AN ANALYSIS OF PULMONARY FUNCTION TESTS, PULSE OXIMETRY, HAEMATOCRIT ABNORMALITIES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE PATIENTS

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CERTIFICATE

This is to certify that the Dissertation entitled **"An Analysis of Pulmonary Function Tests, Pulse Oximetry, Haematocrit Abnormalities in Chronic Obstructive Pulmonary Disease Patients",** herewith submitted by **Dr. R. Arunagiri,** Post graduate in General Medicine, Coimbatore Medical College to the Tamilnadu Dr. M.G.R. Medical University is a record of a bonafide research work carried out by him under my guidance and supervision from January 2006 to June 2007.

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Prof. Dr. K. Umakanthan
Prof. and Head
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DEAN

DECLARATION

I solemnly declare that the Dissertation titled **"An Analysis of Pulmonary Function Tests, Pulse Oximetry, Haematocrit Abnormalities in Chronic Obstructive Pulmonary Disease Patients",** was done by me at Coimbatore Medical College & Hospital during the period from January 2006 to June 2007 under the guidance and supervision of Prof. Dr. K. Umakanthan and Prof. Dr. S.Prabha.

This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch I) in General Medicine.

Place : Coimbatore **Dr. R. Arunagiri**

Date :

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CONTENTS

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the name of a group of chronic and slowly progressive respiratory disorders characterized by reduced maximal expiratory flow during forced expiration[1].

COPD is a common and preventable disease that has great implications on global health. It is the fourth leading cause of death world over, exceeded only by myocardial infarction, malignancy and stroke[2].

COPD is known to cause airflow limitation, impaired gas exchange and it also has effects on the pulmonary circulation.

Pulmonary haemodynamics in patients with COPD depends on the stage of the disease. In patients with mild obstruction and without severe hypoxemia pulmonary arterial pressure is normal at rest. As air flow limitation worsens, along with development of chronic severe hypoxemia and hypercapnea the pulmonary artery pressure is increased at rest.

Spirometers provide quick assessment of expiratory function that corrects with FEV1 & also enable us to differentiate between restrictive, obstructive & proximal air way disease [3].

The combination of Pulse oximetry & spirometry give valuable Information about patient's status.

Long standing COPD disease can lead to exertional & nocturnal hypoxemia.

Frequent Hypoxic episodes & nocturnal hypoxemia leads to the development of secondary polycythemia & its consequences.[4]

AIM OF THE STUDY

To Study the correlation of clinical features, Pulmonary function tests, Pulse oximetry assessment & Haematocrit abnormalities in chronic obstructive pulmonary disease.

To assess the severity of chronic obstructive pulmonary disease by pulmonary function tests.

To correlate the development of hypoxia and polycythemia with respect to severity and duration of the disease.

To study about the Influence of smoking habit in the development & progression of COPD.

Review of Literature

REVIEW OF LITERATURE

HISTORICAL ASPECTS

Arteams (260 AD) had given many clinical descriptions of pulmonary disease of which 'pneumodes' might be the same as chronic bronchitis, emphysema or bronchial asthma leading to congestive cardiac failure.

In Europe, Bodham (1808) and Laennec (1827) made the classic description of chronic bronchitis & emphysema[5].

Laennec in 1926 described the clinical and pathological characteristics of cause heart failure even in otherwise healthy heart.

In 1934 Kountz and Alexander who were studying emphysema stated that "it appears that heart is affected in majority of patients with emphysema".

I.T.T. Higgins in 1959 had studied men between the ages of 25 and 64 to know the relationship between smoking and respiratory symptoms. A clear relationship between smoking and persistent cough and sputum production has been found[6].

- 1960 Benjamin Burrows studies 200 patients and found out mild chronic dyspnea had developed usually at an FEV_1 between 1.5 to

2.0 litres, one would expect progression to more disability in about 10 additional years[7].

- 1963 Christer Larson studies 246 Swedish adults with severe α1 – antitrypsin deficiency, primary emphysema was present in 109 cases. Out of other patients with other types of COPD, few showed signs of emphysema.[8]
- 1959 The Medical Research Council in its publications used the term chronic bronchitis to define expectoration when other causes such as bronchiectasis or tuberculosis have been excluded, to patients who have coughed up sputum on most days during at least for 3 consecutive months in 2 successive years[9].
- 1975, Earliest nocturnal polygraph studies were done
- 1977, Flick MR et al, did a land mark study is to show that Patients with COPD experienced a worsening of hypoxia.[10]
- 1964-1973 Boushy and colleagues published a series of papers on the subject of prognostic factors in COPD and the prognostic values of lung function tests in COPD[11,12,13].
- 1977 Boushy SF et al. described the results of hemodynamic changes in 136 patients with COPD including serial studies, correlating pulmonary function tests, arterial oxygen pressure, and arterial carbon dioxide tension with haemodynamic parameters[14].

- 1983, Catterall TR et al., studied about transient hypoxemia during sleep in COPD patients[15]
- Long-term oxygen in conjunction with pulmonary rehabilitation, it also improves quality of life[16]
- 1991, Weitzen blume et.al. analysed the evaluation of physiological variables in patients with COPD before & during long term oxygen trerpy[17].
- 1991, Swimburn stone TN et al., evaluated the symptomatic benefit of supplemental oxygen in hypoxemic patients with chronic lung disease [18]

DEFINITIONS

Chronic obstructive pulmonary disease (COPD) is the name of a group of chronic and slowly progressive respiratory disorders, characterized by reduced maximal expiratory flow during forced exhalation[1]. Most of the airflow obstructions are fixed.

The American Thoracic Society (ATS) defines COPD as a disease process involving progressive chronic airflow obstruction because of chronic bronchitis, emphysema, or both[19].

COPD comprises emphysema, chronic bronchitis & small air way disease, although they most often present in combinations. The definition

excludes other causes of chronic airflow obstruction such as cystic fibrosis, bronchiolitis obliterans and bronchiectasis.

Chronic bronchitis is defined as the presence of a chronic productive cough on most days, for 3 months, in each of two consecutive years in a patient in who other causes of chronic cough has been excluded.[9]

Emphysema is defined as abnormal permanent enlargement of distal air apices, distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis[20].

EPIDEMIOLOGY

COPD has a chronic protracted course spanning several years or even decades. COPD is responsible for considerable morbidity and mortality in the community especially among the senior citizens. It is of considerable public health importance as there disorders are to a great extent preventable. COPD places an enormous burden on health care resources and it is one of the major causes of working days lost per years. Exacerbations of the disease are more frequent during winter months. Chronic bronchitis and emphysema usually coexist. Those patients with predominant chronic bronchitis or mainly emphysema form a minority at

either end of the disease spectrum of patients with COPD.

PREVALENCE

In India COPD is the second most common lung disorder after pulmonary tuberculosis[21] Overall the prevalence is higher in males due to greater prevalence of smoking. The disease is most often seen in middle aged or elderly people, it is infrequent before the age of 35 years. It has been reported from studies in North India that prevalence of chronic bronchitis may be as high as 15 % in subjects above 40 years from rural areas. Further the prevalence is more in northern India compared to south india[22]. The regional difference has been attributed to climatic conditions, particularly the severe winter in North India. The marked preponderance of males in urban India is less striking in case of rural areas in the country, this has been attributed to greater prevalence of smoking among women in rural areas, and it may also be related to marked indoor air pollution cakes, firewood and fossil fuels.

Bhattacharya et.al. studied chronic bronchitis in rural population aged more than 30 years and found prevalence of chronic bronchitis to be 57/1000[23]. There was male preponderance which was higher with increasing age. Smoking had directmale preponderance which was higher with increasing age. Smoking had direct relation to prevalence was

highest among those whose duration was more than 15 years. They also found out that prevalence was higher among Hukka smokers (85/'1000) as compared to beedi smokers (31/1000).

It is estimated that there are 14 million cases of COPD in the United States. About 12.5 million with chronic bronchitis and 1.65 million with emphysema. The male to female ratio ranges from 4-6 % / 1-4 % with increasing prevalence of smoking among females, prevalence of COPD in females also has increased.

The incidence of COPD has been linked to the prevalence of smoking in the population as this is the most important etiological factor. In a prospective study involving 40,000 medical practitioners in Britain, it was found that the death rate from chronic bronchitis was higher in smokers and increased with amount smoked. It was also found that those who stopped smoking, the mortality after 10 years were much lower than those continued to smoke[24].

AETIOLOGY

COPD is characterized by a reduced Forced Expiratory Volume in

1 second (FEV_1) and an accelerated rate of decline of FEV_1. The reduction in FEV_1 can occur by any of three pathways.

1. Impaired childhood growth and development, with a lower peak in early adulthood and a normal rate of decline with aging eg. Early Childhood infection and passive smoke exposure.
2. Normal growth and development with premature peak but normal subsequent decline eg: asthma and passive smoking.
3. Normal growth and development and peak, with accelerated decline eg: active smoking and to a lesser extent environmental exposures.

SMOKING

Cigarette smoking is the most commonly identified correlate with chronic bronchitis during life[25, 26]. Pipe and Cigar smokers have a higher mortality and morbidity rates for COPD than nonsmokers [27], but lesser than cigarettes smokers. Prevalence of COPD shows a dose response relationship with the number of pack-years of tobacco consumed. The British Thoracic Society guidelines suggest that most patients with COPD have at least 20 pack years smoking history[28]. An average cigarette smoker have high annual rate of decline in FEV_1, of about 50ml, which is nearly double the average value of 30ml in non smokers. In nonsmokers 1the decline in FEV_1 begins at 30-35 years of age and this may occur earlier in smokers. Stopping cigarette smoking does not produce a

substantial improvement in FEV_1 the subsequent but rate of decline is decreased.[30]

Clinically significant COPD develops in 15% of cigarette smokers. Age of initiation of smoking, total pack-years, and current smoking status predict COPD mortality. People who smoke have a greater annual decline in FEV_1. Overall, tobacco smoking accounts for as much as 90% of the risk.[5]

Prolonged cigarette smoking impairs respiratory epithelial ciliary movement, inhibits function of alveolar macrophages and leads to hyperplasia and hypertrophy of mucus secreting glands. Cigarette smoke also inhibits anti-proteases and causes polymorpho nuclear leukocytes to release proteolysis enzymes. Smoking is associated with increased airway responsiveness which is associated with more rapid progression in patients with COPD. Obstruction of small airway is the earliest demonstrable mechanical defect in a smoker. [29]

Secondhand smoke, or environmental tobacco smoke, increases the risk of respiratory infections, augments asthma symptoms, and causes a measurable reduction in pulmonary function[5]

ALPHA 1, ANTITRYPSIN DEFICIENCY

AAT deficiency is the only known genetic risk factor for

developing COPD.

α_1 Anti Trypsin (AT) is a polymorphic glycoprotein responsible for the majority of anti-protease activity in the serum, whose synthesis is governed by a gene on 14q 32 chromosome.

The most common deficient allele termed ZZ (Cor Pi^{zz} Phenotype) results from a single amino acid substitution 342Glu→Lys, which causes spontaneous polymerization of the polypeptide, markedly impairing its release into circulation from the liver. It is commonly seen among people from European descent 1:2000 to 1:7000 people, rare in people from African and Asian lineage.

α_1 AT deficiency accounts for 2% of observed cases of emphysema. Patients present with premature development of emphysema chronic bronchitis or bronchiectasis. The patient usually presents with cough and dyspnea in the fourth decade. Nearly 80% had a family history of lung disease with autosomal recessive inheritance. The average decline of FEV_1 is 100-130ml / yr for smokers and 50 to 80 ml /yr for ex-smokers or lifetime non smokers.

Pathologically panacinar emphysema predominates and radio graphically changes are most marked in lower lobes. Tobacco smoking is

an extremely important cofactor for development of disease in α_1 AT deficiency.

These patients are also at increased risk of hepatic cirrhosis.

AIR POLLUTION

Incidence and mortality rates of both chronic bronchitis and emphysema may be higher in industrialized urban areas. Exacerbation of bronchitis is clearly related to periods of heavy pollution with sulfur dioxide and particulate matter.

In developing countries like India traditional cooking fuels such as wood, cow dung cake, etc, along with poorly ventilated houses are significant risk factors for chronic bronchitis.

OCCUPATION

Chronic bronchitis is more prevalent in workers who engage in occupation exposing them to either inorganic or organic dusts, or to noxious gases. Surveys have found an accelerated decline in lung function in such workers eg, workers in plastic plants to exposure to Toluene etc. Exposure to cadmium can increase the chance of development of emphysema and hence COPD.

