minimised if it can be remembered that about one third of the cases show pathognomic findings one third only suggestive findings, while in remaining one third of the cases the ECG tracings either are normal or show mild nonspecific changes.[32]

Philips and Previdence (1958) described the evolution of ECG changes in RVH as follows. The normal cardiac vector is directed to the left and posteriorly. This is due to the greater weight and thickness of the

left ventricle which is two to three times greater than that of right ventricle. Until the resultant vector remains directed to the left and posteriorly (due to the left ventricular preponderance) there will be no change in the ECG tracing no matter to what extent the hypertrophy of right ventricle has taken place. This has been described as stage I.[33]

In stage II when considerable hypertrophy of right ventricle has already occured the resultant vector now moves to the right but still it is directed to posteriorly due to greater mass of the left ventricle. This produces a left axis deviation in frontal plane and prominent 'S' wave appears in the left sided precardial leads (V4-V6).

In third and final stage the Electro motive force is directed out not only to the right but also anteriorly for by now a markedly hypertrophied right ventricle occupies an extremely anterior position. The patients in this category have the heaviest hearts with the thickened right ventricular walls.[34]

The ECG in these patients showed a tall R wave in the right sided precardial leads including V3 R and V4 R.[29] However at this stage it is to be recollected that not only is the right ventricle becoming hypertrophied during all stages but the left ventricle is also expanding its dimensions at least in one third of the cases of cor pulmonale.

So the ratio of right ventricular and left ventricular thickness or weight along with the displacement and rotational changes in the two chambers will be responsible for the changes seen in the serial electrocardiograms. This explain why even in fatal cases of decompensated cor pulmonale showing definite right ventricular hypertrophy at autopsy the ECG may be entirely normal Similarly even marked enlargement of the right ventricle may not be obvious in ECG tracings in the presence of left ventricular hypertrophy or left ventricular conduction defects.[31]

Some of the commonly used criteria for right ventricular hypertrophy include those of Mayor et al (1948) Sokolow and Lyon (1949) Gold Berger (1953) Scott et al (1955) and Milnor (1957) (1957) Every criteria suggested for ECG diagnosis of RVH has its own merit, but availability of so many criteria itself is a proof of their inadequacy. The criteria adopted by Sokolow and Lyon (1949) which gives highest number of positive results indicative of RVH in ECG also gives the highest number of false positives. Goldberger criteria has shown 68.7% reliability and 6.3% false positive. While Mayer et al criteria gave 50% reliability and only 3.1% false positive.

Sokolow (1949) believed that the best criteria of RVH are,

1. Shift of the mean QRS axis to the right

2. An R : S ratio in lead V1 greater than 1

3. R : S ratio in lead V6 of less than 1.

Despite these criteria correlation of autopsy and ECG changes of cor pulmonale are poor. The reason may be due to part to the altered spatial orientation of the heart within the thorax.[34]

In addition there are other conditions that may make ECG diagnosis difficult for example:

1. Presence of either combined ventricular hypertrophy or right ventricular hypertrophy associated with LBBB.

2. Posterior myocardial infarction with its loss of terminal vectors from the posterior aspect of the left ventricle may resemble the ECG as seen in pulmonary heart disease.

3. In cor pulmonale the evidence of RVH are masked or modified by the presence of emphysema so that there is considerable decrease in the sensitivity and specificity of various criteria.

ECG changes in RVH without emphysema

1. Dominant R in V1 or V3 R, 'R' exceeding 7 mm in height and pattern may be 'R', 'RS', qR, QR, RS, and rSR.

2. RS ratio in V1 greater than 1 with 'S' in V1 not more than 2 mm.

3. R in V5 – V6 less than 3 mm with 'S' in V5, V6 more than 7 mm and R : S ratio is less than one.

4. ST segment depression with T wave inversion in V1, V2, V3.

5. Incomplete RBBB pattern and R greater than 10mm in VI.

6. Intrinsicoid deflection and ventricular activation time of 0.035 secs or more in V1, V2.

7. Sum of R in V1 + S in V5 or V6 are more than 10 mm.

8. Clockwise rotation of heart with right axis deviation.

9. Depression of ST segment with T wave inversion in I, II, III AVF.

10. Wide S in leads, I, II, III with R : S ratio less than one in leads I, II, III (involvement of crista supraventricularis).

