A Dissertation on

A STUDY ON ELECTROCARDIOGRAPHIC AND ECHOCARDIOGRAPHIC CHANGES IN CHRONIC OBSTRUCTIVE AIRWAYS DISEASE

submitted to
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
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in partial fulfillment of the regulations
for the award of the degree of

M.D. (GENERAL MEDICINE)
BRANCH - I

KILPAUK MEDICAL COLLEGE
CHENNAI.

MARCH 2009

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY ON ELECTRO CARDIOGRAPHIC AND ECHOCARDIOGRAPHIC CHANGES IN CHRONIC OBSTRUCTIVE AIRWAYS DISEASE"** is a bonafide work done by **Dr.V.MURUGESAN**, post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision, in partial fulfillment of regulations of **The Tamilnadu Dr.M.G.R.Medical University** for the award of **M.D.Degree Branch I (General Medicine)** during the academic period from May 2006 to March 2009.

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ETHICAL COMMITTEE OF
GOVERNMENT KILPAUK MEDICAL COLLEGE HOSPITAL
KILPAUK, CHENNAI-10.
Venue: Dean Chamber, Date: 3.1.2008

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TO WHOMSOEVER IT MAY CONCERN

Dear Sir / Madam

Sub: Internal Medicine – MD PG's Dissertation Ethical Committee – Reg.

Ref: Requisition from H.O.D. Medicine.

This is in reference to the letter dated 2.1.2008 regarding Ethical committee meeting clearance with regard to the following topics

Sl.No	Name of the Post Graduate	Dissertation Topic
1	Dr. R.Ramprasad	A study on Prevalence and Risk Factors of Diabetic Nephropathy in Newly Detected Type 2 Diabetic Patients
2	Dr. V.Sakthivadivel	A study on Cardiac abnormalities in HIV infected individuals
3	Dr. K.S.Gopakumar	A study on Subclinical Hypothyroidism in females over 50 years of age
4	Dr. T.Karthikeyan	Inflammatory profile in Acute Myocardial Infarction

5	Dr. Maliyappa Vijay Kumar	A study on Prevalence of Microalbuminuria in HIV patients not on ART and Correlation with CD4 count
6	Dr. D.Radha	High sensitivity C - reactive protein as a determinant in the outcome of Acute ischemic stroke
7	Dr. Lakshmi Thampy M.S	A Study on the prevalence of increased LV mass & Proteinuria in newly diagnosed Hypertensive patients.
8	Dr. Manu Bhasker	A study on the effect of Intravenous Metoprolol along with Thrombolysis in Acute Myocardial infarction
9	Dr. P.Mohanraj	Evaluation of Lipid Profile in patients with non diabetic Chronic Kidney Disease Stage 3,4 and 5
10	Dr. P.Dhanalakshmi	A study On Metabolic Syndrome in Young Ischemic Stroke
11	Dr. V.Murugesan	A study on Electrocardiographic and Echocardiographic changes in Chronic Obstructive Airways Disease
12	Dr. M.Seetha	Effect of Right ventricular infarction on the immediate prognosis of Inferior wall Myocardial Infarction

We are glad to inform you that at the EC meeting held on 3.1.08 on the above topics were discussed and **Ethically approved**.

DEAN

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We confirm that no member of the study team is on the Ethics Committee and no member of the study team voted.

The trial will also follow the Ethics Guidelines for Bio-Medical Research On Human subjects issued by ICMR, New Delhi and will not involve any expense to the Government and will not be detrimental to the normal functioning of the Institution.

The study will also satisfy the revised order issued by the Government of Tamil Nadu, Health and Family Welfare Department G.O.MS.No:319, H & FW, Dept. dated 30.11.2001.

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CONTENTS

INTRODUCTION

Chronic Obstructive Airways Disease is acknowledged by the "WHO" as the fourth commonest cause of death worldwide. It is projected to be the third most common cause of death and fifth most common cause of chronic disability by the year 2020.

Earlier in the 20th century TB was the major pulmonary disease in developing countries. Its place has been taken by two diseases in the later half of 20th century namely chronic obstructive airways disease and Carcinoma of lung.

Cigarette smoking is said to have been playing a major role in the etiology of Chronic Obstructive Airways Disease. Knowing the ill effects of smoking many governments including Indian Government have banned smoking in public places. This may in future lead to reduction in number of smokers and its ill effects thus reducing the incidence of COAD in due course.

Chronic Obstructive Airways Disease is known to cause airflow limitations, impaired gas exchange and increased pulmonary artery pressure. According to literatures prevalence of pulmonary hypertension in chronic obstructive airways disease is 18%[1]. Elevated pulmonary artery pressure is a predictor of mortality in chronic obstructive airways disease. "Weitzenblum" and coworkers[2] showed a 72% 4 year survival in patients with normal pulmonary artery pressure compared with 49% survival in patients with

an elevated pulmonary artery pressure in Chronic Obstructive Airways Disease.

Both electrocardiography and echocardiography are very useful in the detection of elevated pulmonary artery pressure. Here an attempt has been made to correlate clinical features, pulmonary function tests, electrocardiography and echocardiography in case of Chronic Obstructive Airways Disease patients.

AIM OF THE STUDY

To analyze various cardiovascular manifestations of “Chronic Obstructive Airways Disease” with the help of Electrocardiography and Echocardiography.

REVIEW OF LITERATURE.

Chronic Obstructive Airways Disease. (COAD)

Chronic Obstructive Airways Disease is defined more clearly using the following,

British Thoracic Society[3]:

Chronic Obstructive Airways Disease is a condition that is characterized by airflow obstruction (FEV1 <80% predicted value & FEV1/FVC < 70%) which shows relentless progression & doesn't change markedly either spontaneously or in response to treatment.

WHO/National heart lung & blood institute GOLD (Global initiative for chronic Obstructive Lung Disease)[4]:

Chronic Obstructive Airways Disease is a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive & associated with an abnormal inflammatory response of the lungs to noxious particles and gases.

The American thoracic society:

The most recent definition, offered by the American Thoracic Society, defines chronic obstructive airways disease as a disease state characterized by the presence of air flow obstruction due to chronic bronchitis or emphysema;

the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity and may be partially reversible[46].

Other names of COAD

-chronic obstructive pulmonary disease (COPD).

-chronic obstructive lung disease (COLD).

-chronic airflow limitation (CAL).

Chronic bronchitis and emphysema both constitute Chronic Obstructive Airways Disease.COAD is present only if chronic airflow obstruction occurs; Chronic bronchitis without chronic airflow obstruction is not included within COAD[17].

Chronic bronchitis:

Chronic bronchitis is defined epidemiologically as a condition in which persistent cough with sputum production is present for at least 3 months for 2 consecutive years[5] .

Prognosis is linked to the presence of airflow obstruction and not sputum production.

Emphysema:

Emphysema is defined pathologically as an abnormal permanent dilatation of air spaces distal to the terminal bronchiole accompanied by destruction of the alveolar walls without obvious fibrosis[6].

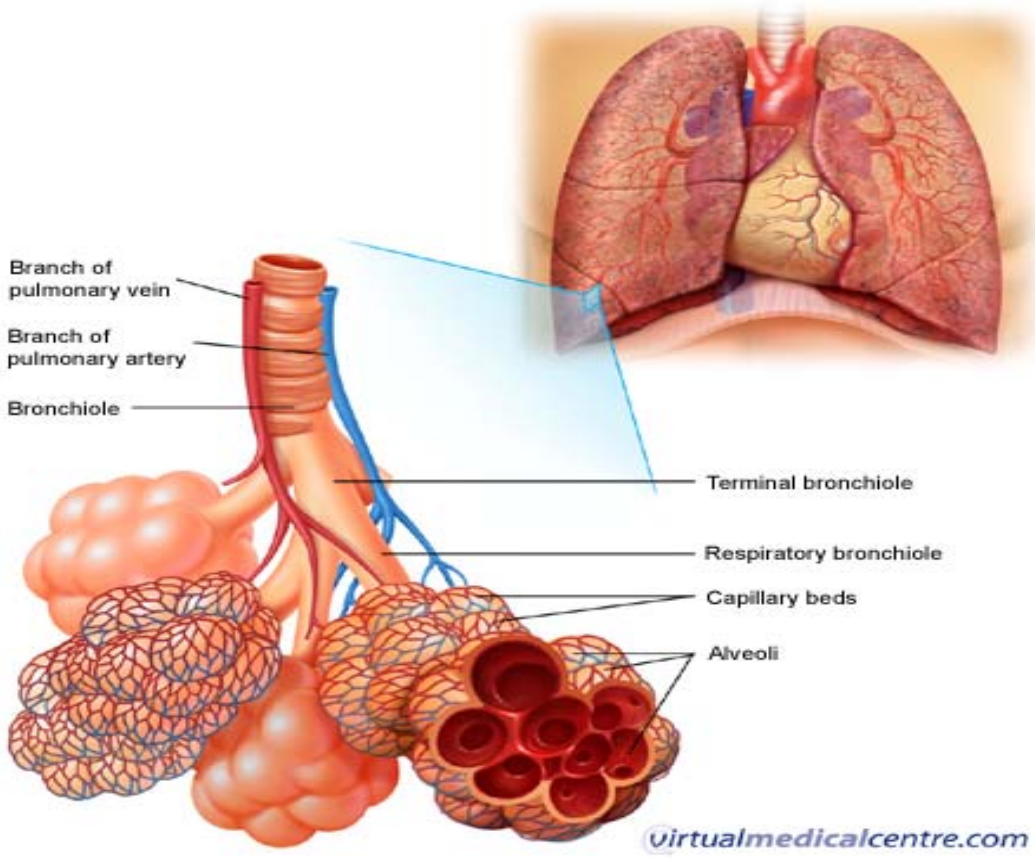

Figure showing areas affected in emphysema.

Etiology of Chronic Obstructive Airways Disease

1) Tobacco use & cigarette smoking (active & passive) –major risk factors.15% of smokers develop COAD.

2) Genetic:

Alpha 1 antitrypsin deficiency is associated with early onset progressive emphysema. This observation made by Laurel & Erickson[11] led to the development of 1st plausible explanation for the pattern of lung destruction seen in Chronic Obstructive Airways Disease.

Alpha – 1- antitrypsin deficiency

- Mutations involving alpha 1 antitrypsin gene.
- Substitution of lysine for glutamic acid.
- Polymerization of alpha 1 antitrypsin as a B-pleated sheet within hepatic mitochondria where it is synthesized.
- No transportation into circulation.
- Young patients affected.
- Predominantly basal panacinar emphysema (smoking- centriacinar emphysema more marked in upper lobes).
- AR disorder

3) Respiratory disease of prematurity, chronic untreated asthma,

childhood malnutrition, and low birth weight may play a role. Data reported by Barker & colleagues[7] shows strong relationship between birth weight & lung function 5-6 decades later.

