A Dissertation on

ECG AND ECHOCARDIOGAPHIC FINDINGS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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CERTIFICATE

This is to certify that this dissertation in **"ECG AND ECHOCARDIOGRAPHIC FINDINGS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE"** was a work done by **Dr.B.ANTONY BENEDICT BABU** under my guidance during the academic year 2004-2007. This has been submitted in partial fulfillment of the award of M.D.Degree in General Medicine (Branch-I) by the Tamil Nadu Dr.M.G.R. Medical University, Chennai - 600 032.

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DECLARATION

I solemnly declare that this dissertation entitled **"ECG AND ECHOCARDIOGAPHIC FINDINGS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE "** was done by me at Madras Medical College and Government General Hospital during the academic year 2004 - 2007 under the guidance and supervision of **Prof.Dr.D.B.SELVARAJ, M.D.** This dissertation is submitted to the Tamil Nadu Dr.M.G.R. Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree in General Medicine (Branch - I).

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) which encompasses both chronic bronchitis and emphysema is one of the commonest respiratory condition of adults in the developing world.

In the western world, COPD is probably the fourth commonest cause of death in middle aged to elderly men. According to estimates, COPD would become the third biggest cause of death in the world by 2020.

Going by the available Indian data for 1996, there were 12.3 million cases of COPD in our country. COPD poses an enormous burden to our society, both in terms of direct cost to health care services and indirect cost to society in terms of loss of production.

Despite the high prevalence and enormous cost to health care and society, COPD has received little attention in respect to other respiratory conditions like Asthma and Lung Cancer.

This is likely to be because COPD is thought of as a self inflicted disease with few effective treatments and mainly affects a more elderly which has less vocal population, which is not true.

REVIEW OF LITERATURE

HISTORICAL PERSPECTIVE

The beginning of modern chest medicine can be traced to the classic volume by Laennec, “A treatise on diseases of the chest” which appeared in 1821 laid the corner stone of modern chest medicine.

In his treatise, Laennec, devoted one chapter to “Pulmonary Catarrh or Bronchitis” and emphysema. The chapter on bronchitis distinguishes between acute and chronic form and sub divides chronic bronchitis into two types – the humid (Copious Expectoration) and dry (Scarcely any Expectoration). He identified “Dilatation of air cells” as the essential feature of emphysema.

Recognition of chronic bronchitis as a potentially grave illness rather than as a trivial but not disabling disease had to wait the “London Fog” of 1953, which was brought about by bad weather and air pollutants, carried with it a surge in morbidity and mortality due to chronic lung disease.

After World War II, clinical investigations of pulmonary disease were provided with a new diagnostic armamentarium; Pulmonary Function tests were extended beyond simple spirometry and innovative techniques were developed for assessing the distribution of gases within the lungs which greatly improved our understanding of COPD.

DEFINITION

The Global Initiative on Obstructive Lung Disease (GOLD) has proposed a definition of COPD that is based on evidence of air flow obstruction. Accordingly it is defined as a “disease state characterized by air flow limitation that is not fully reversible. The air flow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases”.

1. Chronic Bronchitis

It is defined as persistence of cough and excessive mucous secretion on most days over a three month period for at least two successive years.

2. Emphysema

It is defined as abnormal permanent enlargement of the gas exchanging unit of lungs in association with destruction of alveolar walls and without obvious fibrosis.

Burden of COPD

Epidemiology

Most of the information available on COPD prevalence, morbidity and mortality comes from developed countries. Even in these countries, accurate epidemiological data on COPD are difficult and expensive to collect.

Prevalence and morbidity data greatly underestimate the total burden of COPD because the disease is usually not diagnosed until it is clinically

apparent and moderately advanced. The imprecise and variable definitions have made it hard to quantify the morbidity and mortality of this disease in developed and developing countries.

Prevalence

In the “Global burden of disease” study conducted by World Health Organization (WHO) the world wide prevalence of COPD in 2002 was estimated to be 11.6 / 1000 in men and 8.77 / 1000 in women. The prevalence is highest in countries where cigarette smoking has been, or still is very common.

Globally, the study showed COPD results in 2.75 million deaths overall representing 4.8 % of all deaths.

Now COPD is the fourth leading cause of death worldwide and by the year 2020 it will be the third leading cause of death and fifth leading cause of DALY’s lost worldwide (Disability Adjusted Life Years).

Pathogenesis

COPD is characterized by chronic inflammation through out the airways, parenchyma and pulmonary vasculature. Macrophages , T lymphocytes (Predominantly CD 8 +) and neutrophils are increased in various parts of the lung. Activated inflammatory cells release a variety of mediators including leukotriene B4, interleukin 8, tumor necrosis factor (TNF) and others capable of damaging lung structures and / or sustaining neutrophilic inflammation.

In addition to inflammation, two other processes thought to be important in the pathogenesis of COPD are:

(i) imbalance of proteinases and anti proteinases in the lung

(ii) oxidative stress

Pathology

Pathological changes characteristic of COPD are found in the central airways, peripheral airways, lung parenchyma and pulmonary vasculature.

Central airways (> 2-4 mm in internal diameter)

Enlarged mucus secreting glands

Increase in the number of goblet cells

Peripheral airways (< 2 mm in internal diameter)

Structural remodeling of the airway wall, with increasing collagen content and scar tissue formation that narrows the lumen and produces fixed airway obstruction.

Lung parenchyma

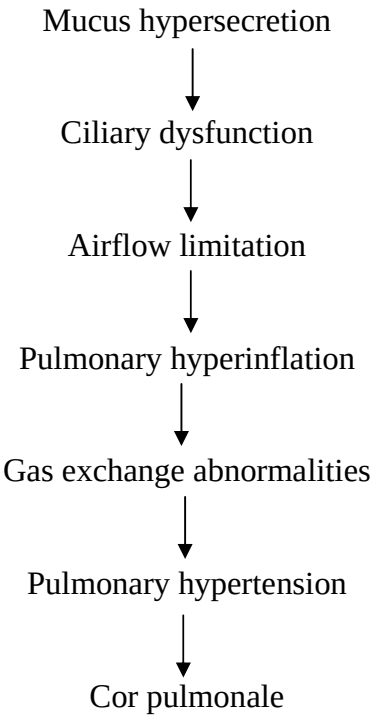
Centri lobular emphysema is predominantly seen in these patients because it is the most frequent type of emphysema seen in smokers. Recent studies indicate increased collagen per unit volume of air space wall in emphysematous lung from smokers.

Pulmonary vasculature

There will be thickening of the intima initially followed by smooth muscle hyperplasia as the disease progresses.

Pathophysiology

Pathological changes in the lungs lead to corresponding physiological changes characteristic of the disease which occur in the following order:



Clinical hallmarks

Table - 1

	Predominant bronchitis	**Predominanat emphysema**
General appearance	Mesomorphic; overweight, dusky with suffused conjunctivae, warm extremities	Thin, often emaciated, pursed lip breathing, anxious, normal or cool extremities
Age, years	40 to 55 years	50 to 75 years
Onset	Cough	Dyspnea
Cyanosis	Marked	Slight to none
Cough	More evident than dyspnea	
Sputum	Copious	Scanty
Upper respiratory infections	Common	Occasional
Breath sounds	Moderately diminished	Markedly diminished
Corpulmonale and right sided heart failure	Common	Only during bouts of respiratory infection and terminal
Other names	Blue bloater	Pink puffer

Pink puffer

Complains of severe dyspnea but has a relatively good blood oxygen level and does not have hypercapnia. Such patients tend to be thin and does not have corpulmonale or right sided heart failure.

Blue bloater

He has peripheral edema caused by right heart failure and has more severe hypoxemia and hypercapnia and less dyspnea.

Cardio vascular manifestations

COPD is associated with several complications, the most serious of which are Pulmonary arterial hypertension, right ventricular hypertrophy (Corpulmonary) and right ventricular failure.

Chronic corpulmonary is defined as " hypertrophy of the right ventricle resulting from the disease affecting the function and / or structure of the lung, except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart or congenital heart disease".

Pathophysiology of pulmonary hypertension

Pulmonary hypertension in COPD is due to multiple factors.:

(i) Pulmonary vasoconstriction caused by alveolar hypoxia, academia and hypercarbia

(ii) Compression of pulmonary vessels by the high lung volume

(iii) Loss of small vessels in the vascular bed in regions of emphysema and lung destruction

(iv) Increased cardiac output and blood viscoscity from polycythemia secondary to hypoxia

Of these, hypoxia is the most important factor and is associated with pathological changes that occur characteristically in the peripheral pulmonary arterial bed.

Intimal thickening appears to be a early event that occurs in association with progressive airflow limitation. The structural rather than hypoxic vasoconstriction is required for the development of sustained pulmonary hypertension in patients with COPD.

With mild to moderate grades of COPD, (FEV_1 40% to 80% of predicted), pulmonary artery pressure (Ppa) is usually normal at rest, but increases with moderate exercise or exposure to cold. In the presence of severe COPD (FEV_1 < 40% of predicted), hypertension is usually present at rest (Mean Ppa > 20 mm Hg) and pulmonary arterial pressure undergoes a disproportionate increase with mild exercise.

Ppa can increase acutely during episodes of hypoxia that occur during sleep and it has been suggested that recurrent nocturnal pulmonary hypertension can eventually lead to “fixed” hypertension as a result of structural changes in the arterial wall.

Pulmonary hypertension in COPD progresses over time and its severity correlates with the degree of airflow obstruction and impairment of pulmonary gas exchange.

Weitzenblum et al studied the evolution of Ppa in a group of 93 patients with COPD for 5 years. They found that Ppa increased at an average rate of 0.08 kPa (0.6mm Hg) per year. The rate of increase of Ppa was slightly higher in patients who did not have pulmonary hypertension at the beginning of the study compared with those who already had pulmonary hypertension.

An intriguing question is when pulmonary hypertension commences in the natural history of COPD. In a recent study, Kessler et al assessed the evolution of pulmonary hemodynamics in a group of 131 patients with moderate COPD who did not have pulmonary hypertension at rest, although 58% developed pulmonary hypertension during exercise.

The results of the study indicates that in COPD, changes in pulmonary circulation may start several years before pulmonary hypertension is apparent at rest and that exercise testing might be useful in showing abnormalities of the pulmonary circulation.

Although left ventricular function is normal in most patients who have COPD, it may deteriorate when corpulmonale and right ventricular failure develop because of right ventricular dilatation, septal shift and left ventricular compliance.

Chronic corpulmonale is associated with a high incidence of cardiac arrhythmias, particulary supra ventricular arrhythmias.

ELECTRO CARDIOGRAPHIC MANIFESTATIONS

Chronic obstructive airways disease influence the electrical events of the heart in the following basic respects:

1. the voluminous lungs have an insulating effect and thereby diminish the transmission of electrical potential to the registering electrodes

2. the heart descends to a lower position within the thorax due to a lowering of the diaphragm. This will alter the position of the heart relative to conventional precordial electrode postions.