11. Tall and Peaked 'P' waves in leads II, III aVF, height exceeding 2.5 mm with axis more than + $60^{0.}$

12. QR, qR pattern in aVR with R more than 5 mm and R : Q ratio more than one.

13. Typical Specific S1, Q3, T3 pattern may be seen specifically in acute cor pulmonale.

14. With marked right atrial dilation Q wave appear in right sided precardial leads.

ECG in emphysema:

Pulmonary emphysema commonly produces changes in the ECG which are secondary to the hyperinflation of the lungs and which need not indicate associated RVH or strain.

1. Low voltage: Low voltage is defined as a total RS magnitude that does not exceed 5mm in any of the frontal leads and 10 mm in any of precardial leads. Since air is a relatively poor conductor of electricity it is understable that the hyperinflated lungs will reduce the electrical potential of the heart as recorded on body surface.

2. The frontal plane QRS axis may be between + 90^0 and + 110^0

3. The terminal QRS forces in the frontal plane may be directed right ward (producing an S wave in I) and superiorly (producing S wave in II, III and aVF). This has been known as S1, S2, and S3 syndrome. The cause for this is not known. Although it is seen in patients with emphysema, it is also seen as a normal variant in children and young adults.

4. An incomplete (or complete) RBBB may be a mainfestation of right ventricular strain.

5. QS waves may be recorded in anterior chest leads.

6. Posterior rotation of heart, which may produce Q in II, III, and aVF.

ECG findings when right ventricular hypertrophy is associated with emphysema

Since in chronic cor pulmonale there is varying degree of emphysema and hypertrophy of right ventricle the following may be taken as,

Early decisive:

1. Rt. axis deviation of 110^0 or more
2. Wide S in lead I, II, III with R : S ratio < 1 in I, II, III

3. Incomplete RBBB with R in VI < 10 mm

4. qR in V1, V3R or QS pattern in V1, V2, V3 aVF like an anteroseptal infarction.

5. P pulmonale in leads II, III a VF and axis greater than 60^0.

Fairly suggestive:

1. Complete RBBB with R in V1, greater than 15 mm

2. S in V5, V6 ,more than 10 mm

3. S in V1 less than 5 mm with R in V1 + S in V5 more than 10 mm

4. Right axis deviation from + 75^0 to + 110^0

5. qR or QR in aVR with R more than 5mm and R : Q ratio more than one or QR less, than one

6. low voltage complex with clockwise rotation with vertical heart

7. Ventricular activation time of more than 0.0355 with ST depression in right sided precordial leads.

ECG findings simulating coronary artery disease:

The various electrocardiographic findings that can simulate mycordial ischemia or old infarction are:

1. Presence of QS pattern either in right sided precardial leads or in all routine precardial leads in the absence of old anteroseptal or anterior wall infarction.

2. Presence of QS complex in aVL due to vertical position of the heart in frontal plane.

3. ST depression and or T wave inversion in leads II, III, aVF and from
   V1 to V6.

Though cardiac arrhythmia's and various conduction defects are quite uncommon in cor pulmonale as compared with primary disease of heart, in case they are present, they can create many problems in the interpretation of the ECG tracing of cor pulmonale.

RADIOLOGICAL FINDINGS IN CHRONIC COR PULMONALE[40 – 43]

Chest roentgenograms including PA; lateral view are most valuable tools for identifying cor pulmonale. In selected cases fluoroscopy, bronchography, pulmonary angiography, computed tomography (CT) and other radiographic procedures may provide useful information.

In cases of emphysema the low position of diaphragm, elongates the heart and makes it appear relatively smaller, inspite of its actual enlargement. It is worth remembering, that the right ventricle when it hypertrophies enlarges first anteriorly. Hence, obliteration of normal retrosternal space and bulging of the pulmonary arterial flow tract, are the earliest sign of right ventricular enlargement. This cannot be made out in the routine PA films. Lateral and right anterior oblique views are more valuable. Apical rib crowding may be seen or there may be widening of the rib spaces with elevation of clavicles.

The x-ray may also reveal the underlying lesions such as pulmonary tuberculosis, diffuse carcinomatosis and diseases causing interstitial pulmonary fibrosis, pneumoconiosis, bronchiectasis etc.