4) Occupational hazards such as toluene di-isocynate, coal dust (mine field workers) & redwood cedar. Occupational dusts appear to be most significant[53, 54, and 55].

5) Indoor pollution particularly cooking performed in poorly ventilated homes.

6) Bronchial hyper responsiveness-(Dutch hypothesis[8])-Chronic Obstructive Airways Disease & Bronchial asthma form part of a spectrum of illness. The asthma in early life can progress to Chronic Obstructive Airways Disease later. The pathological data argues against this type of overlap which was determined by physiological testing. Long term studies, however, such as the Lung Health Study in USA has reported a high incidence of bronchial hyper reactivity to methacholine in smokers with mild Chronic Obstructive Airways Disease especially in women. Recent results from a large 25 year follow up study in the Netherlands indicate that increased airway hyperresponsiveness, defined by measured response to inhaled histamine, is an independent risk factor for an accelerated decline in FEV1[56].

7) Infections-(British hypothesis [9, 10])–recurrent infections with purulent sputum caused progressive Chronic Obstructive Airways Disease. This hypothesis was largely disproven by Fletcher & Peto.

8) Low socieo economic status[47.]

Pathology:

- Persistent inflammation of airways as evidenced by elevated concentration of TNF alpha and IL 8 in induced sputum from patients with stable Chronic Obstructive Airways Disease & by increase in percentage of macrophages and neutrophils in broncho alveolar lavage specimens and induced sputum & by increased numbers of CD 45 lymphocytes with increased CD8 (T-suppressor) cells in endobronchial &peripheral lung biopsies[12]. The number of cells in airway wall is correlated with the FEV1.

- Oxidative stress[13] caused by Reactive Oxygen Species (ROS) in the cigarette smoke. ROS are normally countered by both endogenous antioxidants (glutathione, uric acid, and bilirubin) & exogenous antioxidants (vit C & vit E in diet). Oxidative stress causes stiffening of neutrophil cell wall which reduces deformability & inhibits passage through pulmonary capillary bed.

- Mucus hyper secretion is caused by hypertrophied mucus glands. Loss of ciliated epithelia & short and thickened cilia cause impaired mucociliary

clearance and lead to accumulation of sputum. Increased number &size of goblet cells and degranulation changes that extend to peripheral airways contribute to peripheral airway plugging[14]

- Imbalance between proteinases like human neutrophil elastase & matrix - metallo protease and antiproteinases like alpha 1 AT causes destruction of lung tissue.

Careful quantitative studies suggest that there is diffuse loss of alveolar surface area proceeding in parallel with the more obvious macroscopic damage so that even normal lung adjacent to the damaged areas may not be free from diseases[15]. This helps to explain the relatively brief (i.e.12-24 months) benefits that follow lung volume reduction surgery where macroscopically damaged areas are removed.

Reid Index:

The ratio of the thickness of the submucosal glands to that of the bronchial wall is expressed as Ried index.

In normal individuals, it is 0.44 $\pm$0.09

In chronic bronchitis, it is 0.52$\pm$0.08

If the submucosal layer thickness is >50% of bronchial wall thickness it is highly suggestive of chronic bronchitis.

High index is commonly associated with symptoms.

Pathophysiology:

- Airflow limitations & increased airway resistance may be caused by loss of elastic recoil during passive exhalation due to emphysema, by increased collapsibility of small airways through loss of aerial traction of airways or to increased resistance due to intrinsic narrowing of small airways. As a direct consequence of altered pressure flow relationship, the work of breathing is increased in chronic bronchitis & emphysema.

- Hyperinflation:

Dynamic hyper inflation contributes additionally to the discomfort associated with airflow obstruction by flattening the diaphragm & placing it at a mechanical disadvantage due to shortened diaphragmatic fiber length.

- Inspired air exchange –small airway narrowing causes a decrease in ventilation of their distal alveolar acini when alveolar capillaries remain intact. This result in mismatching of ventilation & blood flow, decreased ventilation: perfusion ratio &mild to moderate hypoxemia.

Pulmonary vascular and cardiac changes:

There is inflammation in the pulmonary arteries even in early Chronic Obstructive Airways disease like that occurring in the adjacent airways. In severe cases thickening of arterial smooth muscle and reduplication of elastic lamina with extensive pulmonary arterial wall remodeling[14, 15] are present. In patients with persistent day time arterial hypoxemia secondary to Chronic Obstructive Airways Disease right ventricular mass is increased. The extent of right ventricular hypertrophy is not however related to pulmonary arterial pressure measured in vivo rather it is correlated with the base line paO2 experienced during the last year. There is hypertrophy of carotid bodies and increase in the size of glomerular tuft.

Clinical features.

Symptoms:

- Cough with or without expectoration.
- Breathlessness
- Chest pain- may be related to intercostal muscle ischemia.
- Rarely haemoptysis.
- Weight loss in advanced disease.

Signs:

- Markers of smoking such as nicotine staining of fingers and teeth.
- Non specific features of over inflation of the lungs with horizontal ribs, splaying of lower costal margin &widened xiphisternal angle.
- Paradoxical inward movement of lower ribs (Hoover's sign).
- Cardiac dullness reduced.
- Generalized reduction in the intensity of breath sounds –crude bed side indicator of reduced peak expiratory flow.
- Occasional crepitations.
- Expiratory wheezes.
- Pursed lip breathing.
- Elevated jugular venous pressure, pedal edema and tender hepatomegaly in case of right heart failure.

Predominant Bronchitis versus Predominant Emphysema[16]

No		Predominant Bronchitis	Predominant Emphysema
1	General appearance	Mesomorphic; Overweight; Dusky with suffused conjunctiva; Warm extremities.	Often emaciated; Pursed lip breathing; Anxious; Prominent use of accessory muscles; Normal or cool extremities
2	Age in years	40-50	50-75
3	Onset of illness	Cough	Dyspnea
4	Cyanosis	Marked	Slight to none
5	Cough	More evident than dyspnea.	Less evident than dyspnea.
6	Sputum	Copious	Scanty
7	Upper RTIs	Common	Occasional
8	Breath sounds	Moderately diminished	Markedly diminished

9	Cor pulmonale and Right heart failure	Common	Only during bout of RTI and also terminally.
10	Radiography	Normal diaphragmatic position; cardiomegaly; lungs normal or having increased bronchovascular markings.	Small tubular heart; low flat diaphragm; Area of increased radiolucency
11	Course of illness	Ambulatory but constantly on verge of right heart failure and coma	Incapacitating breathlessness punctuated by life threatening bouts of upper RTIs; Prolonged course culminating in right heart failure and coma.

Complications of COAD.

- Secondary polycythemia
- Right heart failure

- Pneumothorax
- Respiratory failure.

Prognosis and predictors of survival.

Chronic Obstructive Airways Disease is a progressive disease and a patient's lung function can be expected to deteriorate over time even with best available care. Although pulmonary hypertension progresses slowly in patients with Chronic Obstructive Airways Disease its presence confers a poor prognosis.

- "Weitzenblum and coworkers":

72% 4 years survival rate in patients with normal pulmonary artery pressure compared with a 49% survival rate in those with an elevated pulmonary artery pressure(mean >20mmHg).

- "Professor Richard Doll & colleagues"[9,18].

Mortality from Chronic Obstructive Airways Disease was 17 times greater in smokers compared with non smokers.

- "Fletcher & Peto Classic study of U.K. postal workers"

Rate of decline of lung function (FEV1) was greater in smokers. Normal loss of FEV1 of 15-30ml/year increases to >60ml/year in current smokers and >40ml/year in ex. Smokers. Not all smokers develop this accelerated loss of

lung function & conventional wisdom holds that only 15-20% of smokers are affected.

- Onset of hypercapnia is often first noted during an exacerbation but when present persistently it carries poor prognosis[19].

- Low body mass index (<19) is an independent risk for premature death[20].

- By the time patients are hospitalized with an exacerbation of COPD, they have a poor prognosis especially if they have developed respiratory failure. In one unselected North American series of patients who were followed post discharge from ICU, one third of patients had died within 12 months[21].

- A study with a 10 year follow up conducted on a cohort of 870 patients with severe Chronic Obstructive Airways Disease[22] concluded that patients with Chronic Obstructive Airways Disease have a high mortality rate from acute respiratory failure, cor pulmonale and lung cancer. Patient's age at the time of diagnosis influences death risk. Patients who need long term oxygen treatment have a higher death risk than those who do not need. The higher the FEV1 or paO2 at the time of diagnosis, the lower the death risk.Patients who need and use long term oxygen treatments have a lower death risk than those who need it but do not use it properly. Patients with a partial

reversible airway obstruction who regularly attend the clinic for planned check ups have a lower death risk than those who have the same characteristics but do not show adherence to the care program.

- In another study of 166 patients treated with long term oxygen therapy, the overall survival rates were 78.3 and 67.1 percent at 2 and 3 years, respectively[23].

- A multi variate analysis showed an independent predictive value for right ventricular systolic pressure, age and FEV1[24].

- Once endotracheal intubation is necessary, the prognosis is usually poor and the survival after 1 year is usually lower than 40%[25].

- Pulmonary embolism is a common cause of death with the frequency estimated to be approximately 11 % among patients with Chronic Obstructive Airways Disease in the intensive care unit. **Pulmonary embolism was the most frequent cause of death, at 40.6%[26]**

Investigations:

Respiratory function tests:

These measure the degree of airflow obstruction during forced expiration

Spirometry:

- Robust standard is dry bellows Spirometry such as vitallograph.
- Cheaper alternative - Turbine wane & pneumotachograph – both measure flow and convert it into volume.
- Normal curve has a brisk flow near total lung capacity, then a steady decline down to residual volume. Inspiratory limb is a smooth semicircle.
- With the development of disease in small airways the expiratory limb begins to dip at lower volumes.
- When a large degree of emphysema is present there is a characteristic flow volume loop with a sharp drop in flow after the initial peak as the pressure in the thorax collapses the airway.
- In normal subjects there is little or no difference between the relaxed &forced vital capacity.
- In emphysema forced vital capacity is less than relaxed vital capacity.
- During measurement of FVC the procedure must continue until all possible volume is added.

Staging of severity of COAD

Staging	%Predicted FEV1		
	British thoracic society	European respiratory society	American thoracic society
Mild	60-79%	≥ 70%	≥50%
Moderate	40-59%	50-69%	35-49%
Severe	<40%	<50%	<35%

A ratio of FEV1/ FVC is termed forced expiratory ratio.FEV1 <80% and FER <70% indicates COAD.

Transfer factor:

The transfer factor test for carbon monoxide measures the ability to move carbon monoxide from inhaled gas to hemoglobin in the alveolar capillaries. Carbon monoxide is used as a surrogate for oxygen.