INFECTIONS

Frequency of acute respiratory illnesses is higher in patients with chronic bronchitis. Epidemiological studies however implicate acute respiratory illness as one of the major factors associated with etiology as well as the progression of chronic airway obstruction.

GROWTH AND NUTRITION

Studies have shown that nutrition may affect both the growth and decline in ventilator function. There is also some evidence that severe viral pneumonia early in life may lead to chronic obstruction, particularly in small airways.

PATHOLOGY

The pathologic changes of COPD involve large and small airways and the terminal respiratory unit.

Small airways are the major sites of airflow limitation. Small airways show a variety of lesions narrowing their Lumina, including goblet cell hyperplasia, mucosal and sub mucosal inflammatory cells, edema, peribronchial fibrosis, intraluminal mucus plugs and increased smooth muscle.

In large cartilaginous airways chronic bronchitis is associated with hypertrophy of sub mucosal mucus producing glands. This Mucous gland enlargement is the histological hallmark of chronic bronchitis. Quantization of this anatomic change is known as Reid index is based on thickness of sub mucosal glands to that of bronchial wall. In patients with chronic bronchitis it is 0.44 ± 0.09 otherwise normally 0.52 ± 0.08[4].

Emphysema begins as an increase in the number and sizes of alveolar fenestrate and results in eventual destruction of alveolar septae and their attachments to terminal and respiratory bronchioles.

Emphysema has 3 morphologic patterns :

Centriacinar emphysema is characterized by focal destruction limited to the respiratory bronchioles and the central portions of acinus. This form of emphysema is associated with cigarette smoking and is most severe in the upper lobes.

Panacinar emphysema, involves the entire alveolus distal to the terminal bronchiole. The panacinar type is most severe in the lower lung zones and generally develops in patients with homozygous alpha1-antitrypsin (AAT) deficiency.

Distal acinar emphysema or paraseptal emphysema is the least common form and involves distal airway structures, alveolar ducts, and sacs. This form of emphysema is localized to fibrous septa or to the pleura and leads to formation of bullae. The apical bullae may cause pneumothorax. Paraseptal emphysema is not associated with airflow obstruction.

PATHOPHYSIOLOGY

Airflow limitation: Airflow limitation and 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 radial traction on airways, or to increased resistance due to intrinsic narrowing of small airways.

Hyperinflation: The residual volume and the functional residual capacity (FRC) are almost higher than normal. In addition prolongation of expiration is associated with obstruction which would lead to dynamic increase in FRC (dynamic hyperinflation). Dynamic hyperinflation contributes additionally to discomfort associated with air flow obstruction by flattening the diaphragm fiber length and a perpendicular insertion with the lower ribs.

Impaired Gas Exchange: Maldistribution of inspired air and blood flow is always present. When the mismatching is severe, impairment of gas exchange is reflected in the abnormalities of arterial blood gases. Small airway narrowing causes a decrease in ventilation of their distal alveolar acini. When the alveolar capillaries remain intact, this results in mismatch of ventilation and perfusion leading to mild or moderate hypoxemia.

(i) Abnormal blood gas tensions

Hypoxemia: In COPD there is a negative correlation between oxygen saturation of the blood and pulmonary artery pressure [31,32,33,34]. Hypoxemia is known to be a potent arteriolar constrictor in the pulmonary circulation (As the severity of disease progresses in COPD there is more arterial desaturation correlating with an increase in pulmonary hypertension. Exacerbation of COPD with hypoxemia is associated with acute worsening of pulmonary hypertension[35]. Pulmonary artery pressure can also increase acutely during the episodes of hypoxemia that occur during rapid eye movement of sleep, and it has been suggested that recurrent nocturnal pulmonary hypertension can result in pathologic changes in pulmonary vessels and fixed hypertension[36].

Hypercapnea: In patients with COPD there is a positive correlation between arterial CO2 pressure (PaCO2) and pulmonary artery pressure[33,

[37]. The mechanism could be a change in lung mechanics due to hyperventilation induced by hypercapnea or the potentiating of hypoxic pulmonary vasoconstriction.

NOCTURNAL HYPOXEMIA

Along with FEV1, hypoxemia also one of the strong prognostic markers for COPD.

Healthy individuals have a mean nocturnal SpO2 of above or equal to 96%. Even in them during REM sleep there will be short periods of a physiological drop with a SpO2 between 93 and 96 percent[38] .

COPD Patients may develop substantial decreases in nocturnal PaO_2 during all phases of sleep, but particularly REM phase. These episodes initially are associated with a rise in pulmonary arterial pressures and disturbance in sleep architecture, but they may develop into Pulmonary arterial hypertension and cor pulmonale if hypoxemia remains untreated.

Desaturation episodes can be defined by a fall in pulse (SpO2) of more than 4%, compared to the baseline Level of SpO2 during stable respiration, and immediately preceding the hypoxemic episode.

Patients with severe COPD and with mild to moderate daytime hypoxemia (i.e. PaO2 between 55 and 70 mmHg) have a mean nocturnal SpO2 of approximately 90%. Daytime PaO2 is clearly the best predictive indication of nocturnalSpO2 if daytime hypoxemia is severe, or PaO2 below 55 mm Hg; mean nocturnal SpO2 may reach as low levels as 75 to 80%.

In a community-based study of 884 subjects with a mild obstructive airway disease, by Sanders MH, Newman AB et al showed that 11.4 % of patients had spO2 below 90 %. Which is remarkably high compared to a significantly lower value of 6.3 % of subjects without obstructive airway disease[39].

Two main mechanisms for sleep hypoxemia include alveolar hypoventilation and ventilation-perfusion mismatching.

The deleterious effects of hypoxemia proceed from the effects of tissue hypoxia on cell metabolism and organ function.

CONSEQUENCES OF HYPOXEMIA

Hemodynamic effects in the pulmonary circulation:

Severe episodes of nocturnal desaturation, leading to alveolar

hypoxemia causes vasoconstriction of the small pulmonary arteries and a rise in mean pulmonary arterial pressure (mPAP). [40, 42]

Arrhythmias: Tachycardia's and other cardiac dysrhythmias common in COPD patients during sleep. VPC' s are more frequent during severe nocturnal desaturation ($SpO2 < 80\ \%$) [41]

Secondary polycythemia: chronic hypoxia leads to increased levels of erythropoietin which in turn raises the red blood cell count & haematocrit values Exercise intolerance.

Acidemia: Hypoxia and acidemia act synergistically to produce pulmonary vasoconstriction in patients with CCPD. Thus for a given oxygen saturation, the mean Ppa is higher with increasing arterial hydrogen ion concentration[43].

(ii) Effects of Abnormal pulmonary mechanics :

Changes in airway resistance may augment pulmonary vascular resistance and increase in pulmonary artery pressure, correlating with decrease in FEV_1 [44, 45, 46].

POLYCYTHEMIA

Chronic hypoxia causes stimulation of erythropoietin production leading to compensatory erythrocytosis [60]. Polycythemia defined as

Hb >17 g/dL in men and >15 g/dL in women.

However Polycythemia is uncommon in COPD various studies show that it occurs in 5-8% of cases[48,49] cigarette smoking may determine the severity of secondary polycythemia in patients with hypoxic COPD, and prevent its correction by long-term oxygen therapy.

In a study by Atchison R, Russell N et al showed that commonest physiological abnormality in smokers was a raised red cell mass combined with a low plasma volume.

Six of the 14 patients in their study showed improvement in their haematocrit by reducing their smoking habit.

Another study by Calverley PM, Leggett RJ, et al also stated that Smoking as a major cause of polycythaemia[50] chronic elevation of carboxy haemoglobin to 10 or 15% in smokers can lead to secondary polycythemia.

Symptoms of Secondary Polycythemia include Weakness, Fatigue, Headache, Light-headedness, Shortness of breath, Night sweats, Gastrointestinal, Paresthesias Pruritus, Visual disturbance[59].

CLINICAL FEATURES[29]

Symptoms

The characteristic symptoms of COPD are breathlessness on exertion, mostly accompanied by wheeze and cough, which is often but not invariably productive. Most patients have a smoking history of at least 20 pack years before symptoms develop, commonly in the fifth decade.

Breathlessness is the symptom that causes most disability and is associated with loss of lung function over time. It is usually first noticed on climbing hills or stairs. The appearance of breathlessness indicates moderate to severe impairment of airway function.[51] by the time patient seeks medical help FEV_1 has usually fallen to, 1-1.5 lit in an average male. However, when the FEV-$_1$ has fallen to 30% or less of the predicted values breathlessness is usually present on minimal exertion.

In most patients with COPD cough precedes the onset of breathlessness. Sputum is usually mucoid in character, but becoming purulent during exacerbations. Volume is generally small and less than 60ml per day. As COPD progresses exacerbations becomes more frequent and severe.

Wheezing is present but is neither specific nor indicates the severity of obstruction.

With disease progression, intervals between acute exacerbations become shorter; cyanosis and right heart failure may occur. Anorexia and weight loss often develop and suggest a worse prognosis[9]

Physical Signs

These are not specific to the disease and depend on degree of air flow limitation over inflation. In early disease the only abnormal finding is wheeze on forced expiration and a forced expiratory time prolonged beyond 6 seconds with more advanced disease the breathing pattern is characterised with a prolonged expiratory phase. Some patients adopting pursed-lip breathing on expiration which may reduce expiratory airway collapse. The use of accessory muscles of respiration particularly sternomastiod is seen in advanced disease. These patients adopt the position of leaning forward, supporting themselves with their arms to fix the shoulder girdle and allowing the use of pectoralis and latissimus dorsi to increase chest wall movements. (The tripod position).

In later stages the chest is often barell shaped, an increased anterior posterior diameter, horizontal ribs, prominence of the sternal angle and wide sub costal angle. An inspiratory tracheal tug may be detected. The

horizontal position of diaphragm also acts to pull on the lower ribs during inspiration (Hoover's sign).[52]

On percussion there is decreased cardiac and hepatic dullness, indicating over inflation.

Breath sounds may have a prolonged expiratory phase or may be uniformly diminished.

Table -1 CLINICAL TYPES OF PATIENTS WITH COPD[53]

	Emphysema(Type A) Pink Puffer	**Chronic Bronchitis (Type B) Blue**

		Bloater
Age in years	55-75	45-65
Cough	Short duration (often after dyspnea starts)	Long duration (usually before dyspnea starts
Sputum	Scanty, mucoid	Copious, purulent
Recurrent infection	Mild problem	Severe problem
Chest roentgenogram	Normal or "emphysema"	Normal or "dirty lung"
Diffusing capacity	Low	Variable
Chronic hypercapnea	Unusual	Common
Chronic hypoxemia	Mild to moderate	Often severe
Erythrocytosis	Unusual	Common
Pulmonary artery pressure	Normal or slightly high	Often very high
Chronic cor pulmonale	Unusual	Common
Lung compliance	High	Normal
Recoil pressure	Low	Normal

RADIOLOGY

Chest X-ray: There are no specific features on plain chest-X-ray for chronic bronchitis. The features usually described are for emphysema.

Bronchial wall thickening seen as parallel line opacities on plain chest X-ray has been described in chronic bronchitis.

Radiographic signs for emphysema are:

- Low flattened diaphragm : The border of the diaphragm in the midclavicular line is below the seventh rib.
- Height of patient's lung being greater than 29.9 cm.
- In lateral film increase in the Retrosternal airspace.
- An obtuse costophrenic angle.
- Reduction in size and number of pulmonary vessels particularly in periphery of lung.
- Rapid tapering vascular shadows accompanied by hyperlucency of the lungs
- Heart shadow is vertical and narrow.
- **Computed tomography:** Has greater sensitivity and specificity than plain chest X-ray for emphysema but is rarely necessary except for diagnosis of bronchiectasis, evaluation of bullous lung disease.HRCT scan is highly specific for diagnosing emphysema, and the outlined bullae.

PULMONARY FUNCTION TESTS 26

Because of imprecision of clinical findings, objective evaluation of presence, severity and reversibility of airflow obstruction is essential

in the diagnostic evaluation of COPD.

Spirometry is the most robust test of airflow limitation in patients with COPD. Spirometer provides a graphic output, which enables differentiation between restrictive, obstructive, and proximal airway disease.