The lack of cardiac enlargement in severe emphysema observed by Alexander (1927) was confirmed by Parkinson and Hyle (1937) who noted the prominence of the main pulmonary artery and dilation of main branches at the hilum in over 50% of their patients. In proved instances of cor pulmonale, however the size and shape of the heart shadow vary considerably depending upon the degree of the pulmonary vascular resistance or the absence of heart failure.

The earliest abnormal change is the enlargement of the main pulmonary artery and its branches. Diffuse obstructive emphysema usually causes specific changes in the position of the heart, the heart becoming more vertical and rotated in a clockwise direction about its longitudinal axis. Such positional changes will make the pulmonary artery segment appear slightly more prominent than usual in the both the postero-anterior and right anterior oblique views. In borderline cases, therefore it is sometime difficult to differentiate this increased prominence due to rotation of the heart from that due to slight dilatation of the pulmonary artery. In pectus excavatum and in fibrotic diseases contraction of the left upper lobe of the lung and bulging of the left middle segment often appears like pulmonary artery dilatation and should also be distinguished from that caused by true pulmonary artery dilatation. With the onset of heart failure, the transverse diameter of the

heart is usually increased and dilatation of the right atrium becomes obvious. When there is considerable fusiform dilatation of right pulmonary artery the radiological appearances may strongly suggest a bronchial carcinoma. However the associated dilatation of main pulmonary artery and its left branch which is almost always present should point to the correct diagnosis. When there is associated heart disease involving the left ventricle, cor pulmonale may be present with the clinical and radiological features of left ventricular failure. Pulsation of the right and left main branches of the pulmonary artery may be quite conspicuous on fluoroscopy but it is never florid as in atrial septal defect. Pleural effusion is very uncommon even in heart failure.

MATERIALS AND METHODS

PLACE OF STUDY: Institute of internal medicine, GGH, MMC.

STUDY DESIGN: Observational Study

ETHICAL COMMITEE CLEARANCE: Obtained

PERIOD OF STUDY: Dec 2007 to June 2008

SELECTION CRITERIA: Presence of right ventricular hypertrophy and / or dilatation as observed by Echocardiography along with absence of underlying heart disease (eg Rheumatic, congenital, coronary heart disease) which could have led to right ventricular failure.

STUDY POPULATION

The subjects for the study were selected from cases admitted to the medical wards of Government General Hospital, Chennai during the period from December 2007 to June 2008 who fitted in the criteria described above. The diagnosis of chronic cor pulmonale was made on the basis of history, physical findings and echocardiographic features.

STUDY DESIGN: Observational study

METHODOLOGY

50 representative cases of chronic cor pulmonale which fitted in the criteria were selected. A detailed history was obtained from them and symptom analysis was done. A detailed clinical examination was also done.

A 12 lead electrocardiogram which included right sided chest leads V3 R and V4 R was obtained and analysed.

A chest radiograph which comprised of a posteoanterior chest film was obtained. In selected cases computerised tomogram of the chest was done. Parenchymal lesions in the lung were looked for which would give a clue to the underlying lung disease which caused chronic cor pulmonale. Presence of pulmonary artery dilatation and other features suggestive of cor pulmonale were also looked for.

The clinical profile along with the probable etiology, radiological and electrocardiographic findings were summarised and compared with existing data.

COMPETING INTEREST: NIL

FINANCIAL SUPPORT: NIL

OBSERVATIONS

Out of these 50 patients 40 were male patients and 10 were females. The age of patients ranged from 35 to 80 years.

AGE:

In our present study the following age distribution was observed.

The distribution showes that the peak incidence was in the 5^{th} and 6^{th} decades.

Age distribution in shown in the table below.

Table – 1

AGE DISTRIBUTION

Age groups (in Years)	Number of cases	Percentage
<40 Years	8	16%
40 – 50	12	24%
51-60	20	40%
>60 years	10	20%

SEX:

In our study 40 patients were males and 10 patients were females.

Table – 2

SEX DISTRIBUTION

Sex	Number of cases	Percentage
Male	40	80%
Female	10	20%

SMOKING HABITS:

In our study 38 male patients were smokers and used to smoke more than 10 cigarettes or beedies per day (ranging from 10 to 30 per day). The duration of smoking was more than 10 years in 33 cases where as in 5 cases it was less than 10 years.