The gas mixture inhaled contains a small amount of the inert gas helium. The helium concentration in the expired gas is lower than in inspired gas because of dilution with the residual gas in the lungs.

Carbon monoxide is reduced because of this dilution &transfer across the alveolar capillary membrane into the blood. The combination of the two allows an estimate of the amount of gas transfer into blood.

The result is expressed either as carbon monoxide transfer factor (TLCO) for the lungs or as transfer factor per unit volume accessed (diffusion coefficient or kCO). The kCO adjusts for the size of the lung accessed by the inspired gas, for example a large pleural effusion will reduce lung volume & TLCO but not the kCO since gas transfer is normal within the lung accessed by the inspired gas.

Asthma may reduce TLCO when severe but kCO tends to be high in asthma. Emphysema with its destruction of alveolar capillary membrane results in large air space, decreased area for gas transfer & reduction of TLCO & kCO.

In Chronic Obstructive Airways Disease, the reduction in kCO is a guide to the extent of emphysema. Other restrictive conditions such as fibrosing alveolitis reduce kCO but within the obstructive lung conditions, reduced kCO is a marker for emphysema. There is good direct & indirect evidence that the severity of emphysema is well related to the reduction in TLCO although this relationship is lost when their FEV1 falls below 1 & TLCO becomes more difficult to measure.

Body Plethysmography:

The helium dilution method & the body plethysmograph method can produce different values for lung volumes since the body plethysmograph measures all the gas within the thorax that is subject to the pleural pressure changes.

The helium dilution only measures the volume accessible to inspired air. Poorly ventilated areas such as emphysematous bullae will be included in the plethysmograph volume but may not show on the helium dilution.

This difference, the trapped gas has been used as an estimate of extent of lung damage in Chronic Obstructive Airways Disease. After administration of bronchodilators the over inflation in Chronic Obstructive Airways Disease may be reduced.

This can occur in the absence of significant change in spirometry. Such deflation can reduce symptoms of breathlessness or increase exercise tolerance & can explain subjective benefits found in the absence of any change in spirometry

Blood Gases& Oximetry:

In the chronic stable situation blood gas examination is necessary to guide the provision of long term oxygen treatment. In acute exacerbations,

blood gas analysis helps to evaluate severity, prognosis & the need for oxygen treatment or respiratory support by invasive or non invasive means.

Blood gas analysis is still the only convenient way of assessing paCO2 & should be considered in any patients with an FEV1 below 1.21 or 40% of the predicted value or whose oxygen saturation is less than 92%.

Oximetry provides a measure of oxygen saturation & a portable pulse oximeter is a very useful tool. The relationship between saturation & paO2 is given by the oxygen saturation curve. Oximeter transmits light as set wavelengths through tissue in the finger or ear lobe. Saturated & desaturated hemoglobin absorbs the wave lengths differently & allow a calculation of the percentage of hemoglobin that is saturated. It is accurate in most routine clinical situations although less reliable at saturation below 75% .When there is poor peripheral perfusion the signal may be inadequate or the reading inaccurate. In some circumstances, other pigments such as nail varnish or carboxyhemoglobin produce inaccurate results.

Pulse oximetry provides a very special useful measurement. It is important to remember however that it measures only the oxygen level & doesn’t provide a measurement of the level of carbon dioxide. In acute

exacerbations, patients receiving supplemental oxygen may have marked carbon dioxide retention with inappropriately reassuring oxygen saturation.

In acute exacerbations requiring admission to hospital, baseline arterial blood gas analysis is usually necessary.

Assessment of blood gas is an essential part of the criteria for provision of long term home oxygen. If the baseline oxygen saturation of breathing air is above 92% then arterial pO2 is not likely to meet the criteria for long term home oxygen. Oximetery therefore provides a useful screening test to see whether it is appropriate to go on to measure arterial blood gas.

X-Ray Chest:

- Emphysematous changes
- Increased pulmonary vascular markings
- Pulmonary artery dilatation
- Cardiomegaly

Detection of pulmonary hypertension in COAD.

X-Ray Chest

Chest radiography is poorly sensitive in the diagnosis of pulmonary hypertension.

Electrocardiographic findings in Chronic Obstructive Airways Disease[27].

- P pulmonale- P wave amplitude > 2.5 mm in standard leads together with right p wave axis.
- Right QRS axis deviation directed to the region of +90 degrees clockwise to +150 degrees.
- Left QRS axis deviation in 10% of cases.
- SI, SΠ, SIII syndrome – prominent S waves in standard leads I, Π, and III.This indirectly reflects posterior displacement of the apex. Rather wide and slurred S wave may appear in all three standard leads & leads V4 to V6.
- Complete/ incomplete right bundle branch block.
- R/s >1 in V1 or R wave amplitude in V1 >5 mm.
- r/S < 1 in v6 or r wave in V6 < 5mm,trasition zone is frequently displaced to lead V6 or even further to left.
- Small QS or w shaped complexes in right precardial leads.
- Diminution of QRS magnitude in all the precardial leads. Small r waves may completely disappear in right precardial leads.
- T wave - decrease in amplitude in all leads, may be inverted in right precardial leads.

These abnormalities are usually less pronounced in patients with Chronic Obstructive Airways Disease than in patients with other forms of pulmonary hypertension because of modest degrees of pulmonary hypertension that occurs and because of effects of hyperinflation. ECG changes have a good specificity (85%) but their sensitivity is poor (40%), particularly in patients with mild pulmonary hypertension[65].

Echocardiographic findings of pulmonary hypertension in Chronic Obstructive Airways Disease[23, 31].

Poor window due to over inflation and sub optimal images due to marked respiratory variation in intra thoracic pressure make the performance of ECHO in Chronic Obstructive Airways Disease patients a difficult job. Although the correlation between systolic pulmonary artery pressure measured by echocardiography and that measured at the time of cardiac catheterization was good 52% of pressure measurements were found to be inaccurate. There was more than 10mm Hg difference between the two. Further > 48% of patients were misclassified as having pulmonary hypertension by echocardiography

- Increased pulmonary artery pressure.
- Tricuspid regurgitation.

- Right ventricular wall thickening.
- Increased right ventricular volume.
- Reversed movement of the interventricular septum.
- Enlargement of right atrium and ventricle.
- Normal or small left ventricular dimensions.

Echocardiographic findings that portend a poor prognosis include pericardial effusion, right atrial enlargement and septal displacement.

Measurement of pulmonary artery pressure by echocardiography.

None of the echocardiographic techniques measure the intravascular pressure directly. The use of Doppler echocardiography for determining a pressure involves the use of tricuspid regurgitant jet and the Bernoulli equation. By determining the right ventricular outflow tract one can determine the pulmonary artery systolic pressure. This technique is probably the most accurate for quantification of pulmonary artery pressure.

Doppler recordings of the pulmonary artery velocity can also provide an assessment of pulmonary artery pressure and pulmonary vascular resistance. The measurements that have been used include the pre ejection period which is the time interval from the onset of electrocardiographic QRS to the onset of pulmonary artery systolic flow, the acceleration time which is the time between

the onset of flow to the peak systolic flow and the ejection time which is the interval from the onset to the cessation of flow.

Echo Doppler techniques have become the non invasive standard to detect pulmonary hypertension[68, 70]. These techniques are relatively accurate when pulmonary artery pressure (PAP) is above 30mmHg, but they do not detect milder pathologic pulmonary hypertension. Echo Doppler is useful for longitudinal follow up of pharmacologic treatment of pulmonary hypertension & cor pulmonale. In a recent study the bias of Doppler ECHO in the measurement of systolic PAP compared with right heart catheterization(RHC) was 2.8 mm Hg which is high when one takes into account the modest level of PHT in most COAD patients[71].

B-Type natriuretic peptide (BNP):

B- Type natriuretic peptide (BNP) release is due to increased wall stretch of atria and may have a relatively good sensitivity and specificity for the identification of pulmonary hypertension in COAD patients[66, 67]. However larger studies are needed to determine the benefit of BNP plasma level in the diagnosis of pulmonary hypertension in COAD.

Right heart catheterization.

Right heart catheterization is the **gold standard** for diagnosis of pulmonary hypertension[68, 69]. It allows the direct measurement of pressure in the right atrium, the right ventricle, the pulmonary artery and in the wedge position to estimate left heart filling pressures. RHC is generally carried out using a Swan- Ganz catheter type. RHC has two drawbacks: First it is an invasive procedure having some risk. Next it needs hospitalization which may be inconvenient for patients.

Predominant Bronchitis versus Predominant Emphysema[72].

SI NO		Predominant bronchitis	Predominant emphysema
1	FEV1/FVC	Reduced	Reduced
2	FRC	Mildly increased	Markedly increased
3	TLC	Normal or slightly increased	Considerably increased
4	RV	Moderately increased	Markedly increased
5	Lung compliance	Normal or low	Normal or low
6	Recoil pressure	Normal or high	Low

7	MVV	Moderately decreased	Markedly decreased
8	Airway resistance	Increased	Normal or slightly increased
9	DLCO	Normal or low	Low
10	Arterial Po2	Moderately to severely reduced	Slightly to moderately reduced
11	Arterial hypercapnea	Chronic	Only during acute respiratory infection
12	Hematocrit	Generally high, may reach 70%	Normal or slightly high, rarely above 55%
13	Pulmonary artery pressure	Generally increased	Normal or slightly increased

Management:

The overall approach to the management of stable Chronic Obstructive Airways Disease should revolve around a stepwise increase in treatment, depending on the severity of the disease. Disease severity is determined by the severity of symptoms and air flow limitation as well as other factors, including the frequency and severity of exacerbations, complications, respiratory failure, and comorbid factors, including cardiovascular disease and sleep –related

disorders, in addition to the general health status of the patients. Patient's education is paramount to effective treatment of COAD.

Smoking cessation:

The Lung health study reported that patients who stopped smoking had a small improvement in FEV1 (57ml) after 1 year. Thereafter the rate of decline in lung function is similar to that of age matched non smokers[28].

Numerous studies indicate that nicotine replacement therapy in any form (gum, inhaler, nasal spray, transdermal patch, sublingual tablets, or lozenges) reliably increases long term smoking abstinence rates[29]. The anti depressants bupropion or nortriptyline have also been shown to increase long term smoking cessation rates, although fewer data are available[30].

Pulmonary rehabilitation:

The goals of pulmonary rehabilitation in COAD patients are to reduce symptoms, improve quality of life, and increase physical and emotional participants in everyday activities. Although a large study of 200 patients with disabling Chronic obstructive airways disease demonstrated no difference in hospital admission among the patients randomized to receive rehabilitation versus the control patients, the rehabilitation group showed greater

improvement in walking ability and general and disease –specific health status[32].