Forced expiratory volume in one second (FEV_1) is recommended as the measurement of choice in COPD because,

- FEV_1 is reproducible and objective measurement.
- It is simple and relatively quick to measure and can be measured at all stages of disease.
- The forced expiratory maneuver records not only FEV_1 but also FVC and FEV_1 / FVC ratio less than 70% is diagnostic of airway obstruction
- A decline in the FEV_1 has the most predictive value.[54]
- Serial measurement provides evidence of disease progression. Over time by assessing FEV1. FEV_1 declines with normal ageing at about 30 ml/year and this increase to an average of 45 ml/year in smokers[55].

Table -2 Patterns of Pulmonary Function Abnormality[29]

	Normal* (%)	**Obstructive**	**Restrictive**	**Combined**
VC	> 80	N to decreased	decreased	decreased
FEV_1	> 80	N to decreased	decreased	decreased
FEV_1/FVC%	> 75	decreased	N to increased	decreased
FEF25-75%	> 80	decreased	decreased	decreased
TLC	80-120	N to increased	decreased	Decreased, can be N or increased
RV/TLC%	25-40	increased	increased	increased

Table-3 CLASSIFICATION OF SEVERITY[31]

Stage	Characteristic
0 : At risk	• Normal spirometry • Chronic symptoms (cough, sputum production)
I. Mid COPD	• FEV_1 / FVC < 70% • $FEV_1 \geq$ 80% predicted • With or without chronic symptoms
II. Moderate COPD	• FEV_1 / FVC < 70% • 50% $\leq FEV_1 \geq$ 80% predicted • With or without chronic symptoms
III. Severe COPD	• FEV_1 / FVC < 70% • 30% $\leq FEV_1 \geq$ 50% predicted • With or without chronic symptoms
IV. Very Severe COPD	• FEV_1 / FVC < 70% • $FEV_1 \leq$ 30% predicted • $FEV_1 \geq$ 50% predicted plus respiratory failure.

Flow Volume Loops

Expiratory flow at 75% or 50% of vital capacity have been used as a measure of airflow limitation and provide complementary information to the usual volume time plot. There are problems with the reproducibility of these measurements and hence not preferred for routine clinical use.

Reversibility to bronchodilators

Reversibility tests are important because

1. To help distinguish those patients with marked reversibility (at least 12% or 200ml of FEV_1) who have underlying asthma.
2. To aid with future management.
3. The FEV_1, after bronchodilator is the best predictor of survival.

It is usually recommended that the response to bronchodilator be assessed either using repeated doses from metered dose inhaler or via the nebulised route.

Gas transfer for carbon monoxide

Gas transfer for carbon monoxide values is below normal in many patients with COPD and although there is a relationship between gas transfer and microscopic emphysema the severity of emphysema in an individual patient cannot be predicted from this.

Arterial blood gas analysis

Measurement of arterial blood gas is essential in patients with COPD to confirm the degree of hypoxemia and hypercapnea and in acute exacerbation to determine the hydrogen ion concentration. Hypercapnea commonly is observed as the FEV_1 falls below 1 L/s or 30% of the predicted value[56].

PULSE OXIMETRY

Pulse oximeters are invaluable, non-invasive tools for the assessment of hypoxemia in patients with COPD. However, due to the high proportion of smokers in the COPD population, it could be beneficial to check the amount of carbon monoxide with whole blood oximeters just before starting the sleep study.

Fussell KM, Ayo DS, et al assessed the need for long-term oxygen therapy by ambulatory oximetry monitoring[57].

Pulse oximetry can determine rapidly whether impairment is mild, moderate, or severe. Oximetry is accurate to within 3% to 5% at saturations greater than 70%. When oxygen saturation is less that 65% to

70%, however, the technique is less reliable. Arterial oxygen saturations of less than 92% may be associated with the development of a secondary polycythemia[58.]

Oximetry is useful in checking that the selected oxygen flow will ensure nighttime SpO2 of over 90%.

Patients with pulmonary hypertension or cor pulmonale with normal day time blood gases should be evaluated for nocturnal desaturation by overnight oximetry.

Table - 4 Practical aspects related to nocturnal pulse oximetry in patients with COPD

Benefits of pulse oximetry	Points to note
Non invasive and well tolerated	Check carbon monoxide (CO) levels to ensure SpO2 accuracy with smokers
Monitoring practically in real-Time	Accuracy of devices at low SpO2 values may be variable
Can assess the effect of oxygen therapy	Clip sensors may be dislodged during sleep, adhesive sensors may provide more secure connections
Results may suggest patients requiring polysomnography	

Other tests :

α_1 AT level are not routinely needed but should be obtained for chronic airflow obstruction in non smokers, as well as COPD patients with bronchiectasis, cirrhosis without apparent risks, premature or bibasilar emphysema in patients under 50 years with unremitting disease and in individuals with family history of AT deficiency.

MANAGEMENT OF COPD

The goal of management is to improve daily living and the quality of life by preventing symptoms and the recurrence of exacerbations by preserving optimal lung function.

Health Education: It can play a role in improving skills, ability to cope with illness.Most appropriate topics are,

- Smoking cessation
- Basic information about COPD
- Self management skills
- Strategies to help decrease dyspnea.

SMOKING CESSATION

Smoking cessation in patients with early COPD improves lung function initially and slows the annual decline of FEV_1[61]

BEHAVIORAL TECHNIQUES

A Wide Spectrum of Behavioral Techniques has been used to treat cigarette addiction. These include education, individual & group education, adversive conditioning, psychotherapy, transcental meditation, sensory deprivation, hypnosis and desensitization[62]

Pharmacologic intervention

Nicotine is the ingredient in cigarettes primarily responsible for the addiction. Withdrawal from nicotine may cause unpleasant adverse effects, including anxiety, irritability, difficulty concentrating, anger, fatigue, drowsiness, depression, and sleep disruption. These effects usually occur during the first several weeks.

Nicotine replacement therapies after smoking cessation reduce withdrawal symptom. Transdermal nicotine patches are available readily for replacement therapy. The use of the antidepressant bupropion is also effective for smoking cessation. The most recent drug to receive approval for smoking cessation is varenicline.

Other drugs that can be used include lobeline sulfate, amphetamines, mecamylamine, antidepressants, clonidine [63].

Pharmacologic therapy

This is used to prevent and control symptoms, reduce frequency of exacerbations and improve exercise tolerance.

- Bronchodilators are central to management of COPD. Inhaled therapy is preferred.
- Choice between β_2 agonist, anticholinergic theophylline or combination therapy depends on availability and individual response in terms of symptom relief and side effects.
- In addition to its anti-inflammatory effects, theophylline improves respiratory muscle function, stimulates the respiratory center, and promotes bronchodilation. Adding theophylline to the combination of bronchodilators can result in further benefit in stable COPD.
- Bronchodilators are prescribed or as needed, or on a regular basis to prevent or reduce symptoms depending on singe.
- Long acting bronchodilators are more effective and convenient.
- Combining bronchodilators improve efficacy and decrease the risk of side effects compared to increasing the dose of a single bronchodilator.

Glucocorticosteroids

Inhaled steroids are appropriate for symptomatic COPD patients with an FEV_1<50% predicted (Stage III & IV).Present guidelines recommend a trial of 6 weeks to 3 months with inhaled steroids to identify patients who may benefit from long term inhalation steroid therapy. Long term treatment with oral steroids is not recommended in COPD. However in in acute exacerbation of COPD, use of steroids improves symptoms and lung function.[64]

Phosphodiesterase IV inhibitors

Cilomilast and roflumilast are systemically available, second generation, selective phosphodiesterase 4 inhibitors. They cause a reduction of the inflammatory process in patients with COPD.

Antibiotics

The goal of antibiotic therapy in COPD is not to eliminate organisms but to treat acute exacerbations.

The first-line treatment choices include amoxicillin, cefaclor, or trimethoprim / sulfamethoxazole. Second-line antibiotic regimens are the more expensive antibiotics, including azithromycin, clarithromycin, and

fluoroquinolones.

Diet :

Inadequate nutritional status associated with low body weight in patients with COPD is associated with impaired pulmonary status, reduced diaphragmatic mass, lower exercise capacity, and higher mortality rates. Nutritional support is an important part of their comprehensive care.

SURGICAL CARE

Bullectomy: Giant bullae may compress adjacent lung tissue, thereby reducing the blood flow and ventilation to the healthy tissue. Removal of bullae may result in the expansion of compressed lungs and improved function

Patients who are symptomatic and have an FEV_1 of less than 50% of the predicted value have a better outcome after bullectomy.

Lung volume reduction surgery

Indications for LVRS include, symptoms secondary to severe emphysema, marked hyperinflation, and CT scan evidence of heterogeneous emphysema.

Lung transplantation

Lung transplantation is new therapy for advanced lung disease. Patients with COPD are the largest single category of patients who undergo the process.

Pulmonary Rehabilitation

Rehabilitation exercises improve exercise tolerance[65.] It is a multidisciplinary a team approach, provided by health care professionals who have experience in managing COPD (eg, physician, dietitian, nurse, respiratory therapist, exercise physiologist, physical therapist, occupational therapist, recreational therapist, cardio respiratory technician, pharmacist, psychosocial professionals).

This emphasizes patient and family education, smoking cessation, medical management (eg. oxygen, immunization), respiratory and chest physiotherapy, physical therapy with bronchopulmonary hygiene, exercise, vocational rehabilitation, and psychosocial support. As a result of rehabilitation, improvements occur in the objective measures of quality of life, well being, and health status, including a reduction in respiratory symptoms and an increase in exercise tolerance and functional activities [81].

Home Oxygen Therapy (HOT)

Apart from smoking cessation, supplemental oxygen therapy is the only measure that has been shown to reduce mortality in patients with COPD.[69]Long-term oxygen therapy in conjunction with pulmonary rehabilitation, increase life expectancy & improves quality of life [66, 67]

Supplemental oxygen should be given to patients who are hypoxemic with a Pao_2 of 55 mm Hg or less, or an oxygen saturation of 88 percent or less while sleeping.

Standard long-term oxygen therapy is indicated in patients with a Pao_2 below 55 mm Hg while at rest and awake, and in patients with accompanying polycythemia (haematocrit >55%), pulmonary hypertension, right-sided heart failure or hypercapnea ($Paco_2$ above 45mm Hg). To be effective, long-term oxygen therapy should be applied for at least 16 hours out of 24 hours, and the sleep period must be totally covered [84.] The usual rate of oxygen administration is 1.5 to 3 L/min.

Oxygen therapy also decreases in hospital admissions and length of hospital stays for acute exacerbations of COPD [68].

Nocturnal oxygen therapy

Patients with COPD may develop substantial decreases in nocturnal PaO_2 during all phases of sleep; more in REM phase Nocturnal oxygen therapy might be indicated in nearly all patients of COPD to combat against nocturnal hypoxia[69].

Two landmark trials, The British Medical Research Counsel (MRC study) and the National Heart, Lung, Blood Institutes Nocturnal Oxygen Therapy Trial (NOTT), showed that long-term oxygen therapy improves survival 2-fold or more in hypoxemic patients with COPD.

Benefits of oxygen therapy include amelioration of cor pulmonale, enhanced cardiac function, increased body weight, reversal of polycythemia, improved neuropsychiatric function and exercise performance, reduced pulmonary hypertension, improved skeletal-muscle metabolism[70].

Home oxygen therapy can be supplied in the form of compressed gas cylinders or oxygen concentrators. Oxygen concentrators are most and cost effective.

Other pharmacologic treatment

Vaccines: Influenza vaccines can reduce serious illness and death in elderly COPD patients by about 50%.

Alpha-1 Antitrypsin augmentation therapy: Young patients with severe α-1 AT deficiency and established emphysema may be candidates for the same.

Antitussives: Regular use is contraindicated in stable COPD.

PHLEBOTOMY

Repetitive hemodilution by Phlebotomy causes reduction of blood viscosity & there by improve pulmonary hemodynamics[71], increase oxygen uptake, improve pulmonary gas exchange, improves mental alertness, work performance, increase in arterial oxygen partial pressure (Pa_{O2})[72]. They also improve exercise tolerance[73, 74] and increases the cardiac output.

It also reduces iron levels in the body. Study by Martinez JA, Guerra CC et al showed reduction in iron level with phlebotomy[75.] Phlebotomy also used as an adjunctive therapy of acutely decompensated cor pulmonale in patients with marked polycythemia[76].