Table – 3

Smoking	No. of cases	Percentage
Absent	12	24%
Present	38	76%

Table –4

Smoking duration	No. of cases	Percentage
< 10 Yrs	5	10%
10-20 Yrs	22	44%
21-30 Yrs	9	18%
> 30 Yrs	2	4%

PAST HISTORY OF TUBERCULOSIS:

Past history of tuberculosis was present in nine among the 50 patients. Among them documented sputum positivity and records were available with six of them. Six of them completed treatment and three defaulted. Five out of the nine were non smokers which accounted for more than 50% of them. Only two of them had a history of smoking for more than 10 years.

SYMPTOMATOLOGY:

All the patients with corpulmonale had breathlessness and cough. Most patients presented with pain abdomen and swelling of feet.

Fever was present in 24% percent of patients whereas loss of appetite was in 74 percent, chest pain was noted in 12% and hemoptysis in 16 percent. 20 percent of patients presented with palpitation.

The details about the symptoms are depicted in the table

Table – 5

SYMPTOMATOLOGY

Symptoms	No. of cases	Percentage
Breathlessness	50	100
Cough with expectoration	50	100
Pain abdomen	32	64
Loss of appetite	37	74
Swelling of feet	44	88
Fever	12	24
Haemoptysis	8	16
Palpitation	10	20
Chest pain	6	12

Physical Findings:

All patients had right sided heart failure in the form of raised Jugular venous pressure, tender enlarged liver and bilateral pedal edema. Most commonly observed physical findings in our study are tachypnoea, active accessory respiratory muscles, diminished chest expansion, epigasteric heave, crepitations and rhonchi on auscultation and loud pulmonic sound.

Table – 6

PHYSICAL FINDINGS

Signs	No. of cases	Percentage
Tachycardia	24	48
Tachypnoea	50	100
Active accessory respiratory muscles	35	70
Chest AP diameter equal or greater than transverse diameter	5	10
Downward displacement of liver dullness	23	46
Obliteration of cardiac dullness	9	18
Impalpable apical impulse	11	22
Epigastric pulsation	17	34
Parasternal heave	9	18
Loud pulmonic sound	12	24
Pansystolic murmur at left lower sternal border	9	18
Crepitations and / or rhonchi	50	100
Ascites	5	10

Cardinal signs of right heart failure (pedal edema, palpable tender liver and raised jugular venous pressure) was present in all cases.

RADIOLOGICAL FEATURES:

Majority of the X-ray showed Chronic bronchitis with or without emphysema, the major etiological factor of chronic corpulmonale. 15 cases (30%) showed evidence of cardiomegaly, with pulmonary artery dilatation. However, the type of chamber enlargement could not be accurately predicted in PA view.

Gross pulmonary fibrosis with are without compensatory emphysema was seen in 9 cases. Most the cases had bilateral upper lobe involvement. In our X-ray profile, we had one patient with kyphoscoliosis with gross thoracic deformity. Six cases (12%) showed bilateral basal bronchiectasis and two cases showed reticulo nodular shadow suggestive of interstitial lung disease. CT of the chest was done to confirm the diagnosis.

Radiological features of the lung parenchyma is given in the table below.

Table – 7

RADIOLOGICAL FEATURES

Lung Parenchyma	No. of Cases	Percentage
Gross fibrosis with or without compensatory emphysema	9	18
Increased Broncho vascular markings with or without over inflated lung fields suggestive of COPD	27	54
Bilateral bronchiectasis	6	12
Thoracic cage deformity	1	2
Reticulo nodular pattern	2	4
Normal	5	10

ELECTROCARDIOGRAM:

Among the 50 cases 27 (54%) cases showed evidence of right axis deviation, 24 (48%) cases showed low voltage complexes, 14 (28%) cases showed P-pulmonale and 10 (20%) cases showed right bundle branch block. Eight cases (16%) showed right ventricular hypertrophy. Six cases showed other ECG changes viz ventricular ectopics (1 case), T inversion in II III AVF (4 cases), sinus tachycardia (1 case).