3. the right ventricle and right atrium become compromised due to a reduction of the pulmonary vascular bed. This will result in right ventricular hypertrophy as well as right atrial enlargement.

Decreased magnitude of the electrocardiographic deflexions

The voluminous lungs impair electrical transmission. The QRS and T deflexions are therefore markedly diminished in magnitude.

Lead I sign

In patients with COPD the frontal plane P, QRS and T wave axes are not infrequently all directed at around + 90 degree which are either precisely or almost perpendicular to the standard lead I axis. As a result of this Lead I reflects absent or very low amplitude P, QRS and T wave complexes giving the appearance of minimally disturbed base line. This ECG phenomenon is known as the Lead I sign.

Right atrial enlargement

The frontal plane P wave axis is directed to the right of +60 degree. It is commonly and in a way characteristically directed to + 90 degree

P pulmonale

It is reflected by P waves which are tall and peaked in standard leads II, III aVF, and is the expression of right atrial enlargement.

The combination of right axis deviation and tall peaked P waves is called P pulmonale. A comparison of interstitial pulmonary fibrosis and COPD showed that a deviation of the frontal plane P wave axis to the right of +70 degree only occurs with COPD.

Abnormalities of the QRS complex

Right QRS axis deviation

The frontal plane QRS axis is deviated to the right and commonly directed to +90 degree. When it is deviated further, the frontal plane leads will usually reflect an S1Q3R3 pattern. In very severe cases it may be directed to the “northwest” region.

Left QRS axis deviation

This occurs in about 10% of cases. The mechanism is still speculative.

S1, SII, SIII syndrome

Prominent terminal S waves may appear in standard leads I and II or in I, II and III giving rise to the SI, S II, S III syndrome. This indirectly reflects posterior displacement of the apex.

Posterior displacement of the mean QRS axis

The mean QRS axis may also be displaced somewhat posteriorly so that it tends to be more obliquely oriented to the horizontal plane.

Abnormalities of the precordial QRS form

There is diminution of QRS magnitude in all the leads. It is not uncommon for all the precordial leads to reflect rS complexes. In very severe cases the R : S ratio is usually less than 1 in leads V4 to V6, and R wave amplitude in lead V6 may be less than 5 mm. The transition zone is frequently displaced to V6 or even further to the left.

Right bundle branch block

There may occasionally be transient complete or incomplete RBBB with an exacerbation of the emphysema and increase in oxygen desaturation.

Right ventricle hypertrophy is mainly reflected by:

a. right axis deviation

b. prominent terminal S waves in the left precordial leads

The single most characteristic ECG feature of diagnosis in COPD is said to be P wave axis of +70 to +90 degrees.

Abnormalities of T waves

Frontal T wave axis

It is usually similar in direction to that of QRS axis. The T wave is diminished in amplitude in all leads. The T wave may be inverted in the right precordial leads especially when pulmonary hypertension is marked.

Exacerbation of the disease with an increase in oxygen desaturation may be associated with elevation of ST segment in leads II, III and AVF. This manifestation is reversible.

ECHOCARDIOGRAPHY

Echocardiography is nearly twice as sensitive as clinical examination in detecting corpulmonale. However it may be suboptimal in persons with hyper inflation because of poor transmission of sound waves by the increased retrosternal air space.

Systolic pulmonary artery pressure can be estimated with pulsed Doppler by determining the blood flow velocity in the main pulmonary artery or in the regurgitant jet from the tricuspid valve. The tricuspid valve regurgitant jet can be used to determine the right ventricular – atrial gradient using the modified Bernoulli's equation

$P = 4\ V^2$

Where P = peak pressure difference between the right ventricle (RV) and the right atrium (RA)

V = peak velocity of tricuspid regurgitant jet

This pressure is then added to the mean right atrial pressure to obtain the systolic Ppa.

Sensitivity, specificity and positive and negative predictive values of systolic pulmonary artery pressure estimation for diagnosis of pulmonary hypertension were 85, 55, 52 and 87% respectively.

M Mode, 2D – Echo and Color Doppler features of patients with PAH

Table 2

Variables	Right heart	Left heart
M - mode		
Diminished or absent atrial wave of the pulmonary valve		

2 D Echo		
	RV hypertrophy, RV Dilatation	Reduced LV end diastolic volume
	RV abnormal systolic function	Reduced LV end systolic volume
	RV pressure overload pattern of IVS	LV ejection fraction within normal limits
	Right atrial dilatation	Increased IVS thickness
	Dilated pulmonary artery	IVS / posterior wall thickness ratio more than 1
	Inter atrial septum bows right to left	

Color Doppler		
	Tricuspid regurgitation	
	Pulmonary insufficiency	
	Elevated pulmonary artery systolic pressure	E/A ratio < 1
	RV outflow tract acceleration time < 0.1 seconds	

E/A Ratio

Ratio between early peak transmitral flow velocity and late peak systolic velocity

RV – right ventricle

LV – left ventricle

IVS – inter ventricular septum

AIMS AND OBJECTIVES

To study the electrocardiographic and echocardiographic changes in chronic obstructive pulmonary disease.

MATERIALS AND METHODS

SETTING

The study was conducted in Government General Hospital, Chennai.

DESIGN OF STUDY

It was an observational type of study. Interview technique was used to collect information on a predesigned proforma.

PERIOD OF STUDY

It was conducted in a time period from June 2005 to June 2006.

SAMPLE SIZE

Fifty cases of COPD.

SELECTION OF STUDY SUBJECTS

COPD patients above 30 years of age, attending the outpatient clinic in the admission day of my unit and who were admitted in my ward in Government General Hospital, Chennai were selected for this study.

INCLUSION CRITERIA

All COPD patients above 30 years of age, diagnosed clinically and satisfying the spirometric criteria were included in the study.

A diagnosis of COPD should be considered in any patient who has symptoms of

i. Chronic cough

- Present intermittently or everyday

- Often present throughout the day

- Seldom only nocturnal

ii. Chronic sputum production

- Any pattern of Chronic sputum production may indicate COPD

iii. Dyspnea that is

- Progressive (worsens over time)

- Persistent (present everyday)

- Worse on exercise

- Worsens during respiratory infections

iv. History of exposure to risk factors

RISK FACTORS

Table 3

Environmental	Host -Related
Indoor air pollution from biomass fuel use in setting of inadequate ventilation	Airway hyper responsiveness
Occupational dusts and chemicals	Severe hereditary alpha1antitrypsin deficiency.
History of severe childhood respiratory infections	Low birth weight
Outdoor air pollution	Maternal cigarette smoking during gestation
Low socio economic status	
Intra venous drug use (Methyl Phenidate, Methadone, Talc granulomatosis)	

Of these, cigarette smoking is overwhelmingly the most important risk factor in the development of COPD.

Spirometric criteria

The diagnosis is confirmed by spirometry. The presence of a post bronchodilator FEV 1 < 80% of the predicted value in combination with an FEV 1 / FVC < 70% confirms the presence of air flow limitation that is not fully reversible.

Exclusion criteria

Patients with other respiratory diseases like:

Asthma

Tuberculosis

Bronchiectasis

Lung malignancy were excluded from the study.

Patients with:

Valvular heart diseases

Coronary heart diseases

Systemic hypertension

Cardio myopathies

AIDS were excluded.

All patients were subjected to:

- Routine blood investigations
- Urine analysis
- Sputum analysis for AFB stain, gram stain, culture and sensitivity was done.
- Mantoux test was done

- ELISA for HIV
- Arterial blood gas analysis
- Chest X-ray PA views and lateral views were taken for all patients
- Electrocardiograph was taken
- All patients were subjected to both 2 D and Doppler Echocardiogram and pulmonary artery pressure measured.
- Pulmonary function test was done using spirometry

SPIROMETRY

Several tests are used to study various aspects of pulmonary functions. Amongst these spirometry is the most basic and useful method for evaluating pulmonary function.

Spirometry is a simple expression of a complex process, just like blood pressure. It measures air flow from fully inflated lungs over time in litres. Thus the forced vital capacity (FVC) is the amount of air exhaled from fully inflated lungs and FEV 1 measures the air flow during the first part of vital capacity manoeuvre.

Selecting a Spirometer

The American Thoracic Society (ATS) recommends that the equipment should be such that:

- it can be calibrated with a three litre syringe
- it should record atleast FVC and FEV1
- it should record a flow volume curve or a flow volume loop or both (if possible)

Contra indications for performing Spirometry

It is recommended that patients should not be tested within one month of myocardial infarction.

Patients with any of the conditions listed below are unlikely to achieve optimal or reproducible results.

- Chest or abdominal pain of any cause
- Oral or facial pain exacerbated by a mouth piece
- Stress incontinence
- Dementia or confused state

Performing Spirometry

According to the current ATS statement, spirometry may be performed either in the sitting or standing position. The sitting position is considered safe in order to prevent falling due to syncope. However, in obese subjects, the standing position may be preferred.

- Smoking within one hour of testing
- Consuming alcohol within four hours of testing
- Performing vigorous exercise within thirty minutes of testing

- Wearing clothing that substantially restricts full chest and abdominal expansion
- Eating a large meal within two hours of testing

If it is performed to diagnose airway disorder, avoid:

- Short acting bronchodilators within previous six hours
- Long acting bronchodilators within previous twelve hours
- Slow release theophyllines within the previous twenty four hours

Spirometric manoeuvre

The procedure must be instructed and demonstrated to the patient. The patient then performs spirometry in the following steps:

Expiratory manoeuvre

1. Take a full deep breath away from the spirometer
2. Hold the mouth piece between the lips to create a good seal
3. Expire as fast and as hard as possible for as long as possible until no breath is left

Expiratory and inspiratory manoeuvre

1. Hold the mouth piece between the lips to create a good seal
2. Breathe in and out for two to three tidal breaths
3. Expire as fast and as hard as possible for as long as possible until no breath is left
4. Inspire rapidly to maximum capacity

Spirometry should be recorded fifteen to thirty minutes after administration of a short acting beta-agonist eg. 200 to 400 microgram of salbutamol to check for bronchodilator reversibility.

Acceptable tests

- The effort should be maximal, smooth and cough free
- Exhalation time should be atleast six seconds
- End of test is indicated by a two second volume plateau
- Reproducibility as indicated by FVC should be within 5% or 100 ml between the highest and next best among three acceptable tests
- If three reproducible tests are not available, upto 8 manoeuvres should be attempted
- The best values of three acceptable tests are used for interpretation
- If after 8 manoeuvres, 3 reproducible tests are not available, then the test with highest values may be used.