Study by WEdzicha JA, Rudd RM, Apps MC et al proved that Erythrapheresis significantly improved symptoms, mental function, and work performance in patients with polycythaemia secondary to hypoxic lung disease. The procedure was well tolerated by all patients and no complications occurred. The mean packed cell volume decreased from 0.64 to 0.48 in men and from 0.56 to 0.42 in women, with significant decreases in blood viscosity[77].

Decrease in oxygen transport capacity, hypovolemia, depletion of body iron store are the main complications of phlebotomy.

Intensive care admission:

Indications include confusion, lethargy, respiratory muscle fatigue, worsening hypoxemia, respiratory acidosis (i.e., pH <7.30), newly occurring arrhythmias, Significant co-morbidities, Onset of new physical signs (cyanosis, peripheral edema) ,Failure of exacerbation to respond to initial medical treatment, Severe background COPD or when a patient requires invasive or noninvasive mechanical ventilation. Assisted ventilation Patients may be treated with noninvasive mask ventilation or translaryngeal intubation and mechanical ventilation.

Hemodynamic instability, difficulty with clearing of secretions, and copious secretions are contraindications to noninvasive assisted ventilation.

The main goal of assisted positive pressure ventilation in acute respiratory failure complicating COPD is to rest the ventilator muscles and restore gas exchange. Major risks are ventilator-associated pneumonia, barotrauma, and laryngotracheal complications associated with intubation.

Table – 5[84]

Stages:	Monitor	Recommendations
Stage 0 **At Risk**	Chronic cough and sputum production Lung function is normal	Avoidance of risk factors Annual Influenza Vaccine
Stage 1 **Mild COPD**	FEV1 > 80% FEV1/FVC <70%	Short Acting Bronchodilator when needed
Stage 2 **Moderate COPD**	FEV1/FVC <70% 50%<FEV1 <80% predicted	Short acting Bronchodilators as needed add one or more long acting bronchodilators, Pulmonary Rehabilitation
Stage 3 **Severe COPD**	FEV1/FVC < 70% 30%<FEV1<50% predicted	Short and long acting bronchodilators, Pulmonary Rehabilitation, Inhaled Glucocorticosteroids ,if repeated exacerbations
Stage 4 **Very Severe COPD**	FEV1/FVC < 70% FEV1<30% predicted or FEV1<50% predicted plus respiratory failure,signs of right heart failure	Add long term care oxygen therapy
Patient Education/ Prevention of Complications		Smoking cessation Yearly Influenza & Pneumococcal Vaccine: Increase bronchodilator therapy r antibiotic therapy, Consider corticosteroids if no improvement in symptoms Administer O2 as needed

Prognosis:

The predictors of mortality are aging, continued smoking, accelerated decline in FEV_1,[80] moderate-to-severe airflow obstruction, poor bronchodilator response[82], severe hypoxemia, the presence of hypercapnea, development of cor pulmonale, and Overall poor functional capacity.[78, 83]

The American Thoracic Society (ATS) has recommended the clinical staging of COPD severity according to lung function. Stage I is FEV_1 of equal or more than 50% of the predicted value. Stage II is FEV_1 35-49% of the predicted value, and stage III is FEV_1 less than 35% of the predicted value.

Table-6 Factors Influencing Survival in Patients with COPD[79]

Risk factor	Effect on survival
Post bronchodilator FEV_1	Decreases mortality with increased FEV_1, decreases mortality with reversible component of obstruction
Rate of FEV_1 decline	Decreases mortality with slower decline; FEV_1 <1 L generally considered severe disease
History of atopy	Decreases mortality
Higher diffusion capacity	Decreases mortality
Pao_2 level	Decreases mortality with increased level; Pao_2 <55 mm Hg increases mortality
Age	Increases mortality in older patients
Cigarette smoking	Increases mortality with continued use and greater consumption
Hypercapnea ($Paco_2$ >45 mm Hg)	Increases mortality
Right-sided heart failure	Increases mortality
Malnutrition	Increases mortality
Resting tachycardia	Increases mortality

Materials and Methods

MATERIALS AND METHODS

In the present study 50 cases were selected on the basis of simple random sampling method from the medical wards of CMC Hospital,

Coimbatore.

Study Period: January 2006 to June 2007.

Inclusion Criteria: Adult males and females admitted in the medical wards with symptoms suggestive of airway obstruction of more than 2 years duration and in whom clinical diagnosis of chronic obstructive pulmonary disease was made were included in the study.

All these patients were subjected to clinical examination, chest X-ray, pulmonary function testing, pulse oximetry and Haematocrit analysis

On spirometry the presence of COPD was diagnosed by post bronchodilator values of

(I) Forced expiratory volume in first second / Forced vital capacity = FEV1/FVC less than 70%.

(ii) Forced expiratory volume in first Second ($FEV1_{)}$ less than 80%

All patients were clinically stable at the time of conducting pulmonary function test.

Exclusion criteria: Cases which where excluded form the study were patients with primary diagnosis of bronchial asthma, pulmonary

tuberculosis, Bronchiectasis, cases of sleep apnea syndromes and patients with post infarction failure.

Procedure: A proforma was prepared after applying the above inclusion and exclusion criteria, meeting the objectives of study

For the present study 50 patients were selected, 42 males and 8 females and they were subjected to the following examinations.

1. History and Physical Examination

In every case a detailed history was elicited and thorough clinical examination was done as indicated in the proforma.

2. Radiographic examination

Chest X-ray postero-anterior view and left lateral view were obtained to detect signs chronic bronchitis and emphysema

3. Pulmonary Function Testing (PFT)

PFT was done on computerized spirometer. Spirometery was performed when the patients were clinically stable.

Test was performed with the patient comfortably seated, with

clothes loosened. The patient was instructed to take a deep inspiration then close the lips around the mouth piece and blows out as hard and fast as possible, followed by deep inspiration.

Volume was obtained on the vertical axis of recording paper and time on the horizontal axis. The curve which was obtained is referred to as forced vital capacity curve.

Forced Vital Capacity (FVC) is the volume of air that can be forcibly exhaled (as fast as possible) after a maximal inspiration. It is expressed in litres.

Forced expiratory volume in one second (FEV_1)

It is defined as the volume of air expelled in the first second, from the start of maximum expiratory effort of the forced vital capacity. It is expressed in litres or percentage of predicted value.

Report Date/Time
04/11/2005 16:42:22

Pulmonary Function Report

Subject Data

Name Selvam
IP. No. 51210
Birth Date 26/02/1953 **Sex** Male
Ethnic Gr. South Indian

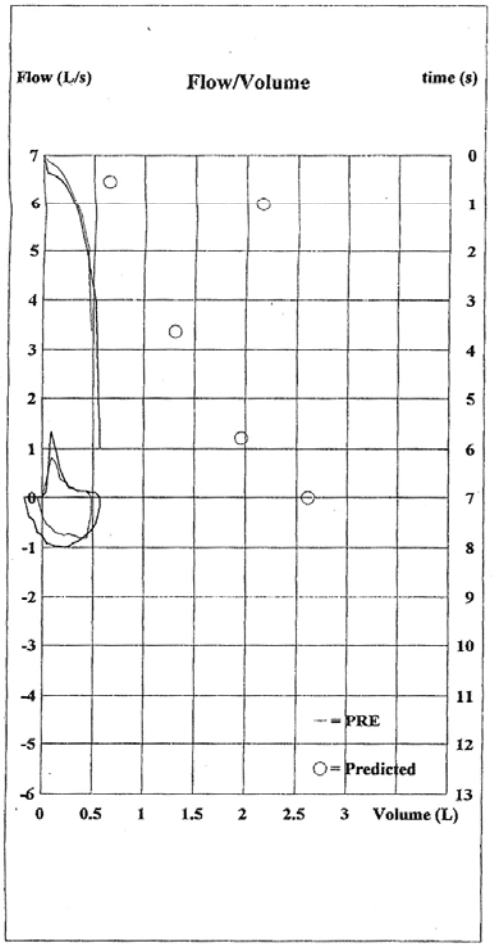
Reference Data

Date	04/11/2005	16:23:21
Age	52	Years
Height	158	cm
Weight	48.5	kg

Smoking History

Smoker - Yes
Pack Years - 25

Symptom



Test Data

Drug Salbutamol Puffs 200 mcg
Administred at 04/11/2005 16:23:04 **Tot. Qty** 200
Predicted Source Knudson

Par. (BTPS)	Pred.	Best	PRE	%Pred.	POST	%Pred.	%PRE
FVC	2.61	0.67	0.56	21.4	0.65	24.9	+16.1
FEV1	2.15	0.32	0.30	13.9	0.33	15.3	+10.0
FEV1%	81.2	53.6	53.6	66.0	50.8	62.5	-5.2
PEF	7.04	1.79	0.81	11.5	1.39	19.7	+71.6
FEF25	6.47	0.38	0.38	5.9	0.55	8.5	+44.7
FEF50	3.36	0.18	0.18	5.4	0.14	4.2	-22.2
FEF75	1.22	0.11	0.11	9.0	0.10	8.2	-9.1
FEF25-75	2.74	0.17	0.17	6.2	0.15	5.5	-11.8
FET		4.30	3.62		6.00		+65.7
FIVC	2.61	0.65	0.60	23.0	0.77	29.5	+28.3
FIV1	2.15	0.64	0.58	26.9	0.77	35.8	+32.8
PIF	7.04	0.90	0.82	11.6	0.99	14.1	+20.7
ELA	52						
FEV1/VC%	81.23		48.39	59.6	55.00	67.7	+13.7
VC	2.61	0.62	0.62	23.7	0.60	23.0	-3.2
MVV	108.0	11.9	11.9	11.0	31.4	29.1	+163.9

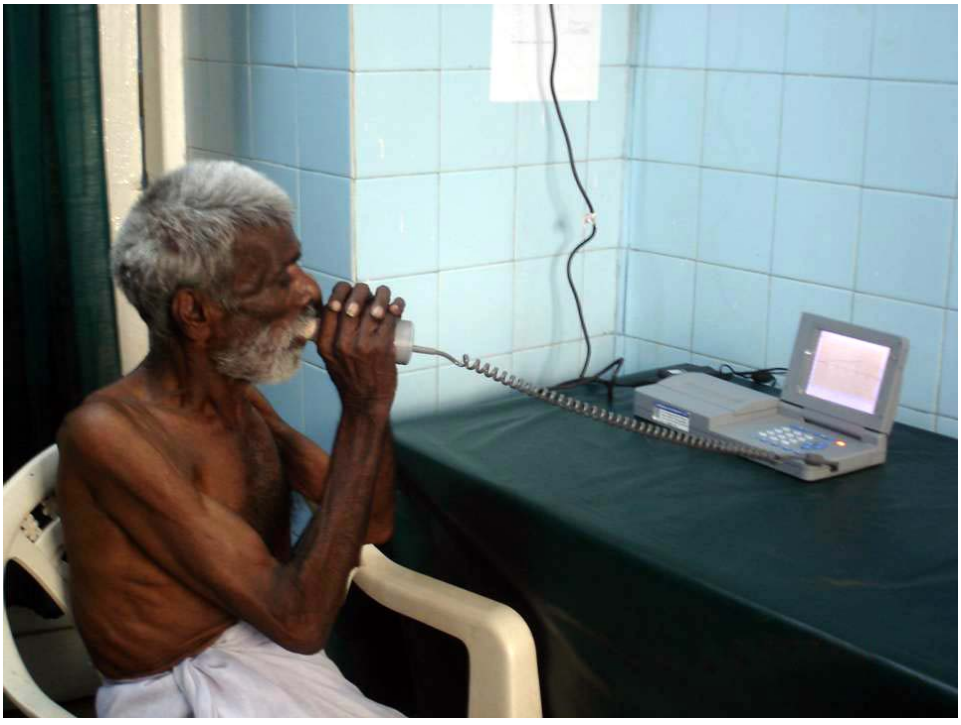
Imp : Very severe obstruction.
Air Trapping +
Gross Small airways dysfunction.
No significant reversibility

Comment

SPACER

SPIROMETER

PATIENT UNDERGOING SPIROMETRY TESTING



PULSE OXIMETER

Forced expiratory volume in one second as a percentage of forced vital capacity (FEV_1/FVC)

It is the percentage of forced vital capacity which is expelled in the first one second of maximal expiratory effort.