Table – 8

ECG CHANGES

ECG Changes	No. of cases	Percentage
Right axis deviation	27	54%
RBBB	10	20%
P-pulmonale	14	28%
Low-Voltage complexes	24	48%
RVH	8	16%
Other changes	6	12%

CAUSES OF CHRONIC CORPULMONALE:

In our study the cause of chronic corpulmonale in majority of the cases was chronic bronchitis and emphysema 54%. In nine of the cases sequelae of pulmonary tuberculosis was the cause. Five cases had bronchial asthma, six cases bronchiectasis one case had kyphoscoliosis with thoracic deformity and two had interstitial lung diseases.

Causes of Chronic corpulmonale are shown in table

Table – 9

CAUSES OF CHRONIC CORPULMONALE

Cause	No. of Cases	Percentage
Chronic bronchitis / emphysema	27	54
Bronchial asthma	5	10
Bronchiectasis	6	12
Sequelae of pulmonary tuberculosis	9	18
Kypho-scoliosis with thoracic deformity	1	2
Interstitial lung disease	2	4

DISCUSSION

Among 50 patients in our study 20% were found to be females and the remaining males. Most of the study population in our present study were in the 40 to 60 year age group. The age and sex ratio in our study is comparable to the studies done previously[35,44]. Shankar et al reported a similar age distribution in his study.[35]

The present study showed that 76% of the patients were smokers as compared to 70% reported by Padmavathi et al[35] and 80% reported by Shankar et al[44]. The importance of smoking has also been stressed in various studies done previously in our country and outside.

The symptoms and physical findings observed did not vary much from studies done previously[35,44]

Comparison of electrocardiographic findings in the present study and studies done previously are given below

Table - 10

COMPARATIVE PERCENTAGE OF P PULMONALE IN DIFFERENT SERIES45

SERIES	PERCENTAGE
Spodick 1959	13.9%
Calatayud 1970	46.2%
Ivan J. Pinto	33.3%
Chappel 1966	29.%
Present study	28%

Table - 11

COMPARITIVE PERCENTAGE OF RIGHT AXIS DEVIATION45

SERIES	PERCENTAGE
Padmavathi	43.4%
Pinto	45.5%
Present study	54%

Table - 12

COMPARITIVE PERCENTAGE OF RBBB45

SERIES	PERCENTAGE
Padmavathi	7.2%
Pinto	13.3%
Present study	20%

Causes of chronic cor pulmonale in our study was COPD in 54%, sequelae of pulmonary tuberculosis in 18% and bronchiectasis in 12%. The etiological causes in Padmavati et al series[44] was chronic bronchitis and emphysema in 50.8% of cases. In Kamal et al series chronic bronchitis and emphysema was the contributor in 60% of cases. Thus the present study and various other studies show that emphysema and chronic bronchitis is major etiological factor in the production chronic cor pulmonale.

In studies done recently from United States and United Kingdom the cause of cor pulmonale was COPD in 85% of cases[9,21]. Pulmonary tuberculosis was very rarely associated with cor pulmonale. But in our study we found an association of 18% with pulmonary tuberculosis,

which formed a significant number. It probably reflects the increased prevalence of pulmonary tuberculosis in our set up as compared to the other parts of the world.

SUMMARY

1. The objective of the study was to know the etiology and clinical profile including radiological features and ECG changes in chronic corpulmonale.

2. 50 cases of chronic corpulmonale, of which 40 were males and 10 were female were included in the study.

3. Chronic corpulmonale was predominantly found to be a disease of middle and older age groups with a peak incidence in the fifth and sixth decades.

4. Smoking plays a significant role in precipitating and aggravating the primary lung disease and hence corpulmonale.

5. Thorough interrogation with reference to symptoms of breathlessness, cough with expectoration swelling of the feet etc, was done.

6. Detailed clinical examination was carried out to confirm the diagnosis of corpulmonale and to find out any associated evidence of lung parenchymatous lesion. Nine cases had sequelae of pulmonary tuberculosis Six cases had bilateral bronchiectasis and 5 cases had bronchial asthma 1 case had kyphoscoliosis and 2 had

ILD. The remaining cases were chronic bronchitis with or without emphysema.

7. Chest X-ray was done in all cases. Chest X-ray showed details of relevant clinical profile. Thus the changes included chronic bronchitis with or without emphysema (54%), braonchiectasis (12%). Bilateral pulmonary tuberculosis with fibrosis with or without compensatory emphysema (18%). One case (2%) showed kyphoscoliosis with gross thoracic deformity. Two (4%) had reticulo nodular pattern suggestive of ILD. CT scan was done in necessary cases.