Reversibility testing

Broncho dilator reversibility

Spirometry is recorded fifteen to thirty minutes after administration of short acting beta agonist eg. 200 to 400 mcg of salbutamol is used.

Calculation of % improvement

$$\frac{\text{FEV1 (post broncho dilator)} - \text{FEV1 (base line)}}{\text{FEV1 (base line)}} \times 100$$

Good bronchodilator reversibility is indicated by improvement in FEV1 by 200 ml and > 12%.

Spirometry in COPD

Gold standard for confirmation of COPD

- Decreased FEV1 with concomitant reduction in FEV1 / FVC ratio with a poor or absent broncho dilator reversibility and normal or reduced FVC

- FEV1 / FVC % < 70 is used to diagnose COPD

For assessment of Severity of COPD by FEV1%

Severity of COPD (GOLD classification)

Table 4

0	**At risk**	**Spirometry normal but symptoms evident**	
I	Mild	FEV1 / FVC % < 70	FEV1 > 80% predicted
II	Moderate	FEV1 / FVC % < 70	FEV1 50 to 80% predicted
III	Severe	FEV1 / FVC % < 70	FEV1 30 to 50% predicted
IV	Very severe	FEV1 / FVC % < 70	FEV1 < 30% predicted (or) FEV1 < 50% predicted with chronic respiratory failure

PROFORMA

Name: Age: Sex:

Height (metres): Weight (Kgs):

Occupation: Income:

Presenting complaints

Cough with expectoration

Dyspnea

Wheeze

Cyanosis

Puffiness of face

Reduced urine output

Swelling of legs

Chest pain

Palpitation

Past history

Tuberculosis

Systemic hypertension

Diabetes mellitus

Asthma

Ischemic heart disease

Previous hospitalization for similar complaints

Personal history

Diet veg or Non-Veg

Smoking–cigarettes or beedis - number of packs per day and duration of smoking

Chewing pan

Alcohol intake

Occupational history

Investigation

Blood hemogram

Blood sugar

Blood urea

Serum creatinine

Lipid profile

Sputum

Gram stain

AFB stain

Culture and sensitivity

Mantoux test

ELISA for HIV

Urine

Albumin

Sugar

Deposits

Chest X-ray

PA view

Lateral view

ECG in all leads

Arterial blood gas (ABG) analysis

Echo cardiography

Pulmonary function test

Percentage predicted

FEV1

FVC

FEV1 / FVC %

PEFR

OBSERVATION

AGE – DISTRIBUTION

Table 5

S. No.	Age (years)	Predominant bronchitis	Predominant emphysema	Total	percentage
1.	30- 39	2	-	2	4
2.	40 - 49	10	2	12	24
3.	50 – 59	16	6	22	44
4.	60 & above	7	7	14	28

Lower age limit of 30 years was selected because this was the most frequently available cut off age in the reported studies. More over COPD is rare below this age.

In my study, it is seen predominantly in persons over 40 years of age

SEX DISTRIBUTION

Table 6

S. No	Sex	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	Male	23	11	34	68
2.	Female	12	4	16	32

The male female ratio is 2.12:1.

SMOKING PATTERN

Table 7

S. No	Pack years	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	> 30	12	6	18	36
2.	20 – 30	8	3	11	22
3.	< 20	3	1	4	8
4.	Non smoker	12	5	17	34

Pack year

It is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked.

Commonly seen in persons with a smoking pattern of more than 20 pack years.

33 of the 34 non smokers in this study were females.

PULMONARY FUNCTION TEST

SPIROMETRY

Table 8

S. No	FEV1 / FVC %	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	30 – 40	3	1	4	8
2.	41 – 50	20	4	24	48
3.	51 - 60	11	8	19	38
4.	61 – 70	1	2	3	6

Table 9

S. No	Post bronchodilator FEV1%	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	Mild (> 80%)	1	2	3	6
2.	Moderate (50 – 80 %)	16	8	24	48
3.	Severe (30 – 50 %)	14	3	17	34
4.	Very severe (< 30) or (< 50% with respiratory failure)	4	2	6	12

FVC (Forced vital capacity)

Maximal volume of air that can be exhaled during a forced manoeuvre

FEV_1

Volume of air expired in the first second of maximal expiration after a maximal inspiration.

This is a measure of how quickly the lungs can be emptied.

FEV1 / FVC %

FEV1 expressed as a percentage of FVC gives a clinically useful index of air flow limitation when it is less than 70 %.

In this study most of the cases had FEV_1 / FVC % in the range between 40 to 60% and most of them had moderate to severe COPD.

CHEST X-RAY FINDINGS

Table 10

S. No	CXR findings	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	Emphysematous changes	4	12	16	32
2.	Increased broncho vascular markings	24	3	27	54
3.	Cardiomegaly	4	2	6	12
4.	Evidence of pulmonary hypertension	6	-	6	12

More than half of them had increased broncho vascular markings. Evidence of pulmonary hypertension was found in six patients.

ARTERIAL BLOOD GAS ANALYSIS

Measurement of PaO2

Table 11

S.No	PaO2 (mmhg)	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	50 - 60	9	4	13	26
2.	40 – 49	6	1	7	14
3.	< 40	2	-	2	4

Measurement of PCO2

Table 12

S. No	PCO2 (mmhg)	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	41 - 50	5	3	8	16
2.	51 – 60	3	-	3	6
3.	> 60	2	-	2	4

22 patients had evidence of hypoxemia and thirteen patients had hypercapnia.

ELECTRO CARDIOGRAPHIC FINDINGS

Table 13

S. No	ECG changes	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	P Pulmonale	6	2	8	16
2.	Right axis deviation	10	3	13	26
3.	Left axis deviation	1	-	1	2
4.	RBBB	-	2	2	4
5.	Low voltage QRS	1	4	5	10
6.	Lead I sign	2	-	2	4
7.	R/S ratio in V6 < 1	8	4	12	24
8.	R/S ratio in V1 > 1	5	1	6	12
9	ST depression in L II, III, AVF	4	3	7	14
10.	T Wave Inversion in V1 – V3	4	2	6	12
11.	S I, S II, S III	1	-	1	2
12.	Ventricular ectopics	1	1	2	4
13.	Multifocal atrial tachycardia	1	-	1	2

In my study of 50 patients with COPD, the following electrocardiographic findings were observed:

Right axis deviation - 13 cases

R/S ratio < 1 in V_6 - 12 cases

"P" pulmonale - 8 cases

ST depression in II, III, aVF - 7 cases

T wave inversion in $V_1 - V_3$	-	6 cases
Low voltage QRS	-	5 cases
Right bundle branch block	-	2 cases
Lead I sign	-	2 cases
Ventricular ectopics	-	2 cases
SI, SII, SIII pattern	-	1 case
Multifocal atrial tachycardia	-	1 case

ECG evidence of right ventricular hypertrophy was found in 28 cases.

MEASUREMENT OF PULMONARY ARTERY PRESSURE BY ECHO-DOPPLER

Table 14

S. No	Systolic pulmonary artery pressure (mmhg)	Predominant bronchitis	Predominant emphysema	Total	Percentage
1.	31 – 40	8	2	10	20
2.	41 – 50	6	1	7	14
3.	51 – 60	2	-	2	4
4.	61 – 70	1	-	1	2
5.	71 – 80	1	-	1	2

21 patients had evidence of elevated pulmonary artery pressure by echo doppler and most of them had systolic pulmonary artery pressure in the range between 30 to 50 mmHg.

DISCUSSION

AGE DISTRIBUTION

I used the lower age of thirty years to calculate my estimate because this was the most frequently available cut off age in the reported studies. Moreover the disease is very rare below this age.

It is found that obstructive air way disease is more common in the middle and old age. This is because

- The risk of COPD increases with increasing age because it is related to smoking pack years and aging per se has a cumulative effect of exposure to environmental stress.

- Mostly, the patients tend to ignore the initial symptoms and with increasing age, the symptoms worsen and they report to the hospital only at this juncture.

- With improving medical care, the life expectancy tends to increase and with it the problem of COPD will also increase with advancing age.

SEX DISTRIBUTION

In my study, the male is to female ratio is 2.12: 1

Sex distribution of COPD in adults (> 30 years of age) in different parts of the country is generally similar.

The following is the male female ratio in the Indian studies:

Table 15: Male female ratio in Indian studies

Authors	Year	Population	M : F
Wig et al	1964	Rural Delhi	1.32 : 1
Viswanathan	1966	Patna	1.59 : 1
Sikand et al	1966	Delhi	1.63 : 1
Bhattacharya et al	1975	Rural UP	1.49 : 1
Viswanathan & Singh	1977	Delhi – Rural Urban	1.34 : 1 1.86 : 1
Thiruvengadam et al	1977	Madras	1.58 :1
Charan	1977	Rural Punjab	1.40 : 1
Radha et al	1977	New Delhi	1.76 : 1
Malik	1986	Chandigarh – Rural Urban	1.92 : 1 2.31 : 1
Jinda	1993	Chandigarh	2.60 : 1
Ray et al	1995	Madras area	1.60 : 1

The prevalence was uniformly higher among male in all reported studies. The male : female ratio vary from 1.32 : 1 to 2.60 : 1 with the median ratio at 1.60 : 1.

From the two south Indian studies which were carried out mainly in Madras region at different times, (1977 and 1995) the male female ratio was fairly constant maintaining at around 1.6: 1 which is that of the national average.

The male female ratio tends to be much higher in case of urban population. Of several possible reasons which might account for a higher prevalence among males, the most important is the habit of smoking of tobacco.

SMOKING PATTERN

The reported smoker: non-smoker prevalence ratio ranged from 61.6 to 91.1 % in ten different population studies. The median value was around 82.3%.

In my study, the ratio is about 66% of smokers, the majority of non – smokers in my study were female population. The greater the pack years, the greater the prevalence of COPD.

Tobacco was introduced in India by the Portugese 400 years ago. Since then, tobacco consumption continued to rise in India. It has been estimated that there are 1.1 billion smokers worldwide and 182 million (16.6 %) of them live in India.

It has been predicted by WHO that more than 500 million people alive today will be killed by tobacco by 2030. Tobacco is used for smoking as well

as in smokeless forms in India. Among the tobacco smokers, beedi smokers constitute 40%, cigarette smokers 20% and those using smokeless forms 40%.

The prevalence of tobacco use during 1993 to 1994 was 23.2% in male (any age) and 4% in female in urban areas, 33.6 % in male and 8.8 % in female in rural areas.

Smokers suffer an irreversible FEV1 loss of 4.4 to 10.4 ml per pack year smoked. Cigarette smoking also retards the normal increase in expiratory flow that occurs during growth in childhood or adolescence. The duration and intensity are of equal importance in determining these effects.

Smoking cessation is associated with a small improvement in lung function, decrease in coughing and sputum expectoration and normalization of the rate of annual decline in lung function.