Pharmacological treatment:

Bronchodilators and corticosteroids:

Bronchodilators are used as either during exacerbations of COAD or on a regular basis to prevent or reduce symptoms. A combination of short acting beta 2 agonist and an anti cholinergic agent in stable chronic obstructive airways disease patients produces greater sustained improvements in FEV1 than either agent alone and does not produce evidence of tachyphylaxsis over 90 days of treatment.

Prolonged treatment with corticosteroids does not modify the long term decline in lung function in patients with Chronic obstructive airways disease and primarily be used in those who have a documented spirometric response to inhaled corticosteroids[33].

Antibiotics:

Antibiotics are very useful during exacerbations of chronic obstructive airways disease.

Vasodilators:

The use of vasodilators has been disappointing in the treatment of chronic obstructive airways disease patients even those with pulmonary hypertension. Because of potential for worsening ventilation-perfusion mismatch vasodilators may even worsen hypoxemia.

Digoxin:

Data recording the use of digoxin in chronic obstructive airways disease patients is insufficient to make recommendations although short term intravenous digoxin improved cardiac output and reduced circulating nor epinephrine levels in patients with right ventricular dysfunction[34].

Vaccination:

Influenza and pnuemococcal vaccines are recommended.

Alpha 1 anti trypsin deficiency:

Weakly or monthly injections of alpha 1 anti trypsin.

Long term home oxygen

In key clinical trials long term oxygen therapy clearly improved the survival of hypoxemic patients with chronic obstructive airways disease[35, 36]. The British study[37] compared the effect of treatment with oxygen approximately for 15 hours per day with the effects of no oxygen therapy. The

NIH study[35] compared nocturnal oxygen therapy (about 12 hours per day) to continuous oxygen therapy atleast 19 hours per day .Oxygen therapy was beneficial in both studies. Continuous therapy is more effective than nocturnal therapy alone.

Haemodynamic effects of oxygen:

1) Oxygen relieves pulmonary vasoconstriction, decreasing pulmonary vascular resistance and thus enabling the right ventricle to increase the stroke volume.

2) Oxygen therapy improves arterial oxygen content, providing enhanced oxygen delivery to the heart, brain and other vital organs. These two hypotheses are not mutually exclusive, and each one has supporting evidence.

I

Indications for Home Oxygen[38]:

Absolute PaO2 < 55mm Hg or SaO2 <88%
PaO2 55-59 mmHg or SaO2 = 89% in the presence of dependent edema, P pulmonale on the ECG, erythrocytosis (hematocrit >56%)
Specific situations During exercisePaO2 < 55mmHg or SaO2 <88% with minimal exertion

During sleep PaO2 <55mmHg or SaO2 <88 % with associated complications like PHT, excessive day time sleepiness and cardiac arrthymias.

Noninvasive ventilation:

Noninvasive positive pressure ventilation has been reported to improve gas exchange, sleep efficiency, quality of life and functional status in patients with restrictive lung disease and chronic respiratory failure; however its usefulness in patients with chronic obstructive airways disease is not as well established. Uncontrolled studies have demonstrated that non invasive positive pressure ventilation used at home may improve oxygenation and reduce hospital admissions in patients with severe chronic obstructive airways disease and hypercapnea and improve long term survival, although large controlled clinical trails are now needed[39]. The combination of noninvasive positive pressure ventilation and long –term O2 therapy may be more effective[40], but again large trails are needed before this approach can be recommended.

Lung volume reduction surgery:

Volume reduction surgery, which was originally described by Brantigan, has been advocated in selected patients with advanced emphysema. The surgical technique involves removing 20-30 % of the volume of each lung by

means of sternotomy, sequential thoracotomy, or thoracoscopy to reduce the severe hyperinflation commonly seen in patients with severe chronic obstructive airways disease.

A randomized trail comparing the results of lung volume reduction with medical therapy for severe emphysema has been completed[41]. A total of 1218 patients with severe emphysema who underwent pulmonary rehabilitation were randomly assigned to undergo lung volume reduction surgery or to receive continued medical therapy. An interim analysis determined that patients with a FEV1 of less than 20 % of predicted and either homogenous distribution of emphysema on CT scan or carbon monoxide diffusing capacity that was 20% or less of the predicted value were at high risk for death after lung volume reduction surgery with a low probability of functional benefit[42.] Among patients with predominantly upper lobe emphysema and low exercise capacity, the mortality rate was lower in the surgery group than in the medical therapy group. Among patients with non –upper lobe emphysema and high exercise capacity, the mortality rate was higher in the surgery group than in the medical therapy group. Lung volume reduction is a palliative procedure that does not halt, but only slows, the rate of functional decline for COAD.The disease will still progress, and symptoms may get worsened.

Lung Transplantation:

Chronic obstructive airways disease is the most common indication for lung transplantation world wide. In 1995, approximately 60% of single lung and 30 % of bilateral lung transplantations were performed on patients with chronic obstructive airways disease[43]. Lung transplantation is a viable treatment option in patients with advanced pulmonary parenchymal or pulmonary vascular disease who have exhausted medical management.

Both single lung transplantation and bilateral lung transplantation result in significant improvement in post operative lung function, exercise capacity, and quality of life[44]. The choice of the procedure needs to be individualized. In general, single-lung transplantation is used for emphysema because of the scarcity of organ donars, lower perioperative morbidity and mortality rates, and comparable improvement in exercise capacity compared with bilateral lung transplantation[45]. However postoperative spirometry, single breath diffusing capacity, and arterial oxygen tension are all significantly higher in bilateral lung transplantation compared with single lung transplantation, which may benefit young patients with emphysema.

STUDY DESIGN

Aim:

To study Electrocardiographic and Echocardiographic changes in patients with “Chronic Obstructive Airways Disease”.

Materials and Methodology:

Population:

Patients attending the out patient clinic &who were admitted in “Government Royapettah Hospital, Chennai” with complaints of persistent cough with expectoration for 3 months in two consecutive years or who have cough, breathlessness with clinical or x-ray evidence of hyper inflation were selected for the study (50 patients).

Period of the study:

January 2008 –July 2008.

Inclusion criteria:

Patients with spirometric evidence of chronic obstructive airways disease.

Criteria for diagnosis of chronic obstructive airways disease - FEV1<80% predicted value & FEV1/FVC < 70%.

Exclusion criteria:

Patients with TB, bronchial asthma, bronchiectasis, bronchogenic carcinoma, interstitial lung disease, coronary artery heart disease, systemic hypertension, aortic & mitral valve disease, right heart failure& patients residing at high altitude were excluded from the study by doing x-ray chest, Mantoux test, sputum for acid fast bacilli & blood pressure measurement.

Performance of spirometry:

Spirometry was done according to the guidelines published by both the British Thoracic Society (BTS) with the Association of Respiratory Technicians & Physiologists (ARTP) & the American Thoracic Society. According to these guidelines production of reliable results of spirometry is very dependent on the person performing the test. They must have the ability to encourage the patients to perform a maximal forced exhalation.

The patients were demonstrated the maneuver first. They were seated in a comfortable stool. They were asked to take deep full inspiration. Good seal was maintained with the mouth piece of spirometry.With maximum effort exhalation was continued for atleast 6 seconds and to a maximum of 15 seconds. The results of the measurement were accepted only if the trace is smooth & cough free. The maneuvers were

done thrice for each patients & the best FEV1 & the best FVC were recorded. Reversibility test with inhaled salbutomal was done to rule out bronchial asthma (reversibility more than 15%).

ECGs were taken and coronary artery heart disease was ruled out.

ECG evidence of pulmonary hypertension in chronic obstructive airways disease.

- P pulmonale
- Right QRS axis deviation
- SI, SΠ, SΙΙΙ syndrome
- Diminution of QRS magnitude in all the precardial leads.
- R/s >1 in V1 or R wave amplitude in V1 >5mm.
- r/S <1 in V5 or V6 or r wave in V6 <5mm.
- Complete/incomplete right bundle branch block.

Echocardiographic evidence of pulmonary hypertension in COAD

- Enlargement of right atrium and ventricle.
- Normal or small left ventricular dimensions.
- Thickened interventricular septum.
- Reversed septal motion.

- Tricuspid regurgitant velocities –contrast enhancement with saline improves accuracy.
- Doppler Echocardiographic quantification of right ventricular systolic hypertension can be obtained by measuring the velocity of the tricuspid regurgitant jet and using the Bernoulli formula.

OBSERVATIONS

Age distribution:

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
AGE	50	50.7000	8.14724	1.15219

Age group	Frequency	Percentage
< 50 years	30	60
51 -60 years	16	32
>61 years	4	8

Chronic obstructive airway disease is most prevalent in persons aged <50 years i.e., 60 % of patients are <50 years of age.

Sex distribution:

Sex group	Frequency	Percentage
Males	49	98
Females	1	2

Chronic obstructive airways disease is most prevalent in males.

Occupation details:

occupation	Frequency	Percentage
Manual laborers	34	68
Factory workers	1(cement factory worker)	2
Shop owners	4	8
Office staffs	10	20
Home makers	1	2

Chronic obstructive airways disease is most prevalent in manual laborers i.e. 2/3 rd of patients are manual laborers.

Socio economic status:

Income group	Frequency	Percentage
Low socio economic group	36	72
Middle class people	14	28

Nearly 3/4th of chronic obstructive airways disease patients are from lower socio economic group.

Smoking pattern:

Smoking pattern	Frequency	Percentage
Smokers	36	72
Non smokers	14	28

72% of chronic obstructive airways disease patients are smokers.83% of smokers (30 persons) still continue to smoke.

Smoking intensity:

Smoking intensity (No of cigarettes / day)	Frequency	Percentage
Non regular smokers	3	8
<5	10	28
6-10	10	28
11-20	6	17
> 20	7	19

64% of smokers smoke <10 cigarettes per day.19% of smokers smoke more than 20 cigarettes per day.

Age at which started smoking:

Age at which started smoking	Frequency	Percentage
< 20 years	18	50
21-30 years	15	42
>31 years	3	8

Most of the patients started smoking at earlier ages that is before 20 years of age.

Air pollution

82% (41 persons) of chronic obstructive airways patients give H/O living in damp housing as well as exposed to passive smoking. One person works in cement factory.

Analysis of etiological factors

Etiology	Frequency	Percentage
Smoking only	5	10
Air pollution only	11	22
Smoking +air pollution	31	62
No aetiology given	3	6

62% of patients give history of exposure to both smoking and air pollution.6% of patients give history of no exposure to risk factors.

X-ray chest findings:

x-ray chest findings	Frequency	Percentage
Emphysematous changes	22 34	44 68
Increased pulmonary vascular markings.	6	12
Pulmonary artery dilatation cardiomegaly	3	6

Pulse oximetry:

SpO2	Frequency	Percentage
More than 88% <87%	42 8	84 16

84% of patients showed no evidence of hypoxemia.16% of patients had pulse oximetry evidence of hypoxemia.