8. In all the clinically proved cases of corpulmonale Electro cardiogram was recorded in all the 12 leads along with V3R, V4R. It was recorded at a speed of 25 mm/sec with standardization of 1 mv – 10 mm.

9. ECG varies between normal (only sinus tachycardia) to evidece of dominant right ventricular activity. The latter was evidenced by RVH (16%). Righ Axis deviation (54%) RBBB (20%) and P – Pulmonale (28%). One case had ventricular ectopics.

10. Response to treatment was better in patients with symptoms of lesser duration compared to patients with symptoms of longer duration.

CONCLUSION

The major cause of chronic cor pulmonale in our study was found to be Chronic Obstructive Pulmonary Disease followed by sequelae of pulmonary tuberculosis. Smoking formed the major and most important causal association in the present study.

PROFORMA

I. General information about the patient:

Name: Date of Admission:

Age: Date of Discharge:

Sex: I.P. No:

Occupation & Income:

II. Complaints with duration:

1. Cough with expectoration and nature of the sputum:
   a. Duration
   b. Quantity: e. Smell:
   c. Quality: f. Relation to posture:
   d. Colour:
2. Breathlessness
   a. At rest:
   b. On exertion:
   c. Constant or paroxysms:
   d. Relation to posture:
3. Swelling
   a. Extremities:
   b. Face:
   c. Abdomen:
4. Fever:
5. Chest pain:
6. Haemoptysis:
7. Palpitation:

8. Headache:

2. Drowsiness:

3. Abdominal pain:

4. Fatigue:

5. Scanty urine:

III. History of present illness:

The mode of onset and appearance of symptoms in chronological order with their duration.

IV. History of previous illness:

a. Chronic bronchitis

b. Bronchial asthma

c. Any suppurative lung disorders

d. Tuberculosis and its treatment details

e. Any other disease, listed under classification.

f. Pneumoconiosis

g. Thoracic operations

h. Malignancy with details

V. Family History:

Parents	Sisters
Brothers	Children

VI. Personal History:

a. Appetite	d. Bowels
b. Sleep	e. Smoking & alcohal
c. Micturation	f. Menstruation

VII. Clinical examination

A. General physical examination:

1. Body built	10. Abdominal distension
2. State of nutrition	11. Jaundice

3. Pallor
4. Cyanosis
5. Clubbing
6. Coldness or warmth of the extremities
7. B.P.
8. Distended peripheral veins Jugular venous pressure : 'a' and 'v' waves.
9. Pulse : Rate, Rhythm, Volume, State of arterial vessels wall and character of the pulse.
12. Peripheral lymphadenopthy
13. Temperature
14. State of consciousness
15. Edema

B. Cardiovascular system:

a. Inspection: 1. Apical impulse
2. Parasternal heave
3. Epigastric pulsation
4. Any other abnormalities

b. Palption: Nature of the special impulse, thrill etc.

c. Percussion: Cardiac dullness
Dullness over the lower end of sternum

d. Auscultation: 3rd Heart sound
P2 accentuation
P2 splitting
Any murmurs

C. Respiratory system:

a. Inspection: Shape of chest
Tracheal position
Kyphoscoliosis
Any other deformities
Rate & Type of respiration

b. Palpation: Tracheal position
Movements
Apex beat
Measurement of chest

c. Percussion:

d. Auscultation: Breath sound, Adventitious sounds

D. Abdomen:

Inspection, Palpation, Percussion & Auscultation

E. Central Nervous System:

HMF, Cr, nerves, Motor & Reflexes, Sensory, Cerebellar, skull & spine, Meningeal signs.

VIII. Electro cardiogram

a. Rate:

a. Rhythm
b. Voltage
c. Axis
d. P – Pulmonale
e. Presence of ventricular hypertrophy
f. Presence of RBBB
g. Other changes