PULMONARY FUNCTION TEST

The primary problem in obstructive lung disease is an increased airway resistance. For each measurement of pulmonary function there is a normal value and range of normal limits. A common method of comparison is to compute a percentage of predicted normal values according to the equation.

% predicted is equal to:

$$\frac{\text{Measured value}}{\text{Predicted normal value}} \times 100$$

Predicted values

Predicted values vary as per age, sex, height and ethnic groups, are obtained by large scale studies in the community and are readily available for use. Values above 80 % of predicted are generally considered as normal. For patients with deformity of the thoracic cage such as Kyphoscoliosis, the arm span from finger tip to finger tip can be used as an estimate of height.

Caucasians have the largest FEV1 and FVC and of the various ethnic groups, Polynesians are among the lowest. There is little difference in PEF between ethnic groups. The values for black Africans are 10 to 15 % lower than the Caucasians of similar age, sex and height because for a given standing height, their thorax is shorter. The Chinese have been found to have an FVC about 20 % lower and Indians about 10 % lower than matched Caucasians.

Approximate conversion factors for adjusting European reference values of FVC and FEV1 for Indians are 0.9 for North Indians and 0.87 for south Indians.

SPIROMETRY AND COPD

- Spirometry is needed to make a firm diagnosis of COPD.
- Together with the presence of symptoms, spirometry helps in staging COPD severity and can be a guide to specific treatment steps.
- The lower the % predicted FEV1 the worse the subsequent prognosis.

- FEV1 declines over time and faster in COPD than in healthy subjects.

- Spirometry can be used to monitor disease progression, buto to be reliable, the intervals between the measurements must be atleast 12 months.

Staging

Once diagnosed, there are no widely accepted staging or severity scoring systems for patients with COPD. At present, we grade the disease based on a single objective physiologic measure such as FEV1.

FEV1 as a % of its predicted value is the best single correlate of mortality in COPD. However, it is not until values fall to < 50% of predicted that mortality begins to increase. It follows that there is a need for a more comprehensive staging system that includes age, FEV1, ABG, body mass index, time walked distance, possible bio markers and genetic markers.

CHEST X-RAY

The main utility of the chest x-ray lies in excluding or suggesting alternate diagnoses that could cause a patient’s respiratory symptoms.

In patients with severe emphysema, the chest x-ray may reveal bilateral lung hyperinflation with flattened diaphragms or the presence of bullae, characterized by thin arcuate lines circumscribing areas of radiolucency .It is important to bear in mind, how ever that a normal chest x-ray does not exclude the presence of emphysema. In one study, spirometry and HRCT were

performed on individuals with more than thirty pack years of smoking and normal chest x-rays .It was found that 58% of these individuals had evidence of significant emphysema.

In chronic bronchitis, the principle abnormalities are bronchial wall thickening and an increase in lung markings which is sometimes termed “dirty chest”, refers to a general accentuation of linear markings throughout the lungs associated with loss of definition of vascular markings. Bronchial wall thickening may be manifested as ring shadows end on or as tubular shadows en face (tram tracks).

The chest x-ray may also reveal radiographic changes of pulmonary hypertension in COPD .Patients can have right main pulmonary artery >16 mm in diameter and left main pulmonary prominence below aortic knuckle with pruning or poorly visualized peripheral vasculature .In lateral view right ventricular encroachment into the retrosternal air space can be seen .

ARTERIAL BLOOD GAS ANALYSIS

In my study, hypoxemia was found in 22 patients and hypercapnia. The hypercapnia was found only in patients having severe or very severe COPD.

Arterial blood gases are commonly abnormal; as a rule, the more the severe the disease, the frequent the hypoxemia and hypercapnia. Arterial hypoxemia is the result of alveolar hypoventilation and ventilation – perfusion mismatching.

In COPD of mild to moderate severity, hypoxemia exits without hypercapnia. Although the V/Q inequality impairs both the uptake of oxygen and elimination of carbon dioxide , the tendency for elevation of $PaCO_2$ is overcome by an increase in alveolar ventilation to well perfused units .However ,the increase in ventilation cannot correct the hypoxemia because of the nonlinear shape of the oxygen dissociation curve .

An increase in arterial $PaCO_2$ does not generally occur until FEV1 is less than approximately 1.2 litres, and the pressure of hypercapnia in a patient with FEV1 > 1.5 litres should raise the possibility of central hypoventilation or obstructive sleep apnea.

Patients with COPD may experience episodic arterial desaturation during sleep, being more severe in patients categorized as blue bloaters than pink puffers. The desaturation is more during the REM sleep, that is partly due to the phasic inhibition of intercostal inspiratory muscle tone that is characteristic of REM sleep.

Approximately 20% of patients with COPD and normal awake arterial Oxygen tension have nocturnal, nonapneic oxygen desatuation. Exertional oxygen desaturation is also common. These episodes are ameliorated with supplemental oxygen.

Consequently, exercise and sleep oximetry should be completed in all patients with pulmonary hypertension. A formal overnight polysomnogram is indicated if the clinical presentation suggests sleep apnea.

In a recent prospective study of 43 patients with pulmonary arterial hypertension, with normal resting oxygenation, it was found that 70% had evidence of nocturnal hypoxemia.

ELECTRO CARDIOGRAPHIC FINDINGS

The ECG signs satisfying the right ventricular hypertrophy (RVH) criteria was found in 28 cases.

Diagnostic criteria for RVH for persons older than 30 years of age

- Right axis deviation > + 110^{o}
- Tall R wave in V_1 > or = 7 mm, S wave in V_1 < or = 2 mm
- R/S ratio in V_1 > 1, R/S ratio in V_5 or V_6 or = 1
- S wave V_5 or V_6 > 2 mm
- qR pattern in V_1 (increases specificity)
- Most important is scrutiny for right atrial enlargement, peaked P waves with an amplitude in V_1, V_2 or V_3 > 1.5 mm or > 2.5 mm in II, III, aVF.
- More than or equal to two criteria are required for the diagnosis of RVH.

In an analysis of the contribution of individual signs in defining the long term prognosis of COPD patients, it was found that P wave axis of more than or equal to + 90^{o} (a sign of severe right atrial over load) and the SI, SII, SIII pattern are independent negative prognostic predictors for survival. In the

patients presenting with one or both of these signs, had a three year survival rate of 44 and 14 % respectively versus 50 and 61% for patients having other or no use ECG signs of chronic corpulmonale. The remaining signs of chronic corpulmonale like RBBB, low voltage QRS, SIQ3 pattern were less consistently associated with poor survival.

A P wave axis > or = 70^0 qualifies as the ECG hallmark of, and thus a screening criterion for COPD. A P wave axis more than or equal to 90^0 probably identifies the stage of lung hyperinflation corresponding to very severe or almost terminal illness. In the two studies assessing this sign, its prevalence was known to be 24 % and 15% respectively.

Although highly specific, ECG is generally insensitive in detecting pulmonary arterial hypertension (PAH). Kilcoyn et al evaluated the ECG of 200 patients with COPD and Corpulmonale and noted at least one of the following changes:

A rightward shift of the mean QRS axis 30 degrees or more from its previous position

- Inverted biphasic or flattened T waves in precordial leads
- ST depression in II, III, AVF
- Right bundle branch block (RBBB)

Incalzi et al reported that an SI, SII, SIII pattern, right atrial overload and alveolar – arterial oxygen gradient more than 48 mmhg during oxygen therapy were the strongest predictors of death.

SI, SII, SIII pattern is a relatively uncommon finding not highly specific for COPD. It reflects an abnormal wave front rightward and superiorly oriented and opposed to the electrical forms of ventricular free wall. Low voltage QRS is frequently associated with chronic corpulmonale from COPD, but not associated with corpulmonale from other pulmonary diseases.

Kok – Jensen studied the ECG of 228 patients between 40 to 69 years of age with COPD. According to him, the survival was very poor in the groups of patients with an ECG showing a QRS axis + 90^0 to 180^0 and a PII amplitude of 0.20 mv or more. Only 37% and 42% of the patients with these changes were alive after four years. Patients with changes only in standard leads had a significantly better survival than those with changes in precordial leads as well.

ECHOCARDIOGRAPHIC FINDINGS

In my study, 21 patients out of 28 who had evidence of right ventricular hypertrophy had elevated pulmonary artery pressure by Doppler echocardiography (> 30 mmhg systolic or > 20 mmhg mean pulmonary artery pressure).

Echocardiographic screening for pulmonary hypertension is based on identification of the tricuspid regurgitant jet (TR), absent in normal individuals. Measurements of TR velocity (m / sec) provides an estimate of the back flow between the right ventricle and the right atrium. The systolic pulmonary artery pressure is estimated by the modified Bernoulli equation which is:

$$P = 4\ V^2$$

where V is the velocity of the tricuspid regurgitant jet. By adding this pressure gradient to an estimate of the right atrial pressure, the right ventricle peak systolic pressure is determined. The right ventricle peak systolic pressure approximates pulmonary artery systolic pressure (PSAP) obtained by right heart catheterization.

Numerous studies have examined the correlation between right ventricular systolic pressure (RVSP) as estimated by Doppler echocardiography and RVSP as directly measured during right heart catheterization, and most of the studies reported a relatively tight correlation (the r value ranged from 0.57 – 0.95). In a study by Hinderliter and colleagues, systolic pulmonary artery pressure was underestimated by atleast 20 mmhg in 31% of patients. Other studies have demonstrated that the concordance between Doppler echocardiography and direct measurement via right heart catheterization worsens as the pressure rises, with poorer correlation when the systolic pulmonary arterial pressure is over 100mmhg.

P Sahoo and Misra et al observed that in their ECG and echocardiographic evaluation of 50 cases, ECG abnormalities were found in 24 cases. Out of these, 20 cases had echocardiographic evidence of raised pulmonary artery pressure.

In addition, 2 dimensional echocardiography can be used to assess RV dimensions and wall thickness. Pulmonary artery hypertension can also be assessed with pulsed Doppler echocardiography from the sub – xiphoid region using a general purpose ultrasound device.

CONCLUSION

In my study of 50 cases of COPD, the conclusions are the following:

- COPD is commonly seen in persons above 40 years of age ie., in the middle and old aged people.

- It is more common among males.

- It is associated with the smoking pattern of more than 20 pack years. Its severity increases with increasing age and duration of smoking.

- ECG abnormalities suggestive of pulmonary hypertension (Right ventricular hypertrophy) was found in more than half of these cases. Poor progression with R/S is < 1 was found in 12 cases, right ventricular strain patterns like ST depression in II, III AVF and T wave inversion was found in 7 and 6 cases respectively, low voltage QRS in 5 cases. Other rare ECG abnormalities like lead I sign and SI, S II, S III was found in 2 and 1 cases respectively. Rhythm abnormalities like ventricular ectopics, RBBB are found in 2 cases and multi focal atrial tachycardia in 1 case.