Pulse oximetry :

Oxygen saturation was assessed by using pulse oximeters. (Future PO, Silicon Lab)

Haematocrit :

Haematocrit was assessed by using automated analyzer

OBSERVATIONS AND RESULTS

OBSERVATIONS AND RESULTS

The data was analysed by using the arithmetic mean, the standard deviation, standard error, and the correlation coefficient.

50 cases of COPD were studied; the results are tabulated as follows:

Table - 7: Age Distribution

Age in years	Male	Percentag	Female	Percentag	Total	Percentage
41-50	3	7.1	1	12.5	4	8
51-60	8	19.0	2	25.0	10	20
61-70	18	42.9	3	37.5	21	42
71-80	12	28.6	1	12.5	13	26
81-90	1	2.4	1	12.5	2	4
Total	42	100	8	100	50	100

From the above table it can be observed that the maximum case among males were between 61-70 years of age constituting 42.9% and the Minimum number cases were in the age group of 81-90 years being 2.4%.

Among females, maximum number of cases were in the age group of 61-70 years constituting 37.5% and the minimum were in the age group of 41-50, 71-80 and 81-90 made up of 12.5% each respectively.

Both sexes put together the maximum cases were in age group of 61-70 years constituting 42% and minimum in the age group of 81-90

years constituting only 4%.

CHART SHOWING AGE DISTRIBUTION IN THE STUDIED POPULATION

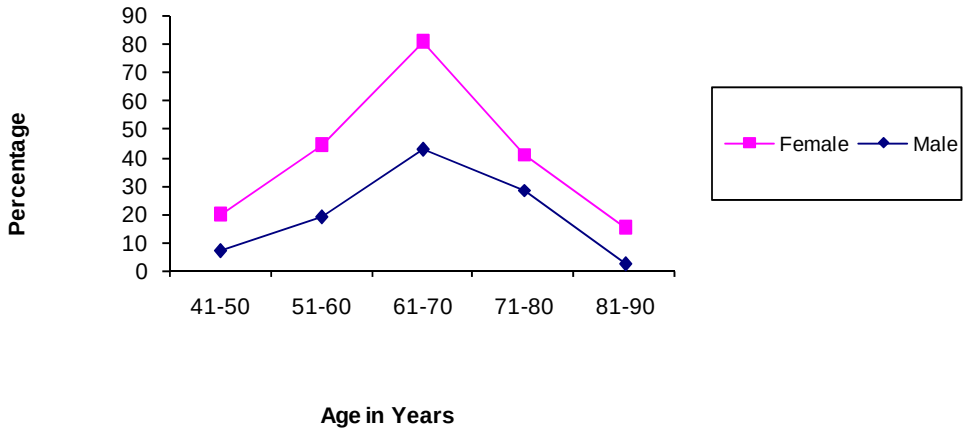


Table - 8: Mean Age and Standard Deviation of the cases

	Mean age in years	Standard deviation
Male	65	9.4
Female	63.75	10.5

P>0.05(NS)

Mean age of the male patient was 65 + 9.4 years and that of the female in the present study was 63.75 + 10.5 years with P > 0.05 statistically not significant.

Table-9: Sex Distribution

Sex	Number of Cases	Percentage
Male	42	84

Female	8	16
Total	50	100

Majority of the patients in the present study were males. The male female ratio was 5.25:1.

PIE DIAGRAM SHOWING SEX DISTRIBUTION OF THE CASES

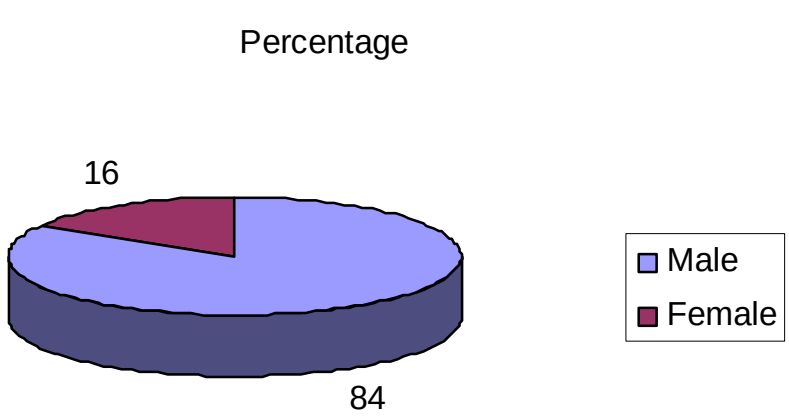


Table-10: Occupations of the patients

Occupation	Number of patients	Percentage
Farmers	37	74
Businessmen	5	10
Housewives	8	16
Total	50	100

In the present study group majority of the patients were farmers accounting to 74%. The remaining were businessmen 10% and

housewives 16% respectively.

Table-11: Urban and Rural distributions

	Number of patients	Percentage
Rural	45	90
Urban	5	10
Total	50	100

It is observed from the above table that 90% of the patients in the present study were from rural background, only 10% of the patients were from the urban locales.

PIE DIAGRAM SHOWING DISTRIBUTON OF CASES IN RURAL AND URBAN POPULATION

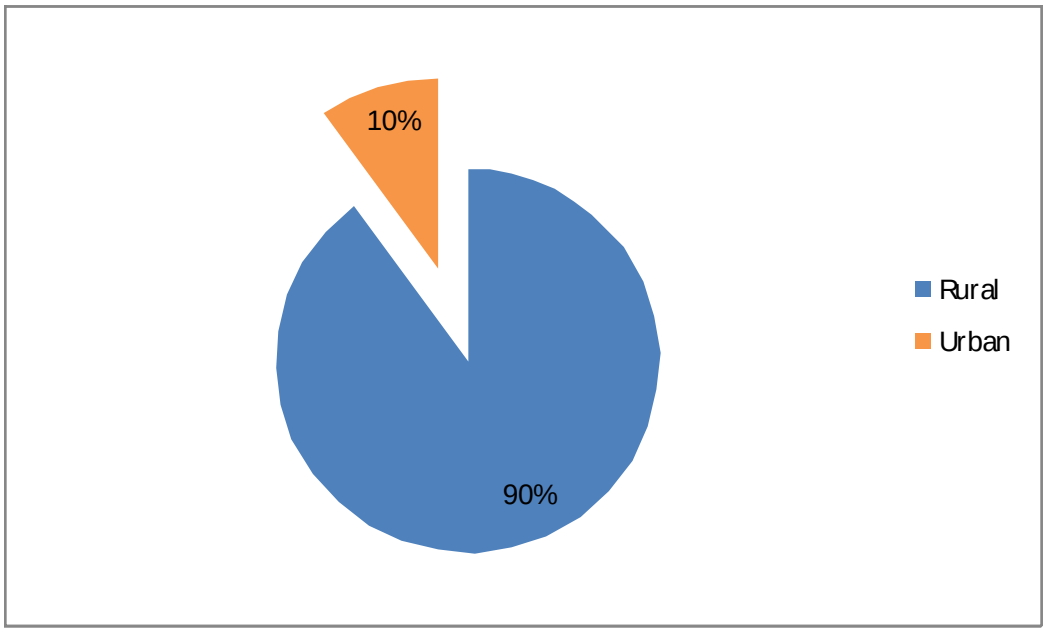


Table-12: Duration of illness

Duration of illness in years	Males	Females	Total	Percentage
2-5	8	0	8	16
6-10	19	2	21	42
11-15	13	6	19	38
16-20	2	0	2	4
Total	42	8	50	100

Table-13: Mean Duration of illness and Standard Deviation according to sex

	Mean (in years)	Standard Deviation
Male	9	4
Female	11.8	2.2

Majority of people in the present study group belonged to more than 5 years duration of illness. The mean duration of illness in males was 9 ± 4 years and in females was 11.8 ± 2.2 years respectively with $P<0.05$ which was statistically significant.

Table-14: Risk factor exposure

	Males	Females	Total	Percentage
History of smoking	42	0	42	84
Exposure to smoke burnt fuels	0	8	8	16
Total	42	8		100

All the male patients were smokers; in female H/o exposure to smoke of burnt fuels was present in all case. $P<0.05$ which is statistically significant.

Table-15: Duration of smoking habits

Pack years	No. of patients	Percentage
20-30	3	7.14
30-40	18	42.85
40-50	16	38.09
50-60	5	11.90
Total	42	100.00

Most of the patients had more than 20 years and majority were in 30-50 pack year exposure duration.

Table 16. Prevalence of COPD and its smoking association in various population studies from India.

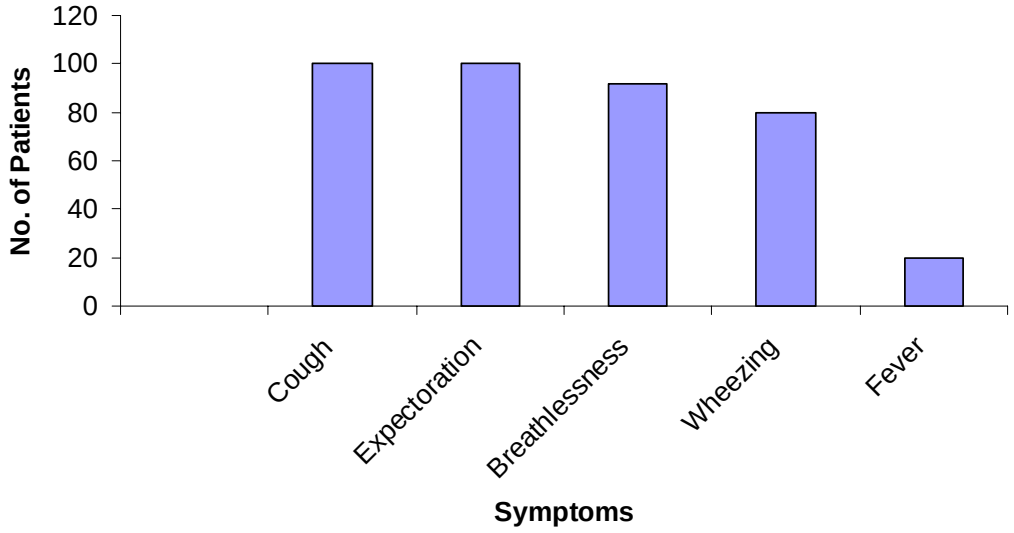
	Population	COPD prevalence (%)			Smoker: Non Smoker Ratio
		Men	Women	M:F Ration	
Wig (1964)	Rural Delhi	3.36	2.54	1.3	2.0
Sikand (1966)	Delhi	7.0	4.3	1.6	2.5
Viswanathan (1966)	Patna	2.12	1.33	1.6	
Bhattacharya (1975)	Rural U.P	6.67	4.48	1.6	
Radha (1977)	New Delhi	8.1	4.6	1.8	1.8
Thiruvengadam (1977)	Madras	1.9	1.2	1.6	10.2
Viswanathan (1977)	Delhi Rural	4.7	3.5	1.3	9.6
	Urban	8.0	4.3	1.9	4.0
Charan (1977)	Rural Punjab	2.28	1.63	1.4	
Malik (1986)	N.India Rural	9.4	4.9	1.9	5.5
	Urban	3.7	1.6	2.3	7.0
Jindal (1993)	N.India Rural	6.2	3.9	1.6	
	Urban	4.2	1.6	2.6	9.6
Ray (1995)	South India	4.08	2.55	1.6	1.6

Table-17: Presenting Symptoms

Symptoms	No. of patients n=50	Percentage
Cough	50	100
Expectoration	50	100
Breathlessness	46	92
Wheezing	40	80
Fever	10	20

All the patients presented with cough and expectoration. Breathlessness and wheezing was present in majority of the patients and fever was present among a small percentage (20%) of patients.

BAR DIAGRAM SHOWING PRESENTING SYMPTOMS IN PERCENTAGES



Vital Statistics:

Pulse Rate : 18 patients had tachycardia, 27 had pulse rate between 80-100/min and 5 had pulse rate below 80/min.