IX. Chest X-ray

a. Lung parenchyma

b. Cardiac shadow

c. Pulmonary Artery

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MASTER CHART

				Symptamatology				Electro Cardiographic Changes						
Sl. No.	Age/Sex	Occup	Smok. Dura. Yrs	B.Ness	Swetl.Limbs	Cgh + Expt Duration	Radiological features	Rt.Ax.Dev.	Low Volt.	P-Pul	RBBB	RVH	Others	Diseases leading to corpulmonale
1	65/M	Agri	15	4 Y	2 Y	10 Y	emph+card.	+	-	-	+	-	T-II III aVF	C.B.+EMP.
2	35/M	Agri	10	2 Y	3 M	3 Y	Card+mpa enlarg_PAH	-	-	+	-	-	T-II III aVF	BR.asthma
3	38/M	Cooli	8	2 Y	6 M	3 Y	Bil.Fibro p.kochs+empy	+	-	+	-	-	-	Bil.pulkochs+emph
4	55/M	Agri	10	20 Y	10 Y	10 Y	INC.br.mark,sugg,ch,bronch	+	-	-	+	-	-	C.B
5	68/M	Agri	10	20 Y	1 Y	20 Y	Bil basalbronch+emph+MPA promi	+	-	+	-	+	-	Bil. bronchiectasis
6	65/M	Agri	20	10 Y	5 Y	7 Y	Cardi. MPA promi +emph	+	-	-	-	+	-	Ch bronch+emph
7	58/M	Clerk	24	2 Y	2 Y	8 Y	Emph + card + promi MPA	-	+	+	+	-	-	C.B +emph
8	80/M	Busi	-	4 Y	5 M	4 Y	Bil.fibro p.kokhs+emph	+	+	-	-	-	-	Bil.P.Kochs+emph
9	54/M	H.Serv	15	4 Y	1 M	15 Y	Card+ccf	+	+	-	-	-	-	Br.asthama
10	50/M	Driver	32	4 Y	4 Y	12 Y	Card+emph+MPA, promi	+	-	-	+	+	-	C.B.+emph
11	37/M	Mason	17	3 Y	6 M	5 Y	Card+Rvconf, P.conus prom, MPA + emph	+	-	+	+	-	-	C.B.+emph
12	85/M	Teacher	20	6 Y	8 M	8 Y	Bil. EMPH	-	-	+	-	+	-	C.B.+emph
13	70/M	Tailor	25	8 Y	1 Y	3 Y	Emp+tub heart	-	+	+	-	-	-	C.B.+emph
14	45/F	H.W.	-	15 Y	3 Y	6 Y	Card+MPA prom	+	+	-	-	-	-	Br.Asthma
15	55/M	Worker	15	2 Y	5 M	5 Y	Bil.EMPH	+	-	-	-	+	-	C.B+emph
16	38/M	Clerk	18	16 Y	6 M	10 Y	Normal	-	+	-	-	-	-	Br.asthma
17	50/F	H.W.	-	15 Y	3 Y	8 Y	Card+RNP	+	-	+	-	-	-	ILD
18	80/M	Agri	35	10 Y	3 Y	8 Y	Bil EMPH+tub heart	-	+	-	+	-	S.Tachy	CB+emph
19	55/F	Worker	-	5 Y	3 M	8 Y	Rt basal bronchiactasis	-	+	-	+	-	Vent ectopic	Bil.Bronchiectasis