- The presence of raised pulmonary artery pressure was confirmed using the Doppler Echo in 75 % of the patients showing ECG evidence of right ventricular hypertrophy.

SUMMARY

COPD is a major and increasing health problem which is predicted to become the third commonest cause of death and fifth commonest cause of disability in the world by the year 2020. Despite its enormous global importance, there has been relatively little research into COPD and it is the most under-funded disease in relation to the global burden of diseases.

This study was carried out to see the ECG and Echocardiographic signs in COPD. It was conducted in 50 cases of COPD diagnosed clinically and confirmed by Spirometry. It is an observational study and the relevant information was obtained in a predesigned proforma using interview technique. All the necessary investigations was done and ECG was done in all cases. 2 D and Doppler Echocardiography was done in cases showing ECG evidence of right ventricular hypertrophy (RVH). The results were carefully documented. ECG evidence of RVH was found in 28 cases, out of which 21 cases was confirmed using echocardiography. Thus, raised pulmonary artery pressure was found in 75 % of cases showing ECG evidence of Right Ventricular Hypertrophy.

LIST OF ABBREVIATIONS

ABG	Arterial Blood Gas
ATS	American Thoracic Society
COPD	Chronic Obstructive Pulmonary Disease
CXR	Chest X-Ray
DALY	Disability Adjusted Life Years
ECG	Electro Cardio Gram
ELISA	Enzyme Linked Immunosorbent Assay
FEV 1	Forced Expiratory Volume in one second
FVC	Forced Vital Capacity
GOLD	Global Initiative on Obstructive Lung Disease
HRCT	High Resonance Computerized Tomogram
HIV	Human Immunodeficiency Virus
IVS	Inter Ventricular Septum
PAP	Pulmonary Artery Pressure
PEFR	Peak Expiratory Flow Rate
PFT	Pulmonary Function Test
RA	Right Atrium
RBBB	Right Bundle Branch Block
RV	Right Ventricle
TNF	Tumor Necrosis Factor
WHO	World Health Organization

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MASTER CHART

S NO.	NAME	AGE / SEX	FVC	FEV1	FEV1/ FVC %	QRS AXIS	P PULM	R/S IN V_1	R/S IN V_6	RBBB	RV STRAIN	PAP (mmhg)	OTHERS
1	Adam mohammed	48 / M	1.8	0.7	44	+90		<1	<1			36	
2	Noor jehan	48/ F	2.1	1.2	57	+80		<1	>1				
3	Daisy Solomon	50/F	1.8	1.0	55	+70		<1	=1				
4	Krishnaiah	35 / M	2.0	1.3	68	+70		<1	>1				Sinus tachycardia
5	Malarkodi	50/F	1.7	0.8	77	+110		=1	<1			34	Low volatage QRS
6	Dhanalakshmi	52/F	1.9	0.9	47	+120	+	>1	>1	+		38	SI, SII, SIII
7	Chakravarthi	45/M	2.1	1.2	27	+80		<1	>1				
8	Sundar	43/M	1.8	1.0	56	+70		<1	>1				
9	Gopalakrishnan	58/M	1.9	1.0	53	+110		=1	>1			36	
10	Govindan	50/M	2.4	1.1	48	+90		<1	<1				
11	Jayanthy	56/F	2.1	1.2	57	+70		<1	>1		+	40	
12	Mariamma	40/F	1.8	0.9	50	+80		<1	>1				
13	Shankar	37/M	2.3	1.9	61	+90		<1	>1		+		Lead 1 sign
14	navaneethan	57/M	2.2	1.1	50	+70		<1	>1				Ventricular ectopics
15	Kandaswamy	64/M	2.1	1.0	47	+90		<1	<1			38	
16	Sankarnarayan	57/M	2.1	1.2	57	-60		<1	>1				
17	Kannammal	75/F	1.4	0.5	36	+140	+	>1	<1		+	64	Low voltage

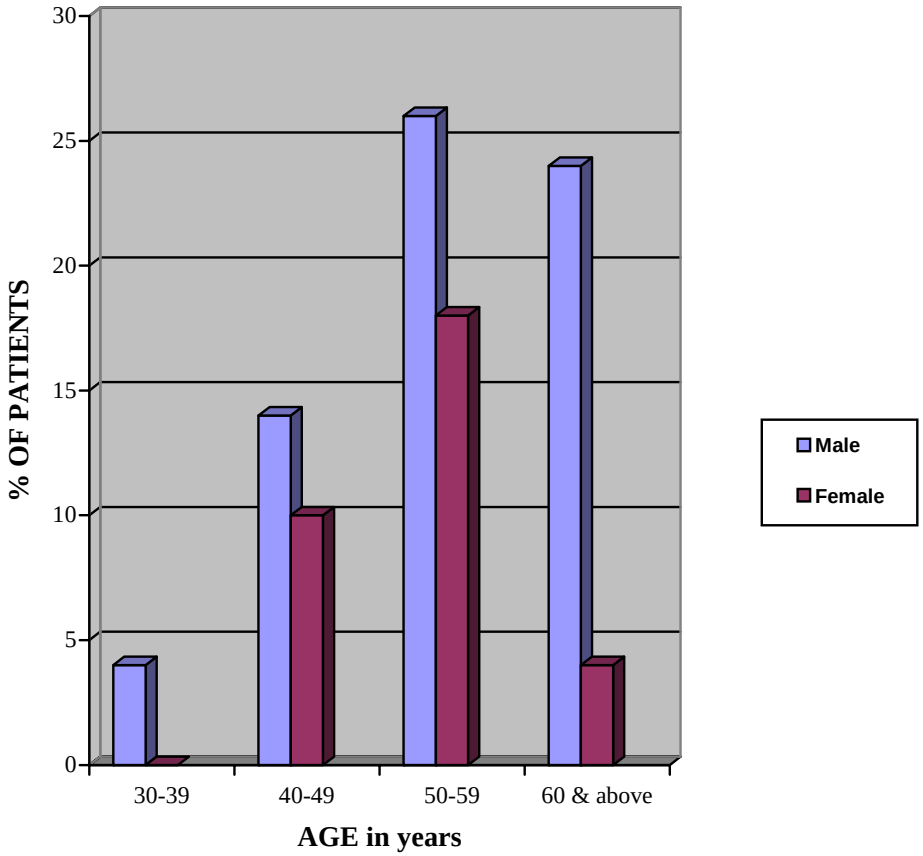
S NO.	NAME	AGE / SEX	FVC	FEV1	FEV1/ FVC %	QRS AXIS	P PULM	R/S IN V_1	R/S IN V_6	RBBB	RV STRAIN	PAP (mmhg)	OTHERS
													QRS
18	Kuppammal	40/F	3.1	2.1	67	+70		<1	>1				Sinus tachycardia
19	Agis Khan	45/M	1.4	0.8	57	+80		<1	>1				
20	Unnamalai	62/F	1.8	0.7	44	+120	+	>1	>1			72	
21	Vasantharaj	42/M	2.3	1.3	56	+80		<1	>1				
22	Jothi	56/M	1.8	1.0	55	+70		<1	>1				
23	Chandran	53/M	2.1	1.2	57	+80		<1	>1				Low voltage QRS
24	Ethiraj	53/M	2.0	1.0	50	+70		<1	>1				
25	Rosamma	55/F	1.8	0.9	50	+90		<1	>1			40	
26	Mahalingam	59/M	1.8	1.0	56	+70		<1	>1		+	42	
27	Kannammal	50/F	1.8	1.0	55	+80		<1	<1				
28	Ramadas	60/M	2.2	1.0	49	+90		<1	>1				Lead 1 sign
29	Arumugam	40/M	1.7	1.0	59	+90		<1	>1				Ventricular ectopics
30	Perumal	55/M	1.8	0.9	50	+70		<1	=1			42	
31	Jayaraman	59/M	2.1	0.9	46	+90		<1	>1				
32	Pachaiammal	40/F	1.8	0.9	50	+90		<1	>1				
33	Raja	42/M	1.7	0.7	41	+120	+	>1	<1			48	
34	Radhakrishnan	56/M	1.8	1.0	56	+70		<1	>1				
35	Ponnusamy	52/M	1.9	0.8	50	+80		<1	=1				

S NO.	NAME	AGE / SEX	FVC	FEV1	FEV1/ FVC %	QRS AXIS	P PULM	R/S IN V_1	R/S IN V_6	RBBB	RV STRAIN	PAP (mmhg)	OTHERS
36	Lalitha	45/F	2.4	1.1	48	+110	+	=1	<1			48	
37	Ranjitham	55/F	1.9	1.0	52	+70		<1	>1				Multifocal atrial Tachycardia
38	Padmavathy	56/F	1.6	0.7	45	+110		=1	>1			46	
39	Kanikannan	82/M	1.6	0.6	37	+110		<1	<1			50	
40	Selvaraj	68/M	1.7	0.7	38	+120	+	>1	>1		+	44	Sinus tachycardia
41	Manickam	60/M	1.8	1.0	55	+90		<1	>1				
42	Sethuraman	75/M	1.7	0.7	41	+120		>1	>1	+		54	
43	Manoharan	60/M	1.9	1.0	52	+90		<1	<1				
44	Subramani	60/M	1.8	1.0	55	-30		<1	=1				
45	Venkatesan	60/M	1.8	0.9	50	+70		<1	>1				Low voltage QRS
46	Stalin Rehman	62/M	1.5	0.6	40	+110	+	=1	>1			56	
47	Satyanarayanan	64/M	1.8	0.7	44	+100		<1	<1			34	Low voltage QRS
48	Abdul khuda	61/M	1.9	0.9	48	+100		<1	<1			56	
49	Kanakamma	55/F	1.7	0.7	41	+120	+	>1	>1		+	38	
50	Ashok	58/M	1.8	0.9	50	+80		<1	>1				

Fig 1. SPIROMETER

Fig 2. PATIENT PERFORMING SPIROMETRY

AGE- SEX DISTRIBUTION



MicroLab Spiro V 1.34

GOVT GENERAL HOSPITAL
DEPT OF GENERAL MEDICINE

Abirami
I.D: 12345678
Sex: Female Age: 30
Factor: 90(Asian)
Height: 152cm Weight: 62kg BMI: 26.8

	FEV1	FVC	PEF	Var	Quality	Time:	Date:
Base	0.49	2.69	10	0%	Slow start	00:50	01-01-08