Respiratory : 22 patients had tachypnoea

Table-18: Physical Signs

Signs	Number of patients n=50	Percentage
Cyanosis	7	14
Flapping tremor	2	4
Pursed lip Breathing	6	12
Intercostals in drawing	6	12

Majority of patients were in the stable state with 10-14% of patients showing signs of distress. Two patients showed features respiratory failure

Table-19: Respiratory signs

Respiratory Sign	Number n=50	Percentage
Decreased chest movements	33	66
Decreased breath sounds	36	72
Crepitations / Rhonchus	44	88
Muffled heart sounds	36	72
Push down liver	36	72
Barrel shaped chest	16	32

BAR DIAGRAM SHOWING RESPIRATORY SIGNS IN PERCENTAGES

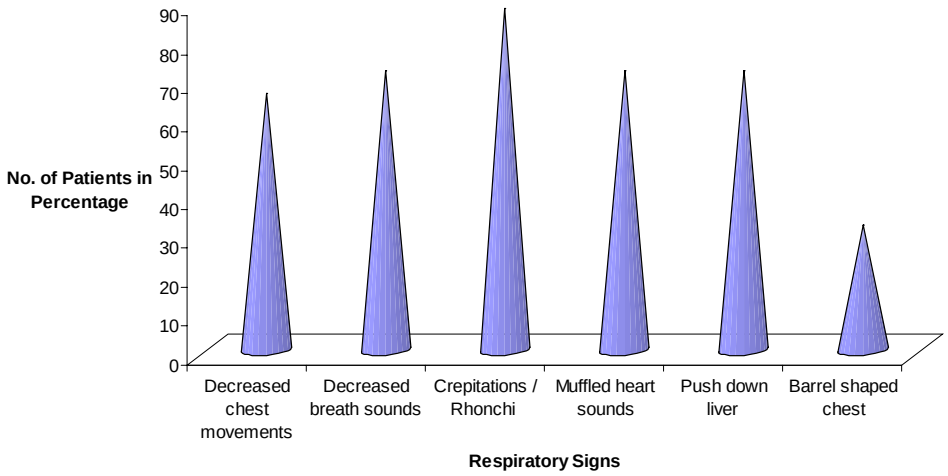
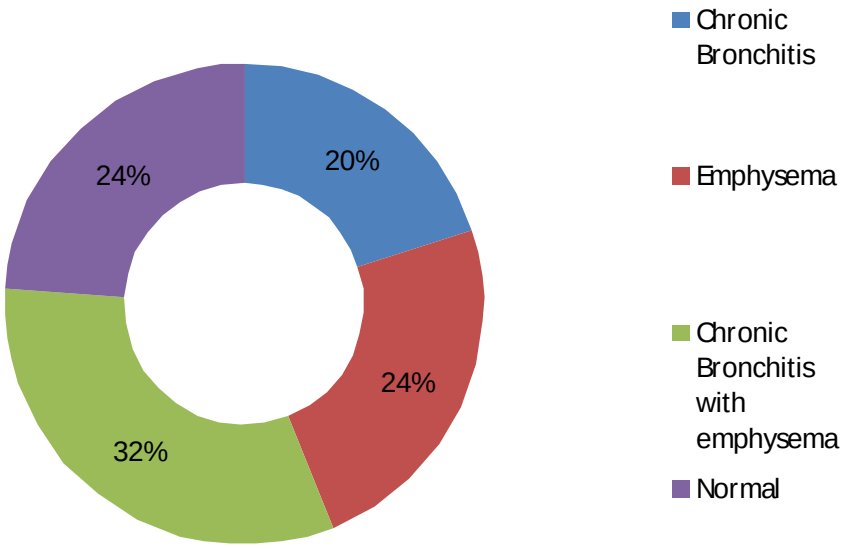


Table-20: Chest X-ray Findings

X-ray Findings	Number of Patients	Percentage
Chronic Bronchitis	10	20
Emphysema	12	24
Chronic Bronchitis with emphysema	16	32
Normal	12	24

In the present study 76% patients had abnormal chest X-ray. Out of which 32% had chronic Bronchitis with emphysema , 22% had chronic bronchitis and 16%had emphysema

DIAGRAM SHOWING CHEST X – RAY FINDINGS



Spirometric values

Though there are many spirometric values FEV_1 and FEV_1, / FVC are often considered as indices of pulmonary function in chronic obstructive pulmonary disease.

1. FEV_1 - reflects the degree of airway obstruction. The mean expected FEV1, among the subjects studied was 2.44 + 0.5 Lt, however the actual mean FEV_1 was 1.11 +0.38 liters. The mean FEV_1 % of expected value in this study was 44.92 + 14.01%.
2. FVC - Reflect the change in vital capacity. The mean expected FVC was 3.03 + 0.42 lt. The actual mean FVC was 2.01 + 0.60 lt.
3. The mean FEV_1 / FVC - was 54.26 $\pm$ 10.12% in the present study.

Table-21: Range and mean value of PFT findings

Test	Range	Mean	Standard deviation
FVC (It)	1.03-3.76	2.01	0.60
FEV_1 (It)	0.60-1.98	1.11	0.38
FEV_1 %	26-78	44.92	14.01
FEV_1 / FVC%	34-73	54.26	10.12

Maximum number of patients in present study were in stage III with 40% of the patients showing severe airflow obstruction with a mean FEV_1 of 45.34 $\pm$ 4.6%, 24% of patients had very severe obstruction with a mean FEV_1 of 28.4 $\pm$ 1.5% and 36% of patients had moderate obstruction of FEV_1 66.9 $\pm$ 6.9%. None of the patients study had mild obstruction.

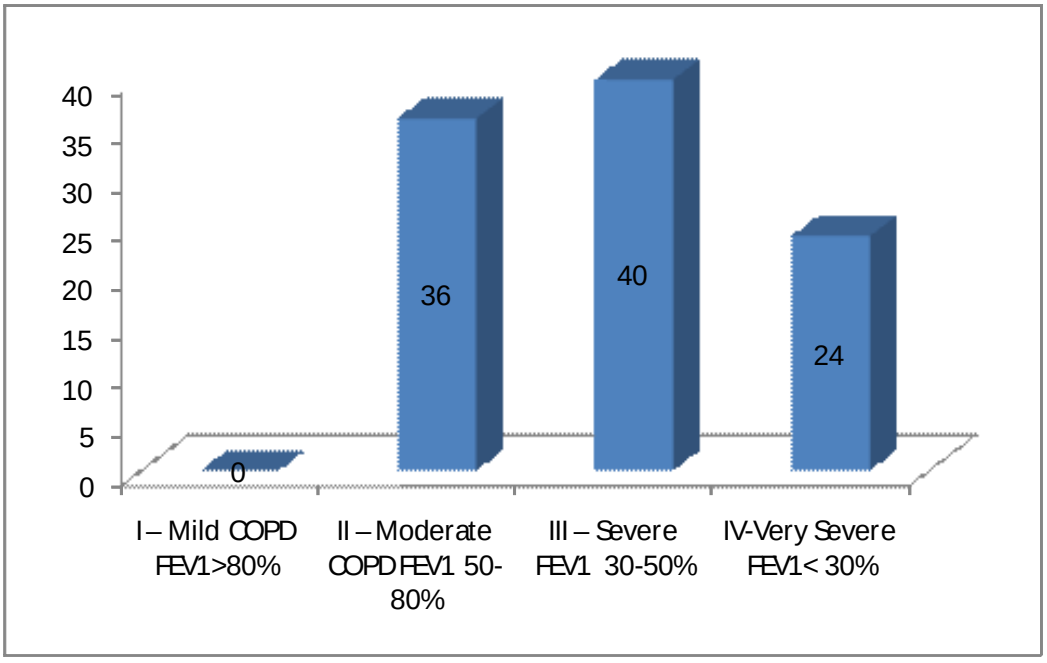
Table-22: Table showing patients in

Different stages as per GOLD staging criteria

Stage	No.	Percentage	Mean FEV in % with SD
I – Mild COPD FEV1>80%	-	-	-
II – Moderate COPD FEV1 50-80%	18	36	66.9 ± 6.9
III – Severe FEV_1 30-50%	20	40	45.34±4.6
IV-Very Severe FEV_1< 30%	12	24	28.4±1.5

BAR DIAGRAM SHOWING DISTRIBUTION OF PATIENTS IN

DIFFERENT STAGES OF COPD



Pulse oximetry assessment:

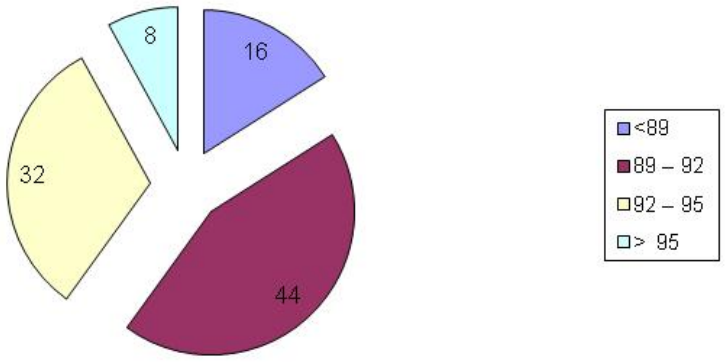
Serum arterial oxygen saturation values are assessed by pulse oximeter in all the patients.

Table -23 Distribution of pulse oximetry values in studied population

SaO2 %	No. of patients	Percentage %
<89	8	16
89 – 92	22	44
92 – 95	16	32
> 95	4	8

In this study patients with H/O chronic smoking (pack years >45) are commonly presented with oxygen saturation of <89%.

PIE DIAGRAM SHOWING DISTRIBUTION OF PULSE OXIMETRY VALUES IN STUDIED POPULATION



Haemoglobin & Haematocrit values

All the patients blood samples were assessed by automated analyzer .the values were correlated with severity and duration of the disease.

Table - 24 Showing Distribution of Haemoglobin Values Among Studied Population

Hb (g/dl)	Male	%	Female	%	Total	%
<10	5	11.9	4	50	9	18
10 -12	6	14.2	2	25.0	8	16
12 – 15	21	50	2	25.0	23	46
15 -17	7	16.6	-	-	7	14
>17	3	7.1	-	-	3	6
Total	42	100	8	100	50	100

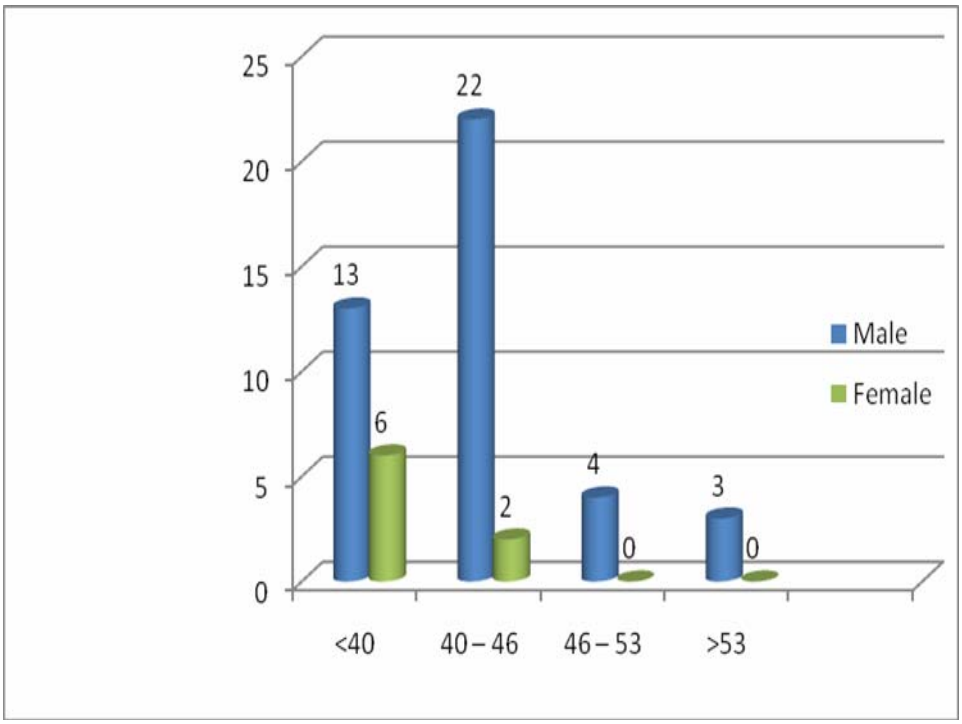
Table - 25 Distribution of Haematocrit Values Among Studied Population

Haematocrit (%)	Male	%	Female	%	Total	%
<40	13	31	6	75	19	38
40 – 46	22	52.4	2	25.0	24	48
46 – 53	4	9.5	-	-	4	8
>53	3	7.1	-	-	3	6
Total	42	100	8	100	50	100

Among the studied population about 3 patients had the values in polycythemic range.