Interpretation of pulmonary function test:

FEV1/FVC	Frequency	percentage
70-61	31	62
60-51	12	24
<50	7	14

One-Sample Statistics

	N	Mean	Std. Deviation	Std. Error Mean
PFT FEVI	50	1.2040	.17723	.02506
PFT FVC	50	2.0280	.16418	.02322
FEV1/FVC	50	59.0400	5.17829	.73232

Interpretation of ECG:

ECG findings	Frequency	Percentage
P pulmonale	17	34
QRS right axis deviation	14	28
Right bundle branch block	6	12
R/s > 1 in V1	4	8
r/s <1 in V6	3	6
Poor progression of R wave in right precordial leads.	5	10
↓ Twave amplitude in right precordial leads.	2	4

ECG evidence of pulmonary hypertension:

ECG evidence of pulmonary hypertension	Frequency	Percentage
Present	23	46
absent	27	54

ECHO evidence of pulmonary hypertension:

ECHO evidence of pulmonary hypertension	Frequency	Percentage
Present	22	44
Absent	28	56

ECHO findings in PHT

ECHO findings	Frequency	Percentage
Elevated PAP	22	44
Tricuspid regurgitation	6	12
Right atrial dilatation	4	8
Pericardial effusion	1	2

Severity of pulmonary hypertension:

Pulmonary hypertension	Frequency	Percentage
Severe(>50mm Hg)	1	2
Moderate(31-50mmHg)	7	14
Mild(<30mmHg)	14	28

Age group * ECHO evidence pulmonary hypertension:

Age group	No Echo E/O PHT	Echo E/O PHT	Total
<50 years	19	11	30
50-60 years	6	10	16
>60 years	3	1	4
Total	28	22	50

p =o.177 not significant

Smoking * ECHO evidence:

Smoking	ECHO evidence of PHT		Total
	Absent	Present	
Absent	13	1	14
Present	15	21	36
total	28	22	50

p= 0.001 significant

Air pollution *Echo evidence of PHT:

Air pollution	Echo evidence of PHT		Total
	Absent	present	
Absent Present	4 24	4 18	8 42
total	28	22	50

p=0.709 not significant

Smoking intensity *Echo evidence of PHT

Smoking intensity(cigarettes /day)	Echo evidence of PHT		Total
	Absent	Present	
No smoking	13	1	14
<5	7	6	13
6-10	6	4	10
11-20	2	4	6
>20	0	7	7
Total	28	22	50

p=0.001 significant.

ECG evidence*ECHO evidence of PHT:

ECG evidence of PHT	ECHO evidence of PHT		Total
	Present	Absent	
Present	18	5	23
Absent	4	23	27
Total	22	28	50

p- Not significant.

Pulmonary artery catheterization is the gold standard for measurement of pulmonary artery pressure. If we take Doppler echocardiogram measurement of pulmonary arterial pressure as the standard procedure, sensitivity and specificity of ECG evidence of pulmonary hypertension can be measured by Wilson score.

Screening test evaluation:

Parameters	Estimates	Lower –upper 95% CI's
Sensitivity	81.82%	(61.48,92.69) Wilson score
Specificity	82.14%	(64.41,92.12) Wilson score
Positive predictive value	78.26%	(58.1,90.34) Wilson score
Negative predictive value	85.19%	(67.52,94.08) Wilson score
Diagnostic accuracy	82%	(69.2,90.23) Wilson score
Likelihood ratio of positive test	4.589	3.022-6.947
Likelihood ratio of negative test	0.2213	0.1331-0.3681
Diagnostic odds	20.7	4.844-88.45

Electrocardiography has high sensitivity and specificity in the detection of pulmonary hypertension.

DISCUSSION

In our study chronic obstructive airways disease is present almost exclusively in males. This is due to increased prevalence of smoking in males. According to literatures after standardization for smoking males are at high risk than females[52].

Most of the chronic obstructive airways disease patients start smoking at an earlier age especially in teen years. Normally lung growth is maximal by the late teenage years, and lung function is relatively constant until the late 20s. Smoking teenagers do not achieve the peak value of their non smoking contemporaries and begin to lose lung function as soon as growth ceases.

ECG and ECHO evidence of pulmonary hypertension is more common in smokers who smoke more than 20 cigarettes/day than those smoke less or those who do not smoke.

The role of smoking in the pathogenesis of chronic obstructive airways disease received considerable attention in the 1960s. By the late 1970s, it was clear that the earliest lesions demonstrable in smokers affected the small airways- i.e., small bronchi and bronchioles[48, 49].

The harmful effects of smoking can be manifested primarily by catarrh as well as by limitation of flow. Of these two types, the catarrhal is more common; it occurs in about 80% of smokers. Also, the volume of sputum

production is not directly related to limitation of airflow[50, 51.] In some persons, the predominant effect of smoking may be hypertrophy of mucous glands and hyper secretion; in others, it may damage small airways, destroy alveolar walls, and modify elastic recoil of the lungs.

In a 40 year follow up of British physicians, the annual mortality per 100,000 from chronic obstructive airways disease was 10 for those who never smoked and 225 for those who smoked more than 25 cigarettes daily; rates were intermediate for former smokers and those who smoked fewer than 25 cigarettes per day[57].Many people with a significant number of pack –years still have a normal or near normal FEV1 while some people have reduced FEV1with relatively modest smoking history. Whether low tar cigarette brands produce less severe chronic obstructive airways disease (analogous to bronchogenic carcinoma) is unclear. Smokers are not a reliable source of information about their own smoke inhalation, and there is no simple means of quantifying smoking patterns to determine the connection between patterns of smoke inhalation and development of chronic obstructive airways disease. It's not possible to predict which 15% of smokers go on to develop chronic obstructive airways disease.

Chronic obstructive airways disease is more common in persons around 50 years of age. This is attributed to age related decline in FEV1.

Chronic obstructive airways disease is more common in manual workers of which most of them have been exposed to smoking since teen years. The high prevalence of smoking among workers has been a major confounding factor.

72% of chronic obstructive airways disease patients are from lower socio economic status. These may be related to the possible low birth weight (Barker hypothesis), childhood malnutrition or damp housing facilities.

In our studies16% of patients have pulseoximetry evidence of hypoxemia. According to American Thoracic Society they are candidates for long term home oxygen.

In our studies 46% of patients has ECG evidence of right ventricular strain .34% of persons have ECG evidence of p pulmonale. According to ATS they are candidates for long term home Oxygen at a higher saO2 level.

Studies involving ECG evidence of pulmonary hypertension in COAD patients

According to literatures [58] many patients have ECG changes suggestive of RV strain but didn't manifest RV strain by Echocardiography.

Fishman-standard criteria for right ventricular hypertrophy in ECGs were absent in 2/3rd of chronic obstructive airways disease patients who had right ventricular hypertrophy on post mortem examination[59].

ECG of prognostic importance in severe chronic obstructive airways disease has been outlined by Kok-Jensaw[60].In a study of 288 patients survival was very poor in individuals with a QRS axis of +90 degrees to +180 degrees and amplitude of p wave in lead Π of 2mm or more. Only 37% & 47% respectively of patients with these changes were alive after 4 years.

In V.K.Singh and S.K.Jain study[61] of 130 patients of chronic obstructive airways disease effect of airflow obstruction on ECG findings such as p>2.5mm, QRS axis equal or more than +90 degrees, R wave inV6 less than or equal to 5mm, R/S ratio in V5, V6 equal to or less than 1 shows a negative correlation with FEV1/FVC ratio. Other features like T wave amplitude, negative "p" wave in avL, S wave more than 5mm in depth in V5, V6 and SI, SΠ, SIII patterns were observed less frequently and correlate weakly with severity of disease.

In a study involving 50 plantation workers attending General Hospital, Tata Tea Ltd, Munnar, Kerala good correlation between severity of COAD and number of abnormal ECG manifestations was present[62].

In a case control study involving 50 cases& 50 controls age and sex matched ECG features of RAD and RV strain, and ECHO features of right chambers and pulmonary artery enlargements were compared. According to the study sub clinical cor pulmonale and severity of right sided haemodynamic disturbances can be measured accurately[63].

In a study involving 25 COAD patients of R G Kar medical college, Calcutta, ECG and ECHO findings were compared. ECG evidence of RVH i.e. P pulmonale, right axis deviation, RBBB, clockwise rotation was present in 50% of patients. ECHO evidence of right ventricular hypertrophy and elevated pulmonary artery pressure were found in 85% of cases[64].

In our study 44% of patients have ECHO evidence of elevated pulmonary artery pressure.4 patients had right artrial dilatation.1 patient had pericardial effusion. Both right atrial enlargement and pericardial effusion show poor prognosis.

Prevention is better than cure.

Primordial prevention.

COAD is more prevalent in smokers: more in those who smoke more than 20 cigarettes/ day: more in those who started smoking in teen years. So health education regarding ill effects of smoking should be given to public especially school children.

Primary prevention.

Avoidance of smoking.

Wearing protective equipments during working hours in dusty occupation.

Secondary prevention.

Early diagnosis of COAD by clinical methods, chest x-ray and pulmonary function test.

Tertiary prevention.

Treatment of acute exacerbations and early detection of pulmonary hypertension by ECG and ECHO. Long term home oxygen is the only proven treatment to arrest pulmonary hypertension. Arterial blood gas analysis is very useful in the selection of candidates for long term home oxygen therapy. If the blood gas analysis facility is not available pulse oximetry can be employed.

CONCLUSION

- In my study Chronic obstructive airways disease is more common in males.
- Smoking is the principle cause of Chronic obstructive airways disease.
- Severity of Chronic obstructive airways disease is directly proportional to intensity of smoking.
- Severity of Chronic obstructive airways disease is associated with earlier age of onset of smoking especially in teen ages.
- Most patients are in low socioeconomic status.
- Electrocardiography has high sensitivity in detecting pulmonary

hypertension in Chronic obstructive airways diseased patients as evidenced by P pulmonale,right axis deviation,right ventricular hypertrophy .Majority of the patients with electrocardiographic evidence of pulmonary hypertension also had Echocardiographic evidence of elevated pulmonary artery pressure. So both Electrocardiography and Echocardiography are very useful in the assessment of severity of chronic obstructive airways disease.