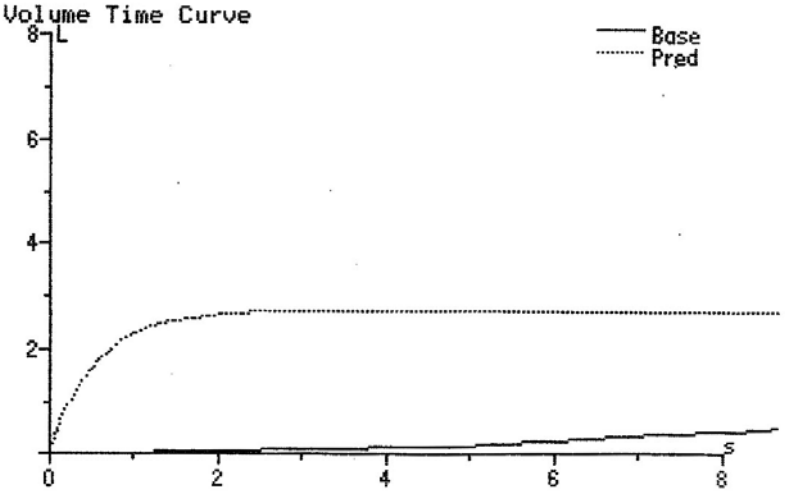
Variation is based on FEV1 + FVC

Best Spirometry Result:

			¦-- Normal --¦¦--- Post1 ---¦					
	Base	%Pr	Min	Pred	Max	Post	%Pr	%Chg
EVC	3.16	104	2.33	3.02	3.71			l
FEV1	0.49	20	1.83	2.39	2.95			l
FVC	2.69	97	2.12	2.76	3.40			l
PEF	9.60	2	292	381	470			l/m
FEV1/VC	15.5							%
FEV1/FVC	18.2	21	72.7	83.4	94.1			%
MVV(ind)	18.4	20	68.6	89.6	110			l/m
PIF	12.6							l/m
FET	22.0							s

Interpretation(Enright): Severe Obstruction.

Volume Time Curve



Micro Medical Ltd

Normal Values: ECCS (Adult);
Zapletal, Solymar, Cogswell (Child)
Results at BTPS.