BAR DIAGRAM SHOWING DISTRIBUTION OF

HAEMATOCRIT VALUES AMONG STUDIED POPULATION



Discussion

DISCUSSION

50 cases of chronic obstructive pulmonary disease were studied.

Mean age in present study was 64.37 $\pm$ 9.7 years

Table-26: Showing means age

Study	Mean age	Standard deviation
Trivedi H.S. et al.,	59.5	4.92
Higham M.A. et al.,	66.7	8.1
Migueres M. et al.,	60.0	10.81
Present Study	64.37	9.7

Table-27 Showing sex distribution

Study	Male %	Female %	Standard deviation
Trivedi H.S. et al.,	80	20	2.0
Migueres et al.,	90	10	6.7
Present Study	84	16	1.6

In the present study males accounted for 84% with a male female ratio of 5.25: 1.

In the studies quoted the maximum incidence of COPD was in males.

In the present study majority of people belonged to more than 5

years duration of illness. The mean duration of illness in males was 9 ± 4 years and females were 11.8 ± 2.2 years.

Table-28: Smoking habits in pack years

Study	Mean (Pack years)	Standard deviation
Migueres M. et al.,	49.0	22
Present Study	39.75	18

In the present study lot of the patients had more than 20 pack years and majority of patients were in 30-50 pack year exposure duration. The mean pack year was 39.75 + 18 years, reinforcing the fact that at least 20 pack year's exposure is necessary for development of COPD.

Presenting symptoms

In the present study, cough with expectoration was present in all the cases and breathlessness was present in 92% of cases, wheezing was present in 80%and fever was present in 20% cases

Physical signs

In the present study cyanosis was present in 7 cases; pursed lip breathing and intercostals in drawing was present in 6 cases each, flapping tremor was present in 2 cases.

Incidence of respiratory signs

In the present study majority of patients had chronic bronchitis with emphysema and 32% of patients had barrel shaped chest.

Chest X-ray

In the present study x-ray showed features of emphysema in 12 cases, features of chronic bronchitis in 10 cases & both emphysematous, bronchitis changes in 16 cases

SPIROMETRY

Table-29 Spirometric study, Distribution of cases

Stage	Hingham M.A. et al., (Percentage)	Present study (Percentage)
I	-	-
II	16.42	36
III	26.00	40
IV	57.58	24
Total	100	100

In the study conducted by Higham M.A. et al majority of patients were in stage IV and constituted about 57.58%, whereas in present study majority of patients were in group III.

PULSE OXIMETRY

In this study majority of patients had oxygen saturation values in the range of 89 -95 %.About 8 patients had hypoxia (<89% saturation).after oxygen administration , treatment with bronchodilators ,and intensive care management they recovered and maintained normal oxygen saturation levels .

POLYCYTHEMIA

In the study, polycythemia was a feature in 6% of cases. In the study by Claudia Cote, MD, Marya Zilberberg, MD et al polycythemia was present in 5% of patients. In another study by C. Cote, S. H. Mody et al, polycythemia was present in 6% of cases.

Table – 30 Prevalence of polycythemia

Study	Percentage of polycythemia
Claudia Cote, MD, Maria Zilberberg, MD et al	5
S. H. Mody, C. Cote et al	6
Present study	6

About 3 patients had polycythemic range of haematocrit values. In these patients phlebotomy was done. Reduction in the haematocrit was observed .Symptomatic improvement was also observed in these patients.

summary

SUMMARY

50 patients of chronic obstructive pulmonary disease were studied.

1. Majority of the patients were in the age group of 50-70 years. COPD was seen predominantly in male patients and majorities were smokers.
2. In the majority of patients the duration of illness was 6-10 years, cough with expectoration was present in all patients.
3. Diminished chest movement, decreased breath sounds, crepitations, rhonchi, muffled heart sounds, and pushed down liver were present in majority of patients.
4. As the number of cigarettes/day and duration increases the severity of the disease also increases in the studied population.
5. In the study ,about 40 % of cases were in stage III disease
6. Computerised spirometry was found to be most sensitive investigation in diagnosing and assessing the severity of the disease in all these cases.
7. As the severity & duration of the disease increases they are more prone to develop hypoxia and polycythemia as a complication. In our study 8 patients had hypoxia, as assessed by pulse oximeter

Conclusion

CONCLUSION

1. Computerized spirometry is a very useful investigation in the management of chronic obstructive pulmonary disease.

Forced expiratory volume in first second (FEV_1) values can be used to diagnose as well as to assess the severity of the disease.

2. Pulse oximetry is a useful tool in diagnosing periods of oxygen desaturation.
3. Pulse oximetry also useful in monitoring the oxygen therapy during management.
4. Haematocrit analysis is a useful adjunct in assessing the severity of the disease.
5. Polycythemia, even though uncommon in chronic obstructive pulmonary disease patients is one of the rare but preventable complication with early cessation of smoking & with oxygen therapy.

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proforma

PROFORMA

‘An analysis of pulmonary function tests, Pulse oximetry and Haematocrit abnormalities in chronic obstructive pulmonary disease patients’

I. Name : 2. Age : 3. Sex : M/F

4. IP NO. : 5. DOA : 6. DOD:

7. Occupation : 8. Economic Status:

9. Address : Poor / Middle / High

10. Presenting complaints

    a. Cough with or without expectoration

    b. Exertional breathlessness

    c. Wheezing

    d. Chest pain

    e. Hemoptysis

    f. Fever

    g. Any other symptoms

II. History of present illness

    a. Cough

    Duration

    Dry / Productive

    If productive i) colour

    ii) odor

iii) Quantity

iv) Type – Mucoid / mucopurulent / purulent

Consistent or continuous

Diurnal / seasonal / postural variation

Any triggering factors - smoke, dust, pollen

Status - Increasing / persistent / decreasing

b. Breathlessness

Duration

Persistent / paroxysmal

Relation to exertion - present / absent

Seasonal variation

Precipitating factors

Duration of attack

History of PND, Orthopnoea - present / absent

c. Wheezing

Duration

Precipitating factors

Duration of attacks

Relieving factors

d. Chest pain

Duration

Onset - acute / insidious

Location

Nature of pain pleuritic / anginal / other types

Radiation neck / shoulder / arm / abdomen

Aggravating factors Exertion / cough / any other

Relieving factors

Duration of each attack

e. Hemoptysis

Duration

Quantity

Type - frank Hemoptysis / blood stained sputum / blood streaked Sputum / rusty sputum Fever Duration

f. Fever

Duration

Degree - Mild/Moderate/High

Type - Continuous/remittent/intermittent/ evening rise of temperature

Associated - Chills - rigor, seating

g. Any other symptoms - Details

12. Past history - Similar complaints / tuberculosis / asthma / exanthematous fevers in childhood / allergy / epilepsy / cardiac

illness / diabetes mellitus.

12. Personal history

Diet	:	Veg / Non-veg/mixed
Sleep	:	Sound/disturbed
Snoring	:	Present / absent
Early morning headache	:	Present / absent
Early morning lethargy	:	Present / absent

Habits

a) Tobacco chewing

b) Tobacco smoking

i) Beedi / Cigarettes-Pack years

Duration

Quantity / day

Current or ex-smoker

c) Snuff inhaler

d) Bowels - regular / constipated / loose stools

e) Bladder-Frequency day / night

f) Appetite :

g) Alcohol - Quantity, duration, type

h) Occupational exposure - Dust / fumes / smoke / chemicals

14. Family history

Tuberculosis	-	Present / absent
Bronchial asthma	-	Present / absent
Allergy like urticaria	-	Present / absent
Rhinitis / eczema	-	Present / absent

15. General physical Examination

a. Comfortable / dysphonic

b. Built: Well built / moderate built / poorly built

c. Nourishment: Well / Moderate / poorly nourished

Height	:	Weight :
Pallor	:	Present / absent
Cyanosis	:	Central / Peripheral / mixed
Icterus	:	Present / absent
Clubbing	:	Present / absent
Halitosis	:	Present / absent
Oedema	:	Present / absent
Lymphacienopathy	:	Present / absent
Koilonychias / platynychia	:	Present / absent
Any other significant signs	:	

16. Vital parameters

Pulse: Rate Rhythm Volume Character

SP

Respiration Rate Rhythm type

Accessory muscle of respiration Acting / normal

Pursed lip breathing Present / absent

16. Examination of nose, oral cavity, pharynx, teeth and gums

17. Systemic system

Respiratory system

Inspection

Shape of the chest : Normal / barrel shaped / pigeon chest / pectus Excavatum / kyphoscoliosis / Harrison's sulcus /any other deformity

Position of the trachea : Central / right / left

Position of the apical impulse :

Any other pulsation

Visible veins over the chest : Present/absent

Chest movement Normal / diminished / symmetrical / asymmetrical

Stridor / Wheeze

Palpation: Position of trachea - Central right / left

Position of apical impulse -

Movement of chest wall - Normal / decreased

Symmetrical /Asymmetrical

Palpable crepitations / Rhonchi

Vocal fremitus: Normal / increase / decrease

Areas:

Chest wall tenderness

Chest measurements

AP diameter	Transverse diameter
Chest circumference	Chest expansion
Percussion note	Normal / impaired / woody dull / stony dull / hyper resonant

Areas:

Liver dullness	Normal / obliterated
Cardiac dullness	Normal / diminished

Auscultation

Breath sounds

Intensity	Normal / diminished / increased / absent

Areas:

Quality Vesicular / bronchial / bronchovesicular

Areas:

Rhonchi Absent / present

High pitched / low pitched

Areas:

Crepitations: Present / absent

Fine / coarse / leathery

Inspiratory / expiratory / post tussive

Areas:

Pleural rub Present / absent

Areas:

Vocal resonance Normal / increased / decreased / absent

Areas : Aegophony / whispering pectiroloquy

Post tussive suction

18. Cardiovascular system

19. Abdominal examination

20. Nervous system

21. Investigations

a) Pulmonary function tests

b) Complete Haemogram

Hemoglobin

Red cell count

Differential count P L E M B

ESR

Haematocrit

c) Biochemistry

Random blood sugar

Urea

Creatinine

d) Urine - Protein / sugar / microscopy

e) Sputum - Microscopy / grams stain

AFB

Abnormal cells including malignant

Cells Culture / sensitivity

f) Chest radiograph - Report

g) Electrocardiogram

h) Pulse oximetry

i) Any other investigations

22. Final diagnosis

Treatment:

Comments

Master Chart

KEY TO MASTER CHART

M	-	Male
F	-	Female
A	-	Agriculture
B	-	Businessman
HW	-	House wife
+	-	Present
-	-	Absent
↓	-	Decreased
N	-	Normal
B	-	Bronchitis
E	-	Emphysematous
B+E	-	Bronchitis with Emphysema
FEV1	-	Forced expiratory volume in first second
FVC	-	Forced vital capacity