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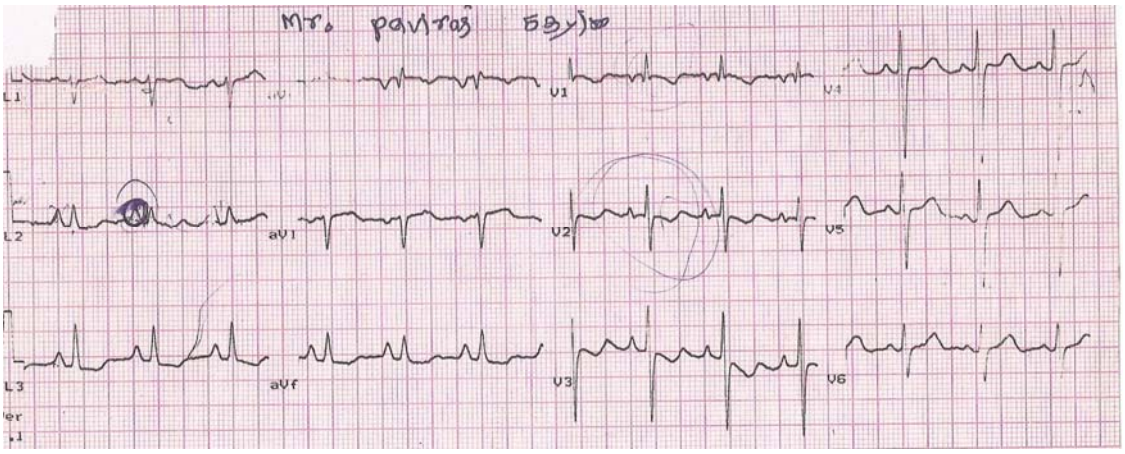
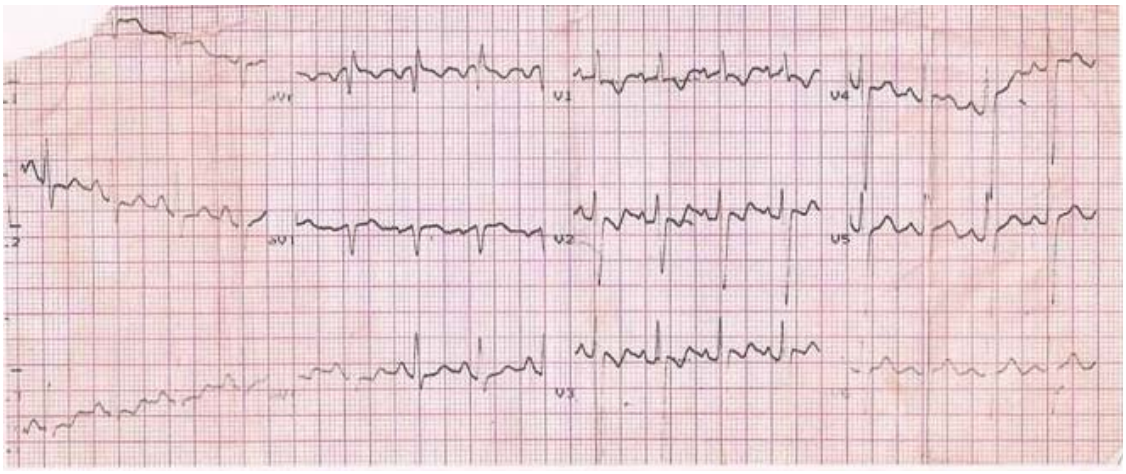
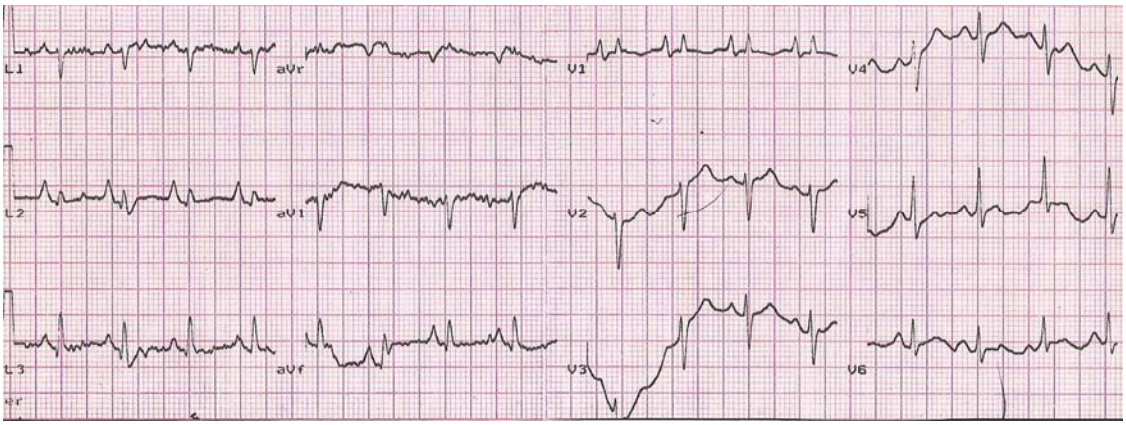


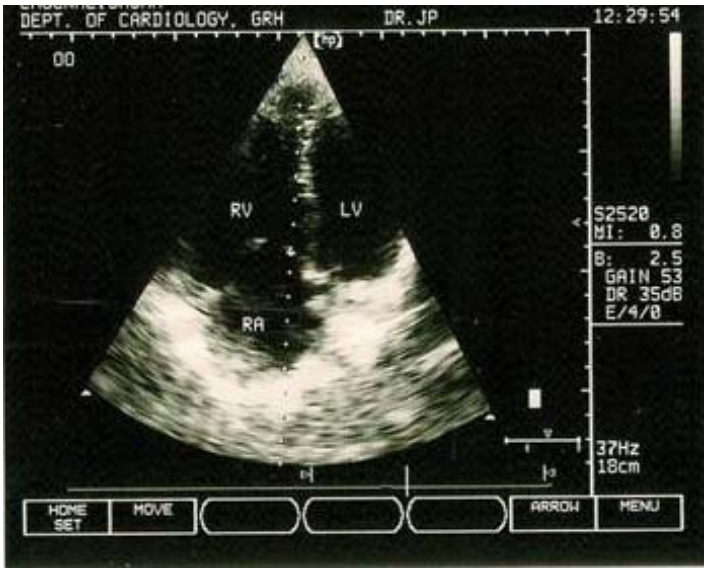
ECG showing P pulmonale, right axis deviation, RVH with strain (R/s > 1 in V1,r/S <1in V5,V6),QR in avR, QR in V1(right atrial enlargement).



ECG showing P pulmonale, RAD, RVH with strain(R/s> 1 in V1,r/S <1in V5,V6), QR in avR.

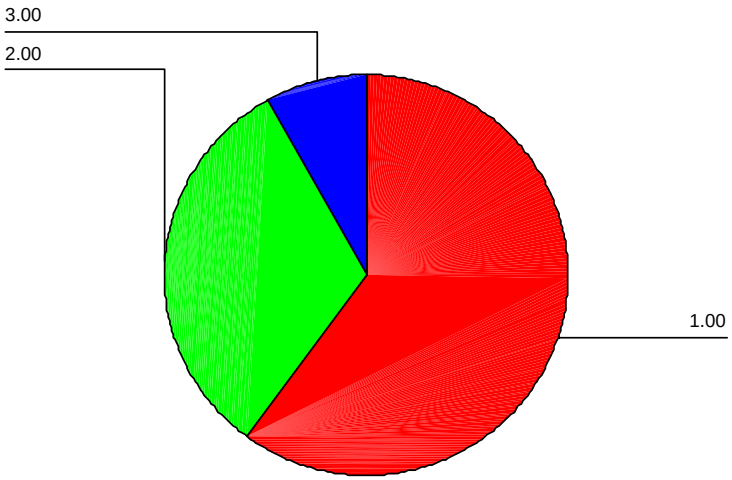


ECG showing P pulmonale, RAD, QR in avR, R/s >1 in V1.



Echo showing right atrial and right ventricular dilatation in a COAD patient.

AGE GROUP

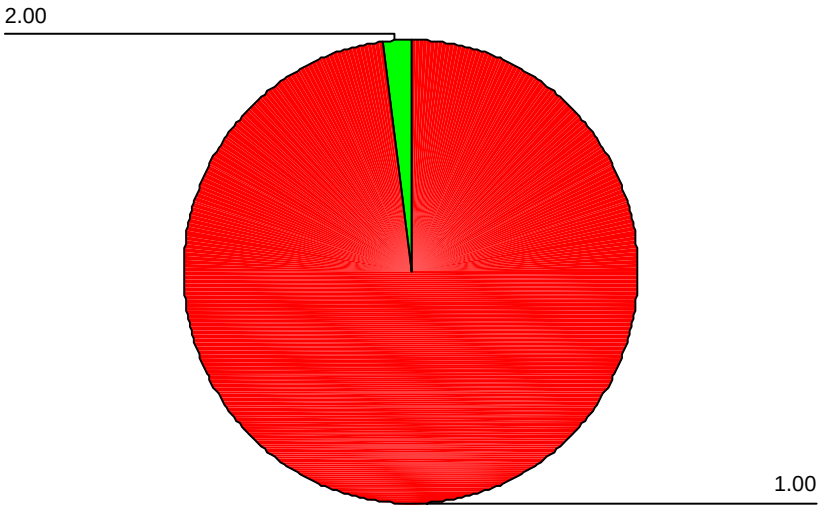


1--<50 Year

2—51-60 years

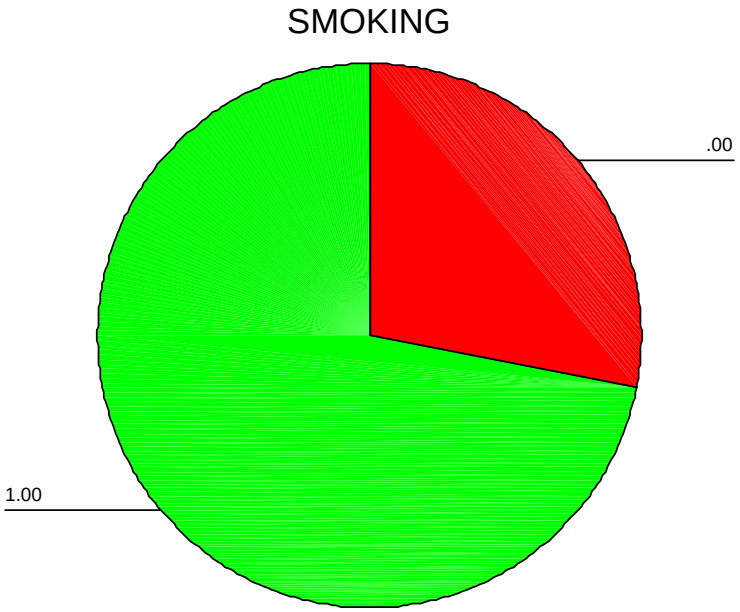
3---<61 years.