Fig 3. SPIROMETRY REPORT

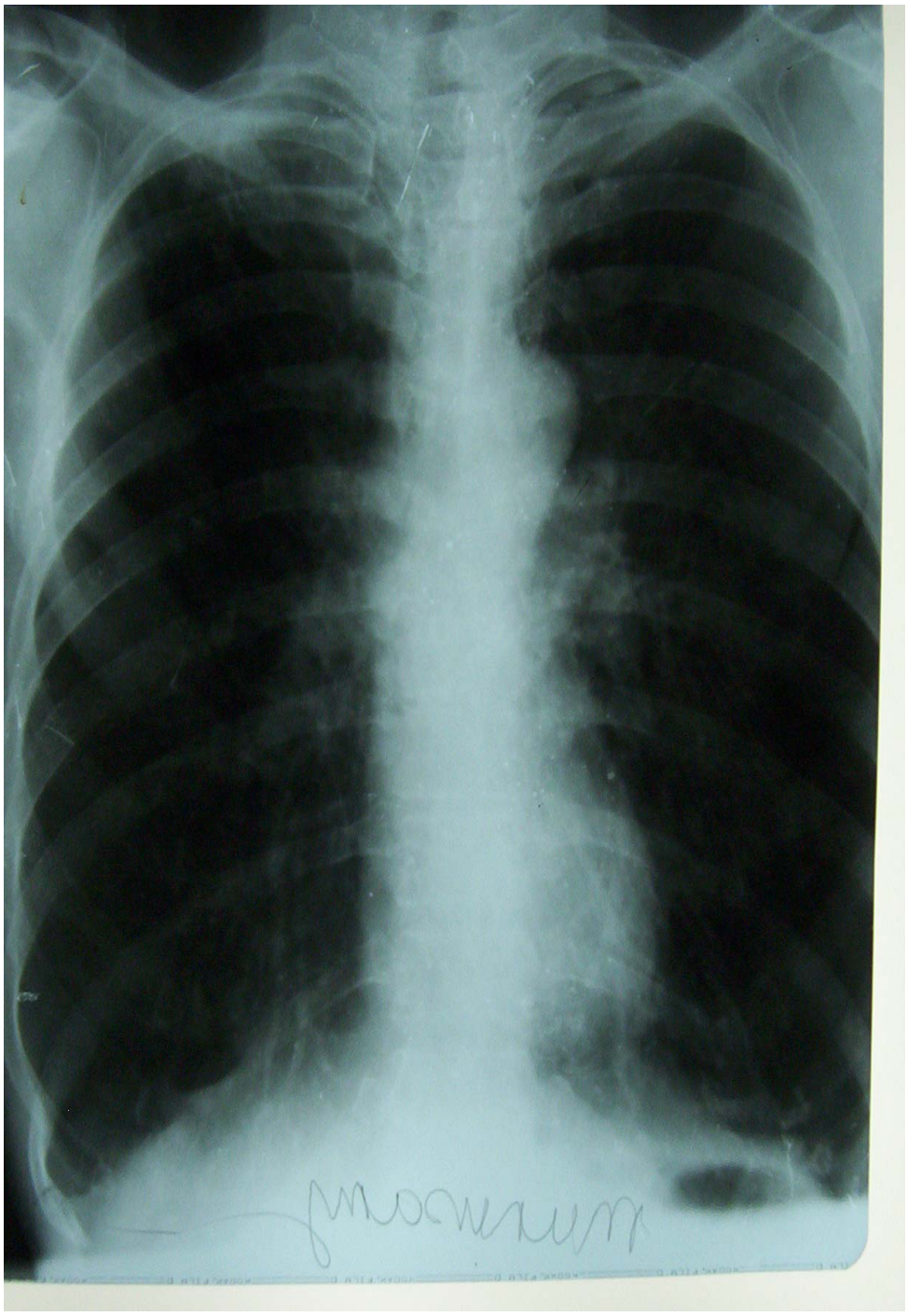


Fig 4.EMPHYSEMA

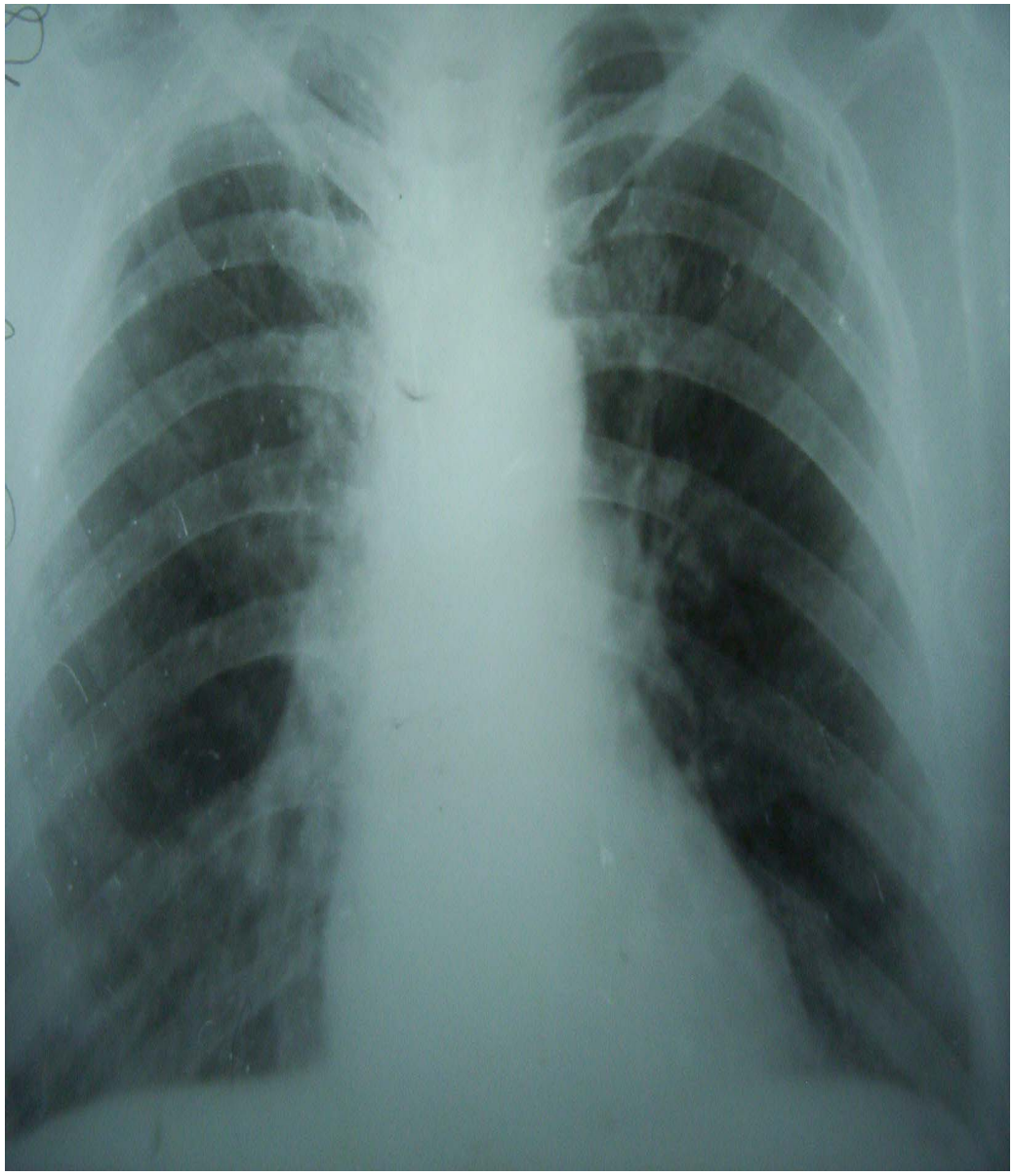


Fig 5.CHRONIC BRONCHITIS SHOWING PROMINENT BRONCHO VASCULAR MARKINGS

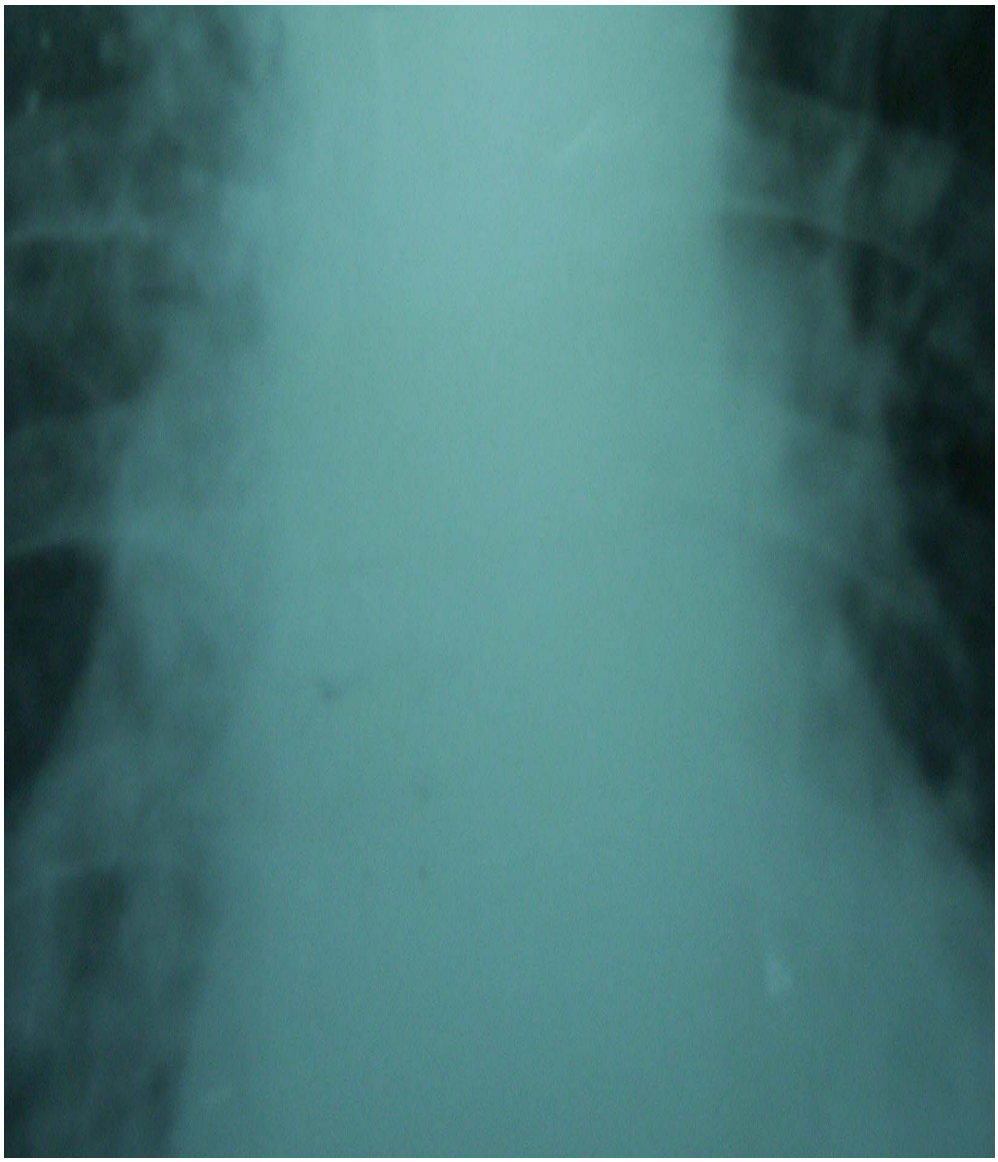


Fig 6.CHEST X-RAY SHOWING PULMONARY ARTERIAL ENLARGEMENT

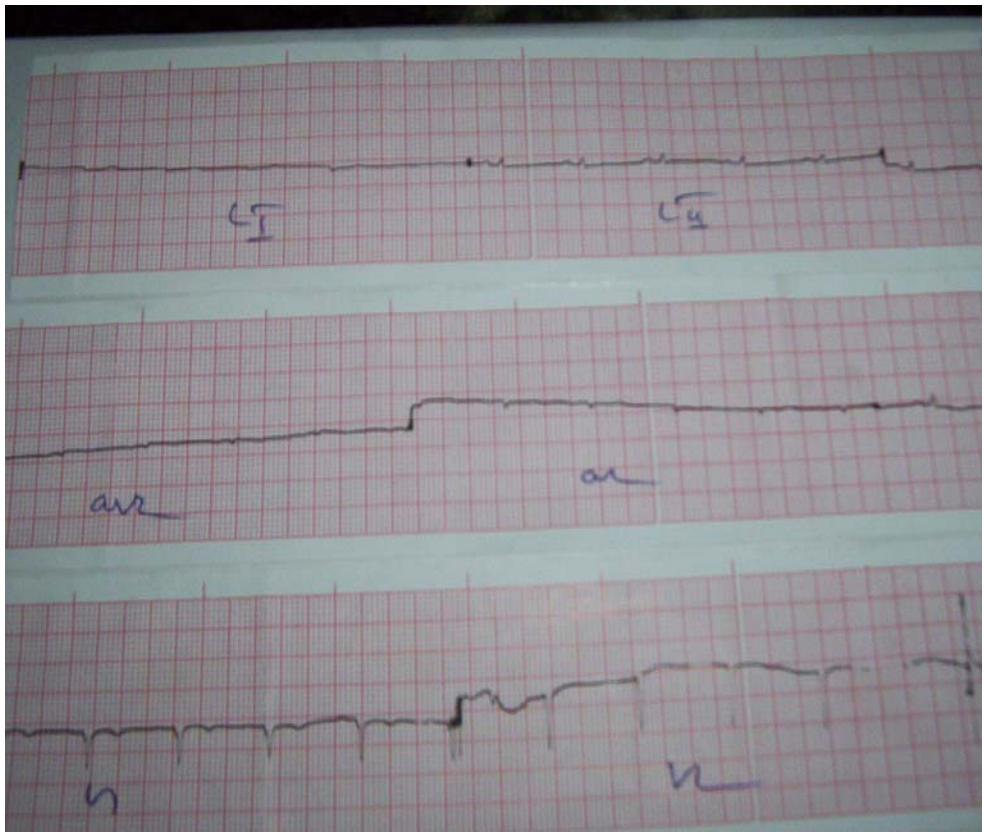


Fig 7.ECG SHOWING LOW VOLTAGE QRS

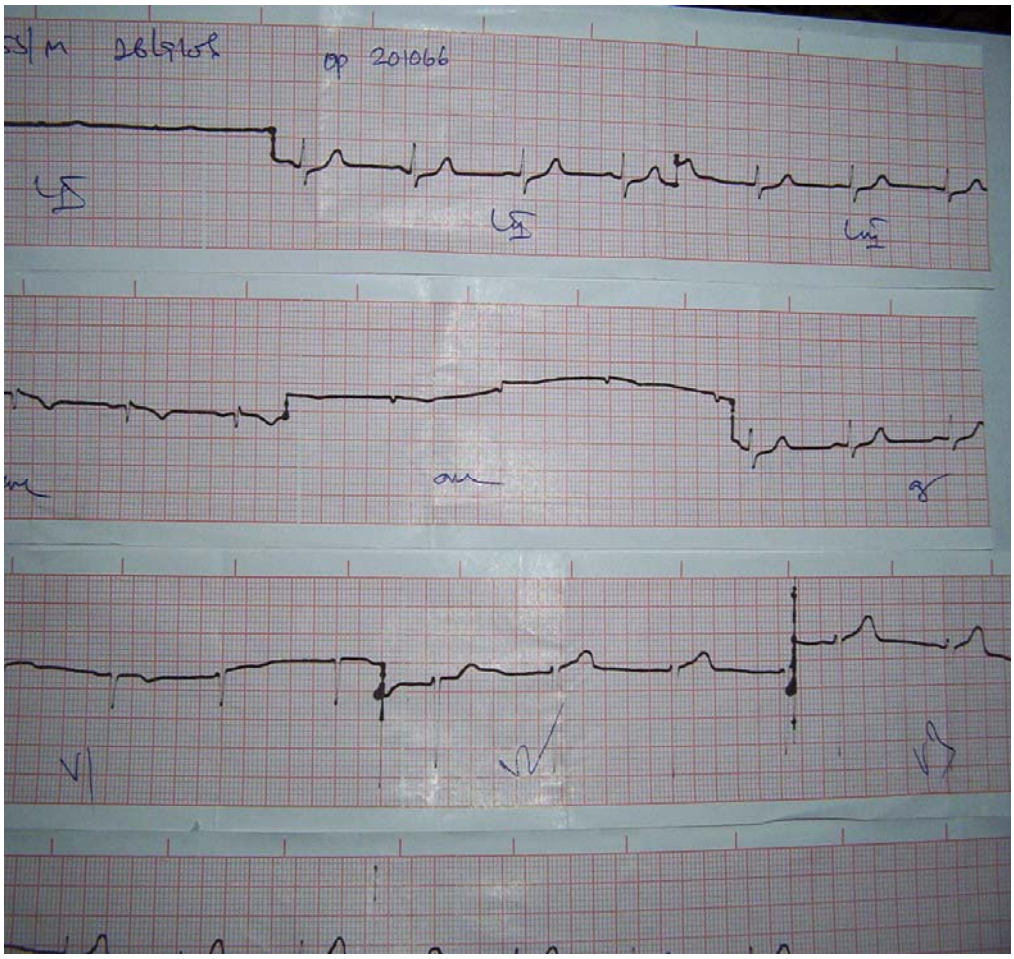