S.No.	I.P. No.	Age (in yrs)	Sex	Occupation	Duration of illness	History						Physical Examination							Respiratory System				
						Cough	Expectoration	Dyspnoea	Wheezing	Fever	H/o smoking (pack years)	Pallor	Cyanosis	Clubbing	Pulse rate / min	Temperature in ^{o}C	Respiratory rate / min	Blood Pressure mmHg	Barrel shaped chest	Chest movement	Breath sounds	Rhonchi	Crepitations
1.	26893	65	M	A	12	+	+	+	+	+	36	-	-	-	86	37.6	26	132/70	+	↓	↓	+	+
2.	26746	72	M	A	14	+	+	+	+	+	45	-	-	-	104	38.0	28	130/80	-	↓	↓	+	+
3.	26946	67	M	A	8	+	+	+	+	-	33	-	-	-	94	37.2	24	136/84	+	↓	↓	+	+
4.	27126	54	M	A	5	+	+	-	-	-	20	-	-	-	76	37.6	21	140/82	-	N	N	+	+
5.	27456	62	M	A	6	+	+	+	+	-	35	-	-	-	96	37.4	24	136/84	-	↓	↓	+	+
6.	27762	57	M	A	5	+	+	+	+	+	25	-	-	-	88	37.6	20	120/82	-	N	↓	+	+
7.	28526	71	M	B	13	+	+	+	+	-	45	-	-	-	110	38.6	30	140/82	-	↓	↓	+	+
8.	28762	53	F	Hw	7	+	+	+	-	-	-	-	-	-	104	38.2	28	124/80	-	N	N	+	+
9.	29486	74	M	A	11	+	+	+	+	-	38	-	-	-	79	37.2	20	136/74	+	↓	↓	+	+
10.	30761	67	M	A	11	+	+	+	+	-	45	+	-	-	87	37.6	22	134/80	+	↓	↓	+	+
11.	30426	49	M	A	2	+	+	+	-	-	25	-	-	-	90	37.8	24	136/84	-	N	N	-	-
12.	30769	76	M	A	10	+	+	+	+	+	45	-	-	-	94	37.4	28	140/84	+	↓	↓	-	-
13.	31264	58	M	A	6	+	+	+	+	-	30	-	-	-	79	37.0	21	140/86	+	N	N	+	-
14.	31424	64	M	A	8	+	+	+	+	-	45	-	-	-	84	37.6	20	132/82	-	↓	↓	+	-
15.	31764	46	F	Hw	5	+	+	-	-	-	-	+	-	-	72	37.6	19	124/76	-	N	↓	-	-
16.	32326	57	M	A	5	+	+	+	-	-	30	-	-	-	82	38	18	128/82	-	N	N	+	-
17.	32124	72	M	A	14	+	+	+	+	-	34	-	-	-	112	38.4	26	140/76	-	↓	↓	-	-
18.	34264	63	M	B	13	+	+	+	+	+	35	-	-	-	84	37.6	20	136/72	+	↓	↓	+	+
19.	34126	57	M	A	5	+	+	+	+	+	30	-	-	-	94	37.8	24	126/78	+	↓	↓	+	+
20.	35762	56	F	Hw	11	+	+	+	+	+	-	-	-	-	106	38.0	28	130/82	-	N	N	-	-
21.	36414	48	M	A	3	+	+	-	-	+	25	-	-	-	78	37.1	22	126/76	-	N	N	+	-
22.	37345	68	M	B	13	+	+	+	+	-	45	-	-	-	112	38.4	30	134/72	-	↓	↓	+	+
23.	37823	84	F	Hw	14	+	+	+	+	+	-	+	-	-	102	37.8	23	140/894	-	N	↓	+	+
24.	38764	47	M	A	4	+	+	-	-	+	20	-	-	-	78	37.6	18	126/74	-	N	N	-	-
25.	45328	79	M	A	12	+	+	+	+	-	55	-	-	-	94	37.6	24	136/74	-	↓	↓	+	+

Sl. No.	Routine Investigation			Chest x-ray			Spirometry						Oxygen saturation	Haematocrit
	Hb in gm%	Red cell count million /cmm	ESR mm/hr	B/E	↑Broncho vascular markings	Hyper translucency	FEV_1 predicted in ltrs	FEV_1 test in ltrs	FEV_1%	FVC Predicted in Ltrs	FVC test in ltrs	FEV_1 / FVC in %	SaO2%	%
1.	15	5.4	11	B+E	-	+	2.36	0.66	28	2.98	1.03	64	88%	45%
2.	13	4.5	12	B	+	-	2.24	1.09	49	2.84	2.01	54	91%	40%
3.	11	4.2	04	B+E	+	+	2.60	1.09	42	3.19	2.27	48	91%	37%
4.	15	5.1	06	B+E	+	+	2.54	1.98	78	3.14	2.71	73	96%	46%
5.	12	4.7	13	B+E	+	+	2.74	2.10	66	3.36	2.58	76	96%	38%
6.	14	4.3	14	N	-	-	2.48	1.83	74	3.09	2.69	72	95%	42%
7.	8	2.5	08	B	+	-	2.40	0.69	29	2.98	1.18	68	89%	32%
8.	11	4.1	15	N	-	-	2.14	1.07	50	2.68	2.43	44	91%	37%
9.	14	4.2	12	B+E	+	+	2.74	1.28	47	3.36	2.78	46	79%	42%
10.	14	4.1	06	B+E	-	+	2.12	0.97	46	2.67	1.86	52	89%	42%
11.	9	3.8	39	N	-	-	2.54	1.98	78	3.14	2.71	73	94%	33%
12.	14	4.3	18	B+E	+	+	2.42	1.21	50	3.02	1.95	62	97%	42%
13.	14.5	4.3	21	B+E	-	+	2.36	0.82	35	2.94	1.17	70	88%	43%
14.	14	4.9	04	B+E	-	+	2.40	0.75	32	2.98	.50	46	80%	49%
15.	8	2.9	40	N	-	-	2.60	1.76	68	3.18	2.75	64	93%	32%
16.	13	4.3	09	N	-	-	2.32	1.55	67	2.89	2.54	61	91%	40%
17.	12	4.2	08	B	+	-	2.42	0.72	30	3.03	1.75	41	89%	38%
18.	13	4.8	17	B+E	+	+	2.10	1.01	48.5	2.64	1.42	71	91%	40%
19.	11.6	4.0	11	B+E	+	+	2.64	1.24	47	3.19	2.69	46	90%	39%
20.	14	4.8	12	N	-	-	2.32	1.16	50	2.92	2.23	52	90%	42%
21.	12	4.03	06	N	-	-	2.48	1.83	74	8.09	2.69	68	90%	38%
22.	15	4.9	08	B	+	+	2.10	0.60	29	2.63	1.30	46	81%	45%
23.	8	2.3	35	B	+	-	2.60	0.78	30	3.22	1.62	48	94%	32%
24.	13	4.5	17	N	-	-	2.36	1.51	64	2.91	2.43	62	94%	40%

S.No.	I.P. No.	Age (in yrs)	Sex	Occupation	Duration of illness	History						Physical Examination							Respiratory System				
						Cough	Expectoration	Dyspnoea	Wheezing	Fever	H/o smoking (pack years)	Pallor	Cyanosis	Clubbing	Pulse rate / min	Temperature in ^{o}C	Respiratory rate / min	Blood Pressure mmHg	Barrel shaped Chest	Chest movement	Breath sounds	Rhonchi	Crepitations
26.	46421	61	M	A	6	+	+	+	+	+	30	-	-	-	96	37.8	28	132/80	+	↓	↓	+	+
27.	47126	68	M	A	9	+	+	+	+	+	45	-	+	-	84	37.8	20	132/76	-	↓	↓	+	+
28.	47742	92	M	A	18	+	+	+	+	-	55	-	-	-	88	37.6	20	140/80	-	↓	↓	+	+
29.	48216	64	F	Hw	12	+	+	+	+	-	-	-	-	-	92	37.8	24	145/70	-	N	N	+	+
30.	48864	73	M	A	12	+	+	+	+	+	45	-	+	-	112	38.7	26	126/72	+	↓	↓	+	+
31.	49312	67	M	A	7	+	+	+	+	-	46	-	-	-	86	37.8	21	134/72	-	↓	↓	+	+
32.	49762	77	M	B	16	+	+	+	+	-	50	-	-	-	94	38.0	26	142/80	-	↓	↓	+	+
33.	50446	72	F	Hw	15	+	+	+	+	-	-	-	-	-	106	39.0	25	136/72	+	N	N	+	+
34.	50876	65	M	A	8	+	+	+	+	-	35	-	-	-	79	37.6	20	126/78	+	↓	↓	+	+
35.	51210	54	M	A	5	+	+	+	-	+	30	-	-	-	75	37.2	20	120/74	-	↓	↓	+	+
36.	52874	64	F	B	7	+	+	+	+	-	20	-	-	-	84	37.8	20	176/72	+	↓	↓	-	-
37.	52976	61	M	Hw	12	+	+	+	+	-	-	-	-	-	86	37.4	21	126/80	-	N	N	-	-
38.	53216	75	M	A	14	+	+	+	+	-	55	-	+	-	92	37.6	24	176/70	-	↓	↓	+	-
39.	33614	68	M	A	10	+	+	+	+	+	45	-	-	-	98	37.2	24	127/72	-	↓	↓	+	-
40.	57364	64	M	A	7	+	+	+	+	-	35	+	-	-	104	38.2	26	132/76	-	↓	↓	-	-
41.	53942	71	M	A	13	+	+	+	+	-	48	-	-	-	82	39.0	22	145/80	-	↓	↓	+	-
42.	54314	65	M	A	6	+	+	+	+	-	40	-	-	-	89	37.0	22	126/70	+	↓	↓	-	-
43.	54411	77	M	A	14	+	+	+	+	-	50	-	+	-	96	37.0	28	128/72	-	↓	↓	+	+
44.	54424	65	F	Hw	12	+	+	+	+	-	-	-	-	-	94	37.4	26	134/76	-	N	N	+	+
45.	54461	68	M	A	9	+	+	+	+	+	42	-	-	-	78	37.2	20	130/82	+	↓	↓	-	-
46.	54824	59	M	A	5	+	+	+	-	-	30	-	-	-	68	37.4	18	134/86	-	N	N	+	-
47.	54562	67	M	A	9	+	+	+	+	-	42	-	-	-	92	37.6	24	136/72	+	↓	↓	+	+
48.	54576	71	M	A	13	+	+	+	+	-	45	+	-	-	81	37.8	20	140/80	-	↓	↓	+	+
49.	54632	58	M	A	4	+	+	+	-	-	30	-	-	-	74	37.2	19	120/76	-	N	N	-	-
50.	54647	62	M	A	5	+	+	+	+	-	25	-	+	-	92	37.6	26	120/80	-	↓	↓	+	+

Sl. No.	Routine Investigation			Chest x-ray			Spirometry						Oxygen saturation	Haematocrit
	Hb in gm%	Red cell count million/cmm	ESR mm/hr	B/E	↑Broncho vascular markings	Hyper translucency	FEV_1 predicted in ltrs	FEV_1 test in ltrs	FEV_1%	FVC Predicted in Ltrs	FVC test in ltrs	FEV_1 / FVC in %	SaO2%	%
25.	14	5.1	16	B+E	+	+	2.16	O.55	26	2.63	1.12	42	89	42
26.	13	4.3	50	N	-	-	2.14	1.24	58	2.60	2.00	62	93	40
27.	18.2	6.4	14	B+E	+	+	2.77	O.72	26	3.36	2.38	52	88	56
28.	15.5	5.4	17	B	+	-	2.60	0.78	28	2.82	1.24	49	89	47
29.	9.5	4.01	08	B	+	-	2.64	0.76	29	3.25	1.26	60	94	33
30.	11.5	4.5	08	E	-	+	2.48	1.65	66	3.08	1.92	62	96	37
31.	14	4.9	16	B+E	-	+	2.46	1.20	49	3.06	2.22	54	92	42
32.	15.5	5.1	17	B	+	-	2.36	O.63	27	2.95	1.28	49	91	46
33.	11.5	4.2	14	E	-	+	2.72	1.03	38	3.27	1.47	70	92	38
34.	14.5	5.1	12	B+E	+	+	2.64	1.28	38.5	3.17	3.76	34	91	44
35.	11	4.02	13	N	-	-	2.15	0.32	15	2.61	0.67	47	92	36
36.	14.2	4.9	14	B+E	+	+	2.74	1.05	60	3.36	2.85	71	95	43
37.	12.4	4.3	12	E	-	+	2.54	0.76	30	3.08	1.40	54	94	39
38.	18.5	6.5	11	B	+	-	2.50	0.67	27	3.03	1.09	61	79	57
39.	14	4.5	08	E	-	+	2.36	1.06	45	2.94	2.16	49	94	42
40.	9	3.9	11	E	-	+	2.54	0.93	37	3.15	1.78	52	90	33
41.	14.2	4.8	12	E	-	+	2.36	1.06	45	2.94	2.16	49	80	43
42.	15	5.2	14	B+E	+	+	2.42	1.18	49	3.02	1.90	62	92	45
43.	18.5	6.6	09	B	+	-	2.62	0.61	26	2.93	1.29	37	86	57
44.	9	4.01	42	B	+	-	2.36	1.14	45	3.13	2.53	45	92	33
45.	11	4.4	12	B+E	+	+	2.46	1.47	60	3.06	2.62	56	91	36
46.	14.5	4.9	13	N	-	-	2.46	1.42	61	2.96	2.52	56	92	44
47.	14.5	4.8	14	B+E	+	+	2.46	1.05	28	3.10	1.51	47	89	44
48.	8	3.8	15	B+E	+	+	2.54	0.71	28	3.10	1.51	47	90	32
49.	14	4.9	16	N	-	-	2.66	1.91	72	3.19	2.80	68	92	42
50.	15.5	5.2	26	B+E	+	+	2.22	0.87	39.2	2.79	1.72	66.5	90	47