SEX



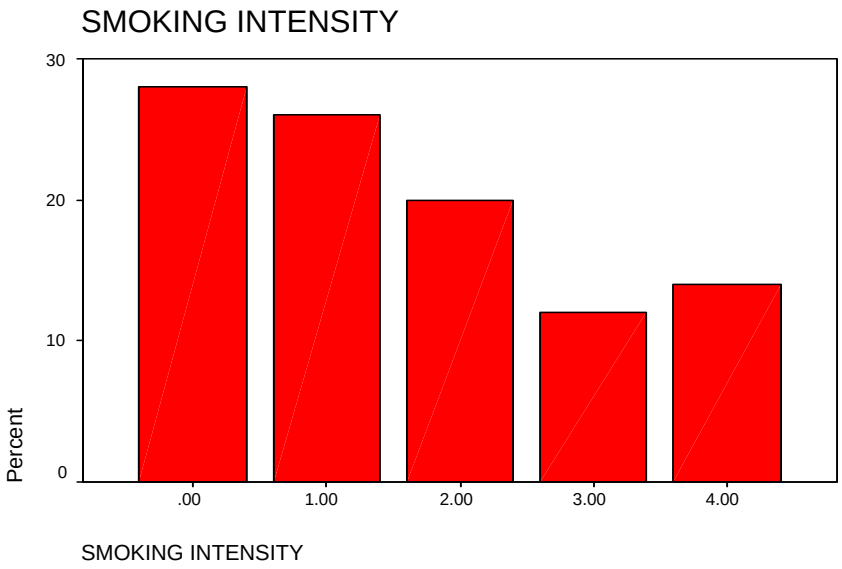
1-male

2-female



0-smoking absent

1-smoking present



0-no smoking

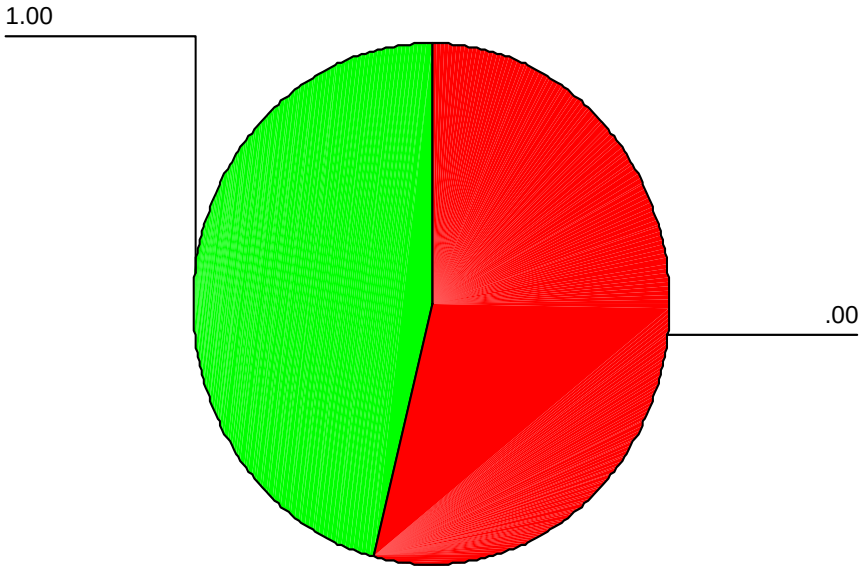
1-<5 cigarettes /day

2-5-10 cigarettes/day

3-10-20 cigarettes/day

4->20 cigarettes /day.

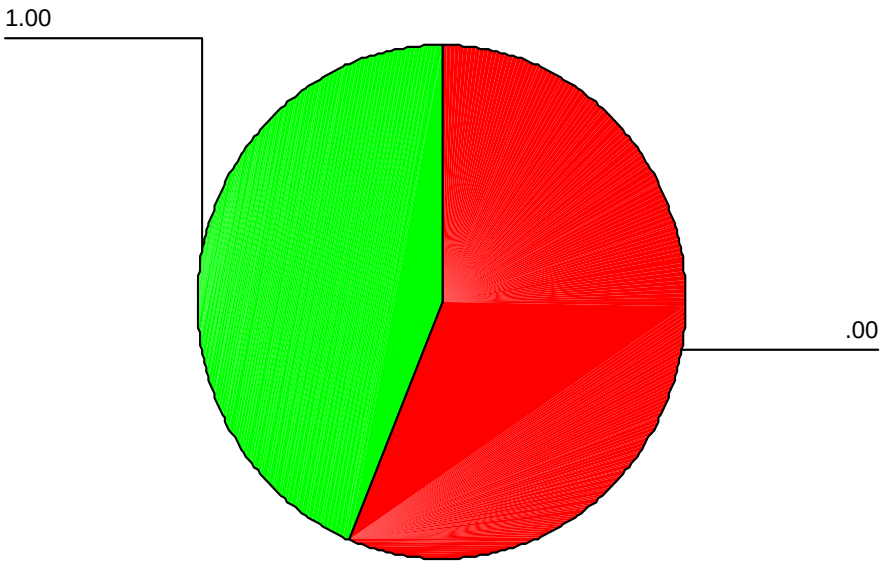
ECG EVIDENCE OF PHT



0-absent

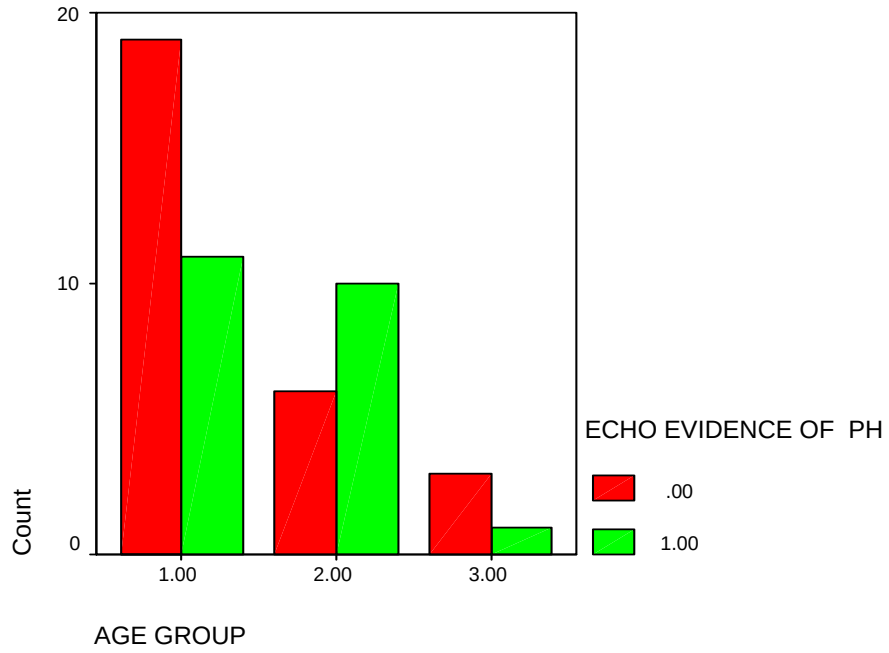
1-present

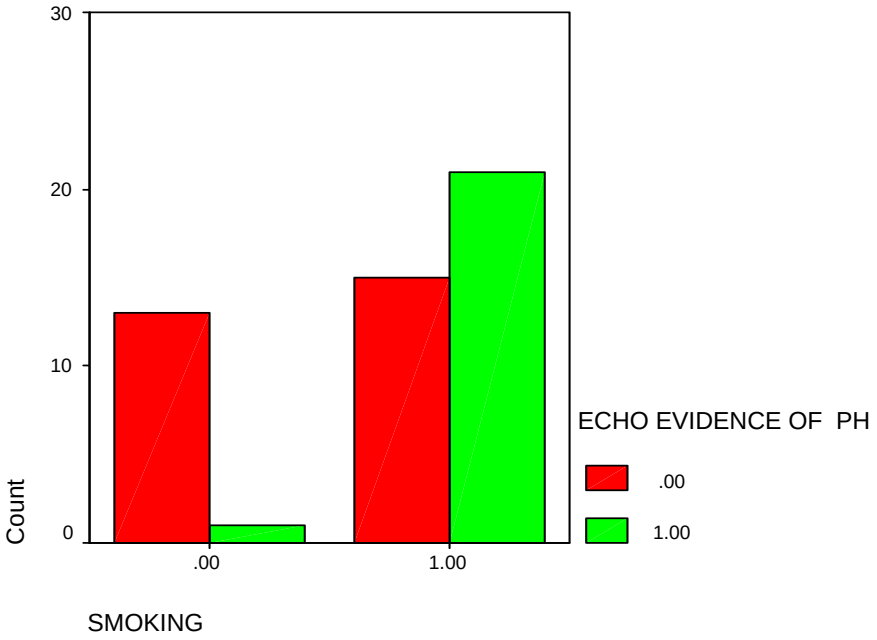
ECHO EVIDENCE OF PHT



0-absent

1- Present


20
10
0
Count
1.00
2.00
3.00
AGE GROUP
ECHO EVIDENCE OF PH
.00
1.00


30
20
10
0
Count
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SMOKING
ECHO EVIDENCE OF PH
.00
1.00

ABBREVIATIONS

COAD—Chronic Obstructive Airways Disease

COPD – Chronic Obstructive Pulmonary Disease.

WHO- World Health Organization.

FEV1- Forced Expiratory volume in one second.

FVC-Forced Vital Capacity.

TNF alpha- Tumor Necrosis Factor alpha.

IL-Interleukin.

CD-Cluster of Differentiation.

ROS-Reactive Oxygen species.

Alpha-1 AT--alpha 1 Anti trypsin.

RTI-Respiratory Tract Infection.

TB-Tuberculosis.

GOLD –Global initiative for Obstructive Lung Disease

AR –Autosomal Recessive.

LHS –Lung Health Study.

SI.NO –Serial Number.

BA-Bronchial Asthma.

H/O-History Of.

U.K-United kingdom.

ICU-Intensive care Unit.

FER-Forced Expiratory Ratio.

TLCO-carbon monoxide transfer factor.

DCO-Diffusing Capacity of Carbon monoxide.

KCO-diffusing coefficient.

PAP- Pulmonary Artery Pressure.

FRC-Functional Residual Capacity.

TLC-Total Lung Capacity.

RV-Residual Volume.

MVV-Maximum Voluntary Ventilation.

LTB-Leukotrine B.

B.M.R.-Basal Metabolic Rate.

B.M.I. - Body Mass Index.

PA- Postero Anterior.

ATS- American Thoracic Society.

ARTP- Association of Respiratory Technicians & Physiologists.

BTS- British Thoracic Society.

PHT- Pulmonary hypertension.

AFB- Acid Fast Bacilli.