Fig 8. ECG SHOWING LEAD I SIGN

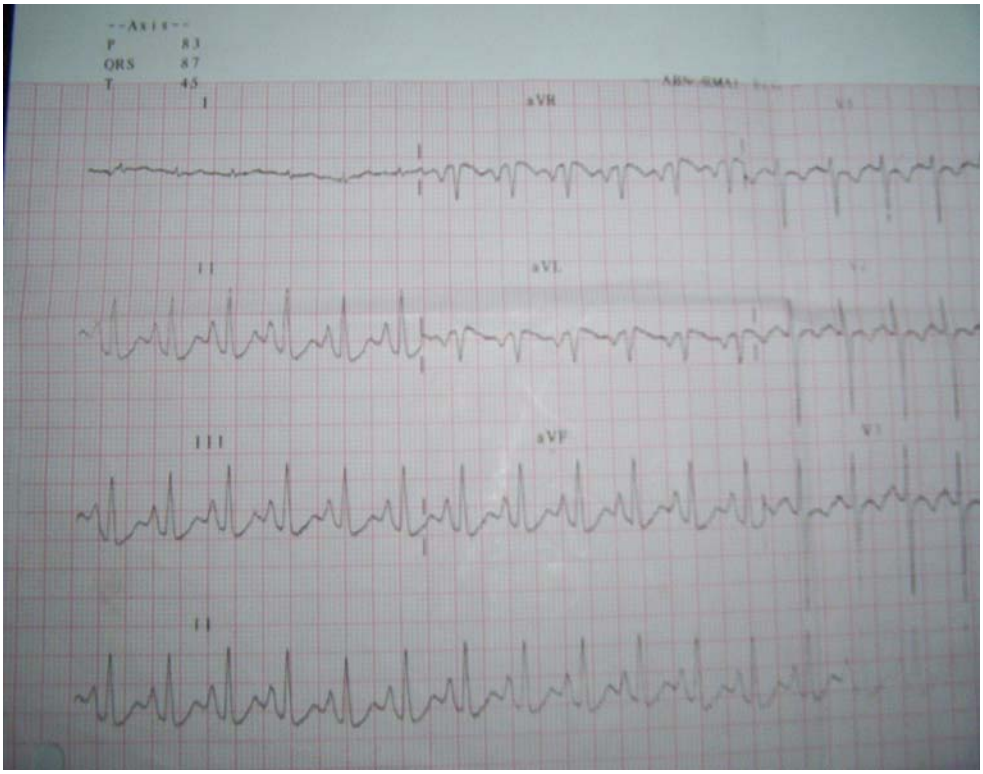


Fig 9.ECG SHOWING P PULMONALE

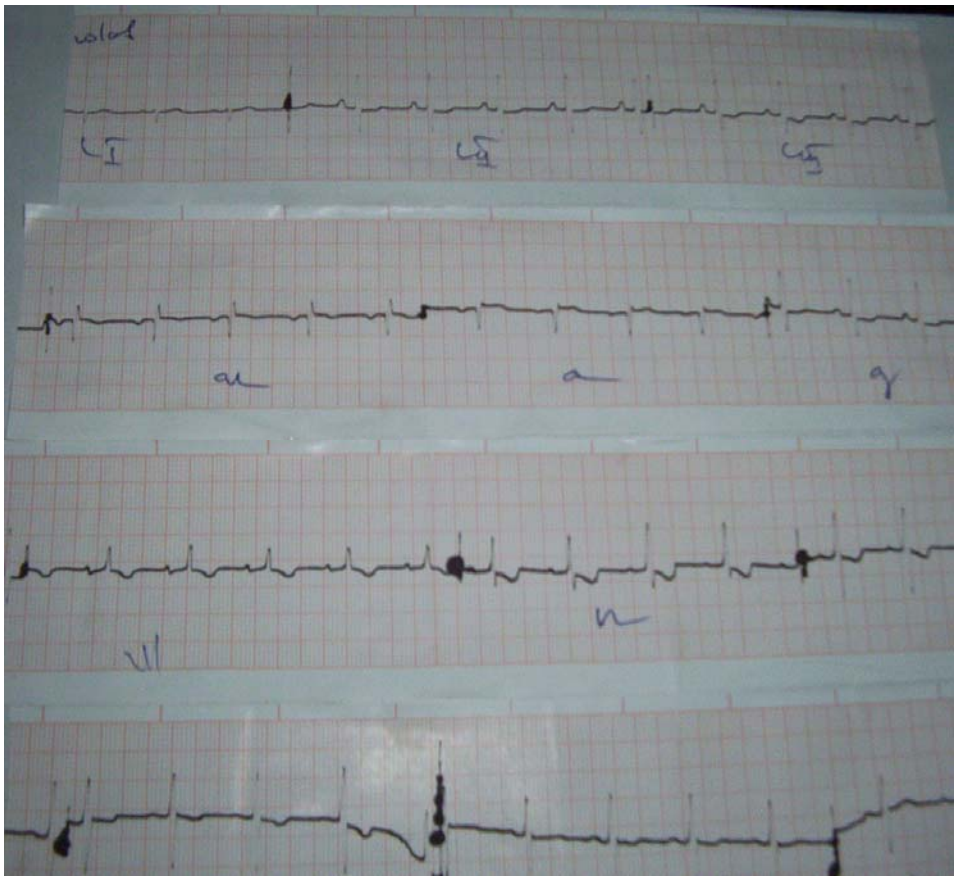


Fig 10. ECG SHOWING RVH

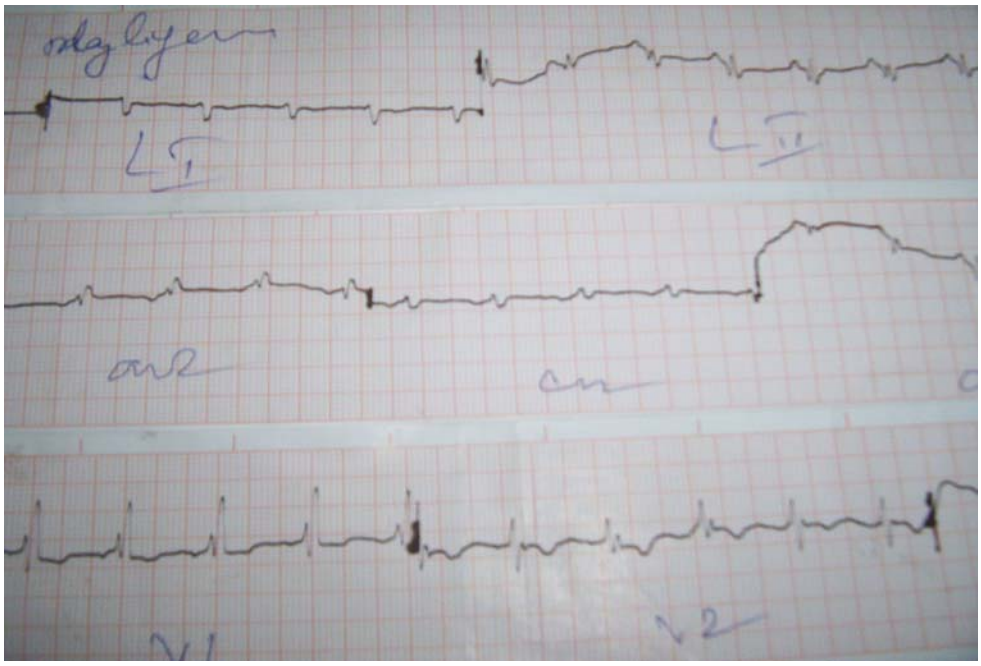


Fig 11.ECG SHOWING RBBB

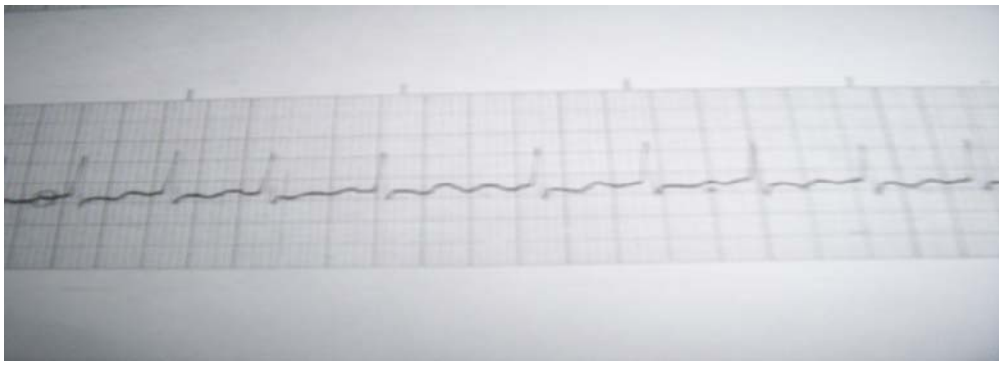


Fig 12.ECG SHOWING MULTIFOCAL ATRIAL TACHYCARDIA

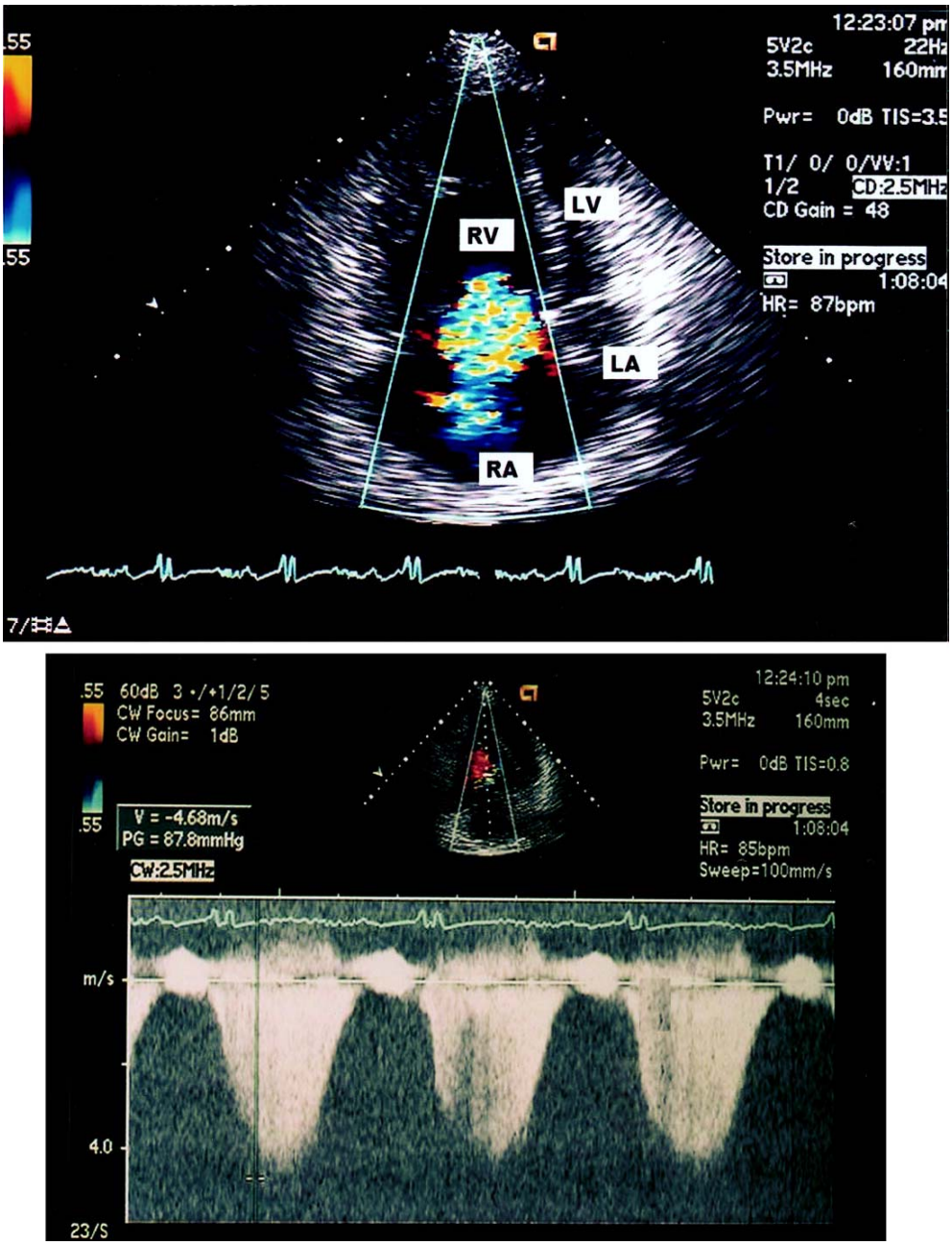


Fig 13. DOPPLER ECHO SHOWING REGURGITANT JET WITH DILATED RA AND RV