20	35/F	H.W.	-	3 Y	2 M	5 Y	Bil basal bronchiactasis	-	-	-	-	-	-	Bil.Bronchiectasis
21	35/M	Weaver	18	8 Y	2 Y	3 Y	Bil emph+low set diaph	-	-	-	-	-	-	C.B.+ emph
22	42/F	H.W	-	2 Y	4 M	2 Y	Bil emph + promi MPA	-	+	-	-	-	-	C.B.+ emph
23	55/M	Agri	20	5 Y	2 M	3 Y	Rt basal bronch	+	+	-	-	-	-	Bronchiectasis
24	48/M	Sh.keep	20	8 Y	2 Y	5 Y	Kyp sco. + eventr. diaph+CCF	+	-	-	+	+	-	Kyphsco+grossthorac.defor
25	40/M	Busi.	8	2 Y	1 M	2 Y	Bil. emph	-	+	-	-	-	T-II III aVF	C.B + emph
26	55/M	Worker	15	5Y	3M	5Y	Bil.emph + tub heart	-	+	-	-	-	-	C.B. + emph
27	35/F	Cooli	-	2Y	6M	2Y	Cav, rt & It uz + cal, spots	-	-	+	-	-	-	Bil p.kochs
28	45/M	Teacher	15	6Y	2Y	8Y	Bil. emph + MPA promi	-	+	-	-	-	-	C.B. + emph
29	60/F	Servent	-	14Y	6M	10Y	Card+ bil fibrosis	-	+	-	-	-	-	BL Old P.Kochs.
30	53/M	Agri	-	8Y	6M	8Y	Card + uz kokhs	+	+	-	-	-	-	BL Old P.Kochs.
31	44/M	Cooli	8	8Y	2M	8Y	Card + uz kokhs	-	-	+	+	-	-	Pul Kochs + emph
32	48/M	Mech	8	5M	4M	6Y	inc Bronch vasc marker	-	+	-	-	-	-	C.B.
33	38/M	Mason	7	3Y	8M	5Y	Bil.emph	+	-	-	-	-	-	C.B. + emph
34	49/F	Servent	-	6Y	1Y	8Y	Bil.uz.kochs + cav. Rt.mz+emph	+	+	+	-	-	-	Bil.p.kochs+emph
35	52/M	Vendor	22	2Y	8M	6Y	Bil.emph+tub heart	-	+	-	-	-	-	C.B. + emph
36	64/M	Agri	24	6Y	8M	8Y	Bil.emph	-	+	-	-	-	-	C.B. +emph
37	53/M	Tailor	14	3Y	2M	4Y	Bil fibro pul kochs	+	-	-	-	-	-	Bil. p. kochs + emph
38.	57/M	Driver	27	4Y	4M	6Y	Emph. + mpa promi	+	-	+	-	-	-	C.B. + emph
39.	55/M	Worker	16	6Y	2M	8Y	Bil.emph	-	+	-	-	-	-	C.B. + emph
40.	59/M	Agri	18	8Y	1Y	10Y	Bil emph+mpa promi	+	-	-	-	+	-	C.B+emph
41.	50/M	Cooli	20	4 Y	6 M	4 Y	Bil.uz kochs+cav.Rt.mz+emph	+	+	+	-	-	-	Bil.p. kochs+emph
42.	44/M	Driver	15	6 Y	8 M	5 Y	Bil. emph	+	-	+	-	-	-	C.B. + emph
43.	35/F	H.W	-	3 Y	2 M	2 Y	RNP+Card + PAD	-	+	-	-	-	-	ILD
44.	64/M	Mil Work	22	8 Y	9 M	6 Y	Rt. Basal. bronch	+	+	-	-	-	-	Bronchiectasis
45.	65/F	Cooli	-	2 Y	1 M	4 Y	Bil. emph	+	-	-	-	-	T-II III aVF	C.B. + emph
46.	54/M	Mason	18	10 Y	2 M	6 Y	Bil emph	+	+	-	-	-	-	emph
47.	58/M	Cooli	16	8 Y	1 M	8 Y	Normal	+	-	-	-	-	-	Br. asthma
48.	56/M	Servent	22	2 Y	6 M	2 Y	Card+rv conf. P. conus prom+ emph	+	-	+	-	-	-	C.B. + emph
49.	38/M	Agri	12	10 Y	3 M	12 M	Bil. emph + lowset diaph	-	-	-	-	-	-	Vent. ectopic+C.B. emph
50.	70/M	Agri	23	6 Y	8 M	4 Y	Rt. Basal. bronchiactasis	-	-	-	-	+	-	Bil. bronchiectasis

ABBREVIATIONS

Bil	-	Bilateral
Rt	-	Right
Cal	-	Calcified
LZ	-	Lower zone
Emph	-	Emphysema
Card	-	Cardiomegaly
MPA	-	Main pulmonary artery
Enlarge	-	Enlarged
P. Koch's	-	Pulmonary Koch's
Inc	-	Increased
Broncho	-	Bronchovascular
Mark	-	Markings
Sugg	-	Suggestive
MZ	-	Midzone
Hil	-	Hilar
Promi	-	Prominent
Cardiomeg	-	Cardiomegaly
CCF	-	Congestive cardiac failure
RNP	-	Recticulonodular pattern
RV Conf.	-	Right ventricular configuration
Br. Asthma	-	Bronchial asthma
P. Conus	-	Pulmonary conus
Dia	-	Diaphragm
Vent Ect	-	Ventricular ectopics
S. Tachy	-	Sinus tachycardia
Fibro	-	Fibrosis
ILD	-	Interstrial lung disease