PFT- Pulmonary Function Test.

NHLBI- National Heart Lung Blood Institute.

RA- Right Atria.

RV- Right Ventricle.

LV- Left Ventricle.

PE- Pericardial Effusion.

TR- Tricuspid Regurgitation.

RBBB- Right Bundle Branch Block.

USA- United States of America.

ECG- Electrocardiography.

ECHO- Echocardiography.

BNP— B-type Natriuretic Peptide.

RHC- Right Heart Catheterization.

CT scan- Computerized Tomographic scan.

AMJ- American Medical Journal.

RAD- Right Axis Deviation.

LAD- Left Axis Deviation.

P2-Pulmonary component of second heart sound.

Ca- Carcinoma.

NA- Normal Axis.

PROFORMA

Biodata:

Name

Age

Sex

Occupation –factory worker, mine worker

Income

Residence

Symptoms:

- Cough with expectoration
- Dyspnea
- Chest pain
- Puffiness of face
- Swelling of legs
- Palpitation
- Syncope

Past medical history:

- Tuberculosis
- Systemic hypertension

- Diabetes Mellitus
- Bronchial asthma
- Previous hospitalization with similar episode

Personal history:

- Smoking –cigarettes –number of packs/day

  -age at which started smoking

  - Passive smoking.
- pan chewing
- alcohol intake
- damp housing

Family history:

- Similar episode in the family

General examination:

- Mental state
- Pallor
- Icterus
- Cyanosis

- polycythemia
- Pedal edema
- Jugular venous pulse & pressure –increased or normal- a wave or v wave
- Pursed lip breathing
- Presence of wasting/Body mass index
- Blood pressure measurement

Examination of Respiratory system:

- Shape of the chest - increased AP diameter
  - Barrel shaped chest
  -horizontal ribs.
  -splaying of lower costal margin.
  -widened xiphisternal angle.
- Paradoxical inward movement of lower ribs during inspiration (HOOVER'S SIGN)
- movements of chest –chest expansion
  -normal or decreased.

- Cardiac dullness –normal or decreased.
- Breath sounds-normal or decreased.

Cardiovascular system:

- Visible pulsation over pulmonary area
- Parasternal heave
- Palpable P2
- Ejection systolic murmur & Early diastolic murmur over pulmonary area
- Pan systolic murmur over Tricuspid area

Abdomen:

- Tender hepatomegaly
- Ascites

Investigations:

- Hemoglobin
- X-ray chest PA view
- Mantoux test
- Sputum for acid fast bacilli

- Pulse oximetry
- Pulmonary function test –spirometry
- Electrocardiography
- Echocardiography

MASTER CHARTS

SI No	Name	Sex	Age	Smoking	Air pollution	PFT		
						FEV1	FVC	FEV1/ FVC
1	Saleem	Male	41	Present	Present	1.2	2.1	57
2	Raji	Female	45	Absent	Present	1.2	2.1	57
3	Meyyappan	Male	42	Absent	Present	1.3	2.1	62
4	Paulraj	Male	53	Present	Present	1	2	50
5	Ezhumalai	Male	60	Present	Present	1.2	2.1	57
6	Joseph	Male	47	Absent	Absent	1.3	2.1	62
7	Santhosh	Male	41	Absent	Present	1,.3	2.1	62
8	Muniyandi	Male	50	Absent	Present	1	1.9	55
9	Ravi	Male	44	Present	Absent	1.2	2.1	55
10	Ashok	Male	43	Absent	Present	1.3	2.1	62
11	Mustafa	Male	48	Present	Present	1.3	2.1	62
12	Jayaram	Male	55	Present	Present	1.3	2.1	62
13	Sekar	Male	47	Present	Present	0.9	1.8	50
14	Chockalingam	Male	60	Present	Present	0.9	1.9	47
15	Vinodh	Male	48	Present	Present	1.3	2.1	62
16	Ibrahim	Male	49	Present	Present	1.4	2.1	67
17	Kanniyappan	Male	55	Absent	Present	1.3	2.1	62
18	Mohammed	Male	60	Present	Present	1.2	2.1	55
19	Govindan	Male	55	Present	Present	0.9	1.8	50
20	Ezhumalai	Male	71	Absent	Absent	1.3	2.1	62
21	Kannan	Male	48	Absent	Present	1.3	2.1	62
22	Jagadesan	Male	59	Present	Present	0.7	1.7	42
23	Govindasamy	Male	55	Present	Present	0.8	1.7	48
24	Aravind	Male	45	Absent	Present	1.3	2.1	62
25	Siva lingam	Male	59	present	Absent	1.3	2.1	62

SI. No	Name	Sex	Age	Smoking	Air pollution	PFT		
						FEV1	FVC	FEV1/ FVC
26	Stephen	Male	52	Present	Present	1.3	2	66
27	Afzal	Male	45	Present	Present	1.3	2.1	62
28	Ramaiah	Male	60	Present	Present	1.3	2.1	62
29	Srinivasan	Male	51	Absent	Absent	1.2	2	60
30	Palani	Male	55	Present	Present	0.6	1.2	50
31	Sankar	Male	47	Present	Present	1.3	2.1	62
32	Sivaram	Male	43	Absent	Present	1.3	2.1	62
33	Vivek	Male	59	Present	Present	1.1	2.1	52
34	Yousuf	Male	45	Present	Present	1.3	2.1	62
35	Noorulla	Male	64	Absent	Present	1	1.8	55
36	Kalai	Male	45	Present	Present	1	1.9	55
37	Sukumar	Male	53	Absent	Absent	1.3	2.1	62
38	Velusamy	Male	47	Present	Present	1.3	2.1	62
39	Karthikeyan	Male	43	Present	Present	1.3	2.1	62
40	Thirupathi	Male	50	Present	Present	1.3	2.1	57
41	Thomas	Male	50	Present	Present	1.3	2.1	62
42	Murali	Male	48	Present	Present	1.3	2.1	62
43	Peermohammed	Male	45	Present	Present	1.2	2.1	55
44	Pachaiyappam	Male	70	Present	Absent	1.3	2.1	62
45	Ramasamy	Male	41	Present	Present	1.3	2.1	62
46	Daniel	Male	43	Present	Present	1.3	2.1	62
47	Deepan	Male	71	Present	Present	1.2	1.9	63
48	Saravanan	Male	35	Present	Present	1.3	2.1	62
49	Paulraj	Male	50	Present	Present	1.3	2.1	62
50	Abdul kadhar	Male	47	present	Absent	1.3	2	65

SI. No	Name	ECG						ECHO			
		P wave	QRS axis	R/S in V1	R/S in V6	RBBB	ECG evidence Of PHT	RA dilatation	TR	PE	Elevated PAP
1	Saleem	3	RAD	<1	>1	present	Present	Absent	Absent	Absent	Present
2	Raji	2.5	RAD	<1	>1	present	Present	Absent	Absent	Absent	Present
3	Meyyappan	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
4	Paulraj	3	RAD	>1	<1	Absent	Present	Absent	Absent	Absent	Present
5	Ezhumalai	1.5	RAD	>1	<1	Absent	Present	Absent	Absent	Absent	present
6	Joseph	1.5	RAD	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
7	Santhosh	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
8	Muniyandi	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
9	Ravi	3.5	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Present
10	Ashok	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
11	Mustafa	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
12	Jayaram	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
13	Sekar	3.5	RAD	<1	>1	Absent	Present	Present	Present	Absent	Present
14	Chockalingam	3	RAD	<1	>1	Absent	Present	Present	Present	Present	Present
15	Vinodh	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	present
16	Ibrahim	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
17	Kanniyappan	2.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
18	Mohammed	2.5	NA	<1	>1	Present	Absent	Absent	Present	Absent	Present
19	Govindan	3.5	NA	<1	>1	Absent	present	Absent	Absent	Absent	Present
20	Ezhumalai	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
21	Kannan	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
22	Jagadesan	3	LAD	>1	<1	Absent	Present	Present	Absent	Absent	Present
23	Govindasamy	3	RAD	<1	>1	Absent	Present	Present	Present	Absent	Present
24	Aravind	1.5	NA	<1	>1	Absent	Absent	Absent	Present	Absent	Absent
25	Siva lingam	3	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Absent

SI. No	Name	ECG						ECHO			
		P wave	QRS axis	R/S in V1	R/S in V6	RBBB	ECG evidence of PHT	RA dilatation	TR	PE	Elevated PAP
26	Stephen	3.5	NA	<1	>1	Absent	Present	Absent	Absent	Absent	present
27	Afzal	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
28	Ramaiah	3	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Absent
29	Srinivasan	3	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Absent
30	Palani	2	RAD	<1	>1	Present	Present	Absent	Present	Absent	present
31	Sankar	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
32	Sivaram	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
33	Vivek	3	NA	<1	>1	Present	Present	Absent	Absent	Absent	present
34	Yousuf	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
35	Noorulla	3	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Absent
36	Kalai	2.5	RAD	<1	>1	Absent	Present	Absent	Absent	Absent	present
37	Sukumar	2.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
38	Velusamy	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	present
39	Karthikeyan	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
40	Thirupathi	2	RAD	<1	>1	present	Present	Absent	Absent	Absent	present
41	Thomas	3	RAD	<1	>1	Absent	Present	Absent	Absent	Absent	Absent
42	Murali	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
43	Peermohammed	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
44	Pachaiyappam	2	RAD	<1	>1	Absent	Present	Absent	Absent	Absent	Present
45	Ramasamy	1.5	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
46	Daniel	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Absent
47	Deepan	3	NA	>1	<1	Absent	Absent	Absent	Absent	Absent	Absent
48	Saravanan	2.5	RAD	<1	>1	Absent	Present	Absent	Absent	Absent	Present
49	Paulraj	3	NA	<1	>1	Absent	Present	Absent	Absent	Absent	Present
50	Abdul kadhar	2	NA	<1	>1	Absent	Absent	Absent	Absent	Absent	Present

KEY TO MASTER CHART

SI. NO- Serial number.
PFT – Pulmonary Function Test.
FEV1- Forced Expiratory Volume in one second. (<80% predicted value is taken into account)
FVC- Forced Vital Capacity.
FER (Forced Expiratory Ratio)-FEV1/FVC (<70% is taken into account)
P wave- > 2.5 mm is taken as p pulmonale.
QRS RAD- QRS Right Axis Deviation.>90°
QRS LAD- QRS Left Axis Deviation. <-30°
QRS NA- QRS Normal Axis-(-30°to +90°)
ECG- Electrocardiography
RBBB- Right Bundle Branch Block
ECG evidence of pulmonary hypertension- p pulmonale, RAD, RBBB, R/s in V1 >1, r/S <1 IN v6.
Elevated pulmonary artery pressure (PAP) - mean PAP 20 mm Hg.